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Chronic wasting disease detection and mortality sources in semi-protected deer population

Krysten L. Schuler, Jonathan A. Jenks, Robert W. Klaver, Christopher S. Jennelle and R. Terry Bowyer

K. L. Schuler (ks833@cornell.edu), J. A. Jenks, Dept of Wildlife and Fisheries Sciences, South Dakota State Univ., Brookings, SD 57007, USA. – R. W. Klaver, US Geological Survey, Iowa Cooperative Fish and Wildlife Unit, Iowa State Univ., Ames, IA, USA. – C. S. Jennelle, Minnesota Dept of Natural Resources, Forests Lake, MN, USA. – R. T. Bowyer, Dept of Biological Sciences, Idaho State Univ., Pocatello, ID, USA.

Surveillance for wildlife diseases is essential for assessing population dynamics of ungulates, especially in free-ranging populations where infected animals are difficult to sample. Chronic wasting disease (CWD) is an emerging infectious disease of concern because of the potential for substantial negative effects on populations of cervids. Variability in the likelihood that CWD is detected could invalidate traditional estimators for prevalence. In some instances, deer located after death cannot be tested for infectious diseases, including CWD, because of lack of availability or condition of appropriate tissues. We used various methods to detect infectious diseases that could cause mortality for deer *Odocoileus* spp. residing in Wind Cave National Park, South Dakota, USA, and we report survival estimates for animals in this population. We included 34 monthly encounters of deer resightings and 67 mortalities. We tested live deer by tonsillar biopsy for CWD and estimated pooled prevalence (mean ± SE) at 5.6 ± 3.0% over the three-year study. Live deer potentially had exposure to several infectious diseases, including bluetongue, epizootic hemorrhagic disease, bovine viral diarrhea, West Nile virus, and malignant catarrhal fever, but no apparent morbidity or mortality from those diseases. We tested survival and influence of covariates, including age and sex, using known-fate analysis in Program MARK. Those data best supported a model with time-invariant encounter probability and an annual survival of 72.8%. Even without direct pressure from hunting within the park, average life expectancy in this population was 3.2 years. Only 68% of mortalities contained sufficient material for CWD sampling (because of predation and scavenger activity) and >42% of these were CWD-positive. These findings underscore the possible biases in postmortem surveillance estimates of disease prevalence because of potential for subclinical infected animals to be removed by predators and not tested.

Assessing effects of disease on free-ranging wildlife is a universally challenging dilemma for wildlife professionals, particularly for emerging infectious diseases in which detection is problematic. Disease status of animals can be difficult to assess when animals are not captured (Brownie et al. 1993, Lebreton and Pradel 2002) or animals are encountered, but the disease state is not observable (Kendal 2004, Conn and Cooch 2009). Most studies of wildlife disease rely on human-derived (e.g. hunter harvest, vehicle-strike) postmortem samples to estimate prevalence. Aside from disease-induced mortality, behavioral changes associated with infection may predispose animals to various causes of mortality (Conner et al. 2000, Krumm et al. 2005). The increased susceptibility of infected animals to non-disease induced mortality can decrease life expectancy leading to population-level effects. For example, infection with chronic wasting disease (CWD), a fatal neurodegenerative prion disease of North American *Cervidae* (Williams et al. 2002), can influence behavior and may exacerbate the likelihood an animal dies from predation (Miller et al. 2008, Edmunds et al. 2018). Like many transmissible spongiform encephalopathies, the duration of CWD infection for deer *Odocoileus* spp. can last up to two years (Williams et al. 2002) during which time they continue to shed prions in their saliva, urine and feces (John et al. 2013, Plummer et al. 2017). Transmission can occur from deer-to-deer or from prions that remain viable in the environment (Mathiason et al. 2009, Almberg et al. 2011). Chronic wasting disease has become endemic in several states and provinces with disease-associated declines in survival in some high-prevalence populations of deer and North American elk *Cervus elaphus* (Miller et al. 2008, Monello et al. 2014, Edmunds et al. 2016, Devivo et al. 2017).

Inconsistent recovery of tissue samples often is overlooked because surveillance methods frequently focus on large numbers of samples from human-derived activities,

such as hunter-harvest or directed collections (Diefenbach et al. 2004, Heisey et al. 2014). Indeed, surveillance and monitoring for CWD in wild cervids largely depends on hunter-harvested animals for postmortem testing (Rees et al. 2012, Evans et al. 2014). Probability of finding and testing dead animals can be contingent upon the source of mortality; a deer that was killed by a vehicle is more likely to be located and tested than one slain by a mountain lion *Puma concolor* in a remote area (Pierce et al. 2000). Only tested animals contribute to determining prevalence levels, which are used to assess influences of harvest (Conner et al. 2000, Grear et al. 2006), transmission (Miller et al. 2006), and population trends (Miller and Conner 2005); such data could contain inherent biases if failures in disease detection are not evaluated (Diefenbach et al. 2004, Heisey et al. 2014). We used both antemortem and postmortem testing for CWD and other infectious diseases: blue tongue, epizootic hemorrhagic disease, bovine viral diarrhea, West Nile virus, and malignant catarrhal fever in mule deer *O. hemionus* and white-tailed deer *O. virginianus* for disease screening. We assessed sources of cause-specific mortality for disease detection (CWD-infected deer, CWD non-detected deer and non-testable deer). We hypothesized there would be an influence of sex and age class on survival, cause-specific mortality, and exposure to infectious diseases. We predicted that deer populations still would have high survival rates despite mortality from CWD, other infectious diseases, predators, and humans.

Material and methods

Study area

We conducted research on mule deer and white-tailed deer at Wind Cave National Park (114.4 km^2), located in the southern Black Hills of South Dakota, USA (43°35′N, 103°30′W). The park is bordered by private ranches to the east and south, by the Black Hills National Forest to the west, and by Custer State Park to the north. The park is fenced but the height of the fence (1–2.5 m) is variable and does not confine deer (Bauman 1998). Hunting is not permitted within park boundaries, and therefore, the age structure may include older individuals than hunted populations. There are no predator control programs for coyotes *Canis latrans* or mountain lions, which are the primary predators of deer. Deer density within the park was unknown. The first CWD case was documented in a clinically ill female elk in November 2002 and subsequently in a female mule deer that was struck by a vehicle in January 2003. Cases of CWD in deer and elk were detected in Pennington, Custer and Fall River counties in southwest South Dakota during our study period.

Deer capture and monitoring

Between February 2003 and December 2005, we radiocollared 74 deer in the study area. We captured deer with a combination of helicopter net-gunning (Krausman et al. 1985), clover trapping (Clover 1956), and remote drug delivery throughout the park. Before processing began, all deer were anesthetized with an intramuscular or intravenous injection combination of ketamine (1.5–7.5 mg kg^{-1}) or tiletamine/zolazepam (4.4 mg kg^{-1}) mixed with xylazine (0.75–2.2 mg kg^{-1}) or medetomidine (0.1–0.2 mg kg^{-1}) (Kreeger 1999). Deer were fitted with VHF or GPS collars, ear-tags, and passive integrated transponder electrochips. Deer were aged by tooth wear and replacement (Severinghaus 1949). All deer were given subcutaneous prophylactic antibiotics (Baytril, 6–10 ml, off-label usage). We tested deer for CWD using an antemortem tonsillar biopsy (Schuler et al. 2005), a reliable and sensitive method of detecting CWD infections in live deer (Wild et al. 2002, Wolfe et al. 2002). At capture, we collected blood (10 ml) to test for disease exposure to bluetongue (BT), epizootic hemorrhagic disease (EHD), bovine viral diarrhea (BVD) disease I and II, Johne's disease, West Nile virus (WNV), and malignant catarrhal fever (MCF) exposure at the South Dakota State Animal Disease Research and Diagnostic Laboratory, Brookings, SD. After processing, most deer received intramuscular or intravenous injection of yohimbine (0.2 mg kg^{-1} IV) or atipamezole (0.5–1.0 mg kg^{-1} IM) to aid in recovery.

We relocated marked deer by VHF beacon strength and direction until the collared deer was observed; deer were observed one to three times per week. Deer locations were recorded by handheld GPS units in Universal Transverse Mercator (UTM), accurate to the nearest 5 m. Mortalities were recovered as soon as possible, ranging from immediate collection to five days after death. All carcasses were recovered, and a field or laboratory necropsy was performed when carcasses had tissues present; heads were removed for CWD testing. When these causes were obvious, death was attributed to vehicle-collision, mountain lion predation, coyote predation, or hunting outside of the park; otherwise, mortality was categorized as 'unknown'. Field investigation of all mortality sites allowed cause of death to be assigned to mountain lions when carcasses were found cached. Attributing mortality to coyotes was difficult when carcasses may have been scavenged; therefore, we conservatively attributed mortality to coyotes only with considerable justification, such as bite wounds (canine width, location on carcass and presence of hemorrhage), prior witnessed attacks, or observation within two days without obvious maladies (White et al. 1987). Deer that tested positive for CWD by tonsillar biopsy at capture were immediately dispatched by park law-enforcement personnel and retested with tonsil, lymph node and brain tissue as confirmatory tests. All tissues were tested with immunohistochemistry (IHC) for positive prion staining by Colorado State University Veterinary Diagnostic Laboratory, Ft Collins, CO (Miller et al. 2000). Some animals captured had tonsillar biopsy samples that were unsuitable for testing and were not included in prevalence calculations (Schuler et al. 2005); a subset of those deer retested in subsequent years were included in later calculations. Results were categorized as CWD-positive (infected) if IHC staining was apparent, CWD non-detect if no staining was present, and not-testable (unknown status) if there were insufficient or deteriorated tissue samples. We used chi-square and odds ratios to determine which mortality sources were most likely to provide a CWD sample, and if there was a sex-based difference in mortalities, respectively. Testing for CWD was grouped

by year from 1 July to 30 June of the following year, with the exception of the first year (2003), which included 17 February to 30 June.

Survival analysis

We included up to 34 monthly periods with a two column format (one column for if the deer was seen alive during that month: 'live-resighting' and one column for if the deer was found dead: 'dead-recoveries'). Of 74 deer captured, we used histories for 67 deer (91%, four white-tailed deer and 63 mule deer; 19 males and 48 females); seven deer (including four CWD-positive deer by tonsillar biopsy) were censored, because they were killed <4-months after initial capture and therefore did not have sufficient encounter histories. Because deer were radiocollared and their fate was known at every sampling occasion encounter probabilities were fixed at one. First, we analyzed survival (*S*) with known-fate analysis with a staggered-entry design (Pollock et al. 1989) with monthly time steps in Program MARK (White and Burnham 2009). We used a binomial model with three possible states: 1) survived; 2) died during the study; and 3) survived to a point where its fate was last known when GPS or VHF collars were released from the animal – such animals were subsequently censored (Cooch and White 2006). This model assumed independent animals that demonstrated homogeneity in survival with mutually exclusive capture histories (Cooch and White 2006).

Although we did not have many white-tailed deer or deer <1-year-old, we explored variation in survival as a function of age (young deer were considered <1.5-years-old and adult deer were ≥1.5-years-old) and sex. The study population did not migrate or have dispersals >5 km, so migration and dispersal were not included in models (Schuler et al. 2014). For these models, sampling probabilities (*p*) and reporting probabilities (*r*) were 1. To account for potential overdispersion in the data, we used the median c-hat approach (Cooch and White 2014) to estimate a variance inflation factor (c-hat) that was subsequently accounted for in variance estimates. The incorporation of c-hat into Akaike's information criteria (AIC) calculations is a standard practice which produces a quasi-likelihood adjusted (QAIC) value (Burnham and Anderson 2002) with similar interpretation to AIC. Given our relatively small sizes, we incorporated a small sample size adjustment, and evaluated our models using $QAIC_c$. Taking into account model selection uncertainty, we report model-averaged survival estimates by age and sex class, unless otherwise indicated.

Results

Captured deer spanned a wide range of ages from 0.5 to 9.5-years-old with the average around 4-years-old (±2 SD). In 2003, we tested 33 live deer (32 mule deer, one white-tailed deer) and had one CWD-positive (tonsillar biopsy confirmed postmortem) 5.5-year-old female mule deer for a sample prevalence of 3.0% (95% CI = 0.1–16.2%). In 2004, of 26 live deer tested by tonsillar biopsy (25 mule deer, one white-tailed deer), there was only one CWD-positive 4.5-year-old female mule deer detected for a sample prevalence of 4.0% (95% CI = 0.1–20.4%). In 2005, sample prevalence was 16.7% (95% CI = 2.1–48.4%) based on two CWD-positive deer tested by tonsillar biopsy (one 5.5 year-old female mule deer and one 1.5-year-old female white-tailed deer) of 12 deer (11 mule deer, one white-tailed deer) tested by tonsillar biopsy. Over three years of study, the pooled sample prevalence was 5.6% (95% CI = 1.6–13.8%) in all female deer. All four deer testing positive for CWD by antemortem tonsillar biopsy were confirmed by postmortem testing of obex and retropharyngeal lymph nodes by immunohistochemistry. Four additional mortalities tested positive for CWD (all mule deer: 1.5- year-old female, 3.5-year-old female, 4.5-year-old female, and 5.5-year-old male) for a total of eight CWD-positive deer. Two had prion staining in both the medulla oblongata and tonsils or lymph nodes; the remaining six deer had staining in the tonsils and lymph nodes only. We did not have deer die within two months of capture from natural causes or capture myopathy.

Deer mortalities resulted from predation (coyote (n = 5) and mountain lion (n = 5)), vehicle collision (n = 3), hunting outside of Wind Cave National Park (n = 5), lethal removal by law enforcement (n = 4), and unknown causes (n = 6). Testability for CWD (19/28, 68%) and number of CWD-positive (8/19, 42%) deer varied by source of mortality (Table 1). Predation by coyotes and mountain lions was the primary mortality factor and also the least likely to be able to be tested for CWD (50%) because tissues necessary for sampling were no longer present due to consumption or scattering. Unknown causes, the second most common source of mortality, resulted from carcasses that were not testable (50%). Deer mortalities from human-derived sources (vehicle collisions and hunting) were more likely than other sources of mortality to provide a sample for CWD testing ($\chi^2 = 4.47$, $p = 0.03$) than those from predation events. Females were six times more likely than males to be killed by a predator than by a human source (OR = 6.67, $p = 0.078$). There was no apparent age bias by mortality type, but our study population was primarily adult animals. If only mortalities were tested, observed CWD prevalence was 42%. Observed prevalence from human-induced mortalities only was 20%. No carcasses had indications of malnutrition, apparent disease, or heavy internal or external parasite loads. A few deer had positive titers for all serum tests, with the exception of Johne's disease, throughout the three years of study (Table 2); there was no difference between sexes. Exposure to EHD (13%) was most commonly identified, followed by WNV (12%), MCF (11%), BT (11%) and 1 individual identified with antibodies to BVD. None of the

Table 1. Cause-specific mortalities of 28 deer (of 67 collared) at Wind Cave National Park, South Dakota, USA, from February 2003 to December 2005 and chronic wasting disease (CWD) status of testable samples, either CWD-positive (+), CWD Non-detect (–), or Non-testable.

MORTALITY	CWD +	CWD –	Non-testable
Coyote	1	3	1
Mountain lion	0	1	4
Vehicle collision	1	2	0
Hunting outside study area	1	3	1
Lethal removal in study area	3	1	0
Unknown	2	1	3

Table 2. Number of deer tested for bluetongue (BT), epizootic hemorrhagic disease (EHD), bovine viral diarrhea (BVD) I and II, Johne's disease, West Nile virus (WNV), and malignant catarrhal fever (MCF) at Wind Cave National Park, South Dakota, USA, from 2003 to 2005 with number of deer testing antibody positive shown in parentheses.

Year	BT	EHD	BVD I	BVD II	JOHNE'S	WNV	MCF
2003	39 (6)	40 (16)	39 (0)	40 (0)	42 (0)	39 (0)	36 (4)
2004	0	0	22 (0)	22 (0)	22 (0)	24 (5)	22 (2)
2005	14 (0)	14 (1)	14 (1)	14 (1)	14 (0)	14 (4)	14 (2)

carcasses examined had gross lesions consistent with these infectious diseases. There was no association between infectious disease exposure based on positive titers and future mortality or CWD infection.

We constructed five known-fate models to examine differences in survival as a function of sex and age (Table 3). Constant survival was the best supported model with 0.36 of the model weight (w), however, all model structures were within four $QAIC_c$ units indicating little power in the data to detect covariate differences. Kaplan–Meier staggered-entry indicated a nearly constant pattern in survival with a linear trend over 34 months ($y = -0.016x + 20.27$, $r^2 = 0.985$); constant survival was estimated at 0.9739 ± 0.0077 SE per month [$S_{annual} = 0.7284$ (± 0.069 SE)] based on the best supported model. Given the expected value of annual survival, the average life expectancy of deer was 3.2 years with a 95% CI [confidence interval] of (1.91, 6.87 years). The model averaged monthly survival estimates by sex and age were: females <1.5 years ($S = 0.963 \pm 0.022$), females ≥1.5 years ($S = 0.977 \pm 0.008$), males <1.5 years ($S = 0.962 \pm 0.022$), and males ≥1.5 years ($S = 0.975 \pm 0.011$); survival estimates had overlapping 95% confidence intervals.

Discussion

CWD is a long-term threat to cervid populations across North America and is increasing in prevalence and spatial distribution. Based on rapidly escalating prevalence rates in the antemortem testing and high observed prevalence in postmortem testing, CWD likely had been in this area for several years before its discovery in 2002. In fact, a private property that shares a fenceline with the park had CWD-positive captive elk reported in 1997. We had to assume all research deer at the beginning of the study were not CWD-positive (Heisey et al. 2006); deer with positive tonsillar biopsies were dispatched and thus not included, which likely increased reported survival and skewed sources of mortality. Deer that were CWD-positive on tonsillar biopsy were comparable in age range to deer that were CWD-positive on postmortem examination, but tooth wear-and-replacement may not be a reliable age indicator (Storm et al. 2014). Mortality was inevitable for CWD-infected deer, but most infected deer did not exhibit staining for prions in the medulla oblongata, indicating early stages of the disease (Williams et al. 2002, Spraker et al. 2003). Therefore, preclinical deer were being removed from the population (Miller et al. 2008, Sargeant et al. 2011), and predators often consumed tissues necessary for testing before the carcass was recovered. Consequently, predators could contribute to lower apparent prevalence by disproportionately taking infected over healthy deer (Miller at al. 2008, Wild et al. 2011) while confounding testing capabilities. Removal of infected deer prior to reaching clinical stages would reduce prion shedding; however, predators or scavenger may widely distribute prions on the landscape following passage through the gastrointestinal tract (Nichols et al. 2015), and subsequently binding to soil particles (Walter et al. 2011) or being absorbed into plant tissues (Pritzkow et al. 2015).

Table 3. Age and sex models for known-fate analysis using Program MARK for survival (S) estimates of white-tailed deer and mule deer in Wind Cave National Park, South Dakota, USA, 2003 to 2005. We used the median c-hat approach (Cooch and White 2014) to estimate the variance inflation factor c-hat (2.44) used to calculate Quasi Akaike information criterion.

Model	ΔQAICc	QDeviance	K[A]	w[B]
S (.)	0.00	39.56	1	0.36
S (age)	0.24	37.79	2	0.32
S (sex)	1.81	39.36	2	0.15
S (sex + age)	2.23	37.76	3	0.12
S (sex x age)	3.78	37.30	4	0.15

[A] the number of estimated parameters in the model
[B] Quasi-Akaike weight

Unknown mortalities resulting from a disease or a predator, could not be accurately identified either because of the poor condition of the carcass or time from last sighting. Acute deaths from CWD were rare (Williams 2005), but deaths may have occurred from other diseases including BT, EHD, BVD I and II, WNV and MCF. Malignant catarrhal fever and hemorrhagic diseases (EHD and BT) can cause sudden deaths, but no associated signs were observed in live deer or carcasses (MCF: erosion of the muzzle, blindness, bloody diarrhea; EHD and BT: swelling of the head, neck, and tongue; depression; and respiratory distress; Davidson and Nettles 1997, Heuschele and Reid 2001, Howerth et al. 2001).

A concern for disease surveillance is that samples obtained from human activities (e.g., hunting) may produce bias in observed CWD prevalence. Predation was the primary documented source of mortality in deer; both mountain lions and coyotes were implicated as major predators of adult deer (Ballard et al. 2001, Pierce et al. 2004). Female deer were taken by predators disproportionately more than males. Mountain lion predation was a primary source of mortality for elk in this study area as well (Sargeant et al. 2011). Mule deer and white-tailed deer likely have similar behavioral changes throughout the disease progression (Spraker et al. 1997), including loss of fear of humans (Williams and Young 1980) and slight motor deficiencies that may not be apparent (Williams and Young 1980, 1982), but confer an advantage to a predator (Edmunds et al. 2018). Carcasses from hunting and vehicle collisions were more likely to have testable samples; whereas, less than one-half of carcasses were

testable for CWD from mortalities because of predation and unknown causes. This bias in recovery of testable tissues may play a considerable role in determining true prevalence and deserves additional consideration (Diefenbach et al. 2004). Substantial variation in prevalence have been noted by sex and age classes (Jennelle et al. 2014, Samuel and Storm 2016), which could be influenced by differential predation or hunting pressure on particular groups of deer (DeYoung 1989, Pierce et al. 2000, Edmunds et al. 2018).

To illustrate variability in prevalence estimates by source, we provide both antemortem and postmortem CWD results (Schaub and Pradel 2004). Testing live deer may reduce some of the potential biases associated with solely testing deer postmortem. If we had only relied on postmortem testing in this study population, the observed prevalence was 7.5 times higher than prevalence by tonsillar biopsy, however, this result is confounded by age of the animals. Clearly, the deer were older at postmortem testing and had longer time (since antemortem testing) to become infected. Prevalence recorded from hunter-harvest and vehicle-killed deer was 3.5 times higher than by live tonsillar biopsy, although hunter-harvested samples have been reported as unbiased in some instances (Conner et al. 2000, Grear et al. 2006). Prevalence in an endemic area of Wyoming was 23.8% from tonsillar biopsies and 35% in adult deer from mortality testing and last tonsil biopsy for surviving deer, including CWD-associated emaciation (Edmunds et al. 2016). This value was higher than our antemortem prevalence and lower than our postmortem observed prevalence (42%). While it is possible that lower antemortem prevalence was due to missed samples (due to insufficient tonsillar follicles) early in the study (Schuler et al. 2005), we were able to successfully retest 1–2 years later and/or confirm survival for ≥10 months post-biopsy in all but two animals. Previous studies have cautioned that prevalence could be biased if only mortalities from hunter-harvest (Conner et al. 2000) or vehicle collisions (Krumm et al. 2005) are recorded, and mortalities from predators or disease are not documented (Schaub and Pradel 2004). Although antemortem testing is not a cost-effective option for statewide surveillance programs, efforts of wildlife agency toward opportunistic collection and testing of predator-killed animals or clinical suspects could provide an enhanced disease-detection strategy (Walsh and Miller 2010).

Survival was high in the park despite having several potential sources of mortality, both natural (predation) and human-induced (hunting, removals, and vehicle-collisions). Our estimate of annual survival ($S = 0.73$) was higher than others for adjacent hunted populations of WTD in South Dakota ($S = 0.50 – 0.60$, DePerno et al. 2000, Klaver et al. 2008) that are also presumably exposed to similar diseases and predators. There were no substantial differences in survival by age or sex, despite unbalanced sample sizes, which could be due to small sample sizes. Because of the similar overlap in space-use by both species, we were comfortable combining data for mule deer and white-tailed deer, which have previously reported similar survival rates between the two species (Whittaker and Lindzey 2001). Complications resulting from migration or movements associated with seasonal home ranges (Miller and Conner 2005) did not occur in our study population (Schuler et al. 2014).

The average life expectancy of individuals in our study population (3.2 years) was intermediate in the range between CWD-infected (1.6 years) and CWD-uninfected (5.2 years) animals in unhunted, but CWD-endemic populations of mule deer (Miller et al. 2008). For CWD management, Miller et al. (2006) projected long-term annual survival rates of about 0.55 are necessary to maintain populations, excluding disease-induced mortality. Our study site provided an opportunity to evaluate CWD as an emerging infectious disease in an unhunted, at least within the park, population of wild deer compared with other areas where CWD is endemic (Osnas et al. 2009) or may involve different survival challenges. An additional benefit of conducting this study in Wind Cave National Park was having a population with an older age structure. Although adult males are reported to have higher rates of infection in most areas (Grear et al. 2006, Potapov et al. 2013, Jennelle et al. 2014, Samuel and Storm 2016), similar to Edmunds et al. (2016), our females had higher prevalence rates that are more likely to affect population growth. Geremia et al. (2015) reported CWD minimally lowered the population growth rate, but did not cause a rapid decline in deer abundance. Given the higher prevalence in adult females in this study area, which may or may not be related to the lack of hunting pressure, we suggest further work to determine the mechanisms that may drive a higher infection rate in females versus males.

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References

Almberg, E. S. et al. 2011. Modeling routes of chronic wasting disease transmission: environmental prion persistence promotes deer population decline and extinction. – PLoS One 6(5): e19896.

Ballard, W. B. et al. 2001. Deer–predator relationships: a review of recent North American studies with emphasis on mule and black-tailed deer. – Wildl. Soc. Bull. 29: 99–115.

Bauman, P. J. 1998. The Wind Cave National Park elk herd: home ranges, seasonal movements, and alternative control methods. – MS thesis, South Dakota State Univ., Brookings, SD.

Brownie, C. J. et al. 1993. Capture–recapture studies for multiple strata including non-Markovian transitions. – Biometrics 49: 1173–1187.

Burnham, K. P. and Anderson, D. R. 2002. Model selection and multimodel inference: a practical information-theoretic approach. – Springer.
Clover, M. R. 1956. Single-gate deer trap. – Can. Fish Game 42: 199–201.
Conn, P. B. and Cooch, E. G. 2009. Multistate capture–recapture analysis under imperfect state observation: an application to disease models. – J. Appl. Ecol. 46: 486–492.
Conner, M. M. et al. 2000. Detection of bias in harvest-based estimates of chronic wasting disease prevalence in mule deer. – J. Wildl. Dis. 36: 691–699.
Cooch, E. and White, G. C. 2014. Program MARK: a gentle introduction, 14th edn. – <www.phidot.org/software/mark/docs/book/>.
Davidson, W. R. and Nettles, V. F. 1997. Field manual of wildlife diseases in the southeastern United States. Southeastern Cooperative Wildlife Disease Study. – Univ. of Georgia.
DePerno, C. S. et al. 2000. Female survival rates in a declining white-tailed deer population. – Wildl. Soc. Bull. 28: 1030–1037.
DeYoung, C. A. 1989. Mortality of adult male white-tailed deer in south Texas. – J. Wildl. Manage. 53: 513–518.
Diefenbach, D. R. et al. 2004. From the field: efficacy of detecting chronic wasting disease via sampling hunter-killed white-tailed deer. – Wildl. Soc. Bull. 32: 267–272.
Devivo, M. T. et al. 2017. Endemic chronic wasting disease causes mule deer decline in Wyoming. – PLoS One 12(10): e0186512.
Edmunds, D. R. et al. 2016. Chronic wasting disease drives population decline of white-tailed deer. – PLoS One 11: e0161127.
Edmunds, D. R. et al. 2018. Chronic wasting disease influences activity and behavior in white-tailed deer. – J. Wildl. Manage. 82: 138–154.
Evans, T. S. et al. 2014. Surveillance and monitoring of white-tailed deer for chronic wasting disease in the northeastern United States. – J. Fish Wildl. Manage. 5: 387–393.
Gannon, W. L. et al. 2007. Guidelines of the American Society of Mammalogists for the use of wild mammals in research. – J. Mammal. 88: 809–823.
Geremia, C. et al. 2015. Age and repeated biopsy influence PrPCWD in mule deer (*Odocoileus hemionus*) in Colorado. – J. Wildl. Dis. 50: 801–810.
Grear, D. A. et al. 2006. Demographic patterns and harvest vulnerability of chronic wasting disease infected white-tailed deer in Wisconsin. – J. Wildl. Manage. 70: 546–553.
Heisey, D. M. et al. 2006. The fitting of general force-of-infection models to wildlife disease prevalence data. – Ecology 87: 2359–2365.
Heisey, D. M. et al. 2014. Using auxiliary information to improve wildlife disease surveillance when infected animals are not detected: a Bayesian approach. – PLoS One 9: e89842.
Heuschele, W. P. and Reid, H. W. 2001. Malignant catarrhal fever. – In: Williams, E. S. and Barker, I. K. (eds), Infectious diseases of wild mammals. Iowa State Press, pp. 157–164.
Howerth, E. W. et al. 2001. Bluetongue, Epizootic Hemorrhagic Disease, and other orbivirus-related diseases. – In: Williams, E. S. and Barker, I. K. (eds), Infectious diseases of wild mammals. Iowa State Press, pp. 77–97.
Jennelle, C. S. et al. 2014. Transmission of chronic wasting disease in Wisconsin white-tailed deer: implications for disease spread and management. – PLoS One 9: e91043.
John, T. R. et al. 2013. Early detection of chronic wasting disease prions in urine of pre-symptomatic deer by real-time quaking-induced conversion assay. – Prion 7: 253–258
Klaver, R. W. et al. 2008. Associating seasonal range characteristics with survival of female white-tailed deer. – J. Wildl. Manage. 72: 343–353.
Kendall, W. L. 2004. Coping with unobservable and mis-classified state in capture–recapture studies. – Anim. Biodivers. Conserv. 27: 97–103.
Krausman, P. R. et al. 1985. Capturing deer and mountain sheep with a net-gun. – Wildl. Soc. Bull. 13: 71–73.
Kreeger, T. J. 1999. Handbook of wildlife chemical immobilization. – Wildlife Pharmaceuticals Inc., Fort Collins, CO.
Krumm, C. E. et al. 2005. Relative vulnerability of chronic wasting disease infected mule deer to vehicle collisions. – J. Wildl. Dis. 41: 503–511.
Lebreton, J. D. and Pradel, R. 2002. Multistate recapture models: modeling incomplete individual histories. – J. Appl. Stat. 29: 353–369.
Mathiason, C. K. et al. 2009. Infectious prions in preclinical deer and transmission of chronic wasting disease solely by environmental exposure. – PLoS One 4: e5916.
Miller, M. W. and Conner, M. M. 2005. Epidemiology of chronic wasting disease in free-ranging mule deer: spatial, temporal, and demographic influences on observed prevalence patterns. – J. Wildl. Dis. 41: 275–290.
Miller, M. W. et al. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. – J. Wildl. Dis. 34: 532–538.
Miller, M. W. et al. 2006. Dynamics of prion disease transmission in mule deer. – Ecol. Appl. 16: 2208–2214.
Miller, M. W. et al. 2008. Lions and prions and deer demise. – PLoS One 3(12): e4019.
Monello, R. J. et al. 2014. Survival and population growth of a free-ranging elk population with a long history of exposure to chronic wasting disease. – J. Wildl. Manage. 79: 214–223.
Nichols, T. A. et al. 2015. CWD prions remain infectious after passage through the digestive system of coyotes (*Canis latrans*). – Prion 9: 367–375
Osnas, E. E. et al. 2009. Spatial and temporal patterns of chronic wasting disease: fine-scale mapping of a wildlife epidemic in Wisconsin. – Ecol. Appl. 19: 1311–1322.
Pierce, B. W. et al. 2000. Selection of mule deer by mountain lions and coyotes: effects of hunting style, body size and reproductive status. – J. Mammal. 81: 462–472.
Pierce, B. M. et al. 2004. Habitat selection by mule deer: forage benefits or risk of predation. – J. Wildl. Manage. 68: 533–541.
Plummer, I. H. et al. 2017. Temporal patterns of chronic wasting disease prion excretion in three cervid species. – J. Gen. Virol. 98: 1932–1942.
Pollock, K. H. et al. 1989. Survival analysis in telemetry studies: the staggered entry design. – J. Wildl. Manage. 53: 7–15.
Potapov, A. et al. 2013. Chronic wasting disease: possible transmission mechanisms in deer. – Ecol. Model. 250: 244–257.
Pritzkow, S. et al. 2015. Grass plants bind, retain, uptake, and transport infectious prions. – Cell Rep. 11: 1168–1175.
Rees, E. E. et al. 2012. Targeting the detection of chronic wasting disease using the hunter harvest during early phases of an outbreak in Saskatchewan, Canada. – Prev. Vet. Med. 104: 149–159.
Samuel, M. D. and Storm, D. J. 2016. Chronic wasting disease in white-tailed deer: infection, mortality and implications for heterogeneous transmission. – Ecology 97: 3195–3205.
Sargeant, G. A. et al. 2011. Implications of chronic wasting disease, cougar predation, and reduced recruitment for elk management. – J. Wildl. Manage. 75: 171–177.
Schaub, M. and Pradel, R. 2004. Assessing the relative importance of different sources of mortality from recoveries of marked animals. – Ecology 85: 930–938.
Schuler, K. L. et al. 2005. Tonsillar biopsy test for chronic wasting disease: two sampling approaches in mule deer and white-tailed deer. – J. Wildl. Dis. 41: 820–824.

Schuler, K. L. et al. 2014. Ad-hoc smoothing parameter performance in kernel estimates of GPS-derived home ranges. – Wildl. Biol. 20: 259–266.
Severinghaus, C. W. 1949. Tooth development and wear as criteria of age in white-tailed deer. – J. Wildl. Manage. 13: 195–216.
Spraker, T. R. et al. 1997. Spongiform encephalopathy in free-ranging mule deer (*Odocoileus hemionus)*, white-tailed deer (*Odocoileus virginianus*) and Rocky Mountain elk (*Cervus elaphus nelsoni*) in northcentral Colorado. – J. Wildl. Dis. 33: 1–6.
Spraker, T. R. et al. 2004. Variable patterns of distribution of PrP(CWD) in the obex and cranial lymphoid tissues of Rocky Mountain elk (*Cervus elaphus nelsoni*) with subclinical chronic wasting disease. – Vet. Rec. 155: 295–302.
Storm, D. J. et al. 2014. Estimating ages of white-tailed deer: age and sex patterns of error using tooth wear-and-replacement. – Wildl. Soc. Bull. 39: 849–856.
Walsh, D. J. and Miller, M. W. 2010. A weighted surveillance approach for detecting chronic wasting disease. – J. Wildl. Dis. 46: 118–135.
Walter, W. D. et al. 2011. Soil clay content underlies prion infection odds. – Nat. Comm. 2: 200–206.
White, G. C. and Burnham, K. P. 2009. Program MARK: survival estimation from populations of marked animals. – Bird Study. 46: S120–S139.
White, G. C. et al. 1987. Survival of mule deer in northwest Colorado. – J. Wildl. Manage. 51: 852–859.
Whittaker, D. G. and Lindzey, F. G. 2001. Population characteristics of sympatric mule and white-tailed deer on Rocky Mountain Arsenal, Colorado. – J. Wildl. Manage. 65: 946–952.
Wild, M. A. et al. 2002. Preclinical diagnosis of chronic wasting disease in captive mule deer (*Odocoileus hemionus*) and white-tailed deer (*Odocoileus virginianus*) using tonsillar biopsy. – J. Gen. Virol. 83: 2629–2634.
Wild, M. A. et al. 2011. The role of predation in disease control: a comparison of selective and nonselective removal of prion disease dynamics in deer. – J. Wildl. Dis. 47: 78–93.
Williams, E. S. 2005. Chronic wasting disease. – Vet. Path. 42: 530–549.
Williams, E. S. and Young, S. 1980. Chronic wasting disease of captive mule deer: a spongiform encephalopathy. – J. Wildl. Dis. 16: 89–98.
Williams, E. S. and Young, S. 1982. Spongiform encephalopathy of Rocky Mountain elk. – J. Wildl. Dis. 18: 465–471.
Williams, E. S. et al. 2002. Chronic wasting disease of deer and elk: a review with recommendations for management. – J. Wildl. Manage. 66: 551–563.
Wolfe, L. L. et al. 2002. Evaluation of antemortem sampling to estimate chronic wasting disease prevalence in free-ranging mule deer. – J. Wildl. Manage. 66: 564–573.