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EVALUATION OF A TEST AND CULL STRATEGY FOR REDUCING PREVALENCE OF CHRONIC WASTING DISEASE IN MULE DEER (*ODOCOILEUS HEMIONUS*)

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ABSTRACT: We evaluated a test and cull strategy for lowering chronic wasting disease (CWD) prevalence in a naturally-infected, free-ranging mule deer (*Odocoileus hemionus*) herd wintering in the town of Estes Park, Colorado, US and in nearby Rocky Mountain National Park. We tested 48–68% of the estimated number of adult (≥1 yr old) deer annually for 5 yr via tonsil biopsy immunohistochemistry (IHC), collecting 1,251 samples from >700 individuals and removing IHC-positive deer. Among males, CWD prevalence during the last 3 yr of selective culling was lower (one-sided Fisher's exact test $P=0.014$) than in the period prior. In contrast, CWD prevalence among females before culling and after culling were equivalent ($P=0.777$). Relatively higher annual testing of males (mean 77%) compared to females (mean 51%) might have contributed to differences seen in responses to management. A more intensive and sustained effort or modified spatial approach might have reduced prevalence more consistently in both sexes. Limitations of this technique in wider management application include cost and labor as well as property access and animal tolerance to repeated capture. However, elements of this approach could potentially be used to augment harvest-based disease management.

Key words: Chronic wasting disease, cull, management, mule deer, *Odocoileus hemionus*, prion.

INTRODUCTION

Chronic wasting disease (CWD; Williams and Young 1980) is an infectious transmissible spongiform encephalopathy of cervid species including North American mule deer (*Odocoileus hemionus*). Areas of relatively high CWD prevalence in mule deer along Colorado's northern Front Range were among the earliest described (Miller et al. 2000). The need for effective strategies to lower CWD prevalence in this area and elsewhere has been recognized for some time (Wolfe et al. 2002, 2004b; Conner et al. 2007; Uehlinger et al. 2016). Unfortunately, progress on understanding how to control CWD has been slow and few empirical data have been reported (Uehlinger et al. 2016; WAFWA 2017).

In the absence of a vaccine or medication to prevent or cure infection with the CWD agent, lethal removal of infected animals or groups are the mainstays of recommended strategies for controlling this disease (WAFWA 2017). Both aggressive sport harvest and professional culling have been used in attempts to lower infection rates or eliminate emergent foci of infection with variable reported success (Conner et al. 2007; Pybus 2012; Mateus-Pinilla et al. 2013). Although professional culling and sometimes sport harvest can be spatially focused on case clusters, or on areas or demographics of relatively high prevalence or risk (Conner et al. 2007; Pybus 2012; Mateus-Pinilla et al. 2013), removals under such programs tend to be random in that both infected and uninfected individuals are killed indiscriminately. Nonetheless, theoretical models and some field data suggest that nonselective culling or harvest of sufficient intensity might be effective in suppressing CWD prevalence among mule deer (Gross and Miller 2001; Wild et al. 2011; Potapov et al. 2016).

In theory, culling that preferentially removes infected animals (selective culling)

should reduce CWD prevalence even more effectively than random culling, provided that infected deer are detected early in the disease course and a substantial proportion of the population is screened annually (Gross and Miller 2001; Wild et al. 2011). More specifically, an early model suggested that CWD prevalence could be reduced by 50% over a 5 yr period via selective culling using a 50% annual testing regimen (Gross and Miller 2001). To that end, we evaluated a test and cull strategy (Wolfe et al. 2004b) for suppressing CWD in a naturally-infected, free-ranging mule deer herd wintering in the town of Estes Park and in Rocky Mountain National Park, Colorado, US. We demonstrated the feasibility of our approach to selective culling in the urban portions of our study area during December 2002–May 2003 (Wolfe et al. 2004b). Here we report the overall outcomes of our 5-yr effort to suppress CWD in free-ranging mule deer via selective culling.

MATERIALS AND METHODS

Study area

Our 84 km^2 study area (Fig. 1) included mule deer winter ranges located in the town of Estes Park (40°23′N, 105°30′W) and in the east side of Rocky Mountain National Park (40°20′N, 105°42′W), located in the western portion of Colorado Game Management Unit 20, which also delimits the approximate range of the Big Thompson deer population (Data Analysis Unit D-10; Conner and Miller 2004; Miller and Conner 2005). During our study, the estimated size of the Big Thompson population averaged about 7,000 individuals. The mule deer herd that wintered in Estes Park and eastern Rocky Mountain National Park represented about 5% of the population total. Hunting occurred each autumn throughout Game Management Unit 20 outside national park boundaries, including some portions of our study area.

Mule deer ranges in our study area encompassed residential and commercial areas developed within native habitats as well as relatively undeveloped wildland settings (Fig. 1). Local native habitats ranged from dense stands of mountain mahogany (*Cercocarpus montanus*) interspersed with grassland openings and small timbered patches of ponderosa pine (*Pinus ponderosa*) to mountain shrub habitat with a ponderosa pine and Douglas-fir (*Pseudotsuga menziesii*) overstory. Elevations ranged from 2,300 to 2,750 m. Varying degrees of human development and habitation occurred in all habitat types outside the park boundaries. Chronic wasting disease occurred in this area for at least two decades before our study began (Spraker et al. 1997).

Test and cull

Field methods generally followed those described previously (Wolfe et al. 2004b). To evaluate a 50% testing level, we tried to sample ≥55% of the estimated number of adult deer of each sex annually, anticipating that about 10% of the biopsies would be deemed unsuitable for lack of at least one discernible lymphoid follicle (Wolfe et al. 2002, 2004b). Target sample sizes were estimated based on annual counts conducted in December (Table 1). We ran two CWD testing campaigns each field season, using deer tested and marked during autumn (September–December) in the annual mark-resight inventory and then sampling the balance needed for annual targets during spring (February–May).

We estimated abundance of deer within study area bounds via mark-resight counts conducted in December of each year (Bowden and Kufeld 1995; White 1996). Counts were timed such that deer marked during autumn were still wearing collars or antler transmitters with unique visible identifiers for the annual inventory.

For testing, deer were chemically immobilized via darting on foot or from a vehicle. Drug combinations varied over time and included 250 mg tiletamine HCl and zolazepam (Telazol®, Fort Dodge, Overland Park, Kansas, USA) and 200 mg xylazine HCl, 10 mg thiafentanil oxalate (A3080, Wildlife Pharmaceuticals, Inc., Fort Collins, Colorado, USA) and 100 mg xylazine HCl (Wolfe et al. 2004a), 14 mg medetomidine and 260 mg ketamine (Wildlife Pharmaceuticals), or 30 mg butorphanol, 25 mg azaperone, and 10 medetomidine (Wildlife Pharmaceuticals). We delivered drugs intramuscularly via darts (Pneu-Dart, Williamsport, Pennsylvania, USA) fired from an adjustable, air-powered rifle (Dan-Inject rifle, Dan-Inject of North America, Fort Collins, Colorado, USA).

We collected samples (tonsil biopsy and blood) and marked each deer with a telemetry device to facilitate tracking down infected individuals (Wolfe et al. 2004b). Most deer received radio collars, but some males caught in autumn were fitted with transmitters attached to an antler or ear tag. Collars were programmed to drop within 6–8 wk for deer captured outside the park, but deer were still identifiable by uniquely numbered plastic tags placed in each ear. Tags were color coded by year of capture and could be seen from

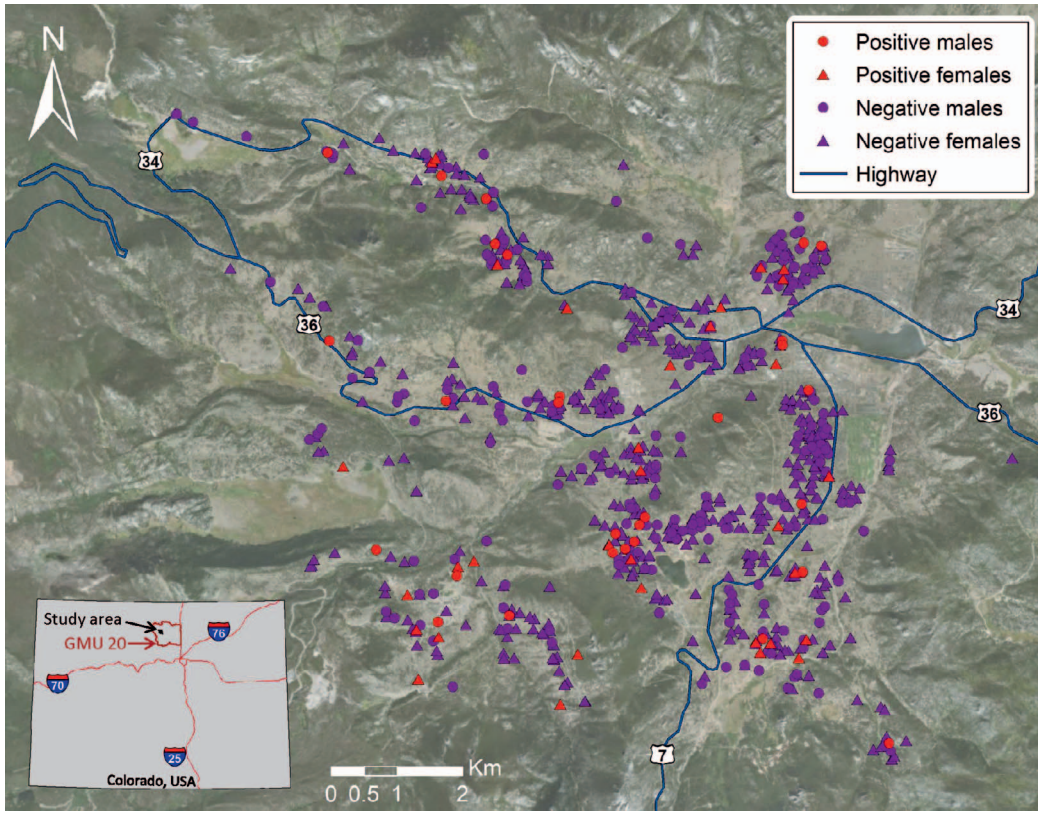

FIGURE 1. Capture locations of male (circles) and female (triangles) mule deer (*Odocoileus hemionus*) tested for chronic wasting disease infection in the Estes Park and Rocky Mountain National Park, Colorado, USA, study area during 2001–07. Our 84 km^2 study area was located in the western portion of Colorado Game Management Unit (GMU) 20, which also delimits the approximate range of the "Big Thompson" deer population (inset). Mule deer winter ranges were located in the town of Estes Park and in the east side of Rocky Mountain National Park. Chronic wasting disease infection status (red=positive; purple=negative) was determined from tonsil biopsy immunohistochemistry. Highways (lines with chevrons) shown for spatial reference.

afar. Recaptured deer received tags colored for the current year of capture but retained their unique identifying number. When encountering a group of deer, we prioritized capturing unmarked individuals or those sampled further in the past based on tag color. We tried to avoid animals 1.5 (±0.5) yr old ("yearlings") because infection rates at that age tend to be low (Miller et al. 2000; Miller and Conner 2005), but yearlings became difficult to distinguish from older individuals by spring. Multiple deer were captured from the same social group on the same day on numerous occasions, particularly in residential areas. We used telemetry to locate test-positive animals and culled them via chemical immobilization as described and intravenous lethal injection (potassium chloride), or via gunshot to the head with a high-caliber rifle.

CWD diagnostics

Tonsil biopsy immunohistochemistry (IHC) was used to detect preclinical CWD in mule deer (Wolfe et al. 2002, 2004b). We used 6-mm, 30-cm biopsy forceps (Sontec Instruments, Centennial, Colorado, USA) and biopsy procedures described by Wolfe et al. (2002, 2004b) to collect tonsil tissue. Lidocaine was applied topically to control pain and Gelfoam® (AmerisourceBergen, Chestbrook, Pennsylvania, USA) was applied to control bleeding. We rotated biopsy instruments and cleaned and soaked mouth gates and other reusable implements in LpH disinfectant (Environ® LpH® Steris Corporation, Saint Louis, Missouri, USA; Race and Raymond 2004) for at least 30 min between uses. In addition to LpH treatment in the field, we sonicated biopsy instruments and other reused implements for 10

TABLE 1. Annual estimated abundance, number of individuals (and percent of estimated total) captured and tested, and chronic wasting disease prevalence among tested female and male mule deer (*Odocoileus hemionus*) wintering in Estes Park and Rocky Mountain National Park, Colorado, USA during 2001–07. Deer abundance (herd size) based on December mark-resighting counts, reported as estimated numbers with 95% confidence limits (CL); observed individuals were classified as female, male, or fawn. Prevalence estimated from tonsil biopsy immunohistochemistry, reported with 95% exact binomial CL (bCL). Active disease management occurred during autumn 2002–spring 2007.[a]

		Females			Males		
	Herd size	No.			No.		
Year	Total (95% CL)	Estimate (95% CL)	Tested (%)	Prevalence (95% bCL)	Estimate (95% CL)	Tested (%)	Prevalence (95% bCL)
Spring 2001	nd	nd	39	0.03 (0.001–0.13)	nd	27	0.07 (0.01–0.24)
2001–02	883 (659–1,058)	389 (363–415)	56 (14)	0 (0–0.06)	168 (136–201)	30 (18)	0.13 (0.04–0.31)
2002–03	619 (554–691)	289 (270–308)	137 (47)[b]	0.04 (0.02–0.09)	125 (101–149)	60 (48)[b]	0.15 (0.07–0.27)
2003–04	565 (501–637)	308 (294–322)	169 (55)[b]	0.05 (0.01–0.08)	103 (91–116)	92 (89)[b]	0.13 (0.07–0.22)
2004–05	597 (533–668)	303 (291–316)	167 (55)[b]	0.04 (0.01–0.08)	90 (74–105)	101 (112)[b]	0.05 (0.02–0.11)
2005–06	499 (442–563)	251 (236–267)	121 (48)[b]	0.06 (0.02–0.12)	103 (86–121)	64 (62)[b]	0.06 (0.02–0.15)
2006–07	572 (500–654)	312 (280–343)	152 (49)[b]	0.05 (0.02–0.09)	116 (97–134)	84 (72)[b]	0.08 (0.03–0.16)

[a] nd = not done.

[b] All test-positive individuals were located and euthanized.

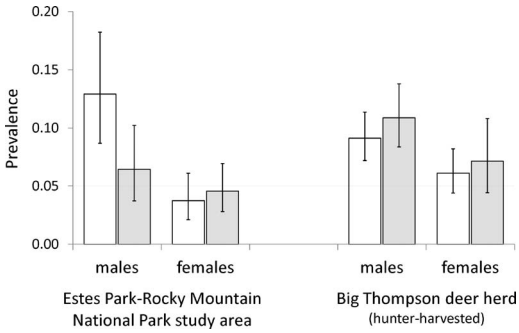

FIGURE 2. Pooled estimates of chronic wasting disease prevalence among tested male and female mule deer (*Odocoileus hemionus*) captured in the Estes Park and Rocky Mountain National Park, Colorado, USA, study area during 2001–07, and from mule deer harvested in the surrounding Big Thompson population unit (Colorado deer Data Analysis Unit D-10) during that same time period. Prevalence for the study area estimated from tonsil biopsy data; prevalence for the surrounding area estimated from hunter-killed deer (see Table S1). Active disease management occurred in the study area during autumn 2002–spring 2007. For analysis, the before culling period (open bars) was defined as spring 2001–spring 2004 (or hunting seasons in autumn 2001–03); the after culling period (shaded bars) was autumn 2004–spring 2007 (or hunting seasons in autumn 2004–06). Capped vertical lines represent upper and lower 95% exact binomial confidence intervals on estimates.

min, manually cleaned biopsy cups, and autoclaved them for 60 min (121 C, 220 kPa) at the end of each day.

Tonsil tissue IHC methods using monoclonal antibody F99/97.6.1 (VMRD Inc., Pullman, Washington, USA) were as described previously (Spraker et al. 2002; Wolfe et al. 2002). Biopsies were evaluated microscopically: those with ≥1 lymphoid follicle were classified as positive (infected) or not detected (negative) based on presence of IHC staining.

Data analysis

We regarded tonsil biopsy samples with ≥1 lymphoid follicle as usable tests. Local data from CWD testing (n=152 individuals) also were available from spring 2001–spring 2002 (Wolfe et al. 2002; Conner and Miller 2004). To assess management effectiveness, we compared pooled estimated prevalence prior to and during the first 2 yr of selective culling ("before;" spring 2001–spring 2004; Tables 1, S1) to prevalence during the last 3 yr of selective culling ("after;" autumn 2004–spring 2007; Tables 1, S1). This breakpoint was based on an assumed 2–3 yr disease course and the first year that recapture and resampling of previously tested deer exceeded 50% (Fig. S1). Proportions are reported with 95% exact binomial confidence limits (bCL). We used one sided Fisher's exact test (α=0.05) to compare prevalence between time periods.

RESULTS

We collected 1,251 biopsies from over 700 individual deer during the autumn 2002–spring 2007 disease management period. Nearly 92% (1,146/1,251) of biopsies were usable, and about 91% (1,041/1,146) of those had ≥5 lymphoid follicles. During the management period we tested an average of 57% of the estimated number of adult deer each year (range 48–68%; Table 1). We culled 34 infected females and 37 infected males during that time, usually within 3–6 wk of sampling and within 2 wk of receiving results; all but two of the culled deer were ≥2 yr old.

Among males, CWD prevalence during the 3-yr after period (0.06) was lower (P=0.014) than during the before period (0.13; Table 1 and Fig. 2). In contrast, prevalence among females during the before and after periods were equivalent (P=0.777; Table 1 and Fig. 2). Estimated prevalence among hunter-harvested, adult male (0.09–0.11) and female (0.06–0.07) mule deer in the surrounding Big Thompson population also remained relatively unchanged during this same time period (Fig. 2 and Table S1).

DISCUSSION

Despite expending considerable field effort and adhering closely to management objectives, we did not uniformly reduce CWD prevalence through selective culling. This might have been in part because we fell short of annual testing targets for females in 3 of the 5 yr (Table 1), largely because deer abundances calculated during the study as the basis for sampling targets were slightly underestimated. The population estimates were corrected post hoc to the values reported in Table 1. Moreover, test sensitivity would have

been lower in the 9% (105/1,146) of biopsies with fewer than five follicles (Geremia et al. 2015a). Repeated sampling and aging of the deer could have contributed to the number of samples with few or no lymphoid follicles (Geremia et al. 2015a). Newer tests (e.g., real time quaking-induced conversion; Hoover et al. 2016) might aid in screening samples unsuitable for IHC.

Relatively higher annual testing of males (mean 77%) compared to females (mean 51%; Table 1) during the management period might have contributed to differences seen in responses. We assumed this was a largely closed deer herd aside from seasonal migration (Conner and Miller 2004). However, recapture rates for males did not exceed 50% until 2006–07 (Fig. S1) and perhaps reflected immigration or underestimated abundance of male deer.

Although we tested ≥48% of adult deer each year throughout the disease management period, the application of testing effort was uneven because there were deer that were undetectable, unapproachable, or inaccessible in some locations. By spring 2007, we had accumulated permissions to access nearly 1,400 separate private property parcels for deer capture and testing in addition to public and municipal lands, but this represented less than 20% (1,387/7,459) of the parcels in the area. Data on individual deer movements to measure home ranges or to identify clear social partitioning were not collected, but anecdotal field observations were consistent with patterns reported for mule deer elsewhere (Cullingham et al. 2011; Mejía-Salazar et al. 2017, 2018).

Continuing the testing and culling over a longer period of time might have been more effective or at least allowed time to detect responses given the likely lag arising from the chronic nature of infection (Geremia et al. 2015b). In light of the observed drop in prevalence among males after annual testing that averaged 77% (vs. 51% for females; Table 1), a more effective approach might have been to sample the entire herd or a vast majority of the herd every other year rather than targeting half of the herd every year. Alternatively, focusing captures on thoroughly sampling about half of the winter range every year, targeting entire social groups, exploiting observed seasonal differences between males and females (below), or following up with more intensive testing of social groups or locations yielding positive deer (Fig. 1) might be worth exploring as variations on our original test and cull strategy (Pybus 2012; WAFWA 2017; Mejía-Salazar et al. 2018). The difference in response between sexes also should serve as a cautionary note that focusing CWD control solely on the male segment of an infected mule deer herd might not suppress infection among females.

While exploring likely spatial variation in our management effort over time, we noticed that females captured in spring unexpectedly tended towards a higher prevalence than did females captured in autumn: during the before culling period, the prevalence among females was 0.05 (95% bCL=0.03–0.09; n=222) in spring and 0.02 (95% bCL=0.003–0.05; n=179) in autumn. This difference seemed likely to be an artifact of the spatial distribution of capture effort or of targeted female deer groups, but illustrated how surveillance based on sampling from females harvested in autumn might underestimate prevalence or distribution in some circumstances. Males showed the opposite pattern: relatively high prevalence in autumn (0.18, 95% bCL=0.11–0.25; n=120) as compared to spring (0.07, 95% bCL=0.03–0.14; n=89). The latter pattern likely reflected older-aged males tending female bands (Mejía-Salazar et al. 2017) and their vulnerability to capture during the autumn breeding season. It follows that disease management directed toward mature males might be most effective in late autumn (Conner et al. 2000), which aligns with traditional sport hunting seasons in most jurisdictions.

Other factors also could have contributed to the disparity between field observations and a priori model projections. The anticipated magnitude of the impact of culling on prevalence seems in retrospect to have been overly optimistic within the time frame we allowed for detecting such a response. The

Gross and Miller (2001) model likely overemphasized the role of infected individuals in CWD transmission—and consequently overestimated the relative impact of their removal—because indirect prion transmission was excluded. But environmental sources of infection apparently can drive CWD epidemics, at least temporarily (Almberg et al. 2011), thereby buffering against the lack of infected animals. Chronic wasting disease became endemic in this area well before disease management began, so it seems plausible that contaminated environments could sustain transmission over several years, despite aggressive removal of infected deer. As suggested by Mejía-Salazar et al. (2018), environmental transmission might have affected female deer more than males in our study area because females tended to spend the majority of autumn and winter within the same home range and about half remained in the same home range all year (Conner and Miller 2004). Some case clusters in our study area (Fig. 1) appeared associated with (illegal) artificial feeding sites that could have exacerbated environmental transmission (Mejía-Salazar et al. 2018).

We considered the possibility that iatrogenic transmission confounded outcomes. This seems unlikely given available data. About half of the infected deer we encountered tested positive upon first capture. Of the deer not testing positive until recapture 274–1,253 days later, fewer than half had been previously handled on the same day as another infected deer. Based on data from a subset of 88 deer with or without potential same-day exposure and with a follow-up test result, we calculated the relative risk of infection for deer previously sampled on the same day as another infected deer to be 1.3 (95% CL=0.5–3.2). However, in the course of reconstructing capture histories, we encountered 13 examples where multiple infected deer were connected to the same social group or location (Fig. 1), consistent with social and spatial exposure risks documented in mule deer elsewhere (Cullingham et al. 2011; Mejía-Salazar et al. 2017, 2018).

Our efforts to "test and cull" CWD-infected mule deer in the Estes Park and Rocky Mountain National Park area minimally stimulated a trend toward decreased prevalence among males and held prevalence static among females. A more intensive and sustained effort or modified spatial approach (e.g., targeting "high risk" environments or locations; Mejía-Salazar et al. 2018) might have reduced prevalence in both sexes. Limitations of this technique in wider management application include cost and labor (Wolfe et al. 2004b), as well as property access and animal tolerance to repeated capture. With some modification, however, elements of this approach could potentially be used to augment harvest-based or environment-based disease management. In some aspects, our culling of known CWD-positive animals simulated the effect of natural predators in the wild that exploit vulnerabilities and weakness when selecting prey. Although we detected some infected individuals well before clinical signs would have been discernible to a predator, at the herd level our testing effort likely was not as persistent or effective as that of natural predators. Our findings could lend credence to the potential role of predation—of sufficiently high intensity and duration—in helping suppress CWD outbreaks if CWD-positive individuals are preferentially targeted by predators (Wild et al. 2011).

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SUPPLEMENTARY MATERIAL

Supplementary material for this article is online at http://dx.doi.org/10.7589/2018-01-015.

LITERATURE CITED

Almberg ES, Cross PC, Johnson CJ, Heisey DM, Richards BJ. 2011. Modeling routes of chronic wasting disease transmission: Environmental prion persistence promotes deer population decline and extinction. *PLoS One* 6:e19896.

Bowden DC, Kufeld RC. 1995. Generalized mark-sight population size estimation applied to Colorado moose. *J Wildl Manag* 59:840–851.

Conner MM, McCarty CW, Miller MW. 2000. Detection of bias in harvest-based estimates of chronic wasting disease prevalence in mule deer. *J Wildl Dis* 36:691–699.

Conner MM, Miller MW. 2004. Movement patterns and spatial epidemiology of a prion disease in mule deer population units. *Ecol Appl* 14:1870–1881.

Conner MM, Miller MW, Ebinger MR, Burnham KP. 2007. A meta-BACI approach for evaluating management intervention on chronic wasting disease in mule deer. *Ecol Appl* 17:140–153.

Cullingham CI, Nakada SM, Merrill EH, Bollinger TK, Pybus MJ, Coltman DW. 2011. Multiscale population genetic analysis of mule deer (*Odocoileus hemionus hemionus*) in western Canada sheds new light on the spread of chronic wasting disease. *Can J Zool* 89:134–147.

Geremia C, Hoeting JA, Wolfe LL, Galloway NL, Antolin MF, Spraker TR, Miller MW, Hobbs NT. 2015a. Age and repeated biopsy influence antemortem PrP^{CWD} testing in mule deer (*Odocoileus hemionus*) in Colorado, USA. *J Wildl Dis* 51:801–810.

Geremia C, Miller MW, Hoeting JA, Antolin MF, Hobbs NT. 2015b. Bayesian modeling of prion disease dynamics in mule deer using population monitoring and capture-recapture data. *PLoS One* 10:e0140687.

Gross JE, Miller MW. 2001. Chronic wasting disease in mule deer: Disease dynamics and control. *J Wildl Manag* 65:205–215.

Hoover CE, Davenport KA, Henderson DM, Pulscher LA, Mathiason CK, Zabel MD, Hoover EA. 2016. Detection and quantification of CWD prions in fixed paraffin embedded tissues by real-time quaking-induced conversion. *Sci Rep* 6:25098.

Mateus-Pinilla N, Weng HY, Ruiz MO, Shelton P, Novakofski J. 2013. Evaluation of a wild white-tailed deer population management program for controlling chronic wasting disease in Illinois, 2003–2008. *Prevent Vet Med* 110:541–548.

Mejía-Salazar MF, Goldizen AW, Menz CS, Dwyer RG, Blomberg SP, Waldner CL, Cullingham CI, Bollinger TK. 2017. Mule deer spatial association patterns and potential implications for transmission of an epizootic disease. *PLoS One* 12:e0175385.

Mejía-Salazar MF, Waldner CL, Hwang YT, Bollinger TK. 2018. Use of environmental sites by mule deer: A proxy for relative risk of chronic wasting disease exposure and transmission. *Ecosphere* 9:e02055.

Miller MW, Conner MM. 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 MW, Williams ES, McCarty CW, Spraker TR, Kreeger TJ, Larsen CT, Thorne ET. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. *J Wildl Dis* 36:676–690.

Potapov A, Merrill E, Pybus M, Lewis MA. 2016. Chronic wasting disease: Transmission mechanisms and the possibility of harvest management. *PLoS One* 11:e0151039.

Pybus MJ. 2012. *CWD program review 2012*. Alberta Sustainable Resource Development, Fish and Wildlife Division, Edmonton, Alberta, Canada. http://aep.alberta.ca/fish-wildlife/wildlife-diseases/chronic-wasting-disease/documents/CWD-ProgramReview-May-2012.pdf. Accessed February 2018.

Race RE, Raymond GJ. 2004. Inactivation of transmissible spongiform encephalopathy (prion) agents by Environ LpH. *J Virol* 78:2164–2165.

Spraker TR, Miller MW, Williams ES, Getzy DM, Adrian WJ, Schoonveld GG, Spowart RA, O'Rourke KI, Miller JM, Merz PA. 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 TR, O'Rourke KI, Balachandran A, Zink RR, Cummings BA, Miller MW, Powers BE. 2002. Validation of monoclonal antibody F99/97.6.1 for immunohistochemical staining of brain and tonsil in mule deer (*Odocoileus hemionus*) with chronic wasting disease. *J Vet Diagn Investig* 14:3–7.

Uehlinger FD, Johnston AC, Bollinger TK, Waldner CL. 2016. Systematic review of management strategies to control chronic wasting disease in wild deer populations in North America. *BMC Vet Res* 12:173.

WAFWA (Western Association of Fish and Wildlife Agencies). 2017. *Recommendations for adaptive management of chronic wasting disease in the West*. WAFWA Wildlife Health Committee and Mule Deer Working Group, Edmonton, Alberta, Canada and Fort Collins, Colorado. https://www.wafwa.org/Documents%20and%20Settings/37/Site%20Documents/Committees/Wildlife%20Health/docs/CWDAdaptiveManagementRecommendations_WAFWAfinal_approved010618.pdf. Accessed February 2018.

White GC. 1996. NOREMARK: Population estimation from mark-resighting surveys. *Wildl Soc Bull* 24:50–52.

Wild MA, Hobbs NT, Graham MS, Miller MW. 2011. The role of predation in disease control: A comparison of selective and nonselective removal on prion disease dynamics in deer. *J Wildl Dis* 47:78–93.

Williams ES, Young S. 1980. Chronic wasting disease of captive mule deer: A spongiform encephalopathy. *J Wildl Dis* 16:89–98.

Wolfe LL, Conner MM, Baker TH, Dreitz VJ, Burnham KP, Williams ES, Hobbs NT, Miller MW. 2002. Evaluation of antemortem sampling to estimate chronic wasting disease prevalence in free-ranging mule deer. *J Wildl Manag* 66:564–573.

Wolfe LL, Lance WR, Miller MW. 2004a. Immobilization of mule deer with thiafentanil (A-3080) or thiafentanil plus xylazine. *J Wildl Dis* 40:282–287.

Wolfe LL, Miller MW, Williams ES. 2004b. Feasibility of "test and cull" for managing chronic wasting disease in urban mule deer. *Wildl Soc Bull* 32:500–505.

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