

# HUNTING PRESSURE MODULATES PRION INFECTION RISK IN MULE DEER HERDS

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**ABSTRACT:** The emergence of chronic wasting disease, an infectious prion disease of multiple deer species, has motivated international calls for sustainable, socially accepted control measures. Here, we describe long-term, spatially replicated relationships in Colorado, US, mule deer (*Odocoileus hemionus*) herds that show hunting pressure can modulate apparent epidemic dynamics as reflected by prevalence trends. Across 12 areas in Colorado studied between 2002–18, those with the largest declines in annual hunting license numbers (pressure) showed the largest increases in the proportion of infected adult ( $\geq 2$ -yr-old) male deer killed by hunters (prevalence); prevalence trends were comparatively flat in most areas where license numbers had been maintained or increased. The mean number of licenses issued in the 2 yr prior best explained observed patterns: increasing licenses lowered subsequent risk of harvesting an infected deer, and decreasing licenses increased that risk. Our findings suggest that harvesting mule deer with sufficient hunting pressure might control chronic wasting disease—especially when prevalence is low—but that harvest prescriptions promoting an abundance of mature male deer contribute to the exponential growth of epidemics.

**Key words:** Chronic wasting disease, control, epidemiology, hunting, mule deer, *Odocoileus hemionus*, prion, risk.

## INTRODUCTION

Chronic wasting disease (CWD; Williams and Young 1980)—an infectious prion disease—now occurs on three continents in at least seven species in the family *Cervidae* (Benestad et al. 2016; Miller and Fischer 2016; Sohn et al. 2016). Its emergence has raised concerns among hunting, conservation, agriculture, and food safety communities worldwide (Miller and Fischer 2016; EFSA Panel on Biological Hazards [BIOHAZ] et al. 2017; Hannaoui et al. 2017; Mysterud and Edmunds 2019). Numerous scattered disease foci of varied size span much of central North America's wildlands, including those in Colorado, US. Despite growing recognition that CWD needs to be controlled, approaches insensitive to the economic and ecological importance of affected host populations have been socially unacceptable and thus unsustainable when applied (Holsman et al. 2010; Miller and Fischer 2016; Mysterud and Edmunds 2019). Hunting can be an effective and socially acceptable tool for managing deer (*Odocoileus* spp.; Krausman and Bleich 2013)

but its effectiveness in controlling this disease has been debated (Manjerovic et al. 2014; Potapov et al. 2016; Uehlinger et al. 2016).

Colorado's recorded history with CWD dates to the 1960s, when a syndrome first noticed by university scientists studying captive mule deer (*Odocoileus hemionus*) was later described as a new transmissible spongiform encephalopathy (now prion disease; Williams and Young 1980, 1992). The disease syndrome initially appeared confined to animals held in captivity in the US and in a Canadian zoo, but by the early 1980s symptomatic cases were being diagnosed in free-ranging deer and wapiti (*Cervus canadensis*) in northcentral Colorado and in southeastern Wyoming (Williams and Young 1992). By the early 1990s the growing number of documented field cases compelled early attempts to estimate infection prevalence (the proportion of animals infected) by sampling hunter- and vehicle-killed deer and wapiti. By the late 1990s those surveys had defined a well-established disease focus involving much of northeastern Colorado and southeastern Wyoming (Miller et al. 2000).

Surveys of three of western Colorado's largest mule deer herds—White River, Middle Park, and Uncompahgre—conducted in 1998–99 revealed no evidence of CWD, supporting the short-lived notion that occurrence in the wild was geographically-confined. But, in early 2002, a cluster of preclinical cases was unexpectedly detected in wild mule deer entrapped in a commercial wapiti facility located near Pagoda, within the range of the White River herd. In autumn 2002, a massive statewide testing campaign screened >25,000 hunter-killed deer and wapiti, revealing that CWD was relatively rare but already far more widespread across northern Colorado than believed just a few years before. In retrospect, the disease likely had been present for a decade or more even in newly discovered locations in Colorado (and elsewhere: Miller et al. 2000; Miller and Fischer 2016). Despite its unexpected geographic distribution, disease prevalence was relatively low (<1–2%) in most affected Colorado deer herds outside the original core endemic area. As a result, attention to CWD occurrence in Colorado waned.

In the course of reviewing changes in annual estimates of disease prevalence among male mule deer harvested from affected Colorado herds more than a decade later, we noticed variability in trends over time since 2002. Prevalence, measured as the proportion of sampled adult ( $\geq 2$ -yr-old) male deer testing positive for abnormal prion protein accumulation, had increased exponentially in some herds but remained relatively stable or declined in others. Changes in prevalence among harvested adult females showed trends paralleling those among sympatric harvested males in herds where females also were harvested, confirming that the trends observed among males were not simply an artifact of sampling or changes in the age structure of harvested individuals.

These patterns had developed over the nearly two decades that followed sweeping changes in the approach to mule deer hunting and harvest management in Colorado (Bergman et al. 2011). Prior to 1999, licenses to hunt male deer were available in unlimited

numbers in most areas but thereafter were issued in limited numbers and assigned to specific geographic areas (Bergman et al. 2011). Consequently, the number of licenses issued and the annual hunting pressure was abruptly halved across most of the state in 1999, and reduced further since, to spare a larger proportion of older adult male deer from harvest (Bishop et al. 2005; Bergman et al. 2011) or to offset the expected effects of especially severe winters on adult male abundance (such as during 2007–08). However, license numbers and hunting pressure were sustained or even increased in a few deer herds (e.g., Red Feather-Poudre Canyon, Big Thompson, Middle Park) to achieve various local herd management goals, including attempts to suppress CWD epidemic growth.

Here we describe long-term, spatially replicated relationships between hunting pressure and CWD dynamics in Colorado mule deer herds that show hunting can modulate disease emergence. Our data offer an empirical basis for developing sustainable programs to suppress this disease in natural systems.

## MATERIALS AND METHODS

### Background on the nuances of deer and deer hunting in Colorado

Two deer species are native to Colorado. Mule deer dominate shrub-steppe and montane habitats throughout the roughly western half of the state—delineated by the Interstate Highway 25 (I-25) corridor—but share the tablelands and riparian habitats in the eastern half of the state more proportionally with white-tailed deer (*Odocoileus virginianus*). For this reason, hunting season timing and structures are different for areas west or east of I-25. The analyses described here focused on mule deer herds located in the northwestern quarter of Colorado, west of I-25 (Fig. 1). Unless otherwise specified, a deer hunting license allowed for the harvest of a single deer of either species in the areas we included in our analyses. However, the vast majority of deer harvested in these areas were mule deer and all of the prevalence data we analyzed were from mule deer. Consequently, throughout the text we have used the terms deer and mule deer interchangeably and have specified our references to white-tailed deer where appropriate.

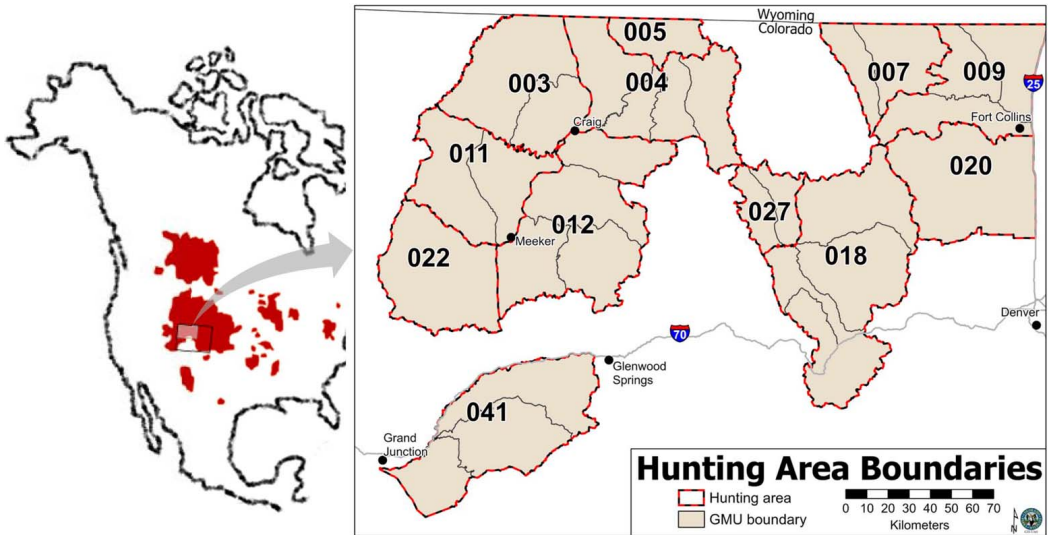


FIGURE 1. Distribution of chronic wasting disease in North American deer (*Odocoileus* spp.) and the relative location of the 12 hunt areas in Colorado, USA, (inset) that were the focus of our analyses. Hunt area (numbered) and associated game management unit (GMU) boundaries are shown. Interstate Highway 25 (I-25) is located in the upper right of the inset panel. North American disease distribution adapted from an online map maintained by the US Geological Survey (2019).

Deer hunting in Colorado occurs during a successive series of 5- to 10-d seasons. Seasons used in the western half of Colorado (west of I-25) during the years covered by our study included a second (mid-October), third (late October–early November), fourth (mid-late November) and in some cases, later management seasons (all seasons after the fourth). All of these seasons precede or overlap the annual mule deer breeding season (mid-November to mid-December in northern Colorado). Hunters could use high-powered rifles in all of these seasons. Each rifle hunting license allowed the holder to hunt deer in a specified geographic area during a specified season.

Population and male-to-female ratio objectives for all Colorado deer herds of both species are set through a public herd management planning process and approved by the Colorado Parks and Wildlife Commission. Wildlife managers then use harvest via hunting to manipulate various aspects of deer population biology, including abundance, age structure, and male to female ratios to manage toward those individualized herd objectives. Managers set annual license quotas to regulate deer hunting across the available seasons. Licenses are apportioned across geographically-defined areas, with each license allowing the holder to hunt deer only during a single season and only within one or more contiguous game management units comprising the prescribed hunting area (specified by

a license code). Managers can modify the recommended quota of available licenses annually toward meeting established herd objectives and in response to constituent input.

Licenses for hunting female deer have been limited in number for decades in Colorado, but licenses for hunting male deer became limited statewide in 1999. Prior to totally limiting licenses for hunting male deer, Colorado had a 5-yr average of 166,000 deer hunters compared to 81,000 in 1999 after the statewide limitation. Mature male mule deer are more vulnerable to harvest in seasons that overlap with breeding activity, in part because they become less wary and in part because they become more readily detected by hunters by joining larger groups residing in lower canopy cover, increasing activity in daylight, and moving closer to roads. Consequently, the number of licenses issued in third, fourth, and late seasons can be used to increase or decrease male:female ratios and the age structure among males; managers decrease the number of licenses issued for those seasons to increase the sex ratio and the number of mature males retained in a herd (Bishop et al. 2005; Bergman et al. 2011).

### Study area

To explore potential mechanisms underlying the relationship between hunting pressure and prevalence, we used data from six mule deer herd

ranges collectively spanning >33,000 km<sup>2</sup> in the northwestern quadrant of Colorado (Fig. 1 and Supplementary Material Table S1). We restricted our analysis to areas located west of I-25 to ensure consistency in hunting seasons. These represented all of the affected Colorado mule deer herds west of I-25 where CWD was known to be endemic in 2002, where mandatory surveys had been conducted in either 2017 or 2018 to confirm prevalence trends, and where the 1–3 hunting areas within each herd ( $n=12$  total; Table S1) had remained consistent from 2000 through 2018. We excluded other affected deer herds a priori because they failed to meet one or more of these criteria.

We analyzed data at the spatial scale of individual hunting areas because they were the basis for setting of license numbers. The 12 hunting areas we compared (Fig. 1 and Table S1) had similar hunting season structures relative to third through late hunting seasons, as well as consistent game management unit compositions from year to year during 2000–18.

### Study design

We explored relationships between the number of hunting licenses issued and subsequent CWD prevalence among harvested adult male mule deer by analyzing annual hunting license sales data and prevalence data in these 12 hunting areas over the 17-yr period from 2002–18 (Fig. 1 and Table S1).

### Hunting license numbers

Deer in all areas were hunted each autumn. Managers apportioned and controlled harvest pressure across these geographically defined areas by establishing annual license quotas for each season in the hunting area covered by a license (Fig. 1 and Table S1). The number of licenses varied among areas and within areas over time. We used the number of licenses sold for the hunting seasons in late October and after (Table S1) from 2000–17 as a direct measure of annual hunting pressure during the seasons when harvest of mature male deer and infected male deer would be most likely (Conner et al. 2000; Bishop et al. 2005; Bergman et al. 2011).

### Disease prevalence

To track CWD occurrence and estimate infection odds, we measured annual prevalence using prion diagnostic data from 19,105 tissue samples collected from adult ( $\geq 2$ -yr-old) male mule deer killed by hunters in these 12 areas during 2002–18. Prevalence as measured in adult males has become our preferred metric for mule

deer because infection is relatively rare in fawns and yearlings and rates among adult males tend to be about twice that measured among adult females in the same herd (Miller et al. 2000, 2008; Miller and Conner 2005; DeVivo et al. 2017). Annual male harvest also tends to provide larger and more consistent sample sizes for assessing trends over time and between Colorado mule deer herds than female harvest. We express prevalence data as percentages for clarity.

Medial retropharyngeal lymph nodes were collected from submitted deer along with data on gender, age (determined by visual inspection for incisor replacement), and harvest date and location. Tissue samples were screened for the presence of disease-associated prion protein using an enzyme-linked immunosorbent assay (Hibler et al. 2003); we classified samples returning an optical density value  $\geq 0.1$  as positive for prion infection (apparent sensitivity=99.6%, apparent specificity=99.7%; Hibler et al. 2003).

### Statistical analyses

We hypothesized that license numbers from the year prior or from both of the prior 2 yr can affect the occurrence of CWD measured in the reference year. Because detected infections are the product of prion exposure months to years earlier (Fox et al. 2006), we anticipated a lagged prevalence response consistent with the >2-yr disease course in naturally infected mule deer (Miller et al. 2008; DeVivo et al. 2017).

We used logistic regression (Hosmer and Lemeshow 1989) to investigate how the number of licenses sold in the prior years correlated with the probability of adult male deer being CWD-negative each year. We used licenses sold because most license groups reach their quota of allocated licenses each year and thus the number sold represented both a known quantity and a management objective rather than an imprecise estimate (such as harvest, density, or total number of deer). We included five predictor variables in the analysis: designated hunting area (license group); the number of third (=late October–early November) season licenses sold the year prior/100 (third); the average of the numbers of third season licenses sold for the 2 yr prior/100 (third 2 yr); and the same two metrics for the sum of third, fourth (=mid–late November), and later management season licenses/100 (TFM, TFM2yr). We express results in two complementary ways. One is the effect that license numbers have on harvested deer being disease-free (i.e., a response variable of one for negative animals in the logistic regression), and the other the effect that license numbers have on prevalence.

Because the 12 areas differed in numbers and trends in licenses sold (Fig. 2 and Table S1), we

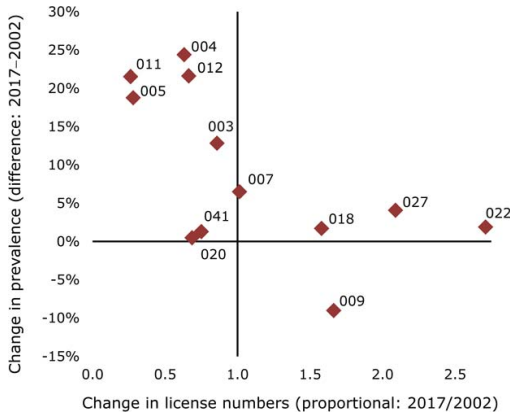


FIGURE 2. Changes in chronic wasting disease prevalence (proportion infected, expressed as a percentage) and number of licenses for hunting during late October–early November (third season) issued annually between 2002 and 2018 across 12 mule deer (*Odocoileus hemionus*) hunting areas in Colorado, USA. In area 020, license numbers were maintained but shifted to a later hunting season. Points are labeled with hunting area number to allow cross-reference to Figs. 1, 3, and Supplementary Material S1.

anticipated that the relationship between licenses and CWD might vary across areas. Consequently, we fit models that included the interaction between licenses sold and hunting area (i.e., license group). We also fit models that included both factors without an interaction, license group only, licenses sold only, and no explanatory factors at all (i.e., a null model). These combinations of different factors yielded 14 competing models, which we compared with Akaike information criterion (AIC; Akaike 1973).

We used PROC LOGISTIC in SAS software (version 9.4, SAS Institute Inc., Cary, North Carolina, USA) with a logit link to conduct the statistical analysis. Although we fit model likelihoods and estimated parameters with a logit link, we derived odds ratios and probabilities from the parameter estimates using standard equations (Hosmer and Lemeshow 1989) and present results on those scales for ease of interpretation.

## RESULTS

Across the 12 hunting areas analyzed, the four with the largest proportional overall declines in the number of hunting licenses issued annually (areas 004, 005, 011, 012) also had the largest increases in disease prevalence between 2002 and 2018 (Fig. 2). In area 020, the overall number of licenses was maintained

but a proportion was shifted to a later hunting season (Table S1). Prevalence at the beginning of the analysis period was relatively low (0.3–1.9%) in all except the three areas (007, 009, 020) where CWD had been enzootic for at least the prior two decades (Table S1). By 2017–18, prevalence had increased about seven-fold or more in five of the nine hunting areas with low starting prevalence (areas 003, 004, 005, 011, 012), ranging from 13% to 25% (Fig. 2 and Table S1); among the other four hunting areas (018, 022, 027, 041) prevalence ranged from 1.6% to 5.2% (Table S1). Prevalence in the three areas with relatively high starting prevalence ranged from 6.3% to 12.6% by 2017, having declined in area 009, increased somewhat in area 007, and remained essentially unchanged in area 020 since 2002 (Fig. 2 and Table S1).

Logistic regression revealed that license group (hunting area) interacting with the average number of late October–early November (third season) hunting licenses sold during the 2 yr prior to a focal prevalence year best explained observed patterns in the prevalence data (Table S2). The closest model was 11.8 AIC units higher (Table S2), placing 99.999% of the AIC weight on our top model. Because it was so well-supported, we present results from only this model.

The average number of licenses sold inversely correlated with prevalence among deer harvested 1–2 yr later in six of the 12 areas (Fig. 3 and Tables S3, S4). In those six areas, changes in license numbers positively correlated with the chance of deer harvested 1–2 yr later being free from apparent infection (i.e., test-negative) with odds ratios (OR) ranging from 1.2 (95% CI: 1.17–1.24) to 17.04 (CI: 5.00–58.10; Fig. S1 and Table S4). As an example, hunt area 004 had an OR of 1.71 (CI: 1.57–1.87), meaning that for every 100 licenses added on average in the prior 2 yr, adult male deer had a 71% greater chance of being disease-free in the year of harvest or, for every 100 licenses subtracted, adult male deer subsequently harvested had a 71% greater chance of being infected. The ORs in four additional areas overlapped an even odds ratio (i.e., no statistical relationship; Fig.

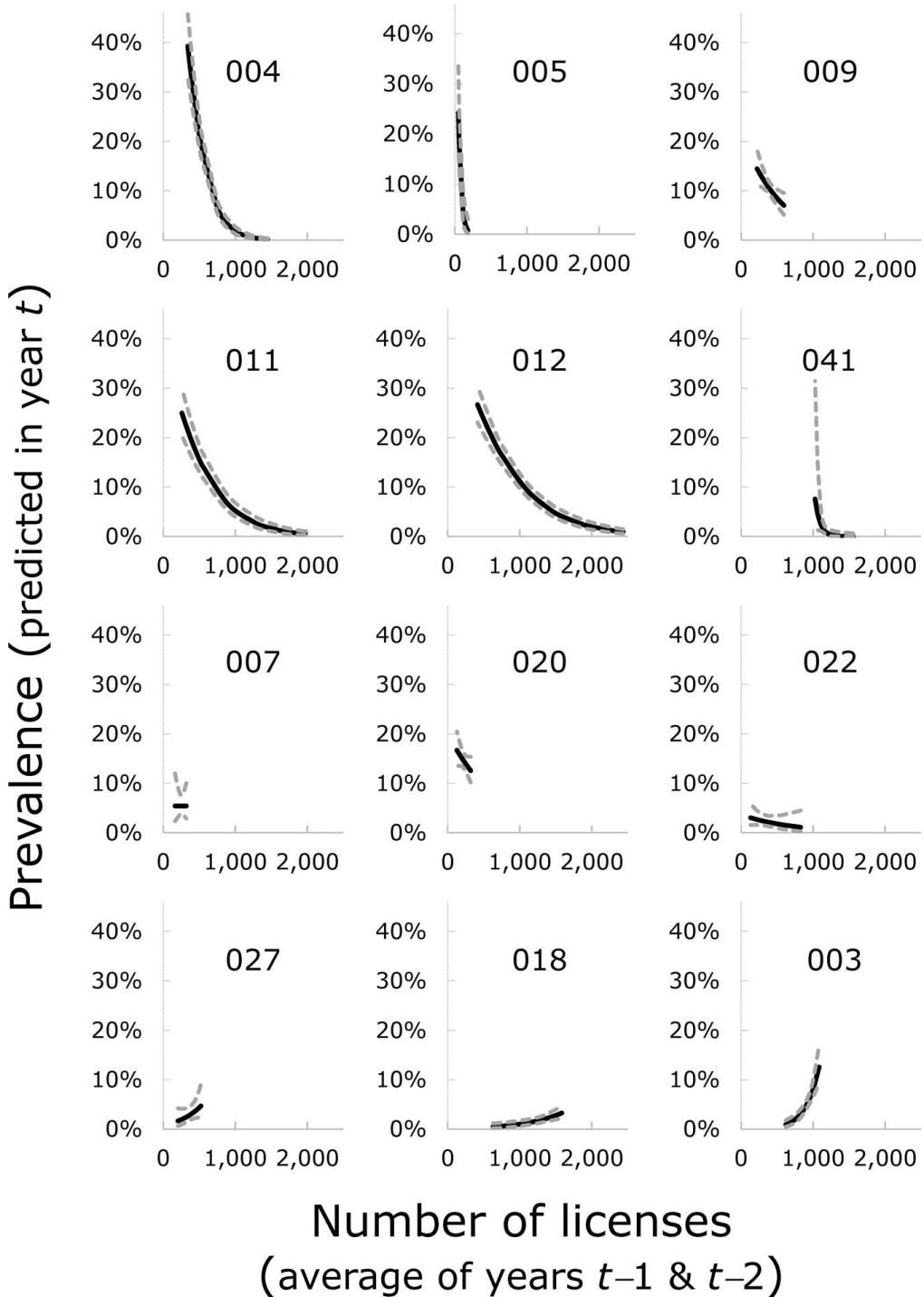


FIGURE 3. Chronic wasting disease prevalence (proportion infected, expressed as a percentage) as a function of the number of hunting licenses issued annually in selected hunting areas in Colorado, USA, 2000–18. In six of the 12 areas (top two rows), the average number of licenses inversely correlated with prevalence among adult male mule deer (*Odocoileus hemionus*) harvested 1–2 yr later. Solid line traces trend in prevalence (in year  $t$ ; y-axis) as predicted from the top logistic regression model over the range of third-season license numbers averaged over the preceding 2 yr (years  $t-1$  and  $t-2$ ; x-axis) in each area. Dashed lines cover 95% confidence limits around prevalence trends. The three-digit number in each panel denotes the hunting area.

S1 and Table S4). Two areas (003, 018) had ORs <1 (Fig. S1 and Table S4). Pooling results over all areas showed a positive relationship between increasing licenses and the odds of harvested deer being uninfected (OR: 1.16, CI: 1.14–1.17), although the associated explanatory model itself was not statistically supported ( $\Delta\text{AIC}=723$ ; Table S2). The relationship between license numbers and predicted prevalence varied among license groups, suggesting local factors also shaped prion disease dynamics in individual hunting areas (Fig. 3).

## DISCUSSION

Our findings suggest that harvesting mule deer with sufficient hunting pressure might affect CWD control—especially when prevalence is low—but that harvest prescriptions promoting an abundance of mature male deer contribute to the exponential growth of epidemics. Chronic wasting disease prevalence and incidence show positive correlation across a wide range of values in field studies where both parameters were measured in the same mule deer herd, with prevalence equaling or somewhat overestimating incidence in both genders (Miller et al. 2008; Geremia et al. 2015; DeVivo et al. 2017). We therefore inferred that observed prevalence trends also reflected underlying epidemic dynamics, and regarded either declining or relatively flat prevalence trends as encouraging evidence of short-term epidemic suppression (Manjerovic et al. 2014). Parallel prevalence trends among male and female deer in five of the herds we studied support such inference (Fig. 4).

The single area (009) showing a decline in prevalence since 2002 had been a focus of disease suppression efforts since 2000 that also included aggressive harvest of female deer and limited, localized professional sharpshooting over the first 5 yr (Conner et al. 2007; Geremia et al. 2015). License numbers for hunting male deer in this area varied but generally had increased over time. In a second area (041) with negative correlation between prior years' license numbers and infection

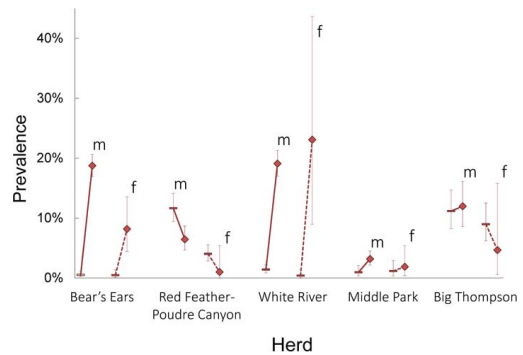


FIGURE 4. Chronic wasting disease prevalence trends for five Colorado, USA, mule deer (*Odocoileus hemionus*) herds harvested by sport hunting and sampled in 2002–04 (dash symbols) or in 2017–19 (diamond symbols). Prevalence (expressed as a percentage) was measured as the proportion of sampled adult ( $\geq 2$  years old) male (m; solid lines) or female (f; dashed lines) deer testing positive for abnormal prion protein accumulation. Because relatively few female deer were harvested in most herds, we pooled data from each 3-yr time period to improve precision of prevalence estimates in showing general trends between the two sampling periods. Vertical lines cover the 95% binomial confidence intervals.

odds among harvested male deer, as well as in three of the four areas with equivocal correlations (areas 020, 022, 027), hunting pressure also varied but had been largely sustained or increased (Fig. 2). Prevalence in these areas remained stable (area 020) or rose <5% over 17 yr, suggesting epidemic growth was relatively slow. Despite the small but positive correlation with license number increases, prevalence in area 018 increased only about 2% over 17 yr.

In contrast, prevalence markedly increased since 2002 in four hunting areas (004, 005, 011, 012) with negative correlations between license numbers and infection odds following overall declines in hunting pressure that began in 1999 when local managers reduced license numbers in those areas to promote mature male deer abundance (Bishop et al. 2005; Bergman et al. 2011). The patterns of annual license numbers varied among areas, including short-term increases in some, but the long-term trends for all four were downward. The relatively sharp rise in prevalence in those areas likely was an unintended

collateral effect of protecting the mature male demographic from harvest (Potapov et al. 2016). We regard the concurrent, parallel rise in observed prevalence among sympatric female deer in the Bear's Ears and White River herds (Fig. 4) as further evidence of local epidemic growth over this period. Prevalence also rose in two areas (003, 007) where license numbers had been largely sustained, suggesting that overall pressure might have been inadequate to affect suppression or that other factors (e.g., immigration; Wolfe et al. 2018) were contributing.

As is inherent in any retrospective, opportunistic analysis of management data, our findings are not without limitations. Absent a designed framework, the trends in license numbers in areas studied were mostly declining or stable rather than offering some ideal mix of increased and decreased hunting pressure over time. Mandatory sampling at regular intervals (e.g., 3–5 yr) would have yielded more refined measures of prevalence trends. Sustained female harvest in the Middle Park, Red Feather-Poudre Canyon, and Big Thompson herds might have contributed to epidemic suppression in associated hunting areas, just as severe winter conditions in 2007–08 variably affected all but the three easternmost areas. Despite these potential limitations, we believe our data offer an empirical basis for developing more refined, adaptive, and sustainable programs to suppress CWD in western, mule deer-dominated natural systems. Whether and how our findings might apply to strategies for suppressing CWD in higher density white-tailed deer herds in eastern North America will require further consideration, although a few key principles seem likely to be relevant.

Prion-infected mule deer show greater vulnerability to predation (Miller et al. 2008; Krumm et al. 2010; DeVivo et al. 2017) and vehicle collisions (Krumm et al. 2005), and also seem more vulnerable to hunter harvest than uninfected individuals (Conner et al. 2000; DeVivo et al. 2017). Removal mechanisms that preferentially target infected animals should be most effective in suppressing epidemic growth (Barlow 1996; Wild et al.

2011; Potapov et al. 2016; Tanner et al. 2019a). Hunting has been used to limit deer abundance (Krausman and Bleich 2013), and harvest might afford compensatory benefits in host populations afflicted with invariably fatal diseases (Tanner et al. 2019b). Our data suggest that hunting can be a partially selective tool for removing infected individuals, provided hunting pressure is of sufficient intensity and duration to exploit the relative vulnerability of infected deer. Hunting pressure applied over a series of successive seasons likely helps expose vulnerable infected deer to harvest in later seasons as learned wariness develops in unaffected individuals (Conner et al. 2000). Hunting applied in this way could more closely mimic predation. Differential vulnerability might have less time to emerge when all of the annual hunting pressure is predictably applied in a single, relatively brief time period. This perhaps explains the lack of apparent benefits of hunting for disease control reported elsewhere (Manjerovic et al. 2014; Uehlinger et al. 2016; Mysterud and Edmunds 2019). As our results show, sustained hunting pressure might afford a level of suppression comparable to that expected from natural predation, but light harvest likely will not stem epidemic growth even when applied over multiple seasons (Figs. 2–4; Wild et al. 2011; Potapov et al. 2016; Tanner et al. 2019a, b).

Our findings offer both optimism and caution to those responsible for controlling CWD. Hunting is a socially accepted tool readily available to wildlife managers in most places where CWD occurs. Measurable disease suppression appears achievable using hunting as the main or only tool, provided interest and willingness of people to hunt in affected areas can be retained long-term. Past surveys indicate that hunters broadly supported taking measures to control CWD (Needham et al. 2004). Such support could be tested by harvest prescriptions that run contrary to those for maintaining older male deer in abundance. If the use of hunting pressure for disease suppression directs decisions on license numbers and season timing, then hunting alone might be sufficient to affect



control, especially when prevalence is low. However, the simple act of hunting a deer herd does not appear universally effective. Moreover, some harvest strategies—for example, those promoting an abundance of mature males—might exacerbate prion transmission and contribute to exponential growth of epidemics (Figs. 2–4). In this light it is not surprising that the emergence of CWD in northwestern Colorado coincided with changes in deer hunting that sought to spare prime-aged male deer, the demographic most likely to be infected (Miller et al. 2000, 2008; Miller and Conner 2005; Potapov et al. 2016).

The conundrum in using hunting to control CWD lies in the collateral exposure of hunters and others to this prion disease via consuming harvested venison (EFSA Panel on Biological Hazards [BIOHAZ] et al. 2017; Hannaoui et al. 2017). Early, sustained intervention when the disease is relatively rare seems a prudent strategy both for minimizing such exposure and for ensuring the greatest likelihood of success in suppressing epidemic growth.

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#### SUPPLEMENTARY MATERIAL

Supplementary material for this article is online at <http://dx.doi.org/10.7589/JWD-D-20-00054>.

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