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# RELATIVE VULNERABILITY OF CHRONIC WASTING DISEASE INFECTED MULE DEER TO VEHICLE COLLISIONS

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ABSTRACT: We estimated chronic wasting disease (CWD) prevalence among vehicle-killed mule deer (*Odocoileus hemionus*) in select data analysis units (DAUs) in northern Colorado, USA, and compared these with estimated CWD prevalence among mule deer of the same sex sampled in the vicinity of collision sites to assess relative vulnerability of CWD-infected individuals to vehicle collisions. Twenty-five of 171 vehicle-killed mule deer tested positive for CWD (overall prevalence=0.146, 95% confidence interval [CI]=0.097–0.208); 173 of 2,317 deer sampled in the vicinity of these vehicle-killed deer tested positive (overall prevalence=0.075, 95% CI=0.064–0.085). In nine of ten DAU×sex comparisons, relative risk of CWD infection tended to be higher among vehicle-killed deer (range of estimated relative risks=1.6–15.9). Spongiform encephalopathy was detected in 12 of 20 (60%; 95% CI=39–81%) CWD-positive deer killed by vehicles and in 79 of 180 (44%; 95% CI=37–52%) CWD-positive deer detected via random sampling (relative risk=1.37; 95% CI=0.92–2.03), suggesting that infected deer killed by vehicles tended to be in later stages of disease than those killed by hunters. Our data offer evidence that CWD-infected mule deer may be relatively vulnerable to vehicle collisions. It follows that sampling of vehicle-killed mule deer may be exploited to increase efficiency of surveillance programs designed to detect new foci of CWD infection; moreover, evidence of increased susceptibility to vehicle collisions may aid in understanding vulnerability of CWD-infected individuals to other forms of death, particularly predation.

*Key words:* Chronic wasting disease (CWD), mule deer, *Odocoileus hemionus*, predator-prey, prion, surveillance, transmissible spongiform encephalopathy, vehicle collision.

## INTRODUCTION

Tens of millions of vertebrates are killed on roadways each year, including an estimated 0.5 to 1.5 million deer (*Odocoileus* spp.) in the United States alone (Clevenger et al., 2002). For certain species, mortality rates attributable to vehicle collisions may exceed natural mortality rates due to predation and disease (Forman, 2003). Previous studies have shown that mortality rates from deer-vehicle collision vary based on factors like migratory routes (Mansfield and Miller, 1975; Reeve and Anderson, 1993), breeding and hunting seasons (Myers, 1969; Bellis and Graves, 1971; Reilly and Green, 1974; Goodwin and Ward, 1976; Sicuranza, 1979; Dusek et al., 1989), and herd composition (Jahn, 1959; Goodwin and Ward, 1976; Romin and Bisonette, 1996). These studies revealed behavioral traits influencing location and timing of deer-vehicle collisions. However, it seems logical that behavioral or cognitive changes due to disease also may influence vulnerability to vehicle collisions and that such changes could be exploited in designing surveillance programs for disease detection.

Chronic wasting disease (CWD) (Williams and Young, 1980), a contagious prion disease of deer and wapiti (*Cervus elaphus nelsoni*), has emerged as an important wildlife health problem in several parts of North America (Williams and Miller, 2002; Williams et al., 2002). Clinical signs of CWD include subtle changes in behavior that are often unrecognizable in early stages. Desensitization to external stimuli and poor body condition become progressively worse in later stages (Williams and Young, 1992). Abnormal behavior and deterioration of body condition are the most apparent clinical signs of end-stage CWD in deer (Williams and Young, 1980, 1992).

Affected animals may show repetitive behaviors or periods of somnolence or depression and may carry their ears and head lowered; various combinations of uncoordination, stumbling, trembling, and hyperexcitability are also displayed (Williams et al., 2002). It follows that CWD-infected deer and elk in later disease stages could be more likely to be struck by vehicles because they are less able to recognize, to avoid, or to respond to vehicles. Here, we investigated whether mule deer (*Odocoileus hemionus*) infected with CWD might be more susceptible to vehicle collisions by comparing prevalence among vehicle-killed deer to prevalence among sympatric deer from the same geographic location.

## MATERIALS AND METHODS

As part of an ongoing CWD surveillance program, tissue samples (tonsil or retropharyngeal lymph node, ±medulla oblongata at the obex; Miller and Williams, 2002) were collected opportunistically from vehicle-killed mule deer throughout Colorado (Fig. 1) during 1996–2004. Sampling effort was more intensive in portions of north-central Colorado where CWD surveillance has been ongoing since the early 1990s (Miller et al., 2000), but vehicle-killed deer from several large mule deer population data analysis units (DAUs) statewide also were sampled. The analyses reported here focused on data from DAUs 4, 7, 9, 10, 17, and 27 (Fig. 1), where numbers of vehicle-killed and other surveillance samples were adequate and CWD had been previously detected. Because CWD prevalence shows considerable spatial heterogeneity (Miller et al., 2000; Conner and Miller, 2004; Miller and Conner, 2005), we used only samples where specific universal transverse mercator (UTM) coordinates describing the geographic location of the collision site had been recorded.

For comparison, we initially used data from ≥1-yr-old mule deer randomly sampled in conjunction with other surveillance, management, and research programs to estimate local CWD prevalence in the vicinity of sampled deer-vehicle collision sites (hereafter referred to as "vicinity-sampled"); we included only samples with corresponding UTM data in analyses. Sources of tissue samples included mule deer killed by hunters from September 1996 to December 2003 (Miller et al., 2000; Miller and Conner, 2005; Colorado Division of Wildlife, unpubl. data), apparently healthy mule deer culled by wildlife managers from December 2001 to December 2003 (Colorado Division of Wildlife, unpubl. data), and mule deer captured and tonsil biopsied from March 2001 to December 2003 (Wolfe et al., 2002, 2004; L. L. Wolfe and M. W. Miller, unpubl. data).

For both vehicle-killed and vicinity-sampled deer, diagnostic methods were as described elsewhere (Miller et al., 2000; Miller and Williams, 2002; Wolfe et al., 2002; Hibler et al., 2003). Sampled mule deer were classified as CWD-positive (=infected) or -negative (=uninfected) on the basis of immunohistochemical (IHC) exam of retropharyngeal lymph node or tonsil tissue (Miller and Williams, 2002). Where appropriate tissue was available, histopathology was performed on brain tissue to determine the presence of spongiform encephalopathy (Williams and Young, 1980, 1993) as an index of disease progression (Miller and Williams, 2002; Spraker et al., 2002). We regarded CWD-positive deer with evidence of spongiform encephalopathy as being in relatively late stages of disease, as compared with positive deer without such lesions (Miller and Williams, 2002). Because IHC and histopathology methods and their interpretation did not change materially over the period when samples were collected, and because most samples (92% of vehicle-kill and 77% of vicinity) were collected from 2001 to 2004, we assumed the foregoing diagnostic criteria to be constant for analysis purposes.

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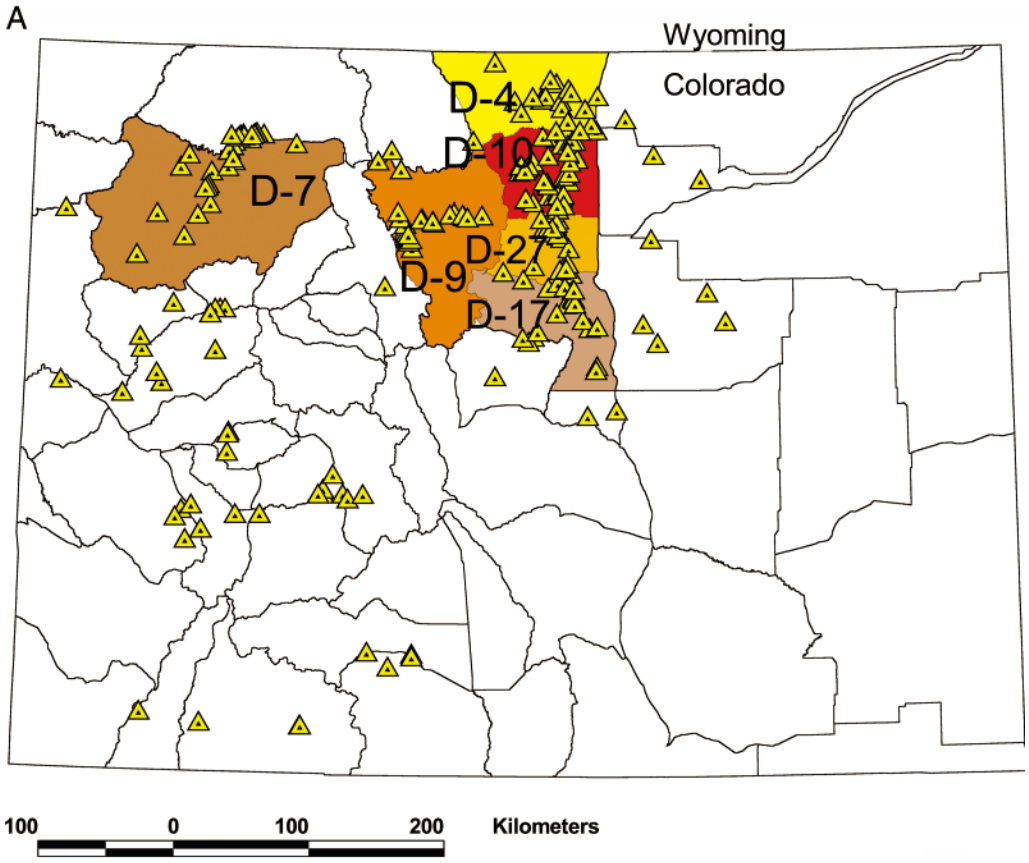
FIGURE 1. Vehicle-killed mule deer sampled in Colorado, USA, for chronic wasting disease (CWD) surveillance. A. Distribution of all vehicle-killed mule deer sampled. Data analysis units (DAUs) used in analyses examining relative vulnerability of CWD-infected animals to collisions (Table 1) are shaded and labeled; yellow triangles are locations of vehicle-killed mule deer. B. Distribution of vehicle-killed mule deer (yellow triangles) in north-central Colorado, USA, illustrating sampling within a 3 km radius (light-blue shading) of collision sites. Red circles in vicinity zones represent vicinity-sampled deer from which prevalence estimates were obtained. Data from the DAU D-4 mule deer population and the Estes Park mule deer population unit residing in the west-central portion of DAU D-10 (Conner and Miller, 2004) and data from DAU D-7 also were used in Table 2.


A
Wyoming
Colorado
D-4
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ESTES PARK
N
Vehicle-kills
Vicinity-kills
DAU boundaries
3km buffer
30 0 30 60 Kilometers

To assess differential vulnerability to vehicle collisions, we compared CWD prevalence among vehicle-killed deer with prevalence among deer sampled in the vicinity of these vehicle-killed deer. Because data from symptomatic animals are highly biased (Miller et al., 2000), we initially excluded emaciated deer and deer showing other signs of CWD from both the vehicle-kill and vicinity data sets before analysis. Then, because previous analyses revealed demographic and spatial influences on CWD prevalence (Miller and Conner, 2005), we used model selection (Burnham and Anderson, 2002) to select an appropriate model representing prevalence patterns in vicinity sample and vehicle-kill data as a basis for making comparisons of vehicle-killed and vicinity prevalence. We used Akaike's Information Criterion (AIC; Akaike, 1973) corrected for small sample bias (AICc) to rank models and select an appropriate "best-approximating" model for these data (Burnham and Anderson, 2002). Explanatory models included age (1 vs. ≥2 yr old), sex, and DAU in various combinations representing potential demographic (sex, age) and spatial (DAU) influences on prevalence. The model (sex+age+DAU) was best by 10 and 1 ΔAIC units for vicinity and vehicle-killed samples, respectively; consequently, we used only data from adult (≥2 yr old) mule deer to minimize age effects on estimated prevalence and made separate comparisons for males and females in each sampled DAU. Because sample distribution was similar across seasons (83% of vehicle kills and 86% of vicinity samples were collected during fall and winter), we assumed no seasonal basis for potential differences between vehicle-kill and vicinity data.

Within the bounds of the foregoing criteria, we used all adult (≥2-yr-old) vehicle-killed mule deer sampled, provided that at least one vicinity sample had been collected within a 3 km radius of the vehicle-killed deer. We used a 3-km radius because a previous study of mule deer winter home range movements showed that 96% of movements made by radiocollared deer were ≤6 km during core winter months when there was no migration (Conner and Miller, 2004). On the basis of these criteria, data from 171 vehicle-killed mule deer (90 males and 81 females) were included in our analyses. For comparison, we used data from 2,317 deer (1,204 males and 1,113 females) sampled within a 3-km radius of vehicle-killed deer. We estimated CWD prevalence among vehicle-killed deer and vicinity-sampled deer as a proportion (CWD-positive samples/total samples) and calculated the relative risk and 95% confidence interval (CI) (Agresti, 1996) for each DAU×sex comparison.

We also calculated the relative risk and 95% CI for occurrence of spongiform encephalopathy among 20 CWD-positive deer killed by vehicles, as compared with 180 CWD-positive, randomly sampled deer where appropriate tissue samples were available for histopathology; case selection criteria were as previously described, except that the set of positive adult deer used in this comparison included but was not limited to those sampled in the vicinity of collision sites. We estimated rates of spongiform encephalopathy among vehicle-killed deer and other positive deer as a proportion (cases with lesions/total cases) and calculated the relative risk and 95% CI (Agresti, 1996).

## RESULTS

Twenty-five of the 171 vehicle-killed mule deer tested positive for CWD (overall prevalence=0.146, 95% CI=0.097–0.208); among the 2,317 deer sampled in the vicinity of these vehicle-killed deer, 173 tested positive (overall prevalence=0.075, 95% CI=0.064–0.085). Where estimable, the relative risk of CWD infection (Table 1) tended to be higher among vehicle-killed deer of both sexes in sampled DAUs, with one exception (male deer in DAU D-4); however, in all but three of the DAU×sex comparisons, the 95% CI for estimated relative risk included 1 (Table 1).

Spongiform encephalopathy was detected in 12 of 20 (60%; 95% CI=39–81%) CWD-positive deer killed by vehicles and in 79 of 180 (44%; 95% CI=37–52%) CWD-positive deer detected via random sampling (relative risk=1.37; 95% CI=0.92–2.03).

## DISCUSSION

Mule deer infected with CWD appear to be more vulnerable to vehicle collisions than otherwise healthy mule deer in sympatric populations. Relatively high CWD prevalence among vehicle-killed deer, as compared with prevalence in the vicinity of collision sites, provides compelling evidence of increased vulnerability, although our small sample sizes and, consequently, the wide confidence intervals on estimates of relative risk (Table 1) precluded unequivocal demonstration of such trends.

TABLE 1. Estimated prevalence (95% confidence intervals [CI]) of chronic wasting disease (CWD), by data analysis unit (DAU) and sex, among vehicle-killed adult mule deer and among adult mule deer sampled in the vicinity of vehicle collision sites from the respective source populations, and estimated relative risk (95% CI) of CWD infection among vehicle-killed deer. (See Fig. 1 for geographic reference and distribution of sampled collision sites within respective DAUs.)

| | | Vehicle-killed deer sampled | | Deer sampled in the vicinity of collision sites | | |
|---|---|---|---|---|---|---|
| DAU | Sex[a] | $n$[b] | Prevalence (95% CI) | $n$[c] | Prevalence (95% CI) | Relative risk (95% CI) |
| D–4 | F | 13 | 0.308 (0.091–0.613) | 420 | 0.069 (0.047–0.098) | 4.5 (1.8–10.8) |
| | M | 10 | 0.10 (0.003–0.443) | 341 | 0.126 (0.093–0.166) | 0.8 (0.1–5.2) |
| D–10 | F | 23 | 0.091 (0.011–0.279) | 506 | 0.057 (0.039–0.081) | 1.6 (0.4–6.1) |
| | M | 31 | 0.290 (0.143–0.479) | 478 | 0.134 (0.105–0.168) | 2.2 (1.2–3.9) |
| D–17 | F | 5 | 0.20 (0.005–0.719) | 18 | 0.056 (0.001–0.275) | 3.6 (0.03–47.3) |
| | M | 18 | 0.167 (0.036–0.413) | 43 | 0.047 (0.006–0.159) | 3.6 (0.7–19.4) |
| D–27 | F | 7 | 0.143 (0.004–0.579) | 107 | 0.009 (<0.001–0.051) | 15.9 (1.1–234.5) |
| | M | 14 | 0.071 (0.002–0.337) | 104 | 0.01 (<0.001–0.052) | 7.1 (0.5–104.8) |
| D–7 | F | 15 | 0.067 (0.002–0.320) | 34 | 0.029 (<0.001–0.153) | 2.3 (0.2–34.8) |
| | M | 10 | 0.10 (0.003–0.443) | 165 | 0.012 (0.001–0.043) | 8.3 (0.8–84.7) |
| D–9 | F | 18 | 0.056 (0.001–0.275) | 28 | 0 (0–0.123) | ne[d] |
| | M | 7 | 0 (0–0.410) | 73 | 0 (0–0.049) | ne[d] |

[a] F = female; M = male.
[b] Sample size used to estimate CWD prevalence among vehicle-killed deer within a DAU.
[c] Sample size used to estimate CWD prevalence in the vicinity of collision sites within a DAU.
[d] ne = relative risk not estimable where denominator equals 0.

The relatively high rate of spongiform encephalopathy among CWD-infected mule deer killed by vehicles is also consistent with disease-related influences on vulnerability to collisions. Altered behaviors (Williams and Young, 1980; Williams and Miller, 2002) that could render deer more vulnerable to collisions become more apparent through the disease course as damage to brain tissue accumulates. Although deer behavior cannot be assessed postmortem, progression of CWD can be staged, at least broadly, through immunohistochemistry and histopathology (Miller et al., 2000; Miller and Williams, 2002; Spraker et al. 2002). Accumulations of CWD-associated abnormal prion protein ($PrP^{CWD}$) are detectable in lymphoid tissue months before detection in brain tissue (Sigurdson et al., 2002; Williams and Miller, 2002) and well in advance of the development of clinical signs of CWD (Wild et al., 2002; Williams and Miller, 2002). In contrast, observations from an experimental study of CWD pathogenesis in orally inoculated mule deer (Williams and Miller, 2002; E. S. Williams and M. W. Miller, unpubl. data) suggested that the appearance of spongiform encephalopathy in sacrificed deer coincided roughly with the onset of subtle clinical signs ≥15 mo after inoculation in surviving individuals. It follows that a somewhat higher proportion of the vehicle-killed deer in our sample may have been suffering from some degree of clinical CWD and thus more vulnerable to collisions.

If CWD-infected deer are more susceptible to death by vehicle collision, as suggested by our data, then it seems logical that infected deer may also be more vulnerable to other forms of mortality such as predation. The knowledge that CWD-infected deer may be more vulnerable to vehicle collisions needs to be considered in studying selective predation, and any evidence of "selection" needs to be interpreted in the context of likely increased vulnerability. Alternatively, because increased vulnerability may foster selective predation, our findings lend some support to the notion that large coursing predators like wolves (*Canis lupus*) could be effective at removing a greater proportion of

TABLE 2. Comparison of estimated sampling effort needed to detect ≥1 case of chronic wasting disease (CWD) with ≥99% detection probability using random sampling of harvested adult mule deer or sampling of vehicle-killed deer. Required sample sizes for detection at three geographic scales were estimated using hypergeometric probability distributions and data from the Estes Park Valley (EPV), data analysis unit (DAU) D–4 populations in north-central Colorado (Fig. 1) and DAU D–7 in northwestern Colorado, representing small, intermediate, and large deer herds and geographic scales, respectively.

| | Random sampling from harvest | | | Sampling from vehicle-killed subpopulation | | | Reduction in sampling effort[e] |
|---|---|---|---|---|---|---|---|
| Population | Population size[a] | CWD prevalence[b] | Required sample[c] | Population size[d] | CWD prevalence | Required sample | |
| EPV | | | | | | | |
| Females | 298 | 0.045 | 81 | 7–22 | 0.25 | 6–11 | 0.86–0.93 |
| Males | 114 | 0.129 | 28 | 3–9 | 0.25 | 3–8 | 0.71–0.89 |
| DAU D–4 | | | | | | | |
| Females | 4,420 | 0.048 | 92 | 99–332 | 0.308 | 13 | 0.86 |
| Males | 2,080 | 0.077 | 56 | 47–156 | 0.10 | 27-37 | 0.34–0.52 |
| DAU D–7 | | | | | | | |
| Females | 47,454 | 0.003 | 1,512 | 1,068-3,559 | 0.067 | 65 | 0.96 |
| Males | 13,762 | 0.011 | 412 | 310–1,032 | 0.10 | 42 | 0.90 |

[a] Population size, estimated from annual inventory, and population modeling (Colorado Division of Wildlife, unpubl. data).
[b] Prevalence = number positive/total sample (Colorado Division of Wildlife, unpubl. data).
[c] Number of samples required to assure ≥99% probability of detecting ≥1 CWD case, given the population size and prevalence estimated, as derived from a hypergeometric probability distribution.
[d] Estimated number of vehicle-killed deer in the population, based on estimated population size, and assuming the mean natural adult (≥1 year old) survival rates = 0.85 (Unsworth et al., 1999) and that 15–50% of "natural" (i.e., nonhunting) adult mortalities were caused by vehicle collisions.
[e] Reduction in sampling effort = 1 − (required number of vehicle-kill samples/required number of random samples).

infected deer from an affected population than random removal via harvest.

Previous studies have suggested that bias may occur in CWD prevalence data from surveillance of hunter-killed deer (Conner et al., 2000) and from "targeted" surveillance (Miller et al., 2000); in both cases, it appeared that bias may have been due to behavioral differences between infected and uninfected deer. On the basis of our findings, abnormal behavior or compromised alertness of infected individuals also may affect the likelihood of being killed by a vehicle collision. Consequently, the potential for bias should be considered before using data from vehicle-killed deer in estimating CWD prevalence. Because original epidemiological studies of CWD in free-ranging mule deer (Miller et al., 2000) assumed that vehicle-killed and harvested animals had equal probabilities of being infected with CWD, some early prevalence estimates may have been biased high relative to true prevalence in affected deer populations. In light of our findings, it seems possible that biases also could arise in data from surveys based on hunting with "primitive weapons" (e.g., long bow, muzzle-loaded rifle), and this potential bias may merit further investigation.

Although problematic in studying CWD epidemiology, the tendency toward bias in prevalence among vehicle-killed deer (Table 1) could be exploited in designing more cost-effective surveys to detect new foci of CWD in free-ranging populations (Table 2). Our data show that the CWD detection probability per animal sampled tends to be higher than for other, more random methods of surveillance such as those based on sampling harvested deer. Because vehicle-killed deer may have a greater probability of being infected with CWD than the population as a whole, fewer samples would be needed to detect at least one CWD-positive deer in an affected target population. In general, surveil-

lance targeting a smaller population subset with relatively high CWD prevalence may be a more efficient detection strategy than sampling the entire population (Miller et al., 2000; Samuel et al., 2003). The efficiency gained through surveillance focused on vehicle-killed deer depends on the ratio of the proportion of the CWD-infected population that is vehicle-killed to the proportion of the entire population that is vehicle-killed (Samuel et al., 2003).

To illustrate the relative efficiency that could be gained by sampling vehicle-killed deer to detect CWD foci, we used the hypergeometric probability distribution (Cochran, 1977) to compute sample sizes needed to detect at least one CWD-infected mule deer, with a ≥99% detection probability in target populations of differing sizes, given underlying CWD prevalence at various levels. For illustration, we used field data from three populations: Estes Park Valley, a relatively small mule deer population residing in and around the town of Estes Park, Colorado; DAU D-4, an intermediate-sized mule deer population residing in northern Larimer County, Colorado (Fig. 1A, B); and DAU D-7, a large mule deer population with relatively low CWD prevalence in northwestern Colorado (Fig. 1A). In all of the example cases we examined, sampling vehicle-killed deer appeared to offer the more efficient approach for detecting cases (Table 2).

Our findings suggest that by sampling vehicle-killed deer, fewer samples would be required in most cases to detect at least one CWD-infected animal than if sampling relied on harvested deer. It follows that the efficiency of surveillance programs with a goal of detecting new CWD foci might be improved by emphasizing approaches that include vehicle-killed deer sampled opportunistically, provided that the size of the vehicle-killed subpopulation is estimable and that such sampling is geographically representative of target populations. Under such approaches, managers must recognize that the assumption of random sampling (equal probability of being sampled between hunter-harvested animals and vehicle-killed animals) is not valid. In addition to improving surveillance efficiency, surveying vehicle-killed deer may be an effective way to identify local areas of relatively high prevalence that could be targeted for management interventions in the face of marked heterogeneity in the geographic distribution of CWD in affected mule deer populations (Wolfe et al., 2002; Conner and Miller, 2004). Although surveying vehicle-killed deer may not yield as many samples as could be obtained via harvest surveys, the former offers year-round surveillance as compared with approaches restricted to relatively brief hunting seasons. Vehicle-kill surveillance could be particularly effective in areas where hunting is limited because of urban development or other constraints. Understanding biases associated with this and other CWD surveillance methods (Conner et al., 2000; Miller et al., 2000; Samuel et al. 2003) should allow managers to use the most efficient and effective combination of techniques to achieve jurisdiction-specific surveillance goals.

The epidemiology of CWD is incompletely understood. However, it is clear that the agent can persist in the environment, and that even decomposed carcasses can be sources of infection (Miller et al., 2004). Therefore, transport and disposal of infected vehicle-killed deer and elk carcasses should be carefully considered to minimize opportunities for spreading CWD. Furthermore, sites where CWD-infected deer have been killed by vehicles but remained in the environment should be monitored as possible areas of emergent CWD foci.

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