

Chronic Wasting Disease and the Greater Yellowstone Area

Prepared for
Greater Yellowstone Coalition, Inc.

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INTRODUCTION

The objective of this review is to provide a concise summary of chronic wasting disease (CWD) in cervids (deer family) that is directly relevant to natural resource managers, policy makers, and conservationists with the hope that this information may lead to better environmental policy. I address this objective in two steps. I first review CWD by discussing the emergence of the disease, its host range, characteristics of the disease in cervids, ecological relationships, and management options. I do not address CWD pathogenesis, histopathology, strain typing, or molecular features of the disease in any detail due to the purpose of this review. Those interested in these aspects of CWD and other transmissible spongiform encephalopathies (TSEs) should see recent reviews by Silveira et al. (2004) and Williams (2005) for details. Second, I address how CWD might become established in the Greater Yellowstone Area (GYA), probable consequences of CWD occurring in the GYA, and options for avoiding this eventuality.

AUTHOR BACKGROUND

Markus J. Peterson is an Associate Professor in the Department of Wildlife and Fisheries Sciences at Texas A&M University. His research interests include the ecology and management of terrestrial vertebrate populations. This research emphasizes factors influencing animal abundance, such as wildlife diseases, predation, weather, habitat conditions, and human exploitation. He also has a keen interest in environmental policy formation and implementation. Markus has published the results of his research on wildlife disease ecology and management in scholarly outlets including the *Birds of North America*, *Ecography*, *Ecological Modelling*, *Journal of the American Veterinary Medical Association*, *Journal of Wildlife Diseases*, *The Journal of Wildlife Management*, and the *Wildlife Society Bulletin* as well as in book chapters, technical reports, and other outlets. Additionally, he has served on external review panels tasked with evaluating CWD and other wildlife disease management programs formulated by both state and federal natural resource management agencies. Markus was raised in Teton counties Idaho and Wyoming and received his B.S. from University of Idaho (1976), D.V.M. from Washington State University (1980), and both M.S. (1990) and Ph.D. (1994) degrees from Texas A&M University. Prior to his current position, he served as Upland Wildlife Program Leader for the Texas Parks and Wildlife Department. For more detail, see attached curriculum vitae.

CWD AND OTHER TSEs

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (TSE). The TSEs are a group of neurologic diseases of mammals that includes scrapie in domestic sheep and goats, CWD in cervids (the deer family), transmissible mink encephalopathy in farmed mink, bovine spongiform encephalopathy (BSE) in cattle, and kuru, Gerstmann-Sträussler-Scheinker syndrome, and Creutzfeldt-Jacob disease (CJD) and all its variants in humans (Collinge 2001, Williams et al. 2001). It now is clear that BSE is associated with a variant of CJD (vCJD) in humans (Will et al. 1996, Lacey 1998, Collinge 2001).

The TSEs are referred to as “prion” diseases because of their association with aberrantly refolded isoforms of the normal prion-related protein or PrP^c (Prusiner 1982, 1991; Williams 2005). The bulk of evidence suggests that abnormal protease-resistant forms (PrP^{res}) of PrP^c in the central nervous system (CNS) and lymphoid tissues cause TSEs (Collinge 2001, Williams et al. 2001). The PrP^{res} proteins arise from post-translational modifications of the three-dimensional structure of PrP^c. Once PrP^{res} enters the host, it catalyses the production of species-specific PrP^{res} in the CNS and lymphoid tissues. The exact biochemical mechanism associated with this change remains unknown. In scrapie, at least, there also is a genetic predilection for this change in some individuals (Hunter et al. 1992, O’Rourke et al. 1997). The PrP^{res} spreads from animal to animal in a manner analogous to that of a microparasite (e.g., pathogenic

viruses or bacteria), and TSEs behave like chronic microparasitic diseases. Because the causative agents are devoid of nucleic acids (i.e., non-living substances), however, TSEs probably should be thought of as a unique class of toxicoses. In fact, PrP^{res} are remarkably resistant to environmental degradation, rendering disposal of carcasses and other contaminated materials difficult. Commonly used procedures, such as cooking, do not destroy the ability of PrP^{res} to cause disease. This requires more costly measures, both in time and other resources, such as incineration or protein digestion.

CHRONIC WASTING DISEASE (CWD)

EMERGENCE

The disease syndrome now known as CWD was first recognized in captive mule deer (*Odocoileus hemionus*) in research facilities in Colorado in 1967 (Williams and Young 1980). In 1978, Williams and Young (1980) determined that CWD was a type of spongiform encephalopathy and soon identified the disease in captive mule deer in a Wyoming research facility. Deer had been moved among these locations. Captive elk (*Cervus elaphus*) in these facilities also were diagnosed with CWD (Williams and Young 1982). The disease was recognized in free-roaming elk in north-central Colorado, including Rocky Mountain National Park, in 1981 (Spraker et al. 1997), and soon was documented in free-roaming mule deer (1985) and white-tailed deer (*O. virginianus*; 1990) in both north-central Colorado and southeastern Wyoming (Williams and Miller 2002, Williams et al. 2002b).

It is not possible to determine whether CWD arose in captive or free-ranging cervids, but it seems certain that it originated within the Colorado–Wyoming endemic area (Williams et al. 2002a). How the deleterious prion associated with CWD (PrP^{CWD}) arose is unknown. It is possible that it resulted from (1) a spontaneous conformational change from PrP^C to PrP^{CWD}, with subsequent transmission throughout the susceptible portions of local deer and elk populations, (2) a strain of the scrapie prion (PrP^{Scrapie}) became adapted to cervids, or (3) it originated from a so-far-unidentified prion strain (Williams and Miller 2002, Williams et al. 2002a). At any rate, PrP^{CWD} is quite different from the prion strains associated with BSE and transmissible mink encephalopathy, but more similar to certain PrP^{Scrapie} strains.

Despite warnings from wildlife disease experts concerned about dissemination of PrP^{CWD} via movement of farmed cervids (Williams and Young 1992), CWD was diagnosed in farmed elk in Saskatchewan, Canada, in 1996 (Williams et al. 2002a, Miller and Williams 2004). It now has been documented in farmed elk herds in Colorado, Kansas, Minnesota, Montana, Nebraska, Oklahoma, South Dakota, Wisconsin, and Alberta, Canada, as well as South Korea. More recently, CWD was documented in captive white-tailed deer in Alberta, New York, and Wisconsin. Since 2000, CWD spread from CWD-infected elk farms to free-roaming cervids in west-central Saskatchewan, northwestern Nebraska, and southwestern South Dakota. Only 1 of 105 (0.95%) free-roaming white-tailed deer sampled in Black Hills of South Dakota (1998–1999) was positive for PrP^{CWD} (Jacques et al. 2003). No positive mule deer ($n = 204$) or elk ($n = 452$) were found in this region. Similarly, it appears that CWD spread from captive to free-roaming white-tailed deer in central New York. The origins of CWD in free-roaming elk and mule deer in northwestern Colorado, white-tailed deer in south-central Wisconsin, north-central Illinois, and eastern West Virginia, and mule deer in southern New Mexico, eastern and central Utah, and the Bighorn Basin of Wyoming are still under investigation. In the case of northwestern Colorado, however, the index cases occurred in free-ranging deer entrapped within an elk farm.

Given the appearance of CWD in both farmed and free-roaming cervids across North America, CWD diagnosis, ecology/epizootiology, and management are now a major concern for wild and captive cervid populations for obvious reasons. Additionally, the spread of CWD raised the public health concern that a human TSE might somehow result from PrP^{CWD} as occurred with vCJD and BSE. For these reasons, excellent reviews targeting different audiences now are available (Williams et al. 2001, 2002a,b; Williams and Miller 2002; Miller and Williams 2004; Williams 2005). Older reviews are less valuable due to the rapid pace of TSE research. Wildlife ecologists and managers should find Williams et al. (2002a) and Miller and Williams (2004) particularly useful.

HOST RANGE

Mule deer, white-tailed deer, and elk were long considered to be the only species naturally susceptible to CWD (Williams et al. 2002a, Williams 2005). Black-tailed deer (*O. h. columbianus*), a subspecies of mule deer, also are susceptible (Williams and Young 1980), and it seems certain that all subspecies of white-tailed deer, mule deer, and elk (includes red deer; *C. e. elaphus*) are susceptible to CWD (Miller and Williams 2004, Williams 2005). Moreover, a moose (*Alces alces*) contracted CWD when given an oral inoculation containing PrP^{CWD} (Williams 2005), and in September, 2005, a free-roaming bull moose taken by a hunter in Colorado tested positive for CWD [Colorado Division of Wildlife (CDW) 2005b). Thus it is likely that all cervid species are susceptible in varying degrees to PrP^{CWD}.

Other North American wild ruminants including pronghorn (*Antilocapra americana*), bighorn sheep (*Ovis canadensis*), and mountain goats (*Oreamnos americanus*) either are resistant, or at least much less susceptible, to CWD. Individuals of these species, as well as domestic cattle, sheep, and goats have been held in PrP^{CWD}-contaminated research facilities in contact with CWD-affected deer and elk for prolonged periods without developing the disease (Williams et al. 2002a, Miller and Williams 2004). Some domestic goats, sheep, and cattle are susceptible to CWD experimentally when exposed via intracerebral inoculation (Williams and Young 1992; Hamir et al. 2001, 2005; Williams 2005). While this route of exposure is useful for experimental studies, it has little bearing on the epizootiology of CWD in nature.

To date, cases of human CJD or its variants have not been tied to CWD even when they occur in unusually young patients who consumed venison (Belay 2001, Williams et al. 2002a). Raymond et al. (2000) demonstrated a molecular barrier that would limit susceptibility of humans, cattle, and sheep to PrP^{CWD}. Similarly, Kong et al. (2005), utilizing transgenic mice, demonstrated a substantial species barrier for transmission of elk CWD to humans. Thus it is likely that humans and cattle are no more susceptible to PrP^{CWD} than to PrP^{Scrapie}. No cases of human TSEs have been tied to scrapie since this disease was first described approximately 200 years ago. This certainly does not mean, however, that we can rule out transmission of some PrP^{CWD} variant to humans.

DISEASE

As the name “chronic wasting disease” implies, deer and elk with end-stage CWD exhibit loss of body condition (Williams et al. 2001, 2002a,b). Changes in behavior, such as repetitive actions, somnolence, depression, and carrying head and ears lowered also are seen. Specific clinical signs are probably a function of exactly where brain lesions occur. The mean incubation period of experimental CWD caused by oral exposure is about 23 months (range = 15 to > 25) in mule deer, while the incubation period is 12–34 months in elk (Williams et al. 2002a). In naturally occurring cases, the youngest animal diagnosed with clinical CWD was 17 months of age, although apparently healthy white-tailed deer fawns as young as 5–6 months have tested positive for PrP^{CWD} in Wisconsin. The mean incubation period probably ranges from 2 to 4 years in naturally occurring cases (Williams 2005). Most cases of CWD in PrP^{CWD}-contaminated research facilities occurred in animals 2–7 years of age (Williams et al. 2002a). Once signs of CWD appear in captive cervids, sick animals live from a few days to a year, with most surviving from a few weeks to 3–4 months. The clinical course in free-roaming deer and elk probably is somewhat shorter. Death is inevitable once clinical signs develop. There is no evidence that deer or elk produce antibody to PrP^{CWD}.

There is a genetic component to susceptibility and development of scrapie in sheep (Hunter et al. 1992, O’Rourke et al. 1997). It is not yet clear whether a genetic predilection to CWD in certain deer or elk occurs, but the potential for differential susceptibility exists at in elk (O’Rourke et al. 1999), white-tailed deer (O’Rourke et al. 2004), and mule deer (Jewell et al. 2005). If these observations are confirmed and expanded, genetic manipulation of farmed elk and deer herds might be useful in managing CWD (Williams 2005). It is difficult, however, to see how this information would markedly change the way CWD is managed in free-roaming cervid populations.

ECOLOGY/EPIZOOTIOLOGY

Transmission

Although CWD is both infectious and transmissible, many details regarding natural transmission have yet to be worked out. Unlike BSE, CWD is not a food-born disease associated with rendered ruminant tissues. Studies of CWD epizootics in captive deer and elk strongly support lateral transmission, as is the case with scrapie in sheep, but not BSE (Williams and Young 1992, Miller et al. 1998, Miller and Williams 2003). Both experimental and ecological modeling studies support lateral transmission as well (Miller et al. 2000, Gross and Miller 2001, Miller and Williams 2003). Maternal transmission cannot be ruled out, but must be rare if it occurs at all, and cannot explain most cases where data are sufficient for evaluation (Miller et al. 1998, 2000). Interspecies transmission among mule deer, white-tailed deer, and elk also has been documented.

Exactly how PrP^{CWD} is shed from affected individuals remains unclear. It is known, however, that PrP^{CWD} accumulates in gut-associated lymphoid tissues such as the tonsils, Peyer's patches, and mesenteric lymph nodes (Sigurdson et al. 1999, Miller and Williams 2002, Spraker et al. 2002). This suggests that PrP^{CWD} probably is shed through the alimentary tract in the feces and/or saliva. A number of CWD epizootics have been attributed to contaminated pastures (Miller et al. 1998, Williams et al. 2002a). Miller et al. (2004) demonstrated experimentally that CWD can be transmitted to susceptible animals indirectly by way of environmental contamination with excreta or decomposed carcasses. They found that mule deer became infected in two of three paddocks containing naturally infected deer. Mule deer also contracted the disease in two of three paddocks where CWD-infected deer carcasses had decomposed in place for about 1.8 years and in one of three paddocks where infected deer had resided 2.2 years previously. No mule deer became infected with CWD in their control paddocks where the disease had never occurred. This study clearly demonstrates that CWD can be transmitted via environmental contamination as well as directly among animals, and that contaminated materials can remain infectious for prolonged periods.

Regardless of transmission route, susceptible individuals are exposed orally. In mule deer at least, the presence of PrP^{CWD} in gut-associated lymphoid tissues early in the incubation period (Sigurdson et al. 1999) suggests that shedding may begin early in the course of the disease. Moreover, it is likely that PrP^{CWD} shedding is progressive (Williams et al. 2002a). For all these reasons, it appears certain that PrP^{CWD} transmission, whether direct, indirect, or both, is dependent to some degree on the density of susceptible hosts.

Prevalence

In situations where high cervid densities are maintained by humans, CWD eventually spreads throughout most of the population. For example, in captive deer herds CWD prevalence can exceed 90% (Williams and Young 1980). Similarly, prevalence demonstrated by PrP^{CWD} immunohistochemistry (IHC) was 52.4% ($n = 88$ of 168) for white-tailed deer inadvertently confined with an affected elk farm in Nebraska (Nebraska Game and Parks Commission 2002; also see O'Rourke et al. 2004). For captive elk, Miller et al. (1998) reported that 4 of 23 (17.4%) animals in a research herd died of CWD, Peters et al. (2000) found 10 of 17 (58.8%) farmed elk positive by IHC at slaughter, and Williams et al. (2002a) found 11 of 54 (20.4%) elk in a research herd positive by IHC. Clearly, when deer or elk are held at high densities in either deer farms or research facilities for a sufficient period, high CWD prevalence should be expected.

Although several regulatory agencies currently are testing free-roaming cervids for PrP^{CWD} by IHC, results for only a few extensive surveys have been published. All of these surveys, however, indicate that foci of high CWD prevalence tend to occur within a matrix of lower prevalence. For instance, soon after CWD was noted in Wisconsin, prevalence was 3% ($n = 476$) for the entire surveillance area, but 9.4% for 127 samples taken near where the index cases were found (Joly et al. 2003). Miller et al. (2000) tested a stratified random sample of 4,108 deer and elk collected from 29 wildlife management units within the CWD-endemic region of southeastern Wyoming and northeastern Colorado (38,137 km²).

between 1996 and 1999 to determine prevalence of PrP^{CWD}. Estimated overall prevalence was 4.9 ($n = 2,836$), 2.1 ($n = 343$), and 0.5% ($n = 929$) for mule deer, white-tailed deer, and elk, respectively. At finer spatial scales, however, estimated prevalence ranged from 0–14.7, 0–15.4, and 0–0.9% for mule deer, white-tailed deer, and elk, respectively. Stated differently, Miller et al. (2000) found marked spatial heterogeneity in CWD prevalence within study regions of $>38,000 \text{ km}^2$. Similar spatial heterogeneity in CWD prevalence also was documented by Wolfe et al. (2002) at much finer spatial scales ($\leq 20 \text{ km}^2$) in north-central Colorado. Conner and Miller (2004) and Miller and Conner (2005) provide further support for spatial heterogeneity in CWD prevalence among wintering mule deer subpopulations in north-central Colorado. Williams (2005) also reported foci of CWD prevalence as high as 30% in local deer populations in Colorado and Wyoming. Clearly, foci of high CWD prevalence within a matrix of lower prevalence are not just characteristic of initial outbreaks, but also are features of long-standing epizootics.

Influence on Cervid Population Dynamics

Miller et al. (2000:676) suggested that, even though CWD probably has occurred in northeastern Colorado and southeastern Wyoming for >30 years, it “may be best represented as an epizootic with a protracted time-scale.” A “slow epizootic” sounds oxymoronic. What the authors meant was that such a disease would gradually rather than rapidly expand its distribution by invading new populations, slowly rather than quickly become more prevalent where it occurs, and after many decades rather than many weeks or months contribute to declining animal abundance. Chronic wasting disease has increased its distribution remarkably across North America (see Emergence, above). Although CWD prevalence is much higher in some portions of the endemic area of Colorado than others (Miller et al. 2000), prevalence has increased in all of these regions over time (Miller and Conner 2005), which also supports the contention that CWD is an epizootic with a protracted time-scale.

Moreover, simulated CWD epizootics in mule deer failed to achieve equilibrium, suggesting that local extinctions of affected deer populations might eventually occur (Gross and Miller 2001). So far, data do not suggest that mule deer populations in this region have been imploding since CWD was first recognized in free-roaming elk in 1981. Thus, either the model used by Gross and Miller (2001) requires modification as Schaubert and Woolf (2003) maintained, or predicted declines may yet occur. At any rate, the existence of foci characterized by high CWD prevalence within a matrix of much lower prevalence, increasing overall prevalence, and the potential for local extinction presents wildlife managers with both an urgent problem and a substantial challenge. The first step toward addressing this problem and meeting the challenge is to develop a better understanding of why the marked spatial heterogeneity in CWD prevalence occurs.

Cervid Movements, Human Development, and CWD Prevalence Patterns

If agencies hope to contain the spread of CWD, it is important to understand cervid movement patterns and reasons for foci of high prevalence within endemic areas. Recent studies of free-roaming cervids illustrate that animal density is a clear contributor to high CWD prevalence in localized areas. Connor and Miller (2004), who conducted a spatially explicit analysis within the CWD endemic area in north-central Colorado, found that migrating mule deer were much more likely to spread CWD among populations than were dispersing deer. Farnsworth et al. (2005) found that CWD prevalence in mule deer was almost twice as high in developed as compared to undeveloped areas in north-central Colorado due to land-use practices that fostered congregation and/or sedentary behavior in urban mule deer. Specifically, they suggested that artificial feeding, ornamental vegetation, decreased predation, and decreased human harvest associated with urbanization, as well as deer congregation in pockets of remaining habitat left behind after development, probably lead to increased CWD transmission. For these reasons, using anthropogenic boundaries (e.g., county lines, game management units based on highways), rather than cervid population units, could obscure the role of cervid movement in the spatial epizootiology of CWD.

Recent research also indicates that deer gender plays a significant role in CWD transmission and prevalence. Farnsworth et al. (2005) found that CWD prevalence was almost twice as high for adult male as compared to adult female mule deer on three study areas in north-central Colorado. This pattern also

was observed at broader spatial scales throughout the region and likely occurs with elk and white-tailed deer as well (Miller and Conner 2005). Further, prevalence in males increased with age and then declined across age classes, peaking at 5–6 years of age. It is logical to assume that gender-specific social behavior accounts for these results because captive male and female mule deer appear equally susceptible to infection (Williams and Young 1980, 1992; Williams and Miller 2002; Miller and Wild 2004; Miller et al. 2004). Thus, adult male mule deer probably have a greater role in CWD transmission within a deer population than do infected females (Miller and Conner 2005).

By extension, it also seems likely that the migratory pathways used by adult male mule deer could help explain the expansion of CWD endemic areas in the Rocky Mountains. After all, CWD prevalence in free-roaming mule deer is much higher than typically seen in free-roaming elk, and prevalence is much higher in adult male as compared to adult female mule deer. Further, anthropogenic factors such as development within mule deer winter range, winter feeding, and reduced human and nonhuman predation undoubtedly ensure a ready source of infected males. For these reasons, spatially explicit analyses such as those conducted by Connor and Miller (2004), Farnsworth et al. (2004), and Miller and Connor (2005) should be conducted for mule and white-tailed deer in other CWD endemic areas. If consistent results are found, much better data would be available upon which to ground management strategies designed to limit the spread of CWD.

MANAGEMENT OPTIONS

Biological

Unfortunately, effective strategies for managing TSEs, including CWD, remain elusive. As Miller and Williams (2004:208) summarized:

Long incubation periods, subtle early clinical signs, absence of a practical antemortem diagnostic test, extremely resistant infectious agent, possible environmental contamination, and incomplete understanding of transmission all constrain options for controlling or eradicating CWD. At present, traditional veterinary approaches to therapy and prevention do not apply to CWD and other prion diseases. No treatment is available to prevent infections or recover affected animals; similarly, no vaccine is available to prevent CWD infection in deer or elk.

For these reasons, there are no clear-cut guidelines for managing CWD in free-roaming cervid populations that are grounded in rigorous experimentation.

For deer farms, management options revolve around restricting the importation of cervids to stock originating from farms with no record of CWD and a long history of intensive and extensive testing. If CWD is detected in a deer farm, depopulation is the only alternative. So far, attempts to eradicate CWD from heavily contaminated facilities have failed (Williams and Young 1992, Miller et al. 1998), probably due to environmental contamination. It is too soon to tell whether depopulation of elk farms and zoological gardens where only a few cases of CWD occurred resulted in eradication. For these reasons, captive deer should not be reintroduced, and free-roaming cervids should be excluded, from contaminated premises for the foreseeable future.

Options for managing CWD once it exists in free-roaming cervid populations are practically nonexistent. Thus the emphasis should be placed on preventing PrP^{CWD} from becoming established in naïve cervid populations. State wildlife agencies have attempted to reach this goal by (1) banning translocation of cervids from endemic areas, (2) banning importation of potentially PrP^{CWD} contaminated tissues collected by hunters in endemic areas (e.g., cervid brain, spinal chord, and certain other tissues), (3) culling animals exhibiting clinical signs consistent with CWD, (4) reducing cervid density by outlawing supplemental feeding, (5) reducing cervid density near CWD foci by culling apparently healthy deer in the vicinity, and (6) aggressively reducing deer density near newly identified CWD foci via culling. These options either attempt to directly limit the spread of PrP^{CWD} from endemic areas to regions where it does not occur or to decrease prevalence, and thus the risk of cervids contracting the disease, so that migratory animals are less likely to bring the disease to areas where it does not yet occur. Although many of these approaches are intuitively appealing, most have not been evaluated experimentally.

Sociological

Not only are there few ecological options for controlling CWD in free-roaming populations, there also are numerous social minefields to negotiate when attempting to manage the disease. Heberlein (2004) provides an insightful case history addressing these issues. He found that the announcement of CWD in the Wisconsin deer herd in February 2002 was treated by the Wisconsin Department of Natural Resources (DNR) as if it were a fire. Without the benefit of having a CWD management plan in place, the DNR quickly decided to (1) reduce deer density statewide, (2) eradicate deer in a 1,065 km² area surrounding where the index cases were found, and (3) outlaw recreational feeding in an effort to stamp out CWD. After two years, the DNR had accomplished neither the biological nor the social goals of their plan for managing CWD (Heberlein 2004). In fact, hunters killed fewer, not more deer; deer densities in the eradication zone remained high; and efforts to end recreational feeding failed. Moreover, deer hunting license sales dropped by over \$90,000 due to an 11% drop in hunter numbers and other programs suffered as money was reallocated to fight CWD. Approximately \$12.6 million of the Wisconsin DNR's budget was spent on CWD during fiscal year 2002–2003 alone (Bishop 2004). Hunters were hardest hit, losing approximately \$60 million annually in recreational benefits, or a 20% decline in the annual surplus value of deer hunting in the state. Of those who quit hunting after CWD was discovered, 52% said they did not participate in the 2002 season because of CWD (Vaske et al. 2004). They perceived higher levels of risk associated with CWD than did those who continued deer hunting or those who quit hunting for other reasons. Dropouts also had much less trust for the Wisconsin DNR than did others. Clearly, social aspects of CWD management were at least as important as the ecological dimensions.

Based on what was learned in Wisconsin (Bishop 2004, Heberlein 2004, Vaske et al. 2004) and elsewhere, state wildlife agencies should consider proactively developing formal CWD management plans. The details of the plans would differ in states with no record of CWD versus those where CWD is endemic in part of the state, but should include adaptive management strategies in both cases (Walters 1986, Heberlein 2004). It would be useful to seek input from relevant state and federal agencies and publics before drafting the plan. After completing a formal public comment process, the final management plan would be available should CWD foci occur in portions of CWD-positive states that formerly were free of the disease or in states where the disease had not previously occurred. These plans would provide a carefully thought-out point of departure for the adaptive management of CWD and would help avoid precipitous and ineffective action to cope with CWD (Heberlein 2004).

CWD AND THE GREATER YELLOWSTONE AREA

Between 1996 and 2002, 1,095 hunter-killed elk from the Jackson herd were tested for chronic wasting disease (CWD) either by histopathologic examination of the brain and/or by immunohistochemistry (IHC) of the brain or lymphoid tissues; none were considered positive [D. Clause, Wyoming Game and Fish Department (WGFD), unpublished data]. Thus, as of 2002, it seems likely that CWD did not yet occur in this elk population or, if it did, prevalence was quite low. The WGFD department has not released sufficient data to calculate the probability that the GYA is CWD-free as of 2005.

Feedground elk in the GYA are highly vulnerable to a CWD outbreak. Elk densities associated with the National Elk Refuge and other Greater Yellowstone Area (GYA) feedgrounds are far higher than in free-roaming elk populations in the Colorado–Wyoming endemic area where CWD prevalence is 1–3%. Instead, functional densities are at least as high as observed in captive elk herds where CWD prevalence was 17.4 to 58.8% (see Ecology/Epizootiology—Prevalence, above). These are nearly ideal conditions for CWD transmission among free-roaming elk. Thus, if CWD becomes established in the Jackson elk herd, for example, one should expect a CWD epizootic with a protracted time-scale (see Ecology/Epizootiology—Influence on Cervid Population Dynamics, above).

Based on what is known about CWD in elk, prevalence in a chronically infected feedground herd could exceed 50% if feeding programs remained unchanged and the disease was allowed to take its

course. The WGFD (2005:5) maintained that “this level of prevalence in captive elk, combined with the density of thousands of elk inhabiting feedgrounds for several months, suggests the possibility of much higher prevalence rates in feedground elk, possibly resulting in a concomitant reduction in elk numbers.” Moreover, because the distributions of most GYA elk herds overlap (Craighead et al. 1972, Rudd et al. 1983, Toman et al. 1997, Smith and Anderson 2001), one would expect that unless managers took decisive action soon after CWD was recognized in a feedground herd, PrP^{CWD} would gradually become established on other feedgrounds and eventually would occur at lower prevalences (1–3%) in non-feedground elk throughout the GYA. Clearly, preventing the introduction of CWD into the GYA is far more desirable than having to live with it.

In this section, I first discuss anthropogenic and non-anthropogenic factors that increase the risk of CWD becoming established in the GYA. I then discuss potential ecological and social consequences of CWD in the region. Last, I outline options for avoiding this eventuality. Rather than integrating myriad technical details here, I refer the reader back to relevant sections of the CWD review already presented.

RISK OF CWD ESTABLISHMENT IN THE GYA

NON-ANTHROPOGENIC RISKS

CWD Distribution Near the GYA

Currently, the most likely sources of PrP^{CWD} for the GYA that do not rely on human activity lie to the east or south, because CWD is not known to occur in free-roaming cervids (members of the deer family) in Montana or Idaho. Mule deer positive for CWD have been documented east of the GYA in the Bighorn Basin of Wyoming, including two positive adult males taken just northwest of Thermopolis in 2005 (WGFD 2005b,c). Similarly, numerous positive mule deer have been killed in several game management units in Natrona and Carbon counties southeast of the GYA (WGFD 2005b). Two CWD-positive elk were collected in central Carbon County in 2004, but most positive elk in Wyoming were found further east. Mule deer positive for CWD also have been documented just south of the Wyoming border in northeastern Utah (Utah Division of Wildlife Resources 2005) and in northwestern Colorado (CDW 2005a). Positive elk have not been found in Utah, but have been taken in northwestern Colorado. The WGFD has not made public sufficient data to calculate the probability that cervids occurring between these known endemic areas and the GYA are free of CWD.

Cervid Movements

Elk and deer herds in the GYA interchange individuals with other herds of the same species to the north, south, east, and west. They also share ranges seasonally. For example, of 41 radio telemetered elk captured on three winter feedgrounds in the Gros Ventre drainage during 2002–2003, 17.1% ($n = 7$) were relocated during summer and fall in the Wind River drainage, while 14.6% ($n = 6$) were relocated in the Green River drainage during this period (B. Smith, unpublished data). Thus, these members of the Jackson elk herd shared summer and fall range with elk and deer that migrate up and down the Wind and Green River drainages. Similar range overlaps among GYA herds, as well as between GYA and other cervid herds in adjoining areas, also occur (Craighead et al. 1972, Rudd et al. 1983, Toman et al. 1997, Smith and Anderson 2001). For these reasons, mountainous terrain, rivers, or other apparent topographic barriers should not be expected to prevent the spread of CWD to the GYA.

Risk to Feedground Elk and Other GYA Cervids

Prevalence of CWD in free-roaming mule deer inhabiting endemic areas is an order of magnitude higher than seen for sympatric elk populations and is roughly twice as high in adult male as compared with adult female deer (see Ecology/Epizootiology—Prevalence, above). Further, migrating mule deer also are much more likely to spread PrP^{CWD} than are dispersing deer (see Ecology/Epizootiology—Cervid Movements, Human Development, and CWD Prevalence Patterns, above). Prevalence of CWD in white-tailed deer also is much higher than typically seen in elk. Based on these ecological relationships, cervid movements in and near the GYA, and the fact that nearly all CWD-positive cervids documented near the

GYA were deer, it logically follows that deer, particularly migrating adult males, are the most likely source of PrP^{CWD} for feedground elk and other cervids in the GYA.

Assuming CWD does not already occur in the heart of the GYA, its most likely point of entry would be via infected cervids moving into Jackson Hole along the Gross Ventre drainage. This could occur if either infected deer or elk immigrated into the Jackson Hole or if elk and/or deer from Jackson Hole commingled with infected deer or elk, then returned to Jackson Hole. For either of these scenarios to occur, infected cervids would have to be present at least seasonally in the upper Green and/or Wind River drainages. This would require continued westward expansion of known CWD foci near Thermopolis and Worland into the Dubois area, or northward expansion of known CWD foci in south-central Wyoming, northwestern Colorado, or northeastern Utah (assuming these expansions have not already occurred). Mule deer, particularly adult males, are more likely carry PrP^{CWD} along these routes than elk.

ANTHROPOGENIC RISKS

Cervid Farms and Translocation

Elk farms or other captive cervid populations with CWD served as the source of PrP^{CWD} for free-roaming cervids in at least four states and two Canadian provinces (see Emergence, above). Although there are no known PrP^{CWD}-positive cervid farms in the GYA, states that allow deer farming in this region should take every precaution to ensure these herds remain CWD-free (see Management Options—Biological, above, and Options for Avoiding CWD in the GYA—Anthropogenic Risks—Cervid Farming, below, for ways this goal could be realized).

If cervids from CWD endemic areas were translocated into the GYA, CWD-positive individuals could result in disease establishment. Because state wildlife agencies have reasonably tight control over cervid translocation and release, this mode of ingress is unlikely. Importation of hunter-collected tissues contaminated with PrP^{CWD} is more realistic concern. Although the risk of seeding new areas with contaminated tissues is unknown, many states (including Wyoming) have taken this risk seriously enough to ban transportation of tissues likely to contain PrP^{CWD} (e.g., brain, spinal chord, certain lymph nodes) taken from cervids killed by hunters in endemic regions to areas or states where CWD does not occur.

Urbanization and Winter Feeding

While it is easy to understand why people should not transport CWD infected cervids or tissues into the GYA, a number of less obvious human activities also could contribute to CWD dissemination should the disease gain a foothold in the GYA. For example, housing developments in mule deer winter range, an increasingly common phenomena on private lands in the region, would be expected to lead to decreased human and nonhuman predation of mule deer, increased deer congregation, and increased CWD prevalence, just as in north-central Colorado (see Ecology/Epizootiology—Cervid Movements, Human Development, and CWD Prevalence Patterns, above).

Similarly, winter feeding of elk and/or deer leads to functional densities of cervids at least as high as those seen in many captive deer or elk herds where CWD prevalences of >50% and even >90% have been documented (see Ecology/Epizootiology—Prevalence, above). If CWD became established in elk populations utilizing winter feedgrounds, prevalences >50% could result. It does not matter ecologically whether elk congregation occurred due to cervid feeding conducted ad hoc by members of the public, approved by the state wildlife agency and conducted by private citizens, or sponsored and conducted by state or federal governments. At any rate, human activities that double CWD prevalence (e.g., urbanization of cervid range) or increase prevalence by an order of magnitude (e.g., winter feeding) would greatly increase the probability that CWD would spread throughout the GYA if the disease ever gained entrance to the region.

CONSEQUENCES OF CWD IN THE GYA

ECOLOGICAL

The ecological consequences of CWD on elk and deer population dynamics in the GYA are unclear, just as are the consequences of the disease within endemic areas (see Ecology/Epizootiology—Influence on Cervid Population Dynamics, above). Ecologically, CWD is probably best characterized as an epizootic with a protracted time-scale (Miller et al. 2000)—a disease that is gradually expanding its distribution by invading new host populations, slowly becoming increasingly prevalent where it occurs, and possibly leading to declining animal numbers in future decades. Miller and Conner (2005) found that CWD prevalence in each mule deer population unit within the endemic region of Colorado was still increasing over time, which lends support to this perspective. Similarly, foci of relatively high CWD prevalence within a matrix of much lower prevalence are consistent with CWD being an epizootic with a protracted time-scale. So far, model predictions of CWD leading to declining mule deer abundance, or even local extinction, have not occurred (Gross and Miller 2001). This could be due to inadequacies of the model (Schauber and Woolf 2003) or that insufficient time has passed to reach conditions necessary for the predicted declines to occur. Hopefully, ongoing ecological research within CWD endemic areas will shed additional light on the ecological consequences of CWD on free-roaming cervid populations.

It is easier to predict the consequences of human activities on CWD transmission and prevalence should the disease become established in the GYA. Housing developments in winter range probably would result in decreased human and nonhuman predation of mule deer, increased deer congregation, and increased CWD transmission and prevalence, just as was observed in north-central Colorado (see Ecology/Epizootiology—Cervid Movements, Human Development, and CWD Prevalence Patterns, above). Moreover, winter feeding of deer and particularly elk often results in functional cervid densities at least as high as those seen in many captive deer and elk herds. High cervid densities would result in PrP^{CWD} transmission rates much higher than typically seen in free-roaming deer or elk, which in turn could result in CWD prevalences in excess of 50% in elk and even higher in deer.

SOCIOLOGICAL

Economic

The most obvious social consequences of CWD in the GYA are centered on the economy. In much of the Colorado–Nebraska–Wyoming endemic area, hunters tend to live in the vicinity, elk are not the primary big game animal, and wildlife viewing is not the economic force it is in Jackson Hole or many other portions of the GYA. In contrast, if CWD becomes endemic in the Jackson elk herd, for instance, the economic repercussions for the guiding, lodging, food service, and sightseeing industries could be catastrophic. While there currently is no evidence that PrP^{CWD} can be spread to humans, fear of such transmission is omnipresent and this risk cannot be ruled out. Wealthy elk hunters, for example, might simply decide to hire guided hunts in northern Idaho, western Montana, or British Columbia. Similarly, tourists currently are drawn to the spectacle of thousands upon thousands of apparently healthy elk in the National Elk Refuge along U.S. Highway 26 north of Jackson, Wyoming. One cannot help but wonder whether this lure would be as great if 50% of the herd carried CWD and animals with clinical CWD occasionally were observed standing by the fence. It seems that the economic threat posed by CWD is sufficient to warrant a detailed economic analysis of the potential impact of CWD on the Jackson Hole and GYA economies. Hard data on the expected economic impact would be important for those making environmental and economic policy for the region.

Barring such a study, one way to obtain a rough notion of what the economic impact of CWD in the GYA is to evaluate the cost of CWD outbreaks in other jurisdictions. In February 2002, hunter-killed white-tailed deer taken by hunters from a relatively small south-central Wisconsin area during 2001 were found positive for CWD (Joly et al. 2003). Bishop (2004) conducted a survey of the economic impact of CWD in Wisconsin associated with deer hunting. He found that hunter losses were between \$53 and \$79 million in 2002 and \$45 and \$72 million in 2003. Because of fewer hunter-days in the field, about \$5

million was lost per year in 2002 and 2003 from the Wisconsin economy due to less spending by hunters. Additionally, the Wisconsin Department of Natural Resources (DNR) spent \$12.6 million and other Wisconsin agencies \$2.1 million on CWD during fiscal year 2002–2003. Because \$12.6 million was a significant portion of the DNR's budget, CWD management came at the expense of other important agency programs.

Another way to evaluate the potential economic impact of a CWD outbreak is to determine how people might respond to this eventuality. Vaske et al. (2004) reported that license sales for the 2002 Wisconsin gun deer hunting season declined approximately 11% following the discovery of CWD. A survey of individuals who quit deer hunting revealed that about half (52%) did not participate in the 2002 season because of CWD. Gigliotti (2004) found that 2 and 4% of hunters surveyed in South Dakota during 2002 and 2003, respectively, would discontinue hunting deer in their hunting unit if CWD was discovered in a single deer there. If CWD prevalence was 5% for the unit, 9 and 15% would discontinue hunting deer there. Because CWD prevalence already is > 5% in some hot spots in the Colorado–Nebraska–Wyoming endemic area, Needham et al. (2004) evaluated how hunters might respond to an array of CWD prevalence levels by surveying deer and/or elk hunters in eight states (Arizona, Colorado, Nebraska, North Dakota, South Dakota, Utah, Wisconsin, and Wyoming). They found that at prevalence levels currently seen in several states, 3% of resident and 5% of nonresident hunters would stop hunting deer and/or elk in the state (See Ecology/Epizootiology—Prevalence, above). At prevalence rates seen in portions of the Colorado–Nebraska–Wyoming endemic area, 10 and 15% of resident and nonresident hunter would stop hunting. If CWD prevalence reached 50% across the state, approximately 42% of resident and 54% nonresident hunters reported that they would stop hunting deer/elk in the state. Clearly, CWD in the GYA would be expected to influence the number of individuals hunting cervids and thus the economy. Unfortunately, no similar data are available to assess the potential economic impact of CWD on non-consumptive users.

Political

Economic problems are not the only societal ramifications of CWD becoming established in the GYA. Political repercussions for state and federal agencies responsible for managing either free-roaming wildlife or livestock health (e.g., cervid farms) could be severe. To many members of the public (both local and national), it will appear that regulatory agencies sat on their hands for 30 years while CWD slowly crept from southeastern Wyoming to the GYA. I expect it will not matter much whether this is an accurate assessment or not; politically, a scapegoat will be needed, and regulatory bureaucracies and key administrators will be easy marks.

More fundamentally, erosion of public trust in natural resource management agencies might be a more serious long-term consequence of CWD establishment in the GYA. If those interested in environmental issues in the GYA—hunters and non-hunters alike—have less trust in the natural resource management agencies they perceive to have been responsible for failing to prevent CWD from reaching the GYA, this could result in fewer management options for these agencies as well as more limited political and financial support.

Paradoxically, many of the political pressures that resulted in widespread winter feeding of GYA elk might cease to be relevant if CWD becomes well established in elk populations associated with feedgrounds (Peterson et al. 2005). Oft cited reasons for winter feeding include (1) enhancing hunting opportunities, (2) making elk more available for viewing, (3) reducing winter mortality to assuage public concerns about animal welfare, (4) keeping elk off roadways for public safety reasons, and (5) keeping elk from damaging crops, orchards, hay stacks and fences (Smith 2001). If CWD became established on GYA feedgrounds, it is probable that fewer people would hunt elk or want to view them (see Sociological—Economic, above). This would negate much of the perceived need for maintaining artificially high elk densities to satisfy hunters or elk watchers. Moreover, as winter feeding would greatly increase the probability that elk would contract and eventually die of CWD, feedgrounds would decrease the probability of death by starvation by unintentionally increasing the probability of death by CWD. This scenario is difficult to justify on humanitarian grounds. Regardless, some of the key reasons for winter

feeding of GYA elk would be much less applicable should CWD become established in GYA cervid populations, and suggests that decreased reliance on feedgrounds should receive serious consideration.

OPTIONS FOR AVOIDING CWD IN THE GYA

Preventing CWD from becoming established in the Jackson elk herd or elsewhere in the GYA should be the first CWD-related priority for wildlife biologists, managers, and policy makers responsible for these populations. Similarly, it should be a high priority for sportsman and environmental groups. If this effort fails, those responsible for GYA cervids would find themselves in the same political quagmire as their peers in northeastern Colorado, southeastern Wyoming, Wisconsin, and other states and provinces where CWD epizootics are occurring in free-roaming cervids. There is no easy solution to this problem (Williams et al. 2002a). Clinical signs of CWD are not diagnostic. There is no treatment. There is no prevention in the sense of vaccination in the face of an outbreak, and CWD is invariably fatal once clinical signs develop. Moreover, because CWD is a chronic disease with a long incubation period, and PrP^{CWD} is so hardy, long-term surveillance is a critical component of both CWD prevention and management. Unfortunately, CWD surveillance is more difficult and expensive than it is for most microparasitic diseases because there is no known antibody response, so serologic techniques have not been developed. Further, because there is no genetic material associated with PrP^{CWD}, PCR methods are not relevant. Other rapid and reasonably priced surveillance techniques are only now becoming available, so few long-term data on CWD prevalence exist.

In this section, I first discuss CWD diagnostic tools and approaches to surveillance. I then address how CWD could become established in the GYA both without and with the help of humans. Lastly, I discuss management of CWD foci, should they occur in the GYA.

DIAGNOSIS AND SURVEILLANCE

Presently, diagnosis of CWD is confirmed by examination of the brain for spongiform lesions and/or the accumulation of PrP^{CWD} in the brain or lymphoid tissues by IHC (Williams and Young 1993, Miller et al. 2000, Peters et al. 2000, Williams et al. 2002a, William 2005). The most useful tissues for diagnosing CWD in deer and elk using these techniques are the dorsal portion of the medulla oblongata at the obex (both histopathology and IHC), and the tonsils and retropharyngeal lymph nodes (IHC). Because PrP^{CWD} accumulates in the retropharyngeal lymph nodes and tonsils of white-tailed and mule deer early in the disease (Sigurdson et al. 1999, Williams et al. 2002a), examination of these tissues using IHC allows reliable clinical and even preclinical diagnosis (Wild et al. 2002, Wolf et al. 2002, O'Rourke et al. 2003). These tissues, as well as the brainstem, are readily collected from the heads of harvested cervids.

Wolfe et al. (2002) developed a technique for collecting tonsillar biopsies from live, free-roaming mule deer and evaluating them via IHC. They obtained similar prevalence estimates in a free-roaming mule deer population using this and traditional postmortem techniques. Wolfe et al. (2004) demonstrated that it was feasible to sample > 50% of the urban mule deer population annually in Estes Park, Colorado, as part of a test-and-cull program. Because this approach requires capturing and anesthetizing deer, collecting biopsies, marking them with radio telemeters prior to release, and relocating positive deer with telemetry and removing them from the population, its applicability is limited to small, urban deer populations. Unfortunately, IHC evaluation of elk lymphoid tissues has not been sufficiently sensitive for reliable antemortem diagnosis to date. Similarly, sampling lymphoid tissue from the nictitating membranes of deer and elk has not proven useful even though this approach works well for scrapie in sheep.

Rapid screening tests for CWD are now available and licensed by the U.S. Department of Agriculture. In a large study of hunter-harvested cervids in Colorado, Hibler et al. (2003) found that when results from an enzyme-linked immunosorbent assay (ELISA; Bio-Rad Laboratories) were compared with results of IHC as the gold standard, the ELISA was 98.3 (59 of 60), 100 (21 of 21), and 100% (3 of 3) sensitive for mule deer, elk, and white-tailed deer, respectively. Specificity was essentially 100% for all three cervid species. Validated rapid assays such as this should prove extremely useful to wildlife

managers conducting CWD surveillance. Their use should result in much larger spatially explicit databases that can be used to determine how confident one can be in CWD prevalence estimates for a given area.

Regardless of the diagnostic techniques used, those managing GYA cervids populations should continue or implement both clinically targeted surveillance and testing a random sample of hunter-killed cervids (Miller et al. 2000). Samples should be collected both within the GYA and between the GYA and areas of Wyoming where CWD is known to be endemic. The goal should be to identify new CWD foci rapidly should they occur (Williams et al. 2002a,b). To be useful in this endeavor, sample sizes must be sufficiently large to detect CWD at low prevalence (e.g., 1–2%) with great confidence (e.g., $\geq 95\%$) in the population to which inferences are to be drawn (Miller and Williams 2004). Not surprisingly, random samples provide a relatively unbiased estimate of prevalence (Conner et al. 2000). Surveillance strategies that meet these objectives also must continue for the foreseeable future to be of value.

NON-ANTHROPOGENIC RISKS

Three issues should be addressed if wildlife managers hope to minimize the risk of free-roaming cervids bringing PrP^{CWD} to the GYA or to halt the spread of the disease should infected cervids establish CWD foci. First, we must know whether migration pathways used by GYA cervids overlap with those of deer in the Bighorn Basin or northeastern Utah, northwestern Colorado, or south-central Wyoming. Sufficient CWD surveillance needs to occur within the GYA as well as in the regions between the GYA and known endemic areas to ensure that CWD foci would be found while they still were small and CWD prevalence low. Finally, wildlife management agencies in the GYA need to have a clear plan in place for managing such foci. I address the first two issues here, and the third in the last section of this essay.

Migration Pathways

Ultimately, migrating mule deer—particularly adult males—are the most likely way CWD would become established in GYA in the absence of human activity (see Risk of CWD Establishment in the GYA, above). For this reason, a better understanding of the migratory habits of mule deer, elk, and other cervids in and around Jackson Hole, and deer within the endemic areas in the Bighorn Basin and south-central Wyoming, northwestern Colorado, and northeastern Utah are badly needed. If no overlap is found, managers could continue surveillance and concentrate their efforts elsewhere. Conversely, if overlap among these cervid populations exists, managers would have the option of decreasing deer density within the endemic area used by these migrating cervids, thereby reducing the probability of PrP^{CWD} transmission to elk and deer in Jackson Hole and eventually to the rest of the GYA.

This objective could be reached using radio-telemetered deer and elk captured in the populations in question in conjunction with CWD surveillance. Conner and Miller (2004) used this approach to delineate (1) mule deer dispersal and migration patterns in north-central Colorado, (2) variation in CWD prevalence among population units, (3) potential exchange rates between populations on winter and summer range, and (4) likely paths of CWD flow based of prevalence and potential exchange rates. These data then were used by Miller and Conner (2005) to evaluate the influence of temporal, spatial, and demographic factors on CWD prevalence in this area. The WGFD (2005a) is supporting a similar study of white-tailed deer. Although this approach would be expensive and take time, it would provide critical data for wildlife managers, policy makers, and the public.

Surveillance

Surveillance in cervids, particularly deer, using samples sizes sufficiently large to detect low CWD prevalence (e.g., 1–2%) with great confidence (e.g., $\geq 95\%$) must occur within the GYA and in areas between the GYA and known endemic areas if managers are to have any certainty that CWD does not already occur in the GYA or in regions between the GYA and endemic areas (see Diagnosis and Surveillance, above). Similarly, assuming these areas are CWD-free, such surveillance is needed to rapidly detect new CWD foci should they occur. If managers are to have any chance of eradicating CWD

foci, they would have to be aware of them soon after establishment. High quality, long term surveillance is required to achieve this goal.

ANTHROPOGENIC RISKS

Although non-anthropogenic risks of CWD establishment in the GYA are theoretically and technically difficult to address, the sociopolitical implementation of measures designed to ensure that humans do not bring PrP^{CWD} to the GYA also are challenging for sociopolitical reasons. The WGFD (2005a) recently produced a draft CWD management plan that acknowledges many of the issues discussed below (deadline for public comments 10 November 2005). Although CWD-positive cervids have not been found in Idaho or Montana, it would be wise for Idaho Fish and Game and Montana Fish, Wildlife and Parks to obtain input from relevant state and federal regulatory agencies and publics, formulate plans for managing CWD in free-roaming cervids in their states should the disease gain entry, obtain public comment on the draft plans, and thus produce formal management plans for their states. If nothing else, this approach would help avoid some of the pitfalls caused by precipitous action elsewhere (Bishop 2004, Heberlein 2004, Vaske et al. 2004). General CWD management plans (Anonymous 2002, 2004), while useful resources, do not take the place of state-specific plans that have received public scrutiny.

Cervid Farming

Although PrP^{CWD} has not been documented in GYA cervid farms, states allowing deer farming in this region should take every precaution to ensure these herds remain CWD-free (Wyoming prohibits deer farming). These efforts should include requiring that (1) all captive cervids that die for any reason be tested for CWD, (2) all slaughtered cervids be tested, and (3) all animals introduced into captive facilities come from certified CWD-free herds. Should CWD be detected in any of these captive herds, depopulation is the only alternative. So far, attempts to eradicate CWD from contaminated facilities have failed (Williams and Young 1992, Miller et al. 1998) or are too recent to evaluate, so both captive and free-roaming cervids should be excluded from potentially contaminated premises for the foreseeable future.

Translocation of Infected Cervids and Tissues

Cervids should not be translocated from CWD endemic areas and released in the GYA. From a wildlife management perspective, it is difficult to envision a valid rationale for translocating deer or elk to the GYA and releasing them into the wild regardless of their origin.

Another potential source of PrP^{CWD} is contaminated tissues collected by hunters in endemic areas. Those having regulatory authority for GYA cervid herds should continue to prohibit importation of cervid brain, spinal chord, and certain other tissues from endemic areas. It also would be useful to conduct a study to estimate compliance rates with these restrictions as agencies largely rely on voluntary compliance. Research designed to determine the risk of seeding new areas with PrP^{CWD}-contaminated tissues also is needed.

Urbanization

The number of households is increasing much faster than the number of people in the United States and most other nations due to fewer generations sharing the same household and proliferation of second homes (Liu et al. 2003). This phenomenon is well established on private lands within the GYA and is likely to continue for the foreseeable future. Because urbanization greatly increases CWD prevalence in mule deer herds—thus increasing the risk of PrP^{CWD} transmission (see Risk of CWD Establishment in the GYA—Anthropogenic Risks, above)—issues surrounding urbanization of cervid winter ranges, particularly, should be addressed during CWD management planning.

Winter Feeding

It is difficult to justify winter feeding that congregates thousands of elk for several months year after year if preventing or managing infectious diseases (e.g., CWD, bovine brucellosis, bovine tuberculosis) is the primary objective. As detailed above, feedgrounds provide nearly ideal conditions for CWD transmission among free-roaming elk. If CWD becomes established in feedground-associated elk populations, managers should expect CWD prevalence to reach at least an order of magnitude higher than typically seen for elk herds in CWD-endemic areas without feedgrounds. There is no question that discontinuing winter feeding now would greatly reduce the risk of CWD transmission among elk in the GYA should elk in these populations be exposed to PrP^{CWD} .

OUTBREAK MANAGEMENT

Ecological options for managing CWD once it occurs in free-roaming cervid populations are few (see Management Options—Biological, above). There are no unambiguous examples of management strategies known to eradicate CWD in free-roaming cervid populations. Most states where CWD is endemic have either explicitly or implicitly followed the lead of the Colorado Division of Wildlife (CDW) regarding planning. Their primary goals are to (1) reduce CWD prevalence within the endemic area, (2) limit expansion of this region, and (3) identify and attempt to eradicate CWD foci found elsewhere in the state (CDW 2002, Peterson et al. 2002).

Concerning new CWD foci, the WGFD (2005a) plans “if possible and appropriate” to implement the following strategy:

Hunter surveillance in the area will be immediately intensified. If hunter samples are unavailable, as feasible the WGFD will attempt to collect and test up to 50 cervids in a 5-mile radius of the index case. If another CWD cervid tests positive based on the first samples, as feasible the WGFD will attempt to collect and test up to 50 additional cervids in a 10-mile radius of the original index case. For each subsequent cervid that tests positive for CWD, as feasible the WGFD will attempt to collect and test up to 50 cervids in a 5-mile radius of the positive animal. If additional cases are identified, the surveillance will be maintained and expanded until the periphery of the CWD area can be defined. This may occur outside of established hunting seasons. Such sampling serves three purposes: 1) to determine the magnitude, if any, of the prevalence; 2) possibly eliminate the only case of CWD in the area to prevent its spread; 3) possibly allow the WGFD to locate an area of infection that it can manage aggressively.

The WGFD (2005a) provided no details regarding what “aggressive management” might entail.

So far, attempts to eradicate CWD from heavily contaminated facilities have failed (see Management Options—Biological, above). If depopulation of elk farms and zoological gardens where only a few cases of CWD occurred proves successful, then it might be possible to eradicate CWD for new foci in the wild. Currently, however, there is no CWD management approach that has been shown to reliably eradicate CWD foci or eliminate the spread of the disease to new areas. Therefore, it is not possible to calculate the probability that CWD management strategies proposed by state wildlife agencies (e.g., CWD 2002, WGFD 2005a) will reach these objectives. Again, preventing CWD from becoming established in the GYA is a far better option than dealing with it once it is there.

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