Captive Deer and Elk as Vectors of Chronic Wasting Disease

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Chronic wasting disease is an incurable, fatal condition that afflicts deer, elk, and moose in North America [1, 2]. These animals are all in the family Cervidae, known as “cervids.” Chronic wasting disease threatens wild and captive populations and regulations are needed to halt this epidemic [3]. The movement of captive animals between game farms plays an important role in the spread of the disease [4, 5]. Game farms, through escaped animals, fence-line transmission, or environmental contamination, spread chronic wasting disease to the wild, where it is predicted to cause dramatic declines of game populations [6, 7].

This review describes the threat of chronic wasting disease to native wildlife and the role of game farms in the origin and spread of the disease. It is organized in three sections that address:

1. The nature of the infectious agent and symptoms of chronic wasting disease;
2. The history and geographic expansion of the chronic wasting disease epidemic in wild and captive cervids; and
3. The unacceptable risks of allowing the continued spread of chronic wasting disease, including the apparently remote but possible risk of transmission to humans.

Chronic wasting disease is “mad deer disease”

Most Americans are now familiar with “mad cow disease” (bovine spongiform encephalopathy). Chronic wasting disease is a similar affliction, which, like mad cow disease, is also caused by an abnormally shaped protein, known as a “prion” [1, 8–10]. Prions cause transmissible spongiform encephalopathy (TSE) [11, 12], different forms of which affect sheep (scrapie) [9], humans (Creutzfeldt–Jakob disease, variant Creutzfeldt–Jakob disease, and kuru) [13], cattle (bovine spongiform encephalopathy), and mink (mink spongiform encephalopathy) [14].

External symptoms of chronic wasting disease in cervids are loss of weight, excessive salivation, weakness, ataxia, behavioral changes such as loss of fear of humans, and depression [15]. Other symptoms include teeth grinding, lowering of the head, and drooping ears [16] (Figure 1).

Prions act by causing the malformation of naturally existing proteins in the host. These malformed prions accumulate in clumps in the brain and also result in development of microscopic
holes in the brain [1, 8, 16]. Most researchers have concluded that prions replicate without RNA or DNA, yet a small minority argue for a virus-like agent that aids in replication [17].

Unlike mad cow disease where brain or spinal material must be consumed to transmit the disease, chronic wasting disease spreads easily between living animals [18–20]. Infective prions are shed in the saliva and blood of infected animals [21]. Bucks are more susceptible to infection than does, probably because of exposure to many females, to other males in buck groups during winter, to rubs frequented by other males during mating season, and to more animals overall because of their larger home ranges and greater geographic movements than does [22]. Animals shed the infective agent before showing symptoms, so the disease can be spread by apparently healthy animals [23].

![Figure 1. Mule deer and elk suffering from chronic wasting disease [8, 16]. Reprinted with permission.](image)

Prions can also be transmitted through the environment so that direct contact between infected animals and healthy animals is not necessary for the disease to spread [24]. Not only is this route possible, but prions in the environment may be more infective because they bind to mineral elements in soil, which increases their infectivity dramatically [25, 26]. Cervids often supplement their diets with soil, so this avenue of infection may be quite significant [27].

**Farmed deer and elk spread chronic wasting disease**

The origin of chronic wasting disease is not definitively known [3, 28]. It was first seen, and subsequently described [8, 16], in captive animals at research farms around Fort Collins, Colorado in the 1960s [19]. The “most plausible” explanation for the origin of chronic wasting disease is that it is derived from scrapie, the prion disease of sheep [1]. Researchers were unable to find distinguishing characteristics that consistently show a difference between the scrapie prion and the chronic wasting disease prion, leading to the conclusion that scrapie was the origin of chronic wasting disease [29]. Scrapie has been found in sheep for over 300 years, but was first documented in North America in 1947 [14, 29]. A scrapie origin is consistent with the first detection of chronic wasting disease at the research farms where deer and elk were not kept isolated from domestic animals such as sheep [8, 16]. The alternative explanation is that the chronic
wasting disease prion arose spontaneously in mule deer and spread to elk and white-tailed deer [1], although this seems highly improbable.

Some have argued that the Fort Collins research farms were not the source of the wild epidemic because the highest infection rates in the wild are not directly around them [19]. However, other factors influence infection rates in the wild that could explain this pattern, including migratory behavior of deer and elk and soil conditions such as absence of copper [19, 30].

![Figure 2. Distribution of chronic wasting disease in free-ranging and captive deer and elk populations in North America (National Wildlife Health Center, USGS, April 2007).](image)

Wild mule deer and elk with chronic wasting disease were recorded first in Colorado in the same region where it was found in captive herds in 1981 [15]. It subsequently spread to the surrounding states of Wyoming [31], Nebraska, South Dakota, and Kansas (Figure 2).

Chronic wasting disease was introduced to Wisconsin “most likely” by farmed animals [32]. The Wisconsin example illustrates how chronic wasting disease can spread from game farms to the wild. Infected elk had been imported to Wisconsin in captive herds in the 1990s and two game farms in Wisconsin were later shown to be positive for chronic wasting disease [33]. Presence of chronic wasting disease in the wild deer population was confirmed in 2001 [32]. Given the infectivity of the disease, it is easy to see how an infected animal could escape from a game farm and transmit the disease to many wild animals and to leave infectious material in the envi-
ronment. Indeed, there is a documented instance of the escape from a game farm of an infected deer, which was recaptured after seven months in the wild [33].

Examples from other locations support this interpretation. In southern Illinois, a free-ranging deer infected with chronic wasting disease was found 30–50 km from an infected captive herd [32]. In western Colorado, chronic wasting disease was first found near a commercial enclosure where animals tested positive for the disease [32]. Introduction of chronic wasting disease to Canada almost certainly was caused by infected captive animals imported from South Dakota [3, 31]. Chronic wasting disease has been found in captive elk in Saskatchewan since 2000 [34]. Although conclusive evidence of transmission to wild elk in Canada is lacking, this is the most rational hypothesis for the current occurrence of the disease there [3]. In New York, chronic wasting disease has been located near an infected game farm (Figure 2). It was exported to Korea in captive herds [4, 35] but has not been transmitted to wild animals there.

In sum, nearly all incidences of chronic wasting disease in the wild are associated with known infections at game farms and the disease has a history of being moved by relocation of captive animals [5].

Allowing further spread of chronic wasting disease poses substantial risks

Chronic wasting disease developed from its epicenter in northcentral Colorado into an epidemic afflicting wild and captive populations of deer and elk in the United States, Canada, and Korea. Further geographic spread of the disease may be inevitable in the wild, but captive populations of cervids pose a particular hazard for the following reasons.

1. **Chronic wasting disease is highly transmissible in captive populations.** Over 90% of animals are infected with chronic wasting disease when it has been in a captive population for more than two years [32]. White-tailed deer transmit the disease faster than mule deer or elk because the prion accumulates in gut-associated lymphoid tissue, causing prions to be shed into the environment in large quantities [36, 37]. Any actions that artificially concentrate animals, either in captivity or at feeding stations such as those set up for elk in the Rocky Mountain west, will increase risk and rate of transmission [15].

2. **Chronic wasting disease goes undetected while it spreads.** The disease has a long latency period, meaning that deer and elk go long periods without showing symptoms while still being infective [23, 38]. In one example, a captive herd of 133 white-tailed deer was 50% infected with chronic wasting disease but neither the owner nor hunters noticed [39]. There is not yet a reliable test for chronic wasting disease in live animals. In free-ranging populations, many sick deer go unreported [40], so it is difficult to detect the disease even when animals become symptomatic.

3. **It is difficult to eliminate contact between confined animals and free-ranging animals.** Fences are not perfect and deer and elk can escape. Furthermore, captive and wild animals interact along fence lines [41]. At an elk farm with a 9-foot fence, video cameras set up along the perimeter recorded 77 instances of nasal or oral contact between wild and captive elk [41]. Elk frequently licked the fence, which could also transmit chronic wasting disease between captive and wild animals [41].
4. **It is nearly impossible to clean areas that have been infected.** Soil enhances transmissibility of chronic wasting disease and is nearly impossible to clean. Paddocks where infected animals died and decayed can transmit the disease [36], as can those where infected animals lived [20, 24, 25]. Prions are resistant to physical and chemical methods of decontamination. For example, formalin and ethylene oxide do not reduce infectivity, nor does autoclaving at 121° C for 15 minutes or high doses of ionizing or ultraviolet radiation [3]. Burial for three years reduces infectivity of the scrapie prion [42], but research on chronic wasting disease shows that the prion binds with soil particles, which makes it more infective [25]. At a game farm in Korea, all sick animals were killed in 2001, but the disease recurred in 2004 at the same game farm because of the failure to remove all of the infectious material [35]. At least two other attempts at eradication at game farms have failed as well [18, 43]. Aggressive treatments to remove the infective agent have been tried on paddocks where soil removal is feasible [35], but this approach is not feasible for larger, topographically diverse game farms. Soil covering has been tried as well, but it may only delay the escape of the infective material.

5. **Chronic wasting disease could devastate wild game populations.** Simulation models suggest that chronic wasting disease is a slow-motion epidemic that will reduce game populations substantially [6, 7]. These models predict that once infection rates reach 5%, the overall population will decline [19]. Because it can take 20–30 years after introduction for infection rates to reach 5%, some management is possible [19]. Researchers agree, however, that eradication is not feasible [1]. Some question these predictions based on the assumptions about transmission rate [44], but long-term population decline of deer and elk can be expected once chronic wasting disease is introduced in a region.

6. **Chronic wasting disease poses an unknown hazard to other species, including humans.** Species other than deer and elk can be infected by cervid prions. Ferrets, raccoons, and ruminants including cattle have been infected by injecting the prions into the brain [10]. However, under the field conditions that have been investigated so far, transmission to cattle seems unlikely [28]. Moose have been infected through oral inoculation in captivity [1], and recently infected wild moose were found in northcentral Colorado [2]. Although there is no strong evidence that transmission to humans has occurred [13], one primate, the squirrel monkey, has been infected experimentally with chronic wasting disease [45]. Some researchers have identified certain molecular barriers to transmission to humans [46, 47], but others have shown that in acidic environments, such as those that occur in some organs and within organelles in cells, the cervid prion is able to transform healthy human proteins into the infective form [48]. Risk of transmission to humans therefore cannot be ruled out. From an epidemiological perspective, not enough humans have yet been exposed to chronic wasting disease prions to determine if the species boundary can be crossed [32].

Chronic wasting disease is an incurable, fatal disease of cervids in North America. Its spread appears to be facilitated and promoted by game farms and by feeding stations for wild animals. Long-distance movement of the disease occurs in commerce of infected animals, but there is currently no method to available to screen living animals for the disease before transporting them. Once introduced, the disease cannot be eliminated in the wild and has adverse consequences for wild cervid populations. States concerned with protecting wild deer, elk, and moose populations from chronic wasting disease should ban the import and captive farming of cervids.
Literature Cited