

Chronic Wasting Disease (CWD)

System: Terrestrial

Kingdom	Phylum	Class	Order	Family
Virus				

Common name Chronic Wasting Disease (English)

Synonym

Similar species

Summary

Chronic Wasting Disease (CWD) is a neurological prion disease affecting cervids. Significant research and attention has been focused on CWD since its infectious agent is similar to that found in other diseases known to affect humans. However, there is no current evidence that CWD is transmissible to humans. The fatalities of cervids, however, can negatively affect cervid populations and impact humans in the form of economic loss. Many questions regarding CWD remain unanswered but the disease's impact on North American cervid populations combined with its continual geographic spread during the past decade has increased the fervour among researchers and managers to develop solutions for the biological and social challenges presented by CWD.



[view this species on IUCN Red List](#)

Species Description

Chronic Wasting Disease is a progressive, fatal disease of the nervous system, affecting cervids, both captive and free-ranging (Alberta Prion Research Institute, 2008). It is classified as a transmissible spongiform encephalopathy (TSE) or prion disease (Belay *et al.*, 2004). The disease was first recognized as a clinical syndrome in mule deer at a research facility in Fort Collins, Colorado, USA in 1967. After initially being considered a disease related to captivity and associated with nutritional deficiency, toxins or stress, it was accurately classified as a TSE in 1978 (Williams & Young, 1980). When considering the entire TSE disease family, only the cervid-specific chronic wasting disease and sheep scrapie are readily transmissible to susceptible hosts through horizontal transmission of infected animals or environmental reservoirs of infectivity (Pilon *et al.*, 2007). CWD is the only known spongiform encephalopathy known to naturally infect both free-ranging and captive animals. This greatly complicates efforts to monitor, control or eradicate it (Bunk, 2004). Of all prion diseases, CWD may be the most efficiently transmitted. In dense free-ranging deer populations prevalence can be as high as 40% and in captive herds it can reach 100% (Sigurdson & Aguzzi, 2007). CWD is caused by "aberrantly refolded isoforms of the prion protein, a normal cellular glycoprotein" (Williams, 2005). Prion proteins convert normal proteins in the host animal's cells resulting in concentrations of refolded isoforms. Over time, these abnormal proteins accumulate in the central nervous and lymphatic systems causing a degenerative lack of body coordination, marked changes in behaviour, emaciations, and a prolonged physical "wasting-away" until death (Williams *et al.*, 2002).

The disease affects elk, mule deer, black-tailed deer, white-tailed deer, white-tailed deer hybrids (Forrest, 2003) and moose (Kreeger *et al.*, 2006). CWD infection-specific protease-resistant prion protein accumulates in gut-associated lymphoid tissues of infected deer, which implicates alimentary shedding of the CWD agent in both feces and saliva. In elk, CWD has been detected by IHC in the myenteric plexus, the vagosympathetic trunk, the cell column of the spinal cord and endocrine glands (Salman, 2003). Because CWD infection-specific protease-resistant prion protein becomes progressively abundant in the nervous system and lymphoid tissues through the disease course, carcasses of cervids succumbing to CWD also likely to serve as foci of infection (Miller *et al.*, 2004). A characteristic of all transmissible spongiform encephalopathy agents is their resistance to conventional disinfectants, high temperatures, and enzymes that normally break down proteins (USDA-APHIS, 2008). The clinical signature of CWD includes several non-diagnostic symptoms such as: weight loss, behavioural changes, excessive water consumption, salivation and urinating, together with erratic teeth grinding (Forrest, 2003). In addition, animals may show repetitive behaviours such as walking set patterns in the pen or pasture, show periods of somnolence or depression from which they are easily roused, and may carry their head and ears lowered. Affected animals continue to eat but consume reduced amounts of feed, leading to gradual loss of body condition. As the disease progresses, many affected animals display polydipsia and polyuria, increased salivation with resultant slobbering or drooling, as well as inco-ordination, posterior ataxia, fine head tremors, and a wide-based stance (Williams & Miller, 2002).

Oregon Invasive Species Council has classified Chronic Wasting Disease as one of 100 most dangerous invaders (2008). Communal use of wallows and mineral licks by elk may increase the risk of CWD transmission by sharing areas contaminated with bodily excretion potentially including CWD prions (VerCauteren *et al.*, 2007a).

Notes

Lateral transmission is significant in the epidemiology of CWD (Kahn *et al.*, 2004). Lateral transmission, compounded by animal movements, is the most important factor in spread of CWD. Indirect transmission via environmental contamination may play a role in natural dynamics and persistence of the disease and thus exacerbate the spread of the disease, and may present an obstacle to eradicating CWD from infected premises (Salman, 2003). It appears that cervids are exposed via ingestion of PrP-CWD from sources in the environment, direct contact with animals that are excreting the agent, or both. Increased population density as a consequence of keeping animals in captivity or artificial feeding of wildlife may increase the likelihood of direct and indirect transmission between individuals. Direct transmission between animals probably requires more than just transient exposure (Kahn *et al.*, 2004). Infectious prions are shed through the alimentary tract via saliva or feces and potentially urine. Prions deposited in soil remain infectious and are extremely resilient, persisting up to 3 years in experimental studies and sometimes for more than 15 years in domestic situations. In addition, fecal-oral transmission is a likely mechanism of disease transmission of CWD. In fact, data shows that deer could not entirely avoid fecal consumption at supplemental feeding sites and even low-level consumption may be important if fecal material contains an infectious dose of disease agent. Consumption of fecal material, contaminated soil, and forage are potential modes of indirect disease transmission (Thompson *et al.*, 2008). In the USA and Canada, the mandatory reporting of CWD is governed by a combination of national and state or provincial legislation (Kellar & Lees, 2003). Species known to be susceptible to CWD by an extreme and unnatural exposure route, i.e. intracerebral inoculation, include ferrets, raccoons, other ruminants, and squirrel monkeys (Sigurdson & Aguzzi, 2007).

Lifecycle Stages

There is no epidemiological evidence that confirms the origin of CWD. There is no evidence to support a feed-borne common source origin of CWD. However, some hypotheses of the origin include: infection of deer by a strain of scrapie that has adapted to cervid hosts, a genetic form of TSE arising in deer, with subsequent natural transmission, exposure to a currently unknown TSE, expressing the possibility, borne particularly out of infancy of study of diseases on wildlife, that there could be undetected TSE or prion disease in other species, and a spontaneous conformational change of the prion protein occurring in mule deer, with subsequent transmission to other deer and to elk. Unfortunately, none of these hypotheses provide a particularly plausible explanation of the origin of CWD (Salman, 2003). Under experimental conditions, minimum incubation was approximately fifteen months and mean time from oral infection to death was approximately twenty-three months in mule deer, and the range of onset of clinical disease in orally infected elk was approximately twelve to thirty-four months (Williams & Miller, 2002). Environmental models are distinct in representing the assumption that infectious material is durable and does not require contact with or the immediate presence of infected deer to perpetuate epidemics (Miller *et al.*, 2006).

Habitat Description

Forecasting by models leads to the assumption that prevalence of CWD increases slowly over several decades. Such increases are uniform across populations and over time, it follows that CWD prevalence should be highest in areas where it has been present longest. Therefore, Wyoming and Colorado have a higher prevalence of CWD where it has been recognized for several decades (Miller *et al.*, 2000).

Reproduction

It is thought that CWD is derived from sheep scrapie. On the other hand, it has been suggested that CWD is derived from the spontaneous generation of infectious prion protein. However, the disease's origin remains known (Masujin *et al.*, 2007).



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General Impacts

There is a significant potential for expansion of the geographic range of CWD, and once established, the disease could be maintained and transmitted through deer to deer interaction and, in some situations environmental contamination for an indefinite period of time (Grear *et al.*, 2006). Exposure to CWD prions could potentially occur through consuming meat or tissues from infected animals, while processing game, or through unusual pathways such as ingesting antler velvet (Anderson *et al.*, 2007). However, evidence strongly suggests a species barrier to transmission of CWD to humans (Bourne, 2004). Although findings consistently suggest that there is no connection between CWD and human TSEs, the possibility that an isolated case of human disease associated with the CWD agent has occurred or may yet occur cannot yet be excluded (MaWhinney *et al.*, 2006). Several states and provinces have banned the importation of cervids in the face of growing knowledge regarding the distribution of CWD within farmed and wild herds on the continent, which could have negative effects on the economies of those who rely on the importation of cervids (Kellar & Lees, 2003). This disease has the potential to cause severe harm to deer populations and thus to disrupt ecosystems where deer occur in abundance (Miller *et al.*, 2006). However, on-going research on the species barrier is indicating that there is a substantial biological barrier to transmission of CWD from deer to cattle under normal conditions (Salman, 2003).



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Management Info

Preventative measures: Regulatory agencies in locations without CWD should plan to prevent its introduction through cervid and cervid part movement restrictions, restrictions on baiting and feeding, risk assessments, population management, and information dissemination (USDA-APHIS, 2008). In particular fawns/calves should never be moved in or out of enzootic and newly infected areas to prevent potential spread. Where possible potentially infected fawns and calves should be euthanized, particularly as most live animal tests for CWD may not be effective in young animals. Carcass disposal in enzootic and newly infected areas should focus on reducing surface contamination by deep burial at approved sites or small numbers can be incinerated (Pybus & Hwang, 2008).

Management Techniques to Eliminate, Contain, or Control CWD: Outbreak surveillance establishes the prevalence, incidence, and distribution of the disease, and allows the evaluation of management actions. Population reduction is a technique that encompasses the depopulation of farmed cervids, or of free-ranging cervids in limited geographical areas. Targeted removal can reduce a specific subset of an affected population (USDA-APHIS, 2008). Surveillance testing and removal can be used to remove CWD affected animals from a population, but this approach may only be appropriate in limited situations. Therapeutics and vaccines could be a future option, but these tools are not currently available. Restrictions on feeding or baiting of free-ranging cervids where appropriate, and regulations for the farmed cervid industry are examples of management tools that may be used to control CWD. The manipulation of environmental factors could limit animal use of areas and potential exposure. These tools are most useful in dealing with environmental contamination. Lastly, agricultural and wildlife agencies should provide scientifically based recommendations for limiting animal movements to prevent the spread of CWD (USDA-APHIS, 2008). Long persistence times or high excretion rates of infectious agent will make it more difficult to reduce or eradicate CWD as compared to the case of direct transmission. Better understanding of agent excretion and persistence are thus needed in order to make realistic assessments of the viability of specific CWD control strategies (Miller *et al.*, 2006).

Integrated Management: In Canada and the USA, financial incentives have been added in the form of compensation for animals slaughtered in national eradication efforts for CWD (Kellar & Lees, 2003). Banning supplemental feeding practices to reduce risks of disease transmission, especially in areas with infectious disease outbreaks, is warranted (Thompson *et al.*, 2008).

Biological: Studies suggest that tonsillar biopsy is a valid method for detecting CWD in live deer during incubation stage, and may be used as an ante-mortem and pre-clinical diagnosis and as an adjunct management tool (Salman, 2003). Current research into rectal biopsies may produce an effective method of ante-mortem CWD diagnosis in elk (USDA, 2008). A CWD-susceptible cell line has also been developed, which is derived from cervid brain fibroblasts, and has been used to screen inhibitors of CWD infection. This is the first CWD specific assay introduced for screening compounds that inhibit CWD propagation (Sigurdson & Aguzzi, 2007).

For more information on response to CWD in at risk, newly infected and enzootic areas please see the [Chronic Wasting Disease Workshop](#) document (Pybus & Hwang, 2008).

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ALIEN RANGE

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