



## CHRONIC WASTING DISEASE (CWD)

ANIMAL GROUP AFFECTED	TRANSMISSION	CLINICAL SIGNS	FATAL DISEASE?	TREATMENT	PREVENTION & CONTROL
Cervidae ( <i>Odocoileus hemionus</i> , <i>Odocoileus virginianus</i> , <i>Cervus elaphus nelsoni</i> )	Mainly horizontally. Presumably, shed in faeces or saliva; environmental contamination	Loss of body condition, changes in behaviour	Yes	No known treatment	<i>In houses</i>  <i>in zoos</i> Prevention difficult; herd certification program in North America.

<b>Fact sheet compiled by</b> Elvira Schettler	<b>Last update</b> January 2004
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<b>Susceptible animal groups</b> The natural host range appears to be limited to mule deer ( <i>Odocoileus hemionus</i> ), white-tailed deer ( <i>Odocoileus virginianus</i> ) and Rocky Mountain elk ( <i>Cervus elaphus nelsoni</i> ). Subspecies of these cervids are probably also susceptible. The disease affects captive as well as free-living animals. Based on sequence analysis of PrP in elk it is recently discussed that polymorphisms in PrP gene could be associated with susceptibility to CWD in this species.	
<b>Causative organism</b> Chronic wasting disease belongs to a group of fatal, neurodegenerative disorders (transmissible spongiform encephalopathies, TSE), thought to be caused by an accumulation of abnormal protease-resistant form (PrP <sup>res</sup> ) of cellular prion protein (PrP <sup>C</sup> ) normally synthesised in the central nervous system (CNS) and lymphoid tissues. On entering a susceptible host, PrP <sup>res</sup> promotes production of species-specific PrP <sup>res</sup> from PrP <sup>C</sup> in lymphoid or CNS tissues. In animals TSE are infectious; spontaneous and familial forms have not been identified, though they may theoretically occur.	
<b>Zoonotic potential</b> Not definitely known. The available epidemiologic evidence indicates that CWD is probably not highly pathogenic for humans. Nevertheless, a level of pathogenicity similar to that displayed by BSE prions is theoretically possible.	
<b>Distribution</b> CWD seems to be restricted to North America. The disease was first recognised in 1967 among captive mule deer at wildlife research facilities in Colorado (USA). However, it also occurs in free-living deer and elk. So far, CWD has been confirmed in deer and elk from the following States in USA: Colorado, Illinois, Kansas, Minnesota, Montana, Nebraska, New Mexico, Oklahoma, South Dakota, Utah, Wisconsin and Wyoming. Moreover, in Canada the disease was reported in free-living deer in Saskatchewan and on deer/elk farms in Alberta. Furthermore, farmed elk in South Korea, imported from North America were found positive for CWD.	
<b>Transmission</b> The exact mode of transmission is still unknown. Based on several epidemiological studies there is strong evidence that CWD is transmitted horizontally from infected to susceptible cervids. Maternal transmission may also occur but seems to play a subordinate role. Accumulation of PrP <sup>res</sup> in gut-associated lymphoid tissues during the disease course suggests agent shedding in faeces and/or saliva as plausible transmission routes. Residual infectivity in contaminated environments, either from excreta or from decomposed carcasses of infected animals, also may be important in sustaining epidemics. There is no evidence that CWD is a food-borne disease as is the case in BSE.	
<b>Incubation period</b> The incubation period is not exactly known. Deer experimentally infected with CWD develop signs of clinical disease $\geq$ 15 months later. The estimated minimum incubation period for natural infections with CWD is 17 months. Maximum incubation period is not known.	
<b>Clinical symptoms</b> The most striking clinical features of CWD in deer and elk are loss of body condition and changes in behaviour. Clinical signs may be more subtle and prolonged in elk than in mule deer. Furthermore, affected	



animals may increase or decrease their interactions with handlers or members of the herd and show repetitive behaviours, such as walking set patterns. Depression and somnolence may be observed and head and ears of animals that suffer from CWD are often carried lowered. Most affected individuals display polydipsia, polyuria, and increased salivation. Moreover, inco-ordination, particularly posterior ataxia, fine head tremors, and wide based stance can be observed as the disease progresses. Death is inevitable.

**Post mortem findings**

Gross lesions of CWD are non-specific. Carcasses are mostly in poor nutritional state or emaciated. However, they may be in fair condition if the animal died of aspiration pneumonia or after only a short clinical course. Aspiration pneumonia may be present in some animals. Rumen contents contain excessive water in those animals displaying polydipsia.

Microscopic lesions of CWD are qualitatively typical of TSE. The most striking change in the brain is a spongiform degeneration of the grey matter. In all cases of clinical CWD, lesions are in the parasympathetic vagal nucleus in the dorsal portion of the medulla oblongata at the obex, in the hypothalamus and thalamus, and in the olfactory tracts and cortex. Other regions of the brain, in particular thalamus and cerebellum, show typical spongiform changes with varying degrees of severity. Lesions are usually mild in the cerebral cortex, hippocampus, and basal ganglia. Plaques composed of PrP<sup>tes</sup> can be seen in most clinically affected white-tailed deer and in a few mule deer but are not obvious in elk. Moreover, scrapie-associated fibrils are found in brains and spleen of deer and elk with CWD.

**Diagnosis**

Clinical signs are non-specific.

Ante mortem: By means of biopsy, tonsil tissue from live deer or elk can be tested using immunohistochemistry or the below listed rapid tests. However, in case of negative test result a CWD infection cannot definitely be excluded.

Post mortem:

Immunohistochemistry (Gold standard).

Histopathology only recommendable as supplementary diagnostic method.

Rapid tests (alphabetically listed):

- BioRad CWD antigen test kit, ELISA (approved for detection of CWD in elk and deer in the USA- in principal identical with BioRad TeSeE Assay approved for detection of BSE in cattle in the EU).
- ENFER (Chemo luminescent-ELISA, approved in the EU for the detection of BSE in cattle).
- IDEXX HerdChek\*CWD Antigen Test Kit (ELISA, not yet licensed).
- Prionics Check Western (Western blot, approved in the EU for the detection of BSE in cattle).
- Prionics check LIA (approved in the EU for the detection of BSE in cattle)
- VMRD "CWD dbELISA" (validated and approved for diagnosis of CWD in elk and deer in the USA).

**Material required for laboratory analysis**

Ante mortem: tonsil tissue

Post mortem: Brain stem (parasympathetic vagal nucleus in the dorsal portion of the medulla oblongata at the obex), medial retropharyngeal lymph node, and tonsil tissue.

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**Treatment**

There is no known treatment for animals that suffer from CWD.

**Prevention and control in zoos**

No case of CWD has been reported in any cervid species (neither in enclosures/ zoos nor from free-living cervids) within Europe. However, it is theoretically possible that CWD can also affect European cervids. Research and surveillance programs on CWD did not exist until recently in free-living or captive cervids from Europe and thus the available data do not yet allow to draw conclusions. Prevention and control of CWD is problematic not only because of the long incubation period of this disease but also because of the absence of reliable and practical ante mortem tests. In North America management in captive cervids currently involves long-term active surveillance to determine distribution and prevalence in free-ranging animals. Moreover, herd certification programs are conducted. Recommendations for North American zoos with respect to CWD are to obtain herd history and source of captive deer and elk (CWD free), minimise contact between captive and wild cervids, and submit tissue material for CWD surveillance from all cervids (> 12 months) within the collection that die or that are euthanized.

**Suggested disinfectant for housing facilities**

2N NaOH or strong sodium hypochlorite.



<b>Notification</b> According to the requirements of the decree V (EG) 999/2001 all TSE suspected animals have to be reported to the local authorities.
<b>Guarantees required under EU Legislation</b>
<b>Guarantees required by EAZA Zoos</b>
<b>Measures required under the Animal Disease Surveillance Plan</b>
<b>Measures required for introducing animals from non-approved sources</b>
<b>Measures to be taken in case of disease outbreak or positive laboratory findings</b>
<b>Conditions for restoring disease-free status after an outbreak</b>
<b>Contacts for further information</b> Elvira Schettler ( <i>Project: TSE in cervids from Germany</i> )
<b>References</b> <ol style="list-style-type: none"><li>1. Anonymous. 2001. Chronic wasting disease- USA: Test. Environmental News Network, 12. Dec. 2001.</li><li>2. Anonymous. 2003. Chronic wasting disease herd certification program and interstate movement of captive deer and elk. US Department of Agriculture, Federal Register 68: 247, pp 74513-74529.</li><li>3. Anonymous. 2003. Chronic wasting disease and tissue that might carry a risk for human food and animal feed chains. Report of the EU Scientific steering committee.</li><li>4. Bosques, P.J. 2002. Bovine spongiform encephalopathy, chronic wasting disease, scrapie, and the threat to humans from prion disease epizootics. Current Neurology and Neuroscience Reports 2: 488-495.</li><li>5. Miller, M.W. and E.S. Williams. 2003. Epidemiology and management of chronic wasting disease in free-ranging cervids. Proceedings of the International CWD Workshop, 15. Aug. 2003, Saskatoon, Canada. Pp 5-6.</li><li>6. Miller, M.W. and E.S. Williams. 2003. Horizontal prion transmission in mule deer. Nature 425: 35-36.</li><li>7. Peters, J., J.M. Miller, A.L. Jenny, T.L. Peterson, K.P. Carmicheal. 2000. Immunohistochemical diagnosis of chronic wasting disease in preclinical affected elk from a captive herd. J. Vet. Diagn. Invest. 12: 579-582</li><li>8. Rourke, K.I., T.E. Besser, M.W. Miller, T.F. Cline, T.R. Spraker, A.L. Jenny, M.A. Wild, G.L. Zebarth, and E.S. Williams. 1999. PrP genotypes of captive and free-ranging Rocky mountain elk (<i>Cervus elaphus nelsoni</i>) with chronic wasting disease). J. Virol. 80:2765—2769.</li><li>9. Williams, E.S., J.K. Kirkwood, and M.W. Miller. 2001. Transmissible spongiform encephalopathies. In: E.S. Williams, and I.K. Barker (eds.) Infectious diseases of wild mammals. Iowa State University press. Pp. 292-297.</li><li>10. Williams, E.S. and S. Young, Neuropathology of chronic wasting disease of mule deer (<i>Odocoileus hemionus</i>) and elk (<i>Cervus elaphus nelsoni</i>). 1993. Vet. Pathol. 30: 36-45.</li></ol>