

# Chronic Wasting Disease (CWD)

## Susceptible species

Numerous cervids such as mule deer, black-tailed deer, white-tailed deer, Rocky Mountain elk, reindeer, wapitis and moose, as well as red deer, which is indigenous in Germany, are susceptible to CWD. Susceptibility of roe deer and sika deer can be assumed.

Fallow deer have only been susceptible to experimental infections, so far.

## Distribution area

The first case of CWD was described in Colorado/USA in 1967 in a mule deer. From there, the disease spread to further U.S. states and eventually reached Canada, due to importation of infected animals. Today CWD is endemic in wide parts of the North American continent. Another import of infected wapitis from the U.S. to South Korea led to the first description in this country in 2001.

In 2016, the disease was detected in a reindeer herd in the Norwegian Nordfjella for the first time. Subsequent eradication of the affected herd and strict surveillance measures yielded additional 19 CWD-positive reindeers. In 2020 another outbreak in the geographically distant Hardangervidda was confirmed. At date, an introduction to other European countries cannot be ruled out completely.

An active surveillance program, including EU states such as Sweden and Finland, detected cases in several moose and one red deer. In September 2021, another red deer was tested positive. However, these cases differ from

those found in reindeer. Based on current knowledge, they are most likely of sporadic origin.

All European CWD cases can be distinguished from the North American outbreaks by further biochemical analysis, suggesting they are not related.

## Causative agent

The disease is classified as a Transmissible Spongiform Encephalopathy. Causative is the conversion of the cellular membrane-bound prion protein ( $\text{PrP}^c$ ) into a pathologic form ( $\text{PrP}^{\text{Sc}}$ ) induced by  $\text{PrP}^{\text{Sc}}$ , leading to a massive protein deposition in the brain.

In many species, susceptibility to TSE depends on the structure of the prion protein gene, leading to variable susceptibility/resistance to CWD. This is also demonstrated in North American cervids. Unfortunately, little is known about the susceptibility of the deer population indigenous in Europe.

## Transmission

CWD is a highly contagious disease transmitted among others by blood, urine, feces, lymph, saliva and semen and is excreted during the incubation period (up to 34 months). The high tenacity of  $\text{PrP}^{\text{Sc}}$  leads to extreme contamination of the environment, in which the prion protein might remain infectious for decades.

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## Clinical picture

Disease manifests usually between the age of two to seven years, with males being affected more frequently. Despite preserved appetite, increasing weight loss leads to cachexia. In addition, affected animals separate themselves, show listlessness and incoordination and lose their natural shyness towards humans. Ruminal atony, excessive salivation, frequent drinking, and increased urination are further symptoms of CWD.

Secondary infection are often fatal. Sudden deaths occur occasionally. An infection with CWD leads to death eventually.

## Diagnostics

PrP<sup>Sc</sup> is diagnosed by a rapid test, biochemical or immunohistological methods. All detection methods require brain stem from the obex (*medulla oblongata*) or a sample from the deep cervical lymph node (*lymphonodus retropharyngeus*).

For further information see:

[Official Collection of Methods of FLI](#)

## Similar clinical pictures

Tuberculosis (*Mycobacterium bovis*), pseudotuberculosis (*Yersinia pseudotuberculosis*), paratuberculosis (*Mycobacterium avium* subsp. *paratuberculosis*) gastrointestinal parasitoses, dental problems, malnutrition, inadequate vitamin and mineral intake lead, like CWD, to weight loss, even cachexia. Rabies, intoxications, infection with *Listeria monocytogenes* and tumors in the brain are associated with incoordination and behavioral changes. Similarly, lameness or trauma may feign incoordination.

## Control

Due to the high tenacity and resistance of the pathogen to environmental influences and disinfection measures, control of CWD is a major challenge. The eradication of the reindeer herd in Norway, where the first CWD case was diagnosed, was initially able to contain the spread of the disease. However, the subsequent active surveillance program detected another case of CWD, indicating the spread of the disease and highlights the importance of minimizing risk factors. The reduction of population density plays a particularly important role. By lowering the animal numbers, in particular males, which are more frequently affected, environmental contamination and the risk of infection, decreases. In addition, human activity in regions affected by CWD should be reduced, so animals have access to larger areas and pathogen spread by humans is avoided. To prevent natural migration of individuals, affected regions should be fenced. Likewise, it is important to reduce lick and feeding stations to avoid gathering of animals. Raising awareness of interest groups (e.g. hunters, veterinary agencies) can also drastically reduce the risk of unintentional introduction and ensure early disease detection.

Further information see:

[German National Reference Laboratory for TSE](#)

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