



Chronic Wasting Disease

A product of the Wildlife Health Committee and Mule Deer Working Group

Fact Sheet #39

CAUSE AND TRANSMISSION

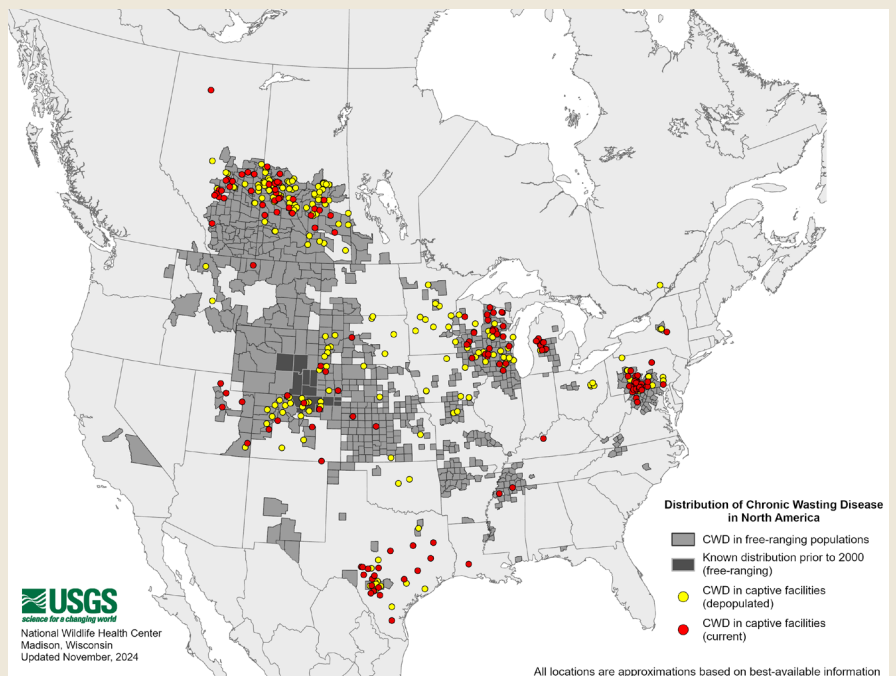
Chronic wasting disease (CWD) is caused by one or more strains of self-propagating proteins called prions (“pree-on”). The prion is transmitted and begins to replicate, first in the tissues of the immune system (lymph nodes and tonsils) and later in the brain, spinal cord and other organs. Infected animals can shed prions in saliva, feces, urine, and possibly after death through their remains. Animals are infected by direct (animal-to-animal) transmission or by indirect transmission from prions deposited in the environment. Once infected, it is always fatal with animals dying, typically within 2-3 years. Individual animal genetics and prion strains play a role in the disease progression

SPECIES AFFECTED

Chronic wasting disease affects species in the deer family (*Family Cervidae*). In North America, mule deer, white-tailed deer, elk, and moose have been infected. Caribou should also be considered susceptible because reindeer have tested positive. CWD has occurred in red deer and sika deer on other continents. Species outside the deer family appear to have natural resistance to infection even though some can be infected experimentally.

GEOGRAPHIC DISTRIBUTION AND OCCURRENCE

Chronic wasting disease occurs primarily in the USA and Canada, with a smaller number of cases reported in Korea and northern European countries. In North America, CWD has a scattered distribution. Disease focal points differ in size, duration, and connectivity among free-ranging deer species. Numerous outbreaks have occurred in commercial captive facilities. Changes in geographic distribution and spread are a result of natural and human-assisted movements of infected live animals. The importance of other potential means of spread remains unclear (transport of infected carcass parts, semen, biological material such as urine, and contaminated forage). Within affected areas, rates of infection and levels of prevalence (the percentage of animals infected) differ widely. The length of time that CWD has been present in an area and harvest management practices influence prevalence, which generally runs higher in deer than in elk or moose in the same location.



WHAT TO LOOK FOR

Adult deer, elk, or moose may appear in poor condition, lack awareness or concern, or otherwise behave oddly. Affected animals in later stages of disease may be thin. Note that animals in early stages of infection may not show visible signs of infection and appear normal.

HERD HEALTH IMPLICATIONS

Infection shortens the lifespan of animals. As an outbreak grows, younger animals are exposed and infected. If infection rates become high enough, CWD can affect a herd's ability to sustain itself. Several western jurisdictions report prevalence among harvested bucks exceeding 10% (1 in 10) in multiple herds and rates approaching 50% (1 in 2) in some populations. A growing body of evidence from studies in heavily infected herds suggests that, at high prevalence, CWD will impair the long-term performance and persistence of deer and elk herds and reduce the amount of harvest they can sustain. Deer herds impaired by CWD will find it harder to be resilient to all the other threats they face.



Photo: George Andrejko

MANAGEMENT

Introductions of CWD in free-ranging populations are very difficult to detect in their earliest stages. Therefore, the majority of “new” CWD areas are well-established when first detected. Increases in disease prevalence and expansion can be slow early on. Individuals genetically predisposed to develop a more rapid form of the disease become less common as prevalence reaches high levels. To date, prevalence has not subsided without active management. Experience suggests CWD can be limited with sustained removal efforts; however, eliminating CWD from the wild is probably not possible with currently available tools. No vaccine or “cure” is available for individual affected animals, so managers rely on hunter harvest or targeted removals to eliminate infected animals, reduce population density and disease prevalence. Data suggest sufficient hunting pressure or removals can suppress CWD prevalence, especially in early stages of the disease progression in a population. Conservative harvest can actually foster growth in disease prevalence and distribution. Measures discouraging deer and elk from artificial congregation in affected populations also may help suppress CWD. Artificial feeding is one of the most likely methods of increasing the prevalence of the disease.

Other measures have been applied – albeit inconsistently – in attempts to prevent further geographic spread of CWD. Stringent rules for monitoring and restricting movements of live deer and elk for commercial and conservation purposes likely help limit new introductions. Controlling the movement and disposal of potentially infectious carcass materials are promoted as important preventive measures. While the role of carcass material in geographic spread remains uncertain, it is important for hunters to follow carcass disposal requirements in areas where they live and hunt. Uneven and inconsistent surveillance in areas where CWD has not been detected hampers assessment of risk, early detection, and preventive measures.

PUBLIC HEALTH CONSIDERATIONS

Minimizing human exposure to CWD seems prudent. Although eating and handling CWD-positive animals has not been associated with any human disease, public health officials advise against consuming meat or any other tissues from infected animals. Hunters should disinfect knives and other equipment, wear gloves when field-dressing animals, avoid handling carcasses of animals that do not appear healthy, and report suspect cases to the responsible state or provincial wildlife management agency so they can be removed from the landscape.