

Chronic Wasting Disease in Deer and Elk in Arkansas

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Chronic wasting disease (CWD) is a neurodegenerative disease found in members of the deer family (**Cervidae**) (Figure 1). It was detected for the first time in Arkansas in a 2.5-year-old female elk (*Cervus canadensis*) legally harvested near Pruitt in Newton County in October 2015. Four months later, in February 2016, a CWD-positive 2.5-year-old female white-tailed deer (*Odocoileus virginianus*) was found dead in Ponca in Newton County. Since then, more elk and deer have tested positive for CWD in Arkansas. CWD is always fatal, though infected animals can carry the disease for many months or years without visible signs. Given the potential negative effects of CWD on deer and elk populations, it is essential that Arkansans understand the nature of CWD, its management and implications for the future. This review highlights current knowledge of CWD in free-ranging deer and elk. Terms in bold print are defined in the glossary.



Figure 1. Animals in latter disease stages often seek water sources. Photo courtesy of Chronic Wasting Disease Alliance and Wisconsin Department of Natural Resources.

What Is CWD?

CWD affects members of the deer family, which in North America includes white-tailed deer, mule deer (*Odocoileus hemionus*), elk, moose (*Alces alces*) and caribou (*Rangifer tarandus*). CWD belongs to a family of unusual diseases known as **Transmissible Spongiform Encephalopathies (TSEs)**, also called prion diseases. CWD is similar to scrapie in sheep and goats, bovine spongiform encephalopathy (mad cow disease) in cattle and Creutzfeldt-Jakob Disease (CJD) and variant CJD in humans. Mad cow disease in humans is commonly called variant CJD.

CWD is caused by prions, which are proteins that multiply by converting normal, nondisease-causing host prion proteins into abnormally shaped, disease-causing forms. Prions are not living organisms like bacteria or viruses. Normal host prion proteins, from which the CWD-causing prions are derived, are called cellular prion proteins (**PrP^C**). PrP^C occur naturally on the cell surfaces of healthy individuals of all mammal species studied to date. PrP^C become CWD-associated prions (**PrP^{CWD}**) when they are folded into an abnormal shape. PrP^{CWD} are relatively resistant to proteases, the cellular enzymes that normally break down proteins. PrP^{CWD} accumulate during the course of the disease in the brain, spinal cord, lymph nodes, spleen, eyes and tonsils in infected cervids. Prion diseases are often called TSEs because nerve tissues accumulate lesions, which are caused by PrP^{CWD} accumulation. The lesions give the

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tissues a spongy appearance when viewed with a microscope. As lesions accumulate in the upper spinal cord, neurodegeneration occurs.

Brief History

CWD was first reported in a captive research mule deer herd in Colorado in 1967 but was not confirmed as a TSE until 1978. Since then, CWD has spread to mule deer, white-tailed deer, elk, red deer (*Cervus elaphus*), sika (*Cervus nippon*), moose and caribou (reindeer) in 24 states, 2 Canadian provinces, South Korea and Norway. By the late 1970s, CWD was detected in captive mule deer and elk in Colorado and Wyoming. In these states, CWD was detected in free-ranging elk in 1981, mule deer in 1985 and white-tailed deer in 1990. In the 1990s, an **endemic zone** for CWD was established in contiguous portions of northeast Colorado, southeast Wyoming and extreme southwest Nebraska. It was not until 1996 that CWD was detected outside this zone. For a complete timeline of the spread of CWD in North America, go to <http://www.cwd-info.org/timeline>.

CWD in Arkansas

The Arkansas Game and Fish Commission has conducted CWD testing on elk since an elk hunting season began in 1998. Statewide surveillance for CWD in white-tailed deer began in 2003. By the time of the first incident of CWD in October 2015, over 200 elk and 7,000 white-tailed deer were tested for CWD in Arkansas. All tested negative for CWD prior to the first case.

In addition to their CWD monitoring efforts, the Arkansas Game and Fish Commission has implemented regulations to prevent CWD from entering Arkansas. Table 1 briefly describes regulations the Arkansas Game and Fish Commission has implemented in recent years to prevent, or postpone as long as possible, CWD from infecting deer and elk in Arkansas.

Given the growing prevalence of CWD nationally, the Arkansas Game and Fish Commission developed a CWD Response Plan in 2006. Goals of the Response Plan are as follows:

1. Determine prevalence and spatial distribution of CWD;
2. Develop and implement management actions to decrease the rate of spread and distribution of CWD;
3. Determine the origin of CWD-positive deer and elk;
4. Continue surveillance statewide to increase the probability of early detection (Figure 2);
5. Provide accurate and relevant information on CWD to Arkansas Game and Fish Commission staff, the public and other stakeholders;
6. Continue to gather and evaluate data that could guide research on CWD, including its epidemiology, to support management of the disease.



Figure 2. By the time of the first incident of CWD in October 2015, over 200 elk and 7,000 white-tailed deer were tested for CWD in Arkansas. All tested negative for CWD prior to the first case. Photo courtesy of Ted Zawislak, Arkansas Game and Fish Commission.

The Arkansas Game and Fish Commission's CWD Response Plan is available for download at <http://www.agfc.com>.

Table 1. Regulations implemented by the Arkansas Game and Fish Commission to postpone chronic wasting disease (CWD) from occurring in Arkansas, 1998-2012. PrPC^{WD} = the prion that causes CWD.

Year	Regulation
1998	CWD testing of harvested elk began.
2002	Importation of live deer banned.
2003	Annual CWD-testing of deer for CWD began.
2005	Importation of deer carcasses from CWD-positive states restricted to deboned meat and cleaned skins and skulls to reduce PrPC ^{WD} transmission. This regulation was implemented because the importation of live deer and deer carcasses originating from CWD-positive states are possible sources of CWD.
2006	Moratorium on breeder/dealer permits and commercial hunting resort permits for new captive deer facilities began.
2012	Restriction on deer carcass importation expanded to include any deer originating outside of Arkansas.
2012	Hand-capturing white-tailed deer prohibited.

Transmission

Research has demonstrated that CWD can be spread directly through animal-to-animal contact and indirectly through prion-contaminated soil or other surfaces. Perhaps the most common mode of transmission from animal-to-animal is saliva, urine and feces. People may have aided the spread of the disease over long distances by moving infected live animals or carcasses. Diseased animals shed infectious prions into the environment where they persist and can be ingested by animals at a later date. Prions originating from infected carcasses are likely an important source of PrP^{CWD}. These prions appear to remain active indefinitely in soil. Additionally, certain plant structures may efficiently bind and retain PrP^{CWD}, thereby preserving them in the environment and acting as reservoirs for the disease. It is unknown, however, how important this reservoir is to the maintenance and spread of CWD.

As previous efforts to eradicate CWD from captive deer facilities have demonstrated, PrP^{CWD} remains infectious in the environment for years and are difficult, if not impossible, to inactivate or remove with practical management and disinfection strategies. Areas of high deer and elk density, such as holding pens in captive facilities or at feeding/bait stations (corn feeders), may serve as sources for infection.

Much is still unknown about CWD. The youngest deer diagnosed with CWD was 17 months old, which suggests the approximate minimum **incubation period**. Without knowledge of when infection takes place, it is impossible to accurately determine the incubation period. Because exposure to prions in natural settings is probably lower than in captive situations, incubation periods in free-ranging deer are probably longer. Once clinical signs appear, a captive deer typically dies within a few weeks to a few months.

Clinical and Pathological Signs

Free-ranging deer and elk in the early stages of CWD may not show obvious **signs** of the disease. In latter stages of the disease, affected animals display abnormal behavior such as uncoordinated stumbling, wide-legged stance and loss of fear of humans (Figure 3). End-stage individuals lose weight rapidly and appear in poor physical condition. Affected deer and elk may also exhibit excessive salivation with slobbering or drooling, excessive urination and excessive drinking. These signs, however, are not diagnostic of CWD; they can be found in other diseases affecting deer and elk, including hemorrhagic disease, brain abscess, meningitis, encephalitis, malnutrition and pneumonia, to name a few.

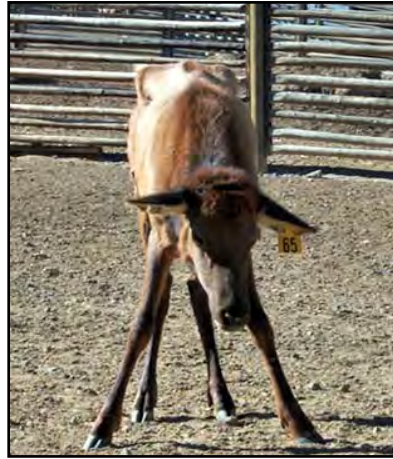


Figure 3. Cervids in the latter stages of CWD exhibit starvation, wide-legged stance, drooping heads and a lack of coordination.
Photo courtesy of CWD Alliance and the Wisconsin Department of Natural Resources.

Identification of lesions and PrP^{CWD} deposition in the **obex** region of the brain stem and retropharyngeal lymph nodes is the only definitive way to diagnosis CWD and rule out other causes. In deer, prions will usually appear in the retropharyngeal lymph nodes before they appear in the obex. Necropsy often indicates aspiration pneumonia as the ultimate cause of death in deer or elk if tissues are not examined specifically for CWD.

CWD Testing

Tests for CWD approved by the USDA APHIS are not food safety tests. Moreover, the tests can be used to establish the presence of PrP^{CWD} only; they cannot be used to establish the absence of PrP^{CWD}. In other words, if examination of the obex and/or retropharyngeal lymph nodes of a deer or elk do not indicate the presence of PrP^{CWD}, that does not mean the animal is CWD-negative. It means that PrP^{CWD} was not detected.

If you have harvested a deer or elk that you wish to have tested for CWD, contact your local Arkansas Game and Fish Commission office (1-800-364-4263). Collect the GPS coordinate where the animal was harvested if possible. (Many phones come equipped with built-in GPS. The Arkansas Game and Fish Commission offers an online locator program [<http://gis.agfc.com/locator/>] for use with cell phones and tablets.) A biologist will collect the appropriate tissues for testing. The head plus 3 to 4 inches of the neck are needed to ensure the sample area won't be destroyed. Antlers can be removed without harming the sample. Do not freeze the head as freezing impedes sampling methods, but the head can be put on ice. Tissues should be sampled within 48 hours for best results.

Treatment and Herd Management

No treatment is available for deer and elk with CWD (Figure 4). While there has been partial preliminary clinical success in vaccination of white-tailed deer in New York, no vaccine has proven effective

thus far). Interestingly, some sheep are immune to scrapie, the TSE that infects domestic sheep and goats. A few researchers have speculated that a natural genetic immunity to CWD probably exists in some deer and elk as well. Unfortunately, no individual deer or elk immune to CWD has been identified to date.



Figure 4. No treatment or vaccine has been developed for cervids exhibiting chronic wasting disease. Photo courtesy of National Park Service.

Although there is no treatment or vaccination for CWD, a number of states and Canadian provinces have adopted management practices aimed at reducing prevalence and limiting distribution. A few of these practices, their benefits, possible problems and implementation are outlined below.

Density Management – This practice focuses on limiting the spread of the disease by lowering animal **density**. While reducing densities cannot eradicate CWD once it is established, it may reduce prevalence and slow the spread of disease. The desired density often reflects scientific estimates of natural (non-human influenced) densities. This may be achieved through removal of animals and/or habitat modification. Although this strategy is likely the most effective at slowing the spread of CWD in a region, several states have indicated difficulty maintaining hunter-performed density-management programs over long periods of time (> 5 years).

Selective Removal – When an animal displaying clinical signs of CWD is observed, it should be killed and tested for CWD. The carcass should be disposed of safely to minimize exposure to other deer. This approach leaves as many deer in the herd as possible while removing those most likely to be infected. Importantly, selective removal will not remove animals before they become able to transmit PrP^{CWD}. Research suggests that this management approach has the potential to significantly reduce disease prevalence, provided infected animals can be identified and removed.

Feed and Bait Management – Reducing the availability and frequency of supplemental feeding or bait stations in order to limit the number of

locations where deer and elk congregate in large numbers may reduce the number of opportunities to transmit PrP^{CWD} from animal to animal. This practice could be implemented through regulatory changes, although it may be unpopular to hunters who have a long history of feeding/baiting. A modification of this regulation involves periodically moving the bait and feeding sites to prevent PrP^{CWD} buildup in the soil. Baiting and feeding could be discontinued during certain times of the year. Additionally, some types of feeders, such as those that do not allow feed to contact the soil, may result in less environmental contamination and disease transmission compared to broadcast feeders.

Human Health and Safety Considerations

For the General Public and Livestock Producers –

There is currently little evidence that CWD can be transmitted to humans. While some researchers have expressed concerns that exposure to CWD-positive deer and elk will increase the prevalence of Creutzfeldt-Jakob Disease in humans, this has not occurred in regions where CWD is considered endemic in deer. There is also no evidence that CWD can be transmitted to domestic livestock such as cattle, sheep or goats. Due to concerns about the development and evolution of **zoonotic diseases**, human exposure to animals potentially infected with any disease should be minimized. Those processing and handling meat from animals killed in areas where CWD is present should limit exposure using the practices outlined below for hunters.

For Hunters – CWD is a disease that affects the nervous system of infected animals. Hunters may feel uneasy about consuming meat from deer and elk in areas where CWD has been found. Whether these prions are capable of mutations that eventually allow it to infect humans is uncertain. However, tens of thousands, possibly hundreds of thousands, of hunters and their families have consumed deer and elk meat from endemic areas over the last several decades without incident. Concerns over the disease should not keep people from hunting. Even in some of the highest CWD-prevalent areas of Wyoming and Colorado, less than 30 percent of free-ranging deer and elk are typically infected. Hunting is one of the best and simplest tools for reducing densities of deer and elk to prevent or slow disease transmission.

Simple health and safety precautions should be employed when handling hunter-harvested deer and elk. By exercising concern over any infectious disease, simple precautions can keep the hunters and their family safe.

- Do not harvest animals exhibiting clinical signs of CWD or any other disease.
- When processing harvested game, hunters should wear gloves and eye protection and

should avoid contact with tissues of the nervous system, especially the brain and spinal cord.

- Do not consume brain or organ meats, especially lymph nodes from the head.
- Bone out the meat carefully minimizing contact with the brain and spinal cord, as they constitute the bulk of the nervous system where PrPC^{WD} accumulate.
- Soak knives and other processing implements in a 40 percent household bleach solution (1/2 cup of bleach in 3/4 cup of water) for one hour. Be aware that bleach will degrade metal surfaces.
- Given the limited health risks to humans, cleaning hands and arms with hot, soapy water is sufficient.
- Using a clean cloth, wipe down processing surfaces with bleach solution followed by hot, soapy water.

Carcass Disposal – Deer or elk exposed to a carcass with CWD could become infected. Particularly in the endemic zone where CWD is found, extra precautions should be taken when disposing of carcasses to prevent transmission to other cervids within and outside the zone. These best practices can be followed statewide to reduce the risk of CWD.

- **Landfills.** Scientific research has shown that prions are extremely unlikely to migrate from a legal landfill disposal site that has a liner. The deer carcass can be taken directly to the landfill or through regular trash pickup service.
- **Bury deep.** Another option is to bury the carcass at least 6 feet deep to prevent scavengers from digging it back up and exposing the carcass to the environment.
- **Leave in place.** The last option is placing the deer carcass waste back on the landscape if taken on your own property. Leave carcass or entrails close to where the deer was found or harvested, ideally at a location where deer and scavengers are unlikely to encounter it.
- **Don't burn.** Open burning, composting, rendering or melting down the carcass will not break down the infectious PrPC^{WD}. High temperature incineration (exceeding 1800-2300 degrees Fahrenheit) is required for destroying PrPC^{WD}. Approved incinerators or tissue digesters may be used by government officials, but they are generally not available to the public.

The Future of Deer Hunting in Arkansas

Due to the environmental resilience of PrPC^{WD}, it is highly unlikely that CWD can be eradicated from Arkansas. Best management practices continue to be those that minimize disease transmission.

Given that deer hunting is culturally and economically important, especially in rural communities, public concern for the future of deer hunting is understandable. In regions where CWD is endemic (i.e., Colorado and Wyoming), deer, elk and moose hunting continues to be a highly sought after commodity whose value has not decreased over time. Deer hunting in Arkansas will continue to be important, although changes in management and hunting practices may become necessary.

If You Observe a Deer or Elk Displaying Signs of CWD

Report any deer or elk showing signs of CWD (Figure 5) to the Arkansas Game and Fish Commission at 1-800-482-9262 or cwdinfo@agfc.ar.gov.



Figure 5. CWD-infected animals may exhibit uncoordinated stumbling, lack fear of humans and excessive slobbering.
Photo courtesy of Warden Micheal Hopper, Kansas Department of Wildlife, Parks and Tourism.

For More Information About CWD

Arkansas Game and Fish Commission
www.agfc.com, 1-800-364-4263

The Chronic Wasting Disease Alliance (a joint project of the Boone and Crockett Club, Mule Deer Foundation and Rocky Mountain Elk Foundation)
www.cwd-info.org

Centers for Disease Control and Prevention
<http://www.cdc.gov>

USDA APHIS Veterinary Services
<https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-disease-information>

USGS National Wildlife Health Center
www.nwhc.usgs.gov

Arkansas Forest Resources Center
www.afrc.uamont.edu

Glossary of Terms

Cervidae – The deer family. North American species in this family include white-tailed deer, mule deer, elk, moose and caribou.

Density – Number of animals per unit area (e.g., the number of deer per square mile).

Endemic zone – An area where a disease is naturally found.

Incubation period – The development of an infectious disease from time of the entrance of the pathogen to the appearance of clinical symptoms.

Obex – A small region in the upper spinal cord close to the brain.

PrP^C – Cellular prion proteins, the normal host prion proteins from which the CWD-causing prions are derived.

PrP^{CWD} – Abbreviation for the malformed proteins called prions that presumably cause CWD.

Signs – The terms *signs* and *symptoms* are often confused. The terms describe the same conditions but are different in several ways. While signs are what you see when you look at an animal (for example, poor body condition, drooling, wide-legged stance), symptoms are what the animal experiences (dizziness, thirst). A symptom can be defined as a characteristic of a disease. A sign, on the other hand, is the physical indication of a disease.

Transmissible Spongiform Encephalopathies

(TSEs) – An unusual family of central nervous system diseases caused by malformed proteins called prions. Prions are characterized by degeneration of the tissues of the central nervous system that leads to neurological dysfunction and death.

Zoonotic disease – A disease that can be transmitted between humans and animals.

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