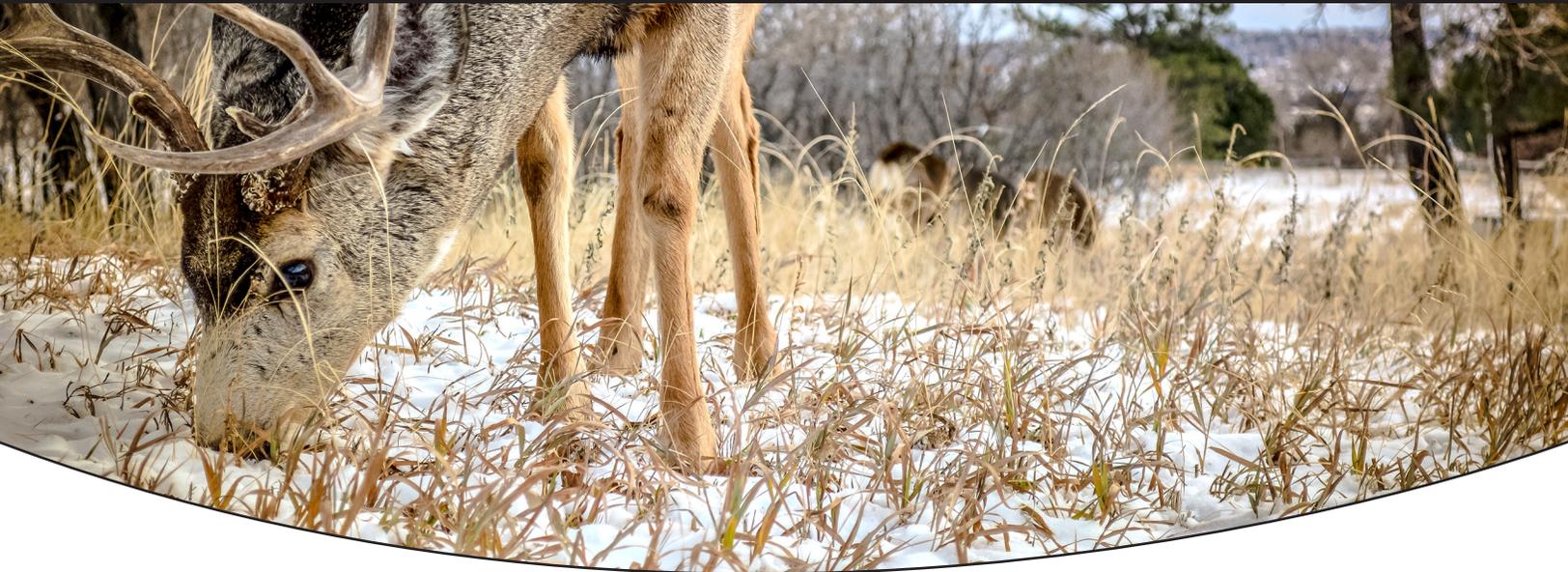


# Chronic Wasting Disease

# FACT SHEET



**C**hronic Wasting Disease (CWD) is an always-fatal nervous system disease found in cervids (deer, elk, moose, reindeer). It can be transmitted through direct animal-to-animal contact, contact with saliva, feces, carcass parts of an infected animal, and can even spread through soil that has been contaminated with any of the above tissues or fluids. Once in the environment, CWD is capable of infecting healthy animals for several years. Below are facts that every sportsmen should know about CWD and information on how you can help prevent the spread of the disease in your area.

- To date, CWD has been found in wild or captive cervids in 25 states, 3 Canadian Provinces, Norway, and South Korea.
- CWD is the result of a naturally occurring protein, called a prion, that becomes misfolded and resists being broken down by the body the way normal proteins are. The disease damages an animal's nervous system and is always fatal because CWD is not caused by a virus or bacteria that can be treated with known vaccines.
- Infected animals are difficult to identify because it may take as long as two years before the animal begins to show outward signs of the disease. It is also rare to see animals exhibiting symptoms of CWD. Animals in the late stages of CWD are often emaciated, show erratic behavior, and exhibit neurological irregularities. Due to the long, slow advancement of the disease, predators, vehicles, and hunters are more likely to kill infected animals well before symptoms of CWD get bad enough to be recognizable.
- There is currently no practical test to determine if a live animal has CWD.
- Besides individual animals, CWD can have negative effects on entire herds of elk, mule deer, and white-tailed deer.
- CWD has not been shown to be infective to humans. Current research indicates that there is a robust species barrier that keeps CWD from being readily transmitted to humans but scientists recommended that humans not consume meat from infected animals.

## COMBATING CWD

Attempts to eradicate the disease have failed, and efforts to control its spread through herd reduction, hunter surveillance, and other methods have had limited success.

The best way to manage CWD is to prevent its introduction into new areas and limit its spread where it is established. To date, CWD has persisted, spread, and increased in prevalence in nearly every area where it has been introduced. The most effective strategies, by far, are those that eliminate ways CWD can travel to new areas. This includes unknowingly transporting infected live animals, or infected animal parts. In places where CWD is present, cervid populations should be managed to reduce their potential to congregate in unnaturally high numbers, or for their populations to increase to unnaturally high totals.

**Hunters! Learn what you can do to help combat CWD.**



# WHAT SPORTSMEN CAN DO

- 1. Keep hunting!** Hunting is critical to the management of CWD, and without the support and participation of hunters, there is little available to wildlife managers to stop the advance of CWD into new herds and new areas. Cervid populations, state wildlife agencies, and all those who care about wildlife depend upon what hunters contribute in the battle to manage CWD. By following carcass disposal guidelines, helping to reduce deer numbers in surveillance areas, and submitting harvested deer for testing, hunters help manage the spread of CWD and inform the science needed to develop more effective CWD management practices.
- 2. CWD is no joke.** It is not a contrived threat that is being overblown. Be aware that much of the information about CWD readily available on the Internet and in popular media is incomplete, heavily biased, or abjectly false. Fortunately, there are numerous sources of reliable, science-based information available. Nearly every state fish and wildlife agency currently managing CWD has an information website where you can learn about what is happening with the disease in your area. These pages can be found by clicking on a state in the interactive map found at the CWD Alliance website. Additionally, both the U.S. Geological Survey National Wildlife Health Center and the U.S. Department of Agriculture's Animal and Plant Health Inspection Service have information websites that host valuable CWD information.
- 3. Get educated and involved.** CWD is an incredibly difficult disease to manage due to its tenacious persistence in the environment, its ease of transmission, its long incubation period, and its difficulty to detect in infected animals. Therefore, the management of this disease demands significant resources and often requires drastic and long-term measures (culling, increased hunter harvest rates, etc.). Your voice is needed to support the agencies and biologists tasked with fighting this disease by advocating for funding and science-based tactics for CWD control.
- 4. Think about your actions.** Remember, CWD can be spread by, 1) animal to animal contact, 2) saliva, feces, and perhaps urine, and 3) contaminated habitats. Any unnatural factor that causes animals to congregate and interact with each other at a higher frequency, a higher density, and a prolonged period of time increases the probability that CWD will be transmitted. Do not feed, bait, or attract cervids to your or your neighbor's property, and if you live in a state or area where such practices are prohibited by law, report any of this behavior to your local wildlife official. How infectious urine can be in transmitting the disease, and to what amount is unknown. To mitigate the risk, several states have banned the use of commercially sold urine-based cover scents and attractants, and others may as well. You should check the laws in your state.
- 5. Help wildlife managers.** Although rare, there have been several cases where concerned wildlife watchers and sportsmen have identified CWD positive animals. If you observe an animal showing emaciation, drooling, or staggering movement, call your state fish and wildlife agency.
- 6. Know the status of CWD regulations** where you hunt, as well as the states you will travel back through with your harvested animal and follow them carefully. Transportation of live animals, infected harvested animals or parts of infected animals is an easy way for CWD to arrive in your neighborhood. Please check with your state fish and wildlife agency on what parts of your harvested animal you are allowed to bring back to your home and how to dispose of them properly. If the state you're hunting in is testing for CWD in cervids, help wildlife managers fight the disease by submitting your harvested animals for testing. Contact the local state fish and wildlife agency for instructions on how to submit a sample.
- 7. Don't use animal attractants** such as grain, other animal feed, or mineral attractants. These and other wildlife feeding practices enhance the risk of spreading CWD. Since infectious prions can persist in the soil and can even be taken up by plants animals feed on, continuing to concentrate animals in one spot only increases the risk of spreading CWD. This may change the way you hunt, but CWD is indifferent to tradition.
- 8. Follow the guidelines** for field-dressing and processing harvested animals in CWD-positive areas. These can be found at <http://cwd-info.org/recommendations-for-hunters-simple-precautions/>.

CWD Fact Sheet  
Provided by



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Visit  
**cwd-info.org**  
for the most up-to-date  
and accurate information

# CHRONIC WASTING DISEASE

## PART 1

This series will give our readers a closer look at chronic wasting disease. It will touch on the various challenges posed by this disease and begin to update you and all hunters about the status of CWD and what science can tell us about it today.

The first part in this series will outline what CWD is and the tangled history of the disease.

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# THE FIRST FIVE (OR MORE) DECADES OF CHRONIC WASTING DISEASE: LESSONS FOR THE FIVE DECADES TO COME

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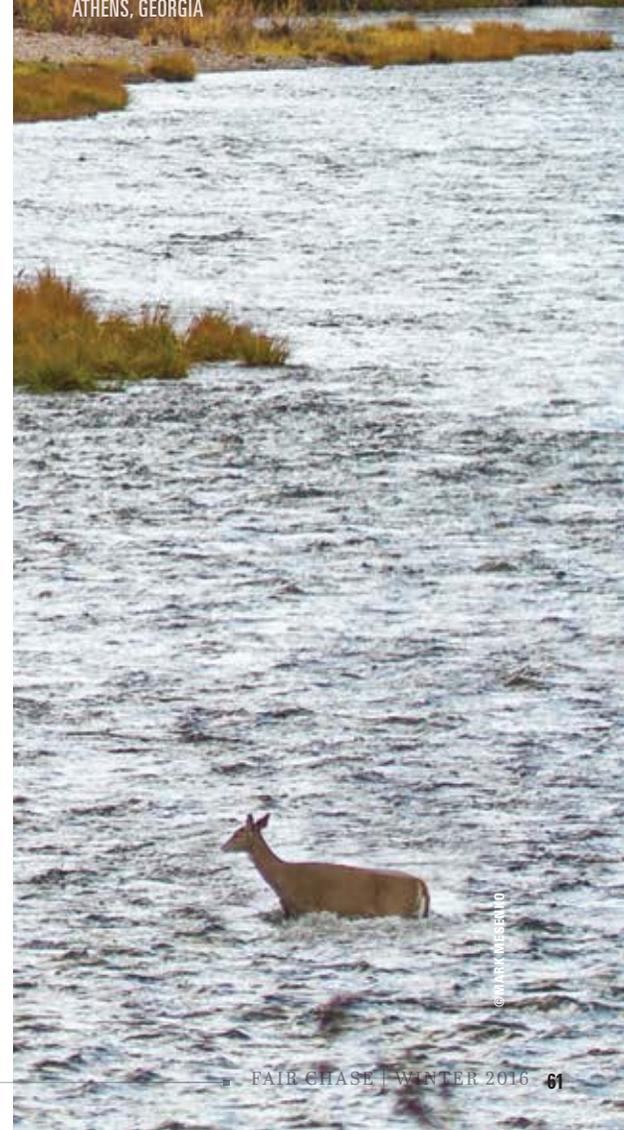
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Chronic wasting disease (CWD), an infectious prion disease of at least five cervid species, has run the gamut from minor scientific curiosity to national crisis since the syndrome was first recognized in the late 1960s.

As of September 2016, CWD had been reported in captive and/or free-ranging cervids in 24 U.S. states, 3 Canadian provinces, South Korea, and Norway. With few exceptions (New York and perhaps Minnesota), the disease has persisted in the wild in the face of widely varied control attempts. Natural and anthropogenic factors have contributed to the geographic spread and persistence of CWD. Natural factors include prolonged incubation, multiple routes of agent shedding, the agent's environmental persistence, and migratory and dispersal movements of wild cervids. Anthropogenic factors include movements of infected live animals (and perhaps infectious tissues and other materials), concentration of normally dispersed

wild cervids, and other artificial wildlife management practices. Many facets of CWD biology and ecology now are well understood, but science-based, effective management and control strategies remain comparatively incomplete. Eradicating CWD appears infeasible given its extensive distribution and other epidemiological attributes. Regardless, adaptive approaches for containing foci and reducing infection and transmission rates have shown some promise and deserve further attention. Such pursuits undoubtedly will be more difficult to champion and garner support for in sociopolitical climates ranging from apathetic to combative, particularly when disease control prescriptions impinge upon or conflict with commercial enclosures or hunting by the general public. We believe there are two important motivations for making progress toward sustainable containment and control strategies for CWD in the coming decades. First, data from several sources suggest that heavily-infected cervid populations will not thrive in the long term. Second, data on CWD prions and experience with other animal prion diseases suggest minimizing human exposure to these agents is prudent.



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## **CHRONIC WASTING DISEASE: LESSONS LEARNED FROM THE FIRST FIVE DECADES**

Chronic wasting disease, an infectious prion disease of at least five cervid species, has run the gamut from minor scientific curiosity to national crisis since the syndrome's first recognition in the late 1960s. Moving forward, we believe this wildlife disease merits attention somewhere between those extremes. Collective experiences and observations made over the last five decades can serve—for better or worse—as a solid foundation for wildlife and animal health professionals to build upon in addressing anticipated challenges posed by CWD in the decades to come. Here we overview what we regard as the key lessons learned over the first five or more decades of North America's experience with CWD.

## **LONGER THAN YOU THINK: BRIEF HISTORY AND KNOWN DISTRIBUTION OF CHRONIC WASTING DISEASE**

That the duration of an outbreak often is underestimated seems perhaps the most important overarching lesson about CWD. Despite its likely occurrence in multiple locations since the 1960s or earlier, many wildlife and animal health professionals, as well as our lay and media publics, perceive CWD as having emerged and spread rapidly only since the early 2000s. This perception has fostered the broader notion that newly discovered disease foci are truly new (very recent) occurrences. To the contrary; given imperfect surveillance approaches, incomplete or inaccurate knowledge about local exposure risks, and the insidious progression of an outbreak in its early stages, the first case detected in a locale is rarely the first case that has occurred. Consequently, on further investigation new foci tend to have larger spatial dimensions and higher prevalence than expected, thereby perpetuating misconceptions about the speed of spread. This lesson has been illustrated by experiences in Colorado and Wyoming, in Saskatchewan, in Wisconsin, and most

recently in Arkansas where expanded surveillance disclosed 79 additional cases within two months after their first case was diagnosed in February 2016.

Chronic wasting disease history remains incompletely documented. The chronic wasting syndrome first was recognized in captive mule deer held for research in Colorado in the 1960s, but unrecognized cases could have occurred in Colorado or elsewhere before that time. Clinical cases also were recognized in captive mule deer in the Denver and Toronto zoos in the 1970s, and in captive Rocky Mountain elk in research and zoological collections in Colorado and Wyoming.

Undocumented involvement of other private collections or menageries during the 1960s and 1970s seems likely. Within little more than the first two decades after its characterization as a transmissible spongiform encephalopathy, CWD cases were diagnosed in wild mule deer, white-tailed deer, and elk in northeastern Colorado and southeastern Wyoming (1980s–1990s), in commercial captive elk facilities in Saskatchewan (1996) and in South Dakota (1997), in commercial captive white-tailed deer facilities in several

jurisdictions (2001–2002), and eventually in wild moose. Cases from what have become recognized as large foci in wild deer in Saskatchewan-Alberta and Wisconsin-Illinois also were first detected in the early 2000s. As of October 2016, cases of CWD had been reported in captive and/or free-ranging cervids in 24 states (77 captive herds in 16 states and free-ranging cervids in 21 states), 3 Canadian provinces (including Ontario's Toronto Zoo in the 1970s), and South Korea. In the spring of 2016, CWD was detected in two free-ranging moose and a single wild reindeer in Norway marking the first detections in Europe. Based on experience to date, the true geographic distribution of CWD likely remains underestimated.

## **TWO GOOD STORIES: THE DRIVERS OF THE SPREAD OF CHRONIC WASTING DISEASE**

A second overarching lesson—a corollary to the first—is that new CWD foci often can be explained by two or more equally plausible (and equally undeniable) origin stories. Distorted temporal perceptions on the likely timing of introduction underlie the plurality of origin stories, as do sociopolitical motivations to deflect or lay blame

**Infected cervids likely shed prions for most of the disease course, thus affording ample opportunities for transmission within and among social groups. Migration movements also have potential for contributing to longer-distance jumps in distribution.**

## CWD THROUGH THE YEARS

YEAR	EVENTS
1967	<ul style="list-style-type: none"> <li>Wasting syndrome observed in captive mule deer at a Colorado wildlife research facility</li> </ul>
1975–81	<ul style="list-style-type: none"> <li>Wasting syndrome observed in Toronto Zoo mule deer that came from the Denver Zoo</li> </ul>
1978	<ul style="list-style-type: none"> <li>“Chronic wasting disease” (CWD) diagnosed as transmissible spongiform encephalopathy (TSE)</li> </ul>
1979	<ul style="list-style-type: none"> <li>Recognized in captive mule deer at Wyoming wildlife research facility</li> </ul>
1981	<ul style="list-style-type: none"> <li>Detected in wild elk in Colorado</li> </ul>
1985	<ul style="list-style-type: none"> <li>Detected in wild mule deer in Colorado and Wyoming</li> </ul>
1996	<ul style="list-style-type: none"> <li>Detected in a captive elk farm in Saskatchewan; 38 other linked farms eventually found positive</li> </ul>
1997	<ul style="list-style-type: none"> <li>Detected in captive elk facilities in South Dakota</li> </ul>
1998	<ul style="list-style-type: none"> <li>Detected in captive elk facilities in Montana and Oklahoma</li> <li><i>Model Program for Surveillance, Control, and Eradication of CWD in Domestic Elk</i> presented at US Animal Health Association to establish monitoring and control standards</li> </ul>
1999	<ul style="list-style-type: none"> <li>World Health Organization indicates no evidence CWD is transmissible to humans, but advises that exposure should be avoided nonetheless</li> </ul>
2000	<ul style="list-style-type: none"> <li>Detected in wild mule deer in Nebraska and Saskatchewan</li> <li>Research: molecular studies compare host ranges for CWD, scrapie, and bovine spongiform encephalopathy prions; environmental contamination and subclinical infection contribute to transmission; prevalence estimates in wild populations in Colorado and Wyoming</li> </ul>
2001	<ul style="list-style-type: none"> <li>Detected in captive elk in Kansas</li> <li>Detected in captive elk in South Korea imported from Saskatchewan</li> <li>Detected in wild white-tailed deer in South Dakota</li> <li>USDA declares CWD emergency in captive elk; funds available for disease control</li> </ul>
2002	<ul style="list-style-type: none"> <li>Detected in captive elk in Minnesota, captive white-tailed deer in Alberta, and wild and captive white-tailed deer in Wisconsin</li> <li>Detected in wild white-tailed deer in Illinois, mule deer in New Mexico, and elk in South Dakota</li> <li>Joint CWD Task Force of USDA/DOI/States/Universities develops <i>Plan for Assisting States, Federal Agencies, and Tribes in Managing CWD in Wild and Captive Cervids</i> (National CWD Plan)</li> <li>Colorado establishes guidelines to minimize transport of high risk carcass materials</li> <li>1st International CWD Symposium (Denver, Colorado)</li> <li>Research: tonsil biopsy as a live animal test; improved high-throughput diagnostics</li> </ul>
2003	<ul style="list-style-type: none"> <li>Detected in wild mule deer in Utah</li> <li>APHIS funds available for CWD work in captive and wild cervids (through 2011)</li> <li>USDA publishes Proposed Rule for CWD herd certification and interstate shipping program (HCP) to eradicate CWD from captive white-tailed deer and elk</li> <li>Research: horizontal transmission of CWD likely important in CWD epidemiology</li> </ul>
2004	<ul style="list-style-type: none"> <li>Detected in wild elk in New Mexico</li> <li>National CWD Plan progress report published and new priorities discussed</li> <li>Research: environmental sources, decomposed carcasses can contribute to transmission</li> </ul>
2005	<ul style="list-style-type: none"> <li>Detected in captive and wild white-tailed deer in New York, wild mule deer in Alberta, moose in Colorado, and white-tailed deer in West Virginia</li> </ul>
2006	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Minnesota and wild white-tailed deer in Kansas</li> <li>USDA publishes CWD HCP Final Rule – never implemented</li> <li>Research: prions in muscles of infected deer; transmitted in saliva and blood</li> </ul>
2007	<ul style="list-style-type: none"> <li>Research: prions in environment more infective in particular (clay) soil types</li> </ul>
2008	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Michigan, wild elk in Saskatchewan, and moose in Wyoming</li> <li>Research: CWD may be a plausible explanation for local deer population declines in Colorado</li> </ul>
2009	<ul style="list-style-type: none"> <li>APHIS plans to withdraw 2006 CWD Final Rule, issue a new rule based on 2006 rule and 2009 proposed rule</li> <li>Research: prions shed in feces from deer in early stages of CWD; prions in urine and saliva</li> </ul>
2010	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Missouri and wild white-tailed deer in North Dakota and Virginia</li> </ul>
2011	<ul style="list-style-type: none"> <li>Detected in wild white-tailed deer in Maryland and Minnesota</li> <li>Severe reduction of USDA funds for CWD work</li> </ul>
2012	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Iowa and Pennsylvania, wild white-tailed deer in Missouri, and wild mule deer in west Texas</li> <li>APHIS Interim Final Rule for CWD Herd Certification and Interstate Movement and CWD Program Standards published</li> <li>Research: possible link between scrapie and CWD</li> </ul>
2013	<ul style="list-style-type: none"> <li>Detected in wild white-tailed deer in Pennsylvania</li> </ul>
2014	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Ohio</li> <li>CWD Program Standards revised</li> <li>APHIS CWD Final Rule implemented</li> <li>Research: plants may play role in CWD transmission and environmental maintenance; experimental aerosol transmission in white-tailed deer</li> </ul>
2015	<ul style="list-style-type: none"> <li>Detected in wild white-tailed deer in Michigan and captive white-tailed deer in Texas</li> <li>Research: plants can bind prions superficially and uptake prions from contaminated soil</li> </ul>
2016	<ul style="list-style-type: none"> <li>Detected in wild elk and white-tailed deer in Arkansas</li> <li>Detected in a wild reindeer in Norway</li> <li>CWD found in two wild moose and a free-ranging reindeer in Norway</li> </ul>

elsewhere when “new” cases arise. But perhaps most pervasive is the lack of complete information on contributory events, particularly for outbreaks involving free-ranging cervids. Although the lack of a singular explanation can be dissatisfying, failing to consider plausible alternative timelines and exposure sources may be more problematic when disease prevention and control efforts are misinformed or misled. For example, the widely held belief that all CWD occurrences can be traced back to a single Colorado research facility has precluded wildlife and animal health professionals from considering that some outbreaks may be arising from unrecognized exposure events that occur repeatedly over time. The recent Norwegian reindeer and moose cases may stimulate broader thinking.

In fact, natural and anthropogenic factors have contributed to the geographic spread and persistence of CWD over the last five decades. Regardless of the

ultimate origin, much of the geographic spread of CWD appears attributable to natural movements in some jurisdictions; Wyoming, for example, has only one private game farm and consequently commercial enterprise is unlikely to have driven the widespread distribution there. Alternatively, the role of commercial elk operations in CWD outbreaks in Saskatchewan and South Korea was well-documented, with inadvertent spillover apparently giving rise to a large free-ranging focus spanning the Saskatchewan-Alberta border. In Colorado, a combination of natural and anthropogenic factors likely contributed in different measures to separate outbreaks along the Front Range and on the Western Slope.

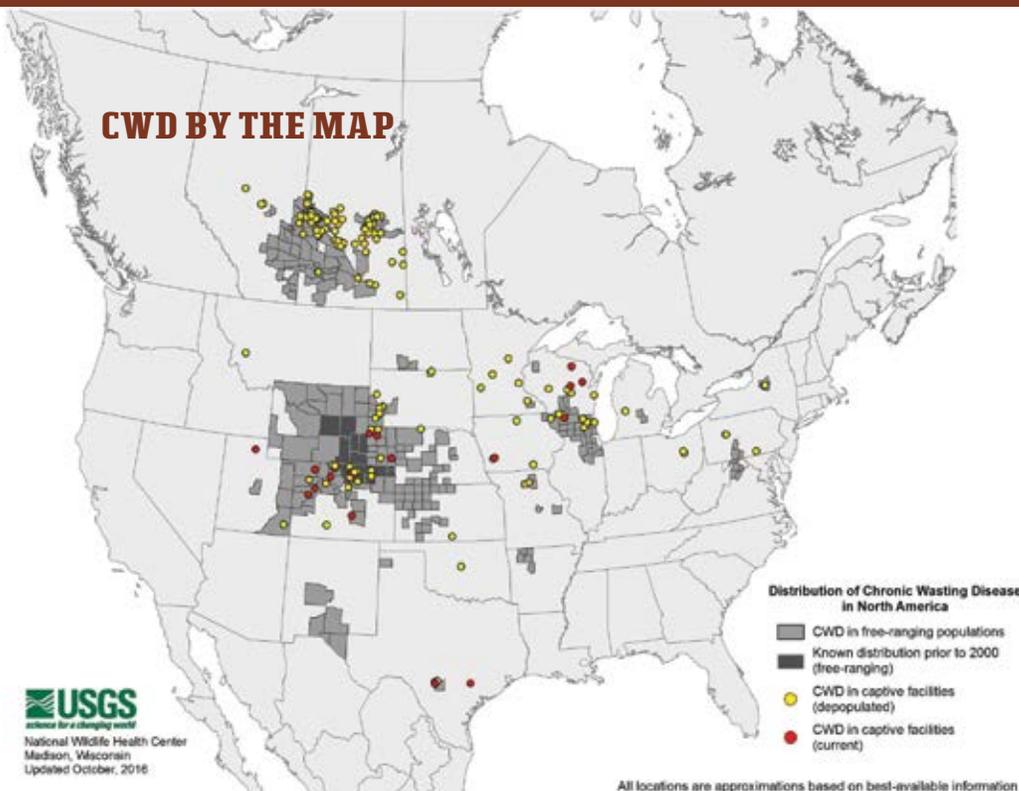
Natural factors contributing to persistence and geographic spread include prolonged incubation, multiple routes of agent shedding, the agent’s environmental persistence, and movements of free-ranging cervids. Infected cervids likely shed

prions for most of the disease course, thus affording ample opportunities for transmission within and among social groups. Migration movements also have potential for contributing to longer-distance jumps in distribution. Because infectivity can be harbored in some environments for an extended time, transmission occurs on overlapping ranges even in the absence of direct interactions between infected and uninfected animals. Indirect transmission also increases the likelihood of interspecies transmission.

The primary anthropogenic factor identified in the dissemination of CWD is human-facilitated movement of live animals, and to date, this is the only confirmed contributing activity linked to CWD’s spread between distant locations. These animal movements typically are fostered by other highly artificial wildlife management activities, such as captive wildlife propagation and high-fenced shooting enclosures. Translocating free-ranging cervids from an

infected source also would present a similar risk for spreading CWD. Local wildlife may be exposed to CWD if infected captive animals escape, or if there is ingress/egress of free-ranging cervids with exposure to infected captive animals or to contaminated environments. Fence-line contact offers another opportunity for direct transmission. (Note that these transmission opportunities are a two-way street, i.e., CWD can move in either direction between captive and wild cervids.) Other possible modes for the anthropogenic spread of CWD include transport of infected carcasses, products manufactured or contaminated with prion-laden deer or elk urine, saliva, or feces, and movement of hay or grain crops contaminated with the CWD agent. None of these has been documented in the field, although proof of concept has been demonstrated experimentally.

In addition, other anthropogenic factors can substantially increase the likelihood of establishing, maintaining, and disseminating CWD and other diseases in free-ranging wildlife. In particular, artificial management activities, such as wildlife baiting and feeding or other practices that congregate normally dispersed wild animals, enhance pathogen transmission opportunities.



Current known distribution of chronic wasting disease (CWD). In addition to North America, cases have been reported in South Korea (captive only) and Norway (free-ranging only). North America map from U.S. Geological Survey (2016).

**THINGS WE NOW KNOW:  
CHRONIC WASTING  
DISEASE BIOLOGY AND  
ECOLOGY**

Many facets of CWD biology and ecology that were mysteries even into the early 2000s now are well understood. For example, notable advances have been made in diagnostics and in our understanding of transmission routes and host factors modulating disease progression that have application in CWD detection and control. These and other advances have been reviewed thoroughly elsewhere; here we offer a brief synthesis of findings most relevant to CWD detection and control, which we will address in the

second article in this series.

Chronic wasting disease appears to be caused by one or more strains of infectious prions. Although the ultimate historical origin never will be known with certainty, we regard exposure of native cervids to the sheep scrapie agent at one or more times and locations as a possible explanation. Regardless of their origin(s), sustained outbreaks now occur as large and small foci in wild cervid populations and in captive wildlife facilities (Fig. 1). Natural cases of CWD have occurred in five host species native to North America: mule deer, white-tailed deer, Rocky Mountain elk, moose, and

reindeer/caribou. No immunity, recovery, or absolute resistance to infection has been documented in any of the susceptible species. However, natural variation in the host gene encoding for cellular prion protein does modulate disease progression, thereby extending survival times and perhaps lowering infection probabilities for relatively resistant genotypes. The disease course typically is measured in years. Clinical signs—altered behavior initially, with body condition declining much later—become progressively apparent relatively late in the disease course. Infection can be detected in carcasses, as well as

in live animals, and diagnostic tests become increasingly reliable in individual animals as the disease progresses.

Chronic wasting disease is infectious. Infected individuals shed prions from several routes during most of the disease course, exposing others either directly or through contamination of shared resources or environments. Shed prions can persist for years in the environment, and their binding to soil elements (e.g., clay) enhances persistence and infectivity. The uncoupling of transmission from the immediate presence of infected animals greatly complicates CWD control. ■

**Part 2 will be featured in the  
Spring 2017 issue of Fair Chase.**

This article is excerpted from the complete paper to be published in the "Transactions of the 81st Wildlife and Natural Resources Conference." It was presented in the special session "Science-based Management Strategies for Fish and Wildlife Diseases" in March 2016. The complete Transactions paper will be available through the website of the Wildlife Management Institute at [wildlifemanagementinstitute.org](http://wildlifemanagementinstitute.org).

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# CHRONIC WASTING DISEASE

## PART 2

This is the second of two *Fair Chase* articles on chronic wasting disease (CWD). The articles are excerpted (and updated) from the complete paper to be published in the "Transactions of the 81st Wildlife and Natural Resources Conference" (Transactions). It was presented in the special session titled "Science-based Management Strategies for Fish and Wildlife Diseases" in March 2016. The complete "Transactions" will be available through the website of the Wildlife Management Institute ([wildlifemanagementinstitute.org](http://wildlifemanagementinstitute.org)).



This series will give our readers a closer look at chronic wasting disease. It will touch on the various challenges posed by this disease and begin to update you and all hunters about the status of CWD and what science can tell us about it today.

# LOOKING HARD- HARDLY LOOKING: DETECTING CHRONIC WASTING DISEASE

Another lesson learned from our first five decades of experience with chronic wasting disease (CWD) is that detecting CWD in captive and wild settings remains difficult despite the considerable effort expended. Most states and provinces have, at least for a time since the early 2000s, engaged in extensive, if not intensive, surveillance to identify affected wild herds. Although these efforts were well-intentioned, many were too flawed or too short-lived to reliably indicate the absence of disease. We briefly review common shortcomings of CWD surveillance as widely practiced to provide a basis for improving the efficiency and effectiveness of future efforts.

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Preferred approaches for detecting CWD in new locations (termed “surveillance” here) differ from approaches for following epidemic trends over time in affected populations (“monitoring”). We recommend that CWD surveillance of wild cervids be an ongoing activity in areas where it has not been detected previously. Monitoring may be more episodic (e.g., at multi-year intervals) when resources are limited because infection rates in wild herds tend to change slowly.

Regardless of the purpose, CWD surveillance and monitoring should be undertaken at a meaningful scale, and any conclusions should reflect the highly patchy distribution of CWD in wild cervids. In our experience, statements indicating that examination of a few hundred (or even a few thousand) harvested animals has proven a state’s freedom from CWD rarely are supported by the data in hand.

In CWD-endemic areas, it has been demonstrated that animals falling into certain categories are more

likely to test positive. These animals may have clinical signs of CWD (emaciation and abnormal behavior), may have been killed by a vehicle or predator, or may be older-age male deer. Consequently, it may be more cost-effective to concentrate testing on animals with a higher probability of infection when surveillance is conducted to detect CWD in new locations than testing large numbers of apparently healthy, hunter-harvested animals. The effectiveness of this type of surveillance assumes relatively even sampling effort over a geographic area, but it does have limitations. For example, clinical disease may not be observed in remote areas, vehicle-killed animals do not occur in roadless areas, and animals killed by predators may be consumed before sampling can occur. In addition to clinical targeting, spatial targeting via risk-based assessments, such as proximity to affected wild populations or captive cervids, also may enhance the effectiveness of CWD surveillance.

For monitoring, random sampling (e.g., from harvested animals) provides relatively unbiased estimates of infection rates. Comparisons over time or between locations should be based on a common denominator (e.g., harvested males aged 2 years or older) to assure that conclusions are reliable. Even though affected areas emerge and grow slowly, infection rates may be remarkably high on first detection when jurisdictions rely on random sampling for surveillance and have not tested adequate numbers of animals at a particular location.

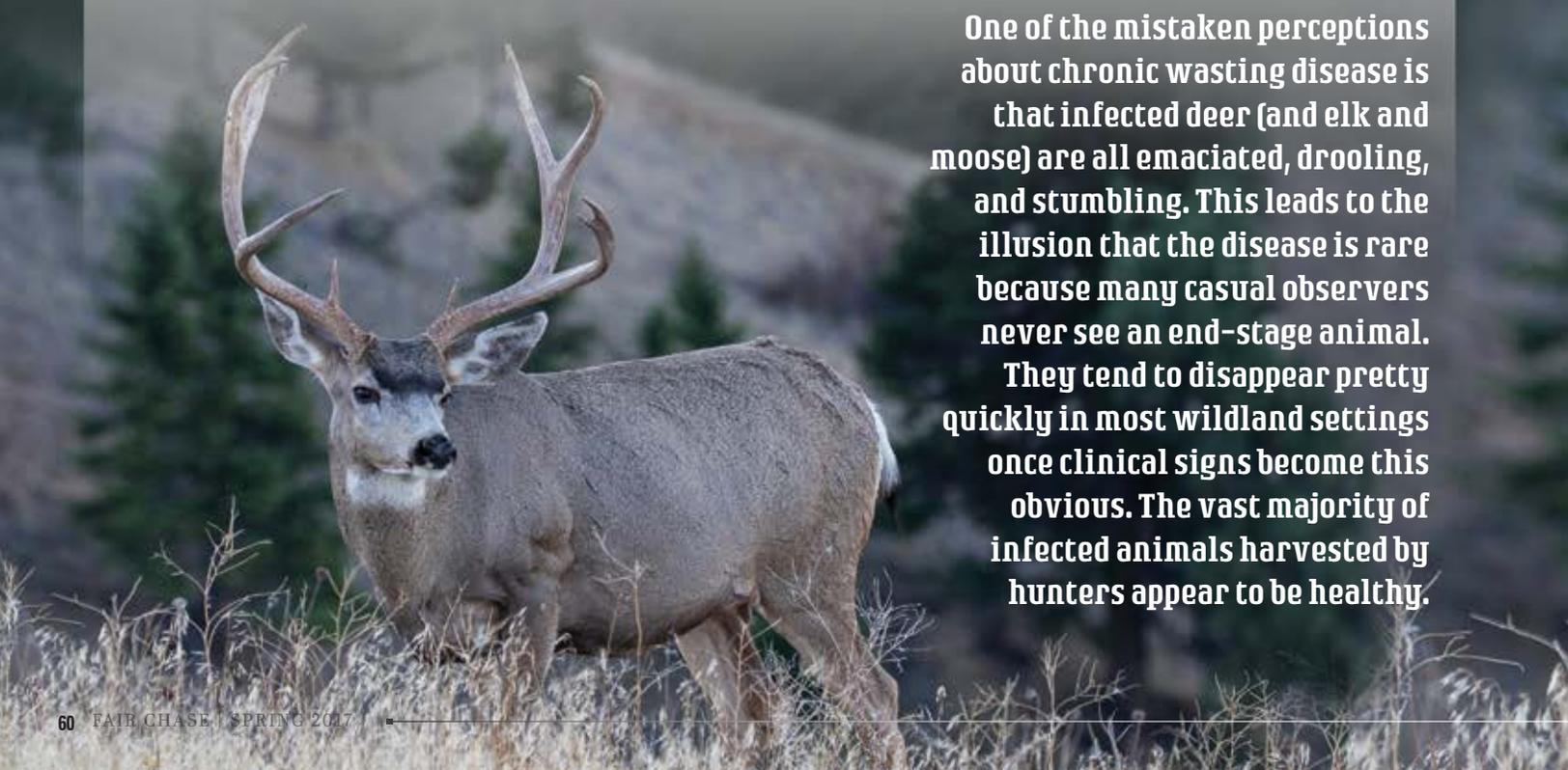
Chronic wasting disease tends to be unevenly distributed in the wild. The notion that a survey sample of 300 assures 95 percent probability of detecting at least one case where prevalence is greater than or equal to 1 percent assumes infection is evenly distributed at that rate throughout the entire target population. However, CWD distribution typically is highly uneven within an affected population, and the target

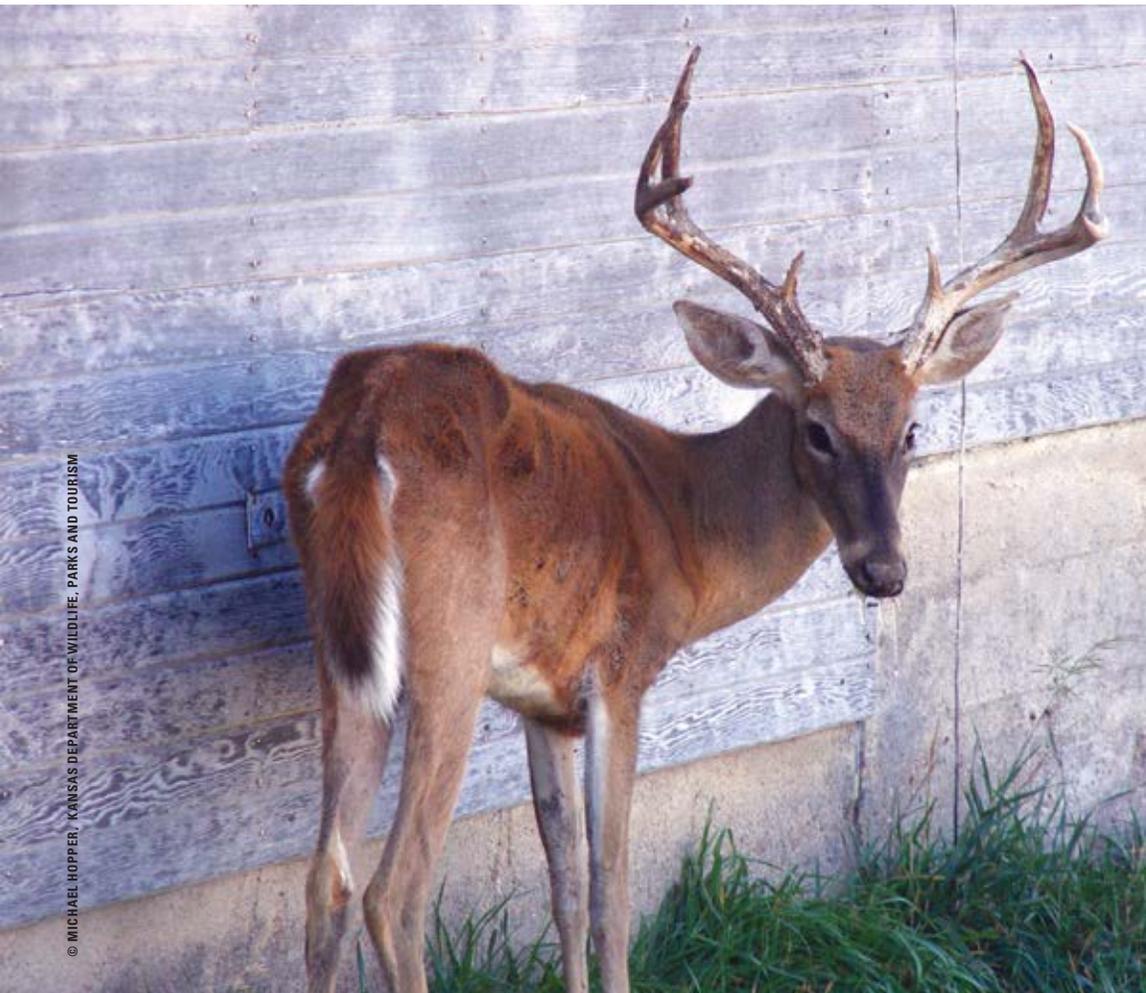
population itself often is distributed unevenly across the area being assessed.

### **TOWARD A SUSTAINED AND SUSTAINABLE EFFORT TO CONTROL CHRONIC WASTING DISEASE**

Eradicating CWD from North America appears infeasible given its extensive distribution and other epidemiological attributes as well as the limited number of available tools. With few exceptions—the detection of two positive deer in New York in 2005 and one positive deer in southeastern Minnesota in 2011 (although CWD has been found in several wild deer in 2016-17 in an adjacent county)—CWD in free-ranging cervids has persisted in affected areas in the face of widely varied control attempts. Faced with dim prospects for eradication, some affected jurisdictions now seem to have abandoned any further consideration of disease management and some have effectively dismantled surveillance and monitoring. In light of numerous

**One of the mistaken perceptions about chronic wasting disease is that infected deer (and elk and moose) are all emaciated, drooling, and stumbling. This leads to the illusion that the disease is rare because many casual observers never see an end-stage animal. They tend to disappear pretty quickly in most wildland settings once clinical signs become this obvious. The vast majority of infected animals harvested by hunters appear to be healthy.**





wildlife conservation needs and ever-dwindling resources, we appreciate the allure but believe this approach should be reconsidered, and we strongly encourage wildlife managers to redouble efforts to collectively develop sustained approaches for CWD surveillance, monitoring, and control.

In contrast to the apparent success in eliminating New York's small free-ranging focus (two wild deer with CWD were detected in 2005 in the vicinity of an affected captive herd), well-publicized early attempts to control CWD in Colorado and Wisconsin yielded little evidence of progress and thus gave initial appearances of failure. In recent years, however, evidence from some control attempts suggests that combinations of intensive deer removal around case clusters,

as well as more sustained reduction of the affected population, may offer some measure of disease suppression. A sustained, localized culling program underway since 2003 has stabilized prevalence in northern Illinois whitetails as compared to the increasing trends in southern Wisconsin where disease control largely was suspended in 2007. Similar divergence in prevalence between deer harvested in Alberta and Saskatchewan may reflect the relative effectiveness of disease suppression efforts in Alberta, but also could be an artifact of more recent CWD emergence there. In north-central Colorado, a combination of focal culling and broader, hunter-harvest population reduction (approximately 25 percent) in the early 2000s appears likely to have contributed to reduced

prevalence, whereas estimated prevalence in other Colorado mule deer herds has increased since 2002.

One of the most common flaws in CWD control efforts to date has been initial underestimation of the affected area (often based on inadequate surveillance and erroneous assumptions about how long CWD has been present). The outcome then gave the appearance that the control attempt had failed when in fact the approach was biologically sound but the application was either too small (spatially) or too short-lived. It follows that acquiring reliable distribution and prevalence data in the planning and early implementation stages may improve the efficacy of future CWD control efforts. Consequently we encourage wildlife managers to set realistic disease-control objectives and to

**Regardless of the purpose, CWD surveillance and monitoring should be undertaken at a sufficient level, and any conclusions should reflect the highly patchy distribution of CWD in wild cervids. In our experience, statements indicating that examination of a few hundred (or even a few thousand) harvested animals has proven a state's freedom from CWD rarely is supported by the data in hand.**

use an adaptive management approach that incorporates future field data to refine objectives and strategies.

In addition to adopting and adaptively assessing approaches for stabilizing or suppressing CWD outbreaks, we encourage wildlife managers to consider how recent trends in cervid management may be contributing to disease establishment. Modeling suggests harvest-based control of CWD may be most effective when focused on male deer, perhaps because infection rates among adult male deer tend to be higher than among adult females. Conversely, then, harvest strategies intended to increase male to female ratios or adult male age structure could inadvertently facilitate CWD persistence. This may explain why the dramatic increases in prevalence observed since 2002 in Colorado in several affected mule deer herds coincide with changes in harvest strategies intended to reduce buck harvest and increase buck to doe ratios over the same period. Given the potential for unintended consequences, we encourage critical assessment of how this and other harvest strategies (e.g., season timing, baiting and/or feeding, "quality deer management") may be affecting CWD dynamics.

Control efforts undoubtedly will be more difficult to champion and garner support for in sociopolitical

climates ranging from apathetic to combative, particularly when control prescriptions impinge upon or conflict with commercial cervid enclosures and/or hunting by the general public. The human dimensions of managing wildlife diseases in general—and CWD in particular—present a substantial challenge for those determining the management objectives and actions. For example, surveys of hunters and landowners in Wisconsin identified several factors that contributed to hunter opposition to the state’s CWD management plan including: opposition to deer

population goals (initially zero); conflicts with traditions; uncertainty about the likelihood of success; questions about agency credibility; and no sense of urgency.

We believe there are two important motivations for responsible wildlife managers to make progress toward sustainable containment and control strategies for CWD in the coming decades. First, data from several sources suggest that an affected whitetail population will not thrive in the long-term. For example, researchers studying of an affected whitetailed-deer population

in Wyoming recently found that CWD-positive deer were 4½ times more likely to die annually than CWD-negative deer, while bucks were 1.7 times more likely to die than does. The researchers concluded that “the strong population-level effects of CWD suggest affected populations are not sustainable at high disease prevalence under current harvest levels.” Second, we believe that existing data on CWD prions and experience with other animal prion diseases suggest minimizing human exposure to these agents would be prudent.

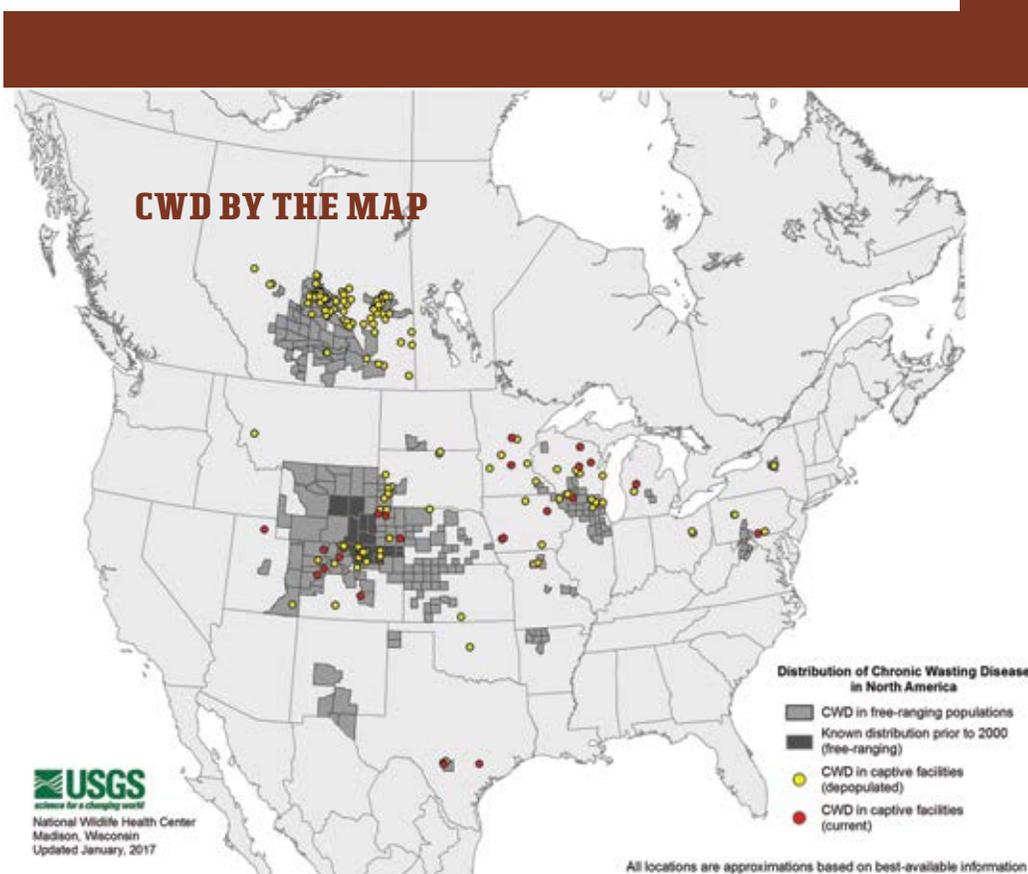
The final overarching

lessons learned over the past five decades relate to how wildlife and animal health professionals should (and probably should not) approach the control of CWD. In contrast to advances in our understanding of CWD biology and ecology, the science informing effective management and control strategies remains relatively incomplete. However, recent insights and modest strides seem to offer a path forward, and adaptive approaches for containing CWD within limited geographic areas and for reducing infection and transmission rates deserve further attention. ■

**The first part of this series was featured in the Winter 2016 issue of *Fair Chase*.**

**CHRONIC WASTING DISEASE: PART 1 REVISITED:**

Chronic wasting disease, an infectious prion disease of at least five cervid species, has run the gamut from minor scientific curiosity to national crisis since the syndrome’s first recognition in the late 1960s. Moving forward, we believe this wildlife disease merits attention somewhere between those extremes. Collective experiences and observations made over the last five decades can serve—for better or worse—as a solid foundation for wildlife and animal health professionals to build upon in addressing anticipated challenges posed by CWD in the decades to come. Many facets of CWD biology and ecology that were mysteries even into the early 2000s now are well understood. For example, notable advances have been made in diagnostics and in our understanding of transmission routes and host factors modulating disease progression that have application in CWD detection and control.



**Current known distribution of chronic wasting disease (CWD). In addition to North America, cases have been reported in South Korea (captive only) and Norway (free-ranging only). North America map from U.S. Geological Survey (2017).**

**This map has been updated since the original production in the Winter 2016 issue of *Fair Chase*.**

## CWD THROUGH THE YEARS

YEAR	EVENTS
1967	<ul style="list-style-type: none"> <li>Wasting syndrome observed in captive mule deer at a Colorado wildlife research facility</li> </ul>
1975–81	<ul style="list-style-type: none"> <li>Wasting syndrome observed in Toronto Zoo mule deer that came from the Denver Zoo</li> </ul>
1978	<ul style="list-style-type: none"> <li>“Chronic wasting disease” (CWD) diagnosed as transmissible spongiform encephalopathy (TSE)</li> </ul>
1979	<ul style="list-style-type: none"> <li>Recognized in captive mule deer at Wyoming wildlife research facility</li> </ul>
1981	<ul style="list-style-type: none"> <li>Detected in wild elk in Colorado</li> </ul>
1985	<ul style="list-style-type: none"> <li>Detected in wild mule deer in Colorado and Wyoming</li> </ul>
1996	<ul style="list-style-type: none"> <li>Detected in a captive elk farm in Saskatchewan; 38 other linked farms eventually found positive</li> </ul>
1997	<ul style="list-style-type: none"> <li>Detected in captive elk facilities in South Dakota</li> </ul>
1998	<ul style="list-style-type: none"> <li>Detected in captive elk facilities in Montana and Oklahoma</li> <li><i>Model Program for Surveillance, Control, and Eradication of CWD in Domestic Elk</i> presented at US Animal Health Association to establish monitoring and control standards</li> </ul>
1999	<ul style="list-style-type: none"> <li>World Health Organization indicates no evidence CWD is transmissible to humans, but advises that exposure should be avoided nonetheless</li> </ul>
2000	<ul style="list-style-type: none"> <li>Detected in wild mule deer in Nebraska and Saskatchewan</li> <li>Research: molecular studies compare host ranges for CWD, scrapie, and bovine spongiform encephalopathy prions; environmental contamination and subclinical infection contribute to transmission; prevalence estimates in wild populations in Colorado and Wyoming</li> </ul>
2001	<ul style="list-style-type: none"> <li>Detected in captive elk in Kansas</li> <li>Detected in captive elk in South Korea imported from Saskatchewan</li> <li>Detected in wild white-tailed deer in South Dakota</li> <li>USDA declares CWD emergency in captive elk; funds available for disease control</li> </ul>
2002	<ul style="list-style-type: none"> <li>Detected in captive elk in Minnesota, captive white-tailed deer in Alberta, and wild and captive white-tailed deer in Wisconsin</li> <li>Detected in wild white-tailed deer in Illinois, mule deer in New Mexico, and elk in South Dakota</li> <li>Joint CWD Task Force of USDA/DOI/States/Universities develops <i>Plan for Assisting States, Federal Agencies, and Tribes in Managing CWD in Wild and Captive Cervids</i> (National CWD Plan)</li> <li>Colorado establishes guidelines to minimize transport of high risk carcass materials</li> <li>1st International CWD Symposium (Denver, Colorado)</li> <li>Research: tonsil biopsy as a live animal test; improved high-throughput diagnostics</li> </ul>
2003	<ul style="list-style-type: none"> <li>Detected in wild mule deer in Utah</li> <li>APHIS funds available for CWD work in captive and wild cervids (through 2011)</li> <li>USDA publishes Proposed Rule for CWD herd certification and interstate shipping program (HCP) to eradicate CWD from captive white-tailed deer and elk</li> <li>Research: horizontal transmission of CWD likely important in CWD epidemiology</li> </ul>
2004	<ul style="list-style-type: none"> <li>Detected in wild elk in New Mexico</li> <li>National CWD Plan progress report published and new priorities discussed</li> <li>Research: environmental sources, decomposed carcasses can contribute to transmission</li> </ul>
2005	<ul style="list-style-type: none"> <li>Detected in captive and wild white-tailed deer in New York, wild mule deer in Alberta, moose in Colorado, and white-tailed deer in West Virginia</li> </ul>
2006	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Minnesota and wild white-tailed deer in Kansas</li> <li>USDA publishes CWD HCP Final Rule – never implemented</li> <li>Research: prions in muscles of infected deer; transmitted in saliva and blood</li> </ul>
2007	<ul style="list-style-type: none"> <li>Research: prions in environment more infective in particular (clay) soil types</li> </ul>
2008	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Michigan, wild elk in Saskatchewan, and moose in Wyoming</li> <li>Research: CWD may be a plausible explanation for local deer population declines in Colorado</li> </ul>
2009	<ul style="list-style-type: none"> <li>APHIS plans to withdraw 2006 CWD Final Rule, issue a new rule based on 2006 rule and 2009 proposed rule</li> <li>Research: prions shed in feces from deer in early stages of CWD; prions in urine and saliva</li> </ul>
2010	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Missouri and wild white-tailed deer in North Dakota and Virginia</li> </ul>
2011	<ul style="list-style-type: none"> <li>Detected in wild white-tailed deer in Maryland and Minnesota</li> <li>Severe reduction of USDA funds for CWD work</li> </ul>
2012	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Iowa and Pennsylvania, wild white-tailed deer in Missouri, and wild mule deer in west Texas</li> <li>APHIS Interim Final Rule for CWD Herd Certification and Interstate Movement and CWD Program Standards published</li> <li>Research: possible link between scrapie and CWD</li> </ul>
2013	<ul style="list-style-type: none"> <li>Detected in wild white-tailed deer in Pennsylvania</li> </ul>
2014	<ul style="list-style-type: none"> <li>Detected in captive white-tailed deer in Ohio</li> <li>CWD Program Standards revised</li> <li>APHIS CWD Final Rule implemented</li> <li>Research: plants may play role in CWD transmission and environmental maintenance; experimental aerosol transmission in white-tailed deer</li> </ul>
2015	<ul style="list-style-type: none"> <li>Detected in wild white-tailed deer in Michigan and captive white-tailed deer in Texas</li> <li>Research: plants can bind prions superficially and uptake prions from contaminated soil</li> </ul>
2016	<ul style="list-style-type: none"> <li>Detected in wild elk and white-tailed deer in Arkansas</li> <li>Detected in wild moose and reindeer in Norway.</li> </ul>

# CHRONIC WASTING DISEASE

## PART 3

This series will give our readers a closer look at Chronic Wasting Disease. It will touch on the various challenges posed by this disease and begin to update you and all hunters about the status of CWD and what science can tell us about it today.



"The disease(s) in question are more than a bit frightening from several standpoints. The disease is not like anything that has been seen before, i.e., it is not a bacteria, not a virus, not a deficiency, not a poison. It is a malformed protein strand that can be transferred somehow between sheep, deer, elk, cattle, and in some rare cases, to people."

Dr. Jack Ward Thomas, 2001

# AN ALLIED FRONT

Until the late 1990s, most hunters, and even wildlife biologists, had very little awareness of what Chronic Wasting Disease was, and nobody was prepared for the impact it would have on hunting and cervid management in the coming decade. But, by 2003, when the disease had been confirmed in eight states and two Canadian provinces, CWD had emerged as an undeniable threat to North America's deer and elk populations. Adding to the growing concern about CWD and its impact to wildlife was the fear that the disease might pose a risk to humans. Media outlets, public health agencies, and sportsmen and sportswomen began asking more questions, but were left only to speculate due to an absence of reliable information and scientifically verified fact.

As CWD was discovered in new areas at an alarming rate during the early 2000s, wildlife management agencies began scrambling to develop techniques to stop or control the disease's spread. At the same time, they were being forced to balance the complex, and often competing or conflicting interests of the general public, hunters, captive cervid industry, traditional livestock industries, and numerous state and federal animal and public health agencies.

**MATT DUNFEE**  
WILDLIFE MANAGEMENT INSTITUTE  
PROGRAM MANAGER  
B&C PROFESSIONAL MEMBER

[www.CWD-Info.org](http://www.CWD-Info.org) has up-to-date information and resources to learn more about CWD in the U.S. and Canada.



**IT WAS IN THIS ENVIRONMENT THAT THE CWD ALLIANCE WAS CREATED.**

As questions, concerns, and fears about CWD and its impacts on wildlife populations and hunting grew, it became clear that strong leadership was needed to ensure that hunters and conservationists had access to timely and accurate information about the disease, as well as a voice in the political debate. In late 2001, three Boone and Crockett Club members provided start-up funds to allow the Club to develop a CWD initiative. Dr. Gary Wolfe, a Professional Member of the Club, was contracted to develop and coordinate the Club's CWD plan. Soon after, the Rocky Mountain Elk Foundation, and the Mule Deer Foundation joined forces with the Club to help fund this important collaborative project. This partnership became known as the CWD Alliance in January of 2002, and the partners agreed to pool resources, share information, and cooperate on projects and activities to positively impact the CWD issue. Since then, over 20 other conservation organizations, sportsmen groups, and industry partners have joined the Alliance.

The early members and partners of the CWD Alliance all recognized that reliable, un-biased public information and education about CWD should be the focus of the Alliance's work. Then, like today, CWD was emotionally and politically charged and inaccurate reporting and sensationalism undermined progress in managing the disease. Thus, the mission of the Alliance was crafted: "To promote responsible and accurate communications regarding CWD, and to support strategies that effectively control CWD to minimize its impact on wild, free ranging cervids."

That mission remains as critical today as it did in 2002. With CWD now found in 24 states and four countries, the concern about the disease's impact to cervid populations has not diminished. Scientific, reliable, and timely information remains at a premium. The flagship project of the Alliance, the website [cwd-info.org](http://cwd-info.org), continues to be the premier information clearing-house for CWD news, updates, and current regulations, ensuring that anyone needing to understand more about CWD has a place to obtain the truth. Over 45 state fish and wildlife agencies link to the site, and it has become a trusted vehicle to disseminate up-to-date information about CWD from a variety of agency partners.

In addition to [cwd-info.org](http://cwd-info.org), the Alliance remains active on numerous other fronts. Over the last decade that Alliance has:

- Provided written and in-person congressional testimony on CWD funding and management issues,
- Helped coordinate and sponsor national CWD symposia,
- Maintained continual media outreach, providing over 120 media interviews,
- Served on dozens of national CWD and wildlife health working groups and committees,
- Produced information brochures and videos on CWD,
- Coordinated CWD information campaigns and resources for other conservation organizations,
- Compiled databases of CWD-related research articles.

**"This is an explosive topic that will, in my opinion, burst onto the ungulate scene (sheep, cattle, deer, elk) and humans within the next year. When it does, there will be dramatic political pressure to do something."**

Past B&C President Earl E. Morgenroth, addressing CWD for the first time in *Fair Chase* magazine, 2001.



In 1981, it was found in the wild elk population in Larimer County, Colorado. The overall infection rate at that time in the area was around four percent, according to Michael Miller of Colorado's Division of Wildlife.

## BOONE AND CROCKETT CLUB'S BOOTS ON THE GROUND.

The Boone and Crockett Club is unique in the fact that many of our members are leading the way or directly involved with CWD research. They work for the state and government agencies trying to understand and manage populations, and are the stakeholders listening to land owners and hunters in the field. Many of our members are board members and active participants of other conservation organizations like the Rocky Mountain Elk Foundation and the Mule Deer Foundation, ensuring that lines of communication stay open and our efforts are organized and cooperative.



In 2002, Fair Chase reprinted an article, *Is it Safe To Hunt?* by David Stalling from the Rocky Mountain Elk Foundation. It was originally written for *Bugle* magazine. The conversation around hunter safety has been going on for 15 years.



Ellis County CWD-Positive WTD, September 19, 2016. PHOTO BY: JASON WAGNER, KDWP

“My experience with CWD includes implementing a statewide surveillance program for a disease considered by many hunters, wildlife managers, policy makers, and researchers as the most devastating disease to have ever affected the nation’s wild cervids. Through national committee work, I help develop strategies to curb CWD’s spread and assist multiple states with legal cases in stopping the movement of this disease. The prion is shed by chronically infected animals that leave habitats infected for years. With no cure or treatment, CWD is spread through animal migrations but more quickly to distant lands in transport trailers. The most effective way to curb its spread is to stop moving animals, both wild caught and owned captive cervids. This one action can slow the spread of CWD to new areas and provide the chance of a healthy wildlife resource for future generations.”

**Colin Gillin serves as the State Wildlife Veterinarian for the Oregon Department of Fish and Wildlife and chairs the Committee on Wildlife Diseases for the US Animal Health Association and the Association of Fish and Wildlife Agencies CWD Standards Working Group.**

“After 20 years of involvement in almost every aspect of CWD, I tend to be more philosophical than scientific in my perspective of this disease. I reflect on human arrogance, which demands a solution to every problem, but knows not the consequences of the solution. I question our empathy for healthy, sentient creatures when our only management solution seems to be a bullet. I despair at how ineffectual human intervention is once a disease takes hold in wildlife. Lastly, I wait to see a comprehensive cost-benefit analysis of current and proposed management efforts to control or eradicate CWD.”

**Terry J. Kreeger, DVM, PhD retired as the State Wildlife Veterinarian with the Wyoming Game and Fish Department. In 2016, he was unanimously elected a lifetime Honorary Member of The Wildlife Society.**

In the state of Kansas it took roughly 7 years after the first detection in 2005 before the first CWD-positive deer with clinical symptoms was observed. On 23 October 2012 in Sherman County, Kansas Department of Wildlife, Parks and Tourism Game Warden Micheal Hopper captured the first photos of a wild, clinical, CWD-positive deer in the state. Reports of clinical CWD-positive deer have increased in the Northwest Zone since Hopper’s encounter. Despite increasing prevalence and geographic spread, no significant deer density declines have been observed in the Northwest Zone using annual distance sampling techniques.

Although interest in CWD among Kansas hunters and the public varies, overall interest in the disease has been stable and slightly increased since CWD was first detected in the state. Even to this day I encounter people who say they have never heard of CWD, or if they have heard of it, are unaware of exactly what the disease is. More importantly, most hunters do not actively pursue having their deer or elk tested for CWD. The reasons for not testing vary, but cost, convenience, access to formalin preservative, knowledge of tissues needed, and lack of concern about CWD are speculated to be the main reasons.

**Shane Hesting, Wildlife Disease Coordinator, Kansas Department of Wildlife, Parks and Tourism**

**2001 - SUMMER ISSUE FROM THE PRESIDENT**

“A small conference of stakeholders is proposed. The objectives of the proposed conference are:

- 1) To foster a free discussion among wildlife veterinarians and a select group of specialists in deer and elk biology in order to relate the etiology of CWD to the ecology and behavior of free-living deer and elk.
- 2) To bring into the discussion representatives of sportsmen organizations and wildlife biologists so as to:
  - (a) develop pilot projects to determine the feasibility of removing sources of CWD infection from the countryside and disposing such safely.
  - (b) develop a system of monitoring to detect infected deer and elk and lead to their removal.

- 3) To develop research projects aimed at rapid decontamination of the countryside.
- 4) To develop a dialogue with agricultural and environmental agencies so as to foster above objectives.
- 5) To organize an administrative structure to continually bring the volunteer potential of organized sportsmen to bear on the problem of CWD.

**2002 - FALL ISSUE FROM THE PRESIDENT**

On May 16th, [2002] the House Resources Subcommittees on Forests and Forest Health and Fisheries Conservation, Wildlife and Oceans held a joint oversight hearing on CWD. The purpose of the hearing was to

probe the growing threat of CWD, and focused on ways federal agencies could support state wildlife managers and other involved state agencies in preventing further spread of CWD to non-infected populations. Additionally, the hearing explored ways in which the federal government could aid and support research and development efforts aimed at containing and, ultimately, eradicating the disease.

Boone and Crockett Club Professional Member, Gary Wolfe, chair of the Club's newly created Wildlife Health Committee was one of ten witnesses invited to present testimony at the congressional hearing. Gary explained the efforts of the Club and the CWD Alliance, and offered specific recommendations for congressional action.



**MESSAGE FROM THE PRESIDENT**  
Chronic Wasting Disease (CWD)



**This is an explosive topic that will, in my opinion, burst onto the ungulate scene (sheep, cattle, deer, elk) and humans within the next year. When it does, there will be dramatic political pressure to do something.**

*Earl E. Morgenroth*

**How They Could Impact Diseases**  
The Future of Big-Game Hunting



In a time when many North American big-game sportsmen are beginning to question the future of big-game hunting, the future of big-game hunting is being questioned. Recent wildlife diseases have caused significant damage to the big-game industry. The future, as projected by the media, seems to change daily with news of new diseases and their potential. It is time to get more knowledge of these diseases, their medical aspects and treatments, and their potential to cross between species. Wildlife management of chronic wasting disease (CWD) and mad cow disease (BSE) and their potential to cross between species are critical to the future of big-game hunting. This issue is a special report on the future of big-game hunting. It is a special report on the future of big-game hunting. It is a special report on the future of big-game hunting.

*Earl E. Morgenroth*



LEFT: Deer are netted and sedated, then fitted with a GPS collar while tests are run and vitals recorded. Winter 2011

<p><b>2001 SUMMER MESSAGE FROM THE PRESIDENT</b> Chronic Wasting Disease Earl E. Morgenroth</p>	<p><b>2002 SUMMER MESSAGE FROM THE PRESIDENT</b> CWD is Topic of Congressional Hearing Earl E. Morgenroth</p>	<p><b>2002 FALL IS IT SAFE TO HUNT?</b> David Stalling Bugle Magazine</p>	<p><b>2003 SPRING DISEASES HOWS THEY COULD IMPACT THE FUTURE OF BIG-GAME HUNTING</b> Michael Lavelle and Kurt VerCauteren USDA/APHIS/WS/National Wildlife Research Center</p>	<p><b>2008 SPRING KNOWLEDGE BASE New Hazards for the Wildlife Biologist</b> Winifred B. Kessler Professional Member</p>
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**2008 -SPRING ISSUE KNOWLEDGE BASE**

Is there some general trend that can explain the emergence of these diseases into the human environment? One theory is that habitat loss and human encroachment into previously wild areas have forced animals

and people into closer contact than ever before. There is greater public awareness about the diseases that jump from animals to people, causing human death and suffering. But zoonoses travel a two-way street. Because they can also jump from humans to animals, zoonoses figure into wildlife conservation and management as well as human health.

**2011 -WINTER ISSUE DEAD DEAR WALKING: CHRONIC WASTING DISEASE IN NEW YORK STATE**

In April 2005 the state of New York got some very, very bad news. A captive whitetailed deer on a farm in central New York tested positive for chronic wasting disease (CWD). Subsequent surveillance identified four more captive deer and two free-ranging deer that tested positive for the fatal disease. In an instant, New York wildlife biologists were faced with one of the most mysterious and unusual wildlife diseases ever reported. CWD had plagued hunters and wildlife professionals in the western U.S. for decades, and in recent years the pernicious disease cropped up in several states and provinces throughout North America. As a newcomer to the east, CWD was unequivocally unwelcome. That's because this tiny, misbehaving protein can cause an awful lot of trouble.

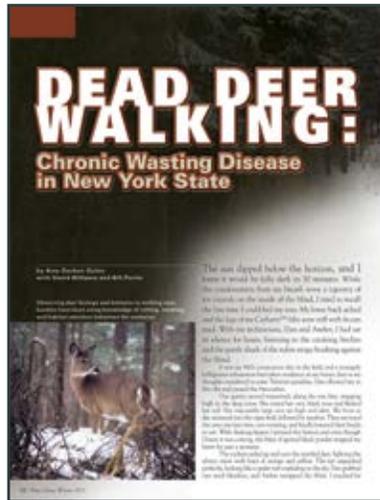
**2014 -SUMMER ISSUE KNOWLEDGE BASE**

A 2011 study by Vaske and Lyon (*Risk Analysis, Vol. 31 No. 3*) looked at CWD-related factors that would influence hunters' decision to give up deer hunting in a state. One factor is CWD prevalence; 52 percent of hunters said they would give up hunting in a state if prevalence of CWD reaches the 50 percent level. Add in the hypothetical situation of a human death due to CWD, and the quit rate jumps to 64 percent. Such studies show how CWD spread undermines the hunting heritage by eroding confidence in a nutritious food source, and by reducing hunter participation and associated revenues needed for wildlife conservation and management.

**2014 -FALL ISSUE FROM THE PRESIDENT**

One key to preventing the spread of wildlife disease is the close monitoring of all captive wildlife. While there is a long history of close inspection and regulation of domestic livestock, captive wildlife generally falls outside of those regulations. The captive wildlife industry is a more recent development. Further, the captive wildlife industry has worked hard to avoid close inspection, which has been one major way that Chronic Wasting Disease has unknowingly spread among captive herds and then to wild herds with devastating effect. Better policy will require changes in legal definitions and jurisdictions concerning

captive wildlife in order to get the right and honest answers on health inspections and control. These are fundamentals that we long ago worked out for other aspects of conservation, and the North American Model must now adapt to address these new developments of wildlife disease.



**What's next? Part four of our CWD series will focus on the disease impacts on cervid populations and the long-term consequences.**

**2010 SPRING KNOWLEDGE BASE Wildlife Diseases**  
Winifred B. Kessler  
Professional Member

**2011 WINTER Dead Dear Walking: Chronic Wasting Disease in New York State**  
Amy Dechen Quinn  
with David Williams and Bill Porter (Professional Member)

**2014 SUMMER KNOWLEDGE BASE CWD: Game Management's Worst Nightmare**  
Winifred B. Kessler  
Professional Member

**2014 FALL FROM THE PRESIDENT Animal Health**  
William A. Demmer

# CHRONIC WASTING DISEASE

## PART 4

This series gives our readers a closer look at Chronic Wasting Disease. It touches on the various challenges posed by this disease and begins to update you and all hunters about the status of CWD and what science can tell us about it today.

We know that male deer become infected with CWD at a higher rate than females. And in whitetail deer, infected males die at a faster rate than infected females. Older deer are also more likely to be infected than young deer. Thus, older males (trophy animals) are the most likely group to be infected with CWD and the quickest to die from the infection.

# PREVENTION EARLY DETECTION CONTROL OF CWD

Chronic wasting disease (CWD) is a newly emerging problem affecting five cervid species that are native to North America (elk, moose, reindeer/caribou, mule deer, and whitetail deer). The disease is caused by a misfolded protein, called a prion, that can be transmitted between animals during contact or by ingestion of prions from a contaminated environment (soil and—potentially—plants are likely sources). Once an animal is infected, the prions propagate and slowly spread throughout its body. They eventually reach the brain where they cause severe neurological damage, clinical signs of disease, and inevitably, death. CWD is considered a chronic disease because this period of disease progression typically takes months to years, depending on factors such as the species and individual's genetic make-up. During most of that period, infected animals look and act completely normal, but they are actively shedding prions that can both infect other animals and contaminate the environment. Unlike most viruses and bacteria, prions are highly resistant to degradation and can persist in the environment for many years, making environmental contamination one of the long-term challenges for CWD management. Currently, we have no cure for CWD or vaccine to prevent infection.

## **MICHAEL D. SAMUEL**

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## **MICHAEL W. MILLER**

SENIOR WILDLIFE VETERINARIAN,  
COLORADO DIVISION OF PARKS AND WILDLIFE,  
FORT COLLINS, COLORADO



In the early stages of a CWD outbreak, the percentage of animals infected (prevalence) is typically quite low—less than 1 percent—and the disease is usually confined to a small geographic area. Over time, usually many years or decades, several patterns typically occur.

CWD belongs to a family of prion diseases that are relatively new to science. The long-term consequences of these diseases are not well understood. However, we have learned a great deal about CWD during the nearly 40 years since it was first described in mule deer. In the early stages of a CWD outbreak, the percentage of animals infected (prevalence) is typically quite low—less than 1 percent—and the disease is usually confined to a small geographic area. Over time, usually many years or decades, several patterns typically occur. In the beginning, prevalence slowly increases as more animals become exposed to CWD by contact with infected individuals or from a contaminated environment. Second, the disease also spreads naturally among animals and expands its distribution across the landscape, facilitated by dispersal of infected juveniles and by migration of herds between summer and winter ranges. Human movement of infected animals or contaminated materials can also contribute to the expanded distribution of CWD. As outbreaks progress, the rate of transmission to susceptible animals accelerates, causing more individuals and younger age classes to become infected, increasing both disease prevalence and the rate of disease spread to new geographic areas. Because clinical CWD is always fatal, increasing prevalence means more animals are infected with CWD and die because of their infection.

Research now clearly shows that heavily infected populations reach a tipping point where CWD infection and mortality causes affected populations to decline. This threshold depends on many factors related to species-specific life history (longevity, birth rate, CWD mortality, and other mortality sources), hunting pressure, and harvest management—and likely varies regionally depending on habitat conditions. Annual herd declines of 10 percent have been found in whitetail deer in Wyoming where CWD prevalence exceeds 40 percent (nearly one in two deer infected). Similar declines have been reported in both hunted and unhunted mule deer herds in Colorado and Wyoming. For an elk herd in Rocky Mountain National Park, researchers reported that losses to CWD can exceed natural mortality, reduce female survival, and cause declines in abundance. These reductions mean that future hunting opportunities also will decline. However, this is not the only important impact of CWD on populations. We know that male deer become infected with CWD at a higher rate than females. And in whitetail deer, infected males die at a faster rate than infected females. Older deer are also more likely to be infected than young deer. Thus, older males (trophy animals) are the most likely group to be infected with CWD and the quickest to die from the infection. Field research in the western U.S. demonstrated that

CWD can reduce the average age of deer (especially males) and dramatically reduce the number of trophy bucks in an affected herd.

CWD has now been found in wild and/or captive cervids in 24 states in the U.S., three Canadian provinces, South Korea, and recently Norway. Given our current scientific knowledge, it is highly unlikely that we can eliminate CWD from North America. Even so, we need management strategies to help control both newly emerging and established outbreaks of this disease to minimize its long-term impacts on our native cervids. The first line of defense against this insidious disease is to prevent it from entering or becoming established in new locations by restricting human activities that might introduce the disease. Many states have implemented preemptive measures designed to reduce this risk, including banning the movement of live animals and contaminated carcasses from infected areas. Some states also have banned urine-based lures, and are evaluating the risk of other materials that may be contaminated with infectious prions.

However, CWD may also spread by natural cervid dispersal or migration. Additional preemptive management approaches, including bans on feeding/baiting and increased male harvest, should be considered in high-risk areas near established outbreaks. These approaches can be combined with rigorous surveillance aimed at

#### Known or Suspected CWD Risk Factors

<b>Exposure Risk Factors</b>	Areas adjacent to CWD-positive wildlife
	Areas adjacent to land on which TSE-positive animals, farmed or wild, have lived
	Areas with CWD-positive farmed or captive herds
	Areas with concentrations of farmed or captive elk or deer
	Areas that have received translocated deer or elk from CWD-affected regions
<b>Amplification Risk Factors</b>	Areas permitting transport of hunter-killed elk or deer carcasses from CWD infected areas
	Areas with high elk or deer population density
	Areas with a history of CWD animals or CWD contaminated environments
	Areas with low abundance of large predators
	Areas where free-ranging elk or deer are artificially concentrated (baiting, feeding, water development, and other human related habitat modifications)

#### Management Goals and Surveillance Objectives

Management Goals	Prevention or Elimination	Elimination, Monitoring, or Control	
	Detection	Assessment	Monitor
<b>Surveillance Objectives</b>	Establish whether CWD occurs in a jurisdiction or part of a jurisdiction; If not detected, estimate likelihood that CWD is absent	Determine the spatial distribution and prevalence of CWD in the target population	Estimate change in prevalence, rate and direction of spread/contraction; Research to understand epidemiology (how CWD is transmitted through a particular target populations); Measure and evaluate the effect of management actions

[www.CWD-Info.org](http://www.CWD-Info.org) has up-to-date information and resources to learn more about CWD in the U.S. and Canada.

early detection of CWD spread into new areas where disease prevalence is still low and before infection is geographically widespread. Early detection, before CWD becomes established, offers the best opportunity to eliminate or control the disease if preventive measures fail. At this early stage of an outbreak, aggressive action should be taken to remove infected animals to reduce transmission to the susceptible population. In addition, early removal of infected animals will help prevent environmental contamination that can infect animals for years into the future. This approach might offer the best hope of completely suppressing a newly emerging CWD outbreak.

Unfortunately, once CWD has become established, control options are limited and so far have proven unsuccessful in eliminating the disease. Current science suggests three theoretically useful strategies to help control CWD once it has become established. First, controlling the rate of disease spread by reducing dispersal of infected juveniles and, where feasible, altering migration patterns. In most cases, changing well-established migration patterns would be a complicated undertaking and may not be feasible or desirable. The obvious approach to reducing dispersal is to cut the size of the affected population, which will mean fewer dispersing juveniles. Second strategy would be to reduce CWD prevalence within an affected population by removing older males, which have high disease prevalence and seem to be an important driver of disease transmission to new animals. Studies on whitetail deer suggest that removal of older males could reduce overall herd prevalence and consequently

reduce the rate of new infection; similar responses would be expected in mule deer. Ideally, this strategy would help control CWD; yet, it will not eliminate it. In contrast, many current deer management strategies encourage higher abundance of older males, which will likely exacerbate CWD infection! Third, localized culling in CWD hot spots with or without more generalized herd reduction also appears to have potential for stabilizing or lowering herd prevalence based on field observations. None of these approaches have yet received widespread application or complete evaluation; therefore, we urge wildlife managers to establish goals for CWD prevention and control, and use adaptive management to evaluate and improve these strategies. We further encourage coordination among different jurisdictions to develop and evaluate alternative CWD management strategies and to assess the effects of past and current management practices on disease trends.

Chronic wasting disease is likely here to stay, and we need to learn how to minimize its future impact on cervid populations and hunting opportunity. We still need continuing research efforts to develop vaccines and other tools to help prevent CWD from becoming established and to control its prevalence and spread when it does, to understand the long-term consequences of the disease on our cervid resources, and to understand several open questions about future trends in CWD infection. Research shows that genetics is an important predictor of CWD infection in cervid populations. While there are no known genotypes that are immune to CWD infection, we are likely to see a long-term shift towards relatively

resistant genotypes in affected populations. Currently we don't understand how this genetic selection will affect the future viability (reproduction and resilience) of our wild populations, whether circulating prion strains may also adapt to shifting host genetics, and whether genotypes with a longer incubation period will also prolong the period to transmission to other animals and deposition in the environment. In addition, there remain many unknowns about prions in the environment: how long do they persist, are they readily absorbed by plants, and what are the main reservoirs that infect susceptible cervids? We know that males become infected with CWD at a much higher rate than females, but we don't know why. Learning how males become infected may help us devise strategies to reduce population prevalence while maintaining a desirable abundance of trophy males. Many of our recent advances were stimulated by the implementation of a national CWD management plan that provided a framework to prioritize and conduct research. Unfortunately, this comprehensive plan has largely been ignored during the past five or more years, particularly in applying research findings to disease management actions. Reviving, funding, and refocusing a national plan, or at least reviving the national conversation about CWD, would provide a crucial catalyst for devising sustainable strategies for a successful long-term battle against CWD in North America and beyond. However, in the long-term, success at preventing and controlling CWD will depend on management actions taken by states and provinces, either individually or collectively. ■

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**We still need continuing research efforts to develop vaccines and other tools to help prevent CWD from becoming established and to control its prevalence and spread when it does, to understand the long-term consequences of the disease on our cervid resources, and to understand several open questions about future trends in CWD infection.**



**The first line of defense against this insidious disease is to prevent it from entering or becoming established in new locations by restricting human activities that might introduce the disease.**

# THINGS YOU MAY HAVE HEARD ABOUT CHRONIC WASTING DISEASE

**“CWD IS NOT NEW NEWS, BUT ITS THREAT TO DEER AND ELK IS INCREASING, WHICH SHOULD BE A CONCERN TO EVERY SPORTSMAN,” SAID BEN B. HOLLINGSWORTH JR., “AS HUNTER-CONSERVATIONISTS, WE’VE HAD TO ADDRESS THREATS TO WILDLIFE BEFORE, BUT NONE LIKE THIS.”**

B&C Press release on Chronic Wasting Disease

**On March 29, 2018, the Boone and Crockett Club officially released a position statement on Chronic Wasting Disease.**

**Read all of our position statements online at [www.Boone-Crockett.org](http://www.Boone-Crockett.org).**

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The prominence of chronic wasting disease (CWD) in the media waxes as new cases are discovered and wanes as the public's interest fades once the disease's presence ceases to be novel. Chronic wasting disease is different from many other deer diseases in that its effects are initially subtle and take months to years to manifest in an animal. The effects of CWD on populations take even longer to become evident. In fact, CWD was first detected in free-ranging deer and elk in the early 1980s and only in the past 10 years have scientists been able to document effects of CWD on populations and even then, only in the areas of Colorado and Wyoming where CWD was first detected.

Chronic wasting disease disrupts the world of people who care about deer and other ungulates. This disruption begins with regulations to determine where the disease is located, to prevent its spread, and sometimes to try to eradicate it before it gains a foothold. Over the long-term, CWD can cause change by decreasing annual survival, thereby reducing the number of deer that can be harvested and eventually lowering deer density. Change is unpleasant and people respond to it in different ways. Some recognize the problem and act to address it. Others deny the change and strive to maintain the world the way it was.



**2) CWD IS NOT A COMMON DISEASE IN THE UNITED STATES. TRUE, BUT MISLEADING.**

People not concerned about CWD state that 97 percent of the 1.17 million deer and elk tested in 14 years had CWD results of “not detected,” and CWD has been detected in only 4 percent of counties in the United States. These facts are not disputed, but their implication is disputed on two accounts.

First, CWD is not common in the United States, but neither is meningococcal disease (human disease with 1.2 cases/1 million people). However, rarity does not mean unimportant. Just as human health officials act quickly when meningitis is detected in the United States, so too should wildlife managers do everything they can to keep from having CWD become established in their ungulate herds. In fact, CWD differs from meningococcal disease in some critical ways. Meningitis outbreaks can be managed and the disease can be eradicated from an area. In contrast, CWD cannot be eradicated once established because the abnormal prion proteins (the disease agent) persist in the environment; all indications are that the disease will be present in the environment for years or even decades. The best offense against CWD is a rock-solid defense to keep CWD from being introduced into a deer population. Thus, the goal of all CWD management programs is to keep the disease rare.

Second, CWD is not equally prevalent across the United States, so defining the population as deer throughout North America is misleading. The disease has not been detected in most places in the United States. But, where CWD has been detected, it can have high prevalence. As might be expected, prevalence increases the longer the disease has been present in an area. Thus, some populations in the endemic areas of Colorado and Wyoming have prevalence ranging from 20 to 40 percent.

There is a campaign, arising from fear of the change caused by CWD, to marginalize CWD as a management issue. The campaign has been intense, occurring in presentations, lay publications, and the internet. Tellingly, the central ideas behind this campaign have never appeared in a publication subject to scientific peer-review, and in fact, are in direct contrast to the recommendations of wildlife veterinarians and epidemiologists. Although CWD has been a topic of intense discussion for several years, we constantly field questions from concerned land stewards about the disease. This article provides a counterweight to things you may have heard about CWD from those who feel CWD is of no significance.

**FOLLOWING ARE 7 STATEMENTS YOU MAY HAVE HEARD ABOUT CWD AND REASONS WHY THESE STATEMENTS ARE MISLEADING AT BEST AND ABSOLUTELY FALSE AT WORST.**

**1) CWD IS NOT A DISEASE.**

Because CWD does not have obvious and immediate impacts and because it is not caused by a typical disease agent, such as a virus or bacterium, deniers suggest that CWD is not a disease, but simply a condition or a syndrome. It is important to note that CWD is always fatal, is caused by a disease agent, can be spread from infected animals to healthy animals, and causes holes to form in the animal’s brain, resulting in the animal progressively losing its ability to avoid danger, eat food, and walk or function normally. CWD is clearly a disease and is listed as a disease by the Centers for Disease Control and Prevention.



**PREVENTING MOVEMENT OF INFECTED DEER AND INFECTED TISSUE INTO AREAS WHERE CWD DOES NOT OCCUR IS THE MOST POWERFUL TOOL IN THE MANAGER’S TOOL KIT.**



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### 3) CWD IS NOT SPREADING RAPIDLY.

**FALSE**

To the contrary, for a disease first detected in free-ranging deer in the 1980s with no natural vector such as an insect to spread it, CWD has spread remarkably fast. From an endemic area in northcentral Colorado and southeast Wyoming, the range of the disease increased over a 20-year period to the lake states, the Midwest, New Mexico, and central Canada. By 2005, the disease was present in the central Appalachians, and around 2010, the number of states in which CWD was detected grew dramatically. The disease has also been found outside of the United States, such as in South Korea and Norway. Some of the spread of the disease outside the endemic area is likely a result of natural deer and elk movements. Other occurrences are best explained by movement of CWD-positive elk or deer or their tissue. Again, the disease has spread remarkably fast, given what is known about its transmission. Responsible wildlife managers should strive to keep CWD from spreading into the herds they manage.

### 4) CWD CANNOT BE PREVENTED OR CONTROLLED, ONLY MONITORED.

Clearly, CWD can be prevented from infecting a population. Preventing movement of infected deer and infected tissue into areas where CWD

**FALSE**

does not occur is the most powerful tool in the manager's tool kit. Once introduced, the options decline dramatically. Managers in New York detected two positive wild deer in the vicinity of an infected captive deer facility in an isolated CWD outbreak in 2005. Acting swiftly, deer managers in New York harvested deer heavily in the outbreak area, and CWD has not been detected in the area since. The case in New York appears unique because CWD was detected early. In most instances, the disease is more well established once detected and there are no viable options for eliminating it. The rate of spread and the rate at which prevalence increases may be slowed by reducing deer density, but the effectiveness of these approaches is still unclear. And, because they must be instituted for years or decades, these approaches may be opposed by groups who desire higher deer densities.

**IT IS IMPORTANT TO NOTE THAT CWD IS ALWAYS FATAL, IS CAUSED BY A DISEASE AGENT, CAN BE SPREAD FROM INFECTED ANIMALS TO HEALTHY ANIMALS, AND CAUSES HOLES TO FORM IN THE ANIMAL'S BRAIN, RESULTING IN THE ANIMAL PROGRESSIVELY LOSING ITS ABILITY TO AVOID DANGER, EAT FOOD, AND WALK OR FUNCTION NORMALLY.**

### 5) NO PEER-REVIEWED PAPER TO DATE CLEARLY SHOWS A CAUSE-AND-EFFECT RELATIONSHIP BETWEEN RECENT DEER DECLINES AND CWD.

This statement harkens back to the tobacco companies' strategy of denying tobacco causes cancer and can shorten people's lives. Cancer may have several causes and it may be difficult to conclude

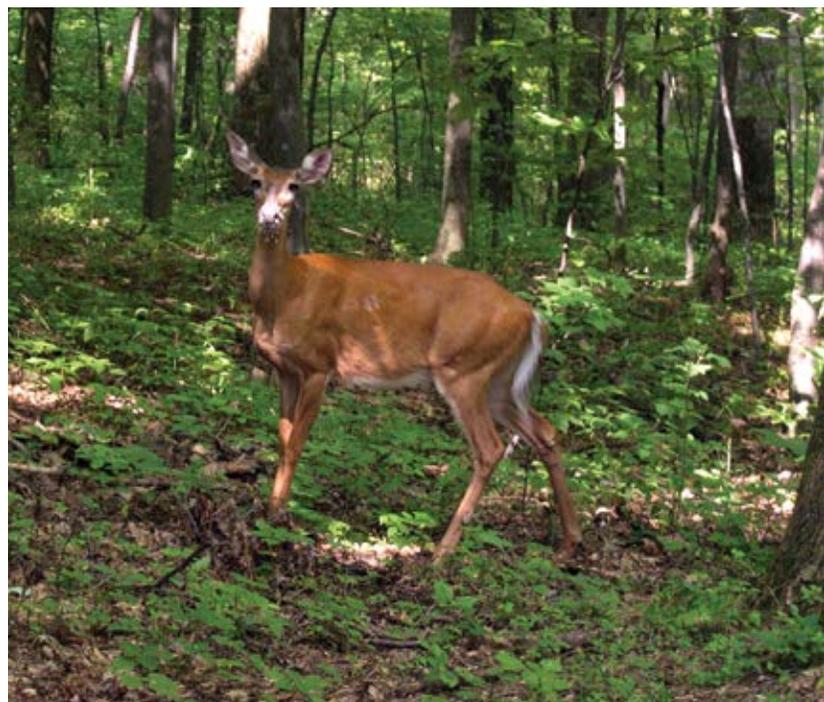
**FALSE**

a given cancer is caused by tobacco use, but that does not mean tobacco is safe to use. To the contrary, the link between premature death and tobacco use is clear. Similarly, deer and elk herds may decline for many reasons, but scientists have techniques that allow them to isolate the cause of population declines. There are now three peer-reviewed studies demonstrating cause and effect relationship between deer declines and CWD. And the effects are not subtle. Population estimates were available for two of the populations, and those populations declined 40-50 percent over 10-20 years. Survival of CWD-negative deer in these studies was 30-40 percent greater than survival of CWD-positive deer (e.g., survival of CWD-negative deer—85 percent vs. CWD-positive deer—50 percent). Some deer populations have high enough reproductive rate to avoid declines, but many do not, especially in the semi-arid south and western Texas (see point 7).

### 6) CWD DOES NOT KILL DEER.

This statement is simply not true because wild and captive deer die of clinical CWD; the disease is always fatal. However, many CWD-positive deer do not die strictly from CWD, just as people positive for AIDS may not die from AIDS. Rather, AIDS-positive people often die of infections that their body could not defeat because AIDS had compromised their immune system. Similarly, deer in free-ranging populations may not die of CWD, but, as might

**FALSE**



be expected of an animal with holes in its brain, these deer are susceptible to other factors. CWD-positive deer are more vulnerable to predators, hunters, and vehicle collisions. Deer with CWD may be less motivated to forage or forage less efficiently. Poor foraging ability makes these deer more susceptible to death during winter or other periods of stress. As an example of the impact of CWD on deer survival, whitetail deer in Wyoming that were positive for CWD, even if they did not show clinical symptoms, had a 40 percent chance of surviving one year, whereas deer without CWD in the same population had an 80 percent chance of surviving one year. These findings are especially meaningful because both the CWD-negative and CWD-positive deer were in the same area and exposed to the same threats of mortality. The only difference was that some had contracted CWD and others had not.

**7) BECAUSE CWD TAKES 14 MONTHS TO 4 YEARS TO DEVELOP AND BECAUSE POPULATION GENERATION TIME IS 3.5 YEARS, THE VAST MAJORITY OF DEER NEVER LIVE LONG ENOUGH TO BE CLINICAL TO CWD. THEREFORE, CWD, BY ITS CLINICAL NATURE, CANNOT BE A FACTOR IN TEXAS DEER HERD MANAGEMENT, PRESENT OR FUTURE.**

This statement is rife with misunderstanding of the importance of adult survival in deer populations, especially deer populations in semi-arid rangelands of Texas. Using South Texas as an example, about 30 percent of the adult deer are typically 6 years old or older in sustainably harvested deer populations. The only way for this age structure to develop is for adult survival to be high. High adult survival is critical to persistence of deer populations in South Texas because fawn-to-doe ratios may average 30 fawns/100 does. This level of fawn production is low and reflects the challenging nutritional environment of South Texas, especially during drought. In fact, fawn production in South Texas is just sufficient to allow the deer population to grow at a slow rate and therefore to support a light recreational harvest. Contrary to the suggestion that there is no problem with deer dying before they reach 4-years of age, such high adult mortality would cause deer populations in South Texas to decline rapidly. To compound the problem of CWD for deer populations in South Texas, female deer rarely raise a fawn until does reach 3 years old. Even though they typically conceive for the first time as yearlings and give birth on their second birthday, autumn lactation rates and maternity determined using genetic techniques show that 2-year-old females rarely raise a fawn. For this reason, high mortality before 4-years of age from CWD would be a big problem for deer populations in South Texas and likely elsewhere in the state.

**FALSE**

Deer managers in South Texas often alleviate the constraints of low fawn production by providing supplemental feed. Supplemental feed makes a huge difference, for example by doubling fawn production and therefore removing many of the concerns described in the preceding paragraph. However, providing supplemental feed is likely to turbo-charge the spread of CWD in a deer herd. For this reason, it would be wise to cease supplemental feeding if CWD may be present.

One final problem with high mortality before deer turn 4-years of age is that antler size, a characteristic of interest to many deer hunters, increases with age through at least 5 years of age, and some bucks show their largest antlers at age 6 or more. An additional problem is that bucks also have higher prevalence of the disease than does. So, not only do deer populations decline with CWD but the number of mature bucks would decline even more dramatically. CWD will be a big problem for hunters who enjoy hunting trophy bucks.

### CONCLUSION

The mass of scientific information and expertise suggests that CWD is a disease we do not want in our deer populations. It has no positive effects and many known and potential negative impacts. In addition, the CDC recommends that hunters not consume meat from animals that test positive for CWD. For all these reasons, it makes sense to take actions to keep CWD from being introduced into deer populations without the disease. Future generations of hunters will thank us for doing all we can to keep CWD rare. ■

**THE BEST OFFENSE AGAINST CWD IS A ROCK-SOLID DEFENSE TO KEEP CWD FROM BEING INTRODUCED INTO A DEER POPULATION.**

