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).

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.

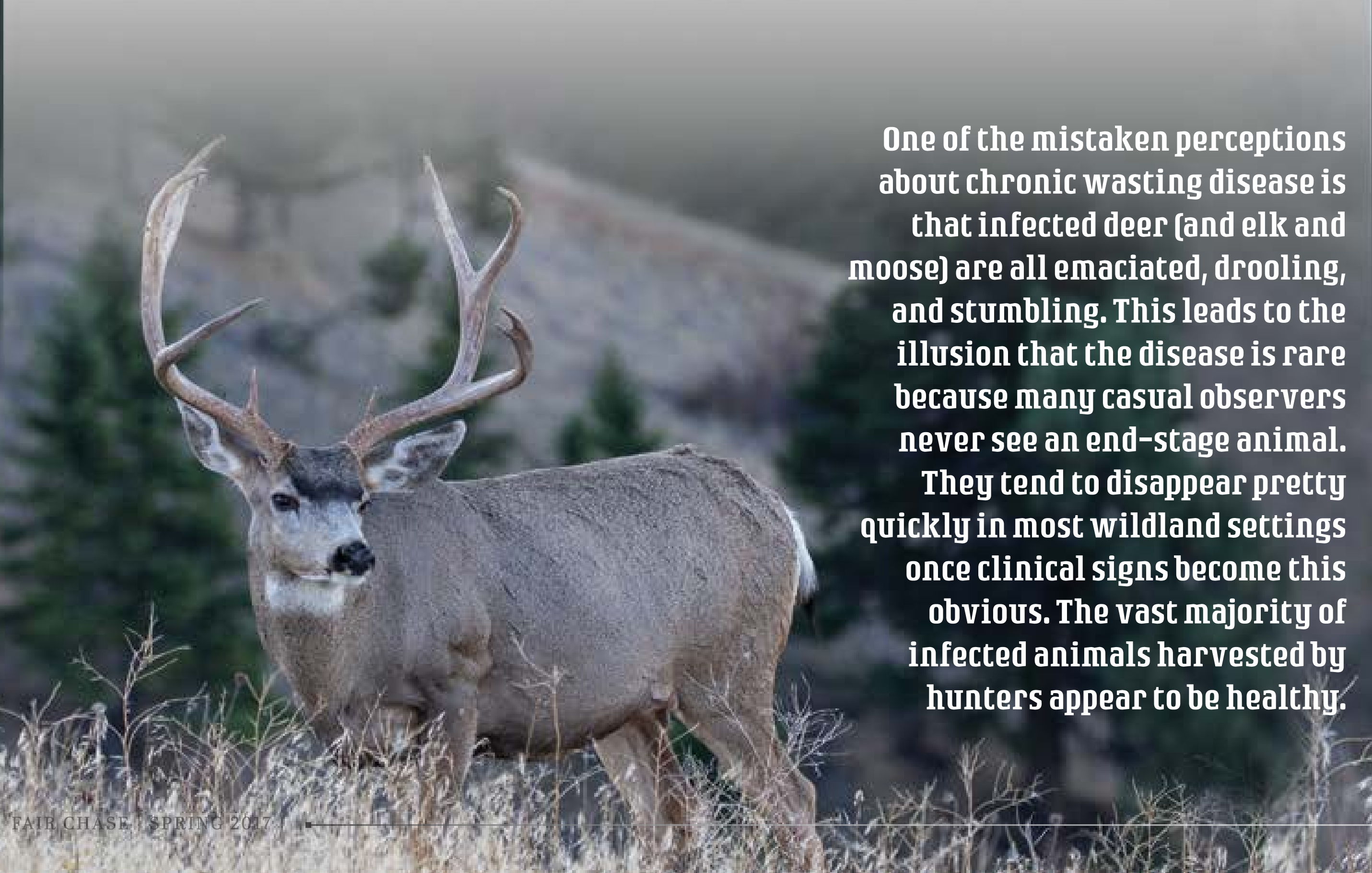
In CWD-endemic areas, it has been demonstrated that animals falling into certain categories are more likely to test positive. These animals may have clinical signs of CWD (emaciation and abnormal behavior), may have been killed by a vehicle or predator, or may be older-age male deer. Consequently, it 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.

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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.

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 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.

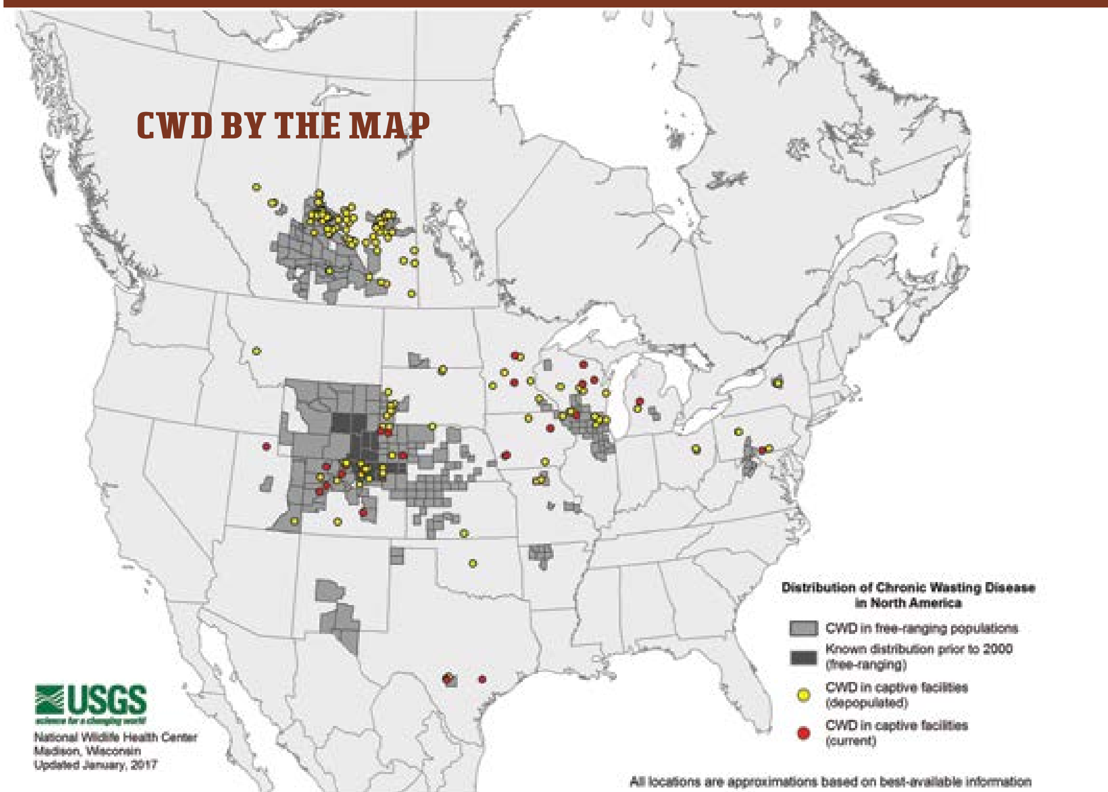
We believe there are two important motivations for responsible wildlife managers to make progress toward sustainable containment and control strategies for CWD in the coming decades. First, data from several sources suggest that an affected whitetail population will not thrive in the long-term. For example, 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 n the Winter 2016 issue of *Fair Chase*.

CWD THROUGH THE YEARS

YEAR	EVENTS
1967	■ Wasting syndrome observed in captive mule deer at a Colorado wildlife research facility
1975–81	■ Wasting syndrome observed in Toronto Zoo mule deer that came from the Denver Zoo
1978	■ "Chronic wasting disease" (CWD) diagnosed as transmissible spongiform encephalopathy (TSE)
1979	■ Recognized in captive mule deer at Wyoming wildlife research facility
1981	■ Detected in wild elk in Colorado
1985	■ Detected in wild mule deer in Colorado and Wyoming
1996	■ Detected in a captive elk farm in Saskatchewan; 38 other linked farms eventually found positive
1997	■ Detected in captive elk facilities in South Dakota
1998	■ Detected in captive elk facilities in Montana and Oklahoma ■ *Model Program for Surveillance, Control, and Eradication of CWD in Domestic Elk* presented at US Animal Health Association to establish monitoring and control standards
1999	■ World Health Organization indicates no evidence CWD is transmissible to humans, but advises that exposure should be avoided nonetheless
2000	■ Detected in wild mule deer in Nebraska and Saskatchewan ■ 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
2001	■ Detected in captive elk in Kansas ■ Detected in captive elk in South Korea imported from Saskatchewan ■ Detected in wild white-tailed deer in South Dakota ■ USDA declares CWD emergency in captive elk; funds available for disease control
2002	■ Detected in captive elk in Minnesota, captive white-tailed deer in Alberta, and wild and captive white-tailed deer in Wisconsin ■ Detected in wild white-tailed deer in Illinois, mule deer in New Mexico, and elk in South Dakota ■ Joint CWD Task Force of USDA/DOI/States/Universities develops *Plan for Assisting States, Federal Agencies, and Tribes in Managing CWD in Wild and Captive Cervids* (National CWD Plan) ■ Colorado establishes guidelines to minimize transport of high risk carcass materials ■ 1st International CWD Symposium (Denver, Colorado) ■ Research: tonsil biopsy as a live animal test; improved high-throughput diagnostics
2003	■ Detected in wild mule deer in Utah ■ APHIS funds available for CWD work in captive and wild cervids (through 2011) ■ USDA publishes Proposed Rule for CWD herd certification and interstate shipping program (HCP) to eradicate CWD from captive white-tailed deer and elk ■ Research: horizontal transmission of CWD likely important in CWD epidemiology
2004	■ Detected in wild elk in New Mexico ■ National CWD Plan progress report published and new priorities discussed ■ Research: environmental sources, decomposed carcasses can contribute to transmission
2005	■ 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
2006	■ Detected in captive white-tailed deer in Minnesota and wild white-tailed deer in Kansas ■ USDA publishes CWD HCP Final Rule – never implemented ■ Research: prions in muscles of infected deer; transmitted in saliva and blood
2007	■ Research: prions in environment more infective in particular (clay) soil types
2008	■ Detected in captive white-tailed deer in Michigan, wild elk in Saskatchewan, and moose in Wyoming ■ Research: CWD may be a plausible explanation for local deer population declines in Colorado
2009	■ APHIS plans to withdraw 2006 CWD Final Rule, issue a new rule based on 2006 rule and 2009 proposed rule ■ Research: prions shed in feces from deer in early stages of CWD; prions in urine and saliva
2010	■ Detected in captive white-tailed deer in Missouri and wild white-tailed deer in North Dakota and Virginia
2011	■ Detected in wild white-tailed deer in Maryland and Minnesota ■ Severe reduction of USDA funds for CWD work
2012	■ Detected in captive white-tailed deer in Iowa and Pennsylvania, wild white-tailed deer in Missouri, and wild mule deer in west Texas ■ APHIS Interim Final Rule for CWD Herd Certification and Interstate Movement and CWD Program Standards published ■ Research: possible link between scrapie and CWD
2013	■ Detected in wild white-tailed deer in Pennsylvania
2014	■ Detected in captive white-tailed deer in Ohio ■ CWD Program Standards revised ■ APHIS CWD Final Rule implemented ■ Research: plants may play role in CWD transmission and environmental maintenance; experimental aerosol transmission in white-tailed deer
2015	■ Detected in wild white-tailed deer in Michigan and captive white-tailed deer in Texas ■ Research: plants can bind prions superficially and uptake prions from contaminated soil
2016	■ Detected in wild elk and white-tailed deer in Arkansas ■ Detected in wild moose and reindeer in Norway.