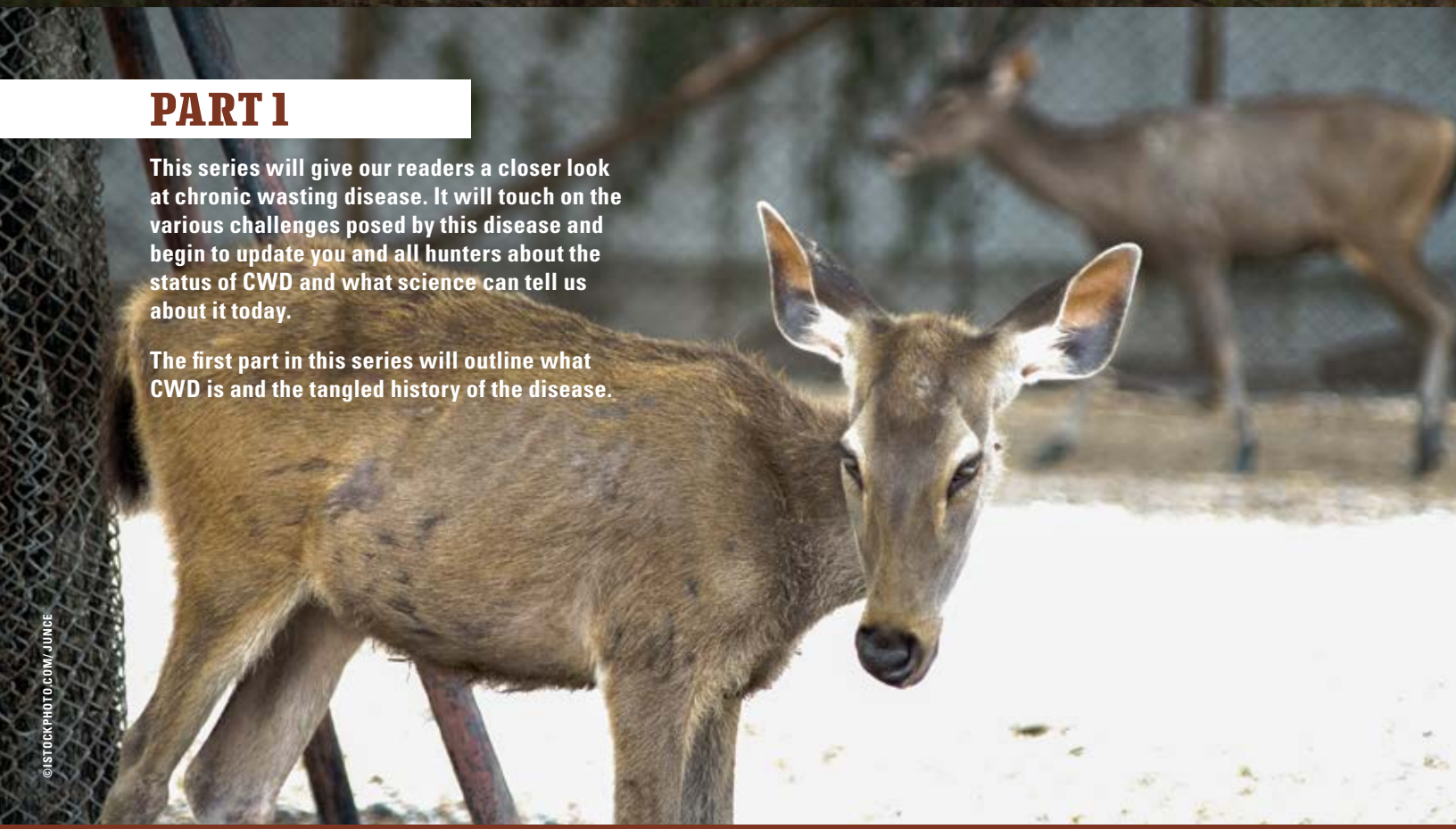
CHRONIC WASTING DISEASE

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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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.

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	■ 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 a wild reindeer in Norway ■ CWD found in two wild moose and a free-ranging reindeer in Norway

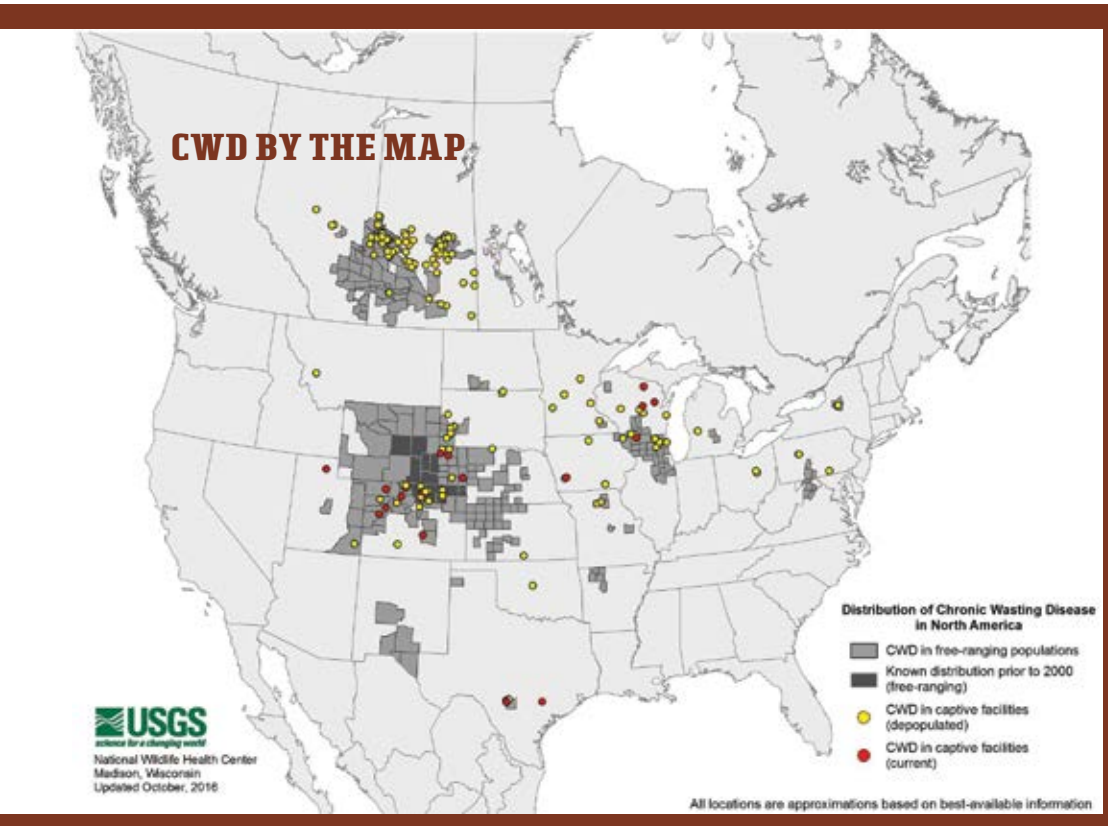
elsewhere when "new" cases arise. But perhaps most pervasive is the lack of complete information on contributory events, particularly for outbreaks involving free-ranging cervids. Although the lack of a singular explanation can be dissatisfying, failing to consider plausible alternative timelines and 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.