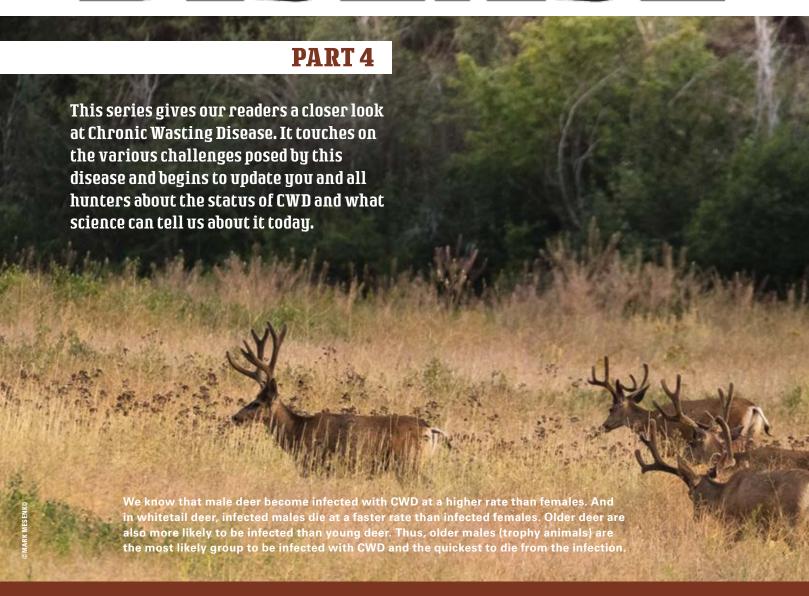
CHRONIC WASTING DISEASE



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.

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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 lowless 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

Exposure Risk	Areas adjacent to CWD-positive wildlife		
Factors	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		
	Areas permitting transport of hunter-killed elk or		
	deer carcasses from CWD infected areas		
Amplification Risk	Areas with high elk or deer population density		
Factors	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)		

www.CWD-Info.org has up-to-date information and resources to learn more about CWD in the U.S. and Canada.

Management Goals and Surveillance Objectives

Management Goals	Prevention or Elimination	Elimination, Monitoring, or Control	
Surveillance Goals	Detection	Assessment	Monitor
Surveillance Objectives	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

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 iuveniles 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 vet 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 bat-

tle 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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