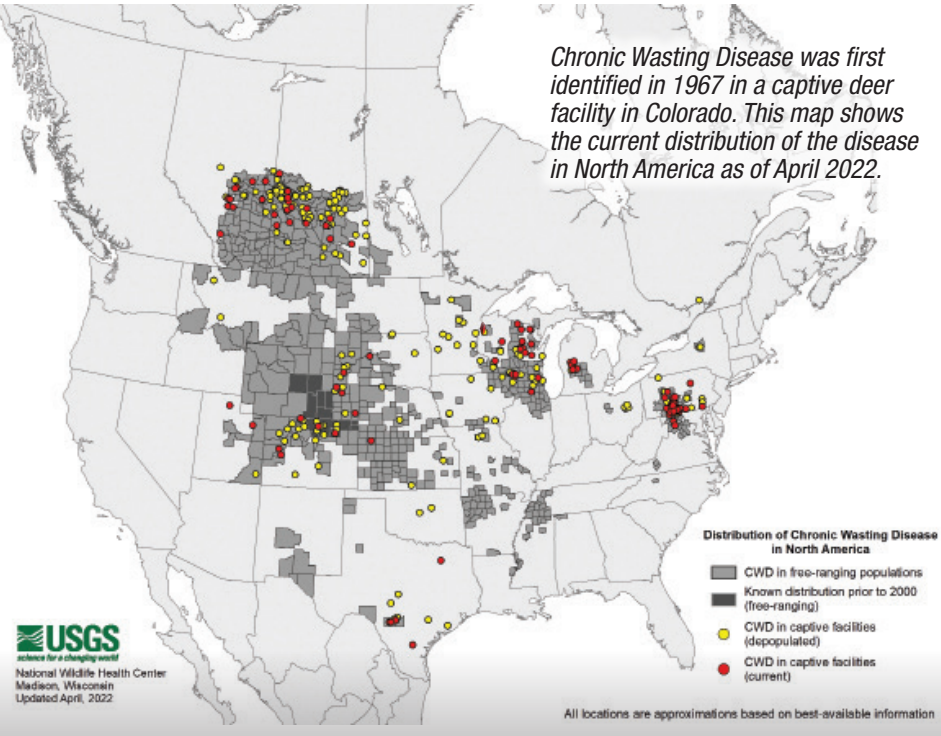
By **JASON SNAVELY**



Chronic Wasting Disease was first identified in 1967 in a captive deer facility in Colorado. This map shows the current distribution of the disease in North America as of April 2022.

MAP COURTESY OF THE U.S. GEOLOGICAL SURVEY

PART 1 OF 3

CHRONIC WASTING DISEASE: THE SAGA CONTINUES

Five Decades In, Many Questions Remain Unanswered

IT HAS NOW been 55 years since the scientific community first identified Chronic Wasting Disease (CWD) — though no one really knows how long it existed before that.

Over the past five-plus decades, CWD has spread throughout much of North America, causing major concern among government wildlife agencies, conservation organizations and hunters as we continue searching for answers about how to effectively deal with a disease that threatens not only whitetails but all cervids (mule deer, elk, moose and caribou).

As I follow the CWD saga, I see a promising area of research that sheds some light on how nature may "manage" this disease: beneficial genetic mutation, through disease-driven natural selection. Perhaps, just as nature intended, these population-level genetic alterations are lowering the number of CWD positives and slowing the progression of CWD's work in deer populations where the disease has been present the longest.

Given that, this seemed like the right time for a three-part series summarizing the highlights of the CWD saga. I'll start this month with the basics of CWD. Part two will cover the latest CWD research, and the final installment will discuss how wildlife managers should develop CWD policy and best-management practices, based on the data we have.

What is CWD?

Explaining exactly what CWD is and how it ultimately kills whitetails has always been challenging, even for the "experts" who study the disease for a living! It is my goal to present a basic, but thorough, understanding of how CWD causes progressive neurological degeneration in whitetails. It's also important to remember that even the experts acknowledge there is much about CWD we simply do not know.

CWD is in a family of rare diseases known as ***transmissible spongiform encephalopathies (TSEs)***. Simplifying the long name, transmissible simply means the disease can be passed from one animal to another. Spongiform refers to the work done by disease-associated prion proteins (more on this later) that accumulate in the brain, where they kill neurons and leave microscopic, "spongy" holes. Encephalopathy is simply a disease of the brain. Therefore, putting it all together the name describes a disease that spreads from animal to animal and causes spongy holes in the brain. The hallmark of all TSEs is progressive neurological degeneration, with infected individuals slowly losing the ability to function and ultimately dying.

CWD affect cervids (deer family). Other TSEs affect different species but are related to CWD. They include bovine spongiform encephalopathy (BSE) in cattle, aka "mad cow disease," scrapie in sheep and goats and Creutzfeldt-Jakob Disease (CJD) in humans.

Transmission and Incubation

CWD is contagious and can be transmitted orally from individual to individual through saliva, urine and feces, though much more needs to be

discovered about the various mechanisms of transmission. Anyone who spends time in the woods knows whitetails are highly social, often grooming one another and sparring. It is hard to believe a highly transmissible disease wouldn't wreak havoc at the population level unless the mere exposure of animals to the disease results in population-level genetic effects that ensure for the proliferation of the species (more on this later). Transmission can occur laterally, from animal to animal, or vertically, from mother to fetus. Animals can also become infected by acquiring the infectious agent from the environment, where disease-associated prions can persist for decades.

Unlike a living bacteria, fungus or virus, CWD is caused by a ***non-living prion protein*** that has, for reasons yet unknown to scientists, gone rogue and "misfolded." A portmanteau (a word blending two words, like breakfast + lunch = brunch), the word ***prion*** is a combination of two words: ***protein and infection***. The term prion is also short for proteinaceous infectious particle, a reference to a prion protein's ability to ***self-propagate*** and transmit its shape to other prion proteins. The fact that a protein, and not a virus or bacteria, appears to cause CWD is, in my experience, what causes so much confusion and misinformation.

All mammals produce normal prion proteins, which are nothing more than a chain of amino acids that fold into a three-dimensional shape. Scientists are not sure what role they perform in the body, but it is clear that normal prion proteins perform their duty and then break down and are recycled in the body within 4-6 hours.

Disease-associated prions, on the other hand, are extremely resilient and have proven to persist in the environment for decades. In fact, state agencies have killed and removed CWD-infected deer from high-fenced enclosures and reintroduced healthy deer many years later only to realize the introduced, CWD-free animals become infected with CWD through environmental transmission.

Scientists recognize that ***disease-associated prion proteins*** have the same sequence of amino acids as normal prion proteins but are folded into ***different shapes,*** with radically different properties. These misfolded prions bind to the normal prions in the body and cause them to misfold too; a process scientists don't fully understand. Eventually, the misfolded, disease-associated prion proteins accumulate in the brain and central nervous system, causing neuronal death. Once a critical threshold is reached in the body, neurological degeneration appears to proceed rather quickly and is followed by death.

One troubling aspect of CWD is how long it takes between infection and the development of clinical symptoms such as weight loss, lack of coordination, excessive thirst, walking in circles, drooling, etc. Research has shown that this gap, known as the incubation period, can last up to four years. This is significant, since we know that many of the deer in huntable whitetail populations never even live to 3 or 4 years of age! B

A whitetail doe exhibiting clinical symptoms of Chronic Wasting Disease.

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