CHRONIC
WASTING DISEASE

The Evolving Challenge

BY LISA BALLARD
THE NEWS shook Carbon County like an earthquake. Last fall, a deer at a hunter check station in this south-central Montana county tested positive for chronic wasting disease (CWD). Hunting households, many of which depend on wild game for affordable meat, ached with anxiety. Should they eat the local deer and elk they had harvested? Would this highly contagious disease decimate Montana herds? If CWD spread throughout the state, the economic impact of losing these animals — and the hunters and wildlife watchers they entice to Montana — would be devastating.

Then another deer tested positive 300 miles away on the northern border of the state near Havre, Montana. Although public concern was palpable, the news was not unexpected — at least not by state wildlife officials. CWD was already present in cervid populations on three sides of Montana: the Canadian provinces of Alberta and Saskatchewan to the north, North Dakota and South Dakota to the east, and Wyoming to the south.

“In my mind, chronic wasting disease was already here. We just hadn’t found it yet,” says Emily Almberg, disease ecologist for Montana Fish, Wildlife and Parks (FWP). “It’s slow moving, and we had a lull in surveillance for a number of years due to a lack of funding. We had already identified high-risk areas close to CWD cases in neighboring states and provinces. Our response planning was laid out.”

Montana’s strategy for controlling CWD mirrored the protocols elsewhere in the country, starting with testing to find what percentage of deer and elk had it and its geographic distribution, which is difficult to determine in the wild. Montana FWP tested more deer at hunter check stations, initially within a 10-mile radius around the spot where the first case was found, and then in a larger area from the Wyoming border in the south to the Yellowstone River about 55 miles to the north. FWP staff also examined hunter-harvested deer in the Havre area. One white-tailed deer and five mule deer tested positive.

In western states, the prevalence of CWD among mule deer tends to be higher initially than among white-tailed deer, says Almberg. “We don’t know why exactly, though it’s probably due to behavioral or aggregation patterns. Elk tend to be a distant third due to a combination of behavioral patterns and biological susceptibility.”

Cervids — deer, elk, moose, reindeer, and caribou — transmit CWD through their bodily fluids (including urine and saliva) and
scat. Game farms, both high-fence hunting facilities and farms where cervids are raised for meat, have grappled with CWD for decades. CWD spreads quickly on these farms because animals eat communally, slobbering on the same feed and bedding in close proximity. Almberg believes that the lack of captive and domestic cervid facilities in Montana (they are illegal in the state now, although a few were grandfathered in) helped wild herds remain CWD free until now. Montana was the 24th state in which the disease had been discovered. This January, Mississippi became the 25th.

Finding cases of CWD among Montana’s wild deer herds triggered a special hunt, with the goal of shooting 200 mule deer and 200 white-tailed deer to get a more precise read on the prevalence of the disease. The special hunt helped map the initial hotspot. Of the 216 mule deer and 123 whitetails harvested, only 8 mule deer and 2 whitetails tested positive for CWD — a 2-percent prevalence. However, in one area close the Wyoming border, the prevalence was 10 percent, and state biologists are working to determine if steps to manage CWD in that area are warranted.

“Realistically, CWD is not going away,” says Almberg. “There’s no hope of eradicating it once you’ve got it. Our goal is to keep it at a low prevalence. It takes decades for population impacts to happen. It took 30 years for the herds to decline in Colorado. We assume it could happen here.”

WHAT IS CHRONIC WASTING DISEASE?

Chronic wasting disease has no known cure and is always fatal. It is not caused by bacteria or a virus. Technically, it’s caused by a “transmittable spongiform encephalopathy” — an abnormal protein more commonly called a prion (pronounced PREE-on). It’s similar to what causes bovine spongiform encephalopathy (mad cow disease) in cattle and scrapie in sheep.

A sick animal sheds prions, primarily through its saliva, urine, and feces. These prions can remain on plants and in the soil for years, allowing other animals to pick them up. This is why CWD spreads quickly among captive deer in enclosures. In the wild, the disease tends to move more slowly because deer congregate in smaller numbers — unless man-made sources of food, such as spilled silage on a farm, bring deer together in large numbers.

At first, it’s difficult to find CWD-infected animals in wild herds, even when the disease is present. Once deer or other cervids contract
the disease, it is virtually undetectable to the naked eye for almost two years, until the animal is within a few months of death. At that point, the symptoms become obvious, including droopy ears and a “wasted” appearance. As abnormal prions become concentrated in the brain and nervous system, the animal functions less normally in every way, including how it eats, causing it to literally waste away.

Which brings up one of the challenges of CWD: it’s hard to detect the disease in live animals until the end, which makes the disease difficult to combat.

COMBATTING CHRONIC WASTING DISEASE

There is no known treatment or cure for chronic wasting disease. Keeping deer and other cervid herds at an appropriate size for their habitat is the only way to limit the spread of CWD. This puts hunters on the front lines of the battle.

For many years, when CWD was discovered in a domestic or wild herd, the accepted method was to kill all the animals in close proximity to the infected cervid and quarantine the area. Today, there’s a more refined, multi-step process that begins with determining the geographic area in which CWD is present and what percentage of the deer population has it.

“Total eradication of a herd or extreme herd depression is an outdated approach,” says Almberg. “It’s hard to accomplish that with wildlife because you might not find all of them. And you can’t determine exactly where all of the prions have been shed. A more practical approach is containment.”

Once a state fish and wildlife agency finds CWD and determines its geographic area and prevalence — either through a special hunt or testing animals harvested through existing hunts — containment involves the following steps:

Reduce buck-to-doe ratios. Bucks have a higher prevalence of CWD, especially when the disease is first discovered in a deer population. This may be because bucks congregate during the rut and young bucks tend to disperse farther away than does when they leave their mothers.

Harvest hotspots. Given that the prevalence of CWD tends to increase among deer in close proximity to each other, when a hotspot is identified, those deer need to be eliminated.
Reduce artificial aggregation of deer. Deer are attracted to places where grain bins have leaked or spilled and where there’s agricultural waste such as beet cuttings. Feeding deer — to watch or to hunt them — also brings large numbers of wild deer together that would otherwise not intermix. Reducing these man-made reasons to gather slows the spread of CWD. Deer-friendly landscaping in your yard may aggregate deer, but this is usually in much lower numbers than the hundreds that might come to a farm field looking for leftovers.

Work across state lines. Deer don’t recognize state borders. State fish and wildlife agencies need to share data and scientific knowledge to help slow the spread of CWD from state to state.

Implement transportation restrictions. Many states already have restrictions on transporting full, dead animals. In other words, you can’t shoot a deer in Wisconsin, throw it in the back of your pickup, and drive it home to North Dakota. Boned-out wrapped meat, hides without heads, and clean antlers are typically okay to transport because they don’t contain neurological matter (brain or spinal material), which has the highest concentrations of CWD prions.

What Is a Prion?

According to the Centers for Disease Control and Prevention, prions are cellular proteins found abundantly in the brain. These proteins appear in other parts of the body but not at the same density. The function of prions is not completely understood. However, scientists do know that when one of these proteins is abnormally folded in structurally distinct ways, it becomes a pathogenic agent called a “transmissible spongiform encephalopathy” (TSE).

TSEs cause diseases that are rare, progressive, neurodegenerative — and often fatal.
Support surveillance. Knowing where and to what extent CWD has infiltrated a geographic region is perhaps the most important information when it comes to containing the threat. Until about a decade ago, the federal government allocated $16 to $19 million each year to the U.S. Department of Agriculture, which passed the funds to state agencies to track CWD. Much of what we know about CWD was discovered thanks to those funds. Unfortunately, the federal government cut those allocations in the 2011-2012 budget, leaving each state to fund its own surveillance programs. This greatly curtailed surveillance efforts. Ongoing and thorough surveillance not only alerts scientists to changes in prevalence in specific areas but also the rate CWD is spreading across the larger landscape.

Given that we have dramatically reduced or eliminated predator populations in many parts of the country, hunting is critical to lowering densities of deer and other cervids to levels appropriate to their habitat. More “natural” density levels slow the rate of CWD infection because animals are not coming together in unnaturally large groups. This is good for deer — and for hunters. “If you control the number of deer, the habitat is higher quality, there are bigger deer, and you’re doing something positive about disease containment,” says Bryan Richards, emerging disease coordinator for the U.S. Geological Survey’s National Wildlife
Disease Center and one of the nation’s leading CWD scientists.

However, the number of hunters across the United States has been in decline for many years. CWD is yet another deterrent to hunter recruitment and retention. Richards calls it “the ick factor.” People who started hunting through the “locavore” movement — one of the key sources of new hunters today — might be less inclined to pursue game that may be tainted with CWD. Unfortunately, fewer hunters also mean fewer hunting license sales, which cuts funding available for research and response to CWD.

“Detractors who do not think CWD is a concern point out that less than one percent of the deer and elk populations in the U.S. have CWD. But that’s not a relevant statistic,” says Richards. A national average is irrelevant because only half of the states across the country have known cases of CWD. Instead, researchers focus on the localized ranges for wild deer. “In Iowa County, Wisconsin, adult male deer in a 12-by-12-mile area have a CWD prevalence of 50 percent,” says Richards. “Now that’s meaningful!”

Richards emphasizes that the total number of deer in a particular habitat is also an important consideration. For example, if there are 100 deer in an area and 50 percent have CWD, that’s 50 cases of the disease. However, if there are 1,000 deer in that same area and 50 percent have CWD, that’s 500 cases — and each of those animals is shedding prions into the landscape, vastly increasing the chances that other deer will contract the disease.

Given the spread of chronic wasting disease to 25 states, should we hope that deer, elk, and other cervids will develop a natural immunity to it? The experts say don’t hold your breath.

**RESISTANCE IS FUTILE**

About five percent of the deer population has shown some “resistance” to CWD, but this does not mean the disease won’t kill them. It just takes longer — five years instead of two — for the animal to die. This means that the animal is shedding abnormal prions for three years longer than non-resistant deer — and likely infecting a greater number of other deer, making “resistant” deer even more problematic.

Deer with these “resistant” genes may have other traits that are less desirable for the long-term survival of the species. Richards believes that the resistant alleles (a variant form of a gene) would be in 75 percent — not just 5 percent — of the deer population if they were truly desirable.
In addition, “the argument that we should just wait and see how these deer do also assumes that the infectious agent stays the same over time,” says Richards. “With scrapie, there are over 30 recognized strains. New strains of CWD could develop that these deer are not resistant to.”

According to Richards, it’s unlikely that natural selection will help eliminate CWD because it can take years and there’s too much hunting pressure. “One researcher modeled [natural selection] and postulated that it will take 150 to 300 years to manifest in Wisconsin,” he says. “Plus, in order for natural selection to occur, you have to stop humans from selecting with their trigger finger... For a specific genotype to prosper, it must place more progeny out there in the next generation. If we kill nearly every deer at younger ages, few will die of disease, and there won’t be much opportunity for natural selection to occur.”

Richards acknowledges that there are genotypes within humans that seem to confer high levels of protection from human TSEs and genotypes within domestic sheep that resist scrapie. So the possibility exists that a genotype that’s fully resistant to CWD could emerge in deer, but Richards isn’t counting on it.

“If we stop hunting deer populations with CWD in the hopes that natural immunity develops, think about what would happen to the ecosystems that these animals live in while we wait,” says Richards. “Deer are called a keystone species for a reason.”

A keystone species is an animal or plant that has a significant impact on its habitat based on its population size. Deer are one such species. If we stop hunting them, deer populations would explode, stripping the landscapes where they — and we — live.

THE HUMAN CONNECTION

There are no known cases of chronic wasting disease having been transmitted to humans. However, the World Health Organization and the Centers for Disease Control and Prevention (CDC) strongly discourage people from eating meat that has tested positive for chronic wasting disease.

This caution is due in part to the possible link between mad cow disease (bovine spongiform encephalopathy), which is a cousin to CWD, and variant Creutzfeldt-Jakob disease in humans. The variant form of Creutzfeldt-Jakob disease appeared in humans about 10 years after an outbreak of mad cow disease in the same area in the mid-1980s. The CDC explains that this timeline is “consistent with known incubation periods for Creutzfeldt-Jakob disease” and reports that
Left: Lisa Ballard  

there is “strong epidemiologic and laboratory evidence for a causal association” between variant Creutzfeldt-Jakob disease in humans and mad cow disease. It is a good reason to be cautious.

Scientists cannot inject CWD-causing prions into live humans to see if the disease will cross the species barrier. In a lab, researchers exposed brain matter from deceased humans to CWD prions — and conversion to those abnormal prions occurred. In mice, scientists replaced the mouse gene with the human gene that produces normal prions, then fed the mice CWD-infected food. At first, there was no evidence of CWD crossing over into the human neural tissue in the mice, but later it did occur in other tissues. In another study, scientists exposed macaques (monkeys), a primate closely related to humans, to CWD by feeding them infected venison. The meat came from deer that were preclinical (meaning they had CWD but still looked healthy). The result: two of the macaques eventually developed a prion disease.

“There’s no evidence that CWD has crossed to humans, but research says there’s a chance,” explains Richards. “Lots of folks think I’m an alarmist, but the research also suggests that the species barrier is not as robust as it was once thought.”

This increases the importance of containing the disease through hunting.

HUNTING IN CWD-INFECTED AREAS

Since CWD first made headlines, hunters have questioned whether to hunt in an area where the disease exists. Those areas need hunters to control cervid populations and slow the spread of the disease.

“In captive areas, we can eliminate a herd, but with wildlife we can’t apply this method,” says Richards. “We’re challenged with CWD. We have only blunt tools for managing wildlife. We can manage habitat or numbers through harvest. We’re a long way from a viable vaccine, and we don’t have the tools to mitigate the prions in the environment.”

If you’re a hunter and concerned about eating CWD-infected venison, Richards recommends having an animal tested through your state fish and wildlife agency. You’ll need to wait before eating the meat — test results can take up to two weeks. Several private companies are trying to develop a rapid field test in which a hunter puts a few drops of blood on a card. Unfortunately, reliable instant tests are not available yet. Other commercially available tests have not yet been vetted by independent scientists (and have the same turnaround time as state fish and wildlife agency tests), which is why Richards urges testing by state agencies.
Most scientists studying CWD agree that caution is prudent when it comes to handling meat after shooting a deer, elk, or moose, especially in a region where CWD is known to exist. Here’s how to stay safe:

1. **Wear rubber gloves** while field dressing game. (You’ll also decrease the chance of contracting a bacterial infection.)
2. **Wear eye protection** for the same reasons.
3. Avoid or minimize handling the brain, spinal tissue, lymph nodes, eyes, spleen, and tonsils. These organs have the highest concentrations of prions. And don’t eat these parts of the animal.
4. Thoroughly wash your knife, game bags, and other items that the animal’s body parts touch.
5. **Have your meat tested** by the state fish and wildlife agency. Don’t eat wild meat if it tests positive for CWD — cooking won’t neutralize or destroy the prions.
6. **Transport only butchered meat** in sealed packages, taxidermied heads, and clean antlers.
7. **Bury unwanted parts** of an animal carcass to prevent possible spread of CWD. (You won’t know until weeks later whether the animal tested positive.)

**THE FUTURE OF HUNTING**

Will chronic wasting disease eventually wipe out all of our deer and elk? No one knows. This is an issue conservation agencies will be working on for many years to come. Colorado Parks and Wildlife has been dealing with the disease for decades. States on the East Coast discovered CWD in their herds in just the past several years.

The challenge is keeping hunters and the general public engaged over time. So far, so good.

Last fall in Missouri, where CWD-infected animals were discovered in 2010, the Missouri Department of Conservation tested tissue from 18,400 wild deer for CWD. The tissue was submitted by hunters and taxidermists and taken from roadkill. The tests uncovered 15 new cases (13 bucks, 2 does) of CWD, including in areas the disease had not been found before. The state immediately took steps to test more deer and thin the herds to limit the spread of the disease.

“We could not accomplish this very important work without the help of the many thousands of hunters and hundreds of landowners around Missouri who brought in their deer for CWD testing to help find and limit the spread of this terrible disease,” said Sara Pauley, director of the Missouri Department of Conservation.
The 15 new CWD positives bring the total number of CWD cases detected in free-ranging deer in Missouri to 57. It’s certainly not devastating to the state’s overall wild deer population — yet. And therein lies another big challenge.

CWD has been in the United States for five decades, and we’ve still got lots of wild deer and elk — in some places, too many. What’s more, people haven’t contracted it. So it is easy not to focus on the disease.

What can we do to ensure the future of deer, elk, and other cervids? “Keep hunting,” says Richards. “From a wildlife management point of view, hunters are how we manage deer and elk populations. In the short term, if you stop hunting whitetails because there’s a high prevalence of CWD in an area, the population will double in two to three years.” As stated earlier, this can decimate habitat and severely impact other wildlife populations.

And then there’s “the ick factor,” which fortunately tends to be temporary. When CWD was first found in Wisconsin in 2002, deer hunting license sales dropped about 10 percent. Since then, license sales have rebounded to pre-CWD levels, even though CWD has now spread to wild deer populations in 27 counties.

“Hunting is still part of the local culture,” says Tamara Ryan, wildlife health section chief at the Wisconsin Department of Natural Resources. “Hunters have grown accustomed to sampling. They incorporate knowledge of CWD into their endeavor. As long as they have the option to have their deer tested, they can make an informed decision... If a deer comes back positive, the hunter gets a replacement tag, so they have another opportunity that season or the next year.”

There are many great reasons to go hunting: It’s a time-honored tradition that binds friends and family. Venison is a healthy, tasty meat. Hunting provides time outdoors and exercise that is good for both physical and mental health. And it helps keep a deadly disease in check.

“CWD will continue to affect a treasured natural resource and might become a human health issue,” says Richards. “We’re not sure we can get ahead of it. But there are also vast areas of the United States where no evidence of CWD exists. It’s up to us to do all we can to keep it that way.”

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