Don't Let CWD Stop Management!

Many hunters and landowners are panicking over the discovery of chronic wasting disease in whitetails. Yes, CWD is a real concern — but could the scary headlines harm deer management more than the disease itself will?

by Dr. James C. Kroll

In 1967, a strange malady showed up among deer at the Colorado Division of Wildlife's Research Facility near Fort Collins. Some of the deer being kept there for nutritional research became lethargic, lost weight, drank excessive amounts of water, staggered, drooled and became disoriented. The disease inevitably was fatal, but no one had a clue as to what was causing it.

Similarly ill animals then appeared in the Wyoming Game and Fish Department's Sybille Research Unit at Wheatland. The facilities apparently had traded animals over the years.

In 1978, Dr. Beth Williams of the Wyoming State Veterinary Laboratory examined brain tissue from some of the stricken animals and discovered that the brains had a "Swiss cheese" or spongy appearance. Something was eating holes in them. For lack of a name, the "disease" was dubbed chronic wasting disease, due to the effect it had on deer.

From 1974-79, of the 66 mule deer and one blacktail housed at the Colorado site, 57 developed CWD. What was really scary was the fact that destroying the infected deer, cleaning the pens and sterilizing the soil did not appear to prevent subsequent infection of newly introduced deer.

It became apparent that CWD was similar in action to a sheep disease called scrapie, which had been known since the 1700s. But no one had identified scrapie's causative agent, either; all researchers knew was that it seemed to have a genetic susceptibility connection. Similar human diseases also had been or were being reported, including Creutzfeldt-Jakobs disease, fatal familial insomnia (Gerstmann-Staussler-Scheinker disease) and kuru.

These diseases were lumped into a general group called transmissible spongiform encephalopathies (TSEs); an impressive name that literally translates to: "a transmissible disease that turns the brain into a sponge." There also were reports of such diseases in mink and some other mammals.

It wasn't until the late 1970s that the ultimate agent was identified as a "rogue" protein, called a prion ("pree-on"). Dr. Stanley Prusiner of the University of California, who identified the agent, received the Nobel Prize for his research team's work.

Prions are normally occurring proteins in mammals' brains. They are thought to protect the brain against deterioration and, in humans, dementia. Then why are they harmful?

Prions are constructed much like a spring. Malformed or mutated prions coil and distort to the point, although identical in molecular composition, their structure renders them dangerous. Rogue prions attack the nervous system, become clumped and physically affect tissue, or are no longer available to protect it. As of yet, we really do not know which.

In 1986, CWD's famous cousin, bovine spongiform encephalopathy (BSE) or "mad cow disease" came to the public's attention. The resulting panic and paranoia over eating beef in England, then France, nearly ruined Europe's livestock industry. By 1992, infection had increased from a few animals to more than 35,000.

To combat BSE, officials relied on what had been learned about a similar human disease, kuru. This TSE occurred among some New Guinea natives (the South Fore), who ritually practiced cannibalism of human brain matter. It thus made sense consuming animal products, especially nerve tissue, might spread the disease, so the European Community banned the use of animal products in feed. Since then, the spread of BSE

Many hunters and their families worry that it's unsafe even to touch a deer, much less eat venison. But as of this writing, there is no evidence humans can contract CWD from any source. Photo by Gordon Whittington.

has slowed dramatically.

Could BSE also move from cattle to humans? When a variant of C-JD showed up in humans in England, the answer seemed obvious. But had these victims contracted the disease as a mutated form of BSE (vBSE)? Possibly, but that still is not certain. Scientists argue both ways. Regardless, the economic damage to British farmers is real. It might take years for them to recover. Twenty-four people contracted vBSE, and during that same span, as many distraught cattle farmers committed suicide.

Now that the agent for CWD had been identified, the logical question arose: Can prions from one deer infect another? The Colorado and Wyoming experience seems to indicate so, but we are not certain. In fact, there is very little scientists *are* certain of when it comes to CWD. We do not know how it spreads, but we do know more cases were reported in Colorado and Wyoming than elsewhere.

Recent reports suggest that TSEs can be caused by many factors, including transmission from infected animals, mineral deficiencies and exposure to environmental contaminants, such as organophosphates.

We have no idea if CWD was out there all along or somehow was created like Frankenstein's monster at the Colorado facility. We probably never will know. Before the culprit was identified, deer from that facility were moved to other areas, including zoos in Colorado and South Dakota. Animals ended up on game farms, as well. The genie was out of the bottle.

CWD was found at the research facility at Wheatland, Wyoming in 1977; in elk at a Colorado research station in 1979; in free-ranging deer in north-central Colorado in 1981; in the first game (elk) farm in 1996; in game farms in South Dakota in 1997; in captive elk in Nebraska and Oklahoma in 1998; in captive elk in Montana, Colorado and Nebraska in 1999; in Saskatchewan elk in 2000; and in Nebraska elk and New Mexico mule deer in 2002.

Now that the agent for CWD had been identified, the logical question arose: Can prions from one deer infect another?

And then there was perhaps the most troubling CWD discovery of all: in southern Wisconsin, almost 900 miles east of the primary zone. (See pages 28-29.) The Wisconsin cases unleashed a storm of controversy being fought in the courts, the popular press, the Internet and in some cases, even through physical confrontation.

How did Wisconsin deer become infected? Again, at this time there is no clear answer. A May 23 press release by the Wisconsin Department of Agriculture (chief Jim Harsdorf) made the following points:

- "There has never been a case of CWD diagnosed on a deer or elk farm in Wisconsin." (Note: This could have changed with the later diagnosis of CWD on deer farms in two counties. However, at the time this issue went to press, those positive test results were in dispute.)
- "Nineteen elk came to Wisconsin legally from a Western ranch where herds were later found to be infected with CWD.

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- "Of the 19, 10 remain alive and healthy. They are no longer under quarantine, because they have been off the infected ranch for five years, the national benchmark. All of the 19 elk went to farms in Chippewa, Outagamie, Waupaca and Washington counties. None went to the Mount Horeb area.
- "Six of the animals died but tested negative for CWD. Three others died but were not tested; two died before the infection was reported, and one was not discovered until the carcass was too decomposed for testing.
- "The agriculture department has tracked the 2,604 deer and elk that have been legally imported into the state since 1995. The majority of those animals have come from Colorado, Nebraska and Montana with nine from Kansas and one each from Oklahoma and South Dakota.
- "To get a scientifically valid sample from Wisconsin's deer and elk farms would require killing at least 35,000 animals."

On May 16, the state legislature voted to spend $4 million to fight CWD, and Wisconsin Gov. Scott McCallum has requested another $18.5 million in federal money over the next four years to aid in the effort.

The state also imposed a dramatic plan to reduce or eradicate the disease in the known area, and that was when the public furor erupted. The plan is to seriously reduce deer populations in a zone about the infected area. Permits are being issued to the public for this eradication program. Obviously, it has led to serious political problems for the DNR.

Secretary Darrell Bazzell issued a statement saying: "We're not sure if we can beat this thing, but we have set a goal trying to eradicate, and we are not going to retreat from that goal. Reducing the deer herd is our next logical — though painful — step. Experts have told us we need to limit deer-to-deer contacts in order to stop transmission of CWD."

There have been threats of lawsuits, as well as public relations battles. Even the U.S. Congress joined the fray. The first committee hearings were held in Washington, D.C., in May to gather information about the disease. Yet public opinion

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appears to be on the side of the Wisconsin DNR; a recent opinion poll supported the eradication program. Only time will tell if it works.

Little is known about how CWD reached Wisconsin. But as of now, here is some of what we *do* know:

TSEs have been reported in a variety of mammals, including deer and elk. The primary focus for the disease seems to be in the Western states, with the exception of Wisconsin.

Several causative agents have been suggested for TSEs. Recent research, for example, suggests that in absence of adequate copper, prions bind to manganese, which distorts them to produce rogue prions. There appears to be a connection between soil copper and the disease in the wild.

Pesticides also have been linked to TSEs. According to elk researcher, Dr. Michael McDonnell, CWD could be caused either by exposure to organophosphates such as Phosmet or by low soil/plant copper. He primarily blames low levels of copper in the soils of northeastern Colorado and in other areas where CWD has been found. He reports on studies that show some protection afforded by copper supplementation.

Dr. Tom Sczwerczek, a livestock nutrition researcher, suggests excessive dietary intake of heavy metals could be a factor. He notes that cadmium in particular has been linked to wasting disease in cattle, horses and some other domestic animals.

I find it disconcerting and suspicious that, to date, the distribution of CWD overlaps the traditional range of domestic sheep, which always seem to be implicated in BSE.

IS THE SKY FALLING?

How serious CWD is depends on whom you ask. Unfortunately, to date scientists have not been of much help.

Professional opinions align with one of two camps. The first takes a more fatalistic view. For example, during testimony before Congress, Research Veterinarian Dr. Michael W. Miller of the Colorado DNR said, "According to published model forecasts of CWD epidemics in deer populations, unmanaged outbreaks will likely devastate infected herds over a period of several decades."

If this claim is correct, it could provide an explanation for the overall decline we have seen in mule deer populations. A recent report indicated that CWD has spread to urban deer around Denver and Boulder.

The second view — which I tend to support — is that CWD is an eruptive disease that might have been around for many years. It appears to me there are two origins of CWD: one in the West and another in farmed elk. But in either case, environmental factors could be at work.

This supports the idea of spontaneity. The apparent genetic links for some TSEs also suggest resistance among some animals, and natural selection forces will solve the problem eventually. The epidemiological models Dr. Miller cites are only as good as the data provided — and frankly, the data are not very good.

Observed infection rates — an important component to any model — often are skewed by sampling procedure. *I suspect a "normal" infection rate of not over two or three percent to be the true figure.* Furthermore, it is known to take three to five years for CWD to become "clinical"

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in deer or elk, and few of them even reach their third birthdays under heavy hunting pressure.

Also, *it has not yet been demonstrated to my satisfaction that CWD always is fatal.* Hopefully, my optimism is well founded. I know Dr. Miller hopes his prediction is too pessimistic.

What are the chances that a human could contract CWD? Recently, some articles in newspapers and outdoor magazines have suggested that humans *have* contracted it. Let me state strongly: *There is absolutely no evidence that a human ever has contracted CWD!*

This is not to say hunters should be careless when handling harvested deer. Because prions primarily inhabit the nervous system, take care not to expose yourself to the animal's brain or spinal fluid. Simply bone out the animal with a knife, rather than use a bone saw.

In reality, there is a far greater threat to humans than contracting CWD: the panic caused by the "mad deer disease" press and the people with anti-hunting/anti-management agendas. These ultimately could have more effect on hunting's future than CWD will. In fact, one study suggests Colorado alone will suffer a $300 million-plus annual hit to its economy as a result of the scare.



Early on, Wisconsin officials decided to move aggressively against CWD, encouraging the eradication of whitetails in one area. Photo courtesy of Wisconsin DNR.

As of press time, the sale of 2002 Wisconsin deer permits was down significantly. With whitetail numbers in southern Wisconsin's core CWD area already too high for the available food supply, any loss of hunters



only figures to add to the management problem.

Venison always has been seen as healthy, but the doubt now being cast on its wholesomeness has made some charitable food distribution organizations re-evaluate their liability exposure. Those using CWD to advance a political agenda could do irreparable harm to hunting and our herds.

WHAT IS NEXT?

CWD and bovine tuberculosis (discussed in detail last month) are not the only infectious diseases affecting whitetails; they harbor a host of bacterial, rickettsial and viral diseases. Some of these are threats to man, but most are communicable only to other deer.

As of this writing, since 1967 fewer than 400 individuals in the deer family are known to have succumbed to CWD. Meanwhile, hundreds of thousands have died from viruses carried by biting midges (gnats). These diseases are known collectively as hemorrhagic diseases, including epizootic hemorrhagic disease (EHD) and blue tongue. They are similar to Ebola virus, which is deadly to humans; however, no human ever has contracted EHD or blue tongue. Viruses can be very specific in their hosts.

WHITETAIL DISEASE INFO

The situation with CWD and other deer diseases changes rapidly. For the latest updates and more background information, try these Web sites:

www.dnr.state.wi.us/org/land/wildlife/whealth/issues/cwd/index.htm
www.michiganfarmbureau.com/bovinetb/
www.wcdefa.org
www.aphis.usda.gove/vs/nahps/cwd/
www.aphis.usda.gove/vs/nahps/cwd/cwd-program.html
www.cvmbs.colostate.edu/dlab/webdocs/special_cases/wdisease.html
www.uwyo.edu/ces/pubs/chronic.htm
www.wildlife.state.co.us/hunt/hunter education/chronic.asp
www.state.sd.us/gfp/hunting/biggame/cwd.htm
www.cwd-info.org
www.defra.gov.uk/footandmouth/
www.vetmed.wisc.edu/pbs/johnes/
www.fao.org/news/2000/000607-e.htm
www.ehs.clemson.edu/forbid.html
www.mc.vanderbilt.edu/peds/pidl/infect/remsf.htm

As of yet, there is no evidence that West Nile virus (spread by mosquitoes) is carried by deer. However, we do know that some other bloodborne diseases *have* infected man, often via tick bites. Among them is Lyme disease, which strikes thousands of humans annually. It is spread by deer ticks, though deer themselves apparently do not contract the disease.

At the Institute for White-tailed Deer Management and Research in East Texas, we conducted a survey that revealed periodic eruptions of ehrlichiosis in our region. This disease was once thought to infect only dogs, but now we have had numerous reports in humans. Like Lyme disease, ehrlichiosis is carried by ticks and is difficult to diagnose. It attacks red and white blood cells and presents symptoms such as fever, headache, nausea and loss of energy. Humans can contract the disease and can die from it.

Rocky Mountain spotted fever also is a dangerous disease spread by ticks. In one East Texas county, we found a very high infection rate among deer ticks, and we do have sporadic reports of RMSF among humans in the area.

Rinderpest, or cattle plague, is a disease of many cloven-hoofed animals. It is caused by a *Morbillivirus* and is related to distemper and measles. It can be transmitted by sneezing and contact with nasal or mouth discharges. This is one of the world's most devastating livestock diseases, and if it shows up in wild ruminants, it could be a problem worse than CWD. It is present in Africa, China, Russia and the Middle East. I am unaware of a human threat from this disease, but its potential impact on livestock and wildlife is considerable.

In 1923, foot-and-mouth disease was discovered in black-tailed deer in California. You surely have heard about this disease and its impact, again, on British agriculture. Reeling from BSE, British farmers are faced with a disease that can move tremendous distances through the air, infecting farms far away.

Foot-and-mouth disease arrived in England by air, all right, but apparently in a different manner: via materials discarded by airline food-service companies. The contaminated food came from Asia and was improperly heat-treated before being fed to British swine. (If BSE ever makes it back into North America, this probably will be the mechanism by which it does so.)

Foot-and-mouth disease is caused by an *Aphthovirus*, and it produces lesions in the mouth and feet, hence the name. It is controllable, however; in fact, California authorities were able to eradicate it from that state, even though it existed in the wild.

I could go on and on, scaring you with tales of one nasty wildlife disease after another, but that would serve no good purpose. My point is: A host of diseases can affect deer and in some cases already have.

The high deer densities we have allowed to develop throughout North America set the stage for many potential disease scenarios. As a result, I predict that the CWD scare is just the first of many. The next "disease of the year" I am betting on is a relative of bovine Tb called Johne's disease (pronounced "yohnees), also known as paratuberculosis (*Mycobacterium avis paratuberulosis* or *M. johnei*). Crohn's disease, a human equivalent, causes chronic intestinal inflammation, diarrhea, weight loss and finally death. I have seen this disease in deer and believe it now exists in the wild.

WHAT CAN WE DO?

The first thing every landowner and hunter should do is step back, take a deep breath and look at wildlife diseases more logically than has tended to be the case lately. We must get away from the mindset that wild animals never are stricken by diseases, parasites or maladies.

The future impact of any of these whitetail diseases will depend on two factors: (1) deer population density and (2) contact with man and his domesticated animals.

I certainly support the return of humans to the countryside, as this restores contact between man and his natural environment. However, we must be vigilant that, as man brings animals with him, he does not infect wildlife with new diseases.

Diseases will be a part of deer management from now on. *My strong opinion is that CWD and other diseases never will be eradicated from wild deer herds. They just become a factor to consider in management.*

Disease transmission can be a two-way street; thus, we also must guard against introducing disease. At present, there are more than 65 reportable livestock diseases. Of these, only a handful have been reported in members of the deer family, and very few are unique to them. (CWD is among the exceptions.)

The first thing every landowner and hunter should do is step back, take a deep breath and look at wildlife diseases more logically than has tended to be the case lately.

Farming deer is now a significant agricultural activity in North America, and most deer-farming organizations have implemented CWD-monitoring programs. For this reason, in my view, deer farms are not a real threat to introduce new diseases.

But what about free-ranging deer? *The best approach is to manage for healthy, productive populations. We must take an ecological approach to management, not one based strictly on maximizing "hunter opportunity."* Well-managed herds are in line with their habitat and have balanced sex ratios and age structures. That is why our "Building Your Own Deer Factory" series focuses on food-source management and proper deer harvest. Managing for healthy deer in good habitat reduces disease problems.

This is where you come in. If we are to continue having good deer hunting, each of us must do his share. Become more actively involved in management where you hunt, and support sound management principles on a broader scale. And, question wildlife agencies if they take a political approach to managing this precious resource, rather than doing what is best for the herd.

(**Editor's note:** Turn the page for a closer look at Wisconsin's approach to its CWD problem.)



