

Chronic Wasting Disease

Still Dying for Answers

By Hal Herring

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Now this deadly disease is on Colorado's West Slope, and in Wisconsin and Nebraska and Saskatchewan . . . what to do?

The mule deer doe, heavy with twin fawns, takes the bullet in the shoulders, falling just as the muffled report from the sound-suppressed .308 reaches the rest of the herd. They stand, ears cupped, in the sagebrush and stunted juniper, trying to find the source of the noise. The rifleman, leaning into his bipod from his position on the hill above, expertly works the bolt on the Savage 110 and fires again, and a young forkhorn tumbles down the steep hill, skidding in a patch of leftover snow. The next to fall is a yearling doe, and then another, and another, before the rest of the herd wheels and runs, bouncing in the peculiar pogo-stick gait that is the trademark of plains game all over the world. It is April, in the dry, cold spring of 2002, and the effort to control chronic wasting disease (CWD) on Colorado's western slope of the Rockies has begun.

It may be impossible to say where this tragic story begins. There is no way to predict how it will end.

We know this much. In the late 1960s, captive mule deer being used in a study of deer feeding habits at wildlife research facilities near Fort Collins, Colorado, and Wheatland, Wyoming, began dying of a disease that researchers had never seen before. The illness was prolonged and ugly, with bizarre symptoms. Infected deer would suddenly become terrified of the most common objects. They staggered, slobbered excessively, drank enormous amounts of water, lost weight and hair. Death came from a kind of pneumonia that resulted from inhaling their own overproduction of saliva. For want of a better term, researchers called it chronic wasting disease. Not one of the infected deer ever recovered, and over a five-year period the disease killed 57 of 66 deer kept in one pen at the Fort Collins facility.

It would be a decade before anyone had the faintest idea what was killing the deer and, by then, some of the elk in the research pens. One night in 1977, at Colorado State University's laboratories, researcher Beth Williams was looking through slides of brain tissue from some of the dead mule deer and noted something very strange. In the tissue was a series of lesions and holes that brought to mind an animal disease she had seen before—scrapie, a devastating brain disease of domestic sheep that has been known for over 200 years.

Williams' suspicions were confirmed by researchers at the National Institute of Health's Rocky Mountain Laboratories. The deer killer was not scrapie, but it was similar, a member of the disease family known as Transmissible Spongiform Encephalopathies, or TSEs. The word "spongiform" refers to the curious pattern of holes, like a sponge, that appears in the brain tissue of infected animals. "Transmissible," of course, means just what it says, able to transmit from animal to animal.

When Williams made her discovery in 1977, TSE diseases were a scientific curiosity. A federal program to try to eliminate scrapie would soon be underway across the U.S., monitoring deaths of domestic sheep and destroying infected herds. A human TSE, called Creutzfeldt-Jacob disease (CJD), had been recognized since 1947, and was believed to be extremely rare, diagnosed in, and killing, about one in 1 million people in the U.S. and Europe. Kuru, a form of CJD associated with ritual cannibalism, killed off a large number of tribespeople in the remote highlands of New Guinea and then fell off slowly as the cause was recognized and stopped. A mysterious TSE had wreaked havoc on commercial mink farms in various parts of the U.S. for more than 50 years. Researchers believed that the diseases could be transmitted through food or, in the case of scrapie, through birth fluids that remained on grass after lambing and were eaten by grazing sheep.

But the TSEs were still too rare and too little understood for anybody but scientists, and the very few victims and their families, to worry about.

In the early 1990s everything changed. “Mad cow disease,” or BSE, for bovine spongiform encephalopathy, appeared in England. Millions of infected or exposed cattle were destroyed, and the outbreak was linked to modern feeding practices—cattle and sheep parts had been rendered into high protein feeds that were then fed back to cattle. Somewhere, it is believed, scrapie-infected sheep, or cows with an unrecognized TSE disease, had been thrown into the mix.

Worse was to come. Although the British government went to great pains to assure consumers that British beef was safe, a new form of CJD soon appeared in several young British people. An immediate warning flag went up. The CJD scientists knew about is a disease that can strike human beings in their old age. They believed that the disease took almost a lifetime to produce symptoms. Suddenly there were people dying in their late teens, and the disease left a slightly different pattern of destruction in brain tissue. Scientists called the new killer vCJD, the “v” for “variant.”

The much-feared epidemic of vCJD has not materialized, although, as of July 2002, 117 people have died of the disease in Europe. The one confirmed victim in the U.S. had lived in England during the height of the BSE crisis. The vCJD is almost certainly a result of BSE jumping what is called “the species barrier”—the natural resistance of one species to diseases of another—to infect human beings. Researchers are quick to point out that while the rate of human infection with vCJD is higher than the traditional one-in-a-million, and the victims are younger, it is estimated that at least 80 million people ate British beef products that may have been contaminated with BSE. With 117 confirmed deaths so far, the disease remains an extremely rare occurrence.

When Bugle first reported on chronic wasting and other TSE diseases in January 2000, the vCJD death toll was 24. We also wrote that all TSEs have long and completely unknown incubation periods, and more people may still be infected. That fact is just as true today as it was then, and there is no reason to downplay it or to lessen the horror of what has befallen these innocent victims.

Among leading researchers, controversy persists over the origins and causes of all TSE diseases. The most widely accepted theory, developed by Dr. Stanley Prusiner, who received the Nobel Prize in medicine for his research on this subject, posits that the diseases are caused by an abnormal type of protein called a prion that acts upon normal

proteins in the brain and causes them to change and form toxic aggregates that destroy brain cells and tissue. The prions may be transmitted from animal to animal by eating them or by some other means that has not been discovered. BSE and human CJD are believed not to be contagious, unless the flesh of infected cows is eaten or the flesh of infected people, as with kuru. Outbreaks of human CJD have occurred that are clearly tied to medical practices—treatment of conditions such as dwarfism with human growth hormone extracted from the pituitary glands of cadavers, and corneal transplants, have both resulted in CJD infection—not vCJD—in human patients. There is a deep, and ugly, mystery here.

In the 230 years that scrapie has been known to affect and kill domestic sheep, there have been no recognized outbreaks of CJD or vCJD in people who have lived in close proximity to the animals—and have eaten every part of them, including brains and spinal tissue—where concentrations of the TSE agent can most readily be found. If scrapie produces CJD in human beings, it probably does so at the rate of about one-in-one-million of the people who come into contact with it.

At this time, scientists believe that chronic wasting disease has not produced CJD or vCJD in people who eat deer or elk meat. No one can say for sure. Researchers have been alert to the possibility that it could ever since the appearance of vCJD in England, and laboratory studies show the possibility exists, although there are substantial natural barriers. In 1999, when CJD was diagnosed in three Americans in their late 20s and 30s, all of whom had eaten venison or elk, a warning flag went up.

Extensive investigation of the cases by Dr. Ermias Belay, of the Center for Disease Control in Atlanta, failed to find any concrete connection to CWD-contaminated meat. “We focused on these three cases because the disease is so rare in these age groups, and because they had venison consumption in common,” said Dr. Belay. “The venison was not from an area where we knew there was CWD, so we collaborated with the USDA to take deer brain samples from the areas where the patients obtained their venison. In over 1,000 samples, we found no CWD. The data that we have argues against a link between venison consumption and these CJD cases.”

Belay said that the science of epidemiology relies on finding “clusters” of disease, and then finding what the victims have in common to determine what is causing the illness. Venison consumption alone was not enough to prove a link, given the fact that an estimated 3 to 4 million Americans eat venison or elk meat every year.

Unfortunately for deer and elk, chronic wasting disease may be the most contagious of any TSE disease yet discovered. The prions may pass from animal to animal through saliva, urine, feces or even skin particles. Nobody knows for certain. It is certain, however, that close contact contributes to the spread of the disease— including confinement as on game farms and feeding game animals in the wild. No one can yet say whether the infective prions linger in soil, the decomposing carcasses of victims or on vegetation.

According to an exhaustive report on chronic wasting disease in the Denver-based Rocky Mountain News, there were scrapie-infected sheep kept in the same pens that were used for the mule deer and elk research. Some scientists speculate that the scrapie crossed the “species barrier” to infect first the mule deer, then the elk. Recent laboratory findings

show that TSEs can be much more destructive when they shift into new species, which could account for the high death rate of the mule deer.

However, says Beth Williams and other researchers, it is just as likely that the study deer, which were captured from the wild, were already infected with the disease. In the close-quarters contact of captivity, the disease may have reached levels of infection that would be impossible in the wild, where predators and harsh winters quickly dispatch weakened animals.

“It is a chicken-or-the-egg question,” said Williams, who is now one of the leading CWD researchers in the world. “We may never know that answer.”

Whatever that answer may be, researchers before 1977 had no reason to take extraordinary precautions with the pens or the research animals. “No one at the time understood that what was killing the deer in the pens was a contagious disease,” Dr. Mike Miller, who is leading the Colorado Division of Wildlife’s CWD control efforts, told Rocky Mountain News. The result was that many research animals were released back into the wild. CWD began appearing in wild mule deer and elk shortly thereafter. Since no one had ever looked for the disease before that, there is another chicken-or-the-egg question here.

In the past 30 years, CWD has spread, with widely varying rates of infection, throughout a roughly 14,600-square-mile area of north-central Colorado, southeastern Wyoming and a small corner of Nebraska. The natural spread of the disease has been very slow, and although the area provides good hunting country, the density of elk and deer is fairly low. Dr. Mike Miller once called CWD “an epidemic in slow motion.” Although wildlife officials in all three affected states monitored hunter kills for the disease, there was no great sense of alarm.

By 1995 however, it was evident that the problem in the wild was getting worse. Surveys of hunter-killed mule deer and random sampling of kills by wildlife officials were showing “hotspots” of infection, ranging up to 15 percent in mule deer.

Such a rate of infection in any other known TSE disease would be a shock—at the height of the BSE epidemic in England, rates of infection in dairy cattle herds averaged only 2 to 3 percent. Wild elk in what Miller and other researchers were now calling the “CWD endemic area” were holding their own, with infection rates of only 1 to 2 percent. The elk seemed to have some natural resistance to CWD, and it seemed possible that they might carry the disease, and transmit the disease to deer, without becoming infected or without displaying any clinical symptoms.

That possibility soon took on grave importance.

In 1996, CWD appeared on a commercial elk farm in Saskatchewan. Echoes of the news were heard in state and provincial agriculture departments and in the offices of wildlife officials all over the U.S. and Canada. The elk farming industry was in a period of frenzied expansion, and its lobbyists had just succeeded, in Colorado and many other states, in removing the industry from the yoke of regulation by wildlife officials and placing it under the sole control of state agriculture officials, a move that researcher Beth Williams calls “a simple failure of common sense.” Many hunters, biologists and other wildlife advocates were horrified. Among them was Dr. Valerius Geist, professor emeritus of environmental science at the University of Calgary, who had been warning the Canadian government that disease would sooner or later sweep the elk industry. “We

had already been through a TB epidemic on game farms here in Canada,” he said. “We knew that CWD existed in the wild in Colorado, and that theft of wild elk was occurring, so it was just a matter of time.”

Many elk and deer farmers and their lobbyists claim that CWD was introduced into their industry by accident. In 1990, Colorado Division of Wildlife officials confiscated a group of elk from a game farmer who had stolen them from the wild. They then held them for several months in the CWD-infected Fort Collins research pens. The wildlife officials then traded these wild elk to game farmers for red deer. At the time, red deer were popular on elk farms for their ease of handling and the supposed “hybrid vigor” they could offer domestic elk herds. But as biologists grew increasingly concerned that these “reds” would escape and genetically contaminate wild elk herds, Colorado joined many other states and provinces in banning them. Mike Miller confirmed to the Rocky Mountain News that these trades did indeed occur in 1991, and said they realized after the trades that “it wasn’t such a good idea,” and tried to buy the elk back. They recovered and killed 13 of them, and all tested negative for CWD. But several of the others disappeared into the elk industry.

Another possibility that both game farmers and wildlife advocates acknowledge is that CWD has been introduced to domestic elk herds through the fences, from the wild. If this is true, then the permitting of over 20 elk ranches by the Department of Agriculture within the CWD endemic area of Colorado was a tremendous mistake. Permitting ranches without double-fencing was a dubious decision as well.

“We [the Colorado Division of Wildlife] asked for a moratorium on elk ranches in the endemic area,” Mike Miller told me in a 1999 interview. “But the Department of Agriculture permitted them anyway. The Department of Agriculture is involved in encouraging this industry. I cannot think of a single permit for an elk ranch that has ever been denied because of wildlife concerns.”

In the same 1999 interview, Miller warned that there were limits to the ability of the Division of Wildlife to deal with the potential effects of the disease on wild big game animals. “If infected animals were brought into the western part of Colorado, where our game herds are much larger, it would be a disaster,” he said. “This is not like brucellosis, where you have tests and vaccines to help you. About the only way to manage CWD is to do a density reduction on the animals that are carrying it, which basically means clobbering the native wildlife.”

By late 1999, CWD had been found on elk farms in five additional states, as well as Saskatchewan. By November of 2001, two CWD-infected mule deer had been shot outside the fences of a heavily infected elk ranch in Saskatchewan, a kind of warning bell of what was to come. In September 2001, 400 domestic elk from an infected ranch deep within the endemic area near Stoneham, Colorado, had been shipped to 15 states across the U.S. One shipment of domestic bulls from the Stoneham ranch ended up at the Trophy Mountain Ranch, a fenced trophy shooting facility on Colorado’s West Slope. After clients at Trophy Mountain shot two of the bulls in October, both tested positive for CWD. The killing of wild mule deer began in November 2001, searching for CWD-positive animals outside the fences of the Trophy Mountain. Miller remained optimistic that the disease could be contained by killing wild deer and elk outside the ranch fences.

“To the best of our knowledge it hasn’t spilled out of any of these farms yet, and we’ll do everything in our power to see that it doesn’t,” he told me shortly thereafter. “We’ve caught it early, and the good news is that we may be able to contain it.”

But the optimism proved to be unfounded. It was not at the Trophy Mountain, but at the Motherwell Elk Ranch, situated in prime big game country near the town of Craig, where the disease was found, first in wild mule deer that had been entrapped when the high fences went up around the ranch, and then in six additional mule deer that lived outside the fences. Colorado Governor Owens stepped in and called a press conference to announce the findings. He said, “I didn’t speak out before because I try to be careful not to use this office to mobilize issues until it’s absolutely necessary. I don’t want to cry wolf.”

Owens said that the finding of CWD in the rich game country of the West Slope of the Rockies could cost Colorado hundreds of millions of dollars, spent by big game hunters and visiting wildlife enthusiasts. He ordered the Department of Agriculture and the Division of Wildlife to lay aside whatever differences they had and work together to solve this new, apparently colossal problem. “We don’t want to face a Colorado without the wild deer and elk herds that are so much a part of our quality of life,” he concluded, calling for aggressive action to fight the spread of the disease. “The danger of not being aggressive enough is far greater than the danger of overreacting.”

For Colorado and other states and provinces that share Owens’ philosophy, the attempt to eradicate the disease in the wild has already proved extremely costly, both in terms of money and in wild deer and elk. Of the 613 wild mule deer shot by state employees outside the Motherwell, 10 tested positive for CWD. None of the 134 wild elk killed proved to be infected. To claims by the ranch owner that the disease must have moved in from the wild, Division of Wildlife spokesman Todd Malmsbury said, “We have tested thousands of animals from that area and from all over Colorado, and we have never found this disease in the wild outside the endemic area until we found it around the Motherwell Ranch.”

Department of Agriculture officials moved in to “depopulate” (slaughter) the domestic elk on the Motherwell, offering a federal indemnity payment of up to \$3,000 per animal, as they have been doing with CWD-infected elk ranches for the past year. But at time of writing ranch owner Wes Adams has refused the deal, asking for more than 1 million dollars, an amount of public money that Colorado agriculture officials say they do not have. The suspect elk remain in the single-fenced facility. Denver attorney Suzanne O’Neill says that she plans to file a lawsuit against Mr. Adams on behalf of the Colorado Wildlife Federation.

The U.S. Department of Agriculture originally allocated \$2.6 million to indemnify elk farmers whose herds were destroyed to try to contain the December 2000 outbreak. Since then the department has spent an additional \$15 million to buy out and destroy the 20 Colorado elk farms within the CWD endemic area.

Colorado wildlife officials have allocated \$300,000 of the public’s money to double-fence the trophy shooting area at the Trophy Mountain ranch, part of which is a section of State Trust Land that the owner leases from the state of Colorado for less than \$1,000 per year. The domestic elk at the Trophy Mountain were slaughtered by agriculture officials after the CWD infection was found, and \$500,000 in USDA indemnities paid to ranch owner Mark Mitchell, who has recently restocked the shooting

enclosure with several hundred new domestic elk. Under current Department of Agriculture regulations, there is no quarantine, no waiting period to restock, and no requirement that the ranch make any attempt to rid its grounds of CWD.

Meanwhile, the killing of Colorado's public wildlife is well underway. The plan, outlined by Malmsbury, is to reduce the density of mule deer and whitetails throughout the endemic area (elk densities are already relatively low). The department hopes to bring the deer population from 25,000 to 20,000 or less, and try to hold it there. Permits will be issued to hunters, and wildlife officials will be in the field trying to kill 5,000 mule deer and whitetails annually for at least the next three years. "What we hope to see," said Malmsbury, "is that a lower density of animals will produce a lower prevalence of the disease." Killing will be concentrated on the "hotspots" in the endemic area, where the highest rates of infection have been found. In the words of one wildlife official, the goal is to bring the disease down from "a brushfire to an ember," wherever possible.

The average infection rate over most of the endemic area (in northeast Colorado) is about 5 percent for mule deer and 1 to 2 percent for elk. A narrow area on the southern perimeter of the endemic area, in Larimer County, showed a possible infection rate of almost 30 percent in wild mule deer, according to Malmsbury. If this proves to be true, it would be one of the highest infection rates ever recorded for a TSE disease. Wildlife officials will concentrate the killing of deer in this area, hoping to slow the southward spread of the disease. No one knows exactly the southern range of the disease—three more positive animals (roadkills) have recently been discovered within the city limits of Boulder, and another just northeast of Denver. Some of the highest infection rates, not surprisingly, are in heavily settled areas where hunting pressure is light, predators are few, and residents have been feeding and concentrating deer.

In October of 2000, Mike Miller and his research colleague, John Gross, published a computer model of what might happen if CWD is left to run its course unchecked in the wild. The model is based, in part, on the severe infectivity that the disease displayed in the corral studies at the Fort Collins and Sybille, Wyoming, research facilities, and on recent studies of "hotspots" found throughout the endemic area. It shows a grim portrait of the future—over a 60 to 100 year period, Colorado's mule deer populations crash.

It's worth noting that the model treats CWD as a new disease, dating back only to the time of its discovery in the late 1960s. Otherwise, clearly, the model would have been already been played out over millennia, rather than the decades that it indicates it will take for the disease to destroy Colorado's wildlife resource. The model also assumes no natural resistance to this TSE disease. There is a rising hum of debate among various groups from researchers to hunters and other wildlife advocates, as to whether either of these assumptions is valid enough to justify the killing of thousands of big game animals.

Most researchers, even those who hate the idea, believe killing deer or elk wherever CWD has or is believed to have been newly introduced is probably the best course of action. A majority support the "density reduction plan" across the endemic area and the heavy culling on the peripheries.

"It is appropriate to try and figure out a way to stop this," said Beth Williams from her office at the Wyoming State Veterinary Laboratories in Laramie, one of the forward posts in the struggle to understand CWD. "Like backfiring a wildfire. I

understand that the model is just that—a model, based on assumption and not the real world, but I am convinced that if we do nothing, the disease will spread.”

Dr. Terry Spraker, a CWD researcher at Colorado State University, is warily supportive of the culling efforts. “I don’t fully understand what the Division of Wildlife people are doing, but I think the idea of culling on the perimeter is a valid management strategy. Now if you ask if you can do that—the shooting, the culling—without causing the deer to disperse and take the disease into new areas, I’m not sure about that either.”

Spraker believes the culling efforts have particular value in the absence of the healthy ecosystems that might well weather a disease event like this one. “The normal, traditional migratory movements of these wild herds would give you a dilution factor for diseases,” he said. “If we had healthy migration patterns, it would be different. But we don’t. We have highways and fences and we have animals concentrated in the last of the habitat, and most of that habitat is stressed, hard. I don’t think you can expect CWD to wear itself out in light of those factors.”

On what is believed to be the southern periphery of the endemic area, the culling efforts are often conducted on private properties and on public open spaces heavily used by people to whom killing such a large number of animals is simply incomprehensible, given the fact that there has been no large-scale die-off due to the disease. The Division of Wildlife efforts have run into some very heavy opposition. Although Malmsbury originally said that opposition to the culling efforts was spearheaded by “Boulder animal rights activists,” it soon became clear that a wide variety of local residents were questioning the department’s plans and activities in the area.

“They are talking about killing 15,000 deer,” said Marcia Barber, who lives outside of Boulder in an area where heavy culling efforts have been conducted. “You don’t have to be an animal rights activist to object to that, especially since it’s based on no evidence whatsoever. I mean, what the heck are they doing?”

Barber and other residents opposed to the actions were soon joined by Charles Southwick, a former professor of pathobiology at Johns Hopkins University and professor emeritus of environmental, population and organismic biology at the University of Colorado. Southwick has been conducting a population study of mule deer in an area west of Boulder for over 20 years, an area that is now considered one of the “hotspots” on the perimeter of the endemic area.

“We found one wild mule deer, five years ago, in the study area that tested positive for CWD,” Southwick said. “Since then there has been no die-off that we can see. Mule deer are tough, adaptable animals, always on the move. The DOW did one cull in our area around Rabbit Mountain and said they found two positives, then they came back, killed 12, and found three positives. Then they came back, took 30 animals and found 10 positives. If that’s true, they must be causing dispersal, opening holes, and animals, maybe infected animals, are coming in from other areas looking for forage. The plan couldn’t be more counterproductive.”

Southwick believes that too much is being made of the connection between CWD and mad cow disease. “Everybody is scared by that connection, which means plans are made in haste,” he said. “And the animals are not the same. The genetic variability of wild animals is many times greater than that of domestic cattle or sheep. The model that the Division of Wildlife is working from is flawed, assuming no genetic or other resistance. Elk show resistance. Some mule deer show resistance—obviously, they are

still alive and healthy—and they are killing them off, the healthy ones with the sick, killing the very animals that could bring us out of this situation once and for all.”

When Todd Malmsbury was asked the Division of Wildlife’s opinion of Dr. Southwick’s theories, he was respectful, but succinct. “Based on the data we have, we just don’t believe that letting this thing run its course is in anybody’s best interests.”

For Tony Peri, a Grand Junction trophy mule deer hunter, it is difficult not to be extremely discouraged. “You hear about them killing 1,000 mule deer and getting just a few positives, and you have to wonder about it. Everybody is really concerned about it, but nobody knows what the alternatives would be.”

North of the Colorado state line, in the CWD endemic area of Wyoming, no culling or killing is planned, even though one of the major “hotspots”—an area where the mule deer show a 15 percent rate of infection—is known to be in the rough country near the Sybille research facility. Beth Williams and her husband Tom Thorne, Wyoming Fish and Game’s interim director, have a cabin at Sybille Canyon, where they have hunted and watched mule deer for the past 10 years. “Last year, Tom found a dead deer in Sybille Canyon,” said Williams, “but it is not something we see all the time. There’s no big die-off with CWD—it is very slow, but steady. I think that we may be seeing the turning point in that Sybille country now, where there are just less and less deer.”

Williams also said that she and other longtime CWD researchers never expected their work, and this disease, to be so much in the news. “In Wyoming, there just hasn’t been that much change in the seriousness of the situation,” she said. “It is still kind of a slow-moving problem. All of us are kind of amazed at how high-profile this whole thing has become.”

In Cheyenne, Tom Thorne said that Wyoming will act as a “control” in the experiment of culling and testing deer and elk—in other words, there will be none. “We are doing everything that Colorado is doing except for killing deer,” he said. “One of the things we hope to learn from this whole thing is whether the killing will work—and that depends on how much of the infective agent persists in the ground, or wherever it is found. If the agent is a persistent part of the environment, then the killing will not work.

“We are working closely with Colorado—we always have—and I believe they are doing the right thing.” Thorne notes, “Many of the outbreaks we know about in free ranging wildlife are linked to the game farm industry, and we don’t have that here. I wouldn’t have much enthusiasm for destroying our deer herds while the same disease is being hauled down the highway in a truck.”

Whatever actions Wyoming decides to take in the future, the entire CWD story took an abrupt and bracing shift with the discovery of the disease on a mixed whitetail and elk farm in Sioux County, Nebraska, far to the east of the known endemic area. Captive whitetails showed extraordinary rates of infection—over 52 percent—and CWD prions appeared in the lymphatic and brain tissue of young fawns, causing researchers to step back and look again at the assumptions of long incubation periods for all TSEs. According to Bruce Morrison, assistant administrator of wildlife for the state, wild deer outside the fences also showed high rates of infection.

“We killed 104 wild whitetails out from the fencelines of the game farm to five miles, and we got seven positives so far. Out to 10 miles, two positives out of 57, 20 miles, one positive out of 176. We’ve never seen the disease here before, so we are assuming the game farm is the source.”

Morrison said seven out of 54 of the captive elk on the game farm tested positive for CWD. “We did trace-backs on all those elk, and none of them traced back to a known infected farm. But that just doesn’t mean much anymore.”

Nebraska will use its October 28-February 13 regular deer season to try to reduce the population of deer in the region. There will be no bag limits, and so far, eight out of 10 landowners say they will help as much as they can. “The hunting was just now getting good in there,” said Morrison. “We spent 30 years trying to get it to where it is now, and we are going to destroy it. Based on what I know, we don’t have a choice.”

Just as the Nebraska findings hit the news, testing of white-tailed deer in south-central Wisconsin revealed 18 CWD-positive whitetails north of the community of Mount Horeb, near Madison. Wisconsin Department of Natural Resources and the vast community of serious Wisconsin hunters were shocked. No one had ever seen the disease across the Mississippi River before, and the Nebraska whitetail infection was fresh in everyone’s minds. And here was genuine mystery, because, although there are 947 deer and elk farms scattered across Wisconsin, none lie close enough to the findings area to be seen as the source of this infection. Furthermore, the places where the sick deer were killed were in a tantalizing circle about 12 miles across, as if the solution to the puzzle must surely rest at the dead center, as if at the very point of a protractor. But the world is not so exact, and there was no smoking gun there, centered in those quietly beautiful farmlands and long, heavily forested ridgelines—a whitetail hunters’ dream country. The mystery remains, but with the manpower that Wisconsin is bringing to bear, there may well be answers one day. The man who may find them is Thomas C. Solin, the supervisor of wardens.

“We are looking at this as a wildlife event, in the absence of that smoking gun on a game farm,” said Solin. “There is one game farm that has had some traffic, near the community of Arena, but there’s no confirmed cases of CWD there. We are looking at feed blocks, at deer feeding stations and the feed they are using in that area to see if there is animal protein in it.” Solin and many others are now trying to track deer and elk feeds to see how much meat and bone meal (MBM), may be in the mix to build bigger and stronger trophy antlers. Following the mad cow epidemic in England, the U.S. banned the feeding of MBM to all ruminants, which would include deer and elk, but enforcement, and even labeling of the feeds, which are still legal for pigs and chickens, has been haphazard. Wisconsin quickly passed a ban on all deer feeding in the state after CWD was found.

Solin said that two years ago, an ear-tagged mule deer—which are not native to Wisconsin—was seen in the Mount Horeb area. “We also have reports, maybe rumors is a better word, that people have imported white tails and released them in that area to try and improve the herd. That is a big QDM, or Quality Deer Management area, and something like that might have happened. The whole idea of feeding deer was a bad idea to start with. It is a piss-poor way to manage wildlife, for every reason.”

According to Solin, Wisconsin’s game farm industry has not been as adversarial to the wildlife officials as in many other states. “They don’t give us that much trouble. We do have escapes though, and we’ve had six since CWD was discovered. Two of those were more like releases—the owners looked at the new regulations and just turned them out, one elk and the rest whitetails, none of them with ear tags.”

The Wisconsin Department of Natural Resources plans the most ambitious attempt to destroy wild deer in the history of the U.S., in an all-out attempt to stop CWD before it can gain a foothold in what may be the richest and most densely populated whitetail habitat on the continent. Officials hope to cull 25,000 deer from the area over the next two years.

How to dispose of their carcasses is the subject of intense debate. Wisconsin food pantries have made it clear that they will accept “none of the deer ever.” The state has concluded that putting the carcasses in rubber-lined landfills is not a suitable solution. They’re currently investigating the possibility of a mobile incinerator large enough to handle the volume and capable of reaching temperatures hot enough to potentially destroy or neutralize the infectious CWD agent. Whatever option Wisconsin officials ultimately choose will certainly carry a multi-million dollar price tag.

As in Colorado, they face growing opposition, coalescing around a group called Citizens Against Irrational Deer Slaughter (CAIDS). As in Colorado, officials are moving ahead with their plans, convinced that, as difficult and awful as it may be, they must make the attempt. Speaking of CAIDS, Julie Langenburg, of the Department of Natural Resources, said, “I appreciate their input, but we’re going to stand tall and go with the best science. We realize this is a grand experiment, no guarantees. But we think we have a good chance, and we’re going to take it, right now. Stand tall and try to stop this thing rather than living with the consequences of not trying.”

Langenburg lives on her own small farm in the area and said she understands the dissent. “What we are seeing here is brand new—wildlife management driven by disease control, not hunting or recreation, not even driven by the needs of communities. Nobody likes it.”

Hal Herring previously wrote about chronic wasting disease in our January-February 2000 and January-February 2002 issues. He has published articles on the topic in *Field & Stream*, *The Economist*, *High Country News* and elsewhere.

CWD: The Elk Foundation’s Role
by David Stalling

With presentations such as, “Can TSE Diseases of Ruminants Be Distinguished Based on PrP-res Glycoform Profiles?” and “Molecular Epidemiology of Creutzfeldt-Jakob Disease in the U.S.,” the Elk Foundation-sponsored Chronic Wasting Disease Symposium held in Denver, Colorado, August 6-7, was hardly for the scientifically disinclined. It was, however, a chance for more than 400 wildlife professionals, government officials, scientists and others from throughout North America (and a few from other Continents) to share knowledge about the disease and begin developing consistent responses.

“I now ask each of you—did you learn?” said Len Carpenter, of the Wildlife Management Institute, during a summation of the conference. “I would answer yes. I am sure each of you will do the same.” However, we also learned that there is a lot more to

be learned. We are far from knowing everything we need to know to successfully manage this disease. Awareness of what we do not know is as important as what we know, and I think this conference helped us recognize our knowledge gaps.”

Jack Ward Thomas, Elk Foundation board member and Boone and Crockett Professor of Wildlife Conservation at the University of Montana, summed up a common theme at the conference in his call for a well-coordinated, national response to the disease, and the need for accurate, consistent communications to overcome hype and misconceptions sometimes found in national media.

“Clearly, appropriately qualified professionals should take the lead in these efforts,” Thomas said. “However, citizens groups with interest in CWD—such as the CWD Alliance founded by the Boone and Crockett Club, the Rocky Mountain Elk Foundation and the Mule Deer Foundation—have responsibility to assure that improved, coordinated and appropriately funded actions take place.”

With just such a goal in mind, the Elk Foundation not only helped fund the CWD conference and found the CWD Alliance, but has also published dozens of timely, informative and accurate articles in *Bugle* and *Wapiti*. In addition, the Elk Foundation continues to work with the University of Wyoming, Wyoming Game and Fish Department, Colorado Division of Wildlife, the Agriculture Research Service and others to assist in research that will increase our knowledge of the disease and help managers respond.

Is it Safe To Hunt?
by David Stalling

Deer hunting may be as sacrosanct in Wisconsin as dairy farming and the Green Bay Packers. More than 700,000 people hunted deer in Wisconsin last year, contributing more than \$25 million to state wildlife programs through license fees and pumping an estimated \$250 million into local economies. But a poll conducted last May indicates that 248,000 people—36 percent of the state’s deer hunters—may not hunt next year. Why? Fear of Chronic Wasting Disease (CWD), a form of Transmissible Spongiform Encephalopathy (TSE) that infects deer and elk.

Hunters worry that CWD could jump the species barrier, infecting humans with another deadly type of TSE called Creutzfeldt-Jakob Disease (CJD). Recently, Wisconsin health officials found a possible correlation between a deer hunter who died of CJD back in 1993 and two of his friends who, within six years, also died of rare brain disorders: they all partook of venison feasts. There is no definitive link between the deaths and CWD, but the news may further the resolve of many hunters to stay out of the woods come fall.

“If more than one-third of hunters do not hunt, imagine the impacts on the economy and on wildlife management,” said Gary Wolfe, a longtime elk biologist and former executive director of the Elk Foundation, during a Chronic Wasting Disease Symposium held August 6-7 in Denver, Colorado. Wolfe now heads up the Chronic Wasting Disease Alliance—a cooperative venture between the Elk Foundation, Mule Deer Foundation and Boone and Crockett Club.

“We need to be honest with the public, and tell them what we know,” Wolfe said. “But we need a consistent message; we need to keep this disease and its risks in

perspective, and not overreact.” Here’s Wolfe’s perspective: the risk of getting into an automobile accident on the way to the trailhead, getting shot, thrown off a horse or having a heart attack while hunting are all greater than the risk of contracting a human variant of CWD.

“Nothing in life is risk free,” Wolfe said. “I plan to minimize risk and continue to hunt.”

People are sometimes justifiably cynical of attempts by scientists and government to placate the public. After all, British officials spent 10 years assuring people that another form of TSE, known as Mad Cow Disease, could not jump from bovine to humans. And then it happened. But the science on CWD is thorough and, according to nearly a dozen scientists at the CWD symposium speaking as part of a panel called “Assessing the Potential for Interspecies Transmission,” it is also reassuring.

Beth Williams, a professor of veterinary sciences at the University of Wyoming who first discovered CWD in 1977, examined the susceptibility of cattle exposed to CWD by intracerebral inoculation, oral inoculation and by contact with CWD-infected cervids in endemic facilities. Three of 13 cattle inoculated intracerebrally developed evidence of CWD, Williams reports. But cattle exposed via more natural routes of exposure have shown no evidence of CWD.

In parts of Wyoming and Colorado where CWD has existed for at least 30 years, an average of less than 6 percent of deer are infected. Infection rates for deer in endemic areas of Colorado vary from less than 1 percent to 13 percent. CWD is far less prevalent in elk than deer. Less than 1 percent of elk found in areas where the disease occurs in northeastern Colorado are infected. If you hunt in these areas, the chances of killing an infected animal are slim. If by chance you do, there is currently no scientific evidence that CWD has or can spread to humans, either through contact with infected animals or by eating meat of infected animals. The Center of Disease Control states: “the risk of infection with the CWD agent among hunters is extremely small, if it exists at all,” and “it is extremely unlikely that CWD would be a food borne hazard.”

In presentation after presentation, researchers concurred that cattle are difficult to infect with CWD in intentional, controlled situations and nearly impossible under natural conditions. Sheep, mice and raccoons could not even be infected on purpose. As for humans? Chances are greater that you’ll be hit by lightning. Precautions seem wise, and cynicism may be justified, but to not hunt at all?

“I hunt elk in an endemic area of Colorado,” Wolfe said. “I’m not planning to have any scrambled eggs and elk brain for breakfast, but I’m going to continue to hunt in my favorite place. Certainly, I’ll take precautions to minimize any risk. I’ll be careful, but I’m going hunting.”

Precautions:

Public health and wildlife officials advise hunters to take the following precautions when pursuing or handling deer and elk that may have been exposed to CWD:

- 1 Do not shoot, handle or consume any animal that is acting abnormally or appears to be sick. Contact your state game and fish department if you see or harvest an animal that is unhealthy.
- 1 Wear latex or rubber gloves when field dressing your deer

or elk.

- 1 Bone out the meat from your animal. Don't saw through bone, and avoid cutting through the brain or spinal cord (backbone).
- 1 Minimize the handling of brain and spinal tissues.
- 1 After field dressing an animal, wash hands thoroughly and clean knives and saws with strong chlorine bleach.
- 1 Avoid consuming brain, spinal cord, eyes, spleen, tonsils and lymph nodes of harvested animals. (Normal field dressing coupled with boning out a carcass will remove most, if not all, of these body parts. Cutting away all fatty tissue will remove remaining lymph nodes.)
- 1 If you keep the entire skull (for a European mount), you should insure all meat is removed, wearing rubber or latex gloves while doing this, and soak the head in strong chlorine bleach.
- 1 If you have your deer or elk commercially processed, request that your animal is processed individually, without meat from other animals being added to meat from your animal.

Most states with CWD in wild deer and elk populations are offering hunters an opportunity to have their game tested for CWD. To learn where to get your animal tested, or if you have specific questions regarding the status of CWD where you are planning to hunt, please contact your state game and fish department.

Precautions and questions/answers provided by the Chronic Wasting Disease Alliance at <http://www.cwd-info.org>