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CHRONIC WASTING DISEASE:
What You Should Know

By Dr. James C. Kroll



In Part I of this series, I pointed out that the field of wildlife diseases is relatively new. We know so little about the diseases affecting wild animals. As human populations continue to grow—and wildlife is crowded into less and less space—opportunities for cross-species infection significantly increase. Also, as we learn more and we monitor more extensively for diseases, we are discovering a host of "organisms" affecting and endangering game animals.

In this installment, I will try to bring you up to date on the "new kid on the block," chronic wasting disease (CWD). As noted last issue where I discussed tuberculosis (Tb) in deer, the facts bear out wildlife diseases originate from one of two sources: they either arise by trans-species infection (one species to another) or they exist naturally at some low level until the right conditions occur.

Tuberculosis developed in the wild Michigan deer herd as a result of the mixing of infected cattle with deer. Recent outbreaks of anthrax in western Texas illustrated the sporadic eruption of a disease occurring naturally. In the case of CWD, as you will learn in this article, it is not so clear.

I want to warn you ahead of time, this discussion may become a bit "technical" at times, but I'll ask you to hang in there and read every word; it's important to the future of deer and deer hunting. There are a lot of rumors floating around about CWD and the more you learn about this and other diseases, the better equipped you are to judge fact from fiction, genuine concern from irrational panic.

[CONTINUED ON PAGE 70]

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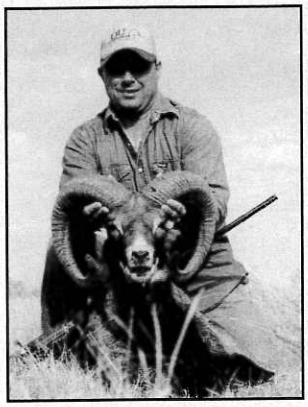


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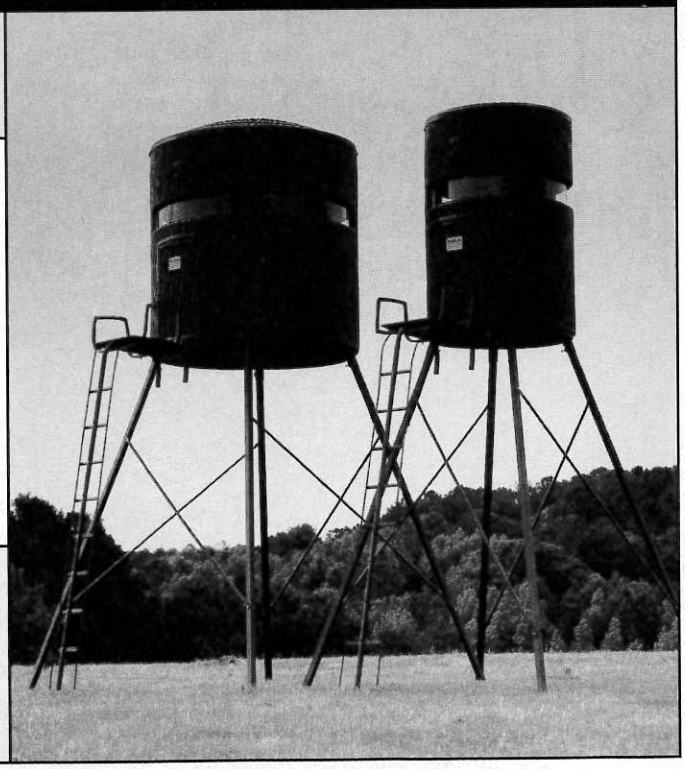
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Chronic Wasting Disease: What You Should Know

[CONTINUED FROM PAGE 68]

What is it?

No doubt you are aware of the panic that spread across Europe, especially the U.K., about a decade ago when a new and mysterious disease showed up in cattle. Although called by various names, most commonly "mad cow" disease, it was given the official name Bovine Spongiform Encephalopathy or BSE. It is one of a group of diseases called Transmissible Spongiform Encephalopathies or TSEs. The name is long and technical, but it simply means a disease than can be transmitted which turns the brain into a "Swiss cheese" of spongy holes.

Cattle infected by the disease show a host of symptoms, including emaciation, staggering, drooling and erratic behavior. Safe to say, its appearance in the U.K. virtually destroyed the cattle industry there. Normally big beef eaters, Englishmen looked longingly toward the vegetarian way of life. Farmers committed suicide by the droves as their farms failed and panic set in.

Spurred on by the media, the entire European community closed its borders to importation and instituted plans to keep the disease out of their countries. Sales of beef plummeted in Europe and sales of alternative red meat sources such as horse meat and venison increased. English farmers were effectively cut off from the world beef market. Consumer confidence throughout Europe declined as media coverage intensified.

Actually, this disease, or at least the presumed causative agent, is not new to mankind. The first known form of these diseases appeared about 250 years ago in sheep and was given the name scrapie. It always is fatal and is a long, degenerating disease, apparently spreading by moving flocks from one farm to the next.

Scrapie first appeared in the U.S. at the end of World War II in Michigan. A flock of sheep had been imported from England through Canada. To date, more than 1,000 flocks have been reported with the disease, according to the USDA. The disease appears to concentrate within one variety, the Suffolk breed and a host of cross-bred animals. (This point will be important later when I discuss possible genetic relationships in deer.) Only Australia and New Zealand are now considered "free" of this disease.

Sheep and cattle are not the only victims of TSEs. Similar diseases have been reported in mink, cats, monkeys and man. Man seems first to have contracted a variant of TSE in New Guinea. It was called Kuru.

First reports of Kuru came in the early 1900s among aborigines known as South Fore. These folks inhabited the Okapa Subdistrict of New Guinea, numbering about 8,000. The disease peaked in the 1960s when over 1,000 South Fore died from the disease. But, what had caused it?

These primitive peoples practiced a unique form of cannibalism! When someone died, the women of the group would meticulously strip the body of all

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meat and even clean out the skull cavity for the brains. These "morsels" then were shared with the family in a bizarre ritual. Apparently, it served two purposes. First, protein was scarce so it provided critical nourishment; and second, it must have been part of a religious ritual.

Subsequently, additional human variants of these diseases were identified in other portions of the world. They include Creutzfeldt-Jakob disease (CJD) (pronounced Kroytzfelt-Yaakabs), Gerstmann-Straussler-Scheinker Syndrome (GSS), Familial Insomnia (FFI) and nvCJD a "new variant" possibly derived from BSE.

In 1969, Kuru's apparent causative agent was shown to be one of the most unique disease agents currently known: the prion (pronounced pree-on). Prions are unique since they are not a bacteria, not a virus or even a retrovirus. They seem to be simple molecules of proteins (assemblages of amino acids) and may be part of a virus.

What's interesting is prions normally are found in the brains and nervous systems of all mammals. Their function is not certain, but some believe them to be important in protecting the sensitive neural tissue from harmful agents. They may even be involved in preventing dementia and Alzheimer's disease. This protective system can go seriously awry when these little molecules turn into "rogue" proteins, which explains what may be happening in the TSE diseases.

Rogue prions can be likened to the little toys kids bought by the thousands a few years back. They might look like a car now, but a child can rearrange the pieces to "morph" them into a doll or a train. The number of pieces never changed, just the way the pieces were arranged. So it is with the bad prions. They simply are rearrangements of the same 250 or so amino acids that make them up.

There are cells in the brain called Purkinje cells, which require healthy prions to survive. Purkinje cells are very important to our brains, aiding in memory. In TSE cases, Purkinje cells are conspicuously absent or reduced. There may be a genetic predisposition to TSEs,

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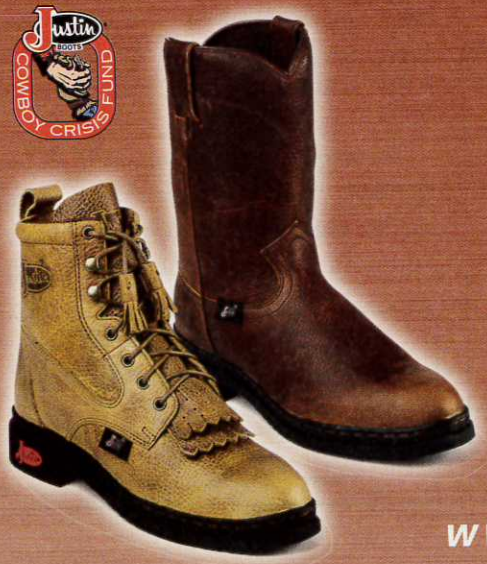
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Chronic Wasting Disease: What You Should Know

[CONTINUED FROM PAGE 71]

since some research in mice have documented a genetic relationship; hence, the prevalence of scrapie in Suffolk sheep. Some cases of CJD in humans also have been shown to be hereditary.

How did we find it?

With such a weird and exotic disease, how did we ever find it? Scientists always have followed a process taught long ago by a medical scientist named Koch (pronounced "coke"), who came up with a series of rules to be followed when identifying a disease: Koch's Postulates.

Essentially, he suggested the following questions:

Is the causative agent present in every case of the disease?

Can the agent be cultured?

Is the agent unique to this disease condition?

Can it be reproduced experimentally?

Can it be cultured from diseased animals?

Koch's Postulates served medicine for many years, but recently we have learned some diseases cannot be proven using them. TSEs are such a group. They just don't fit Koch's Postulates. In fact, we still are not sure prions even are the causative agent. Some scientists think there still may be viruses or retroviruses involved.

To further complicate the issue, recent research indicated not only a genetic predisposition to TSEs, but also possible involvement of environmental pollutants. At first discredited, now taken more and more seriously, a British amateur scientist (Mark Purdey) came up with a very interesting, yet plausible, theory on how at least BSE may have developed.

It seems the U.K. mandated applications of massive amounts of organophosphate pesticides in cattle to control the warble fly. A concentrated mixture of a pesticide Maneb (or Mancozeb) was poured along the backs of cattle to control the parasite. These pesticides were derivatives of the nerve gas poisons developed by the Germans for use in World War I. In peacetime, organophosphates became commonly used after pesticides such as DDT were made illegal in many countries.

The current thought is, organophosphates can alter the relationship between two important minerals: copper and manganese. Prions have a tendency to bind with manganese (Mn) to form rogue prions. This theory is gaining momentum. There also appears to be a link between some cases of CJD and lotions used to treat lice in humans.

A paper published recently by Dr. Larry Barger of the University of Illinois concludes: "The final chapter in the BSE story has not been written. The author acknowledges that some of the information included here is controversial...However, the role of copper and manganese in the development of prion diseases is gaining increasing credibility."

So, how did we find it? We don't even know what "it" is! Certainly, Koch's Postulates fail us in this mysterious disease. Is it caused by several factors, some of which are environmental? We honestly don't know. Since scrapie has been around for around 250 years (that we know), that disease could not have been caused by organophosphates.

We do, however, find a link in some of these TSEs to local soil conditions, notably those either deficient in copper or high in manganese. Is it always transmissible? Again, we really don't know. Is there a genetic link? There may be in some species.

The only thing I can state here without doubt is we have a mysterious, deadly disease that has been around for many years and we are now finding it in other

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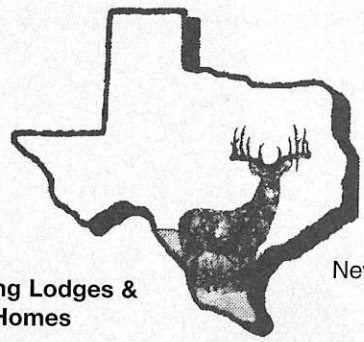
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species, which brings us probably to the most important thing on your mind—how about deer?

Where did it show up in deer?

The time line on the discovery of CWD in deer is somewhat short. Not all the facts are in and there are many rumors and undocumented facts. Yet, we do know that a mysterious disease showed up in the Colorado Department of Natural Resources Research Facility near Fort Collins in 1967. Sadly, it took 10 years to identify the causative agent, a type of spongiform encephalopathy.

How it got into the state's facility is not known—and may never be known. There are rumors sheep were also maintained there. Others say animal product feed was the source, while still others say the disease always had been in the wild. It was just the bad luck of the draw. At any rate, in spite of the presence of an obviously virulent disease, little action was taken for 10 years.

During this time, animals from the facility unwittingly were moved to zoological parks in Colorado and South Dakota. Others reportedly were used to mitigate losses of animals due to Tb testing. The zoos sold or gave deer to private landowners. Animals from the original herd ended up in other research facilities of western states. A pattern of potential transmission, just as we saw with the Michigan scrapie sheep, emerged. Later generation animals also were sold to ranches in Texas, Utah, Idaho, Nebraska and Pennsylvania. Fortunately, there have been no signs of the disease in Texas.

The "genie was out of the bottle!" The disease showed up in 1977 at a state deer research facility at Wheatland, Wyoming; in 1979 in elk at a Colorado research station; in free-ranging deer in north-central Colorado in 1981; the first game (elk) farm in 1996; in game farms in South Dakota in 1997; captive elk in Nebraska and Oklahoma in 1998; captive elk in Montana, Colorado and Nebraska in 1999; Saskatchewan in 2000; and in a Nebraska elk herd in 2002. Do you see a pattern?

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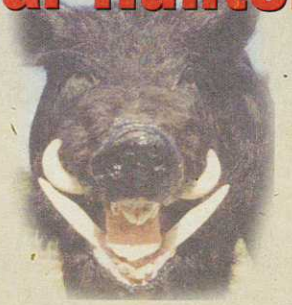
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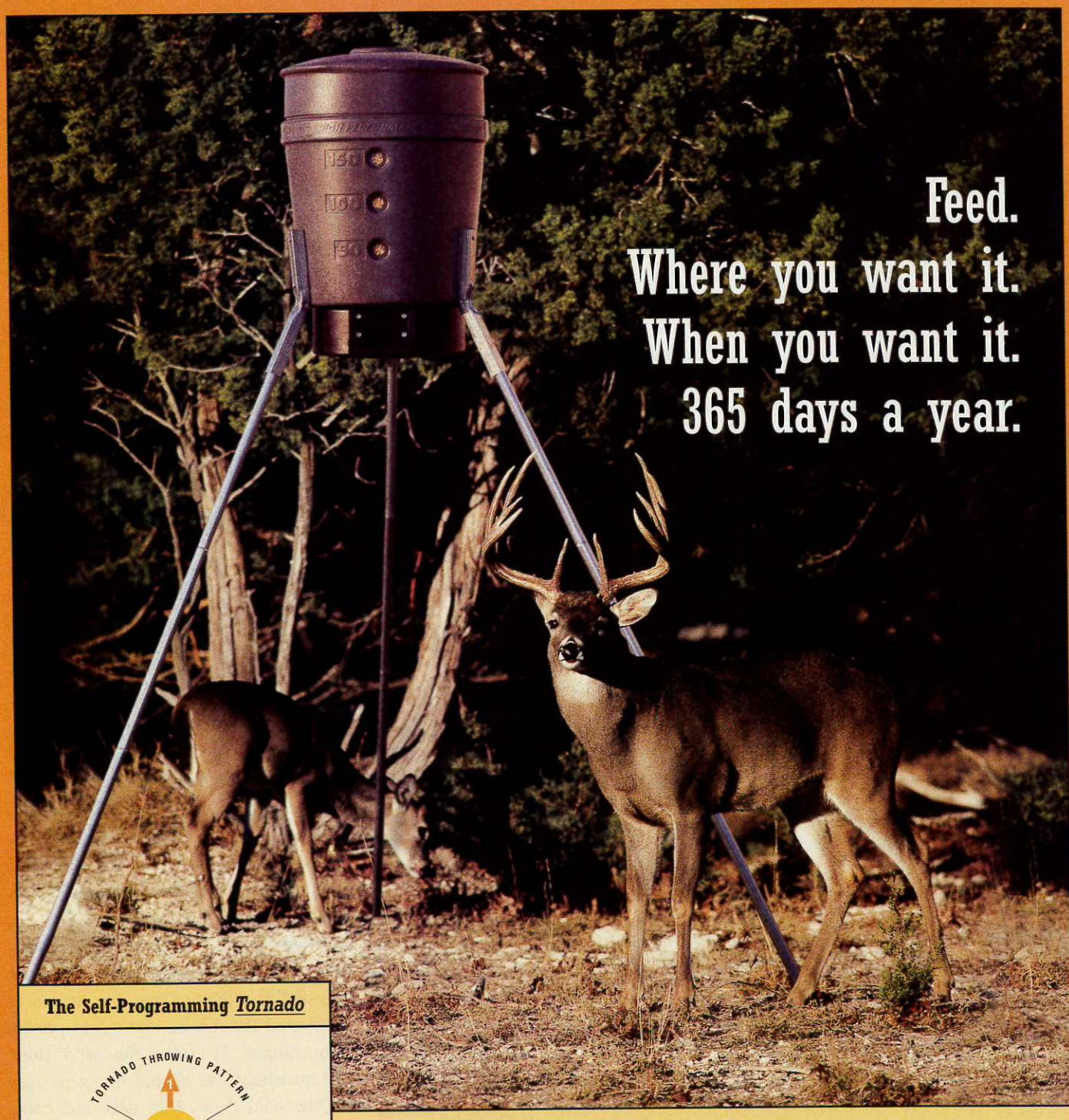
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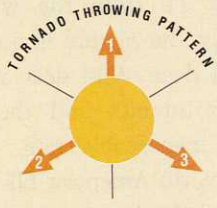
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[CONTINUED FROM PAGE 75]

interesting, particularly in that the elk involved came from captive herds with no history of the disease. Subsequent follow-ups on the sources for these animals have also shown no disease. Then, where did it originate? We only recently discovered that mule deer trapped within the fence have the disease at a very high rate. Many now think the elk got CWD from these animals. That means either 1) the disease has spread into the wild or 2) it has been present all along as some claim.

At the time of this writing, CWD has been reported in 14 wild white-tailed deer in Wisconsin. How these animals—some 900 miles away—came down with CWD is uncertain. Recent investigations suggest homemade mineral supplements high in manganese or food supplements made from chicken by-products may be implicated. Birds are notorious for passing high amounts of manganese through their systems into their feces. Others suggest the wild deer caught the disease from fenced ranches or deer farms nearby. Hopefully, we will soon find out.

Ok, where do we go from here?

Almost every state now has at least temporarily closed its borders to importation of deer, and it is my opinion this is a good idea. The Texas Deer Association has worked with TP&WD and the Texas Animal Health Commission in closing Texas' border. It just makes good sense to everyone concerned.

Arkansas Wildlife Director Hugh Durham put it best: "At least for now, we feel it is prudent to take a wait and see position, that's why we temporarily closed the Arkansas border to importation. That gives us time to catch our breath and put together a sound plan for dealing with the situation." Solid reasoning in my book! But what about the state where the disease originated?

Colorado, in spite of taking a very long time to react to the disease, has produced a "plan" for management. They actually tailored three plans: Big Thompson Plan, Red Feather-Poudre Canyon Plan and the South Platte River Plan. Although supposedly tailored to each situation, each plan is the same.

In essence, these plans are aimed at "...reducing the prevalence of chronic wasting disease (CWD) and keep it from spreading to disease-free areas." The operative phrase here folks is "reducing the prevalence."

The standard procedure for controlling CWD, Tb or any other disease in fenced deer has been total eradication, not reducing prevalence. The targeted goal in Colorado is to reduce prevalence to less than one percent. That means the disease will remain out there for the remainder of time! Is it then a "ticking time bomb?" This was the approach tried by Michigan for Tb in wild deer and officials now are willing to admit it will always be an issue in wild deer. What about fenced deer?

As I reported in Part I, the convenient thing about fenced herds is disease can be eradicated from them. The horrible situation involving Tb in Michigan deer has turned out to be a "win-win" for Michigan deer breeders. After six years of testing, the industry has been shown to be free of this disease. Only one herd ever contracted the disease and it was proven they caught Tb from the wild deer. Unfortunately, the picture is not so rosy for the wild deer. Wild deer and cattle continue to interact and the disease persists at a "manageable" rate.

Both the North American Elk Breeders (NAEBA) and the North American Deer Farmers (NAdeFA) Associations have taken a proactive stance on CWD, as has the Texas Deer Association (TDA). NAEBA worked closely with the USDA and their Canadian equivalent on eradication plans. Numbers of cases in farmed elk have been reduced significantly, as a consequence.

The plan focuses on eradication, not management. Likewise, Canadian farmers

Chronic wasting disease is far less prevalent in elk than deer. Less than one percent of elk in the endemic area have been found to be infected. Historical infection rates in deer herds vary from less than one percent to a range from three percent to 15 percent. The chart below indicates specific infection rates in mule deer harvested by hunters by game management unit. Monitoring of game management units varies between years and location. The information presented is based on a running total of hunter-killed mule deer tested since 1996.

Unit	Percentage of Infected Deer	Animals Tested
7	1.0%	97
8	2.8%	352
9	14.2%*	324
19	5.0%	418
20	5.0%	959
29	0%+	142
87	results pending	results pending
88	results pending	results pending
89	results pending	results pending
90	results pending	results pending
91	0%+	54
93	0%+	98
94	0%+	76
95	2.4%	248
96	3.5%	57
191	7.1%	771
951	3.0%	99

**Additional testing results pending*
+Infected animals found in unit, but through hunter surveys

have pushed for eradication, not control of the disease when or if it is found. The bottom line is CWD, as with Tb, is a controllable disease in fenced situations. Herds can be monitored, animals tracked and actions taken to eliminate the disease. Already, there are positive signs of a significant turnaround. What happens in the wild is quite another issue.

State game agencies depend heavily on sales of licenses and fees to support their operations. Anything that threatens income raises a "red flag" for DNRs. Many state biologists within the Rocky Mountain CWD core area will not support total eradication of deer and elk within the infected regions. That would bankrupt their agency.

License sales have declined in many of the western states, especially Colorado. I receive dozens of calls each month from frightened hunters saying they will not return to the west next season. So, there is not a whole lot of support for eradication, only in privately-owned herds.

How big is the problem?

It would be irresponsible to understate the seriousness of the CWD situation. But, as Director Durham suggested, we must take a reasoned and prudent approach to the situation. We need a national, as well as state plan to eradicate the disease—and that's going to be a formidable task. Some say wild animals always have had these and other diseases and we are tampering with nature to control them. One of the best (and few) texts on the subject of wildlife diseases was written by Dr. Gary Wobeser of the University of Saskatchewan. He notes:

"The reasons for undertaking any type of disease manipulation in wild animals are essentially anthropomorphic, i.e., management is usually done to benefit humans in some way. Since this is the case, a hands-off approach may be entirely appropriate where the consequences of disease are perceived to be of little or no consequence…This approach would be even more appropriate if the animals were unaffected by human activities and lived in a pristine environment where disease was truly a natural event."

[CONTINUED ON PAGE 80]



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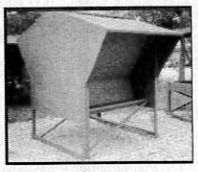
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Chronic Wasting Disease: What You Should Know

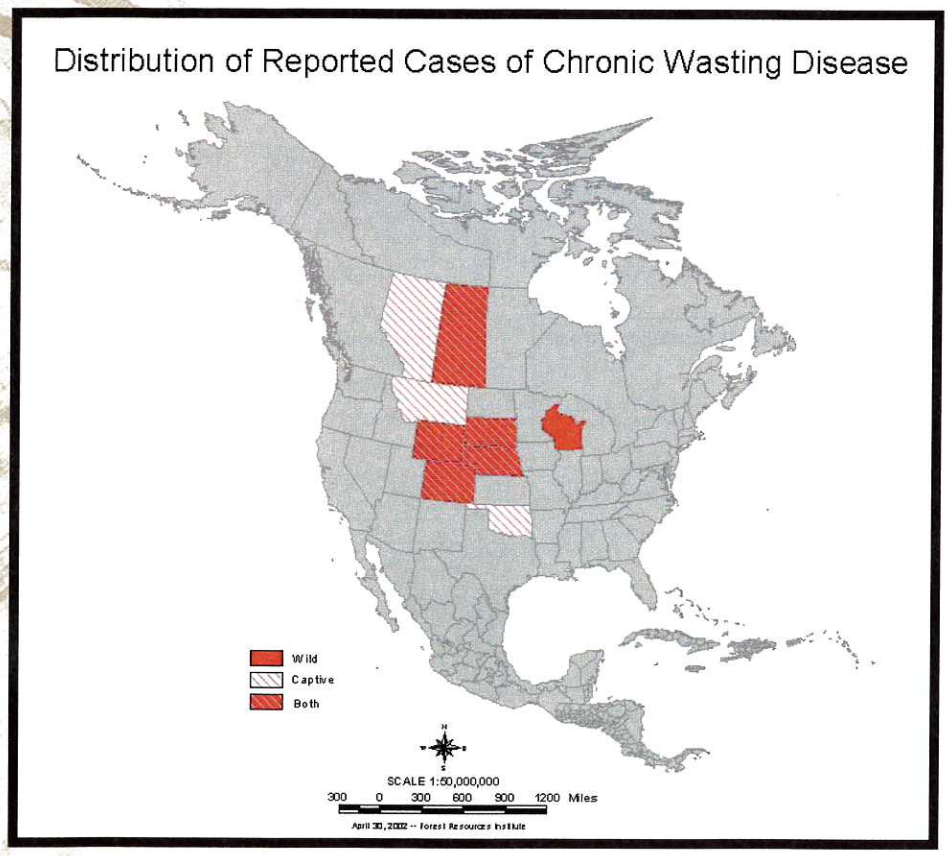
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Aldo Leopold, the father of wildlife management in this country, left little doubt about his opinion on this subject in his landmark book, "Game Management." "In its more advanced stages, game management is in effect the art of maintaining a population which is vigorous and healthy in spite of its density."

Managing game for man's benefit, as well as the various associated species, is the cornerstone of modern game management. Since we don't know if man or nature created this situation, I for one think it prudent to follow Leopold's guidance on this issue.

Can CWD, as may be the case now with BSE, spread to other non-deer species? The "official" position of everyone involved—agencies, organizations, private owners and scientists—is that the disease cannot jump species. Again, I think it irresponsible and unethical to suggest otherwise at this time. There is no credible scientific evidence that eating venison from an infected animal will give you CWD or a related TSE disease. But, again, we must be vigilant and prudent.

We closed the borders to movement of live deer for the immediate future. But, what about carcasses of animals harvested from infected regions. A fellow can shoot a mule deer in Colorado that is emaciated in appearance but has a fine rack and take it home to Texas. Later, the taxidermist can cut the skull cap off and, because no processor will take the remains for rendering, throw the remains into a trash pit. The hunter decides the venison is not all that good and it also ends up in the trash. Could animals scavenge on the remains? Deer are notorious for eating bones and antlers for the calcium and phosphorus. Again, there is no evidence this could lead to transmission, and the probability is low, but we should at least consider the possibility.



The above map shows the spread of reported cases of CWD across North America.

Media and other agendas

As I noted earlier, BSE caused a veritable panic in Europe. Fear of getting the disease from eating red meat literally bankrupt an entire agricultural industry. Yet, today the problem is much less than originally thought. It appears to be on the decline and consumer confidence is increasing.

Scrapie, in spite of being around for more than two centuries, has not stopped folks from enjoying lamb. But, the power of the media to influence public opinion continues to be frightening.

Outdoor writers have jumped on the CWD issue like the proverbial "duck on a junebug!" One of the most prominent outdoor magazines published at least two pieces stating without a doubt three hunters had contracted and died from CWD. How irresponsible!

As I noted in Part I, a ruthless gang of facts has murdered many a beautiful theory. There are no cases documented in which humans either have contracted or died from CWD.

There is a great debate raging among wildlife professionals concerning private management of game animals, especially deer. It is a philosophical debate that will, in my opinion, play out shortly. But, there are some who have used disease issues to bolster their positions against intensive deer management.

It is interesting to note most of the monitoring efforts for CWD have occurred only around privately fenced properties. The standard protocol for investigating wildlife diseases, however, requires a systematic search for the disease over the entire landscape in order to assess distribution.

The Facts

What I hope you have learned from this two-part series is that wildlife, like any other group of animals, has diseases. The source(s) for these diseases are many and complex. They may come, as with Tb, from domestic livestock or, as with anthrax, they have always been out there waiting for an opportunity. As may be the case with CWD, they even can develop

from, or be encouraged by, a weird mix of environmental factors acting on natural body processes.

There is also another important point. There is no evidence fencing deer causes diseases. If a disease shows up it's because it was there in the first place or contracted through contact with other species. It does, however, provide higher probabilities of detecting the presence of the disease. That also leads to easier control or eradication.

Tuberculosis came about through livestock co-mingling with wild deer. Chronic wasting disease was either present in the wild or expressed itself as above or was created like a modern day Frankenstein monster in a state-owned research facility. Whatever the cause, it does no good to fight among ourselves over who is to blame. Unfortunately, much of the motivation for this anti-management mentality is a genuine concern for the future of hunting—one that we all share.

Ironically, though, in their zeal to support their position with the latest "evidence," opponents to intensive management may have inadvertently "thrown the baby out with the bath water!" The hunting public, as with the English consumer, seemed to have panicked to the point long-term economic impact may destroy what we all love: deer hunting.

I learned recently that several of the national movements to distribute venison to the hungry are now thinking about closing down or discontinuing their efforts until this "blows over," a tragic consequence of the panic unnecessarily generated. In my opinion, we all need to take a deep breath and pull together. CWD is still a relatively rare disease condition and should be treated as such.

The Future

CWD appears to be localized at this point, primarily in some western states. The Wisconsin outbreak has yet to be investigated thoroughly. It may turn out unique environmental conditions favor the disease. CWD is indeed a manageable disease, provided appropriate plans are put into place.

In 1923, hoof-and-mouth disease broke out in California deer. A plan was organized and the disease was eradicated. We can do the same with CWD. These plans should involve solid, scientific research into the causal agent and underlying factors behind TSE-type diseases. Fenced deer will be the easiest to free from CWD, as was the case with Tb. Wild deer will be much more difficult.

In spite of the furor, CWD remains a rare disease, which could have been present all along. We do know that, at least in elk, there are genetically resistant animals. There are herds that have been exposed to the disease without contracting it. So, I think there is a bright future for dealing with this disease.

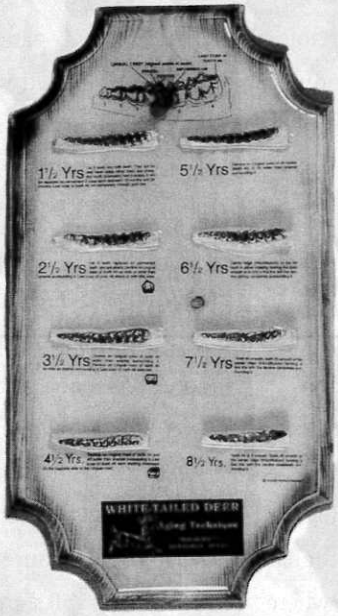
We have come to this point, however, not because people intensively managed deer. Rather, by allowing wild deer herds to reach un-natural population levels and un-natural age and sex structure, we have set the stage for disease. When you combine these facts with ever-increasing interactions between man, his livestock and wild animals, it seems logical to me there will be disease challenges.

Tb and CWD certainly are not the last diseases we will encounter with game animals. There are currently over 100 reportable diseases in livestock—and only a handful in wildlife. Why would we not expect this to change? The paradox is that humans, in particular wildlife professionals, tend to think of wild animals as "perfect." They never have an accident; they never contract diseases. Yet, my prediction many years ago about the eventual appearance of new and old diseases among wildlife populations are coming true.

What's next? Well, Johne's disease or paratuberculosis is hiding out there somewhere. So is pseudorapies in wild hogs and brucellosis both in cattle and deer. Then there are many more that have not even been described yet. But, we will deal with each as they appear. The animals will do their part, and I firmly believe so should we.



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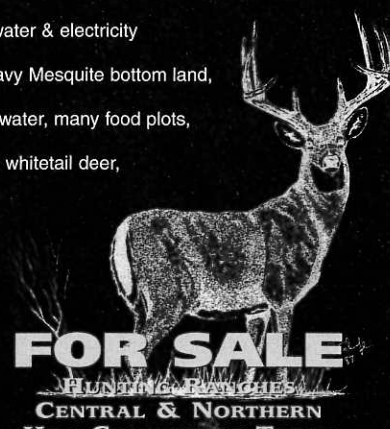
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