

# Dr. Deer's PRESCRIPTION



## Breakthroughs For Chronic Wasting Disease?

By Dr. James C. Kroll

I am a scientist, and I have been one for four and a half decades. Even before I officially became a scientist, it was all I ever wanted to be. Growing up in Waco, Texas, on Baylor Avenue, I would ride my bike to the biology building on the Baylor campus to hang around the professors and graduate students. By the time I entered Baylor, I was already involved in research with professors, Julian F. Watkins III and Fred Gehlbach.

These were exciting times, coming on the heels of the newly created National Science Foundation. President Kennedy vowed to put us on the moon in a decade and we succeeded. Whereas scientists had been considered for decades as “egg heads,” I became part of a new generation of scientists—more akin to fighter pilots than stodgy, pipe-smoking old men in lab coats. The National Science Foundation, and other related agencies, quickly became the source for easy money to support almost any project. I even obtained a grant as a graduate student to work on snakes.

The scientists who trained me were among the top naturalists in the world. Men like Drs. Harley W. Reno, James R. Dixon, Donald Clark, Jr., W. B. Davis, and others at Baylor and Texas A&M who were my mentors—men who viewed their role as standing between ignorance and truth via the scientific method. I learned early on a true scientist *never* tried to prove anything; the goal was to prove yourself wrong. In order to have a paper published in a scientific journal, you had to undergo intense scrutiny by a select group of your peers. The rejection rate for most manuscripts was very high in those days. To be a professor was akin to taking a vow of poverty. A good scientist could not be bought, and public opinion in the early

1970s ranked professors right up there with parents and clergy as people you could trust.

It has been said too often the two primary corruptors of humans are power and greed. “Big League” science created a whole new generation of scientists. Media and entertainment shared in responsibility, as TV created science celebrities, captured public attention. Professors were no longer evaluated by the quality of their students; rather by how much money generated and how many publications they had each year.

Complicating the picture was a growing environmental movement, and unfortunately, I was deeply involved in its origins. Many of us young scientists organized the first Earth Day, leading ultimately to the Environmental Protection Agency, the Clean Water Act, and the Endangered Species Act. No one can argue these programs were not effective in their early days; yet, it can be argued the bureaucracies they fostered have led the original intent far off the goal. At the same time, social movements and civil disobedience were growing as fast as the hair and beards of the “hippies” that fired the movement. It was not long before the striking barrier between cold, hard science and political activism began to blur.

I remained a naïve purest. A critical turning point in my career came at a meeting of deer biologists organized to promote exchange of new findings in the southeastern U.S.; a region where deer management was gaining public support. For over a decade, I looked forward to the annual meeting of the Southeast Deer Study Group to find out about the latest discoveries by my colleagues, to share mine, and to argue and discuss scientific theories openly and honestly. The second decade of its existence, however, saw a striking change in the

presentations and the quality of “science” being reported. More and more philosophy crept into presentations, and less and less hypothesis testing.

The last straw for me came when a respected colleague stood at the podium, waved a worn copy of *A Sand County Almanac* (written in the '40s by the “Father” of wildlife management, Aldo Leopold), and said: “This is my bible and I am speaking to our father in heaven, Aldo!” I seldom returned, and later discovered there were other scientists who left that meeting with the same concerns.

*What does all this have to do with new discoveries about Chronic Wasting Disease (CWD), a neurological disease of certain deer species? The story behind the disease, its discovery, and subsequent impacts on deer management and hunting, has a great deal to do with my personal history.*

There has been so much written about CWD in the last 15 years, there is no reason to clutter this article with a rehash of its history. Suffice it to say CWD first was noticed among deer and elk being held in captivity in a government research facility by the Colorado Parks & Wildlife Department, near Ft. Collins. I first became aware of the disease when I attended a talk by Dr. Elizabeth S. Williams (Wyoming State Veterinary Laboratory) at a meeting of the North American Deer Farmers. There were only a handful of attendees, as in those days the organization was concerned more with red deer and fallow deer than whitetails or mule deer. I was impressed with the objectivity of Dr. Williams and the quality of her science. I went away thinking that CWD was an interesting malady of deer and elk; one worth watching. Until her untimely death, Dr. Williams remained THE expert on CWD.

CWD remained mostly unknown to deer managers and hunters until February 28, 2002, when some white-tailed deer tested positive for the disease around Mount Horeb, Wisconsin. The discovery was significant, since it had been thought to be

found only in the Rocky Mountains and surrounding western states. Since its “discovery” by Dr. Williams, a growing number of scientists had been studying the disease, supported by increased funding stimulated by media hype for the so-called “mad cow” disease; the bovine cousin to CWD.

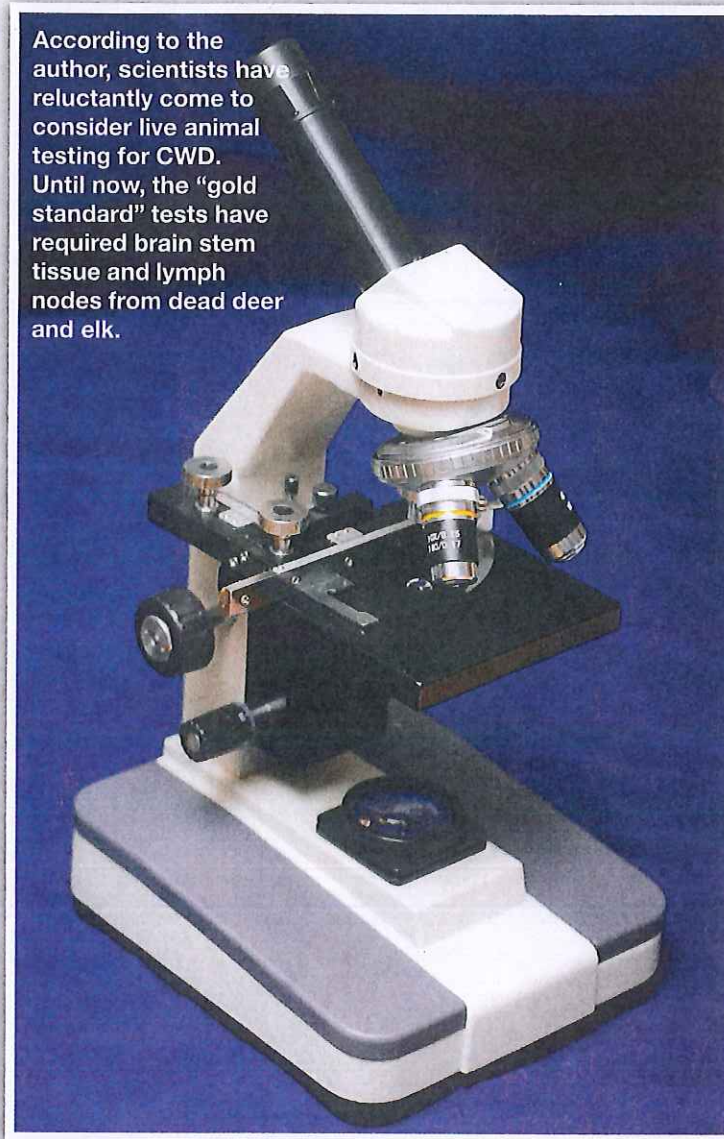
At least three outdoor publications immediately jumped on the discovery of CWD at Mount Horeb, falsely reporting that three individuals had contracted and died from the disease by eating venison. Fortunately, *The Journal* was NOT one of these

outlets. As if it were not bad enough for the media to make such reports, scientists and several state game agencies immediately jumped on the band wagon, warning that white-tailed deer and hunting soon would be extinct. State and federal money began to flow freely, as scientists anxious for support and fame showed up like blowflies at a deer carcass.

The decade following the Mount Horeb discovery saw over \$40 million spent on research and “control” in Wisconsin alone. Dozens of scientists padded their résumés and received awards and raises for their discoveries about CWD. Unfortunately, however, efforts to eradicate this disease in Wisconsin failed; and the Wisconsin legislature recommended other strategies. Wisconsin Gov. Scott Walker appointed me “Deer Trustee” in 2011 to deal with growing public distrust of the Wisconsin DNR and its programs.

The Deer Trustee Review Committee soon discovered questionable science behind eradication efforts and reports. After spending millions of the public’s dollars, we had learned a great deal about CWD in general, but almost nothing about its origins or how to manage the disease—in my mind what should have been the goal of such efforts. Early on, a handful of scientists had gained control over research priorities and funding. Agenda-driven science prevented the kind of scientific scrutiny needed for such efforts.

The scientific method became a casualty in recent years as socially minded scientists push agendas ranging from global



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warming to genetically engineered crops to, yes, CWD. No group should be more culpable than the wildlife management community in all this. Over 80 percent of wildlife biologists work for government agencies or universities. Over time, the type of individual entering the profession has become more urban than the farm and ranch kids to which I belong.

Game management has become a "step child" in most wildlife programs; in favor of endangered and non-game species. A minority of wildlife scientists hunt, and a frightening number are anti-hunting. Fact is many professional wildlife professionals do not like hunters. When you add in the recent trend toward private lands wildlife management by the "unwashed" public, these individuals feel threatened and are philosophically opposed to intensive wildlife management. CWD became a "hammer" to use against successful and ever-more popular wildlife management techniques such as supplemental feeding, genetic im-

provement and, God forbid, high fences. It did not matter there is no good science to support any of these concerns.

Understanding this explains the battles currently being waged in the popular media about intensive white-tailed deer management. CWD and the fear of losing deer became the perfect weapon, and research priorities and dollars were monopolized by poorly planned projects aimed at "proving" feeding deer spreads disease and intensive deer management destroys

ecosystems. In short, there is a situation in which there is a premise in search of a conclusion.

Official science quickly closed the door on the most critical issues of dealing with CWD; namely what causes the disease (or condition) and how can it be managed

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The first casualty in all this was any scientist daring enough to question the disease itself, *in spite of the fact it did not fit scientific facts about what constitutes a disease.* There is no doubt proteins called prions (pronounced "precons") that in themselves are



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MARTY BERRY PHOTO

important to the nervous systems of all mammals, are involved. Prions normally protect against neurodegeneration and neuronal cell death; and become a problem only when they change shape like “transformers” caused by a host of factors, including mineral deficiencies.

I was raised academically to synonymize scientist with skeptic; it’s what made science so pure in the old days. Good scientists do not believe what they see and hear, no matter how unpopular the truth is. Yet, in modern times “official” science no longer can be questioned; and God save anyone who dares to do so. TTHA was the first to raise questions about the doom and gloom of CWD through its TV program. In 2003 I stuck my neck out on the show, when I took a very unpopular stance in predicting the Wisconsin efforts would fail. By 2013 it was clear my predictions had come true. It was not that I was clairvoyant; rather, I based my prediction on plain old scientific facts about deer and common sense. As with its human equivalent, Cruetzfeldt-Jakob disease, CWD takes time to manifest itself in an infected individual. Individuals do not become infectious until clinical signs appear—most often years after exposure. In the case of whitetails, by the time an individual becomes clinical, it not only has reproduced several times, but probably has died from something else.

The next problem in all of this is that there has been little, if any effort to develop ways to manage the disease, or even to identify susceptibility. From the outset, it was dogma that all deer could get the disease and that once a deer had the disease, it would die. Again, a little basic biological knowledge would lead you to question this hypothesis. Natural selection works best when the species’ population is quite large; increasing the probability resistant individuals exist. With more than 25 million deer on the North American landscape, surely I thought there had to be resistant genes out there.

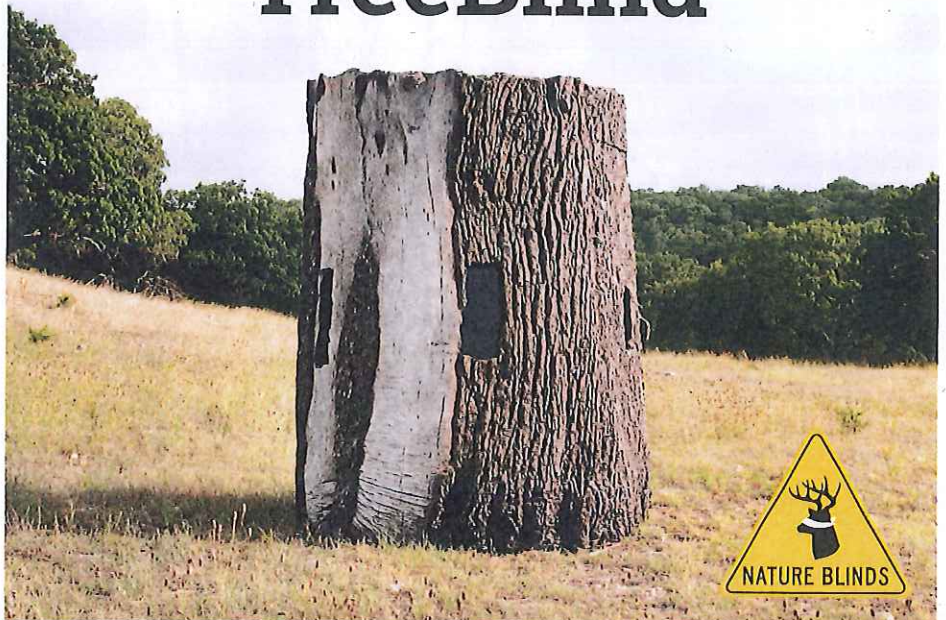
My thinking about solutions to CWD focused on the following. First, continue to conduct surveillance on the geographic distribution of the disease to define the extent of potential damage. Second, look for existence of resistant individuals and identify markers for which genes are responsible for resistance. Third, examine potential for immunization, if CWD is a true disease. Finally, develop ways to manage the disease, rather than eradicating it by integrating the above. In all of history, I am familiar with only one wildlife disease (hoof-and-mouth) that was eradicated, and that was for a geographically restricted species (blacktail deer). Unfortunately, the philosophical-political

climate dealing with CWD does not support this approach. The premise has been that there has to be a man-caused origin for the disease, and the conclusion is that intensive deer management and breeding is the cause. Any research not aimed at “proving” this cannot be tolerated by official science. Most important, the idea of developing an effective CWD management program would enable, not curtail deer management, reduce funding and loss of power for select scientists.

### Getting to the point

Given this long discourse on the political and academic climate surrounding CWD, let’s look at what has been done to reach a point where we might be able to manage it. First, we still are in the dark as to the causal factors for the appearance of distorted prions. One alternative hypothesis published by

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Purdey (2000) suggests a nutritional deficiency involving copper and manganese may be one factor, supporting the "spontaneous" appearance in some areas such as New Mexico. Even grass mites and spirochaetes have been blamed for the disease. Second, my idea about genetic resistance is supported by work done on the domestic sheep version of CWD, *Scrapie*, in which identification of a susceptible gene allowed breeders to significantly reduce susceptibility.

It was not until later in the Wisconsin researchers and others began to look at possible genetic markers for susceptibility to CWD. A resistant gene (genotype 96SS) has been identified, with non-resistance for the 96GG genotype, and intermediate

*partially protects white-tailed deer from chronic wasting disease,"* presented results of an oral recombinant *Salmonella* (bacteria) vaccine trial that appears promising. The authors reported that control deer exposed orally to CWD developed clinical signs of the disease in a median time of 602 days; while the experimental group went 909 days, with one still disease-free. That means an immunized deer might become clinical almost a year later. I offer a caveat, however, since only 11 deer were involved in the study, five treated and six control, we need more studies. I might wonder what would be the results if genetic resistance were included?

The second paper, also accepted for publication in 2014,

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resistance for the 96GS genotype. This is encouraging because it will allow identification of more resistant individuals for selective breeding for resistance.

Reluctantly scientists have come recently to consider live animal testing for CWD. Until now, the "gold standard" tests such as immunohistochemistry (IHC) have required brain stem tissue and lymph nodes from dead deer and elk. The search for a live animal test, which would be important for deer breeders, has been slow for obvious reasons! At this time, combining both genetic and live animal testing is very encouraging, but regulatory agencies are slow to accept advances. The reason relates both to attitudes toward and lack of understanding of deer management. Agency biologists and researchers have a regulatory mentality, rather than the facilitation mentality held by their agricultural commodity counterparts.

If, in spite of the slow pace for development of management tools for CWD, we do develop ways to use animal husbandry to manage the disease, what good will this do for free-ranging deer? Yes, reintroduction of genetically resistant deer could be part of the solution; yet a faster approach would include immunization of free-ranging deer against the disease. To do so, there are two hurdles: 1) development of an immunizing agent, and 2) an effective inoculation vehicle other than injection. You cannot expect deer to line up for shots.

Two promising papers were published late in 2014 (available in pre-publication form at this time), both dealing with immunization methods. The first (Goni, et al. in press) was by a long list of 17 authors (part of that resume building I discussed earlier), representing a variety of interests. This paper, entitled "*Mucosal immunization with an attenuated Salmonella vaccine*

was authored by eight Canadian scientists (Marciniuk, et al. in press) and presents a similar approach, but did not deal directly with CWD. The scientists examined efficacy of prion protein antibodies in mice and sheep. Even though it does not deal with deer as subjects, it has direct application to CWD. The bottom line to both of these studies is that solutions to CWD are within reach, in spite of the dire predictions of agenda-driven scientists.

CWD first came on the scene in 1967, but was not formally considered a unique disease (condition) until 1979. In 2003, CWD suddenly emerged among free-ranging deer in southwestern Wisconsin, sparking an ill-fated attempt to eradicate the disease. Over the subsequent decade, thousands of deer were slaughtered in Wisconsin to eliminate a disease that could not be eliminated. It now is commonly acknowledged that CWD has been around for at least 100 years, and we still have deer. Had deer and elk been accepted agricultural crops or commodities, the approach would have been much different. As we have learned with other diseases such as bovine tuberculosis, you can only manage a disease once it is established across the broad landscape.

In regard to an important disease issue for our most economically and historically important game species, deer and elk, political-philosophical agendas caused professional wildlife scientists to lose sight of the scientific method and its approach to problem solving. In my mind, efforts to eradicate CWD have diverted our attention away from more pressing issues related to whitetail population declines in recent years, which are much more important and relative to CWD. 