Dr Deer's™ PRESCRIPTION

Chronic Wasting Disease: The Issues At Hand

A White Paper

By Dr. James C. Kroll

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (Williams 2005, Williams and Young 1980), first recognized among deer and elk residing in research facilities located near Fort Collins, Kremmling, and Meeker, Colorado, and Wheatland, Wyoming (plus two zoological collections). CWD is one of the many Transmissible Spongiform Encephalopathies (TSEs) affecting mammals, including cattle, sheep, goats, and mink. TSEs represent a family of rare, progressive neurodegenerative disorders affecting animals and humans. These diseases are characterized by long incubation periods, and caused by abnormal pathogenic agents called "prions," which exhibit abnormal folding of cellular proteins commonly occurring in the brain. Apparently, as the disease progresses, these proteins accumulate in the brain, causing destruction and deterioration of critical brain functions.

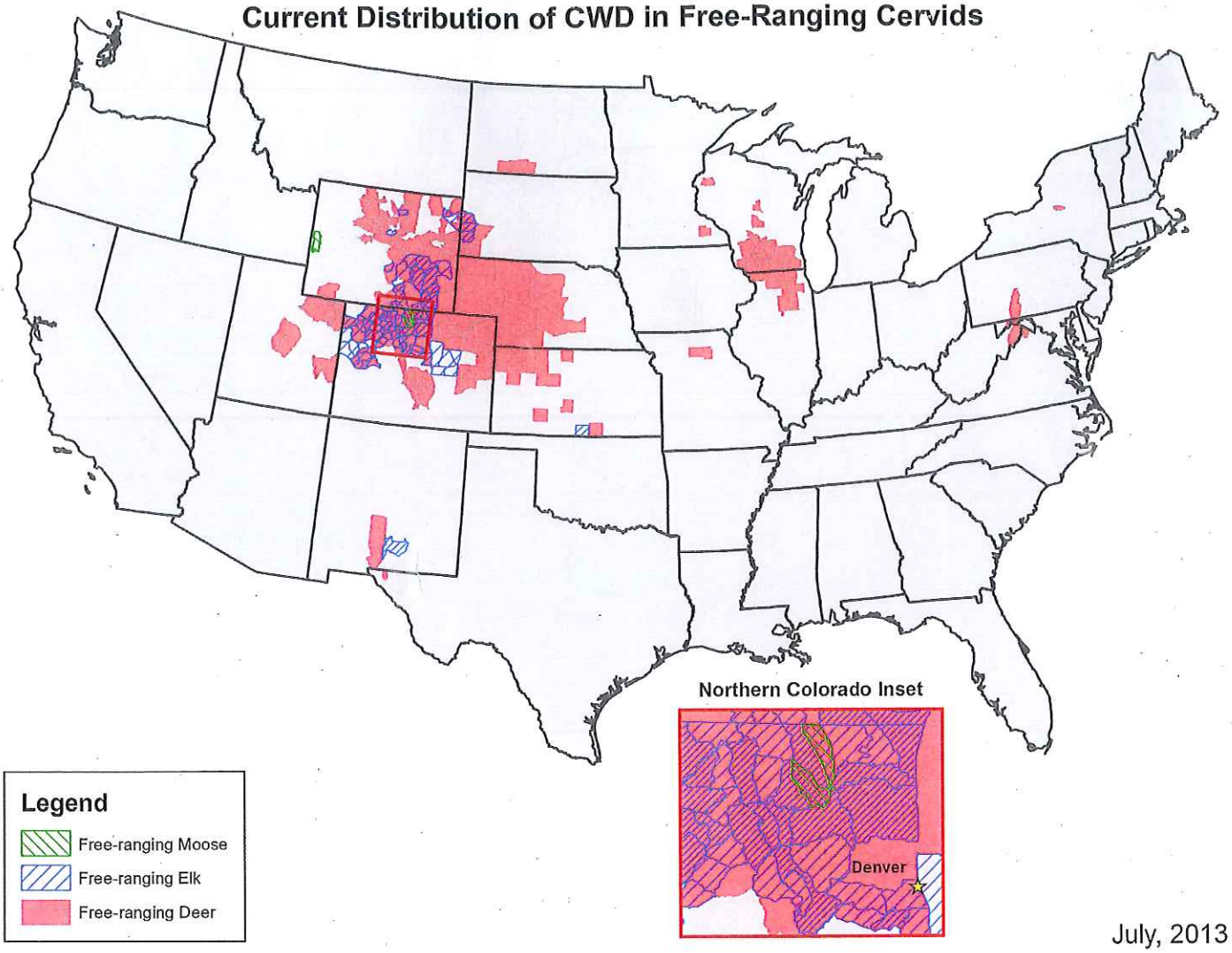
The occurrence rates for these diseases are relatively low, but may be influenced by the biology and behavior of the affected species. For example, published occurrence rates for Creutzfeld-Jakob Disease (CJD) in humans is less than 2 per million individuals.

Since its discovery, there have been numerous publications and research projects on this disease. It can be said we know a great deal about the disease, but know little about some of the most critical information needed to manage it. Some examples of knowledge gaps include: how the disease actually originated; the causal factors behind the appearance of the disease, both historically and currently; how the disease can be managed and at what levels and goals; and, most importantly, what is and will be the true impact of the disease on white-tailed deer populations? In this section, I will limit my comments to those I feel directly related to the issues of this paper.

CWD is a reportable disease, just as with bovine tuberculosis and anthrax. A "reportable" disease in my judgment is one that has the potential to, 1) significantly increase mortality of the species affected, 2) have the potential to be zoonotic, particularly to humans and livestock, and 3) have a demonstrable economic impact on livestock agriculture or, in this case, recreational hunting.

Impact of CWD on White-tailed Deer Populations?

Almost from the very beginning, the appearance of CWD triggered a landslide of dire predictions about its impact on whitetail, mule deer and elk populations. A University of Wisconsin-Madison (UW-M) professor quickly developed a



USDA-APHIS Maps

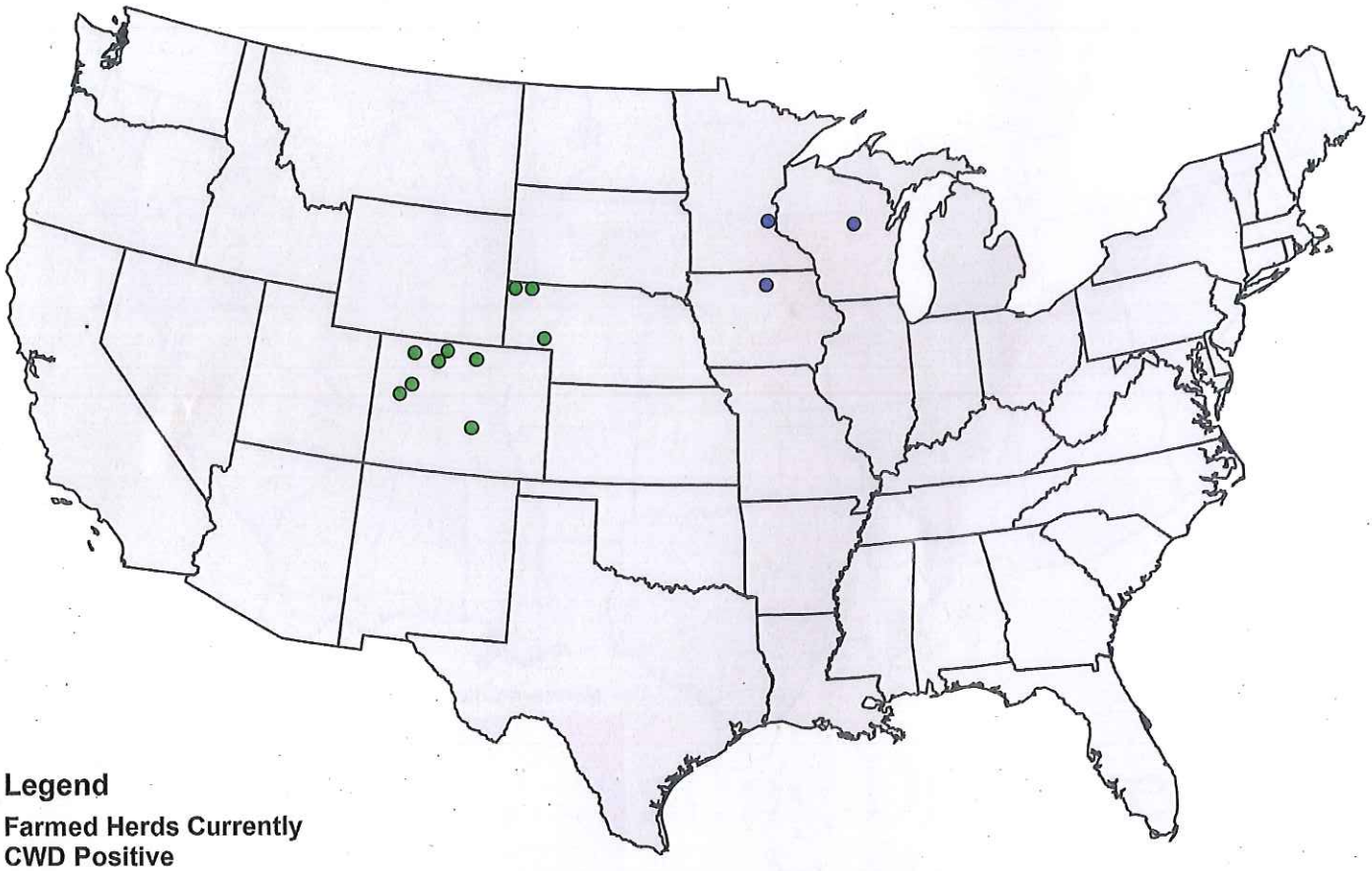
computer model, virtually predicting the time of day white-tailed deer would go extinct in Wisconsin! Yet, to this day no one knows how long CWD has existed within white-tailed deer populations, or if the disease originated near the Colorado or Wyoming research facilities. Epidemic modeling by Miller et al., 2000 suggested CWD might have been present among free-ranging animals in some portions of the disease-endemic area decades before it was initially recognized. In addition, the CWD "outbreak" in Wisconsin may have gone unnoticed for years prior to discovery. Hence, I feel there has been enough time to detect negative population dynamics impacts from the disease. So, let's see.

In 2010, Colorado State University researchers estimated CWD effects on mule deer recruitment and population growth during 2006-2008. Infected does weaned 0.95 fawns (0.56-1.43), and uninfected does weaned 1.34 fawns (1.09-1.61); suggesting higher recruitment from healthy does. However, they concluded: **"We conclude that although CWD may affect mule deer recruitment, these effects seem to be sufficiently small that they can be omitted in estimating the influences of CWD on population growth rate."**

Earlier, researchers at UW-M in 2006 examined demographic patterns and harvest vulnerability of CWD infected deer in Wisconsin; an area now infamous for its high concentration of CWD positive deer. They concluded: ***"We found no difference in harvest rates between CWD infected and non-infected deer. Our results show that the probability of infection increased with age and that adult males were more likely to be infected than adult females."*** A radio-telemetry study involving 160 deer over a 6-year period within the highest prevalence CWD management units found that, excluding hunting mortality, male survival ranged 0.83 (yearlings)-0.89 (mature), while females were 0.88 for yearlings and 0.83 for adults. They concluded, **"We find no evidence that CWD was substantially increasing mortality rates during the duration of our study from 2003-2007, though the disease is relatively new to this area."** Other Wisconsin scientists reported in 2003 on the fate of 179 deer (110 does and 69 bucks), with 4 testing CWD-positive (3 females, one male, 2.2%). Only 8% of mortalities involved CWD in some manner: one adult doe died of CWD; one yearling was mistaken to have CWD and euthanized (negative test); and 4 tested positive and later euthanized.

These studies tend to support my

Current Distribution of CWD among Farmed Cervid Herds



Legend

Farmed Herds Currently CWD Positive

- Captive Elk
- Captive Deer
- Captive Elk and Deer

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opinion there is no evidence CWD has an impact on the population dynamics of whitetails or mule deer. I must point out, even allowing for a shorter incubation period, by the time a doe becomes clinical for CWD, or even begins to be affected by it, she probably has reproduced at least twice. Researchers in 1971 reported that 77% of Ohio fawn does ovulated; and, precocial fawn breeders averaged 1.29 fetuses. Even allowing for a doe being infected early in its life, it is not logical to conclude impacts on reproductive performance.

Lastly, the average age of deer in most herds is well beneath the incubation time for the disease; and most studies note the greatest mortality factor for white-tailed deer is hunting, followed by accidents and predation. CWD infection rates for U. S. testing to date (USDA/APHIS) total 164,500 captive deer and elk and 775,000 free-ranging deer and elk, with only 171 captive (1/1000) and 3,130 (4/1,000) free-ranging animals testing positive.

As part of the review process as Deer Trustee of Wisconsin, the review panel examined harvest and population estimate data from CWD eradication zone in southern Wisconsin. During a 10-year period, 172,000 whitetails were removed from the eradication zone, 1,800 (1%) tested positive for the disease. In spite of the heavy harvest and presence of the disease, the deer herd actually grew during eradication efforts, significantly exceeding population goals for the zone.

Testimony by Dr. Michael Miller of the Colorado Division of Wildlife Research Center during an Iowa lawsuit confirmed there is no credible scientific evidence CWD has had any negative impact on deer populations. ***"Overall, at a large scale, so at a population level it's been difficult to demonstrate any effect over the period of time. In part because there are a number of other things that influence particularly deer population dynamics a lot more than a disease like this would in the short-term: weather events, our own hunting practices, predation."***

In a white paper, Dr. Don Davis, Wildlife Disease Scientist at Texas A&M University College of Veterinary Medicine, noted that although CWD has been found in 20 (40%) of the 50 states (USDA/APHIS records), "This is somewhat misleading because there are about 3,500 counties in the US and CWD has been found in about 175 of them or in 5%. Five percent is a closer figure to the actual occurrence and distribution of CWD." I concur with Dr. Davis' finding.

Impact of CWD on livestock and agriculture?

The zoonotic (transmission from animals to other animals) potential for CWD, especially for livestock species, is an important concern and consideration.

The early recognized CWD authority, Beth Williams concluded in 2005, **"Studies have not documented that livestock are susceptible to CWD." Miller et al. (2000) earlier had made the same conclusion. CWD has been experimentally transmitted to cattle and sheep through intracerebral injection; however, this is not a normal or even feasible means of naturally transmitting the disease."** The lengths scientists have gone to in trying to demonstrate an ability of CWD to infect livestock has been exceptional. After 6 years of trying, researchers could not establish the disease in cattle or sheep, even by injecting their brains with prions. It is now thought that there is a molecular barrier to the disease in cattle and sheep. Although it may be possible to artificially induce limited response, I must conclude there is no evidence to date of likely CWD transmission from deer to livestock.

CWD Health Hazards to Humans

There are published studies that monitored human populations for signs of CWD infection. Numerous studies have been conducted on the possible trans-species infection between deer and humans, using human models such as primates or transgenic mice (genetically engineered mice to mimic humans, a scary thought in itself). In 2007, scientists reported no convincing cases of CWD transmission to humans. Shortly after the report of CWD in deer, at least three outdoor magazines (but not TTHA) reported erroneously that three men had died from the disease after eating venison. The obvious damage to deer hunting as a recreational activity was staggering! Wisconsin lost a significant number of its hunters, and at this time has not gotten them back!

Yet, again science showed no such relationship. In 2001 three young patients who had contracted Creutzfeldt-Jakob's Disease or CJD (a human prion disease) and who had eaten venison, were shown not to have contracted either CWD or a variant of CWD. A 2006 examination of data from the Colorado Department of Public Health and Environmental Human Prion Disease Surveillance Program, concluded no convincing cases of CWD transmission to humans were detected. Similar results have been reported by other research groups. Studies involving other primates and transgenic mice) found higher primates and transgenic mice also apparently had a molecular barrier to infection. The normal occurrence rate of CJD in humans is about 1 case per 1,000,000 persons and this rate has not appeared to have increased.

It has been reported that humans in the United Kingdom also are resistant to some extent to variant CJD. As of June 2006, there were only 161 cases of vCJD in the UK, as compared to the millions of residents and the obvious high numbers eating beef. Over a million infected cattle may have entered the UK human food chain since BSE (aka mad cow disease) first appeared. Researchers have concluded that the inherent ability of these infectious prions to affect humans may be low. Therefore, it is my opinion CWD does not pose a threat to human health.

Origin of CWD

It still is unclear as to the original source or cause of CWD, and I could find no credible scientific work supporting any theory. We do know that abnormal prions are, 1) infectious agents, and 2) they are capable of reproducing themselves. CWD prions are mis-folded through some unknown mechanism. There has not been a great deal of research on alternative explanations for causes of CWD. One alternative hypothesis suggested a nutritional deficiency involving copper and manganese may be one factor, supporting the "spontaneous" appearance in some areas such as New Mexico. I am inclined to agree with this hypothesis. I also can say with full confidence deer breeding had nothing to do with the appearance or transmission of CWD. Again, there is no credible evidence to support any claim to the contrary. Deer breeders have been the victim rather than the source or perpetrator of the disease. According to USDA, from 775,000 free-ranging cervids tested for CWD from 2002-2011, 3,130 tested positive (0.4%); while 164,500 farmed cervids, with 0.1% testing positive.

Genetics and CWD

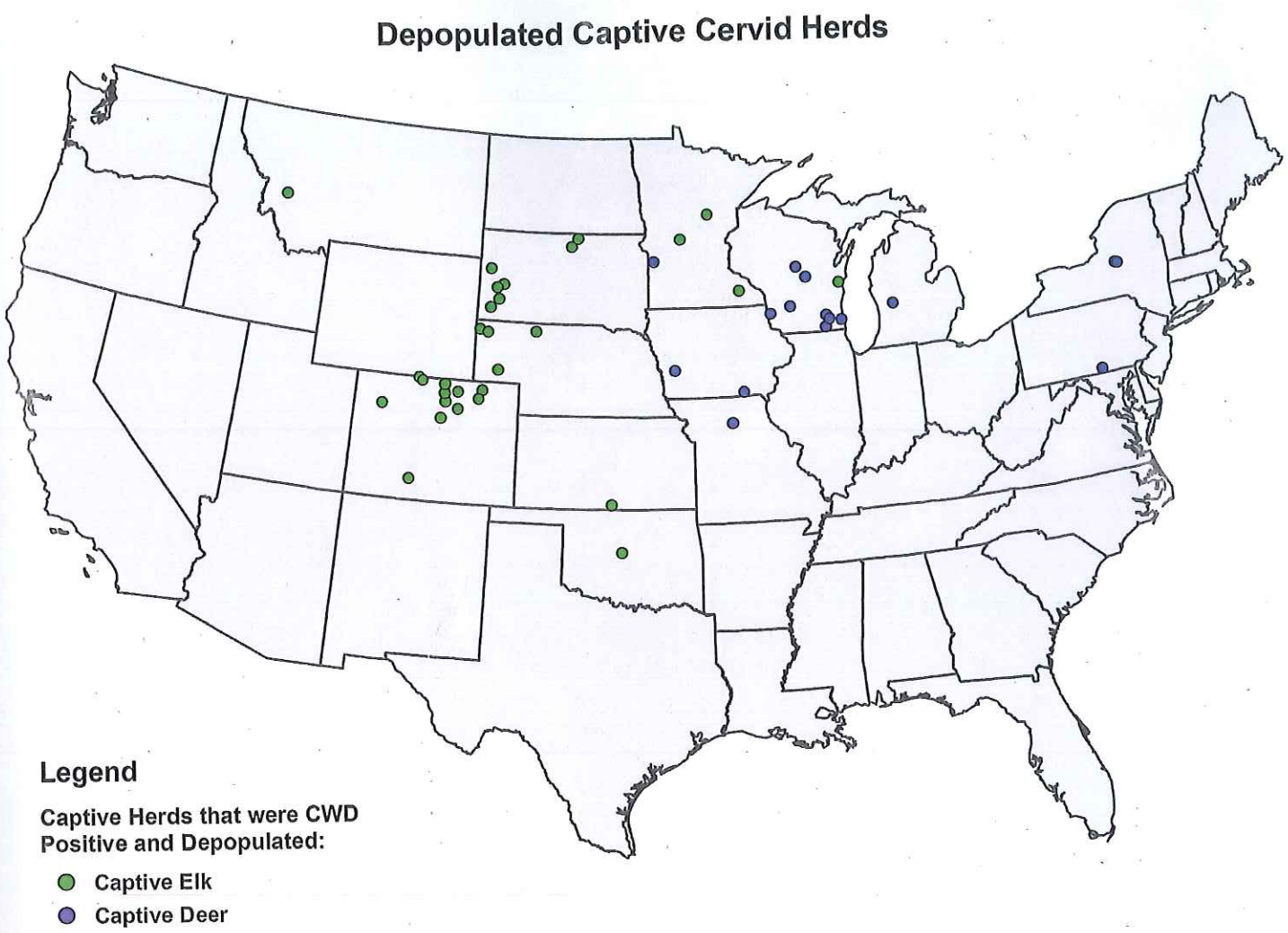
Suffolk sheep are particularly susceptible to scrapie (the sheep variety of the disease), with a reported 87% of cases attributed to this breed. Experiments involving injections of scrapie prions into sheep, followed for 10 years revealed genotypes susceptible to the disease and those resistant to it. This led scientists to suggest selection of breeding stock with the resistant gene would reduce the incidence of clinical scrapie in Suffolk sheep.

Following this line of thought, researchers at UW-M analyzed the PRNP gene in Cervids naturally infected with CWD, and reported that PRNP polymorphisms do affect susceptibility to infection. They reported:

"To test this effect, we orally inoculated 12 white-tailed deer with CWD agent. Three different PRNP alleles, wild-type (wt; glutamine at amino acid 95 and glycine at 96), Q95H (glutamine to histidine at amino acid position 95) and G96S (glycine to serine at position 96) were represented in the study cohort with 5 wt/wt, 3 wt/G96S, and 1 each wt/Q95H and Q95H/G96S. Two animals were lost to follow-up due to intercurrent disease. The inoculum was prepared from Wisconsin hunter-harvested homozygous wt/wt animals. All infected deer presented with clinical signs of CWD; the orally infected wt/wt had an average survival period of 693 days post inoculation (dpi) and G96S/wt deer had an average survival period of 956 dpi. The Q95H/wt and Q95H/G96S deer succumbed to CWD at 1,508 and 1,596 dpi respectively. These data show that polymorphisms in the PRNP gene affect CWD incubation period. Deer heterozygous for the PRNP alleles had extended incubation periods with the Q95H allele having the greatest effect."

My interpretation of these findings is that a resistant gene does indeed exist for CWD in deer. Additional research comparing CWD infectivity in deer with genetic variation concluded:

"Susceptibility to prion infections, including CWD, can be dependent on the amino acid sequence of the host prion protein (PrP). Here, CWD agent obtained from a deer expressing the 96SS genotype, associated with partial resistance to CWD, was used to infect transgenic (tg) mice expressing either 96GG or 96SS deer PrP. Transgenic mice expressing 96GG deer PrP succumbed to this agent, but tg mice expressing 96SS deer PrP did not. Additional studies using inocula from 96GG deer showed no transmission to 96SS PrP mice and delayed disease in 96GS mice. Thus, 96S PrP played an inhibitory role in disease progression in tg mice."



Let me explain this quote. There is a gene identified in deer (position 96 PrP) that when homozygous for 96GG (identical gene pairs) is more susceptible to CWD agent. When deer had the other homozygous gene (96SS) there was no transmission. When there was a heterozygous pairing (96GS), there was a delay in the disease. Hence, given the huge current population of deer in North America, there is potential for, at the least, deer developing resistance. Furthermore, it is possible to effect this through management.

To support my position, computer modeling by other Wisconsin scientists concluded:

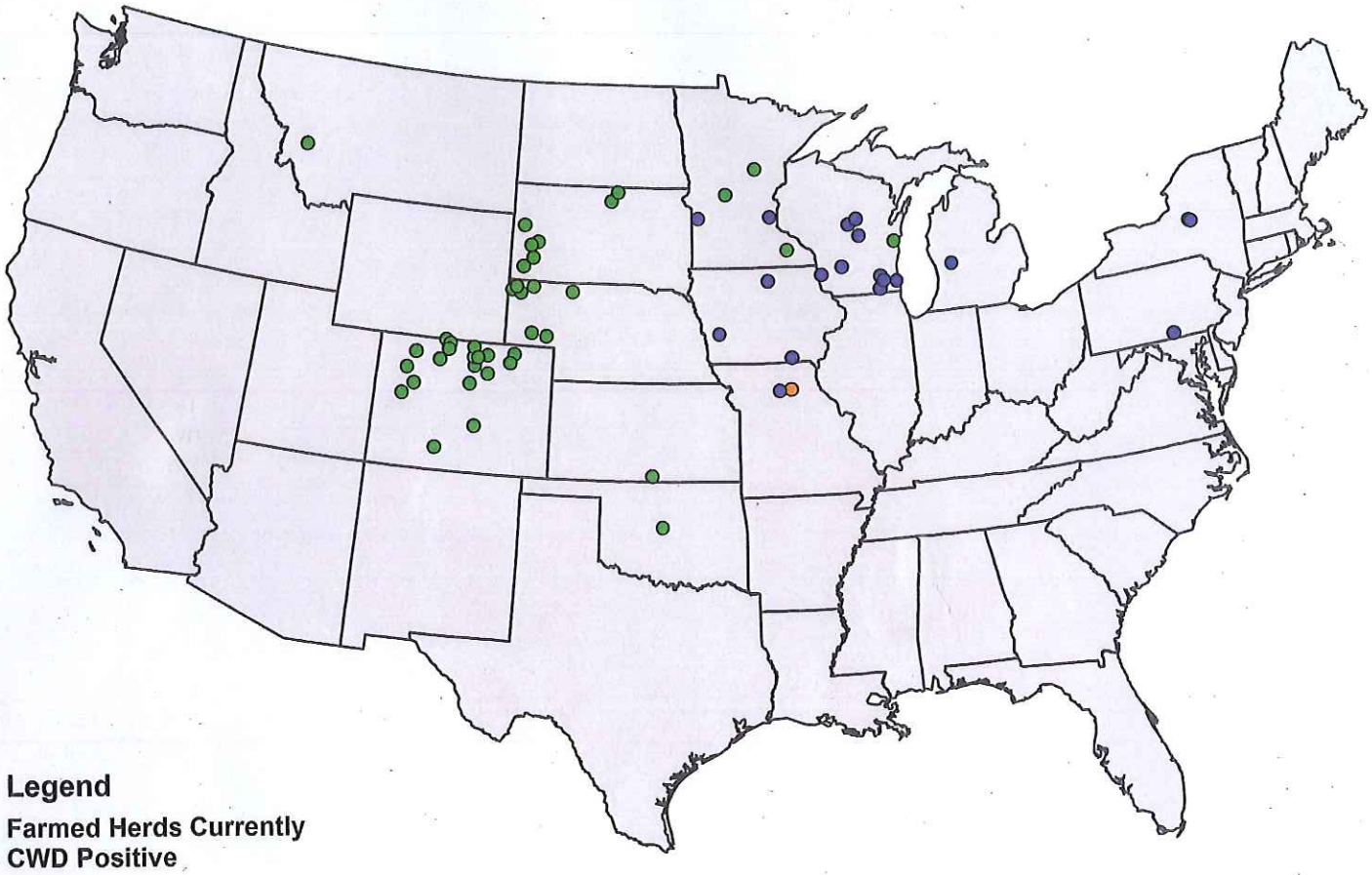
"The differences in disease infection and mortality rates allowed genetically resistant deer to achieve higher population growth and obtain a long-term fitness advantage, which translated into a selection coefficient of over 1% favoring the CWD-resistant genotype. The selective pressure suggests that the resistant allele could become dominant in the population within an evolutionarily short time frame. ... White-tailed deer with at least one copy of the serine-coding allele may resist infection or delay the clinical stages of CWD compared to deer homozygous for glycine (referred to here as '96GG'). Gly-Gly homozygotes constituted 59.4% of our study population. ... These genetic differences in disease dynamics have not been previously documented, accounted for in management planning, or considered in current CWD models."

Hence, as with sheep it also appears to be possible to identify resistant and susceptible deer through genetic analysis. When combined with a live animal test (see below), this could lead to a management solution to CWD. Unfortunately, there is little interest in developing management strategies to increase the occurrence of resistance in whitetails.

Testing for CWD, Post-Mortem and Ante-Mortem

The traditional method in testing for presence of CWD is a post-mortem (after death) test, using the extracted brain stem (obex) and retropharyngeal lymph nodes. These procedures are considered as the "Gold Standard" for testing, yet as with all clinical tests, they do not elicit 100% certainty. A typical CWD test result never reports the animal as not having the disease, only that "CWD was not detected." Sensitivity of these tests is reported as about 80% and specificity was 100. Specificity refers to the proportion of samples of uninfected animals that test negative with the test. Sensitivity is the calculated proportion of samples of infected animals that are tested positive by the assay. These numbers are only slightly

Cumulative Distribution of CWD among Farmed Cervid Herds



Legend

Farmed Herds Currently CWD Positive

- Captive Elk
- Captive Deer
- Captive Elk and Deer

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better than other livestock disease tests. In comparison, the cattle BSE "Rapid Tests" range 97.9-100% sensitivity and 99.3-100% specificity. This explains why a typical CWD test result report uses the words "not detected" instead of negative. No test is 100% effective.

Using a test that has to be conducted after death presents obvious challenges to the deer industry; and even more to wildlife management per se. Other diseases such as tuberculosis commonly are tested for using live animal procedures. Again, there has not been a great deal of interest in live animal testing for CWD. Recently, however, there has been progress in this area, with scientists comparing results from the Gold Standard test to those produced from biopsies of lymph tissue in the rectums of Rocky Mountain Elk. Postmortem comparisons of retropharyngeal (back of the throat) and rectal lymphoid tissue tests from 308 elk killed as part of depopulation of two private herds yielded encouraging results. Seven elk were positive from the brainstem, retropharyngeal lymph nodes and palatine tonsil; while 6 were positive using the postmortem rectal mucosal sections.

Live animal test for CWD?

There clearly is a need for an effective live-animal test for CWD. As with other reportable livestock diseases, testing without having to euthanize the animal is the preferred method. Unfortunately, however, only recently have researchers turned their attention toward this need. In 2007, researchers discovered CWD-associated prion protein deposits in the rectal lining from 19 inoculated mule deer. Detectable protein was evident within 381 days post inoculation; and, 45 of 50 naturally infected mule deer had PrPCWD in their rectal mucosa. Incidentally, the presence of Glycine (G) and serine (S) at condon 96 PrP (i.e., 96GG, 96 GS or 96SS) seemed to influence deposition patterns. These findings supported both a genetic link and the potential use of rectal tissue (lymph tissue) in a live animal test.

By 2009 progress had been made toward a live animal test. A significant relationship was reported between CWD infection status on codon 96 for deer in 2 out of 3 Canadian herds. The study included 600+ clinically normal whitetails from 4 genetically and geographically source herds. Odds ratios relative to 96GG deer were significant for both 96GS and 96SS deer, with 96GS deer 3 times less likely and 96 SS deer about 19 times less likely to be CWD positive than 96GG. Scientists concluded: "...rectal biopsy sample testing was an effective method in detecting subclinical CWD infection;" the most significant finding of this research. Yet, again did you hear anything about these results?

Subsequently, scientists at the USDA National Veterinary Services Laboratories at Ames, Iowa, compared results from retropharyngeal lymph node and palatine tonsil to rectal mucosa biopsy samples. As the experimental infection progressed, rectal biopsy sensitivity increased from 36% for early CWD stage deer to 100% for the last two stages of preclinical disease. Overall specificity was 99%.

My finding is that the rectal mucosa biopsy, when done in conjunction with genetic testing is a viable alternative to postmortem brain stem, retropharyngeal lymph node and palatine tonsil IHC testing, and could be used to reduce the spread of the disease.

Apparently there is agreement with my finding, as several states have been relocating Rocky Mountain Elk as part of restoration activities in conjunction with the Rocky Mountain Elk Foundation. Hundreds of elk have been moved and released in several states, including Missouri, Michigan, Wisconsin, Kentucky, Tennessee, Virginia (pending), and Pennsylvania. In most cases, when asked about health issues, state agencies claim that stocker animals come from disease free herds. Virginia reports sourcing their animals from Kentucky under conditions set by the state veterinarian, and mortalities necropsied and tested for tuberculosis, brucellosis and CWD for at least 5 years after translocation. Yet, it is my opinion finding a positive animal in these translocation projects is tantamount to the "horse out of the barn." The question arises: Should not the regulations for translocation be applied equally for public and private movement of animals?

The Missouri state website claimed in 2010: *"Since 2000, there has been significant progress made in our understanding of chronic wasting disease (CWD), including a live-animal test for elk. Our extensive animal health protocols include testing all elk for chronic wasting disease."* http://mdc.mo.gov/discover-nature/wildlife-restoration/elk-restoration/elk-restoration-background/elk-restoration-plan. It is interesting to note, I recently testified before the Missouri Legislature, appearing just after the State Veterinarian and head of their game agency. Their testimony was that a live animal test was not reliable enough to implement. I pointed out they needed to read their own web site.

It is my opinion, results from rectal mucosa biopsy tests, plus the acceptance by a state agency (Missouri) of this test prior to relocating Rocky Mountain elk, validates the use of these procedures for private deer/elk breeding operations; especially when combined with genetic testing.

What do we know about CWD transmission?

It generally is considered that CWD prions are difficult to remove from a site where infected deer have existed. Although this is considered as fact, methodologies for detecting CWD contamination has not been developed. Most of the positions taken by disease and wildlife professionals are based on computer modeling studies with "expert" assumptions.

Research has shown it is possible to transmit the disease through blood, saliva, urine-feces and environmental exposure; however, in most cases exposure must be continuous and take place for an extended time period, ranging up to several months.

I have arrived at the opinion CWD can be transmitted from indirect contact with various waste products, including blood, tissues, saliva, urine and feces. However, there are two important points to make at this juncture: 1) it appears from the literature, infection occurs after considerable frequent exposure; and, 2) there are no data or publications on probability versus possibility, and due to the tendency to rapidly depopulate any facility where CWD tests positive there is little information about the dynamics of the disease in a "natural" context.

Adequacy of Fencing to Prevent Contact

Over the last four decades, I have considerable experience in the fencing of white-tailed deer, either to contain them or to exclude them from specific properties. This experience includes both high, netwire fencing and electric fencing; studying the efficacy of the latter most recently. There also have been some scientific studies on efficacy of different fence designs, and I will present the state of knowledge below.

In 2012 the USDA (2012) published chronic wasting disease program standards, including fencing requirements. Appendix II of this document states that a 2.4 m (8 feet) tall netwire fence is adequate to reduce contact between farmed and free-ranging deer and elk, but cite a 2010 study that recommended a 10-foot high fence, based on 6 opinions from a survey of 150 wildlife biologists that deer could jump an 8 feet tall fence. My experience has been this is possible, but deer habituated to a fence rarely challenge unless forced to do so.

From 2007 to 2011, three papers were published on the effectiveness of deer-fencing in controlling movements. These studies attempted to measure behavioral response of confined and free-ranging deer to fences; primarily to determine the amount of contact between these two types of deer. The studies were conducted in Colorado and Michigan, and involved white-tailed deer, mule deer and elk. Fencing in this case refers to 8 ft. (2.4 m) high wire netting in various configurations. Electric fencing refers to a 3-wire design erected on the netwire fences as an offset.

In 26 trials (11 without electric fences over 48.2 days; and 15 trials with electric fences over 63.7 days), there were 426 documented contacts between elk without the electric fence; yet, 0 contacts between adult elk when the electric fence was present. Twenty-four of 25 elk during the electric fence study were "completely deterred."

I have worked with electric fence configurations to control white-tailed deer since the late 1980s, most recently directing a master's thesis on efficacy of a three-wire system to protect agricultural crops and food plots. I must agree with the findings of the above studies that electric fences are efficacious as a barrier between deer, but also add that additional experience supports that even free-ranging, naïve cervids can be controlled.

Hence, it is my professional opinion, an 8 feet tall, netwire fence, with a 3-wire outrigger on each side would reduce nose to nose contact between deer at a reliable level.

Summation of Professional Opinions

The white-tailed deer is a highly prolific, adaptable ungulate that exists in

a vast array of habitats, including urban environments. Although nearly extirpated by the turn of the 20th Century, these animals have made a remarkable comeback, with populations now estimated at over 30 million. In fact, it is my opinion (and that of most deer biologists) whitetail populations have exceeded carrying capacity in most of the areas where they reside, and may be declining in some areas. We already are seeing significant habitat degradation, disease and economic and human health impacts (accidents, Lyme disease, agricultural damage, landscape damage, etc.) from this species. At the same time, deer are the most popular and economically important game species in the US, if not the world; fostering concerns both by sportsmen and game agencies when populations appear to be threatened. Deer not only generate significant incomes and value-added products to the private sector, but deer hunting licenses, fines and fees provide a great deal of financial support to game agencies. At the same time, the deer farming industry has grown exponentially over the last two decades, and now ranks high in agricultural commodities. In Texas, deer farming and ranching now ranks as a top 10 agricultural commodity. So, it is understandable when a disease such as CWD seems to appear from "nowhere," alarms are sounded!

Whitetails endure a great number of diseases, predators, parasites and environmental stressors annually, yet continue to thrive. The population size, plus the inherent genetic variability of the species pre-adapt them to change whenever a significant mortality agent appears. The idea any disease will totally extirpate whitetail populations is unwarranted and not supported by scientific data or facts. Indeed, although millions of dollars have been spent on research and "control" of CWD, scientists only recently have considered the potential for genetic resistance within the genome of deer and elk. Yet, genetic resistance has been used to advantage by the sheep industry to produce scrapie resistant herds. Scientists at the University of Wisconsin-Madison and Stevens Point have suggested in the long-term deer may make genetic shifts to deal with the disease, and I am in agreement.

After 10 years of dealing with CWD issues, my experiences in Wisconsin, and after my review of knowledge relating to this white paper, I feel concerns for devastating impacts of CWD on the nation's deer herds needs to be revisited! Understanding the biology of white-tailed deer as I do, and combining this knowledge with that discussed in this report, I must conclude the long-term impact of CWD will not be significant declines in deer populations. **I must question the rationale behind CWD as a serious reportable disease.** As noted in this report, studies support:

1. CWD has not been demonstrated to have significant impacts on deer population dynamics.
2. CWD has not been demonstrated to have a significant human health concern.
3. CWD has not been demonstrated to affect other species, particularly livestock.

I did not come to these opinions lightly. These are strong statements and I make them after a great deal of thought. However, I am not alone in my skepticism. The white paper ("What we know about CWD that isn't so?" 2013) by my respected colleague, Dr. Don Davis (Texas A&M University Veterinary College) clearly states:

"It is often stated that CWD is a threat to the natural resource, and 'is a devastating disease to wild populations'. This belief again is not supported either by scientific data or empirical observations through time. The USDA/APHIS data based on a huge sample collected over 10 years indicates that CWD occurs at a very low prevalence. The loss of 10-40 animals per one million animals due to CWD is not significant in wild populations when those populations number in the 100,000s or in the 1,000,000s. Therefore clearly CWD is neither an epidemic nor is it population limiting. Losses at a level of 1-4 per 1000 are hardly 'devastating'. Populations of game animal species such as deer and elk are routinely harvested by hunters at a level of 10% to 25% without deleterious effects on those populations. A disease such as CWD with a prevalence rate of 0.1 to 0.4% will not have a significant effect on those populations. These levels of morality loses, even if the losses are additive instead of compensatory, will be insignificant. Despite some reports of population losses due to CWD in isolated or local instances, there is no supporting scientific data to either support these speculative hypotheses or rule out other possible causes for the population decrease such as drought, other diseases, or loss of suitable habitat.

Another popular misconception about CWD is that it is highly contagious. The definition of a highly contagious disease is a disease that is rapidly and easily transmitted from infected individuals to susceptible individuals, and usually with a short incubation period. An example of a highly contagious disease is plague in the pneumonic form in humans. The common cold is another example of a contagious disease. CWD is a chronic disease (hence the name) and not an acute disease. CWD can have an incubation period of many years, it can be transmitted from one individual to another but not often or easily. If indeed, CWD is a contagious disease then why does it exist with a very low prevalence rate and rare occurrence? If CWD is a contagious disease then why with a history of more than 45 years in North Eastern Colorado has CWD not completely eliminated populations of elk and deer in that area? If CWD is easily and often transmitted, why is the disease only found in 1-4 animals per 1,000? The answer is obvious. Many of the widespread and accepted 'facts' about the prevalence of CWD and the host distribution of CWD are erroneous, illogical, and untrue.

It is notable that Dr. Beth Williams stated that CWD 'is more correctly perceived and classified as a special type of toxicity' than as an infectious disease."

I stand firmly in support of Dr. Davis' conclusions, and have arrived at the exact same positions independently. In a 2001 TV program (Journal of the Texas Trophy Hunters) on the Outdoor Channel I correctly predicted the outcome of the ill-conceived, yet well-meaning, "eradication" program in Wisconsin. Ten years later, my predictions were mostly validated.

These are my professional opinions, given with a great deal of thought and review of current information. Knowledge never is perfect or complete, but decisions have to be made based on the best available science.

References for all research findings included in this article can be had by request to www.drdeer.com.