The Prion Protein

The prion protein occurs in most species of mammals, including humans. The biologic activity of any protein depends on its 3-dimensional shape, much like a lock and key must have a complementary shape to mesh together properly. In its natural form, the prion protein influences neural activity, memory retention, and sleep patterns. Normal prion proteins are synthesized, serve their function, and are broken down specifically, a misfolded form of a protein known as a prion. Chronic wasting disease affects members of the deer family, including elk, mule and white-tailed deer, moose, and caribou. The disease was first diagnosed in captive mule deer in the 1960’s, and in free-ranging Colorado mule deer in 1980. Since that time, CWD has spread into free-ranging populations of mule and white-tailed deer and elk in 24 US states and two Canadian provinces.
and recycled in the body or excreted as waste. However, the prion protein can become bent or misfolded in such a way that it becomes resistant to natural breakdown. Furthermore, these abnormal, misfolded prions can convert "normal" prion proteins to the misfolded form, resulting in a chain reaction and accumulation of the misfolded prions. The misfolded prions clump together in the brain, which creates holes in the tissue and impairs brain function, leading to weight loss—the "wasting" symptom of chronic wasting disease—lack of coordination, drooling, excessive thirst or urination, lack of fear of people, and eventually death. Infected individuals die between 2 weeks to 8 months after showing symptoms. Many CWD-positive animals die from secondary diseases or illnesses, predation, or road mortality prior to the onset of obvious symptoms.

The conversion of normal to abnormal prions occurs over months or years after exposure. Therefore, it may be 1-2 years or more before any physical symptoms begin to show. During this time, the infected individuals shed infectious prions into the environment via body fluids and excretions. Misfolded, infectious prions are found in urine, feces, saliva, blood, antler velvet, and carcasses of infected deer. The misfolded prions are spread through either direct contact with an infected individual or contact with prions in the environment. The misfolded prions are so resistant to breakdown that they can remain in the environment for years. Studies of scrapie, a closely related prion disease in sheep, have shown that infectious prions may remain in the soil at least 16 years after infected individuals were removed. There is no indication that CWD can be spread to humans. However, the Centers for Disease Control and World Health Organization recommend against eating meat from infected animals. Despite widespread beliefs, the misfolded prions cannot be cooked out of meat.

**Chronic Wasting Disease in Texas**

Chronic wasting disease was detected in Texas mule deer in 2012 in far west Texas. The disease probably spread to that part of Texas via natural movements of mule deer from an adjacent, CWD-endemic area in New Mexico. Since that time, CWD has been found in free-ranging mule deer, white-tailed deer, and elk in the Panhandle and Trans-Pecos regions of the state. In Texas, CWD remains rare, but the long-term prognosis is troubling, as the population-level impacts of CWD do not become apparent for years or decades. In areas of Wyoming, where CWD has been endemic for 30 years or more, 31% of harvested deer tested positive for CWD during 2001–2009, and the population has declined by over 50%.

It appears older animals, particularly bucks, are most likely to become infected with CWD. Mature bucks are important to both the Texas hunting industry and economy of the state as a whole. In addition to this, mule deer fawn survival is highly variable in Texas because of inconsistent rainfall, which leads to dependence on older individuals to maintain population levels. If CWD continues to spread, Texas mule deer are vulnerable to population declines and decreased buck age structure.

**Controlling the Spread**

There is no vaccine or treatment for CWD. Because of this, management of CWD has focused on early detection and containment as the most realistic and economically feasible option. Therefore, natural movement of deer across the landscape may provide an understanding of how the disease spreads geographically. Both dispersal (movement from the area of birth) and migration (seasonal movements) increase the possibility of CWD transmission into new areas.

Texas mule deer populations are non-migratory and so dispersal is the main way for the disease to naturally spread. Mule deer populations may become isolated by landscape features that serve as barriers to movement, such as major roads, mountain ranges, and rivers. Populations that are physically isolated can be detected using genetic markers. Understanding of corridors or barriers to movement of mule deer across Texas could be used to predict the spread of CWD. This leads to an important question: Could CWD in
Mapping Genetics of Mule Deer

The Texas Parks and Wildlife Department paired with researchers at the Caesar Kleberg Wildlife Research Institute at Texas A&M University-Kingsville and the Borderlands Research Institute at Sul Ross State University to conduct genetic analyses of Texas mule deer. The Department collected tissue samples and data from hunter-harvested mule deer throughout the range at hunter check stations during 2012–2016.

We extracted DNA from the tissue of about 500 mule deer harvested across their range in Texas. We compared genetic similarities and differences among mule deer and used this information to infer past movements of deer across the landscape.

Hybridization

Our early analyses revealed some individual mule deer were quite genetically different from the others. There was no geographic pattern, as the genetic outliers occurred throughout our sampling range. Because these deer were spread across the landscape and could not be explained by physical barriers alone, we wondered if the genetic anomalies could be from hybridization with white-tailed deer. All samples were collected at check stations by trained personnel, and all had the phenotypic appearance of mule deer. However, hybrids cannot always be diagnosed by appearance alone, especially back-crosses. Back-crossing occurs when a hybrid breeds back to one of the parental species. First generation (F1) hybrids are a 50-50 mix of mule and white-tailed deer, while back-crossed individuals (F2) that breed to mule deer would be 25% white-tailed deer; further back-crossing (F3) would be 12.5% white-tailed deer, and so on. Previous studies have documented hybrids where the range of mule and white-tailed deer overlap, so we sampled and analyzed west Texas white-tailed deer to confirm our suspicions. Through genetic analyses and computer simulation of hybridization, we determined that our genetic outliers were indeed hybrids. Overall, about 5% of our mule deer samples had some ancestry from white-tailed deer; most were back crosses (F2 or F3), as first-generation (F1) crosses were rare. This suggests that hybridization has occurred over multiple generations and throughout a wider geographic area of Texas than previously found. While interesting from an ecological point of view, the presence of hybrids could influence our overall landscape analysis. We identified and removed all detectable hybrids from the analysis so that the only factor that would influence the genetic results was landscape features.

Do Landscape Features Influence Deer Movements?

The next step was to investigate potential barriers to movement of Texas mule deer. The genetic analyses revealed only minor differences among mule deer across their vast range in Texas.
The biggest factor that explained genetic differentiation of Texas mule deer was simply the geographic distance between individuals. Deer harvested from the same ranch were more genetically similar than deer harvested 20-50 miles or more away. We calculated genetic similarity between all pairs of mule deer and analyzed the map distance between harvest locations. This allowed us to estimate the size of a local population of deer that were genetically non-independent of each other—not closely related, but not so different as to be separated by more than a few generations of movement and dispersal. We found that mule deer within about 93 miles of each other were more genetically similar than expected compared to randomly chosen pairs of deer. If we imagine a population of mule deer as a circle with a diameter of 93 miles, then the geographic size of the population translates to a whopping 4.3 million acres! This result tells us that mule deer can move through most of their range with few or no barriers.

We then analyzed the data separately by region to determine if there were any differences between mule deer from the Trans-Pecos region in far west Texas and the Panhandle. We found that a Panhandle mule deer population covers a smaller area, about one-fourth the geographic area of a Trans-Pecos population. However, “small” is a relative term, as a Panhandle population still covers about 1 million acres! Deer in the Panhandle region face more local barriers than Trans-Pecos deer due to greater land fragmentation in parts of the Panhandle, where deer must cross many more roads, fences, and farm land.

We found no effects of elevation, rivers, or major highways on Texas mule deer. Studies in Canada and Wisconsin found that rivers and mountains blocked movement of deer, but within Texas this does not appear to be the case. We did find some slight genetic differences between the Trans-Pecos and Edward’s Plateau ecoregions and between the High Plains and Rolling Plains. We believe that land fragmentation in the southeastern Panhandle and cliffs and canyons of the lower Pecos River may exert subtle effects on mule deer movements.

Implications for Management
There appear to be no barriers to movement throughout much of the range of mule deer in Texas. We found only subtle effects of landscape on deer populations, and these were small enough that they may not form firm barriers to movement. Therefore, CWD might eventually spread throughout the region via natural movements of deer, though our results predict that the disease may spread slower in the Panhandle region than the Trans-Pecos due to landscape fragmentation. It is important to continue monitoring and testing of deer in the region. Those who manage deer in the range of CWD should stay informed and re-consider any management practice that concentrates deer or may facilitate the spread of the disease. Other management actions, such as restricting live deer movements, intact carcass restrictions, and strategic harvest strategies will help contain the disease until the time when more effective treatment or control measures are found.

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~Thanks to all Sportsmen~