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# GENETICS IN WHITETAIL DEER:

## IT'S ABOUT MORE THAN JUST LARGE ANTLERS

When we think about genetics of whitetail deer, we almost always think about the large-antlered buck we see walking by our tree stand. In fact, there is a booming business in the deer-farming industry to share genetic materials to breed those bucks that give the highest score. Over years or decades, mating these large-antlered bucks in captivity has resulted in deer farms having deer with antlers that we would never see in the wild deer we hunt. This form of breeding bucks for big antlers over many generations coincidentally provides us with a useful tool for another type of deer research—landscape genetics of wild deer. The use of genetics in the deer farming industry is different than how we might use genetics to manage or understand the ecology of wild whitetail deer. Genetics have become even more important recently due to the wild deer interface with farmed deer as it relates to chronic wasting disease.

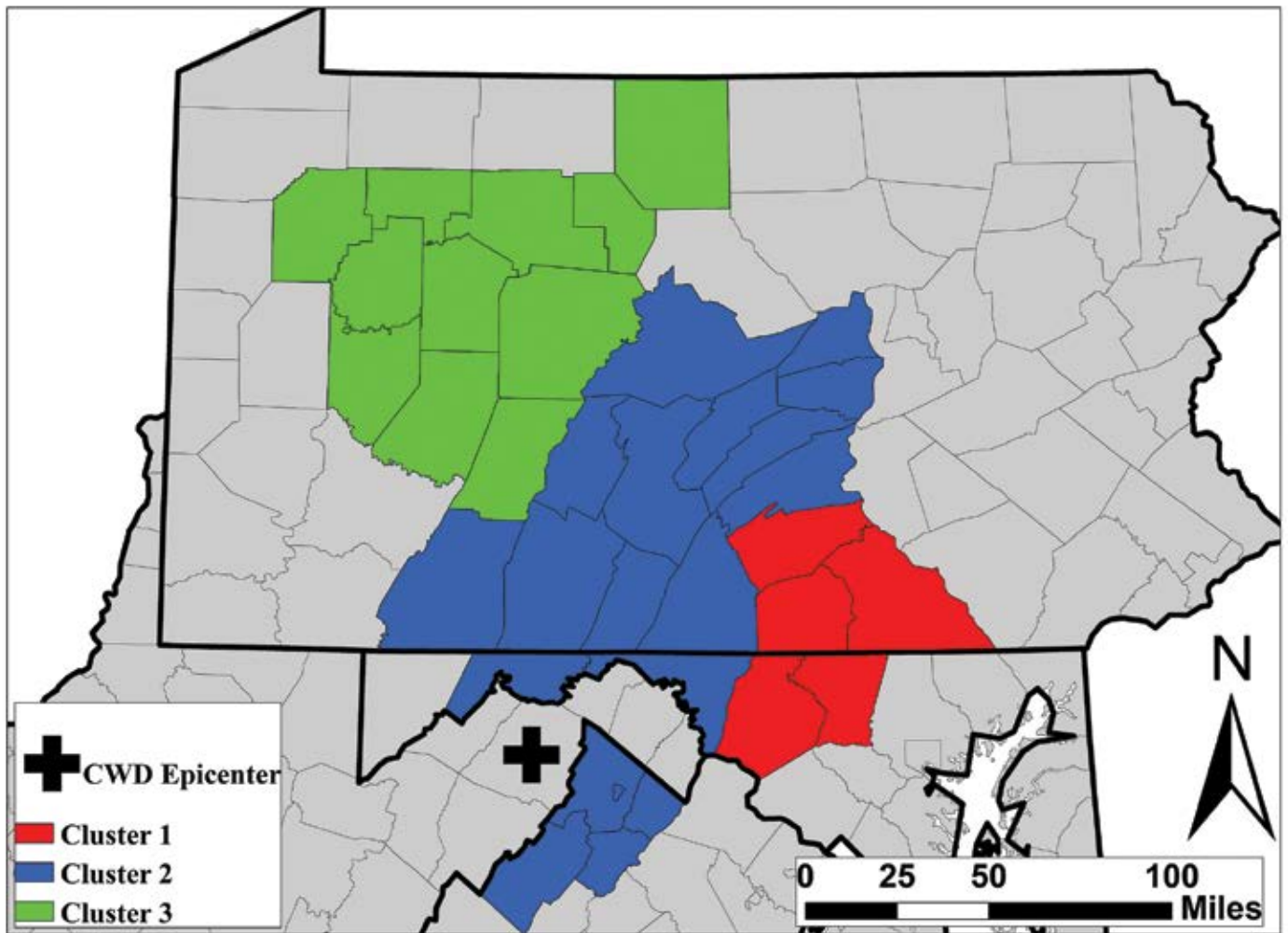


FIGURE 1. Relative relatedness of deer based on three subpopulation clusters in the Mid-Atlantic region based on genetic markers for over 2,000 whitetail deer sampled. The epicenter of chronic wasting disease in the region is in West Virginia where it was first detected in 2005. It is indicated on the map with a plus (+) symbol.

Chronic wasting disease (CWD) is what we call a transmissible spongiform encephalopathy that is characterized by abnormal deposits of the misfolded prion protein throughout the brain and central nervous system of infected cervids. It is similar to mad cow disease that infected European cattle and Creutzfeld-Jacobs disease in humans. While research suggested that CWD poses little risk to agriculturally important species (e.g. cattle) and human health, recent experimental infection of a primate species crab-eating macaque (*Cynomolgus macaques*) has raised concern of CWD overcoming the species barrier and posing a risk to human health in the future.

Genetics offers a few pathways of insight to understanding CWD as it pertains to movements of deer around the landscape and susceptibility to disease.

#### MOVEMENTS

Deer behavior can facilitate disease spread, as dispersing males have been documented to move more than 100 kilometers and the tendency of females to remain in a local area can exacerbate direct transmission within family groups. Male dispersal distance was greater for deer in more open than forested landscapes (determined from deer equipped with satellite collars by researchers in Pennsylvania); however, limited information can be gleaned from studies

that require capturing and collaring deer. Genetics provides data collected over a broad geographic scale—especially through collection of tissue samples provided by hunters or via other means that supply voluntary sampling. With this information, we would be able to map with considerable detail the landscape genetics of deer and not rely on capturing and monitoring deer with GPS technology.

To this end, my colleagues and I initiated a study to see how chronic wasting disease could travel across a landscape after starting in an epicenter in West Virginia in 2005. We have collected tissue samples from hunters in the Mid-Atlantic region that includes Maryland,

Pennsylvania and Virginia to assess genetic markers called “microsatellites” for each deer. Combinations of these markers are unique to individual deer and are shared between family groups or will be shared by others when a male or female disperses and breeds with another subpopulation of deer. Using these markers, we can infer subpopulations of deer across a large area of the Mid-Atlantic based on relatedness of these subpopulations in the areas we received genetic samples.

Landscape-level sharing of genetics can also provide insight on potential barriers to movement by deer across the region. This, in turn, can show us potential transmission pathways of disease

because not only is genetic material shared, but disease may be shared as well. As you can see, deer share genes across a large area in the region (Figure 1), although this is not surprising considering what we know about how far deer move across an area.

There is some isolation of deer subpopulations as a result of highways, rivers, and high ridges in the region (Figure 2). Can we answer the question in this area about whether CWD was started in the more than 1,000 deer-farm operations in the region, or did it expand from the epicenter in West Virginia where it was first detected (Figure 1) in 2005? Stay tuned.

### SUSCEPTIBILITY

Rare differences of the prion protein gene, called “polymorphisms,” have been investigated for their link to potential reduced susceptibility to CWD infection across the range of whitetail deer. The possibility that there is a less-susceptible genotype that shows a difference in infection rates when compared to deer with more susceptible genotypes requires further research. Any indication that genotype-based susceptibility is a potential factor affecting shedding of infective disease agents or increases the lifespan of the infected deer would be valuable information in our fight

against this disease. Several investigations on the proportions of the prion protein in whitetail deer that identified a predominant genotype was observed throughout North America (50–87 percent). Understanding if there is a link between CWD susceptibility and particular genotypes of the prion protein gene would allow wildlife managers to develop more targeted mitigation strategies accounting for underlying genetic risk factors. Mitigating the effects of this disease is particularly important, given the economic, social, and ecological importance of whitetail deer. ■

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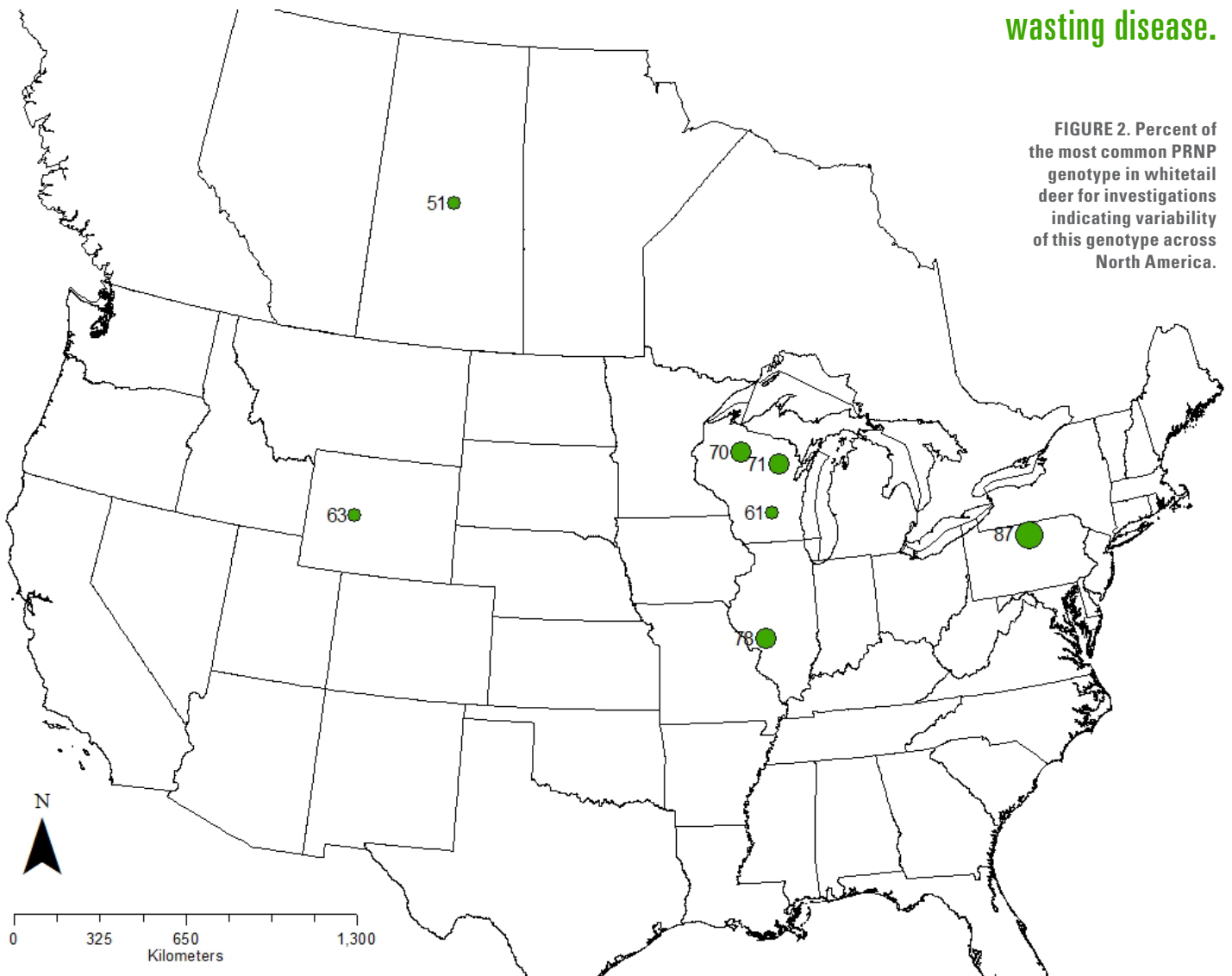


FIGURE 2. Percent of the most common PRNP genotype in whitetail deer for investigations indicating variability of this genotype across North America.