

## **Landscape Patterns of Chronic Wasting Disease**

### **Introduction:**

Chronic Wasting Disease (CWD) is a prion disease that affects members of the Cervidae family such as deer, elk, and moose. It was discovered in Wyoming and Colorado in the late 1970s and has spread at a tremendous rate since then. (Gilch et al. 2011) Prions are the infectious agents responsible for transmissible spongiform encephalopathies (Mad Cow's Disease, Creutzfeldt-Jakob's Disease). They are an aggregate of a misfolded protein that can seed normally folded proteins to misfold and cause a chain reaction of protein replacement in the brain that leads to neurodegeneration and ultimately death (Escobar et al. 2020). CWD is incredibly contagious, with multiple pathways of infection, from mother to child, or horizontally to unrelated deer through many fluids and tissues that contain the prion combined with high persistence in the environment (Escobar et al. 2020). Since prions are not destroyed by conventional techniques like heat, UV radiation, or chemical disinfectants they can survive in soil, food, water, or bedding used by infected individuals for a long time. This causes reservoirs of potential CWD transmission to uninfected deer when they share habitat, foraging space, and water with infected deer, even at different times. This stresses the necessity to understand this disease and its geographic distribution, but it also shows that it is near impossible to destroy these infectious agents and prevent wild deer from coming in contact with them in the environment. (Gilch et al. 2011) Multiple and creative management strategies need to be implemented to properly control CWD.

Since infectious diseases are not distributed randomly throughout landscapes, differences in environmental conditions must be accounted for when modeling distribution or spread. Identifying key landscape features that facilitate CWD transmission is a high priority, features like vegetation communities, soil composition, local and seasonal climate, and land-use patterns can identify high risk hotspots, studies at the landscape-level of CWD occurrence remain neglected and are critically important (Escobar et al. 2020). If environmental conditions that increase prion transmission are identified, the environmental niche of this disease can be expressed in terms of geography, and more powerful predictive models can be created (Escobar and Craft 2016).

### **Background Literature**

Disease transmission and biogeography can be extremely complicated and influenced by an unimaginable number of factors, so it is imperative the proper environmental variables are selected for the specific study system (Escobar and Craft 2016). These variables may directly influence deer contact with contagious CWD material in infected individuals or the environment, and have received relatively little study since CWD's recent discovery. For example, differences in soil and vegetation characteristics may increase the lifespan of prions in the environment (Bartel-Hunt and Bartz 2013, Pritzkow et al. 2018), while harsh winter climates and limited food availability force deer into constricted winter ranges, increasing deer density and contact (Conner et al. 2008). Environmental factors may also indirectly influence CWD distribution through limiting the habitable deer range and creating bottlenecks for deer dispersal. Topography, elevation, and landscape connectivity influence deer habitat and deer densities. Features of the terrain such as these have been explored as covariates in evaluating the effect of deer reduction on CWD spread, but have not been fully studied in terms of CWD transmissibility (O'Hara Ruiz et al. 2013, Mateus-Pinila et al. 2013). Human land-use and changes in land use have both been

highly implicated in CWD prevalence in other study systems (Farnsworth et al. 2005) and exploring trends in CWD and land-use in Wisconsin can lead to better management decisions.

For the regression Professor Raynor and I decided to begin with white-tailed deer population, human land use, and deer habitat as basic predictors of CWD at the quarter section scale. We thought about adding other environmental variables, but the southern part of Wisconsin is an interesting area for deer, and we wanted to not overload the regression with predictor variables. Since there is so much agriculture, the deer do not have the same ecological pressures as white-tailed deer in the north. We hypothesized CWD would mainly vary based on land use and the amount of deer habitat in an area, especially because the abundance of food and homogenous topography would cause any area of “deer habitat” to be essentially equal.

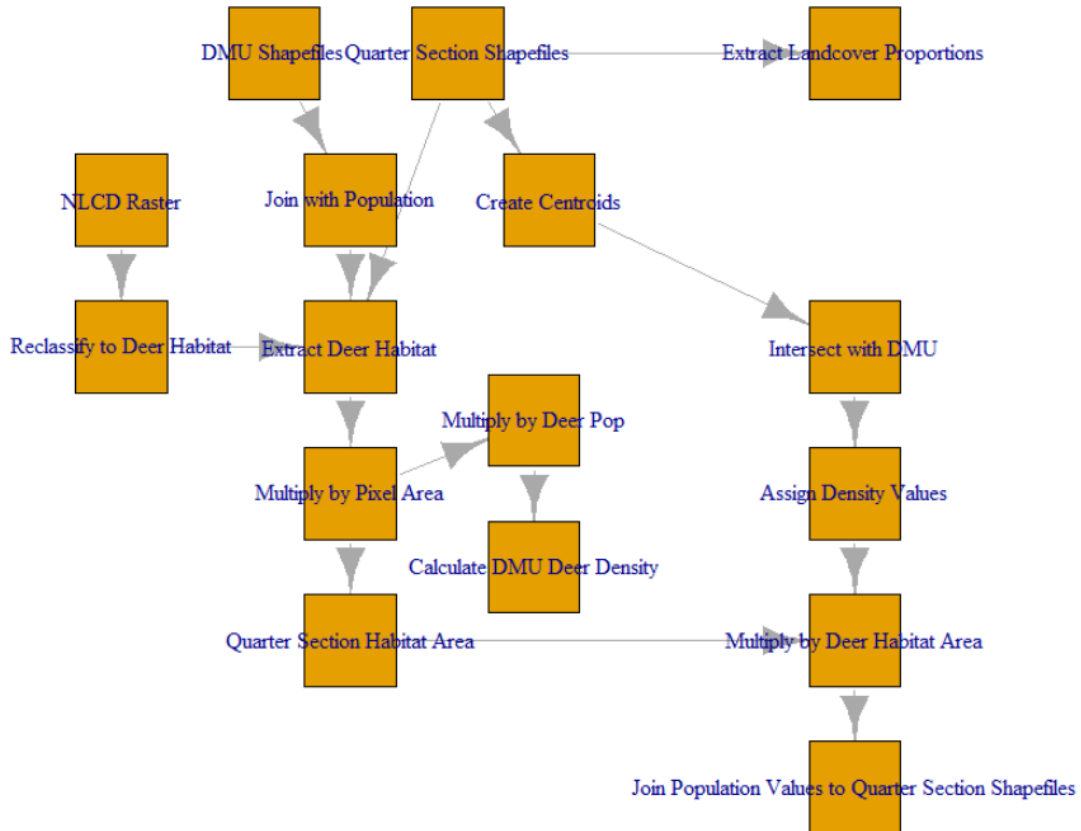
**Procedure:** (Note: The network package used to create the data analysis flowchart in R did not allow for annotations and many steps were done simultaneously, so I have included all my code in my ArcPro project package with more details on each piece of the data analysis).

In our effort to model landscape relationships of CWD, we will begin with white-tailed deer population, human land use, and deer habitat as basic predictors of CWD. Deer population data was provided at the Deer Management Unit (DMU) scale, and this data was joined to shapefiles. There is no data collected for metro areas or native lands so those DMUs are excluded. Since deer population data at the scale of deer management units does not truly represent the heterogeneity of these management units, we quantified deer population at the finer Public Land Survey System quarter section scale that the CWD data is provided in. The population for each deer management unit is assumed to be distributed throughout the suitable deer habitat in a management unit. A binary deer habitat raster was created by reclassifying forest, shrubland, and agricultural values of the USGS National Land Cover Database 2016 raster. Then I limited the deer habitat raster to continuous patches greater than or equal to four hectares (Figure 1). The deer habitat values are summed for each DMU, and the deer population is divided by the total deer habitat to create a deer density value for every DMU. The deer habitat values are then extracted and summed for each quarter section polygon. The centroid of each quarter was used to assign a deer density value from the DMU's, and this was multiplied by the deer habitat sum to estimate deer population at the quarter section scale. Using NLCD 2016 the proportion of each land cover category for all quarter sections was calculated and added as an attribute to the corresponding polygons. These quarter section polygons with predictor variables as attributes will eventually have CWD data joined from the Wisconsin Department of Natural Resources and used in a geographically weighted regression.

## Results

Without the regression there are little results and even fewer conclusions to report from this project, but I have included some preliminary figures. Figure 2 is a map from the WIDNR, and it shows the cumulative CWD counts for southern Wisconsin at the quarter section scale. Most cases are clustered on either side of the Wisconsin River. If we move down to Figure 3 and 4, its clear that deer populations and forested area are high around the Wisconsin River. Since these CWD tests and population estimates are conducted opportunistically from hunter harvested deer, this pattern may also just be a result of people hunting near or in forests more than farmland.

The main output of my project is the dataset “quarter\_pop” which contains all of Wisconsin at the quarter section scale with all our predictor variables. It is saved in the dataEdited directory as a csv, rds, and shapefile.



*Data Analysis Flow Chart:* the top of each path is a shapefile or raster input, and the geoprocessing was done chronologically from left to right in this diagram. (e.g., Extracting landcover proportions and calculating quarter section population values was done simultaneously after creating centroids and calculating DMU deer density.).

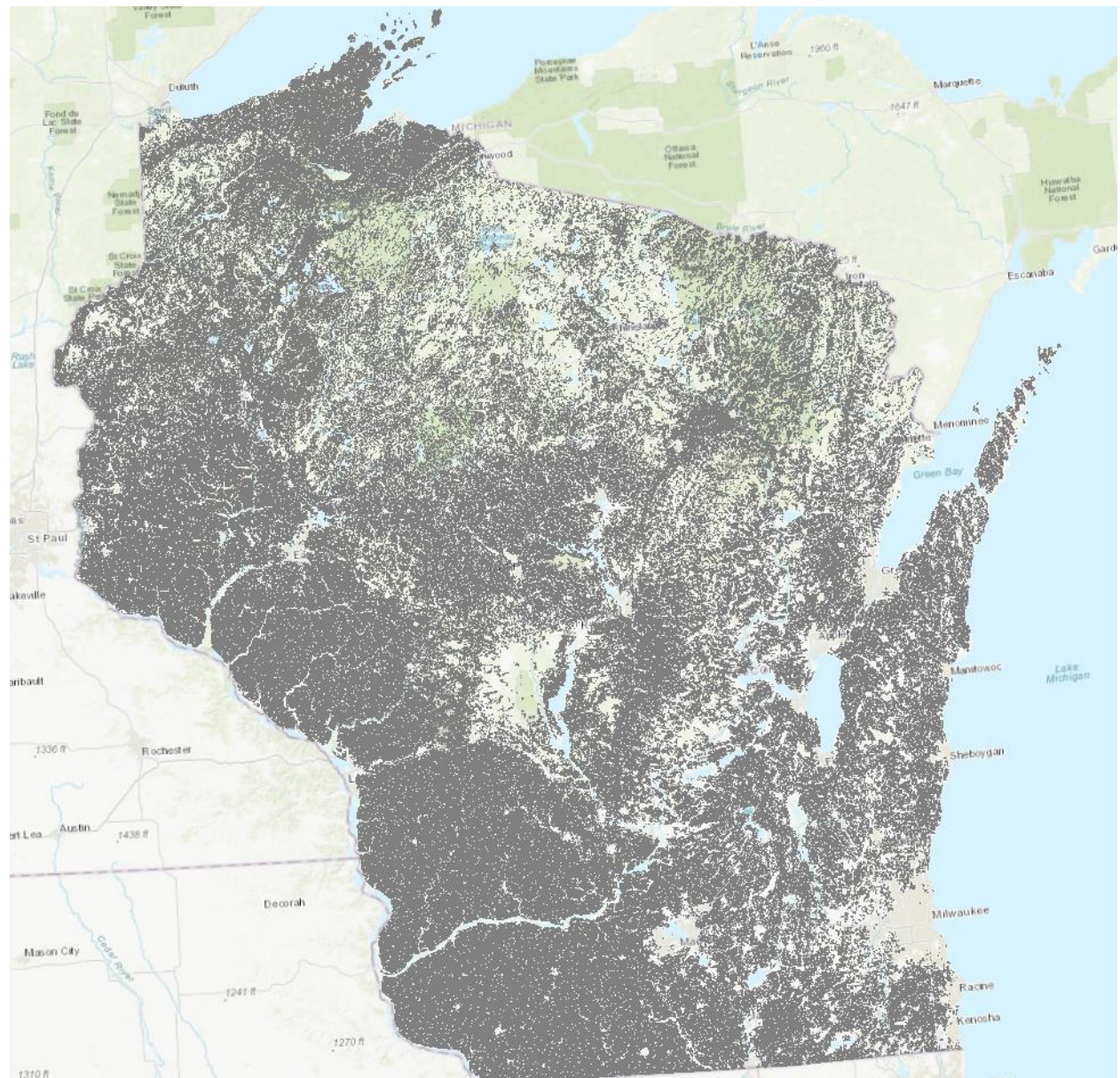


Figure 1: Deer habitat binary raster, grey pixels indicate suitable deer habitat at the 30m x 30m resolution.

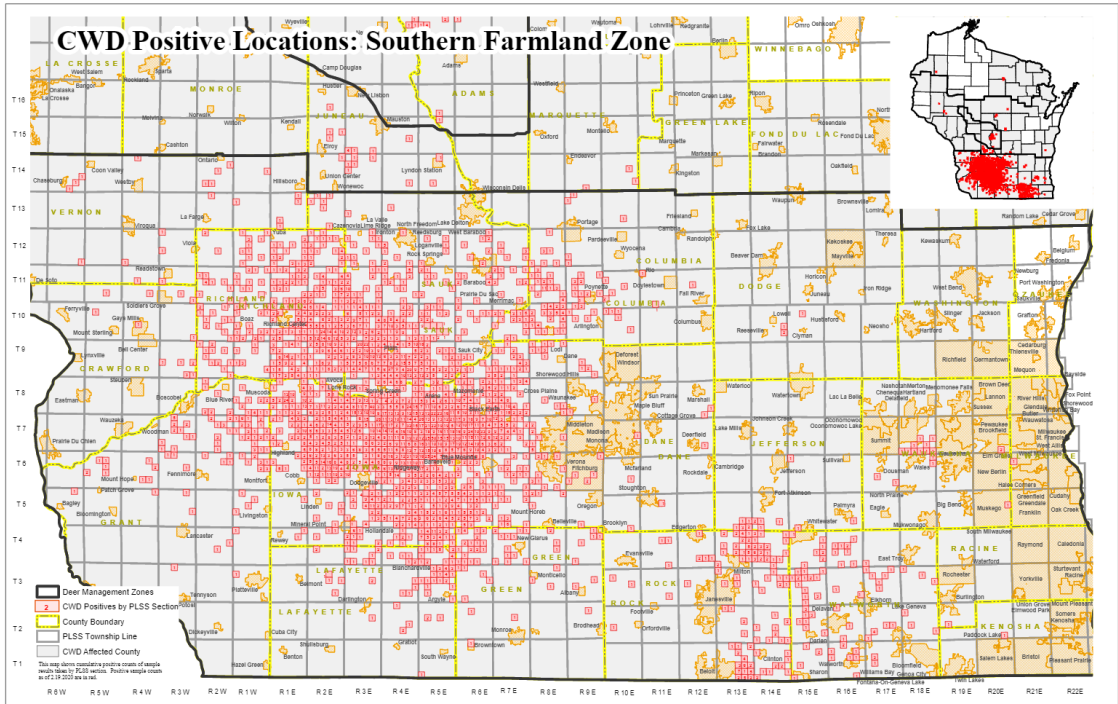


Figure 2: CWD positive test counts for southern Wisconsin PLSS quarter sections, a preview map of the WIDNR data we have applied for.

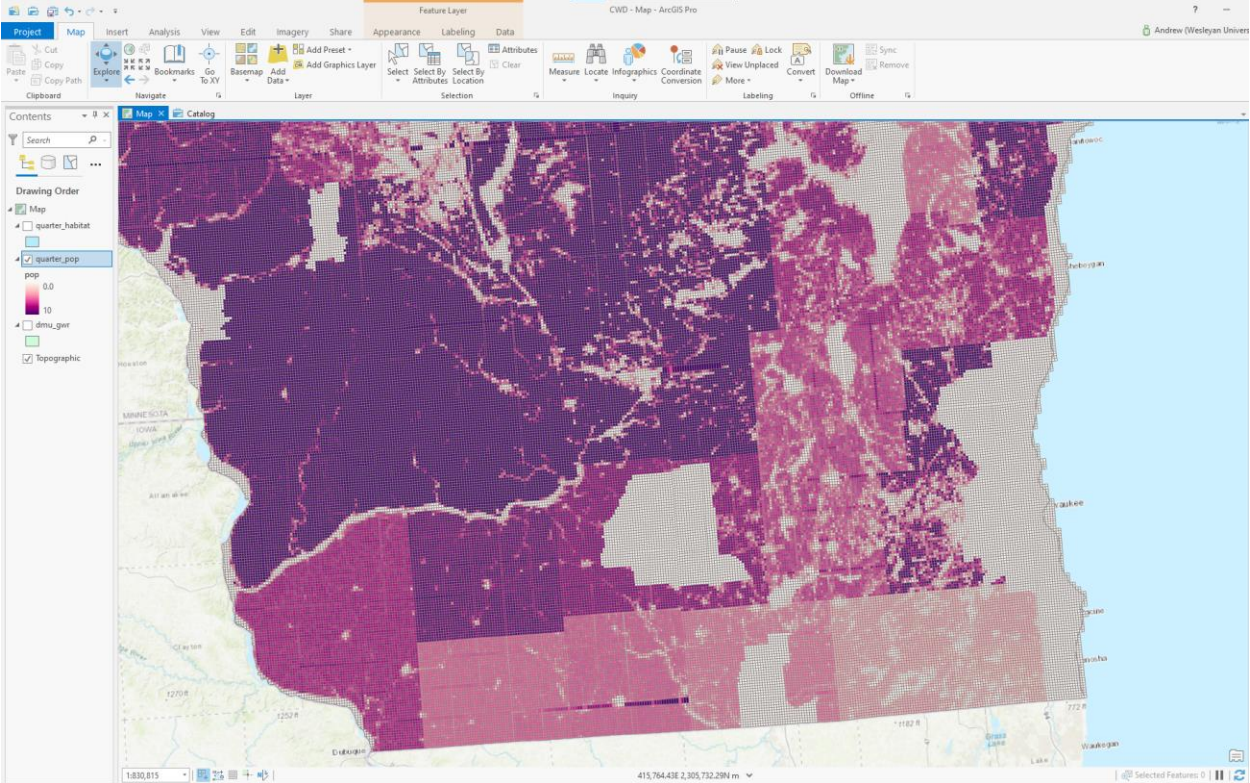


Figure 3: Southern Wisconsin PLSS quarter sections colored by their estimated deer population.

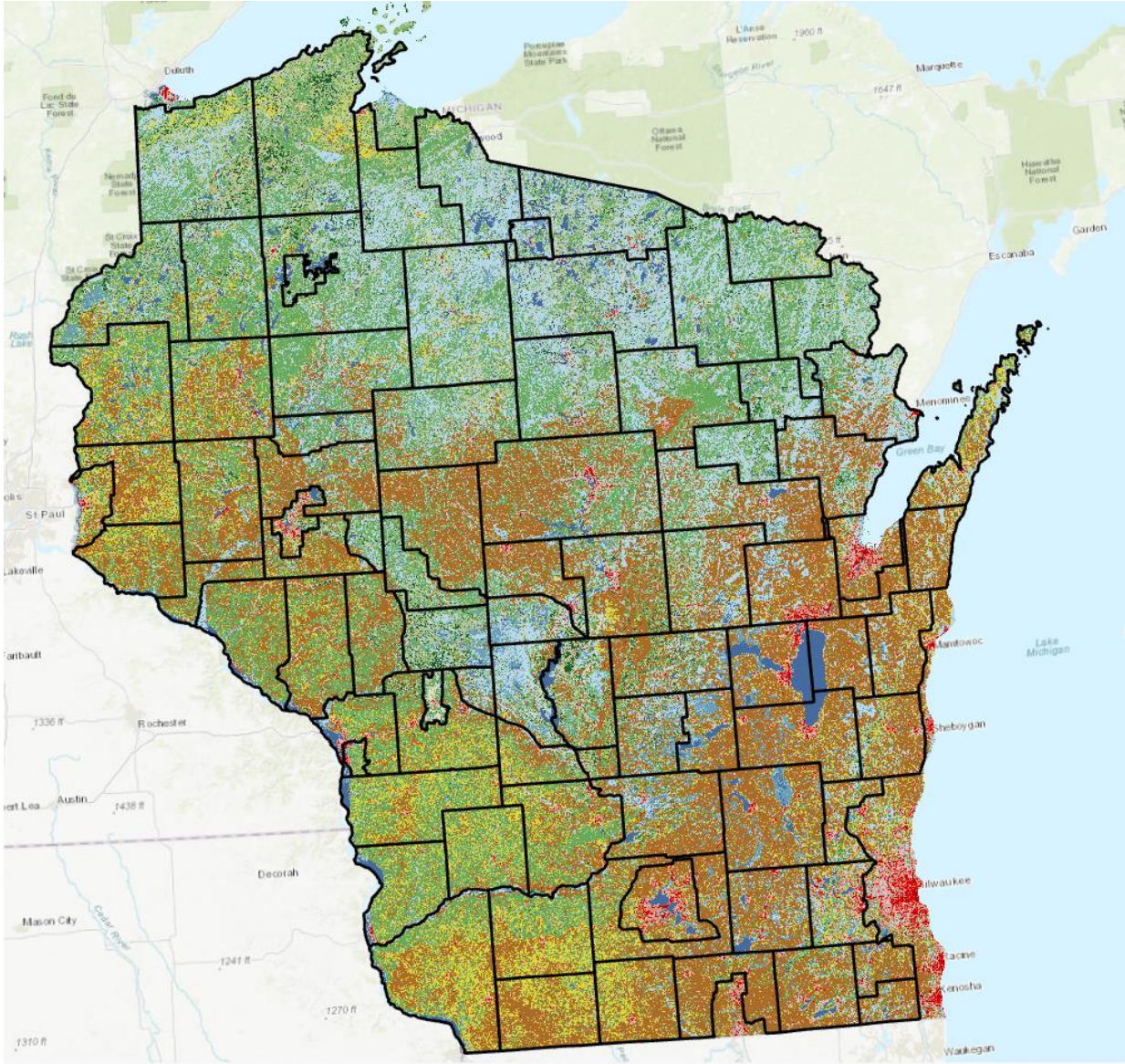


Figure 4: Wisconsin Deer Management Units overlain on top of the National Landcover Database 2016 raster.

## References:

Bartelt-Hunt, S.L. and Bartz, J.C., (2013). "Behavior of prions in the environment: implications for prion biology." *PLoS Pathog*, 9(2).

Conner, M.M., et al. (2008). "Infectious disease in cervids of North America: data, models, and management challenges." *Annals of the New York Academy of Sciences*, 1134: 146.

Edmunds, D.R., et al. (2018). "Chronic wasting disease influences activity and behavior in white-tailed deer." *The Journal of Wildlife Management*, 82(1): 138-154.

Escobar, L. E., et al. (2020). "The ecology of chronic wasting disease in wildlife." *Biological Reviews* 95: 393 - 408.

Escobar, L.E. and Craft, M.E., (2016). "Advances and limitations of disease biogeography using ecological niche modeling." *Frontiers in microbiology*, 7: 1174.

Farnsworth, M.L. et al. (2005). "Human land use influences chronic wasting disease prevalence in mule deer." *Ecological Applications*, 15(1): 119-126.

Joly, D. O., et al. (2006). "Spatial epidemiology of chronic wasting disease in Wisconsin white-tailed deer." *J Wildl Dis* 42(3): 578-588.

Mateus-Pinilla, N., et al. (2013). "Evaluation of a wild white-tailed deer population management program for controlling chronic wasting disease in Illinois, 2003–2008." *Preventive Veterinary Medicine*, 110(3-4): 541-548.

O'Hara Ruiz, M., et al. (2013). "Influence of landscape factors and management decisions on spatial and temporal patterns of the transmission of chronic wasting disease in white-tailed deer." *Geospatial health*, 8(1):215-227.

Pritzkow, S., et al. (2018). "Efficient prion disease transmission through common environmental materials." *Journal of biological chemistry*, 293(9): 3363-3373.

Winter, S.N. and Escobar, L.E., (2020). "Chronic wasting disease modeling: An overview." *Journal of Wildlife Diseases*, 56(4): 741-758.