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CHRONIC WASTING DISEASE IN THE CENTRAL APPALACHIAN REGION OF THE UNITED STATES

A Thesis in

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by

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ABSTRACT

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy that was first detected in white-tailed deer (*Odocoileus virginianus*) in the northeastern United States (Northeast) in 2005. Maintaining a healthy population of white-tailed deer is important to states in the Northeast for numerous recreational and economic reasons. The Midwest documented a decline in hunter participation and hunter-associated revenue following detection of CWD, and the presence of CWD in the Northeast could have a similar impact on local economies and wildlife management efforts. Considering CWD is a new and emerging disease in the Northeast, I investigated sampling strategies by state, ecology of deer, and environmental drivers of disease to better understand CWD in the region.

I surveyed 14 state agencies responsible for CWD surveillance in the Northeast to identify how surveillance efforts have changed following detection of CWD in the region. Annual surveillance efforts throughout the region initially exceeded 15,000 deer per year. Loss of federal funding in 2012, however, led to a drastic reduction in these efforts (<9,000 deer per year), with Pennsylvania alone accounting for ~4,000 samples. This reduction in sampling requires states to allocate resources into areas that are at greatest risk for CWD to maximize efficiency of these limited resources. Literature pertaining to state responses to CWD had been nonexistent for the Northeast and these surveys were the initial effort for collaboration between adjacent states that had detected CWD. I compiled data that have been shown to influence movements of deer and epidemiology of CWD in other regions. This was particularly important because all previous analyses had been performed in regions with landscapes that were dominated by agriculture or rangeland in contrast to the predominantly forested landscape of the Northeast.

Spatial processes, including spread of disease, rely on the scale of deer movement (e.g., home range), and this scale can be related to landscape. Therefore, I estimated size of home range in relation to landscape for 45 white-tailed deer in Pennsylvania to identify the spatial scale that described movements and home range of deer in the region. I documented that size of home range was related to connectivity of forest, with contiguous forest associated with expansive size of home range. In areas with high levels of fragmentation or less contiguous forest, home ranges were small (e.g., 0.63 km^2 for females) likely because deer were able to fulfill daily requirements for forage intake over a smaller area. Differences in size of home range by landscape likely would result in differences in the distribution of disease due to increased contact rates among matriarchal groups of deer that occupy these fragmented landscapes. Conversely, deer that occupy expansive tracts of contiguous forest may establish home ranges of larger size (e.g., 2.65 km^2 for females) due to foraging and breeding requirements and also may introduce disease into new areas. These concepts suggest that surveillance in highly fragmented areas should be concentrated locally to reflect less expansive home ranges that may inhibit expansive spread of disease, whereas surveillance in contiguously forested areas should be applied to a broad scale to reflect expansive home ranges that may allow disease to spread into new areas.

To further understand these concepts, I incorporated landscape into my analysis of CWD by linking location-based surveillance data to environmental and spatial factors that could influence the distribution of CWD in Maryland, Virginia, and West Virginia. I identified a clustering of CWD in an area where forested habitat was sparse and open (e.g., agricultural) and developed (e.g., suburban) habitats were more prominent. I identified areas of elevated risk that included a potential corridor east of the study area where spread of CWD may be expedited among several states. This corridor was at elevated risk because it was dominated by an

anthropogenic landscape where forests were even less prominent than in the core of the study area where CWD was most prevalent. These open and developed landscapes may promote contact between deer, and proactively sampling these areas may improve feasibility of containment efforts if CWD is detected. Since my research concluded, a free-ranging white-tailed deer tested positive for CWD in this corridor in 2013 so state agencies should consider collaborating to further monitor spread of CWD in this corridor and other high-risk landscapes.

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Chapter 1

Surveillance and monitoring of white-tailed deer for chronic wasting disease in the northeastern United States

Chapter 1 was written in collaboration with Krysten L. Schuler and W. David Walter. This chapter was accepted for publication in the peer-reviewed *Journal of Fish and Wildlife Management* and will appear in the December 2014 issue. I have included proofs of this manuscript on the following pages.

Notes

Surveillance and Monitoring of White-Tailed Deer for Chronic Wasting Disease in the Northeastern United States

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Abstract

Chronic wasting disease (CWD) is a prion disease that affects both wild and captive cervid populations. In the past 45 y, CWD has spread from northern Colorado to all bordering states, as well as the midwestern United States (Midwest) and northeastern United States (Northeast), Canada, and South Korea. Because CWD is a relatively new issue for wildlife management agencies in the Northeast, we surveyed a representative (e.g., cervid biologist, wildlife veterinarian) from 14 states to gain a better understanding of state-specific surveillance measures. Between 2002 and 2012, New York (37,093) and Pennsylvania (35,324) tested the greatest number of harvested white-tailed deer *Odocoileus virginianus* in the Northeast. Additionally, the 14 states surveyed have tested 121,730 harvested deer, or approximately 15,216/y, since CWD was first detected in 2005. The most common tissues used by agencies in the Northeast for testing were retropharyngeal lymph nodes, which have been determined to be the most reliable in detecting CWD in cervids. Understanding CWD surveillance efforts at a regional scale can help to provide guidance for the development of new surveillance plans or the improvement of existing ones. Furthermore, collaborations among state and regional agencies in the Northeast may attempt to identify deficiencies in surveillance by state or subregion.

Keywords: cervid; chronic wasting disease; retropharyngeal lymph node; surveillance

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Introduction

Understanding tools used to monitor diseases when they are new and emerging is integral to surveillance and containment efforts at local and regional scales. Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy that was first detected in 1967 in a captive research facility in Fort Collins, Colorado (Williams and Young 1980). Chronic wasting disease is unique to members of the Cervidae family, including white-tailed deer *Odocoileus virginianus*, mule deer *O. hemionus*, elk *Cervus elaphus*, and moose *Alces alces*. Since initial detection, CWD has spread to more than 22 states, as well as two Canadian provinces and South Korea (Sohn et al. 2002; Rees et al. 2012). Of the states affected by CWD, five are in the northeastern United States (Northeast; Figure 1). By the turn of the 21st century, many states had adopted surveillance plans as CWD became increasingly prevalent. Additionally, most states banned importation of farmed or captive cervids from states with CWD to prevent the disease from being introduced into wild populations (Salman 2003). Movement of infected, captive cervids from South Dakota game farms has been identified as the cause of CWD in Canada (Kahn et al. 2004; Argue et al. 2008), and these transfers are also the most commonly accepted explanation for the presence of CWD in Wisconsin (Joly et al. 2003).

Increasing concerns over captive elk importation from infected game farms in the west prompted the state of Wisconsin to implement a CWD surveillance plan in 1999 (WDNR 2010). During the 2001 hunting season, three hunter-harvested male deer in the south-central region of the state tested positive for CWD. The earliest estimate of overall prevalence of CWD in Wisconsin's herd reduction zone was 1.5% from 21,285 deer tested between April 2002 and January 2004 (Joly et al. 2003; Grear et al. 2006). The most recent estimates in Wisconsin show prevalence as high as 23% in the CWD management zone (WDNR 2009). In Illinois, surveillance did not begin until after the disease crossed the northern border and was identified 2 wk before the firearms season in November 2002 (Miller 2003). Several measures have been taken to contain the spread of CWD in Illinois, including unlimited bag limits for antlerless deer. However, the increase in hunting opportunities for antlerless deer has not resulted in higher harvest rates in those areas. Additionally, sharpshooting—defined as the targeted culling of deer exhibiting signs characteristic of CWD—has been unsuccessful in preventing the spread of CWD (Shelton and McDonald 2012; Manjerovic et al. 2014).

Unlike the endemic areas of Colorado, Wyoming, and the Midwest, there has been no reputable determination of the mechanism that resulted in the presence of CWD in the Northeast in 2005, and there is no published information on CWD occurrence since its first detection in a captive deer herd in New York. These facts and the varied success that states in other regions have had in preventing the introduction of CWD and containing spread in local populations prompted us to conduct surveys of state agencies in the Northeast to gain a better understanding of surveillance measures taken to detect CWD. The objectives of our survey were to determine 1) the number of hunter-harvested deer tested for CWD by year in each state, 2) tissues that were most commonly submitted for testing, and 3) the laboratory used by each state to test CWD samples. Our results provide valuable information for agencies responsible for CWD testing regarding issues of sample size, appropriate tissues, and laboratories available for CWD diagnosis.

Methods

We initiated surveys of state agencies in June 2012 to gain a better understanding of the surveillance measures taken by 14 states in the Northeast. The survey area included Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Vermont, Virginia, and West Virginia. An agency representative (e.g., cervid biologist, wildlife veterinarian) from each state provided information via email regarding their state's surveillance efforts for detecting CWD. This information included the number of deer harvested and tested for CWD each year, the tissue(s) collected, and the United States Department of Agriculture (USDA)–certified laboratory at which the samples were tested. Although many states use a variety of testing protocols that include hunter-harvested, road-killed, and targeted (e.g., culled) deer, we chose to focus on the hunter harvest for means of comparison.

Results and Discussion

Intensive CWD surveillance in the Northeast did not begin until 2002, but all states had established surveillance plans by 2005 (Table 1). The number of harvested deer collected for testing ranged from 41 in Rhode Island in 2003 to 8,164 in New York in 2005. The intensive surveillance efforts in New York were in response to the discovery of CWD in two captive herds and two wild deer in Oneida County, New York. Between 2005 and 2012, states in the Northeast tested 121,730 harvested deer, or approximately 15,216/y. However, recent surveillance efforts have decreased sample sizes (e.g., 9,778 for all states in 2012) because of the use of alternate methods (e.g., road-kill) of surveillance in many states. Prevalence (proportion of positive samples to total number of samples tested) for all years combined was highest in West Virginia (0.71%) using hunter-harvested deer, whereas no other state had prevalence that exceeded 0.07%.

Alternate methods included use of weighted surveillance in New York (e.g., testing of high-risk deer based on sex and age; Walsh and Miller 2010), testing of road-killed deer, and targeted surveillance of deer exhibiting signs characteristic of CWD. West Virginia and Pennsylvania were the only states in the Northeast that detected CWD in road-killed deer, totaling three positive cases since 2005. West Virginia also identified 63 positives using targeted surveillance and was the only state in the

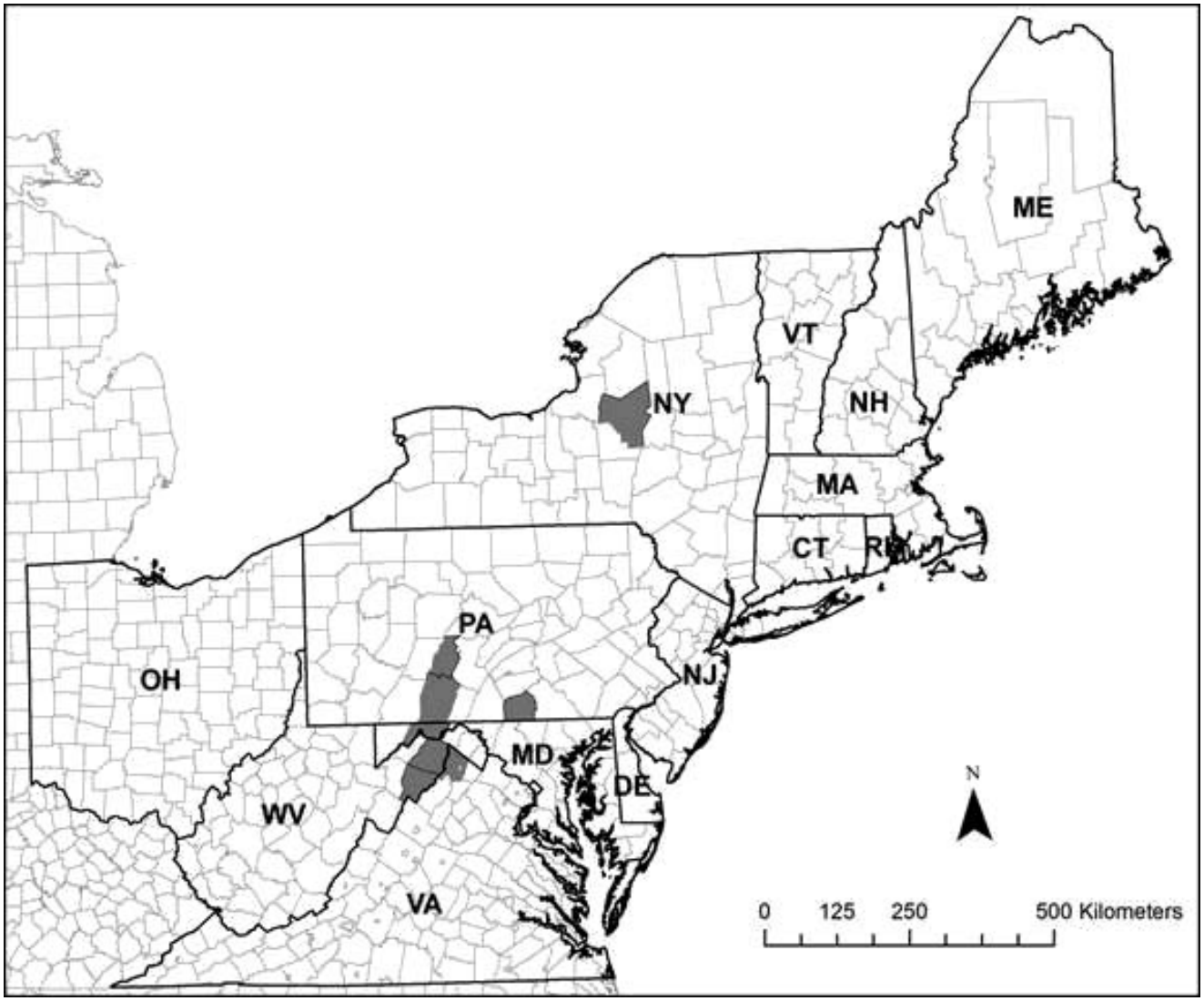

Figure 1. Map of the northeastern United States with shading of counties in states with captive or wild white-tailed deer *Odocoileus virginianus* diagnosed as positive for chronic wasting disease (CWD), 2005–2012.

Northeast that detected CWD using this method. Because of high costs associated with processing thousands of samples, weighted surveillance became the preferred method for detecting CWD in New York, where it is unknown whether CWD still persists in the state's deer herd (NYSDEC 2013). All states dealing with CWD in the Northeast drastically increased surveillance efforts after initial detection of CWD in their respective states, but later reduced these efforts after either failing to detect additional cases of CWD in large sample sizes (e.g., New York) or implementing alternate methods of surveillance. Pennsylvania also followed this trend by increasing surveillance efforts after detecting CWD in both captive and free-ranging deer in 2012, but it remains to be seen how sampling will change over time.

Tissues submitted for CWD testing included retropharyngeal lymph nodes, tonsil lymph nodes, and the medulla oblongata sectioned at the obex. Retropharyngeal lymph nodes were most common, as 13 of 14 states (92.9%) in the Northeast submitted this type of tissue. However, some states, including Maryland and Pennsylvania, also extracted the obex from hunter-harvested deer, and others extracted the obex only from deer exhibiting signs characteristic of CWD. Connecticut was the only state surveyed that relied primarily on testing of tonsil tissue (Table 2).

Miller and Williams (2002) found that immunohistochemistry staining of the three major tissues collected from infected deer resulted in detection inconsistencies. Deer in the early stages of infection stained positive only in retropharyngeal lymph nodes and tonsil lymph nodes but the obex provided negative results in the same deer. In a different study, 80% of 269 infected deer tested positive based on prion detections in both the retropharyngeal lymph nodes and obex, but retropharyngeal lymph nodes were the only indicators of CWD in the remaining 55 positive deer (Keane et al. 2008). The effectiveness of using retropharyngeal lymph nodes to

Table 1. Summary of surveillance efforts to detect chronic wasting disease (CWD) in white-tailed deer *Odocoileus virginianus* harvested by hunters in the northeastern United States by year, with number of positive cases in parentheses.

Year	CT[a]	DE	MA[a,b]	MD[b]	ME[d]	NJ	NH	NY[c]	OH[a]	PA	RI	VA[a,b]	VT[a]	WV
1997	—	—	—	—	—	502	—	—	—	—	—	—	—	—
1999	—	—	—	—	299	—	—	—	—	—	—	—	—	—
2000	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2001	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2002	—	—	87	304	830	900	259	1,194	500	—	160	1,112	251	—
2003	239	300	301	542	804	51	388	988	500	500	41	32*	297	—
2004	317	300	294	872	747	364	385	551	500	2,003	160	90*	323	—
2005	643	625	577	999	819	505	402	8,164 (2)	737	3,833	183	700	276	996
2006	667	615	464	982	909	537	460	7,907	1,097	4,334	158	899	363	1,336 (1)
2007	623	600	487	983	848	339	405	7,473	941	3,944	180	1,098	407	1,272 (6)
2008	632	487	400	997	791	374	426	2,971	1,021	4,224	196	433	403	1,349 (6)
2009	623	592	489	1,130	699	384	439	2,682	571	4,029	150	286 (1)	410	1,084 (15)
2010	615	582	627	369 (1)	717	392	405	1,792	588	3,882	225	592 (1)	7*	1,054 (10)
2011	565	605	615	305	702	360	431	1,807	n/a	3,766	198	1,588 (2)	8*	1,111 (9)
2012	2*	663	*	278	412	398	384	1,564	*	4,809 (3)	279	333 (1)	8*	658 (16)
Total	4,924 (0)	5,369 (0)	4,341 (0)	7,761 (1)	8,577 (0)	5,106 (0)	4,384 (0)	37,093 (2)	6,455 (0)	35,324 (3)	1,930 (0)	7,041 (5)	2,730 (0)	8,860 (63)

[a] For the states of Connecticut, Massachusetts, Ohio, Vermont, and Virginia, asterisks (*) indicate exclusive use of either a targeted surveillance for deer exhibiting clinical signs of CWD or a protocol of testing road-killed deer.
[b] In addition to harvested deer, the annual surveillance figures for the states of Massachusetts, Maryland, and Virginia included road-killed and targeted individuals.
[c] In 2011, New York changed hunter-harvest surveillance to a point system weighted by sex and age class (Walsh and Miller 2010).
[d] In 2012, Maine changed surveillance protocols in favor of testing a higher number of moose *Alces alces* in the northern region of the state.

detect CWD in nearly all stages of infection is presumably the reason that this tissue was most commonly submitted by state agencies in the Northeast.

The Wisconsin Veterinary Diagnostic Laboratory in Madison, Wisconsin, was the most-used USDA-certified laboratory prior to 2012, and was still used by 29% of states (4 of 14) in the Northeast to analyze CWD samples in 2012. Six states sent CWD samples to laboratories within the Pennsylvania Animal Diagnostic Laboratory System, consisting of the Pennsylvania Veterinary Laboratory in Harrisburg and New Bolton Center Veterinary Laboratory in Kennett Square (Table 2). Location and lower overall costs associated with processing samples were identified as primary reasons for using these Pennsylvania laboratories. Certified laboratories in other states, including the Animal Disease Diagnostic Laboratory in Reynoldsburg, Ohio, and the Colorado State University Diagnostic Laboratory in Fort Collins, Colorado, also were used to analyze samples from the Northeast. The laboratory chosen by each state was not as important as the tissue submitted for detection of CWD, because all states sent samples to laboratories that were certified by the USDA and therefore appropriate for processing CWD samples.

Given the postulated spread of CWD from captive facilities to wild populations that is believed to have occurred in the CWD endemic region of Colorado and Wyoming (Miller et al. 2000), this seemed likely to be the case elsewhere. New York confirmed CWD in two captive herds in March 2005 and later in two wild deer in April 2005. However, investigations into the causes of these cases were inconclusive because of complexities that occurred within the captive herd in which CWD was first detected. Complexities ranged from captive deer escaping the fenced facilities to undocumented transfers between facilities. Surveillance efforts have continued in New York since 2005 but no additional cases have been found in wild or captive herds.

West Virginia had its first case of CWD in a wild, road-killed deer in Hampshire County in 2005. Between 2005 and 2012, testing of harvested deer in West Virginia has yielded 63 CWD-positive cases out of 8,860 test samples (Table 1). All but two of these deer were collected in Hampshire County, with the remaining deer found in bordering Hardy County. Despite the proximity between Hampshire County, West Virginia, and bordering states (such as Virginia, Maryland, and Pennsylvania), CWD was not detected in these states until Virginia confirmed its first case in 2009 in a wild deer that was harvested $<$2 km from the West Virginia state line. Similarly, Maryland's only two cases to date were detected in deer harvested in 2010 and 2013 in Allegany County, just north of the initial Hampshire County outbreak in West Virginia. In October 2012, Pennsylvania's first case of CWD was detected in a captive deer from a farm in Adams County, and CWD was also found in three wild deer that were harvested during the 2012 rifle season. Two of these deer were harvested in Blair County and the other came from Bedford County. In November 2013, Bedford County's second case was found in a road-killed deer. Although it is possible that CWD crossed the Potomac River into these bordering areas, studies examining movements of deer between populations are needed to determine routes of transmission. There is also a difference between

Table 2. Summary of tissues submitted and the United States Department of Agriculture–certified laboratory used by each state in the Northeast for chronic wasting disease (CWD) testing.

State	Tissue(s) submitted			Most recent laboratory used
	Obex	Retropharyngeal lymph nodes	Tonsil	
CT	X		X	Wisconsin Veterinary Diagnostic Laboratory (Madison, WI)
DE	X	X		Pennsylvania Animal Diagnostic Laboratory (Harrisburg, PA)
MA	X	X		Pennsylvania Animal Diagnostic Laboratory (Kennett Square, PA)
MD	X	X		Pennsylvania Animal Diagnostic Laboratory (Harrisburg, PA)
ME		X		Colorado State University Diagnostic Laboratory (Fort Collins, CO)
NH[a]		X		Pennsylvania Animal Diagnostic Laboratory (Kennett Square, PA)
NJ[b]		X		Pennsylvania Animal Diagnostic Laboratory (Kennett Square, PA)
NY[a]		X		New York State Veterinary Diagnostic Laboratory (Ithaca, NY)
OH	X	X		Animal Disease Diagnostic Laboratory (Reynoldsburg, OH)
PA	X	X		Pennsylvania Animal Diagnostic Laboratory (Harrisburg, PA)
RI		X		Wisconsin Veterinary Diagnostic Laboratory (Madison, WI)
VA[b]		X		Wisconsin Veterinary Diagnostic Laboratory (Madison, WI)
VT[a]		X		Wisconsin Veterinary Diagnostic Laboratory (Madison, WI)
WV[b]		X		Animal Disease Diagnostic Laboratory (Reynoldsburg, OH)

[a] Earlier surveillance protocols (e.g., 2002–2010) in these states included submission of the obex and/or tonsils but now retropharyngeal lymph nodes are the only tissues submitted.
[b] The obex is also submitted in captive cervids and/or deer exhibiting signs characteristic of CWD.

CWD not being ''detected'' and CWD not being ''present,'' and the dates of initial detection in each state may not provide an accurate timeline of CWD in the Northeast.

As of 2002, Wisconsin and Illinois were the only states east of the Mississippi River that had confirmed cases of CWD (Saunders et al. 2012). However, CWD has been found in five states in the Northeast during the past decade. New York is the only state that has detected CWD but not redetected it in subsequent sampling efforts. In New York, a containment area 16 km in diameter was established around the 2005 index cases with the following emergency regulations to prevent further spread of the disease: mandatory check stations and testing of all harvested deer within the containment area; bans on deer rehabilitation, movement of intact carcasses, use of deer or elk urine, and possession of deer killed by motor vehicles in the containment area. Requirements were also increased for taxidermist record keeping and reporting. All states dealing with CWD in the Northeast have containment and/or management plans with similar regulations.

Potential spread of CWD into new regions, specifically if expedited by transfer of captive cervids, may be a serious issue for additional states in the future. As of fiscal year 2012, the USDA provided the primary source of funding for CWD surveillance taking place at the state level. However, lack of USDA funding since 2012 has been problematic for states currently dealing with CWD or those at risk to have CWD in populations of wild cervids in the future. The loss of funding provided by the USDA for surveillance suggests that each state's future plans will require state funds or some other source to continue to monitor for CWD. Pennsylvania, West Virginia, and Virginia spend considerable state funding on CWD sampling, but this is not the case with many other states in the Northeast. After years of USDA-funded testing, Colorado documented a 90% reduction in samples submitted for CWD testing in response to a sample fee of US$25 that was imposed on hunters that wanted their deer tested (CDPW 2011). States in the Northeast may alter surveillance efforts with an emphasis on weighted (Walsh and Miller 2010) or targeted surveillance to reduce sample size and cost, and others that are at risk for CWD infection may not continue surveillance without the availability of federal funding.

Weighted sampling efforts appear to be warranted in areas that are at the greatest risk for CWD infection based on associations of CWD with environmental and landscape covariates (Osnas et al. 2009; Walsh and Miller 2010; Walter et al. 2011). For example, a 1% increase in clay particle content increased odds of infection by up to 8.9% in mule deer in Colorado, because clay soil binds the infectious prion that causes CWD, making it bio-available for long durations (Walter et al. 2011). Other studies have documented that the presence of landscape characteristics such as low-lying grasslands that provide high-quality winter habitat, as well as riparian ecosystems, concentrate deer and increased the odds of CWD infection in north-central Colorado and Saskatchewan, Canada, respectively (Farnsworth et al. 2006; Rees et al. 2012). Assessing variables specific to the Northeast, such as forest cover linked to deer dispersal and habitats that concentrate deer during the winter, may provide the information needed to improve surveillance for CWD throughout the Northeast and is currently ongoing (T. S. Evans, The Pennsylvania State University, unpublished data). Furthermore, with new financial constraints,

collaborations among state agencies have been initiated in the Northeast and may improve efficiency of disease surveillance at a regional scale.

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References

Argue CK, Ribble C, Lees VW, McLane J, Balachandran A. 2008. Epidemiology of an outbreak of chronic wasting disease on elk farms in Saskatchewan. Canadian Veterinary Journal 48:1241–1248.

[CDPW] Colorado Division of Parks and Wildlife. 2011. Chronic wasting disease in Colorado: 2010–2011 surveillance update. Colorado Division of Parks and Wildlife. Available: http://cpw.state.co.us/Documents/Hunting/BigGame/CWD/PDF/TestResults/CWDReport2010-2011.pdf (April 2014).

Farnsworth ML, Hoeting JA, Hobbs NT, Miller MW. 2006. Linking chronic wasting disease to mule deer movement scales: a hierarchical Bayesian approach. Ecological Applications 16:1026–1036.

Grear DA, Samuel MD, Langenberg JA, Keane D. 2006. Demographic patterns and harvest vulnerability of chronic wasting disease infected white-tailed deer in Wisconsin. Journal of Wildlife Management 70:546–553.

Joly DO, Ribic CA, Langenberg JA, Beheler K, Batha CA, Dhuey BJ, Rolley RE, Bartelt G, Van Deelen TR, Samuel MD. 2003. Chronic wasting disease in free-ranging Wisconsin white-tailed deer. Emerging Infectious Diseases 9:599–601.

Kahn S, Dube C, Bates L, Balachandran A. 2004. Chronic wasting disease in Canada: part 1. Canadian Veterinary Journal 45:397–404.

Keane DP, Barr DJ, Keller JE, Hall SM, Langenberg JA, Bochsler PN. 2008. Comparison of retropharyngeal lymph node and obex region of the brainstem in detection of chronic wasting disease in white-tailed deer (*Odocoileus virginianus*). Journal of Veterinary Diagnostic Investigation 20:58–60.

Manjerovic MB, Green ML, Mateus-Pinilla N, Novakofski J. 2014. The importance of localized culling in stabilizing chronic wasting disease prevalence in white-tailed deer populations. Preventive Veterinary Medicine 113:139–145.

Miller C. 2003. Hunter perceptions and behaviors related to chronic wasting disease in northern Illinois. Human Dimensions of Wildlife 8:229–230.

Miller MW, Williams ES. 2002. Detection of PrPCWD in mule deer by immunohistochemistry of lymphoid tissues. Veterinary Record 151:610–612.

Miller MW, Williams ES, McCarty CW, Spraker TR, Kreeger TJ, Larsen CT, Thorne ET. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. Journal of Wildlife Diseases 36:676–690.

[NYSDEC] New York State Department of Environmental Conservation. 2013. Surveillance plan for chronic wasting disease in New York State 2013–2014. New York State Department of Environmental Conservation. Available: http://www.dec.ny.gov/docs/wildlife_pdf/cwdsurplan13web.pdf (May 2014).

Osnas EE, Heisey DM, Rolley RE, Samuel MD. 2009. Spatial and temporal patterns of chronic wasting disease: fine-scale mapping of a wildlife epidemic in Wisconsin. Ecological Applications 19:1311–1322.

Rees EE, Merrill EH, Bollinger TK, Hwang YT, Pybus MJ, Coltman DW. 2012. Targeting the detection of chronic wasting disease using the hunter harvest during early phases of an outbreak in Saskatchewan, Canada. Preventive Veterinary Medicine 104:149–159.

Salman MD. 2003. Chronic wasting disease in deer and elk: scientific facts and findings. The Journal of Veterinary Medical Science 65:761–768.

Saunders SE, Bartelt-Hunt SL, Bartz JC. 2012. Occurrence, transmission and zoonotic potential of chronic wasting disease. Emerging Infectious Diseases 18:369–376.

Shelton P, McDonald P. 2012. Illinois chronic wasting disease: 2011–2012 surveillance/management summary. Illinois Department of Natural Resources. Available: http://www.dnr.illinois.gov/programs/cwd/documents/cwdannualreport20112012.pdf (April 2014).

Sohn HJ, Kim JH, Choi KS, Nah JJ, Joo YS, Jean YH, Ahn SW, Kim OK, Kim DY, Balachandran A. 2002. A case of chronic wasting disease in an elk imported to Korea from Canada. The Journal of Veterinary Medical Science/The Japanese Society of Veterinary Science 64:855–858.

Walsh DP, Miller MW. 2010. A weighted surveillance approach for detecting chronic wasting disease foci. Journal of Wildlife Diseases 46:118–135.

Walter WD, Walsh DP, Farnsworth ML, Winkelman DL, Miller MW. 2011. Soil clay content underlies prion infection odds. Nature Communications 2(200):1–6.

[WDNR] Wisconsin Department of Natural Resources. 2009. Prevalence of CWD in the CWD management zone. Wisconsin Department of Natural Resources.

Available: http://dnr.wi.gov/topic/wildlifehabitat/documents/prev.pdf (April 2014).
[WDNR] Wisconsin Department of Natural Resources. 2010. Wisconsin's chronic wasting disease response plan: 2010–2025. Wisconsin Department of Natural Resources. Available: http://datcp.wi.gov/uploads/About/pdf/FinalCWDResponsePlan2010-2025.pdf (April 2014).
Williams ES, Young S. 1980. Chronic wasting disease of captive mule deer: a spongiform encephalopathy. Journal of Wildlife Diseases 16:89–96.

Chapter 2

Home range is related to landscape heterogeneity in deciduous forests

Chapter 2 was written in collaboration with David Stainbrook, Bret Wallingford, Chris Rosenberry, Duane Diefenbach and W. David Walter. I have included this manuscript on the following pages as formatted for the *Journal of Wildlife Management*.

ABSTRACT

Spatial heterogeneity, or composition and configuration of a landscape, plays a role in many biological and ecological processes. In spatial ecology, understanding movements of a species in relation to the landscape can assist wildlife managers in better understanding other processes, including habitat use and disease transmission. In the northeastern United States (Northeast), chronic wasting disease has been detected in populations of white-tailed deer (*Odocoileus virginianus*), and understanding the relationship between landscape and size of home range may provide a basis for disease surveillance and containment efforts. The objectives of our study were to (1) compare size of home range between sexes and among study areas for white-tailed deer occupying a continuum of forested landscapes from highly fragmented to contiguous and (2) investigate relationships between size of home range and measures of landscape composition and configuration. We observed differences in size of 95% home range between males (3.77 km^2) and females (1.83 km^2) across all study areas, as well as between deer in highly fragmented and contiguous landscapes. We developed 20 linear regression models that contained measures of landscape that were correlated with size of home range, and the best model showed that size of home range increased with connectivity of forest cover. Understanding this relationship may provide a foundation for disease surveillance efforts, because size of home range may represent the scale at which disease will spread. Therefore surveillance should be conducted in a manner that reflects connectivity of landscape at a local scale.

Spatial patterns of a landscape play a role in many biological and ecological processes. These patterns, referred to as spatial heterogeneity, are described best by measures of landscape composition and configuration (Li and Reynolds 1994). Composition includes numbers and proportions of distinct patches of land cover, and configuration includes shapes, arrangements, and contrast between patches. Landscapes also can be defined by spatial categories including patch, edge, diversity, contagion, and shape (McGarigal and Marks 1995).

Studies in the western (West) and Midwestern (Midwest) United States identified characteristics of landscapes (e.g., edge density, patch shape) that were related to size of home range for mule deer (*Odocoileus hemionus*; Kie et al. 2002) and white-tailed deer (*Odocoileus virginianus*; Walter et al. 2009). Studies in the northeastern United States (Northeast) have shown that features of the landscape, such as terrain, roads, rivers, and forest cover influenced dispersal behaviors of white-tailed deer (Long et al. 2005, Long et al. 2010). Assessments of spatial heterogeneity and how it relates to size of home range, however, are lacking in the Northeast.

Increased use of Global Positioning System (GPS) technology for tracking wildlife movements has resulted in development of estimators of home range for serially correlated locations. In comparison with traditional estimators that include only location-based parameters (e.g., fixed kernel density estimators), movement-based kernel density estimators take advantage of greater amounts of data available with GPS technology that was not available with transmitters that required locations estimated via triangulation (Horne et al. 2007, Benhamou and Cornelis 2010). Movement-based kernel density estimators (hereafter referred to as MKDE) incorporate serially correlated locations, duration between locations, positional error of GPS technology, and habitat and provide estimates of home range that better account for movements

that relate to the landscape (Benhamou and Cornelis 2010). Applied use of MKDE, however, has not been reported in the literature for cervids in North America.

Understanding the relationship between landscape and movements of white-tailed deer can assist wildlife managers with alleviating issues that include forest regeneration, crop damage, and disease transmission (Alverson et al. 1988, Vecellio et al. 1994, Conner and Miller 2004). The spatial distribution of chronic wasting disease was related to features of the landscape in the West and Midwest (Farnsworth et al. 2006, Walter et al. 2011a, Storm et al. 2013). Movements of deer (e.g., dispersal and home range) also were related to landscape in these regions and the Northeast (Long et al. 2005, Walter et al. 2009, Long et al. 2010). Furthermore, it is likely that the spatial distribution of chronic wasting disease in the Northeast is related to both movements of deer and landscapes that relate to these movements, and identifying the scale at which deer establish home range can provide a basis for disease surveillance and containment. The objectives of our study were to (1) compare size of home range between sexes and among study areas for white-tailed deer occupying a continuum of forested landscapes from highly fragmented to contiguous and (2) investigate relationships between size of home range and measures of landscape composition and configuration.

STUDY AREA

We estimated home range for white-tailed deer in 6 study areas in Pennsylvania, USA (Fig. 1). Each area was classified based on a continuum of forested landscapes ranging from highly fragmented to contiguous (Table 1). The *highly fragmented* area was located in the Gettysburg National Military Park in central Adams County and elevation ranged from 87 m to 236 m. Pasture and cropland were dominant classes of land cover throughout the area, and the town of Gettysburg was located in the center. Forest cover was sparse and highly fragmented in

this area due to the dominant presence of anthropogenically-modified habitats. The 2 *moderately fragmented* areas were located approximately 50 km northeast of Pittsburgh and in the northeastern region of the state, respectively (Table 1). These areas represented rural and moderately fragmented landscapes where open (e.g., pasture, cropland) and forested classes created a mosaic, and elevation in these areas ranged from 225 m to 819 m. The 2 *evenly divided* areas were located in central Pennsylvania and were characterized by contiguous forests along ridgelines and contiguous distributions of pasture and cropland in valleys. Elevation in these areas ranged from 111 m to 737 m. The *contiguous* area was located in the north-central region of the state in homogeneous forests that represented the dominant class of land cover. Although sparse, other classes (e.g., pasture) were present and elevation ranged from 406 m to 785 m (Table 1).

METHODS

Home range estimation.– We captured and equipped 45 white-tailed deer with GPS collars across the study areas for various projects on white-tailed deer movements and survival between 2009 and 2013 (Stainbrook 2011, Buderman et al. 2014, Lutz et al. 2014). We captured deer using a combination of rocket nets (Beringer et al. 1996), single-gate Clover traps (Clover 1956), and drop nets (modified from Ramsey 1968). All capture and handling methods were in accordance with protocols approved by the Pennsylvania State University Institutional Animal Care and Use Committee (IACUC No. 29677 and 34910) and within guidelines of the American Society of Mammalogists (Sikes et al. 2011). We used *adehabitatLT* and *adehabitatHR* packages (Calenge 2011, Calenge 2014) in program R (R Foundation for Statistical Computing, Vienna, Austria) to estimate mean daily distance traveled and home range for each deer, respectively. We

estimated mean distance traveled by each deer across a 24-hour period, provided ≥3 recorded locations were available (range: 3–7 locations; Appendix A).

We incorporated duration of time between recorded locations (1–10 hr), a minimum distance of 30 m that needed to be traveled between consecutive locations to be considered active, and landscape-specific diffusion coefficients into the MKDE to estimate 95% and 99% isopleths of annual home range for each deer. We defined annual as late winter of one year through late winter of the following year with no overlapping dates (e.g., 1 February–31 January), because dates of capture varied for each deer. We grouped deer according to sex and landscape (e.g., *highly fragmented*) and used a single-factor ANOVA with a confidence level of 95% (α=0.05) to assess differences in size of home range between sexes, as well as pairwise t-tests with Bonferroni correction (Rice 1989) to assess differences among all sex-landscape comparisons.

Land cover reclassification.– We reclassified the 2006 National Land Cover Database with 30 m resolution into 5 classes: developed, forested, open, water, and wetland (Fry et al. 2011). Prominent water sources and wetlands were present in only 2 study areas and represented less than 1% of the landscape in home ranges of 5 deer in these areas. Therefore we did not consider these 2 classes in our analysis and reclassified water as open (e.g., pastures, grasslands and croplands) and wetland as forested, given the association of woody wetlands with forest vegetation (Fry et al. 2011). The developed class contained roads and all intensities of development, including urban, suburban, and exurban.

Landscape metrics.– We used the *SDMTools* package to calculate measures of landscape configuration and connectivity for each class of land cover and the *plyr* package to automate the process for each deer within 2 spatial scales (95% and 99% isopleths of home range) in program

R (VanDerWal et al. 2012, Wickham 2014). We calculated 37 measures for each class within each spatial scale and subsequently subset each class and conducted Pearson correlation matrices between our response variable, natural log of home range size, and each measure. We used natural log of home range because the original distribution was positively skewed (Kie et al. 2002, Walter et al. 2009), and we also used a Bonferroni correction to account for Type I error in multiple comparisons (Rice 1989).

We retained measures that were correlated ($r > 0.5$) with natural log of home range but not correlated with each other. Some measures were not correlated with home range for all 3 classes of land cover, however, and we prioritized correlations between the forested class and home range as most important. We believed the forested class would be most influential given the association of forest with movement and disease epidemiology in white-tailed deer in the Northeast and Midwest (Long et al. 2005, Nixon et al. 2007, Kelly et al. 2014). We selected 4 covariates that included *proportion of landscape* (proportion of each class), *patch density* (number of patches/km^2), *edge density* (m/ha), and *patch cohesion index* (measure of physical connectivity for each class). We selected these covariates due to their relationships with definitions of spatial heterogeneity (Li and Reynolds 1994, McGarigal and Marks 1995) and also with size of home range in previous studies of cervids (Kie et al. 2002, Anderson et al. 2005, Walter et al. 2009). We chose covariates that were relative (e.g., edge density) rather than absolute (e.g., total edge) because we were comparing landscapes with varying extents of configuration and connectivity occupied by white-tailed deer.

Linear regression modeling.– We created 20 linear models with combinations of the 4 covariates as independent variables and natural log of home range as the response variable for each spatial scale (Table 2). We used covariates that corresponded to each spatial scale to

determine if changing the scale of analysis from 95% to 99% would influence model selection results (Kie et al. 2002, Walter et al. 2009). Each model contained covariates specific to one class of land cover (e.g., forested), and we used Aikake's Information Criterion with correction for small sample size to evaluate the set of models (AIC_c; Burnham and Anderson 2002). We did not use hypothesis testing because we were interested only in determining which covariates were related to size of home range.

RESULTS

Mean distance traveled by females ranged from 179.5 m in the *highly fragmented* area to 287.1 m in an *evenly divided* area (Appendix A). Similarly, mean distance traveled by males ranged from 222.1 m in the *highly fragmented* area to 445.1 m in an *evenly divided* area. Size of 95% home range for females ranged from 0.40 km^2 in the *highly fragmented* area to 5.96 km^2 in a *moderately fragmented* area (Table 3). Size of 95% home range for males ranged from 1.12 km^2 in the *highly fragmented* area to 8.55 km^2 in an *evenly divided* area.

Males established 95% home ranges (3.77 km^2) that were more than twice the size of home ranges established by females (1.83 km^2) across all study areas ($F_{1,43} = 8.82$, $P < 0.005$). Mean size of 99% home range also varied between males (7.73 km^2) and females (3.37 km^2; $F_{1,43} = 12.53$, $P < 0.001$). We observed differences in 95% home range for females in *highly fragmented* (0.63 km^2) and *contiguous* (2.65 km^2) areas ($P < 0.05$; Fig. 2). Males from *contiguous* (5.61 km^2) and *evenly divided* (5.78 km^2) areas established home ranges that were 3 times greater in size than home ranges for males in the *highly fragmented* area (1.84 km^2; $P < 0.02$).

All 45 deer established home ranges that encompassed forest cover. Developed and open classes of land cover, however, were present in home ranges of only 42 and 37 of these deer,

respectively. Patch density was inversely related to size of home range for all 3 classes (forested: $r = –0.66$, $P < 0.001$; developed: $r = –0.55$, $P < 0.001$; open: $r = –0.68$, $P < 0.001$). Edge density also had an inverse relationship with home range for all classes (forested: $r = –0.54$, $P < 0.001$; developed: $r = –0.57$, $P < 0.001$; open: $r = –0.55$, $P < 0.001$). Patch cohesion index ($r = 0.79$, $P < 0.001$) and proportion of landscape ($r = 0.50$, $P < 0.001$) were correlated with home range for only the forested class, but the positive relationship between patch cohesion index and size of home range appeared to be stronger than any other relationship.

The same model, containing forest edge density and patch cohesion index (physical connectivity of forest), best described the relationship between landscape and size of home range at our 2 spatial scales. This model accounted for 89% and 76% of AIC_c weights at 95% and 99% scales, respectively (Tables 4–5). Forest edge density was inversely related to size of home range with parameter estimates of –0.250 and –0.270 at 95% and 99% scales, respectively (Figs. 3–4). Patch cohesion index was positively related with estimates of 1.341 and 1.618 at these 2 scales, respectively (Figs. 5–6). At each spatial scale, the second best model differed only in that edge density was not included.

DISCUSSION

We expected size of home range to vary between sexes, given associations of expansive home range with males, as well as increased movements by males for dispersal, migration, and breeding (Beier and McCullough 1990, McCoy et al. 2005, Nixon et al. 2007, Long et al. 2008, Long et al. 2010). Expansive home range for males in the Northeast was consistent with findings in the Midwest that identified several factors that influenced differences in size between sexes, including increased nocturnal movement by males during the breeding season, decreased diurnal movement by females during the growing season, and higher site fidelity by females across all

seasons (Beier and McCullough 1990, Walter et al. 2011b). Increased rates and distances of dispersal were observed for males in the Northeast and Midwest and supported our findings of expansive home range and greater daily distance traveled by males than females in each study area in which both sexes were included for analysis (Nixon et al. 2007, Long et al. 2008).

Home ranges for males and females in the *highly fragmented* area were less expansive than home ranges in all other study areas. Kie et al. (2002) observed a negative relationship between areas of high fragmentation and size of home range for female mule deer. It is possible that home ranges that were less expansive in the *highly fragmented* area were related to the landscape comprised of small woodlots and high intensities of open and developed classes that may provide forage for white-tailed deer (Vecellio et al. 1994, Grund et al. 2002). Our *highly fragmented* area more closely resembled an agricultural landscape than any other study area, and size of home range for females in this area (0.63 km^2) was more comparable to home ranges reported in agricultural landscapes of the Midwest (0.99–1.47 km^2; Walter et al. 2009) than to any of our other study areas (Fig. 2). In areas where open and developed classes were as prominent as they were in the *highly fragmented* area, deer likely were able to obtain suitable forage without being required to traverse a large distance (Nixon et al. 1991).

Deer located in contiguously forested areas established home ranges that were largest of all deer in our analysis. Areas that are predominantly forested may be viewed as less productive, because deer that rely on food sources (e.g., mast and browse) that can vary on a seasonal or annual basis may be required to establish home ranges that are expansive to ensure access to sufficient resources (Alverson et al. 1988, McShea and Schwede 1993, Rooney and Waller 2003). In areas that are predominantly forested, mast can comprise $\geq$76% of the diet of deer in these areas (Harlow et al. 1975). Female deer in Virginia expanded seasonal home ranges into

oak (*Quercus* spp.) stands during years in which production of mast exceeded 300 kg/ha, whereas size of home range remained unchanged during years of poor mast production (<100 kg/ha; McShea and Schwede 1993). Production in this area varied greatly from year to year (e.g., 396 kg/ha to 3 kg/ha) and suggested that shifts in home range by deer in predominantly forested landscapes are in response to availability of forage.

In the *highly fragmented* area, changing the spatial scale from 95% to 99% encompassed greater amounts of edge and other patches that were occupied less often by deer. Conversely, changing the scale in the *contiguous* area encompassed only greater amounts of forest rather than other classes of land cover given the homogeneously forested landscape in this area. Our model selection results, however, remained the same without regard to the scale that was used. This was inconsistent with findings in female mule deer in the West, where measures of landscape that influenced size of home range changed as the scale of analysis was increased from 250 m to 2,000 m from the centroid location of each home range (Kie et al. 2002). In a similar study of female white-tailed deer in the Midwest, measures of landscape that influenced size of home range changed as the spatial scale was modified (Walter et al. 2009). Although changing spatial scale yielded similar results in our study, further research using other extents (e.g., buffered circles around locations; Kie et al. 2002) would be necessary to further evaluate influence of spatial scale in the Northeast.

A caveat of our study was that we were unable to assess deer densities across our study areas. High deer densities in our *highly fragmented* area (>40 deer/km^2; Stainbrook 2011) were linked to crop damage (Vecellio et al. 1994), and similar densities (47–51 deer/km^2) were identified as the cause of forest regeneration issues on a predominantly forested landscape in Connecticut (Kilpatrick et al. 1997). Deer in high-density areas also have been shown to exhibit

greater site fidelity, especially during winter months (Lesage et al. 2000). Reduction in deer density appears to have varying effects on expansion of home range. Seasonal home range expanded by 30% in a developed area in South Carolina following a 50% reduction in herd size (Henderson et al. 2000), however, home ranges remained unchanged in Connecticut following reduction of the deer herd from 88 to 17 deer/km^2 (Kilpatrick et al. 2001). Although assessments of home range expansion in response to herd reduction are not feasible in Pennsylvania, lower densities (~18 deer/km^2; Tilghman 1989) in contiguously forested areas in the state may require males to traverse greater distances in search of females during the breeding season. Therefore landscape is one of many factors that likely play a role in differences in size of home range between highly fragmented and contiguously forested landscapes.

Epidemiology of disease is likely to vary in areas with differing landscapes and deer densities, because rates of contact also differ in these areas (Schauber et al. 2007, Kelly et al. 2014). In fragmented areas with high deer densities, contact rates are likely higher at a local scale due to frequent interactions among social groups and therefore may limit spread of disease to a local scale. Increased dispersal rates and distances among juvenile males in these areas, however, also may influence outward expansion of disease into previously unaffected areas (Long et al. 2005). In contiguously forested areas, spread of disease may reflect expansive home ranges that influence contact rates or deposition of infectious disease agents at a broad scale. Interactions between deer in these areas may be lower, however, due to low densities and greater isolation of philopatric groups (Kelly et al. 2014).

Spread of disease likely is related to the scale at which deer establish home range, and our findings show that size of home range varies in fragmented and contiguously forested landscapes that also likely contain deer densities that differ. State agencies that are responsible

for disease surveillance should sample deer at a scale that reflects size of home range established by deer in each type of landscape, because size of home range likely best represents the scale at which disease will spread. Surveillance efforts in highly fragmented areas should be concentrated locally to reflect concentrated movements and home ranges that lead to higher contact rates between social groups that likely utilize similar resources. Conversely, surveillance in contiguously forested areas should be conducted at a broader scale to reflect home ranges that are more expansive and dispersed due to limitations in foraging and breeding opportunities at the local scale. Most states in the Northeast are characterized by a continuum of fragmented and contiguous landscapes, thus the scale of disease surveillance should account for variability in landscape that also likely relates to the scale at which deer may transmit disease.

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REFERENCES

Alverson, W. S., D. M. Waller, and S. L. Solheim. 1988. Forests Too Deer: Edge Effects in Northern Wisconsin. Conservation Biology 2:348-358.

Anderson, D. P., J. D. Forester, M. G. Turner, J. L. Frair, E. H. Merrill, D. Fortin, J. S. Mao, and M. S. Boyce. 2005. Factors influencing female home range sizes in elk (*Cervus elaphus*) in North American landscapes. Landscape Ecology 20: 257-271.

Beier, P., and D. R. McCullough. 1990. Factors influencing white-tailed deer activity patterns and habitat use. Wildlife Monographs 109:1-51.

Benhamou, S., and D. Cornelis. 2010. Incorporating movement behavior and barriers to improve kernel home range space use estimates. Journal of Wildlife Management 74:1353-1360.

Beringer, J., L. P. Hansen, W. Wilding, J. Fischer, and S. L. Sheriff. 1996. Factors affecting capture myopathy in white-tailed deer. Journal of Wildlife Management 60:373-380.

Bowyer, R. T., K. M. Stewart, S. A. Wolfe, G. M. Blundell, K. L. Lehmkuhl, P. J. Joy, T. J. McDonough, and J. G. Kie. 2002. Assessing sexual segregation in deer. Journal of Wildlife Management 66:536-544.

Buderman, F. E., D. R. Diefenbach, C. S. Rosenberry, B. D. Wallingford, and E. S. Long. 2014. Effect of hunter selectivity on harvest rates of radio-collared white-tailed deer in Pennsylvania. The Journal of Wildlife Management 78:1456-1465.

Burnham, K. P., and D. R. Anderson. 2002. Model selection and multimodel inference: a practical information-theoretic approach. Volume 2nd.Springer-Verlag, New York.

Calenge, C. 2011. adehabitatHR: Home range Estimation. R package (Version 0.4.11).

Calenge, C. 2014. adehabitatLT: Analysis of animal movements. R package (Version 0.3.16).

Clover, M. R. 1956. Single-gate deer trap. California Fish and Game 42:199-201.

Conner, M. M., and M. W. Miller. 2004. Movement patterns and spatial epidemiology of a prion disease in mule deer population units. Ecological Applications 14:1870-1881.

Farnsworth, M. L., J. A. Hoeting, N. T. Hobbs, and M. W. Miller. 2006. Linking chronic wasting disease to mule deer movement scales: a hierarchical bayesian approach. Ecological Applications 16:1026-1036.

Fry, J., G. Xian, S. Jin, J. Dewitz, C. Homer, L. Yang, C. Barnes, N. Herold, and J. Wickham. 2011. Completion of the 2006 National Land Cover Database for the Conterminous United States. Photogrammetric Engineering & Remote Sensing 77:858–864.

Grund, M. D., J. B. McAninch, and E. P. Wiggers. 2002. Seasonal movements and habitat use of female white-tailed deer associated with an urban park. Journal of Wildlife Management 66:123-130.

Harlow, R. F., J. B. Whelan, H. S. Crawford, and J. E. Skeen. 1975. Deer foods during years of oak mast abundance and scarcity. The Journal of Wildlife Management 39:330-336.

Henderson, D. W., R. J. Warren, J. A. Cromwell, and R. J. Hamilton. 2000. Responses of urban deer to a 50% reduction in local herd density. Wildlife Society Bulletin 28:902-910.

Horne, J. S., E. O. Garton, S. M. Krone, and J. S. Lewis. 2007. Analyzing animal movements using Brownian bridges. Ecology 88:2354-2363.

Kelly, A. C., N. E. Mateus-Pinilla, W. Brown, M. O. Ruiz, M. R. Douglas, M. E. Douglas, P. Shelton, T. Beissel, and J. Novakofski. 2014. Genetic assessment of environmental features that influence deer dispersal: implications for prion-infected populations. Population Ecology 56:327-340.

Kie, J. G., R. T. Bowyer, M. C. Nicholson, B. B. Boroski, and E. R. Loft. 2002. Landscape heterogeneity at differing scales: effects on spatial distribution of mule deer. Ecology 83:530-544.

Kilpatrick, H. J., S. M. Spohr, and G. G. Chasko. 1997. A controlled deer hunt on a state-owned coastal reserve in Connecticut: controversies, strategies, and results. Wildlife Society Bulletin 25:451-456.

Kilpatrick, H. J., S. M. Spohr, and K. K. Lima. 2001. Effects of population reduction on home ranges of female white-tailed deer at high densities. Canadian Journal of Zoology 79:949-954.

Lesage, L., M. Crete, J. Huot, A. Dumont, and J. Ouellet. 2000. Seasonal home range size and philopatry in two northern white-tailed deer populations. Canadian Journal of Zoology 78:1930-1940.

Li, H., and J. F. Reynolds. 1994. A simulation experiment to quantify spatial heterogeneity in categorical maps. Ecology 75:2446-2455.

Long, E. S., D. R. Diefenbach, C. S. Rosenberry, and B. D. Wallingford. 2008. Multiple proximal and ultimate causes of natal dispersal in male white-tailed deer. Behavioral Ecology 19:1235-1242.

Long, E. S., D. R. Diefenbach, C. S. Rosenberry, B. D. Wallingford, and M. D. Grund. 2005. Forest cover influences dispersal distance of white-tailed deer. Journal of Mammalogy 86:623-629.

Long, E. S., D. R. Diefenbach, B. D. Wallingford, and C. S. Rosenberry. 2010. Influence of roads, rivers, and mountains on natal dispersal of white-tailed deer. Journal of Wildlife Management 74:1242-1249.

Lutz, C. L., D. R. Diefenbach, and C. S. Rosenberry. 2014. Population density influences dispersal in female white-tailed deer. Journal of Mammalogy: *In press*.

McCoy, J. E., D. G. Hewitt, and F. C. Bryant. 2005. Dispersal by yearling male white-tailed deer and implications for management. Journal of Wildlife Management 69:366-376.

McGarigal, K., and B. J. Marks. 1995. General Technical Report PNW-351, U.S. Forest Service, Corvallis.

McShea, W. J., and G. Schwede. 1993. Variable acorn crops: responses of white-tailed deer and other mast consumers. Journal of Mammalogy 74:999-1006.

Nixon, C. M., L. P. Hansen, P. A. Brewer, and J. E. Chelsvig. 1991. Ecology of white-tailed deer in an intensively farmed region of Illinois. Wildlife Monographs 118:1-77.

Nixon, C. M., P. C. Mankin, D. R. Etter, L. P. Hansen, P. A. Brewer, J. E. Chelsvig, T. L. Esker, and J. B. Sullivan. 2007. White-tailed deer dispersal behavior in an agricultural environment. American Midland Naturalist 157:212-220.

Ramsey, C. W. 1968. A drop-net deer trap. Journal of Wildlife Management 32:187-190.

Rice, W. R. 1989. Analyzing tables of statistical tests. Evolution 43: 223-225.

Rooney, T. P., and D. M. Waller. 2003. Direct and indirect effects of white-tailed deer in forest ecosystems. Forest Ecology and Management 181:165-176.

Schauber, E. M., D. J. Storm, and C. K. Nielsen. 2007. Effects of joint space use and group membership on contact rates among white-tailed deer. Journal of Wildlife Management 71:155-163.

Sikes, R. S., W. L. Gannon, and The Animal Care and Use Committee of the American Society of Mammalogists. 2011. Guidelines of the American Society of Mammalogists for the use of wild mammals in research. Journal of Mammalogy 92:235-253.

Stainbrook, D. P. 2011. Methods of estimating white-tailed deer abundance at Gettysburg National Military Park: Testing Assumptions of Distance Sampling. Thesis, Pennsylvania State University, University Park, PA, USA.

Storm, D. J., M. D. Samuel, R. E. Rolley, P. Shelton, N. S. Keuler, B. J. Richards, and T. R. Van Deelen. 2013. Deer density and disease prevalence influence transmission of chronic wasting disease in white-tailed deer. Ecosphere 4:art10.

Tilghman, N. G. 1989. Impacts of White-Tailed Deer on Forest Regeneration in Northwestern Pennsylvania. The Journal of Wildlife Management 53:524-532.

VanDerWal, J., L. Falconi, S. Januchowski, L. Shoo, and C. Storlie. 2012. SDMTools: Species Distribution Modelling Tools: Tools for processing data associated with species distribution modelling exercises. R package (Version 1.1–13).

Vecellio, G. M., R. H. Yahner, and G. L. Storm. 1994. Crop damage by deer at Gettysburg park. Wildlife Society Bulletin 22:89-93.

Walter, W. D., J. Beringer, L. P. Hansen, J. W. Fischer, J. J. Millspaugh, and K. C. VerCauteren. 2011b. Factors affecting space use overlap by white-tailed deer in an urban landscape. International Journal of Geographical Information Science 25:379-392.

Walter, W. D., K. C. VerCauteren, H. Campa, III, W. R. Clark, J. W. Fischer, S. E. Hygnstrom, N. E. Mathews, C. K. Nielsen, E. M. Schauber, T. R. Van Deelen, and S. R. Winterstein. 2009. Regional assessment on influence of landscape configuration and connectivity on range size of white-tailed deer. Landscape Ecology 24:1405-1420.

Walter, W. D., D. P. Walsh, M. L. Farnsworth, D. L. Winkelman, and M. W. Miller. 2011a. Soil clay content underlies prion infection odds. Nature Communications 2:1-6.

Wickham, H. 2014. plyr: Tools for splitting, applying and combining data. R package (Version 1.8.1).

FIGURE LEGENDS

Figure 1. Study areas used to assess the relationship between heterogeneity of landscape and size of home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania.

Figure 2. Mean size of 95% home range by sex and study area for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania.

Figure 3. Relationship between forest edge density and size of 95% home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. Males (M) are denoted above each corresponding symbol.

Figure 4. Relationship between forest edge density and size of 99% home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. Males (M) are denoted above each corresponding symbol.

Figure 5. Relationship between patch cohesion index, or physical connectivity of forest cover, and size of 95% home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. Males (M) are denoted above each corresponding symbol.

Figure 6. Relationship between patch cohesion index, or physical connectivity of forest cover, and size of 99% home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. Males (M) are denoted above each corresponding symbol.

Table 1. Forest type, physiographic province[1], and proportions of 3 classes of land cover summarized within the extent of each study area used in analysis of home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania.

Study Area	Forest type	Physiographic province	Developed	Forested	Open
Highly Fragmented	Appalachian oak	Gettysburg-Newark Lowland	0.162	0.221	0.617
Moderately Fragmented	Appalachian oak	Pittsburgh Low Plateau	0.100	0.623	0.277
Moderately Fragmented	Northern hardwoods	Glaciated Low Plateau	0.037	0.717	0.246
Evenly Divided	Appalachian oak	Appalachian Mountain	0.064	0.723	0.213
Evenly Divided	Appalachian oak	Appalachian Mountain	0.073	0.676	0.251
Contiguous	Northern hardwoods	Deep Valleys	0.018	0.893	0.089

[1] Bureau of Topographic and Geologic Survey, Commonwealth of Pennsylvania Department of Conservation and Natural Resources.

Table 2. Candidate set of models investigating the relationship between heterogeneity of landscape and size of home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. The first term in some models refers to proportion of landscape (e.g., *FOR* = proportion of forested). Subscripts for forested (F), open (O), and developed (D) classes refer to covariates pertaining to that class of land cover, including *PCOH* (patch cohesion index), *EDGE* (edge density), and *PDEN* (patch density).

Covariates	Explanation
FOR + --------- + $EDGE_F$ + --------	Forest model with patch cohesion index and patch density removed
------- + $PCOH_F$ + $EDGE_F$ + --------	Forest model with proportion of forest and patch density removed
------- + --------- + $EDGE_F$ + $PDEN_F$	Forest model with proportion of forest and patch cohesion index removed
FOR + --------- + ---------- + --------	Proportion of forest retained
------- + $PCOH_F$ + --------- + --------	Forest patch cohesion index retained
------- + --------- + $EDGE_F$ + --------	Forest edge density retained
------- + --------- + --------- + $PDEN_F$	Forest patch density retained
OPEN + -------- + --------- + $PDEN_O$	Open model with patch cohesion index and edge density removed
------- + $PCOH_O$ + --------- + $PDEN_O$	Open model with proportion of open and edge density removed
------- + --------- + $EDGE_O$ + $PDEN_O$	Open model with proportion of open and patch cohesion index removed
------- + $PCOH_O$ + --------- + ---------	Open patch cohesion index retained
------- + --------- + $EDGE_O$ + ---------	Open edge density retained
------- + --------- + -------- + $PDEN_O$	Open patch density retained
DEV + $PCOH_D$ + ------- + $PDEN_D$	Developed model with edge density removed
DEV + $PCOH_D$ + -------- + ---------	Developed model with edge density and patch density removed
DEV + ---------- + -------- + $PDEN_D$	Developed model with patch cohesion index and edge density removed
------- + --------- + $EDGE_D$ + $PDEN_D$	Developed model with proportion of developed and patch cohesion index removed
DEV + ---------- + --------- + ---------	Proportion of developed retained
------- + ---------- + $EDGE_D$ + --------	Developed edge density retained
------- + ---------- + -------- + $PDEN_D$	Developed patch density retained

Table 3. Number of locations and size of 95% and 99% home range (km^2) for female (F) and male (M) white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania.

Study area	Sex	Locations	95%	99%
Highly Fragmented	F	1,784	0.701	1.071
Highly Fragmented	F	987	0.414	0.679
Highly Fragmented	F	1,031	0.922	1.671
Highly Fragmented	F	833	0.568	0.887
Highly Fragmented	F	872	0.546	0.872
Highly Fragmented	F	983	0.500	0.812
Highly Fragmented	F	994	0.398	0.536
Highly Fragmented	F	831	0.610	1.001
Highly Fragmented	F	847	0.430	0.713
Highly Fragmented	F	928	0.584	0.810
Highly Fragmented	F	1,966	1.226	2.358
Highly Fragmented	M	1,622	1.524	3.067
Highly Fragmented	M	1,016	2.621	3.951
Highly Fragmented	M	1,727	1.119	2.615
Highly Fragmented	M	1,796	2.086	8.005
Moderately Fragmented	F	3,320	1.154	2.167
Moderately Fragmented	F	3,032	1.231	2.315
Moderately Fragmented	F	3,367	5.963	11.087
Moderately Fragmented	F	3,029	4.396	7.858
Moderately Fragmented	F	3,341	1.249	2.139
Moderately Fragmented	F	3,270	0.997	1.578
Evenly Divided	F	1,484	1.495	2.092
Evenly Divided	F	1,752	1.672	3.216
Evenly Divided	F	1,732	1.858	4.670
Evenly Divided	F	1,665	1.560	2.174
Evenly Divided	F	1,710	3.035	4.603
Evenly Divided	F	1,728	2.871	4.371
Evenly Divided	M	1,688	8.552	14.034
Evenly Divided	M	1,653	3.010	5.921
Evenly Divided	F	3,238	1.812	5.738
Evenly Divided	F	2,824	1.480	2.253
Evenly Divided	F	2,336	0.874	1.559
Evenly Divided	F	3,161	0.707	1.188
Evenly Divided	F	3,422	4.786	8.527
Evenly Divided	F	3,405	2.450	5.471
Contiguous	F	1,737	3.637	5.748
Contiguous	F	1,743	2.969	6.557
Contiguous	F	1,714	1.512	2.324

Contiguous	F	1,702	2.728	3.840
Contiguous	F	1,565	1.193	2.642
Contiguous	F	1,686	2.355	6.399
Contiguous	F	1,699	1.716	3.164
Contiguous	F	1,613	5.086	9.624
Contiguous	M	1,502	4.950	9.248
Contiguous	M	1,721	6.269	15.014

Table 4. Model selection results for the candidate set of models investigating the relationship between heterogeneity of landscape and size of 95% home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. The first term in some models refers to proportion of landscape (e.g., *FOR* = proportion of forested). Subscripts for forested (F), open (O), and developed (D) classes refer to covariates pertaining to that class, including *PCOH* (patch cohesion index), *EDGE* (edge density), and *PDEN* (patch density).

Model Terms	*K*	AIC_c	ΔAIC_c	Weight
------- + $PCOH_F$ + $EDGE_F$ + --------	4	64.295	0.000	0.886
------- + $PCOH_F$ + --------- + --------	3	68.534	4.240	0.106
------- + $PCOH_O$ + --------- + $PDEN_O$	4	75.018	10.724	0.004
------- + ---------- + --------- + $PDEN_O$	3	76.725	12.430	0.002
------- + ---------- + $EDGE_O$ + $PDEN_O$	4	78.128	13.834	0.001
OPEN + ---------- + ---------- + $PDEN_O$	4	78.851	14.557	0.001
------- + --------- + $EDGE_O$ + ---------	3	85.923	21.629	0.000
------- + --------- + --------- + $PDEN_F$	3	87.712	23.417	0.000
------- + --------- + $EDGE_F$ + $PDEN_F$	4	89.138	24.844	0.000
------- + --------- + $EDGE_D$ + $PDEN_D$	4	89.367	25.072	0.000
DEV + ---------- + --------- + $PDEN_D$	4	91.277	26.982	0.000
------- + --------- + $EDGE_D$ + --------	3	91.714	27.419	0.000
------- + --------- + --------- + $PDEN_D$	3	92.599	28.305	0.000
DEV + $PCOH_D$ + --------- + $PDEN_D$	5	92.986	28.691	0.000
FOR + --------- + $EDGE_F$ + --------	4	93.996	29.702	0.000
------- + $PCOH_O$ + --------- + ---------	3	94.676	30.381	0.000
DEV + --------- + --------- + ---------	3	97.513	33.218	0.000
------- + --------- + $EDGE_F$ + --------	3	97.618	33.323	0.000
DEV + $PCOH_D$ + --------- + ---------	4	98.147	33.853	0.000
FOR + --------- + ---------- + --------	3	100.229	35.934	0.000

Table 5. Model selection results for the candidate set of models investigating the relationship between heterogeneity of landscape and size of 99% home range for white-tailed deer (*Odocoileus virginianus*) between 2009 and 2013 in Pennsylvania. The first term in some models refers to proportion of landscape (e.g., *FOR* = proportion of forested). Subscripts for forested (F), open (O), and developed (D) classes refer to covariates pertaining to that class, including *PCOH* (patch cohesion index), *EDGE* (edge density), and *PDEN* (patch density).

Model Terms	*K*	AIC_c	ΔAIC_c	Weight
------- + $PCOH_F$ + $EDGE_F$ + ----------	4	81.469	0.000	0.763
------- + $PCOH_F$ + --------- + ----------	3	83.822	2.353	0.235
------- + ---------- + --------- + $PDEN_O$	3	95.779	14.310	0.001
------- + ---------- + $EDGE_O$ + $PDEN_O$	4	96.079	14.609	0.001
OPEN + --------- + --------- + $PDEN_O$	4	96.916	15.447	0.000
-------- + $PCOH_O$ + --------- + $PDEN_O$	4	97.339	15.870	0.000
-------- + --------- + $EDGE_O$ + ----------	3	98.087	16.617	0.000
-------- + --------- + $EDGE_D$ + ----------	3	105.068	23.598	0.000
-------- + -------- + $EDGE_F$ + $PDEN_F$	4	106.252	24.783	0.000
-------- + -------- + ---------- + $PDEN_F$	3	106.632	25.163	0.000
-------- + -------- + $EDGE_D$ + $PDEN_D$	4	107.477	26.008	0.000
DEV + $PCOH_D$ + ---------- + ----------	4	107.715	26.246	0.000
FOR + --------- + $EDGE_F$ + ----------	4	108.839	27.370	0.000
DEV + --------- + ---------- + ----------	3	109.942	28.473	0.000
-------- + -------- + $EDGE_F$ + ----------	3	109.990	28.521	0.000
DEV + $PCOH_D$ + --------- + $PDEN_D$	5	110.018	28.549	0.000
-------- + $PCOH_O$ + --------- + ----------	3	111.441	29.972	0.000
DEV + ---------- + --------- + $PDEN_D$	4	111.864	30.395	0.000
-------- + ---------- + -------- + $PDEN_D$	3	111.994	30.525	0.000
FOR + ---------- + --------- + ----------	3	112.136	30.667	0.000

Figure 1.

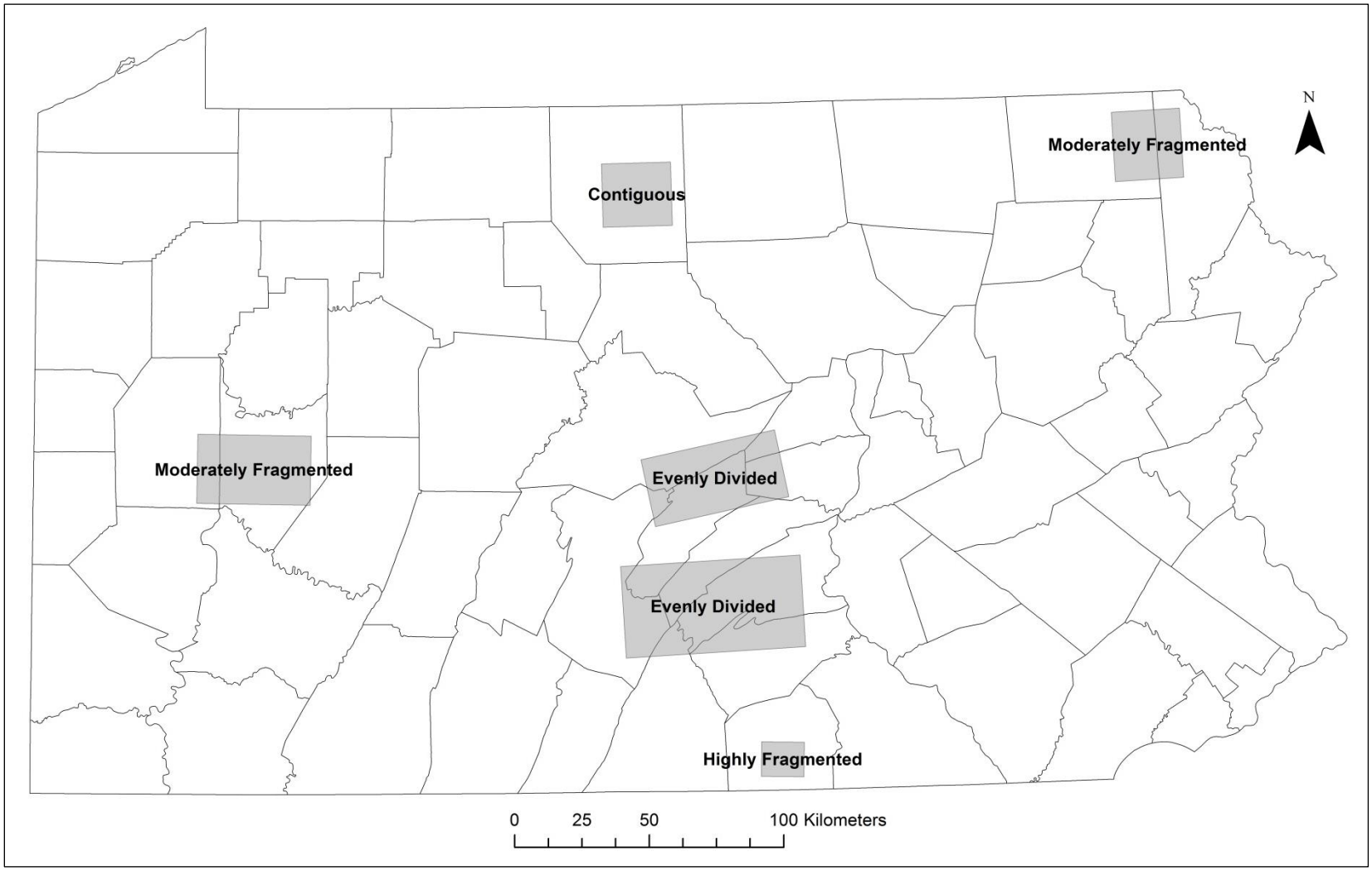

N
Moderately Fragmented
Contiguous
Moderately Fragmented
Evenly Divided
Evenly Divided
Highly Fragmented
0
25
50
100 Kilometers

Figure 2.

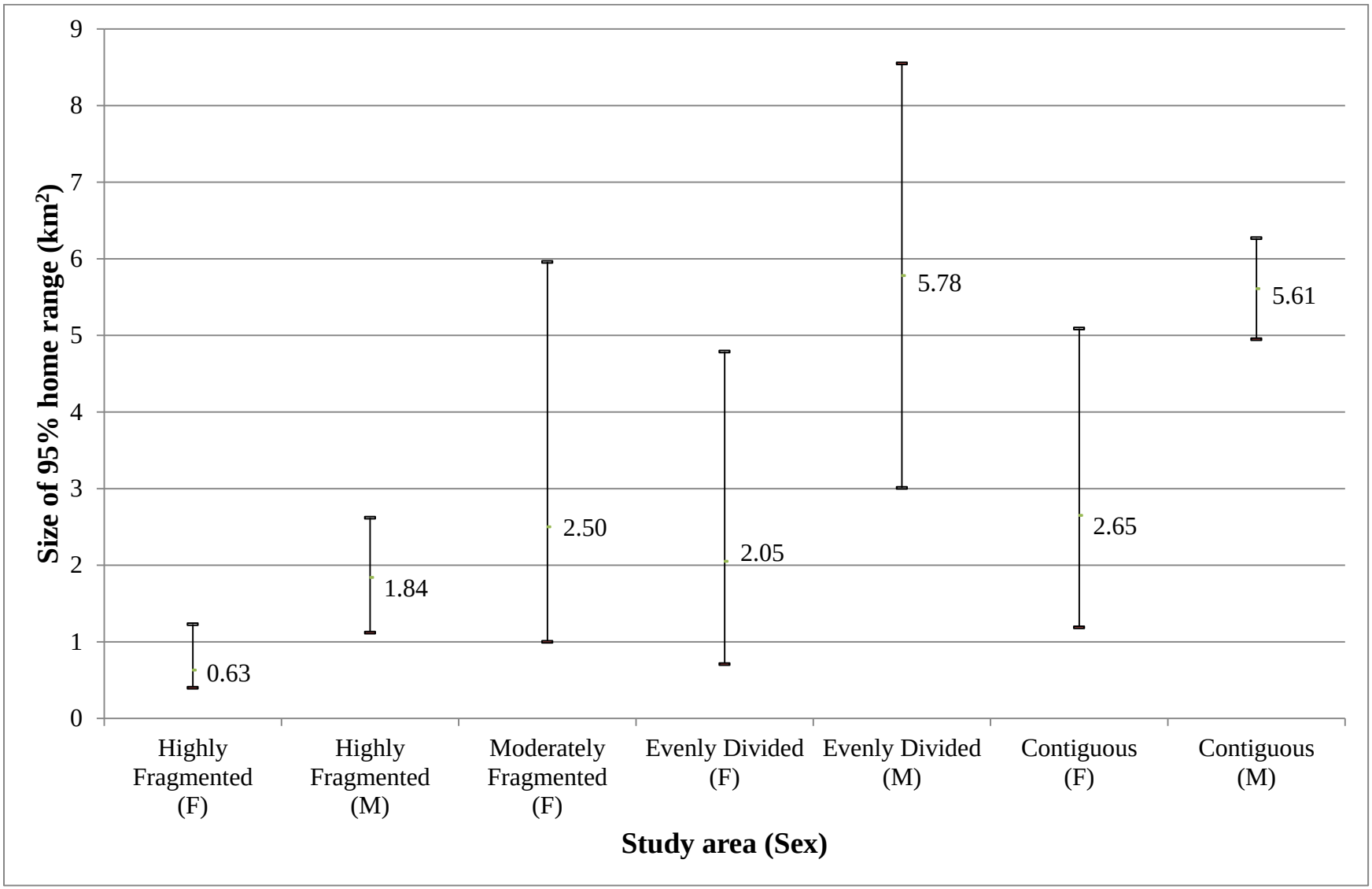

9
8
7
6
5
4
3
2
1
0
Size of 95% home range (km²)
0.63
1.84
2.50
2.05
5.78
2.65
5.61
Highly Fragmented (F)
Highly Fragmented (M)
Moderately Fragmented (F)
Evenly Divided (F)
Evenly Divided (M)
Contiguous (F)
Contiguous (M)
Study area (Sex)

Figure 3.

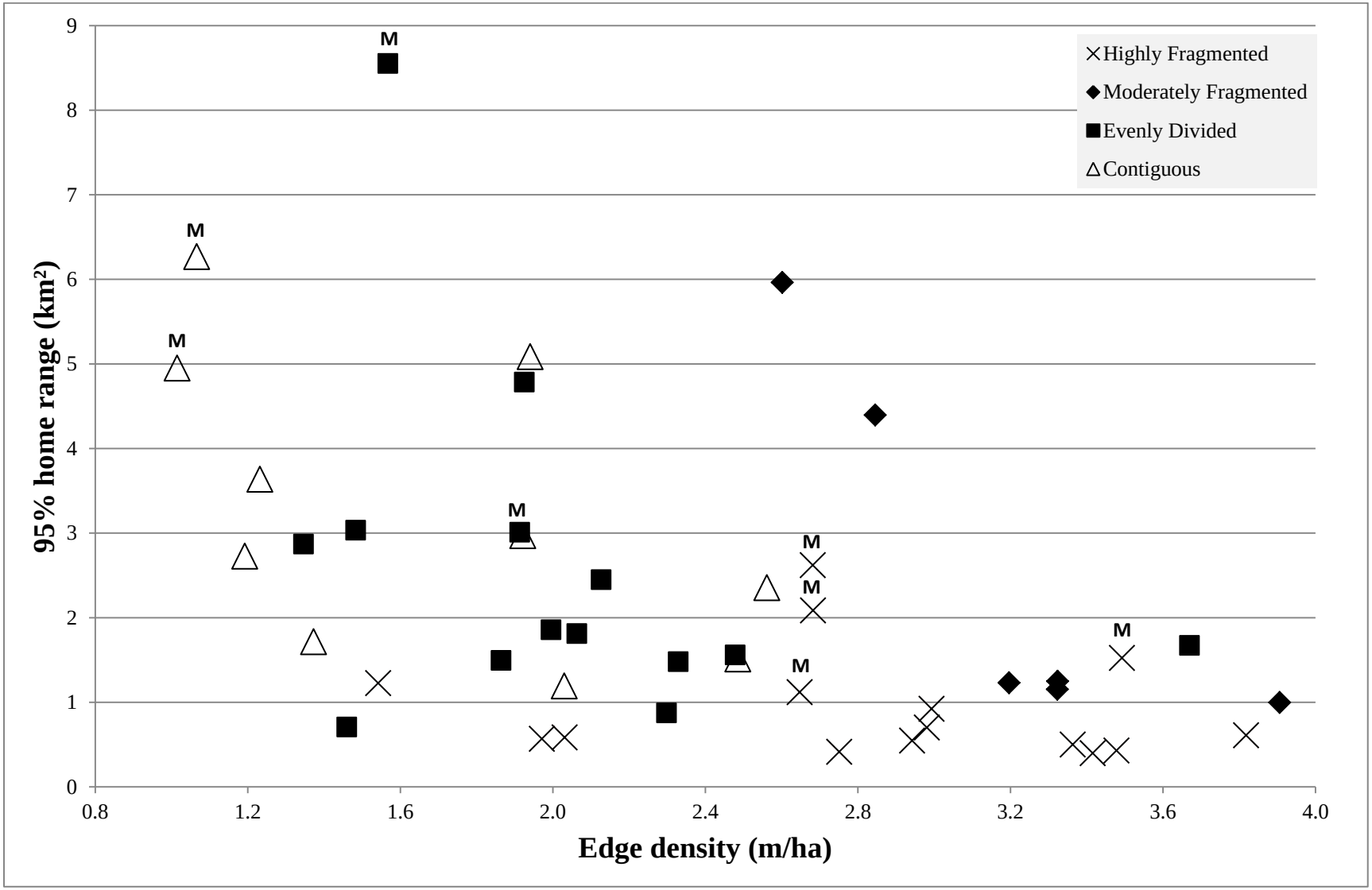

Highly Fragmented
Moderately Fragmented
Evenly Divided
Contiguous
95% home range (km²)
Edge density (m/ha)
0
1
2
3
4
5
6
7
8
9
0.8
1.2
1.6
2.0
2.4
2.8
3.2
3.6
4.0
M

Figure 4.

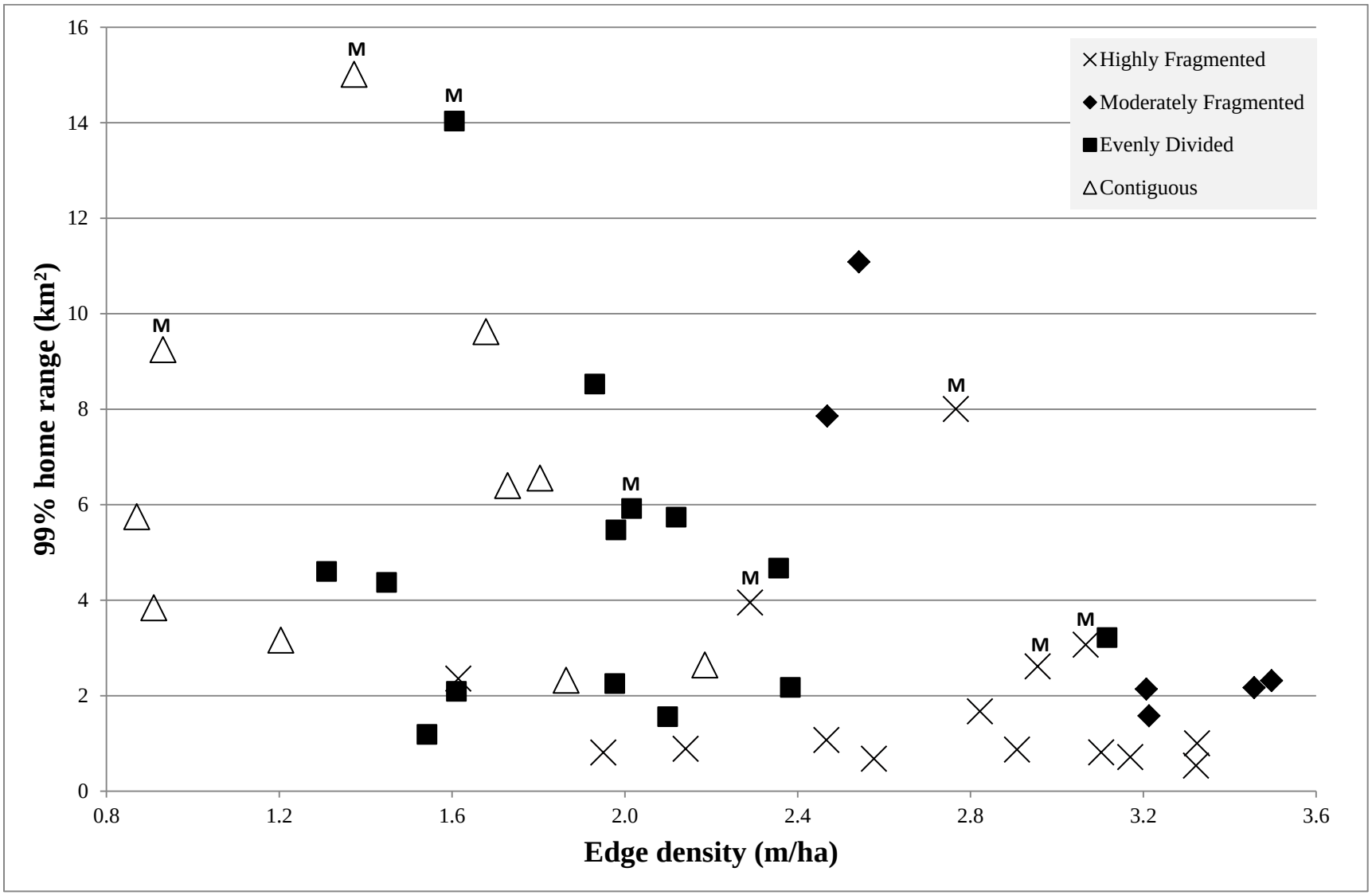

Highly Fragmented
Moderately Fragmented
Evenly Divided
Contiguous
99% home range (km²)
Edge density (m/ha)
0
2
4
6
8
10
12
14
16
0.8
1.2
1.6
2.0
2.4
2.8
3.2
3.6
M

Figure 5.

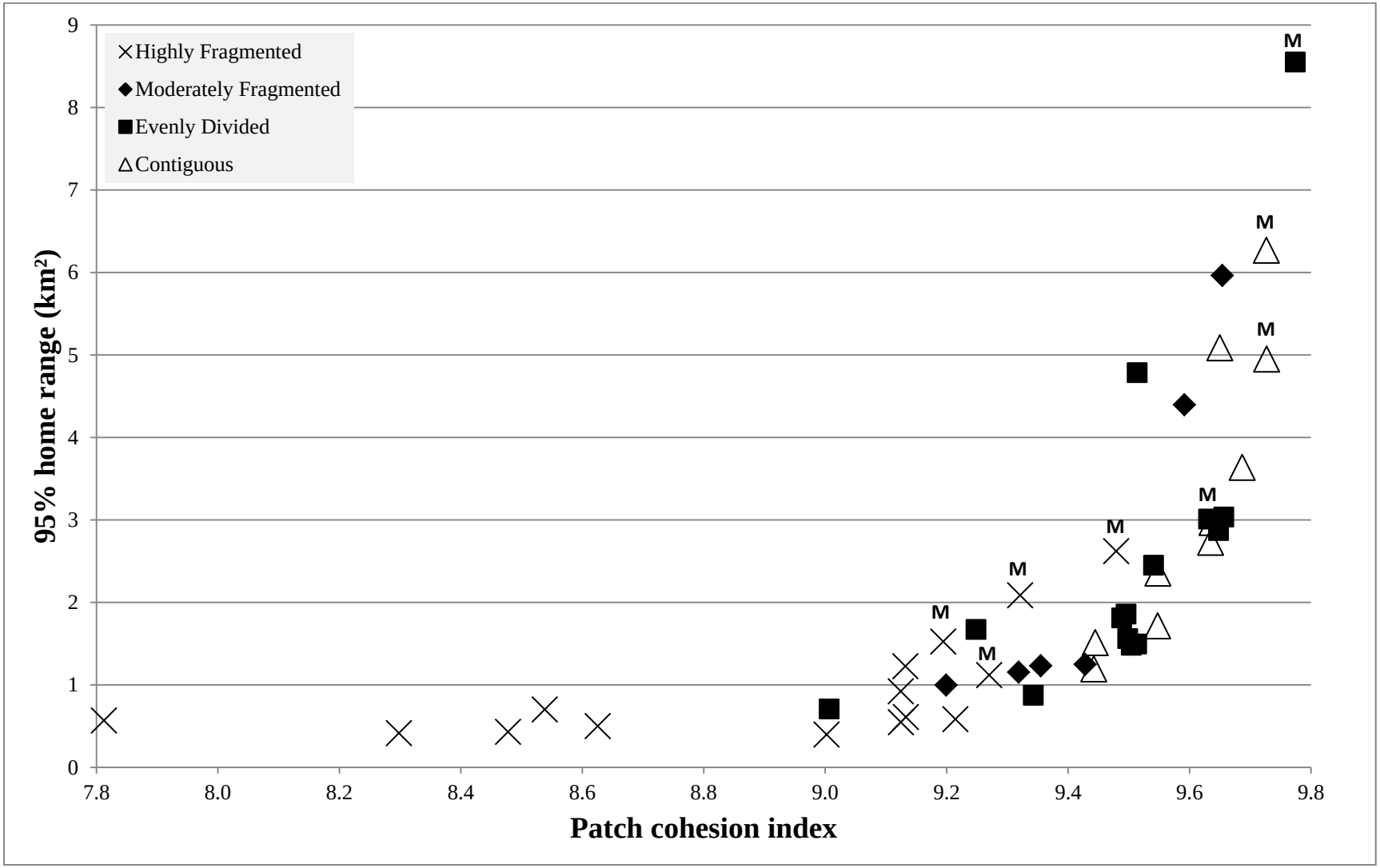
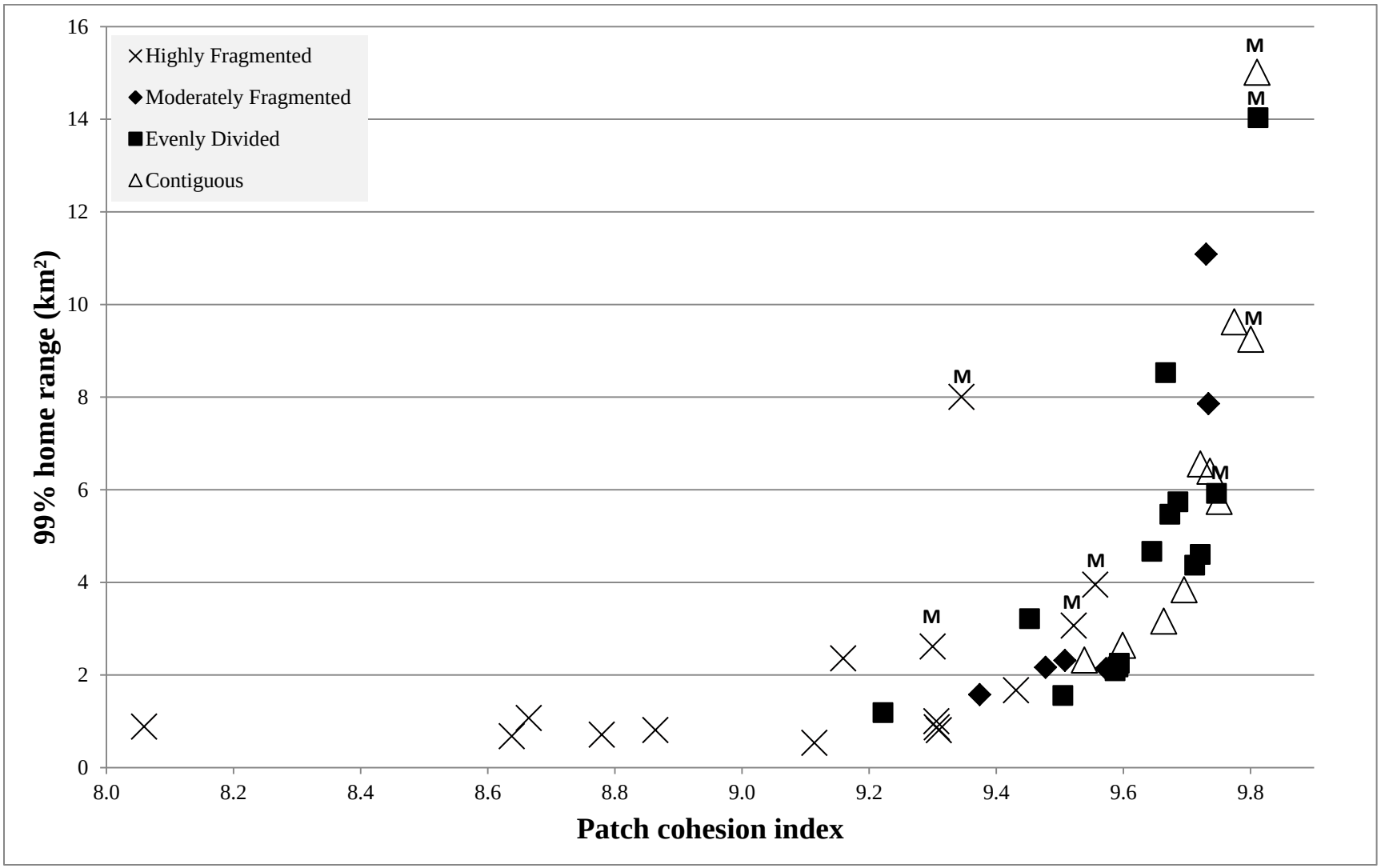

Highly Fragmented
Moderately Fragmented
Evenly Divided
Contiguous
95% home range (km²)
Patch cohesion index
0
1
2
3
4
5
6
7
8
9
7.8
8.0
8.2
8.4
8.6
8.8
9.0
9.2
9.4
9.6
9.8
M

Figure 6.


Highly Fragmented
Moderately Fragmented
Evenly Divided
Contiguous
99% home range (km²)
Patch cohesion index
0
2
4
6
8
10
12
14
16
8.0
8.2
8.4
8.6
8.8
9.0
9.2
9.4
9.6
9.8
M

Chapter 3

Odds of exposure to chronic wasting disease driven by habitat occupied by white-tailed deer

Chapter 3 was written in collaboration with Megan S. Kirchgessner, Brian Eyler, Christopher W. Ryan, and W. David Walter. I have included this manuscript on the following pages as formatted for the *Journal of Wildlife Management*.

ABSTRACT

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy that was first detected in 1967 in a captive research facility in Colorado. In the northeastern United States (Northeast), CWD was first confirmed in white-tailed deer (*Odocoileus virginianus*) in 2005. Because CWD is a new and emerging disease with a spatial distribution that had yet to be assessed in the Northeast, we examined fixed (demographic and environmental) and random effects to determine how each related to this spatial distribution. The objectives of our study were to (1) identify fixed and random effects that best described the spatial distribution of CWD in free-ranging white-tailed deer and (2) identify areas at risk for CWD infection in the Northeast. Demographic covariates included sex and age, and environmental covariates included elevation, slope, riparian corridor, percent clay, and proportion of 3 habitat types (developed, forested, and open). The model with the most support contained habitat covariates and random spatial effects that represented clustering of CWD in adjacent grid cells. Forested habitat had the strongest relationship with the distribution of CWD, with increased risk of CWD occurring in areas that had lesser amounts of forest. Our results will assist resource managers in understanding the spatial distribution of CWD not only within the study area, but also in surrounding areas where CWD has yet to be found. Efficiency of disease surveillance and containment efforts can be improved by allocating resources used for surveillance into areas that are at greatest risk for infection.

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy that was first detected in captive mule deer (*Odocoileus hemionus*) in Colorado (Williams and Young 1980). Other members of the Cervidae family, including white-tailed deer (*Odocoileus virginianus*), elk (*Cervus canadensis*), and moose (*Alces alces*), also are affected by CWD. In 2005, CWD was first detected in the northeastern United States (Northeast) in white-tailed deer in New York and West Virginia and since has been found in Virginia, Maryland and Pennsylvania. Studies in other regions identified demographic and environmental covariates that influenced the spatial distribution of CWD (Farnsworth et al. 2006, Walter et al. 2011, Storm et al. 2013). An assessment of the relationship between these covariates and the distribution of CWD is lacking, however, in the Northeast where the disease is still new and emerging.

Bayesian methods are used in a variety of fields to explain epidemiology of disease and resulting spatial patterns (Levin 1992). Bayesian hierarchical modeling provides a framework that can be used to examine disease presence or absence across the landscape, as well as fixed and random effects that explain the spatial distribution of disease (Clayton and Kaldor 1987, Waller et al. 1997). Random effects account for spatial autocorrelation because estimates of fixed effects could be biased and inappropriately precise otherwise, leading to increased Type I error rates (Diniz-Filho et al. 2008). In comparison with earlier methods of disease mapping (e.g., Empirical Bayes) that incorporate less information, fully Bayesian approaches provide estimates of disease risk that account for variability and randomness at local scales (Bernardinelli et al. 1995, Elliott and Wartenburg 2004). Bayesian hierarchical modeling allows for simultaneous investigation of fixed effects that pertain to environmental covariates as well as variability caused by random effects that can be either spatially dependent or independent

(Farnsworth et al. 2006, Osnas et al. 2009). Combined with demographic covariates, any of these fixed or random effects or combinations of effects can influence the spatial distribution of disease across the landscape.

In a predominantly agricultural-forest matrix, fixed effects (e.g., forest cover, edge) promoted high deer densities and CWD prevalence in Wisconsin and Illinois, and these fixed effects likely contributed to spread of CWD at a local scale (Storm et al. 2013, Kelly et al. 2014). We would expect the epidemiology of CWD to differ in the Northeast because the landscape is composed predominantly of large homogeneous forests. The Northeast presents a new environment in which CWD has yet to be assessed and an opportunity to better understand the epidemiology of CWD in white-tailed deer. As CWD continues to expand in the region, an assessment of its spatial distribution is needed for improvement of surveillance and containment strategies for not only the Northeast but also other regions (e.g., southeastern U.S.) with predominantly forested landscapes. In this study, we examined factors to determine how each related to the spatial distribution of CWD and potentially controlled odds of CWD infection in the central Appalachian region of the Northeast. The objectives of our study were to (1) identify fixed and random effects that best described the spatial distribution of CWD in free-ranging white-tailed deer and (2) identify areas at risk for CWD infection in the Northeast.

STUDY AREA

Our study area consisted of 2,340 km^2 in northwestern Maryland, northern Virginia, and eastern West Virginia (Fig. 1). The core was located in West Virginia, where CWD was first confirmed in a deer killed by a vehicle in 2005 and since has been found in deer harvested in nearby counties and in adjacent states (Evans et al. 2014). Primary land cover consisted of

deciduous oak-hickory forests with sparse stands of conifers. Pastures and croplands were prominent land uses in the area, and intensities of urban development also were present in the form of roads and small communities. Elevation ranged from 131 m to 875 m, and mean annual snowfall ranged from 57 cm to 163 cm.

METHODS

Sampling grid.– We used estimates of annual home range for 45 white-tailed deer in the region to determine the spatial resolution for our analysis (Chapter 2). We used a movement-based kernel density estimator to estimate 99% isopleths of home range for each deer. The combined mean size of home range for males (7.7 km^2) and females (3.4 km^2) was approximately 6 km^2, and this constituted the resolution of a spatial sampling grid (e.g., size of each cell) over the study area (hereafter referred to as *sampling grid*). Within each cell of our sampling grid, data for environmental covariates was calculated for each deer in our dataset. We assumed that each deer's location of harvest reflected its natural home range within the sampling grid.

Demographic covariates.– We received geo-referenced data for 11,357 hunter-harvested white-tailed deer that were tested for CWD between 2005 and 2012 from the Maryland Department of Natural Resources, Virginia Department of Game and Inland Fisheries, and West Virginia Division of Natural Resources. We retained data for deer (n = 7,427) that were located within the extent of the outermost CWD-positive deer (Fig. 1), due to computing limitations with Bayesian models over large spatial dimensions. All locations of harvest were reported by hunters in accordance with each state's surveillance protocol (Evans et al. 2014). Locations of harvest in West Virginia and Virginia were provided on grids with 2.6 km^2-cells, and we extracted centroid

locations from each grid cell. Exact coordinates were provided for all locations of harvest in Maryland. In addition to location of harvest, the sex, age, and test result for CWD (positive or negative) also were provided for each deer. We aggregated all data collected between 2005 and 2012, because temporal trends were not found to be significant in other studies of CWD (Farnsworth et al. 2006, Osnas et al. 2009). We removed fawns (n = 8) from our dataset, because fawns were removed from past analyses due to low prevalence (<0.005) in the age class and none of the hunter-harvested fawns in our study area tested positive for CWD (Grear et al. 2006).

Given the association of CWD with males and increased prevalence of CWD in older deer of both sexes, sex and age were considered as covariates (Miller and Conner 2005, Grear et al. 2006). Assuming that these associations could be used to describe demographic trends of CWD in our study area, all male deer were coded as one and females as zero in the sex category. Several detections of CWD in yearling males (<2.5 years) in the region prompted us to code both yearling and adult males (≥2.5 years) as one in the age category. Additionally, all females that were ≥2.5 years of age at time of harvest also were coded as one in this category. Therefore yearling females were the baseline demographic, providing an average infection risk that would be represented by the intercept in each model (Osnas et al. 2009, Walsh and Miller 2010).

Environmental covariates.– Environmental covariates were based on location of harvest, landscape covariates specific to the central Appalachian region, and findings from studies of CWD in other regions (Farnsworth et al. 2006, Rees et al. 2011, Walter et al. 2011). We calculated data for environmental covariates using soil, land cover, elevation, and stream layers in ArcMap 10.1 (ArcMap; Environmental Systems Research Institute, Redlands, CA, USA). We included a covariate representing percent of clay-sized particles in the soil (hereafter referred to

as *percent clay*) that was found to be most important in explaining the distribution of CWD in mule deer in Colorado (Walter et al. 2011). We extracted percent clay within the Soil Data Viewer using the Soil Survey Geographic database (SSURGO; USDA 2007), and we estimated an area-weighted mean of percent clay for each cell of our sampling grid using the Geospatial Modeling Environment (Beyer 2012).

We used the 2006 National Land Cover Database to estimate proportion of 3 habitat types (developed, forested, and open) within each cell of the sampling grid (Fry et al. 2011). The developed class included roads and urban, suburban, and exurban levels of development. All types of forested habitat (deciduous, coniferous, and mixed) were reclassified as forested, and the open class included all pastures, grasslands, and croplands. We chose classes of habitat that covered broader spectrums, because we viewed sub-habitats (e.g., cropland and pasture) similarly in regard to basic movements and function to white-tailed deer.

We used a 30 m digital elevation model provided by the United States Geological Survey in the National Elevation Dataset to estimate mean slope (degrees) and elevation (m) in each cell of our sampling grid (Gesch 2007). We estimated percent of riparian corridor for each cell in our sampling grid using a stream layer that was generated with data collected by the United States Geological Survey (National Atlas of the United States 2012). We created buffered corridors around each stream, and the size of each buffer (274 m on each side of the stream) was based on mean daily distances traveled by deer used to estimate home range (Appendix A). We examined distributions of each environmental covariate, log-transformed riparian corridor and habitat to follow normal distributions, and assigned data to each deer in our dataset based on location of harvest within the sampling grid.

Bayesian hierarchical modeling.– We created 25 logistic regression models in a Bayesian hierarchical modeling framework and compared the fit of each model to the data. Models were additive and represented 25 combinations of demographic and environmental covariates, as well as spatial and non-spatial random effects (Table 1). We included sex and age in 23 models to account for presumed effects of each on occurrence of CWD (Miller and Conner 2005, Grear et al. 2006). We grouped covariates from the digital elevation model (slope and elevation) and land cover layer (habitat) as DEM and HAB, respectively, such that they were either included or removed as one set of covariates in each model (Table 1).

We addressed random effects, represented by local clustering of disease in adjacent grid cells (CAR) and region-wide heterogeneity (HET), by creating a spatial adjacency matrix using our sampling grid and *spdep* package (Bivand et al. 2011) in program R (R Foundation for Statistical Computing, Vienna, Austria). Relationships between neighboring cells were represented using an intrinsic Gaussian conditional autoregressive model that specified a dependent relationship between neighboring grid cells but conditional independence between non-neighboring cells (Besag et al. 1991).

We assumed that each deer's CWD infection status (coded one for positive and zero for negative) was Bernoulli distributed and conditionally independent given the probability of infection. We used the logit-link function to describe probability of infection as a function of the covariates and effects incorporated within each model (Besag et al. 1991). After testing prior distributions from other studies in our full model (Appendix B), we estimated gamma-distributed priors that represented our study area with the following equation:

$$SD(\delta_j) = \frac{1}{\sigma_\delta} \approx \frac{1}{0.7\sigma_\gamma \overline{m}} \approx SD(\gamma_j), \qquad (1)$$

where σ_γ refers to the prior conditional standard deviation, $\overline{m}$ refers to mean number of neighbors, and $SD(\gamma_j)$ refers to the prior marginal standard deviation of γ_j parameters (Bernardinelli et al. 1995, Banerjee et al. 2004). Hyperparameters of 17.04 and 4.13 were assigned to the random effect capturing region-wide heterogeneity (HET) for shape and scale parameters, respectively, and hyperparameters of 1.0 were assigned to both shape and scale parameters for the random effect capturing local clustering (CAR). We calculated lambda, or the ratio of the standard deviation of CAR to combined standard deviations of CAR and HET, to assess how each random effect accounted for variability that was not accounted for by fixed effects.

We performed all modeling in program R and used the *R2WinBUGS* package to call Markov Chain Monte Carlo simulation methods within program WinBUGS (Spiegelhalter et al. 2003, Sturtz et al. 2005). These simulations were used to estimate posterior distributions for all model parameters. We used initial values of zero for all beta parameters, as these starting values were not considered detrimental to convergence rates (Eberly and Carlin 2000). For each model, we ran 3 independent chains for 250,000 iterations, while discarding the first 100,000 and thinning each chain by keeping every twentieth iteration (Appendix C).

Model evaluation.– We used the deviance information criterion (DIC) to compare fit of each model to the data, and we recorded complexity of each model using the effective number of parameters (p_D) produced in the summary output (Spiegelhalter et al. 2002). The model with the most support had a DIC that was lowest of all candidate models. We used DIC weights (W_{DIC}) to

estimate model selection uncertainties while providing a measure of model strength given the data (Farnsworth et al. 2006). Weights were estimated with ΔDIC referring to difference in DIC between the model with the lowest DIC and the model of interest (Table 2). We assessed chain convergence using the Bayesian analysis package *boa* (Smith 2007). After examining several graphical tools, including autocorrelation and trace plots, we calculated univariate corrected scale reduction factors and a multivariate potential scale reduction factor to ensure that all parameters had converged simultaneously (Brooks and Gelman 1998).

Predicting risk of CWD.– We used land cover data that was reclassified into developed (Dev), forested (For), and open (Open) classes to identify areas at risk for CWD in the Northeast. We created 3 separate raster layers with each class of habitat coded as one in a layer while the other classes were coded as zero. We applied parameter estimates from the best supported model to each corresponding layer using a resource selection function represented by the following equation:

$$Risk = \frac{Exp((Dev*1.06)+(For*-6.50)+(Open*-0.86))}{1+Exp((Dev*1.06)+(For*-6.50)+(Open*-0.86))}, \qquad (2)$$

where each class of habitat was represented with its corresponding estimate (Manly et al. 2002). We used results to identify areas at high (50–75%), medium (25–50%), and low (0–25%) risk for CWD occurrence in the Northeast.

RESULTS

Of the 7,427 samples analyzed from harvested white-tailed deer, 69 tested positive for CWD and yielded a prevalence of 0.93% within our sampling grid. The adult male class contained the greatest number of positive cases (n = 50), followed by yearling males (n = 9), adult females (n = 8), and yearling females (n = 2). The adult male class also contained the

greatest number of negatives (n = 3,457), followed by yearling males (n = 2,082), adult females (n = 1,339), and yearling females (n = 480).

Bayesian hierarchical modeling showed strong support for models containing environmental and spatial components. With exceptions made for sex and age, which were included in all but 2 models, the model with the most support contained habitat (HAB) and the local clustering effect (CAR) and accounted for 80.3% of the overall weight (Table 2). The top 5 models contained the local clustering effect and received greater than 99% of the combined weight for all models considered, suggesting that random spatial effects (clustering of disease among neighboring grid cells) combined with habitat (proportion of developed, forested, and open) better accounted for the spatial distribution of CWD than any other random or fixed effects. In the second best model (14.5%), mean slope was the only influential covariate and shared a slightly inverse relationship (–0.32; 95% CI = –0.07 to –0.56) with the spatial distribution of CWD. Model results and parameter estimates suggested that other environmental covariates (elevation, riparian corridor and percent clay) did not influence odds of CWD infection in our study area.

The value of lambda was 0.78 in the best model, suggesting that random effects captured by local clustering of CWD better accounted for variability not described by other random or fixed effects (Fig. 2). Specifically, local clustering accounted for between 71% and 84% of this variability (Table 3). Models that contained either or both random effects (n = 18) had estimates of lambda that ranged from 0.53 to 0.80, suggesting that local clustering better accounted for this variability in all models that incorporated random effects.

Among all habitat covariates, forested habitat had the strongest relationship with odds of CWD infection (odds ratio = 0.9371; 95% CI = 0.8864 to 0.9932), indicating that a decrease in forest increased odds of CWD infection. Open and developed habitats also were influential and had slightly inverse and positive relationships with odds of CWD infection, respectively (Table 3). The influence of habitat on odds of infection ranged from 0.1% to 8.3% within all cells of our sampling grid.

Our analysis showed that effects of sex (odds ratio = 1.0056; 95% CI = 0.9982 to 1.0142) and age (odds ratio = 1.0063; 95% CI = 0.9906 to 1.0263) could not be identified, as both had 95% credible intervals that contained zero (Table 3). The intercept (–7.62; 95% CI = –10.30 to –4.99) quantified average risk of infection throughout the study area among yearling females, which represented the baseline demographic in our analysis.

DISCUSSION

Spatial effects, represented as clusters of neighboring cells that contained CWD-infected deer, influenced odds of infection in the central Appalachian region in a similar manner to results in other regions of the U.S. (Farnsworth et al. 2006, Osnas et al. 2009, Walter et al. 2011). A clustering of CWD-positive samples was identified in the center of the study area where CWD was first detected in 2005 and has been detected every year since (6–15 samples/year) using samples from the hunter harvest (Fig. 2; Evans et al. 2014). This clustering, possibly representing an effect of increased horizontal transmission between deer occupying the same social groups, also was apparent when choosing prior distributions for random effects describing local clustering and region-wide heterogeneity. If effects of clustering and heterogeneity described by CAR and HET had been equal, then estimates of lambda would have been approximately 0.5.

Estimates were consistently above 0.75, however, regardless of the set of gamma-distributed priors tested (Appendix B).

The effect of local clustering on the distribution of CWD may be explained by presumed methods of direct and indirect transmission that occur in cervid populations affected by CWD. The CWD prion has been found in several secretions, including saliva, blood, urine, and feces (Sigurdson 2008). Prions also are found in high concentrations at mortality sites and these areas enable potential uptake by other deer (Jennelle et al. 2009). Increased deposition and environmental persistence of prions was identified during early stages of CWD infection in the Midwest and may play a similar role in the central Appalachian region (Almberg et al. 2011). Deposition of prions in areas where CWD has been present for several years may explain the clustered distribution of CWD within a 1,671 km^2 county where 61 of 69 positive cases (via hunter harvest) were found between 2005 and 2012 in the central Appalachian region. Of the other 8 cases, 5 were detected 14 km southeast in Virginia (2009–2012), 2 were 25 km south in West Virginia (2010, 2012), and the remaining case was 21 km north in Maryland (2010). An additional 5 positive deer were identified >85 km north in Pennsylvania from 2012 to 2014, however, these cases were not included in our analysis. This may describe a gradual spread outward from this cluster that is more dependent on spatial effects rather than a temporal component (Osnas et al. 2009). However, the role that captive cervid facilities play in CWD transmission in Pennsylvania, second in numbers only to Texas, has not been evaluated and requires further research.

We observed an inverse relationship between forested habitat and odds of CWD infection in the central Appalachian region. Long et al. (2005) found that distance of deer dispersal in the

region was influenced by forest cover, with greater distances associated with areas that had lesser amounts of forest cover. Similar behaviors of dispersal have been identified in Illinois, a state infected with CWD since 2002, where increased forest cover negatively influenced dispersal of white-tailed deer (Nixon et al. 2007). In the core of our study area in West Virginia, open and developed habitats appear to be more prominent and CWD has spread rapidly since 2005. Detection of CWD did not occur in Maryland until 2010, however, suggesting that the contiguously forested landscape in this area may limit dispersals of infected deer to a local scale. However, recent detections of CWD in Pennsylvania (e.g., 5 positive cases in free-ranging deer tested between 2012 and 2014) suggest that sparse cases found in contiguously forested areas outside of the core area may play a role in the spatial distribution of CWD in the central Appalachian region. Cases detected in free-ranging deer in Pennsylvania may or may not be related to this core, and determining the origins of these cases will require further research that identifies measures of genetic relatedness and structuring of subpopulations.

Other habitat types were not as highly associated with odds of CWD infection. Farnsworth et al. (2006) found a strong relationship between low-elevation grassland habitat and odds of CWD infection in mule deer. In Colorado, this habitat is used as core wintering habitat where higher interactions are likely to occur. In our study area, open habitat such as pasture, grassland, and cropland had a slightly inverse relationship with odds of CWD infection (odds ratio = 0.9914; 95% CI = 0.9843 to 0.9990). Similar to wintering habitat in Colorado, we expected an increase in open habitat to have a positive relationship with odds of CWD infection due to increased interactions during winter months. Milder winters (e.g., lower snow depths and warmer temperatures) in the central Appalachian region (Horsley et al. 2003), however, as well

as behavioral differences between white-tailed deer and mule deer, may explain why open habitat was less influential in our study area than others.

The habitat type representing intensities of urban development positively influenced odds of CWD infection (odds ratio = 1.0106; 95% CI: 1.0004 to 1.0233). Although developed habitats in our study area had less of an effect on odds of CWD infection than in other regions where urban landscapes influenced CWD and even doubled odds of infection, the positive relationship we observed was consistent with findings from those studies (Wolfe et al. 2004, Farnsworth et al. 2005). Because open and developed habitats could be misclassified in some cases, especially when dealing with land cover data, we combined these classes and repeated our modeling procedure with a new habitat covariate consisting only of the forested class and this combined class (i.e., open and developed). The strong inverse relationship between forested habitat and odds of infection persisted. Combining classes, however, did not result in the strong positive relationship that we expected. This also could be an effect of both open and developed classes occurring at lower elevations, thus providing a similar niche to ecology of white-tailed deer.

Because we did not find sex and age to be as important in controlling odds of infection as in previous studies, we examined the effect of changing our baseline infection class and found that age became significant when we retained the age effect for adult males only and designated all other classes as the baseline for age. Adult females are presumed to be at elevated risk due to increased time of exposure, however, and we used recent detections of CWD in yearling males in the Midwest and Northeast as a basis for including the age effect for these classes (Miller and Conner 2005, Grear et al. 2006, Storm et al. 2013). Regardless of the baseline infection class assigned for demographic covariates in our analysis, estimates for environmental and random

effect parameters did not change. Further analysis of the relationship between demographic covariates and epidemiology of CWD is needed in the central Appalachian region and will require age-specific data collected using a method (e.g., sharpshooting or road-kill) that is less biased than the hunter harvest (Conner et al. 2000).

The region east of our study area is characterized by sparse forested habitat and high intensities of developed and open habitats. As of 2012, CWD had not been detected in this area, however, a yearling male that was harvested during the fall of 2013 tested positive for CWD approximately 16 km to the southeast of the eastern-most deer included in our analysis. Predictions of risk for CWD suggested that this area was not only at elevated risk for CWD exposure, but also may expedite spread into northern Virginia and southeastern Pennsylvania (Fig. 3). With CWD now present, future surveillance should focus on this expedited front, as well as in bordering areas with similar landscapes.

The spatial distribution of CWD in the central Appalachian region yielded insights into roles played by covariates that had not been observed in other regions as well as other anecdotal observations (Farnsworth et al. 2006, Osnas et al. 2009, Walter et al. 2011). For example, 140 positive cases were detected over a 7-year span in the central Appalachian region from samples collected by all methods of surveillance (i.e., hunter harvest, road-kills, and sharpshooting). Over a comparable time period using the same methods of surveillance, more than twice and nearly 3 times the number of positives were detected in Wisconsin's disease eradication zone (n = 316; Grear et al. 2006) and along the Wisconsin-Illinois border (n = 382; O' Hara Ruiz et al. 2013), respectively. The lower number of positives in the homogenously forested region of the Northeast (Evans et al. 2014) compared to the agricultural-forest dominated landscape of the

Midwest with comparable sampling efforts would appear to support our assumptions of a lower potential for spread of CWD due to greater isolation of deer matriarchal groups in large homogenous forests. Further research on genetic relatedness and subpopulation structuring is underway and will provide additional information on movements of deer in this region similar to previous research in the Midwest (Kelly et al. 2014). In any region, understanding movements of hosts and distribution of disease in relation to factors that influence odds of disease presence or absence allows surveillance and containment measures to be improved by allocating resources into areas that are determined to be at greatest risk for infection.

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REFERENCES

Almberg, E. S., P. C. Cross, C. J. Johnson, D. M. Heisey, and B. J. Richards. 2011. Modeling routes of chronic wasting disease transmission: environmental prion persistence promotes deer population decline and extinction. PLoS ONE 6:e19896.

Banerjee, S., B. P. Carlin, and A. E. Gelfand. 2004. Hierarchical modeling and analysis for spatial data. Chapman and Hall/CRC, New York.

Bernardinelli, L., D. Clayton, and C. Montomoli. 1995. Bayesian estimates of disease maps: how important are priors? Statistics in Medicine 14:2411-2431.

Besag, J., J. York, and A. Mollie. 1991. Bayesian image restoration, with two applications in spatial statistics. Annals of the Institute of Statistical Mathematics 43:1-59.

Beyer, H. L. 2012. Geospatial Modelling Environment (Version 0.6.0.0).

Bivand, R., M. Altman, L. Anselin, R. Assuncao, and O. Berke. 2011. spdep: spatial dependence, weighting schemes, statistics and models. R package (Version 0.5–71).

Brooks, S. P., and A. Gelman. 1998. General methods for monitoring convergence of iterative simulations. Journal of Computational and Graphical Statistics 7:434-455.

Clayton, D., and J. Kaldor. 1987. Empirical Bayes estimates of age-standardized relative risks for use in disease mapping. Biometrics 43:671-681.

Conner, M. M., C. W. McCarty, and M. W. Miller. 2000. Detection of bias in harvest-based estimates of chronic wasting disease prevalence in mule deer. Journal of Wildlife Diseases 36:691-699.

Cross, P. C., D. M. Heisey, B. M. Scurlock, W. H. Edwards, M. R. Ebinger, and A. Brennan. 2010. Mapping brucellosis increases relative to elk density using hierarchical Bayesian models. PLoS ONE 5:e10322.

Del Rio Vilas, V. J., S. Ancelet, J. J. Abellan, C. P. D. Birch, and S. Richardson. 2011. A Bayesian hierarchical analysis to compare classical and atypical scrapie surveillance data: Wales 2002-2006. Preventive Veterinary Medicine 98:29-38.

Diniz-Filho, J., T. Rangel, and L. Bini. 2008. Model selection and information theory in geographical ecology. Global Ecology and Biogeography 17:479-488.

Eberly, L. E., and B. P. Carlin. 2000. Identifiability and convergence issues for Markov chain Monte Carlo fitting of spatial models. Statistics in Medicine 19:2279-2294.

Elliott, P., and D. Wartenburg. 2004. Spatial Epidemiology: Current Approaches and Future Challenges. Environmental Health Perspectives 112:998–1006.

Evans, T. S., K. L. Schuler, and W. D. Walter. 2014. Surveillance and monitoring of white-tailed deer for chronic wasting disease in the northeastern United States. Journal of Fish and Wildlife Management 5: *In Press*.

Farnsworth, M. L., J. A. Hoeting, N. T. Hobbs, and M. W. Miller. 2006. Linking chronic wasting disease to mule deer movement scales: a hierarchical bayesian approach. Ecological Applications 16:1026-1036.

Farnsworth, M. L., L. L. Wolfe, N. T. Hobbs, K. P. Burnham, E. S. Williams, D. M. Theobald, M. M. Conner, and M. W. Miller. 2005. Human land use influences chronic wasting disease prevalence in mule deer. Ecological Applications 15:119-126.

Fry, J., G. Xian, S. Jin, J. Dewitz, C. Homer, L. Yang, C. Barnes, N. Herold, and J. Wickham. 2011. Completion of the 2006 National Land Cover Database for the Conterminous United States. Photogrammetric Engineering & Remote Sensing 77:858–864.

Gesch, D. 2007. The National Elevation Dataset. Pages 99-118 *in* D. Maune, editor. Digital Elevation Model Technologies and Applications: The DEM Users Manual. American Society for Photogrammetry and Remote Sensing, Bethesda.

Grear, D. A., M. D. Samuel, J. A. Langenberg, and D. Keane. 2006. Demographic patterns and harvest vulnerability of chronic wasting disease infected white-tailed deer in Wisconsin. Journal of Wildlife Management 70:546-553.

Horsley, S. B., S. L. Stout, and D. S. DeCalesta. 2003. White-tailed deer impact on the vegetation dynamics of a northern hardwood forest. Ecological Applications 13:98-118.

Jennelle, C. S., M. D. Samuel, C. A. Nolden, and E. A. Berkley. 2009. Deer carcass decomposition and potential scavenger exposure to chronic wasting disease. Journal of Wildlife Management 73:655-662.

Kelly, A. C., N. E. Mateus-Pinilla, W. Brown, M. O. Ruiz, M. R. Douglas, M. E. Douglas, P. Shelton, T. Beissel, and J. Novakofski. 2014. Genetic assessment of environmental features that influence deer dispersal: implications for prion-infected populations. Population Ecology 56:327-340.

Kelsall, J. E., and J. C. Wakefield. 1999. Discussion of ‘‘Bayesian models for spatially correlated disease and exposure data’’ by Best et al. Page 151. *in* B. J.M., J. O. Berger, A. P. Dawid, andA. F. M. Smith, editors. Bayesian Statistics 6. Oxford University Press, Oxford, UK.

Levin, S. A. 1992. The problem of pattern and scale in ecology. Ecology 73:1943-1967.

Long, E. S., D. R. Diefenbach, C. S. Rosenberry, B. D. Wallingford, and M. D. Grund. 2005. Forest cover influences dispersal distance of white-tailed deer. Journal of Mammalogy 86:623-629.

Manly, B. F. J., L. L. McDonald, and D. L. Thomas. 2002. Resource selection by animals: statistical design and analysis for field studies. Volume 2nd.Kluwer Academic Publishers, Dordrecht.

Miller, M. W., and M. M. Conner. 2005. Epidemiology of chronic wasting disease in free-ranging mule deer: spatial, temporal, and demographic influences on observed prevalence patterns. Journal of Wildlife Diseases 41:275-290.

National Atlas of the United States. 2012. One Million-Scale Streams of the United States. National Atlas of the United States. http://www.nationalatlas.gov/mld/1strmsl.html.

Nixon, C. M., P. C. Mankin, D. R. Etter, L. P. Hansen, P. A. Brewer, J. E. Chelsvig, T. L. Esker, and J. B. Sullivan. 2007. White-tailed deer dispersal behavior in an agricultural environment. American Midland Naturalist 157:212-220.

O' Hara Ruiz, M., A. C. Kelly, W. M. Brown, J. Novakofski, and N. E. Mateus-Pinilla. 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 8:215-227.

Osnas, E. E., D. M. Heisey, R. E. Rolley, and M. D. Samuel. 2009. Spatial and temporal patterns of chronic wasting disease: fine-scale mapping of a wildlife epidemic in Wisconsin. Ecological Applications 19:1311-1322.

Rees, E. E., E. H. Merrill, T. K. Bollinger, Y. T. Hwang, M. J. Pybus, and D. W. Coltman. 2011. Targeting the detection of chronic wasting disease using the hunter harvest during early phases of an outbreak in Saskatchewan, Canada. Preventive Veterinary Medicine 104:149-159.

Sigurdson, C. J. 2008. A prion disease of cervids: chronic wasting disease. Veterinary Research 39:1-12.

Smith, B. J. 2007. boa: an R package for MCMC output convergence assessment and posterior inference. Journal of Statistical Software 21:1-37.

Spiegelhalter, D., A. Thomas, N. Best, and D. Lunn. 2003. WinBUGS Version 1.4 user manual. MRC Biostatistics Unit, Cambridge.

Spiegelhalter, D. J., N. G. Best, B. P. Carlin, and A. van der Linde. 2002. Bayesian measures of model complexity and fit. Journal of the Royal Statistical Society.Series B (Statistical Methodology) 64:583-639.

Storm, D. J., M. D. Samuel, R. E. Rolley, P. Shelton, N. S. Keuler, B. J. Richards, and T. R. Van Deelen. 2013. Deer density and disease prevalence influence transmission of chronic wasting disease in white-tailed deer. Ecosphere 4:art10.

Sturtz, S., U. Ligges, and A. Gelman. 2005. R2WinBUGS: a package for running WinBUGS from R. Journal of Statistical Software 12:1-16.

USDA, NRCS. 2007. Soil Data Viewer 5.2 User Guide. *in* United States Department of Agriculture, Natural Resources Conservation Service.

Waller, L. A., B. P. Carlin, H. Xia, and A. E. Gelfand. 1997. Hierarchical spatio-temporal mapping of disease rates. Journal of the American Statistical Association 92:607-617.

Walsh, D. P., and M. W. Miller. 2010. A weighted surveillance approach for detecting chronic wasting disease foci. Journal of Wildlife Diseases 46:118-135.

Walter, W. D., D. P. Walsh, M. L. Farnsworth, D. L. Winkelman, and M. W. Miller. 2011. Soil clay content underlies prion infection odds. Nature Communications 2:1-6.

Williams, E. S., and S. Young. 1980. Chronic wasting disease of captive mule deer: a spongiform encephalopathy. Journal of Wildlife Diseases 16:89-96.

Wolfe, L. L., M. W. Miller, and E. S. Williams. 2004. Feasibility of "test-and-cull" for managing chronic wasting disease in urban mule deer. Wildlife Society Bulletin 32:500-505.

FIGURE LEGENDS

Figure 1. Study area in the central Appalachian region of the northeastern United States. The inset identifies the sampling grid and locations of all positive (+) and negative (·) samples used to model chronic wasting disease between 2005 and 2012.

Figure 2. Estimates of the spatial effect capturing local clustering. Estimates are from the best model investigating the effect of demographic and environmental covariates on odds of chronic wasting disease infection between 2005 and 2012 in the central Appalachian region.

Figure 3. Chronic wasting disease (CWD) infection risk in the northeastern United States. Infection risk is represented by low (0–25%), medium (25–50%), and high (50–75%) classes. Risk was estimated using a resource selection function that incorporated region-wide land cover data and parameter estimates from the best model describing the spatial distribution of CWD between 2005 and 2012 in the central Appalachian region.

Table 1. Candidate set of models investigating the effect of demographic and environmental covariates on odds of chronic wasting disease infection from 2005 to 2012 in the central Appalachian region. In addition to sex and age, *Clay* represents the area-weighted mean of percent soil clay content in the 6 km^2 grid cell containing each sampled deer. *DEM* represents mean slope and elevation, *RIP* represents riparian corridor, and *HAB* represents proportion of 3 habitat types (developed, forested, and open). Random effects include the effect capturing local clustering (*CAR*) and the effect capturing region-wide heterogeneity (*HET*).

Covariates	Explanation
Sex + Age + Clay + DEM + RIP + HAB + CAR + HET	Full model of all covariates including random effects
Sex + Age + Clay + DEM + RIP + ------- + CAR + ------	Full model with habitat covariates and heterogeneity effect removed
Sex + Age + Clay + DEM + ----- + HAB + CAR + ------	Full model with the riparian covariate and heterogeneity effect removed
Sex + Age + Clay + DEM + ----- + ------ + CAR + HET	Full model with riparian and habitat covariates removed
Sex + Age + Clay + ------- + RIP + HAB + ------- + HET	Full model with elevation covariates and local clustering effect removed
Sex + Age + Clay + ------- + RIP + ------- + CAR + HET	Full model with elevation and habitat covariates removed
Sex + Age + ------ + DEM + RIP + ------- + CAR + HET	Full model with percent clay and habitat covariates removed
Sex + Age + ------ + DEM + ----- + HAB + CAR + HET	Full model with percent clay and riparian covariates removed
Sex + Age + ------- + ------ + RIP + HAB + CAR + HET	Full model with percent clay and elevation covariates removed
Sex + Age + Clay + ------- + ----- + HAB + ------ + HET	Percent clay, habitat covariates and heterogeneity effect retained
Sex + Age + Clay + ------- + ------ + ----- + CAR + HET	Percent clay and both random effects retained
Sex + Age + ------ + DEM + RIP + ------- + CAR + -----	Elevation, riparian covariates and local clustering effect retained
Sex + Age + ------- + ------ + RIP + HAB + CAR + ------	Riparian, habitat covariates, and local clustering effect retained
Sex + Age + ------- + ------ + RIP + ------ + CAR + HET	Riparian covariate and both random effects retained
Sex + Age + Clay + ------ + ------ + HAB + ------ + -----	Percent clay and habitat covariates retained
Sex + Age + ------ + DEM + RIP + ------- + ------ + -----	Elevation and riparian covariates retained
Sex + Age + ------- + DEM + ----- + HAB + ------ + -----	Elevation and habitat covariates retained
Sex + Age + ------- + DEM + ----- + ------ + ------ + HET	Elevation covariates and heterogeneity effect retained

Sex + Age + ------- + ------- + RIP + HAB + ------ + -----	Riparian and habitat covariates retained
Sex + Age + ------- + ------- + ----- + HAB + CAR + -----	Habitat covariates and local clustering effect retained
Sex + Age + ------- + ------- + ----- + HAB + ------ + HET	Habitat covariates and heterogeneity effect retained
Sex + Age + ------- + ------- + ----- + HAB + ----- + ------	Habitat covariates retained
Sex + Age + ------- + ------- + ----- + ------ + ------ + -----	Sex and age retained
----- + ----- + ------- + ------- + ----- + ------ + CAR + HET	Local clustering and heterogeneity effects retained
Intercept Only	Intercept retained

Table 2. Model selection results for the candidate set of models investigating the effect of demographic and environmental covariates on odds of chronic wasting disease infection from 2005 to 2012 in the central Appalachian region. In addition to sex and age, *Clay* represents the area-weighted mean of percent soil clay content in the 6 km^2 grid cell containing each sampled deer. *DEM* represents mean slope and elevation, *RIP* represents riparian corridor, and *HAB* represents proportion of 3 habitat types (developed, forested, and open). Random effects include the effect capturing local clustering (*CAR*) and the effect capturing region-wide heterogeneity (*HET*).

Model Terms	$\overline{D}$ [1]	$\widehat{D}$ [2]	pD [3]	DIC [4]	ΔDIC [5]	W_{DIC} [6]
Sex + Age + ------ + ------- + ----- + HAB + CAR + -----	601.93	541.79	60.13	662.06	0.00	0.803
Sex + Age + ------ + DEM + RIP + ------ + CAR + -----	605.67	545.86	59.81	665.48	3.42	0.145
Sex + Age + ------ + ------- + RIP + HAB + CAR + -----	604.14	540.34	63.80	667.93	5.87	0.043
Sex + Age + Clay + DEM + RIP + ------- + CAR + -----	609.13	546.62	62.51	671.64	9.58	0.007
Sex + Age + Clay + DEM + ----- + HAB + CAR + -----	607.51	541.64	65.87	673.38	11.32	0.003
---- + ----- + ------ + ------- + ------ + ------- + CAR + HET	607.78	539.07	68.70	676.48	14.42	0.000
Sex + Age + ------ + ------- + ----- + HAB + ------ + HET	649.27	618.31	30.96	680.23	18.17	0.000
Sex + Age + Clay + ------- + ----- + HAB + ------ + HET	654.61	627.45	27.16	681.77	19.71	0.000
Sex + Age + Clay + ------- + ----- + ------- + CAR + HET	610.16	537.96	72.20	682.35	20.29	0.000
Sex + Age + ------ + ------- + RIP + ------- + CAR + HET	609.48	536.41	73.07	682.55	20.49	0.000
Sex + Age + Clay + ------- + RIP + HAB + ------- + HET	655.10	627.16	27.95	683.05	20.99	0.000
Sex + Age + ------ + DEM + ----- + ------ + ------- + HET	655.43	627.60	27.83	683.26	21.20	0.000
Sex + Age + Clay + DEM + ----- + ------ + CAR + HET	615.73	542.88	72.86	688.59	26.53	0.000
Sex + Age + Clay + ------- + RIP + ------ + CAR + HET	612.87	536.84	76.03	688.90	26.84	0.000
Sex + Age + ------ + DEM + RIP + ------ + CAR + HET	616.69	541.82	74.87	691.56	29.50	0.000
Sex + Age + ------- + DEM + ----- + HAB + CAR + HET	614.72	536.89	77.82	692.54	30.48	0.000
Sex + Age + ------- + ------- + RIP + HAB + CAR + HET	614.67	536.72	77.95	692.61	30.55	0.000
Sex + Age + Clay + DEM + RIP + HAB + CAR + HET	620.23	537.39	82.85	703.08	41.02	0.000
Sex + Age + Clay + ------- + ----- + HAB + ------- + -----	723.92	717.17	6.75	730.66	68.60	0.000
Sex + Age + ------ + DEM + ----- + HAB + ------- + -----	728.94	721.27	7.67	736.60	74.54	0.000
Sex + Age + ------ + DEM + RIP + ------ + ------- + -----	736.80	730.94	5.86	742.66	80.60	0.000
Sex + Age + ------ + ------- + ----- + HAB + ------- + -----	742.83	737.03	5.80	748.63	86.57	0.000

Sex + Age + ------ + ------- + RIP + HAB + ------- + -----	742.06	735.24	6.82	748.87	86.81	0.000
Sex + Age + ------ + ------- + ----- + ------ + ------- + -----	781.64	778.68	2.96	784.60	122.54	0.000
Intercept Only	784.04	783.03	1.00	785.04	122.98	0.000

[1] $\overline{D}$ is the posterior mean of the deviance.
[2] $\widehat{D}$ is a point estimate of the deviance.
[3] p*D* is the effective number of parameters.
[4] DIC is the Deviance Information Criterion.
[5] Δ DIC is the difference between each model's DIC value and the smallest DIC value among models compared.
[6] W_{DIC}, or weights, were calculated by dividing the likelihood value for each model by the sum of likelihood values for all candidate models in the set.

Table 3. Parameter estimates, odds ratios and 95% credible intervals for the best-fitting model investigating the effect of demographic and environmental covariates on odds of chronic wasting disease infection from 2005 to 2012 in the central Appalachian region. In addition to sex and age, μ defines the baseline infection probability for a yearling female white-tailed deer. The covariate *HAB* represents 3 habitat types (developed, forested, and open), and *CAR* represents the spatial effect capturing local clustering.

Parameter	Mean	Standard deviation	Monte Carlo error	2.50%	Median	97.50%	Odds ratio	95% CI
μ	–7.62	1.36	0.02	–10.30	–7.61	–4.99	0.9266	0.9021 to 0.9513
Sex	0.56	0.40	0.00	–0.18	0.54	1.41	1.0056	0.9982 to 1.0142
Age	0.62	0.90	0.01	–0.94	0.56	2.60	1.0063	0.9906 to 1.0263
Developed	1.06	0.58	0.01	0.04	1.02	2.30	1.0106	1.0004 to 1.0233
Forested	–6.50	2.89	0.03	–12.06	–6.52	–0.69	0.9371	0.8864 to 0.9932
Open	–0.86	0.37	0.00	–1.58	–0.87	–0.10	0.9914	0.9843 to 0.9990
CAR	0.78	0.03	0.00	0.71	0.78	0.84	1.0078	1.0071 to 1.0085

Figure 1.

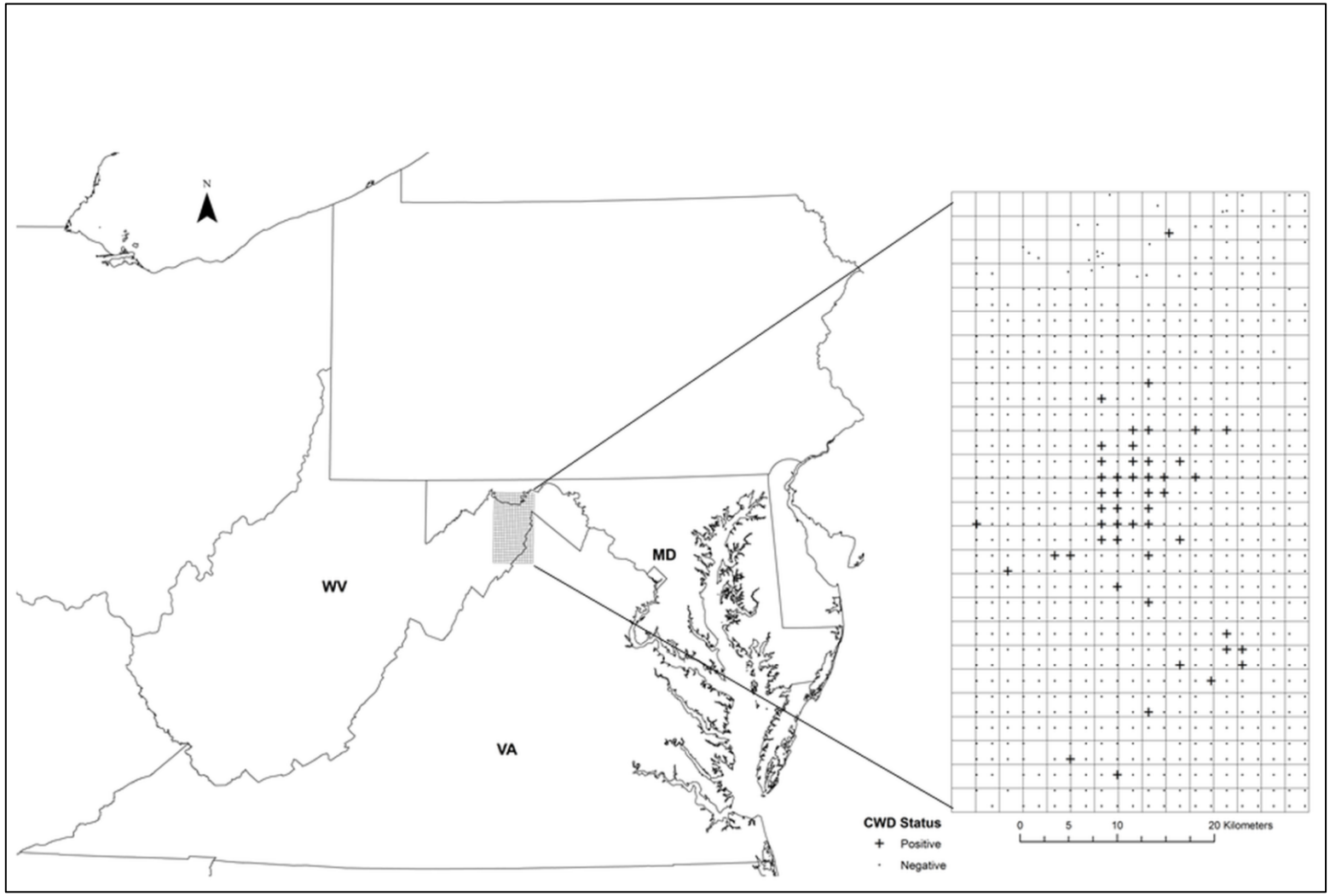


Figure 2.

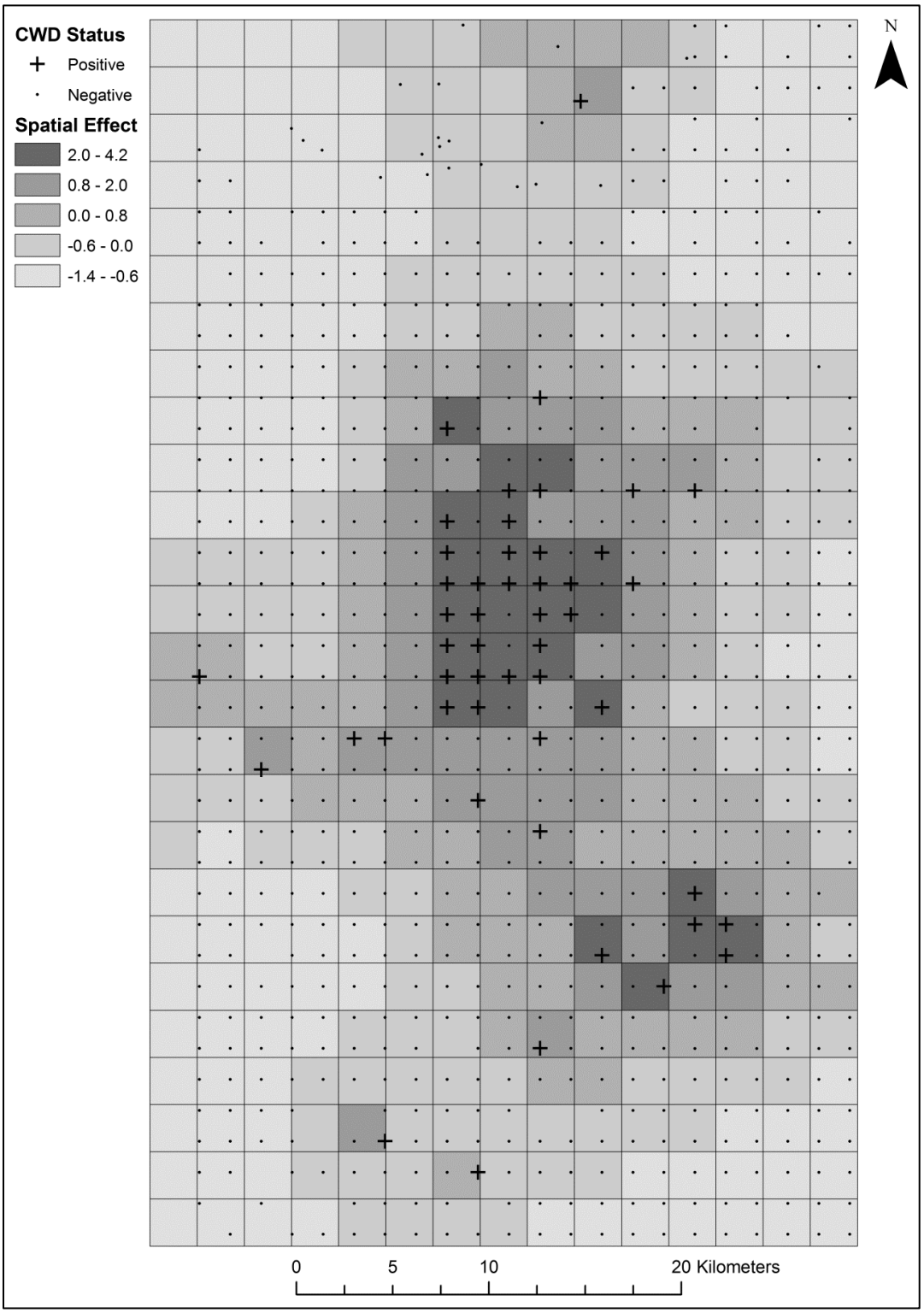
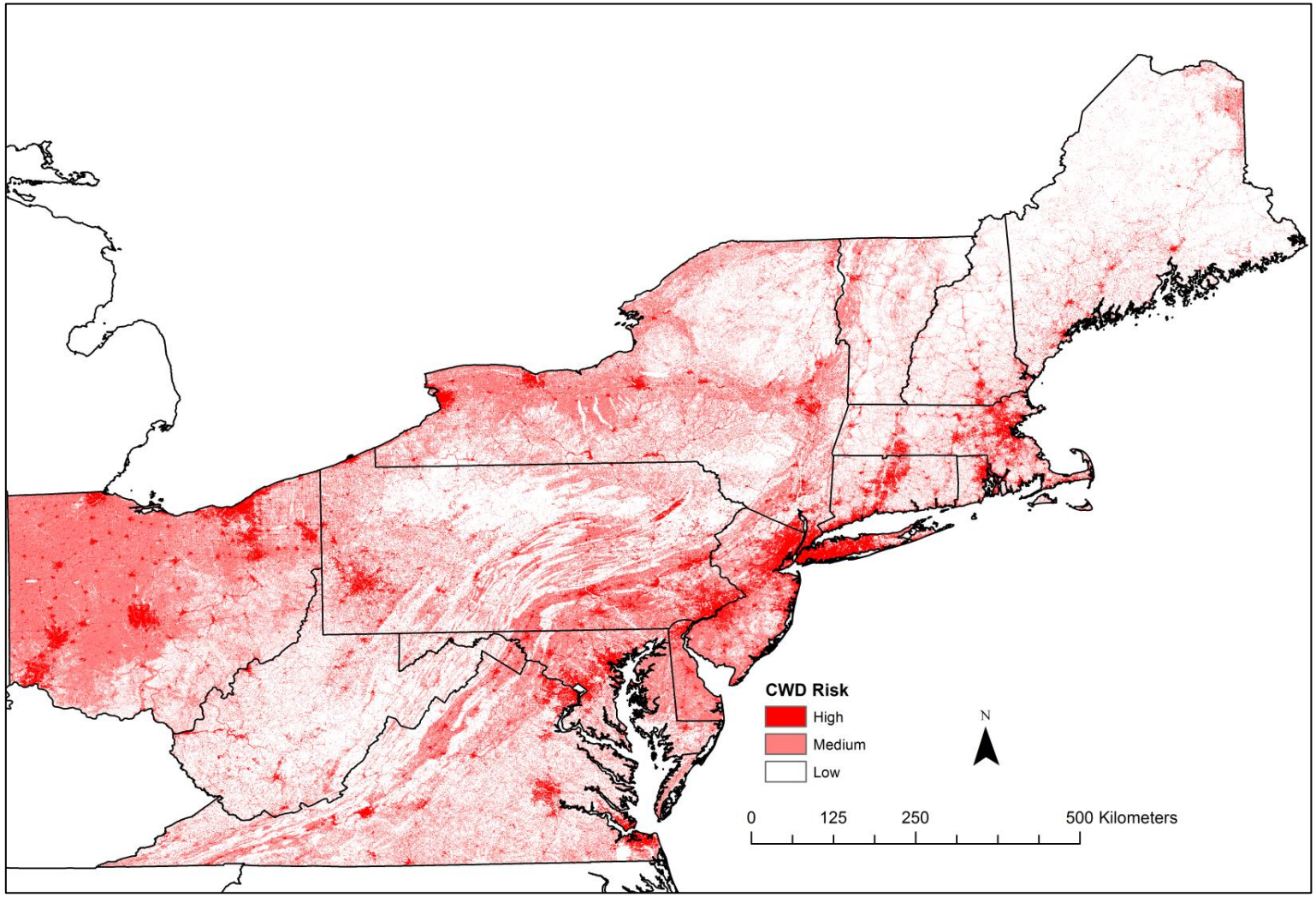

CWD Status
Positive
Negative
Spatial Effect
2.0 - 4.2
0.8 - 2.0
0.0 - 0.8
-0.6 - 0.0
-1.4 - -0.6
N
0
5
10
20 Kilometers

Figure 3.



APPENDICES

Appendix A. Estimates of mean daily distance in meters traveled by male (M) and female (F) white-tailed deer (*Odocoileus virginianus*) in six study areas in Pennsylvania. Outer percentiles (0% and 100%) refer to minimum and maximum distances, respectively, and the median (50%) and quartiles (25% and 75%) also are shown.

						Percentiles				
Study area	Sex	No. Deer	n[1]	Mean	SD	0%	25%	50%	75%	100%
Highly Fragmented	F	11	1,583	179.45	139.89	72.74	113.81	157.88	230.54	316.29
Highly Fragmented	M	4	919	222.09	161.06	99.05	150.81	206.14	281.98	368.75
Moderately Fragmented	F	2	645	223.41	181.09	65.59	116.17	180.88	291.87	510.53
Moderately Fragmented	F	4	1,229	234.75	208.10	59.26	111.46	184.88	307.42	583.16
Evenly Divided	F	6	1,983	287.09	203.08	111.08	183.16	260.48	372.03	500.96
Evenly Divided	M	2	666	445.06	327.43	156.03	277.50	413.26	590.56	783.89
Evenly Divided	F	6	1,755	250.84	231.30	58.02	111.53	190.46	327.76	651.74
Contiguous	F	8	2,690	266.65	198.26	100.05	167.72	241.85	346.44	471.55
Contiguous	M	2	658	404.05	307.06	144.31	244.15	360.44	531.46	727.20

[1] Number of 24-hr periods.

Appendix B. Estimates of lambda, or the ratio of spatial effects to total random effects, corresponding to each set of priors tested in the full model containing all covariates and random effects.

Fair priors	Tau.car	Tau.epsi	Lambda	Source
Evans[1]	dgamma(1.0, 1.0)	dgamma(17.04, 4.13)	0.7821	This study
Del Rio Vilas	dgamma(0.1, 0.01)	dgamma(0.1, 0.01)	NA[2]	Del Rio Vilas et al. 2011
Walter	dgamma(0.5,0.015)	dgamma(0.5, 0.0005)	0.9605	Kelsall and Wakefield 1999
Farnsworth	dgamma(1.0, 1.0)	dgamma(10.37, 3.22)	0.7621	Farnsworth et al. 2006
Cross	dgamma(0, 20)	dgamma(0, 20)	NA[3]	Cross et al. 2010

[1] These priors were used in all models that incorporated random effects.

[2] Tau.car priors (0.1, 0.1) resulted in a trap window and would not run in WinBUGS.

[3] Tau.car priors (0, 20) would not load initial values in WinBUGS.

Appendix C. R script for full CWD model.

```
##### LIBRARIES #####
library(R2WinBUGS)
library(spdep)
library(maptools)
gpclibPermit()
library(maptools)

##### DATA #####
df<-read.table('Smp1205.txt', na.strings='NA', header=T)
summary(df)

Result <- df$Result
Grid_ID <- df$Grid_ID
Sex <- df$Sex
Age <- df$Age
Clay <- df$Clay
Elev <- df$Elev
Slope <- df$Slope
Rip <- df$Rip
Dev <- df$Dev
For <- df$For
Open <- df$Open

##### ADJACENCY MATRIX FOR CAR MODEL #####
Shape <- readShapePoly("H:\\New_Data\\Grid.shp", IDvar = "ID")
shape_nb <- poly2nb(shape)
NumCells = length(shape_nb)
num = sapply(shape_nb,length)
adj = unlist(shape_nb)
sumNumNeigh = length(unlist(shape_nb))


##### SPATIAL MODEL #####
sink("globalmodel.bug")
cat("model {
###PRIORS FOR CAR MODEL ###
b.car[1:NumCells] ~ car.normal(adj[], weights[], num[], tau.car)
for (k in 1:sumNumNeigh)
{
    weights[k] <- 1
}
for (j in 1:NumCells)
{
epsi[j] ~ dnorm(0,tau.epsi)
}
```

```
###OTHER PRIORS ###
alpha ~ dflat()
beta1 ~ dnorm(0,1.0E-5)
beta2 ~ dnorm(0,1.0E-5)
beta3 ~ dnorm(0,1.0E-5)
beta4 ~ dnorm(0,1.0E-5)
beta5 ~ dnorm(0,1.0E-5)
beta6 ~ dnorm(0,1.0E-5)
beta7 ~ dnorm(0,1.0E-5)
beta8 ~ dnorm(0,1.0E-5)
beta9 ~ dnorm(0,1.0E-5)
tau.car ~ dgamma(1.0,1.0)
tau.epsi ~ dgamma(17.0393,4.1279)

sd.car<-sd(b.car[])
sd.epsi<-sd(epsi[])
lambda <- sd.car/(sd.car+sd.epsi)

for (i in 1 : 7427)
{
Result[i] ~ dbern(pi[i])
logit(pi[i]) <- mu[i]
mu[i] <- alpha + beta1*Sex[i] + beta2*Age[i] + beta3*Clay[i] + beta4*Elev[i] + beta5*Slope[i]
+ beta6*Rip[i] + beta7*Dev[i] + beta8*For[i] + beta9*Open[i] + b.car[Grid_ID[i]] +
epsi[Grid_ID[i]]
}
}", fill=TRUE)
sink()


##### BUNDLE DATA #####
bugs.data <- list(Result = Result, Grid_ID = Grid_ID, NumCells = NumCells, sumNumNeigh =
sumNumNeigh, num = num, adj = adj, Sex = Sex, Age = Age, Clay = Clay, Elev = Elev, Slope =
Slope, Rip = Rip, Dev = Dev, For = For, Open = Open)


##### SPECIFY INITIAL VALUES #####
inits1 <- list(alpha =0, beta1 =0,  beta2 =0, beta3 =0, beta4 =0, beta5 =0, beta6 =0, beta7 =0,
beta8 =0, beta9 =0)
inits2 <- list(alpha =0, beta1 =0,  beta2 =0, beta3 =0, beta4 =0, beta5 =0, beta6 =0, beta7 =0,
beta8 =0, beta9 =0)
inits3 <- list(alpha =0, beta1 =0,  beta2 =0, beta3 =0, beta4 =0, beta5 =0, beta6 =0, beta7 =0,
beta8 =0, beta9 =0)
inits <- list(inits1, inits2, inits3)
```

```
##### PARAMETERS TO ESTIMATE #####
parameters <- c("alpha","beta1","beta2", "beta3", "beta4", "beta5", "beta6", "beta7", "beta8",
"beta9", "lambda")

##### SETTINGS FOR MCMC #####
niter <- 250000
nthin <- 20
nburn <- 100000
nchains <- 3


##### LOCATE WINBUGS #####
bugs.dir <- "C:\\WinBUGS14"


##### CALL WINBUGS FROM R #####
out <- bugs(data = bugs.data, inits = inits, parameters.to.save = parameters, model.file =
"globalmodel.bug", n.chains = nchains, n.thin = nthin, n.iter = niter, n.burnin = nburn, debug =
TRUE, bugs.directory = bugs.dir)

print(out, 3)
```