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Deer density and disease prevalence influence transmission of chronic wasting disease in white-tailed deer

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Abstract. Host-parasite dynamics and strategies for managing infectious diseases of wildlife depend on the functional relationship between disease transmission rates and host density. However, the disease transmission function is rarely known for free-living wildlife, leading to uncertainty regarding the impacts of diseases on host populations and effective control actions. We evaluated the influence of deer density, landscape features, and soil clay content on transmission of chronic wasting disease (CWD) in young (<2-year-old) white-tailed deer (*Odocoileus virginianus*) in south-central Wisconsin, USA. We evaluated how frequency-dependent, density-dependent, and intermediate transmission models predicted CWD incidence rates in harvested yearling deer. An intermediate transmission model, incorporating both disease prevalence and density of infected deer, performed better than simple density- and frequency-dependent models. Our results indicate a combination of social structure, non-linear relationships between infectious contact and deer density, and distribution of disease among groups are important factors driving CWD infection in young deer. The landscape covariates % deciduous forest cover and forest edge density also were positively associated with infection rates, but soil clay content had no measurable influences on CWD transmission. Lack of strong density-dependent transmission rates indicates that controlling CWD by reducing deer density will be difficult. The consequences of non-linear disease transmission and aggregation of disease on cervid populations deserves further consideration.

Key words: chronic wasting disease (CWD); density dependence; disease management; disease transmission; frequency dependence; *Odocoileus virginianus*; prion disease; white-tailed deer; wildlife disease; Wisconsin, USA.

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Introduction

The impact of diseases on host populations and optimal disease management strategies depend on the functional relationship between disease transmission rates and host density. When disease transmission is dependent on host density, contact rates may not sustain disease transmission when host density drops below a disease-specific threshold (McCallum et al. 2001, Deredec and Courchamp 2003, Lloyd-Smith et al. 2005). This phenomenon potentially allows for host-pathogen coexistence and disease management via generalized culling to reduce host density. When disease transmission rates are independent of density (frequency-dependent), host-pathogen coexistence is uncertain and generalized culling is unlikely to be successful, because transmission, and hence, disease-induced mortality, is maintained at low host densities (Getz and Pickering 1983, de Castro and Bolker 2005, McCallum et al. 2009). While frequency- and density-dependent transmission have received the bulk of attention, they represent opposite extremes, and intermediate forms of transmission may be more realistic for natural systems (Barlow 2000, McCallum et al. 2001, Schauber and Woolf 2003, Smith et al. 2009). Knowledge of the correct transmission function is essential to properly forecasting disease-host dynamics and devising appropriate management strategies.

Numerous populations of mule deer (*Odocoileus hemionus*), white-tailed deer (*O. virginianus*), and elk (*Cervus elaphus*) in North America are affected by chronic wasting disease (CWD; Williams et al. 2002), a fatal, neurodegenerative prion disease with potential impact on cervid populations with high disease prevalence (Gross and Miller 2001, Wasserberg et al. 2009). In addition, there is uncertainty regarding the health risk to humans and livestock (Williams et al. 2002, Belay et al. 2004). Vertical transmission (mother to offspring) does not appear to be important, thus deer are born susceptible (Miller and Williams 2003). Additionally, there is a substantial incubation period between infection and clinical signs (≈15 months) and death (≈23 months post-infection; Williams et al. 2002). CWD prions are shed in saliva (Mathiason et al. 2006, Haley et al. 2009), urine (Haley et al. 2009), feces (Tamgüney et al. 2009), and infected carcasses (Miller et al. 2004), and other prions readily bind to soil and remain infective (Johnson et al. 2007).

Research on captive mule deer has demonstrated both direct (animal-to-animal) CWD transmission (Miller and Williams 2003) and indirect transmission from prion-contaminated environments (Miller et al. 2004). Indirect transmission was considered the primary driver of CWD epidemics in captive mule deer herds (Miller et al. 2006). Laboratory research has demonstrated that prions bind particularly well to clay minerals such as montmorillonite (Johnson et al. 2006), and transmissibility increases when prions are bound to soils with high clay content (Johnson et al. 2007). Because cervids consume soils during gazing, grooming, and from mineral licks (Beyer et al. 1994, Atwood and Weeks 2002), soils may serve as an important reservoir for CWD transmission (Schramm et al. 2006). Walter et al. (2011) found that the soil clay content was associated with CWD infection in mule deer in Colorado. Investigation of the role of soil clay content in CWD infection risk in the Wisconsin white-tailed deer epidemic is warranted because montmorillonite is a common component of soils in the region of the epidemic (Kaddou 1960). Schauber and Woolf (2003) argued that the presence of indirect transmission likely precludes strict frequency-dependent transmission.

The composition and configuration of the landscape can also influence the spatial distribution and density of disease agents, vectors, reservoirs, and hosts (Allan et al. 2003, Brownstein et al. 2005, Ostfeld et al. 2005), and may also influence disease transmission through facilitation or hindrance of contact between individuals or groups (Habib et al. 2011). Aggregation of individuals and groups increases potential disease transmission (Cross et al. 2007), and seasonal aggregation of white-tailed deer groups occurs more frequently in open agricultural landscapes than in heavily forested landscapes (Hirth 1977, Nixon et al. 1991, Mandujano and Gallina 1996, Lingle 2003). Additionally, deer home range size is inversely related to habitat heterogeneity (Kie et al. 2002, Saïd and Servanty 2005, Skuldt 2005, Walter et al. 2009), and overlap of social groups may decrease as home range size

decreases, potentially reducing infectious contact.

Female white-tailed deer form small matriarchal groups rather than herds (Hawkins and Klimstra 1970, Marchinton and Hirth 1984), and contact rates are much higher within groups of related females than between groups (Schauber et al. 2007; S. B. Magle, M. D. Samuel, T. R. Van Deelen, S. J. Robinson, and N. E. Mathews, *unpublished manuscript*). Grear et al. (2010) found that risk of CWD transmission was much higher for related female deer presumably within the same social group. This matriarchal group structure has been suggested as evidence that CWD transmission in deer populations may be frequency-dependent (Gross and Miller 2001, Grear et al. 2010). However, Joly et al. (2006) found a positive relationship between CWD prevalence and the amount of white-tailed deer habitat in Wisconsin (a surrogate for deer density), which appeared to be consistent with density-dependent transmission. In addition, perpetuation of CWD requires transmission between female groups, the sexes, and/or among males, all of which may be density-dependent. Habib et al. (2011) used a mechanistic model of deer contact rates to suggest that CWD transmission could be intermediate between density- and frequency-dependent because contact rates are a saturating function of density.

Transmission functions are rarely estimated in wild populations (but see Caley and Ramsey 2001, Smith et al. 2009). Instead, they are inferred when disease and host dynamics are consistent with a particular transmission function (McCallum et al. 2009) depending on how social organization and behavior of the host (e.g., group size, rate of exchange between groups, between-group contact rate) are related to host density (Altizer et al. 2003, Cross et al. 2009, Habib et al. 2011), or based on the characteristics of the pathogen (e.g., transmission mode, duration of infectivity). A simulation model by Gross and Miller (2001), assuming frequency-dependent transmission, predicted that CWD could drive mule deer populations to extinction. Wasserberg et al. (2009) evaluated both density- and frequency-dependent transmission models of CWD epidemics in white-tailed deer. They found that non-selective culling could eradicate CWD if transmission was density-dependent, but not if it was frequency-dependent. Deer abundance also was projected to be lower under a frequency-dependent scenario (Wasserberg et al. 2009). Thus, whether transmission is frequency- or density-dependent is important for management of CWD in cervids.

Few studies have examined the relationship between host density and disease transmission, and few have considered how landscape variation or environmental factors may influence disease transmission. Understanding this relationship will help guide management decisions, which is especially important in light of the public controversy over the effects of culling and other regulations intended to reduce deer populations (Holsman et al. 2010). Using data on CWD-infection and deer density, we investigated whether CWD transmission in white-tailed deer is better described as frequency-dependent, density-dependent, or by an intermediate form. We used logistic regression models to relate CWD transmission (incidence of CWD in yearling deer) to the frequency (apparent prevalence of CWD in adult deer) and density of infected deer. Additionally, we used a number of landscape metrics to assess whether variation in landscape composition and configuration, and soil clay content influence CWD transmission.

Study Area and Methods

Study area

We studied CWD transmission in a white-tailed deer population in south-central Wisconsin. The outbreak is west of Madison, WI and is centered in western Dane and eastern Iowa counties (Grear et al. 2006, Joly et al. 2006). We focused data collection and analysis within the Wisconsin Department of Natural Resources (WIDNR) designated 'western core area' which was 210 sections (544 km^2) and generally encompassed the areas of highest CWD prevalence (Joly et al. 2006) in the outbreak. The landscape has a rolling topography with deciduous forest on hillsides and hilltops and agricultural crops or pasture in small valleys.

CWD-infection data

Our data on CWD infection in deer came from deer harvested by hunters or professional sharpshooters during February 2002–March 2008. Deer

harvested by sharpshooters constituted a small proportion of the total harvest (≈7%). Registration of harvested deer was mandatory and demographic (sex and age class) data and location of harvest to the quarter section, (0.65 km^2, United States public land survey system) were recorded for each harvested deer. Chronic wasting disease status was determined for deer ≥1 year of age and for most fawns. To test for CWD, WIDNR staff removed the retropharyngeal lymph nodes and obex from harvested deer and personnel at the Wisconsin Veterinary Diagnostic Laboratory used immunohistochemistry or plate ELISA to determine CWD status (Keane et al. 2008). We used the proportion of CWD-positive yearling deer (>1, <2 years of age at harvest) harvested in section *i* (2.59 km^2) as a measure of incidence (probability of acquiring detectable infection) from birth to harvest. This was appropriate because deer were born uninfected (Miller and Williams 2003) and yearlings had not been infected long enough to experience disease-induced mortality (Williams et al. 2002). While exposure time for each individual varied, most deer were harvested during fall, thus the average exposure was approximately 1.5 years. We calculated incidence for each section because harvested deer and deer abundance (below) were recorded at the section scale. This scale also approximated the home range size of deer in this area (<3 km^2; Skuldt 2005). Additionally, these deer were non-migratory, females were highly philopatric, and <50% of yearling males dispersed a relatively short distance (8 km; Skuldt et al. 2008). Thus, we believe the incidence and covariates calculated for a given section approximated the incidence of yearlings living in that area and risk factors of those individuals. Deer spend most, if not all of the first 1.5 years of life in matriarchal groups, thus our findings are most relevant to CWD transmission to deer in matriarchal groups and least relevant to adult males, which have very different social behavior and organization.

The number of yearling deer sampled annually in each section was insufficient to calculate year-specific incidence rates with appropriate precision, so we calculated incidence across the entire, 6-year sample period. We also calculated yearling incidence rates by pooling sexes because CWD-prevalence was similar for male and female yearlings (Grear et al. 2006; Henaux et al., *unpublished*).

The number of adults (≥2 years of age at harvest) that were tested for CWD and the number that were CWD-positive in each section provided the basis for measuring frequency-dependent CWD-infection risk for yearlings in that section. We used both males and females because any infected deer can pose an infection risk, and considering the separate roles of males and females in CWD epidemics requires not only sex-specific prevalence, but also data on the sex structure of the population within each section. As with incidence, we calculated average adult prevalence across the 6-year sample period, because there was no evidence of temporal trend in prevalence during 2002–2007 (Osnas et al. 2009). Harvest vulnerability likely did not differ between uninfected and CWD-infected deer, so prevalence estimates derived from harvested samples were likely unbiased (Grear et al. 2006).

Deer-density

WIDNR staff conducted helicopter counts to determine deer density for each section. All sections were comprehensively surveyed once each year during 3 winters (2006–2008) using standard survey protocols (Storm et al. 2011). Adequate snow cover was required to determine deer abundance during helicopter surveys (Storm et al. 2011). In 2006, 146 of 210 sections were surveyed with inadequate snow cover, which substantially reduced the number of deer counted (Storm et al. 2011). Therefore, deer abundance data from sections with poor snow cover were not included in any analysis. We reconstructed pre-hunt deer densities by adding deer harvested prior to the helicopter survey to the number of deer counted in each section. We found no deer population trend in total helicopter counts for the core from 2006–2008, and thus considered them representative of the deer densities in those sections over the study period.

Landscape

We used landcover data (raster-format; 30-m^2 resolution) obtained from the National Land Cover Database 2001 (NLCD 2001) derived from Landsat 5 and 7 imagery (Homer et al. 2004). We extracted the landcover data and calculated landscape metrics for each section. We used

Fragstats (McGarigal and Marks 1995) to calculate the following landscape metrics as potential explanatory variables for CWD incidence: % deciduous forest, edge density of forest cover, mean shape index of forest patches. Edge density of forest cover is the length of edge between forest cover and other landcover types, relative to the total area analyzed (McGarigal and Marks 1995). Mean shape index measures patch compactness, and is the patch perimeter relative to the perimeter of a maximally compact patch of the same size, averaged across forest patches (McGarigal and Marks 1995). Both indices are negatively related to home range size of deer (Kie et al. 2002, Skuldt 2005) and likely relate to the degree of resource interspersion.

Soil clay content

We obtained soil data from NRCS Soil Survey Spatial and Tabular Data for counties around the CWD core area (Soil Survey Staff 2012), and used the Soil Data Viewer tool for ArcMap to query the soil dataset and summarize the percent clay content in the soil surface layer by soil map unit. Because soil map units were irregular in shape and size, we used the Feature to Raster tool to create a raster dataset of percent clay values (cell size 200 × 200 m). Raster cells were then averaged for each section (using the Zonal Statistics as Table tool) to provide a measure of mean clay content on a scale comparable with the CWD data.

Analysis

We used a Bayesian framework to evaluate alternative models reflecting hypotheses of disease transmission and the potential influence of landscape characteristics on incidence of CWD in yearling white-tailed deer. Measurement error in covariates is common for ecological data and ignoring this uncertainty in regression analyses can result in biased parameter estimates with underestimated variances (Gustafson 2003, Denham et al. 2011). We incorporated this uncertainty into our regression analyses, by treating key covariates as random variables which were estimated from our data. Our approach is similar to that taken by Kamarainen et al. (2008), who modeled measurement error in zooplankton biomass from replicate measurements. We used the logit link to model the relationship of CWD incidence for yearlings in section i for density-dependent, frequency-dependent, and non-linear (intermediate transmission) models.

Our intermediate model was:

$$\text{logit}(\pi_i) = \beta_0 + \frac{\beta_1 I_i}{1 - \beta_2 + \beta_2 d_i} + \beta_3 x_i$$

$$\Pr(\boldsymbol{\beta}, \boldsymbol{\pi}, \mathbf{P}, \mathbf{d}, \sigma, I | \mathbf{y}, \mathbf{n}, \mathbf{A}, \mathbf{T}, \mathbf{D}, \mathbf{x}) \propto$$

$$\prod_{i=1}^{205} \text{binomial}(y_i | \pi_i, n_i) \prod_{i=1}^{205} \text{binomial}(A_i |\ P_i, T_i)$$
$$\prod_{i=1}^{205} \prod_{j=1}^{3} \text{normal}(D_{ij} |\ d_i, \sigma_i)$$

$$\times$$

$$\prod_{i=1}^{205} \text{uniform}(P_i |\ 0, 1) \text{uniform}(d_i |\ 1, 100)$$
$$\text{uniform}(\sigma_i |\ 0, 100)$$

$$\times$$

$$\text{normal}(\beta_0 | 0, 1000) \text{normal}(\beta_1 | 0, 1000)$$
$$\text{uniform}(\beta_2 | 0, 1) \text{normal}(\beta_3 | 1, 1000)$$

where $\boldsymbol{\beta}$ is the vector of regression coefficients; $\boldsymbol{\pi}$, $\mathbf{P}$, and $\mathbf{d}$ are vectors of the incidence of infected yearling deer, prevalence of adult deer, and mean deer density for sections ($i = 1, \ldots, 205$), respectively. $\mathbf{I}$ is the independent variable vector of the number of infected deer ($I_i = P_i \times d_i$). The vectors $\mathbf{y}$, $\mathbf{n}$, $\mathbf{A}$, $\mathbf{T}$, and $\mathbf{x}$ are the observed number of infected yearlings, number of CWD-tested yearlings, number of infected adults, number of CWD-tested adults, and a specific landscape covariate, respectively. $\mathbf{D}(D_{ij})$ is the matrix of the number of deer counted in section i during 3 years ($j = 1, ..., 3$). β_2 is a dimensionless scaling constant between 0 and 1. In this model $\beta_2 = 0$ corresponds to density-dependence and $\beta_2 = 1$ corresponds to frequency-dependence. Intermediate values of β_2 indicate how quickly an asymptote is reached, with the exact shape determined by plotting model predictions. This model allows for nonlinear relationships, intermediate between linear density-dependence and density-independence, and is similar in form to the contact rate function found in Roberts (1996) and McCallum et al. (2001).

We used a logit model with adult CWD prevalence (P_i) as the linear covariate to repre-

sent a frequency-dependent transmission hypothesis:

$$\text{logit}(\pi_i) = \beta_0 + \beta_1 P_i + \beta_3 x_i,$$

and a logit model with density of infected deer (I_i) to represent density-dependent transmission:

$$\text{logit}(\pi_i) = \beta_0 + \beta_1 I_i + \beta_3 x_i.$$

We fit models in which each of the landscape metrics (including soil-clay content) was a covariate along with each of the 3 transmission hypotheses. We fit models using WinBUGS 1.4 (Lunn et al. 2000). We sampled from 4 Markov chains with widely divergent starting values, used a 40,000 iteration burn-in period, and sampled from an additional 10,000 iterations. We examined MCMC chains, Brooks-Gelman-Rubin plots, and autocorrelation plots to ensure MCMC chains converged. We summarized posterior distributions of the model parameter estimates using the mean of the posterior and the 95% Bayesian credible interval (BCI). We used Deviance Information Criteria (DIC; Spiegelhalter et al. 2002) to determine which models had the greatest support for our data. The model with the lowest DIC value is deemed the 'best' of the model set (Spiegelhalter et al. 2002), models with $\Delta\text{DIC} < 2$ are nearly as well supported, and model support declines with increasing ΔDIC. The Deviance information criteria is considered a Bayesian analogue to Akaike's information criterion (AIC) (Spiegelhalter et al. 2002), and like AIC can favor models with more parameters over simpler models (Link and Barker 2006). We used DIC because of ease of interpretation, widespread familiarity with information-theoretic methods, and the use of DIC in similar investigations (Farnsworth et al. 2006, Osnas et al. 2009, Heisey et al. 2010, Walter et al. 2011). We assessed whether 95% BCIs overlapped 0. We used the Bayesian P-value to evaluate goodness-of-fit of our models, based on the proportion of times the discrepancy measure (Pearson's residual) of a simulated data (generated from posterior distributions of parameter estimates) exceeded the discrepancy measure from our observations (Kéry 2010). Bayesian P-values near 0 or 1 indicate serious lack of fit (Gelman et al. 2004). In regression models of spatial data, residual error may be spatially autocorrelated, causing overly precise parameter estimates and increased Type I error (Diniz-Filho et al. 2008). We tested for the presence of spatial autocorrelation of Pearson residuals using Moran's I.

Results

Ninety-five of 3,901 yearlings (2.44%) tested CWD-positive during 2002–2007. Mean number of yearlings per section was 18.58 (SE = 0.82, range = 0–56). Four sections did not have any yearling deer sampled during our study. Mean adult prevalence (P) was 6.14% (SE = 0.49, range = 0.00–50.00%), and mean number of infected adults (I) was 2.14 (SE = 0.19, range = 0.00–14.71). Mean sampling intensity of adult deer was 32.05 per section (SE = 1.46, range = 0–121). Mean (SE) % deciduous forest, edge density of forest cover, mean shape index of forest patches, and mean % soil clay content were 40.22 (1.53), 62.26 (1.70), 1.74 (0.03), and 14.70 (0.42), respectively.

The intermediate model with % deciduous forest had the lowest DIC value followed by the intermediate model with edge density and the frequency-dependent (P) model with % deciduous forest ($\Delta\text{DIC} < 2$: Table 1). Among the 3 transmission models without landscape variables, the intermediate model had the lowest DIC and the frequency-dependent had the least support. Models including % deciduous forest and edge density of forest always had lower DIC values than the corresponding transmission models without landscape variables. The 95% BCIs of % deciduous forest and edge density did not overlap 0 in the intermediate and frequency-dependent models, but did in the density-dependent model. DIC values suggested similar data prediction from intermediate models with % deciduous forest cover or with edge density; however, these landscape variables are highly correlated (r = 0.69), indicating both models are likely identifying related landscape characteristics. The intermediate models tended to have the lowest DIC values and the shape parameter estimates in intermediate models were not close to 0 or 1. For these reasons, we concluded that yearling incidence is a complex function of the density of infected deer and CWD prevalence (β_1 = 3.861, 95% BCI = 0.651, 7.62; β_2 = 0.591, 95% BCI = 0.080, 0.983), and is positively related to % deciduous forest (β_3 = 0.018, 95% BCI = 0.005,

Table 1. Results of model selection to identify which candidate model best explains incidence of chronic wasting disease (CWD) in yearling white-tailed deer during 2001–2007 in Wisconsin, USA.

Model	pD†	DIC‡	ΔDIC
Intermediate	8.85	308.19	2.56
Intermediate + % deciduous forest	11.94	305.63	0.00
Intermediate + edge density of forest cover	10.98	307.00	1.37
Intermediate + mean shape index of forest patches	9.58	309.71	4.08
Intermediate + % clay content	8.98	308.51	2.88
I	11.92	309.33	3.70
I + % clay content	11.53	309.06	3.43
I + % deciduous forest	11.80	308.48	2.85
I + edge density of forest cover	11.99	308.10	2.47
I + mean shape index of forest patches	13.16	310.92	5.29
P	9.85	312.66	7.03
P + % deciduous forest	11.85	306.63	1.00
P + edge density of forest cover	10.85	308.32	2.69
P + mean shape index of forest patches	10.68	314.20	8.57
P + % clay content	9.40	311.09	5.46

†pD is a Bayesian measure of the effective number of model parameters.
‡DIC is the deviance information criteria.

0.032). Bayesian P-values were not close to 0 or 1, suggesting our models did not suffer from serious lack of fit. Moran's I of the Pearson's residuals for the top model ($P > 0.56$) indicated that there was not significant spatial autocorrelation of residual errors.

Discussion

An intermediate transmission model with deciduous forest cover best predicted CWD transmission in yearling white-tailed deer. Our intermediate model indicated that yearling incidence is a non-linear function of both frequency of disease and density of infected deer (Fig. 1). Yearling incidence is a saturating function of the density of infected individuals, but the strength of this relationship depends on prevalence. At low deer density (e.g., <5–10 deer/mi^2), disease transmission increases in a density-dependent manner until it reaches a saturation (or frequency-dependent) level. Disease transmission as a saturating function of density has been suggested as an alternative to linear density- and frequency-dependence (Antonovics et al. 1995, Roberts 1996), and has empirical support from other systems (Cross et al. 2009, Smith et al. 2009, Habib et al. 2011). Our findings support the notion that classic frequency- and density-dependent models inadequately represent disease transmission processes in hosts with complex social behavior.

Our results generally correspond with current knowledge about deer social organization and behavior. Rates of between-group contacts increase with deer density (Habib et al. 2011) because spatial overlap of social groups increases and contact rates increase with spatial overlap (Schauber et al. 2007). While rates of between-group contacts (and thus probably CWD transmission) are a positive function of deer density, total contact rates are non-linearly related to density (Habib et al. 2011). The strong matriarchal social structure of white-tailed deer and other cervids prevents homogenous mixing of individuals (Schauber et al. 2007) and homogeneous CWD transmission (Grear et al. 2010), which also precludes classic density-dependent disease transmission. The relative importance of between- and within- group transmission (or infectious contact) can also influence the transmission function. When within-group contacts constitute a large proportion of the infectious contacts, the contact rate-density relationship saturates more quickly and becomes independent of density for a larger portion of the density spectrum. For white-tailed deer, within-group contact rates may be 10–30 fold higher than between groups (Schauber et al. 2007; S. B. Magle, M. D. Samuel, T. R. Van Deelen, S. J. Robinson, and N. E. Mathews, *unpublished manuscript*). In contrast, Grear et al. (2010) reported that CWD transmission probabilities were ≥100 fold higher for related female deer

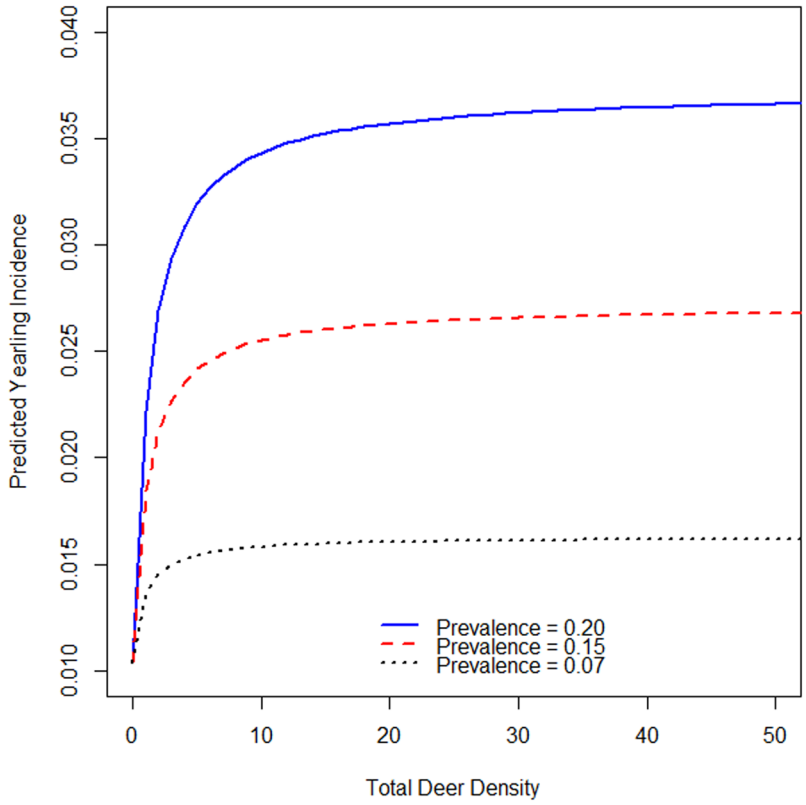

Fig. 1. Predicted incidence of CWD in yearling white-tailed deer during 2001–2007 in Wisconsin from the intermediate transmission model, as a function of deer density (deer/mile^2), at 3 levels of disease prevalence and median % deciduous forest cover (44.2%).

within 3.2 km (same social group) than for unrelated deer in the same area, indicating that within group contacts have a higher probability of producing infection than contacts between members of different social groups. Our observation of quick saturation in the incidence-density relationship may be further evidence that infectious contact rates are much higher within groups than between groups.

Transmission that is influenced simultaneously by prevalence and density is not a usual feature of disease models. Rather, this pattern likely reflects how deer and infection are distributed in social groups, and how infectious contact influences the immediate infection risk for a yearling deer. Females and young white-tailed deer live in small social groups where contact rates with group members are much higher than with other deer (Schauber et al. 2007; S. B. Magle, M. D. Samuel, T. R. Van Deelen, S. J. Robinson, and N. E. Mathews, *unpublished manuscript*), and infected individuals pose the greatest risk to those in the same social group (Grear et al. 2010). Thus, at higher deer density and low prevalence, most of the infection risk likely results from within social group contact; however, as prevalence increases the risk of infectious contact with deer outside the social group also contributes to the total risk of infection. This relationship may also be affected by deer density. Consider a single infected deer in a low density population (higher apparent prevalence) versus an infected deer in a high density population (lower apparent prevalence). While the total contact rates may be lower in the low density population, the infection risk of a random susceptible deer is likely higher in the low density population, because it is more likely to share a group with the infected deer. This highlights the importance of heterogeneity of infectious contact in disease transmission (Drewe 2010), in particular higher probability of CWD transmission because of the more intense

nature of contacts among related deer (Hirth 1977, Nelson and Mech 1999, Grear et al. 2010), and suggests research will be needed to understand how CWD infection occurs within and between social groups and how these factors change with population density.

We used incidence in yearlings to measure CWD transmission because this represents a discrete period of disease risk from birth of susceptible fawns to yearling harvest that is not likely complicated by disease mortality. Yearlings have lower prevalence of infection than adults (Miller and Conner 2005, Grear et al. 2006), which may be due partly to cumulative risk of infection, but could also result from different transmission rates or mechanisms. Because there is a 3–4 month lag between infection and detection of CWD (Sigurdson et al. 1999), CWD positive yearlings likely became infected while in their mother's social group and prior to dispersal, which occurs near their first birthday (spring) or the following fall. Social interactions among deer vary by sex, age, and season; thus CWD transmission likely results from a complex of mechanisms, including between-group and within-group transmission, transmission between and within sexes, and indirect transmission from an environmental reservoir. While we cannot evaluate these mechanisms separately, their composite effect is that CWD transmission to yearlings does not conform to either classic frequency- or density-dependence. Because young deer primarily interact with deer in their mother's social group (S. B. Magle, M. D. Samuel, T. R. Van Deelen, S. J. Robinson, and N. E. Mathews, *unpublished manuscript*), patterns of yearling infection are likely similar to those found among adult female deer.

Using methods similar to those described in this paper, we also analyzed data from a CWD outbreak in a white-tailed deer population in southeastern Wisconsin and northeastern Illinois (Storm 2011). In this analysis, regression coefficients indicated no statistically significant relationship between yearling incidence and the covariates for any of the models, and for some models, the posterior distribution of parameters closely matched their prior distributions (Storm 2011). These results suggest that the data from this outbreak were not sufficient to answer our research questions, which highlights the data-intensive nature of this type of analysis.

We hypothesized that landscape variation on the scale of our sampling units (approximate deer neighborhoods) would affect CWD transmission by influencing grouping behavior, spatial overlap, and contact rates. We expected incidence would be negatively related to % deciduous forest and edge density of forest because smaller home range size (Kie et al. 2002, Walter et al. 2009) and group-size (Hirth 1977, Nixon et al. 1991) would reduce between-group contact rates (Habib et al. 2011). Instead, we found evidence that incidence was positively related to % deciduous forest and edge density of forest. These landscape variables are closely related to deer habitat quality, which Joly et al. (2006) found was related to CWD prevalence. Further research will be needed to understand how landscape features affect deer behavior and risk of CWD transmission.

In contrast to Walter et al. (2011), we did not find that soil clay content was related to CWD incidence. However, soil clay content in our study area was not highly variable (half of sections had mean soil clay content values between 12% and 19%), thus our study may not be well suited to evaluating how clay soils might influence indirect transmission of CWD. Whether soils play an important role in facilitating indirect transmission of CWD in our study area remains an unresolved issue. In general, the relative importance of direct and indirect transmission (via an environmental reservoir) of CWD in wild deer populations remains unknown.

The rationale for disease eradication through host culling derives from the concept of a host density threshold, below which a disease cannot persist (Barlow 1996, Smith and Cheeseman 2002). Our study provides evidence that CWD transmission in yearling white-tailed deer increases substantially with population prevalence, but rapidly saturates with increasing density, which lowers the host-density threshold compared to linear density-dependent transmission (Barlow 1996). Based on infection patterns in young deer, density reduction of white-tailed deer in the WI outbreak through non-selective culling may reduce incidence, but primarily when deer densities are relatively low. In contrast, selective removal of infected deer would simultaneous reduce density of infected deer and

prevalence, and hence, CWD incidence. Our model suggests that management strategies to reduce population prevalence will be more effective in reducing CWD transmission than efforts to reduce deer abundance. Selective culling strategies, however, such as "test-and-cull" (Wolfe et al. 2004) are impractical on large spatial scales. Alternatively, focused culling in areas with high densities of infecteds, while non-selective at local scales, could impact outbreaks by reducing local transmission rates, numbers of infected dispersers, and deposition of prions into the environment. In addition, efforts to reduce the size of female social groups (Grear et al. 2010) or to remove entire female groups which have infected deer (Schauber et al. 2007, Habib et al. 2011) are potential management strategies that deserve further evaluation.

Few studies have evaluated the functional relationship between host density and infectious contacts (Cross et al. 2009) and evidence for host population thresholds in wildlife disease remains limited (Lloyd-Smith et al. 2005). This paucity of evidence in support of density- dependence or population thresholds is not surprising considering the difficulties in collecting data on disease transmission across a range of densities in wildlife populations (Cross et al. 2009). Our study provides critical information regarding the relationship between CWD transmission, deer density, and disease prevalence. Our results indicate that CWD transmission in yearlings increases substantially with population prevalence. However, this transmission patterns also has density-dependent components, which saturate quickly with deer density. It seems likely that these non-linear patterns of disease transmission in young deer are driven by three interacting factors: limitation of infectious contacts between female social groups, non-linear increases in contact rates with population density (Habib et al. 2011), and the distribution of infection among social groups. Although the social organization and behavior of deer have been extensively studied (Hirth 1977, Forand and Marchinton 1989, Ozoga 1989), knowledge about these processes in the context of changing density and environmental gradients is limited (but see Silbernagel et al. 2010, Habib et al. 2011).

Many recent studies have added to our understanding of CWD prevalence and transmission patterns in cervids, and as a result to our general understanding of how social factors, host density, environmental reservoirs, and host demographics affect wildlife disease patterns. Despite these advances, critical gaps are evident in our understanding of CWD, which can be transmitted from both direct contact and an environmental reservoir. Despite the importance of multiple transmission routes to future CWD infection patterns (Wasserberg et al. 2009, Almberg et al. 2011), we do not know the relative contributions of these factors in free-ranging populations. If infectious contacts are driven by contaminated environments, more information is required regarding the likely sources of concentrated prion contamination (e.g., mineral licks, scrapes, bait sites). Sampling the environment for prions (Maddison et al. 2010), especially across a range of densities of infected deer, could help determine the availability and importance of environmental sources of prions to CWD transmission. In general, we believe the role of environmental transmission in wild deer populations is a critical area of needed research to determine the future dynamics of CWD epidemics (Wasserberg et al. 2009, Almberg et al. 2011). Additionally, we know little about how CWD is transmitted between social groups of female deer or in adult male deer, despite their higher rates of prevalence (Miller and Conner 2005, Grear et al. 2006, Osnas et al. 2009). Whether male deer become infected by females, from males in bachelor groups, or via environmental routes (Grear et al. 2006) is unknown but important to developing potential disease management strategies.

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