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Effects of Chronic Wasting Disease on Reproduction and Fawn Harvest Vulnerability in Wisconsin White-Tailed Deer

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Abstract

Chronic wasting disease (CWD) is a fatal, transmissible spongiform encephalopathy that affects free-ranging and captive North American cervids. Although the impacts of CWD on cervid survival have been documented, little is known about the disease impacts on reproduction and recruitment. We used genetic methods and harvest data (2002–04) to reconstruct parentage for a cohort of white-tailed deer (Odocoileus virginianus) fawns born in spring 2002 and evaluate the effects of CWD infection on reproduction and fawn harvest vulnerability. There was no difference between CWD-positive and CWD-negative male deer in the probability of being a parent. However, CWD-positive females were more likely to be parents than CWD-negative females. Because our results are based on harvested animals, we evaluated the hypothesis that higher parentage rates occurred because fawns with CWD-positive mothers were more vulnerable to harvest. Male fawns with CWD-positive mothers were harvested earlier (.1 mo relative to their mother's date of harvest) and farther away from their mothers than male fawns with CWDnegative mothers. Male fawns with CWD-positive mothers were also harvested much earlier and farther away than female fawns from CWD-positive mothers. Most female fawns (86%) with CWD-positive mothers were harvested from the same section as their mothers, while almost half of male and female fawns with CWD-negative mothers were farther away. We conclude that preclinical stages of CWD infection do not prohibit white-tailed deer from successfully reproducing. However, apparently higher harvest vulnerability of male fawns with CWD-positive mothers suggests that CWD infection may make females less capable of providing adequate parental care to ensure the survival and recruitment of their fawns.

Keywords

Chronic wasting disease, harvest vulnerability, microsatellites, Odocoileus virginianus, parentage, white-tailed deer

Comments

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EFFECTS OF CHRONIC WASTING DISEASE ON REPRODUCTION AND FAWN HARVEST VULNERABILITY IN WISCONSIN WHITE-TAILED DEER

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ABSTRACT: Chronic wasting disease (CWD) is a fatal, transmissible spongiform encephalopathy that affects free-ranging and captive North American cervids. Although the impacts of CWD on cervid survival have been documented, little is known about the disease impacts on reproduction and recruitment. We used genetic methods and harvest data (2002–04) to reconstruct parentage for a cohort of white-tailed deer (*Odocoileus virginianus*) fawns born in spring 2002 and evaluate the effects of CWD infection on reproduction and fawn harvest vulnerability. There was no difference between CWD-positive and CWD-negative male deer in the probability of being a parent. However, CWD-positive females were more likely to be parents than CWD-negative females. Because our results are based on harvested animals, we evaluated the hypothesis that higher parentage rates occurred because fawns with CWD-positive mothers were more vulnerable to harvest. Male fawns with CWD-positive mothers were harvested earlier (>1 mo relative to their mother's date of harvest) and farther away from their mothers than male fawns with CWD-negative mothers. Male fawns with CWD-positive mothers were also harvested much earlier and farther away than female fawns from CWD-positive mothers. Most female fawns (86%) with CWD-positive mothers were harvested from the same section as their mothers, while almost half of male and female fawns with CWD-negative mothers were farther away. We conclude that preclinical stages of CWD infection do not prohibit white-tailed deer from successfully reproducing. However, apparently higher harvest vulnerability of male fawns with CWD-positive mothers suggests that CWD infection may make females less capable of providing adequate parental care to ensure the survival and recruitment of their fawns.

Key words: Chronic wasting disease, harvest vulnerability, microsatellites, *Odocoileus virginianus*, parentage, white-tailed deer.

INTRODUCTION

Infectious diseases are now recognized for their important roles in natural systems, with potential impacts on biological conservation and biodiversity (Daszak et al., 2000; Harvell et al., 2002). Disease can have significant direct and indirect impacts on wildlife by affecting both mortality and fecundity rates (Wobeser, 2006). Determining disease dynamics such as annual or seasonal rates of transmission and associated demographic impacts in wildlife populations, however, presents significant ecologic and epidemiologic challenges (McCallum et al., 2001; Wobeser, 2008). Additionally, it can be especially difficult to determine whether pathogens indirectly affect recruitment of young animals into a population by affecting breeding success or behavior associated with caring for young (Wobeser, 2006). Nonetheless, this information is essential for understanding population-level impacts of diseases and formulating disease management plans.

Chronic wasting disease (CWD) is a fatal, transmissible spongiform encephalopathy affecting free-ranging and captive North

American cervids including elk (*Cervus elaphus*), moose (*Alces alces*), mule deer (*Odocoileus hemionus*), and white-tailed deer (*Odocoileus virginianus*; Miller et al., 2000; Baeten et al., 2007). The probable causative agent of CWD is an abnormal prion protein (PrP^{CWD}). The course of CWD varies among animals and prion protein (Prnp) genotypes but averages approximately 2 yr from infection to death (Williams et al., 2002; Fox et al., 2006). In captive mule deer PrP^{CWD} accumulates in lymph nodes about 1.5–3 mo after infection (Sigurdson et al., 1999) and roughly 6 mo later becomes detectable in the obex portion of the brainstem (Williams and Miller, 2002). Within 10–12 mo after obex infection, clinical signs become apparent (Williams and Miller, 2002) including emaciation; loss of fear of humans; drooping head and ears; excessive drinking, urination, and salivation; dehydration; lethargy; and ataxia (Spraker et al., 1997; Williams, 2005). Once clinical signs appear, death occurs within a few weeks to 1 yr (Williams et al., 2002; Fox et al., 2006).

Because CWD is always fatal it can directly impact cervids by significantly reducing survival of infected individuals (Edmunds, 2008) and cause population declines where the disease is at high prevalence (Miller et al., 2008). However, the disease threshold required to reduce cervid populations likely varies based on species demography, CWD prevalence, and other sources of mortality (Miller et al., 2000; Schuler, 2006; Edmunds, 2008; Wasserberg et al., 2009). Another important, but poorly explored, mechanism by which CWD could affect cervid populations is by reducing reproduction and recruitment (Dulberger et al., 2010). Breeding in white-tailed deer occurs in the fall. Male deer are polygamous and compete with each other for access to females often through vigorous sparring. During courtship, a male deer chases a female, tends her over a roughly 24-hr estrous period, and defends her against other males (Marchinton and Hirth, 1984). CWD may negatively affect a male deer's ability to successfully compete for and defend females. In the case of infected females, although it is unlikely that CWD is transmitted to prenatal offspring (Williams, 2005), young cervids could become infected from close contact with their infected mothers (Argue et al., 2007). In addition, indirect effects of CWD on recruitment are plausible because behavioral changes during the clinical stages of CWD infection (brain spongiform lesions) could reduce the quality of maternal care a female provides. Subtle behavioral changes in preclinical deer may also affect an animal's ability to care for its offspring leading to reduced fawn recruitment as recently reported for mule deer (Dulberger et al., 2010). This lack of care may result in increased mortality for fawns with CWD-positive mothers through mechanisms such as increased vulnerability to predation or harvest.

We hypothesized that CWD infection in white-tailed deer would negatively affect reproduction and increase harvest vulnerability of fawns with CWD-positive mothers. We used genetic methods to reconstruct parentage for a cohort of fawns conceived during the 2001 breeding season, born in spring 2002, and subsequently harvested in south-central Wisconsin where CWD is endemic. Our specific objectives were to 1) compare the probability of being assigned as a parent between CWD-positive and CWD-negative deer and 2) compare characteristics associated with harvest vulnerability (i.e., timing, distance separating fawn and mother) between fawns with CWD-positive mothers and fawns with CWD-negative mothers. Given the behavioral changes associated with CWD infection, we hypothesized 1) that a deer's ability to produce offspring would be lower for CWD-positive deer relative to CWD-negative deer and 2) that fawns with CWD-positive mothers would be harvested earlier and farther away from their

mothers relative to fawns with CWD-negative mothers.

MATERIALS AND METHODS

Sample and genetic data collection

We collected skeletal muscle and retropharyngeal lymph node tissues from hunter-harvested deer within a 285-km^2 region of south-central Wisconsin (centered on 43°6′39.5995″N, 89°50′52.7994″W) where CWD prevalence in adult deer was 6–7% (Grear et al., 2006; Joly et al., 2006). Deer were sampled during extended hunting seasons (September–March) instituted by the Wisconsin Department of Natural Resources (WDNR) to reduce overall deer abundance following the discovery of CWD. Specifically, we selected deer harvested during the 2002, 2003, and 2004 seasons that, based on their age at harvest, were likely participants in the 2001 fall breeding season (age in 2001: fawns and older for females, yearlings and older for males) or were offspring born in spring 2002 (henceforth referred to as "fawns"). Animals that were harvested by WDNR sharpshooters were excluded from this analysis because this type of harvest was biased toward areas with high CWD prevalence or animals showing clinical signs associated with CWD. Tissues were placed in 95% ethanol in 1.5-ml microcentrifuge tubes and stored frozen at −20 C until processing.

We identified sex, age, and location of harvest to the section (2.6 km^2) for all deer. Deer age was determined by tooth replacement and wear (Severinghaus, 1949). We converted deer age categories (fawn, 1.5, 2.5, 3.5, 4.5–5.5, 6.5–8.5, 9.5–11.5, >12.5) to a continuous variable by assuming all deer were born on 15 May, and calculating the amount of time in years between 15 May of the birth year and date of death (Osnas et al., 2009). For deer whose age category spanned multiple years, we used the midpoint of the age interval (e.g., deer in the age category 6.5–8.5 were assigned an age of 7.5). Aging deer by tooth wear has errors (Storm, 2011) so that some adult animals may have been aged incorrectly, causing us to misclassify them with respect to whether they were "potential parents" or "potential fawns." However, fawns and adults can be reliably distinguished by tooth wear and physical appearance; therefore, we are very confident in the categorization of fawns and parents harvested in 2002 (the first harvest season following the fall 2001 breeding season). To account for the potential errors associated with incorrect aging, we conducted our analyses 1) using animals harvested across all years and 2) using only animals harvested in 2002.

Retropharyngeal lymph nodes and the obex portion of the brainstem for all adults were sent to the Wisconsin Veterinary Diagnostic Laboratory, where CWD-infection status was determined using immunohistochemistry (Johnson et al., 2006; Keane et al., 2008a). A deer was determined to be CWD-positive at harvest if abnormal prions (PrP^{CWD}) were detected in lymph nodes or obex. In white-tailed deer, PrP^{CWD} accumulates in retropharyngeal lymph nodes prior to invasion and accumulation in the central nervous system (Keane et al., 2008b). Based on this progression, the approximate stage of infection at harvest was determined for each CWD-infected animal as follows: 1) accumulation of PrP^{CWD} in lymph node tissue only (henceforth referred to as lymph only); 2) accumulation of PrP^{CWD} in lymph node tissue and the dorsal motor nucleus of the vagal nerve, but not in surrounding tissue (henceforth referred to as obex +); or 3) accumulation of PrP^{CWD} in lymph node and throughout the obex (henceforth referred to as obex ++). Obex + is equivalent to obex scores 1 and 2; obex ++ is equivalent to obex scores 3 and 4 of Keane et al. (2008a). To our knowledge, none of the CWD-positive deer in our study exhibited clinical signs of disease.

We extracted DNA from deer tissue samples using Qiagen DNEasy Blood and Tissue Kits. We used methods described by Grear et al. (2010) to genotype deer at 11 highly polymorphic, biparentally inherited microsatellite loci (BM1225, BM4107, BM4208, BM6506, Bishop et al., 1994; Cervid1, Cervid2, DeWoody et al., 1995; IGF1, Kirkpatrick, 1992; RT7, RT9, RT23, RT27, Wilson et al., 1997).

Parentage reconstruction

We determined maternity and paternity of fawns using the likelihood-based parentage reconstruction program Cervus 3.0 (Marshall et al., 1998; Kalinowski et al., 2006). Program inputs included the approximate number of candidate mothers (fathers) in the population, the approximate proportion of females (males) in the population that were sampled, the proportion of loci successfully typed, and an estimate of genotyping error rate. For each fawn, we first calculated the likelihood ratio of parentage for each candidate parent. This ratio was based on the probability of the fawn's genotype given the candidate parent was the true parent divided by the probability of the

TABLE 1. Number of male and female candidate parents classified as chronic wasting disease negative (CWD−) or CWD-positive (CWD+) at breeding (2001) as a function of harvest year and disease stage at the time of harvest, for white-tailed deer (*Odocoileus virginianus*) in Wisconsin.

	CWD− at breeding[a]					CWD+ at breeding[a]		
CWD status at harvest	Not infected	Lymph only	2003 Obex+	2004 Obex+, Obex++	Total	2002 Obex+, Obex++	2003 Obex++	Total
Males	446	6	2	2	456	41	5	46
Females	1,210	15	5	16	1,246	43	7	50

[a] Obex+ indicates accumulation of PrP^{CWD} in lymph node tissue and the dorsal motor nucleus of the vagal nerve, but not in surrounding tissue; Obex ++ indicates accumulation of PrP^{CWD} in lymph node tissue and throughout the obex.

fawn's genotype given the candidate parent was an unrelated individual of the same multilocus genotype in the population. We calculated LOD (natural logarithm of the odds ratio) scores for each potential parent and classified the candidate parent with the highest positive LOD score as the most likely parent of a given fawn (Marshall et al., 1998; Kalinowski et al., 2006).

We also calculated delta scores, the difference in LOD scores between the most likely parent and the second most likely parent, to evaluate the reliability of our LOD assigned parentage. We simulated a distribution of delta scores for tests in which the most likely parent was the true parent and a distribution of delta scores for tests in which the most likely parent was not the true parent (Marshall et al., 1998; Kalinowski et al., 2006). For parentage analysis, we estimated the significance of individual delta scores by comparing them against the delta distributions obtained by these simulations.

For each fawn we conducted two parentage analyses, one to assign maternity and one to assign paternity. In each case the parent of the opposite sex was considered unknown. We assigned mothers/fathers to fawns for which two criteria were met: 1) a parent was assigned with ≥80% confidence and 2) a fawn and assigned parent were separated by ≤6 km, the maximum distance over which female deer in our study area are genetically spatially autocorrelated (i.e., significantly higher interindividual relatedness than random; Grear et al., 2010). We chose an 80% confidence threshold to identify enough female parent-offspring pairs to provide sufficient statistical power for subsequent analyses of fawn harvest vulnerability. This 80% confidence threshold increases our chances of including erroneously identified parent-offspring pairs but also increases our chances of including true parent-offspring pairs. The inclusion of erroneous parent-offspring pairs likely dilutes our ability to find significant differences between CWD-positive and CWD-negative deer by adding additional variation (noise) to our data, making our test more conservative. The distance criterion was important to enhance our confidence in mother-fawn pairs used to evaluate fawn harvest vulnerability.

CWD status at breeding

CWD status at the time of breeding (fall 2001) was determined by backward calculation from a candidate parent's CWD status, stage of infection, and the year of harvest (Table 1). For all harvest seasons (2002–04), CWD-negative deer were categorized as CWD-negative at breeding in 2001 because no animals have ever been demonstrated to clear the infection (Williams and Miller, 2002). CWD-positive lymph-only deer were also categorized as CWD-negative at breeding because PrP^{CWD} accumulation in the obex is detectable roughly 6 mo after lymph node infection (Williams and Miller, 2002). Because CWD-positive deer generally do not survive more than 2 yr after infection, all candidate parents harvested in the 2004 season were also categorized as CWD-negative at breeding in 2001. Candidate parents harvested in the 2002 season with obex + or obex ++ positive were categorized as CWD-positive at breeding. Candidate parents harvested in the 2003 season with obex + positive were categorized as CWD-negative at breeding because PrP^{CWD} accumulation in the obex generally occurs within 12 mo after infection (Sigurdson et al., 1999; Fox et al., 2006), making it unlikely that CWD-positive deer in fall 2001 would be at an early stage of PrP^{CWD} accumulation (obex +) 2 yr later. Candidate parents harvested in the 2003 season with obex ++ positive at harvest were categorized as CWD-positive at breeding, although we acknowledge this classification is somewhat subjective. Several studies have demonstrated that Prnp genotype influences CWD progression in mule and white-tailed deer (Fox et al.,

2006; Johnson et al., 2006; Wolfe et al., 2007; Johnson et al., 2011). There are no specific data published, however, about how CWD progresses over time in the different Prnp genotypes in white-tailed deer except for time to clinical disease and death (Wilson et al., 2009; Johnson et al., 2011). In this study, therefore, we used average rates of disease progression that are believed to be common in our study area (Wasserberg et al., 2009).

CWD status and parentage

We used logistic regression to predict the probability of being assigned as a parent depending on CWD status at the time of breeding, age at breeding, and the number of deer harvested from the section. We conducted analyses separately for candidate female parents and candidate male parents. Because retrospective determination of CWD status is partly subjective and Prnp genotype or dose may be related to the rate of disease accumulation in the obex (Johnson et al., 2006; Keane et al., 2008a), we also conducted these analyses using only candidate parents and fawns harvested during the 2002 season. We were most confident in our classifications of infection status of these deer at breeding (i.e., lymph only positive in 2002=CWD-negative in 2001 [n=16] and obex + and obex ++ positive in 2002=CWD-positive in 2001 [n=84]).

We included age as a covariate because studies on cervids have demonstrated that breeding opportunities and number of offspring for males and females generally increase with age (e.g., San Jose et al., 1999; DeYoung et al., 2006). We also expected older females would be more likely to be assigned as parents because their previous maternal experience might increase the chances their offspring survived to the harvest season, where we sampled them, relative to fawns of younger, more inexperienced females. A deer's age at breeding in 2001 was calculated by subtracting its age at harvest by the number of years after 2001 that it was harvested (e.g., a 4.5-yr-old deer harvested in the 2003 season was 2.5 yr old at breeding in 2001). We also included a covariate for the number of deer harvested from the section (2.6 km^2) from which the candidate parent was harvested because a section with a higher harvest may increase the likelihood that both a parent and its fawn were harvested. Ideally, we would use the proportion of deer harvested from a section to address this question, but we do not have data on deer density at the section level.

Fawns with CWD-infected and noninfected parents

To determine whether CWD-positive or CWD-negative parents were more likely to be assigned as parents of male or female fawns, we used chi-square analyses to evaluate the proportion of male and female fawns assigned to male and female parents, respectively.

Because fawns are associated with, and reliant upon, their mothers during their first year of life (Marchinton and Hirth, 1984), we used female parents and fawns harvested in the 2002 season (true fawns) to compare harvest characteristics between fawns with CWD-positive and CWD-negative female parents. We used analysis of variance (ANOVA) to evaluate whether the number of days separating a fawn and its mother's harvest was related to the CWD status of its mother, the sex of her fawn, and the interaction between these factors. We used a Tobit (censored regression; Tobin, 1958) analysis to determine whether the harvest distance between fawns and their mothers was predicted by CWD status of the mother, the sex of her fawn, or the interaction of these covariates. The Tobit analysis simultaneously tests whether 1) the probability of a fawn being harvested from the same section as its mother (actual harvest distance between fawn and mother was considered censored because it was smaller than the section scale for our harvest locations and thus not measured) and 2) the distance (natural log) between a fawn and its mother harvested in different sections were significantly related to our covariates.

RESULTS

We obtained multilocus genotypes for 1,468 fawns (born in 2002), 1,296 candidate female parents, and 502 candidate male parents. Within this dataset, CWD prevalence was 6.6% for candidate female parents and 11.2% for candidate male parents. The proportion of candidate female and male parents determined to have been CWD-positive during the 2001 breeding season was 3.9% and 9.2%, respectively (Table 1). For both maternity and paternity analyses, we believe that approximately 50% of 2001 candidate parents were sampled based on the declining number of deer of both sexes (especially males) harvested from the 2002–04 seasons, and missed sampling of some males that were harvested in 2001

TABLE 2. Numbers (percentages) of candidate male and female Wisconsin white-tailed deer (*Odocoileus virginianus*) assigned as parents categorized by their chronic wasting disease (CWD) status (positive [+] vs. negative [−] at breeding).

	Candidate males			Candidate females		
	Total No.	CWD+	CWD−	Total No.	CWD+	CWD−
Assigned	171	20 (11.7%)	151 (88.3%)	269	17 (6.3%)	252 (93.7%)
Not assigned	331	26 (7.9%)	305 (92.1%)	1027	33 (3.2%)	994 (96.8%)

shortly after they bred. The genotyping error rate was 1.69% (range 0–3.86% per locus) (estimated from the data by re-genotyping ~7% of the animals). The average number of alleles for the 11 microsatellites used in this study was 16.82 (range 10–22). Average heterozygosity (H_e) was 0.82, and the polymorphic information content (PIC) was 0.80. Using these markers, the probability of identity was 2.86^{-15}, and the probability of sibling identity was 1.05^{-5}.

For maternity analyses, 269 (20.8%) females were assigned as parents, 17 (6.3%) of which were classified as CWD-positive at the time of breeding. For paternity analyses, 171 (34.1%) males were assigned as parents, of which 20 (11.7%) were classified as CWD-positive at the time of breeding.

CWD status and parentage assignment

For males, the probability of being assigned as a parent was significantly related to their age at breeding. Older males were more likely to be assigned as parents than younger males (odds ratio [OR]=1.655, SE=0.217, $P<0.001$). CWD status at breeding was not significantly related to the probability that a male would be assigned as a parent (OR=1.484, SE=0.475, $P=0.217$; Table 2). The number of deer harvested from the same section as a candidate male parent was not significantly related to the probability of parentage assignment (OR=0.996, SE=0.004, $P=0.252$), indicating that results were not strongly biased by spatial variation in harvest intensity. However, some areas likely have a higher probability of harvesting both a parent and its fawn. When considering only candidate male parents and fawns harvested during the 2002 season, the results were similar (i.e., all parameter estimates were in the same direction [positive vs. negative], significance of variables did not change [at $P<0.05$]).

For females, the probability of being assigned as a parent was significantly related to both CWD status and age at breeding. CWD-positive females were more likely to be assigned as parents than CWD-negative females (OR=2.016, SE=0.621, $P=0.023$; Table 2). Older females were more likely to be assigned as parents than younger females (OR=1.148, SE=0.039, $P<0.001$). The number of deer harvested from the same section as a candidate female parent was not significantly related to the probability of parentage assignment (OR=1.001, SE=0.002, $P=0.762$). When considering only candidate female parents and fawns harvested during the 2002 season, the results were similar (i.e., all parameter estimates were in the same direction [positive vs. negative], significance of variables did not change [at $P<0.05$]).

Differences between fawns of CWD infected and noninfected parents

Overall, 13% (35 of 262) of fawns assigned to male parents had CWD-positive fathers, and 6.7% (21 of 313) of fawns assigned to female parents had CWD-positive mothers. Chi-square analyses indicated these proportions were not different (all $P>0.10$; Table 3). These rates were similar to the CWD prevalence found in male and female parents (above).

TABLE 3. Numbers of male and female fawns of Wisconsin white-tailed deer (*Odocoileus virginianus*) assigned to male and female parents. The percentage of assigned fawns whose parents were chronic wasting disease positive (CWD+) is indicated in parentheses. (The sex of one fawn was unknown; it was assigned to a CWD+ male parent.)

	Total No.	Male parents	Female parents
Male fawns	663	112 (14.3%)	130 (8.5%)
Female fawns	804	150 (12.7%)	183 (5.5%)

With respect to harvest characteristics, we found a significant interaction between sex of the fawn and the CWD status of the mother and the days separating a fawn and its mother's harvest ($F_{1,159}=5.09$, $P=0.026$). Male fawns with CWD-positive mothers were harvested earlier than their mothers compared (all $P<0.06$) to all other fawns, regardless of the mother's CWD infection status. Specifically, male fawns with CWD-positive mothers ($n=11$) were harvested before ($\bar{x}=48.1$ days, SE=15.6) their mothers and male fawns with CWD-negative mothers ($n=119$) were harvested after ($\bar{x}=9.5$ days, SE=4.8) their mothers. Female fawns with CWD-positive mothers ($n=10$) were harvested after ($\bar{x}=11.0$ days, SE=8.9) their mothers, and female fawns with CWD-negative mothers ($n=173$) were harvested before ($\bar{x}=4.7$ days, SE=5.2) their mothers. In terms of harvest location, male fawns with CWD-positive mothers were less likely ($t=-1.67$, $P=0.09$) to be harvested from the same section as their mothers (25%) compared to male fawns with CWD-negative mothers (56%). Female fawns with CWD-positive mothers were more likely to be harvested from the same section as their mothers (86%) than male fawns ($t=2.35$, $P=0.02$) with CWD-positive mothers (25%), female fawns ($t=2.02$, $P=0.04$) with CWD-negative mothers (44%), or male fawns ($t=1.70$, $P=0.09$) with CWD-negative mothers (56%). Neither sex of fawns nor CWD status of the mother was related to the distance separating the harvest location of fawns harvested from a different section than their mothers (all $P>0.10$; Table 4), although our samples were limited for female fawns with CWD-positive mothers harvested from different sections.

DISCUSSION

Our results suggest that preclinical CWD infection in white-tailed deer does not significantly interfere with the ability of males or females to successfully breed and produce offspring. CWD-positive female deer were more likely than CWD-negative females to be assigned as parents of fawns in our dataset. This higher rate of parentage assignment for CWD-positive females may be because these mothers are more vulnerable to harvest and thus over-represented in our sample. A previous study in this region, however, concluded that CWD-positive deer were not more vulnerable to harvest (Grear et al., 2006) unlike what has been documented for mule deer (Conner et al., 2000). In addition, we found no difference in average harvest date between female parents based on their CWD status at harvest in the 2002 or 2003 hunting seasons, while CWD-positive deer were harvested somewhat later than CWD-

TABLE 4. Number of Wisconsin white-tailed deer (*Odocoileus virginianus*) mother-fawn pairs and mean (SE in parentheses) distance (km) separating harvest location of females and their fawns harvested from different sections as a function of fawn sex and the mother's chronic wasting disease (CWD) status (positive [+] or negative [−] at breeding).

	CWD+ mother	CWD− mother
Male fawns	6, 2.4 (0.69)	31, 2.9 (0.23)
Female fawns	1, 1.6 (na)[a]	41, 3.1 (0.23)

[a] na = not appropriate; SE cannot be estimated from single sample.

negative deer in the 2004 hunting season (results not shown). Another explanation for the higher rate of parentage assignment for CWD-positive female deer may be that their fawns are more vulnerable to harvest. For example, CWD infection may not negatively affect a deer's ability to reproduce, but it may interfere with the ability of a female to provide parental care, and thus ensure the survival of her fawns to adulthood.

Our findings provide support for this alternate hypothesis because male fawns with CWD-positive mothers were harvested, on average, more than a month before their mothers. In addition, despite fawns of both sexes generally associating with their mothers for their first full year of life (Marchinton and Hirth, 1984), male fawns with CWD-positive mothers were less likely to be harvested from the same section as their mothers compared to male fawns with CWD-negative mothers. Perhaps, because most male white-tailed deer disperse, male fawns with CWD-positive mothers are more independent and engage in more exploratory movements making them more vulnerable to harvest than male fawns with CWD-negative mothers. Most female fawns with CWD-positive mothers (86%), on the other hand, were harvested from the same section as their mothers, while almost half of female fawns with CWD-negative mothers were farther away. Further investigation is necessary to identify the reason why female fawns were harvested closer to their CWD-positive mothers than all other fawn groups.

Negative impacts of CWD infection on harvest vulnerability could impact white-tailed deer population demographics as disease prevalence increases, although this effect is likely less important than direct impacts on adult survival (Dulberger et al., 2010). Because fawns are most dependent on their mothers for survival in the first 8–10 wk of life (Marchinton and Hirth, 1984) we expect differences in parental care between CWD-positive and CWD-negative mothers on fawn survival to be most pronounced during this time. In addition, if CWD-positive females provide inadequate parental care, one might predict differences between fawns of CWD-positive and CWD-negative mothers in developmental rates. The relationship between clinical CWD infection, fawn recruitment, and body condition should be investigated directly to evaluate early survival and development of fawns (e.g., the first 2 mo postparturition; all of the fawns in our dataset had survived from birth in late spring until the fall harvest season). It is also important to identify differences in dispersal and recruitment into adulthood between fawns with CWD-positive versus CWD-negative mothers.

Recent data suggest that disease transmission from CWD-positive mothers to their offspring may be more common than previously thought (Argue et al., 2007). Because dispersing male deer could be responsible for CWD spread, higher harvest vulnerability or lower survival rates for male fawns with CWD-positive mothers may be mechanisms that reduce disease spread. Alternatively, earlier dispersal or more wide ranging movement of potentially infected male fawns with CWD-positive mothers may promote disease spread. The importance of CWD transmission from mothers to offspring and the role of dispersing, potentially CWD-positive male fawns should be considered in understanding and managing CWD spread.

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LITERATURE CITED

ARGUE, C. K., C. RIBBLE, V. W. LEES, J. MCLANE, AND A. BALACHANDRAN. 2007. Epidemiology of an outbreak of chronic wasting disease on elk farms in Saskatchewan. Canadian Veterinary Journal 48: 1241–1248.

BAETEN, L. A., B. E. POWERS, J. E. JEWELL, T. R. SPRAKER, AND M. W. MILLER. 2007. A natural case of chronic wasting disease in a free-ranging moose (*Alces alces shirasi*). Journal of Wildlife Diseases 43: 309–314.

BISHOP, M. D., S. M. KAPPES, J. W. KEELE, R. T. STONE, S. L. F. SUNDEN, G. A. HAWKINS, S. S. TOLDO, R. FRIES, M. D. GROSZ, J. YOO, AND C. W. BEATTIE. 1994. A genetic linkage map for cattle. Genetics 136: 619–639.

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.

DASZAK, P., A. A. CUNNINGHAM, AND A. D. HYATT. 2000. Emerging infectious diseases of wildlife: Threats to biodiversity and human health. Science 287: 443–449.

DEWOODY, J. A., R. L. HONEYCUTT, AND L. C. SKOW. 1995. Microsatellite markers in white-tailed deer. Journal of Heredity 86: 317–319.

DEYOUNG, R. W., S. DEMARAIS, R. L. HONEYCUTT, K. L. GEE, AND R. A. GONZALES. 2006. Social dominance and male breeding success in captive white-tailed deer. Wildlife Society Bulletin 34: 131–136.

DULBERGER, J., N. T. HOBBS, H. M. SWANSON, C. J. BISHOP, AND M. W. MILLER. 2010. Estimating chronic wasting disease effects on mule deer recruitment and population growth. Journal of Wildlife Diseases 46: 1086–1095.

EDMUNDS, D. R. 2008. Epidemiology of chronic wasting disease in white-tailed deer in the endemic area of Wyoming. MS Thesis, University of Wyoming, Laramie, Wyoming, 137 pp.

FOX, K. A., J. E. JEWELL, E. S. WILLIAMS, AND M. W. MILLER. 2006. Patterns of PrP^{CWD} accumulation during the course of infection in orally inoculated mule deer (*Odocoileus hemionus*). Journal of General Virology 87: 3451–3461.

GREAR, D. A., M. D. SAMUEL, J. A. LANGENBERG, AND D. KEANE. 2006. Demographic patterns and harvest vulnerability of CWD infected white-tailed deer in Wisconsin. Journal of Wildlife Management 70: 546–553.

———, ———, K. T. SCRIBNER, B. V. WECKWORTH, AND J. A. LANGENBERG. 2010. Influence of genetic relatedness and spatial proximity on chronic wasting disease infection among female white-tailed deer. Journal of Applied Ecology 47: 532–540.

HARVELL, C. D., C. E. MITCHELL, J. R. WARD, S. ALTIZER, A. P. DOBSON, R. S. OSTFELD, AND M. D. SAMUEL. 2002. Climate warming and disease risk for terrestrial and marine biota. Science 296: 2158–2162.

JOHNSON, C., A. HERBST, C. DUQUE-VELASQUEZ, J. P. VANDERLOO, P. BOCHSLER, R. CHAPPELL, AND D. MCKENZIE. 2011. Prion protein polymorphisms affect chronic wasting disease progression. PLoS ONE 6: e17450.

———, J. JOHNSON, J. P. VANDERLOO, D. KEANE, J. M. AIKEN, AND D. MCKENZIE. 2006. Prion protein polymorphisms in white-tailed deer influence susceptibility to chronic wasting disease. Journal of General Virology 87: 2109–2114.

JOLY, D. O., M. D. SAMUEL, J. A. LANGENBERG, J. A. BLANCHONG, C. A. BATHA, R. E. ROLLEY, D. P. KEANE, AND C. A. RIBIC. 2006. Spatial epidemiology of chronic wasting disease in Wisconsin white-tailed deer. Journal of Wildlife Diseases 42: 578–588.

KALINOWSKI, S. T., M. L. TAPER, AND T. C. MARSHALL. 2006. Revising how the computer program CERVUS accommodates genotyping error increases success in paternity assignment. Molecular Ecology 16: 1099–1106.

KEANE, D. P., D. J. BARR, P. N. BOCHSLER, S. M. HALL, T. GIDLEWSKI, K. I. O'ROURKE, T. R. SPRAKER, AND M. D. SAMUEL. 2008b. Chronic wasting disease in a Wisconsin white-tailed deer farm. Journal of Veterinary Diagnostic Investigation 20: 698–703.

———, ———, J. E. KELLER, S. M. HALL, J. A. LANGENBERG, AND P. N. BOCHSLER. 2008a. 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.

KIRKPATRICK, B. W. 1992. Identification of a conserved microsatellite site in the porcine and bovine insulin-like growth factor-I gene 5′ flank. Animal Genetics 23: 543–548.

MARCHINTON, R. L., AND D. H. HIRTH. 1984. Behavior. *In* White-tailed deer ecology and management, L. K. Halls (ed.). Stackpole, Harrisburg, Pennsylvania, pp. 129–168.

MARSHALL, T. C., J. SLATE, L. E. B. KRUUK, AND J. M. PEMBERTON. 1998. Statistical confidence for likelihood-based paternity inference in natural populations. Molecular Ecology 7: 639–655.

MCCALLUM, H., N. D. BARLOW, AND J. HONE. 2001. How should pathogen transmission be modeled? Trends in Ecology and Evolution 16: 295–300.

MILLER, M. W., H. M. SWANSON, L. L. WOLFE, F. G. QUARTARONE, S. L. HUWER, C. H. SOUTHWICK, AND P. M. LUKACS. 2008. Lions and prions and deer demise. PLoS ONE 3: e4019.

———, E. S. WILLIAMS, C. W. MCCARTY, T. R. SPRAKER, T. J. KREEGER, C. T. LARSEN, AND E. T. THORNE. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and

Wyoming. Journal of Wildlife Diseases 36: 676–690.

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.

SAN JOSE, C., F. BRAZA, AND S. ARAGON. 1999. The effect of age and experience on the reproductive performance and prenatal expenditure of resources in female fallow deer (*Dama dama*). Canadian Journal of Zoology 77: 1717–1722.

SCHULER, K. L. 2006. Monitoring for chronic wasting disease in mule deer and white-tailed deer at Wind Cave National Park: Investigating an emerging epizootic. PhD Dissertation, South Dakota State University, Brookings, South Dakota, 173 pp.

SEVERINGHAUS, C. W. 1949. Tooth development and wear as a criteria of age in white-tailed deer. Journal of Wildlife Management 13: 195–216.

SIGURDSON, C. J., E. S. WILLIAMS, M. W. MILLER, T. R. SPRAKER, K. I. O'ROURKE, AND E. A. HOOVER. 1999. Oral transmission and early lymphoid tropism of chronic wasting disease PrP^{res} in mule deer fawns (*Odocoileus hemionus*). Journal of General Virology 80: 2757–2764.

SPRAKER, T. R., M. W. MILLER, E. S. WILLIAMS, D. M. GETZY, W. J. ADRIAN, G. G. SCHOONVELD, R. A. SPOWARDT, K. I. O'ROURKE, J. M. MILLER, AND P. A. MERZ. 1997. Spongiform encephalopathy in free-ranging mule deer (*Odocoileus hemionus*), white-tailed deer (*Odocoileus virginianus*) and Rocky Mountain elk (*Cervus elaphus nelsoni*) in northcentral Colorado. Journal of Wildlife Diseases 33: 1–6.

STORM, D. J. 2011. Chronic wasting disease in white-tailed deer: Evaluation of aerial surveys; age-estimation; and the role of deer density and landscape in disease transmission. PhD Dissertation, University of Wisconsin, Madison, Wisconsin, 87 pp.

TOBIN, J. 1958. Estimation of relationships for limited dependent variables. Econometrica 26: 24–36.

WASSERBERG, G., E. E. OSNAS, R. E. ROLLEY, AND M. D. SAMUEL. 2009. Host culling as an adaptive management tool for chronic wasting disease—A modeling study. Journal of Applied Ecology 46: 457–466.

WILLIAMS, E. S. 2005. Chronic wasting disease. Veterinary Pathology 42: 530–549.

———, AND M. W. MILLER. 2002. Chronic wasting disease in deer and elk in North America. Revue Scientifique et Technique de l'Office International des Epizooties 21: 305–316.

———, ———, T. J. KREEGER, R. H. KAHN, AND E. T. THORNE. 2002. Chronic wasting disease of deer and elk: A review with recommendations for management. Journal of Wildlife Management 65: 551–563.

WILSON, G. A., S. M. NAKADA, T. K. BOLLINGER, M. J. PYBUS, E. H. MERRILL, AND D. W. COLTMAN. 2009. Polymorphisms at the PRNP gene influence susceptibility to chronic wasting disease in two species of deer (*Odocoileus* spp.) in western Canada. Journal of Toxicology and Environmental Health: Part A: Current Issues 72: 1025–1029.

———, C. STROBECK, L. WU, AND J. W. COFFIN. 1997. Characterization of microsatellite loci in caribou, *Rangifer tarandus*, and their use in other artiodactyls. Molecular Ecology 6: 697–699.

WOBESER, G. A. 2006. Essentials of disease in wild animals. Blackwell, Ames, Iowa, 243 pp.

———. 2008. Parasitism: Costs and effects. *In* Parasitic diseases of wild birds, C. T. Atkinson, N. J. Thomas and D. B. Hunter (eds.). Wiley Blackwell, West Sussex, UK, pp. 3–9.

WOLFE, L. L., T. R. SPRAKER, L. GONZALEX, M. P. DAGLEISH, T. M. SIROCHMAN, J. C. BROWN, M. JEFFREY, AND M. W. MILLER. 2007. PrP^{CWD} in rectal lymphoid tissue of deer (*Odocoileus* spp.). Journal of General Virology 88: 2078–2082.

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