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Chronic wasting disease: Possible transmission mechanisms in deer

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ABSTRACT

We develop a model for the spread of chronic wasting disease (CWD) in a mule deer (*Odocoileus hemionus*) population to assess possible mechanisms of disease transmission and parameterize it for the mule deer population in Alberta, Canada. We consider seven mechanisms of disease transmission corresponding to direct and indirect contacts that change with seasonal distribution and groupings of deer. We determine the minimum set of mechanisms from all possible combinations of mechanisms with different weights for duration of seasonal segregation of sexes that are able to reproduce the observed ratio of CWD prevalence in adult males and females of ~2 and greater. Multiple mechanisms are likely to produce the ratio of male:female prevalence levels and include: (1) environmentally mediated transmission associated with higher food intake by males, (2) female to male transmission during mating of this polygamous species, (3) increased male susceptibility to CWD and (4) increased intensity of direct contacts within male social groups. All of these mechanisms belong to the class of frequency-dependent transmission. Also important is seasonality in deer social structure with an increasing ratio of prevalence in males:females under all mechanisms as the duration of sexual segregation increases throughout a year.

1. Introduction

Chronic wasting disease (CWD) is a fatal disease of cervids, including white-tailed (*Odocoileus virginianus*) and mule deer (*Odocoileus hemionus*), elk (*Cervus elaphus*) and moose (*Alces alces*) (Williams, 2005), which belongs to a class of prion diseases called transmissible spongiform encephalopathies (TSEs). Along with the other well-known TSEs, such as BSE or "mad-cow disease" and Creutzfeldt–Jakob disease in humans, CWD is characterized by the accumulation of an abnormal misfolding of normal forms of proteins, called prions, in lymphatic and neural tissues. The disease was first recognized as a clinical "wasting" syndrome in 1967 in mule deer at a wildlife research facility in northern Colorado, USA, but was later identified as a TSE (Williams and Young, 1980). The disease has since spread or been translocated to over fifteen US states and two Canadian provinces.

The exact routes of CWD transmission remain unclear. There is evidence that infection is transmitted horizontally directly from individual to individual during close contact via saliva, urine and feces (Mathiason et al., 2006, 2009), or indirectly through the environment (Miller et al., 2004; Johnson et al., 2007). Environmental transmission may occur via ingestion of soils or plants previously contaminated by an infected animal and the prions may accumulate in the environment and remain infectious for a long time (Schramm et al., 2006; Genovesi et al., 2007,). Once contracted, the incubation period for the disease is about 18 months (Tamgueney et al., 2009), and only in the later, clinical stages is CWD typified by the chronic weight loss and behavioral changes that eventually lead to death. Because infected deer cannot be distinguished from healthy ones during initial stages of the disease, even though they may already be spreading the disease, the primary information about disease infection comes from post-mortem examination of tissues. To develop CWD a deer must contact a sufficient number of prions, although the minimum dosage needed to contract the disease is unknown. Vertical transmission from mother to fawn before or at birth appears to play only a minor role (Miller and Williams, 2003).

Because the transmission of infectious diseases in wildlife populations typically is complex (Keeling and Rohani, 2008), the problem of deriving adequate models to help guide management of wild populations remains a challenge. The first models describing CWD (Miller et al., 2000; Gross and Miller, 2001) included only a basic disease transmission function common to all individuals, and assumed that the number of contacts encountered by an infectious individual was density independent. Two more

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recent papers illustrate contrasting approaches to CWD modeling. In Wasserberg et al. (2009), a population projection model of white-tailed deer consisting of 160 compartments (20 age classes, two sexes, and 4 disease stages) was developed. The authors considered outcomes of two types of disease transmission, frequency dependent (FD) and density dependent (DD) transmission (McCallum et al., 2001; Begon et al., 2002), but did not include environmental transmission. When they fitted the transmission coefficient from CWD prevalence data in Wisconsin, FD and DD terms fit the observed disease pattern almost equally well. In contrast, Miller et al. (2006) used a simple Kermack–McKendrick type model with minimum population details parameterized by cumulative mortality data from two small captive herds. These authors compared 6 models including different number of disease stages and direct (deer to deer) and indirect (through the environment) transmission and showed that the best two models corresponded to both indirect and direct transmission without explicitly accounting for disease stages. Their study likely reflects realistic DD disease transmission because small numbers of deer were in pens with close contact.

In the case of CWD, sources of complexity in determining transmission include variable contact rates due to seasonal movement, social aggregations, habitat selection and landscape structure (Carnes, 2009; Habib et al., 2011). Limited information about potential deer contacts can be obtained using GPS collars (Kjaer et al., 2008; Schauber et al., 2007) or proximity detectors (Prange et al., 2006). However, these studies do not provide population-level transmission, and have not yet been used to infer contact with environmental contamination in wildland situations, despite the potential for environmental persistence to shape deer-CWD dynamics (Almberg et al., 2011; Sharp and Pastor, 2011). Inherent differences in susceptibility among individuals of different age, sex, and genetic strains further complicate our understanding.

In this paper we address seven hypothesized mechanisms for CWD transmission. Our approach takes advantage of the consistent evidence that CWD prevalence is about two times higher in adult male deer than in adult females across regions (e.g., Miller and Conner, 2005; Heisey et al., 2010; Rees et al., 2012). Our goal, therefore, is to select a number of transmission mechanisms capable of producing high enough male to female prevalence ratio as a set of competing hypotheses reflecting the most important transmission paths, and evaluate whether they can produce the observed discrepancy in male and female prevalence. We use a continuous-time population SI model with three categories of both susceptible and infected animals: male adults, female adults, and juveniles (fawns) with density-dependent fawn mortality, density-independent adult mortality, and hunting with different hunting preferences for males, females and juveniles. We incorporate seasonality in grouping patterns among the sexes and explore the effect of duration of sexual segregation across the year. The general scheme of the model is shown in Fig. 1. After addressing mechanisms of transmission, we study the sensitivity of the results to model parameters including hunter harvest and the relative susceptibility of males and females given contact with an infected individual.

2. Model of deer population

In this section we develop the basic model of deer population dynamics with a very general description of disease transmission, which is considered in more detail in Section 3. Notation for model variables and parameters is defined in Table 1. Details of components of the model are found in Appendices A–D.

2.1. Population structure, vital rates and density dependence

The model has two disease-related stages: susceptible (S) and infected (I) deer. Each of the stages includes the simplest sex/age structure commonly used in deer management: adult males (m), adult females (f) and juveniles (j); the latter are assumed to have a 50:50 sex ratio at birth. This gives six compartments for population outputs: three densities of susceptible deer, S_j, S_f, S_m, and three densities of infected ones, I_j, I_f, I_m. The model includes juvenile birth and maturation, natural mortality, harvest and disease transmission:

Rate of change of deer class	Juvenile birth (B, B_{IS}, B_{II}) or maturation at rate τ^{-1}	Natural mortality	Harvest	Disease transmission	
$\frac{dS_j}{dt} =$	$BS_f + B_{IS}I_f - \tau^{-1}S_j$	$-(m_{0S,j} + Vm_{1S,j})S_j$	$-h_jS_j$	$-\lambda_jS_j$	(1)
$\frac{dS_f}{dt} =$	$0.5\tau^{-1}S_j$	$-m_{0S,f}S_f$	$-h_fS_f$	$-\lambda_fS_f$	(2)
$\frac{dS_m}{dt} =$	$0.5\tau^{-1}S_j$	$-m_{0S,m}S_m$	$-h_mS_m$	$-\lambda_mS_m$	(3)
$\frac{dI_j}{dt} =$	$B_{II}I_f - \tau^{-1}I_j$	$-(m_{0I,j} + Vm_{1I,j})I_j$	$-h_jI_j$	$+\lambda_jS_j$	(4)
$\frac{dI_f}{dt} =$	$0.5\tau^{-1}I_j$	$-m_{0I,f}I_f$	$-h_fI_f$	$+\lambda_fS_f$	(5)
$\frac{dI_m}{dt} =$	$0.5\tau^{-1}I_j$	$-m_{0I,m}I_m$	$-h_mI_m$	$+\lambda_mS_m$	(6)

The model is general enough, but we parameterize it for mule deer, the species in which the most CWD cases occur in free-ranging deer in Alberta. In this paper we do not consider the effects related to deer harvest (see Potapov et al., 2012). However, we parameterized the model from the data for a harvested population, and hence harvest component is present in the model as well.

Birth and mortality rates are the key components of deer population dynamics models because they describe population self-regulation. We incorporated density-dependent fawn survival but not fecundity rate because density–fecundity relationships for mule deer are not as well developed in the literature as density-dependent juvenile mortality (Bartmann et al., 1992; Gaillard et al., 1998; Unsworth et al., 1999; Heffelfinger et al., 2003). Although birth rates could decline if there were not enough males to fertilize all the females, we assume there are always sufficient males because a threshold in buck:doe ratios below which recruitment declines rapidly has not been reported for mule deer (White et al., 2001; Erickson et al., 2003; Bishop et al., 2005). For example, the data in White et al. (2001) show only a minor decline in fawn:doe ratio with a major decline of buck:doe ratio, whereas the effect of other factors was much more prominent. Furthermore, very low buck to doe ratio never occurred in our results.

For modeling density-dependent mortality we used an approach similar to Powers et al. (1995) that relates mortality to the available food, where the amount of required food in a critical season (assumed to be winter in Alberta) is proportional to densities in the deer sex and age groups. For the sake of simplicity we do not include stochasticity in summer food (Hurley et al., 2011) and in snow accumulation in winter that influences energy expenditures for locomotion (Parker et al., 1984) and reduces forage availability (Visscher et al., 2006). Hence, we scale mortality as a simple starvation index V, which depends on the ratio of available winter food F_A and required food F_R:

$$V = \max\left\{0,\ \ 1 - \frac{F_A}{F_R}\right\}. \tag{7}$$

If there is excess food, i.e., the population is below winter carrying capacity, then $F_A > F_R$ and $V = 0$. If F_A is much less than F_R and starvation rates are high, V approaches 1. When the population is at a food-based equilibrium (at carrying capacity), V takes some value V_0 between 0 and 1, corresponding to partial food limitation.