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Modeling seasonal behavior changes and disease transmission with application to chronic wasting disease

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HIGHLIGHTS

- A new model is built to study spread chronic wasting disease in free-ranging deer.
- The model employs two modes of transmission based on seasonal behavior.
- Birth and change in seasonal home range are impulsive.
- The basic reproduction number and stability of disease-free equilibrium are studied.
- Under certain conditions, culling can eradicate the disease.

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ABSTRACT

Behavior and habitat of wildlife animals change seasonally according to environmental conditions. Mathematical models need to represent this seasonality to be able to make realistic predictions about the future of a population and the effectiveness of human interventions. Managing and modeling disease in wild animal populations requires particular care in that disease transmission dynamics is a critical consideration in the etiology of both human and animal diseases, with different transmission paradigms requiring different disease risk management strategies. Since transmission of infectious diseases among wildlife depends strongly on social behavior, mechanisms of disease transmission could also change seasonally. A specific consideration in this regard confronted by modellers is whether the contact rate between individuals is density-dependent or frequency-dependent. We argue that seasonal behavior changes could lead to a seasonal shift between density and frequency dependence. This hypothesis is explored in the case of chronic wasting disease (CWD), a fatal disease that affects deer, elk and moose in many areas of North America. Specifically, we introduce a strategic CWD risk model based on direct disease transmission that accounts for the seasonal change in the transmission dynamics and habitats occupied, guided by information derived from cervid ecology. The model is composed of summer and winter susceptible-infected (SI) equations, with frequency-dependent and density-dependent transmission dynamics, respectively. The model includes impulsive birth events with density-dependent birth rate. We determine the basic reproduction number as a weighted average of two seasonal reproduction numbers. We parameterize the model from data derived from the scientific literature on CWD and deer ecology, and conduct global and local sensitivity analyses of the basic reproduction number. We explore the effectiveness of different culling strategies for the management of CWD: although summer culling seems to be an effective disease eradication strategy, the total culling rate is limited by the requirement to preserve the herd.

1. Introduction

In temperate climates, seasonal variation in environmental and climatic conditions is closely mirrored by behavioral responses in animal populations, including aggregation, separation, hibernation, and reproduction (Begon et al., 2002; Lloyd-Smith et al., 2005). Such seasonal variation needs to be accurately represented in models aimed at informing wildlife managers to obtain realistic predictions about the efficiency and effectiveness of human intervention. Such considerations are particularly crucial in the case of managing diseases in wild animal populations where seasonally varying behavior can drive seasonally varying force of infection.

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Most existing mathematical models incorporated seasonal variation as time-dependent coefficient functions (Altizer et al., 2006; Bolzoni et al., 2008; Liu et al., 2010; Buonomo, 2011; Zhang et al., 2012; Hu, 2012; Wesley and Allen, 2009), thereby assuming that the disease transmission mechanism itself remains constant in time. Here, we argue that certain seasonal behavioral changes can lead to a qualitative shift in the mechanism of transmission (rather than only a quantitative adjustment to its strength), and that such shifts require novel modeling approaches.

A fundamental question in disease modeling is whether transmission is represented as frequency (FD) or density dependent (DD), particularly in wild animal populations (Lloyd-Smith et al., 2005; McCallum et al., 2001). These two options represent two different mechanistic assumptions. Whereas FD assumes that the rate of contact between individuals is independent of population density, DD assumes that this rate increases linearly with density (Begon et al., 2002; Lloyd-Smith et al., 2005), see also Fig. 1 in Almberg et al. (2011). Since the rate of contact depends on social behavior, and since social behavior differs between seasons, the mechanism of disease transmission may vary seasonally. Specifically, seasonal differences in behavior can lead to a qualitative shift from FD to DD transmission. For example, wild cervids roam freely over large geographic areas during the summer, and are more aggregated in winter due to snow cover and location of feeding sites. Hence, while FD maybe more appropriate to describe transmission during the summer, the high spatial density of individuals in the winter could lead to DD transmission. The choice of transmission modality is of fundamental importance since, for example, models with DD transmission exhibit a population threshold below which a disease will die out, whereas corresponding FD models have no such threshold (Lloyd-Smith et al., 2005; McCallum et al., 2001). In particular, the predicted efficiency of culling as a disease control strategy depends on whether DD or FD transmission terms are used, see e.g. Beeton and McCallum (2011). We derive and analyze a strategic (sensu Pielou, 1977) two-season model, wherein disease transmission is FD in the summer season and DD in winter. Finally, we explore the implications of this model for the seasonal dynamics of chronic wasting disease in North America.

Chronic wasting disease (CWD) is a well-characterized prion disease (Williams and Young, 1980) that belongs to the family of transmissible spongiform encephalopathies (TSEs), which include diseases like scrapie in sheep, bovine spongiform encephalopathy (BSE) in cattle, transmissible mink encephalopathy (TME) in Mink, and both Creutzfeldt–Jakob disease (CJD) and variant Creutzfeldt–Jakob disease (vCJD) in humans. Since its initial discovery in captive mule deer in 1967 (Williams and Young, 1980; Sigurdson, 2008), CWD has appeared in many species of cervids, such as white-tailed deer (*Odocoileus virginianus*), black-tailed deer (*Odocoileus columbianus*), mule deer (*Odocoileus hemionus*), Rocky Mountain elk (*Cervus elaphus nelsoni*), and Shira's moose (*Alces alces shirasi*) (Sigurdson, 2008; Gilch et al., 2011). CWD is transmitted upon direct contact between deer (Miller and Williams, 2003), vertically, from mother to offspring (Mathiason et al., 2010), and indirectly through the environment (Miller et al., 2004). The incubation period is relatively long (12–34 months, Kahn et al., 2004), and the disease can be transmitted during at least part of this period (Mathiason et al., 2009; Safar et al., 2008; Aspinall, 2011). In the final (and relatively short) clinical stages of CWD, a diseased deer typically exhibits physical wasting, increased thirst and urination, excessive salivation, difficulty in swallowing, trouble walking, dropping of ears, and changes in behavior (Gilch et al., 2011). At present, there is no (prophylactic or therapeutic) vaccination or cure for CWD. In addition to the decimation of cervid herds across North America, there is concern about CWD spreading to other species, including humans (Belay et al., 2004), although there is currently no strong evidence to support the hypothesis CWD is a zoonotic disease (Sandberg et al., 2010).

Social behavior in cervids changes throughout the year. After fawns are born in spring (Kie and Bowyer, 1999), females rear their young in separate groups, while males tend to flock in stag groups (Kjaer et al., 2008; Silbernagel et al., 2011). The rut period in the fall is characterized by increasing interactions between males and by polygynous mating. Large mixed-sex groups form during the subsequent period of gestation in winter. This winter aggregation can result as a social response to low temperatures, a migration away from snow covered higher elevations, and an attraction to accessible feeding sites. The temporal patterns of these events depend on species and location, e.g. Kjaer et al. (2008), Silbernagel et al. (2011), Kjaer (2010), Lingle (2003). Contact rates between individuals vary considerably between seasons (Kjaer et al., 2008; Kjaer, 2010; Habib et al., 2011). We argue that the fundamental differences between confinement in the winter and expansion during the summer, see e.g. Darling (1964), could lead to a qualitative change in contact rate between individuals, and thus also in force of infection.

Current models for CWD dynamics range from relatively simple, analytically tractable models with few compartments (Miller et al., 2006) to highly complex simulation models with tens or even hundreds of compartments (Almberg et al., 2011). Although disease transmission has been modeled with FD and DD terms, it is not entirely clear which is more appropriate (Wasserberg et al., 2009), and some authors have used various interpolations between the two (Almberg et al., 2011; Habib et al., 2011). Some authors generally favor FD models for wildlife disease, but the contact rate data by Habib et al. (2011) show that contact rate of deer during the winter season does scale with density, so that a pure FD model for the winter season is unrealistic.

In this paper, we develop an analytically tractable two-season model for CWD that incorporates FD transmission during the summer and DD transmission during the winter season. FD transmission is appropriate in the summer when the herd is spread out and an increase in density does not lead to an increase in contact rate, whereas DD transmission is appropriate in winter when crowding effects occur and an increase in density results in higher contact rate, as illustrated in Fig. 1 in Almberg et al. (2011). Not only do deers exhibit a change in behavior between seasons, but they also shift in the range of habitat (Dahlberg and Guettinger, 1956; Darling, 1964; Sorensen and Taylor, 1995). Therefore, we account also for possible differences in the spatial extent of the range of the herd in the two seasons, and include density-dependent reproduction. We reduce the model to a system of two coupled impulsive differential equations and use the theory of Floquet multipliers to determine the stability of the disease-free state and the basic reproduction number R_0. We determine values for the parameters in our model from published data and perform global and local sensitivity analyses of R_0 using Latin hypercube sampling, partial rank correlation analysis, and elasticity coefficients.

2. Materials and methods

2.1. Model derivation

We divide the year into two seasons ('summer' and 'winter') that last a fraction of τ and $1-\tau$ of the year, respectively. Year n begins, biologically, with the summer season when fawns are born. The nth summer lasts from time n to time $n+\tau$, whereas the nth winter lasts from $n+\tau$ to $n+1$. The spatial extent of suitable habitat during the summer (winter) is A_s (A_w) (dimension: area). We split the total population density $N=S+I$ into susceptible and infectious individuals (dimension: density=number per area).