Modeling and Conservation of Wildlife Populations in Managed Landscapes:
A Trade-Off Between Effort and Results

A DISSERTATION
SUBMITTED TO THE FACULTY OF THE GRADUATE SCHOOL
OF THE UNIVERSITY OF MINNESOTA
BY

Sergey Sergeyevich Berg

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

James D. Forester

August 2016

Acknowledgements

I would first and foremost like to thank my wife Sarah for being there for me through every challenge, failure, and success of my journey to earn my Ph.D. Your support and encouragement are the reason I am here, and I am forever grateful that I have you by my side. I am also thankful for the support of my parents, sister, and extended family as I decided to pursue one degree after another instead of looking for a real job.

I am deeply indebted to James Forester for being willing to take me on as a graduate student over five years ago and for helping me navigate all the challenges and complexities of doing good research. This has been one of the most rewarding journeys of my life and I will never forget all the suggestions, contributions, and mentoring that helped yield this final produce. Thank you for showing me how to be a scientist.

I would also like to thank Todd Arnold, John Fieberg, and Meggan Craft for serving as on my Ph.D. committee. Your feedback and guidance have helped transform this dissertation from what I envisioned when I first applied to graduate school into the final product it is today. Thank you for believing in me.

I am deeply indebted to the staff at the Minnesota Department of Natural Resources (DNR) for providing the fisher and marten data that served as the foundation for much of this dissertation, and particularly to John Erb and his team for providing invaluable feedback and support during every stage of my research. None of this would have been possible without the ongoing effort or Pam Coy, Barry Sampson, and Carolin Humpal to collect and process the data, and the many DNR pilots who assisted with the monitoring of animals.

Dedication

This dissertation is dedicated to the memories of my grandparents, Ivan and Taisiya

Gornak and Isaac Berg, and to my grandmother Lidiya Berg, who was there to see me

finally achieve my goals.

Table of Contents

List of Tables

List of Figures

Introduction

The mathematical modeling of ecological interactions is an essential tool in predicting the

behavior of complex systems across managed landscapes (Laguna et al. 2015). The

literature abounds with examples of models used to explore predator-prey interactions

(Berryman 1992, Swihart et al. 2001, Kalinkat et al. 2013), resource selection (Weir and

Harestad 2003, Zielinski et al. 2004, Meyer and Thuiller 2006), population growth

(Leslie 1945, 1948, Harris et al. 2007, Dillingham 2010), and the relationship between

population density and disease transmission (Miller et al. 2006, Wasserberg et al. 2009,

Habib et al. 2011). These models provide managers with an efficient alternative means of

testing new management and control strategies without resorting to empirical testing that

is often costly, time-consuming, and impractical (Caswell 2001, Bolzoni 2008, Bos and

Ydenberg 2011, Wood et al. 2013). However, because models are abstractions of reality

that make a large number of simplifying assumptions, their results are substantially less

accurate than those of empirical testing (Bunnell and Tait 1980, Grimm 1994, Johnson

2001). This illustrates a fundamental challenge in conservation biology – the trade-off

between effort and the validity or impact of results. Because wildlife managers and

researchers have a limited number of resources with which to conserve and study wildlife

populations (e.g., time, funding, number of field technicians), this trade-off is a matter of

efficiency. Using mathematical models to test a hypothesis, for example, requires

substantially less effort and fewer resources than empirically testing, but at the expense of

the validity of the results. Understanding the dynamics of this trade-off in different

situations and landscapes can help managers and researchers better allocate their limited

resources. My intention is to explore this trade-off in addressing questions on individual-level resource selection, state-level estimates of population abundance, and population-level assessments of alternative management strategies in controlling the spread of infectious diseases.

In chapter two, I start by investigating this trade-off in the use of mathematical models to study and predict the spread of infectious disease by presenting a comprehensive review of the main types of disease models found in the wildlife literature. Mathematical models are some of the most widely used tools in the study of wildlife infectious diseases, and have played an integral part in the management and control of diseases such as rabies (Källen et al. 1985), bovine tuberculosis (Smith and Cheeseman 2002), conjunctivitis (Hosseini et al. 2004), and plague (Webb et al. 2006). I review the five main types of models used in studies of wildlife infectious diseases (i.e., compartmental, metapopulation, lattice-based, reaction-diffusion, and contact network; Keeling and Eames 2005, Keeling and Rohani 2008, Kawata 2010) and discuss their uses, limitations, and contributions to the understanding of infectious diseases. I also present several potential avenues for the use of such models in future studies of infectious diseases.

In chapter three, I investigate how the effort put into identifying available habitat influences conclusions about habitat selection by evaluating the selection of den structures by female fishers (*Martes pennanti*) in northern Minnesota. Although previous studies have shown the importance of these structures for maintaining fishers populations (Paragi et al. 1996, Lofroth et al. 2010, Raley et al. 2012, Weir et al. 2012, Zhao et al. 2012), a concise theory of den site selection has been hampered by the inconsistency in

how different studies approach selection. Analyzing habitat selection typically involves comparing used sites and associated characteristics to unused sites that are deemed available to the animal (Johnson 1980, Manly et al. 2002, Meyer and Thuiller 2006), and the definition of what is "available" can drastically alter the conclusion drawn (Johnson 1980). Previous studies of fisher denning habitat selection have used different criteria for defining what is available, including trees used as rest sites (e.g., Thompson et al. 2010), trees of the same species within the same plot (e.g., Weir and Harestad 2003), random points within the home ranges (e.g., Davis 2009), and random points centered on trees within the study area (e.g., Zhao et al. 2012). Because of inconsistencies in how availability is defined between these and other studies, I propose two adjustments to future and ongoing research of denning habitat selection by fishers: (1) the spatial extent of what is available should be defined separately for each animal based on movement behavior (e.g., home range estimates, movement rates), and (2) only trees that meet certain criteria (e.g., presence of a suitable cavity) should be classified as available. I apply these two recommendations to assess whether the nearby abundance of suitable cavity trees influences the selection of den sites by female fishers in northern Minnesota. Using conditional logistic regression with model averaging, I identified tree health (i.e., alive, alive but declining, or dead) and the abundance of other cavity trees within 600m as the most important predictors of den-site selection by female fishers. Fishers in northern Minnesota used live ($\beta = 1.283$, SE = 0.021) and declining ($\beta = 1.254$, SE = 0.025) cavity trees disproportionately to their availability, and selected trees with a higher surrounding abundance of other cavity trees ($\beta = 0.0030$, SE = 0.0001). I suggest that female fishers select for these high abundance areas because it allows them to be more

selective in the types of trees they use for establishing subsequent maternal dens, which can help improve offspring survival and reproductive success. These results affirm the need to retain large-diameter cavity trees, illustrate the benefits of retaining them in high-density patches throughout the environment, and caution against creating a landscape with a large number of highly dispersed denning options.

In chapter four, I investigate how the amount of radio-telemetry data collected influences the performance of statistical population reconstruction in accurately estimating the abundance of American marten (*Martes americana*) in northeastern Minnesota. Statistical population reconstruction using age-at-harvest and catch-effort data has recently emerged as a robust and versatile approach to estimating the demographic dynamics of harvested wildlife populations (Skalski et al. 2007, Fieberg et al. 2010, Schaub and Abadi 2011, Clawson et al. 2013). Although there are clear benefits to incorporating radio-telemetry data into reconstruction efforts (Gove et al. 2002, Broms et al. 2010, Clawson et al. 2013), these data are costly and time-consuming to collect. Managers that consider collecting these data alongside existing efforts could benefit from a comprehensive examination of how such benefits are influenced by the amount of radio-telemetry data collected. Using American marten as a case study, I investigate the performance of population reconstruction using information on natural, harvest, or combined mortality derived from radio-telemetry data collected over different numbers of years and with different numbers of animals collared each year. I simulated populations under a range of conditions and found that incorporating radio-telemetry data on natural and harvest mortality significantly improved model precision, and that each additional animal collared per year yielded a 0.62% (SE = 0.20) improvement in precision, while

every additional year of radio-telemetry data resulted in a 3.11% (SE = 0.98) improvement. Thus, including another year of radio-telemetry resulted in similar gains in precision as including approximately five additional animals collared per year. In the applied marten example, I found that incorporating radio-telemetry data resulted in a substantially higher estimate of trapping vulnerability (0.23 vs. 0.086) and an overall smaller population size than reconstruction based solely on age-at-harvest and trapper effort data. These results illustrate the benefits of performing auxiliary studies, caution against relying on the results of population reconstruction based solely on age-at-harvest and hunter-effort data, and demonstrate that improvements from incorporating radio-telemetry data become evident even after as few as two years of data collection.

In chapter five, I use simulation models to investigate how the number and location of culled individuals influences the spread of chronic wasting disease (CWD) in a population of white-tailed deer (*Odocoileus virginianus*). Chronic wasting disease is a fatal prion disease of deer, elk, and moose that is transmitted through direct animal-to-animal contact and indirectly through contact with prions deposited by infected animals (Miller et al. 2004, Mathiason et al. 2006, Schramm et al. 2006, Haley et al. 2009*a*, *b*, Tamgüney et al. 2009, David Walter et al. 2011). Because prions can remain bio-available and infectious in the environment for at least two years after being deposited (Miller et al. 2004), herd reduction via wide-scale culling has thus far proven to be insufficient to halt the spread of CWD. Localized management in the form of intensive, non-selective culling of deer over a small geographical area (e.g., $<5km^2$), may provide a suitable alternative for management of CWD in white-tailed deer. By creating within the population persistent, low density areas that are not encroached upon by neighboring

animals for at least five years (Porter et al. 1991, Oyer and Porter 2004), I believe that localized management can limit the number of susceptible deer that are exposed to a prion-contaminated environment until those prions degrade and are no longer infectious. I use simulation models to compare the effectiveness of different scales of localized management to wide-scale herd reduction in controlling the spread of CWD through deer populations of varying densities. My results suggest that intensive removal of deer over a small area may provide a more effective control strategy than the broad-scale approaches currently used to manage CWD, without severely reducing overall abundance. Although this method was very efficient at preventing the spread of CWD in low-density populations, it was less effective at higher deer densities, although still more efficient than other control strategies. I recommend that this method be incorporated into ongoing adaptive management of CWD in white-tailed deer and stress the importance of improving our understanding of prion persistence in the environment and the relative importance of direct and indirect modes of transmission in driving observed disease dynamics.

Mathematical Models in Studies of Infectious Diseases in Wildlife Populations: A

Literature Review

2.1 Introduction

Infectious diseases of both humans and animals have played a large role in shaping the

world as it is today, from the role of smallpox in the fall of the Aztec (McNeil 1998) to

the 2001 foot-and-mouth disease epidemic that cost the United Kingdom $16 billion

(Keeling and Rohani 2008), and will continue to do so in the future. Although most

research into this topic has focused on human and domesticated animals as hosts, the

study of wildlife diseases has been growing rapidly since the 1980s (Anderson et al.

1981, Keeling and Rohani 2008, Kawata 2010). This interest has been driven by desires

to minimize risks to human health (e.g., zoonotic diseases such as leptospirosis; Munoz-

Zanzi et al. 2014), decrease loss of livestock (e.g., transmission of brucellosis from elk to

cattle; Xie and Horan 2009), and preserve native or endangered species (e.g., canine

distemper in the black-footed ferret; Thorne and William 1988).

Mathematical models have been one of the most commonly used tools in the

study of wildlife infectious diseases. These models have been used for tasks as varied as

predicting the spatial spread of rabies in Eastern Europe (Källen et al. 1985), comparing

the benefits of vaccination versus culling in eradicating bovine tuberculosis from England

(Smith and Cheeseman 2002), investigating the role of bird-feeders in promoting the

spread of conjunctivitis in house finches in North America (Hosseini et al. 2004), and

even to show that flea-borne transmission is insufficient to maintain plague epizootics in

Colorado prairie dogs (Webb et al. 2006). Here I present a review of the five main types

of models used in studies of wildlife infectious diseases – compartmental,

metapopulation, lattice-based, reaction-diffusion, and contact network – and discuss their

uses, limitations, and contributions to the understanding of infectious diseases (Keeling

and Eames 2005, Keeling and Rohani 2008, Kawata 2010).

2.2 Compartmental Models

2.2.1 Model Overview

The majority of disease models are based on categorizing hosts within a population based

on their disease status. The most basic form of such a model describes the proportions of

the population that are susceptible to (S), infected with (I), and recovered from (R) a

particular disease. For a pathogen that confers lifelong immunity from future infections,

this compartmentalization results in the following set of differential equations that

describe how individuals transition between different disease categories:

$$\frac{dS}{dt} = v - \beta SI - \mu S,$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I,$$

$$\frac{dR}{dt} = \gamma I - \mu R,$$

where v is the influx of new susceptible individuals through births, μ represents the

removal of individuals through death, β is the product of contact rate and transmission

probability, and γ is the recovery rate. This formulation is known as the susceptible-

infected-recovered (SIR), which was originally studied in depth by Kermack and

McKendrick (1927). A slightly different version of these equations can be used to model

the number of individuals in each disease category:

$$\frac{dX}{dt} = v - \beta XY/N - \mu X,$$

$$\frac{dY}{dt} = \beta XY/N - \gamma Y - \mu Y,$$

$$\frac{dZ}{dt} = \gamma Y - \mu Z,$$

where X, Y, and Z are the *number* of susceptible, infectious, and recovered individuals

and N is the total number of individuals ($N = X + Y + Z$). Such compartmentalization

ignores many details of the progression of the disease, as well as heterogeneity in

individual responses and contact probabilities (i.e., all individuals have the same

probability of contacting every other individual in the entire population), but nonetheless

provides some very important insights into disease progression (Keeling and Eames

2005, Keeling and Rohani 2008, Kawata 2010).

Many biologically motivated modifications have been made to this basic

framework, usually involving further subdivision of the disease classifications to reflect

either more complex pathogen biology (e.g., waning immunity, fatal infection, or the

presence of a latent period), multiple pathogens (e.g., canine distemper and sarcoptic

mange in fishers; Keller et al. 2012), multiple hosts (e.g., louping-ill virus in grouse, hare,

and deer; Gilbert et al. 2001), or greater structure within the host population (e.g., age,

sex, or social status). These modifications include the susceptible-infectious (SI) and

susceptible-exposed-infectious-recovered (SEIR) models, among others. For structured

host populations, differences in contact rates between different subgroups often become

important, replacing the single parameter β with a matrix that describes the transmission

of infection between and within different groups. It is important to note that the

assumption of random mixing still remains despite these modifications, although now it

is limited to transmission within each subgroup separately (Keeling and Eames 2005,

Keeling and Rohani 2008).

2.2.2 Uses and Limitations

Some of the first and most influential uses of compartmental models involved

establishing the conditions necessary to ensure disease persistence or eradication (Smith

and Cheeseman 2002, Keeling and Rohani 2008). Two of the most important quantities

in epidemiology emerged from such analyses. The first, R_0, defines the average number

of secondary cases than would arise from an average primary case in an entirely

susceptible population. Although the exact formulation of this variable is highly

dependent on the type of model used (i.e., SIR versus SEIR), for the SIR model described

above it is given by $R_0 = \frac{\beta}{\gamma+\mu}$. The force of infection, λ, on the other hand, defines the

per capita rate at which susceptible individuals contract the infection and is equal to βI

and $\beta Y/N$, for the proportions and number of individuals formulations, respectively. The

values that these quantities take in different host-pathogen systems in many ways

determine the final outcome of the disease (e.g., R_0 has to be greater than unity for a

disease to spread and less than unity to ensure eventual disease eradication; Keeling and

Eames 2005, Keeling and Rohani 2008, Kawata 2010).

   Anderson et al. (1981), for example, used a compartmental model to demonstrate

that vaccinating at random at least a proportion $p = 1 - 1/R_0$ of the population ensures

the eventual eradication of rabies from foxes. Although numerous methods have been

devised to improve the calculation of this value (e.g., incorporating age or transmission

heterogeneity), the original formulation remains one of the most fundamental and often-

used discoveries in epidemiology (Keeling et al. 2003, Garnett 2005, Yip et al. 2007).

Multi-host models, on the other hand, have been used to investigate the effects of multiple hosts on maintaining infections in situations where a single host would be unable to (Dobson and Meagher 1996, Gilbert et al. 2001).

The main limitation of these models is the mean-field assumption, wherein all individuals in the population contact each other with equal probability. Although certain modifications have been devised to address this concern (e.g., introducing host structure such that certain groups mix more preferentially with each other), it still remains the main drawback of compartmental models. Additionally, because such models are aspatial, they cannot be used to investigate the dynamics of host populations that exhibit significant spatial heterogeneity or to model the spatial spread of an invading disease (Keeling and Eames 2005, Keeling and Rohani 2008, Kawata 2010).

Another issue with compartmental models has centered on how best to model the transmission between susceptible and infected individuals. For most of the field's history, density-dependent transmission was used because of its clear analogy to how the rate of interaction between two types of molecules is directly proportional to their densities. While this framework, which yields the familiar βSI transmission term, has been widely applied with great success, certain populations may not display such a characteristic. For a sexually-transmitted disease in humans, for example, the number of transmissible contacts (i.e., sexual partners) is often independent of population density (Lloyd-Smith et al. 2004, Ryder et al. 2007). For such situations, a frequency-dependent rate of transmission is required, which alters the transmission term to $\beta SI/N$ (McCallum et al. 2001, Begon et al. 2002, Keeling and Rohani 2008).

Although these examples may give the impression that transmission operates only at two different extremes (i.e., either independent or as a function of host density), in most situations it is in fact a continuum of values. Antonovics et al. (1995), for example, modeled this continuum based on the Holling Type II functional response curve, wherein contacts at low densities are proportional to the host density but eventually reach a maximum at higher densities. Because the exact formulation of the transmission term is crucial to a model's predictive and descriptive capabilities, as well as the qualitative behavior of the system itself, the relationship of contact rates to density is still an area of active research. In general, however, frequency-dependent transmission is used primarily in models of vector-borne pathogens and human populations while density-dependent transmission is generally considered to be more applicable to plant and animal diseases (McCallum et al. 2001, Begon et al. 2002, Keeling and Rohani 2008).

Another common use of compartmental models has been to investigate the role of seasonality in producing annual, semiannual, or even chaotic patterns of disease prevalence. Many wildlife populations, for example, experience significant changes in transmission rates throughout the year as a result of flocking behavior (Hosseini et al. 2004), seasonal migration (Bradley and Altizer 2005), or congregation during the breeding and molting season (Swinton et al. 1998). An additional source of seasonality that is almost exclusive to wildlife populations arises from birth pulses which recruit a large number of susceptible individuals into the population at approximately the same time each year (Gremillion-Smith and Woolf 1988, Keeling and Rohani 2008). By incorporating a time-dependent component into the transmission term, Ireland et al. (2004), for example, demonstrated that increasing the amplitude of seasonality increased

the number of prevalence peaks throughout the year, eventually resulting in chaotic dynamics.

2.2.3 Specific Applications

The earliest application of compartmentalized models to infectious diseases in wildlife was presented in the seminal work of Anderson et al. (1981), who developed a deterministic SEI (susceptible-exposed-infectious) model for the fatal rabies virus in European red foxes (Kawata 2010). This model demonstrated that the latent period of rabies can act as a time-delayed density-dependent regulator of fox population growth and result in the four-year oscillations in population size and disease prevalence observed in fox populations throughout Europe. The authors also concluded that culling alone is unlikely to be effective once rabies becomes established within a fox population. Vaccination, on the other hand, significantly increased the odds of disease eradication, especially when combined with culling in regions with low fox density (Anderson et al. 1981).

Gilbert et al. (2001) used a multi-host approach to investigate how the persistence of a tick-borne virus (louping ill-virus) is affected by the presence of grouse, hare, and deer in upland Britain. This model demonstrated that neither deer nor grouse alone could sustain such a virus, but that the combination of the two could, and that the inclusion of hare into this system greatly increased the likelihood of virus persistence (Gilbert et al. 2001). These results were later used by Laurenson et al. (2003) to investigate whether decreasing hare density could be used as a strategy to decrease virus prevalence in grouse. They discovered that not only would this strategy decrease infections in grouse, but it would also increase their fecundity as a consequence of increased life expectancy.

The authors concluded that mountain hares serve as a reservoir species that can maintain the virus as long as hare density remains above 6.5 individuals per km.

Packer et al. (2003) used a simple compartmentalized SI model to demonstrate a very interesting principle – that predator control programs aimed at increasing prey abundance could prove harmful to prey populations that are regulated by infectious diseases rather than by predation. The authors showed that because infected prey live longer in the absence of predators, they are able to infect many more susceptible animals than they would be to otherwise. Although this effect is more pronounced if predators selectively remove infected prey (e.g., if the infection makes prey easier to capture or locate), nonselective predation is still beneficial to such populations by reducing the lifespan of infectious individuals. This principle was used to retroactively explain how the introduction of private gamekeepers (who removed predators) in red grouse habitats destabilized the grouse population in North Yorkshire, England (Hudson et al. 1992, Packer et al. 2003).

As illustrated above, mean-field compartmental models have been used in a wide variety of settings and provided the foundation for much of the epidemiological literature and theory. Despite their extensive application, however, there are still many more aspects of wildlife diseases that could be investigated with these models. For example, although compartmental models have been used to understand the role of seasonal variation in births and transmission rates, the role of seasonality in mortality has yet to be explored. This avenue for future research would be particularly appealing because newly developed theory could be tested by manipulating the timing, extent, and demographic characteristics of mortality (e.g., harvest regulations; Hickling 2002, Erb et al. 2013).

2.3 Metapopulation Models

2.3.1 Model Overview

Although the spread of infectious diseases is predominantly a localized process (i.e., between individuals in the same location), the movement of individuals between aggregate groups can help to facilitate the geographical spread of the disease. Metapopulation models, originally developed for application in ecology, provide a simple but powerful means of capturing this spatial structure by subdividing the entire population into distinct groups known as subpopulations. Although there is some limited interactions between these subpopulations, the separation between them allows each one to have its own independent dynamics (Jesse et al. 2008, Keeling and Rohani 2008, Beyer et al. 2012).

In such a framework, the original SIR equations can be modified as

$$\frac{dX_i}{dt} = v_i N_i - \lambda_i X_i - \mu_i X_i,$$

$$\frac{dY_i}{dt} = \lambda_i X_i - \gamma_i Y_i - \mu_i Y_i,$$

$$\frac{dZ_i}{dt} = \gamma_i Y_i - \mu_i Z_i,$$

where i refers to parameters that are particular to subpopulation i and may vary substantially between subpopulations due to differences in the local environment (Broadfoot et al. 2001, Langlois et al. 2001). Although most of the useful quantities derived from mean-field compartmental models still apply, they must now incorporate, ρ_{ij}, the relative strength of transmission from one subpopulation to another (e.g., $\lambda_i = \beta_i \sum_j \rho_{ij} Y_j / N_i$ vs $\lambda = \beta Y / N$; Keeling and Rohani 2008).

Although metapopulation structure is inherently based on the host population of interest, these models usually fall into one of three categories. Wind- or vector-borne models, for example, assume that subpopulations are epidemiologically sessile, forcing coupling to decrease with distance. Such models have been used extensively to model the spatial dynamics of livestock diseases by treating each farm as a sessile subpopulation (e.g., Ferguson et al. 2001, Keeling 2001) Commuter models, on the other hand, are based on the premise that permanent relocation between subpopulations is rare but temporary travel between them is sufficient for disease spread. Finally, most models of animal diseases rely on the migration or permanent movement of individuals to spread the pathogen (Keeling and Rohani 2008).

Once the appropriate relations are defined, the system of equations can be solved in either a deterministic or stochastic manner. Because this framework is able to capture the spatial clustering of the host population, it has been used extensively not only for the study of infectious diseases, but also in the ecological literature to derive such concepts as the extinction-colonization balance, rescue effect, and isolation paradigm (Hanski 1998, 1999).

2.3.2 Uses and Limitation

One of the most common uses of metapopulation models is to investigate the influence of substructure on disease thresholds and persistence. Group size heterogeneity, for example, can increase disease persistence by allowing smaller subpopulations where the pathogen would normally be unable to persist to continually become reinfected from larger ones where the disease is maintained (Broadfoot et al. 2001). The level of substructure has also been shown to be an important consideration, with higher numbers

of subdivision leading to longer disease outbreaks and increased persistence (Swinton et al. 1998). Similar approaches have also been used to understand the rate and heterogeneity of the spatial spread of a pathogen (e.g., Langlois et al. 2001, López et al. 2009).

A general trend that has emerged from this modeling framework is that given certain levels of aggregation and coupling, a pathogen can persist within the host population for much longer than would be predicted by a mean-field compartmental model (Swinton et al. 1998, Fulford et al. 2002, Keeling and Rohani 2008). Although many modeling studies have concluded that persistence increases with the inter-group connectivity, Jesse et al. (2008) demonstrated that the relationship is actually highly non-linear. More specifically, a peak in epidemic duration appears at small movement rates (due to asynchrony in the disease dynamics between subpopulations), followed by a global maximum at larger rates (when the pathogen "perceives" the metapopulation as a single well-mixed population). These results were particularly applicable to outbreak management because they demonstrated that while restricting animal movement (a common response to disease outbreaks in cattle farms) will reduce the total number of subpopulations that experience infection, it may also prolong epidemic duration instead of decreasing it. Another important conclusion is that although increasing mixing between subpopulations via habitat corridors is an effective means of species conservation (Hanski 1999), their potential as conduits for the spread of infectious diseases should be carefully considered before implementation (Hess 1994, 1996).

Metapopulation models have also been used to evaluate the benefits of different control strategies that take into account the local nature of spatial transmission. Beyer et

al. (2012), for example, demonstrated that rabies fade-out in Tanzania became increasingly likely once vaccination reduced the number of susceptible dogs in each village subpopulation below 150. Fulford et al. (2002) used a similar approach to show that for subpopulations that occur in a chain configuration (e.g., along rivers or roads), limited culling applied to alternate patches was significantly more effective in reducing disease prevalence than extensive culling applied in large batches along the chain.

Apart from the fact that metapopulation models still assume homogeneous mixing (although at the more realistic intra-subpopulation level), their main limitation is that they require detailed information on the number of individuals in each subpopulation, which can be difficult to collect and computationally intensive to model explicitly (Keeling and Rohani 2008). An alternative approach, originally proposed by Levins (1969), models each subpopulation as being either pathologically empty (disease-free) or occupied (having infection). The intuitive way to conceptualize this is to assume that localized extinctions and successful recolonization events are extremely rare, so that each subpopulation spends the vast majority of its time either disease-free or close to the endemic equilibrium. For a large number of subpopulations with equal probabilities of infecting each other, the proportion of infected subpopulation is governed by

$$\frac{dP}{dt} = \rho(1 - P)P - eP,$$

where ρ measures the re-infection (coupling) rate from an infected subpopulation to an uninfected one, and e is the rate of local extinction (Keeling and Rohani 2008). Although this approach has been used much less extensively than the previously described standard approach, it has nonetheless been used to derive some interesting results (e.g., see Smith et al. 2002 below).

2.3.3 Specific Applications

Rushton et al. (2000) created a metapopulation model to investigate the role of parapoxvirus in the observed population and distribution decline of the red squirrel following the introduction of gray squirrels in Great Britain. Although both species can become infected with the virus, gray squirrels show no sign of the disease and as such can act as a reservoir host. By incorporating a disease component into a previously-developed metapopulation model of gray and red squirrels in Norfolk, Rushton et al. (2000) demonstrated that the transmission of parapoxvirus resulted in faster and more sporadic localized extinctions of the red squirrel population than would be expected from inter-specific competition alone. This result called into question proposed conservation management in which animals were to be translocated or given supplementary feeding, actions that would enhance disease spread by increasing contact rates of infected individuals. These actions likely would have accelerated the decline of the red squirrel population as opposed to reversing it, prompting efforts toward vaccine development instead (Rushton et al. 2000).

A very interesting application of this modeling framework was recently employed by Plowright et al. (2011) to provide a mechanistic understanding of the causal links between anthropogenic change and emergence of new zoonoses from wildlife in Australia. As urbanization destroyed most of the contiguous forest cover on the eastern coast, fruit bats began switching their diet from the more patchily distributed nectar and fruit sources to the year-round abundant food in urban gardens. Unlike the old diet that required long-distance foraging and migration, the new one required minimal migration, which decreased the connectivity between daytime roost (camp) subpopulations. By

incorporating data on within- and between-camp transmission, the authors demonstrated that the decreased migration led to increased epidemic sizes and divergence in both amplitude and frequency from epidemics in rural camps. This was due to the fact that as fewer individuals migrated, the probability of infected hosts moving between camps decreased, which lowered the probability of camps becoming reinfected after local viral extinction and increased the time over which subpopulations could recruit susceptible individuals via births. This resulting decline in herd immunity across the metapopulation shifted disease dynamics toward sporadic, shorter, and more intense local epidemics with larger numbers of infected individuals. However, if this connectivity were to drop below a certain threshold level, the resulting movement would become insufficient to reinfect distant camps. Thus, the same conditions that are currently responsible for large and sporadic epidemics could eventually lead to viral extinction, rendering the entire population vulnerable to a large synchronized outbreak (Plowright et al. 2011).

When a large wave of rabies infections began on the Virginia-West Virginia border in the mid-1970s, Smith et al. (2002) used a Levins-type model to study a small portion of this wave as it traveled through raccoons in 169 townships across Connecticut from 1991-1996, concentrating on the underlying spatial heterogeneity of the habitat. This spatial SI model (because there is no recovery from infection at the township-level) was compared to the observed data of reported rabies outbreaks, and resulted in a best fit model where rivers reduced transmission by 87% compared to land boundaries, local transmission accounted for most but not all of the spatial spread, and that human population density played a small but positive role in transmission (Smith et al. 2002). Although this formulation ignored within-subpopulation dynamics (i.e., a newly-infected

township can transmit infection as strongly as when endemic equilibrium is reached), this type of Levins approximation allowed for far richer parameterization and even circumvented the difficulty of estimating the raccoon population levels within each township (Keeling and Rohani 2008).

As illustrated above, metapopulation models have provided important insights into the complexities and spatial variation observed in disease prevalence and outbreaks. An exciting avenue for future research is to devise a practical framework for ascertaining the optimum timing and duration of vaccination pulses to promote the extinction of a pathogen at the population-level. Although theoretical research has demonstrated that pulsed vaccination has the ability to synchronize epidemic behavior and thereby limit pathogen rescue effects, understanding how this benefit interacts with the resulting buildup of susceptibles between pulses can have enormous public and animal health consequences (Keeling and Rohani 2008).

2.4 Lattice-Based Models

2.4.1 Model Overview

Lattice-based models subdivide the area occupied by the host species into a grid, and thus, provide a way to model situations where the spatial location of the host is important but there is no natural way to divide the population into distinct groups. These models traditionally fall into two main categories – coupled lattice and cellular automata (Keeling and Rohani 2008).

Coupled lattice models can be thought of as a special case of metapopulation models, where subpopulations are arranged on a homogeneous grid and interactions

occur only between neighboring adjacent grid cells, and as such are governed by a very

similar set of equations:

$$\frac{dX_i}{dt} = \upsilon - \beta X_i \frac{(1 - \sum_j \rho_{ji})Y_i + \sum_j \rho_{ij}Y_j}{(1 - \sum_j \rho_{ji})N_i + \sum_j \rho_{ij}N_j} - \mu X_i,$$

$$\frac{dY_i}{dt} = \beta X_i \frac{(1 - \sum_j \rho_{ji})Y_i + \sum_j \rho_{ij}Y_j}{(1 - \sum_j \rho_{ji})N_i + \sum_j \rho_{ij}N_j} - \gamma Y_i - \mu Y_i,$$

$$\frac{dZ_i}{dt} = \gamma Y_i - \mu Z_i,$$

where ρ_{ij} is equal to one if i and j are neighbors and zero otherwise (Keeling and Rohani

2008).

Cellular automata models, on the other hand, typically treat each grid cell as being

big enough to contain only a single individual (a subpopulation of one). This results in a

finite number of states for these subpopulations, wherein each cell is either empty or

occupied by a susceptible, infectious, or recovered individual (Keeling and Rohani 2008,

Miksch et al. 2013). Grid cell transitions are evaluated stochastically using transition

probabilities in either a contact (for an SIS pathogen) or forest-fire (for an SIRS

pathogen) process (Keeling and Rohani 2008). The latter of these follows a framework

originally developed by Bak et al. (1990), where each cell was either empty or occupied

by a healthy or burning tree (reflecting a recovered, susceptible, and infectious host,

respectively). Burned trees that died left empty spaces which could then be colonized

(reflecting recovery and births), and fire (reflecting infection) could spread between

neighboring trees.

2.4.2 Uses and Limitations

Like all mathematical models, lattice-based models are a clear abstraction of reality, because except for in agricultural settings such as orchards (e.g., Gibson 1997, Poggi et al. 2013), individuals do not exist on a regular grid. Because of this, these models are seldom used to provide accurate predictions of disease spread but are instead used to investigate how the effects of spatial separation and non-random mixing cause disease dynamics to deviate from mean-field approximations. Coupled lattice models, for example, provide a definite method of including the spatial location of individuals, thereby allowing researchers to predict the expected spread of an infection. The most notable feature of this model is that an invading infection is characterized by a clear wave-like spread because the disease must spread to neighboring sites before reaching the rest of the population.

Cellular automata models, on the other hand, are used mainly to understand how the stochastic inclusion of individuals influences disease spread. Two general trends have emerged from the use of these models. The first is that the early rate of growth and spread of an epidemic is significantly lower than during later phases. This is caused by the rapid depletion of the locally available pool of susceptible individuals, and is illustrated by the fact that although the initial seed infection has four neighbors to which it can spread, the next infected cell will only have three (four minus the already-infected seed cell; Keeling and Rohani 2008). The second trend is that the frequency distribution of different sized epidemics follows an approximate power-law relationship (i.e., frequency is inversely proportional to size). Although this relationship holds only if the rates of transmission, recovery, births, and random imports of seed infections are in descending order, this

happens to be the case in many epidemiological studies (e.g., Bak et al. 1990, Rhodes et al. 1997).

Although lattice-based models are a clear misrepresentation of the spatial structure of most host populations, certain types of wildlife populations lend themselves very naturally to be modeled using such an approach. Populations that occur in a one-dimensional chain (e.g., along a river) or loop (e.g., surrounding an impassable urban center) can be thought of as having only nearest-neighbor connectivity, and as such can easily be modeled using either the coupled lattice or cellular automata approach. More generally, lattice models have also been used to evaluate the broad-scale consequences of different control efforts including vaccination (e.g., Tischendorf et al. 1998) and culling (e.g., White and Harris 1995, Deal et al. 2004).

Despite these uses, the separation of populations into a lattice framework has several drawbacks. Imposing a square grid onto a habitat, for example, forces interactions in particular directions (e.g., north, east, south, or west) to play a disproportionately larger role in disease transmission than interactions in other directions, thereby removing the property of isotropy (Keeling and Rohani 2008). The use of a hexagonal grid, however, helps to alleviate this problem and often better recreates observed spatial patterns (Anneville et al. 1998, Van Baalen and Rand 1998, Kao 2003, Birch et al. 2007). A more general issue is that the discretization of space forces intra-grid interactions to be far stronger than inter-group ones, regardless of the actual distance between individuals in each cell. Two individuals that occupy adjacent corners of two separate cells, for example, would have weaker interaction than two who occupy the same cell but are at

opposite ends, despite the fact that the former are closer to each other than the latter (Keeling and Rohani 2008).

Another limitation of these models is that the small number of neighbors with which individuals can interact cannot capture the complex and often heterogeneous contact structure in wildlife populations through which a pathogen can spread (Keeling and Rohani 2008). The use of higher dimensionality (i.e., >2-dimensional grids) has been proposed as a means of capturing this clustering and heterogeneity. Although this approach has yet to be applied in the wildlife disease literature, Rhodes et al. (1997) used it to demonstrate that a five-dimensional lattice was necessary to recreate the observed patterns of mumps outbreaks in humans living in the Fareo Islands, while only three dimensions were required for measles and whooping cough. This helped to reinforce the notion that even diseases spreading through the same host population by similar means may operate on significantly different levels of interactions, suggesting that multiple lattice dimensions need to be tested before a conclusion can be drawn.

2.4.3 Specific Applications

In 1997, a large-scale and long-term immunization of European foxes against rabies was under consideration to be terminated or cut back on due to diminishing returns and despite lasting sporadic incidences. Tischendorf et al. (1998) used a modified lattice-based model to investigate the effectiveness of this program and assess the consequences of diminishing it on rabies persistence. This approach involved simulating the mating, dispersal, and vaccination of fox social groups across a lattice grid, as well as the contact between and within them. Unlike the traditional approach, each grid was able to hold one of six possible states – the three 'simple' states of infectious, empty, and susceptible, and

three new 'mixed' states of susceptible plus immune, infectious plus immune, and empty plus immune (meaning that only immune foxes are present). The results of this model showed that even in a highly immunized fox population such as the one throughout Europe, rabies can still persist in the form of spatio-temporal moving infection clusters. The probability of disease eradication rises sharply after a mean immunization rate threshold of 70%, although a further six years of maintaining this level was required to guarantee rabies eradication (Tischendorf et al. 1998).

In Southwest England, efforts to eradicate bovine tuberculosis from cattle continue to be complicated by the presence of a wildlife reservoir for the disease in European badgers (*Meles meles*). White and Harris (1995) developed a stochastic lattice-based model that incorporated density-dependent fecundity and cub survival and used badger social groups as the basic unit of measurement. The major result of this model showed that although bovine tuberculosis could persist for a long time in populations with a disease-free equilibrium group size of only four individuals, a group size threshold of six was required for the disease to become endemic. Additionally, increasing intergroup contact rates significantly improved the probability and rate of spread of infection and lowered the group size threshold required for endemicity. As such, perturbations of badger social groups caused by control operations could actually increase the probability of persistence (or spread) of an infection instead of preventing it (White and Harris 1995).

In the mid-1980s, Mollison and Kuulasmaa (1985) used a similar modeling approach to demonstrate a very interesting phenomenon: that distinct pockets of infection could form despite the homogeneity of the imposed landscape. These authors showed that

although a rabies epidemic begins by advancing in a fairly regular manner, after a certain threshold the wave front often breaks out into an endemic pattern of "wandering" patches of infection. Any environmental heterogeneity would then act not only to increase the probability of formation of such patches, but also to increase their spatio-temporal stability (Mollison and Kuulasmaa 1985). This theoretical pattern was later used to explain how increased environmental heterogeneity resulted in an increased risk of transmission of bovine tuberculosis from badgers to cattle (White et al. 1993), as well as an increased potential for disease spread through the badger population itself (White and Harris 1995).

As illustrated above, the use of lattice-based models has provided managers with not only the tools to evaluate different control strategies, but also with explanations for observed disease distribution. Although the use of higher dimensionality in lattice-based models has thus far been applied only to human populations (e.g., Rhodes et al. 1997), exploring its application to host-pathogen systems in wildlife can be an exciting area for future research.

2.5 Reaction-Diffusion Models

2.5.1 Model Overview

The main disadvantage of the lattice-based approach is that dividing the space occupied by a population into discrete cells limits how precisely we can model the location of each individual (i.e., we only know which cell an individual is in but not where inside the cell they are). By instead treating this space as continuous, reaction-diffusion models allow us to model the *exact* location of each individual in the population. This approach is essentially an extension of the lattice-based framework (where the size of each cell has

becomes infinitely small) and can be described using either partial differential equations
(PDEs) or integro-differential equations (IDEs; Schofield 2002, Ruan and Xiao 2004,
Keeling and Rohani 2008).

The PDE approach assumes that individuals more randomly throughout the
landscape and that infections are only transmitted by individuals sharing the same
location (Källen et al. 1985, Murray et al. 1986, Yachi et al. 1989, Kawata 2010),
resulting in the following set of equations:

$$\frac{\partial X}{\partial t} = \upsilon - \beta XY/N - \mu X + D_X \nabla^2 X,$$

$$\frac{\partial Y}{\partial t} = \beta XY/N - \gamma Y - \mu Y + D_Y \nabla^2 Y,$$

$$\frac{\partial Z}{\partial t} = \gamma Y - \mu Z + D_Z \nabla^2 Z,$$

where X, Y, and Z are now functions of both space and time, ∇^2 describes the local
diffusion of individuals through space, and D_X, D_Y, and D_Z, are the diffusion rates for the
three classes of individuals (Keeling and Rohani 2008, Kawata 2010).

The standard IDE approach expands upon this formulation by incorporating a
transmission kernel which models how transmission risk decreases with distance. This
kernel acts to relax the assumption that transmission is solely a localized process,
allowing individuals not in the immediate vicinity of an infected individual to still
become infectious, albeit at a significantly lower rate (Schofield 2002, Keeling and
Rohani 2008).

These standard approaches can be expanded upon by incorporating spatial
covariates into the diffusion rates for the three classes of individuals, such that certain

landscape characteristics (e.g., ridges and valleys) impede or accelerate the diffusion of individuals. The resulting diffusion surface yields disease dynamics that no longer conform to the homogeneous point diffusion process, but instead spread out faster in some directions and areas of the landscape than in others (Moore 1999).

2.5.2 Uses and Limitations

Because reaction-diffusion models provided a very broad-scale view of disease outbreaks, their application has foremost been limited to investigating epidemics that span large spatial scales (Conner and Miller 2004). One of the most common uses of PDE models, for example, is to identify how landscape-level heterogeneity in the environment affects disease spread dynamics. Because the pathogen spreads as a growing circular wave of constant velocity in the simplest of formulations, such an approach provides the perfect means of evaluating how valleys, mountain ranges, or even elevation in general influence the speed and direction of disease spread (e.g., Källen et al. 1985, Moore 1999).

IDE models, on the other hand, are more often used to investigate the presence and role of rare but long-term disease transmission (e.g., dispersing animals) in accelerating the spatial diffusion of a newly-introduced pathogen. The major trend that has emerged from such studies is that the tail of the kernel distribution, which determines the frequency and extent of long-range transmissions, ultimately determines the spatial pattern of the disease spread. The exact shape of this tail (e.g., height and length) can yield patterns ranging from the simple wavelike spread seen in PDE models to scattered loci and a highly heterogeneous wavefront (Schofield 2002, Keeling and Rohani 2008).

Both the PDE and IDE formulations have also been used extensively to study the spread of vector-borne diseases and their carriers. Caraco et al. (2002), for example,

developed a stage-specific reaction-diffusion model to study the rate at which emerging

diseases advance, demonstrating that the spread of Lyme disease is highly correlated with

the density of infectious vectors. Ruan and Xiao (2004), on the other hand, took a broader

approach and used PDEs to investigate the dynamics of a host-vector-pathogen system

with heterogeneous latent periods.

One of the main drawbacks of the reaction-diffusion approach is that except for in

the simplest situations, the defined system of equations cannot be solved analytically.

This forces scientists to resort to numerical simulations which require discretization of

space, breaking the continuous-space foundation of such models. In this way, most

reaction-diffusion models are approximated by very fine-scaled lattice-based models

(Keeling and Rohani 2008).

2.5.3 Specific Applications

In 1977, a single rabies-positive raccoon was identified in Pennsylvania and determined

to be a byproduct of a restocking program that transported raccoons from Florida to

Virginia and West Virginia in the mid-1970s. By 1996, the rabies virus spread to almost

all counties in Pennsylvania, resulting in 3912 confirmed cases of infected raccoons and a

further 2137 cases in other animals. Moore (1999) used the timing of first cases in each

county to construct a diffusion surface for the entire state in order to explore what factors

influenced the speed and direction of the spread. The resulting contours revealed that the

infection first spread northward along the corridors of the Appalachian Mountain and

Great Valley sections of the state before twisting to the west once it reached the high

plateau areas in the north. These results differed significantly from the homogeneous

point diffusion process originally proposed for the rabies outbreak and were used to inform a strategic oral-bait vaccination strategy (Moore 1999).

Sayers et al. (1985) used a similar approach to investigate how the diffusion of fox rabies through cities in Germany was determined by geographic features. The derived pathogen velocity vectors demonstrated that propagation was fastest along a broad range of limestone with elevations up to 600m and diverse land-cover and vegetation. These results were later used by Källen et al. (1985) to design a deterministic model for the spread of this epizootic front westward across Europe in order to help identify the width of a control barrier that would prevent the disease from entering the rabies-free United Kingdom. This model predicted a front wave speed of 50km/year, consistent with empirical data from the German-Polish border, and resulted in an estimated barrier width of 15km (Källen et al. 1985). This westward spread was eventually halted through the delivery of a highly effective oral vaccine (Jackson and Wunner 2002).

Schofield (2002), on the other hand, compared the ability of PDE and IDE frameworks in recreating the observed spatio-temporal spread of Wolbachia in a species of fly. The authors demonstrated that although the IDE approach provides a better approximation, its accuracy is highly dependent on the exact shape of the transmission kernel. This study helped to conclude that despite their intuitive appeal, normally-distributed kernels are often very inaccurate representations of reality. Additionally, the authors provided a framework for the inclusion of imperfect maternal transmission of a disease in reaction-diffusion modeling.

Despite their limited use, reaction-diffusion models have provided invaluable insights into the dynamics of point diffusion and traveling wave fronts in disease

progression. Recent interest in these models has focused on the existence of travelling

wave solutions, where the spread of vector-borne pathogens that confer no immunity can

give rise to a moving zone of transition from an infective to a disease-free state. Although

Ruan and Xiao (2004) have established the existence and dynamics of such waves for

some specific types of diffusion kernels, their dynamics in other situations is an area for

future investigation. Additionally, the increasing interest paid to vector-borne diseases in

general, driven by such recent outbreaks as that of the Chikungunya virus in France

(Cavrini et al. 2009), provides an excellent basis from which to explore multi-vector

pathogen diffusion in a variety of landscapes.

2.6 Network Models

2.6.1 Model Overview

Although many infectious disease models assume random mixing either within the host

population as a whole or within each subgroup separately, the number of contacts each

individual has in a real population is significantly smaller than the (sub)population size

and is highly heterogeneous between individuals. The contact network approach,

originally developed for application in statistical physics, provides a natural means of

studying disease dynamics in such situations (Craft and Caillaud 2011). In this

framework, individuals or groups of individuals are represented as nodes, and

connections between them are referred to as edges, which can be either undirected (for

infections that can pass in both directions with equal probability), or directed (for

infections that can pass better in one direction than the other; Keeling and Eames 2005,

Keeling and Rohani 2008). These connections may also be binary (representing whether

any contact has occurred or not) or weighted (representing the duration or frequency of contact) depending on the host-pathogen system of interest (Godfrey 2013).

The simplest way to represent a contact network is to construct an $N \times N$ adjacency matrix, $\boldsymbol{G}$, where N is the number of individuals in the study population and $\boldsymbol{G_{ij}}$ represents the presence, duration, or frequency of contact between individuals i and j. Although this a matrix is usually symmetric (i.e., $\boldsymbol{G_{ij}} = \boldsymbol{G_{ji}}$), directed networks yield non-symmetric matrices (i.e., $\boldsymbol{G_{ij}} \neq \boldsymbol{G_{ji}}$; Keeling and Eames 2005). A number of useful quantities have been derived using adjacency matrices to describe how connections within a network are structured. These include simple measures such as degree (number of edges connected to a single node; Craft and Caillaud 2011) and clustering coefficient (the extent to which an individual's neighbors are connected to each other; Godfrey 2013), alongside newer measures such as "small-world-ness" (the interaction of local clustering and average path-length; Humphries and Gurney 2008).

Although networks are inherently host-pathogen-specific, they usually fall into one of five main categories. Random networks, for example, ignore the spatial position of individuals and randomly generate connections, while lattices are based on regularly-spaced individuals with a fixed number of contacts (usually the four or eight nearest neighbors). Small-world networks expand upon the lattice formulation by also allowing a small number of long distance connections. Spatial networks, on the other hand, use a kernel to calculate the probability of contact between two individuals based on the distance between them. Finally, scale-free networks capture the heterogeneity in contacts (i.e., most individuals having relatively few contacts and a couple having significantly more) by adding new individuals one at a time and connecting them to the existing

network based on observations of (social) contact structures (Keeling and Eames 2005, Keeling and Rohani 2008, Craft and Caillaud 2011).

Once the appropriate contact network and associated adjacency matrix is constructed, transitions between disease statuses are evaluated in discrete time steps (e.g., once every three months) on an individual basis using predetermined probabilities (e.g., a susceptible individual who is in contact with an infectious one has a 0.4 probability of becoming infected, while the infectious individual has a 0.2 probability of recovering; Keeling and Rohani 2008). This framework is able to capture complex individual-level structure and heterogeneity in a relatively simple manner and as such has been used extensively not only for wildlife diseases, but also for a variety of human ones such as HIV (Sloot et al. 2008) and SARS (Small and Tse 2005). Network models have also been used in other disciplines, such as sociology, to measure actor prestige (Korfiatis and Sicilia 2007) and to investigate dynamics of rumor spreading (Moreno et al. 2004).

2.6.2 Uses and Limitation

One of the most common uses of network models is to investigate the role of degree heterogeneity and well-connected individuals (i.e., super-spreaders) in driving disease dynamics (Böhm et al. 2009, Craft and Caillaud 2011, Hamede et al. 2012). For example, because these super-spreaders can have a disproportionately high effect on the basic reproductive rate, R_0, and the epidemic threshold, network models are often used to evaluate how vaccinating or quarantining them will influence disease spread (Craft and Caillaud 2011, Tompkins et al. 2011). Normally, such well-connected individuals would be considered to be at highest risk of infection (Godfrey 2013). However, while this relationship does exist for many wildlife populations (e.g., brushtail possums; Leu et al.

2010), it is entirely absent in others (e.g., meerkats; Drewe 2009). It is also important to remember that although super-spreaders have been identified in many wildlife populations, they appear to be absent in others (e.g., Tasmanian devils; Hamede et al. 2009).

A general trend that has emerged from analyzing network models is that high variation in the degree distribution tends to increase the epidemic threshold and basic R_0 when compared to mean-field compartmental models that use the average degree as a proxy for transmission rates (Hamede et al. 2012). Although several researchers have proposed methods to account for such heterogeneity in mean-field models (e.g., Anderson et al. 1986, Newman 2002), their explicit inclusion via network modeling still provides the best predictor of disease spread (Hamede et al. 2012).

The use of alternative networks, on the other hand, has been used to gain insights into the importance of different types of animal behavior in disease transmission and to evaluate support for alternative working hypotheses (Godfrey 2013). Drewe (2009), for example, developed separate networks for grooming and aggressive interactions in meerkats and found that not only are the two highly directional (e.g., groomers had a higher risk of tuberculosis infection than groomees), but also that grooming was the most important type of social interaction. Fenner et al. (2011) used a similar approach to demonstrate that connectivity to dispersers was more important than connectivity to resident individuals in predicting nematode loads in gidgee skinks.

Networks have also been used to show that group size alone does not necessarily reflect transmission rates (Godfrey 2013). Godfrey et al. (2009), for example, found no relation between group size and risk of infection in gidgee skinks, while Griffin and

Nunn (2011) demonstrated that larger primate groups offset parasite risk by imposing within-group substructure. Network models have also been used to show that social interactions are often more important in disease spread than spatial proximity or home range overlap (Bull et al. 2012), address the issue of using different spatial scales (Davis et al. 2008), and investigate if density influences contact rates (Ji et al. 2005). Alongside these more specific applications, network models have of course been used in much the same way as earlier model types – to evaluate a wide range of disease control measures such as oral vaccines (Delahay et al. 2009) and culling (Ramsey et al. 2002).

As has been illustrated above, one of the main advantages of network models is their versatility, allowing them to be applied to virtually any host-pathogen system (e.g., both directly- and indirectly-transmitted viruses, bacteria, and parasites; Godfrey 2013). In fact, they can be parameterized to recreate almost any other model type (e.g., a compartmental model is simply a fully-connected network, while a lattice-based model can be replaced by a lattice network with nearest-neighbor connectivity; Craft and Caillaud 2011). The main limitation of such models, however, is that accurate parameterization requires detailed data on the contact and movement patterns of every individual within the study population at a time scale appropriate for the transmission process (Godfrey 2013). Although ongoing advances in GPS and proximity sensor technology is helping to alleviate this problem (e.g., Leu et al. 2010, Hamede et al. 2012), accurate sampling of every individual is still financially and logistically unfeasible except for the smallest of wildlife populations (Craft and Caillaud 2011, Tompkins et al. 2011, Godfrey 2013). Because of this, most studies (e.g., Craft et al. 2010, Hamede et al. 2012) use a "representative" subset of the population to generate contact networks that recreate

observed measures of connectivity (e.g., average number of contacts, degree distribution, or amount of clustering; Keeling and Eames 2005, Craft and Caillaud 2011, Tompkins et al. 2011).

Another challenge in using contact networks is defining what constitutes a transmissible contact (e.g., what proximity or duration is required to transmit an aerosol pathogen; Craft and Caillaud 2011, Tompkins et al. 2011). Because it is often impossible to determine exactly how transmission occurs without resorting to controlled transmission experiments that are often infeasible, most studies use spatial proximity (e.g., Fenner et al. 2011) or home range overlap (e.g., Godfrey et al. 2010) as proxies of contact and transmissible events (Craft and Caillaud 2011, Godfrey 2013). Additionally, wildlife networks often, if not always, exhibit temporal variation (e.g., higher contact rates while females are in estrous; Rushmore et al. 2013) that can drastically alter disease dynamics, requiring long-term observations for accurate parameterization (Volz and Meyers 2007, 2009, Hamede et al. 2012).

2.6.3 Specific Applications

Although most contact networks are extrapolated from a supposedly representative subset of the study population (Craft and Caillaud 2011), certain situations (e.g., endangered or very large animals) may still allow researchers to explicitly capture the full network of contacts. Leu et al. (2010), for example, constructed a directed transmission network for a subpopulation of sleepy lizards in order to determine whether asynchronous use of overnight refuges (a form of indirect and directional transmissible contact) could account for observed ectoparasite loads in individual lizards. Simulating disease spread on this network provided a powerful predictor of ectoparasite loads and revealed that individuals

who use many refuges had significantly lower loads as a result of a lower chance of using a previously occupied refuge. This study helped to not only identify increasing the number of available refuges as the optimum strategy for reducing disease in sleepy lizards, but also provided one of the first frameworks for using contact networks to investigate the dynamics of indirectly transmitted diseases (Leu et al. 2010).

Network models have been used extensively to demonstrate that heterogeneity in the connectivity or degree of individuals can have drastic consequences for the epidemic threshold and rate of spread of a disease. However, it is important to realize that a population's contact structure often operates on multiples hierarchical scales (i.e., individuals form groups that exhibit their own heterogeneous contact network; Craft and Caillaud 2011). Caillaud et al. (2013), for example, constructed an undirected, two-level (i.e., individuals interacting within groups alongside inter-group connectivity) hierarchical model for a hypothetical infectious disease to demonstrate that variation in group size significantly reduces the epidemic thresholds, as well as increases the mean and variance of small outbreaks sizes. This suggests that incorporating group size heterogeneity into disease models can greatly improve their predictive capabilities, much like the incorporation of degree heterogeneity described previously. The authors also introduced the concept of an epidemiological effective group size, defined as the group size in a homogeneous population that would result in the same epidemic thresholds as the heterogeneous one. By applying this concept to lions in the Serengeti National Park, Caillaud et al. (2013) demonstrated that the observed network, which exhibits highly variable pride sizes, is comparable to a homogeneous population with approximately 10-20% larger prides.

Recognizing the importance of temporal variation in contact structure, Hamede et al. (2012), used proximity-sensing radio collars to derive seasonal patterns of contact in a distinct population of Tasmanian devils. These patterns were then used to generate sets of contact networks with suitable characteristics (i.e., mean degree and transitivity) that regenerated associations once every three months. By stochastically simulating the spread of devil facial tumor disease through these dynamic contact networks, the authors demonstrated that failure to account for seasonal contact structure significantly overestimated the time to host extinction as well as the transmissibility threshold necessary for an epidemic to occur. Interestingly, the differences in time to host extinction between network and compartmental models became negligible for transmission rates close to zero or one, suggesting that contact heterogeneity has little effect on the dynamics of host extinction from disease with either very low or very high transmissibility. Unfortunately, because transmission rates are likely to be intermediate for most wildlife diseases, ignoring contact structure can have drastic consequences for wildlife conservation (Hamede et al. 2012).

As illustrated above, the use of network models to study the patterns of and driving forces behind disease spread has been growing in the wildlife literature despite many challenges. As technological advances continue to lessen these challenges, network models can provide the means to investigate many new facets of disease ecology, including how infection alters network topography (Croft et al. 2011) or how parasites influence the evolution of social organization (Godfrey 2013). Another potential avenue for future research is to develop generalities about what ecological variables (e.g., resource or predator distribution) govern the establishment of contact structure, allowing

39

researchers to extrapolate their findings to other areas or even time periods (Craft and Caillaud 2011, Tompkins et al. 2011).

2.7 Management Implications

The use of epidemiological models in the study of wildlife diseases began in the early 1980s and has since then come to dominate the wildlife disease literature. Although mean-field compartmental models laid much of the foundation for current epidemiological practices (e.g., the basic reproductive ratio and vaccination threshold), their inability to capture spatial dynamics led to the use and evolution of metapopulation, lattice-based, and reaction-diffusion models (Keeling and Rohani 2008, Kawata 2010). More recently, the use of contact networks has emerged as an intuitive and highly versatile means of studying disease dynamics. As ongoing advances in GPS and proximity sensor technology make it more feasible to capture the level of data required to accurately parameterize contact structure, network modeling will continue to rise to the forefront of the infectious disease literature (Keeling and Eames 2005, Tompkins et al. 2011, Godfrey 2013).

Although much research has evaluated the uses, limitations, and dynamics of different infectious disease models, many potential avenues for future research still exist for each one. For example, although previous research using compartmental models has assessed the role of birth and transmission seasonality in producing annual, semiannual, and even chaotic patterns of disease prevalence (Hosseini et al. 2004, Ireland et al. 2004), research investigating the role of seasonal patterns of mortality has yet to be undertaken. The development of generalities about what ecological variables (e.g., resource or

predator distribution) govern the establishment of contact structure is also of high priority (Craft and Caillaud 2011, Tompkins et al. 2011).

The ultimate goal of epidemiological models such as those discussed in this paper is to allow scientists to understand the mechanisms behind and predict the future course of disease spread. As the use and advancement of these models continues, they will undoubtedly continue to aid efforts to minimize risks to human health, decrease loss of livestock, and preserve native or endangered species.

CHAPTER THREE

The role of nearby large-diameter cavity trees in the selection of den sites by female fishers in northern Minnesota

3.1 Introduction

The fisher (*Martes pennanti*) is a medium-sized carnivore native to the boreal forests of North America whose pre-European distribution encompassed much of the northeastern and western United States, parts of the Great Lakes and Rocky Mountains, and southern portions of Canada (Gibilisco 1994, Zhao et al. 2012). However, ongoing changes to forest structure caused by extensive logging and altered fire regimes, unregulated trapping, and habitat loss associated with urban and recreational development resulted in the fisher being extirpated from many areas of the United States by the early 1930s (Powell 1982, Powell and Zielinski 1994, Purcell et al. 2009, Zhao et al. 2012). Although many remnant populations throughout the United States have now recovered due to trapping restrictions, reintroductions, and reforestation (Gibilisco 1994, Purcell et al. 2009), continued concerns over their limited abundance and extent throughout parts of their range have prompted ongoing research aimed at identifying limiting factors and habitat requirements (e.g., Purcell et al. 2009, Zielinski et al. 2010, Zhao et al. 2012).

Studies have shown that structures suitable for resting and denning are crucial for maintaining fisher populations (Lofroth et al. 2010, Weir et al. 2012, Zhao et al. 2012), and that fisher selection for these components is much stronger than selection for foraging locations (Kelly 1977, Arthur et al. 1989, Jones and Garton 1994, Powell and Zielinski 1994, Zielinski et al. 2006). Although selection of rest sites and structures has been extensively studied at multiple spatial and temporal scales (e.g., Kilpatrick and

Rego 1994, Zielinski et al. 2004, 2006, Purcell et al. 2009), research on multi-scale selection of natal (i.e., those used for parturition) and maternal (i.e., those subsequently used for rearing) dens is more scarce in the literature (Zhao et al. 2012). Reproductive female fishers are obligatory cavity users, exclusively using cavities in large-diameter trees for birthing and rearing kits between late February and mid-June (Coulter 1966, Leonard 1980, Paragi 1990, Paragi et al. 1996, Raley et al. 2012). Because fisher kits are altricial, females select den structures to minimize predation, infanticide, and wind exposure while maximizing capture of solar radiation (Powell 1982, Paragi et al. 1996, Raley et al. 2012). Female fishers can use up to five different cavities during a single rearing season, relocating kits between them until they are weaned at five months of age (Paragi et al. 1996, Zhao et al. 2012).

Although several studies have evaluated fisher selection of den structures across their natural range (e.g., Paragi et al. 1996, Weir and Harestad 2003, Weir and Corbould 2008, Davis 2009, Higley and Matthews 2009, Thompson et al. 2010, Raley et al. 2012), a concise theory of den site selection is hampered by the inconsistency in how these studies approach selection. Analyzing habitat selection typically involves comparing used sites and associated characteristics to unused sites that are deemed available to the animal (Johnson 1980, Manly et al. 2002, Meyer and Thuiller 2006, Northrup et al. 2013), and the definition of what is "available" can drastically alter the conclusion drawn (Johnson 1980, Northrup et al. 2013). Previous studies of fisher denning habitat selection have used different criteria for defining what is available, including trees used as rest sites (e.g., Thompson et al. 2010), trees of the same species within the same plot (e.g., Weir and Harestad 2003), random points within the home ranges (e.g., Davis 2009), and

random points centered on trees within the study area (e.g., Zhao et al. 2012). Because of inconsistencies in how availability is defined between these and other studies, we propose two adjustments to future and ongoing research of denning habitat selection by fishers: (1) the spatial extent of what is available should be defined separately for each animal based on movement behavior (e.g., home range estimates, movement rates), and (2) only trees that meet certain criteria (e.g., presence of a suitable cavity) should be classified as available.

Habitat selection is a hierarchical process in which the selection and availability of resources at finer scales is dependent upon choices made at larger scales (Johnson 1980, Weir and Harestad 2003, Meyer and Thuiller 2006, DeCesare et al. 2012). One method of accounting for this hierarchy in studies of habitat selection is to constrain the availability of resources to the extent of the home range (e.g., resources outside an animal's home range should not be considered available once the home range is established; Arthur et al. 1996, Dickson and Beier 2002, Rhodes et al. 2005, Simcharoen et al. 2008). Only considering resources that fall within some buffer of animal locations as available would yield similar results. These methods help ensure that resources far outside the area an animal moved through (i.e., resources the animal could not have reached or been aware of given previous locations and movement characteristics) would not be used as a comparison to what the animal selected. As such, we recommend that the spatial extent of what is available in terms of fisher den sites be constrained by either the home range or estimated locations of each individual animal, instead of comparing used to unused sites throughout an entire study area or other larger extent.

Further, because of the obligatory use of large-diameter cavity trees as den sites by female fishers (Coulter 1966, Leonard 1980, Paragi 1990, Paragi et al. 1996, Raley et al. 2012), comparing used cavity trees to trees that have no cavity suitable for establishing dens is not very informative. To make more meaningful inferences about den selection, we recommend that not all trees be included in the definition of what is available (i.e., trees that are too small in diameter or that do not have at least one cavity large enough to contain a fisher den should be excluded). Although there are no clear requirements as to what characteristics determine the suitability of trees for denning by fishers, numerous studies (e.g., Paragi et al. 1996, Weir and Harestad 2003, Higley and Matthews 2009, Thompson et al. 2010, Niblett et al. 2015) have reported characteristics of trees that have been used in the past (e.g., size, species, and health). We recommend using these characteristics as a starting point to restrict availability to a particular set of characteristics such as the presence of a large cavity and a minimum size based on the smallest tree fishers have been recorded using.

We apply these two recommendations to assess whether the nearby abundance of suitable cavity trees influences the selection of den sites by female fishers. Fishers relocate kits multiple times throughout the rearing season, and although the exact cause of den relocations is unknown, females of other mammalian species such as Canada lynx (*Lynx canadensis*), striped skunk (*Mephitis mephitis*), and arctic fox (*Alopex lagopus*) relocate dens in order to gain access to areas with increased prey availability (Laurenson 1993, Olson et al. 2011), reduce the accumulation of excrement and parasites (Butler and Roper 1996, Lariviere and Messier 1998), avoid attracting predators (Prestrud 1992), and in response to anthropogenic disturbance (Linnell et al. 2000, Argue et al. 2008). In

fishers, these relocations expose kits to increased predation by ground-dwelling predators such as bobcats, as well as infanticide by adult male fishers (Hodgson 1937, Leonard 1980, Powell 1982). We believe that because of the need to relocate kits to different maternal dens several times before weaning, females will select landscapes within their home range that have a higher abundance of cavity trees suitable for den establishment. Such selection would not only give a fisher more options when she needs to relocate dens, but should also lead to shorter relocation distances between subsequently used dens, thereby minimizing the amount of time kits are exposed to an increased risk of predation while being relocated.

We evaluated the selection of natal and maternal dens by female fishers within their home ranges in northern Minnesota by comparing cavity trees used as dens to other large-diameter cavity trees within each animal's home range that could have been used as dens. We hypothesized that (1) fishers would select cavity trees in areas with a high abundance of other cavity trees suitable for denning, and (2) the abundance of other suitable cavity trees around used dens would be negatively correlated with the relocation distance to subsequently used dens. We also evaluated whether tree type and health, slope, or stand type influenced den selection, and attempted to identify at what spatial scale, if any, fishers perceive nearby abundance of cavity trees as important for den selection.

3.2 Study Area

Data were collected across three study sites in the Laurentian Mixed Forest Province of Northeastern Minnesota (Figure 3.1). This area is largely under public ownership and includes Superior National Forest as well as state and county lands. Northeastern

Minnesota's climate is characterized by short summers and long, cold winters, with temperatures averaging 18.6°C in July and -14.7°C in January. Fishers in this region occupy habitats at elevations between 274 and 700m above sea level, with annual precipitation between 53 and 81cm. The northern portion of the area receives an average of 142.7cm of snowfall annually, with persistent snow cover between mid-November and early April (Kapfer 2012, Joyce 2013).

The first site ("Remer") encompasses parts of Chippewa National Forest and Land O' Lakes State Forest near Remer, MN (47.06°N, 93.91°W) and is composed primarily of deciduous forest (74%) with smaller proportions of open water (11%), wetlands (10%), and regenerating and mixed forest (5%) (Erb et al. 2009). The second site ("Hoyt") encompasses a southern portion of Superior National Forest near Hoyt Lakes, MN (47.52°N, 92.14°W) and is primarily mixed forest (69%) interspersed with wetlands (15%), upland coniferous forest (5%), gravel pits and open mines (4%), and a combination of regenerating forest, shrubby grassland, open water, and deciduous forest (7%) (Erb et al. 2009). The third site ("Ripley") encompasses the northern portion of the Camp Ripley Military and Civilian Training Facility (46.18°N, 94.37°W) and is composed primarily of deciduous forest (55%) with the remainder almost equally divided between wetlands, dry open grass, and military installations (Dirks and Dietz 2009).

3.3 Methods

3.3.1 Fisher Trapping and Telemetry

Fishers were live-trapped, radio-marked, and monitored from 2009 to 2016 as part of a larger project on *Martes* ecology in Minnesota (hereafter "*Martes* study"; Erb et al. 2009). We located possible fisher den structures using standard ground telemetry

techniques (White and Garrott 1990, Berg 2015) between early March and mid-April

(Powell 1982, Paragi et al. 1996, Zhao et al. 2012). When a suspected den was located,

we deployed remotely-activated cameras (Reconyx PC-85, RC-55, and HC600; Reconyx,

Inc, Holmen, WI) to monitor female activity and subsequent relocation to maternal dens.

We excluded dens from our analysis if we were not able to confirm kits via sound or

camera, or if no evidence of kits (e.g., obvious lactation, placental scarring, or kit bite

marks on collar; Erb et al. 2013) was present when the animal was subsequently handled

as a mortality or recapture.

3.3.2 Cavity Tree Sampling

We located unused large-diameter trees with at least one cavity large enough to contain a

fisher den (hereafter "cavity trees") within a 25-km^2 circular area surrounding each

confirmed natal den (hereafter "den range"), an area approximating the home range of an

individual fisher based on previous observations of fisher home range size and hunting

excursions (Leonard 1980, Aubry and Raley 2006, Erb et al. 2009). To ensure a non-

clumped, random sample of cavity trees available to each animal, we used hierarchical

random sampling to place 637 25-m fixed-radius plots throughout each den range (Figure

3.2). At the broadest scale, we established an 875-m fixed-radius plot around each den

and around six random locations throughout the 25-km^2 den range of each fisher (Figure

3.2a). The radii of these plots encompass the maximum relocation distance from natal to

maternal dens observed in female fishers used in this study (range = $20.52 - 769.01$m),

and are similar in size to areas used for broad-scale analyses of natal den selection in

fishers in the past (e.g., Aubry and Raley 2006). We then divided each plot into 37 125-m

fixed-radius tangent circles laid out in four concentric rings, and used multi-stage cluster

sampling to randomly select one, two, four, and six circles to sample from each ring (i.e., six circles from the outermost ring, four circles from the next ring, etc.; Figure 3.2b). We further divided selected circles into 19 25-m fixed-radius tangent plots laid out in three concentric rings, and randomly selected one, two, and four plots from each ring to sample (i.e., four plots from the outer ring, two from the middle ring, and one from the inner ring; Figure 3.2c).

At each sampled plot, we recorded the Universal Transverse Mercator (UTM) coordinates and characteristics of every cavity tree we located. Recorded characteristics included species, diameter at breast height (DBH), presence-absence of a broken top, and tree health (i.e., alive, alive but declining, or dead; Paragi et al. 1996, Weir and Corbould 2008, Davis 2009). We excluded trees less than 37cm DBH (the minimum DBH of a used cavity tree in our study) or where the size and suitability of the cavity could not be confirmed. We included trees that had cavities accessible from a broken top because one of the fishers in this study used such a cavity for a maternal den.

We estimated the nearby abundance of other cavity trees around each located tree by dividing the total number of trees by the proportion of area sampled within 350, 600, 850, and 1050m of each sampled tree. These distances were based on the 50, 75, 90, and 100% quantiles of fisher den relocation distances observed in *Martes* study (n = 30; Figure 3.3), and were identified as potential scales at which fishers perceive nearby abundance of cavity trees as important for den selection.

3.3.3 Denning Habitat Selection

We used a hierarchical framework to understand fisher selection of den sites, where the selection of a natal den determines the availability of trees that can be used for

subsequent maternal dens. In this framework, a fisher can select any cavity tree within its den range to establish a natal den, at which point only trees within 1050m (the maximum relocation distance between dens observed in our study) are available for establishing a secondary den. Similarly, only trees within 1050m of the secondary den are available to the fisher for establishing a tertiary den.

We evaluated fisher den site selection by comparing the suite of vegetative resources and characteristics of used cavity trees relative to other cavity trees available to each fisher using an information theoretic criterion approach (AIC; Burnham and Anderson 2002, Arnold 2010) in three steps. First, we developed conditional logistic regression models for each combination of measured covariates, (i.e., the four scales at which the nearby abundance of cavity trees was measured as well as characteristics previously shown to be important in the selection of natal and maternal dens – slope, stand type, and the health, type [coniferous or deciduous], and DBH of the tree itself; Paragi et al. 1996, Weir and Harestad 2003, Weir and Corbould 2008, Davis 2009, Higley and Matthews 2009, Thompson et al. 2010, Zhao et al. 2012, Niblett et al. 2015). Second, we used all-subset model averaging to identify which covariates best describe the selection of den sites by female fishers, using sum of weights (SWs) for determining variable importance (Giam and Olden 2016). We ranked models using Akaike's Information Criterion adjusted for small sample size (AICc; Burnham and Anderson 2002), and calculated slope coefficients (β_i) and associated standard errors using standard model averaging techniques (Burnham and Anderson 2002, Lukacs et al. 2010, Symonds and Moussalli 2011). Finally, we tested whether the nearby abundance of cavity trees resulted in shorter relocation distances between consecutive dens using linear regression.

50

All analyses were done in Program R (R Core Team 2015) using the *survival* (Therneau

and Lumley 2015) and *MuMIn* (Bartoń 2015) packages for fitting conditional logistic

regression models and model averaging, respectively.

3.4 Results

3.4.1 Used and Unused Tree Characteristics

We located 35 natal and 20 maternal dens used by 26 female fishers between 2009 and

2016 ($\bar{x}$ = 1.62 dens per female per year). Ten of the 26 females used in this study were

tracked to dens over two consecutive years, totaling 36 unique instances of fishers using

dens to raise kits during a single season (hereafter "denning occasions"). We were able to

locate maternal dens for 15 of these 36 denning occasions. An additional 23 dens (11

natal and 12 maternal) located during the *Martes* study were excluded from our analysis

due to extensive logging that occurred throughout the surrounding area prior to our

vegetation sampling (1 natal and 3 maternal dens) and a high abundance of private

property (9 natal and 9 maternal) and excessive remoteness (1 natal) that precluded

access. We also excluded one maternal den because we suspected that the relocation from

the natal den was due to extensive disturbance caused by military activity and not natural

causes. This maternal den's relocation distance was also a clear outlier, almost twice the

relocation distance of any other maternal den in our study (1831m vs. a maximum of

1036m).

Females mainly established natal and maternal dens in aspen (*Populus* spp.; 38 of

55), although maple (*Acer* spp.), oak (*Quercus* spp.), pine (*Pinus* spp.), and cedar (*Thuja*

spp.) trees were also used (Table 3.1). We did not detect a significant difference between

the types of trees used for natal and maternal dens (chi-squared test of independence, χ^2 =

4.90, df = 4, simulated p = 0.324), although oaks and cedars were used for natal but not for maternal dens. The types of trees used did, however, differ significantly between study sites (χ^2 = 19.54, df = 8, p = 0.015), with a smaller proportion of aspen used in Ripley (42.9%) than in Remer (75.9%) or Hoyt (68.4%). Females rarely denned in declining trees (4 of 55), and used live trees more often than dead trees for natal, maternal, and all dens combined (Table 3.1). Mean DBH of used trees was 51.9cm (SE = 1.5cm, range = 37.6 – 95.8cm) and we did not detect a significant difference in DBH between natal and maternal dens (52.5cm vs. 50.7cm; ANOVA, p = 0.550) or between study sites (54.4cm, 48.1cm, and 51.5cm for Remer, Hoyt, and Ripley, respectively; p = 0.140).

We located a total of 1602 unused large-diameter trees with suitable cavities across the den ranges of the 36 denning occasions in our three study sites ($\bar{x}$ = 98.9 trees per occasion, SE = 9.5). Suitable cavities were primarily found in aspen (57.7%), with smaller quantities found in oak (12.2%), maple (10.6%), and pine (8.1%), and very few (<4.4%) in cedar, ash (*Fraxinus* spp.), basswood (*Tilia* spp.), birch (*Betula* spp.), elm (*Ulmus* spp.), fir (*Abies* spp.), hemlock (*Tsuga* spp.), spruce (*Picea* spp.), and tamarack (*Larix* spp.; Table 3.1). An additional 20 dead cavity trees (1.2% of all trees) that we located were removed from the analysis because we were unable to determine genus due to extensive deterioration of the bark. The majority of unused cavity trees were dead (67.5%), with most of the remaining trees being alive (28.2%) and very few in the alive but declining stage (4.2%). The mean DBH of unused cavity trees was 48.1cm, with a maximum DBH of 108.9cm. The distribution of the number of suitable cavity trees found in each of the 13,281 25m-radius plots sampled followed a negative binomial distribution

(Figure 3.4), with a mean of the non-zero values of 1.36 trees per plot (SE = 0.02, range = 1 – 6) and 75.16% of plots containing no cavity trees.

Both the composition and relative abundance of available cavity trees varied significantly across our three study sites (chi-squared test of independence, χ^2 = 361.99, df = 24, p < 0.001), although all sites were consistently dominated by aspen (48.9, 75.1, and 49.4% for Remer, Hoyt, and Ripley, respectively). Health of available trees also differed among study sites (χ^2 = 109.92, df = 4, p < 0.001), with a higher proportion of live trees in Remer (39.5%) than in Hoyt (13.8%) or Ripley (23.0%). Additionally, available trees in Hoyt were smaller than those in Remer or Ripley (mean DBH of 45.4cm vs. 49.7 and 48.9cm, respectively; ANOVA, df = 2 and 1599, p < 0.001).

3.4.2 Denning Habitat Selection

Model averaging using conditional logistic regression identified tree health and the abundance of cavity trees within 600m as the two most important predictors of den-site selection by female fishers (Table 3.2). Model-averaged coefficients indicate that fishers used live (β = 1.283, SE = 0.021) and declining (β = 1.254, SE = 0.025) cavity trees disproportionately to their availability within their den ranges. As hypothesized, females also selected trees with a higher surrounding abundance of other suitable cavity trees (β = 0.0030, SE = 0.0001). This surrounding abundance, however, did not significantly affect relocation distance to subsequently used trees (p = 0.144).

3.5 Discussion

3.5.1 Drivers of Den Site Selection

Our observations of den use by female fishers in northern Minnesota are consistent with previous findings that fishers exclusively use cavities in large-diameter trees for whelping

and rearing kits (Coulter 1966, Leonard 1980, Paragi 1990, Paragi et al. 1996, Raley et al. 2012), and that deciduous trees such as aspen are used more often than coniferous trees (Paragi 1990, Weir and Harestad 2003, Weir and Corbould 2008, Higley and Matthews 2009, Niblett et al. 2015). When compared to unused cavity trees, however, this high use of deciduous trees did not translate into selection of deciduous trees (i.e., use was proportional to availability). Instead, fishers appear to select den cavities primarily on the basis of tree health, selecting for live and declining cavity trees while avoiding dead trees. These results emphasize the need to treat with caution any conclusions drawn about habitat selection or preference without an accurate comparison to what is available.

Although selection for declining trees is well supported by the literature (e.g., Weir and Harestad 2003, Weir and Corbould 2008, Davis 2009), previous studies have resulted in conflicting accounts about whether fishers prefer live (e.g., Higley and Matthews 2009, Thompson et al. 2010) or dead (e.g., Paragi et al. 1996, Weir and Corbould 2008) trees. One potential reason for this inconsistency is that apart from Weir and Corbould (2008), most conclusions have been based on the percentage of used dens that were in live or dead trees without a comparison to what was available. Our analysis, on the other hand, evaluates whether fishers select for living or dead cavity trees by comparing used trees to unused cavity trees that could have supported a fisher den. Our conclusions concerning the role of tree type and health in fisher den selection support the conclusions drawn by Raley et al. (2012) in their synthesis of the habitat associations of fishers in western North America.

Although our results suggest that fishers have no preference for particular types of trees, certain species of trees are more susceptible to cavity formation throughout their

life. Aspen, for example, are more likely to develop cavities earlier in life because of heart rot infection, and subsequently experience a higher rate of vertical decay than other deciduous trees (Silverborg 1959, Boyce 1961, Paragi et al. 1996, Weir et al. 2012). This results in a high abundance of aspen with cavities suitable for fisher dens, and helps to explain why the majority of used and unused cavities in this study and used cavities in other studies (e.g., Leonard 1980, Arthur 1987, Paragi et al. 1996) have been located in aspen. As such, although fishers may not prefer aspen over other types of cavity trees, the propensity for aspen to develop suitable cavities still makes them an important target for conservation.

In addition to preserving large-diameter cavity trees because of their exclusive use for natal and maternal dens, our results suggest that simply preserving a few suitable trees throughout each fisher's home range may not be sufficient to ensure adequate denning habitat. Fishers in northern Minnesota selected landscapes with a higher abundance of suitable trees, even though other areas of their range often contained enough cavity trees to ensure a sufficient number of options for future relocations (i.e., at least five). This higher abundance, however, did not result in shorter relocations to subsequently used dens as we had originally hypothesized. This suggests that although fishers select den sites based partially on the surrounding abundance of nearby cavities, the need to minimize the amount of time kits are exposed to an increased risk of predation while being relocated is not the major driving force behind this selection.

An alternative explanation for why female fishers select for these high abundance areas is that it allows them a larger number of options from which to choose the most optimum cavity trees. For example, having a larger number of options could allow fishers

to establish subsequently used maternal dens on particular types of slopes, which has been identified as an important component of den site selection in previous studies (e.g., Thompson et al. 2010, Zhao et al. 2012). Although slope only displayed moderate support in our analysis (Table 3.2), the abundance of nearby cavity trees was highly correlated with the slope of subsequently used trees (p = 0.017). As such, we suggest that maintaining higher densities of cavity trees may allow fishers to be more selective in the types of trees they use for establishing subsequent maternal dens, which can help improve offspring survival and eventual recruitment into the population.

3.5.2 Causes of Den Relocation

Our results also provide some novel insights into the potential causes of den relocations by female fishers. Instead of relocating to nearby cavity trees, fishers often established subsequent dens in trees that were further away than expected, based on the suite of available trees. Other predators are known to relocate their dens to gain access to areas with increased prey availability (e.g., cheetahs (*Acinonyx jubatus*; Laurenson 1993) and Canada lynx (Olson et al. 2011)). This may also be the case for fishers because male fishers provide no parental care, so female fishers must hunt to meet the energetic and nutritional demands of lactation (Powell 1982, Powell and Leonard 1983, Roy 1991). Further, female fishers spend very little time outside of the den while rearing kits, especially during the first few weeks of the kits' life (Leonard 1980, Powell 1982), implying that most hunting occurs in close proximity to the den itself. This behavior can lead to a local depletion of prey, forcing the female to either spend increasingly more time away from her kits to reach more distant areas with higher prey abundance, or to relocate her kits to these areas instead. Relocating the kits would allow female fishers to

spend more time with the kits, which may increase juvenile survival (Powell 1982). In such situations, relocating kits to nearby dens would not alleviate the problem of locally depleted prey availability, forcing fishers to use cavity trees that are further away. This avoidance of nearby cavity trees in favor of more distant ones is consistent with our observations, and we recommend that future research attempt to link the timing of relocations with the local availability of common fisher prey species such as snowshoe hare (*Lepus americanus*), red squirrels (*Tamiasciurus hudsonicus*), and southern red-backed voles (*Myodes gapperi*; Powell 1982, Raine 1987, Weir et al. 2005).

Our study also provides some support for the notion that fishers may relocate dens in response to excessive anthropogenic disturbance, as has been observed in brown (*Ursus arctos*) and black (*Ursus americanus*) bears (Linnell et al. 2000), as well as in eastern wolves (*Canis lycaon*; Argue et al. 2008). One of the relocations between a natal and maternal den observed during our study coincided with extensive disturbance of the area by military activity. The distance of this relocation was almost twice that of any relocation in our study, and was subsequently removed from our analysis. This fisher later relocated her kits to a different maternal den, and this second relocation was well within the range of relocation distances observed in our study, suggesting that the first relocation was caused by outside factors. Although this was an isolated incidence, it does support the suggestion that disturbance of den sites should be minimized, particularly while the mother is present (Paragi et al. 1996, Aubry and Raley 2006, Weir and Corbould 2008, Davis 2009, Zhao et al. 2012).

3.5.3 Caveats

Although our definition of availability excluded trees that could not be used by female fishers (e.g., those with no cavities), it is important to note that some of the included trees were potentially also unusable. Presence of cavities, for example, was determined mainly by visually locating cavity entrances. Due to logistical constraints, we were unable to measure the size or depth of the cavity itself. As such, it is possible that some of the trees we classified as available had cavities that were too small to house fisher dens. Similarly, we cannot be certain that we were able to locate all cavity trees within a given plot. Fishers have used cavities with entrances as high as 30m off the ground (Weir and Harestad 2003), which may be difficult to spot. We do, however, believe that our list of cavity trees provided a representative, if not exhaustive, sample of the types of trees fishers could have used instead.

Another caveat of our findings is that our definition of availability was based largely on observations of used dens in our own study, and may not directly apply to other parts of the fisher's range. For example, we considered trees less than 37-cm DBH as unavailable because that was the minimum DBH of a used cavity tree in our study. Although this lower limit is comparable to that observed by Higley and Matthews (2009) (35 cm), several studies have reported lower limits (25-27.5 cm; Paragi et al. 1996, Davis 2009), and at least one study documented a higher limit (62 cm; Aubry and Raley 2006). Similarly, we included cavities that were accessible from a broken tree top because one of the fishers in this study used this type of a cavity for a maternal den. Whether similar cavities have been used by fishers throughout other parts of their range is unclear, and we cannot rule out that this was an isolated incident unrepresentative of overall fisher denning preference.

We also note that the covariates considered here are by no means an exhaustive list of factors that could influence the suitability of a cavity tree as a potential fisher den site. Other characteristics such as canopy cover (Aubry and Raley 2006, Zhao et al. 2012), proximity to riparian zones (Weir and Harestad 2003, Davis 2009), orientation of the cavity (Paragi et al. 1996), size and height of cavity entrances (Leonard 1980, Raley et al. 2012), and dimensions of the cavity itself (Weir and Corbould 2008, Raley et al. 2012) may also influence den site selection by female fishers. Smaller cavity openings, for example, may be important for excluding potential predators and deterring infanticide by larger male fishers (Leonard 1980, Aubry and Raley 2006, Raley et al. 2012), suggesting that the size of the cavity opening may influence whether or not a cavity is selected. Orientation and canopy cover, on the other hand, can influence the thermal properties of den sites (e.g., the amount of sunlight a den is exposed to), which may also be important (Paragi et al. 1996, Raley et al. 2012). Although quantitative evidence of the importance of these other factors is lacking (Raley et al. 2012), future studies of habitat selection should attempt to include these and other potential covariates in order to provide a more comprehensive assessment of what drives den site selection by female fishers.

3.6 Management Implications

To ensure adequate fisher denning habitat, forest management needs to provide for retention of appropriate structural elements from multiple stages of forest development. Our results affirm the need to retain large-diameter cavity trees, but also suggest that the species of retained trees is not as important as retaining live and declining trees. However, because aspen are more likely to develop suitable cavities, we recommend that

they be the first target for retention where they exist. Further, younger aspen that are showing signs of early decay or heart rot infection should also be retained because they are likely to grow into suitable cavity trees later in life and ensure that falling cavity trees will be replaced.

In addition to simply maintaining an adequate number of suitable cavity trees throughout the fisher range, our results suggest that a higher local abundance of these trees is an important driver of den site selection. Higher local abundance of cavity trees may allow fishers to be more selective in the types of trees and cavities they later relocate kits to, thereby improving juvenile survival and reproductive success. We recommend forest managers retain large-diameter cavity trees in high-density patches throughout the environment and avoid creating a landscape with a large number of highly dispersed denning options. These recommendations should be implemented alongside an adaptive management approach to provide a better assessment of the denning habitat requirements of fishers throughout their natural range.

Comparing the Utility of Varying Amounts of Radio-Telemetry Data for Improving Statistical Population Reconstruction of American Marten in Northern Minnesota

4.1 Introduction

Accurately estimating the population dynamics of furbearer species such as bobcats (*Lynx rufus*), fishers (*Martes pennanti*), and American marten (*Martes americana*) is challenging because their elusive behavior, solitary lifestyle, and relatively low densities make direct monitoring costly and impractical (Becker et al. 1998, Heilbrun et al. 2006, Williams et al. 2009, Skalski et al. 2011). Given these difficulties, most abundance estimates have relied on track surveys (e.g., Zielinski et al. 2001), noninvasive hair sampling using snares (e.g., Williams et al. 2009), and camera traps (e.g., Heilbrun et al. 2006). Unfortunately, these methods do not provide a cost-effective means of estimating annual abundance over larger spatial scales (e.g., across an entire state, at which most harvest-related management occurs). Age-at-harvest data commonly collected at hunter check stations often provides the only information on catch per unit effort and overall age-structure of a population across broad spatial extents (Gove et al. 2002, Broms et al. 2010, Skalski et al. 2012*b*). Early on, these data were used simply to estimate minimum population size based on cohort-specific sums of harvested animals (Fry 1949, Skalski et al. 2007). In rare cases, age-at-harvest data were also used to estimate state-level abundance under restrictive assumptions of stable and stationary populations (Millspaugh et al. 2009, Broms et al. 2010).

More recently, statistical population reconstruction using integrated population models (IPMs) has emerged as a robust and more versatile alternative for estimating

population abundance using age-at-harvest and annual catch-effort data (Skalski et al. 2007, Fieberg et al. 2010, Schaub and Abadi 2011, Clawson et al. 2013). These methods simultaneously estimate several demographic parameters (e.g., annual abundance and recruitment, natural survival, and harvest mortality), offer confidence intervals on estimated values, and provide a flexible framework for incorporating auxiliary information from index surveys, mark-recapture, or radio-telemetry studies (Gove et al. 2002, Skalski et al. 2007, 2011, Broms et al. 2010, Fieberg et al. 2010, Clawson et al. 2013). Such models have been used to estimate population abundance and trends of harvested furbearers such as American marten (Skalski et al. 2011) and black bears (*Ursus americanus*; Fieberg et al. 2010), as well as black-tailed deer (*Odocoileus hemionus*; Skalski et al. 2007), elk (*Cervus elaphus*; Gove et al. 2002), and greater sage-grouse (*Centrocercus urophasianus*; Broms et al. 2010).

Several types of auxiliary data have been used to improve population reconstruction models in the past, including a yearly browse damage index (Skalski et al. 2007), intermittent statewide mark-recapture estimates of abundance (Fieberg et al. 2010), and, most commonly, information on natural or harvest mortalities from radio-telemetry studies (Gove et al. 2002, Broms et al. 2010, Clawson et al. 2013). Radio-telemetry data are commonly collected by management agencies throughout the United States, and have been consistently shown to significantly improve reconstructed estimates of population abundance (e.g., Gove et al. 2002, Broms et al. 2010, Clawson et al. 2013) Although there are clear benefits to incorporating telemetry data into statistical population reconstruction, these data are costly and time-consuming to collect. Management agencies that consider starting a radio-telemetry study alongside existing

efforts to collect age-at-harvest data must make decisions regarding the number of collars to deploy each year and how often and for how long to monitor collared animals. To this end, a more quantitative understanding of how these factors influence the accuracy of population reconstructions is needed.

We use simulated populations with varying demographic characteristics (e.g., population size, survival, and harvest mortality) to compare statistical population reconstruction results derived using different amounts of radio-telemetry data. We evaluated how the number of years and average number of new animals collared each year influence model accuracy and precision. For each case, we incorporated information on natural, harvest, and combined mortality, and compared these three approaches to a standard reconstruction that used no auxiliary data (i.e., only age-at-harvest and catch-effort data), a method that has been shown to give unbiased but sometimes very imprecise results (Skalski et al. 2012*b*). We illustrate these different techniques and corresponding improvements using age-at-harvest, trapper effort, and radio-telemetry data from a harvested population of American marten in northeastern Minnesota.

4.2 Study Area

To demonstrate the effects of the amount of available radio-telemetry data on population reconstruction, we used age-at-harvest data collected from a population of American marten in the Laurentian Mixed Forest Province of Northeastern Minnesota, USA. This area is largely under public ownership and includes Superior National Forest as well as state and county lands. Northeastern Minnesota's climate is characterized by short summers and long, cold winters, with temperatures averaging 18.6°C in July and -14.7°C in January. Marten in this region occupy habitats at elevations between 450 and 600m

above sea level, with annual precipitation between 53 and 81cm. The area receives an average of 142.7cm of snowfall annually, with persistent snow cover between mid-November and early April (Kapfer 2012, Joyce 2013). The terrain is composed primarily of mixed forest (69%) interspersed with wetlands (15%), upland coniferous forest (5%), gravel pits and open mines (4%), and a combination of regenerating forest, shrubby grassland, open water, and deciduous forest (7%) (Erb et al. 2008).

4.3 Methods

4.3.1 Available Demographic Data

From 2007 to 2014, all marten harvested in Minnesota were recorded through mandatory furbearer registration (estimated compliance rate of 90%) by the Minnesota Department of Natural Resources (MN DNR) and aged using a combination of microscopic counts of cementum annuli and x-ray examination of pulp cavities (Erb 2015). Because not all reported marten were successfully aged due to information loss and non-extraction (mean proportion aged annually = 70%; Erb 2015), age-at-harvest numbers were inflated accordingly by assuming that the aged sample is representative of the overall harvest (Fieberg et al. 2010). The MN DNR also used annual postcard mail-in surveys (mean response rate = 75%, range = 58-84%; Dexter 2014) to estimate yearly trapper effort (i.e., trap nights).

In addition to these data, we utilized marten survival data collected via radio-telemetry from 2008 to 2014 (Erb et al. 2014). Animals were captured using Tomahawk cage traps (Models 106 and 108; Tomahawk Live Trap, Hazelhurst, WI, USA) during winter after the conclusion of the trapping season. Captured animals were weighed and sexed, tagged on both ears, and fitted with Holohil Systems Ltd MI-2 collars (~31g;

Carp, Ontario, CA). The first pre-molar was also removed and used for aging. Collared

animals were monitored from fixed-wing aircraft at approximately weekly intervals until

harvest, natural mortality, or collar failure (see Erb et al. 2014 for more detail). Animals

with collar failures were excluded from the data in the year of collar failure (i.e., right-

censored; Gove et al. 2002), as were animals who died shortly after capture. A total of

189 animals from the radio-telemetry study were used in our analysis.

4.3.2 Likelihood Model

4.3.2.1 Age-at-Harvest Likelihood

We used a multinomial likelihood to describe the expected harvest numbers for each

combination of age-class and year, an approach similar to that used by Skalski et al.

(2012b). Consider a hypothetical population of animals divided into three age-classes

(juveniles, yearlings, and adults) harvested over Y consecutive years, where N_{ij} and h_{ij}

are, respectively, the abundance immediately before harvest and the number subsequently

harvested, of animals of age-class j (j = 1, 2, 3) in year i (i = 1,…, Y) (Figure 4.1). Under

this framework, all individuals born during the same year constitute a single cohort that is

subsequently subjected to annual mortality from harvest and natural causes. Using an

age-at-harvest matrix (Table 4.1), we can represent each cohort as a separate diagonal,

where the observed counts, h_{ij}, are a function of the initial abundance of the

corresponding cohort and annual rates of harvest mortality and natural survival (Gove et

al. 2002, Skalski et al. 2007, 2012b, Clawson et al. 2013). We model each of these

diagonals as independent multinomial distributions, and the joint likelihood for the entire

matrix is

$$L_{Age-at-harvest} = \prod_{i=1}^{Y} l_{i1} \times \prod_{j=2}^{3} l_{1j},$$

(1)

where l_{ij} is the cohort-specific age-at-harvest likelihood (see Skalski et al. 2012b for more details).

This multinomial distribution requires the simplifying assumption that the fates of every animal are independent and identically distributed. Additional model assumptions include a geographically closed population (i.e., no immigration or emigration), an unbiased harvest reporting rate, and a precise measurement of trapper effort (Gove et al. 2002, Gast 2012).

4.3.2.2 Catch-Effort Likelihood

The current model is over-parameterized and incapable of estimating parameters for initial abundance, recruitment into the trappable population, and separate values for both survival and harvest vulnerability; at least one of these parameters must be estimated independently of the age-at-harvest likelihood with the use of some additional source of field information (Gove et al. 2002, Skalski et al. 2007, 2012a, Clawson et al. 2013). One of the most common sources of this additional information is catch per unit effort (CPUE). Skalski et al. (2007) used CPUE data to model the total annual harvest as a binomially-distributed function of the yearly trapper effort:

$$L_{Catch-effort} = \prod_{i=1}^{Y} \left(\frac{\sum_{j=1}^{A} N_{ij}}{\sum_{j=1}^{A} h_{ij}} \right) (1 - e^{-cf_i})^{\sum_{j=1}^{A} h_{ij}} (e^{-cf_i})^{\sum_{j=1}^{A}(N_{ij} - h_{ij})},$$

(2)

where f_i is the trapper effort in year i, and c represents the trapping vulnerability coefficient that converts trapper effort to harvest probability (Skalski et al. 2007, 2011,

Broms et al. 2010, Clawson et al. 2013, Gast et al. 2013*a*). Under this framework, the full

standard reconstruction model may be written as

$$L_0 = L_{Age-at-harvest} \times L_{Catch-effort}.$$ (3)

Unfortunately, even this model may be insufficient to accurately estimate all

demographic parameters unless trapper effort is highly variable (Laake 1992, Clawson et

al. 2013). For example, Skalski et al. (2011) used a nearly five-fold increase in trap-

nights over eight years to recreate population trends of American marten in the Upper

Peninsula of Michigan. A similar approach was used to estimate abundance of female

black-tailed deer in Pierce County, Washington between 1979 and 2002, where the

number of hunter-days varied from a low of 92 to a high of 629, a nearly seven-fold

increase (Skalski et al. 2007). By comparison, our population experienced only a three-

fold difference in estimated hunter-days (Table 4.1). For this and other populations

experiencing less dramatic changes in trapper effort over time (e.g., Broms et al. 2010),

additional auxiliary data may be required (Clawson et al. 2013).

4.3.2.3 Radio-Telemetry Likelihoods

Radio-collared individuals with known fates can be used to help estimate harvest

probability and natural survival. The simplest way to incorporate radio-telemetry data is

to model the total number of natural mortalities per year as a binomial process of the

form

$$L_{Radio-telemetry1} = \prod_{i=Y-y+1}^{Y} \binom{n_i}{v_i} (1-S)^{v_i} (S)^{n_i-v_i},$$ (4)

where S is the natural survival probability (i.e., the complement of the probability of

mortality from non-harvest, natural causes; Gove et al. 2002), n_i is the number of

collared animals alive at the beginning of year i, v_i is the number of collared animals that die from natural causes in year i, and y is the total number of years spanned by the radio-telemetry study (which may or may not equal Y). Broms et al. (2010) focused instead on the total annual radio-collared harvest and proposed a different formulation:

$$L_{Radio-telemetry2} = \prod_{i=Y-y+1}^{Y} \binom{n_i - v_i}{u_i} (1 - e^{-cf_i})^{u_i} (e^{-cf_i})^{n_i - v_i - u_i},$$

(5)

where $n_i - v_i$ is the number of collared animals alive at the beginning of the harvest season in year i. Gove et al. (2002) used a combination of the two approaches to help estimate both natural survival and harvest probability, modeling the two as joint binomial process:

$$L_{Radio-telemetry3}$$

$$= \prod_{i=Y-y+1}^{Y} \binom{n_i}{v_i, u_i} (1$$

$$- S)^{v_i} [S(1 - e^{-cf_i})]^{u_i} [Se^{-cf_i}]^{n_i - v_i - u_i}.$$

(6)

In our simulations, we assumed the radio-telemetry study covered the last y years of the available harvest data, representing a situation in which a wildlife manager has historical age-at-harvest data but has only recently started conducting a radio-telemetry study. Note that to maintain consistent notation among likelihoods, the variables and names used in equations (5) and (6) differ from those originally used by Gove et al. (2002) and Broms et al. (2010) (e.g., the harvest probability, p_i, has been replaced with a function of the yearly trapper effort, $1 - e^{-cf_i}$). Additionally, these two likelihoods were originally based on radio-telemetry studies where animals were collared immediately

prior to harvest, and as such have been modified to be used for the American marten data where animals were collared immediately after harvest.

Using these radio-telemetry likelihoods, we can now construct three different versions of the full reconstruction model (i.e., models that incorporate radio-telemetry data on natural survival, harvest mortality, or both). The three likelihoods can be written as

$$L_1 = L_{Age-at-harvest} \times L_{Catch-effort} \times L_{Radio-telemetry1}, \tag{7}$$

$$L_2 = L_{Age-at-harvest} \times L_{Catch-effort} \times L_{Radio-telemetry2}, \tag{8}$$

and

$$L_3 = L_{Age-at-harvest} \times L_{Catch-effort} \times L_{Radio-telemetry3}. \tag{9}$$

Because the radio-telemetry data used in equation (9) includes additional information on twice as many parameters as those data used in equations (7) and (8) (i.e., information on natural survival and harvest probability as opposed to only one or the other), we expect L_3 to perform significantly better than either L_1 or L_2.

4.3.3 Simulation Study

We used a Monte Carlo simulation to determine the accuracy and precision of population reconstruction based on the four different formulations of the likelihood model and varying amounts of radio-telemetry data. We developed a demographically-stochastic version of a Leslie matrix model (Leslie 1945, 1948, Berg 2012, Skalski et al. 2012*b*, Clawson et al. 2013) in Program R (R Core Team 2015) to generate age-at-harvest data for a population with a maximum age of 15 (as observed in the marten age-at-harvest data; J. Erb, unpublished data), exhibiting different levels of natural survival and average harvest rate (see Appendix S1 at the Data Repository for U of M (DRUM) for full code).

Recruitment was generated using a Poisson process and adjusted to produce populations with stationary abundance in expectation that fluctuated as a result of stochasticity (Skalski et al. 2012*b*, Clawson et al. 2013). Natural survival and harvest were modeled as binomial processes, and trapper effort was generated by re-sampling with replacement the relative trapper effort observed in Minnesota over the last eight years (Table 4.1). The resulting age-at-harvest data were pooled into three age classes (juveniles, yearlings, and adults) prior to analysis.

For each simulation, we generated 50 years of data to establish demographic trends, with the last eight years used for the population reconstruction. We also simulated a radio-telemetry study during the last seven years to mimic the data available for the marten reconstruction. The number of newly-collared animals each year was generated by re-sampling the observed number of animals collared each year by the MN DNR from 2008 to 2014 (mean = 27, range = 12 − 38), with an annual 33% chance of collar failure (i.e. loss of signal due to collar damage or dead battery; J. Erb, unpublished data).

To minimize the number of demographic scenarios investigated, we assumed survival and trapping vulnerability did not vary by age-class (Skalski et al. 2012*b*, Clawson et al. 2013). Scenarios were designed to represent a wide variety of harvested populations, simulating natural survival rates of 0.75 and 0.90, harvest rates of 0.10 and 0.25, and low (i.e., between 1,000 and 4,000 individuals) and high (i.e., 10,000 to 15,000) population sizes. To evaluate how the amount of radio-telemetry data affected population reconstruction, each simulation was analyzed using the full seven years of radio-telemetry data, removal of the first one, three, and five years of data, and 33 and 67%

reductions in the number of newly-collared animals each year. A total of 1,000 simulations were analyzed per demographic scenario.

4.3.4 Case Study: Population Reconstruction of American Marten in Minnesota

For each of the four likelihood formulations, we examined nine alternative models for the actual (i.e., not simulated) marten population reconstruction, testing whether trapping vulnerability or natural survival were age-class-specific, age-constant, or different between juveniles and older individuals. We also evaluated how our reconstruction estimates were affected by the removal of the first one, three, and five years of radio-telemetry data (Table 4.2), using Akaike's Information Criterion (AIC; Burnham and Anderson 2002, Skalski et al. 2011) for model selection.

4.3.5 Parameter Estimation

We used a bounded form of the Nelder-Mead algorithm in the "dfoptim" package (Varadhan and Borchers 2011) of Program R to numerically solve for the maximum likelihood estimates, which allowed us to directly estimate natural survival, harvest vulnerability, initial age-class abundances in year 1 (i.e., N_{11}, N_{12}, N_{13}), and recruitment in subsequent years (i.e., $N_{21}, ..., N_{Y1}$). All other year- and age-class specific abundances were estimated based on the invariance property of the maximum likelihood estimation (Skalski et al. 2007, 2012b), where

$$N_{ij} = N_{i-1,j-1}e^{-c_{j-1}f_{i-1}}S. \tag{10}$$

We calculated standard errors from a numerical estimate of the inverse Hessian (Skalski et al. 2012b, Gast et al. 2013a, b) using the "numDeriv" package (Gilbert and Varadhan 2012).

Using our simulated data, we compared the accuracy of the four different likelihood models by calculating the mean relative bias of the cohort-specific abundance estimates for each scenario, defined as

$$B = \frac{1}{1000}\sum_{k=1}^{1000}\frac{1}{Y \times A}\sum_{i=1}^{Y}\sum_{j=1}^{A}\frac{\widehat{N}_{ij} - N_{ij}}{N_{ij}},$$

(11)

where N_{ij} is the true abundance of age-class j in year i and $\widehat{N}_{ij}$ is the associated model-derived estimate in the kth simulation (k=1,…, 1000) (Skalski et al. 2012b). Precision was examined using scenario-specific coefficients of variation (CVs), where 95% of the time the reconstruction estimates of yearly population size fell within two CVs of the true values (Skalski et al. 2012b, Clawson et al. 2013). We used linear models to evaluate the effects of adding more animals or years to the radio-telemetry study, and to quantify the average improvement in precision gained by using the three alternative likelihood models over the standard approach.

4.4 Results

4.4.1 Simulation Study

We simulated twelve different demographic scenarios 1,000 times each, reconstructing population abundance over an eight-year period using the four different formulations of the likelihood model and varying amounts of radio-telemetry data. The relative bias for the standard reconstruction (i.e., no auxiliary radio-telemetry data; L_0) ranged from -0.114 to +0.204, with a CV between 0.156 and 0.517 (Table 4.3).

Using all seven years of radio-telemetry data on natural survival (L_1), the relative bias and CV for the reconstruction ranged from -0.038 to +0.005 and 0.048 to 0.473, respectively. The relative bias for reconstruction using seven years of telemetry data on

harvest probability (L_2) ranged from -0.011 to +0.003, with a CV between 0.074 and 0.189. The relative bias and CV for the reconstruction using auxiliary data on both survival and harvest rates (L_3) ranged from -0.011 to +0.004 and 0.043 to 0.160, respectively.

Including additional collared animals and years of study from radio-telemetry data in population reconstruction consistently improved the CV of the model estimates (Figure 4.2). On average, CV decreased by 0.62% (SE = 0.20) for a one-unit increase in the average number of animals collared per year and by 3.11% (SE = 0.98) for every additional year of study (Figure 4.2). All else held constant, incorporating radio-telemetry data on natural survival resulted in a 2.19% (SE = 13.43) decrease in CV, compared to an 8.47% (SE = 13.43) decrease from incorporating radio-telemetry data on harvest. Using radio-telemetry data on both natural survival and harvest mortality resulted in a 17.01% (SE = 13.43) decrease in CV.

4.4.2 Case Study: Population Reconstruction of American Marten in Minnesota

Incorporating the full extent of the available radio-telemetry data (i.e., information on both natural survival and harvest rates over seven years of study), the best available population reconstruction (i.e., the model with the smallest AIC) modeled trapping vulnerability and natural survival as age-constant (next best model $\Delta AIC > 6$; Table 4.4). Using this model, we estimated a trapping vulnerability of 0.23 ($\widehat{SE} = 0.023$) and a yearly survival rate of 0.70 ($\widehat{SE} = 0.018$). Using these estimates of vulnerability alongside annual changes in trapper effort, we estimated that harvest rates decreased from 0.249 in 2007 to 0.110 in 2014. Pre-harvest marten abundance estimates showed a slight overall downward trend from an estimated 10,188 (95% CI = 9,062 − 11,314) animals in 2007 to

9610 (95% CI = 8,325 − 11,003) in 2014, with a very pronounced four-year cycle in abundance (Figure 4.3). We estimated the average annual finite rate of population change to be $\hat{\lambda}$ = 0.993 (95% CI = 0.971 − 1.015) and annual recruitment between 2,353 ($\widehat{SE}$ = 203) and 6,283 ($\widehat{SE}$ = 570) animals, with an overall trend similar to that of abundance (Figure 4.3). All of the alternative models showed similar overall trends but different estimates of absolute abundance.

Abundance estimates from alternative formulations of the likelihood model deviated from the estimates derived from the optimum approach (i.e., best-fit reconstruction using telemetry data on both survival and harvest rates) in a manner consistent with our simulation results (i.e., larger deviations for reconstructions using no radio-telemetry data and smaller ones for reconstructions using auxiliary data on either survival or harvest rates; Figure 4.3). The best-fit model determined from the full data likelihood (equation (9)) had a single survival parameter (0.70 for all age classes), whereas the optimal model from the no-telemetry-data likelihood (equation (3)) included different survival rates for juveniles and older individuals (0.56 and 0.63, respectively). In addition, the estimated trapping vulnerability was substantially lower for the no-telemetry-data model (0.086 vs. 0.23). The two models resulted in similar annual growth rates over the eight-year period, but the no-telemetry-data model had a 248% higher estimate of annual abundance (Figure 4.3). Excluding the first one, three, and five years of radio-telemetry data from the reconstruction resulted in increasingly divergent estimates, also consistent with our simulated results (Figure 4.3).

4.5 Discussion

Our simulation study reaffirms that statistical population reconstructions based solely on age-at-harvest and catch-effort data provide mostly unbiased but sometimes extremely imprecise results. Incorporating auxiliary data from simulated radio-telemetry studies significantly improved model precision while still providing an overall unbiased estimate of cohort abundance (Figure 4.2). As expected, analyses that used ratio-telemetry data to simultaneously help estimate both natural survival and harvest mortality yielded the biggest improvements, followed by those that only helped estimate one or the other. These results were consistent across a wide range of harvest intensity, natural mortality, and population abundance. This consistency can be explained by the simple fact that a study that collects data on both harvest and natural mortality collects data that help estimate twice as many parameters as a study that collects data on only one or the other. As such, we encourage managers to conduct radio-telemetry studies that assess both natural and harvest mortality whenever possible and to treat any results derived from reconstructions based solely on age-at-harvest and catch-effort data with extreme caution.

As the amount of radio-telemetry data available for reconstruction decreased, so did the precision of the reconstruction models. This trend followed a fairly linear pattern over the range of analyzed demographic parameters and was consistent across all simulated scenarios (Figure 4.2). On average, every additional year of study improved precision by as much as the addition of five newly-collared animals per year would have. The consistency of this trade-off for both large and small populations experiencing varying levels of survival and harvest can provide management agencies with a useful way to determine the number of animals and years needed from a proposed radio-telemetry study to achieve some desired level of improvement in population

reconstruction. Additionally, our results demonstrate that improvements in precision are clearly visible even after only two years of tracking eleven animals, meaning that managers do not need to wait several years into a study before seeing significant improvements in their reconstruction estimates.

Use of statistical population reconstruction with the full set of radio-telemetry data (i.e., information on both harvest mortality and natural survival collected over seven years) suggests that the northern Minnesota marten population (measured pre-harvest) experienced very pronounced four-year oscillations around a stable overall abundance of approximately 9,500 animals (Figure 4.3a). This abundance and associated estimates of trapping vulnerability and survival are very similar to the demographic parameters used in our fifth set of simulated reconstructions (HHL; Table 4.3), leading us to conclude with 95% certainty that the actual marten abundance in Minnesota is within approximately 9% of our estimated values.

Exclusion of radio-telemetry data from this reconstruction model resulted in a substantially lower estimate of trapping vulnerability and natural survival, and a substantially higher estimate of overall population abundance (Figure 4.3c). Further, exclusion of one, three, five, and all seven years of radio-telemetry data produced estimates progressively more deviant from those obtained from the best-fit, full data model (Figure 4.3d). These results corroborate conclusions drawn from our simulation study and further demonstrate not only the need to perform auxiliary studies to help estimate harvest and natural mortality, but that even a couple of years of data can significantly improve model accuracy. Interestingly, our case study results also suggest that estimated trends in relative population abundance are somewhat robust to whether or

76

not auxiliary data are included, and that the main benefit of these data is to provide more accurate estimates of absolute abundance, trapping vulnerability, and natural survival.

Although the estimated abundance compares favorably with the results of independent population modeling conducted by the MN DNR (Erb 2015), our reconstructed estimates do not corroborate the overall population decline observed in that model (Figure 4.3a). An alternative method of estimating overall population trends, which involves comparing the changes in trapper effort to changes in annual harvest numbers, supports our conclusion that the marten abundance in Minnesota has been generally stable during the past eight years. Using this method, higher increases in effort accompanied by smaller increases in harvest, for example, would indicate a declining population (Skalski et al. 2011). During the eight years of population reconstruction, marten harvest decreased by slightly more than 60%, a trend that was closely followed by the approximately 60% decrease in trapper effort. This suggests that the marten population was not in decline from 2007 to 2014, but experiencing either no or positive population growth.

The statistical population reconstruction also estimated more extreme oscillations between years of high and low abundance than that predicted by the MN DNR model (approximately 3,500 vs. 1,500 animals; Figure 4.3a). These oscillations match closely with those observed in the annual recruitment into the trappable population (Figure 4.3b), neither of which appear to be strongly influenced by the observed trends in trapper effort. These results are inconsistent with marten reconstruction conducted in Michigan, USA (Skalski et al. 2011), leading us to conclude that harvest rates do not play as large a role in governing the growth of the marten population in Minnesota as they do in Michigan.

Whether the observed trends in annual recruitment are caused by fluctuations in juvenile survival or reproductive success is difficult to discern using the reconstruction results. These trends do, however, appear to coincide with observed fluctuations in small mammal abundance between 2007 and 2014 (MN DNR, unpublished data), which can have a strong influence on juvenile survival or reproductive success. Several previous studies, for example, have suggested that observed variations in marten pregnancy rates and fecundity were caused by changes in small mammal abundance (Weckwerth and Hawley 1962, Thompson and Colgan 1987, Flynn and Schumacher 2009). Future reconstruction efforts should aim to incorporate auxiliary data on annual pregnancy rates, litter size (e.g., placental scarring data from harvested animals), or juvenile survival in order to attempt to separate the effects of juvenile survival and reproductive success on annual recruitment, thereby allowing us to better identify the driving forces behind observed population trends.

Although real populations can experience significant variation in survival and trapping vulnerability between years due to changes in environmental conditions or trapper behavior (Skalski et al. 2007, Gast et al. 2013*a*), the population reconstruction we considered here assumed constant values for these parameters. Previous simulation studies suggest that non-constant survival would tend to overestimate total abundance, whereas non-constant vulnerability would underestimate abundance (Skalski et al. 2011). If inter-annual variability exists in both natural survival and trapping vulnerability, however, these opposing effects can cancel each other out to produce largely unbiased results (Skalski et al. 2011). Although not evaluated here, recent and ongoing advances in reconstruction methods that allow for the incorporation of such variability (Gast 2012,

Gast et al. 2013*a*) provide a promising avenue for evaluating these assumptions and incorporating relevant auxiliary data (e.g., winter severity or hunter skill) in future research.

One overall caution is that incorporating auxiliary data from radio-telemetry studies is based on the assumption that the parameters they help estimate (i.e., natural mortality and harvest probability) are the same for both collared and un-collared animals. Although a significant amount of effort has been put into minimizing the effects of handling and radio-collaring on animal behavior and survival (e.g., Beale and Smith 1973, DelGiudice et al. 1986, Larsen and Gauthier 1989, Cote et al. 1998, Nussberger and Ingold 2006), some populations of animals may still experience significant effects (either positive or negative) from radio-collaring. Black bears in northern Minnesota, for example, are monitored using bright-colored, conspicuous radio-collars and ear-tags that hunters are encouraged to avoid (Minnesota Department of Natural Resources 2013). Using radio-telemetry data on harvest probability in a statistical population reconstruction of this population would tend to underestimate the population-level coefficient of vulnerability and result in significantly higher estimates of population size. In such situations, radio-telemetry data should only be used to help estimate natural survival, even though on average this approach performs worse than one that helps estimate both survival and trapping vulnerability.

It is also important to note that the accuracy and precision of reconstruction based solely on age-at-harvest and catch-effort is positively correlated with the amount of year-to-year variability in catch-effort (Laake 1992, Clawson et al. 2013). Our simulated and case study data, for example, were based on a barely three-fold difference in trapper

effort, compared to the five- and seven-fold differences successfully used before (Skalski et al. 2007, 2011). Our simulation results also demonstrate that reconstruction precision (in terms of CV) is substantially lower for smaller populations than for large ones (Table 4.3). As such, using the standard reconstruction without incorporating radio-telemetry data may still be appropriate for larger-sized populations where catch-effort varies more drastically over the reconstructed time period, such as those studied by Skalski et al. (2007) and Skalski et al. (2011).

Additionally, although the incorporation of radio-telemetry data into statistical population reconstructions yielded fairly predictable improvements in precision over the standard approach, the level of precision displayed by the standard approach is highly dependent on the population of interest. Different levels of natural survival, harvest intensity, and overall population size can drastically alter the accuracy and precision of reconstructed population estimates, as we saw in our simulation study (Table 4.3). For example, even though incorporating a five-year radio-collar study of harvest mortality may decrease the CV by 30%, whether that is a decrease from a CV of 0.15 or 0.90 is a big difference. Additionally, statistical reconstruction of populations experiencing variations in trapper effort different from those analyzed here will undoubtedly be affected differently by the incorporation of radio telemetry data, although we expect the overall patterns (e.g., better improvements from using radio-telemetry data on both survival and harvest rates than from using one or the other) to remain qualitatively the same. This highlights the potential benefits of performing simulation studies for each population (i.e., using the observed variability in trapper effort and rough estimates of

overall abundance, survival, and harvest rates) before relying on the results of population reconstruction or undertaking radio-telemetry studies to improve those results.

4.6 Management Implications

Statistical population reconstruction using integrated population models can provide managers with a flexible and robust means of estimating abundance of harvested species across large geographical areas by using commonly-collected age-at-harvest data. Auxiliary data from radio-telemetry studies can drastically improve the precision of population reconstruction, and these results become evident even with only a couple of years of data collection. These improvements can not only provide more accurate yearly estimates of cohort abundance, but also help managers better discern the influence of natural versus harvest mortality on observed population trends. With this information, management agencies can be better equipped to set effective season lengths and limits, and better manage wild resources that are in high demand by the public.

Intensive Localized Culling as a Management Tool for Chronic Wasting Disease in White-Tailed Deer

5.1 Introduction

Emerging wildlife diseases such as bovine tuberculosis (bTB), avian malaria, and leptospirosis pose a serious threat to the survival and persistence of their natural hosts, and can have significant economic and public health repercussions for incidental hosts such as livestock and humans (Daszak 2000, Cleaveland et al. 2001, Ricklefs and Fallon 2002, Wasserberg et al. 2009, Munoz-Zanzi et al. 2014, Ramsey et al. 2014). Unfortunately, management and eradication of these and other diseases is often hampered by disagreement among stakeholders and uncertainties surrounding estimates of disease prevalence, underlying rates and mechanisms of transmission, and the effectiveness of alternative management strategies (Peterson 1991, Wasserberg et al. 2009, Carstensen et al. 2011, Manjerovic et al. 2014). Because of this, most outbreaks are managed in a *post hoc* or crisis fashion (Peterson 1991, Wasserberg et al. 2009), resulting in measures that are often extremely costly and not always capable of preventing the ongoing spread of the diseases to neighboring areas (e.g., Carstensen et al. 2011). Successful management of future and ongoing wildlife disease outbreaks requires a detailed understanding of not only the underlying dynamics of the disease itself, but also of the effects different management strategies will have on them.

The effects of alternative management strategies on the spread of chronic wasting disease (CWD) in cervids are of current interest due to ongoing and persistent outbreaks of the disease throughout North America (Williams et al. 2002, Haley and Hoover 2015).

Chronic wasting disease is a fatal, transmissible spongiform encephalopathy (TSE), or prion disease, and is the only "infectious" TSE found in free-ranging animals (Williams et al. 2002, Farnsworth et al. 2006, Mathiason et al. 2009, Wasserberg et al. 2009). Similar to other TSEs such as sheep scrapie, transmissible mink encephalopathy, and bovine spongiform encephalopathy (BSE or "mad cow disease"), CWD is caused by an infectious, abnormal isoform of the normal cellular prion protein (PrP^C), referred to as a prion (Mathiason et al. 2006, Tamgüney et al. 2009, Almberg et al. 2011, Haley and Hoover 2015). The disease is characterized by the accumulation of prions in the brain, lymphoid tissue, and central nervous system, which eventually and inevitably leads to host death (Schramm et al. 2006, Collinge and Clarke 2007, Almberg et al. 2011). Cervids infected with CWD can take over a year to display neurodegenerative symptoms, although they become infectious significantly sooner (Williams and Miller 2002, Osnas et al. 2009, Tamgüney et al. 2009, Almberg et al. 2011). The disease was originally thought to be transmitted primarily by direct contact; however, indirect modes of transmission through contact with saliva (Mathiason et al. 2006, Haley et al. 2009*b*), urine (Haley et al. 2009*a*, *b*), feces (Haley et al. 2009*a*, Tamgüney et al. 2009), blood (Mathiason et al. 2006), infected carcasses (Miller et al. 2004), or prion-contaminated environments (Schramm et al. 2006, David Walter et al. 2011) have also been shown to play a large role in CWD transmission. Additionally, CWD prions can remain bio-available and infectious in the environment for at least two years (Miller et al. 2004) and closely related sheep scrapie prions can persist for over 16 years (Georgsson et al. 2006), which severely complicates ongoing management efforts.

Chronic wasting disease was first recognized in a captive population of mule deer (*Odocoileus hemionus hemionus*) in Colorado during the 1960s (Williams and Young 1980), and has since then been identified in at least 15 states and two Canadian provinces (Schramm et al. 2006, Haley et al. 2009*a*, *b*, Mathiason et al. 2009), as well as in at least 30 farms throughout the Republic of Korea (Sohn et al. 2002, Kim et al. 2005). Aside from mule deer, the disease has been detected in white-tailed deer (*Odocoileus virginianus*; Osnas et al. 2009), black-tailed-deer (*Odocoileus hemionus columbianus*; Williams and Young 1980), elk (*Cervus elaphus*; Miller et al. 2000), and moose (*Alces alces*; Baeten et al. 2007). Although prevalence of CWD varies greatly across North America (Haley et al. 2009*a*), estimates as high as 30% have been reported in Colorado (Williams 2005), with annual growth rates between 1.08 and 1.25 (Miller and Conner 2005, Heisey et al. 2010, Almberg et al. 2011). The detrimental effects on cervid populations (e.g., coincidental population declines of up to 45%; Miller et al. 2008), possible ramifications for human and domestic animal health (e.g., spillover events), and uncertain economic consequences have increased the need for effective management strategies to control and prevent the spread of CWD (Williams et al. 2002, Wasserberg et al. 2009).

Because there is no current vaccine or treatment for CWD, herd reduction via wide-scale selective (i.e., deer showing clinical symptoms) or non-selective (i.e., all deer) culling and increased harvest rates by hunters remains the main tool available to management agencies (Williams et al. 2002, Joly et al. 2006, Wasserberg et al. 2009, Haley and Hoover 2015). By simultaneously reducing overall population density, which often decreases the rate of contact between individuals, and removing infectious

individuals from the population altogether, managers use herd reduction to lower the rate at which susceptible individuals are exposed to infectious ones (Lloyd-Smith et al. 2005, Joly et al. 2006, Wasserberg et al. 2009). Although this method has proven to be effective for managing diseases that are primarily transmitted by direct animal-to-animal contact (e.g., bTB; Carstensen et al. 2011), it cannot address the indirect transmission component of a disease such as CWD. As such, attempts to use widespread herd reduction have thus far proven to be insufficient to halt the spread of CWD, with mostly limited or no effect on prevalence in affected populations (Williams et al. 2002, Haley and Hoover 2015). Although studies of herd reduction have occasionally produced favorable results (e.g. Manjerovic et al. 2014), a strategy that addresses both the direct and indirect modes of CWD disease transmission is still needed.

Localized management in the form of intensive, non-selective culling of deer over a small geographical area (e.g., $<5km^2$), may provide a suitable alternative to more broad-scale approaches currently used to manage CWD, especially for white-tailed deer. Populations of white-tailed deer consist mainly of small and geographically distinct social groups of related females (Mathews and Porter 1993, Aycrigg and Porter 1997, McNulty et al. 1997) and larger "bachelor" groups of unrelated males (Hirth 1977, Nixon et al. 1994, Kjaer 2010). Although individual males may move between several groups throughout their lives and the groups themselves are disbanded during the rut (Hirth 1977, Marchinton and Hirth 1984, Nixon et al. 1994, Kjaer 2010), membership in female groups tends to be quite stable (Nixon et al. 1991, Aycrigg and Porter 1997, McNulty et al. 1997, Laseter 2004). Additionally, female white-tailed deer are highly philopatric, with as many as 97% remaining on their natal range for life (Tierson et al. 1985, Aycrigg

and Porter 1997, Campbell et al. 2004). As such, removing all individuals in a social group by aggressively targeting all deer in a small geographical area may create persistent, low-density areas within the population (Porter et al. 1991, McNulty et al. 1997, Oyer and Porter 2004). Previous tests of this method have successfully created low-density areas that were only occasionally visited by dispersing males, and were not encroached upon by females from nearby social groups (McNulty et al. 1997, Oyer and Porter 2004). Because these low-density areas have been shown to persist for at least five years without additional culling (Oyer and Porter 2004) and are projected to persist for up to ten to fifteen years (Porter et al. 1991), we hypothesize that they would be effective in reducing direct contact between susceptible and infectious deer and limiting the number of susceptible deer exposed to a prion-contaminated environment until those prions degraded and were no longer infectious.

Experimental testing of alternative management strategies is often costly, time consuming, and impractical; however, mathematical models provide managers with an efficient alternative means of testing new disease control strategies (Caswell 2001, Bolzoni 2008, Bos and Ydenberg 2011, Wood et al. 2013). Models have been used to not only evaluate the potential effectiveness of different CWD control strategies (Wasserberg et al. 2009, Kjaer 2010, Almberg et al. 2011), but also to compare evidence for different underlying mechanisms of disease transmission (i.e., density- vs. frequency-dependent (Wasserberg et al. 2009, Habib et al. 2011) and direct vs. indirect (Miller et al. 2006, Almberg et al. 2011)) and time since disease introduction (Wasserberg et al. 2009). Due to uncertainties over the relative importance of direct versus indirect transmission, however, most earlier modeling attempts (e.g., Gross and Miller 2001, Wasserberg et al.

2009, Habib et al. 2011) were based on the *a priori* assumption that CWD is primarily directly transmitted (Almberg et al. 2011). As the importance of indirect transmission has become more apparent, more and more modeling attempts (e.g., Miller et al. 2006, Kjaer 2010, Almberg et al. 2011) have attempted to incorporate environmental contamination as an additional source of infection. Unfortunately, these attempts have mostly omitted the spatially-explicit nature of environmental contamination (i.e., deer can only get indirectly infected by visiting areas contaminated with prions), modeling it instead as a non-spatial process (e.g., Miller et al. 2006, Almberg et al. 2011, but see Kjaer 2010). We believe that incorporating the spatial aspect of both direct and indirect transmission can provide novel insights into ongoing CWD outbreaks and proposed methods of disease management.

In this study, we combine empirical data on movement, modes of infection, and disease progression of free-ranging cervids to evaluate the use of localized culling as an alternative method for controlling CWD outbreaks. To this end, we developed a spatially-explicit, individual-based mathematical model to simulate the initial spread, eventual detection, and subsequent management of CWD in a harvested population of white-tailed deer. Because of ongoing uncertainties surrounding the exact details of CWD transmission and persistence in the environment, we tested alternative management strategies across a wide range of direct and indirect transmission rates, functional forms of infection risk (i.e., density- versus frequency-dependent), and rates of environmental prion degradation. Aside from comparing the results of alternative strategies on overall disease prevalence, we also demonstrate how failing to account for the spatial nature of

indirect transmission can drastically alter model results and corresponding conclusions about the effectiveness of localized culling on stopping the spread of CWD.

5.2 Methods

5.2.1 Model Structure

We developed a spatially-explicit, individual-based, SEIC (susceptible-exposed-infectious-clinical) model to explore the effectiveness of localized culling on the spread of CWD in a geographically closed population of 2000 to 7000 white-tailed deer (see Appendix S2 at the Data Repository for U of M (DRUM) for full code). The model keeps tracks of each individual's sex, age, matriarchal lineage, home range, spatial location, behavioral state, disease stage, and time since infection, and operates on a weekly time step. We modeled events during each time step in the following order: movement, births, transition between behavioral states, disease transmission, transition between disease stages, host survival, shedding of prions, and the "survival" or persistence of prion infectivity in the environment. We also separated each year into four seasons applicable to deer biology (Kjaer 2010), each with its own unique adjustments to movement and disease transmission: pre-rut (Sep 1 – Oct 31), rut (Nov 1 – Dec 31), gestation (Jan 1 – Apr 30), and fawning (May 1 – Aug 31). We coded and ran all simulations and associated analyses using Program R (R Core Team 2015).

5.2.2 Movement

We based movement of individual animals on a behavioral state principle, wherein each animal is considered to be in a specific behavioral state at each point in time, based on season, age, sex, and the behavioral states of nearby deer. Different behavioral states are associated with different types of movements, and transitions between states are

88

associated with certain events such as mating or parturition (Fryxell et al. 2008, Nathan et al. 2008, Gurarie et al. 2009). Based on previous modeling attempts of white-tailed deer movement (Kjaer 2010), we modeled three unique behavior states: normal, dispersal, and mating.

We used correlated random walks (CRWs) to model the movement of individual deer, using different combinations of step lengths and turn angles for each behavioral state. Weekly step lengths and turn angles were calculated by re-sampling published estimates of the same parameters measured at 2hr time intervals (Kjaer 2010). More specifically, we simulated the movement of 1,000,000 individual deer over a period of one week at 2hr time intervals using the movement rates and methods of Kjaer (2010), and derived weekly step lengths and turn angles from the resulting data. We then fit Weibull and wrapped Cauchy distributions to the resulting step length and turn angle data using the *fitdistrplus* (Delignette-Muller and Dutang 2014) and *CircStats* (Lund and Agostinelli 2012) packages in Program R, respectively. Although this conversion from 2hr to weekly time-steps was conducted separately for each of the three behavioral states, the resulting distributions for normal and mating behaviors were virtually identical, and as such we used the average of the two in our simulations and named it "Normal' (Table 5.1; Step length and Turn angle).

To account for the formation of matriarchal groups by related female deer and loose bachelor packs by males during most of the year (Hirth 1977, Marchinton and Hirth 1984, Nixon et al. 1994, Kjaer 2010), we incorporated sex- and season-specific group behavior explicitly into our model. We used a maximum group size of 20 and 12 for males and females, respectively, based on published estimates of female group size (5-17;

McNulty et al. 1997, Laseter 2004, Oyer and Porter 2004) and observations that male

groups tend to be larger than female groups (Kjaer 2010). At the start of each simulation,

nearby individuals of the same sex are grouped together and assigned a leading agent that

does not contribute to group size or disease dynamics (Langrock et al. 2014). Using the

framework of Langrock et al. (2014), this agent moves independently, and all other

members of the group follow the leading agent at distances drawn from an exponential

distribution with a mean of 225m (Kjaer 2010). This modeling approach results in

overlapping home ranges consistent with the rose petal hypothesis (Mathews 1989, Porter

et al. 1991, McNulty et al. 1997, Schauber et al. 2015) and eliminates the need to

designate new group leaders in the event of group leader death or dispersal.

Each group is assigned a home range center based on where its leading agent is

located at the start of each simulation. Although movement is random, we included a

group-based probability of turning toward the home range center that increases the

further the agent moves from it:

$$P = 1 - e^{-\left(\Delta x^2/2\varphi_x{}^2 + \Delta y^2/2\varphi_y{}^2\right)},$$

where Δx and Δy are the x and y distance to the home range center and φ_x and φ_y are

group-specific distance boundaries (Kjaer 2010). These boundaries are drawn from sex-

specific uniform distributions based on home range sizes used in other movement models

of white-tailed deer (150-450 and 100-300m for males and females, respectively;

Cosgrove et al. 2012, Ramsey et al. 2014) (Table 5.1; Circular home range dimensions).

Although matriarchal groups are quite stable (Nixon et al. 1991, Laseter 2004),

we included a 0.01 yearly probability of a group member dispersing from the group,

evaluated at weekly time intervals. Because membership in male bachelor groups is

significantly more fluid (Hirth 1977, Nixon et al. 1994, Kjaer 2010), males dispersed from their group at a higher rate of 0.10 (Table 5.1; Group adhesion). An animal in the dispersal state moves independently based on state-specific movement patterns (Table 5.1; Step length and Turn angle) until it encounters and joins another group that is below its maximum group size. Dispersing animals that reached the outer boundaries of our simulated study area were turned around by 90° to maintain a geographically closed population.

During the mating season (Nov 1 to Dec 31), males separate from their respective groups and search their home range for females (Marchinton and Hirth 1984, Nixon et al. 1994) using the Normal distributions of step length and turning angle. Upon locating an adult female that has not already mated and is not being tended by another male, the male follows her for one week before mating (Marchinton and Hirth 1984, Kjaer 2010). Because males stay close to potential mates during this time (Marchinton and Hirth 1984), we modeled the movement patterns of mating pairs analogously to group movement. In this framework, the female moves independently, and the corresponding male follows at a distance drawn from an exponential distribution with a mean of 10m (Kjaer 2010). Once mated, the male and female revert to their normal rut movements, with the female giving birth 27 to 31 weeks later (week randomly chosen; Verme 1965, 1969, Marchinton and Hirth 1984). The number of offspring is drawn from a multinomial distribution based on observed litter sizes in the wild (Verme 1965, 1989, Ozoga et al. 1982) (Table 5.1; Litter size distribution). To account for yearly shifts in home range, each group's home range center is re-calculated at the end of the mating season as the group-average of individual spatial positions on the last day of the rut.

Sex of fawns is assigned at birth (50:50 sex ratio; Verme 1983), although fawn behavior is identical for males and females until one year of age. To account for the mostly sedentary lifestyle of fawns and the frequent visits by the mother (up to three to four times per day) during the first three weeks of their life (Marchinton and Hirth 1984, Kjaer 2010), we modeled both the fawns and their mothers as stationary during this time. After the fawn(s) reach three weeks of age, the female reverts to her normal movements, and the fawn(s) follow her at a distance drawn from the same exponential distribution used to model mating pair movements (mean = 10m). At six months of age, the fawn takes on the movement characteristics of a regular group member (Kjaer 2010). We included a 20% chance of dispersal for female fawns when they reach one year of age (Nixon et al. 1991, Nelson and Mech 1992, Nelson 1998), and for simplicity assumed that all male fawns dispersed at one year of age (Table 5.1; Chance of dispersal at one year of age).

5.2.3 Disease

We modeled disease transmission to include both direct (i.e., animal to animal) and indirect (i.e., environmental contamination) modes of transmission, with the probability of a susceptible individual becoming infected being dependent on proximity to infectious or clinical deer and prions still bio-available in the environment. Although the model records the exact spatial location of every individual at the end of each time step, we aggregated disease transmission to operate on a lattice framework. To this end, we modeled our hypothetical study area as a raster with a pixel size of 100x100m. We chose a study area size of 400 km^2 to mimic the size of deer permit areas (DPAs) over which management decisions concerning white-tailed deer are often made (range = 119 –

4714km^2; (MNDNR 2016*a*) At the end of each movement and shedding phase, the

location of every deer and prion is converted into its corresponding grid location. Using

this framework, susceptible deer can only become infected from individuals or prions that

occupy the same cell. To account for the increased risk of infections associated with

higher abundances of prions and infectious or clinical deer, we used a similar force-of-

infection,λ, as Almberg et al. (2011), adopted into a cell-specific format:

$$\lambda = \frac{ln[1 + \beta_d \times Z + \beta_i \times V]}{1 - \varepsilon + \varepsilon \times N},$$

where Z is the combined number of infectious and clinical deer, V is the number of

prions, and N is the total number of deer in a given cell. β_d and β_i represent the direct

and indirect transmission coefficients, respectively, and ε allows us to model

transmission modes between density- ($\varepsilon = 0$) and frequency-dependent ($\varepsilon = 1$). We

converted λ into a cell-specific infection probability equal to $1 - e^{-\lambda}$ (Almberg et al.

2011), and modeled infection as a binomial process. All simulations were run over a

range of ε values (0, 0.0001, and 1; Almberg et al. 2011) to provide a more robust

evaluation of potential management strategies.

We used a compartmental approach to separate individuals into different stages of

infection, each with a different duration and probability of transitioning to the next stage

(Almberg et al. 2011). Upon infection, susceptible individuals transitioned into a series of

consecutive, week-long, exposed stages (i.e., one week since infection, two weeks, etc.)

before becoming infectious (mean duration of exposure = 26.6 weeks, range = 16 − 35).

Once infectious, an individual transitioned through a series of infectious stages (mean

duration of infectious phase = 34.8 weeks, range = 24 − 44), followed by a series of

clinical stages (mean duration of clinical phase = 18.2 weeks, range = 1 − 36), and then

death. Transitions between stages were evaluated at weekly time intervals using binomial processes based on stage-specific transition probabilities (Table 5.2) estimated from previous studies by Almberg et al. (2011).

Host survival was modeled as a binomial process and was evaluated at each time-step using published estimates of white-tailed deer annual survival (Nelson and Mech 1986, Ballard et al. 1999, Vreeland et al. 2004, Ramsey et al. 2014) (Table 5.1; Natural mortality). Additionally, 44% of antlered (yearling and adult males) and 21% of antlerless (fawns of both sexes and yearling and adult females) deer were harvested each year during a two-month-long hunter harvest during the rut season, modeled as a binomial process (Wasserberg et al. 2009, Ramsey et al. 2014). We assumed that dependent fawns died if their mother died until they became functional ruminants at nine weeks of age (Marchinton and Hirth 1984, Kjaer 2010). Based on previous modeling attempts (Wasserberg et al. 2009, Almberg et al. 2011), only individuals in the clinical stage experienced a disease-associated increased risk of mortality (Table 5.2). Individuals in the infectious and clinical stages shed two "prions units" (hereafter prions) per week, and released 100 prions upon death (Almberg et al. 2011). For simplicity, we assumed that all 100 prions were released immediately upon death, that individuals in the exposed stage shed no prions upon death, that hunter-harvested individuals released no prions upon death, and that the number of prions released was independent of the time since infection.

Prion "survival" (i.e., whether a prion remains infectious and bio-available in the environment) was evaluated on an individual basis using a binomial process. Although pen studies of CWD prions suggest the infectivity can persist for over two years (Miller

et al. 2004), the exact rate of prion survival is unknown. As such, we ran simulations over a range of prion half-lives (i.e., the average time it takes for half of the prion load to cease being infectious) between six months and four years.

5.2.4 Simulated Scenarios

Disease outbreaks were initiated by introducing one infectious male individual into the population at a random location along the study area boundary, three years after the start of the simulation. The infectious individual was considered to be in the "dispersal" state, simulating the introduction of CWD into the population from an outside source. Because the probability of infection given contact is unknown, we adjusted β_d and β_i to produce disease dynamics that fell within certain boundaries deemed reasonable based on empirical data from ongoing CWD epidemics in Wisconsin, Colorado, and Wyoming (Almberg et al. 2011). These boundaries included an annual population growth rate greater than or equal to 0.85, annual prevalence growth less than 1.25 with disease control and 1.45 without disease control, and a peak prevalence no greater than 0.5. We also adjusted the probability of an unsuccessful pregnancy (i.e., litter size of 0) to produce an annual host population growth rate of approximately 1.0 in the absence of disease and between 1.35 and 1.45 in the absence of hunting or disease (Wasserberg et al. 2009).

We simulated a 0.02 probability of a harvested deer in the infectious and clinical stages being detected during the two-month-long hunting season. Although this surveillance rate is lower than that currently employed in Minnesota (8%; MNDNR 2016*b*), it resulted in an average prevalence at detection (between 0.009 and 0.011) that was more consistent with published estimates of early outbreak prevalence in white-tailed

deer (0.007 – 0.021; Miller et al. 2000, Manjerovic et al. 2014). This prevalence at detection was also closer to detection thresholds used in previous models of CWD (0.03; Almberg et al. 2011).

Once an outbreak was detected, we simulated intensive culling of the area surrounding the detected individual as a potential tool for managing CWD outbreaks. We tested intensive non-selective culling across square areas of 1, 3, and 5km^2 for a period of two months. To account for the fact that not all deer can be successfully located and removed during culling, we modeled individual-based culling as a binomial process with a probability of 0.8, based on the removal success rate documented in field tests of localized deer management (McNulty et al. 1997, Oyer and Porter 2004). We also simulated no culling and non-selective widespread culling of the entire study area to provide comparisons to our proposed strategy.

Because both the effectiveness of localized management (Laseter 2004, Miller et al. 2010) and the dynamics of CWD spread (Conner and Miller 2004, Joly et al. 2006, Habib et al. 2011) may be influenced by host density, we simulated three different starting population densities (6, 12, and 18 deer/km^2) for each control strategy based on published estimates of white-tailed deer density (2.3 – 20 deer/km; Tierson et al. 1985, Lesage et al. 2000, Miller et al. 2010, Cosgrove et al. 2012). At the start of each simulation, we first calculated the expected number of male and female social groups as a function of starting population density and the average number of individuals in each social group (10 and 6 for males and females, respectively). We then assigned random home range centers to each group while maintaining a minimum distance of 450 and 300m between centers for males and females, respectively. These values were selected to

generate variable deer density across the modeled landscape, mostly geographically distinct group home ranges at low population densities, and significant home range overlap among groups at higher densities, consistent with the results of previous studies (McNulty et al. 1997, Laseter 2004, Oyer and Porter 2004).

Simulations were run 250 times for each unique combination of deer density, transmission parameters, and control strategy. Presented results include median and 95% quantiles of CWD prevalence, abundance of infectious and bio-available prions in the environment, and the total number of deer culled, evaluated ten years after the disease was first detected. We also report the probability of disease establishment (i.e., proportion of simulations where the disease spread enough to be detected) for each of the modeled deer densities.

5.3 Results

Because of ongoing uncertainties regarding the relative strength of direct and indirect transmission as well as the rate of prion shedding and persistence in the environment, a wide range of variable combinations (i.e., β_d, β_i, ε, and prion half-life) produced reasonable disease dynamics. Although results differed substantially between different variable combinations, the overall qualitative patterns and conclusions discussed here were consistent across all tested combinations (Appendix A). As such, we only discuss a subset of our results, using a prion half-life of 0.5 years (Kjaer 2010) and a relative strength of direct versus indirect transmission of approximately 4.6 (Table 5.1; Transmission coefficients).

As expected, overall deer density had a substantial effect on the dynamics of CWD spread. The probability of disease establishment given the introduction of a single

infectious individual increased from 11.5% at low densities to 36.0% and 56.8% at

medium and high densities, respectively. Disease prevalence ten years after disease

detection in the absence of disease management followed a similar pattern (Figure 5.1).

Localized management created persistent areas of low density by removing entire

social groups (or most members of social groups) in affected areas. At low deer densities,

these areas were not substantially encroached upon by members of neighboring social

groups for up to ten years after disease detection, but were occasionally visited and

eventually repopulated by dispersing deer. At higher deer densities, however, these areas

were encroached upon and visited by dispersing deer at a much higher rate and were

repopulated much sooner.

Localized management consistently performed better than no management in

terms of decreasing the final prevalence of CWD across all modeled deer densities, with

larger culling scales (i.e., 1, 3, and 5km^2) producing increasingly better results (Figure

5.1). However, the overall effectiveness (i.e., disease prevalence ten years of detection) of

localized management in controlling the spread of CWD was significantly affected by

deer density. At low deer densities (e.g., 6 deer/km^2), for example, localized culling at a

scale of 5km^2 resulted in a prevalence of 0 ten years after disease detection in 83% of

simulations, a prevalence less than 0.01 in 95% of simulations, and negligible effects on

population density (mean final density of 6.1 ($\widehat{SD} = 2.3$) deer/km^2). At higher population

densities, however, the same scale of localized culling resulted in a median prevalence of

0.04 ten years post-detection, and a prevalence greater than 0.06 in 25% of simulations.

Using widespread culling to remove the same number of deer as was done with

localized management produced inconsistent results in terms of controlling the spread of

CWD. At low and medium deer densities, wide-spread culling performed substantially worse than localized culling of almost any scale. At high deer densities, however, widespread culling outperformed all scales of localized management, resulting in a median prevalence of 0.01 ten years post-detection. However, this reduction in prevalence coincided with a 75% decrease in overall population size, and a final deer density of 4.7 ($\widehat{SD} = 0.9$) deer/km^2. Although localized management was not as effective at controlling CWD at these higher densities, it was able to achieve its results while maintaining a substantially higher deer density of 13.4 ($\widehat{SD} = 4.7$) deer/km^2.

5.4 Discussion

Our simulation study demonstrates that localized management in the form of intensive, non-selective culling of deer over a small geographical area provides a more effective control strategy than the broad-scale approaches currently used to manage CWD, especially at low to medium deer densities. By removing most if not all individuals in a social group by aggressively targeting all deer in a small geographical area surrounding a detected infection, localized management creates areas of significantly lower deer density that can persist for several years (Porter et al. 1991, McNulty et al. 1997, Oyer and Porter 2004). The creation of these areas increases the probability that prions deposited by infected individuals throughout their home range would degrade or otherwise cease to be bio-available before they come into contact with susceptible deer moving into or through the area. Aggressively culling deer over larger areas increases the probability that all prions deposited by infected deer and deer from neighboring social groups that were already exposed to those prions are contained within the culling area, thereby increasing the probability of disease eradication.

Our results suggest that although aggressively culling a 5km^2 area around detected infections is sufficient to prevent the spread of CWD in low-density populations of white-tailed deer, the effectiveness of this method decreases at higher deer densities. Higher population densities result in substantial home range overlap among groups that effectively connect groups that would otherwise be geographically isolated at lower densities (Laseter 2004). This decreases the probability that localized management can successfully remove all individuals that frequently use areas where prions have been deposited by infectious deer (Miller et al. 2010). Additionally, our results affirm the concern that higher densities increase the rate at which dispersing deer and deer from neighboring social groups repopulate the low-density areas created by localized management (McNulty et al. 1997, Miller et al. 2010), decreasing the amount of time prions are allowed to degrade before coming into contact with susceptible deer from outside the culling area. Despite this limitation, however, localized management was still effective at limiting the spread of CWD while maintaining relatively stable deer densities across the study area.

By explicitly incorporating the spatial aspect of both direct and indirect modes of CWD transmission, our results demonstrate that successful management of CWD is influenced more by the location (i.e., where on the landscape deer are removed) rather than the number of deer removed. At lower deer densities, for example, removing 6.5% of the overall population by intensively culling areas of 5km^2 around detected infections resulted in substantially lower disease prevalence after ten years than removing the same proportion of the population randomly throughout the entire study area (Figure 5.1). Because the management of CWD and other wildlife diseases is often hampered by

disagreement among stakeholders as well as logistical and monetary constraints (Peterson 1991, Carstensen et al. 2011, Manjerovic et al. 2014), these results have important implications for how managers can maximize the efficiency of deer removal (e.g., number of infections prevented per number of deer removed) to prevent the spread of CWD.

The main advantage of localized management over more wide-spread control measures is that the removal of entire social groups creates areas of low density that are not encroached upon by members of neighboring social groups for up to five to fifteen years (Porter et al. 1991, Oyer and Porter 2004), allowing enough time for deposited prions to degrade or otherwise cease to be bio-available. However, as more time passes, deer from other social groups will eventually repopulate the low-density area, which can significantly decrease the effectiveness of localized management if prions persist for longer than five years. Unfortunately, very little is currently known about the rate of prion degradation, except that infectivity can persist for over two years (Miller et al. 2004). Because the rate at which deposited prions are no longer capable of infecting susceptible individuals can have significant effects on the effectiveness of localized culling and other CWD management strategies, we recommend future studies attempt to estimate the maximum time prions can persist in the environment under a variety of circumstances (e.g., temperature, moisture, and soil composition).

Our results also support a mechanistic explanation for the observation that widespread herd reduction is insufficient to halt the spread of CWD (Williams et al. 2002, Haley and Hoover 2015), especially without severely decreasing deer abundance. Herd reduction aims to reduce the rate at which susceptible individuals are exposed to

infectious ones (Lloyd-Smith et al. 2005, Joly et al. 2006, Wasserberg et al. 2009), which is extremely effective for managing diseases that are transmitted primarily by direct animal-to-animal contact. When applied to CWD, however, this strategy was not able to address the fact that because prions can persist in the environment for at least two years (Miller et al. 2004), susceptible individuals were able to become infected long after infectious individuals were removed. Although herd reduction proved to be an ineffective tool once CWD became established, our results suggest that preemptively reducing deer density can significantly decrease the probability of disease establishment. As expected, our simulation study confirmed that lower population densities reduce the probability that infectious adults and the prions they deposit come into contact with susceptible deer while the prions are still infectious. Managers concerned about the risk of CWD introduction into previously disease-free regions could consider reducing deer density to decrease such risks.

One aspect of our modeling approach that may limit the applicability of our results is the temporal aggregation of movement and disease spread. Because we used a weekly time step, we were unable to include events that occurred at shorter intervals, such as occasional excursions made by deer outside of their home range (Inglis et al. 1979, Nelson 1993, 1998). These excursions may increase the rate of indirect contact between otherwise geographically isolated social groups and thereby complicate management efforts. However, because our results were robust to increases in deer density and the amount of inter-group home range overlap, we believe this omission did not drastically alter model results. Similarly, we were unable to include variability in the amount of times males follow females during the rut (Marchinton and Hirth 1984, Kjaer

102

2010) or instances of sparring and dominance fights during the pre-rut and rut seasons (Marchinton and Hirth 1984), both of which can influence CWD spread. As finer-scale estimates (e.g., daily) of transition rates between stages of infection become available, we recommend future modeling attempts to evaluate how these and other fine-scale interactions and heterogeneities influence CWD dynamics and control.

Additionally, our discretization of space forced us to evaluate all instances of direct and indirect contact on a strictly cell-by-cell basis, obscuring potentially important heterogeneities in contact within each cell. This can be alleviated by using finer pixel grain (e.g., 25m x 25m; Kjaer 2010), but requires more detailed information about movement rates and disease transmission. A more general issue with this lattice-based approach is that two individuals 5m apart who occupy adjacent corners of two neighboring cells, for example, would have weaker (in our approach, zero) interaction than two individuals who occupy the same cell but are 95m apart at opposite ends, despite the fact that the former are closer to each other than the latter (Keeling and Rohani 2008). Despite these limitations, the use of lattice models in evaluating different control strategies is well-supported by the literature (e.g., White and Harris 1995, Tischendorf et al. 1998, Deal et al. 2004, Kjaer 2010), and we believe that our model provides a reasonable abstraction of reality for our purposes.

Previous studies have identified significant genetic heterogeneity in susceptibility to CWD between individual white-tailed deer, which undoubtedly play an important role in the transmission of the disease throughout the population (O'Rourke 2004, Mathiason et al. 2006, 2009). Because we lack data that would allow us to model this heterogeneity, however, we assumed all individuals were equally susceptible to infection. This

assumption should be adjusted in the future as more data on genetic and other forms of

individual heterogeneity become available. Environmental factors such as soil properties

may also play an important role in the transmission and persistence of prions in the

environment. David Walter et al. (2011), for example, found that the odds of CWD

infection in mule deer increased by up to 8.9% for every 1% increase in the proportion of

soil clay (i.e., soil particles $< 2\mu$m in size) within each animal's home range. This and

other soil attributes (e.g., abundance of organic matter) can not only enhance the

infectivity of deposited prions, but can also help maintain them close to the surface where

they can be easily ingested by herbivores (Johnson et al. 2006, 2007, Schramm et al.

2006, David Walter et al. 2011). Although we did not include the spatial heterogeneity in

prion persistence and infectivity associated with different soil attributes throughout the

study site, future research could help identify particular areas where increased rates of

indirect transmission require more intensive and prolonged control efforts.

Finally, ongoing uncertainties regarding the shedding rate of prions during

different stages of the disease, including death, and the rate at which shed prions cease to

be bio-available or infectious raises the question as to whether our parameterization of

these variables is reasonable. Despite the documented resistance of prions to a variety of

standard decontamination procedures such as proteases, chemical disinfection, dry heat,

and ultraviolet and microwave irradiation (Taylor 1999, 2000, Johnson et al. 2006,

Schramm et al. 2006), the bioavailability of prions likely declines over time as shed

prions are covered over by soil and debris or otherwise rendered unavailable to cervids

(Schramm et al. 2006, Almberg et al. 2011). Although the exact distribution of this

decline is unknown, an exponential decay rate seems plausible because the actual

distribution likely has a long and skinny right-tail (Almberg et al. 2011). Ongoing research on this and other aspects of prion biology, such as the relative amount of prions deposited by living deer versus decaying carcasses or whether the rate of shedding increases as the disease progresses as has been previously suggested (Miller et al. 1998), will undoubtedly alter the quantitative results presented here, but we believe that our overall conclusions about the relative effectiveness of localized management will remain unchanged.

5.5 Management Implications

Our results highlight the importance of explicitly accounting for the spatial nature of indirect transmission in the management and control of CWD. Incorporating this spatial nature into disease models allowed us to demonstrate that localized management in the form of intensive, non-selective culling of deer over a small geographical area can provide a suitable alternative to more broad-scale approaches currently used to manage CWD in white-tailed deer, especially at low to medium deer densities. We recommend wildlife managers implement localized culling of areas surrounding detected infections alongside an adaptive management approach to provide an empirical test of our simulation results. Given the clear dependence of localized management success on the rate of prion degradation in the environment, we also recommend improving our understanding of prion persistence and other characteristics of CWD transmission. In the absence of this information, wildlife managers and other stakeholders will continue to be faced with limited confidence in model predictions and the merits of alternative control strategies.

Conclusion

Conservation biology is, to a large extent, a matter of maximizing the efficiency with which managers and researchers spend the limited resources they have available. To this end, a more quantitative understanding of the trade-offs between effort and the validity or impact of results can help with the allocation of said resources. This dissertation explored this trade-off in a variety of systems. The results of chapter three demonstrated that as more effort is put into accurately identifying available habitat, the results of habitat selection analyses can change drastically. Chapter four, on the other hand, demonstrated that although incorporating radio-telemetry data drastically improves the accuracy of population reconstruction, this benefit becomes less pronounced as more data are collected. Identifying this point of diminishing returns in other settings can help managers determine the optimum time to switch efforts from one task (e.g., collecting radio-telemetry data) to another (e.g., sampling vegetation for habitat selection analyses).

It is important to remember, however, that there are many outside factors that may hinder efforts to maximize this efficiency. Chapter five demonstrated that aggressively removing deer from small geographical areas is a more effective strategy to control the spread of chronic wasting disease than removing the same number throughout a larger area. Although this strategy may be the most efficient in terms of impact on disease prevalence per deer removed, social and political considerations may make such a strategy impractical. Local hunters, for example, may not appreciate the removal of so many deer from their region, regardless of the effect the removal might have on controlling the spread of the disease. These and other considerations make navigating the

trade-off between effort and results substantially more difficult and should be explored further in future research.

Finally, it is important to note that mathematical models, apart from being a prime example of the trade-off between effort and results, are an excellent tool with which to explore these trade-offs in other systems. Chapter four used simulation modeling to quantify how much improvement in reconstruction accuracy can be gained per extra year of radio-telemetry data. Performing similar simulations and analyses before beginning data collection can help managers and researchers identify areas where efficiency can be improved. With this information, management agencies can be better equipped to manage a range of socially and ecologically important natural resources.

Figure 3.1. Distribution of study sites across Northeastern Minnesota.
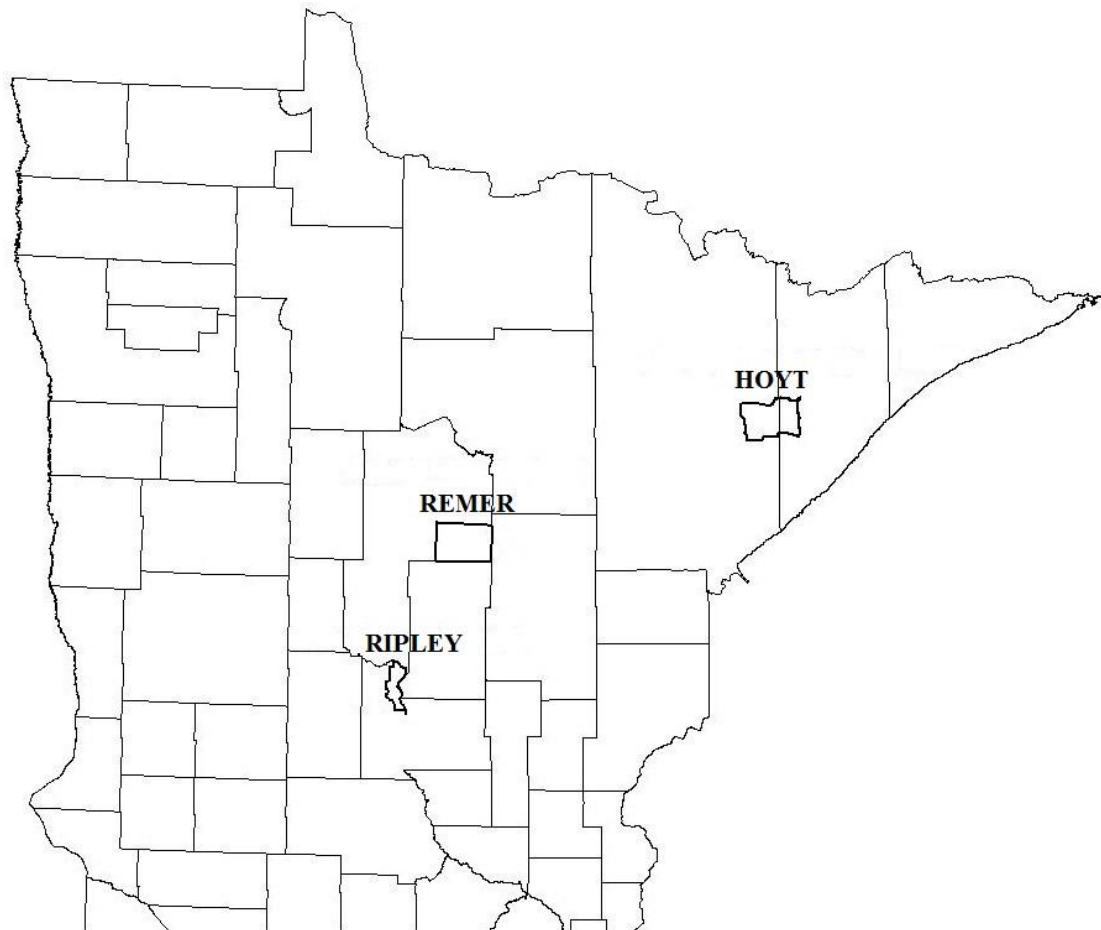
Figure 3.2. An example of the hierarchical random sampling used to place 637 25-m fixed-radius plots throughout a 25-km^2 circular area surrounding each confirmed natal den in order to locate large-diameter trees that could have been used by fishers as den sites.
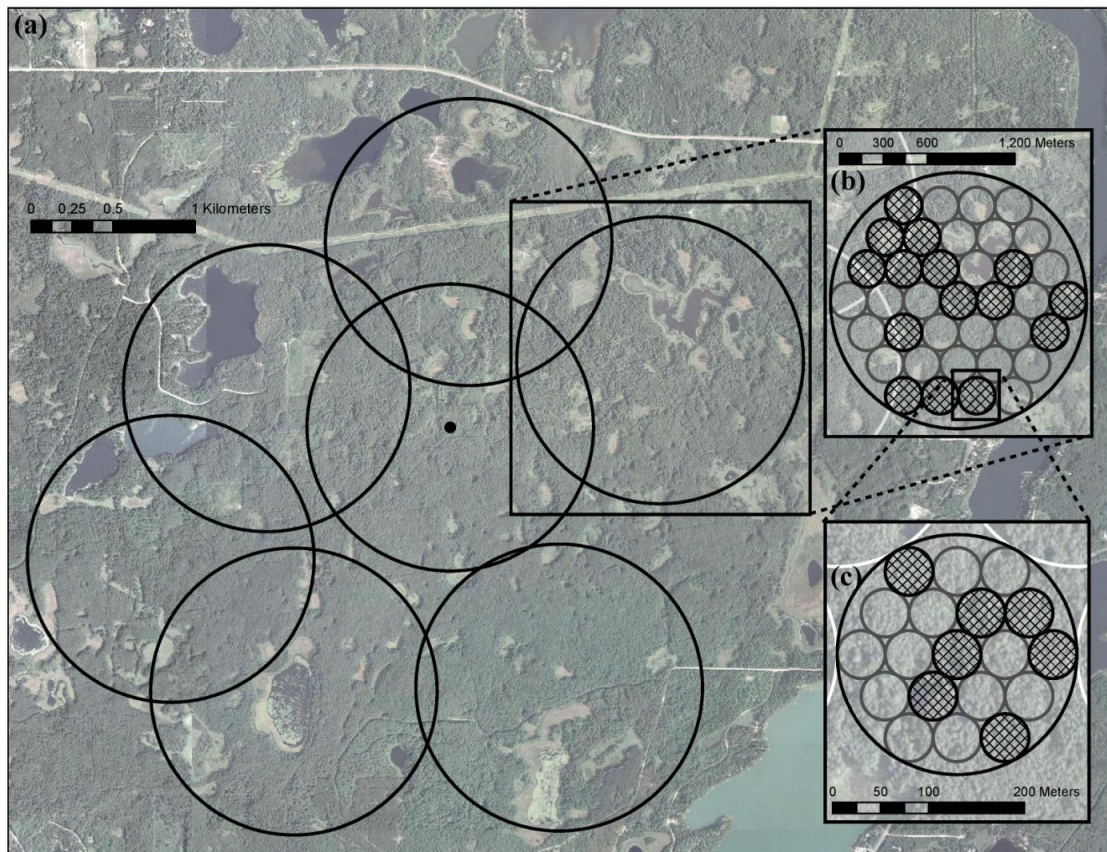
Figure 3.3. The distribution of relocation distances between consecutively used den sites by female fishers in Northeastern Minnesota observed in the larger project on *Martes* ecology (J. Erb, unpublished data).
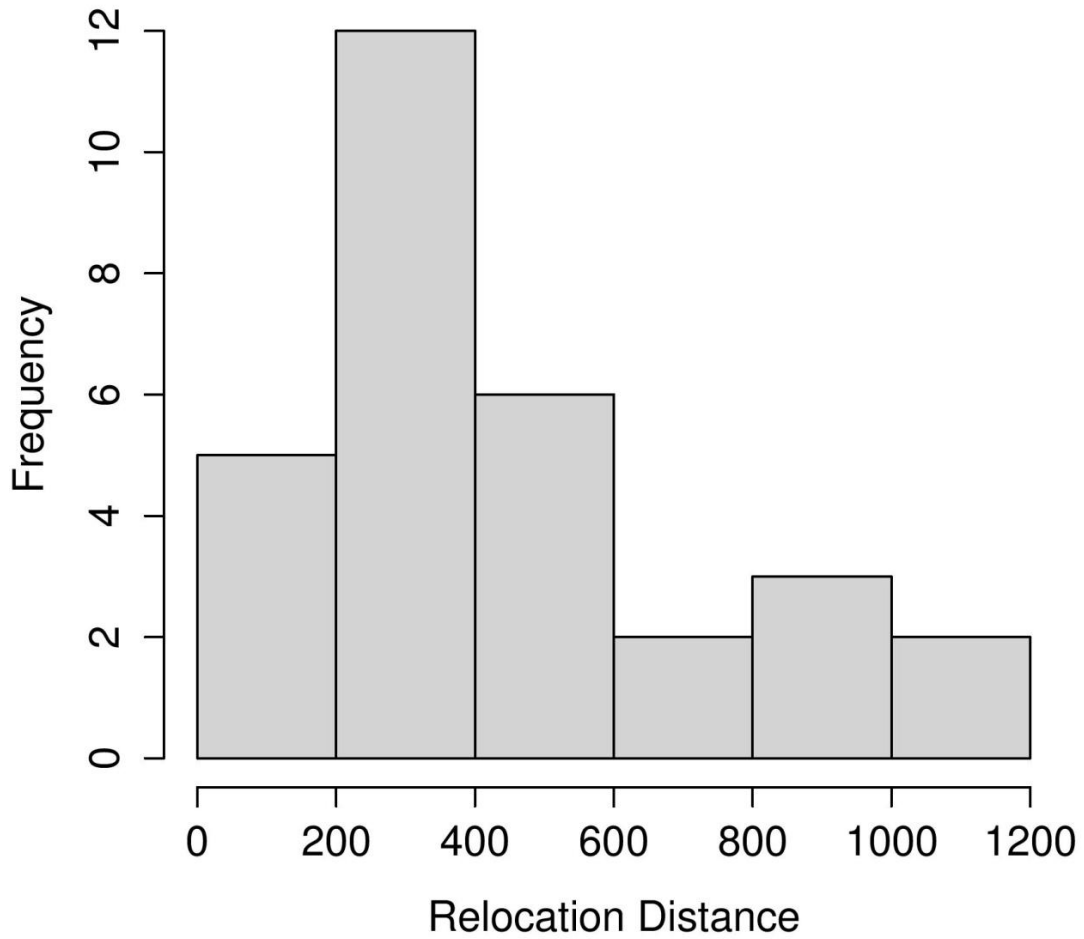
Figure 3.4. The distribution of the number of suitable cavity trees found in each of the 13,281 25-m-radius plots sampled throughout the 25-km^2 circular area surrounding each confirmed fisher natal den used in our study.
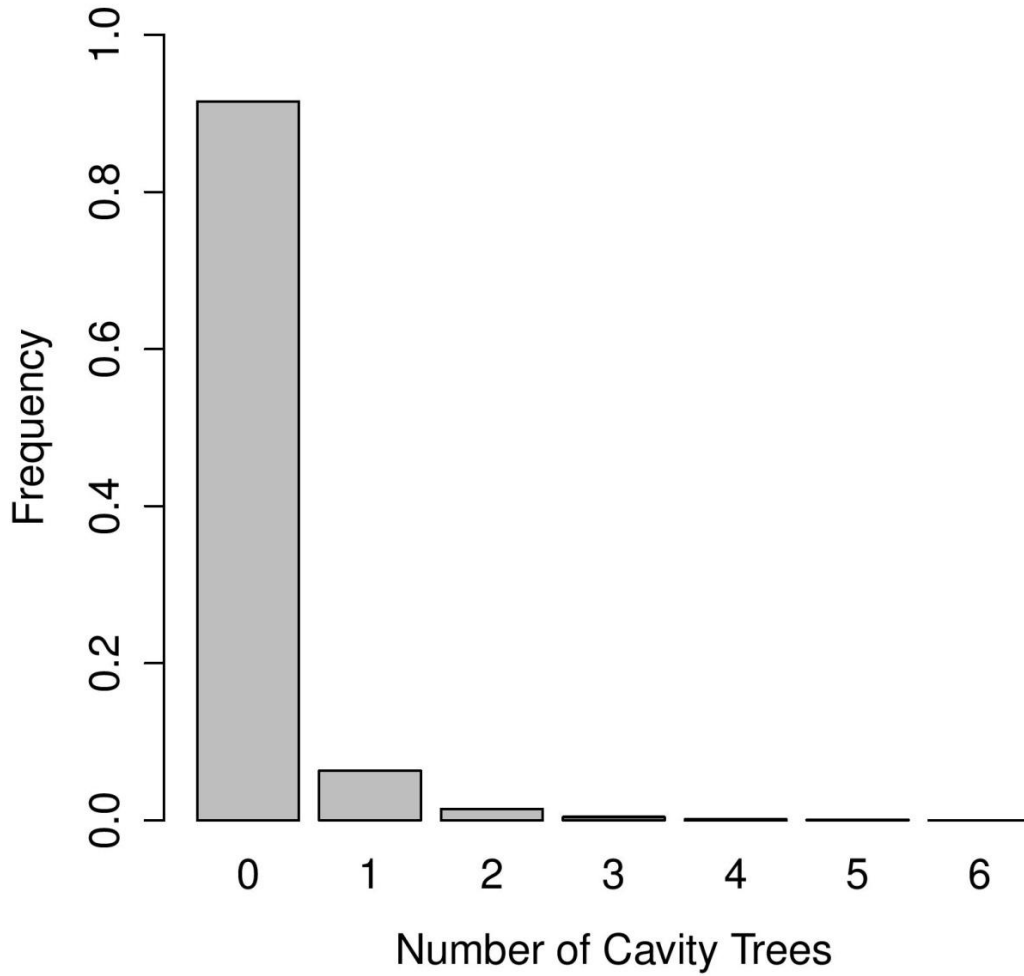
Figure 4.1. A visual depiction of the timeline (left) and demographic processes (right) governing population growth in the modeled marten population, where N_{ij} is the abundance in year i of age-class j immediately before the onset of harvest, the top row (i.e., N_{11}, N_{12}, N_{13}) represents the initial age-class abundances, and the left column represents recruitment of juveniles into the population in subsequent years (i.e., $N_{21}, \dots, N_{Y1}$).

Figure 4.2. The effects of increasing (a-c) the average number of animals collared per year and (d-f) the number of years of a radio-telemetry study on reducing reconstruction bias when using radio-telemetry data on natural survival (left), harvest mortality (middle), and both (right), as compared to using no radio-telemetry data (left-most item in each plot). See Appendix B for data values.
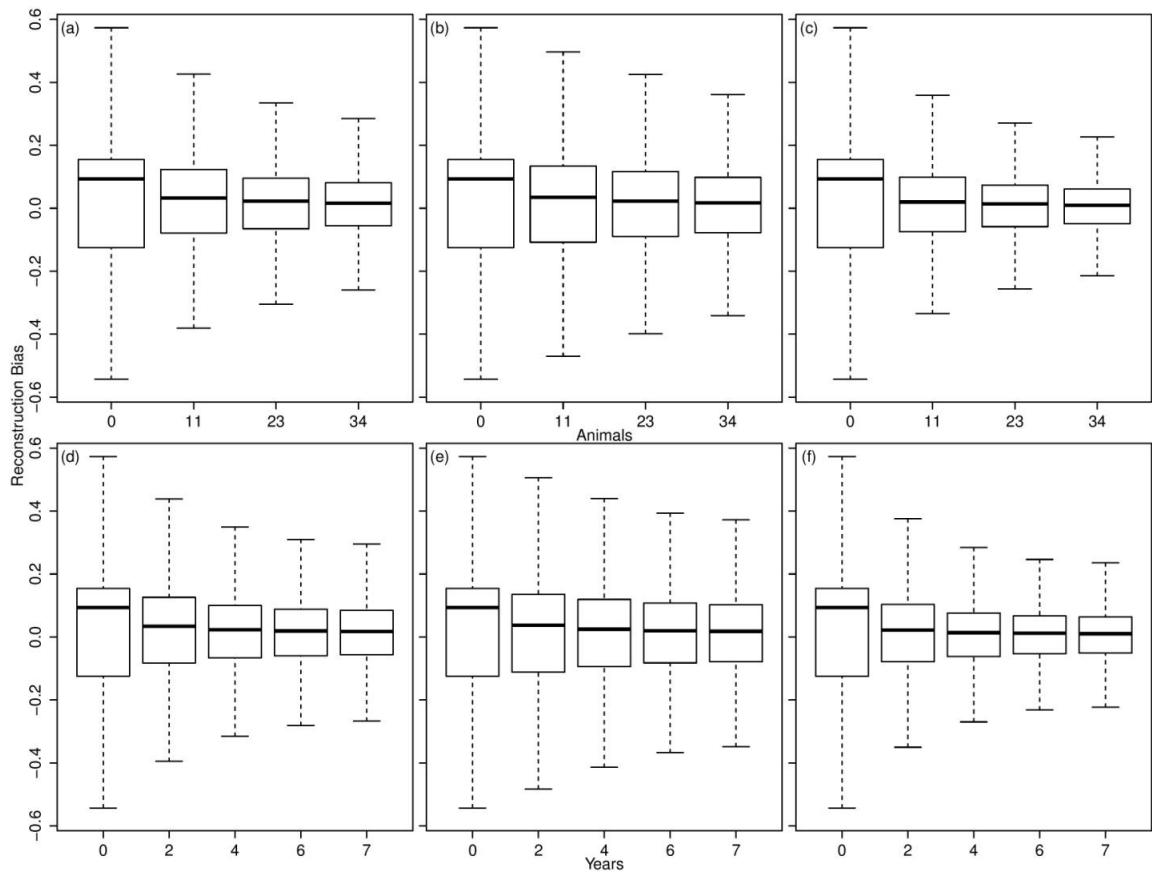
Figure 4.3. Estimated trends in (a) abundance and (b) recruitment into the trappable population of American marten in northern Minnesota (thick solid line) between 2007 and 2014 based on the best available population reconstruction model, along with associated confidence intervals (dotted lines), compared to the results of independent population modeling conducted by the MN DNR (thin dashed line; Erb 2015). Alternative population reconstructions of the American marten populations in northern Minnesota using (c) radio-telemetry data on harvest mortality (dashed line), natural survival (dashed-dotted), and neither (dotted), and (d) the full likelihood model with the first one (dashed), three (dashed-dotted), and five (dotted) years of radio-telemetry data removed, compared to the best-fit model (solid). See Appendix B for data values.
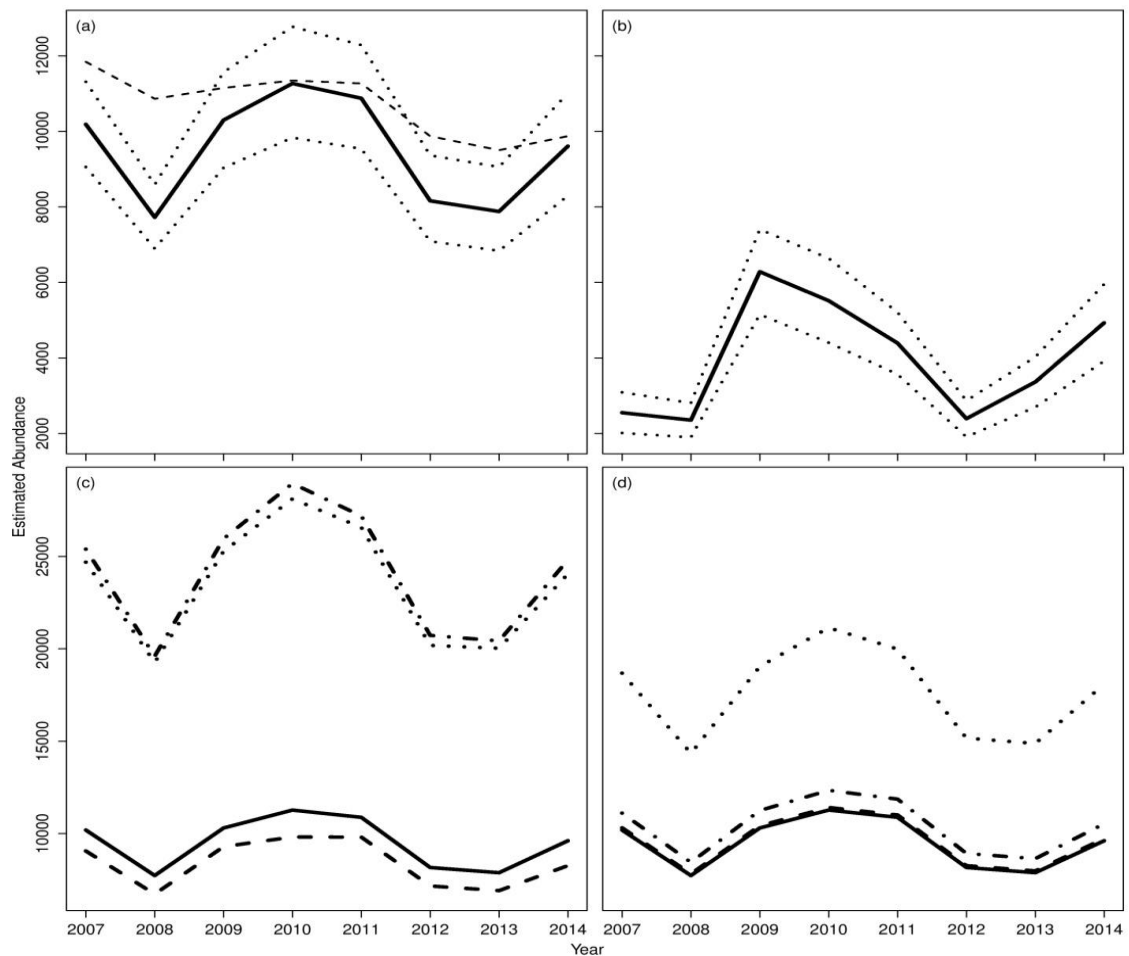
Figure 5.1. Disease prevalence ten years after disease detection in populations of white-tailed deer experiencing densities of 6, 12, and 18 deer/km^2 (left, middle, and right panel, respectively). Management scenarios include no culling (N), intensive non-selective culling across square areas of 1, 3, and 5km^2 for a period of two months (L, M, and H, respectively), and widespread, non-selective culling of the entire study area (W). See Appendix B for data values.
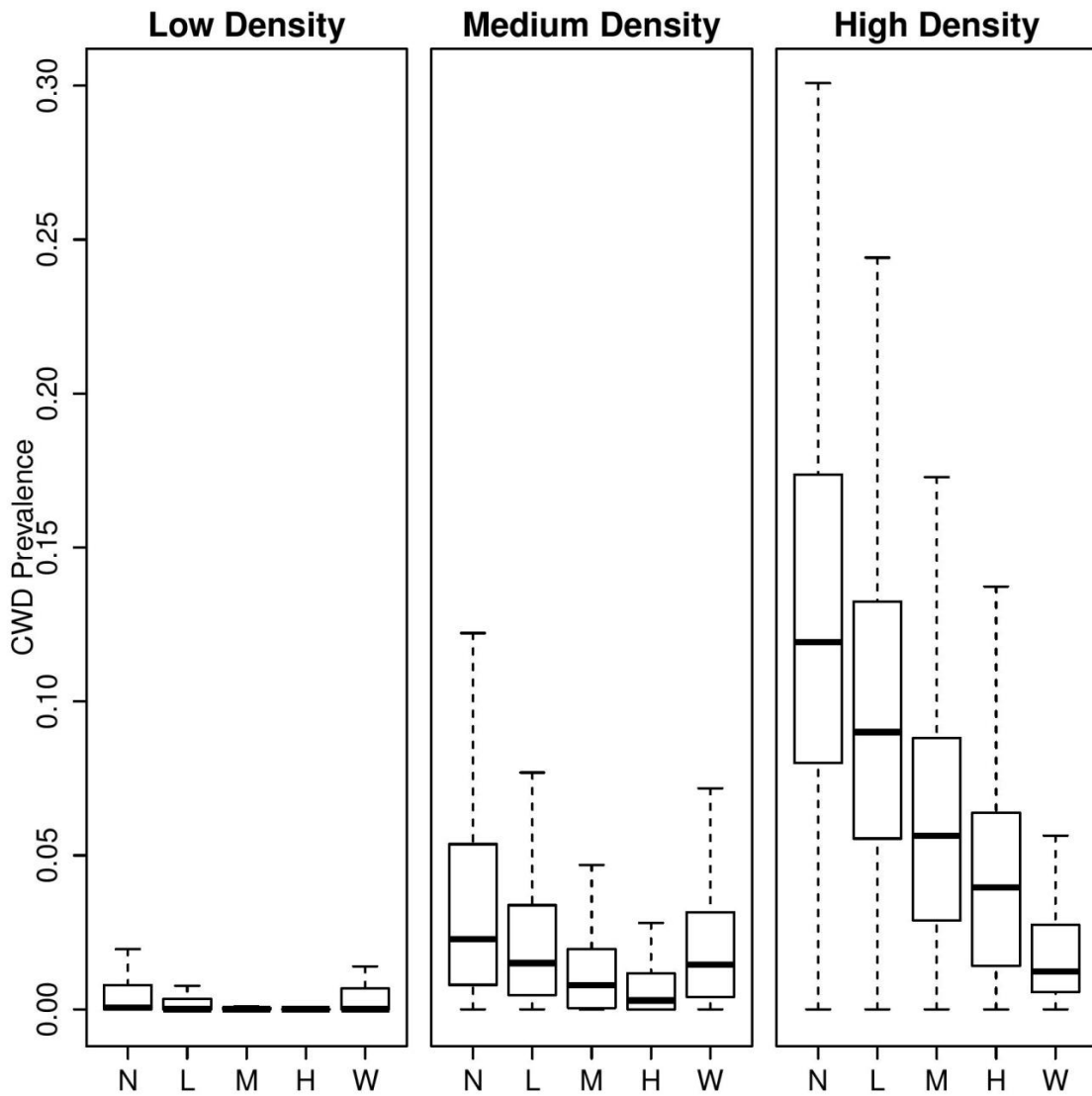
Table 3.1. Characteristics of large-diameter cavity trees used as reproductive dens by female fishers in Northeastern Minnesota and of cavity trees deemed available to the animals.

Characteristic	Natal Dens	Maternal Dens	All Dens Combined	Available Trees
Species				
Aspen	23	15	38 (69.1%)	925 (57.7%)
Maple	3	4	7 (12.7%)	170 (10.6%)
Oak	3	0	3 (5.5%)	196 (12.2%)
Pine	4	1	5 (9.1%)	130 (8.1%)
Cedar	2	0	2 (3.6%)	7 (0.4%)
Other	0	0	0 (0.0%)	174 (10.9%)
Health				
Alive	20	10	30 (54.5%)	1082 (67.5%)
Declining	3	1	4 (7.3%)	68 (4.2%)
Dead	12	9	21 (38.2%)	452 (28.2%)
Mean DBH (cm)	52.5	50.7	51.9	48.1

Table 3.2. Comparison of AICc-based variable sum of weights (SW), estimates, and standard errors derived using all-subset model averaging with conditional logistic regression in the study of den site selection by female fishers in northern Minnesota.

Variable	SW	Estimate
Health – Declining	0.999	1.2536 (0.0245)
– Live		1.2827 (0.0214)
Type – Deciduous	0.415	-0.2157 (0.0139)
Slope	0.545	-0.0253 (0.0012)
DBH	0.574	0.0121 (0.0006)
Stand – Deciduous	0.186	0.1321 (0.0190)
– Mixed		0.0089 (0.0056)
– Other		0.1846 (0.0267)
Cavity Abundance at 350m	0.379	0.0005 (0.0001)
Cavity Abundance at 600m	0.679	0.0030 (0.0001)
Cavity Abundance at 850m	0.371	-0.0001 (0.0001)
Cavity Abundance at 1050m	0.556	-0.0015 (0.0001)

Table 4.1. Age-at-harvest data and estimated trapping effort (in terms of total trap-nights, scaled to a mean of one) for American marten in northeastern Minnesota from 2007 to 2014.

Year	Age class (yr)			Trapper effort
	0.6	1.6	2.6+	
2007	753	715	1013	1.260
2008	783	402	768	1.323
2009	1250	344	656	1.007
2010	930	486	561	0.877
2011	1069	680	995	1.235
2012	545	489	576	1.049
2013	567	259	496	0.736
2014	576	140	275	0.513

Table 4.2. Age-specific radio-telemetry data on natural (v_j) and harvest mortalities (u_j) and associated at-risk counts (n_j) for juvenile, yearling, and adult (j = 1, 2, and 3, respectively) American marten in northeastern Minnesota from 2008 to 2014.

Year	n_1	v_1	u_1	n_2	v_2	u_2	n_3	v_3	u_3
2008	1	0	0	9	2	3	2	1	0
2009	6	2	1	3	1	1	20	4	5
2010	21	6	5	3	2	0	10	5	2
2011	26	8	11	14	1	5	8	3	3
2012	15	9	2	11	2	1	16	6	3
2013	3	0	0	16	5	3	16	7	2
2014	5	1	0	7	4	2	28	7	2

Table 4.3. Simulation results for population reconstructions using no radio-telemetry data (L_0) and radio-telemetry data on natural survival, harvest mortality, and both (L_1, L_2, and L_3, respectively) in terms of mean bias, B, and coefficient of variation, CV. Simulations were performed at low (L) and high (H) levels of population size, N, harvest rate, H, and natural survival, S.

					B				CV	
N	H	S	L_0	L_1	L_2	L_3	L_0	L_1	L_2	L_3
L	L	L	0.204	-0.032	-0.011	-0.007	0.517	0.473	0.189	0.160
L	H	L	0.008	-0.004	0.003	-0.002	0.156	0.097	0.096	0.071
L	H	H	-0.114	0.005	0.002	0.003	0.464	0.055	0.084	0.049
H	L	L	-0.009	-0.038	-0.011	-0.011	0.517	0.372	0.184	0.147
H	H	L	-0.029	0.003	0.003	0.003	0.186	0.048	0.074	0.043
H	H	H	-0.034	0.004	0.000	0.004	0.206	0.049	0.077	0.043

Table 4.4. Likelihood and Akaike's Information Criterion (AIC) for alternative population reconstruction models of American marten in northeastern Minnesota using both natural and harvest mortality data from a seven year radio-telemetry study.

Model	ln L	No. of parameters	AIC
McS	-235.491	12	494.982
$McS_{0.6,1.6+}$	-237.909	13	501.818
$McS_{0.6,1.6,2.6+}$	-247.347	14	522.693
$Mc_{0.6,1.6+}S$	-307.156	13	640.313
$Mc_{0.6,1.6+}S_{0.6,1.6+}$	-317.777	14	663.554
$Mc_{0.6,1.6+}S_{0.6,1.6,2.6+}$	-326.566	15	683.132
$Mc_{0.6,1.6,2.6+}S$	-395.347	14	818.694
$Mc_{0.6,1.6,2.6+}S_{0.6,1.6+}$	-402.799	15	835.599
$Mc_{0.6,1.6,2.6+}S_{0.6,1.6,2.6+}$	-411.805	16	855.610

Table 5.1. Demographic, movement, and disease parameters used to model the spread of CWD in white-tailed deer.

Parameter	Model Value	Source
Chance of dispersal at one year of age	Males: 100%[a] Females: 20%	(Nixon et al. 1991, Nelson and Mech 1992, Nelson 1998)
Group adhesion[b]	Males: 90% Females: 99%	(Kjaer 2010)
Maximum group size (adults)	Males: 20[c] Females: 12	(McNulty et al. 1997, Laseter 2004, Oyer and Porter 2004)
Circular home range dimensions (m)	Males: 150-450 Females: 100-300	(Cosgrove et al. 2012, Ramsey et al. 2014)
Gestation period (weeks)	27-31	(Verme 1965, 1969, Marchinton and Hirth 1984)
Probability of failed pregnancy	Yearlings: 25% Adults: 10%	
Litter size distribution	1 fawn: 25% 2 fawns: 75% 3 fawns: 5%	(Verme 1965, 1989, Ozoga et al. 1982)
Natural mortality	Juveniles: 0.20 Yearlings: 0.10	(Nelson and Mech 1986, Ballard et al. 1999,

	Adults: 0.05	Vreeland et al. 2004, Ramsey et al. 2014)
Following distance	Normal: $X \sim$ Exponential($\lambda = 1/225$) Mating: $X \sim$ Exponential($\lambda = 1/10$)	(Kjaer 2010, Langrock et al. 2014)
Step length	Normal: $X \sim$ Weibull(3.62, 256.01) Dispersal: $X \sim$ Weibull(3.70, 1000.00)	(Kjaer 2010)
Turn angle	Normal: $X \sim$ WrpCauchy(5.94, 0.21) Dispersal: $X \sim$ WrpCauchy(0.12, 0.01)	(Kjaer 2010)
Prion half life (years)	0.5^{d}	(Kjaer 2010)
Transmission coefficients	Direct (β_d) = 5.5e-3[d] Indirect: (β_i) = 1.2e-3[d] Functional form ($\varepsilon = 0.0001$)[d]	

[a] For simplicity we assumed that all male fawns dispersed at one year of age.

[b] Yearly probability of an adult not dispersing from their social group.

[c] Based on observations that male groups tend to be larger than female groups (Kjaer 2010).

[d] These values are only a subset of those tested, and are presented here because the overall patterns and conclusions discussed in this paper were consistent across all tested combinations.

Table 5.2. Probabilities used to model weekly transition between exposed (E), infectious (I), and clinical (C) stages in white-tailed deer.

Week	$P(E{\rightarrow}I)$	$P(I{\rightarrow}C)$	$P(C{\rightarrow}death)$ for juveniles	$P(C{\rightarrow}death)$ for yearlings	$P(C{\rightarrow}death)$ for adults
1	0	0	0.005	0.003	0.002
2	0	0	0.008	0.006	0.005
3	0	0	0.010	0.008	0.007
4	0	0	0.013	0.011	0.010
5	0	0	0.016	0.014	0.013
6	0	0	0.019	0.017	0.016
7	0	0	0.023	0.021	0.020
8	0	0	0.027	0.025	0.024
9	0	0	0.032	0.030	0.029
10	0	0	0.037	0.035	0.034
11	0	0	0.042	0.040	0.039
12	0	0	0.048	0.046	0.045
13	0	0	0.055	0.052	0.051
14	0	0	0.061	0.058	0.057
15	0	0	0.067	0.064	0.063
16	0.010	0	0.074	0.072	0.071
17	0.014	0	0.081	0.079	0.078
18	0.019	0	0.089	0.087	0.086
19	0.026	0	0.097	0.095	0.094

20	0.035	0	0.105	0.103	0.102
21	0.046	0	0.113	0.111	0.110
22	0.060	0	0.121	0.119	0.118
23	0.075	0	0.130	0.128	0.127
24	0.093	0.009	0.140	0.138	0.137
25	0.113	0.013	0.150	0.148	0.147
26	0.135	0.018	0.160	0.158	0.157
27	0.159	0.025	0.172	0.170	0.169
28	0.185	0.034	0.185	0.183	0.182
29	0.214	0.045	0.199	0.197	0.197
30	0.247	0.058	0.217	0.216	0.215
31	0.287	0.073	0.240	0.239	0.238
32	0.340	0.091	0.273	0.271	0.270
33	0.419	0.110	0.317	0.315	0.315
34	0.567	0.131	0.390	0.389	0.388
35	1	0.154	0.542	0.541	0.54
36		0.179	1	1	1
37		0.205			
38		0.234			
39		0.267			
40		0.306			
41		0.356			
42		0.432			

43	0.577
44	1

LITERATURE CITED

Almberg, E. S., P. C. Cross, C. J. Johnson, D. M. Heisey, and B. J. Richards. 2011. Modeling Routes of Chronic Wasting Disease Transmission: Environmental Prion Persistence Promotes Deer Population Decline and Extinction. A. Yates, editor. PLoS ONE 6:e19896.

Anderson, R. M., H. C. Jackson, R. M. May, and A. M. Smith. 1981. Population dynamics of fox rabies in Europe. Nature 289:765–771.

Anderson, R. M., G. F. Medley, R. M. May, and A. M. Johnson. 1986. A preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV), the causative agent of AIDS. Journal of Mathematics Applied in Medicine and Biology 3.

Anneville, O., P. Cury, C. Le Page, and J. P. Treuil. 1998. Modelling fish spatial dynamics and local density-dependence relationships: detection of patterns at a global scale. Aquatic Living Resources 11:305–314.

Antonovics, J., Y. Iwasa, and M. P. Hassell. 1995. A generalized model of parasitoid, venereal, and vector-based transmission processes. The American Naturalist 145:661–675.

Argue, A. M., K. J. Mills, and B. R. Patterson. 2008. Behavioural response of eastern wolves (Canis lycaon) to disturbance at homesites and its effects on pup survival. Canadian Journal of Zoology 86:400–406.

Arnold, T. W. 2010. Uninformative Parameters and Model Selection Using Akaike's Information Criterion. Journal of Wildlife Management 74:1175–1178.

Arthur, S. M. 1987. Ecology of Fishers in South-Central Maine. Ph.D., University of Maine, Orono, Maine, USA.

Arthur, S. M., W. B. Krohn, and J. R. Gilbert. 1989. Home Range Characteristics of Adult Fishers. The Journal of Wildlife Management 53:674.

Arthur, S. M., B. F. J. Manly, L. L. McDonald, and G. W. Garner. 1996. Assessing Habitat Selection when Availability Changes. Ecology 77:215–227.

Aubry, K. B., and C. Raley. 2006. Ecological Characteristics of Fishers (Martes pennanti) in the Southern Oregon Cascade Range.

Aycrigg, J. L., and W. F. Porter. 1997. Sociospatial Dynamics of White-Tailed Deer in the Central Adirondack Mountains, New York. Journal of Mammalogy 78:468–482.

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.

Bak, P., K. Chen, and C. Tang. 1990. A forest-fire model and some thoughts on turbulence. Physics letters A 147:297–300.

Ballard, W. B., H. A. Whitlaw, S. J. Young, R. A. Jenkins, and G. J. Forbes. 1999. Predation and Survival of White-Tailed Deer Fawns in Northcentral New Brunswick. The Journal of Wildlife Management 63:574.

Bartoń, K. 2015. MuMIn: Multi-Model Inference.

Beale, D. M., and A. D. Smith. 1973. Mortality of Pronghorn Antelope Fawns in Western Utah. The Journal of Wildlife Management 37:343.

Becker, E. F., M. A. Spindler, and T. O. Osborne. 1998. A Population Estimator Based on Network Sampling of Tracks in the Snow. The Journal of Wildlife Management 62:968.

Begon, M., M. Bennett, R. G. Bowers, N. P. French, S. M. Hazel, and J. Turner. 2002. A clarification of transmission terms in host-microparasite models: numbers, densities and areas. Epidemiology and infection 129:147–153.

Berg, S. 2012. Using demographically stochastic modeling to study the effects of cub survival on Amur leopard population trends. Proceedings of the Stochastic Modeling Techniques and Data Analysis International Conference 2:37–42.

Berg, S. 2015. The package "sigloc" for the R software: A tool for triangulating transmitter locations in ground-based telemetry studies of wildlife. Bulletin of the Ecological Society of America 96:500–507.

Berryman, A. A. 1992. The origins and evolution of predator-prey theory. Ecology 73:1530–1535.

Beyer, H. L., K. Hampson, T. Lembo, S. Cleaveland, M. Kaare, and D. T. Haydon. 2012. The implications of metapopulation dynamics on the design of vaccination campaigns. Vaccine 30:1014–1022.

Birch, C. P. D., S. P. Oom, and J. A. Beecham. 2007. Rectangular and hexagonal grids used for observation, experiment and simulation in ecology. Ecological Modelling 206:347–359.

Böhm, M., M. R. Hutchings, and P. C. L. White. 2009. Contact Networks in a Wildlife-Livestock Host Community: Identifying High-Risk Individuals in the Transmission of Bovine TB among Badgers and Cattle. D. M. Ojcius, editor. PLoS ONE 4:e5016.

Bolzoni, L. 2008. Seasonality and Adaptive Dynamics in Host-Parasite Systems in Wildlife. Ph.D., Università degli Studi di Parma, Parma, Italy. <http://dspace-unipr.cineca.it/handle/1889/948>. Accessed 3 Mar 2016.

Bos, D., and R. Ydenberg. 2011. Evaluation of alternative management strategies of muskrat Ondatra zibethicus population control using a population model. Wildlife Biology 17:143–155.

Boyce, J. S. 1961. Forest Pathology. 3rd edition. McGraw-Hill, New York, New York, USA.

Bradley, C. A., and S. Altizer. 2005. Parasites hinder monarch butterfly flight: implications for disease spread in migratory hosts: Parasites and monarch flight. Ecology Letters 8:290–300.

Broadfoot, J. D., R. C. Rosatte, and D. T. O'Leary. 2001. Raccoon and skunk population models for urban disease control planning in Ontario, Canada. Ecological Applications 11:295–303.

Broms, K., J. R. Skalski, J. J. Millspaugh, C. A. Hagen, and J. H. Schulz. 2010. Using Statistical Population Reconstruction to Estimate Demographic Trends in Small Game Populations. Journal of Wildlife Management 74:310–317.

Bull, C. M., S. S. Godfrey, and D. M. Gordon. 2012. Social networks and the spread of *Salmonella* in a sleepy lizard population. Molecular Ecology 21:4386–4392.

Bunnell, F. L., and D. E. N. Tait. 1980. Bears in Models and in Reality: Implications to Management. Bears: Their Biology and Management 4:15.

Burnham, K. P., and D. R. Anderson. 2002. Model selection and multimodel inference. Springer-Verlag, New York, New York, USA.

Butler, J. M., and T. J. Roper. 1996. Ectoparasites and sett use in European badgers. Animal Behaviour 52:621–629.

Caillaud, D., M. E. Craft, and L. A. Meyers. 2013. Epidemiological effects of group size variation in social species. Journal of The Royal Society Interface 10:20130206–20130206.

Campbell, T. A., B. R. Laseter, W. M. Ford, and K. V. Miller. 2004. Feasibility of localized management to control white-tailed deer in forest regeneration areas. Wildlife Society Bulletin 32:1124–1131.

Caraco, T., S. Glavanakov, G. Chen, J. E. Flaherty, T. K. Ohsumi, and B. K. Szymanski. 2002. Stage-Structured Infection Transmission and a Spatial Epidemic: A Model for Lyme Disease. The American Naturalist 160:348–359.

Carstensen, M., D. J. O'Brien, and S. M. Schmitt. 2011. Public acceptance as a determinant of management strategies for bovine tuberculosis in free-ranging U.S. wildlife. Veterinary Microbiology 151:200–204.

Caswell, H. 2001. Matrix Population Models: Construction, Analysis, and Interpretation. Second edition. Sinauer Associates, Inc. Publishers, Sunderland, Massachusetts.

Cavrini, F., P. Gaibani, A. M. Pierro, G. Rossini, M. P. Landini, and V. Sambri. 2009. Chikungunya: an emerging and spreading arthropod-borne viral disease. Journal of infection in developing countries 3.

Clawson, M. V., J. R. Skalski, and J. J. Millspaugh. 2013. The utility of auxiliary data in statistical population reconstruction. Wildlife Biology 19:147–155.

Cleaveland, S., M. K. Laurenson, and L. H. Taylor. 2001. Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. Philosophical Transactions of the Royal Society B: Biological Sciences 356:991–999.

Collinge, J., and A. R. Clarke. 2007. A General Model of Prion Strains and Their Pathogenicity. Science 318:930–936.

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

Cosgrove, M. K., H. Campa, D. S. L. Ramsey, S. M. Schmitt, and D. J. O'brien. 2012. Modeling vaccination and targeted removal of white-tailed deer in Michigan for bovine tuberculosis control. Wildlife Society Bulletin 36:676–684.

Cote, S. D., M. Festa-Bianchet, and F. Fournier. 1998. Life-History Effects of Chemical Immobilization and Radiocollars on Mountain Goats. The Journal of Wildlife Management 62:745.

Coulter, M. W. 1966. Ecology and management of fishers in Maine. Ph.D., Syracuse University, New York, USA.

Craft, M. E., and D. Caillaud. 2011. Network Models: An Underutilized Tool in Wildlife Epidemiology? Interdisciplinary Perspectives on Infectious Diseases 2011:1–12.

Craft, M. E., E. Volz, C. Packer, and L. A. Meyers. 2010. Disease transmission in territorial populations: the small-world network of Serengeti lions. Journal of The Royal Society Interface 8:776–786.

Croft, D. P., M. Edenbrow, S. K. Darden, I. W. Ramnarine, C. Oosterhout, and J. Cable. 2011. Effect of gyrodactylid ectoparasites on host behaviour and social network structure in guppies Poecilia reticulata. Behavioral Ecology and Sociobiology 65:2219–2227.

Daszak, P. 2000. Emerging Infectious Diseases of Wildlife-- Threats to Biodiversity and
    Human Health. Science 287:443–449.

David Walter, W., D. P. Walsh, M. L. Farnsworth, D. L. Winkelman, and M. W. Miller.
    2011. Soil clay content underlies prion infection odds. Nature Communications
    2:200.

Davis, L. R. 2009. Denning Ecology and Habitat Use by Fisher (Martes pennanti) in Pine
    Dominated Ecosystems of the Chilcotin Plateau. Masters, Simon Fraser
    University, Burnaby, British Columbia.

Davis, S., P. Trapman, H. Leirs, M. Begon, and J. A. P. Heesterbeek. 2008. The
    abundance threshold for plague as a critical percolation phenomenon. Nature
    454:634–637.

Deal, B., C. Farello, M. Lancaster, T. Kompare, and B. Hannon. 2004. A dynamic model
    of the spatial spread of an infectious disease: the case of fox rabies in Illinois. R.
    Costanza and A. Voinov, editors. Landscape Simulation Modeling: A Spatially
    Explicit, Dynamic Approach. Springer-Verlag, New York, New York, USA.

DeCesare, N. J., M. Hebblewhite, F. Schmiegelow, D. Hervieux, L. E. Morgantini, M.
    Wheatley, and M. Musiani. 2012. Transcending scale dependence in identifying
    habitat with resource selection functions. Ecological Applications 22:1068–1083.

Delahay, R. J., G. C. Smith, and M. R. Hutchings, editors. 2009. Management of Disease
    in WildMammals. Springer, New York, New York, USA.

DelGiudice, G. D., L. D. Mech, W. J. Paul, and P. D. Karns. 1986. Effects on fawn
    survival of multiple immobilizations of captive pregnant white-tailed deer.
    Journal of Wildlife Diseases 22:245–248.

Delignette-Muller, M. L., and C. Dutang. 2014. fitdistrplus: An R Package for Fitting
    Distributions. Journal of Statistical Software 64:1–34.

Dexter, M. 2014. Trapping Harvest Statistics. Division of Fish and Wildlife, St. Paul,
    Minnesota, USA.

Dickson, B. G., and P. Beier. 2002. Home-Range and Habitat Selection by Adult Cougars
    in Southern California. The Journal of Wildlife Management 66:1235.

Dillingham, P. 2010. Generation time and the maximum growth rate for populations with
    age-specific fecundities and unknown juvenile survival. Ecological Modelling
    221:895–899.

Dirks, B. J., and N. J. Dietz. 2009. Camp Ripley and Arden Hills Minnesota Army
    National Guard Training Sites Conservation Program Report - 2008 Annual
    Report. Camp Ripley Series Report, Minnesota Department of Natural Resources,
    St. Paul, Minnesota, USA.

Dobson, A., and M. Meagher. 1996. The population dynamics of Brucellosis in the Yellowstone National Park. Ecology 77:1026–1036.

Drewe, J. A. 2009. Who infects whom? Social networks and tuberculosis transmission in wild meerkats. Proceedings of the Royal Society B: Biological Sciences 277:633–642.

Erb, J. 2015. Registered Furbearer Population Modeling 2015 Report. Minnesota Department of Natural Resources, Grand Rapids, Minnesota, USA.

Erb, J., P. Coy, and B. Sampson. 2013*a*. Reproductive ecology of fisher and marten in Minnesota. Minnesota Department of Natural Resources Summaries of Wildlife Research Findings 2013 100–111.

Erb, J., P. Coy, and B. Sampson. 2014. Survival and causes of mortality for fishers and martens in Minnesota. Pages 83–92 *in* L. Cornicelli, M. Carstensen, M. D. Grund, M. A. Larson, and J. S. Lawrence, editors. Summaries of Wildlife Research Findings 2014. Minnesota Department of Natural Resources, St. Paul, Minnesota, USA.

Erb, J., B. Sampson, and P. Coy. 2008. Fisher and Marten Demography and Habitat Use in Minnesota. Pages 51–60 *in* M. W. DonCarlos, R. O. Kimmel, J. S. Lawrence, and M. S. Lenarz, editors. Summaries of Wildlife Research Findings 2008. Minnesota Department of Natural Resources, St. Paul, Minnesota, USA.

Erb, J., B. Sampson, and P. Coy. 2009. Fisher and marten demography and habitat use in Minnesota. Minnesota Department of Natural Resources Summaries of Wildlife Research Findings 2008 51–60.

Erb, J., B. Sampson, and P. Coy. 2013*b*. Survival and causes of mortality for fisher and marten in Minnesota. Minnesota Department of Natural Resources Summaries of Wildlife Research Findings 2013 112–121.

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

Fenner, A. L., S. S. Godfrey, and C. Michael Bull. 2011. Using social networks to deduce whether residents or dispersers spread parasites in a lizard population: Social networks and parasite transmission. Journal of Animal Ecology 80:835–843.

Ferguson, N. M., C. A. Donnely, and R. M. Anderson. 2001. The foot-and-mouth epidemic in Great Britain: pattern of spread and impact of interventions. Science 292:1155–1160.

Fieberg, J. R., K. W. Shertzer, P. B. Conn, K. V. Noyce, and D. L. Garshelis. 2010. Integrated Population Modeling of Black Bears in Minnesota: Implications for Monitoring and Management. W. M. Getz, editor. PLoS ONE 5:e12114.

133

Flynn, R. W., and T. V. Schumacher. 2009. Temporal Changes in Population Dynamics of American Martens. Journal of Wildlife Management 73:1269–1281.

Fry, F. E. J. 1949. Statistics of a Lake Trout Fishery. Biometrics 5:27.

Fryxell, J. M., M. Hazell, L. Börger, B. D. Dalziel, D. T. Haydon, J. M. Morales, T. McIntosh, and R. C. Rosatte. 2008. Multiple movement modes by large herbivores at multiple spatiotemporal scales. Proceedings of the National Academy of Sciences 105:19114–19119.

Fulford, G. R., M. G. Roberts, and J. A. P. Heesterbeek. 2002. The Metapopulation Dynamics of an Infectious Disease: Tuberculosis in Possums. Theoretical Population Biology 61:15–29.

Garnett, G. P. 2005. Role of herd immunity in determining the effect of vaccines against sexually transmitted disease. Journal of Infectious Diseases 191:S97–S106.

Gast, C. M. 2012. Fixed and Random Effects Models and Multistage Estimation Procedures for Statistical Population Reconstructions. Ph.D., University of Washington, Seattle, Washington, USA.

Gast, C. M., J. R. Skalski, and D. E. Beyer. 2013*a*. Evaluation of fixed- and random-effects models and multistage estimation procedures in statistical population reconstruction: Fixed- and Random-Effects Models. The Journal of Wildlife Management 77:1258–1270.

Gast, C. M., J. R. Skalski, J. L. Isabelle, and M. V. Clawson. 2013*b*. Random Effects Models and Multistage Estimation Procedures for Statistical Population Reconstruction of Small Game Populations. S. D. Peddada, editor. PLoS ONE 8:e65244.

Georgsson, G., S. Sigurdarson, and P. Brown. 2006. Infectious agent of sheep scrapie may persist in the environment for at least 16 years. Journal of General Virology 87:3737–3740.

Giam, X., and J. D. Olden. 2016. Quantifying variable importance in a multimodel inference framework. Methods in Ecology and Evolution 7:388–397.

Gibilisco, C. J. 1994. Distributional dynamics of modern Martes in Northern America. S. W. Buskirk, A. S. Harestad, M. G. Raphael, and R. A. Powell, editors. Martens, Sables, and Fishers: Biology and Conservation. Cornell University Press, Ithaca, New York, USA.

Gibson, G. J. 1997. Markov chain Monte Carlo methods for fitting spatiotemporal stochastic models in plant epidemiology. Applied Statistics 46:215–233.

Gilbert, L., R. A. Norman, M. K. Laurenson, H. Reid, and P. J. Hudson. 2001. Disease Persistence and Apparent Competition in a Three-Host Community: An Empirical

and Analytical Study of Large-Scale, Wild Populations. Journal of Animal Ecology 70:1053–1061.

Gilbert, P., and R. Varadhan. 2012. numDeriv: Accurate Numerical Derivatives. R package version 2012.9-1. <http://cran.r-project.org/package=numDeriv>. Accessed 21 Sep 2015.

Godfrey, S. S. 2013. Networks and the ecology of parasite transmission: A framework for wildlife parasitology. International Journal for Parasitology: Parasites and Wildlife 2:235–245.

Godfrey, S. S., C. M. Bull, R. James, and K. Murray. 2009. Network structure and parasite transmission in a group living lizard, the gidgee skink, Egernia stokesii. Behavioral Ecology and Sociobiology 63:1045–1056.

Godfrey, S. S., J. A. Moore, N. J. Nelson, and C. M. Bull. 2010. Social network structure and parasite infection patterns in a territorial reptile, the tuatara (Sphenodon punctatus). International Journal for Parasitology 40:1575–1585.

Gove, N. E., J. R. Skalski, P. Zager, and R. L. Townsend. 2002. Statistical Models for Population Reconstruction Using Age-at-Harvest Data. The Journal of Wildlife Management 66:310.

Gremillion-Smith, C., and A. Woolf. 1988. Epizootiology of skunk rabies in North America. Journal of W 24:620–626.

Griffin, R. H., and C. L. Nunn. 2011. Community structure and the spread of infectious disease in primate social networks. Evolutionary Ecology 26:779–800.

Grimm, V. 1994. Mathematical models and understanding in ecology. Ecological Modeling 75/76:641–651.

Gross, J. E., and M. W. Miller. 2001. Chronic Wasting Disease in Mule Deer: Disease Dynamics and Control. The Journal of Wildlife Management 65:205.

Gurarie, E., R. D. Andrews, and K. L. Laidre. 2009. A novel method for identifying behavioural changes in animal movement data. Ecology Letters 12:395–408.

Habib, T. J., E. H. Merrill, M. J. Pybus, and D. W. Coltman. 2011. Modelling landscape effects on density–contact rate relationships of deer in eastern Alberta: Implications for chronic wasting disease. Ecological Modelling 222:2722–2732.

Haley, N. J., and E. A. Hoover. 2015. Chronic Wasting Disease of Cervids: Current Knowledge and Future Perspectives. Annual Review of Animal Biosciences 3:305–325.

Haley, N. J., C. K. Mathiason, M. D. Zabel, G. C. Telling, and E. A. Hoover. 2009a. Detection of Sub-Clinical CWD Infection in Conventional Test-Negative Deer

Long after Oral Exposure to Urine and Feces from CWD+ Deer. J. Ma, editor. PLoS ONE 4:e7990.

Haley, N. J., D. M. Seelig, M. D. Zabel, G. C. Telling, and E. A. Hoover. 2009*b*. Detection of CWD Prions in Urine and Saliva of Deer by Transgenic Mouse Bioassay. M. R. Cookson, editor. PLoS ONE 4:e4848.

Hamede, R., J. Bashford, M. Jones, and H. McCallum. 2012. Simulating devil facial tumour disease outbreaks across empirically derived contact networks: Simulating DFTD outbreaks in contact networks. Journal of Applied Ecology 49:447–456.

Hamede, R. K., J. Bashford, H. McCallum, and M. Jones. 2009. Contact networks in a wild Tasmanian devil (*Sarcophilus harrisii*) population: using social network analysis to reveal seasonal variability in social behaviour and its implications for transmission of devil facial tumour disease. Ecology Letters 12:1147–1157.

Hanski, I. 1998. Metapopulation dynamics. Nature 396:41–49.

Hanski, I. 1999. Metapopulation Ecology. Oxford University Press, Oxford, United Kingdoms.

Harris, R. B., G. C. White, C. C. Schwartz, and M. A. Haroldson. 2007. Population Growth of Yellowstone Grizzly Bears: Uncertainty and Future Monitoring. Ursus 18:168–178.

Heilbrun, R. D., N. J. Silvy, M. J. Peterson, and M. E. Tewes. 2006. Estimating bobcat abundance using automatically triggered cameras. Wildlife Society Bulletin 34:69–73.

Heisey, D. M., E. E. Osnas, P. C. Cross, D. O. Joly, J. A. Langenberg, and M. W. Miller. 2010. Linking process to pattern: estimating spatiotemporal dynamics of a wildlife epidemic from cross-sectional data. Ecological Monographs 80:221–240.

Hess, G. R. 1994. Conservation Corridors and Contagious Disease: A Cautionary Note. Conservation Biology 8:256–262.

Hess, G. R. 1996. Disease in Metapopulation Models: Implications for Conservation. Ecology 77:1617–1632.

Hickling, G. J. 2002. Dynamics of bovine tuberculosis in wild white-tailed deer in Michigan. Wildlife Division Report, Michigan Department of Natural Resources, Michigan, USA.

Higley, J. M., and S. Matthews. 2009. Fisher habitat use and population monitoring on the Hoopa Valley Reservation, California Final Report USFWS TWG U-12-NA-1.

Hirth, D. H. 1977. Social behavior of white-tailed deer in relation to habitat. Wildlife Monographs 3–55.

Hodgson, R. G. 1937. Fisher Farming. Fur Trade Journal of Canada 1–104.

Hosseini, P. R., A. A. Dhondt, and A. Dobson. 2004. Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of Mycoplasma gallisepticum in house finches. Proceedings of the Royal Society B: Biological Sciences 271:2569–2577.

Hudson, P. J., A. P. Dobson, and D. Newborn. 1992. Do parasites make prey vulnerable to predation? Red grouse and parasites. Journal of Animal Ecology 61:681–692.

Humphries, M. D., and K. Gurney. 2008. Network "Small-World-Ness": A Quantitative Method for Determining Canonical Network Equivalence. O. Sporns, editor. PLoS ONE 3:e0002051.

Inglis, J. M., R. E. Hood, B. A. Brown, and C. A. DeYoung. 1979. Home Range of White-Tailed Deer in Texas Coastal Prairie Brushland. Journal of Mammalogy 60:377–389.

Ireland, J. M., R. A. Norman, and J. V. Greenman. 2004. The effect of seasonal host birth rates on population dynamics: the importance of resonance. Journal of Theoretical Biology 231:229–238.

Jackson, A., and W. Wunner, editors. 2002. Rabies. Academic Press, London, United Kingdoms.

Jesse, M., P. Ezanno, S. Davis, and J. A. P. Heesterbeek. 2008. A fully coupled, mechanistic model for infectious disease dynamics in a metapopulation: Movement and epidemic duration. Journal of Theoretical Biology 254:331–338.

Ji, W., P. C. L. White, and N. M. Clout. 2005. Contact rates between possums revealed by proximity data loggers. Journal of Applied Ecology 42:595–604.

Johnson, C. J., J. A. Pedersen, R. J. Chappell, D. McKenzie, and J. M. Aiken. 2007. Oral Transmissibility of Prion Disease Is Enhanced by Binding to Soil Particles. PLoS Pathogens 3:e93.

Johnson, C. J., K. E. Phillips, P. T. Schramm, D. McKenzie, J. M. Aiken, and J. A. Pedersen. 2006. Prions Adhere to Soil Minerals and Remain Infectious. PLoS Pathogens 2:e32.

Johnson, D. H. 1980. The Comparison of Usage and Availability Measurements for Evaluating Resource Preference. Ecology 61:65.

Johnson, D. H. 2001. Validating and Evaluating Models. T. M. Shenk and A. B. Franklin, editors. Modeling in Natural Resource Management: Development, Interpretation, and Application. Island Press, Covelo, California, USA.

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.

Jones, J. L., and E. O. Garton. 1994. Selection of successional stages by fishers in north-central Idaho. Pages 377–387 *in* S. W. Buskirk, A. S. Harestad, M. G. Raphael, and R. A. Powell, editors. Martens, Sables, and Fishers: Biology and Conservation. Cornell University Press, Ithaca, New York, USA.

Joyce, M. J. 2013. Space use behavior and multi-scale habitat selection of American marten (Martes americana) in northeastern Minnesota. Masters, University of Minnesota, St. Paul, Minnesota, USA.

Kalinkat, G., F. D. Schneider, C. Digel, C. Guill, B. C. Rall, and U. Brose. 2013. Body masses, functional responses and predator-prey stability. S. Navarrete, editor. Ecology Letters 16:1126–1134.

Källen, A., P. Arcuri, and J. D. Murray. 1985. A simple model for the spatial spread and control of rabies. Journal of theoretical biology 116:377–393.

Kao, R. R. 2003. The impact of local heterogeneity on alternative control strategies for foot-and-mouth disease. Proceedings of the Royal Society B: Biological Sciences 270:2557–2564.

Kapfer, P. M. 2012. Bobcat (Lynx rufus) spatial ecology and harvest in Minnesota. Ph.D., University of Minnesota, St. Paul, Minnesota, USA.

Kawata, Y. 2010. Susceptible-Infective-Recovered Type Epidemiological Models in Wild Animal Management: A Literature Review. Research Bulletin of Obhiro University 31:1–12.

Keeling, M. J. 2001. Dynamics of the 2001 UK Foot and Mouth Epidemic: Stochastic Dispersal in a Heterogeneous Landscape. Science 294:813–817.

Keeling, M. J., and K. T. D. Eames. 2005. Networks and epidemic models. Journal of The Royal Society Interface 2:295–307.

Keeling, M. J., and P. Rohani. 2008. Modeling Infectious Diseases in Humans and Animals. Princeton University Press, Princeton, New Jersey, USA.

Keeling, M. J., M. E. J. Woolhouse, R. M. May, G. Davies, and B. T. Grenfell. 2003. Modelling vaccination strategies against foot-and-mouth disease. Nature 421:136–142.

Keller, S. M., M. Gabriel, K. A. Terio, E. J. Dubovi, E. VanWormer, R. Sweitzer, R. Barret, C. Thompson, K. Purcell, and L. Munson. 2012. Canine Distemper in an Isolated Population of Fishers (Martes pennanti) from California. Journal of Wildlife Diseases 48:1035–1041.

Kelly, G. M. 1977. Fisher (Martes pennanti) Biology in the White Mountain National Forest and Adjacent Areas. Ph.D., University of Massachusetts, Amherst, Massachussets, USA.

Kermack, W. O., and A. G. McKendrick. 1927. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society of London A: Mathematical, Physical, And Engineering Sciences 115:700–721.

Kilpatrick, H. J., and P. W. Rego. 1994. Influence of season, sex, and site availability on fisher (Martes pennanti) rest-site selection in the central hardwood forest. Canadian Journal of Zoology 72:1416–1419.

Kim, T. Y., H. J. Shon, Y. S. Joo, U. K. Mun, K. S. Kang, and Y. S. Lee. 2005. Additional cases of Chronic Wasting Disease in imported deer in Korea. Journal of Veterinary Medical Science 67:753–759.

Kjaer, L. J. 2010. Individual-based modeling of white-tailed deer (Odocoileus virginianus) movements and epizootiology. Ph.D., Southern Illinois University, Carbondale, Illinois, USA.

Korfiatis, N., and M.-A. Sicilia. 2007. Social measures and flexible navigation on online contact networks. Pages 1–6 *in*. Proceedings of the IEEE international conference on fuzzy systems (FUZZ-IEEE). IEEE, London, United Kingdom.

Laake, J. F. 1992. Catch-effort models and their application to elk in Colorado. Ph.D., Colorado State University, Fort Collins, Colorado, USA.

Laguna, M. F., G. Abramson, M. N. Kuperman, J. L. Lanata, and J. A. Monjeau. 2015. Mathematical model of livestock and wildlife: Predation and competition under environmental disturbances. Ecological Modelling 309:110–117.

Langlois, J. P., L. Fahrig, G. Merriam, and H. Artsob. 2001. Landscape structure influences continental distribution of hantavirus in deer mice. Landscape Ecology 16:255–266.

Langrock, R., J. G. C. Hopcraft, P. G. Blackwell, V. Goodall, R. King, M. Niu, T. A. Patterson, M. W. Pedersen, A. Skarin, and R. S. Schick. 2014. Modelling group dynamic animal movement. N. Yoccoz, editor. Methods in Ecology and Evolution 5:190–199.

Lariviere, S., and F. Messier. 1998. Denning ecology of the striped skunk in the Canadian prairies: implications for waterfowl nest predation. Journal of Applied Ecology 35:207–213.

Larsen, D. G., and D. A. Gauthier. 1989. Effects of Capturing Pregnant Moose and Calves on Calf Survivorship. The Journal of Wildlife Management 53:564.

Laseter, B. R. 2004. Socio-spatial characteristics and genetic structure of white-tailed deer in the central Appalachians of West Virginia. Ph.D., University of Georgia, Athens, Georgia, USA. <https://getd.libs.uga.edu/pdfs/laseter_benjamin_r_200412_phd.pdf>. Accessed 9 Mar 2016.

Laurenson, M. K. 1993. Early maternal behavior of wild Cheetahs: implications for captive husbandry. Zoo Biology 12:31–43.

Laurenson, M. K., R. A. Norman, L. Gilbert, H. Reid, and P. J. Hudson. 2003. Identifying Disease Reservoirs in Complex Systems: Mountain Hares as Reservoirs of Ticks and Louping-Ill Virus, Pathogens of Red Grouse. Journal of Animal Ecology 72:177–185.

Leonard, R. D. 1980. The winter activity and movements, winter diet, and breeding ecology of the fisher (Martes pennanti) in Southeastern Manitoba. University of Manitoba.

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

Leslie, P. 1945. On the use of matrices in certain population mathematics. Biometrika 33:183–212.

Leslie, P. 1948. Some further notes on the use of matrices in populations mathematics. Biometrika 35:213–245.

Leu, S. T., P. M. Kappeler, and C. M. Bull. 2010. Refuge sharing network predicts ectoparasite load in a lizard. Behavioral Ecology and Sociobiology 64:1495–1503.

Levins, R. 1969. Some demographic and genetic consequences of environmental heterogeneity for biological control. Bulletin of the Entomological Society of America 15:237–240.

Linnell, J. D., J. E. Swenson, R. Andersen, and B. Barnes. 2000. How vulnerable are denning bears to disturbances? Wildlife Society Bulletin 28:400–413.

Lloyd-Smith, J. O., P. C. Cross, C. J. Briggs, M. Daugherty, W. M. Getz, J. Latto, M. S. Sanchez, A. B. Smith, and A. Swei. 2005. Should we expect population thresholds for wildlife disease? Trends in Ecology & Evolution 20:511–519.

Lloyd-Smith, J. O., W. M. Getz, and H. V. Westerhoff. 2004. Frequency-dependent incidence in models of sexually transmitted diseases: portrayal of pair-based

transmission and effects of illness on contact behaviour. Proceedings of the Royal Society B: Biological Sciences 271:625–634.

Lofroth, E. C., C. Raley, J. M. Highley, R. L. Truex, J. S. Yaeger, J. C. Lewis, P. J. Happe, L. L. Finley, R. H. Naney, R. H. Hale, A. L. Krause, S. A. Livingston, and A. M. Myers. 2010. Conservation of Fishers (Martes pennanti) in Southern-Central British Columbia, Western Washington, Western Oregon, and California. Volume I: Conservation Assessment. USDI Bureau of Land Management, Denver, Colorado, USA.

López, G., M. López-Parra, L. Fernández, C. Martínez-Granados, F. Martínez, M. L. Meli, J. M. Gil-Sánchez, N. Viqueira, M. A. Díaz-Portero, R. Cadenas, H. Lutz, A. Vargas, and M. A. Simón. 2009. Management measures to control a feline leukemia virus outbreak in the endangered Iberian lynx. Animal Conservation 12:173–182.

Lukacs, P. M., K. P. Burnham, and D. R. Anderson. 2010. Model selection bias and Freedman's paradox. Annals of the Institute of Statistical Mathematics 62:117–125.

Lund, U., and C. Agostinelli. 2012. CircStats: Circular Statistics.

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

Manly, B. F. J., L. L. McDonald, D. L. Thomas, T. L. McDonald, and W. P. Erickson. 2002. Resource Selection by Animals: Statistical Design and Analysis for Field Studies. 2nd edition. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Marchinton, R. L., and D. H. Hirth. 1984. Behavior. L. K. Halls, editor. White-Tailed Deer: Ecology and Management. Stackpole Books, Harrisburg, Pennsylvania, USA.

Mathews, N. E. 1989. Social structure, genetic structure, and anti-predator behavior of whitetailed deer in the Central Adirondacks. Ph.D., State University of New York, Syracyse, New York, USA.

Mathews, N. E., and W. F. Porter. 1993. Effect of Social Structure on Genetic Structure of Free-Ranging White-Tailed Deer in the Adirondack Mountains. Journal of Mammalogy 74:33–43.

Mathiason, C. K., S. A. Hays, J. Powers, J. Hayes-Klug, J. Langenberg, S. J. Dahmes, D. A. Osborn, K. V. Miller, R. J. Warren, G. L. Mason, and E. A. Hoover. 2009. Infectious Prions in Pre-Clinical Deer and Transmission of Chronic Wasting Disease Solely by Environmental Exposure. P. Westermark, editor. PLoS ONE 4:e5916.

Mathiason, C. K., J. G. Powers, S. J. Dahmes, D. A. Osborn, K. V. Miller, R. J. Warren, G. L. Mason, S. A. Hays, J. Hayes-Klug, D. M. Seelig, M. A. Wild, L. L. Wolfe, T. R. Spraker, M. W. Miller, C. J. Sigurdson, G. C. Telling, and E. A. Hoover. 2006. Infectious Prions in the Saliva and Blood of Deer with Chronic Wasting Disease. Science 314:133–136.

McCallum, H., N. Barlow, and J. Hone. 2001. How should pathogen transmission be modelled? Trends in Ecology & Evolution 16:295–300.

McNeil, W. H. 1998. Plagues and Peoples. Anchor, New York, USA.

McNulty, S. A., W. F. Porter, N. E. Mathews, and J. A. Hill. 1997. Localized Management for Reducing White-Tailed Deer Populations. Wildlife Society Bulletin 25:265–271.

Meyer, C. B., and W. Thuiller. 2006. Accuracy of resource selection functions across spatial scales. Diversity and Distributions 12:288–297.

Miksch, F., C. Haim, G. Schneckenreither, and F. Breite-necker. 2013. Comparison of differential equations and cellular automata for epidemic simulation. ERK 2013 Proceedings 22:137–140.

Miller, B. F., T. A. Campbell, B. R. Laseter, W. M. Ford, and K. V. Miller. 2010. Test of Localized Management for Reducing Deer Browsing in Forest Regeneration Areas. Journal of Wildlife Management 74:370–378.

Miller, M. W., and M. M. Conner. 2005. Epidemiology of Chronic Wasting Disease in Free-Ranging Mule Deer: Spatial, Temporal, and Demographic Influences on Observed Prevalence Patterns. Journal of Wildlife Diseases 41:275–290.

Miller, M. W., N. T. Hobbs, and S. J. Tavener. 2006. Dynamics of prion disease transmission in mule deer. Ecological Applications 16:2208–2214.

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.

Miller, M. W., M. A. Wild, and E. S. Williams. 1998. Epidemiology of Chronic Wasting Disease in Captive Rocky Mountain Elk. Journal of Wildlife Diseases 34:532–538.

Miller, M. W., E. S. Williams, N. T. Hobbs, and L. L. Wolfe. 2004. Environmental sources of prion transmission in mule deer. Emerging Infectious Diseases 10:1003–6.

Miller, M. W., 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.

Millspaugh, J. J., J. R. Skalski, R. L. Townsend, D. R. Diefenbach, M. S. Boyce, L. P. Hansen, and K. Kammermeyer. 2009. An Evaluation of Sex-Age-Kill (SAK) Model Performance. Journal of Wildlife Management 73:442–451.

Minnesota Department of Natural Resources. 2013. DNR asks bear hunters not to shoot radio-collared bears. News Release.27 August 2013.

Minnesota Department of Natural Resources [MNDNR]. 2016*a*. Deer Hunting Season. <http://dnr.state.mn.us/hunting/deer/index.html>. Accessed 14 Jul 2016.

Minnesota Department of Natural Resources [MNDNR]. 2016*b*. Managing Chronic Wasting Disease. <http://www.dnr.state.mn.us/hunting/deer/cwd/index.html>. Accessed 15 Jul 2016.

Mollison, D., and K. Kuulasmaa. 1985. Spatial epidemic models: theory and simulations. P. J. Bacon, editor. Population Dynamics of Rabies in Wildlife. Academic Press, London, United Kingdoms.

Moore, D. A. 1999. Spatial diffusion of raccoon rabies in Pennsylvania, USA. Preventive veterinary medicine 40:19–32.

Moreno, Y., M. Nekovee, and A. F. Pacheco. 2004. Dynamics of rumor spreading in complex networks. Physical Review E 69:66130.

Munoz-Zanzi, C., M. Mason, C. Encina, M. Gonzalez, and S. Berg. 2014. Household Characteristics Associated with Rodent Presence and Leptospira Infection in Rural and Urban Communities from Southern Chile. American Journal of Tropical Medicine and Hygiene 90:497–506.

Murray, J. D., E. A. Stanley, and D. L. Brown. 1986. On the Spatial Spread of Rabies among Foxes. Proceedings of the Royal Society B: Biological Sciences 229:111–150.

Nathan, R., W. M. Getz, E. Revilla, M. Holyoak, R. Kadmon, D. Saltz, and P. E. Smouse. 2008. A movement ecology paradigm for unifying organismal movement research. Proceedings of the National Academy of Sciences 105:19052–19059.

Nelson, M. E. 1993. Natal Dispersal and Gene Flow in White-Tailed Deer in Northeastern Minnesota. Journal of Mammalogy 74:316–322.

Nelson, M. E. 1998. Development of migratory behavior in northern white-tailed deer. Canadian Journal of Zoology 76:426–432.

Nelson, M. E., and L. D. Mech. 1986. Mortality of White-Tailed Deer in Northeastern Minnesota. The Journal of Wildlife Management 50:691.

Nelson, M. E., and L. D. Mech. 1992. Dispersal in Female White-Tailed Deer. Journal of Mammalogy 73:891–894.

Newman, M. E. 2002. Spread of epidemic disease on networks. Physical review E 66:16128.

Niblett, M. R., S. H. Sweeney, R. L. Church, and K. H. Barber. 2015. Structure of Fisher (Pekania pennanti) Habitat in a Managed Forest in an Interior Northern California Coast Range. Forest Science 61:481–493.

Nixon, C. M., L. P. Hansen, P. A. Brewer, and J. E. Chelsvig. 1991. Ecology of white-tailed deer in an intensively farmed region of Illinois. Wildlife monographs 3–77.

Nixon, C. M., L. P. Hansen, P. A. Brewer, J. E. Chelsvig, J. B. Sullivan, T. L. Esker, R. Koerkenmeier, D. R. Etter, J. Cline, and J. A. Thomas. 1994. Behavior, dispersal, and survival of male white-tailed deer in Illinois. Illinois Natural History Survey Biological Notes 139:1–29.

Northrup, J. M., M. B. Hooten, C. R. Anderson, and G. Wittemyer. 2013. Practical guidance on characterizing availability in resource selection functions under a use–availability design. Ecology 94:1456–1463.

Nussberger, B., and P. Ingold. 2006. Effects of radio-collars on behaviour of alpine chamois Rupicapra rupicapra rupicapra. Wildlife Biology 12:339–343.

Olson, L. E., J. R. Squires, N. J. DeCesare, and J. A. Kolbe. 2011. Den use and activity patterns in female Canada lynx (Lynx canadensis) in the Northern Rocky Mountains. Northwest Science 85:455–462.

O'Rourke, K. I. 2004. Polymorphisms in the prion precursor functional gene but not the pseudogene are associated with susceptibility to chronic wasting disease in white-tailed deer. Journal of General Virology 85:1339–1346.

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.

Oyer, A. M., and W. F. Porter. 2004. Localized management of white-tailed deer in the central Adirondack Mountains, New York. Journal of Wildlife Management 68:257–265.

Ozoga, J. J., L. J. Verme, and C. S. Bienz. 1982. Parturition Behavior and Territoriality in White-Tailed Deer: Impact on Neonatal Mortality. The Journal of Wildlife Management 46:1.

Packer, C., R. D. Holt, P. J. Hudson, K. D. Lafferty, and A. P. Dobson. 2003. Keeping the herds healthy and alert: implications of predator control for infectious disease. Ecology Letters 6:797–802.

Paragi, T. F. 1990. Reproductive biology of female fishers in southcentral Maine. M.S., University of Maine, Orono, Maine, USA.

Paragi, T. F., S. M. Arthur, and W. B. Krohn. 1996. Importance of tree cavities as natal dens for fishers. Northern Journal of Applied Forestry 13:79–83.

Peterson, M. J. 1991. Wildlife Parasitism, Science, and Management Policy. The Journal of Wildlife Management 55:782.

Plowright, R. K., P. Foley, H. E. Field, A. P. Dobson, J. E. Foley, P. Eby, and P. Daszak. 2011. Urban habituation, ecological connectivity and epidemic dampening: the emergence of Hendra virus from flying foxes (Pteropus spp.). Proceedings of the Royal Society B: Biological Sciences 278:3703–3712.

Poggi, S., F. M. Neri, V. Deytieux, A. Bates, W. Otten, C. A. Gilligan, and D. J. Bailey. 2013. Percolation-Based Risk Index for Pathogen Invasion: Application to Soilborne Disease in Propagation Systems. Phytopathology 103:1012–1019.

Porter, W. F., N. E. Mathews, H. B. Underwood, R. W. Sage Jr, and D. F. Behrend. 1991. Social organization in deer: implications for localized management. Environmental Management 15:809–814.

Powell, R. A. 1982. The Fisher. University of Minnesota Press, Minneapolis, Minnesota, USA.

Powell, R. A., and R. D. Leonard. 1983. Sexual Dimorphism and Energy Expenditure for Reproduction in Female Fisher Martes pennanti. Oikos 40:166.

Powell, R. A., and W. J. Zielinski. 1994. The fisher. Pages 38–73 *in*. The Scientific Basis for Conserving Forest Carnivores: American Marten, Fisher, Lynx, and Wolverine in the Western United States. General Technical Report RM-254. USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colorado, USA.

Prestrud, P. 1992. Denning and home-range characteristics of breeding arctic foxes in Svalbard. Canadian Journal of Zoology 70:1276–1283.

Purcell, K. L., A. K. Mazzoni, S. R. Mori, and B. B. Boroski. 2009. Resting structures and resting habitat of fishers in the southern Sierra Nevada, California. Forest Ecology and Management 258:2696–2706.

R Core Team. 2015. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria. <http://www.R-project.org>.

Raine, R. M. 1987. Winter food habits and foraging behaviour of fishers (Martes pennanti) and martens (Martes americana) in southeastern Manitoba. Canadian Journal of Zoology 65:745–747.

Raley, C. M., E. C. Lofroth, R. L. Truex, J. S. Yaeger, and J. M. Higley. 2012. Habitat Ecology of Fishers in Western North America. Pages 231–254 *in* K. B. Aubry, W. J. Zielinski, M. G. Raphael, G. Proulx, and S. W. Buskirk, editors. Biology and Conservation of Martens, Sables, and Fishers: A New Synthesis. Cornell University Press, Ithaca, New York, USA.

Ramsey, D. S. L., D. J. O'brien, M. K. Cosgrove, B. A. Rudolph, A. B. Locher, and S. M. Schmitt. 2014. Forecasting eradication of bovine tuberculosis in Michigan white-tailed deer: Forecasting bTB in White-Tailed Deer. The Journal of Wildlife Management 78:240–254.

Ramsey, D., N. Spencer, P. Caley, M. Efford, K. Hansen, M. Lam, and D. Cooper. 2002. The effects of reducing population density on contact rates between brushtail possums: implications for transmission of bovine tuberculosis. Journal of Applied Ecology 39:806–818.

Rhodes, C. J., H. J. Jensen, and R. M. Anderson. 1997. On the critical behaviour of simple epidemics. Proceedings of the Royal Society B: Biological Sciences 264:1639–1646.

Rhodes, J. R., C. A. McAlpine, D. Lunney, and H. P. Possingham. 2005. A spatially explicit habitat selection model incorporating home range behavior. Ecology 86:1199–1205.

Ricklefs, R. E., and S. M. Fallon. 2002. Diversification and host switching in avian malaria parasites. Proceedings of the Royal Society of London B: Biological Sciences 269:885–892.

Roy, K. D. 1991. Ecology of reintroduced fishers in the Cabinet Mountains of northwest Montana. M.S., University of Montana, Missoula, Montana, USA.

Ruan, S., and D. Xiao. 2004. Stability of steady states and existence of travelling waves in a vector-disease model. Proceedings of the Royal Society of Edinburgh: Section A Mathematics 134:991–1011.

Rushmore, J., D. Caillaud, L. Matamba, R. M. Stumpf, S. P. Borgatti, and S. Altizer. 2013. Social network analysis of wild chimpanzees provides insights for predicting infectious disease risk. D. Tompkins, editor. Journal of Animal Ecology 82:976–986.

Rushton, S. P., P. W. W. Lurz, J. Gurnell, and R. Fuller. 2000. Modelling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the UK? Journal of Applied Ecology 37:997–1012.

Ryder, J. J., M. R. Miller, A. White, R. J. Knell, and M. Boots. 2007. Host-parasite population dynamics under combined frequency- and density-dependent transmission. Oikos 116:2017–2026.

Sayers, B. M., A. J. Ross, P. Saengcharoenrat, and B. G. Mancourian. 1985. Pattern analysis of the case occurrences of fox rabies in Europe. P. J. Bacon, editor. Population Dynamics of Rabies in Wildlife. Academic Press, London, United Kingdoms.

Schaub, M., and F. Abadi. 2011. Integrated population models: a novel analysis framework for deeper insights into population dynamics. Journal of Ornithology 152:227–237.

Schauber, E. M., C. K. Nielsen, L. J. Kjær, C. W. Anderson, and D. J. Storm. 2015. Social affiliation and contact patterns among white-tailed deer in disparate landscapes: implications for disease transmission. Journal of Mammalogy 96:16–28.

Schofield, P. 2002. Spatially Explicit Models of Turelli-Hoffmann Wolbachia Invasive Wave Fronts. Journal of Theoretical Biology 215:121–131.

Schramm, P. T., C. J. Johnson, N. E. Mathews, D. McKenzie, J. M. Aiken, and J. A. Pedersen. 2006. Potential Role of Soil in the Transmission of Prion Disease. Reviews in Mineralogy and Geochemistry 64:135–152.

Silverborg, S. B. 1959. Rate of Decay in Northern Hardwoods Following Artificial Inoculation with Some Common Heartrot Fungi. Forest Science 5:223–228.

Simcharoen, S., A. C. D. Barlow, A. Simcharoen, and J. L. D. Smith. 2008. Home range size and daytime habitat selection of leopards in Huai Kha Khaeng Wildlife Sanctuary, Thailand. Biological Conservation 141:2242–2250.

Skalski, J. R., M. V. Clawson, and J. J. Millspaugh. 2012*a*. Model evaluation in statistical population reconstruction. Wildlife Biology 18:225–234.

Skalski, J. R., J. J. Millspaugh, and M. V. Clawson. 2012*b*. Comparison of Statistical Population Reconstruction Using Full and Pooled Adult Age-Class Data. G. Lythe, editor. PLoS ONE 7:e33910.

Skalski, J. R., J. J. Millspaugh, M. V. Clawson, J. L. Belant, D. R. Etter, B. J. Frawley, and P. D. Friedrich. 2011. Abundance trends of American martens in Michigan based on statistical population reconstruction. The Journal of Wildlife Management 75:1767–1773.

Skalski, J. R., R. L. Townsend, and B. A. Gilbert. 2007. Calibrating Statistical Population Reconstruction Models Using Catch-Effort and Index Data. Journal of Wildlife Management 71:1309–1316.

Sloot, P., S. Ivanov, A. Boukhanovsky, D. van de Vijver, and C. Boucher. 2008. Stochastic simulation of HIV population dynamics through complex network modelling. International Journal of Computer Mathematics 85:1175–1187.

Small, M., and C. K. Tse. 2005. Small world and scale free model of transmission of SARS. International Journal of Bifurcation and Chaos 15:1745–1755.

Smith, D. L., B. Lucey, L. A. Waller, J. E. Childs, and L. A. Real. 2002. Predicting the spatial dynamics of rabies epidemics on heterogeneous landscapes. Proceedings of the National Academy of Sciences 99:3668–3672.

Smith, G. C., and C. L. Cheeseman. 2002. A mathematical model for the control of diseases in wildlife populations: culling, vaccination and fertility control. Ecological Modelling 150:45–53.

Sohn, H. J., J. H. Kim, K. S. Choi, J. J. Nah, Y. S. Joo, Y. H. Jean, S. W. Ahn, O. K. Kim, D. Y. Kim, and A. Balachandran. 2002. A case of chronic wasting disease in an elk imported to Korea from Canada. Journal of Veterinary Medical Science 64:855–858.

Swihart, R. K., Z. Feng, N. A. Slades, D. M. Mason, and T. M. Gehring. 2001. Effects of Habitat Destruction and Resource Supplementation in a Predator-Prey Metapopulation Model. Journal of Theoretical Biology 210:287–303.

Swinton, J., J. Harwood, B. T. Grenfell, and C. A. Gilligan. 1998. Persistence Thresholds for Phocine Distemper Virus Infection in Harbour Seal Phoca vitulina Metapopulations. Journal of Animal Ecology 67:54–68.

Symonds, M. R. E., and A. Moussalli. 2011. A brief guide to model selection, multimodel inference and model averaging in behavioural ecology using Akaike's information criterion. Behavioral Ecology and Sociobiology 65:13–21.

Tamgüney, G., M. W. Miller, L. L. Wolfe, T. M. Sirochman, D. V. Glidden, C. Palmer, A. Lemus, S. J. DeArmond, and S. B. Prusiner. 2009. Asymptomatic deer excrete infectious prions in faeces. Nature 461:529–532.

Taylor, D. M. 1999. Inactivation of prions by physical and chemical means. Journal of Hospital Infection 43 (Supplement):S69–S76.

Taylor, D. M. 2000. Inactivation of Transmissible Degenerative Encephalopathy Agents: A Review. The Veterinary Journal 159:10–17.

Therneau, T. M., and T. Lumley. 2015. A Package for Survival Analysis in S.

Thompson, C., K. L. Purcell, J. Garner, and R. E. Green. 2010. Kings River Fisher Project Progress Report 2007-2010. USDA Forest Service, Pacific Southwest Research Station, Fresno, California, USA.

Thompson, I. D., and P. W. Colgan. 1987. Numerical Responses of Martens to a Food Shortage in Northcentral Ontario. The Journal of Wildlife Management 51:824–835.

Thorne, E. T., and E. S. William. 1988. Disease and Endangered Species: The Black-Footed Ferret as a Recent Example. Conservation Biology 2:66–74.

Tierson, W. C., G. F. Mattfeld, R. W. Sage, and D. F. Behrend. 1985. Seasonal Movements and Home Ranges of White-Tailed Deer in the Adirondacks. The Journal of Wildlife Management 49:760.

Tischendorf, L., H.-H. Thulke, C. Staubach, M. S. Muller, F. Jeltsch, J. Goretzki, T. Selhorst, T. Muller, H. Schluter, and C. Wissel. 1998. Chance and risk of controlling rabies in large-scale and long-term immunized fox populations. Proceedings of the Royal Society B: Biological Sciences 265:839–846.

Tompkins, D. M., A. M. Dunn, M. J. Smith, and S. Telfer. 2011. Wildlife diseases: from individuals to ecosystems: Ecology of wildlife diseases. Journal of Animal Ecology 80:19–38.

Van Baalen, M., and D. A. Rand. 1998. The unit of selection in viscous populations and the evolution of altruism. Journal of Theoretical Biology 193:631–648.

Varadhan, R., and H. W. Borchers. 2011. dfoptim: Derivative-free Optimization. R package version 2011.8-1. <http://cran.r-project.org/package=dfoptim>. Accessed 19 Apr 2015.

Verme, L. J. 1965. Reproduction Studies on Penned White-Tailed Deer. The Journal of Wildlife Management 29:74.

Verme, L. J. 1969. Reproductive Patterns of White-Tailed Deer Related to Nutritional Plane. The Journal of Wildlife Management 33:881.

Verme, L. J. 1983. Sex Ratio Variation in Odocoileus: A Critical Review. The Journal of Wildlife Management 47:573.

Verme, L. J. 1989. Maternal Investment in White-Tailed Deer. Journal of Mammalogy 70:438–442.

Volz, E., and L. A. Meyers. 2007. Susceptible-infected-recovered epidemics in dynamic contact networks. Proceedings of the Royal Society B: Biological Sciences 274:2925–2934.

Volz, E., and L. A. Meyers. 2009. Epidemic thresholds in dynamic contact networks. Journal of The Royal Society Interface 6:233–241.

Vreeland, J. K., D. R. Diefenbach, and B. D. Wallingford. 2004. Survival rates, mortality causes, and habitats of Pennsylvania white-tailed deer fawns. Wildlife Society Bulletin 32:542–553.

Wasserberg, G., E. E. Osnas, R. E. Rolley, and M. D. Samuel. 2009. Host culling as an adaptive management tool for chronic wasting disease in white-tailed deer: a modelling study. Journal of Applied Ecology 46:457–466.

Webb, C. T., C. P. Brooks, K. L. Gage, and M. F. Antolin. 2006. Classic flea-borne transmission does not drive plague epizootics in prairie dogs. Proceedings of the National Academy of Sciences 103:6236–6241.

Weckwerth, R. P., and V. D. Hawley. 1962. Marten Food Habits and Population Fluctuations in Montana. The Journal of Wildlife Management 26:55–71.

Weir, R. D., and F. B. Corbould. 2008. Ecology of fishers in the sub-boreal forests of north-central British Columbia, final report. Peace/Williston Fish and Wildlife Compensation Program, Prince George, British Columbia, Canada, Report 315:1–178.

Weir, R. D., and A. S. Harestad. 2003. Scale-dependent habitat selectivity by fishers in South-Central British Columbia. The Journal of Wildlife Management 67:73–82.

Weir, R. D., A. S. Harestad, and R. C. Wright. 2005. Winter Diet of Fishers in British Columbia. Northwestern Naturalist 86:12–19.

Weir, R. D., M. Phinney, and E. C. Lofroth. 2012. Big, sick, and rotting: Why tree size, damage, and decay are important to fisher reproductive habitat. Forest Ecology and Management 265:230–240.

White, G. C., and R. A. Garrott. 1990. Analysis of Wildlife Radio-Rracking Data. Academic Press, San Diego, California, USA.

White, P. C. L., J. A. Brown, and S. Harris. 1993. Badgers (Meles meles), Cattle and Bovine Tuberculosis (Mycobacterium bovis): A Hypothesis to Explain the Influence of Habitat on the Risk of Disease Transmission in Southwest England. Proceedings of the Royal Society B: Biological Sciences 253:277–284.

White, P. C. L., and S. Harris. 1995. Bovine Tuberculosis in Badger (Meles meles) Populations in Southwest England: The Use of a Spatial Stochastic Simulation Model to Understand the Dynamics of the Disease. Philosophical Transactions of the Royal Society B: Biological Sciences 349:391–413.

Williams, B. W., D. R. Etter, D. W. Linden, K. F. Millenbah, S. R. Winterstein, and K. T. Scribner. 2009. Noninvasive Hair Sampling and Genetic Tagging of Co-Distributed Fishers and American Martens. Journal of Wildlife Management 73:26–34.

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

Williams, E. S., and M. W. Miller. 2002. Chronic wasting disease in deer and elk in North America., 21, 305–316. Revue Scientifique et Technique de l Office International des Epizooties 21:305–316.

Williams, E. S., M. W. Miller, T. J. Kreeger, R. H. Kahn, and E. T. Thorne. 2002. Chronic Wasting Disease of Deer and Elk: A Review with Recommendations for Management. The Journal of Wildlife Management 66:551.

Williams, E. S., and S. Young. 1980. Chronic Wasting Disease of Captive Mule Deer: A Spongiform Encephalopathy. Journal of Wildlife Diseases 16:89–98.

Wood, K. A., R. A. Stillman, F. Daunt, and M. T. O'Hare. 2013. Evaluating the Effects of Population Management on a Herbivore Grazing Conflict. M. Krkosek, editor. PLoS ONE 8:e56287.

Xie, F., and R. D. Horan. 2009. Disease and behavioral dynamics for Brucellosis control in elk and cattle in the Greater Yellowstone Area. Journal of Agriculture and Resource Economics 34:11–33.

Yachi, S., K. Kawasaki, N. Shigesada, and E. Teramoto. 1989. Spatial patterns of propogating waves of fox rabies. Forma 4:3–12.

Yip, P. S. F., R. Watson, and Q. Chen. 2007. Estimation of vaccine efficacy and the vaccination threshold. Statistics in Medicine 26:4475–4488.

Zhao, F., R. A. Sweitzer, Q. Guo, and M. Kelly. 2012. Characterizing habitats associated with fisher den structures in the Southern Sierra Nevada, California using discrete return lidar. Forest Ecology and Management 280:112–119.

Zielinski, W. J., J. R. Dunk, J. S. Yaeger, and D. W. LaPlante. 2010. Developing and testing a landscape-scale habitat suitability model for fisher (Martes pennanti) in forests of interior northern California. Forest Ecology and Management 260:1579–1591.

Zielinski, W. J., K. M. Slauson, C. R. Carroll, C. J. Kent, and D. G. Kudrna. 2001. Status of American martens in coastal forests of the Pacific states. Journal of Mammalogy 82:478–490.

Zielinski, W. J., R. L. Truex, J. R. Dunk, and T. Gaman. 2006. Using forest inventory data to assess fisher resting habitat suitability in California. Ecological Applications 16:1010–1025.

Zielinski, W. J., R. L. Truex, G. A. Schmidt, F. V. Schlexer, K. N. Schmidt, and R. H. Barrett. 2004. Resting habitat selection by fishers in California. Journal of Wildlife Management 68:475–492.

Disease prevalence ten years after disease detection in populations of white-tailed deer with a density of 6 deer/km^2 under a range of different variable combinations (i.e., β_d, β_i, ε, and prion half-life). Management strategies include no culling (N), localized management at scales of 1, 3, and 5km^2 (L, M, and H, respectively), and widespread, non-selective culling (W). Left panel corresponds to the variable combination discussed in chapter 5.

Figure A1. Impacts of increasing prion half-life while holding β_d constant on the effectiveness of different control strategies in a low-density population.

Figure A2. Impacts of increasing β_d while holding β_i constant on the effectiveness of different control strategies in a low-density population.
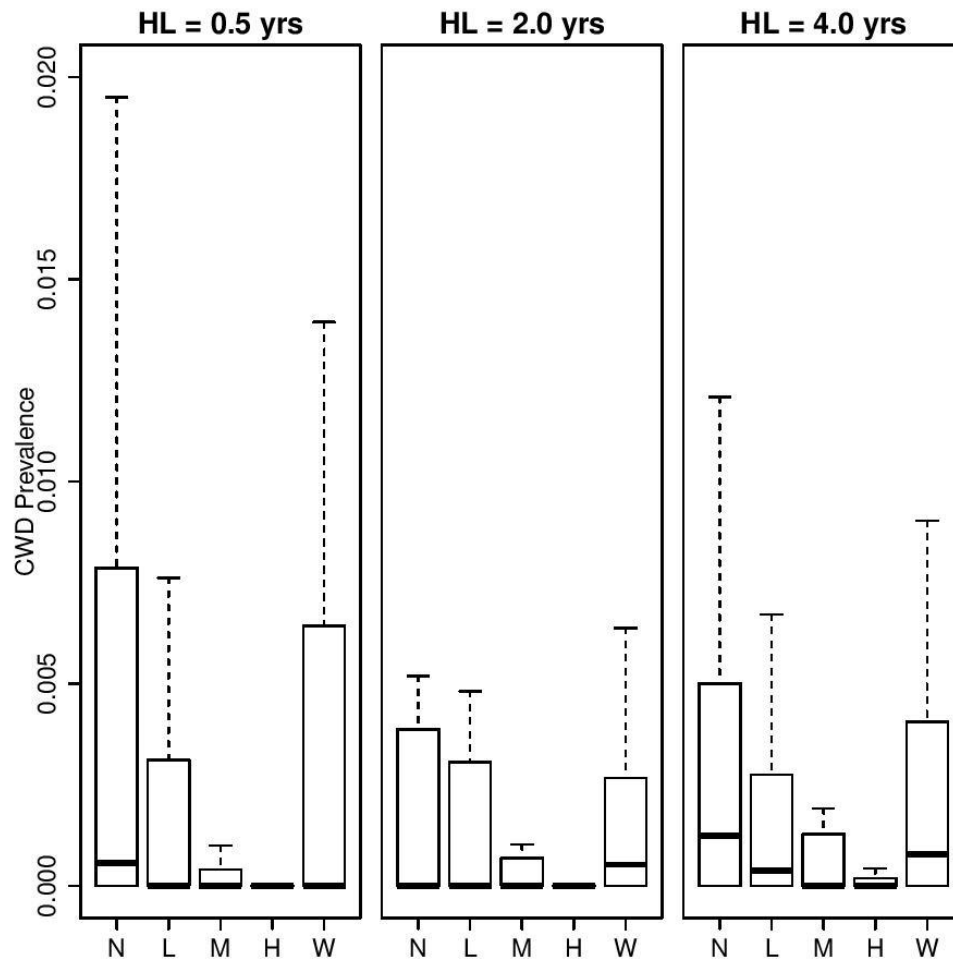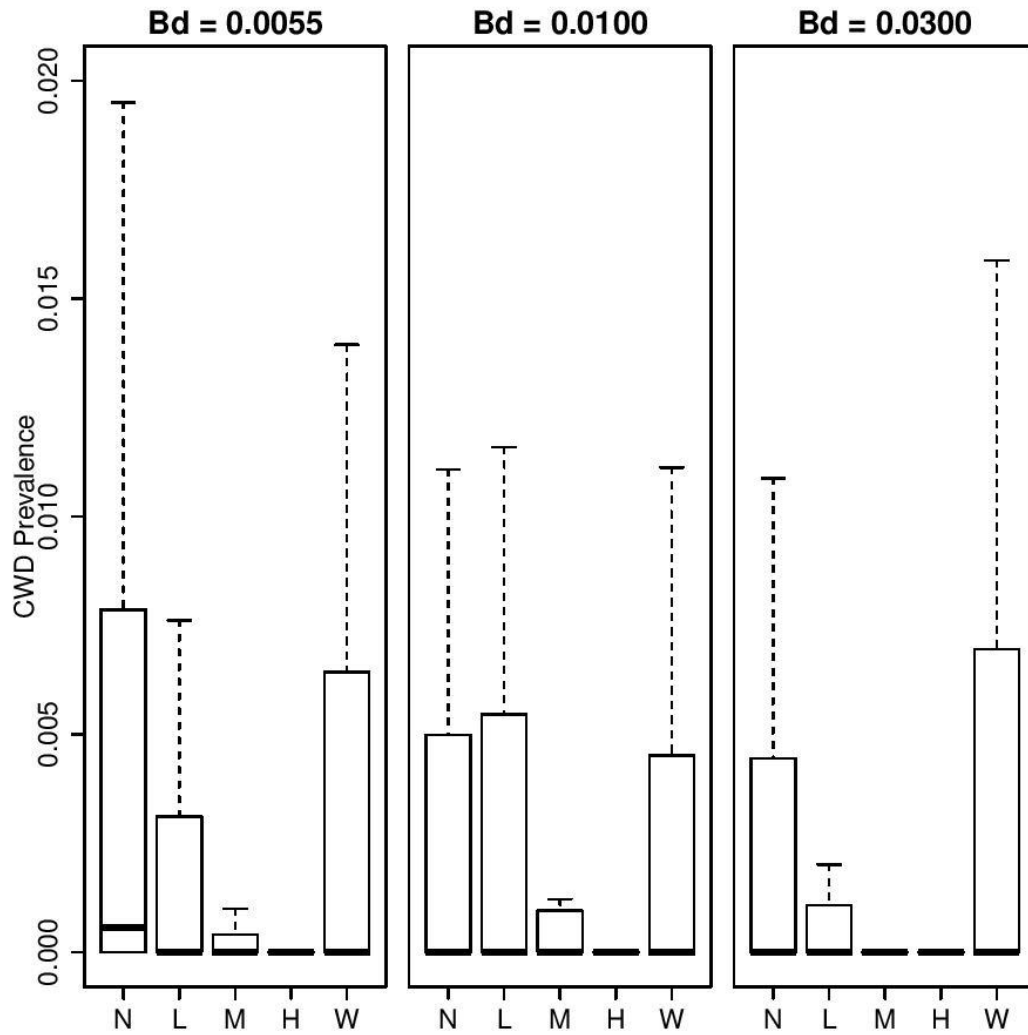
Figure A3. Impacts of increasing β_i while holding prion half-life constant on the

effectiveness of different control strategies in a low-density population.
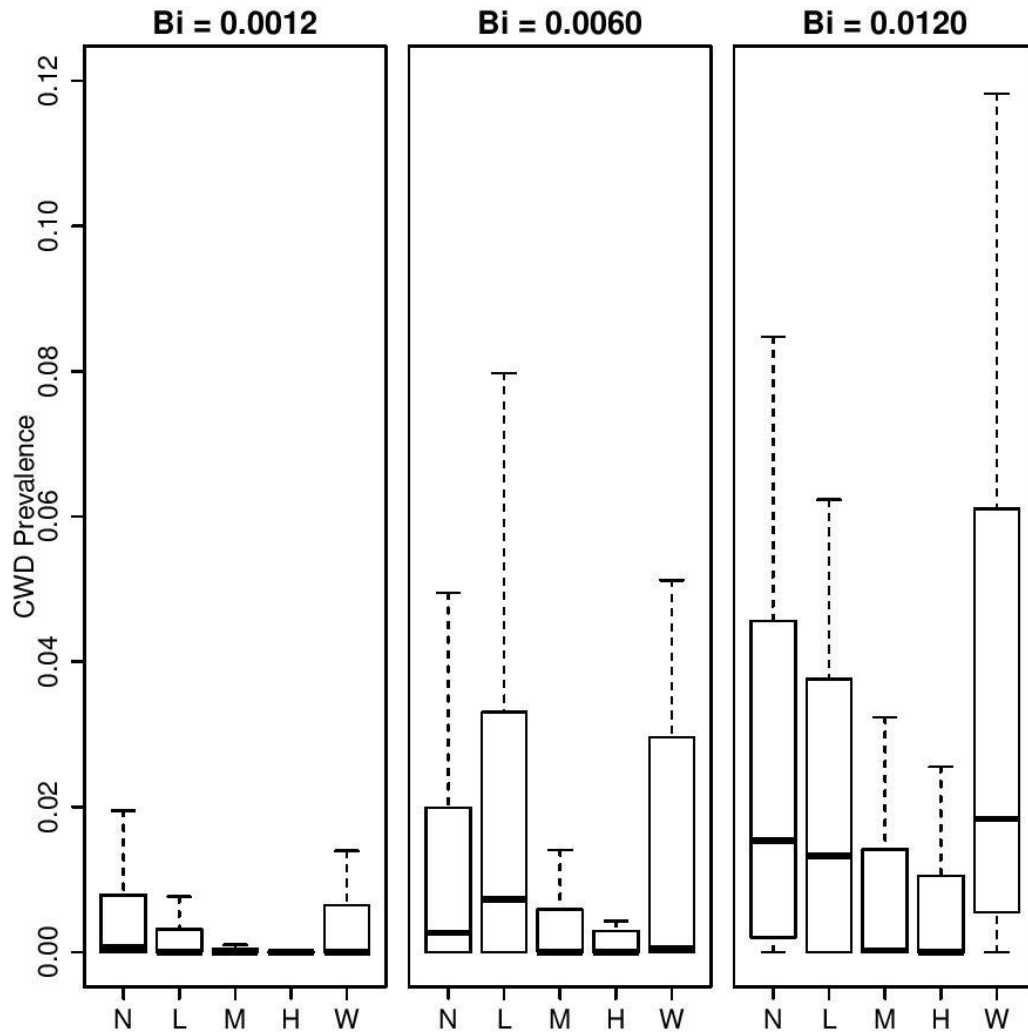
Figure A4. Impacts of changing ε while holding β_d, β_i, and prion half-life constant on the

effectiveness of different control strategies in a low-density population.
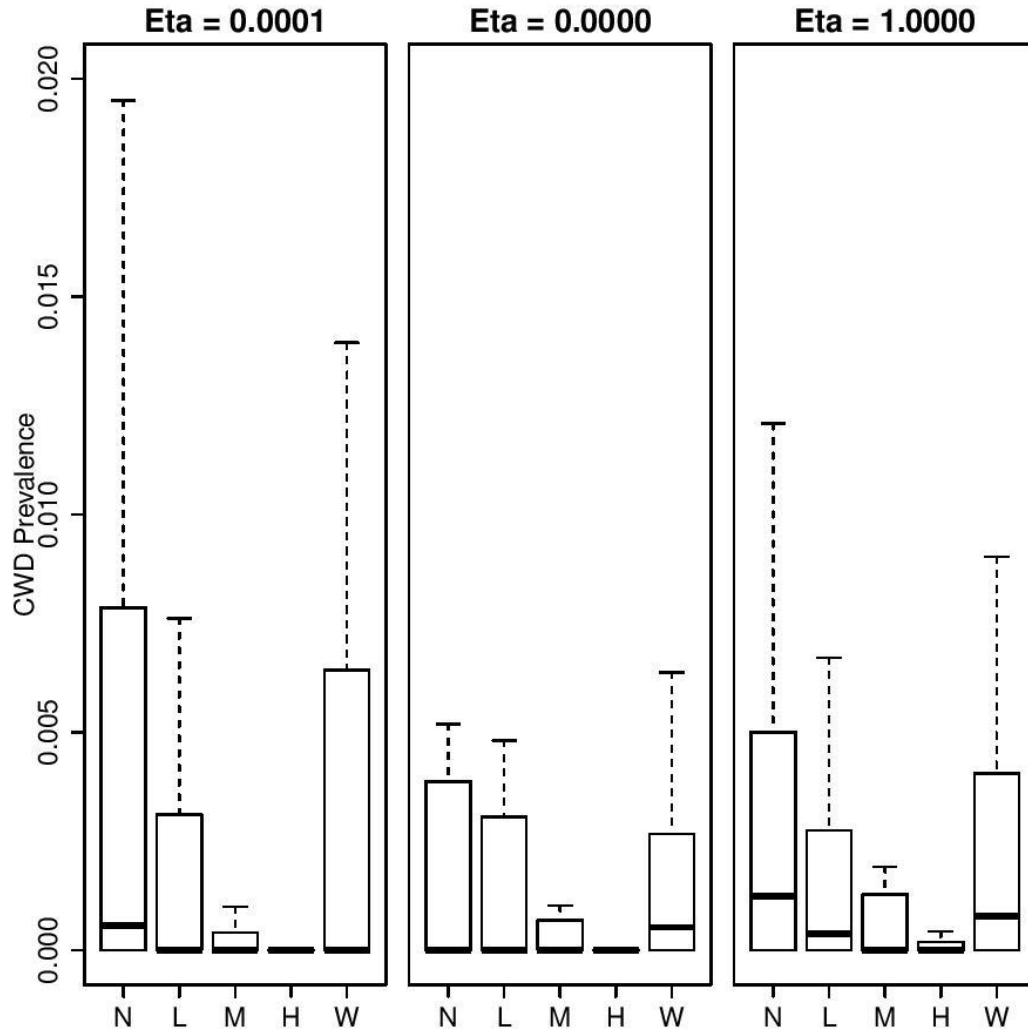
Table A1. Variable combinations used in Figures A1–4 to examine the robustness of model results to changes in variable values.

	Parameter				
Prion half-life	β_d	β_i	ε	Figure	Panel
0.5	5.5e-3	1.2e-3	0.0001	A1–4	Left
2	5.5e-3	3.1e-4	0.0001	A1	Middle
4	5.5e-3	2.1e-4	0.0001	A1	Right
0.4	1.0e-2	1.2e-3	0.0001	A2	Middle
0.25	3.0e-2	1.2e-3	0.0001	A2	Right
0.5	4.0e-3	6.0e-3	0.0001	A3	Middle
0.5	2.0e-3	1.2e-2	0.0001	A3	Right
0.5	5.5e-3	1.2e-3	0.0000	A4	Middle
0.5	5.5e-3	1.2e-3	1.0000	A4	Right

List of values used to construct Figures 4.2, 4.3, and 5.1

Table A2. List of boxplot summaries used in Figure 4.2 to compare population reconstruction bias as a function of the type and amount of radio-telemetry data incorporated.

Min	Q1	Median	Q2	Max	Type of radio-telemetry data used	Number of animals	Number of years
-0.543	-0.125	0.093	0.155	0.573	None	0	0
-0.381	-0.079	0.032	0.123	0.426	Survival	11	7
-0.305	-0.065	0.022	0.095	0.335	Survival	23	7
-0.259	-0.055	0.016	0.081	0.285	Survival	34	7
-0.470	-0.108	0.035	0.134	0.496	Harvest	11	7
-0.398	-0.090	0.023	0.116	0.425	Harvest	23	7
-0.341	-0.077	0.017	0.098	0.361	Harvest	34	7
-0.335	-0.075	0.020	0.099	0.359	Both	11	7
-0.256	-0.058	0.014	0.073	0.271	Both	23	7
-0.214	-0.049	0.009	0.061	0.227	Both	34	7
-0.395	-0.082	0.037	0.126	0.438	Survival	34	2
-0.315	-0.066	0.023	0.100	0.349	Survival	34	4
-0.281	-0.060	0.019	0.088	0.310	Survival	34	6

-0.267	-0.059	0.018	0.085	0.296	Survival	34	7
-0.483	-0.112	0.038	0.136	0.516	Harvest	34	2
-0.414	-0.094	0.025	0.119	0.440	Harvest	34	4
-0.368	-0.082	0.019	0.108	0.393	Harvest	34	6
-0.348	-0.078	0.018	0.102	0.372	Harvest	34	7
-0.350	-0.078	0.022	0.103	0.375	Both	34	2
-0.270	-0.062	0.014	0.076	0.284	Both	34	4
-0.232	-0.053	0.012	0.067	0.246	Both	34	6
-0.223	-0.051	0.010	0.064	0.236	Both	34	7

Table A3. List of abundance estimates used in Figure 4.3 to compare trends in abundance of and recruitment in Minnesota fishers based on different types and amounts of radio-telemetry data incorporated into statistical population reconstruction.

			Year						
2007	**2008**	**2009**	**2010**	**2011**	**2012**	**2013**	**2014**	**Panel**	**Line**
10188	7726	10300	11270	10877	8161	7879	9610	(a)	Solid
9057	6898	9037	9835	9540	7092	6837	8298	(a)	Dashed
11314	8591	11572	12773	12286	9365	9057	11014	(a)	Dotted
11843	10864	11152	11348	11270	9874	9503	9875	(a)	Dotted
2550	2353	6283	5516	4394	2392	3364	4930	(b)	Solid
2014	1898	5152	4405	3567	1909	2697	3916	(b)	Dotted
3090	2806	7408	6638	5210	2870	4025	5954	(b)	Dotted
24689	19261	25275	28117	26570	20183	20027	24052	(c)	Dotted
25393	19601	25959	28949	27191	20728	20426	24807	(c)	Dot-Dashed
9056	6722	9291	9813	9807	7160	6911	8261	(c)	Dashed
10187	7726	10300	11270	10877	8161	7879	9610	(c)	Solid
18682	14343	19040	21142	19982	15164	14875	18084	(d)	Dotted
11106	8443	11246	12339	11862	8921	8639	10529	(d)	Dot-Dashed
10308	7819	10424	11410	11006	8260	7978	9730	(d)	Dashed
10188	7726	10300	11270	10877	8161	7879	9610	(d)	Solid

Table A4. List of boxplot summaries used in Figure 5.1 to compare disease prevalence ten years after disease detection in populations of white-tailed deer under a range of control strategies and population densities.

Min	Q1	Median	Q2	Max	Strategy	Density
0.000	0.000	0.001	0.008	0.020	N	Low
0.000	0.000	0.000	0.003	0.008	L	Low
0.000	0.000	0.000	0.000	0.001	M	Low
0.000	0.000	0.000	0.000	0.000	H	Low
0.000	0.000	0.000	0.007	0.014	W	Low
0.000	0.008	0.023	0.054	0.122	N	Medium
0.000	0.005	0.015	0.034	0.077	L	Medium
0.000	0.000	0.008	0.020	0.047	M	Medium
0.000	0.000	0.003	0.012	0.028	H	Medium
0.000	0.004	0.014	0.031	0.072	W	Medium
0.000	0.080	0.119	0.174	0.301	N	High
0.000	0.055	0.090	0.132	0.244	L	High
0.000	0.029	0.056	0.088	0.173	M	High
0.000	0.014	0.040	0.064	0.137	H	High
0.000	0.006	0.012	0.027	0.056	W	High