MANAGEMENT OF FREE-RANGING WHITE-TAILED DEER WITH CHRONIC WASTING DISEASE IN MICHIGAN

By

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ABSTRACT

Chronic wasting disease (CWD) continues to spread among wild populations of cervids across North America and where endemic can result in long-term population declines. With no treatment, vaccine, nor environmental decontamination methods forthcoming, identifying and implementing effective management strategies to limit its growth and spread is critical. I focus on three challenges associated with managing CWD in my dissertation. First, I expand our knowledge of CWD dynamics in deer, particularly on indirect disease transmission. Second, I provide insights into heterogeneity observed in disease dynamics in response to local population and landscape conditions. Last, I describe sources of uncertainty surrounding implementation of CWD management that affect our ability to: 1) implement management effectively, 2) measure management success and, 3) learn from past experiences.

A contribution of this work is an individual-based model (IBM) that depicts CWD dynamics in free-ranging deer. The model is spatio-temporal and can: 1) incorporate local conditions and individual deer variation into estimates of disease dynamics, 2) simulate and assess management scenarios, and 3) integrate and account for aspects of uncertainty, such as with management implementation. In Chapter 1, I present the model and explore how interactions among deer and interactions with their environment affect CWD dynamics. Chapter 2 focuses on construction and evaluation of localized and fine-scale deer management strategies for CWD using this model. In the third chapter, I incorporate sources of implementation uncertainty into management scenarios to assess impacts on management outcomes. I worked closely with Michigan Department of Natural Resources, U.S. Department of Agriculture, and U.S. Geological Survey to develop this model and construct realistic management scenarios.

The IBM reproduced individual deer, population, and disease processes observed in

Midwestern deer populations. A sensitivity analysis revealed that fall migration, disease mortality, and fawn mortality rates had the largest impact on CWD prevalence 20 years post initial introduction of CWD. Prion half-life, prion shedding rate, and deer group membership had minimal influence. Chapter 2 model results suggest that CWD outbreaks are more likely to persist in exurban areas compared to suburban landscapes. Additionally, CWD prevalence rate increased faster in exurban areas. Initial density of the affected deer population did not influence long-term CWD prevalence nor the probability of CWD persisting in the population. Localized deer removal by means of ring culling and removing deer closer to the disease origin were the most effective management methods for reducing CWD persistence and long-term prevalence rate. Current agency removal goals of 20–30% of the population were not enough to reduce disease persistence below 10% except for in one of the 30 management scenarios. The level of deer removal required (>60%) to reduce persistence to <10% may not be feasible for wildlife agencies nor socially acceptable by the public. In Chapter 3, model results suggest that partial controllability, i.e., discrepancy between management decisions and their realization, affects success of deer removal efforts to control CWD dynamics. The IBM revealed thresholds for deer removal and land access rates required to influence persistence and long-term prevalence. Land access and deer removal rates as low as 20% could reduce long-term prevalence of CWD. Conversely, at least one of these rates had to be ≥70% and the other ≥30% to reduce probability of CWD persisting in the population.

I conclude this dissertation with a discussion of my results, management implications, and future directions for the IBM. By inputting region-specific data and developing management scenarios relevant to management objectives, wildlife managers can use this IBM to make more informed decisions about managing cervid populations for CWD.

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INTRODUCTION

Chronic wasting disease (CWD) is a fatal prion disease transmitted among members of the *Cervidae* family (Williams and Young 1992). The disease has occurred in free-ranging cervids for over four decades and continues to grow and spread across North America (31 U.S. states and four Canadian provinces; USGS 2023), and occurring internationally in Norway, Sweden, Finland, and South Korea (Sohn et al. 2002, EFSA BIOHAZ Panel 2023). In the western US where the disease is endemic, CWD is contributing to >10% population declines in mule deer (*Odocoileus hemionus hemionus*) and elk (*Cervus elaphus nelsoni*) (Edmunds et al. 2016). No known individual treatment or cure for CWD exists (Smith et al. 2011, Xu et al. 2013), and vaccine development has proven difficult because the misfolded CWD prion resembles its native form, thus not triggering an immune response within the host (Goni et al. 2015). Occurrence of CWD threatens the foundation of wildlife conservation in North America by discouraging hunters from harvesting deer in affected regions, thereby reducing the number of hunting licenses, deer tags, and hunting equipment purchased and, ultimately, the amount of Pitman-Robertson Act funds generated and distributed to each U. S. state (Needham et al. 2004, Vaske and Lyon 2011). Chronic wasting disease is referred to as "the greatest contemporary threat to free-ranging deer herds" by the Association of Fish and Wildlife Agencies (Gillin and Mawdsley 2018).

Chronic wasting disease is transmitted directly through animal to animal contact and indirectly through animal contact with contaminated environments (Miller et al. 2004). Environmental contamination occurs as infected individuals begin to shed prions as early as three months post-infection (Henderson et al. 2015). Infectious deer shed prions through bodily fluids and excreta such as blood, saliva, urine, feces, and antler velvet (Miller et al. 2004,

Mathiason et al. 2006, Safar et al. 2008, Haley et al. 2009). Prions are nonobligatory pathogens that remain infectious outside the host and are resilient to many regular treatment methods for infectious agents (Colby and Prusiner 2011). Consequently, researchers have made slow progress toward finding an effective environmental decontamination technique.

Cervids become infectious before they show outward signs of CWD (e.g., emaciation, disorientation, fearlessness, paralysis; Henderson et al. 2015). Therefore, infected deer should be removed from the environment before clinical disease signs are expressed to prevent direct and indirect transmission (Gillin and Mawdsley 2018). With no vaccine nor treatment for this disease available, managers have limited options for preventing and controlling CWD in free-ranging deer populations.

Past CWD management approaches have focused largely on implementing regulations that prevent artificial congregation of deer and transport of live deer and carcasses (Thompson et al. 2023). Additionally, managers have removed deer in infected and surrounding populations with the goal of reducing deer density (Thompson et al. 2023). It is not clear whether transmission of CWD among hosts is frequency- and density-dependent of falls in between these two transmission paradigms; however, it had been suggested that fewer disease transmission events are expected to occur where fewer deer are present (Schauber and Wolf 2003, Jennelle et al. 2014). Past deer removal attempts have generally failed to eradicate CWD in endemic regions. Eradication has been successful at eliminating CWD only in free-ranging deer in a four-county (6,200 km^2) area surrounding an infected captive elk facility in southeastern Minnesota and a 4,113 km^2 area surrounding an infected captive white-tailed deer facility in New York (Miller and Fischer 2016). In both of those states, CWD was detected early, and wildlife managers were able to perform localized culling soon after detection (CWDA 2008, Miller and

Fischer 2016). Given the general failure of eliminating CWD in endemic populations, social nature of deer, and expansive range of cervids throughout North America, some wildlife managers now consider eradication of endemic CWD an unattainable goal (Miller and Fischer 2016).

Like eradication, previous management by agencies to control endemic CWD (e.g., reduce prevalence or geographic spread of CWD) have had limited success. Past strategies have focused on deer population reduction to reduce prevalence and minimize geographic spread of CWD (Uehlinger et al. 2016, Thompson et al. 2023). However, deer population reduction had no effect on CWD prevalence rates in free-ranging deer populations in Colorado, Wyoming, West Virginia, or Wisconsin (Conner et al. 2007, DeVivo et al. 2017, WDNR 2018, WV DNR 2018 but see Conner et al. 2021). Conversely, slower growth in CWD prevalence, but not geographic spread, in Illinois may be attributed to a localized removal program conducted annually by the Illinois Department of Natural Resources (Mateus-Pinilla et al. 2013, Manjerovic et al. 2014).

Given these studies are largely observational, there is uncertainty regarding effective management strategies for controlling CWD in free-ranging deer populations. Reasons for absence in knowledge on effective management strategies stem from the expense, time, and risk of applying rigorous experimentation on live deer populations. Additionally, although mathematical modeling has a role in evaluating CWD control strategies without field experimentation, accurately projecting CWD dynamics within a modeling environment has proven difficult because of the many uncertainties associated with CWD dynamics (e.g., transmission rate and the basic reproductive number, R_o for the disease (Dietz 1993)). Furthermore, past modeling studies that evaluated management strategies for CWD did not account for implementation uncertainty, the idea that management strategies are rarely

implemented with 100% success, which limits relevance of model results for wildlife managers.

The state of Michigan detected CWD in its free-ranging white-tailed deer (*Odocoileus virginianus*) population in 2015. Not only does CWD cause long-term population declines for deer (Edmunds et al. 2016, DeVivo et al. 2017), it can also discourage hunters from harvesting deer in affected areas (Needham et al. 2004, Vaske and Lyon 2011). These consequences can lead to significant declines in number of deer licenses purchased by hunters each year. Michigan has nearly 590,000 deer hunters and annual revenue from license purchases supports 57% of state-based wildlife and fisheries programs (Gwizdz 2018, Frawley 2021). Therefore, it is critical for wildlife managers to prevent further spread of CWD in Michigan to protect the hunting culture and to conserve species in addition to white-tailed deer.

My goal for this dissertation was to develop a comprehensive modeling tool that provides wildlife agencies and researchers the ability to assess CWD dynamics in free-ranging cervid populations and estimate impacts of various management actions on those disease dynamics. I targeted my research and results for practical use by wildlife agencies having to manage CWD in free-ranging deer herds while also having to consider social and financial constraints and implementation uncertainty.

CHAPTER 1 AN INDIVIDUAL-BASED MODEL FOR DIRECT AND INDIRECT TRANSMISSION OF CHRONIC WASTING DISEASE IN FREE-RANGING WHITE-TAILED DEER

1.1 Introduction

Chronic wasting disease (CWD) is an emerging infectious disease spreading among cervid (i.e., species of the *Cervidae* family) populations across North America (Williams and Young 1992, USGS 2023). This disease has infected or currently infects cervids across 31 states in the United States, four provinces in Canada, Finland, Norway, Sweden, and South Korea (Sohn et al. 2002, BIOHAZ Panel 2023, USGS 2023). Eradicating CWD seems unlikely, as researchers have made little advancement toward finding a vaccine or treatment (Colby and Prusiner 2011, Xu et al. 2013, Goni et al. 2015, Napper and Schatzl 2023). Similarly, field and research testing of prevention strategies have generated minimal successes towards limiting CWD introductions to new free-ranging cervid populations, and management strategies for controlling the disease once an outbreak occurs have generally been ineffective (Uehlinger et al. 2016).

Mathematical modeling is a useful tool for studying infectious diseases and their management because methods such as field or clinical studies are often cost-prohibitive, put scientists and the public at risk of pathogen exposure, and potentially cause localized deer population declines where the disease persists or prevalence increases. While mathematical models are useful, understanding and predicting wildlife disease dynamics in free-ranging cervid populations is often challenging because disease systems are complex and not fully understood. This challenge is apparent for CWD. Researchers and wildlife agencies have monitored and studied the CWD disease system for four decades (Gross and Miller 2001, Samuel and Storm

2016, Uehlinger et al. 2016), yet few advancements in quantitative modeling have occurred until recently. Uncertainty associated with pathogen parameters (e.g., indirect and direct transmission rates, R_0, and others; Uehlinger et al. 2016) and high variation associated with the host and environment (Ketz et al. 2019) pose challenges to CWD modeling.

Most (6 of 9) field-based studies that evaluated management strategies for CWD were based on mathematical modeling that predicted management effects on CWD dynamics (Uehlinger et al. 2016). No single optimal management strategy emerged from the modeling nor field studies, as findings varied considerably given study-specific contexts. Additionally, the studies acknowledged limitations to their work. For example, 5 of 6 modeling studies were unsure of how to accurately parameterize CWD transmission mode as a model variable (Uehlinger et al. 2016).

Improved models for CWD dynamics are needed that can readily incorporate individual heterogeneity in deer behavior and local deer, management, and landscape information. Deer behavior highly depends on habitat characteristics of occupied landscapes. For example, in the Midwest US, higher birth and death rates are observed annually compared to other US regions, resulting in a higher turnover rate for cervid populations inhabiting this region (Nixon et al. 1991, Hewitt 2011). Additionally, year-round food abundance and landscapes with fragmented habitat associated with agriculturally dominated areas affect home-range sizes, movement rates, and contact rates among deer (Hewitt 2011). Furthermore, differences in CWD and deer population outcomes due to variation in deer and disease management practices among local jurisdictions are expected, furthering the need to account for local conditions when modeling CWD in free-ranging cervid populations.

Individual-based models (IBMs) are useful when working with complex systems that

contain high uncertainty and variation, such as the deer-CWD system (Bonabeau 2002). In addition, IBMs are becoming increasingly applied for epidemiology because they incorporate population processes including individual and group movements, social behaviors, and local interactions with the environment, all of which affect transmission of infectious pathogens (Ramsey and Efford 2010, Ramsey et al. 2014, Merler et al. 2015). Researchers can incorporate real landscapes into IBMs, typically by coupling the IBM with geographic information systems software, which allows for exploration of disease transmission over space and the effects of landscape characteristics on the disease system (Perez and Dragicevic 2009). An individual-based framework is more amenable to incorporating individual variation, complex social structure among hosts, and spatially explicit pathogen transmission; properties of CWD transmission that have not been included in past models (Uehlinger et al. 2016; Belsare and Stewart 2020). Further, long-lasting yet unverified assumptions about the disease system, such as primary transmission pathways, can be tested through simulation (Kelly et al. 2013). I introduce a stochastic, spatially explicit epidemiological IBM of CWD within a free-ranging white-tailed deer (*Odocoileus virginianus*) population in Michigan to explore and predict spatiotemporal dynamics of CWD. Specifically, my IBM integrates deer population dynamics and individual deer movements and behaviors to produce the complex dynamics associated with free-ranging white-tailed deer populations and CWD.

1.2 Methods

I developed a stochastic, spatially explicit IBM of a free-ranging white-tailed deer population in Michigan, USA, to simulate deer population dynamics. I then introduced CWD into the deer population using a Susceptible-Exposed-Infectious-Dead (SEID) epidemiological framework (Anderson and May 1991). I parameterized the IBM with values reported in the

CWD literature and by wildlife agencies to assess disease dynamics in the modeled deer population through time and space. The model provides a bottom-up approach to understanding CWD dynamics because it incorporates variation in individual deer behaviors and movements. For example, model users can account for heterogeneous epidemiologic processes, such as super-spreaders in a population, and avoid homogenous spatial mixing among members (Anderson and May 1991, Lloyd-Smith et al. 2005, Jankowski et al. 2013).

My IBM includes three main model components (Appendix A): a deer population model, a deer movement model, and an epidemiological model. Individual deer interact with each other and with the environment in the model. The spatial extent of interactions occurs within a landscape, which may range in size from a Public Land Survey System section (i.e., 2.6 km^2) to a deer management unit (100-1000s km^2) or U.S. state. The model also includes an environmental prion (i.e., the infectious agent that causes CWD) spatial layer. I initiate an outbreak of CWD by introducing ≥1 infected deer into the modeled population, which may then spread within the population directly (via direct contact between deer) and indirectly (through deposition of prions into the landscape by infectious deer). I developed each module of my IBM in Python (v 3.7) and corresponding analyses in program R (v 3.5.2). The movement module was adapted from a deer movement model developed by Butts et al. (2022), which applied an exploratory data analysis approach to develop a Langevin model that describes movement patterns trained on GPS location data obtained from white-tailed deer inhabiting central New York. I use a standardized and familiar protocol, the Overview, Design concepts, and Details (ODD) protocol, to describe my IBM (Fig. 1.1; Grimm et al. 2006, 2010, 2020). The purpose of my IBM is provided in the introduction.

1.2.1 Overview – Entities, state variables, and scales

In an IBM, state variables influence how entities interact over spatial and temporal scales (Grimm and Railsback 2005). Entities in my IBM are free-ranging white-tailed deer that interact with each other and the landscape. Each deer has 10 entity-level state variables that influence its behavior and characterize its physical and biological properties (Table 1.1). State variables age and sex define the demographic group for each deer (Table 1.1). I randomly assign a social group number to each deer at model initialization, with number of groups based on average group size reported from observational data collected within the study area (S. Courtney, unpublished data), which guides movements (Appendix A Section 5) and probability of contacting other deer (Appendix A Section 6). The location state variable indicates the GPS location of each deer within the study area, and the covariance matrix represents distances a deer can move in the next time step based on its current location (Table 1.1). These distances are drawn from a zero-mean Laplace distribution, which was trained on the dataset applied by Butts et al. (2022; Table 1.2). Each deer is assigned a vital status, indicating alive or dead during a time step (Table 1.1).

Deer may demonstrate three disease states in this model: susceptible, exposed, or infectious (Table 1.1). I consider every uninfected deer in the model as susceptible to CWD, as no immunity has been reported to date (Brandt et al. 2018, Napper and Schatzl 2023). Between three and six months following initial infection of a deer, the deer changes disease state from exposed (i.e., infected but not yet infectious) to infectious (Henderson et al. 2015). The exact day that a deer switches disease states and begins shedding prions is modeled as a stochastic process; the model randomly chooses an integer that falls between 90 and 180 days (i.e., 3–6 months; Henderson et al. 2015) after initial infection of each deer.

Lastly, each deer has state variables representing the time since infected with CWD to

allow for time-dependent disease processes that include disease state switching from exposed to infected, time since the deer last gave birth, and time since the deer last dispersed to a new social group (prevents these processes from occurring twice in one season; Table 1.1). My model also includes state variables that apply to individual deer, deer populations, and CWD in the landscape (Table 1.2). Entity based age and sex (Table 1.1) determine demographic group and results in corresponding daily probabilities (converted from yearly estimates) for parturition, mortality, movement, and disease (Table 1.2; Appendix A).

I used a spatial extent of 93-km^2 at 30m resolution. Each 30m grid cell is assigned two values retrieved from underlying maps to guide deer movement: a habitat use value (i.e., can a deer occupy this grid cell?) and a habitat suitability value (i.e., likelihood a deer moves into this cell; Appendix A Section 5). A proportion of young deer are allowed to move into and away from the assessment area (movement; Table 1.2). Excluding this subset of dispersing deer, if the movement module generates a new location for a deer that is outside of the study boundary, the model will generate another new location for that deer until one within the study boundary is identified. This process repeats up to 100 times to identify a new location that is within the study boundary. However unlikely, if a deer cannot move into a habitable cell, it will not move during that time step. One time step in the model represents one day. Each month is 30 days (i.e., time steps) in the model and thus each year is 360-time steps. The model can be run for any number of time steps, although typical simulations are for 5–20 years (1800–7200-time steps) given that computation time is a limiting factor.

1.2.2 Overview – Process overview and scheduling

For each daily time step, all deer move, have probabilities of dying from various causes, and have a probability of contacting other deer that are within 100 meters of them (Table 1.2).

Susceptible deer can become infected with CWD directly by contacting an infectious deer (i.e., direct pathogen transmission) or indirectly by inhabiting a cell that contains deposited environmental prions (i.e., indirect pathogen transmission). Infectious deer deposit prions into their residing cell during each time step. The processes to which deer are subjected depend on time of year, but the order of daily processes is the same for each day in the simulated year (Fig. 1.2).

Some population variables are seasonal (Fig. 1.2) and each deer is assessed by season for spring dispersal and migration by male fawns and yearlings (May), fawn births (May–June), hunting mortality (September–January), and fall dispersal and migration by male fawns and yearlings (October; Table 1.2). In general, CWD processes are not seasonal and can occur during any time step in the year. I multiply the daily probability of direct transmission by a monthly probability of contact throughout the year to account for seasonal changes in group membership and sex-specific differences in contact rates (S. Courtney, unpublished data, Williams et al. 2014, Tosa et al. 2015; Table 1.3). For example, the chances of male-male direct contact in August and September is >2X the chances in May, June, or July (Table 1.3). Disease expression in an individual results from three stages: an exposed period, an infectious period, and a clinical period (Table 1.1). Deer are only infectious during the infectious and clinical periods, but I do not model the behaviors of deer in these two stages differently.

1.2.3 Design Concepts

1.2.3.1 Basic Principles

Movements and behaviors of white-tailed deer differ by sex and age-class (Nixon et al. 1991), and individual variation in movements and behaviors within sex and age classes have been documented (Fieberg et al. 2008). Incorporating individual variation associated with

different sex and age classes of deer into models is critical for understanding infectious disease transmission within and among white-tailed deer populations. Incorporating individual variation also allows for inclusion of super-spreaders and super-shedders in diseased populations (Lloyd-Smith et al. 2005). Seasonality affects white-tailed deer behaviors, movements, and contact rates (Nixon et al. 1991, 2007; Schauber et al. 2007; Williams et al. 2014). I incorporate both individual and seasonal variation in the IBM by assigning different parameter values or coefficients for each sex and age-class of deer during the different seasons of the year where appropriate (Tables 1.2, 1.3).

1.2.3.2 Emergence

Contact rates among deer emerge from this model based on deer density, movements, and number of groups specified by the user. Contact rates will be greater for populations at higher densities as more deer will likely be in the same and neighboring landscape grid cells. Further, there is a higher probability that group members will be close to one another; all deer within a group experience the same attraction toward the center of that group in addition to random variation of their movements and positions around group center (Butts et al. 2022). If fewer groups are simulated while population size is held constant, there will be larger group sizes and more contacts among simulated deer.

1.2.3.3 Sensing

Individual deer respond to the location of their group center during every time step. Before any movement, the model will assess habitat usability for each deer in available grid cells based on their group center location. During a dispersal movement, the model assesses habitat suitability values of grid cells located in the area occupied by a new group into which it can disperse. Before initiating a group movement, the model assesses usability and suitability values

for the group from potential cells selected by the movement module. The usability map prevents the model from placing deer and group centers in a grid cell that cannot be occupied by deer (e.g., large body of water; Appendix A Section 5). Once in a new cell, deer are no longer affected by its suitability and usability values.

1.2.3.4 Interaction

Individual deer can interact with other deer within 100 m and the landscape within their current cell during any given time step. Interactions between deer depend on proximity, which is affected by group status. Deer in the same group will be more likely to interact because group members are closer to one another. I do not track nor record direct interactions (i.e., direct contact) among deer in the model, but I record direct transmission events of CWD, which occur via direct contact. If two deer reside within 100 meters of each other during a single time step (i.e., one day), there is a probability of interaction and direct transmission of CWD (see Appendix A Section 6). Although density-dependent effects on birth, mortality, or contact rates have been reported in free-ranging white-tailed deer populations (McCullough 1999; Schauber et al. 2007), I do not incorporate density dependence explicitly in this model because annual density of deer fluctuates minimally during model runs. Only when CWD becomes established in the deer population does the model begin to show an annual decline in the population.

A second modeled interaction occurs between individual deer and prions deposited across the modeled landscape. Infectious deer shed prions into occupied cells during each time step and the model records the density of prions in each cell**.** This prion density directly affects the probability that a susceptible deer becomes infected indirectly while inhabiting that cell (see Appendix A Section 8).

1.2.3.5 Stochasticity

Life events for each deer are probabilistic in the model and based on mean (and associated uncertainty) daily values reported in the literature (Table 1.2). I incorporate uncertainty by stochastically varying state variable values within the ranges of uncertainty associated with each mean (Table 1.2). This variability allows the model to mimic stochasticity observed among individual deer and among age classes and sexes of deer. Variables within each of the model modules (i.e., population, movement, and disease) incorporate stochasticity (Table 1.2). Furthermore, initial properties (e.g., sex, disease status, age in months) for each deer and its location are assigned randomly during initialization of the model.

1.2.3.6 Collectives

The deer movement module aggregates deer into social groups that vary in size and location across the study area (Butts et al. 2022). Deer social groups create intermediate levels of organization within the modeled deer population, where deer in the same group share the same general home range area and, thus, interact at higher frequencies than with deer from other groups. The number of groups is user-defined and specified at the beginning of each simulation. Conversely, the number of deer within each group is not user-defined but is an emergent model property resulting from initial user-defined number of deer and groups in the simulation. Social groups may consist of any age and sex of deer.

1.2.3.7 Observation

Output files are generated after each simulation that include: daily numbers of total deer, total exposed deer, total infectious deer, and percent of cells with deposited prions. I also record number of deer that reached their maximum lifespan, deer that reach five years post initial CWD infection (i.e., maximum disease lifespan), deer that succumb to CWD-related mortality, infected

deer that emigrate from the study area, and daily direct and indirect disease transmission events after each time step (see Section 1.2.6).

1.2.4 Initialization

Initial population size and size of the simulated landscape are user-defined but should align with deer density estimates reported in the literature for the region of interest. I initialized my model with 1,250 deer in a 93-km^2 study area in Clinton County, Michigan, USA (~13.5 deer/km^2 if uniformly distributed across the landscape) with a sex-age structure of 15% adult males, 45% adult females, 7% yearling males, 8% yearling females, and 25% fawns. This density and sex-age distribution was taken from a sex-age-kill model developed by Michigan Department of Natural Resources (unpublished) for the mid-Michigan region.

Habitat usability and suitability for the modeled landscape are depicted in two spatial layers. These habitat variables guide placement of deer and deer group centers simulated in the model (Appendix A Section 5). I seed deer and group centers across the landscape by first using the binary habitat usability map (described in Appendix A Section 5) to identify usable grid cells (Fig. 1.3). Among the usable cells, the model selects three cells at random where each group center can be placed. The model then places the group center in the cell that has the highest habitat suitability value out of the three options (Fig. 1.4). Once the model has identified initial starting locations for all deer group centers, the model gives each deer a location in proximity to the group center to which it was randomly assigned at model initialization.

I started the model with composition values for the deer population estimated by the Michigan Department of Natural Resources for each sex and age class: 15% adult males, 45% adult females, 7% yearling males, 8% yearling females, and 25% fawns. I then assessed how those values changed after each year as an emergent property of the model. Once the

composition remained relatively constant (< ±1%) for each sex and age class across years, I considered population composition stabilized and used those values as initial composition values of the modeled deer population for all analyses (Table 1.2). For each variable in the model, default values were based on literature for free-ranging deer populations in Michigan or other agriculture-forested regions (Table 1.2). When uncertainty was reported, I incorporated that variability into my IBM by drawing values from uniform distributions to obtain daily probabilities of each variable occurring during a time step, where the distribution was bounded by one standard deviation or error (Table 1.2).

I initialize an outbreak of CWD by introducing a CWD-infected deer into the simulated population at model initialization (i.e., time step 0). The time step an infected deer is introduced, the number of infected deer to introduce, and the type of deer that is infected (e.g., sex and age-class) can be specified by the user. The user may also define for how long the CWD-infected deer has been infected at the time it is introduced into the simulation.

1.2.5 Input data

I developed the habitat use layer using publicly available urban imperviousness data and open water data published by the National Landcover Database (NLCD; Fig. 1.3). I assigned the 30-m grid cells with an urban imperviousness percentage ≥ 75% and cells labeled as open water a value of 0 indicating that individual deer and group centers cannot occur in those cells. All other cells were assigned a value of one and are available for deer to occupy. I chose 75% as the value for urban imperviousness after examining local areas in Michigan that deer will not or cannot use to meet life requisites. These areas are typically situated within highly urban settings that deer may cross through but will not inhabit for extended periods (e.g., parking lots).

I also input a habitat suitability map developed from a white-tailed deer resource

selection function to characterize the landscape, with each cell assigned a habitat suitability value (Fig. 1.4). I derived these values and built this map in ArcGIS using a step-selection function (Quinn 2010) and publicly available NLCD landscape data (ESRI ArcMap 10.4.1). I conducted a moving window analysis over the habitat suitability map to estimate and account for suitability values of proximal cells, recognizing that habitat suitability surrounding a deer influences movement decisions. The moving window analysis computes the suitability value of each cell as the average value of cells, including its own, within a 0.56-km radius from the cell of interest, which corresponds to average home range size.

1.2.6 Variable estimation, verification, and validation metrics

Individual-based models that are parameterized, verified, and validated at the individual level and emergent collective levels better identify probable values for unknown variables and determine the realism and applicability of the model (Dion et al. 2011). As a verification step, I monitored the number of deer that reached their maximum lifespan (i.e., 12 years) and the number of deer that reached maximum disease duration (i.e., five years) after each simulation. The literature reports that few to no deer should reach these maximum life and disease spans in Midwestern regions of the USA (Table 1.2).

To understand disease dynamics, I calculated prevalence as the number of infectious and exposed deer divided by the total population size during a given time step. I also calculated sex-specific prevalence rates. I recorded the total number of direct and indirect transmission events (Appendix A Sections 6 and 8) that occurred after each time step and the number of CWD-related mortality events. As a proxy for geographic spread of CWD, I calculated the proportion of cells with shed prions (i.e., where infected deer inhabited the study area; Appendix A Section 7). I verified the disease model at the emergent collective level by calculating prevalence of

CWD after each year and compared the model predictions to annual apparent prevalence rates reported by wildlife agencies within the Midwestern USA. I developed a range of possible estimates for each disease variable by excluding values that led to population extirpation, prevalence rates greater than observed by wildlife agencies, or consistent disease extirpation in the model. I then chose the best estimate within each range based on reported annual prevalence rates from field studies.

Based on verification results of disease dynamics produced by the model, I calibrated estimates for the unknown parameters in the model: direct and indirect transmission rate and disease mortality rate (Table 1.2). I refer to these estimates as default values and used these values in the baseline scenarios. I also identified a range of possible values around the default value for each parameter (Table 1.2). Once I obtained best estimates for the unknown disease parameters, I calculated Spearman correlation coefficients to quantify the degree of association between observed and model-predicted annual rates of CWD prevalence.

I identified the number of repetitions needed for model convergence by evaluating stability of average CWD prevalence rate at year 50 as the model output. I produced 500 model repetitions and calculated the average value of the output iteratively across repetitions, starting with two and ending at the 500^{th} repetition. Successful stabilization of model output was achieved once changes in prevalence rate at year 50 were less than 0.001 after adding subsequent model repetitions.

1.2.7 Global sensitivity analysis

I quantified uncertainty and sensitivity of variables in the model via a global sensitivity analysis. I employed a mixed-method technique recommended for computationally expensive IBMs with many (10+) variables and interactions among those variables (Campolongo et al.

2011, Pianosi et al. 2016, Ligmann-Zielinska et al. 2020). To save on computation time, I assessed model sensitivity for 15 of 18 parameters (Table 1.4). I chose to include the seasonal dispersal and migration variables for yearling males in the sensitivity analysis but exclude these movement parameters for adult (2-2.5 years) males with the assumption that model sensitivity to these movement parameters would be similar between age classes, if not lower for adults given that they occur less frequently in the model. I also chose to exclude the mortality parameter for fawns aged 0-2 months and assume that the model would be similarly sensitive to this parameter for fawn mortality at 2-12 months. The model output I used to conduct the sensitivity analysis was prevalence of CWD at year 20 following initial introduction of CWD.

I used an elementary effects method (i.e., Morris method) as a screening method to initially identify order of variable influence on estimates of CWD prevalence at year 20, identify variables involved in input interactions, and identify which variables were non-influential inputs and could be omitted from further sensitivity analyses (Morris 1991, 2006). The Morris method approximates a global measurement of the elementary effects by locally estimating the elementary effects throughout the input space. Each local estimate utilizes 16 sets of inputs (1 + total number of input variables), with one being the baseline model applying the most likely value for all model variables, referred to as a trajectory. For each trajectory, the first set of inputs was randomly sampled from triangle distributions, which allowed me to specify a minimum, maximum, and most likely values for each of the input variables, thus avoiding unphysical regions of the input space (Table 1.4; Kotz and van Dorp 2004). The remaining 15 sets of input variables used a one-factor-at-a-time experimental design that increased a single variable by 10%; this was done such that only a single variable would differ between any pair of variables. I chose to use 79 trajectories because that maximized the number of parallel simulations I could

execute at a single time using high performance computing.

For each of the 79 trajectories, I estimated the elementary effect, $EE_y(x_i)$, for each input variable (x_i) on CWD prevalence after 20 years (y). The elementary effect for an input variable is the derivative of the model output with respect to that variable,

$$EE_y(x_i) = \frac{y(x_1 \dots x_i+\Delta,\dots,x_n)-y(x_1 \dots x_i,\dots,x_n)}{\Delta x_i},$$

Where x_i is the difference in variable x_i between runs in a trajectory that correspond to changing variable x_i, Δ is the 10% change in parameter value, and n is the total number of variables assessed (Morris 1991). With these local estimates of the elementary effects, I estimated the total effect measure (μ_i^*) and non-linear and interaction effects (σ_i) for each input using the following equations:

$$\mu_i^* = \sum_{r=1}^{R} \left| \frac{EE_i(x_r)}{R} \right|,$$

$$\sigma_i = \sqrt{\sum_{r=1}^{R} \frac{(EE_i(x_r)-\mu_i)^2}{R}},$$

where R represents the number of trajectories (Morris 1991). The elementary effects value for each parameter did not deviate ≥ 5% after 45 trajectories, indicating that the values converged and that I met the number of trajectories needed to cover the high-dimensional input space of this model adequately.

I used an additional method to assess model sensitivity on influential variables identified via the elementary effects method to assess influence of individual inputs in relation to the other input factors, such as characterizing the extent of *how* individual interactions affect model output (Campolongo et al. 2011, Ligmann-Zielinska et al. 2020). I conducted a global variance-based decomposition, which decomposes the output variance and assigns partial variances to the inputs and their interactions (Saltelli et al. 2008, Ligmann-Zielinska and Sun 2010, Zhang et al. 2019).

The variance decomposition method can be applied to nonlinear models (i.e., nonlinear relationships between model inputs and outputs) and fully assesses interactions among inputs (Ligmann-Zielinska et al. 2020). Variance decomposition produces two sensitivity indices for each input variable: a first order index (S_i) and a total effect index (ST_i). The first order and total effect indices of input variable *i* on total model variance, *V*, are defined as:

$$S_i = \frac{V_i}{V},$$

$$ST_i = \frac{V - V_{C_i}}{V},$$

where V_i is the variance of model output *Y* in response to the variability of variable *i* alone, and V_{C_i} is the conditional variance resulting from all input variables except *i*.

The first order index measures the independent contribution of each individual input variable to output variance. The total effect index measures overall influence of each variable on output variance by including the effect of that variable associated with variable interactions. If the sum of the S_i values for all input variables equal 1, the model is additive without interactive effects and ST_i does not need to be calculated. If ST_i is greater than S_i, for the i^{th} input variable, that variable is involved in model interactions (Ligmann-Zielinska and Sun 2010). This method is computationally expensive and requires systematic sampling of input variables (Sobol′ 1993), grouping of variables (Ligmann-Zielinka 2018), or high-performance computing (Tang et al. 2011). I opted for systematic sampling and high-performance computing.

I employed Sobol′ quasi-random sampling, also referred to as radial sampling, to reduce computational cost by producing more evenly distributed samples and to ensure that the entire model input space was assessed (Sobol′ 1993, Saltelli et al. 2008). Quasirandom sampling avoids inherent clusters and gaps of values within the distributions of values for each input variable,

particularly when sample size is low (Saltelli et al. 2010). Sobol′ sampling uses a Monte Carlo integration method to achieve multidimensional integration of the sensitivity indices. It decomposes the variance of model output into summands of variances of the input factors in increasing dimensionality (Sobol′ 1993). I generated variable sets using a Sobol′ Sequence and ran those variable sets within the IBM.

Each variable set is composed of $2+k$ sets of inputs, referred to as 'radials,' where k is the number of input variables in the model. The first two radials (A and B) are independent random vectors generated from sampling the distributions in Table 1.4. The remaining k samples (AB_1 AB_2,…,AB_k) are created by combining the first two samples. The i^{th} radial is made by copying A, then replacing the i^{th} location with the i^{th} location in B.

1.3 Results

My IBM reproduced short- and long-term population dynamics characteristic of Midwestern white-tailed deer, as represented by seasonal population fluctuations within each year and stability of the population across years (Fig. 1.4). In addition, my model reproduced long-term CWD dynamics observed in field observations reported from endemic areas of Wisconsin (WDNR 2023; Fig. 1.6). Visual displays for three time periods illustrate deer locations and density of prion deposition (Fig. 1.7). My assessment of model convergence identified stabilization of model output after 322 repetitions (Fig. 1.8). Therefore, I report results that are averaged across 350 model repetitions to account for stochasticity in the model. The proportion of deer in each sex and age class in the simulated deer population took three years to stabilize when CWD was not introduced in the model. The population stabilized at 33% adult females, 12% adult males, 10% yearling females, 9% yearling males, 18% female fawns, and 18% male fawns.

The high-performance computing center (HPCC) at Michigan State University took about 30 hours for a full model to run for 50 simulated years with a starting population of 1250 deer and to extract 14 output values after each time-step. Twenty-year model runs took 11–14 hours. The HPCC allowed me to run all 350 repetitions of the model in parallel, which reduced computation time exponentially.

Out of 350 model repetitions, CWD persisted (i.e., prevalence >0.1% at year 50) in 100 (28.6%) model simulations after the introduction of one infected deer. For the model runs where CWD persisted, total population size decreased 0.5% by year 10, 7.7% by year 25, and 87.1% by year 50 following initial introduction of CWD in the population (Fig. 1.9). In 102 of the 350 model repetitions (29.1%), the outbreak of CWD ended at least once (i.e., 0 infected deer by the end of a year) after persisting for at least the first year. In those 102 model repetitions, there were 118 individual events where the outbreak ended, indicating that the outbreak ended and restarted multiple times during some model runs. The percentage of outbreaks that ended but led to reintroduction of CWD via indirect transmission through environmental prions was 23%. CWD was reintroduced and faded out up to three times within an individual model run. Year 5 was the mean year when outbreaks were most likely to end (mean=5.0, median=3.0, SD=4.2, range=1-23 years). For outbreaks that ended after persisting for at least one year, highest prevalence rate of CWD reached was 1.47%. Reintroduction events via indirect transmission ranged between 1 and 17 years following the last year of the previous outbreak of CWD.

After 50 years following introduction of the disease and for the 29% of cases where CWD persisted in the landscape (93 km^2), final deer population size averaged 140 (a population decline of 87%). Mean prevalence of CWD was 0.4%, 3.4%, 27.7%, 58.9%, 60.7%, and 51.8% after the first, tenth, twentieth, thirtieth, fortieth and fiftieth years, respectively (Fig. 1.10). The

difference between prevalence rates for males and females decreased over time, with 5.3% and 2.3% after year 10, 37.0% and 22.4% after year 20, 69.5% and 52.9% after year 30, 69.5% and 55.5% after year 40, and 59.1% and 47.3% after year 50, respectively (Fig. 1.10). The Spearman correlation coefficient assessing the degree of association between observed prevalence values reported annually by the Wisconsin Department of Natural Resources (2023) and my model-predicted prevalence values was 0.9, 0.988, 0.985, and 0.994 when assessing the first 5, 10, 15, and 20 years, respectively, following initial introduction of CWD (Fig. 1.6). Apparent prevalence rates are reported for only 21 years in most counties in Wisconsin, so I could not compare reported rates to the model-derived rates beyond 21 years. Although wildlife agency reports of sex-specific prevalence rates differ in their extent spatially and temporally, Illinois Department of Natural Resources (2023), Government of Alberta (2023), and Saskatchewan Government (2023) report 40%, 54% and 67% higher prevalence rates, respectively, in male white-tailed deer than females.

1.3.1 Variable calibration and model verification metrics

In addition to prevalence of CWD, I calculated summary statistics for ten other outputs after each simulated day to verify individual deer, population, and disease model processes: total deer, exposed deer, infectious deer, deer that reach their maximum lifespan and disease span, direct and indirect transmission events, CWD-associated deaths, emigrated deer that were infected, and the proportion of cells with shed prions (Table 1.5). I found that direct transmission of CWD was higher than indirect transmission as time progressed (but direct transmission estimates were highly uncertain; Fig. 1.11). Numbers of exposed and infectious deer, prevalence, the proportion of the study area affected by prions, and the numbers of direct and indirect transmission events were calculated only for model runs where CWD persisted in the population

(29% of total runs). The average (lower 2.5% and upper 97.5% quantiles) number of deer that reached their maximum lifespan after running the model 350 times across 50 years was 134 (128, 181). The average (2.5%, 97.5% quantiles) number of infected deer that reached five years post-initial infection (i.e., maximum disease timeline) was 470 (2, 590), which was 14.9% of the total number of deer that became infected with CWD throughout the 50-year simulation.

The model estimated an average (2.5%, 97.5% quantiles) of 245 (2, 335) CWD-related mortalities, and 120 (1,183) infected deer emigrated away from the study area (Table 1.5). The mean proportion of grid cells making up the study area that contained shed prions at 50 years post-initial infection was 0.57 (0.02, 0.66; Table 1.5). My model predicted 3,027 (102, 5,395) direct transmission events and 538 (18,773) indirect transmission events after 50 years (Table 1.5).

1.3.2 Global sensitivity analysis results

The Morris screening method identified three variables that did not influence prevalence of CWD after 20 years based on their mean total effect values and, thus, were excluded from further sensitivity analyses: group number, prion half-life, and prion shedding rate (Table 1.6). To reduce computation time, I also excluded spring dispersal rate, which ranked fourth lowest (Table 1.6). CWD mortality rate had the largest total and interaction effect values on CWD prevalence at year 20 (Table 1.6). Indirect and direct transmission rates ranked second and third, respectively, for total and interaction effects (Table 1.6).

I completed 95 Sobol′-derived sampling trajectories across the factor input space to assess variance decomposition for the first 20 years in the IBM (Table 1.7). I could not complete enough trajectories to account for all model variation and, thus, achieve model convergence necessary to calculate first-order S_i indices for each factor. Therefore, I was able to calculate the

amount of model variance contributed by each variable but not the extent of interactive effects driving the variance. I normalized the S_T values by dividing the value generated for each factor by the sum of S_T values for all input factors. Adult female harvest was the largest contributor to variation in CWD prevalence for all model years assessed (i.e., years 5, 10, 15, and 20; Fig. 1.12). Yearling female harvest rate accounted for the second most variation observed in CWD prevalence at years 10, 15, and 20. Fall immigration and emigration rate was second most influential on CWD prevalence in model year 5 (Fig. 1.12). Yearling and adult male harvest rates were least influential on CWD prevalence for model years 5, 10, 15, and 20.

1.4 Discussion

The IBM for predicting CWD dynamics in free-ranging deer is based on deer and disease model components. The deer component includes modules that represent population-level processes of demographics, parturition, mortality, and movement. The disease component includes modules for disease-related mortality, and direct and indirect transmission. Complexities of the deer-CWD system make modeling challenging (Uehlinger et al. 2016), but development of IBMs and high-performance computing has advanced our ability to portray these systems. The IBM described herein mimicked CWD disease dynamics in deer to year 21 (temporal extent of available validation data) and indicated that fall fawn immigration and emigration rate, disease mortality rate, and fawn mortality rate had the largest impact on CWD prevalence. The model also indicated that prion half-life, prion shedding rate, and deer group membership (a variable representing the social structure of deer sub-groups) had minimal influence on CWD prevalence at year 20. Furthermore, sensitivity analysis indicated that adult and yearling female harvest rates explained the greatest amount of variation in CWD prevalence at year 20. Collectively, model results indicate that females are a demographic group that has a

large effect on CWD dynamics in Michigan and this effect increases age (i.e., adult females affect CWD prevalence most and fawn females affect prevalence least).

An important part of model development includes verification and validation (Augusiak et al. 2014). The IBM reproduced individual-level deer processes and macro-level population and disease dynamics typically observed in Midwestern white-tailed deer populations for 20 years. My IBM found that a small proportion of simulated deer remained alive for 12 years, consistent with observed longevity for Midwestern deer (Michigan Department of Natural Resources, unpublished). Furthermore, I demonstrated that total number of modeled deer in the absence of CWD fluctuated annually and remained relatively stable over the 50-year model horizon. This pattern corresponds to population dynamics commonly observed in white-tailed deer populations inhabiting mixed agricultural and forested regions, particularly within the U.S. (Rosenberry et al. 2011). Thus, the deer demographic component of the IBM functioned as expected.

I validated individual-level CWD processes by comparing the number of modeled deer that died from CWD-associated mortality and reached their maximum disease timeline (i.e., five years post initial infection) to published literature or independently collected field data from Wisconsin. Deer apparently do not live ≥5 years after CWD initial infection (Williams 2005). In the IBM, I observed <15% of infected deer reach this maximum disease duration, providing evidence that the epidemiological processes in the model are reasonable.

I hypothesized that direct CWD transmission events primarily drive CWD dynamics as the disease emerges in a population, whereas indirect transmission events remain low and are less influential until the disease becomes endemic in later years (>50 years). Modeled disease dynamics reflected these patterns. The proportion of modeled grid cells containing shed prions

increased through time along with annual CWD prevalence rate (Spearman correlation coefficient = 0.863 for the first 50 years). Thus, it appears that indirect transmission grows proportionally with direct transmission over time.

I reported CWD prevalence values after each time step and compared those values to annual prevalence rates reported by the Wisconsin Department of Natural Resources. My model CWD prevalence values were highly correlated (i.e., r = 0.994) with prevalence rates reported at the township level and averaged over six counties in Wisconsin for 21 years. The correlation between modeled and observed CWD prevalence rates was higher for the 21-year period compared to shorter duration (5-15 years). The lower correlation for earlier years suggests that my model is slightly better at predicting long-term CWD dynamics rather than dynamics occurring soon after initial infection. However, this part of the model validation is complicated by potential variations in field data collection over time. In the years following initial detection of CWD in Wisconsin, surveillance and sampling efforts focused in the immediate area where CWD was first detected, which was labeled the CWD eradication zone (Heberlein 2004). As CWD spread into new townships and counties, I suspect that apparent prevalence rates were generated from smaller sample sizes until sampling capacity increased.

Annual CWD prevalence growth rate was 1.1% for infected Wisconsin deer populations when apparent CWD prevalence rates were 5-15% (Heisey et al. 2010). In my model, prevalence rates between 5 and 15% were observed for model years 12–16, with a mean annual CWD growth rate of 1.9%. In the western U.S., annual growth rates of 1.15% (Kreeger 2008, Binfet 2009) and 1.2-1.25% have been estimated (Miller and Conner 2005). Trends in reported prevalence rates in Colorado indicate slow increases until an inflection point is reached at a rate of approximately 5%. Once reached, exponential acceleration in prevalence occurs (Miller et al.

2000, Colorado Parks and Wildlife 2018). In an area endemic with CWD in Wyoming, the state agency reported an increase in prevalence from 11% to 36% in ten years (Kreeger 2008, Binfet 2009). It took my model eight years (model year 15 to year 22) for prevalence of CWD to grow from 11% to 35%.

Many states within the U.S. report prevalence of CWD in male cervids as 2- to 3-times greater than prevalence rate in females within a population (Miller and Conner 2005, DeVivo et al. 2017, Samuel and Storm 2016; but see Edmunds et al. 2016). By accounting for differences in monthly contact rates within and between different sexes of deer in the modeled population and applying a correction factor to males for indirect and direct CWD transmission, my goal was to reproduce these sex-specific trends in prevalence of CWD. I was able to produce higher prevalence rates among males consistently in my model, although the difference between male and female rates shrunk through time. Most wildlife agencies do not report male versus female prevalence within a population. For the agencies that do, rates are often aggregated at larger spatial scales. Illinois Department of Natural Resources (IDNR) reports a ~75% higher statewide prevalence rate for males during a 20-year period (2003-2023; IDNR 2023). For the 2022-2023 hunting season, the Government of Alberta reported 54% higher prevalence statewide in male white-tailed deer than females (Government of Alberta 2023). Similarly, in a subset of wildlife management zones chosen based on sufficient surveillance sampling (>5 males and >5 females), three-year pooled prevalence estimates measured a 67% higher prevalence for male white-tailed deer (Saskatchewan Government 2023).

It remains relatively unknown how differences in prevalence rates among male and female white-tailed deer within a population change over time. Illinois Department of Natural Resources (2023) reports this difference for each hunting season, and it appears the statewide

male prevalence rate has remained about 40% greater than the female prevalence rate for 20 years (2003-2023) with annual fluctuations but no long-term trends. I provide three hypotheses for observing a shrinking difference in male and female prevalence rates in my model. My first hypothesis is that my model calculates true prevalence rates whereas wildlife agencies calculate and report apparent rates that are biased by sampling strategy and amount. It may be the case that deer populations are also experiencing this decreased difference in male and female prevalence rates through time but cannot detect it using their current surveillance methods. Second, my model predicts differences in prevalence rates for 50 years following the initial introduction of CWD into a deer population. Most wildlife agencies, such as those in the Midwest, have only detected the disease within the last 20-25 years. These agencies may start to observe a decline in sex-specific differences in prevalence rate later in the disease epidemic. Last, if male prevalence does remain consistently higher than female prevalence through time, I expect that I underestimated the magnitude of male-male interactions or overestimated the magnitude of male-female interactions each year despite generating monthly contact coefficients that were based on observational field data within the study area. To force male prevalence at least 1.5 times greater than female prevalence, I included a correction factor, which was multiplied by direct transmission and contact rates when a susceptible male made contact with an infected individual, regardless of the sex of the infected deer, in September through January. I chose to add this correction factor during September through December because this period captures rut behavior and poor body condition by adult males, which are two possible reasons for higher prevalence rates reported for males (Hewitt 2011). However, this correction factor was not sufficient to keep male prevalence at least 1.5 times greater than female prevalence across 50 years.

In recent years, researchers are finding that scrapes, rubs, and licking branches serve as reservoirs for prions in the environment and may play an important role in indirect transmission of CWD among male white-tailed deer during the rut (Egan et al. 2023, Hearst et al. 2023). It is possible to account for this chemical communication behavior among males during the rut and explore their effects on CWD dynamics by incorporating seasonal sex-specific indirect transmission rates into the model (Alexy et al. 2001).

A limitation to consider when comparing prevalence rates generated by a model to rates reported by wildlife agencies is that I am comparing a true rate to an apparent one. Agencies cannot compute actual prevalence of a wildlife disease; they rely on apparent rates, which are estimated from a subsample of the affected population. This subsample is typically the number of deer that are harvested or collected via other disease sampling methods and tested for CWD each year. The total size of the subsample used to calculate the apparent prevalence rate and, thus, its precision changes each year in response to sampling intensity. Factors affecting sampling intensity include hunter participation, such as the number of deer harvested that year, and harvest-associated regulations, such as mandatory testing of harvested deer.

When calculating and reporting apparent prevalence rates of CWD, we risk either under or over reporting the extent of the disease in an area (i.e., directional bias), and the direction of this bias is scale dependent. Chronic wasting disease in free-ranging cervid populations is spatially clustered and not uniformly distributed across the population, as is the case with most transmissible diseases (Joly et al. 2006, Osnas et al. 2009, Walsh 2012, Hedman et al. 2020). If deer tested for CWD are not located within a clustered diseased area, which can be the case when relying on hunter-harvested and roadkill deer, the disease will be underreported. If a wildlife agency is successful in its ability to locate the origin or center of the outbreak and tests deer from

that area more but reports it at a larger scale, such as at a county level, there is an opportunity to over report the disease. The former scenario is a common issue for agencies. Chronic wasting disease is rarely detected soon after its introduction to a population, therefore making it difficult to locate the center of the outbreak (Hefley et al. 2017, Cook 2020). In addition, CWD is often managed and reported at the county scale or greater. If CWD is detected in a localized area within a county but sampling is not targeted and conducted arbitrarily across the entire county, the apparent prevalence rate will be an underestimate of true prevalence.

The Morris screening method for model sensitivity testing revealed that most of the variables associated with indirect transmission of CWD, but not indirect transmission rate itself, were not influential on prevalence of CWD after 20 years. This implies that direct transmission of CWD still has a greater influence on disease dynamics 20 years into the epidemic. As the disease enters an endemic state and as more deer become infected with CWD and advance into later stages of infection, more prions are shed into the environment and create additional opportunities for indirect disease transmission. At that time, shedding rate and half-life of prions may become more influential on disease dynamics. However, results from the global variance decomposition analysis complicates this finding. Indirect transmission rate had greater influence than direct transmission on prevalence rate at year 20, albeit the values were similar (0.089 and 0.085).

The sensitivity indices calculated for each model input by the Morris method and the variance decomposition analysis do not provide the same results. For example, the Morris method revealed prevalence of CWD at year 20 as most sensitive to CWD mortality rate whereas variance decomposition identified this variable as 8^{th} most influential. The Morris method cannot adequately account for interactive effects among input variables. Therefore, although the Morris

method is sufficient in identifying which input variables do not influence model output, its results should not be used to rank variables according to their sensitivity indices. Alternatively, variance decomposition can decompose and quantify variable interactions and, thus, can be used to rank the influence of each input variable on model output.

Researchers and wildlife managers have estimated that, at some point, indirect transmission becomes more important than direct transmission on CWD dynamics in free-ranging cervid populations endemic with the disease. This estimate stems from the ideas that: 1) CWD reduces population size and deer density over time, which reduces the number of contacts made and, thus, direct transmission events among deer, and 2) prions accumulate in the soil within affected areas over time, which increase the probability of local deer becoming infected indirectly through the environment (Edmunds et al. 2016, DeVivo et al. 2017). The sensitivity analysis I performed on the IBM supports this transition of influence between transmission modes and suggests that the transition occurs between 15 and 20 years post initial introduction of CWD.

The Morris screening method identified number of deer groups to be uninfluential on disease dynamics after 20 years. One hypothesis for this finding is that the number of deer (i.e., deer density) within the study area, which was held constant at initialization of my model, is more influential on disease dynamics than total number of groups of deer. Although studies report that deer in different social groups are less likely to interact when in proximity, this appears to hold true for females and juveniles only (Magle et al. 2013, Schauber et al. 2015, Grear et al. 2010). Furthermore, transmission of CWD may be driven by males in the deer population given their higher prevalence rates in most reported regions of the U.S. (Miller and Conner 2005, DeVivo et al. 2017, Samuel and Storm 2016). Males form looser social groups

with other members of the same sex (Nixon et al. 1991) and interactions with other male and female deer prior to and during the rut (e.g., sparring, scraping, and breeding) are not known to be influenced by group membership.

Variance decomposition of model output identified that CWD prevalence was most sensitive to harvest rates of adult and yearling females over time, despite including a correction factor to ensure higher prevalence in males. Adult females made up the highest proportion of the population (i.e., 33%), and were responsible for producing the most fawns each year, which is likely why the harvest rate of adult females is more influential on disease dynamics than harvest of other demographic groups. Yearling females age into adults and produced the second most fawns each year, which may explain the influence of their harvest on model output.

Increasing model sensitivity to the harvest rate of deer indicates that deer removal, regardless of method (i.e., hunter harvest or agency culling), as a management strategy for reducing CWD prevalence becomes more effective over time (i.e., the effect size increases with time). However, due to political and financial constraints, population reduction efforts as a method for controlling the growth and spread of CWD are often terminated within the first few years of their initiation (Heberlein 2004, Holsman et al. 2010). My results emphasize a need for continued population reduction throughout the disease epidemic and not just the first few years after disease detection.

My goal was to provide researchers and wildlife managers with a model that serves as a tool to investigate and predict spatiotemporal dynamics of CWD in white-tailed deer populations. This model simulates CWD in accordance with our current knowledge of CWD dynamics and Midwestern white-tailed deer behavior using available epidemiological and ecological field data. Moreover, I developed this tool using a framework that can easily be

adapted to address other cervid species, geographic locations, and infectious disease systems. However, model complexity quickly grew during the development phase, which increased computation time of the model exponentially. My next steps for this model include streamlining many of the population and disease functions to achieve my goal of providing a user-friendly tool for wildlife agencies and others to use without access to high-performance computing systems.

Wildlife managers and researchers may apply this model to assess disease management interventions, surveillance methods, or the effects of environmental conditions on direct and indirect transmission of CWD. In addition, wildlife agencies can incorporate this model into adaptive management plans for CWD to aid in decision-making by testing alternative management scenarios. Given the expenses and risks involved in the investigation of surveillance and management methods for CWD in free-ranging cervid populations, I suggest that wildlife agencies use this model to assess potential actions prior to decision making and implementing management in the field.

Table 1.1. State variables assigned to each white-tailed deer (entities) in the individual-based model (IBM) for estimating chronic wasting disease (CWD) dynamics.

Variable	Description
Age	Deer age in months
Sex	Deer sex
Group number	Group number of the deer
Location	X (i.e., UTM Easting of a deer) and Y (i.e., UTM Northing of a deer) position of a deer
Covariance matrix	Covariance matrix of UTM Easting and UTM Northing location data
Vital status	Identifies whether the deer is alive or dead during a time step
Disease status	Deer disease status indicating no exposure to CWD (i.e., susceptible), exposure to CWD (i.e., exposed and infected but not yet infectious; yes/no) or infected by CWD (i.e., infectious; infected and infectious; yes/no)
Time since infected	A counter tracking the number of months since a deer was initially infected with CWD
Birth interval	A counter tracking number of months since a deer last gave birth
Dispersal interval	A counter tracking number of months since a deer last dispersed

Table 1.2. State variables assigned to model components in the individual-based model for CWD dynamics in free-ranging deer populations.

Model Component	Model Module	State Variable	Default Value(s)
Deer	Demographic	Maximum Age	12 yrs [a]
		Population proportion Adult Males	12% [b]
		Population proportion Adult Females	33% [b]
		Population proportion Yearling Males	9% [b]
		Population proportion Yearling Females	10% [b]
		Population proportion Fawns	36% [b]
		Average group size	5 [c]
	Parturition	Adult Birth Rate	1.8 fawns/doe (range = 0-3 fawns) [a. d]
		Yearling Birth Rate	1.25 fawns/doe (range = 0-3 fawns) [a]
		Fawn Birth Rate	0.4 fawns/doe (range = 0-1 fawns) [a]
		Fetal Sex Ratio	0.5 [d, e, f]
	Mortality	Adult Male Baseline Non-harvest Rate	0.24 deer/yr [g]
		Adult Female Baseline Non-harvest Rate	0.075 deer/yr [g]
		Yearling Male Baseline Non-harvest Rate	0.28 deer/yr [g]
		Yearling Female Baseline Non-harvest Rate	0.28 deer/yr [g]
		Fawn < 2 Months	0.34 deer/yr [h]
		Fawn 2-12 Months	0.08 deer/yr [h]
		Adult Male Harvest Rate	0.42 deer/yr (97.5% CI = 0.28-0.56) [g]
		Adult Female Harvest Rate	0.16 deer/yr (97.5% CI = 0.10-0.22) [g]
		Yearling Male Harvest Rate	0.36 deer/yr (97.5% CI = 0.21-0.50) [g]
		Yearling Female Harvest Rate	0.17 deer/yr (97.5% CI = 0.07-0.28) [g]
	Movement	Fall Male Adult (2-2.5 yr) Dispersal	0.09 deer/yr [g]
		Fall Male Yearling Dispersal	0.13 deer/yr [g]
		Spring Male Adult (2-2.5 yr) Dispersal	0.27 deer/yr [g]
		Spring Male Yearling Dispersal	0.38 deer/yr [g]
		Spring Male Yearling Immigration	0.08 deer/yr [g]

Table 1.2 (cont'd).

Model Component	Model Module	State Variable	Default Value(s)
		Spring Male Yearling Emigration	0.08 deer/ yr [g]
Disease	General	CWD-associated mortality	0.055 (range = 0.004 – 0.1) [b]
	Direct Transmission	Direct Transmission	0.017 (range = 0.1 – 0.2) [b]
	Indirect Transmission	Prion Half-life	48 months [i, j]
		Indirect Transmission	0.0003 (range = 0.0001 – 0.001) [b]

[a] Michigan Department of Natural Resources (unpublished).
[b] Model-derived or estimated value.
[c] S. Courtney (unpublished).
[d] Green et al. 2017.
[e] Mori et al. 2022.
[f] Verme 1983.
[g] J. Trudeau (unpublished).
[h] Rohm et al. 2007.
[i] Miller et al. 2004.
[j] Tenant et al. 2020.

Table 1.3. Monthly coefficients used to adjust the constant monthly rate for direct transmission of chronic wasting disease (0.017) to account for changes in the probability of two deer contacting each other by season and sex. Coefficients are estimated from empirical deer observations in the southern portion of the Lower Peninsula of Michigan, USA (S. Courtney, unpublished data), where lower coefficients correspond to lower likelihoods of direct contact. Females are further categorized as group and non-group members to account for potential differences in contact rates.

	Jan	**Feb**	**Mar**	**April**	**May**	**June**	**July**	**Aug**	**Sept**	**Oct**	**Nov**	**Dec**
Male-male	0.034	0.034	0.034	0.034	0.051	0.051	0.051	0.119	0.119	0.017	0.017	0.017
Male-female	0.034	0.034	0.034	0.017	0.017	0.017	0.034	0.034	0.034	0.085	0.034	0.034
Female-female												
Group members	0.119	0.119	0.119	0.068	0.034	0.017	0.017	0.068	0.068	0.068	0.068	0.068
Non-group members	0.051	0.051	0.051	0.017	0.017	0.017	0.017	0.017	0.017	0.017	0.017	0.017

Table 1.4. Triangular distributions of variable estimates specified for each of 15 model variables assessed by the Morris screening method within the global sensitivity analysis (Kotz and van Dorp 2004). Initial variable values were drawn at random from the listed range of values. I report daily rates in this table except for deer group number and prion half-life, which is in months.

	Minimum value	Maximum value	Most likely value
Deer group number	50	630	251
Direct transmission rate	0.005	0.03	0.017
Indirect transmission rate	0.0001	0.00065	0.0003
Prion shedding rate	0.001	20	1
Prion half-life (months)	3	120	48
Disease mortality rate	0.00005	0.002	0.00015
Harvest mortality rate			
Adult male	0.0002	0.007	0.0025
Adult female	0.0002	0.007	0.001
Yearling male	0.0002	0.007	0.003
Yearling female	0.0002	0.007	0.00144
Fawn mortality (2-12 months)	0.0002	0.003	0.00087
Immigration & emigration rate (male yearlings)			
Spring	0	0.03	0.00267
Fall	0	0.03	0.00267
Dispersal rate (male yearlings)			
Spring	0	0.03	0.01267
Fall	0	0.03	0.00433

Table 1.5. Chronic wasting disease (CWD) outcomes produced by the individual-based model after model year 50 summarized with means, standard deviations, medians, and upper (97.5%) and lower (2.5%) quantiles.

Model output	Mean	SD	Median	2.50%	97.50%
CWD prevalence (%)	52	19	53	20	81
Male CWD prevalence (%)	59	20	63	18	88
Female CWD prevalence (%)	47	18	47	16	79
Total deer	163	107	115	54	523
Total CWD-related deaths	245	103	269	2	335
Total deer that reached maximum disease lifespan	470	162	532	2	590
Proportion of cells with shed prions	0.57	0.13	0.60	0.02	0.66
Total direct transmission events	3,027	1,141	4,714	102	5,395
Total indirect transmission events	538	169	645	18	773
Total number of infected deer that emigrated out of study area	120	43	133	1	183

Table 1.6. Mean total effect (μ^*) and interaction effect (σ) measures calculated for each input variable in the individual based model used for predicting CWD prevalence at year 20 in deer assessed using the Morris screening method. Variables are ranked from highest to lowest μ^* value. Variables in bold were determined to be uninfluential input parameters based on their low μ^* value and removed from further model sensitivity analyses.

Parameter	μ^*	σ
CWD mortality	3871	10699
Indirect CWD transmission	2589	4977
Direct CWD transmission	2322	4224
Fawn (2-12 months) mortality	1856	3892
Adult female harvest	1519	2871
Yearling female harvest	1131	2579
Adult male harvest	699	1402
Yearling male harvest	655	1361
Fall immigration & emigration	469	1043
Spring immigration & emigration	337	725
Fall dispersal	240	569
Spring dispersal	182	330
Prion shedding rate	0.595	2.135
Prion half life	0.054	0.151
Group number	0.007	0.014

Table 1.7. Total effect indices (i.e., normalized S_T values) derived from the global sensitivity analysis of the individual based model for predicting chronic wasting disease (CWD) dynamics. The analysis uses a variance decomposition approach based on Sobol′ sampling. Table values represent total amount of variance in predictions of CWD prevalence at model years 5, 10, 15, and 20, attributed to each model input factor. S_T values are ordered from highest to lowest based on model year 20 values generated by the individual-based model.

	S_T values			
Model factor	Year 5	Year 10	Year 15	Year 20
Adult female harvest	0.194	0.186	0.160	0.150
Yearling female harvest	0.102	0.128	0.115	0.123
Fawn (3-12 months) mortality	0.082	0.091	0.111	0.101
Fall immigration & emigration	0.126	0.098	0.095	0.093
Indirect CWD transmission	0.068	0.065	0.076	0.089
Direct CWD transmission	0.073	0.124	0.111	0.085
Adult male harvest	0.056	0.042	0.055	0.078
CWD mortality rate	0.082	0.082	0.073	0.076
Fall dispersal	0.107	0.084	0.087	0.072
Spring immigration & emigration	0.063	0.061	0.062	0.070
Yearling male harvest	0.048	0.040	0.056	0.065

Figure 1.1. The structure of model descriptions for individual-based models (IBM) following the Overview, Design, Concepts, and Details (ODD) protocol. This figure is adapted from the figure provided by Grimm et al. (2020). Not all components are relevant to each IBM; thus, some of the design concepts listed here are not included in the methods section, as suggested by Grimm et al. (2020).

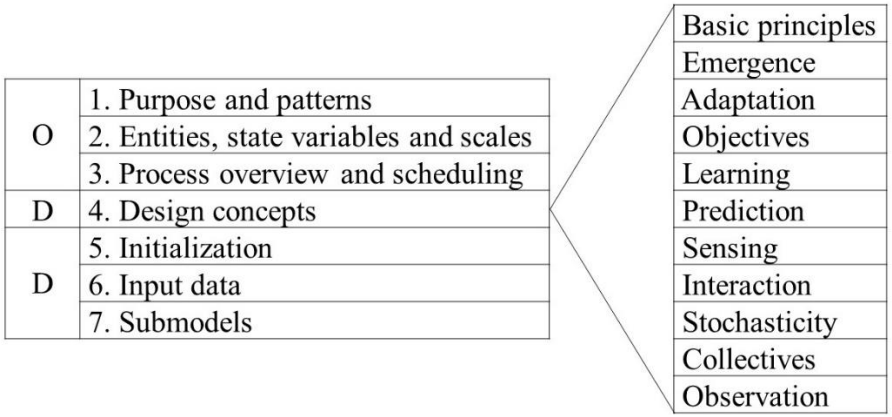


Figure 1.2. Scheduling daily and monthly deer population state variables for simulating chronic wasting disease (CWD) dynamics in a white-tailed deer population using an individual-based model.

Daily functions

Move
Increase time since infected (in days)
Shed prions
Become infected indirectly
Become infected directly
Die from CWD
Die from natural causes (baseline mortality)
Give birth
Die as young fawn
Die from hunting
Spring dispersal and migration
Fall dispersal and migration

Monthly functions

Increase age (in months)
Increase time since given birth (in months)
Environmental prion decay

Seasonal functions

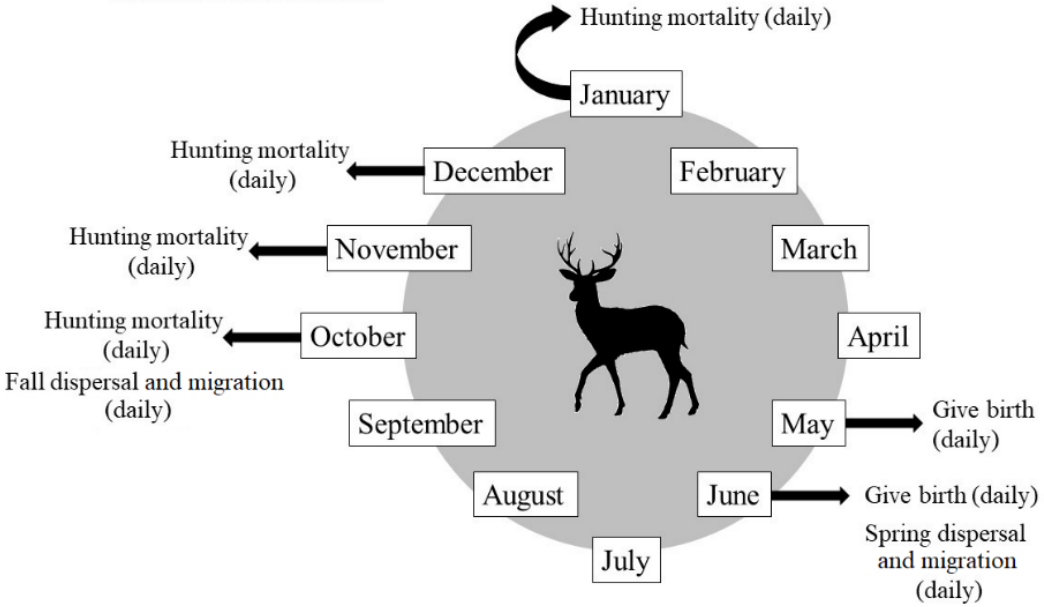


Figure 1.3. Map of usable habitat for deer across the 93-km^2 modeled landscape. Cells with a value of 0 (i.e., white cells) indicate unusable areas and cells with a value of 1 (i.e., black cells) indicate usable areas. Deer and group centers cannot be located within a cell that has a value of 0.

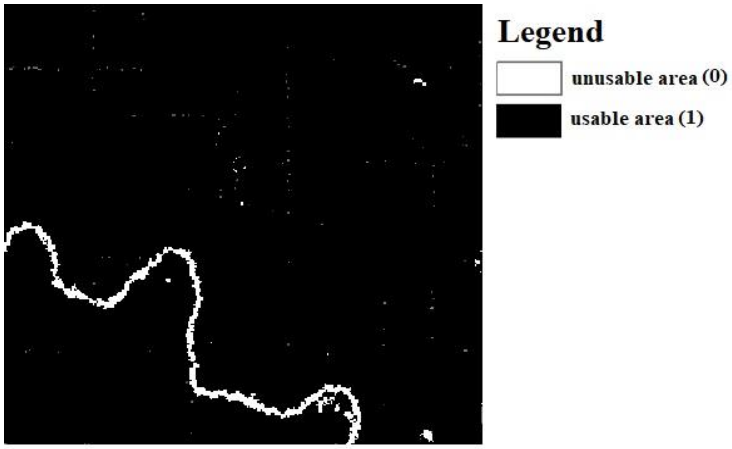


Figure 1.4. Map of habitat suitability for deer across the 93-km^2 modeled landscape. The left panel shows the original resource selection map based on Quinn (2010). The right panel shows a smoothed version using moving window analysis. For both maps, white indicates highly suitable areas for deer and black indicates low suitability areas.

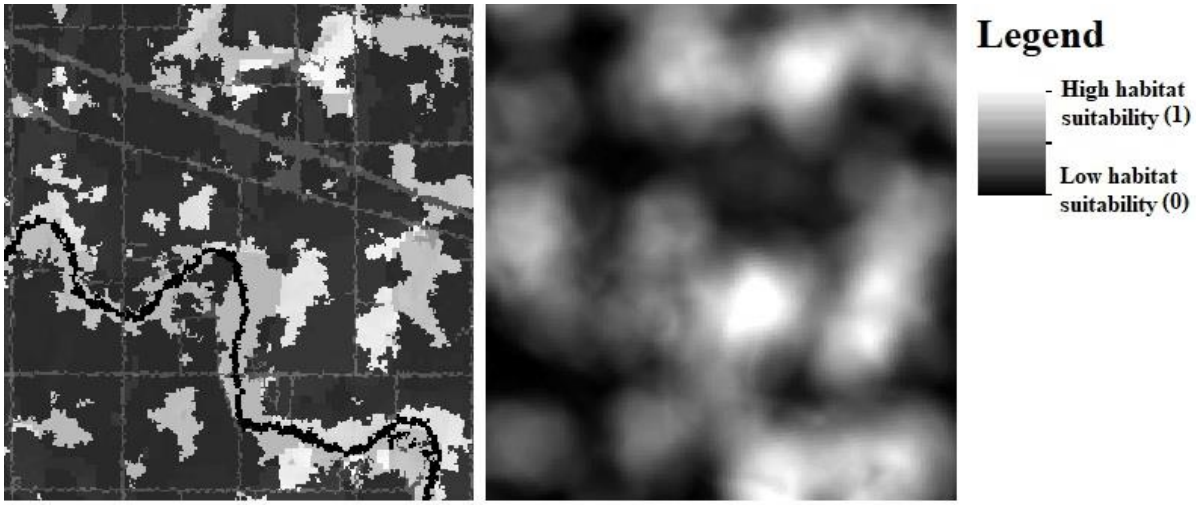


Figure 1.5. Average (with 95% confidence intervals) total population size (deer/93km^2) for 50 years predicted by the individual-based model for a Michigan white-tailed deer population unaffected by chronic wasting disease estimates from 350 model runs.

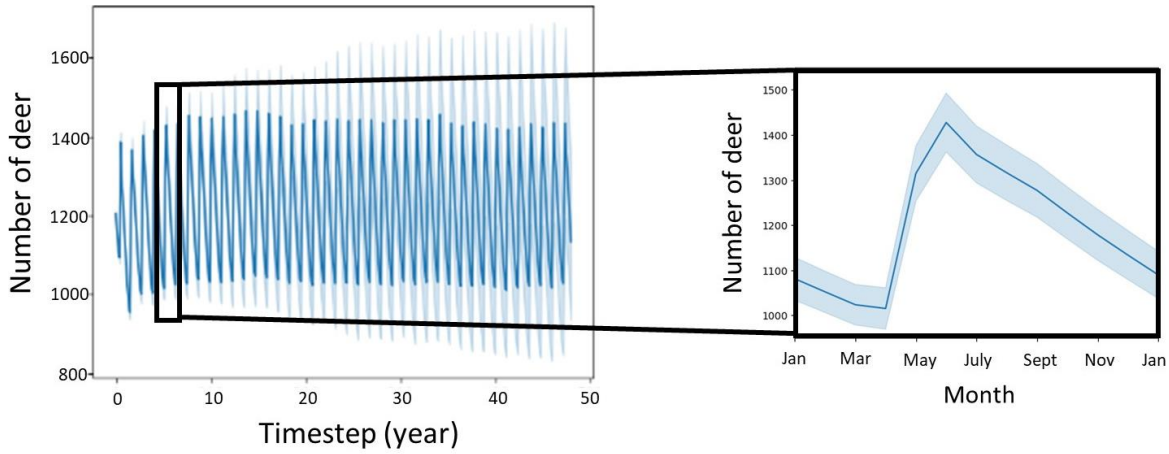


Figure 1.6. Annual prevalence rates of chronic wasting disease (CWD) in a Michigan white-tailed deer population simulated by the individual-based model and average apparent prevalence rates calculated for CWD-affected counties in Wisconsin. I calculate an average annual rate at the county level for each year following initial detection for Columbia, Dane, Green, Iowa, Lafayette, Richland, and Sauk counties (r=0.994; Fig. 1.6A). I calculate average annual rates at the township level for Dane, Iowa, and Sauk counties (r= 0.923–0.990; Fig. 1.6B). I calculated mean prevalence rates and 95% confidence intervals for the 100 simulated model repetitions that resulted in an outbreak of disease. The shaded areas indicate 95% confidence intervals for each dataset.

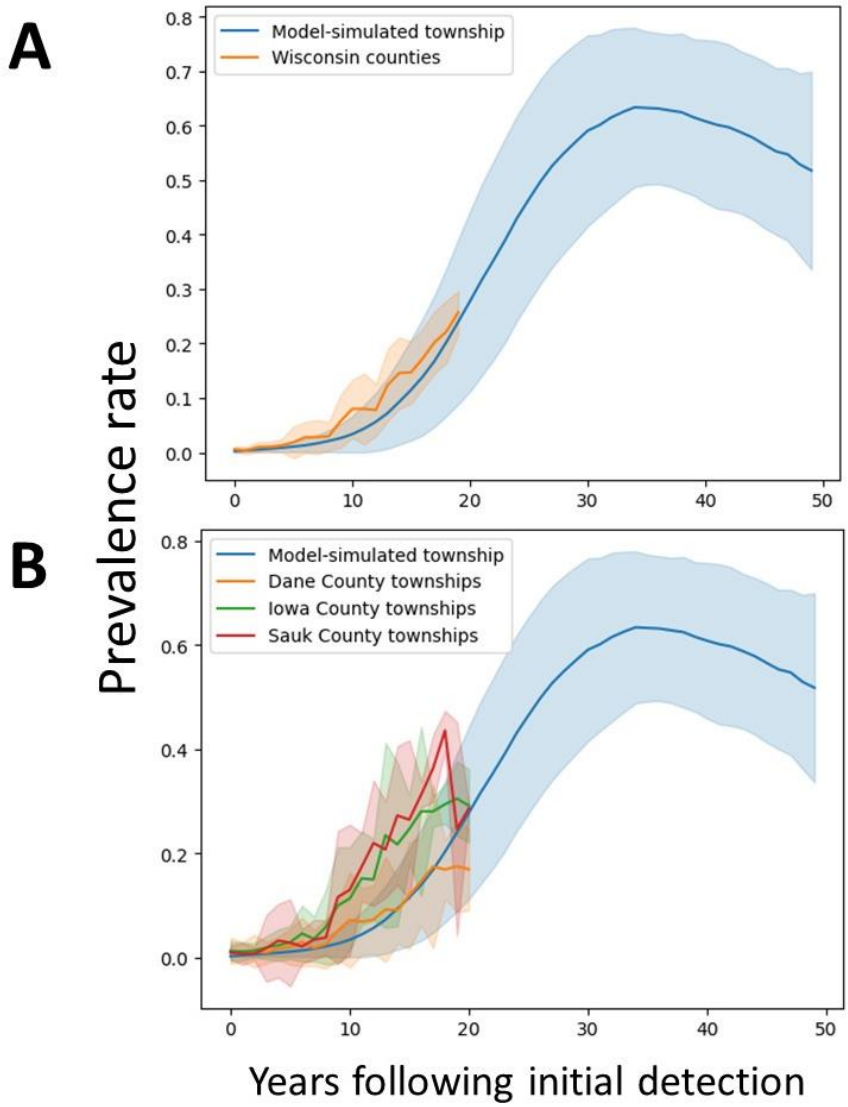


Figure 1.7. Location of modeled deer after the fifth, twenty-fifth, and fiftieth model year on the habitat selection (left panel) and environmental prion map (right panel). Deer are indicated by points on each map. Green points indicate deer unaffected by but susceptible to chronic wasting disease (CWD). Newly infected deer that are not yet infectious are indicated by orange points and infected and infectious deer are colored red. Infectious deer shed prions into the landscape, indicated by the change in colors of cells on the prion map. Cells change from black to dark brown to light brown in cells where prions are shed.

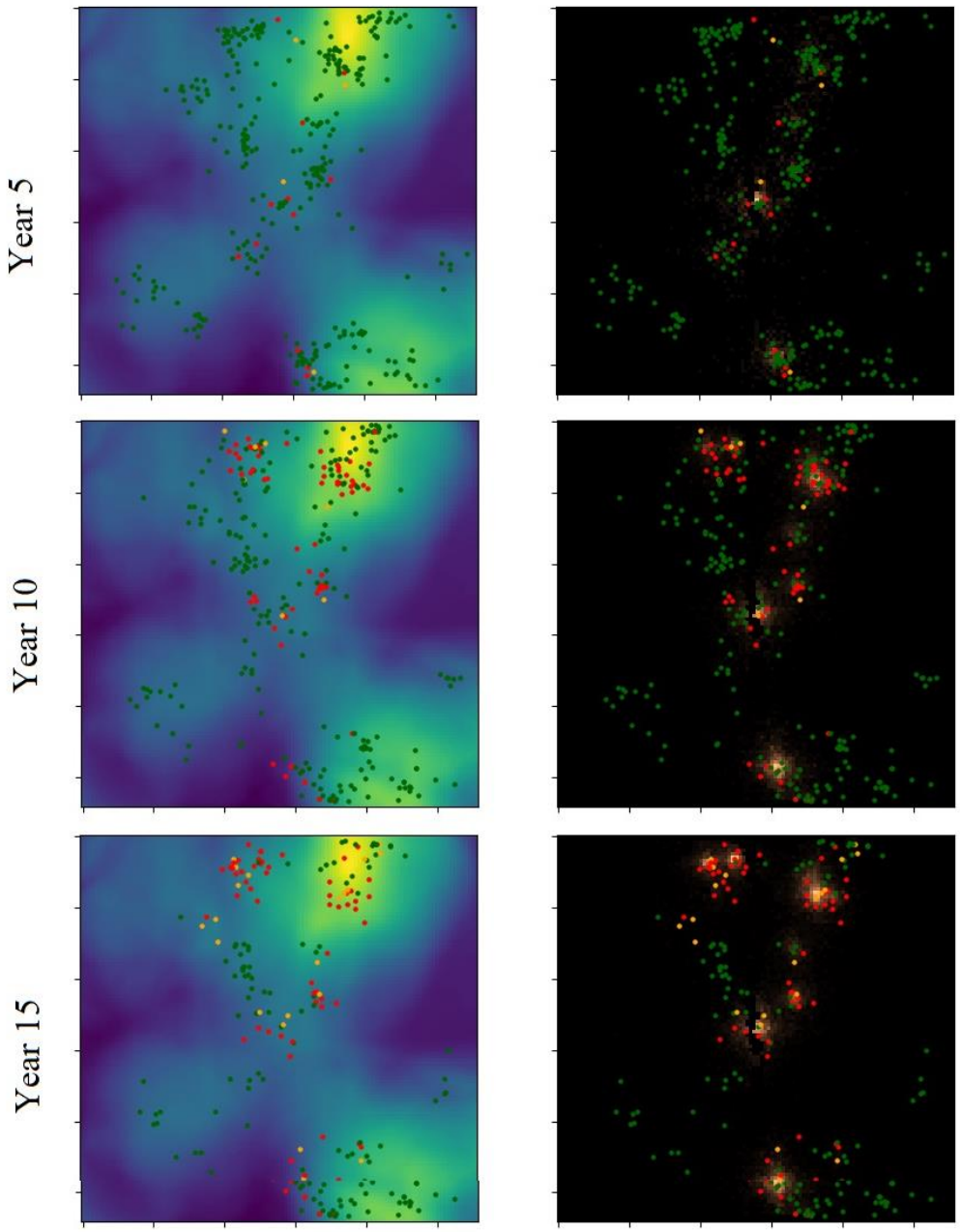


Figure 1.8. Average prevalence rate of chronic wasting disease (CWD) after year 50 in the individual-based model calculated iteratively across 500 model repetitions to assess convergence (i.e., stability) of model output. Model output stabilizes (i.e., average model output does not change by >0.001 with subsequent repetitions) at 322 model repetitions.

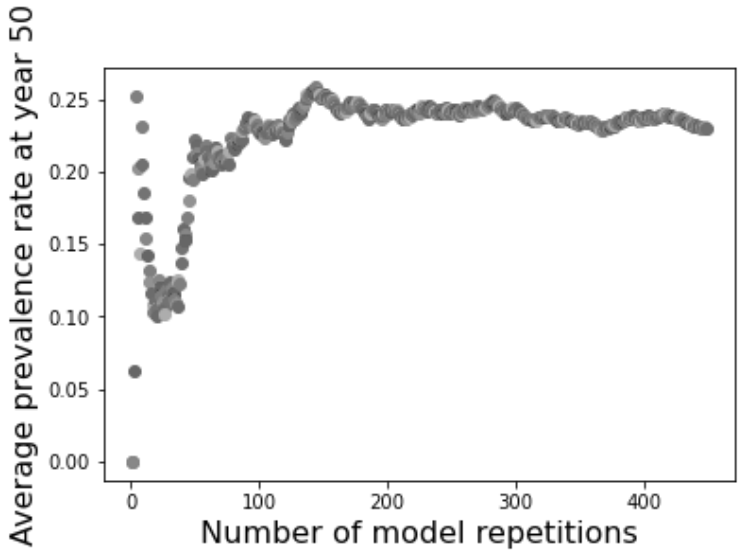


Figure 1.9. Annual mean values (and 95% confidence intervals) for total deer with chronic wasting disease (CWD) present in the population, total deer in an unaffected population, total infected deer, and total exposed deer simulated and averaged over the 100 simulations where CWD persisted in the modeled population in the individual-based model. The shaded areas indicate 95% confidence intervals around the annual mean values.

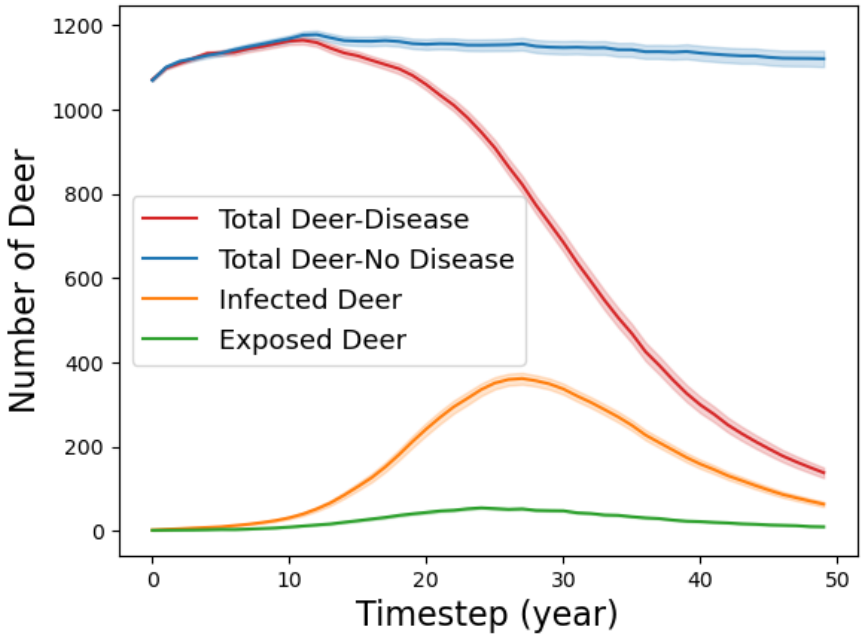


Figure 1.10. Annual sex-specific prevalence rates of chronic wasting disease in the white-tailed deer population simulated in the individual-based model. The shaded areas indicate 95% confidence intervals around the annual mean values.

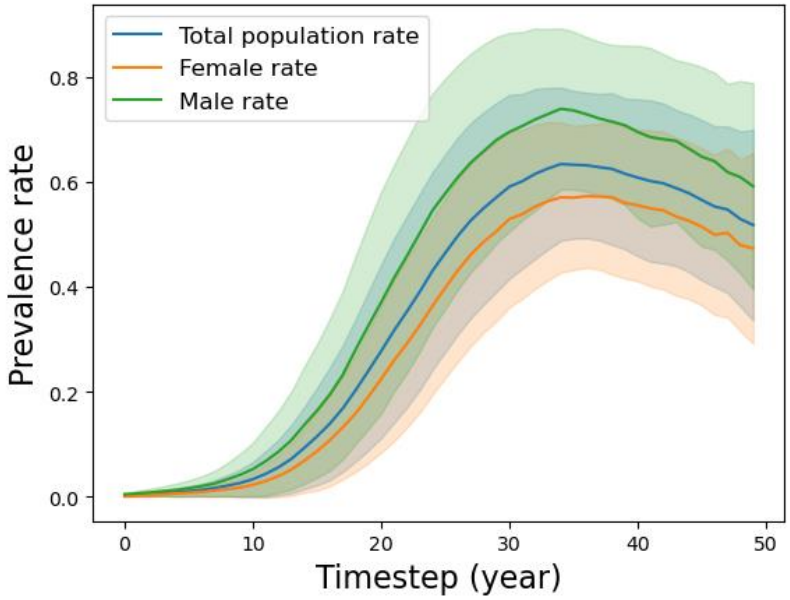


Figure 1.11. Cumulative number of direct and indirect transmission events occurring in a population of free-ranging white-tailed deer infected with chronic wasting disease (CWD) in the individual-based model. The solid lines indicate the mean total number of events after each year across the 100 repetitions of the model where persistence of CWD occurred. The shaded areas indicate 95% confidence intervals around the annual mean values.

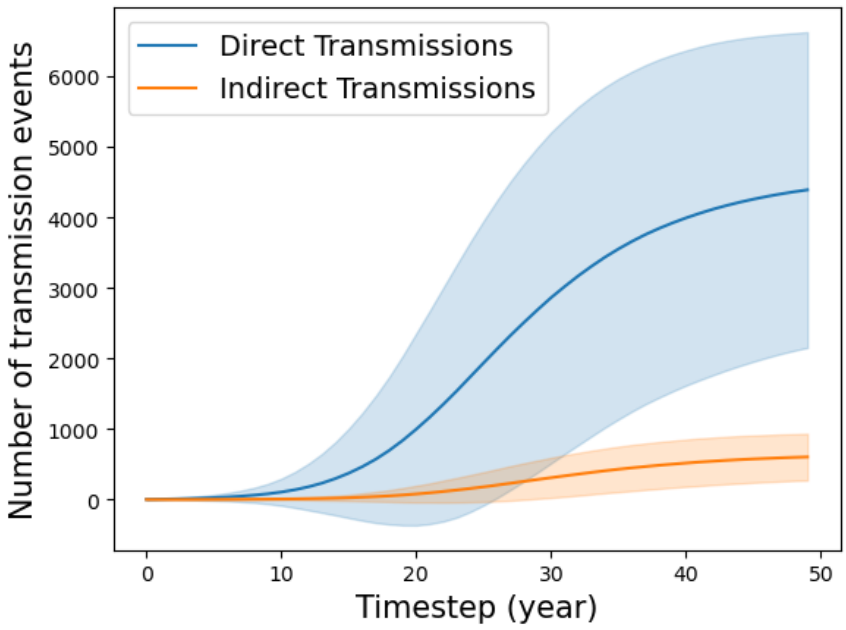
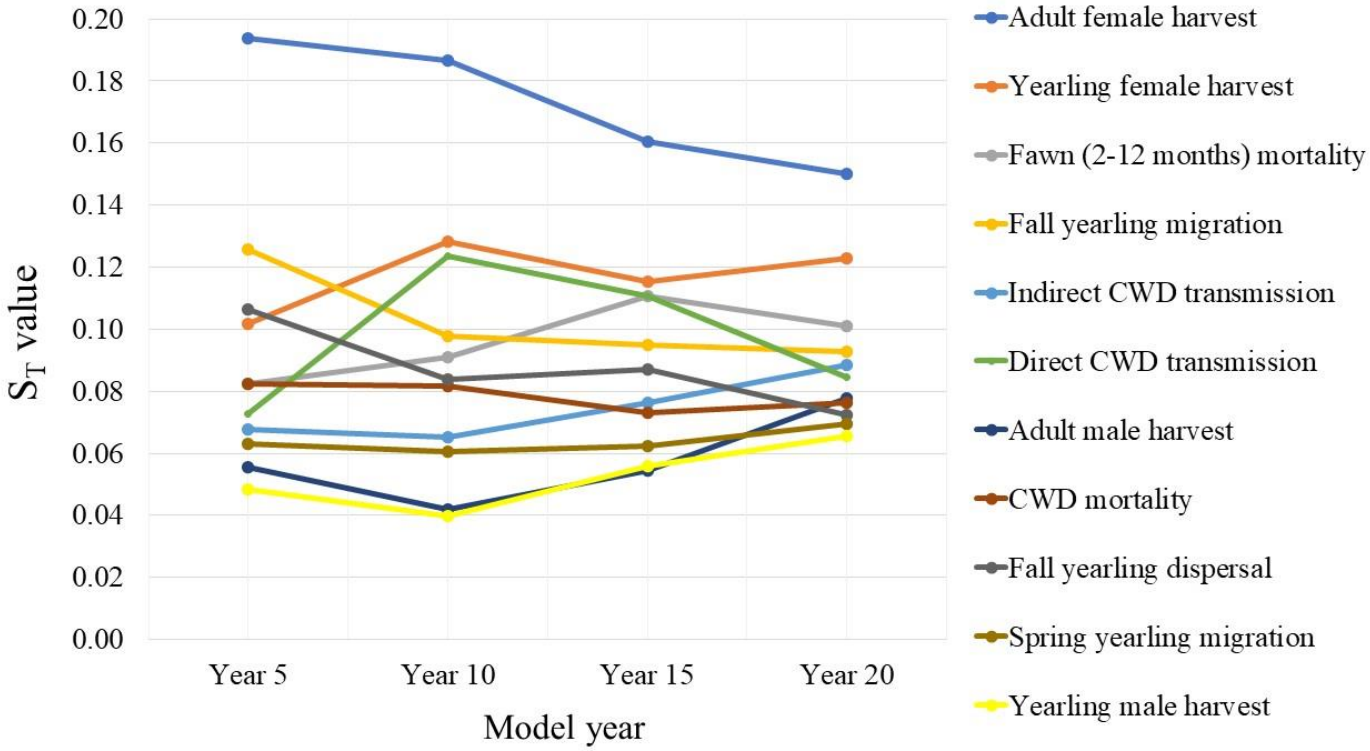


Figure 1.12. Changes in total effect indices (i.e., normalized S_T values) derived from the global sensitivity analysis, which applied a variance decomposition approach using Sobol′ sampling, indicating the total amount of variance in model output, chronic wasting disease (CWD) prevalence, attributed to each input factor across model years 5, 10, 15, and 20.



CHAPTER 2 ASSESSING LOCALIZED DEER REMOVAL STRATEGIES FOR MANAGING CHRONIC WASTING DISEASE

2.1 Introduction

Infectious diseases threaten wildlife populations across the world and are being detected at an increasing rate (Jones et al. 2008, Smith et al. 2009, Hatcher et al. 2012). To exacerbate this issue, identifying effective management for infectious wildlife diseases remains problematic because of limited understanding of these disease systems and ethical and financial constraints surrounding research and application of management options (Wobeser 2002, Cowled et al. 2012). Moreover, wildlife infectious diseases that have an environmental transmission component are particularly under-researched, are more difficult to control, and are becoming more prevalent (Tompkins 2015, Beeton et al. 2019).

Chronic wasting disease (CWD) is a disease within the family of transmissible spongiform encephalopathies (TSEs) that includes scrapie in sheep and goats (Detwiler 1992), bovine spongiform encephalopathy in cattle ("mad cow disease"; Holt and Phillips 1988), Creutzfeldt-Jakob disease in humans (Matthews 1978), and a newly identified prion disease in camels (Babelhadj et al. 2018). Chronic wasting disease is the only TSE that affects free-ranging wildlife, making management of this TSE particularly problematic. To add to this difficulty, there remains no treatment or vaccine (Pilon et al. 2013, Goni et al. 2015), surveillance in free-ranging cervids generally relies on post-mortem testing (Monello et al. 2013), there is a long incubation period (Williams and Miller 2002), and there is environmental transmission with the disease agent remaining stable outside of its host for numerous years (Miller et al. 2004).

Many of the cervid species susceptible to CWD, such as white-tailed deer (*Odocoileus virginianus;* hereafter "deer"), are generalists (Baker 1984, Long et al. 2005). The ability of deer

to adapt to rapidly changing environments creates dissimilarities in behavior among populations inhabiting different regions or landscapes. Differences in behavior typically include diet (Putnam 1988), movement (Verme 1983, Geist 1974, Marchinton and Hirth 1984), and space and resource use (Marchinton and Hirth 1984, Putman 1986). As a result of this heterogeneity, effective management of these populations often depends on understanding local differences in behavior and should not be generalized across an entire species or even multiple subpopulations. Therefore, because population management is context dependent, so is management of infectious disease transmission (Foster et al. 1997, Wobeser 2002, 2007, Fattorini et al. 2020).

Management to reduce transmission of CWD depends on understanding local deer contact structure, space use, and other behaviors unique to each host population (Ketz et al. 2019). As a result, success of a management action varies depending on local conditions of affected populations. However, past modeling efforts to assess management of CWD have not incorporated local contexts (Uehlinger et al. 2016, Rivera et al. 2019). For example, Jennelle et al. (2014) developed a multi-state deterministic matrix model that did not have a spatial component, nor did they incorporate environmental transmission. Similarly, a review conducted by Uehlinger et al. (2016) of studies that assessed management of CWD revealed that only 1 of 6 models incorporated demographic-specific parameter rates. Models used to predict management outcomes should incorporate or account for variability associated with local populations and the landscape in which they reside.

I use a spatially explicit individual-based model described in Chapter 1 to assess localized, fine scale deer removal strategies for CWD in a Midwestern white-tailed deer population inhabiting mid-Michigan, USA. The model describes the population and CWD dynamics within a free-ranging deer population including direct and indirect transmission of

CWD across real landscapes defined using geographic information systems software. For this study, I collaborated with the Michigan Department of Natural Resources (MDNR) and the United States Department of Agriculture's (USDA) Animal and Plant Health Inspection Service Wildlife Services and used the model to evaluate a suite of realistic management scenarios. My management categories included: 1) nonselective removal of deer around a center location (i.e., ring culling), 2) deer removal on pre-specified land parcels, and 3) deer removal on areas of highest-quality deer habitat. I then assessed how variability in landscape and deer density interacted with management to influence disease and population dynamics. Lastly, I was interested in determining what level of management was needed following CWD detection to reduce the probability of outbreak persistence to below either 1% or 10%.

2.2 Methods

I used an individual-based model that projects daily indirect and direct transmission of CWD to infer overall disease dynamics in free-ranging white-tailed deer populations, which is described in Chapter 1 of this dissertation. My study landscape consists of a 23.3-km^2 suburban area (43.2% developed, 32.5% wetlands, 8.6% agriculture, and 6.5% forested; NLCD 2020) and a 23.3-km^2 exurban area (81% agriculture, 10.5% wetlands, 5.3% developed, and 2.6% forested; NLCD 2020) in Ingham County, Michigan, USA (Fig. 2.1). These study areas are similar in size to those delineated by USDA Wildlife Services for localized removal of deer in response to spark occurrences of CWD (i.e., detections of CWD in new areas) in Michigan (E. Krom, personal communication).

At model initialization, the user defines the number of deer and groups to simulate. The centers of deer groups are placed across the study area randomly but guided by habitat criteria. Deer habitat for this area is based on a resource selection function developed from 2020 National

Landcover Database data (NLCD; Quinn 2010). Habitat usability and habitat suitability influence locations of group centers. Usability of each grid cell defines where deer and group centers can potentially occur and is based on a combination of NLCD urban imperviousness and open water classifications. Habitat suitability in each grid cell guides deer selective use of the landscape, with each grid cell assigned a habitat suitability value ranging from 0 to 1 (poor to high suitability; Quinn 2010; Fig. 1.3).

Each deer in the population is randomly assigned to a group and assigned a location in proximity to the center specified for that group. In the model, regular day-to-day home range movements by deer are not influenced by habitat suitability but the occasional shifting of deer groups is affected by habitat suitability, with groups being attracted to higher suitability habitats.

For all model scenarios and repetitions, a single time step is one day, and each month consists of 30 days. I introduced CWD in the 4th month of the first year (i.e., 120th time step) because spring dispersal most often occurs during the month of June for fawn and yearling males inhabiting mid-Michigan (Trudeau, unpublished). This dispersal event provides a mechanism for geographic spread of CWD into new areas. To simulate disease introduction, I randomly chose one deer group to place in the center of the study area and one deer in that center group to change disease status from susceptible to exposed during the 120th time step. An exposed deer is a deer that has been infected with CWD but is not yet infectious. Exposed deer turn infectious between the 3rd and 6th months (i.e., 90–180 days) following infection, with the specific day chosen at random by the model (Henderson et al. 2015).

For each management strategy assessed, deer removal (i.e., ‘culling’) was initiated in January of the year immediately following the year CWD was detected. Disease detection occurred when a deer infected with CWD was removed from the model by the hunter-harvest

mortality function with the assumption that all deer harvested in the study area were tested for CWD. The hunter-harvest mortality function introduces an additional daily probability of death between October 1 and January 31 for each deer equivalent to age-class and sex-specific rates reported in the literature (Table 1.2; Van Deelen et al. 1997).

I simulated deer removal daily for up to 90 days from January 1st through March 30th, the period during which USDA Wildlife Services is contracted to remove deer in the winter by MDNR (C. Stewart, personal communication). For each time step in the model (i.e., one day), a random number between 0 and 7 was chosen to determine the number of deer removed that day. The model chose deer at random to be culled and this probability was the same for each sex and age class of deer, unlike the hunter-harvest mortality function. I chose this range of numbers based on the average and range of numbers of deer Wildlife Services typically culls in a week (E. Krom, personal communication). I simulated removal of deer daily until March 30th or until approximately 75% of the original number of deer (i.e., the deer population at time 0) were left in the population. This corresponds to the estimated percentage of the population Wildlife Services removes in a season when contracted to perform localized culling of deer (E. Krom, personal communication). This also matches deer removal efforts conducted in Illinois, where the goal is to remove 25% of the deer from local populations (C. Jacques, personal communication). Culling was simulated only in the first year following CWD detection.

I assessed three types of localized deer removal methods with the objective to reduce deer abundance as management strategies for CWD on each study area: 1) ring culling at two scales (i.e., 1.6 and 2.4-km radii ring removal), 2) removal of deer in specific land parcels (i.e., parcel removal), and 3) removal of deer in high-quality deer habitat (i.e., high-quality habitat removal; Fig. 2.2). I also ran a baseline scenario where only deer removal via hunter-harvest was

performed each year using annual harvest rates reported for Michigan (0.16–0.42 depending on sex and age; J. Trudeau, unpublished). I applied the baseline scenario and each of the three deer removal scenarios to deer populations at three initial densities: 10, 14, and 19 deer/km^2. Hereafter, I refer to these density levels as low, medium, and high, respectively. I produced 350 repetitions for each of the 30 scenario combinations (5 removal strategies × 3 deer densities × 2 landscapes) to incorporate stochasticity in the model. Underlying population variables and demographics and model functions were held constant across the different deer removal scenarios and repetitions.

For the ring culling scenarios, the center of the ring was placed at the center of the study area (Fig. 2.2C). This ensured that the entire ring would fall within the study area, which improved consistency of deer removal across model repetitions. Depending on size specified for the ring, any deer within 1.6 or 2.4 km of the center of the study area could be culled and removed from the model at any time step during the removal period from January through March.

I used land parcel data for Ingham County, Michigan for the parcel deer removal scenarios. For both suburban and exurban study areas, I selected parcels randomly across the 23.3-km^2 area that covered 9.3-km^2 area, or 40% of the total area (Fig. 2.2A). Wildlife Services estimates a 20-50% success rate when seeking landowner permission to remove deer in a localized area (E. Krom, personal communication).

For the high-quality habitat deer removal scenarios, I used the underlying habitat suitability map divided into 30-meter grid cells to identify 40% of the cells with the highest habitat suitability values (Fig. 2.2; Quinn 2010). I chose 40% of cells for consistency in the amount of area exposed to deer removal across the parcel and high-quality habitat removal

scenarios. For each daily time step during the removal period, the model chose any deer located within cells with the highest suitability values, regardless of sex or age of the deer, to be culled.

For each removal strategy, landscape, and deer density combination, I documented the number of model repetitions out of 350 that resulted in persistence of the disease. For this study, I defined disease persistence as having at least one infected deer by the end of the 10-year model run, however, I acknowledge that identifying disease thresholds is rarely relevant for wildlife diseases because it is typically not possible for wildlife managers to measure the true extent of disease in wildlife populations. Therefore, policies should not be centered on pre-defined thresholds (Lloyd-Smith et al. 2005). In addition, I identified the number of repetitions where the disease was detected and, thus, culling was performed the following year. I also identified how frequently disease detection occurred via the hunter-harvest function during each model year.

For each model repetition, I extracted daily prevalence of CWD but calculated average prevalence rates for each scenario only using the repetitions that resulted in disease persistence. I also calculated average values for the total number of direct transmission events, indirect transmission events, infected deer that dispersed outside of the study area (i.e., emigrants), deer culled, final deer population size, and proportion of cells in the study area that contained deposited prions from infected deer after model year 10.

Based on preliminary results of the deer removal scenarios where 25% deer removal rates were applied in the model, I investigated population reduction incrementally at three additional levels to identify the proportion of the deer population needing to be removed to reduce probability of disease persistence to 1% and 10%. I removed 33.3%, 50%, and 66.7% of the population in January of the first year following the year of disease detection for a subset of removal scenarios (i.e., the 2.4-km ring and high-quality habitat removal types) at the high and

low deer density levels and for each landscape type. I calculated disease persistence rate, final prevalence rate for those repetitions when disease persisted, and the failed cull rate, which I define as the percentage of repetitions where deer removal was performed but the disease persisted in the population.

Using Program R version 4.3.1 (R Core Development Team 2022), I applied a conditional negative binomial modeling approach using a Bayesian framework to assess the effects of landscape type, initial deer density, and deer removal strategy on two disease outcomes of interest: 1) disease persistence and 2) the number of infected individuals given persistence of CWD. Preliminary results indicated that, on average, more than half of model repetitions resulted in extirpation of the disease. Thus, I expected the response variable data, both disease persistence and number of infected deer, to have excess zeros and be of little value in assessing disease outcomes where repetitions did not result in CWD persistence.

I treated the first response variable, persistence of CWD, as binary. Model repetitions where CWD persisted were given a '1' whereas repetitions where the disease was extirpated resulted in a value of '0.' The second response variable, number of infected deer, was conditional on the first: whether the disease persisted in the population and, thus, produced a non-zero value for the first outcome. Hurdle models are ideal for datasets where one outcome depends on the presence or absence of another (i.e., the 'hurdle') and for datasets that produce excess zeros because it separates the outcomes by creating two datasets based on whether the criteria considered the hurdle was 'crossed' (Mullahy 1986). Outcomes where the hurdle condition was met and non-zero values were produced for both disease response variables were subset and modeled separately (Dalrymple et al. 2003).

To assess the data within a Bayesian framework, I first created a design matrix for the

predictors and simulated datasets in the Bayesian hurdle model by developing a zero-truncated negative binomial regression model that applies the conditional ('hurdle') approach within a frequentist framework. I developed this model using the hurdle function provided by the PSCL package (v 1.5.5.1; Jackman 2020) in R and specified a negative binomial data distribution:

$$P(Y_i = y_i) = \begin{cases} p_i & if\ y_i = 0 \\ \frac{1-p_i}{1-\left(\frac{r}{\mu_i+r}\right)^r} \frac{\Gamma(y_i+r)}{\Gamma(r)y_i!} \left(\frac{\mu_i}{\mu_i+r}\right)^{y_i} \left(\frac{r}{\mu_i+r}\right)^r & if\ y_i \geq 1, \end{cases}$$

$$\log(\mu_{ij}) = \beta_0 + \beta_1 D_{ij} + \beta_2 L_{ij} + \beta_3 R_{ij} + \beta_4 D_{ij} L_{ij} + \beta_5 L_{ij} R_{ij} + \beta_6 D_{ij} R_{ij} + \beta_7 D_{ij} L_{ij} R_{ij} + \log(T_{ij}),$$

$$\text{logit}(p_{ij}) = \beta_0 + \beta_1 D_{ij} + \beta_2 L_{ij} + \beta_3 R_{ij} + \beta_4 D_{ij} L_{ij} + \beta_5 L_{ij} R_{ij} + \beta_6 D_{ij} R_{ij} + \beta_7 D_{ij} L_{ij} R_{ij} + \log(T_{ij}),$$

where y_{ij} represents the response of the first disease outcome, disease persistence, for the i^{th} model repetition in the j^{th} deer density-study area-deer removal method scenario combination; Y_{ij} represents the response for the second disease outcome, number of infected deer; p_i indicates the probability that $y_{ij} = 0$ given Y_{ij}; D_{ij} indicates the deer density level; L_{ij} indicates the study area type; R_{ij} indicates the deer removal method; T_{ij} indicates an offset variable, the natural log of the total number of deer; and $\beta_0 - \beta_7$ are regression coefficients. Negative binomial regression models require response variables in the form of count data. Thus, I used the number of CWD-infected deer as the response variable and offset those values by the natural logarithm of the total number of deer in the model to assess final prevalence rate.

I implemented the model within a Bayesian framework by using a Markov Chain Monte Carlo method and simulated posterior distributions using the Nimble package in R (NIMBLE Development Team 2023). For each model, I sampled from three Markov chains that ran for 50,000 iterations each and excluded a burn-in period of 10,000 iterations. I looked for evidence

of non-convergence of each chain by examining standard diagnostic plots for each parameter.

2.3 Results

Chronic wasting disease was most often detected through hunter harvested deer (27.5% of model runs) in year 2 of the model, which was the year immediately following its introduction into the deer population (Table 2.1). Given that culling was initiated the year following CWD detection, culling most often occurred in model year 3. After initial detection, CWD continued to be detected from hunter harvested deer for the next nine years of the model across the 30 scenario combinations (Table 2.1). The proportion of detection events occurring in each year decreased with time, with the second year consisting of the second most frequent detection events (mean=24.4%, 95% CI=23.9–31.1%). Detection of CWD was possible in the first year but never occurred (Table 2.1). Disease detection occurred more frequently in later years in scenarios with greater deer density levels.

For management scenarios in an exurban landscape, average final CWD prevalence rate (i.e., prevalence rate at the end of model year 10) ranged from 4.0% (1.6-km ring cull for medium deer density) to 11.5% (no culling for high deer density; Table 2.2). In a suburban landscape, average 10-year CWD prevalence ranged from 1.6 % (1.6-km ring cull for medium deer density) to 6.1% (parcel-based culling for medium deer density; Table 2.3). For all scenarios in the exurban landscape, 1.6-km ring culling resulted in lowest CWD prevalence for 10 years (Fig. 2.3). In the suburban landscape, the 1.6-km ring resulted in lowest 10-year CWD prevalence at low and medium deer densities, but 2.4-km ring culling resulted in lowest prevalence at high deer density (Table. 2.3).

Percentage of model repetitions resulting in persistence of CWD (i.e., at least one infected deer by the end of the 10-year model run) ranged from 2% to 53% (Tables 2.2, 2.3) in

an exurban landscape. In the exurban landscape, 1.6-km ring culling produced the lowest CWD persistence rates for each deer density (11–18%) whereas no culling produced the greatest rates (45–53%; Table 2.2). For the suburban area, 1.6-km ring culling produced the lowest CWD persistence rates for each deer density (2–6%; Table 2.3). The highest rates were produced by no culling (33% and 34% for low and medium deer density, respectively) and high-quality habitat culling (33% for high deer density; Table 2.3).

The average percentage of the study area affected by CWD (i.e., containing shed prions) was highest when no culling occurred for suburban and exurban landscapes and all three deer densities (5.2–11.7%; Tables 2.4, 2.5). Ring culling at 1.6-km produced the lowest percentage of affected study area for all densities and landscapes (3.7–6.2%; Tables 2.4, 2.5) except at high deer density in the suburban study area (4.8%; Table 2.5). At high deer densities, 2.4-km ring-culling resulted in lowest percent of CWD in the landscape (Table 2.5).

Study Area

Disease persistence was always greater for model scenarios in the exurban study area (relative to the suburban study area; Fig. 2.3). Similarly, final prevalence of CWD was greater in the exurban study area for 13 out of the 15 deer density-removal method combinations (Fig. 2.3). Mean total number of direct transmission events was always greater in the exurban study area (Tables 2.2, 2.3), even for the few scenarios where final prevalence rate was greater in the suburban landscape. Mean numbers of indirect transmission events were greater in the exurban study area for 13 of 15 scenarios, although these averages were more similar between the two study area types than observed for direct transmission events (Tables 2.4, 2.5).

Mean percentage of the study area containing shed prions after 10 years was small (<15%) for all 30 scenario combinations but was always greater in the exurban study area

(Tables 2.4, 2.5). Mean values for total number of deer culled for each scenario were similar between the two study areas with differences ≤ 4 deer, although standard deviation was lower in the suburban study area (Tables 2.2, 2.3). Mean number of infected deer that dispersed away from the study area (i.e., infected emigrants) was minimal for all scenarios (≤ 3 deer; Tables 2.4, 2.5). However, for scenarios without culling and scenarios applying the parcel culling method, more infected emigrants were reported in the exurban study area for 2 of 3 deer density levels (Tables 2.4, 2.5).

Results of the Bayesian hurdle model revealed that study area influenced probability of disease persistence and final prevalence rate. The suburban study area had a lower probability of CWD persistence than the exurban study area (Table 2.6). Similarly, when CWD persisted, prevalence rates were lower in the suburban study area (Table 2.7). Diagnostic plots and statistics calculated using the Gelman and Rubin approach (GELMAN.DIAG; Gelman and Rubin 1992) to assess within- and among-chain variance for each parameter in the hurdle model are provided in Appendix C.

Deer Density

In general, a greater number of repetitions resulting in persistence of CWD and larger mean values for final prevalence rates were observed in scenarios with medium and high deer density levels for each management scenario and study area (Tables 2.2, 2.3; Figs. 2.3, 2.4). The range of mean values for final prevalence rate among management strategies was greatest for populations at the highest density level (3.1–11.5%; Tables 2.2, 2.3).

The number of direct transmission events increased with increasing deer density for 8 of 10 management scenario-study area combinations (Tables 2.2, 2.3). For the baseline scenario with no deer culling, the mean number of direct transmission events increased by 55% and 19%

in the exurban and suburban study areas, respectively. Doubling of deer density (from low to high in this study) resulted in more direct transmission events by 11–55% for each of the scenarios (Tables 2.2, 2.3). For the scenarios with culling, increases in direct transmission events ranged from 11–47%, with larger increases in the exurban study area for each deer removal method (Tables 2.2, 2.3).

Doubling deer density (from low to high in this study) increased the number of indirect transmission events for 4 of 5 deer removal scenarios in the exurban study area, and for 2 of 5 scenarios in the suburban study area (Tables 2.4, 2.5). For scenarios resulting in more indirect transmissions of CWD, mean number of events increased by 36% (n=4) and 44% in the exurban study area and 10% and 11% in the suburban study area.

Mean values for percentage of the study area containing shed prions also increased with increasing deer density levels (Tables 2.4, 2.5). The percentage was always greater for scenarios at the high deer density level compared to the low-density level. However, this trend was not consistent between the low and medium deer density levels (Tables 2.4, 2.5).

The posterior distribution summary statistics produced by the Bayesian hurdle model generally indicated that deer density alone did not affect CWD persistence or prevalence rate (Tables 2.6, 2.7). The exception was for higher prevalence rates for medium compared to low deer density (Table 2.7).

Deer Culling Method

Average 10-year CWD prevalence rate was consistently highest (9.5–11.5% prevalence) for scenarios without culling in the exurban study area (Table 2.2). The 10-year CWD prevalence for culling scenarios ranged from (4.0–9.0%; Table 2.2). This pattern was not observed for scenarios from the suburban area. At low deer density, the lowest mean 10-year CWD

prevalence (5.1%) was observed for the baseline scenario, whereas the parcel culling method resulted in the highest rate (6.1%; Table 2.3). At medium deer density in the suburban area, parcel and high-quality habitat culling resulted in highest 10-year CWD prevalence rates (4.6%), whereas the 1.6-km ring cull method produced the lowest rate (1.6%; Table 2.3). At high deer density, 10-year CWD prevalence was highest for the high-quality habitat cull (5.1%) and lowest for the 2.4-km ring cull (3.1%). The 1.6-km ring cull consistently produced the lowest prevalence rates across deer density levels in the exurban study area (Table 2.3).

The 1.6-km ring cull resulted in lower disease persistence for both study areas and across deer density levels (2–18% of repetitions; Tables 2.2, 2.3). The baseline no culling method resulted in highest disease persistence in both study areas and across all deer densities, except for the suburban study area at high deer density. For this scenario, the high-quality habitat cull generated 10-year CWD persistence in 33% of simulations, whereas CWD persistence for the no-cull appeared in 31% of simulations (Table 2.3).

The fewest mean number of direct and indirect transmission events consistently occurred for the 1.6-km ring cull method (Tables 2.2, 2.3). Direct and indirect transmission were most frequent in scenarios where culling was not implemented (Tables 2.2, 2.3). The 1.6-km ring cull always resulted in the smallest percentage of study area affected by CWD (3.7–6.2%) and no culling always produced the largest percentage (5.2–11.7%; Tables 2.2, 2.3).

Relative to the baseline deer removal treatment, the four deer cull methods had a negative influence on CWD persistence, with the 1.6-km ring cull having the largest negative impact (Table 2.6). Final prevalence rate of CWD was influenced by 3 of 4 culling methods when compared to the baseline scenario; the land parcel cull did not differ from baseline (Table 2.7).

Interactions among factors

Two-way interactions between culling method, study area, and deer density were influential on final prevalence rate of CWD (Table 2.7) but had no effect on CWD persistence (Table 2.6). The parcel and high-quality habitat culling methods interacting with high deer density were the only 2 of 8 interactions that influenced CWD prevalence and persistence (Tables 2.6, 2.7). None of the interactions among culling methods and study area were influential on probability of disease persistence but they all influenced prevalence rate (Tables 2.6, 2.7).

Increasing the proportion of the deer population culled from 25% to 66% consistently lowered CWD persistence rate when applying 2.4-km ring culling for suburban and exurban landscapes and high and low deer densities and for high-quality habitat-based culling in the exurban landscape (Fig. 2.5). Conversely, increasing the proportion of the population culled did not decrease the probability of CWD persistence for scenarios applying the high-quality habitat-based culling method in the suburban study area (Fig. 2.5). Similarly, increased deer removal rates that applied high-quality habitat culling in the suburban area did not reduce the cull failure rate (i.e., culling still resulted in CWD persistence), whereas the ring cull method (both densities and landscapes) was able to reduce the probability that CWD persisted following culling (Fig 2.6). Increased proportions of the deer population culled decreased 10-year prevalence rates for both deer removal methods and deer densities in the exurban area except at the 67% removal rate, where sample size was small (Fig. 2.7). Prevalence rates increased with increased deer removal rates in the suburban landscape (Fig. 2.7).

Removing at least 50% of the deer population reduced probability of CWD persistence and cull failure rate to approximately 10% or less for the ring cull method (both deer densities and landscape) and high-quality habitat-based cull method in the exurban area. Removing 67% of the population was required to reduce the probability of disease persistence and cull failure

rate to ≤ 1%. Increasing the proportion of the population culled with the high-quality habitat cull method in the suburban area could not reduce the probability of CWD persistence nor cull failure rate (Figs. 2.5, 2.6).

2.4 Discussion

The simulation model indicated that culling deer, when added to hunter harvest, can reduce CWD prevalence over time and reduce probability of a CWD persistence. However, culling method has significant effects on CWD outcomes. For example, a ring-cull method was consistently more successful than parcel or habitat quality-based culls. Additionally, local conditions such as deer density and landscape type affect the success of CWD management regardless of cull strategy. For example, less culling effort was required in the suburban landscape to lower CWD prevalence over time. Despite culling more deer, CWD persistence and prevalence were marginally affected by high-quality habitat culling in the suburban study area. Conversely, removing more deer reduced CWD persistence and prevalence in the exurban study area regardless of cull method.

Extent of management needed to affect CWD dynamics depends on landscape context. Baseline model scenarios without culling revealed that persistence and prevalence of CWD across years was consistently lower in the suburban study area. Underlying population and disease processes and management scenarios were modeled identically between study areas, so differences in CWD dynamics can be attributed to how simulated deer are using the landscape. In the suburban study area, patches of unusable or low-quality areas existed and can constrain deer movements resulting in CWD persistence (Crawford et al. 2018). O'Hara Ruiz et al. (2013) found higher risk of CWD for deer occupying areas with larger and more intact forests (and correspondingly less urban and agricultural lands). The exurban study area in my simulations

was 2.6% forested, whereas the suburban study area was 6.5% forested; however, both areas assessed in this model consisted of less forested areas than the study area in Illinois (~27%; O'Hara Ruiz et al. 2013). Deer group members were modeled as more concentrated in the suburban study area because of lower amounts of usable habitat compared to exurban areas, but interactions among these deer groups were potentially reduced by the fragmented landscape, resulting in slower growth and geographic spread of CWD and lower persistence. Model results on percentage of the study area affected by CWD support this hypothesis; scenarios from the suburban study area always resulted in a smaller affected area. Another hypothesis for lower CWD persistence and spread in suburban areas is that despite more patches of unusable habitat in the suburban study area, more high-quality habitat concentrated deer in smaller areas. The simulation model results indicate that regardless of the reason, landscape context affects the spatial structure of deer and deer groups, which in turn affects growth and spread of CWD. It is more important to consider how patch configuration and quality of the landscape facilitates or hinders concentration of deer and group overlap across the entire area of interest (Tian et al. 2022).

Modeling indicated that increasing deer density did not influence prevalence of CWD nor its persistence in the population. I only investigated relatively high deer densities in this study where even the 'low' deer density level (10 deer/km^2) may already saturate the landscape with deer to the extent that higher densities become indistinguishable in the IBM. Statistically significant negative interactions between density and deer removal method compared to the intercept (low density) for prevalence but not persistence of CWD produced by the hurdle model support this explanation. Culling may be able to reduce prevalence rate over time but not affect the probability that CWD will persist because it cannot remove enough deer to reduce the

population to a level where CWD is likely to be extirpated.

When comparing deer culling strategies, the 1.6-km ring cull was most likely to reduce CWD prevalence and probability of persistence in exurban and suburban landscapes for all deer density levels. The ring cull methods are less geographically restricted compared to land parcel and high-quality habitat culls; it appears that deer culling is more effective at managing CWD when efforts remove deer uniformly across the targeted area. I hypothesized that high-quality habitat culling would be as effective, if not more, than ring culls. In the model, dispersing and immigrating deer and shifting social groups are more likely to move into areas containing higher-quality habitat, but habitat quality does not affect daily movements, as observed in the field data on which the movement model was trained (see Chapter 1; Butts et al. 2022). The influence of habitat quality on daily deer movement remains unclear (Massé and Côté 2013). If habitat quality does affect short-term deer movements, the utility of culling based on deer habitat quality may be disadvantaged in my simulation model.

Simulation model results also indicate that when responding to an emergent, localized case of CWD, chances of controlling CWD prevalence and reducing persistence increase if culling focuses on deer closer to the origin of the disease outbreak as opposed to removing deer from a larger area. The 1.6-km ring cull outperformed the 2.4-km ring cull in both exurban and suburban landscapes. When the general location of an outbreak is known, culling closer to this location likely results in higher probability that infected deer will be removed. Chronic wasting disease likely diffuses from the original infection site; thus, infected deer are more likely to be closer to the center of the outbreak in early years (Hefley et al. 2017). In addition to being more effective at mitigating the spread of CWD, performing localized culling across a smaller area may save time and resources. However, return on culling investment decreases at a faster rate as

deer numbers get lower, and costs per culled deer peak at extremely low density (Van Deelen and Etter 2003).

My simulation model indicated that current localized deer culling efforts performed by Midwestern wildlife agencies in response to new detections of CWD are not sufficient to reduce the probability of CWD persistence below 10%. Similarly, model simulations identified that the culling method wildlife agencies implement most often cannot reduce prevalence rates below rates expected when culling is not performed. Land parcel-based culling (culling delineated by property boundaries on 40% of study area with 25% deer removal) was ineffective at reducing CWD prevalence, yet this is often the only culling option available to wildlife agencies in new CWD outbreak areas. When CWD is detected in free-ranging deer populations that inhabit private lands, landowner permission is required by agency staff to lethally remove deer from their property. If the ring cull method is not feasible for an agency, I suggest increased effort towards obtaining land access and rotating removal efforts among accessible properties. As such, agencies will achieve greater spatial coverage where deer are culled and have a higher probability of culling deer that are infected. Although spreading out deer removal efforts across a greater number of properties requires more resources, it is likely more socially acceptable because fewer deer are culled from any given property. Wildlife Services staff indicate that it is difficult to maintain landowner permission for culling over time as landowners notice fewer deer on their properties and become increasingly concerned with local extirpation (E. Krom, personal communication).

In this model, 100% of deer harvested by hunters are tested for CWD, likely allowing for quicker detection of the disease than what is realistic for wildlife agencies. With earlier detection of CWD, management can begin sooner which can lead to higher probabilities of local

eradication of CWD and lower prevalence rates over time (Samuel 2023). In addition, disease dynamics for CWD remain generally unknown (Jenelle et al. 2014, Haley and Hoover 2015, Uehlinger et al. 2016), which creates uncertainty in disease parameter estimates and inter-variable relationships within the model. Aspects of the simulation model were verified and validated (see Chapter 1), and the model reasonably predicted CWD prevalence measured from field data. The model includes the most up-to-date information on CWD and allows specification of local deer population size, landscape context, and disease characteristics, providing a useful framework to assess management outcomes across various scenarios.

Management of CWD is costly, requires significant personnel time, and is often politically charged (Heberlein 2004, Holsman et al. 2010, Wolfe et al. 2004, 2018). As a result, field studies to compare localized deer removal techniques on CWD dynamics have not been conducted. For the same reasons identifying effective management strategies through trial and error in the field is generally not a sustainable option. The ability to predict management outcomes for CWD through application of models prior to implementing management can save wildlife agencies time, money, and potentially political tumult.

To my knowledge, this is the first modeling study to compare efficacy of fine-scale, localized management scenarios for CWD. Although this study is not the first to model management scenarios for CWD, previous efforts focused on hunter harvest-based deer removal (Al-Arydah et al. 2016, Potapov et al. 2016), did not include both direct and indirect transmission of disease in their model (Jenelle et al. 2014, Oraby et al. 2014, Belsare and Stewart 2020), and did not compare outcomes across multiple management strategies (Uehlinger et al. 2016, Winter and Escobar 2020). Similar to findings from this study, Al-Arydah et al. (2016) found that low levels of deer removal are not effective in controlling CWD. Al-Arydah et al.

(2016) identified a range of harvest rates that controlled CWD without extirpating local deer populations.

To date, there have been one prospective and few retrospective studies that investigated localized deer removal efforts as a method for CWD management in free-ranging cervid populations. A 'test-and-cull' strategy for reducing CWD in mule deer in the western U.S. was found to be ineffective (Wolfe et al. 2018). A long-term localized deer removal program in Illinois was evaluated in two retrospective studies. Both studies found that the removal program was effective at reducing prevalence of CWD in free-ranging white-tailed deer herds but did not reduce persistence or eliminate the disease entirely (Mateus-Pinilla et al. 2013, Manjerovic et al. 2014). Contrary to the findings from Illinois, a before-after-control-impact study performed in Colorado to compare impact of localized culling on mule deer populations determined that localized culling was ineffective at reducing prevalence of CWD (Conner et al. 2007).

My analysis provides an initial perspective for assessing management strategies for CWD in free-ranging cervid populations. Next steps may include investigating selective removal of adult male deer, which have highest documented prevalence of CWD (Grear et al. 2006, Osnas et al. 2009). I was able to account for reported differences in prevalence of CWD between sexes of white-tailed deer and corresponding movement and disease transmission behaviors (Jenelle et al. 2014, Oraby et al. 2014) by including a correction factor multiplied by the direct transmission rate for male deer (see Chapter 1). The simulation model can also be used to assess other disease management scenarios, such as changes to hunter-harvest regulations and regulations banning the baiting and feeding of cervids.

Table 2.1. Means and 95% confidence intervals for percentage of model repetitions (n=350) when chronic wasting disease detection occurred through hunter-harvested deer by year for 30 deer management scenario treatments (3 deer densities*2 landscapes*5 deer cull methods).

Model Year	Mean	95% Confidence Interval
Year 1	0%	0%
Year 2	27.5%	23.9–31.1%
Year 3	24.4%	23.2–25.6%
Year 4	11.7%	10.8–12.6%
Year 5	5.8%	5.2–6.3%
Year 6	2.8%	2.4–3.1%
Year 7	1.6%	1.5–1.9%
Years 8-9	< 1%	< 1%

Table 2.2. Percent of individual-based model repetitions (n=350) from an exurban landscape resulting in chronic wasting disease (CWD) being detected in at least one deer at the end of a 10-year model simulation. Each deer population was infected with CWD in year 1 of model simulations. Model scenarios for three deer density levels: Low (10 deer/km^2), Medium (14 deer/km^2), and High (19 deer/km^2). Mean (standard deviation) for CWD prevalence rate after model years 5 and 10, total numbers of direct transmission events, and deer culled for those model repetitions where CWD persisted for each management scenario.

Model Output	Baseline (No Culling)	High Quality Habitat Culling	1.6-km Ring Culling	2.4-km Ring Culling	Parcel-based Culling
Low Deer Density					
Disease persistence (%)	45	28	11	28	27
Year 5 CWD prevalence (%)	3.4 (2.9)	2.7 (2.8)	1.2 (1.2)	2.6 (2.3)	2.6 (1.7)
Year 10 CWD prevalence (%)	9.2 (6.8)	7.4 (6.3)	4.2 (1.9)	6.5 (4.8)	9.0 (5.9)
Direct transmissions	34 (33)	13 (18)	5 (6)	12 (15)	15 (11)
Deer removed	0	58 (6)	59 (1)	58 (1)	58 (1)
Deer population size	139 (16)	78 (9)	68 (9)	79 (8)	78 (12)
Medium Deer Density					
Disease persistence (%)	53	32	18	30	32
Year 5 CWD prevalence (%)	2.9 (2.9)	2.2 (1.8)	1.1 (1.6)	2.4 (2.2)	2.0 (2.0)
Year 10 CWD prevalence (%)	9.5 (8.0)	7.6 (6.1)	4.0 (2.8)	6.3 (5.0)	7.1 (5.7)
Direct transmissions	50 (49)	18 (25)	6 (9)	16 (21)	19 (24)
Deer removed	0	86 (1)	85 (4)	86 (1)	86 (1)
Deer population size	219 (20)	127 (12)	118 (13)	121 (15)	122 (13)

Table 2.2 (cont'd)

Model Output	Baseline (No Culling)	High Quality Habitat Culling	1.6-km Ring Culling	2.4-km Ring Culling	Parcel-based Culling
High Deer Density					
Disease persistence (%)	50	38	12	39	40
Year 5 CWD prevalence (%)	2.4 (2.3)	1.9 (1.7)	0.8 (0.8)	1.9 (1.6)	2.2 (1.9)
Year 10 CWD prevalence (%)	11.5 (7.8)	6.4 (4.8)	4.4 (3.9)	6.5 (4.8)	7.9 (6.3)
Direct transmissions	76 (71)	23 (28)	6.7 (12)	21 (27)	28 (36)
Deer removed	0	113 (1)	113 (1)	113 (7)	112 (7)
Deer population size	299 (23)	184 (16)	150 (11)	178 (16)	182 (14)

Table 2.3. Percent of individual-based model repetitions (n=350) from a suburban landscape resulting in chronic wasting disease (CWD) being detected in at least one deer at the end of a 10-year model simulation. Each deer population was infected with CWD in year 1 of model simulations. Model scenarios for three deer density levels: Low (10 deer/km^2), Medium (14 deer/km^2), and High (19 deer/km^2). Mean (standard deviation) for CWD prevalence rate after model year 5 and 10, total numbers of direct transmission events, and deer culled for those model repetitions where CWD persisted for each management scenario treatments.

Model Output	Baseline (No Culling)	High Quality Habitat Culling	1.6-km Ring Culling	2.4-km Ring Culling	Parcel-based Culling
Low Deer Density					
Disease persistence (%)	33	24	6	14	21
Year 5 CWD prevalence (%)	1.8 (1.5)	3.5 (3.4)	2.3 (1.7)	2.4 (2.2)	3.7 (1.4)
Year 10 CWD prevalence (%)	5.1 (7.9)	5.9 (5.1)	5.7 (5.4)	5.8 (7.3)	6.1 (4.3)
Direct transmissions	16 (16)	11 (12)	5 (4)	77 (11)	13 (14)
Deer removed	0	78 (4)	58 (5)	59 (4)	58 (1)
Deer population size	136 (12)	78 (11)	65 (16)	67 (7)	79 (12)
Medium Deer Density					
Disease persistence (%)	34	30	2	14	30
Year 5 CWD prevalence (%)	1.9 (1.7)	2.3 (1.8)	0.4 (0.6)	1.8 (1.6)	2.2 (2.2)
Year 10 CWD prevalence (%)	4.4 (3.5)	4.6 (3.8)	1.6 (0.4)	3.4 (2.4)	4.6 (3.6)
Direct transmissions	20 (24)	14 (15)	3 (3)	6 (8)	13 (15)
Deer removed	0	85 (10)	83 (4)	84 (7)	85 (8)
Deer population size	210 (20)	129 (13)	100 (6)	113 (15)	128 (12)

Table 2.3 (cont'd).

Model Output	Baseline (No Culling)	High Quality Habitat Culling	1.6-km Ring Culling	2.4-km Ring Culling	Parcel-based Culling
High Deer Density					
Disease persistence (%)	31	33	2	20	29
Year 5 CWD prevalence (%)	1.4 (1.2)	2.4 (1.9)	0.9 (0.37)	1.2 (0.9)	2.1 (1.5)
Year 10 CWD prevalence (%)	4.4 (3.5)	5.1 (4.4)	5.6 (5.5)	3.1 (3.4)	4.3 (2.8)
Direct transmissions	20 (25)	18 (21)	5 (7)	9 (13)	14 (17)
Deer removed	0	113 (1)	111 (5)	113 (7)	112 (10)
Deer population size	280 (15)	147 (8)	132 (11)	156 (15)	175 (14)

Table 2.4. Mean (standard deviation) for total numbers of indirect transmission events of CWD, infected deer that emigrated, and percent of the study area affected by CWD for model repetitions where CWD persisted (detected in at least one deer at the end of a 10-year model simulation) for each management scenario in an exurban landscape. Model scenarios for three deer density levels: Low (10 deer/km^2), Medium (14 deer/km^2), and High (19 deer/km^2). Each deer population was infected with CWD in year 1 of model simulations.

Model Output	Baseline (No Culling)	High Quality Habitat Culling	1.6-km Ring Culling	2.4-km Ring Culling	Parcel-based Culling
Low Deer Density					
Indirect transmissions	7 (6)	3 (3)	2 (2)	3 (2)	9 (3)
Infected emigrants	2 (2)	1 (1)	1 (1)	1 (1)	1 (1)
Study area affected (%)	7.4 (3.1)	5.6 (2.2)	4.1 (1.1)	5.0 (1.6)	6.2 (2.2)
Medium Deer Density					
Indirect transmissions	9.6 (8)	5 (4)	2 (2)	5 (4)	4 (4)
Infected emigrants	2 (2)	1 (1)	1 (1)	1 (1)	2 (1)
Study area affected (%)	9.2 (3.9)	6.4 (2.8)	4.9 (2.7)	6.4 (2.2)	6.3 (2.3)
High Deer Density					
Indirect transmissions	12 (9)	5 (4)	2 (2)	5 (4)	14 (6)
Infected emigrants	3 (2)	2 (1)	1 (1)	1 (1)	2 (1)
Study area affected (%)	11.7 (5.1)	7.6 (2.9)	6.2 (2.0)	7.4 (2.7)	7.9 (2.4)

Table 2.5. Mean (standard deviation) for total numbers of indirect transmission events and infected deer that emigrated and percent of the study area affected by CWD for model repetitions where chronic wasting disease (CWD) persisted (detected in at least 1 deer at the end of a 10-year model simulation) for each management scenario in a suburban landscape. Model scenarios for 3 deer density levels: Low (10 deer/km^2), Medium (14 deer/km^2), and High (19 deer/km^2). Each deer population was infected with CWD in year 1 of model simulations.

Model Output	Baseline (No Culling)	High Quality Habitat Culling	1.6-km Ring Culling	2.4-km Ring Culling	Parcel-based Culling
Low Deer Density					
Indirect transmissions	4 (3)	4 (3)	2 (2)	3 (2)	4 (3)
Infected emigrants	1 (1)	1 (1)	1 (1)	1 (1)	1 (1)
Study area affected (%)	5.2 (3.5)	4.5 (1.6)	4.0 (0.6)	4.3 (1.4)	4.8 (1.4)
Medium Deer Density					
Indirect transmissions	5 (5)	4 (3)	1 (1)	3 (2)	4 (3)
Infected emigrants	2 (1)	2 (2)	1 (0)	1 (0)	1 (1)
Study area affected (%)	6.2 (2.8)	5.1 (1.6)	3.7 (0.6)	4.3 (1.1)	5.0 (1.5)
High Deer Density					
Indirect transmissions	5 (4)	4 (3)	2 (2)	3 (2)	4 (3)
Infected emigrants	1 (1)	2 (1)	1 (0)	1 (1)	1 (1)
Study area affected (%)	6.2 (2.9)	6.1 (2.0)	5.6 (0.9)	4.8 (1.8)	5.8 (2.1)

Table 2.6. Posterior distributions of model parameters and two-way interactions summarized with means, standard deviations (SD), medians, and upper (97.5%) and lower (2.5%) highest posterior density intervals for predicting chronic wasting disease (CWD) persistence (detected in at least one deer at the end of a 10-year model simulation) generated by the individual-based model.

Bayesian Negative Binomial-logit hurdle					
	CWD Persistence				
Model Parameters	Mean	SD	Median	2.5%	97.5%
Intercept	-0.19	0.11	-0.19	-0.41	0.02
Medium (14 deer/km^2) Deer Density	0.29	0.16	0.29	-0.01	0.60
High (19 deer/km^2) Deer Density	0.01	0.16	0.00	-0.28	0.33
Suburban Study Area	-0.53	0.16	-0.53	-0.83	-0.20
Parcel-based Culling	-0.85	0.16	-0.85	-1.16	-0.52
1.6-km Ring Culling	-1.93	0.21	-1.92	-2.34	-1.53
2.4-km Ring Culling	-0.77	0.16	-0.77	-1.10	-0.45
High-quality Habitat Culling	-0.81	0.17	-0.82	-1.13	-0.48
Medium Density*Suburban Study Area	-0.28	0.23	-0.28	-0.73	0.15
High Density*Suburban Study Area	-0.13	0.23	-0.12	-0.59	0.30
Medium Density*Parcel-based Culling	-0.03	0.23	-0.03	-0.48	0.42
High Density*Parcel-based Culling	0.62	0.22	0.63	0.17	1.04
Medium Density*1.6-km Ring Culling	0.24	0.29	0.24	-0.33	0.80
High Density*1.6-km Ring Culling	-0.01	0.29	-0.01	-0.58	0.56
Medium Density*2.4-km Ring Culling	-0.21	0.23	-0.21	-0.66	0.24
High Density*2.4-km Culling	0.48	0.23	0.48	0.04	0.92
Medium Density*High-quality Habitat Culling	-0.10	0.23	-0.10	-0.56	0.34
High Density*High-quality Habitat Culling	0.49	0.23	0.50	0.04	0.93
Suburban Study Area*Parcel-based Culling	0.21	0.21	0.24	-0.26	0.66
Suburban Study Area*1.6-km Ring Culling	-0.13	-0.13	0.33	-0.78	0.49
Suburban Study Area*2.4-km Culling	-0.41	-0.41	0.26	-0.93	0.08
Suburban Study Area*High-quality Habitat Culling	0.31	0.31	0.24	-0.18	0.75

Table 2.7. Posterior distributions of model parameters and two-way interactions summarized with means, standard deviations (SD), medians, and upper (97.5%) and lower (2.5%) posterior density intervals for predicting number of deer infected with chronic wasting disease (CWD) after model year 10 generated by the individual-based model.

Bayesian Negative Binomial-log link hurdle					
	Number of CWD-Infected Deer				
Model Parameters	Mean	SD	Median	2.5%	97.5%
Intercept	-2.60	0.02	-2.60	-2.64	-2.57
Medium (14 deer/km^2) Deer Density	0.04	0.02	0.04	0.00	0.08
High (19 deer/km^2) Deer Density	0.00	0.02	0.00	-0.05	0.05
Suburban Study Area	-0.68	0.03	-0.68	-0.74	-0.61
Parcel-based Culling	0.00	0.03	0.00	-0.07	0.07
1.6-km Ring Culling	-0.97	0.07	-0.97	-1.10	-0.84
2.4-km Ring Culling	-0.40	0.04	-0.40	-0.47	-0.33
High-quality Habitat Culling	-0.36	0.04	-0.36	-0.43	-0.29
Medium Density*Suburban Study Area	-0.26	0.04	-0.26	-0.35	-0.18
High Density*Suburban Study Area	-0.38	0.04	-0.38	-0.47	-0.30
Medium Density*Parcel-based Culling	-0.39	0.04	-0.39	-0.47	-0.30
High Density*Parcel-based Culling	-0.20	0.04	-0.20	-0.28	-0.12
Medium Density*1.6-km Ring Culling	-0.40	0.09	-0.40	-0.57	-0.22
High Density*1.6-km Ring Culling	-0.20	0.08	-0.20	-0.37	-0.04
Medium Density*2.4-km Ring Culling	-0.14	0.05	-0.14	-0.23	-0.05
High Density*2.4-km Culling	-0.04	0.05	-0.04	-0.13	0.05
Medium Density*High-quality Habitat Culling	0.04	0.05	0.04	-0.05	0.13
High Density*High-quality Habitat Culling	-0.12	0.04	-0.12	-0.21	-0.04
Suburban Study Area*Parcel-based Culling	0.24	0.24	0.06	0.13	0.35
Suburban Study Area*1.6-km Ring Culling	0.86	0.87	0.11	0.66	1.07
Suburban Study Area*2.4-km Culling	0.35	0.35	0.07	0.21	0.48
Suburban Study Area*High-quality Habitat Culling	0.49	0.49	0.06	0.37	0.60

Figure 2.1. National land cover database (NLCD) maps of the 23.3-km^2 suburban (A) and exurban (B) study areas in Ingham County, Michigan, USA. The black lines overlaying the NLCD layer indicate individual land parcels.

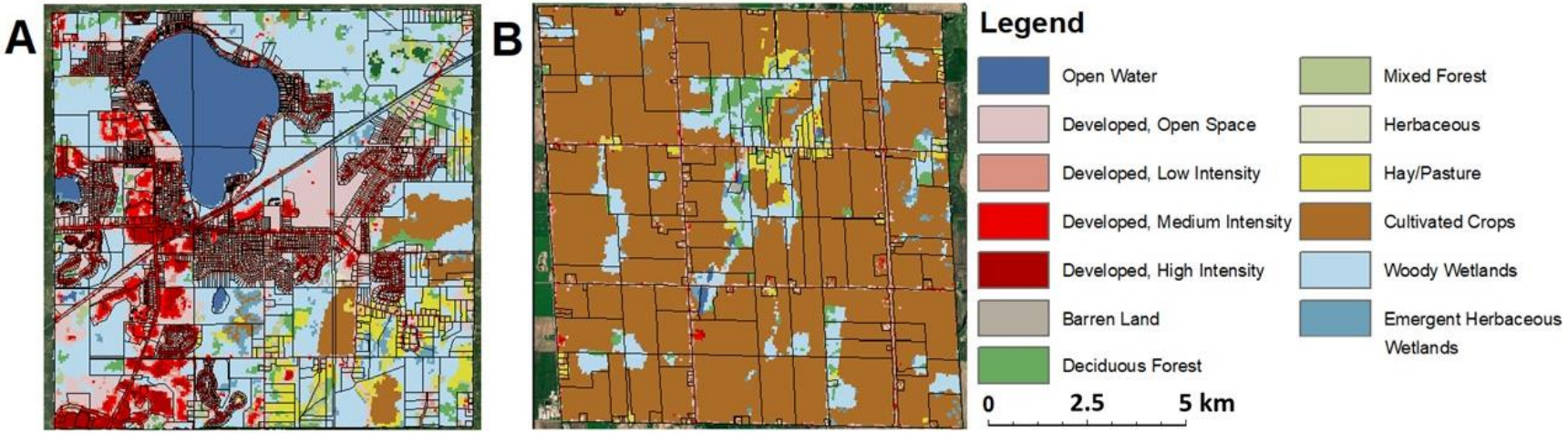


Figure 2.2. Maps indicating where deer were removed in the suburban (left) and exurban (right) study areas for the three deer culling scenarios: land parcel-based culling (A), high quality habitat culling (B), and 2.4-km radius ring culling (C).

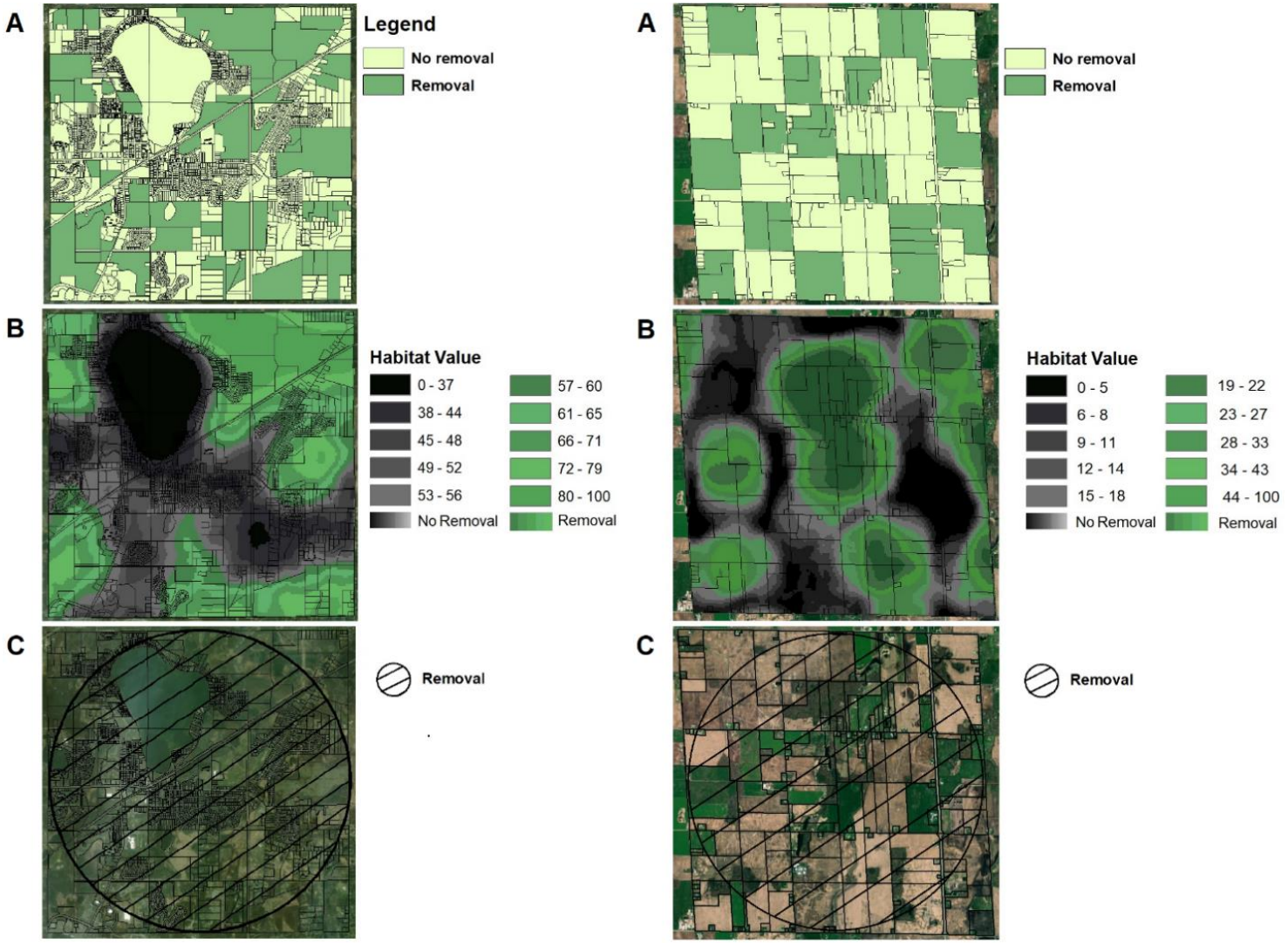


Figure 2.3. Estimated annual prevalence rate of chronic wasting disease (CWD) throughout a 10-year model simulation for each deer culling method that resulted in persistence of CWD (i.e., at least one infected deer after year 10). Landscape types in columns and deer density levels in rows.

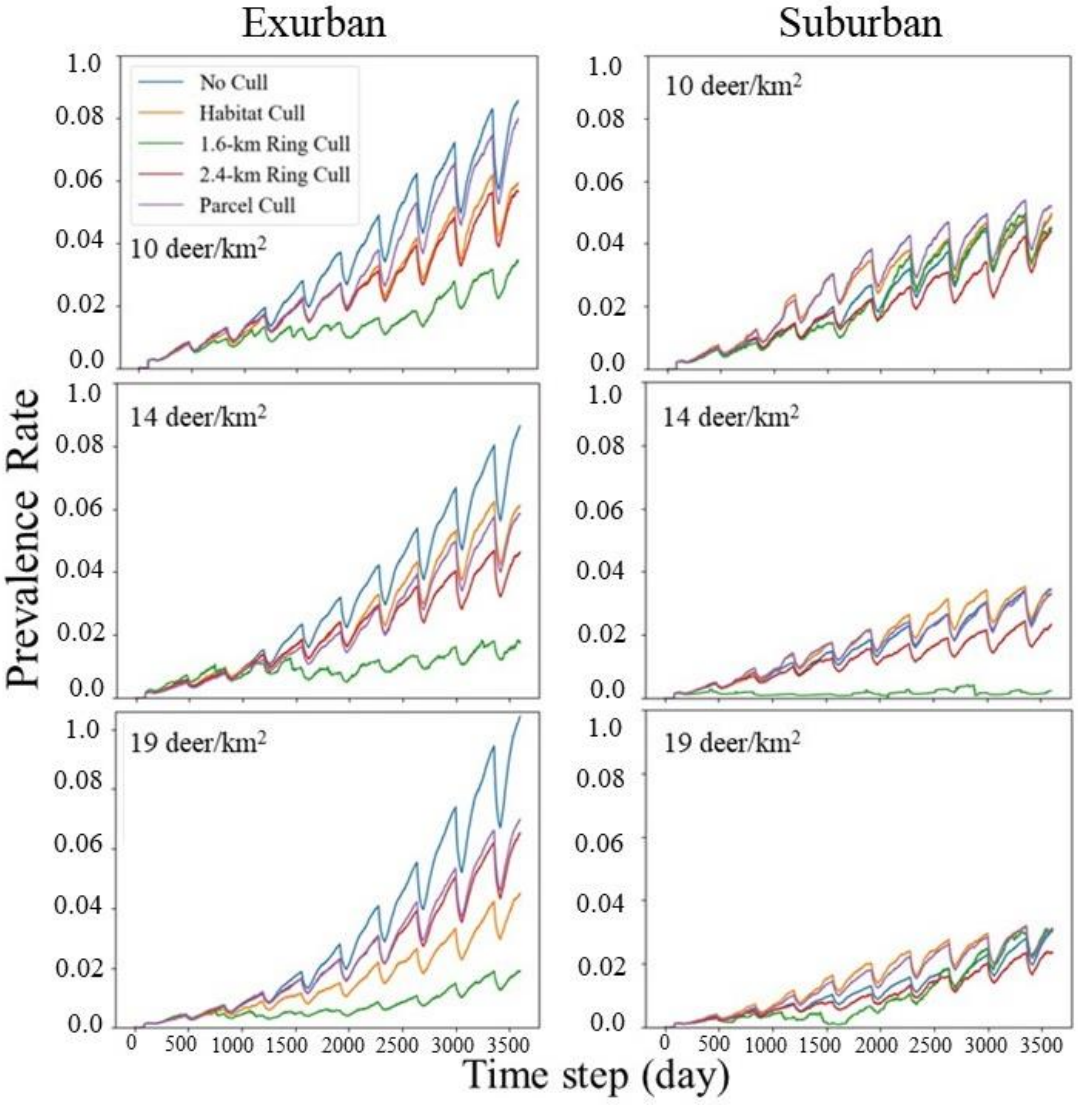


Figure 2.4. Estimated mean annual prevalence rate of chronic wasting disease (CWD) and 95% confidence interval throughout a 10-year model simulation for baseline deer management scenario without supplemental deer culling that resulted in persistence of CWD (i.e., at least one infected deer after year 10). Deer density levels are portrayed in rows. Percentages in parentheses beside each entry in the legend indicate percentage of model repetitions (n=350) where CWD persisted in the deer population after 10 years.

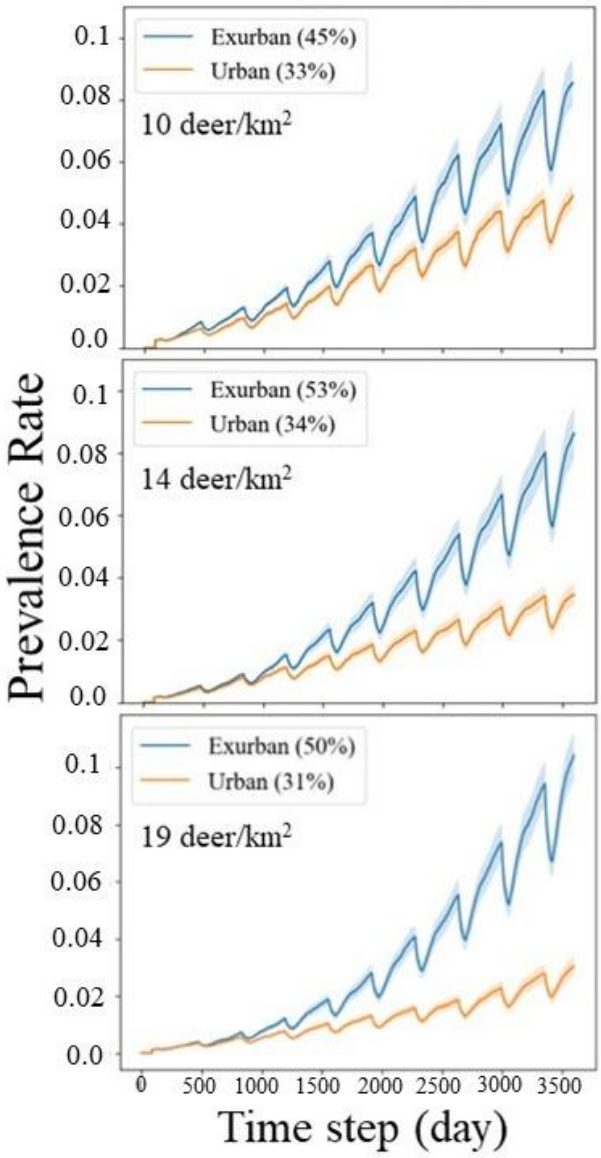


Figure 2.5. Proportion of model repetitions (out of 350) resulting in persistence of chronic wasting disease (i.e., at least one infected deer in the population by the end of the 10-year model simulation) in a simulated free-ranging deer population compared across four deer removal rates, two culling scenarios, two deer densities, and two landscape types.

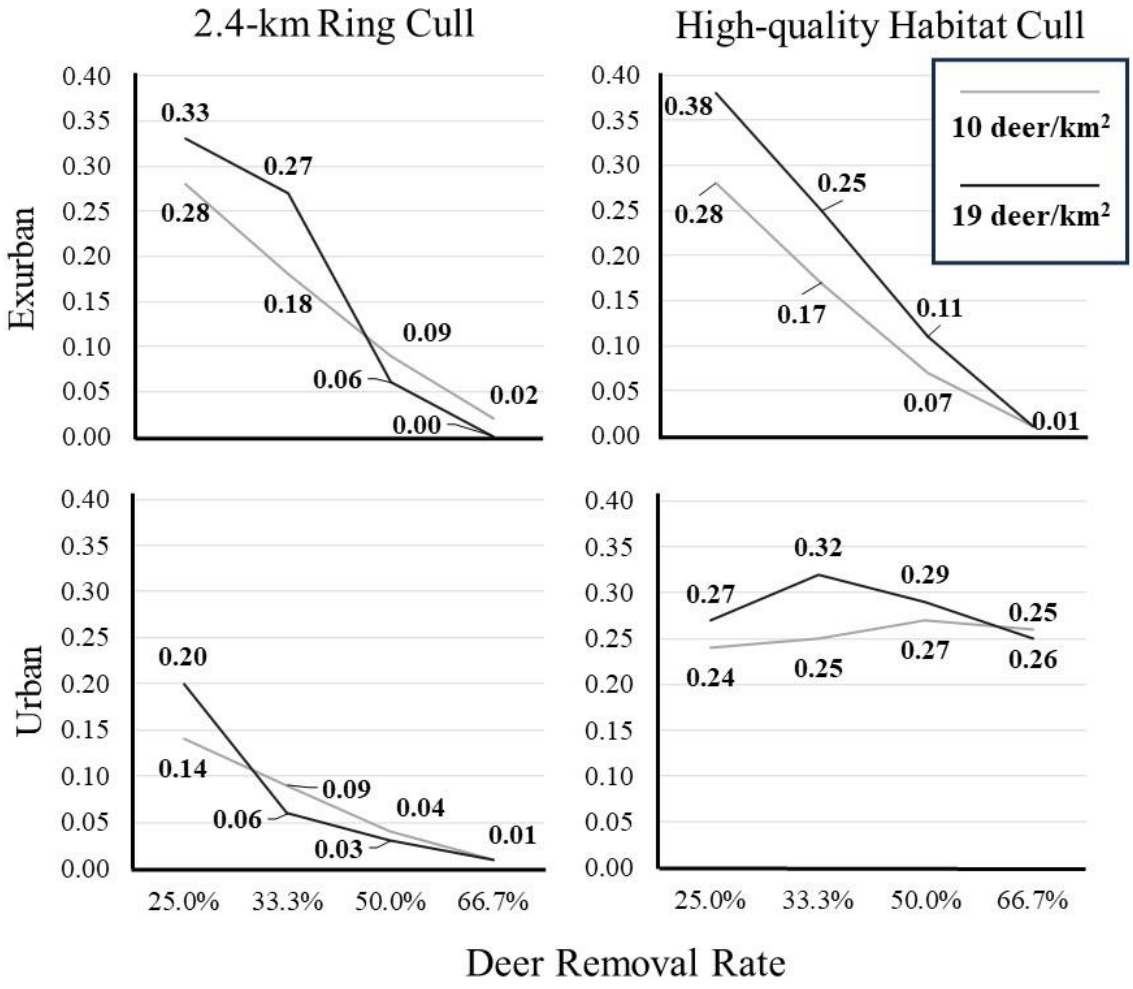


Figure 2.6. The percentage of model repetitions where deer removal was implemented but chronic wasting disease persisted in the population (i.e., at least one infected deer in the population by the end of the 10-year model simulation) in a simulated free-ranging deer population compared across four deer removal rates, two removal scenarios, two deer densities, and two landscape types.

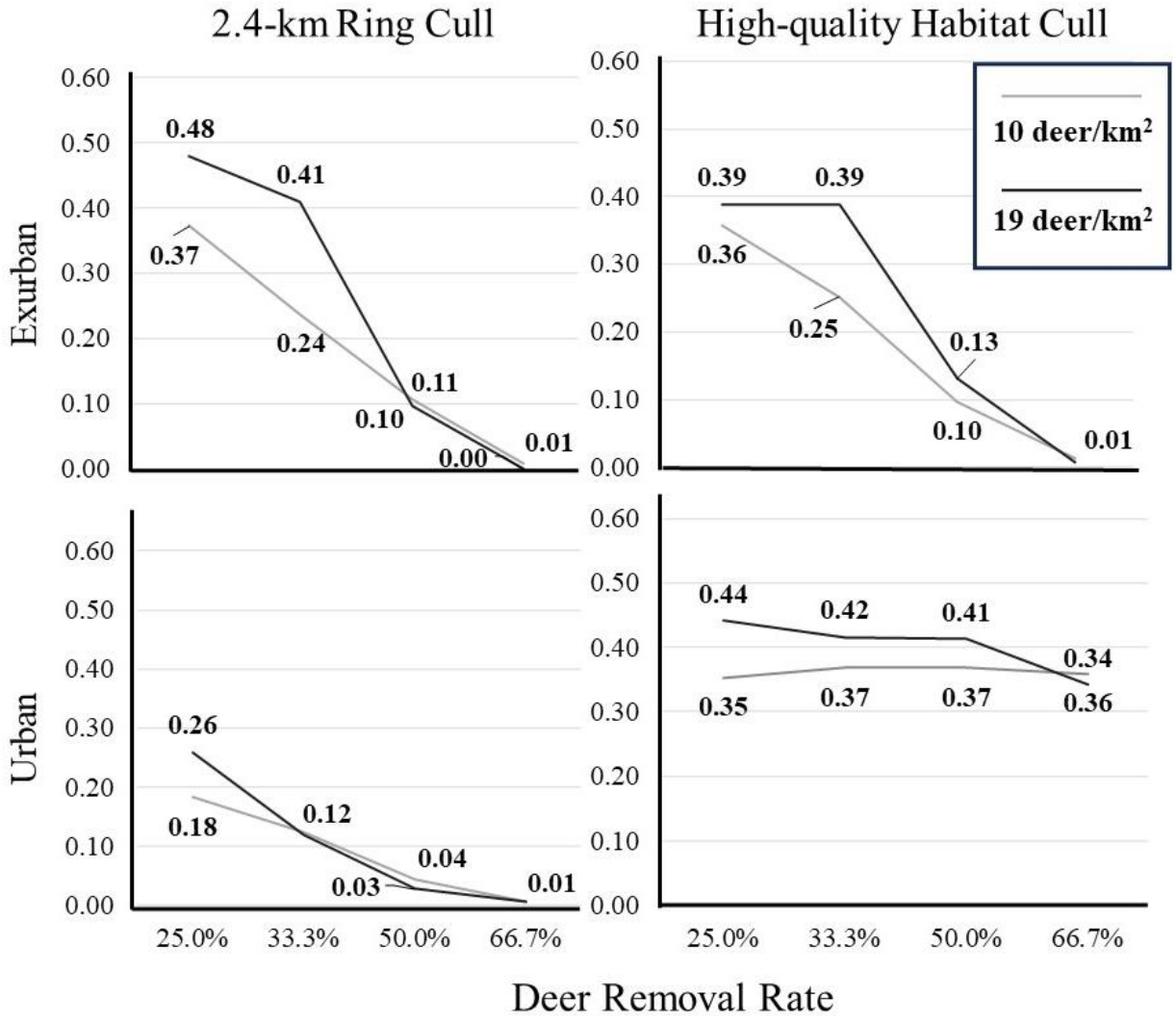
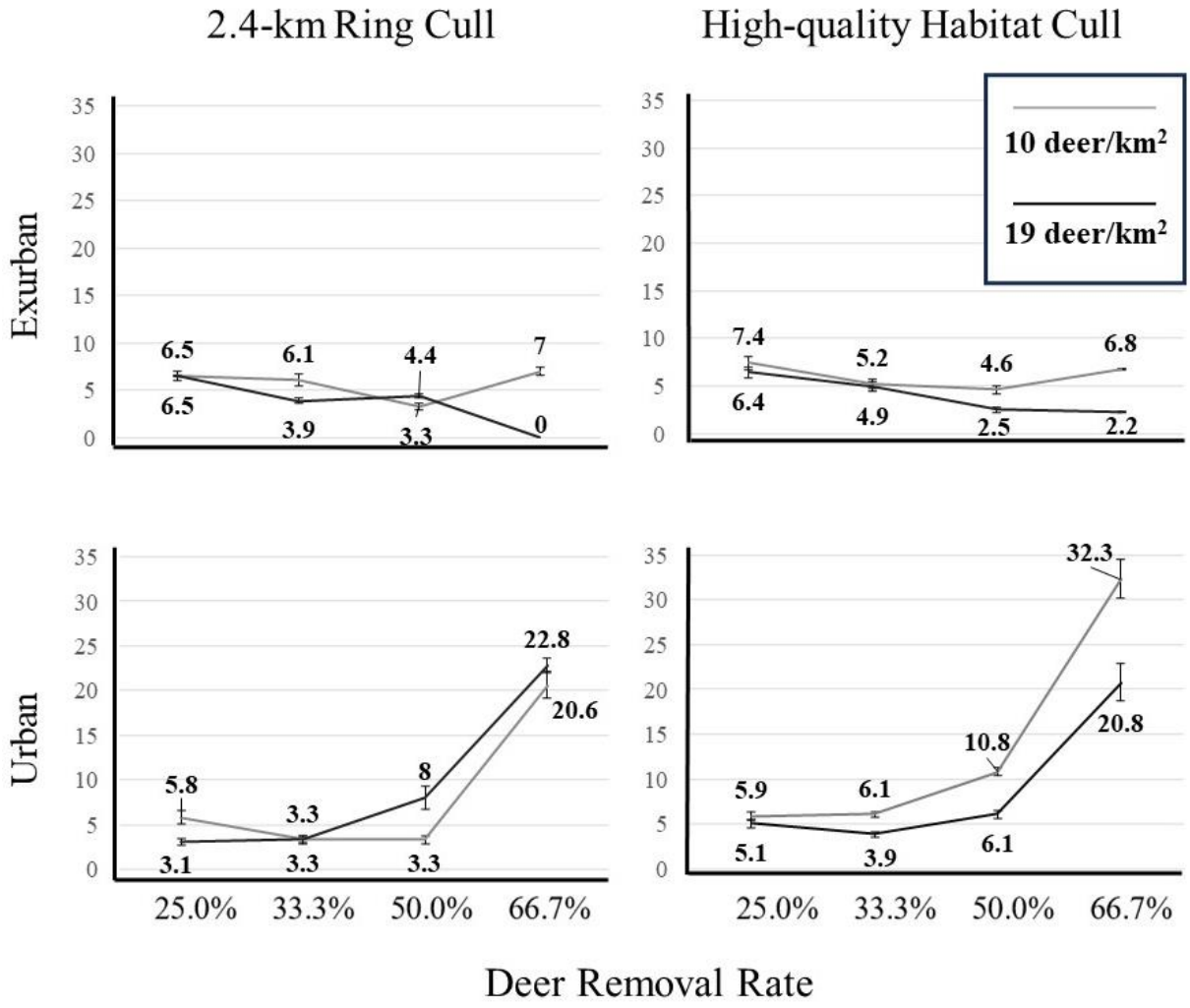


Figure 2.7. Final mean prevalence rate of chronic wasting disease after year 10 and 95% confidence interval for model repetitions where deer removal was implemented but chronic wasting disease persisted in the population (i.e., at least one infected deer in the population by the end of the 10-year model simulation) in a simulated free-ranging deer population compared across four deer removal rates, two removal scenarios, two deer densities, and two landscape types.



CHAPTER 3 INCORPORATING UNCERTAINTY INTO MODELING MANAGEMENT OF WHITE-TAILED DEER POPULATIONS FOR CHRONIC WASTING DISEASE

3.1 Introduction

Management decisions by North American fish and wildlife agencies are under continual scrutiny by scientists and lay publics. Soundness, defensibility, and transparency of management decisions are particularly important and necessary when the subject is highly polarized, complex, and incorporates multiple sources of uncertainty (Nichols et al. 1995). Fish and wildlife agencies that strive to identify, measure, and account for all sources of uncertainty affecting the system or population are more likely to arrive at sound and defensible decisions (Murphy and Noon 1991). Two categories of uncertainty afflicting complex systems, including ecological systems, are linguistic and epistemic uncertainty (Regan et al. 2002). Linguistic uncertainty is uncertainty that arises from natural language and communication failure, such as vagueness, ambiguity, and context-dependence in vocabulary terms or the changing of the meaning of words over time (Burgman 2005, Gregory et al. 2012). Linguistic uncertainty surfaces when scientists or other experts communicate with specialized terminology, even when discussing seemingly common terms such as wildlife health (Hanisch et al. 2014). The second form of uncertainty, epistemic uncertainty, occurs from incomplete knowledge of the system (Regan et al. 2002). There are numerous sources of epistemic uncertainty, but four commonly considered classes include environmental variation, partial observability, partial controllability, and structural uncertainty (Nichols et al. 1995, Williams 1997, Regan et al. 2002).

Chronic wasting disease (CWD) in free-ranging cervid (*Cervidae* family) populations is an example of a polarized complex system in which numerous uncertainties are prevalent (Haley and Hoover 2015, Gillin and Mawdsley 2018). Cervid species are some of the most widespread

and hunted wildlife species in North America of which white-tailed deer (*Odocoileus virginianus*) are the most popular big game (Hewitt 2015). Other economically and traditionally important species affected by CWD in North America include mule deer (*Odocoileus hemionus*) and elk (*Cervus canadensis nelsoni*; Hewitt 2015). Cervids are ecologically important as prominent species that alter structure and composition of ecological communities (Rooney and Waller 2003). Thus, cervids have diverse stakeholders and great economical and traditional importance (Hewitt 2015). Chronic wasting disease is always fatal once a cervid becomes infected (Williams and Young 1992). Field studies in the western United States revealed population declines caused by CWD for these species (Edmunds et al. 2016, DeVivo et al. 2017). Given the importance of cervids and potential detrimental effects of CWD on cervid populations, it is critical for wildlife agencies to execute CWD management actions as initially intended, such as to their full extent and duration. However, previous management attempts were often terminated prior to their intended duration with reasons including stakeholder disapproval and depleted resources (Miller and Fischer 2016). Creating a decision space that is rational and defensible by measuring and accounting for sources of uncertainty affecting CWD management allows wildlife agencies to circumvent many issues that cause premature termination of management plans. Terminology exists for reducing linguistic uncertainty when discussing CWD ecology and management (e.g., Thompson et al. 2023). Similarly, past modeling efforts focused on addressing and reducing epistemic uncertainty surrounding transmission of CWD typically by conducting sensitivity analyses to assess how uncertainty affects parameter values (i.e., structural uncertainty; Wasserberg et al. 2009, Al-Arydah et al. 2012, Kjaer and Schauber 2022). Less attention has been given to addressing epistemic uncertainty associated with management implementation for CWD control (Lischka et al. 2010). To support the claim that

understanding sources and effects of uncertainty on management of CWD is lacking, a recent review of CWD modeling efforts suggested that future research focus on assessing sources of uncertainty within the disease system, such as partial observability associated with CWD surveillance and detection bias (Winter and Escobar 2020).

In this chapter, I focus on assessing the effects of partial controllability as a source of uncertainty in management of CWD. Partial controllability, also referred to as implementation uncertainty and outcome uncertainty, arises when management results on the ground do not match the original management goals or predicted outcomes (Bischof et al. 2012, Link et al. 2012). A mismatch between objectives and outcomes is common when managers rely on recreational hunters to achieve wildlife population goals, such as assuming fulfillment of annual harvest quota to reduce population size. Management related to CWD has focused largely on removal or reduction of cervids in or surrounding CWD affected areas, which is attempted via hunter harvest during the deer hunting season and often supplemented by agency-led culling (Mateus-Pinilla et al. 2013, Uehlinger et al. 2016, Thompson et al. 2023).

When predicting management outcomes for CWD, particularly in a model setting, researchers often fail to incorporate partial controllability (Schauber and Woolf 2003, Winter and Escobar 2020). However, when speaking to wildlife professionals who perform on-the-ground management for CWD, partial controllability varies given the management context but is always present (E. Krom, personal communication; Fulton et al. 2011); i.e., the objectives seldom, if ever, can be flawlessly achieved. There are two major sources of partial controllability associated with lethal removal of deer as a management method: deer removal success and land access. I define deer removal success as the number or proportion of deer an agency or contractor can successfully remove from a targeted area relative to the intended amount defined by the

management objective. However, removal success may also be defined as the participation or willingness of hunters to harvest deer when hunter-harvest is used as a method of management for CWD (Rudolph and Riley 2017, Carstensen et al. 2011). I define land access as the amount or proportion of land on which the agency or contractor is allowed to remove deer.

I use the spatially explicit individual-based model developed in Chapter 1 to simulate direct and indirect transmission of CWD and its management in free-ranging white-tailed deer populations to assess the effects of partial controllability on management of CWD. I developed management scenarios focused on localized deer removal at various levels of deer removal success and land access rates to understand how sources of partial controllability may affect success of management objectives. I model the uncertainties and variability associated with deer removal for management of CWD and report minimum thresholds that allow for disruption of disease dynamics and reduction of prevalence over time.

3.2 Methods

I used an individual-based model (IBM) that projects daily indirect and direct transmission of CWD in free-ranging white-tailed deer populations. The IBM is described in the first chapter of this dissertation. My study area consisted of a 23.3-km^2 (9-mi^2) exurban area (81% agriculture, 10.5% wetlands, 5.3% developed, and 2.6% forested; NLCD 2020) in Ingham County, Michigan, USA (Fig. 3.1). The deer population was initially set at a density of 14 deer per square kilometer (35 deer per square mile). This study area is similar in size to those where U. S. Department of Agriculture's (USDA) Animal and Plant Health Inspection Service Wildlife Services typically perform localized removal of deer in response to detections of CWD in Michigan (E. Krom, personal communication).

At model initialization, the location of group centers of a user-defined number of deer

groups were placed based on habitat usability and habitat suitability. Usability of each 30m grid cell by deer was determined from a binary habitat map produced using NLCD 2020 urban imperviousness and open water data (Fig. 1.2). Habitat quality in each grid cell was estimated from a habitat suitability map developed from a white-tailed deer resource selection function, with each usable cell assigned a habitat suitability value ranging from 0 to 1 (Quinn 2010, Fig. 1.3). A user-defined number of deer were then assigned randomly to these groups based on average social group size reported from mid-Michigan (5; Courtney, unpublished data), and each group member was given a location in proximity to the center location specified for that group at random as defined by the deer movement model developed by Butts et al. (2022). Regular day-to-day home range movements by deer were not influenced by habitat quality but occasional shifting of deer group locations was.

For all model scenarios and repetitions, a single time step represented one day, and each month consisted of 30 days. I introduced CWD in the 4^{th} month of the first year (i.e., 120^{th} time step), which is the time of year when young males are most likely to disperse and spread CWD to new geographic areas (Trudeau, unpublished data, Samuel 2023). To simulate disease introduction, I randomly chose one deer group to occupy the center of the study area and one deer in that group to change disease status from susceptible to exposed during the 120^{th} time step. An exposed deer is a deer that was infected with CWD but not yet infectious. Exposed deer turned infectious 90-180 days post initial infection (Henderson et al. 2015).

Detection of CWD occurred when a deer infected with CWD was harvested by a hunter-harvest function in the simulation model, which was invoked daily between October and January each year and assumed all deer harvested were tested for CWD. I initiated culling of deer on January 1 each year immediately following the year CWD was detected. I simulated daily culling

of deer for up to 90 days from January 1st through March 30th. For each time step (i.e., one day) during the culling period, a random number between 0 and 7 was chosen to determine the number of deer culled that day. I chose this range of numbers based on average and range of numbers of deer Wildlife Services typically culls in a week during localized culling (E. Krom, personal communication). The model assumed all deer on accessible properties had an equal probability of being culled.

I adjusted the number of individual land parcels on which deer were removed across the study area as a technique to assess variability in land access rates for localized lethal culling of deer. I used land parcel data for Ingham County, Michigan, to identify and map each individual parcel (i.e., property) across the study area. I began by randomly selecting a sample of parcels that covered 10% of the study area that served as baseline access for culling. I then added to this baseline for different access scenarios. For example, for scenarios that applied a 20% land access rate I included the baseline 10% and 10% new properties chosen at random. For all time steps during the deer culling period, any deer located within those parcels could be chosen for culling. I adjusted percentage of land parcels on which deer were culled in intervals of 10% from 10% - 50%. I also ran simulations that removed deer on 70% and 100% of the parcels. This resulted in seven different land access scenarios. Deer were culled daily from the model population until the total number of deer needing to be culled for that scenario was reached or until March 30th.

To assess variability in deer removal success rates, I adjusted percentage of deer culled from parcels between 10–100% using the same rates as used for land access rates for a total of seven culling scenarios (i.e., 10–50%, 70%, and 100% of the deer population removed). With seven land access rates, seven deer culling success rates, and a baseline scenario where no deer culling was implemented, I had a total of 49 management scenarios to evaluate in this study. I

did not simulate 100% removal on 100% of the parcels because that would remove all deer from the model. For each scenario, I ran the model for 10 years and 350 repetitions.

I report prevalence of CWD, number of direct and indirect transmission events, and percentage of study area with shed prions after each time step (i.e., one day) in the model. For each management scenario, I calculated average values at the end of year 10 for each outcome using only the repetitions where disease persisted in the population. I defined disease persistence as at least one infectious deer in the population by the end of the model simulation. I also calculated the percentage of repetitions where detection of disease and, thus, deer culling occurred, the total number of deer culled, and percentage of repetitions where deer removal occurred and CWD was extirpated (i.e., "successful culling") by model year 10. I constructed 3-dimensional (3D) surface plots with 10% contour lines to visualize the change in prevalence of CWD and the number of repetitions that resulted in persistence of CWD across deer removal and land access rates.

I used Program R version 4.3.1 (R Core Development Team 2022) to develop a negative binomial hurdle model to estimate the influence of deer removal rate, land access rate, and their interaction on probability of CWD persistence and the number of infected deer given disease persistence. The model is fully described in Chapter 2. For this current chapter, the model covariates were land access rate and deer removal success rate and were treated as categorical. I excluded scenarios that included a deer removal or land access rate of 100% because I did not have a scenario with 100% deer removal and 100% land access rates, which was required for model fitting in order to be able to include this rate in the model assessment. I further assessed these covariates by applying a Bayesian regression approach as reported in Chapter 2 and used the non-Bayesian hurdle model to generate a design matrix. I looked for evidence of non-

convergence of each Markov chain by producing standard diagnostic plots and Brooks-Gelman-Rubin diagnostic statistics for each parameter.

3.3 Results

Of 350 repetitions, CWD was most frequently detected in model year 2, which was the year immediately following introduction into the deer population. Thus, deer culling was most often initiated in model year 3 (mean percentage of repetitions=27.5%, 95% CI=23.9–31.1%). Disease detection occurred throughout the first nine years of the model across the 49 scenario combinations, and this variability was caused by the random chance of a hunter harvesting a CWD positive deer. The proportion of detection events occurring in each year decreased with time (Table 3.1). Within the first two years of introduction, CWD was detected from hunter harvested deer (with 100% testing) in over 50% of model simulations. Detection of CWD was possible in the first year but never occurred.

Without localized deer culling simulated in the model, mean prevalence (95% confidence interval) of CWD increased to 9.5% (8.7–10.3%) by the end of the 10-year model simulation, and 47% of the scenarios resulted in persistence of CWD. For deer culling scenarios, mean prevalence rates ranged from 4.4% (100% land access, cull 40-50% of deer population) to 9.8% (10% land access, cull 30% of deer population; Table 3.1). Except for the scenario with low (10%) land access, increasing land access generally resulted in lower CWD prevalence for most levels of deer culling (Fig. 3.2A). Lowest CWD prevalence rates occurred for 70% and 100% land access where at least 30% of the deer population was culled (Fig. 3.2A). According to the simulation model, CWD prevalence rate was lowest for 40-50% deer population removal at 70-100% land access (Fig. 3.2B). Collectively, model simulations indicate that high land access and moderate deer population control most effectively reduced CWD prevalence (Fig. 3.2).

The mean number of direct CWD transmissions varied from 8 (70% land access, 70% deer cull rate) to 116 (20% land access, 10% deer cull rate), but the estimates were highly uncertain (Table 3.1). Mean number of indirect transmissions ranged from 3 (100% land access, 70% cull rate) to 22 (10% land access, 50% cull rate), and these estimates were also highly uncertain (Table 3.1). Mean percentage of the study area with shed prions was consistently <10%, ranging from 4% (100% land access, 70% deer cull rate) to 9.4% (10 and 20% land access, 10% deer cull rate; Table 3.1).

Probability of disease persistence ranged from 1.2% (70% land access, 100% deer population cull) to 51.7% (10% land access, 10, 70, and 100% deer population cull; Table 3.2). Chronic wasting disease was detected, and culling initiated, in >70% of model simulations for land access and deer cull combinations (Table 3.2). Culling resulted in disease extirpation a minimum of 35% of the simulations (10% land access, 40 and 70% deer cull) and up to 99% (70 and 80% land access, 70 and 100% deer cull; Table 3.2).

My 3D surface plots of CWD prevalence rate and proportion of model repetitions resulting in disease persistence (Fig. 3.3) for all land access and deer culling rates indicated that disease eradication is possible with high land access (>70% of the study area) and high deer cull rates (>70%; Fig. 3.3) Prevalence of CWD declines sharply at 50–70% for both deer cull and land access rates. Collectively, these simulation results indicate that access to at least 50% of a landscape and culling 50% of the deer population are needed to substantially reduce CWD prevalence.

Compared to a no culling scenario, the Bayesian hurdle model identified all levels of land access and deer removal rates as influential on the number of deer infected with CWD after model year 10 when compared to the baseline management scenario involving no culling (Table

3.3). However, the covariates, regardless of rate, were not influential on their own on persistence of CWD. The interaction between deer removal and land access rates was influential on CWD prevalence for 46 of 48 deer cull scenarios (Table 3.3). Probability of CWD persistence was different from the baseline scenario with no culling for 8 of 48 culling scenarios (Table 3.3). For 7 of these 8 scenarios, land access rate and/or deer removal rate was at least 70%, with the exception being a 30% deer removal rate and a 40% land access rate (Table 3.3). Diagnostic plots and statistics calculated using the Gelman and Rubin approach (GELMAN.DIAG; Gelman and Rubin 1992) to assess within- and among-chain variance for each parameter in the hurdle model are provided in Appendix D.

3.4 Discussion

My main objective for research reported in this chapter was to assess impacts of common sources of uncertainty (i.e., partial controllability) associated with management of CWD in free-ranging cervid populations using an IBM. Results indicate that the ability for wildlife managers to reduce probability that an outbreak of CWD persists in a localized area requires high rates of land access and deer culling success, whereas CWD prevalence rate can be reduced when low rates are applied. Deer culling and land access rates as low as 10% reduced CWD prevalence in a newly infected deer population after 10 years for some model scenarios. Two scenarios that did not result in a prevalence rate statistically lower than baseline no-culling scenarios without deer culling included 10% land access (and 10 and 40% deer cull rates). Conversely, reducing the probability that emergent CWD persisted required 40 and 70% land access, and cull rates ≥30%. The simulation model also suggested that increasing rates of deer culling and land access produces a cumulative negative effect on CWD persistence and prevalence that is not linear. Thus, when combined, efforts to increase land access and deer removal rates have a greater

negative effect on disease outcomes than changes in either rate individually.

My results suggest a threshold for deer culling rate to effectively disrupt CWD transmission and reduce prevalence over time. Moreover, there appears to be thresholds for deer culling and land access rates to reduce the CWD persistence in a localized area. If slowing prevalence of CWD is the objective, wildlife managers should strive for ≥20% for deer culling to negatively impact long-term prevalence rates. Wildlife agencies in the Midwest have reported deer culling goals of 25% of the targeted population in localized areas in response to CWD detection (E. Krom and C. Jacques, personal communication). Based on the results of my model, this culling rate will reduce CWD prevalence by up to 30% over 10 years compared to not culling (i.e., relying solely on hunter harvest of deer). However, even when conducted immediately after initial detection of CWD, a 25% deer culling rate is not enough to reduce the probability of the outbreak persisting in the population.

Findings from other studies that assessed management of wildlife diseases also support the principle that removal thresholds exist. A field study conducted in New Zealand by Tweddle and Livingstone (1994) measured a decline in tuberculosis in cattle when greater than 70% of the local brushtail possum (*Trichosurus vulpecula*) population was removed and maintained at a lower density. Roberts (1996) modeled bovine tuberculosis in possums in New Zealand and estimated that a 57% reduction in possum density would extirpate the disease from the local population. Ramsey (2000) suggested that tuberculosis could be eradicated from a population long-term if the population could be reduced and maintained at 30% of its original size. Although it is possible to identify removal thresholds required for effective disease control via field studies, this study illustrates the ease of doing so via simulation modeling.

The results of my simulation model would be more defensible if compared to field data

(e.g., apparent prevalence rates) reported by Midwestern wildlife agencies. However, I ran the simulation model and assessed management outcomes across a highly localized area (23.3 km^2). Although deer removal efforts in response to CWD detections are often constrained to limited geographic areas, it is difficult to estimate prevalence at this scale. The Illinois Department of Natural Resources collects localized deer removal and CWD data at the 1-mi^2 section level. The data, however, are shared only at broader scales (e.g., 93-km^2 township level). Thus, I cannot directly compare my model results to field studies or results obtained from other modeling efforts because of scale mismatch. I also expect my semi-closed study area affects estimates of prevalence rates and geographic spread of CWD. In the model, only dispersing fawn and yearling males were allowed to emigrate out of the study area in spring and fall with new males immigrating in at similar rates. The remainder of the population was forced to move within the study area boundaries. These movement restrictions may have inflated growth of the disease within the constrained area by preventing any geographic spread, thereby artificially increasing prevalence rate over time. However, small percentages of the study area affected by CWD (i.e., 4–10%) across management scenarios suggest that this was not an issue.

In the model, I assume that CWD is detected locally as soon as an infected deer is harvested during the hunting season and deer removal begins in the subsequent year. This assumption implies that every deer that is harvested by a hunter gets tested for CWD, which is improbable, even when the area is designated a CWD surveillance zone with mandatory check station requirements. Compliance rate to regulations is always less than 100%, creating partial controllability to the studied system (Nichols et al. 1995, Rudolph and Riley 2018). Similar to land access and deer removal success rates, the effects of variable public compliance rate to regulations on the disease system can be assessed with this IBM.

By assuming rapid disease detection and management implementation, I expect that my model provides a best-case scenario for management response, and the simulated management was more effective at impeding disease progression than would be expected in real deer populations. Delaying deer culling encourages greater transmission of CWD by providing more time for infected individuals to contact susceptible deer and shed prions into the environment before being exposed to lethal culling from the population. Additionally, delays in detection of CWD and management response by wildlife agencies can be visualized as a temporal lag in the model. True to all systems, temporal lags introduce system chaos and create additional instability by increasing the tendency of the system to oscillate with greater amplitude because of the lagged response (Sterman 2002). Accordingly, I expect that the model would predict higher mean prevalence rates and probabilities of persistence and more variation around those values in response to delayed management.

Wildlife managers base decisions on management objectives and these objectives can guide use of my model for specific situations. For example, if extirpation of a local outbreak is the management objective, then managers who focus on that outcome (i.e., probability of persistence) when using the model results for guidance can expect to improve their chances of achieving the objective. If slowing transmission of the disease is the objective, then managers may want to focus on prevalence rate and the total number of transmission events for each management scenario. If limiting geographic spread to surrounding areas is the goal, the percentage of study area affected by CWD (i.e., containing shed prions) should be the primary outcome of interest.

Although I report rates of deer culling and land access that are statistically different (i.e., probability of persistence and final 10-year prevalence rate) from the scenario without culling,

statistical significance has limited meaning to wildlife managers trying to control CWD in free-ranging deer populations. An increase in deer removal rate by 10% may produce a statistically significant reduction in prevalence rate when compared to the scenario without management, but if that 10% increase in deer removal only reduces annual prevalence of CWD by 2% after 10 years, managers must decide if the added effort and cost to remove more deer is worth their time.

To avoid using statistical significance as guidance for which rates agencies should target, response plans for CWD are often generated by wildlife state agencies to publicize how and why they are responding to CWD and can provide guidance (Thompson et al. 2023). If prevalence rate thresholds are listed in an agency CWD response plan as a management objective or metric for successful management, application of that objective to results of this model will facilitate decision-making. For example, Pennsylvania Game Commission (PGC; 2020) lists a measure of success as "a decline and maintenance of CWD sample prevalence to ≤ 5%" in their CWD response plan. After running the model using their study area, applying deer population parameters relevant to Pennsylvania, and assessing management scenarios with different rates of deer removal and land access, PGC wildlife managers can aim for deer removal and land access rates that are predicted by the model to maintain prevalence of CWD to ≤ 5% in the model.

Wildlife agency personnel have limited capacity to substantially reduce deer populations to control CWD, whether those limitations are cost (Thompson and Mason 2022), time, stakeholder acceptance (Holsman et al. 2010), or the general inability to control deer population size, particularly through the inability to affect harvest by hunters (Brown et al. 2000, Riley et al. 2003, Triezenberg et al. 2016). Previous CWD models indicated the efficacy of localized culling on reducing disease transmission but rates of deer removal and other management specifics were unspecified and remain unclear in the literature (Wasserberg et al. 2009, Potapov et al. 2012,

Oraby et al. 2014). By identifying minimum land access and deer removal rates needed to reduce disease transmission, I provide wildlife agencies with a better idea of expected outcomes and the ability to make more informed decisions about CWD management.

Table 3.1. Mean (standard deviation) prevalence rate of chronic wasting disease (CWD), total numbers of direct and indirect CWD transmission events, and percentage of the study area with shed prions after model year 10 by land access and deer removal rate. Means calculated only for model repetitions (n=350) resulting in persistence of CWD (i.e., number of infected individuals ≥ 1 after model year 10) for each land access and deer removal rate scenario. Values in bold indicate mean year-10 prevalence rate not statistically different from the baseline scenario with no deer culling. Scenarios that included deer culling or land access rate of 100% were not assessed.

	Deer cull rate						
10% Land access	**10%**	**20%**	**30%**	**40%**	**50%**	**70%**	**100%**
Year 10 prevalence (%)	9.3 (15.6)	9.3 (14.7)	9.8 (15.8)	9.3 (14.8)	9.7 (15.0)	9.1 (14.2)	9.6 (16.3)
Direct transmissions	52 (56)	49 (48)	51 (48)	50 (46)	54 (50)	49 (43)	26 (52)
Indirect transmissions	10 (8)	10 (7)	9 (7)	10 (7)	11 (7)	9 (6)	9 (8)
Affected area (%)	9.4 (8.3)	9.2 (7.3)	9.1 (7.5)	8.8 (7.2)	9.3 (7.5)	8.9 (7.1)	9.1 (7.6)
20% Land access							
Year 10 prevalence (%)	**9.5 (15.2)**	7.6 (12.8)	7.8 (13.3)	7.9 (12.4)	7.6 (11.6)	7.1 (12.8)	6.9 (12.7)
Direct transmissions	**56 (54)**	41 (39)	38 (40)	39 (38)	37 (36)	28 (33)	23 (29)
Indirect transmissions	**10 (9)**	8 (6)	7 (6)	8 (6)	7 (5)	6 (5)	5 (4)
Affected area (%)	**9.4 (8.6)**	8.4 (6.6)	8.2 (6.8)	8.3 (6.8)	7.8 (6.3)	7.4 (5.9)	7.0 (5.3)
30% Land access							
Year 10 prevalence (%)	8.2 (13.5)	8.0 (12.8)	8.0 (13.0)	6.4 (10.8)	6.3 (12.5)	5.6 (10.9)	5.1 (9.9)
Direct transmissions	46 (47)	41 (42)	38 (37)	39 (32)	24 (29)	18 (24)	11 (15)
Indirect transmissions	8 (7)	8 (6)	8 (6)	6 (5)	6 (5)	5 (4)	3 (2)
Affected area (%)	8.7 (7.5)	7.4 (5.9)	8.3 (6.8)	7.6 (5.6)	7.1 (5.8)	6.9 (5.9)	5.6 (2.5)

Table 3.1 (cont'd).

	Deer cull rate						
40% Land access	**10%**	**20%**	**30%**	**40%**	**50%**	**70%**	**100%**
Year 10 prevalence (%)	8.2 (13.7)	7.4 (11.8)	7.5 (12.0)	7.5 (13.4)	5.6 (11.3)	5.6 (11.4)	6.4 (15.0)
Direct transmissions	46 (45)	35 (35)	36 (36)	31 (36)	21 (27)	16 (24)	7 (11)
Indirect transmissions	8 (7)	7 (5)	7 (6)	6 (6)	5 (4)	4 (3)	4 (3)
Affected area (%)	8.9 (7.4)	8.0 (6.3)	8.0 (6.9)	7.7 (6.6)	6.7 (5.2)	6.0 (5.0)	5.0 (3.1)
50% Land access							
Year 10 prevalence (%)	**9.3 (14.7)**	8.4 (13.8)	8.2 (13.9)	7.5 (12.7)	7.4 (13.9)	7.1 (13.3)	7.4 (16.9)
Direct transmissions	**50 (48)**	40 (44)	34 (38)	26 (29)	25 (32)	15 (22)	9 (15)
Indirect transmissions	**9 (29)**	7 (6)	7 (5)	6 (5)	5 (5)	4 (5)	3(3)
Affected area (%)	**9.4 (7.2)**	8.4 (7.2)	7.8 (6.7)	7.0 (5.6)	6.9 (6.0)	6.4 (4.8)	5.6 (3.8)
70% Land access							
Year 10 prevalence (%)	7.5 (12.6)	6.8 (11.2)	7.0 (12.0)	4.5 (9.2)	5.4 (10.4)	5.4 (11.3)	7.8 (18.6)
Direct transmissions	36 (37)	29 (32)	23 (26)	12 (16)	12 (15)	6 (8)	2 (4)
Indirect transmissions	7 (5)	7 (5)	5 (4)	3 (3)	4 (3)	2 (2)	1 (1)
Affected area (%)	8.2 (6.4)	7.4 (5.5)	7.0 (5.1)	5.4 (4.0)	5.5 (3.1)	4.6 (2.4)	4.1 (0.0)
100% Land access							
Year 10 prevalence (%)	8.3 (15.0)	6.8 (13.3)	6.2 (11.5)	4.4 (8.6)	4.4 (9.4)	5.3 (6.8)	NA
Direct transmissions	40 (41)	27 (30)	21 (24)	11 (13)	8 (9)	4 (4)	NA
Indirect transmissions	8 (6)	5 (4)	5 (4)	3 (3)	2 (2)	1 (1)	NA
Affected area (%)	8.4 (7.5)	7.1 (5.9)	6.2 (4.7)	5.4 (3.7)	4.6 (2.1)	4.0 (0.7)	NA

Table 3.2. Percentage of model repetitions (n=350) that resulted in chronic wasting disease (CWD) persistence (i.e., number of infected individuals ≥ 1) by model year 10, percentage of repetitions resulting in CWD detection and, thus, deer culling. percentage of repetitions resulting in culling performed and extirpation of CWD (i.e., successful culling), and means (standard deviation) for total deer culled for repetitions where culling was performed according to land access and deer removal rate combinations from the individual-based model. Values in bold indicate that the probability of CWD persistence for that scenario was statistically different from the baseline no culling scenario. Scenarios that included deer culling or land access rate of 100% were not assessed.

	Deer cull rate						
10% Land access	**10%**	**20%**	**30%**	**40%**	**50%**	**70%**	**100%**
Disease persistence (%)	49.4	51.7	49.7	53.1	50.3	51.7	51.7
Culling performed (%)	76.6	77.4	77.7	78.0	76.9	76.6	75.1
Successful culling (%)	63.4	38.4	40.1	35.5	37.9	35.4	36.9
Total deer culled	2 (1)	7 (1)	10 (1)	13 (1)	17 (1)	24 (1)	35 (2)
20% Land access							
Disease persistence (%)	50.1	50.0	45.1	48.9	47.7	38.3	33.1
Culling performed (%)	75.9	77.7	80.0	78.9	79.1	76.6	78.9
Successful culling (%)	35.5	41.2	48.2	42.8	42.6	55.2	62.3
Total deer culled	7 (1)	14 (1)	21 (1)	14 (1)	17 (3)	24 (4)	35 (1)
30% Land access							
Disease persistence (%)	47.1	46.3	46.6	44.9	40.3	**31.2**	18.9
Culling performed (%)	79.1	75.7	77.7	78.0	79.7	**77.4**	75.4
Successful culling (%)	42.6	43.4	43.8	47.3	54.8	**64.8**	78.4
Total deer culled	11 (1)	21 (1)	31 (1)	41 (1)	51 (4)	**71 (4)**	101 (9)
40% Land access							
Disease persistence (%)	45.4	40.9	**45.7**	43.5	42.7	**33.7**	13.4
Culling performed (%)	74.9	74.3	**76.6**	80.1	81.3	**78.0**	80.0
Successful culling (%)	43.1	48.8	**43.7**	51.4	54.5	**63.0**	89.3
Total deer culled	14 (1)	28 (2)	**41 (3)**	55 (3)	68 (4)	**95 (6)**	154 (21)
50% Land access							
Disease persistence (%)	47.7	50.0	45.3	41.4	37.9	**26.4**	17.1
Culling performed (%)	72.3	81.1	77.9	76.6	80.9	**76.3**	77.7
Successful culling (%)	41.5	43.0	47.3	50.7	59.6	**71.2**	89.4
Total deer culled	17 (1)	35 (2)	51 (1)	67 (1)	85 (1)	**118 (10)**	168 (14)

Table 3.2 (cont'd).

	Deer cull rate						
70% Land access	**10%**	**20%**	**30%**	**40%**	**50%**	**70%**	**100%**
Disease persistence (%)	48.0	40.9	**36.0**	**29.1**	**28.3**	**10.3**	1.2
Culling performed (%)	78.3	80.3	**83.4**	**79.4**	**77.4**	**80.0**	78.3
Successful culling (%)	43.8	53.0	**59.6**	**69.1**	**69.4**	**92.9**	99.3
Total deer culled	24 (2)	48 (3)	**72 (1)**	**96 (1)**	**119 (7)**	**166 (1)**	229 (17)
100% Land access							
Disease persistence (%)	47.1	40.0	43.1	29.7	24.0	1.7	NA
Culling performed (%)	76.9	78.6	82.3	75.7	78.9	82.9	NA
Successful culling (%)	43.1	63.7	63.4	70.6	79.3	99.3	NA
Total deer culled	34 (1)	68 (4)	102 (1)	136 (1)	169 (1)	229 (10)	NA

Table 3.3. Posterior distributions of Bayesian model parameters summarized with means, standard deviations (SD), medians, and upper (97.5%) and lower (2.5%) highest posterior density intervals for the two disease outcomes of the individual-based model: probability of chronic wasting disease (CWD) persistence and CWD prevalence rate at model year 10 given CWD persistence.

Bayesian negative binomial hurdle										
	CWD Persistence (logit link)					**Number of CWD-Infected Deer (log link)**				
	Mean	SD	Median	2.5%	97.5%	Mean	SD	Median	2.5%	97.5%
Intercept	0.12	0.10	0.12	-0.08	0.32	-2.36	0.01	-2.36	-2.38	-2.33
Land Access Rate										
10%	0.07	0.15	0.07	-0.22	0.35	0.17	0.02	0.17	0.14	0.20
20%	-0.23	0.14	-0.23	-0.52	0.05	0.12	0.02	0.12	0.08	0.15
30%	-0.28	0.15	-0.28	-0.57	0.01	0.03	0.02	0.03	0.00	0.06
40%	0.06	0.15	0.06	-0.23	0.34	0.17	0.02	0.17	0.14	0.20
50%	0.00	0.15	0.00	-0.28	0.28	0.03	0.02	0.03	0.00	0.06
70%	-0.28	0.15	-0.28	-0.57	0.01	0.03	0.02	0.03	0.00	0.06
Deer Removal Rate										
10%	0.00	0.15	0.00	-0.29	0.29	0.03	0.02	0.03	0.00	0.06
20%	-0.24	0.15	-0.24	-0.52	0.05	0.12	0.02	0.12	0.08	0.15
30%	0.07	0.15	0.06	-0.22	0.35	0.17	0.02	0.17	0.14	0.20
40%	-0.23	0.14	-0.23	-0.51	0.05	0.12	0.02	0.12	0.08	0.15
50%	-0.28	0.14	-0.28	-0.56	0.00	0.03	0.02	0.03	0.00	0.06
70%	-0.01	0.15	-0.01	-0.29	0.28	0.03	0.02	0.03	0.00	0.06
Interactions										
10% Removal*10% Access	-0.21	0.21	-0.21	-0.62	0.21	-0.11	0.02	-0.11	-0.16	-0.07
10% Removal*20% Access	0.12	0.21	0.13	-0.29	0.53	-0.04	0.02	-0.04	-0.08	0.01
10% Removal*30% Access	0.05	0.21	0.05	-0.37	0.46	-0.09	0.02	-0.09	-0.14	-0.05
10% Removal*40% Access	-0.36	0.21	-0.36	-0.77	0.05	-0.22	0.02	-0.22	-0.27	-0.18
10% Removal*50% Access	-0.20	0.21	-0.20	-0.62	0.21	0.02	0.02	0.02	-0.02	0.07
10% Removal*70% Access	0.08	0.21	0.08	-0.33	0.49	-0.22	0.02	-0.22	-0.26	-0.17
20% Removal*10% Access	0.12	0.21	0.12	-0.29	0.54	-0.21	0.02	-0.21	-0.25	-0.17

Table 3.3 (cont'd).

Bayesian negative binomial hurdle										
	CWD Persistence (logit link)					**Number of CWD-Infected Deer (log link)**				
	Mean	SD	Median	2.5%	97.5%	Mean	SD	Median	2.5%	97.5%
20% Removal*20% Access	0.35	0.21	0.35	-0.06	0.77	-0.36	0.02	-0.36	-0.41	-0.32
20% Removal*30% Access	0.25	0.21	0.25	-0.17	0.66	-0.23	0.02	-0.23	-0.27	-0.18
20% Removal*40% Access	-0.32	0.21	-0.32	-0.73	0.09	-0.45	0.02	-0.45	-0.49	-0.40
20% Removal*50% Access	0.12	0.21	0.12	-0.29	0.54	-0.14	0.02	-0.14	-0.19	-0.09
20% Removal*70% Access	0.02	0.21	0.02	-0.39	0.44	-0.39	0.03	-0.39	-0.44	-0.34
30% Removal*10% Access	-0.26	0.21	-0.26	-0.67	0.15	-0.21	0.02	-0.21	-0.25	-0.16
30% Removal*20% Access	-0.15	0.21	-0.15	-0.56	0.26	-0.41	0.02	-0.41	-0.45	-0.36
30% Removal*30% Access	-0.04	0.21	-0.04	-0.45	0.37	-0.28	0.02	-0.28	-0.33	-0.23
30% Removal*40% Access	-0.42	0.21	-0.42	-0.83	-0.01	-0.49	0.02	-0.49	-0.54	-0.45
30% Removal*50% Access	-0.37	0.21	-0.37	-0.79	0.04	-0.27	0.02	-0.27	-0.32	-0.22
30% Removal*70% Access	-0.48	0.21	-0.48	-0.90	-0.07	-0.41	0.03	-0.41	-0.46	-0.36
40% Removal*10% Access	0.17	0.21	0.17	-0.24	0.59	-0.22	0.02	-0.22	-0.26	-0.17
40% Removal*20% Access	0.30	0.21	0.30	-0.10	0.71	-0.31	0.02	-0.31	-0.36	-0.26
40% Removal*30% Access	0.19	0.21	0.19	-0.22	0.60	-0.44	0.03	-0.44	-0.49	-0.39
40% Removal*40% Access	-0.21	0.21	-0.21	-0.62	0.20	-0.42	0.03	-0.42	-0.47	-0.37
40% Removal*50% Access	0.12	0.21	0.11	-0.29	0.52	-0.55	0.03	-0.55	-0.60	-0.50
40% Removal*70% Access	-0.50	0.22	-0.50	-0.92	-0.08	-0.86	0.03	-0.86	-0.92	-0.79
50% Removal*10% Access	0.13	0.21	0.13	-0.28	0.53	-0.07	0.02	-0.07	-0.12	-0.03
50% Removal*20% Access	0.30	0.21	0.30	-0.10	0.71	-0.27	0.02	-0.27	-0.31	-0.22
50% Removal*30% Access	0.04	0.21	0.05	-0.37	0.45	-0.39	0.03	-0.39	-0.44	-0.34
50% Removal*40% Access	-0.20	0.21	-0.20	-0.62	0.22	-0.69	0.03	-0.69	-0.74	-0.63
50% Removal*50% Access	-0.03	0.21	-0.03	-0.45	0.38	-0.44	0.03	-0.44	-0.49	-0.39
50% Removal*70% Access	-0.50	0.21	-0.50	-0.91	-0.08	-0.60	0.03	-0.60	-0.67	-0.53
70% Removal*10% Access	-0.09	0.21	-0.09	-0.50	0.33	-0.14	0.02	-0.14	-0.19	-0.10
70% Removal*20% Access	-0.36	0.21	-0.36	-0.77	0.05	-0.35	0.03	-0.35	-0.40	-0.30

Table 3.3 (cont'd).

Bayesian negative binomial hurdle										
	CWD Persistence (logit link)					**Number of CWD-Infected Deer (log link)**				
	Mean	SD	Median	2.5%	97.5%	Mean	SD	Median	2.5%	97.5%
70% Removal*30% Access	-0.63	0.22	-0.63	-1.05	-0.21	-0.53	0.03	-0.53	-0.59	-0.47
70% Removal*40% Access	-0.85	0.21	-0.85	-1.27	-0.43	-0.65	0.03	-0.65	-0.71	-0.59
70% Removal*50% Access	-1.14	0.22	-1.14	-1.57	-0.71	-0.28	0.03	-0.28	-0.34	-0.22
70% Removal*70% Access	-2.01	0.25	-2.01	-2.51	-1.52	-0.71	0.06	-0.71	-0.83	-0.59

Figure 3.1. National land cover database (NLCD) maps of the 23.3-km^2 study area in Ingham County, Michigan, USA, used to model chronic wasting disease dynamics in white-tailed deer. Black lines overlaying the NLCD layer indicate individual land parcels used to assess land access rates.

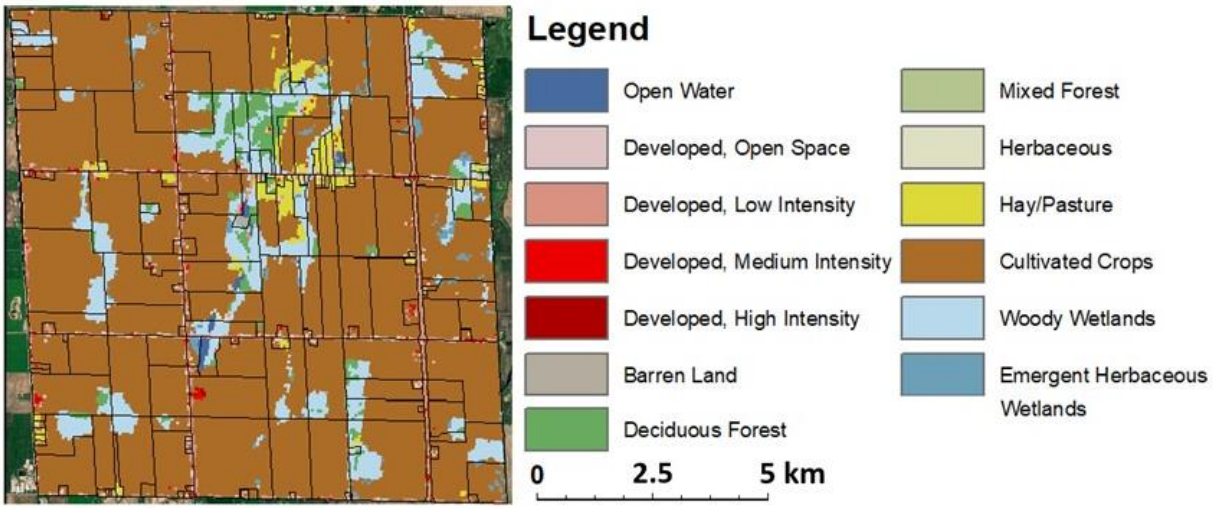


Figure 3.2. Mean prevalence rate of chronic wasting disease (CWD) after modeled year 10 (350 model simulations) for land access and deer removal combinations (n=48) clustered by A) land access rate and B) deer removal rate.

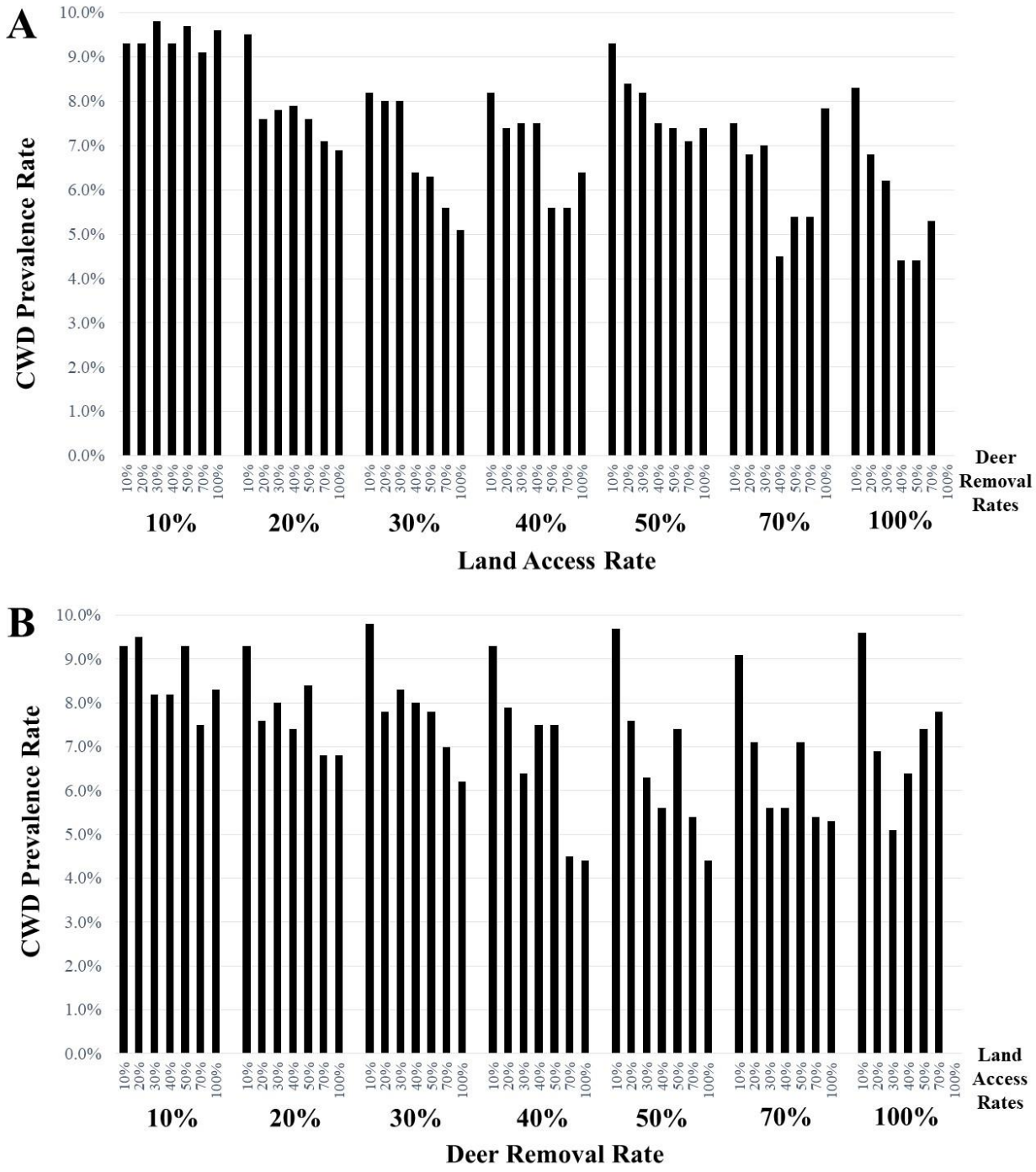
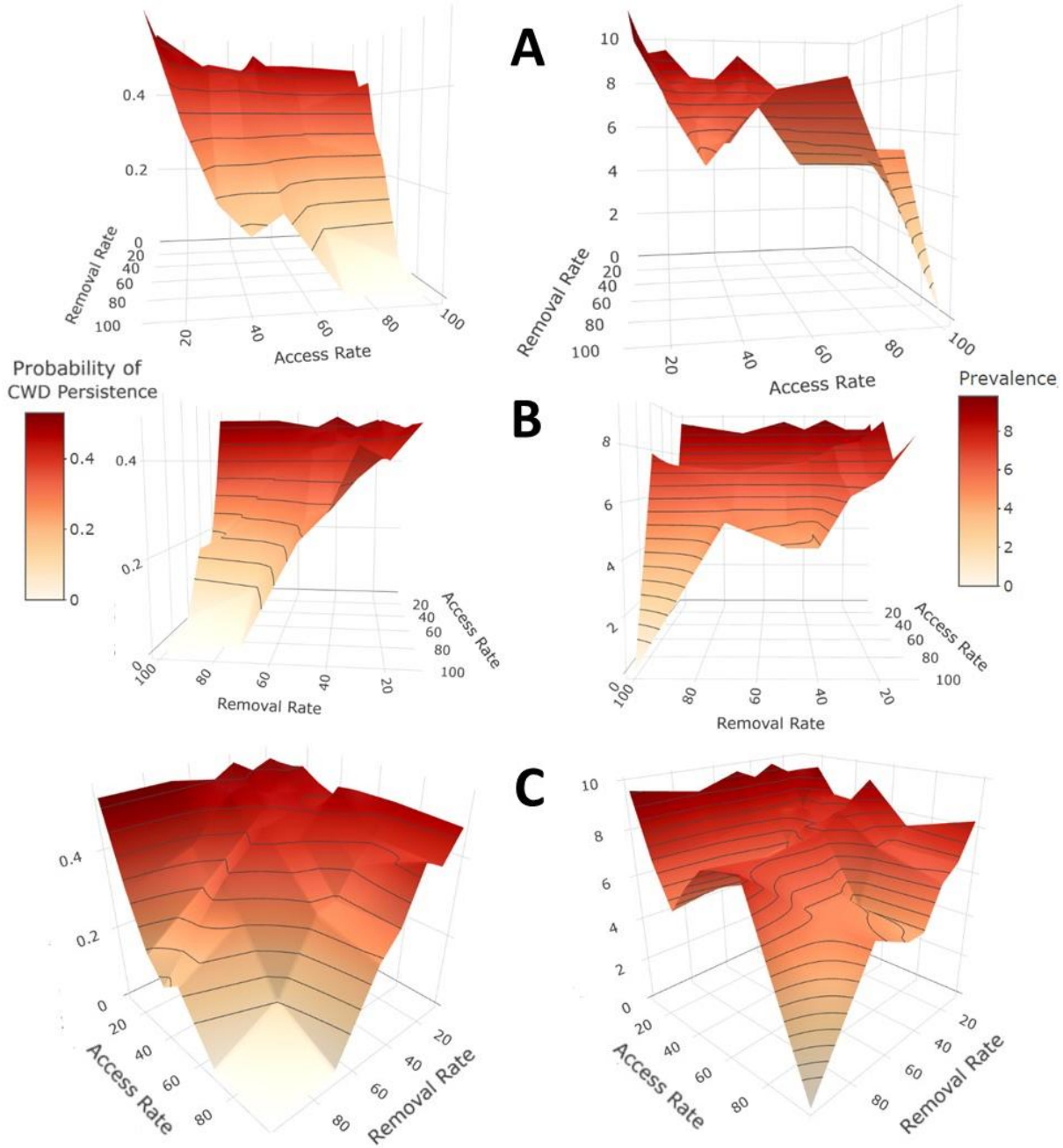


Figure 3.3. Three-dimensional surface plots representing mean probability of chronic wasting disease (CWD) persisting to model year 10 (left column) and CWD prevalence rate after model year 10 (right column) for different deer removal and land access rates estimated from an individual-based model for free-ranging deer in Michigan. Probability of CWD persistence is calculated by dividing the number of model repetitions where CWD persisted to model year 10 (potential range 0–350) by the total number of repetitions for each deer removal and land access scenario (n=350). The horizontal contour lines indicate 10% increments in probability of CWD persistence and CWD prevalence rate. A–C are the same figures from three different angles.



CONCLUSION

A purpose of my research was to better understand and elucidate dynamics of CWD transmission and management of the disease and to provide wildlife managers with a tool that allows them to make more informed decisions about managing local free-ranging deer populations for CWD. To achieve this purpose, I developed an individual-based model to simulate direct and indirect transmission of CWD across space and time in free-ranging white-tailed deer populations using our best knowledge of the disease system to date. Beyond developing a comprehensive model of the disease, I sought to create this model using an adaptable and user-friendly framework that provides wildlife agencies with the ability to input their own estimates of deer populations and landscape characteristics to assess local disease dynamics. White-tailed deer and other species of the *Cervidae* family are highly adaptable. Documentation of their ability to alter their behaviors in response to local environmental conditions is vast (Nixon et al. 1991, Massé and Côté 2013, Weiskopf et al. 2019). Although relatively less is known about CWD in comparison to deer ecology (Miller and Fischer 2016), it is understood that CWD dynamics must also be context dependent. Variability in CWD dynamics, such as population prevalence rates and rates of spread, have been documented across different regions of North America, supporting this association (Farnsworth et al. 2005, Ruiz et al. 2013, Evans et al. 2016).

In my first chapter, I present the individual-based model I developed in detail and assess the sensitivity of CWD dynamics to the model input factors. The ability of my model to reproduce short and long-term CWD dynamics observed in affected deer populations in Wisconsin implies that the model captures the necessary details associated with white-tailed deer populations and CWD processes. For this reason, I feel it was satisfactory to serve as the

foundation to conduct further analyses and address active research areas.

For Chapter 2, I sought to assess localized, fine-scale deer removal strategies for CWD by using an individual-based modelling approach. I also estimated the effects of local conditions, such as landscape type and deer density level, on management outcomes in this chapter. Results from model simulations demonstrated how landscape characteristics may affect the probability that an outbreak persists and the trajectory of its prevalence. The model also suggested that current deer removal rates of 20–30% of the local population often set by wildlife agencies is effective at reducing prevalence rate through time but is ineffective at reducing the likelihood of outbreak persistence. Deer density was not found by the model to be influential, but I suspect that we did not test a wide enough range of densities and further investigation is warranted. I found localized removal of deer using a ring cull technique at a small (1.6-km radius) spatial scale was the most effective management scenario under all landscape and deer density conditions.

In Chapter 3, my objective was to assess the effects of two sources of partial controllability, landowner access and deer removal success rates, on the ability of localized management strategies to affect disease dynamics. The results from this chapter suggest that there are thresholds for deer removal and land access rates, where any management below these thresholds are ineffective. Land access and deer removal success rates greater than 10% are necessary to affect long-term prevalence of CWD in a localized population. Conversely, one of the two rates had to be greater than 50% and the other greater than 20% to statistically reduce the probability that CWD persists in the deer population.

4.1 Management Implications

I provide a modeling framework that enables wildlife agencies to explore the effects of

management actions on localized outbreaks of CWD and assess conditions unique to individual deer herds. The method of deer removal and local conditions, such as landscape configuration, affect CWD dynamics and management success.

My model assessment of localized management strategies indicated that removing deer by: 1) applying a ring cull formation technique around the location of disease detection and 2) concentrating removal efforts closer to the site of disease detection (i.e., 1.6-km^2 radius instead of 2.4-km^2) was most effective at reducing the probability of disease persistence and long-term prevalence rates. The model identified strategies with greater land coverage as more effective than spatially-concentrated efforts. For example, culling restricted to property boundaries (i.e., less land coverage) is less effective than culling without property restrictions, even when the same number of deer are culled. Management was less effective when implementing a strategy that removed deer from areas considered high-quality deer habitat. If CWD is detected on private land and culling is restricted to within the boundaries of properties from which wildlife managers receive landowner access, the model identified scenarios that removed fewer deer on more properties more effective than scenarios that removed more deer on fewer properties.

The results produced in my third chapter corroborated conclusions made in Chapter 2. To assess the effects of partial controllability on CWD management success, I varied the rates of deer removal and land access in the model. When land access rate (i.e., the number of properties on which deer were removed) was increased, the disease was less likely to persist in the population and long-term prevalence rate was reduced. Moreover, my assessment of variability in management implementation indicated that deer removal was only effective at reducing the probability of CWD persistence and long-term prevalence rates when specific rates of land access and deer removal were achieved. Land access and deer removal rates of at least 20% were

effective at reducing prevalence rate in the population, whereas reductions in the probability of disease persistence required greater rates. To reduce the likelihood that the disease would persist in the population, either of the two rates had to be greater than 50% and the other had to be at least 30%.

If the goal is to slow the spread of CWD through time, current localized removal efforts with deer removal goals of ≥25% of the targeted local population may be seen as beneficial. However, this rate will not reduce the likelihood of disease persistence. Rates greater than 50% for deer removal or land access in localized areas are needed to functionally disrupt disease dynamics and reduce the probability of CWD persistence. If resources are limited, as is nearly always the case, it would be beneficial to determine whether obtaining more land access or increasing the total number of deer removed is more cost efficient to accomplish.

Lastly, managers that are prepared and enact deer removal soon after the disease is detected are most likely to control an outbreak of CWD. My model simulates deer removal in the January immediately following a hunting season (Oct–Jan) during which CWD was detected. Thus, management was always implemented no later than three months following disease detection. In the past, state wildlife agencies have waited until after a second hunting season to initiate agency-led deer removal in the detected area (Thompson et al. 2023). This may be because allowing hunters the first opportunity to control CWD through increased deer removal (e.g., increased bag limits) is more favorable to hunters and the general public than agency-led culling (Needham et al. 2004, Durocher et al. 2022). Postponing removal efforts, however, can be expected to have less of an effect on reducing the probability of disease persistence and prevalence rates. As such, greater rates of deer removal and land access may be needed to achieve the same success in reducing disease transmission when management is delayed.

4.2 Future Directions

The individual-based model I developed may serve as a foundation to conduct further analyses, address active research areas, and reveal knowledge gaps in the literature. For example, this model may serve as a tool for additional parameter calibration or assessing the sensitivity of the system to individual deer, population, or environmental parameters. Management strategies for CWD beyond localized deer removal may also be assessed, such as hunter-harvest regimes or regulations aimed at banning the baiting of cervids or antler-point restrictions (Wallingford et al. 2017). Lastly, by inputting genotypic variation for individual deer in the modeled population, the effects of genotypic variation on CWD processes at the population level can be assessed, which is just one of many research areas currently being investigated and of great interest.

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APPENDIX A
INDIVIDUAL-BASED MODEL SUBMODELS

1. Aging

This process increments age in months of individual deer after 30 daily time steps (i.e., one month). If a deer reaches 144 months (i.e., 12 years) of age, the vital status state variable is set to dead and the individual is removed from the simulation.

2. Births

The number of fawns born each year is probabilistic for each female deer, independent of the number or proximity to male deer in the model. Births occur between May 1 and June 30 each year. I chose a daily probability that summed to one by June 30 to ensure that each female deer gave birth every year while still allowing for variation in the day that parturition occurred. Adult female deer (deer greater than or equal to 22 months of age) can birth 0 to 3 fawns each year, with a population-level mean of 1.8 fawns per adult female (Table 1.2; Green et al. 2017). Yearling females (deer between the ages of 13 and 22 months old) average 1.25 fawns with a range of 0 to 3 fawns each year (Table 1.2). In the Midwestern United States, fawns (i.e., female deer less than 13 months of age) may also give birth (average=0.4; Green et al. 2017). Each birthed fawn has a 50/50 chance of being born a male or female (Verme 1983, Mori et al. 2022). Fawns are introduced into the simulation in the same cell that is occupied by their mother and join the deer group that their mother is a member of at the time of birth.

3. Birth interval

Female deer in the model are assigned a state variable that tracks the time since last birth to prevent a female deer from giving birth twice within the same birthing period (May–June) each year. The counter is reset after she gives birth. Female deer cannot give birth again if they already gave birth in the last ten months.

4. Mortality

4.1. Harvest mortality

In this model, deer face a daily probability of being harvested between October 1 and January 30 each year corresponding to most deer hunting seasons for Midwestern states. I applied sex- and age class-specific harvest rates obtained from the literature for Michigan white-tailed deer (Table 1.2; Van Deelen et al. 1997).

4.2. Baseline mortality

Deer experience constant low probability of mortality each day throughout the year. This category of mortality accounts for deer-vehicle collisions, predation, and other mortality events. The daily probability of a deer dying from these causes was determined based on values reported in the literature and identification of values that produced a stationary deer population over time within the model, a feature regularly observed in white-tailed deer populations unaffected by CWD (Table 1.2; Rosenberry et al. 2011).

4.3. Fawn mortality

Fawns are exposed to an additional fawn-specific mortality function each day during their first year of life (Table 1.2). Fawns ≤ 60 days of age have a higher daily probability of mortality than fawns aged 61–360 days (daily rates of 0.0055 and 0.000325, respectively; Rohm et al. 2007).

4.4. Disease mortality

Deer infected with CWD are subject to a daily probability of dying from the disease (Table 1.2; Samuel and Storm 2016). I assume this added mortality begins once the disease has advanced enough to begin impacting neurologic function. Therefore, I apply this daily probability of disease-associated mortality to simulated deer that have been infected with

CWD for more than 10 months, as there is no published evidence that disease-associated mortality occurs in deer that are newly infected with the disease. This submodel also sets infected deer to dead after they have been infected with CWD for five years, as five years appears to be the maximum amount of time reported in the literature that a cervid has survived following initial infection of CWD (Williams 2005, Argue et al. 2007).

5. Movement

I incorporated a data-driven deer movement model developed by Butts et al. (2022) using GPS location data from white-tailed deer in central New York (Quinn et al. 2013), which provides foundational rules for how deer move across the modeled landscape. Individual deer are randomly assigned a social group at the beginning of each simulation (see Section 1.2.5). Members of the same group move independently but their movements are biased towards the center of their group. I call this movement 'within-basin movement,' as each group center acts as a basin within which the movement of group members occurs. Landscape factors, such as habitat suitability of grid cells do not influence basin shape or size for deer in this model. I made this decision after exploring potential relationships among the parameters that define space use in the movement model derived by Butts et al. (2022) and common landscape configuration metrics including patch density, contagion, interspersion, juxtaposition index, and cohesion (Kindlmann and Burel 2008). I did not identify any significant correlations between landscape characteristics and inhabited areas by deer when compared to uninhabited areas in the model.

In addition to within-basin movement, the model produces basin hopping movements indicating the shift of entire social groups to a new area. I refer to this group movement as 'basin hops,' as the deer GPS data used to train the movement model revealed that groups can shift locations throughout the year (Butts et al. 2022; Quinn 2010). Thus, group location is dynamic

through time. Unlike within-basin movement, the habitat suitability value of cells influences where deer groups relocate within the study area when they execute a basin hop. Specifically, the model identifies three cells at random using a range of distances estimated from the GPS location data as potential new locations for the basin center. Out of those three cells, the model chooses the cell with the highest habitat suitability value to become the new center location for the group.

5.1 Seasonal dispersal

I added a component to the foundational movement model produced by Butts et al. (2022) that I refer to as 'basin switching,' which allows fawns and yearling males to disperse to new social groups during the spring and fall, as commonly reported in Midwestern regions (Table 1.2; Nixon et al. 1991). During the months of June and October of each year, each fawn and yearling male has a daily probability of changing social groups. The probability is user-defined. For my simulated population, I chose values that randomly select a percentage of deer of each age class to undergo dispersal that is similar to the percentage of Michigan deer reported to seasonally disperse an average distance that is less than the spatial extent of the modeled study area (e.g., < 15.5 km for this example) each season (Table 1.2). The remaining dispersing deer (i.e., deer that disperse beyond the length of the study area) will emigrate from the population (see Section 1.2.7.7). If a deer is chosen by the model to disperse within the study area, a new group will be randomly selected for it to join.

5.2 Emigration and Immigration

I mimic an open population by allowing male yearlings and young adults (age 2–2.5 years) to move in and out of the study landscape during the months of June and October, when young deer are most likely to leave their social groups and search for new ones

(Trudeau, unpublished data; Nixon et al. 1991). I select yearlings and adults to emigrate from or immigrate into the population that equals the percentage of male yearlings and adults reported to disperse an average distance greater than the length of the modeled study area annually in Michigan (Table 1.2; J. Trudeau, unpublished data). For yearlings immigrating into the study area, I did not restrict the area where those deer can be placed initially (i.e., within a certain distance from a border) given the current study area (15.5 km^2) and average dispersal distances of white-tailed deer in mid-Michigan (12.3 ± 2.35 km; J. Trudeau, unpublished data). However, if larger spatial extents are analyzed in the model, this function would have to be adapted to prevent unrealistic travel distances within a single time step (i.e., one day).

6. Direct disease transmission

If two deer are positioned within 25 meters of one another during a given time step (i.e., one day) and one is infectious while the other is neither infectious nor exposed, there is a probability that direct disease transmission occurs. My model does not incorporate the seasonal dissolution and reformation of social groups each year nor seasonally dependent contact rates between deer within and among social groups (Schauber et al. 2007, Silbernagel et al. 2011, Williams et al. 2014). To account for these seasonal differences in social behavior and contact rates among deer, I multiply the constant direct transmission rate by a monthly coefficient to account for varying probability of contact given proximity based on the time of year and sex and group membership of the two deer (Table 1.3). I derived these coefficients based on observational field studies conducted within the study area that reported contact rates and group size among free-ranging white-tailed deer (S. Courtney, unpublished; Table 1.3). The monthly contact coefficients did not replicate a pattern commonly observed by state wildlife agencies that

apparent prevalence rate of males is often 2-3x high than the female rate within a population, so I included a correction factor of six during the months of September through December (i.e., the time of year when male deer are more likely to interact with other males and females and have lower body condition; Hewitt 2011, Egan et al. 2023, Hearst et al. 2023) to force this trend in my modeled population. Direct transmission rate remains unknown for CWD, so I identified a range of possible values using model output verification (i.e., how well model output matches observations) with annual prevalence rates reported by a Midwestern state wildlife agency (Table 1.2; see Section 1.2.8; Augusiak et al. 2014).

7. Prion deposition

Deer infected with CWD for longer than 90 days become infectious during a randomly determined time step that falls between day 90 and 180 post-infection (Henderson et al. 2015). Once infectious, a deer will shed prions into grid cells it occupies during each time step. In addition, death of an infectious deer results in deposition of prions into the grid cell where it died. The number of prions deposited from a carcass or live deer at each time step is a function of amount of time the deer has been infected; infectious deer shed higher concentrations of prions over time (Henderson et al. 2015)**.** Deposited prions serve as an indirect source of disease transmission for susceptible deer residing in those cells during later time steps (Miller et al. 2004, Mathiason et al. 2009).

There are few estimates for the quantity of prions that deer shed during a given time and it is unknown how number of prions shed by live deer compares to prions bioavailable in a carcass after an infected deer has died (Tamgüney et al. 2009; Davenport et al. 2015; Denkers et al. 2020). For each deer *x* in a given grid cell during a given time step *t*, I estimate the number of prions deposited into that cell as:

$$p_L(x,t) = p_L(x,t) + \left[p_L(x,t) \times \left(\frac{t_{DPI}}{30}\right)\right], \text{ and}$$

$$p_D(x) = 50 * \left(p_L(x,t) + \left[\, p_L(x,t) \times \left(\frac{t_{DPI}}{30}\right)\right]\right),$$

where $p_L(x)$ is the daily number of prions shed by a live deer, $p_D(x)$ is the number of prions shed by a dead deer during the time step its death occurred, and t_{DPI} is the total number of days that the deer has been infected with CWD (i.e., days post initial infection). I divide t_{DPI} by 30 to allow the number of prions shed by deer to increase monthly instead of daily. I assume that deer shed 50 times the number of prions into the environment during one time step at death than when alive.

8. Indirect disease transmission

If a susceptible deer moves into a contaminated grid cell where infectious deer deposited prions during a previous time step, there is a probability that the susceptible deer becomes infected with CWD. I apply a negative exponential relationship to represent the probability of indirect transmission during a given time step given the density of prions in each cell:

$$p_{IT}(\propto, n, dt, v) = 1 - e^{-\alpha N/V},$$

where α represents the user-defined rate at which prions affect deer, N represents the number of prions in the cell, and V represents the area of the cell. Rates of indirect disease transmission for CWD remain unknown so I ran the model using a wide range of parameter values and applied model output verification with reported annual prevalence rates to determine a range of suitable values (Table 1.2; see Section 1.2.8).

9. Prion decay rate

The literature suggests prions can remain infectious in the environment for multiple decades (Georgsson et al. 2006, Seidel et al. 2006, Smith et al. 2011); however, freeze-thaw cycles and clay content in soil can affect prion infectivity and bioavailability (Wyckoff et al.

2016, Tennant et al. 2020). Thus, I allow prions that have been deposited into the environment to decay over time with a half-life of four years (i.e., 48 months), which produces a monthly decay rate (r) of 0.0144 using the following equation:

$$r = -\frac{\ln\left(\frac{1}{2}\right)}{t},$$

where t represents prion half-life in months. To implement this decay function in the model, I multiply the prion concentration of each cell by $(1–r)$ after each month.

APPENDIX B
PYTHON CODE FOR INDIVIDUAL-BASED MODEL

```
#################################
## Importing necessary packages ##
#################################

import numpy as np
import matplotlib.pyplot as plt
import scipy as sp
from scipy import stats
from scipy.stats import multivariate_normal
import pandas as pd
from itertools import combinations
from sklearn.neighbors import KernelDensity
import random
import copy
import rasterio
import itertools
import time
import mpi4py
from mpi4py import MPI
import os
import math

#################################
## Define output folder pathway ##
#################################

PATH = './PATH_FILE'

#####################################################################
## Designate each model repetition with a separate 'rank' value ##
#####################################################################

comm = MPI.COMM_WORLD
rank = comm.Get_rank()

#######################################################
## Create output subfolders for each output metric ##
#######################################################

if rank == 0:
    os.mkdir(PATH)
    os.mkdir(PATH+"/Total_deer")
    os.mkdir(PATH+"/Total_exp_deer")
    os.mkdir(PATH+"/Total_infect_deer")
    os.mkdir(PATH+"/Prevalence")
    os.mkdir(PATH+"/Direct_trans")
    os.mkdir(PATH+"/Indirect_trans")
    os.mkdir(PATH+"/Max_life")
    os.mkdir(PATH+"/Max_disease")
    os.mkdir(PATH+"/Disease_mort")
    os.mkdir(PATH+"/Emigrants")
    os.mkdir(PATH+"/Deer_culled")
    os.mkdir(PATH+"/Prion_cells")
```

```
#####################################################################
## Specify basemaps by directing to a map configuration folder ##
#####################################################################

maps = np.loadtxt('./config_twp',dtype=str)

###################################
## Specify variable values (daily) ##
###################################

DIR_TRANS = 0.017
MALE_ADDED_DT = 6
DIS_MORT = 0.00015
GROUPS = 251
IND_TRANS = 0.0003
HALF_LIFE = 48

decay_rate = (-np.log(0.5)/HALF_LIFE)

BIRTH_RATE = 0.0167

ADULT_MALE_HARVEST_RATE = 0.0025
ADULT_FEMALE_HARVEST_RATE = 0.001
YEARLING_MALE_HARVEST_RATE = 0.003
YEARLING_FEMALE_HARVEST_RATE = 0.00144
ADULT_MALE_BASE_MORT_RATE = 0.001
ADULT_FEMALE_BASE_MORT_RATE = 0.00033
YEARLING_MALE_BASE_MORT_RATE = 0.0023
YEARLING_FEMALE_BASE_MORT_RATE = 0.0025
FAWN_TWO_MONTH_MORT_RATE = 0.0055
FAWN_TEN_MONTH_MORT_RATE = 0.00087

#Number of young males leaving and entering the study area
YEARLING_FALL_EM_RATE = 0.00267
YEARLING_SPRING_EM_RATE = 0.00267
ADULT_FALL_EM_RATE = 0
ADULT_SPRING_EM_RATE = 0

#Number of young males changing groups within study area
YEARLING_FALL_DISP_RATE = 0.00433
YEARLING_SPRING_DISP_RATE = 0.01267
ADULT_FALL_DISP_RATE = 0
ADULT_SPRING_DISP_RATE = 0

#############################################################
## Specify map details for underlying layers (e.g., RSF) ##
#############################################################

bm = rasterio.open(maps[0])

bm_rows = bm.height #rows
bm_cols = bm.width #cols
bm_cs = bm.res[0] #cell size
bm_xllc = bm.bounds[0] #xllcorner
bm_yllc = bm.bounds[1] #yllcorner
```

```
BINARY_MAP = bm.read()[0,:,:]
BINARY_MAP[BINARY_MAP == -9999] = 0

rm = rasterio.open(maps[1])

rm_rows = rm.height #rows
rm_cols = rm.width #cols
rm_cs = rm.res[0] #cell size
rm_xllc = rm.bounds[0] #xllcorner
rm_yllc = rm.bounds[1] #yllcorner

RSF_MAP = rm.read()[0,:,:]
RSF_MAP[RSF_MAP == -9999] = 0

rem = rasterio.open(maps[2])

REMOVE_MAP=rem.read()[0,:,:]
REMOVE_MAP[REMOVE_MAP == -9999] = 0

rem_rows = rem.height
rem_cols = rem.width
rem_cs = rem.res[0]
rem_xllc = rem.bounds[0]
rem_yllc = rem.bounds[1]

################################################
## Defining a distance function between 2 deer ##
################################################

def dist(p1, p2):
    (x1, y1), (x2, y2) = p1, p2
    return np.sqrt((x2 - x1)**2 + (y2 - y1)**2)

################################################
## Initializes a world for the deer to exist on ##
################################################

class World():
    def
__init__(self,total_timesteps,suscept_yrling_males=0,infected_yrling_m
ales=0,suscept_yrling_females=0,infected_yrling_females=0,suscept_adul
t_males=0,infected_adult_males=0,suscept_adult_females=0,infected_adul
t_females=0,suscept_fawn_males=0,infected_fawn_males=0,suscept_fawn_fe
males=0,infected_fawn_females=0,groups=0,params='',xtrds='',ytrds='',c
ov_mats=''):

        self.y_size = bm_rows
        self.x_size = bm_cols

        self.days = 0

        self.dir_transmissions = 0
        self.ind_transmissions = 0
        self.max_life = 0
        self.max_disease = 0
```

```
        self.dz_mort = 0
        self.emigrants = 0
        self.groups = groups

        self.culled_deer = 0
        self.infectious_culled = False
        self.infectious_harvested = False

        self.prion_deposited_cells=0
        self.prion_map = np.zeros((self.y_size,self.x_size))
        self.deer_list = []
        self.male_list = []
        self.female_list = []
        self.herd_list = []
        self.cov_mats = cov_mats
        self.params = params

        self.group_pos = [(681636,4742528)]
        self.dz_detected= False

        while len(self.group_pos) < groups:
            possible_locs = []
            possible_loc_vals = []
            for n in range(3):
                rand_loc =
np.random.randint(len(np.where(BINARY_MAP==1)[0]))

                pos_x,pos_y = np.where(BINARY_MAP==1)[1][rand_loc],
np.where(BINARY_MAP==1)[0][rand_loc]

                possible_locs.append((pos_x,pos_y))

                possible_loc_vals.append(RSF_MAP[pos_y,pos_x])

            x_loc = rm_xllc + possible_locs[np.argmax
(possible_loc_vals)][0]*rm_cs
            y_loc = rm_yllc + possible_locs[np.argmax
(possible_loc_vals)][1]*rm_cs

            distances_from_groups = [np.inf]
            for other in self.group_pos:

distances_from_groups.append(dist((x_loc,y_loc),other))

            if np.min(distances_from_groups) > 150:
                self.group_pos.append((x_loc,y_loc))


        for h in range(groups):
            loc = np.random.randint(params.shape[0])

self.herd_list.append(Herd(self.group_pos[h],params[loc,:],xtrds[loc],
ytrds[loc],total_timesteps))
```

```
        for m in range(suscept_yrling_males):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Male',inf=Fal
se,exp=False,age=np.random.randint(17,21),params=params[loc,:],cov_mat
=cov_mats[loc,:,:],herd=g))
        for f in range(suscept_yrling_females):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Female',inf=F
alse,exp=False,age=np.random.randint(17,21),params=params[loc,:],cov_m
at=cov_mats[loc,:,:],herd=g))
        for m in range(infected_yrling_males):
            loc = np.random.randint(params.shape[0])
            g =0

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Male',inf=Fal
se,exp=True,age=np.random.randint(17,21),params=params[loc,:],cov_mat=
cov_mats[loc,:,:],herd=g))
        for f in range(infected_yrling_females):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Female',inf=F
alse,exp=True,age=np.random.randint(17,21),params=params[loc,:],cov_ma
t=cov_mats[loc,:,:],herd=g))
        for m in range(suscept_adult_males):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Male',inf=Fal
se,exp=False,age=np.random.randint(29,65),params=params[loc,:],cov_mat
=cov_mats[loc,:,:],herd=g))
        for f in range(suscept_adult_females):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Female',inf=F
alse,exp=False,age=np.random.randint(29,65),params=params[loc,:],cov_m
at=cov_mats[loc,:,:],herd=g))
        for m in range(infected_adult_males):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Male',inf=Fal
```

```
se,exp=True,age=np.random.randint(29,65),params=params[loc,:],cov_mat=
cov_mats[loc,:,:],herd=g))
        for f in range(infected_adult_females):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Female',inf=F
alse,exp=True,age=np.random.randint(29,65),params=params[loc,:],cov_ma
t=cov_mats[loc,:,:],herd=g))
        for m in range(suscept_fawn_males):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Male',inf=Fal
se,exp=False,age=np.random.randint(5,8),params=params[loc,:],cov_mat=c
ov_mats[loc,:,:],herd=g))
        for f in range(suscept_fawn_females):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Female',inf=F
alse,exp=False,age=np.random.randint(5,8),params=params[loc,:],cov_mat
=cov_mats[loc,:,:],herd=g))
        for m in range(infected_fawn_males):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Male',inf=Fal
se,exp=True,age=np.random.randint(5,8),params=params[loc,:],cov_mat=co
v_mats[loc,:,:],herd=g))
        for f in range(infected_fawn_females):
            loc = np.random.randint(params.shape[0])
            g = np.random.randint(0,groups)

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),gender='Female',inf=F
alse,exp=True,age=np.random.randint(5,8),params=params[loc,:],cov_mat=
cov_mats[loc,:,:],herd=g))

###################################
## World directs how deer move ##
###################################

    def move_deer(self):

        # non-stationary movements
        b_hops = []
        for h in self.herd_list:
            b_hops.append(h.basin_hop(self.days))
```

```
        for d in self.deer_list:

d.move(b_hops[d.herd_id],self.herd_list[d.herd_id].cx,self.herd_list[d
.herd_id].cy)

    def initialize_infection(self):
        group_zero = [d for d in self.deer_list if d.herd_id ==0]
        np.random.choice(group_zero).infectious=True

    def fall_dispersal(self):
        for d in self.deer_list:
            if d.gender=='Male' and 12<=d.age<24 and d.disp_int >= 5:
                if np.random.uniform(0,1) < YEARLING_FALL_DISP_RATE:
                    tot_groups = list(range(len(self.herd_list)))
                    tot_groups.remove(d.herd_id)

                    new_group = np.random.choice(tot_groups)

                    d.herd_id = new_group
                    d.disp_int = 0

            if d.gender == 'Male' and 24<=d.age<36 and d.disp_int>=5:
                if np.random.uniform(0,1) < 0.003:
                    tot_groups = list(range(len(self.herd_list)))
                    tot_groups.remove(d.herd_id)

                    new_group = np.random.choice(tot_groups)

                    d.herd_id = new_group
                    d.disp_int = 0

    def spring_dispersal(self):
        for d in self.deer_list:
            if d.gender == 'Male' and 12<=d.age<24 and d.disp_int>=5:
                if np.random.uniform(0,1) < YEARLING_SPRING_DISP_RATE:
                    tot_groups = list(range(len(self.herd_list)))
                    tot_groups.remove(d.herd_id)

                    new_group = np.random.choice(tot_groups)

                    d.herd_id = new_group
                    d.disp_int = 0

    def spring_emigration(self):
        loc=np.random.randint(self.params.shape[0])
        g=np.random.randint(0,self.groups)
        for d in self.deer_list:
            if d.gender == 'Male' and 11<=d.age<=22:
                if np.random.uniform(0,1) < YEARLING_SPRING_EM_RATE:
                    d.alive = False
                    if d.exposed == True or d.infectious == True:
                        self.emigrants += 1

    def spring_immigration(self):
```

```
        loc=np.random.randint(self.params.shape[0])
        g=np.random.randint(0,self.groups)
        if np.random.uniform(0,1) < YEARLING_SPRING_EM_RATE:

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),
gender='Male',inf=False,exp=False,age=np.random.randint(11,13),params=
self.params[loc,:],cov_mat=self.cov_mats[loc,:,:],herd=g))

    def fall_emigration(self):
        loc=np.random.randint(self.params.shape[0])
        g=np.random.randint(0,self.groups)
        for d in self.deer_list:
            if d.gender == 'Male' and 14 < d.age <= 18:
                if np.random.uniform(0,1) < YEARLING_FALL_EM_RATE:
                    d.alive = False
                    if d.exposed == True or d.infectious == True:
                        self.emigrants += 1

    def fall_immigration(self):
        loc=np.random.randint(self.params.shape[0])
        g=np.random.randint(0,self.groups)
        if np.random.uniform(0,1) < YEARLING_FALL_EM_RATE:

self.deer_list.append(Deer(x=self.group_pos[g][0]+np.random.normal(10,
1),y=self.group_pos[g][1]+np.random.normal(10,1),
gender='Male',inf=False,exp=False,age=np.random.randint(14,18),params=
self.params[loc,:],cov_mat=self.cov_mats[loc,:,:],herd=g))

    def incr_age(self):
        for d in self.deer_list:
            d.age_deer()

            if d.age >= 216 and d.alive:
                d.alive = False
                self.max_life += 1

    def incr_time_inf(self):
        for d in self.deer_list:
            d.infected_count()

    def incr_birth_int(self):
        for d in self.deer_list:
            d.birth_interval()

    def incr_disp_int(self):
        for d in self.deer_list:
            d.disp_interval()

    def birth(self):
        for d in self.deer_list:
            if d.age>=22 and d.gender=='Female' and d.birth_int>=5:
            #Adults have a mean litter size of 2.0
                if BIRTH_RATE > np.random.uniform():
```

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                    litter_size = np.random.choice([0,1,2,3],
p=[0.070747,0.158505,0.670748,0.1])
                    d.birth_int = 0
                    for f in range(litter_size):
                        loc = np.random.randint(self.params.shape[0])
                        self.deer_list.append(Deer(x=d.x,y=d.y,
inf=False,exp=False,gender=random.choice(['Male','Female']),age=0,
params=self.params[loc,:],cov_mat=self.cov_mats[loc,:,:],herd=d.herd_i
d))

            if 13<=d.age<22 and d.gender=='Female' and d.birth_int>=5:
            #Yearlings have mean of 1.8 fawns/litter
                if BIRTH_RATE > np.random.uniform():
                    litter_size = np.random.choice([0,1,2,3],
p=[0.05,0.45,0.45,0.05])
                    d.birth_int = 0
                    for f in range(litter_size):
                        loc = np.random.randint(self.params.shape[0])
                        self.deer_list.append(Deer(x=d.x, y=d.y,
inf=False,exp=False,gender=random.choice(['Male','Female']),age=0,
params=self.params[loc,:],cov_mat=self.cov_mats[loc,:,:],herd=d.herd_i
d))

            if d.age<13 and d.gender=='Female' and d.birth_int>=5:
            #Fawns have mean of 1.2 fawns
                if BIRTH_RATE > np.random.uniform():
                    litter_size = np.random.choice([0,1,2,3],
p=[0.5,0.5,0,0])
                    d.birth_int = 0
                    for f in range(litter_size):
                        loc = np.random.randint(self.params.shape[0])
                        self.deer_list.append(Deer(x=d.x, y=d.y,
inf=False, exp=False, gender=random.choice(['Male','Female']), age=0,
params=self.params[loc,:],cov_mat=self.cov_mats[loc,:,:],herd=d.herd_i
d))


    def shed_prions(self):
        for d in self.deer_list:
            if d.infectious == True and d.alive == True:
                self.prion_map[int((d.y-rem_yllc)/rem_cs),int((d.x-
rem_xllc)/rem_cs)] += 1*(int(d.time_infected/30)+1)

            if d.infectious == True and d.alive == False:
                self.prion_map[int((d.y-rem_yllc)/rem_cs),int((d.x-
rem_xllc)/rem_cs)] += 50*(int(d.time_infected/30)+1)

    def prion_decay(self):
        self.prion_map *= 1-decay_rate

    def ind_trans(self):
        for d in self.deer_list:
            if d.infectious == False and d.exposed == False:
                if np.random.uniform() < 1 - np.exp(-
```

```
IND_TRANS*self.prion_map[int((d.y-rem_yllc)/rem_cs),int((d.x-
rem_xllc)/rem_cs)]/rem_cs**2):
                    d.exposed = True
                    self.ind_transmissions += 1

    def dir_trans_jan(self,dir_radius):
        '''
        January direct transmission events
        For every pair of deer where one is infectious and one is
susceptible (exposed==False)
        '''
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
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                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_feb(self,dir_radius):
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*3 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
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                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*3 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_mar(self,dir_radius):
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*3 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
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                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*3 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1


    def dir_trans_apr(self,dir_radius):
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
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                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_may(self,dir_radius):
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*3 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
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                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*3 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_june(self,dir_radius):
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*3 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
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                            deer_2.exposed=True
                            self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*3 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1

    def dir_trans_july(self,dir_radius):

        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if DIR_TRANS*3 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if DIR_TRANS*1 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
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deer_2.gender=='Male':
                            if DIR_TRANS*3 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.gender=='Male' and
deer_2.gender=='Female':
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.gender=='Female' and
deer_2.gender=='Male':
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.gender=='Female' and
deer_2.gender=='Female':
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_aug(self,dir_radius):

        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male': # M-M
                            if DIR_TRANS*7 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.gender=='Male' and
deer_2.gender=='Female':
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.gender=='Female' and
deer_2.gender=='Male':
                            if DIR_TRANS*2 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.gender=='Female' and
deer_2.gender=='Female':
                            if deer_1.herd_id != deer_2.herd_id:
                                if DIR_TRANS*1 > np.random.uniform():
                                    deer_2.exposed=True
                                    self.dir_transmissions += 1
                            if deer_1.herd_id == deer_2.herd_id:
                                if DIR_TRANS*4 > np.random.uniform():
                                    deer_2.exposed=True
                                    self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
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deer_2.gender=='Male':
                        if DIR_TRANS*7 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female': # F-F
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_sept(self,dir_radius):

        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*7 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*2 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
```

```
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*7 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*2 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_oct(self,dir_radius):

        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*1 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*5 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*5 >
```

```
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*1 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*5 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*5 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_nov(self,dir_radius):

        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*1 >
np.random.uniform():
                            deer_2.exposed=True
```

```
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*2 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*1 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*2 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def dir_trans_dec(self,dir_radius):
```

```
        for deer_1,deer_2 in itertools.combinations(self.deer_list,2):
            if (deer_1.infectious ^ deer_2.infectious) and (deer_1 -
deer_2 < dir_radius):
                if deer_1.infectious==True and deer_2.exposed==False:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*1 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*2 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

                if deer_1.infectious==False and deer_2.exposed==True:
                    if deer_1.gender=='Male' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*1 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Male' and
deer_2.gender=='Female':
                        if DIR_TRANS*2 > np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Male':
                        if MALE_ADDED_DT*DIR_TRANS*2 >
np.random.uniform():
                            deer_2.exposed=True
                            self.dir_transmissions += 1
                    if deer_1.gender=='Female' and
deer_2.gender=='Female':
                        if deer_1.herd_id != deer_2.herd_id:
                            if DIR_TRANS*1 > np.random.uniform():
```

```
                                deer_2.exposed=True
                                self.dir_transmissions += 1
                        if deer_1.herd_id == deer_2.herd_id:
                            if DIR_TRANS*4 > np.random.uniform():
                                deer_2.exposed=True
                                self.dir_transmissions += 1

    def disease_mort(self):
        '''
        This function simulates CWD-associated mortality
        Sets infected deer to dead after 5 years post-initial
infection. Daily probabilities
        '''
        for d in self.deer_list:
            if d.time_infected > 300 and np.random.uniform() <
DIS_MORT:
                d.alive = False
                self.dz_mort += 1
            if d.time_infected >= 1800: #(exposed+infectious periods)
                d.alive = False
                self.max_disease += 1

    def harvest_mort(self):
        '''
        This function simulates harvest mortality for each deer
demographic
        '''
        for d in self.deer_list:
            if d.gender == 'Female' and 11 < d.age < 23 and
np.random.uniform() < YEARLING_FEMALE_HARVEST_RATE:
                d.alive = False
            if d.gender == 'Male' and 11 < d.age < 23 and
np.random.uniform() < YEARLING_MALE_HARVEST_RATE:
                d.alive = False
            if d.gender == 'Male' and d.age >=23 and
np.random.uniform() < ADULT_MALE_HARVEST_RATE:
                d.alive = False
            if d.gender == 'Female' and d.age >=23 and
np.random.uniform() < ADULT_FEMALE_HARVEST_RATE:
                d.alive = False

    def fawn_mort(self):
        '''
        This function simulates increased mortality of fawns during
their first 12 months of life (predation, abandonment, etc.)
        Fawn harvest mortality included in this parameter
        '''
        for d in self.deer_list:
            if 0 <= d.age < 2 and np.random.uniform() <
FAWN_TWO_MONTH_MORT_RATE:
                d.alive = False
            if 2 <= d.age <= 12 and np.random.uniform() <
FAWN_TEN_MONTH_MORT_RATE:
                d.alive = False
```

```
    def baseline_mort(self):
        '''
        This function simulates baseline mortality of deer during the
non-hunting season and to keep population stationary (i.e., not
growing; predation, DVCs, etc.)
        '''
        for d in self.deer_list:
            if d.gender == 'Male' and d.age >= 23 and
np.random.uniform() < ADULT_MALE_BASE_MORT_RATE:
                d.alive = False
            if d.gender == 'Female' and d.age >= 23 and
np.random.uniform() < ADULT_FEMALE_BASE_MORT_RATE:
                d.alive = False
            if d.gender == 'Male' and 11 < d.age < 23 and
np.random.uniform() < YEARLING_MALE_BASE_MORT_RATE:
                d.alive = False
            if d.gender == 'Female' and 11 < d.age < 23 and
np.random.uniform() < YEARLING_FEMALE_BASE_MORT_RATE:
                d.alive = False

    def male_comp(self):
        self.male_list = [d for d in self.deer_list if d.alive == True
and d.gender == 'Male']

    def female_comp(self):
        self.female_list = [d for d in self.deer_list if d.alive ==
True and d.gender == 'Female']

    def list_comp(self):
        self.deer_list = [d for d in self.deer_list if d.alive==True]

    def increment_day(self):
        self.days += 1

###############################################
## Calculate statistics for each time step ##
###############################################

    def stats(self):
        total_deer = len(self.deer_list)
        exposed_deer = 0
        infectious_deer = 0

        total_males = len(self.male_list)
        exposed_males = 0
        infectious_males = 0

        total_females = len(self.female_list)
        exposed_females = 0
        infectious_females = 0

        total_cells = int(rm_rows*rm_cols)
        prion_deposited_cells = (self.prion_map!=0).sum()
```

```
        for d in self.deer_list:
           if d.infectious == True:
               infectious_deer += 1
           elif d.exposed == True:
               exposed_deer += 1
           else:
               pass

        for d in self.male_list:
            if d.infectious == True:
                infectious_males += 1
            elif d.exposed == True:
                exposed_males += 1
            else:
                pass

        for d in self.female_list:
            if d.infectious == True:
                infectious_females += 1
            elif d.exposed == True:
                exposed_females += 1
            else:
                pass

        return total_deer, exposed_deer, infectious_deer,
((exposed_deer+infectious_deer)/total_deer),self.dir_transmissions,
self.ind_transmissions, self.max_life, self.max_disease,
self.dz_mort,self.emigrants,((exposed_males+infectious_males)/
total_males), ((exposed_females+infectious_females)/
total_females),(prion_deposited_cells/total_cells)

######################################################################
## Initializes the agents (deer) and functions happening to them ##
######################################################################

class Deer():
    def
__init__(self,x=None,y=None,inf=False,exp=False,gender='Male',age=0,ti
me_infected=0,birth_int=0,disp_int=0,params='',cov_mat='',herd=0):
        self.x = x
        self.y = y

        self.infectious = inf
        self.exposed = exp
        self.gender = gender
        self.alive = True
        self.age = age
        self.time_infected = time_infected
        self.birth_int = 0
        self.disp_int = 0
        self.cov_mat = cov_mat
        self.params = params
        self.herd_id = herd
```

```
    def move(self,b_hop,cx,cy):

        has_moved = False

        attempts = 0
        while has_moved == False and attempts < 100:
            rand = self.rng(1)

            new_x=self.x-self.params[0]*(self.x-cx) + rand[0][0] +
b_hop[0]
            new_y=self.y-self.params[1]*(self.y-cy) + rand[1][0] +
b_hop[1]

            if int((new_y - bm_yllc)/bm_cs) < bm_rows and int((new_y -
bm_yllc)/bm_cs) >= 0 and int((new_x - bm_xllc)/bm_cs) < bm_cols and
int((new_x - bm_xllc)/bm_cs) >= 0:

                if BINARY_MAP[int((new_y - bm_yllc)/bm_cs), int((new_x
- bm_xllc)/bm_cs)]:
                    # stationary part
                    self.x = new_x
                    self.y = new_y
                    has_moved = True

                else:
                    attempts += 1
            else:
                attempts += 1

    def rng(self,n):
        mvnorm = stats.multivariate_normal(mean=[0,0],
cov=self.cov_mat)

        x = mvnorm.rvs(n).reshape(n,2)

        norm1 = stats.norm(0,np.sqrt(self.cov_mat[0,0]))
        norm2 = stats.norm(0,np.sqrt(self.cov_mat[1,1]))

        x_unif1 = norm1.cdf(x[:,0])
        x_unif2 = norm2.cdf(x[:, 1])

        bx = self.params[5]
        by = self.params[6]

        m1 = stats.laplace(0,bx)
        m2 = stats.laplace(0,by)

        x1_trans = m1.ppf(x_unif1)
        x2_trans = m2.ppf(x_unif2)

        return x1_trans,x2_trans

    def age_deer(self):
        self.age += 1
```

```
    def infected_count(self):
        if self.exposed or self.infectious:
            self.time_infected += 1

        if self.time_infected > np.random.randint(90,180):
            self.infectious = True
            self.exposed = False

    def birth_interval(self):
        self.birth_int += 1

    def disp_interval(self):
        self.disp_int += 1

    def __sub__(self, other):
        return np.sqrt((self.x - other.x)**2 + (self.y - other.y)**2)

#################################################################
## Initializes the deer groups and functions happening to them ##
#################################################################

class Herd():
    def __init__(self,pos,params,xtrds,ytrds,total_timesteps):

        self.params = params
        self.trd_x = xtrds
        self.trd_y = ytrds
        self.kde = self.make_kde()

        self.cx = pos[0]
        self.cy = pos[1]

        self.lasso_times = self.lasso_times(total_timesteps)

    def make_kde(self):

        maxCol=lambda x: max(x.min(), x.max(), key=abs)

        smth_lasso_jumps =
np.stack([pd.DataFrame(np.diff(self.trd_x)).rolling(3,center=True).app
ly(maxCol).dropna().to_numpy().flatten(),pd.DataFrame(np.diff(self.trd
_y)).rolling(3,center=True).apply(maxCol).dropna().to_numpy().flatten(
)],axis=1)

        pts = smth_lasso_jumps

        points = list(zip(np.diff(pts[:,0]),np.diff(pts[:,1])))
        distances = [dist(p1,p2) for p1,p2 in combinations(points, 2)]
        avg_distance = sum(distances) / len(distances)

        lasso_pdf = KernelDensity(kernel='gaussian',
bandwidth=avg_distance*.5)
        lasso_pdf.fit(smth_lasso_jumps)
```

```
        return lasso_pdf


    def basin_hop(self,time):

        if np.any(self.lasso_times == time):

            jump = self.kde.sample(3)

            possible_jumps = []
            attempts = 0
            while len(possible_jumps) < 3:
                new_x,new_y = jump[len(possible_jumps)] +
[self.cx,self.cy]

                if int((new_y - rm_yllc)/rm_cs) < rm_rows and
int((new_y - rm_yllc)/rm_cs) >= 0 and int((new_x - rm_xllc)/rm_cs) <
rm_cols and int((new_x - rm_xllc)/rm_cs) >= 0:

                    possible_jumps.append(RSF_MAP[int((new_y-
rm_yllc)/rm_cs),int((new_x-rm_xllc)/rm_cs)])

                else:
                    attempts += 1

                if attempts > 100:
                    return [0,0]

            x_jump_ = jump[np.argmax(possible_jumps)][0]
            y_jump_ = jump[np.argmax(possible_jumps)][1]

            # move the center
            self.cx += x_jump_
            self.cy += y_jump_

            return [x_jump_,y_jump_]

        else:
            return [0,0]

    def lasso_times(self,total_timesteps):

        num_crit_jumps_data = self.params[3]

        jump_times = [np.random.exponential(self.params[4])]
        while np.cumsum(jump_times)[-1] <= total_timesteps:
            jump_times.append(np.random.exponential(self.params[4]))
        return jump_times

fits = np.load('./Data/fits.npy')
xtrends = np.load('./Data/xtrends.npy',allow_pickle=True)
ytrends = np.load('./Data/ytrends.npy',allow_pickle=True)
cov_matrices = np.load('./Data/covar_matrices.npy')
```

```
#################################
## Initializes Simulation Trial ##
#################################

def disease(timesteps):
    np.random.seed()

    data = np.zeros((13,timesteps))

    dir_radius = 100

    T_total_deer=np.zeros(timesteps)
    T_exposed_deer=np.zeros(timesteps)
    T_infectious_deer=np.zeros(timesteps)
    T_prevalence=np.zeros(timesteps)
    T_dir_trans=np.zeros(timesteps)
    T_ind_trans=np.zeros(timesteps)
    T_max_life=np.zeros(timesteps)
    T_max_disease=np.zeros(timesteps)
    T_dz_mort=np.zeros(timesteps)
    T_emigration=np.zeros(timesteps)
    T_m_prevalence=np.zeros(timesteps)
    T_f_prevalence=np.zeros(timesteps)
    T_shed_prions=np.zeros(timesteps)

    trial =
World(total_timesteps=timesteps,suscept_yrling_males=111,infected_yrli
ng_males=1,suscept_yrling_females=138,infected_yrling_females=0,suscep
t_adult_males=134,infected_adult_males=0,suscept_adult_females=413,inf
ected_adult_females=0,suscept_fawn_males=229,infected_fawn_males=0,sus
cept_fawn_females=231, infected_fawn_females=0,groups=GROUPS,
params=fits,xtrds=xtrends,ytrds=ytrends,cov_mats=cov_matrices)

    month = 1
    day = 1
    year = 1

    for s in range(1, timesteps+1):
       trial.move_deer()
       trial.incr_time_inf()
       trial.shed_prions()
       trial.ind_trans()
       trial.disease_mort()
       trial.fawn_mort()

       if not s % 30:
           trial.incr_age()
           trial.incr_birth_int()
           trial.incr_disp_int()
           trial.prion_decay()

           day=1
           month += 1
```

```
    if month > 12:
        year+=1
        month=1
        day = 1

if month == 1:
    trial.dir_trans_jan(dir_radius)
    trial.harvest_mort()
if month == 2:
    trial.baseline_mort()
    trial.dir_trans_feb(dir_radius)
if month == 3:
    trial.baseline_mort()
    trial.dir_trans_mar(dir_radius)
if month == 4:
    trial.baseline_mort()
    trial.dir_trans_apr(dir_radius)
if month == 5:
    trial.baseline_mort()
    trial.dir_trans_may(dir_radius)
    trial.birth()
if month == 6:
    trial.baseline_mort()
    trial.spring_dispersal()
    trial.spring_emigration()
    trial.spring_immigration()
    trial.dir_trans_june(dir_radius)
    trial.birth()
if month == 7:
    trial.baseline_mort()
    trial.dir_trans_july(dir_radius)
if month == 8:
    trial.baseline_mort()
    trial.dir_trans_aug(dir_radius)
if month ==9:
    trial.baseline_mort()
    trial.dir_trans_sept(dir_radius)
if month == 10:
    trial.fall_dispersal()
    trial.fall_emigration()
    trial.fall_immigration()
    trial.dir_trans_oct(dir_radius)
    trial.harvest_mort()
if month == 11:
    trial.dir_trans_nov(dir_radius)
    trial.harvest_mort()
if month ==12:
    trial.dir_trans_dec(dir_radius)
    trial.harvest_mort()

trial.list_comp()
trial.male_comp()
trial.female_comp()
```

```
        trial.increment_day()
        day += 1

t_deer,e_deer,i_deer,prev,d_trans,i_trans,m_life,m_disease,d_mort,emig
rant,m_prev,f_prev,prion_cells=trial.stats()
        T_total_deer[s-1]+=t_deer
        T_exposed_deer[s-1]+=e_deer
        T_infectious_deer[s-1]+=i_deer
        T_prevalence[s-1]+=prev
        T_dir_trans[s-1]+=d_trans
        T_ind_trans[s-1]+=i_trans
        T_max_life[s-1]+=m_life
        T_max_disease[s-1]+=m_disease
        T_dz_mort[s-1]+=d_mort
        T_emigration[s-1]+=emigrant
        T_m_prevalence[s-1]+=m_prev
        T_f_prevalence[s-1]+=f_prev
        T_shed_prions[s-1]+=prion_cells

        data[0,:] = T_total_deer
        data[1,:] = T_exposed_deer
        data[2,:] = T_infectious_deer
        data[3,:] = T_prevalence
        data[4,:] = T_dir_trans
        data[5,:] = T_ind_trans
        data[6,:] = T_max_life
        data[7,:] = T_max_disease
        data[8,:] = T_dz_mort
        data[9,:] = T_emigration
        data[10,:] = T_m_prevalence
        data[11,:] = T_f_prevalence
        data[12,:] = T_shed_prions

    return data

res = disease(3600)   #total timesteps
np.savetxt(PATH+'/Total_deer/rank_'+str(rank)+'_total_deer',res[0,:],d
elimiter=',')
np.savetxt(PATH+'/Total_exp_deer/rank_'+str(rank)+'_exposed_deer',res[
1,:],delimiter=',')
np.savetxt(PATH+'/Total_infect_deer/rank_'+str(rank)+'_infect_deer',re
s[2,:],delimiter=',')
np.savetxt(PATH+'/Prevalence/rank_'+str(rank)+'_prev',res[3,:],delimit
er=',')
np.savetxt(PATH+'/Direct_trans/rank_'+str(rank)+'_dir_trans',res[4,:],
delimiter=',')
np.savetxt(PATH+'/Indirect_trans/rank_'+str(rank)+'_ind_trans',res[5,:
],delimiter=',')
np.savetxt(PATH+'/Max_life/rank_'+str(rank)+'_max_life',res[6,:],delim
iter=',')
np.savetxt(PATH+'/Max_disease/rank_'+str(rank)+'_max_dz',res[7,:],deli
miter=',')
np.savetxt(PATH+'/Disease_mort/rank_'+str(rank)+'_dz_mort',res[8,:],de
limiter=',')
```

```
np.savetxt(PATH+'/Emigrants/rank_'+str(rank)+'_emigration',res[9,:],de
limiter=',')
np.savetxt(PATH+'/Prevalence_male/rank_'+str(rank)+'_m_prev',res[10,:]
,delimiter=',')
np.savetxt(PATH+'/Prevalence_female/rank_'+str(rank)+'_f_prev',res[11,
:],delimiter=',')
np.savetxt(PATH+'/Prion_cells/rank_'+str(rank)+'_prion_cells',res[11,:
],delimiter=',')
```

APPENDIX C
DIAGNOSTIC OUTPUT FOR MONTE CARLO MARKOV CHAIN CONVERGENCE IN CHAPTER 2

Table C1. Gelman and Rubin diagnostic statistics for each parameter of the Bayesian hurdle model performed on the output generated by the individual-based model in Chapter 2.

	CWD Persistence		CWD Prevalence	
	Mean	Upper CI	Mean	Upper CI
Intercept	1.07	1.21	1.01	1.04
Medium Density	1.06	1.20	1.01	1.04
High Density	1.05	1.15	1.01	1.03
Suburban study area	1.07	1.23	1	1.01
Parcel cull method	1.04	1.13	1	1.02
1.6-km ring cull method	1.02	1.07	1	1
2.4-km ring cull method	1.04	1.13	1	1.02
Habitat cull method	1.03	1.11	1.01	1.03
Medium Density*Suburban study area	1.06	1.18	1	1.01
High Density*Suburban study area	1.05	1.15	1	1
Medium Density*Parcel cull	1.04	1.14	1	1.02
High Density*Parcel cull	1.03	1.1	1	1.02
Medium Density*1.6-km ring cull	1.02	1.07	1	1
High Density*1.6-km ring cull	1.01	1.05	1	1
Medium Density*2.4-km ring cull	1.03	1.12	1	1.01
High Density*2.4-km ring cull	1.03	1.1	1	1.02
Medium Density*Habitat cull	1.03	1.11	1.01	1.03
High Density*Habitat cull	1.02	1.08	1.01	1.02
Suburban study area*Parcel cull	1.04	1.15	1	1
Suburban study area*1.6-km ring cull	1.02	1.07	1	1
Suburban study area*2.4-km ring cull	1.03	1.11	1	1
Suburban study area*Habitat cull	1.04	1.14	1	1.01
Medium Density*Suburban study area*Parcel cull	1.04	1.13	1	1
High Density*Suburban study area*Parcel cull	1.03	1.1	1	1
Medium Density*Suburban study area*1.6-km ring cull	1.01	1.05	1	1
High Density*Suburban study area*1.6-km ring cull	1.01	1.03	1	1
Medium Density*Suburban study area*2.4-km ring cull	1.02	1.09	1	1
High Density*Suburban study area*2.4-km ring cull	1.02	1.08	1	1
Medium Density*Suburban study area*Habitat cull	1.04	1.12	1	1.01
High Density*Suburban study area*Habitat cull	1.03	1.1	1	1.01

Figure C1. Gelman and Rubin diagnostic plots for the intercept on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

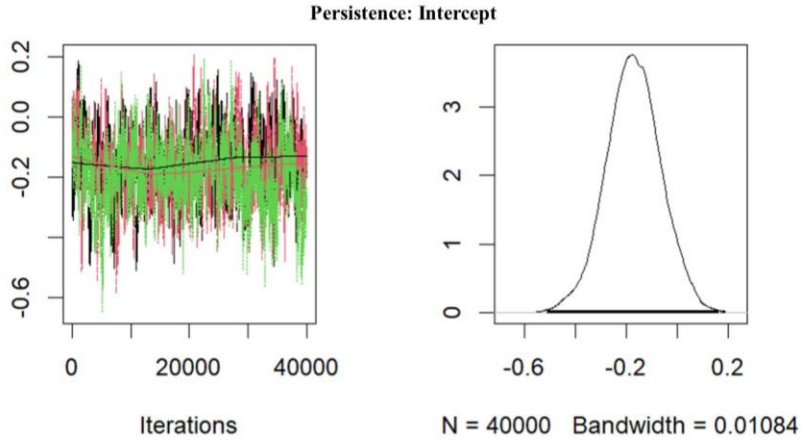


Figure C2. Gelman and Rubin diagnostic plots for the medium deer density parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

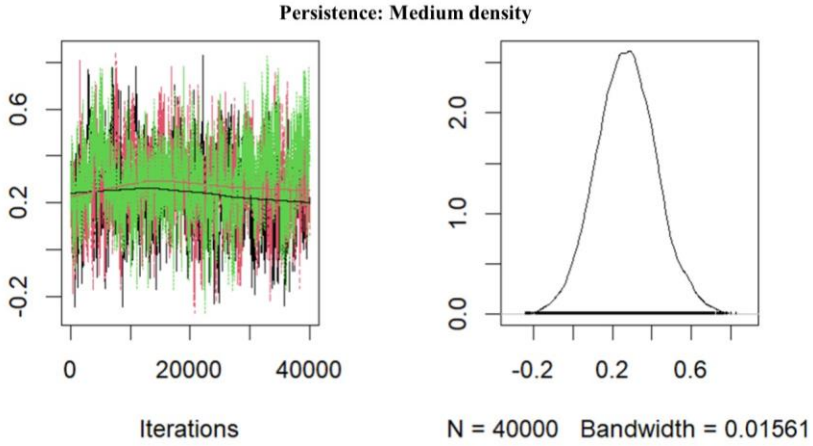


Figure C3. Gelman and Rubin diagnostic plots for the high deer density parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

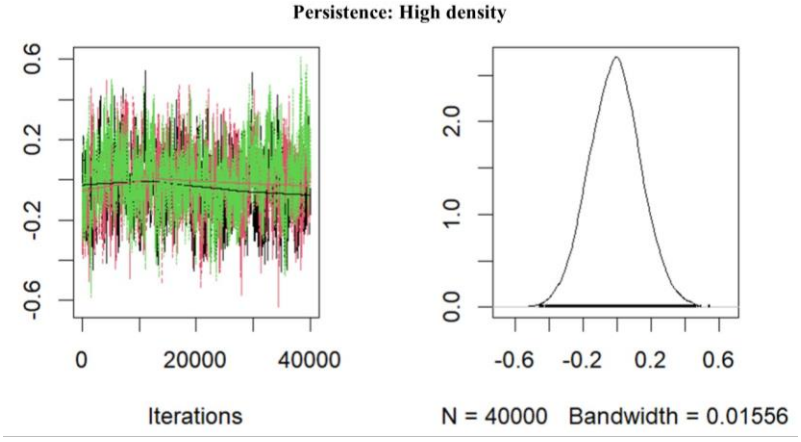


Figure C4. Gelman and Rubin diagnostic plots for the suburban study area parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

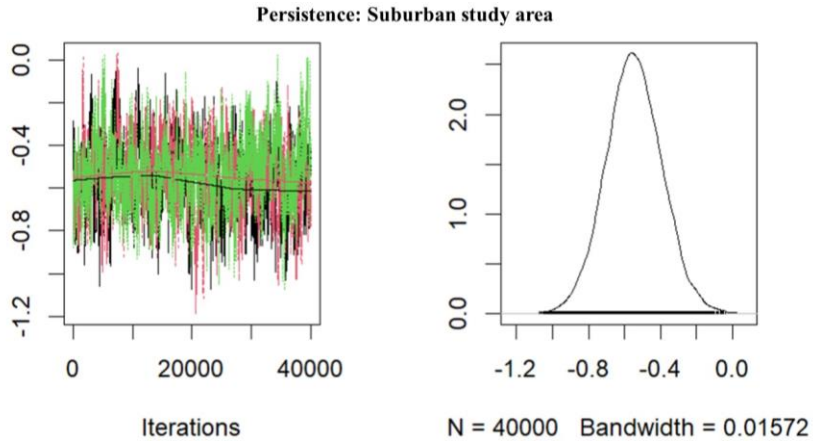


Figure C5. Gelman and Rubin diagnostic plots for the parcel cull method parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

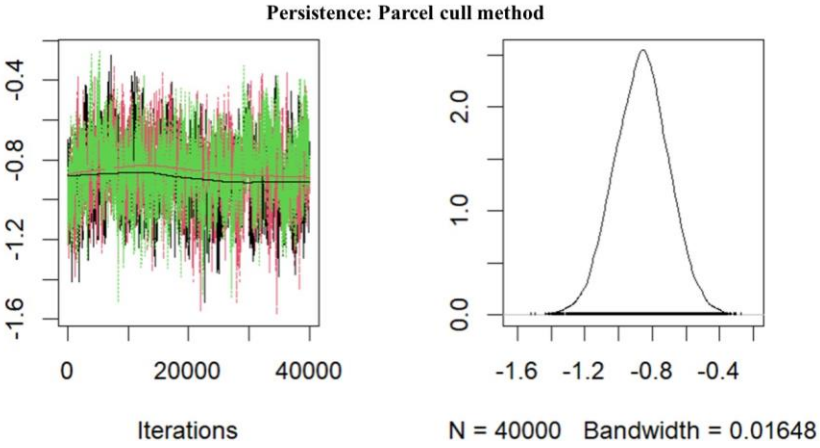


Figure C6. Gelman and Rubin diagnostic plots for the 1.6-km ring cull method parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

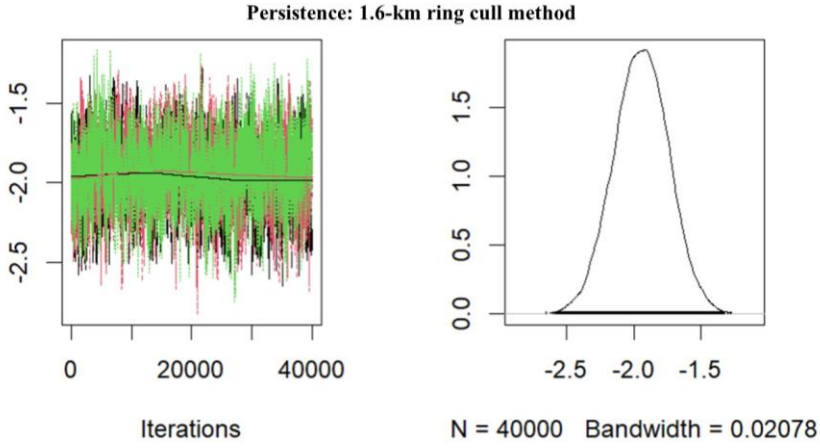


Figure C7. Gelman and Rubin diagnostic plots for the 2.4-km ring cull method parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

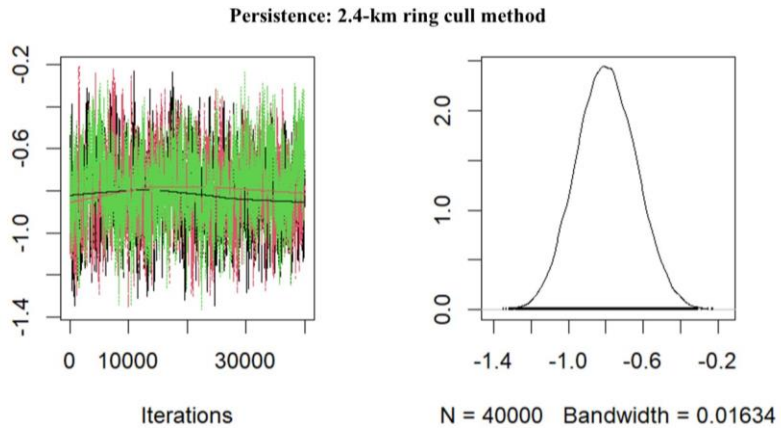


Figure C8. Gelman and Rubin diagnostic plots for the high quality habitat cull method parameter on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

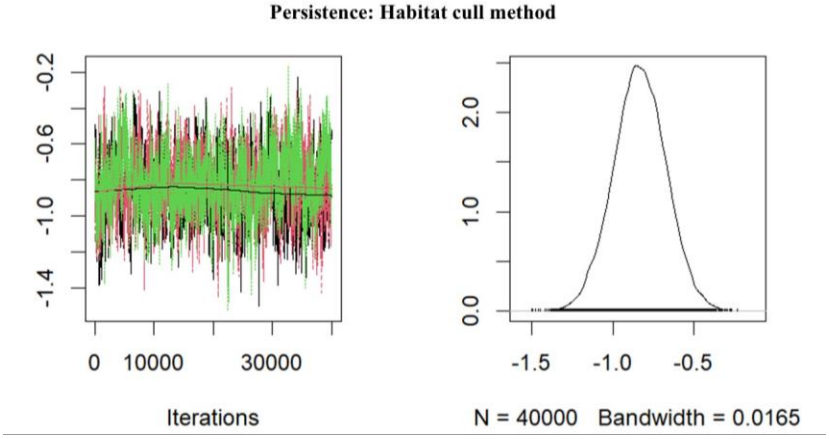


Figure C9. Gelman and Rubin diagnostic plots for the interaction between medium deer density and suburban study area on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

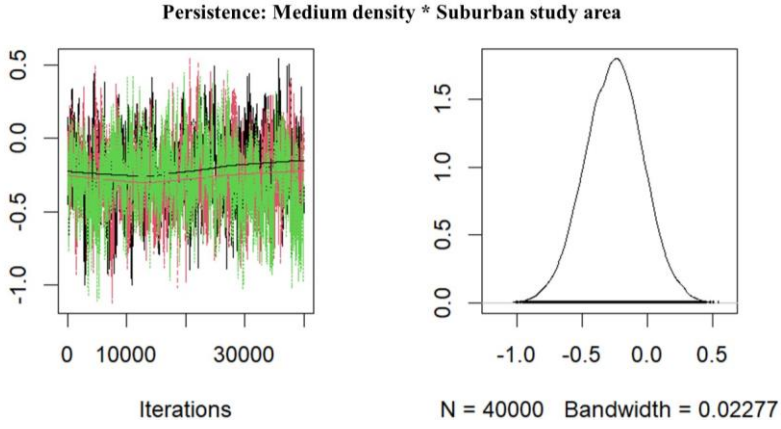


Figure C10. Gelman and Rubin diagnostic plots for the interaction between high deer density and suburban study area on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

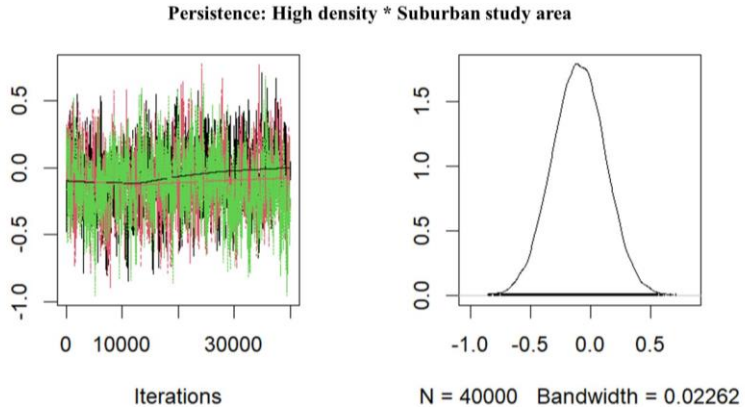


Figure C11. Gelman and Rubin diagnostic plots for the interaction between medium deer density and the parcel cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

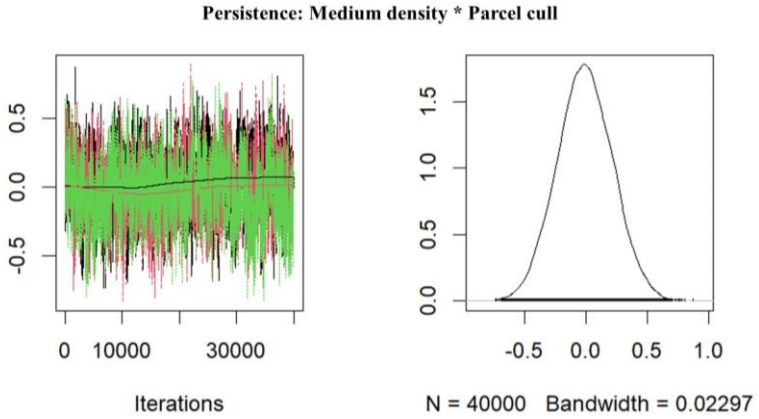


Figure C12. Gelman and Rubin diagnostic plots for the interaction between high deer density and parcel cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

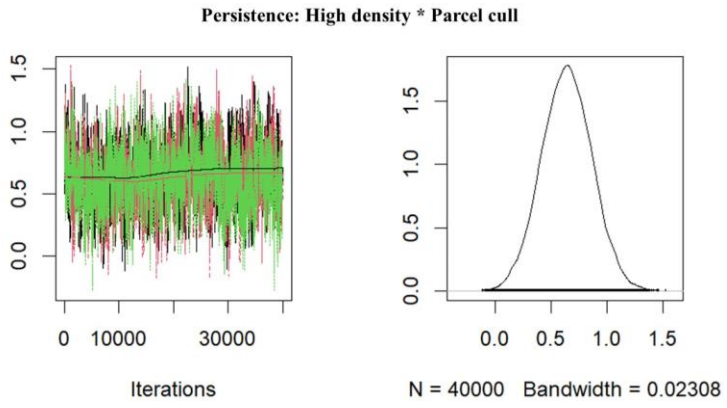


Figure C13. Gelman and Rubin diagnostic plots for the interaction between medium deer density and 1.6-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

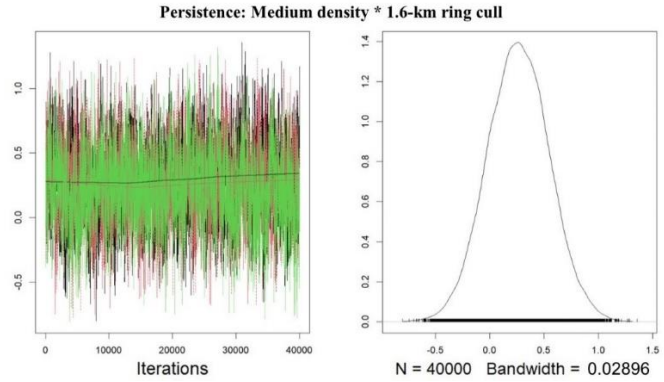


Figure C14. Gelman and Rubin diagnostic plots for the interaction between high deer density and 1.6-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

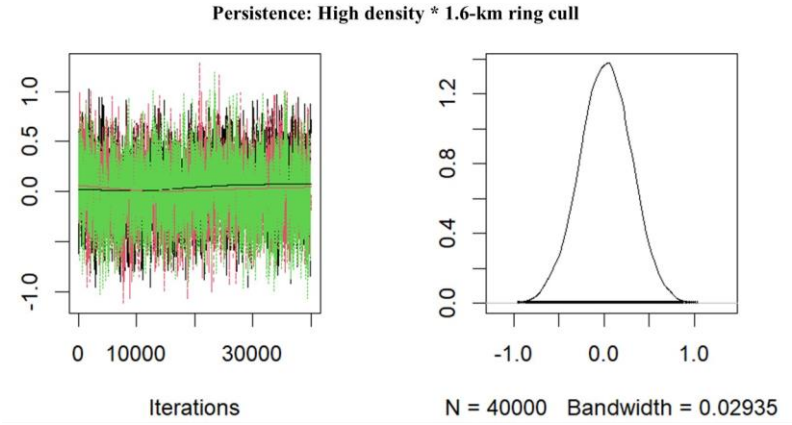


Figure C15. Gelman and Rubin diagnostic plots for the interaction between medium deer density and 2.4-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

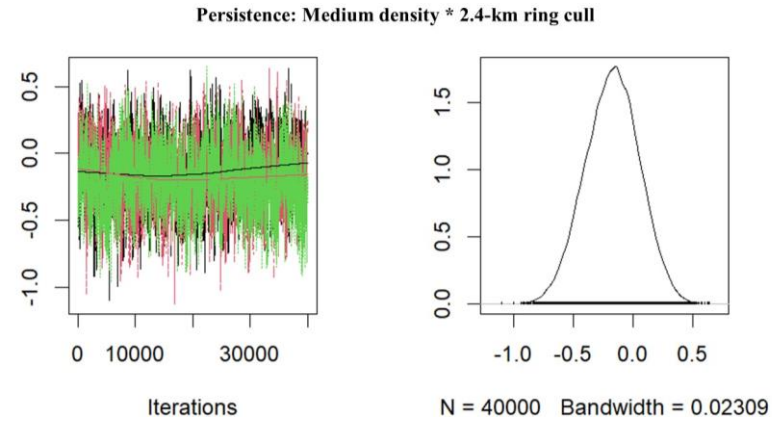


Figure C16. Gelman and Rubin diagnostic plots for the interaction between high deer density and 2.4-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

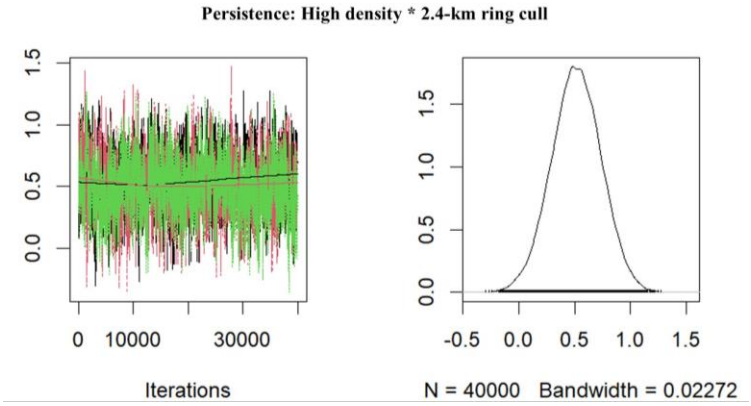


Figure C17. Gelman and Rubin diagnostic plots for the interaction between medium deer density and high quality habitat cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

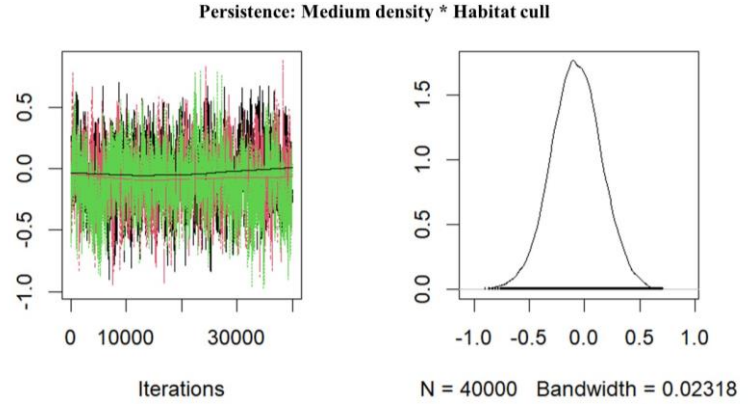


Figure C18. Gelman and Rubin diagnostic plots for the interaction between high deer density and high quality habitat cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

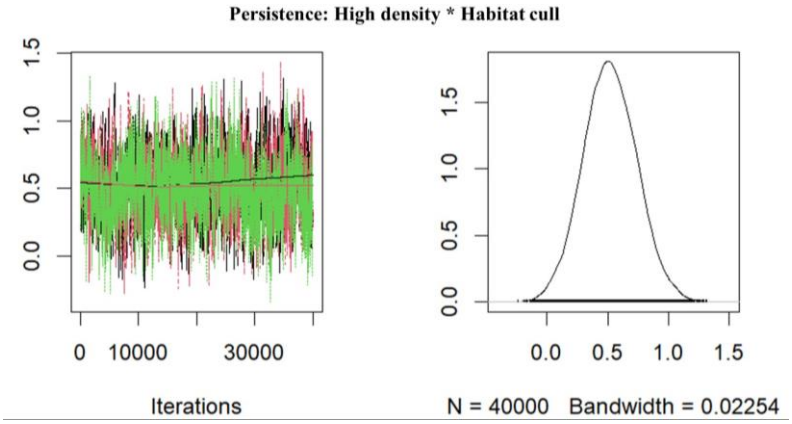


Figure C19. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and parcel cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

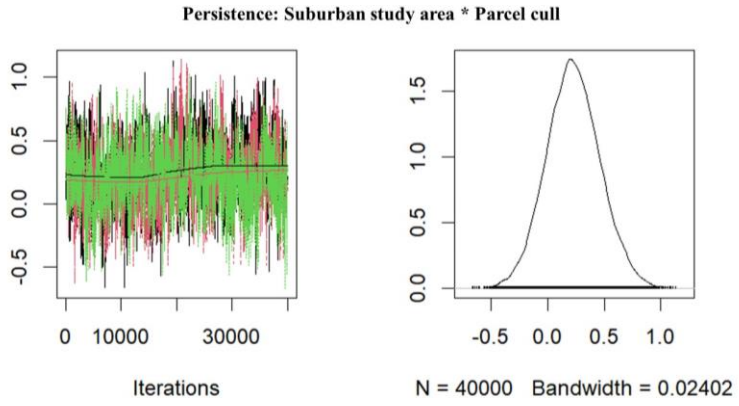


Figure C20. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and 1.6-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

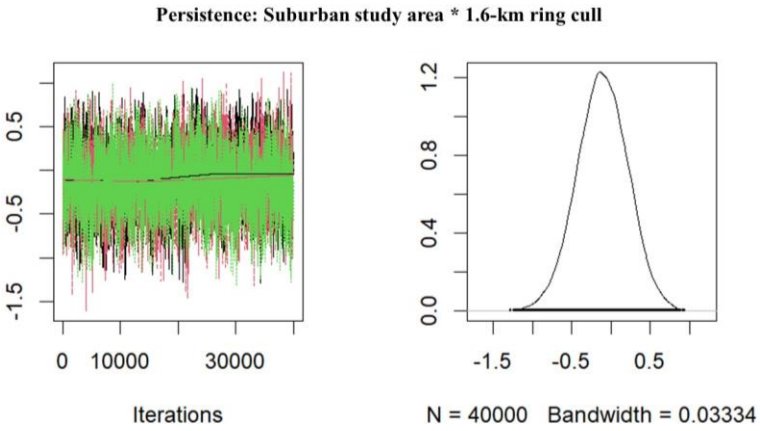


Figure C21. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and 1.6-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

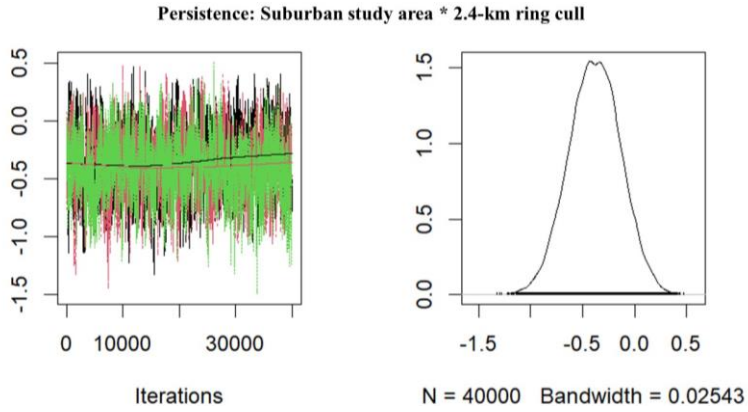


Figure C22. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and high quality habitat cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

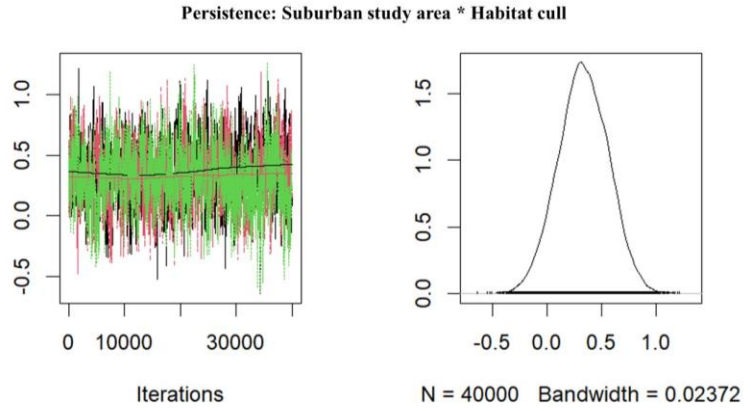


Figure C23. Gelman and Rubin diagnostic plots for the interaction among medium deer density, suburban study area, and parcel cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

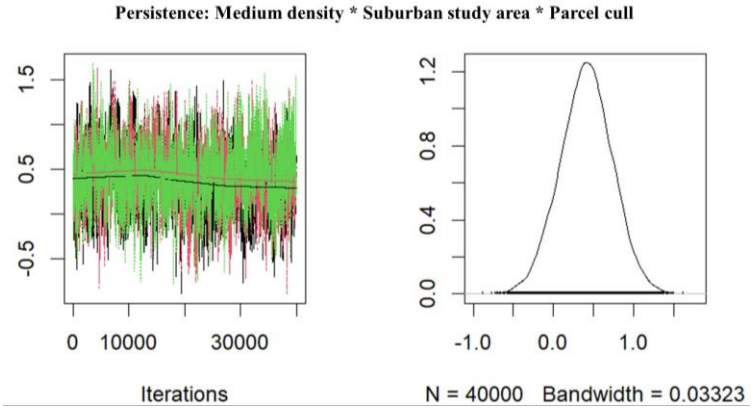


Figure C24. Gelman and Rubin diagnostic plots for the interaction among high deer density, suburban study area, and parcel cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

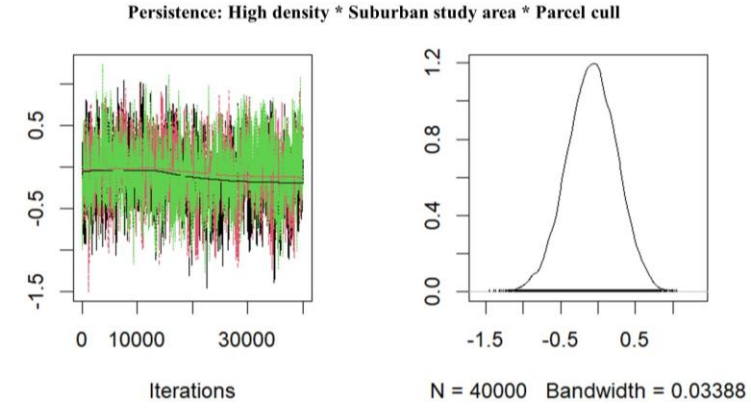


Figure C25. Gelman and Rubin diagnostic plots for the interaction among medium deer density, suburban study area, and 1.6-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

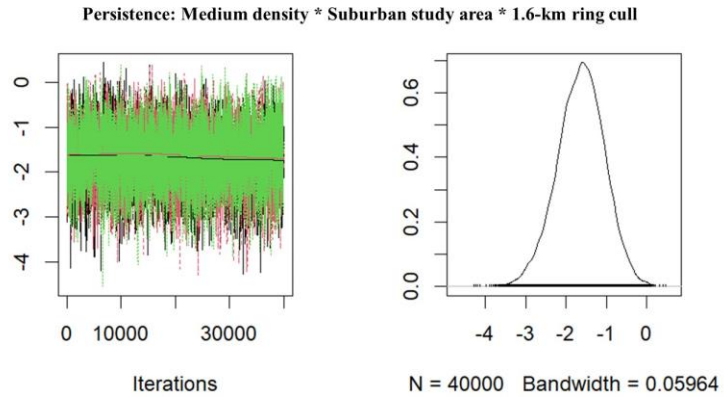


Figure C26. Gelman and Rubin diagnostic plots for the interaction among high deer density, suburban study area, and 1.6-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

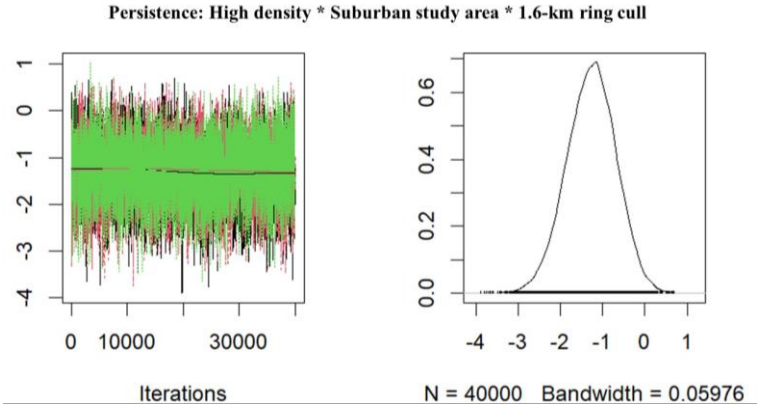


Figure C27. Gelman and Rubin diagnostic plots for the interaction among medium deer density, suburban study area, and 2.4-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

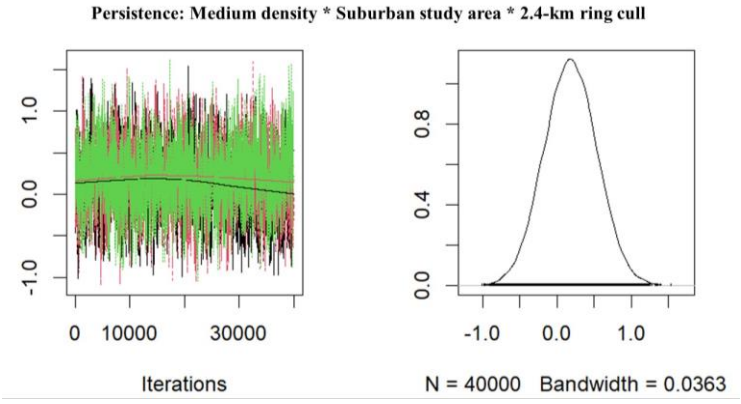


Figure C28. Gelman and Rubin diagnostic plots for the interaction among high deer density, suburban study area, and 2.4-km ring cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

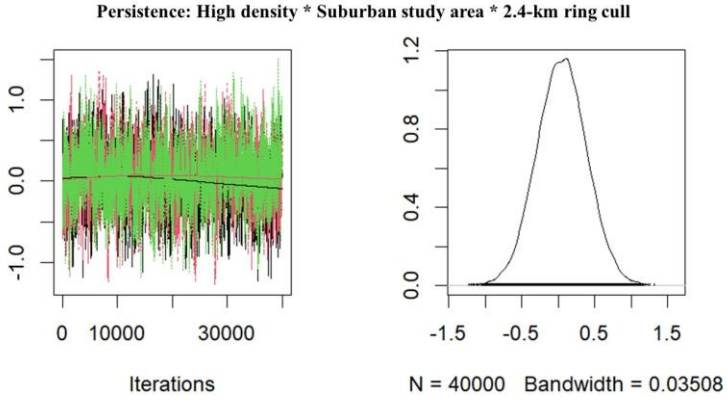


Figure C29. Gelman and Rubin diagnostic plots for the interaction among medium deer density, suburban study area, and high quality habitat cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

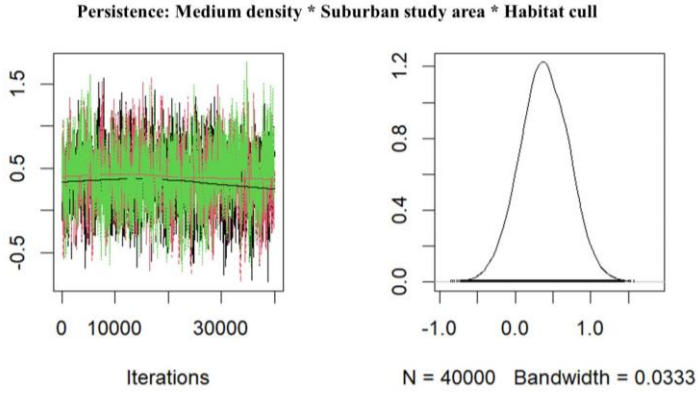


Figure C30. Gelman and Rubin diagnostic plots for the interaction among high deer density, suburban study area, and high quality habitat cull method on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 2.

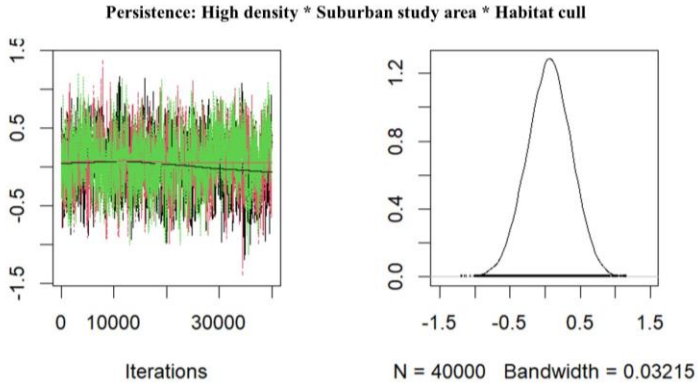


Figure C31. Gelman and Rubin diagnostic plots for the intercept on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

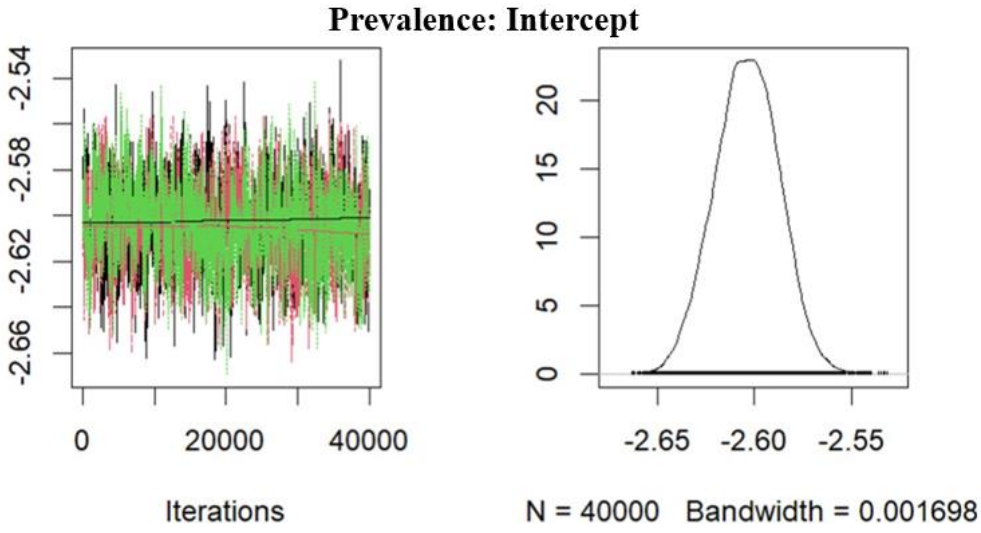


Figure C32. Gelman and Rubin diagnostic plots for the medium deer density parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

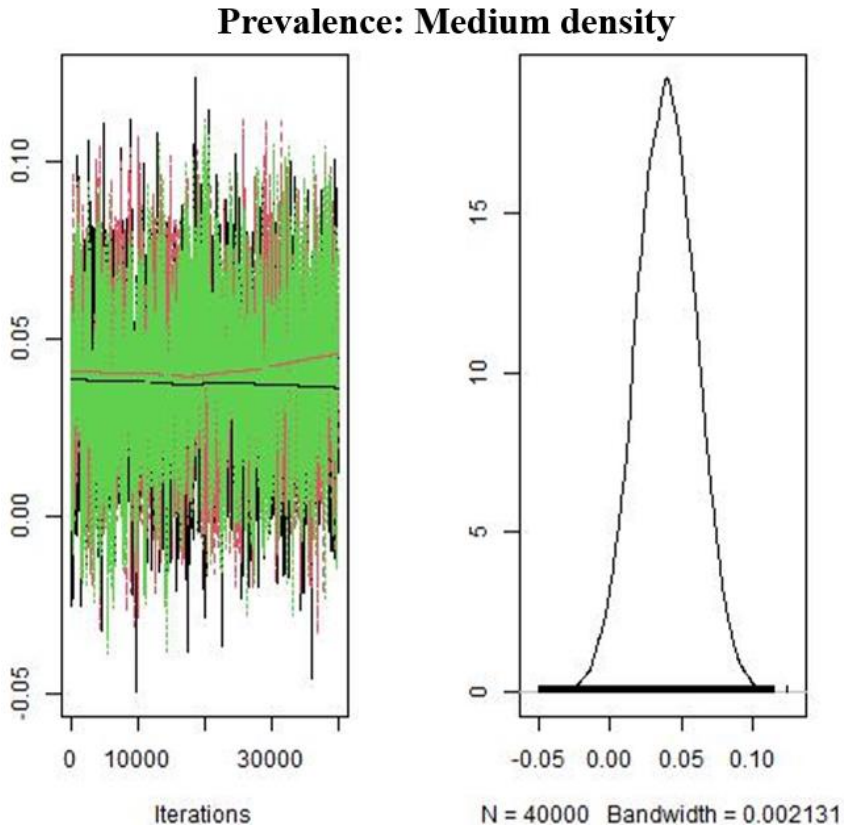


Figure C33. Gelman and Rubin diagnostic plots for the high deer density parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

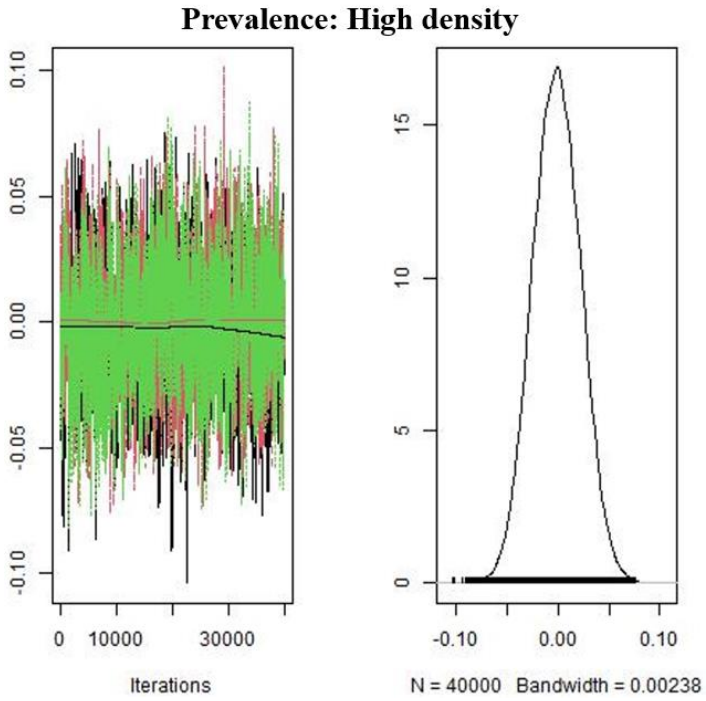


Figure C34. Gelman and Rubin diagnostic plots for the suburban study area parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

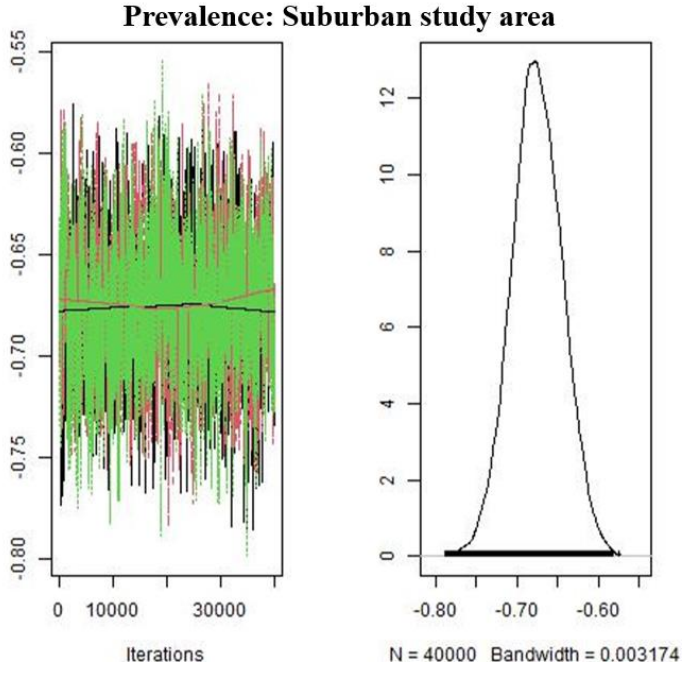


Figure C35. Gelman and Rubin diagnostic plots for parcel cull method parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

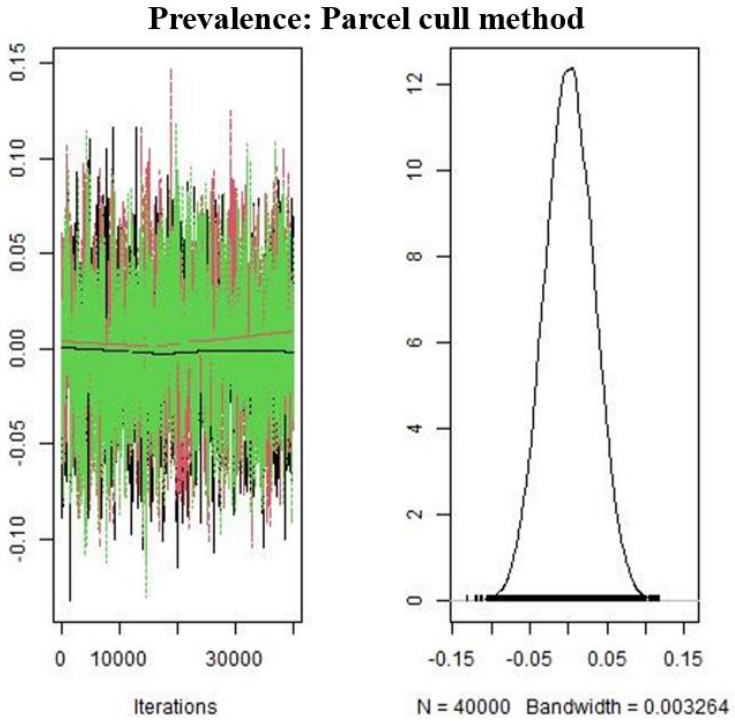


Figure C36. Gelman and Rubin diagnostic plots for the 1.6-km ring cull parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

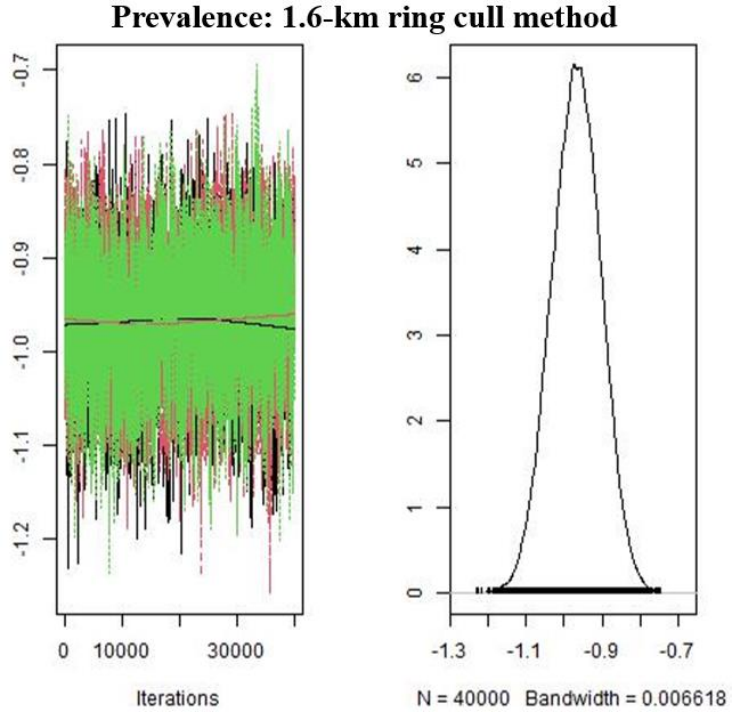


Figure C37. Gelman and Rubin diagnostic plots for the 2.4-km ring cull parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

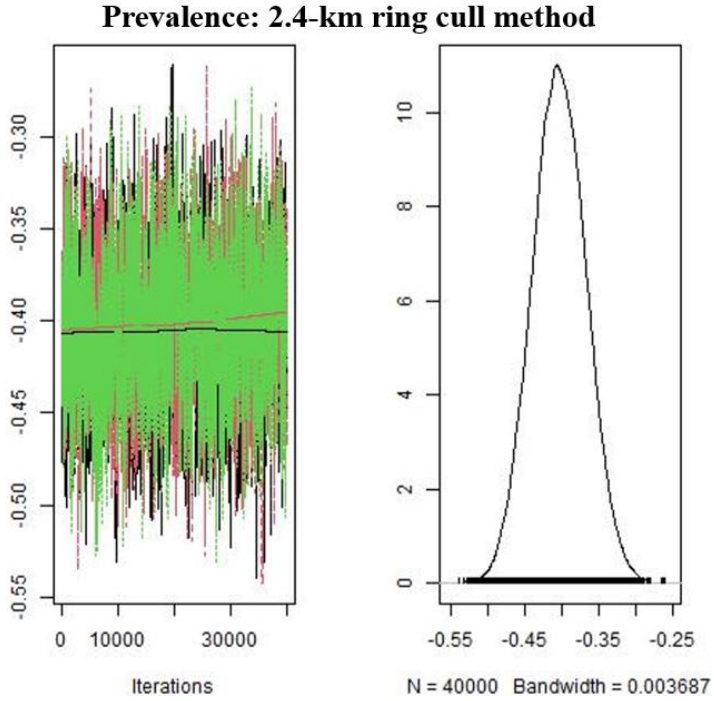


Figure C38. Gelman and Rubin diagnostic plots for the high quality habitat cull parameter on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

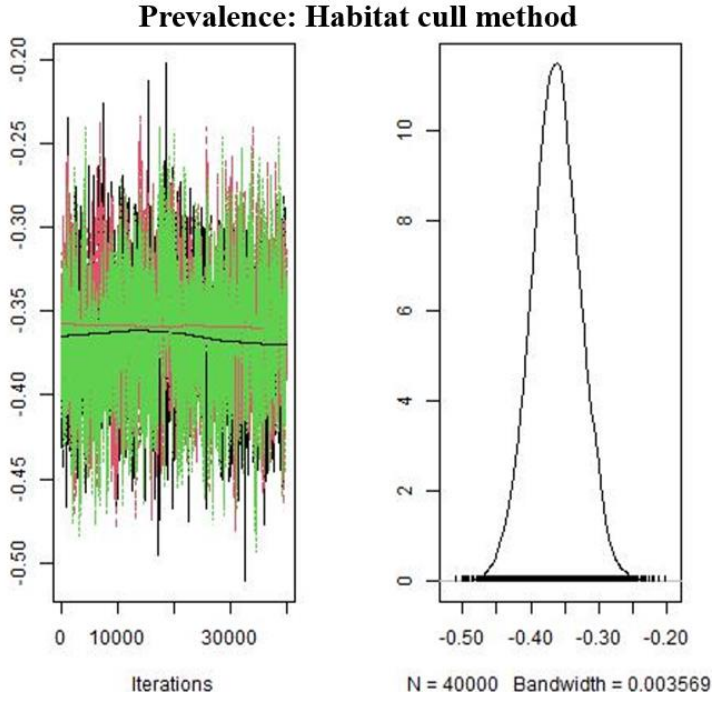


Figure C39. Gelman and Rubin diagnostic plots for the interaction between medium deer density and suburban study area on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

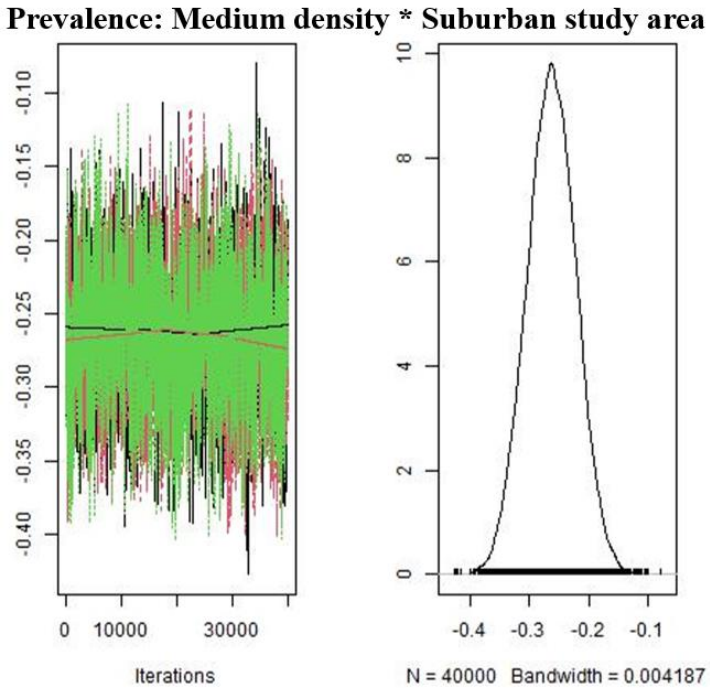


Figure C40. Gelman and Rubin diagnostic plots for the interaction between high deer density and suburban study area on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

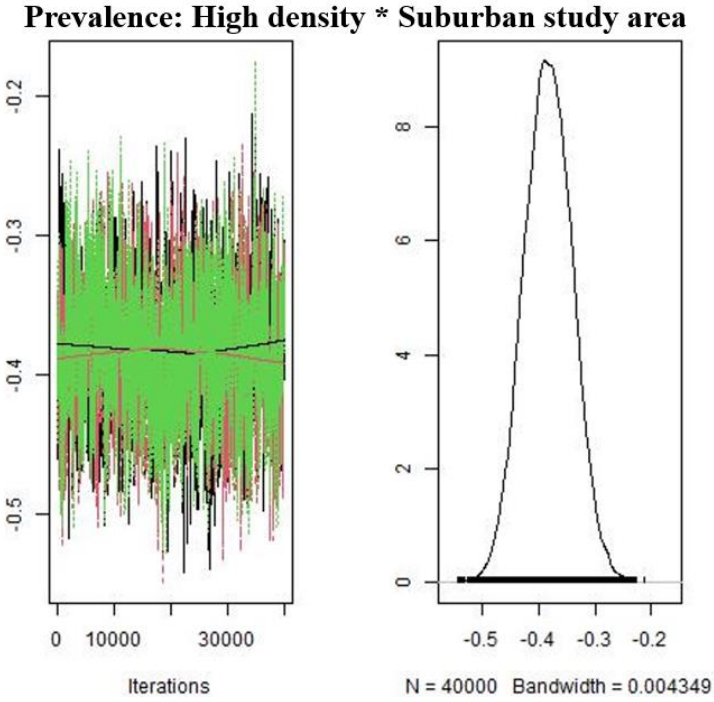


Figure C41. Gelman and Rubin diagnostic plots for the interaction between medium deer density and parcel cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

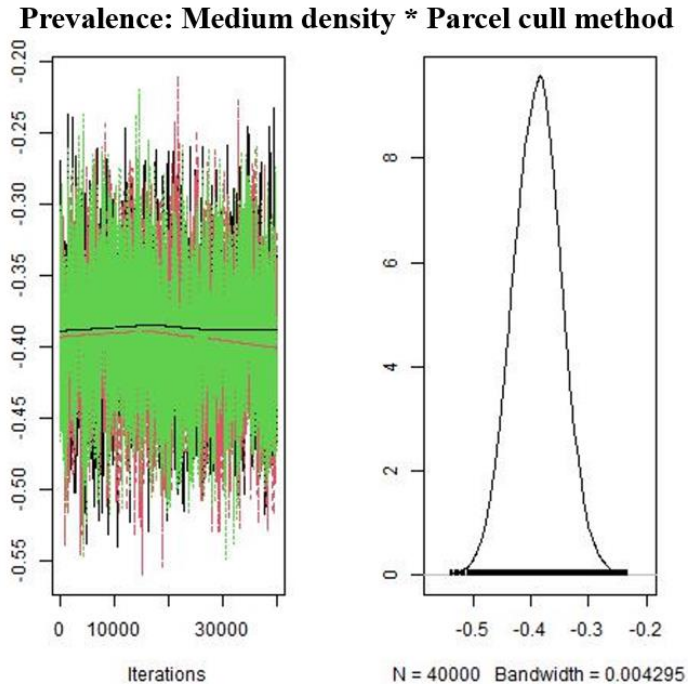


Figure C42. Gelman and Rubin diagnostic plots for the interaction between high deer density and parcel cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

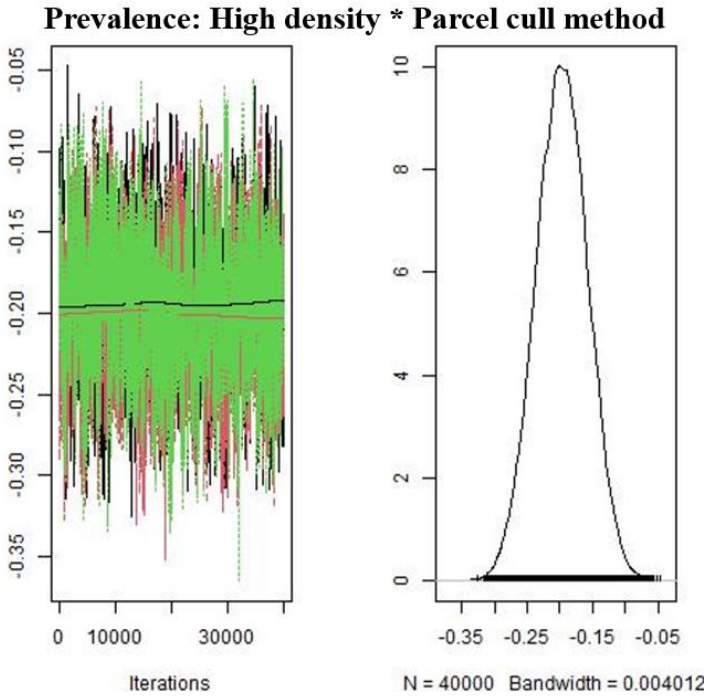


Figure C43. Gelman and Rubin diagnostic plots for the interaction between medium deer density and 1.6-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

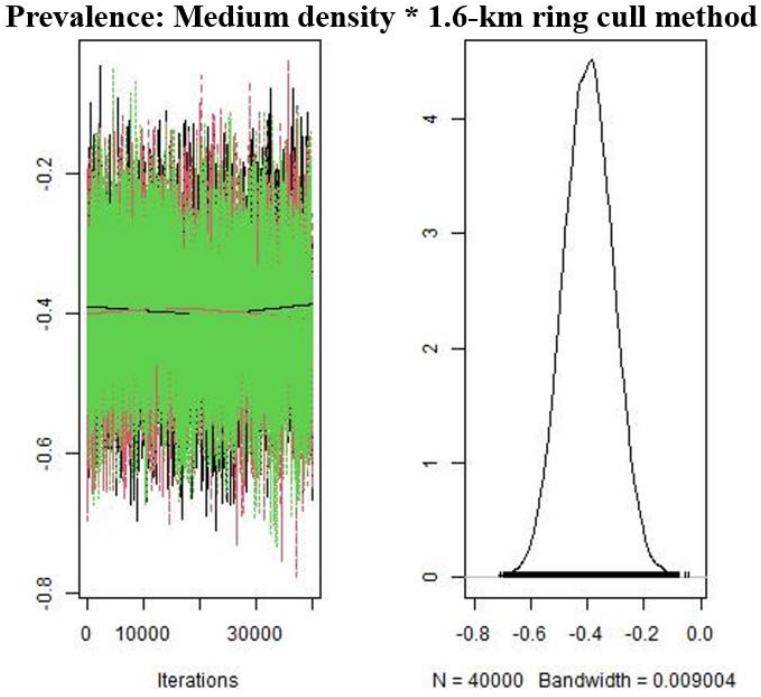


Figure C44. Gelman and Rubin diagnostic plots for the interaction between high deer density and 1.6-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

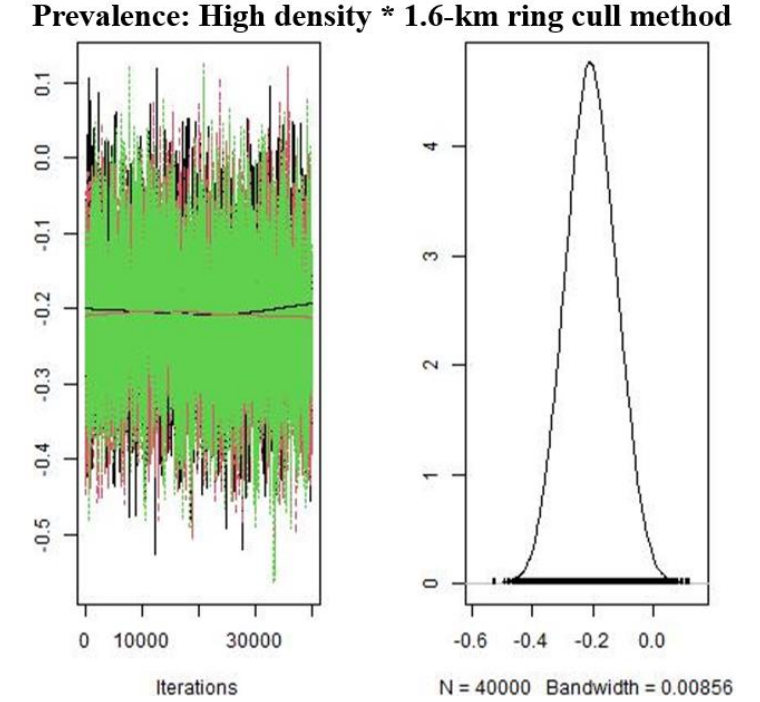


Figure C45. Gelman and Rubin diagnostic plots for the interaction between medium deer density and 2.4-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

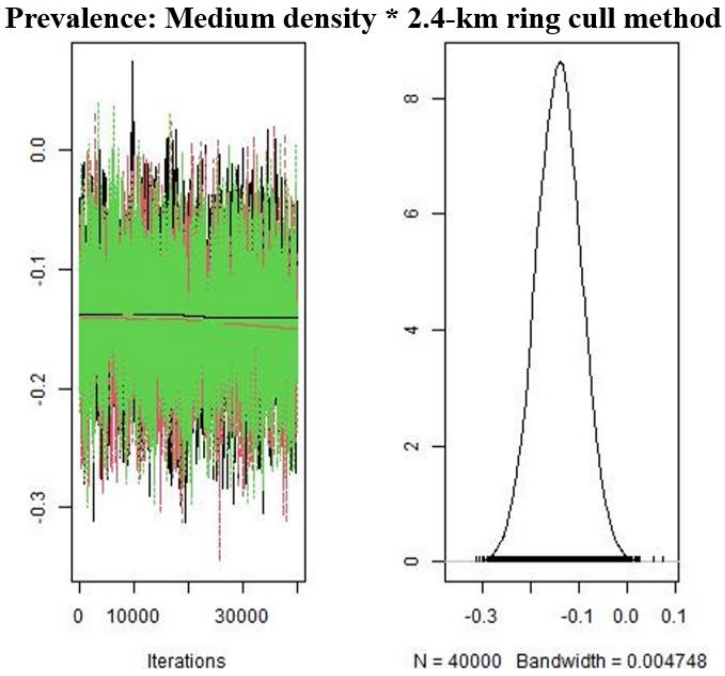


Figure C46. Gelman and Rubin diagnostic plots for the interaction between high deer density and 2.4-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

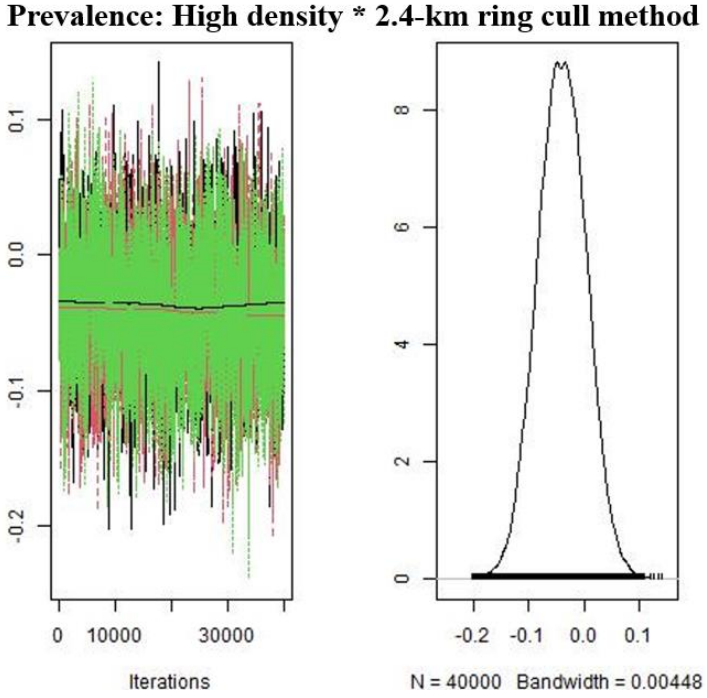


Figure C47. Gelman and Rubin diagnostic plots for the interaction between medium deer density and high quality habitat cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

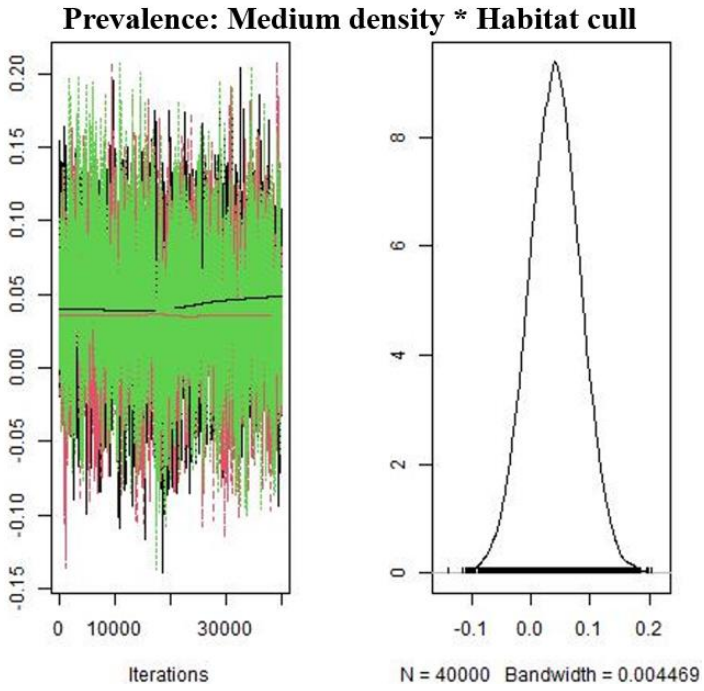


Figure C48. Gelman and Rubin diagnostic plots for the interaction between high deer density and high quality habitat cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

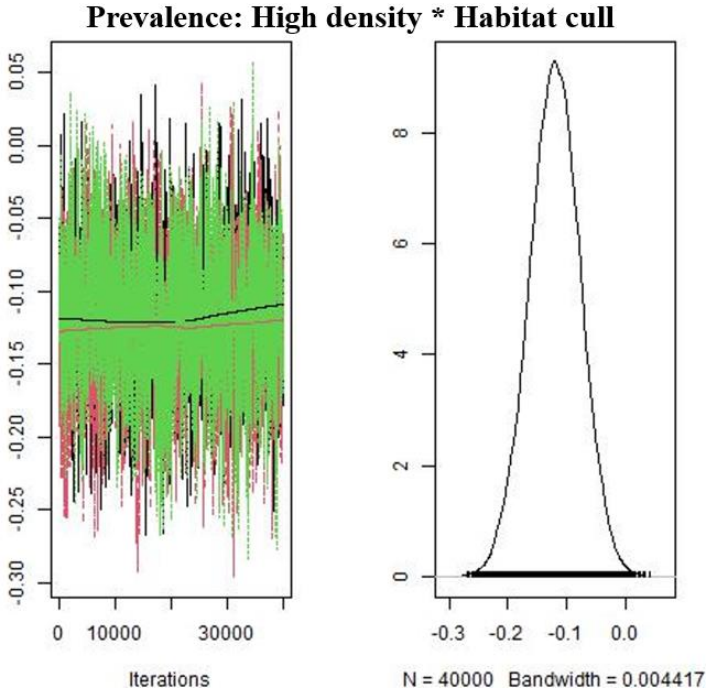


Figure C49. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and parcel cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

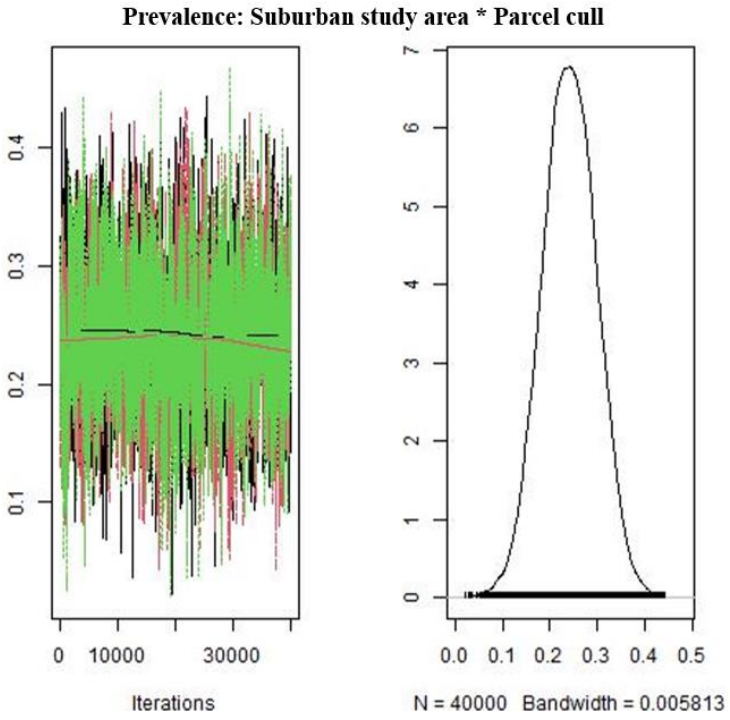


Figure C50. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and 1.6-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

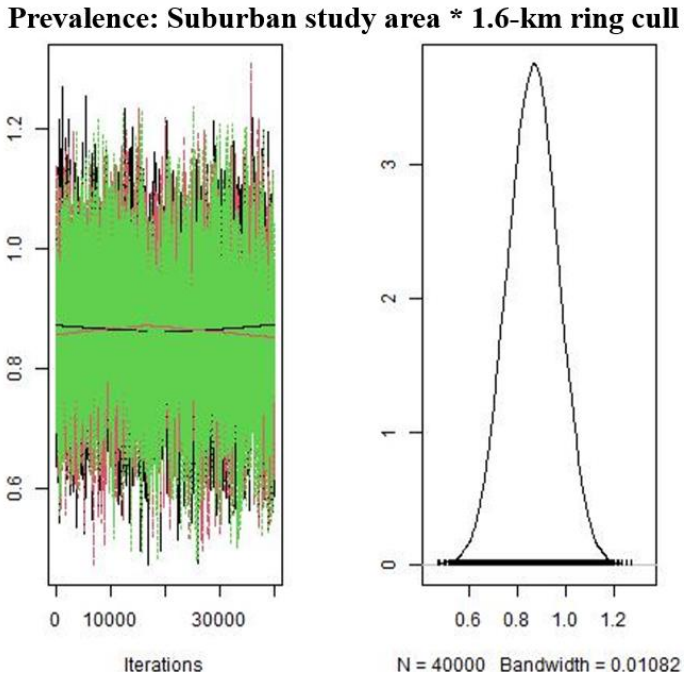


Figure C51. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and 2.4-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

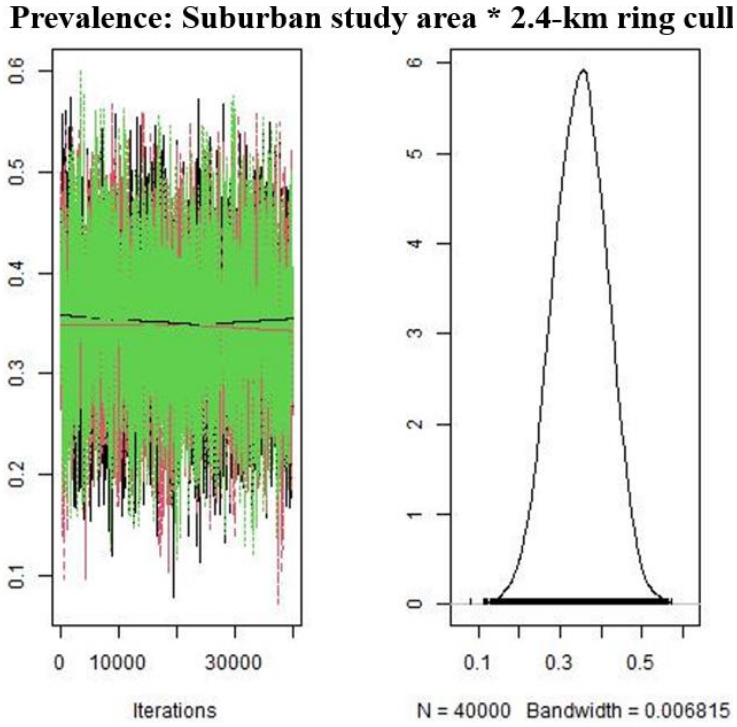


Figure C52. Gelman and Rubin diagnostic plots for the interaction between the suburban study area and high quality habitat cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

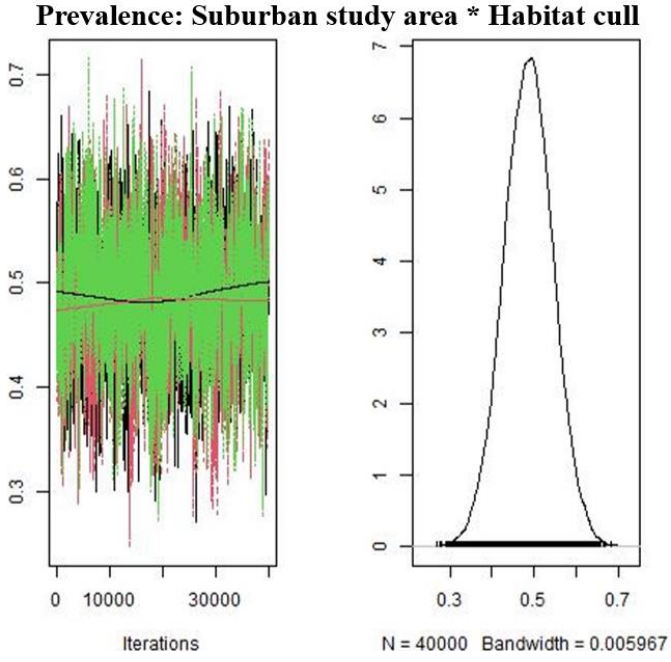


Figure C53. Gelman and Rubin diagnostic plots for the interaction among medium deer density, the suburban study area, and parcel cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

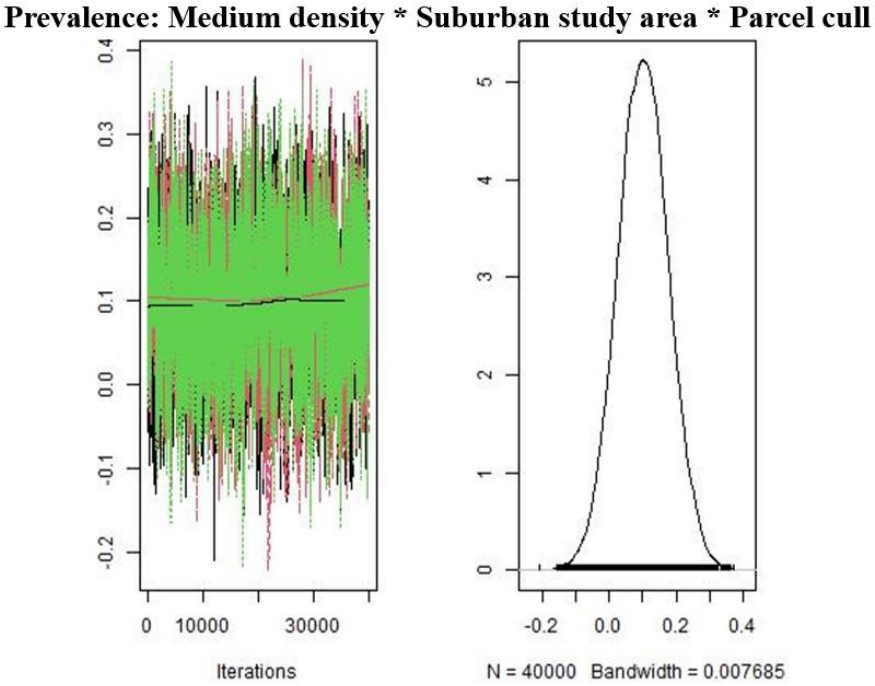


Figure C54. Gelman and Rubin diagnostic plots for the interaction among high deer density, the suburban study area, and parcel cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

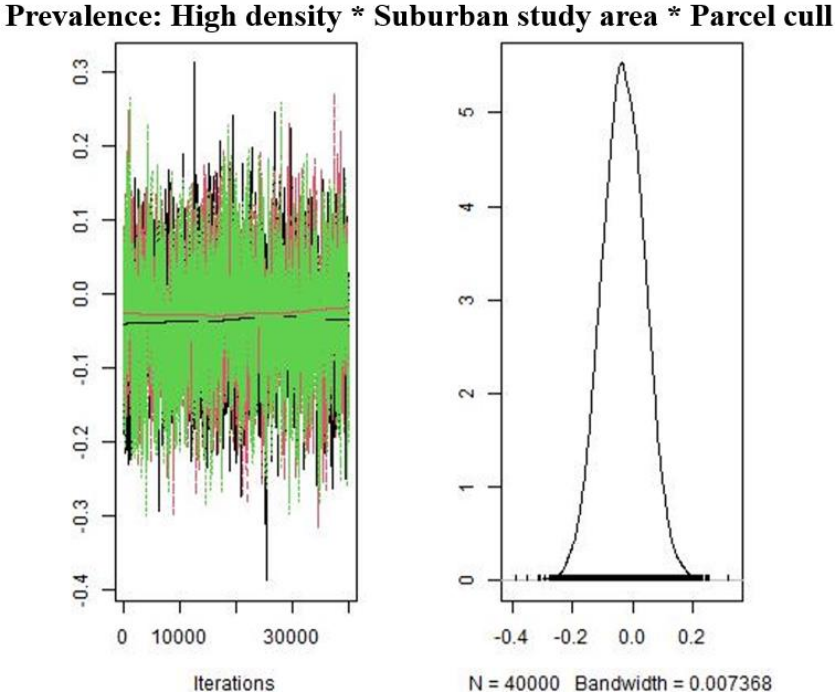


Figure C55. Gelman and Rubin diagnostic plots for the interaction among medium deer density, the suburban study area, and 1.6-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

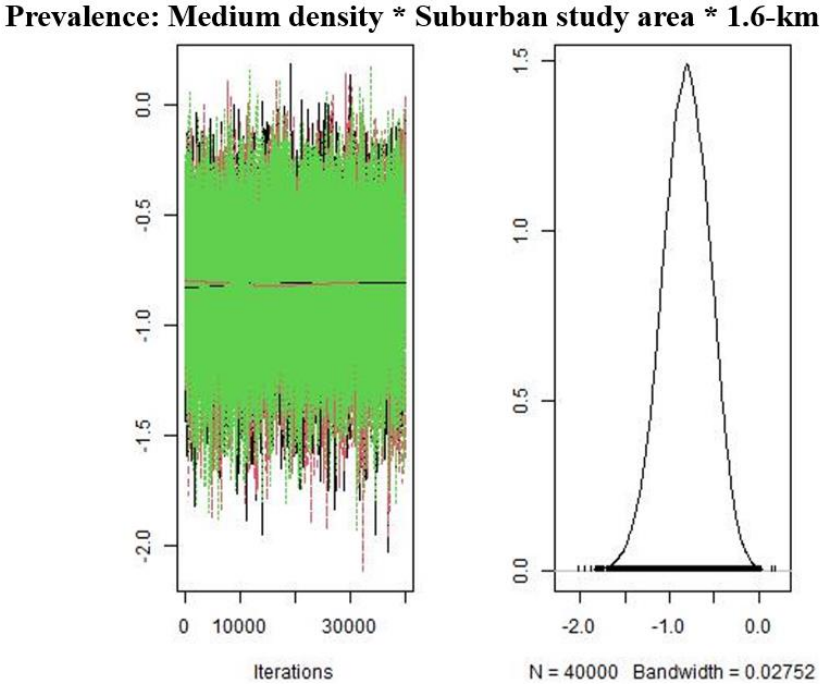


Figure C56. Gelman and Rubin diagnostic plots for the interaction among high deer density, the suburban study area, and 1.6-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

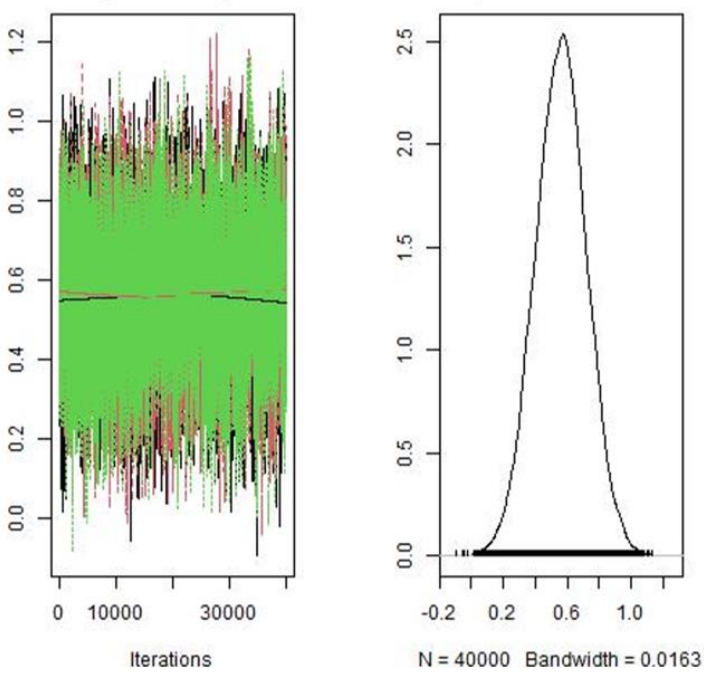


Figure C57. Gelman and Rubin diagnostic plots for the interaction among medium deer density, the suburban study area, and 2.4-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

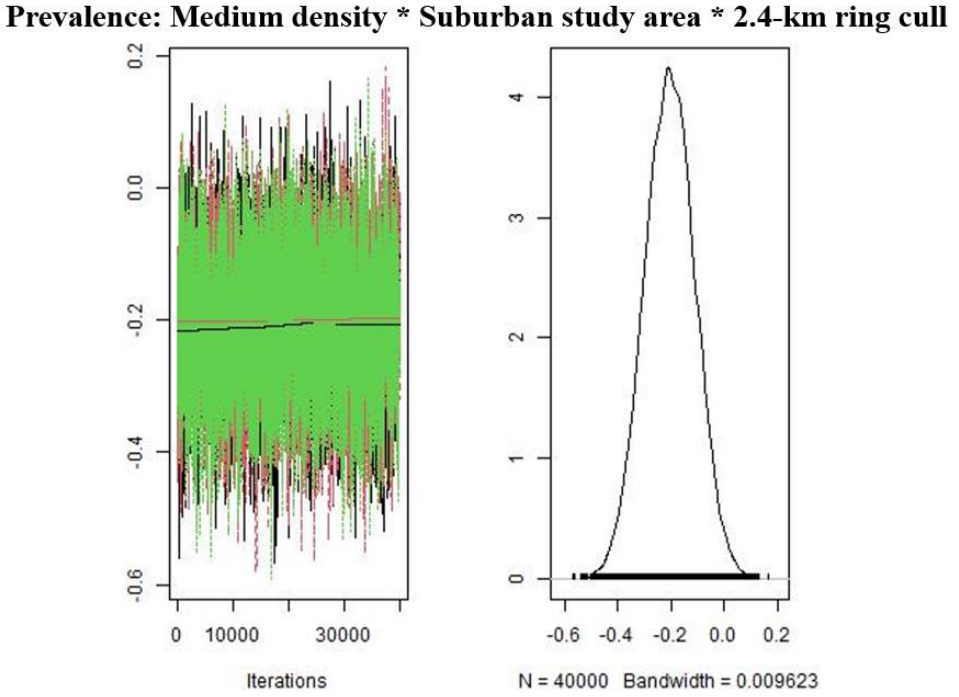


Figure C58. Gelman and Rubin diagnostic plots for the interaction among high deer density, the suburban study area, and 2.4-km ring cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

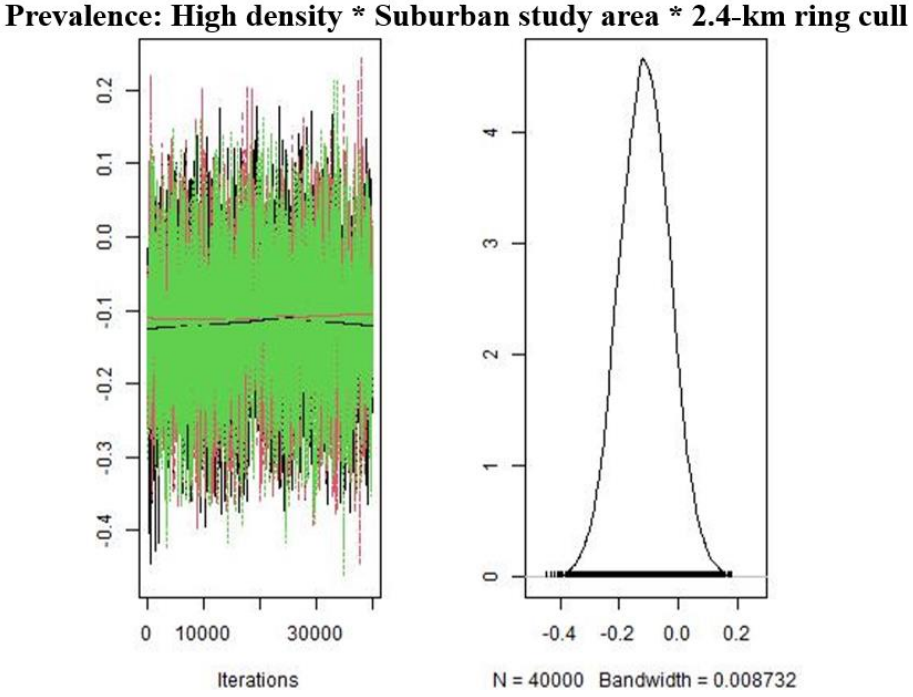


Figure C59. Gelman and Rubin diagnostic plots for the interaction among medium deer density, the suburban study area, and high quality habitat cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.

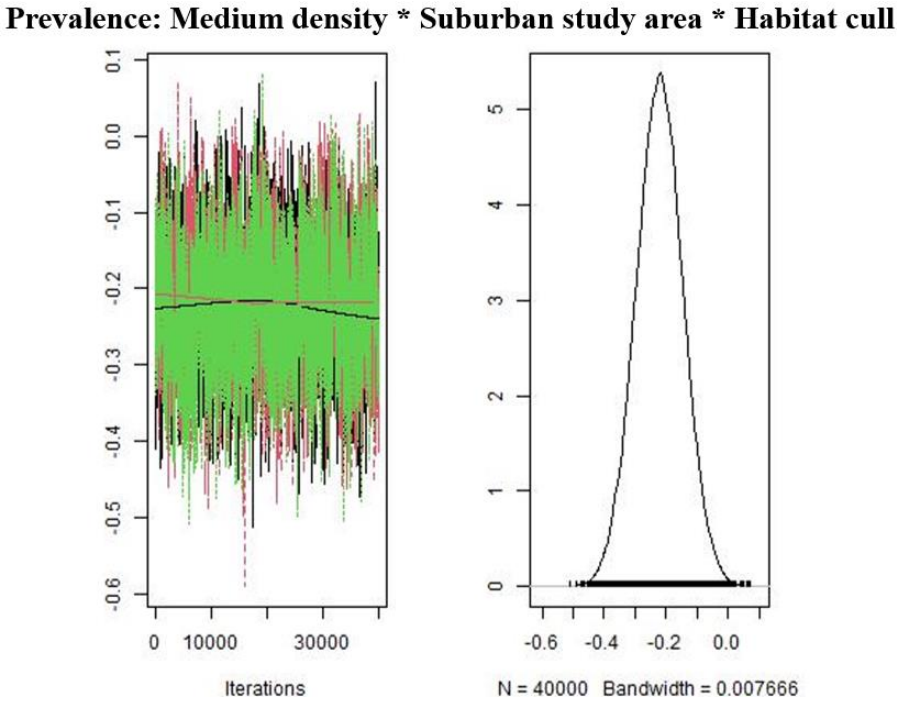
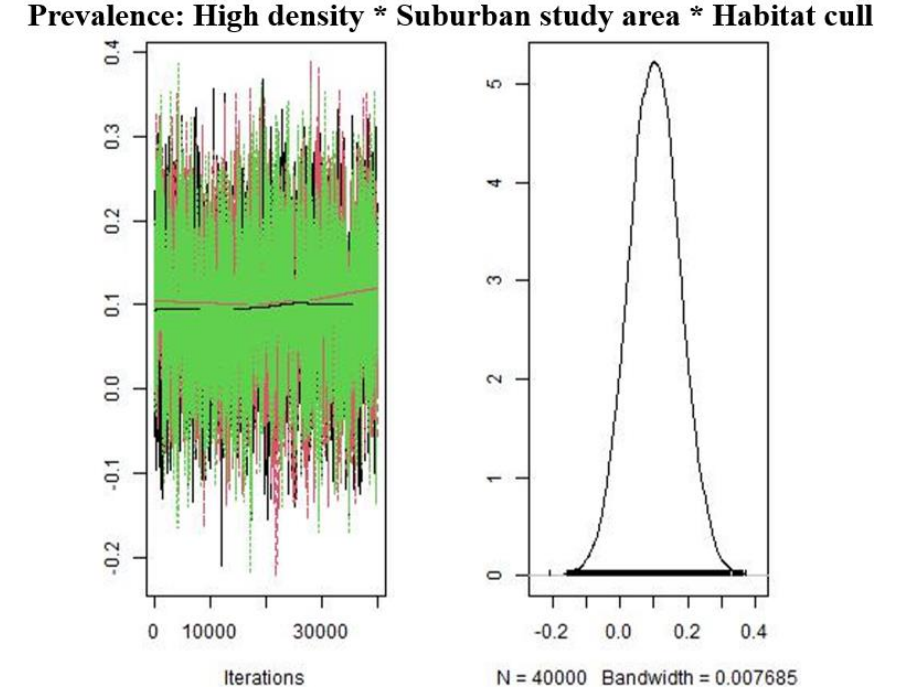


Figure C60. Gelman and Rubin diagnostic plots for the interaction among high deer density, the suburban study area, and high quality habitat cull method on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 2.



APPENDIX D
DIAGNOSTIC OUTPUT FOR MONTE CARLO MARKOV CHAIN CONVERGENCE IN CHAPTER 3

Table D1. Gelman and Rubin diagnostic statistics for each parameter of the Bayesian hurdle model performed on the output generated by the individual-based model in Chapter 3.

	CWD Persistence		CWD Prevalence	
	Mean	Upper CI	Mean	Upper CI
Intercept	1.01	1.04	1.02	1.05
10% Access Rate	1.01	1.04	1.01	1.03
20% Access Rate	1	1.01	1.01	1.03
30% Access Rate	1	1.01	1.01	1.02
40% Access Rate	1.01	1.02	1.01	1.03
50% Access Rate	1.01	1.03	1.01	1.03
70% Access Rate	1.01	1.03	1.01	1.03
10% Removal Rate	1.01	1.02	1.01	1.04
20% Removal Rate	1.01	1.04	1.01	1.02
30% Removal Rate	1.01	1.03	1.01	1.03
40% Removal Rate	1.01	1.03	1.01	1.04
50% Removal Rate	1.01	1.02	1.01	1.03
70% Removal Rate	1	1.02	1.01	1.02
10% Access Rate*10% Removal Rate	1.01	1.02	1.01	1.02
20% Access Rate*10% Removal Rate	1	1.01	1.01	1.02
30% Access Rate*10% Removal Rate	1	1.01	1.01	1.02
40% Access Rate*10% Removal Rate	1	1.01	1	1.02
50% Access Rate*10% Removal Rate	1	1.01	1.01	1.03
70% Access Rate*10% Removal Rate	1.01	1.02	1.01	1.03
10% Access Rate*20% Removal Rate	1.01	1.04	1	1.01
20% Access Rate*20% Removal Rate	1	1.02	1	1.01
30% Access Rate*20% Removal Rate	1	1.01	1	1.01
40% Access Rate*20% Removal Rate	1.01	1.03	1	1.01
50% Access Rate*20% Removal Rate	1.01	1.03	1	1.01
70% Access Rate*20% Removal Rate	1.01	1.03	1	1.01
10% Access Rate*30% Removal Rate	1.01	1.03	1.01	1.02
20% Access Rate*30% Removal Rate	1	1.01	1	1.01
30% Access Rate*30% Removal Rate	1	1.01	1	1.01
40% Access Rate*30% Removal Rate	1	1.02	1	1.01
50% Access Rate*30% Removal Rate	1.01	1.02	1	1.01
70% Access Rate*30% Removal Rate	1.01	1.02	1	1.01
10% Access Rate*40% Removal Rate	1.01	1.03	1.01	1.03

Table D1 (cont'd).

	CWD Persistence		CWD Prevalence	
	Mean	Upper CI	Mean	Upper CI
20% Access Rate*40% Removal Rate	1	1.01	1.01	1.02
30% Access Rate*40% Removal Rate	1	1.01	1	1.01
40% Access Rate*40% Removal Rate	1.01	1.02	1	1.02
50% Access Rate*40% Removal Rate	1.01	1.02	1.01	1.02
70% Access Rate*40% Removal Rate	1.01	1.02	1	1.01
10% Access Rate*50% Removal Rate	1.01	1.02	1.01	1.02
20% Access Rate*50% Removal Rate	1	1.01	1.01	1.02
30% Access Rate*50% Removal Rate	1	1.01	1	1.01
40% Access Rate*50% Removal Rate	1	1.01	1	1.01
50% Access Rate*50% Removal Rate	1	1.02	1	1.02
70% Access Rate*50% Removal Rate	1	1.01	1	1.01
10% Access Rate*70% Removal Rate	1.01	1.02	1	1.01
20% Access Rate*70% Removal Rate	1	1.01	1	1.01
30% Access Rate*70% Removal Rate	1	1	1	1.01
40% Access Rate*70% Removal Rate	1	1.01	1	1.01
50% Access Rate*70% Removal Rate	1	1.01	1	1
70% Access Rate*70% Removal Rate	1	1.01	1	1

Figure D1. Gelman and Rubin diagnostic plots for the intercept on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

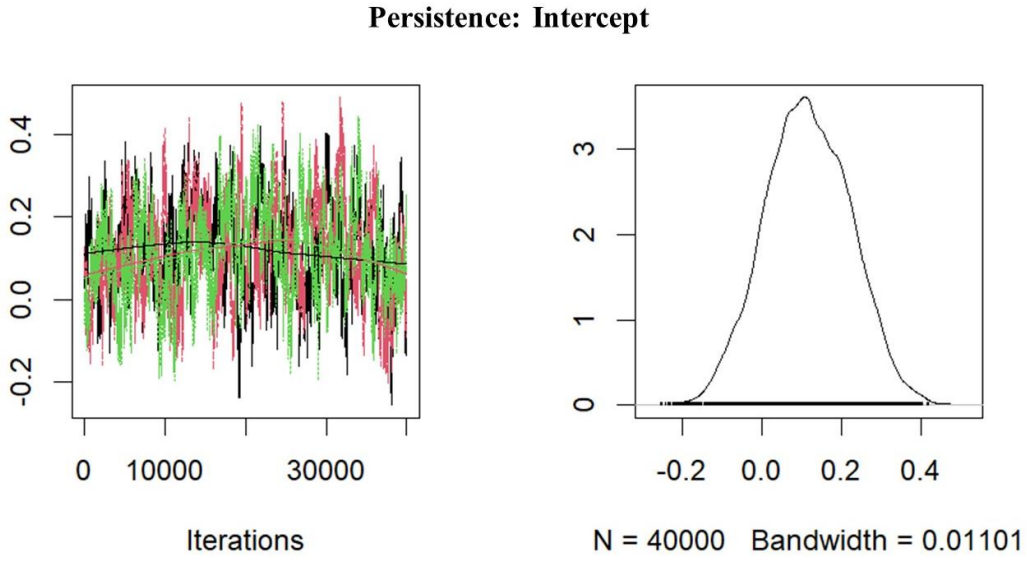


Figure D2. Gelman and Rubin diagnostic plots for the 10% land access rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

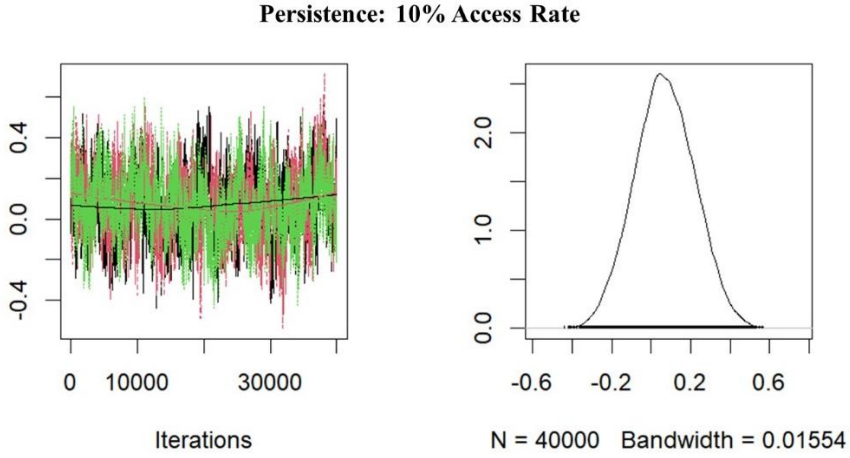


Figure D3. Gelman and Rubin diagnostic plots for the 20% land access rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

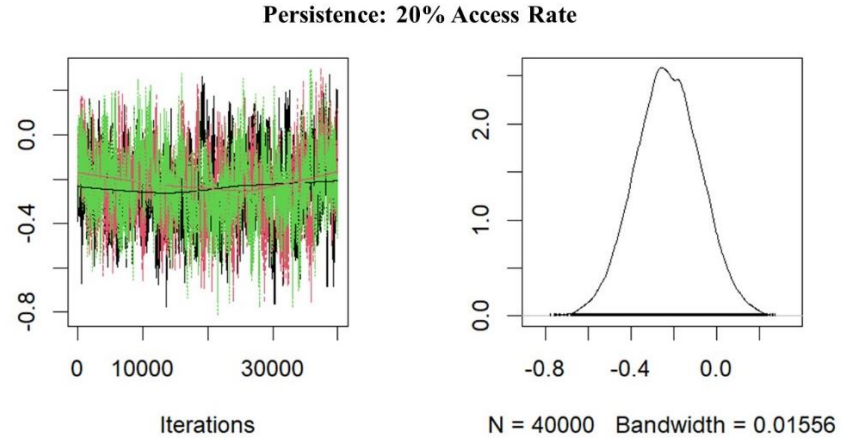


Figure D4. Gelman and Rubin diagnostic plots for the 30% land access rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

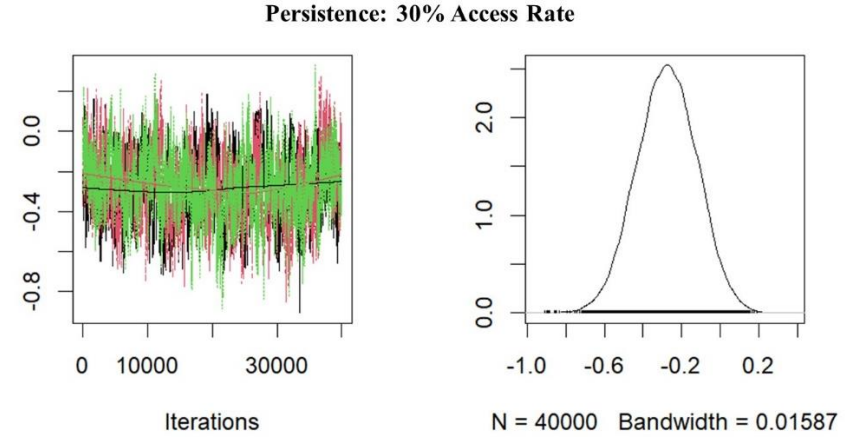


Figure D5. Gelman and Rubin diagnostic plots for the 40% land access rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

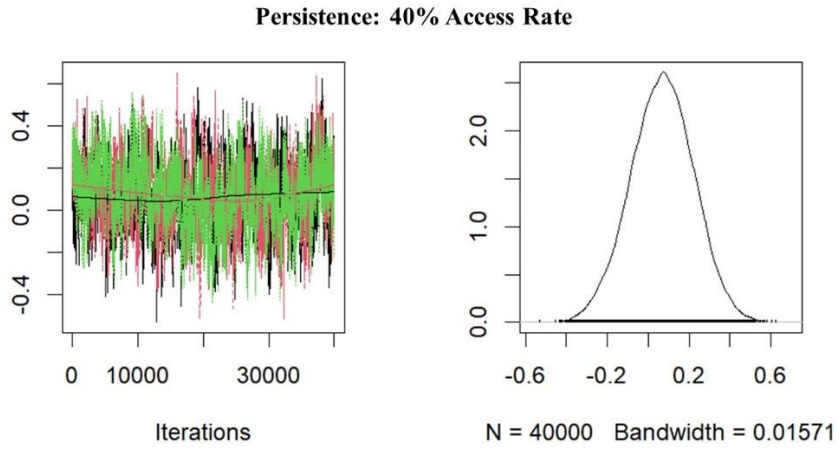


Figure D6. Gelman and Rubin diagnostic plots for the 50% land access rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

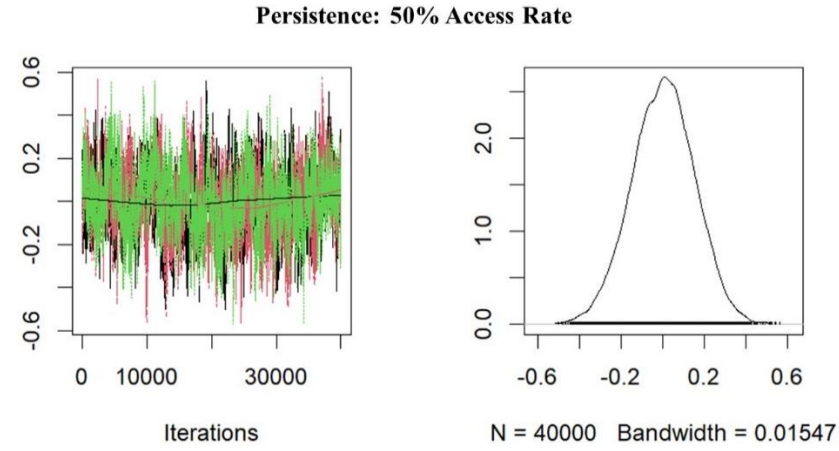


Figure D7. Gelman and Rubin diagnostic plots for the 70% land access rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

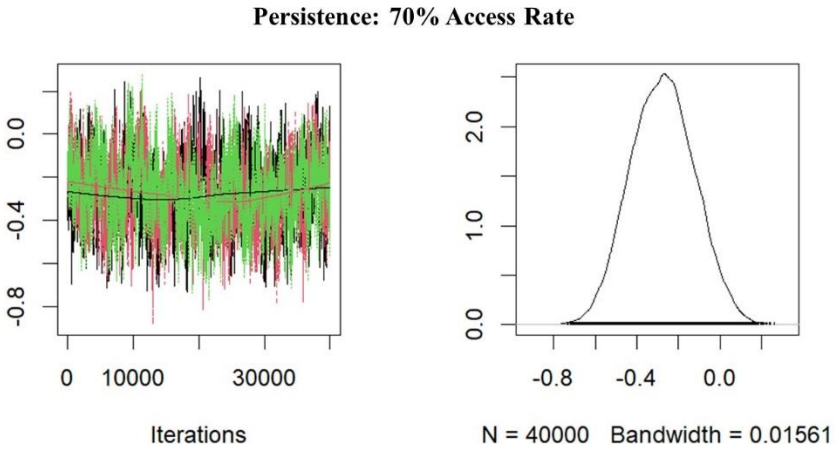


Figure D8. Gelman and Rubin diagnostic plots for the 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

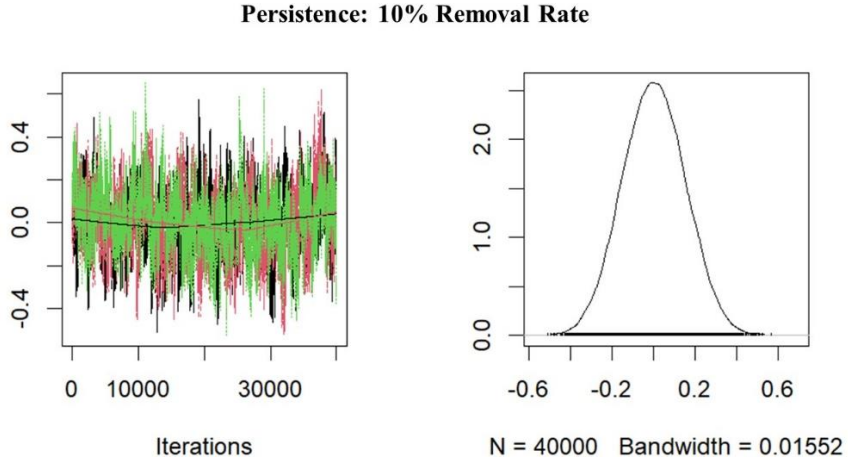


Figure D9. Gelman and Rubin diagnostic plots for the 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

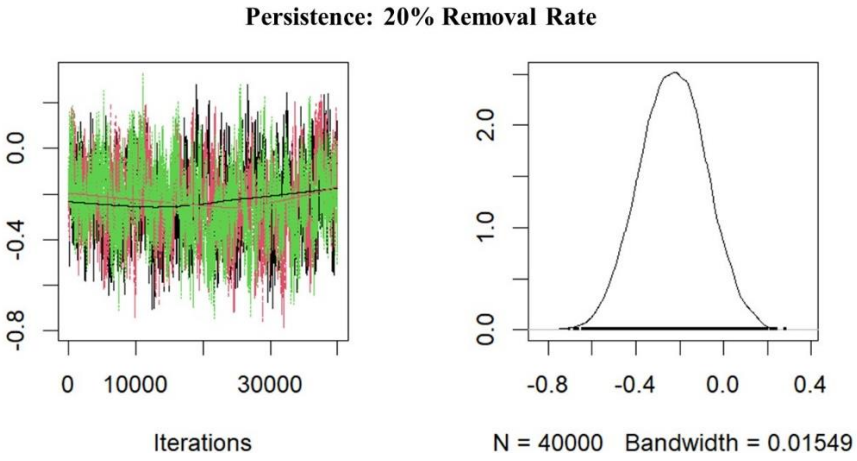


Figure D10. Gelman and Rubin diagnostic plots for the 30% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

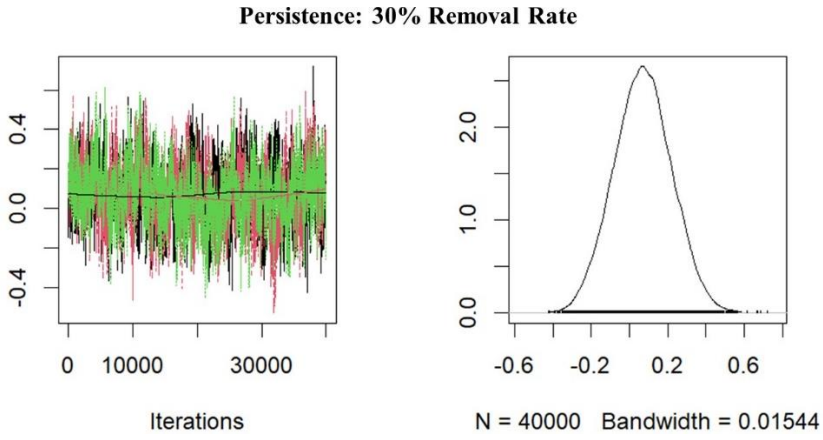


Figure D11. Gelman and Rubin diagnostic plots for the 40% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

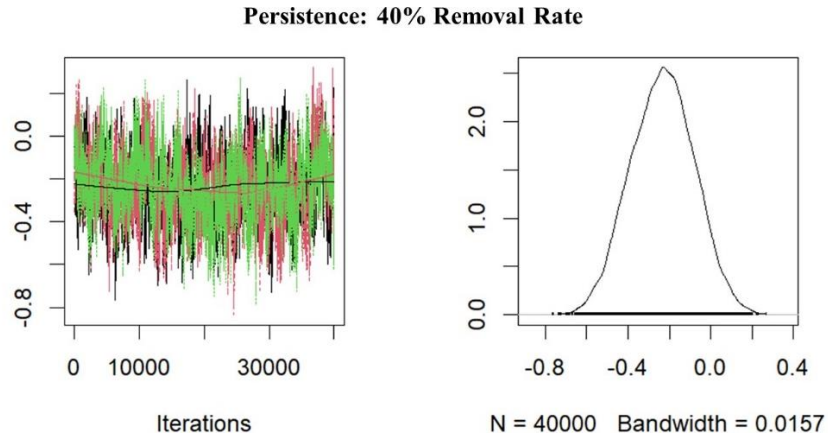


Figure D12. Gelman and Rubin diagnostic plots for the 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

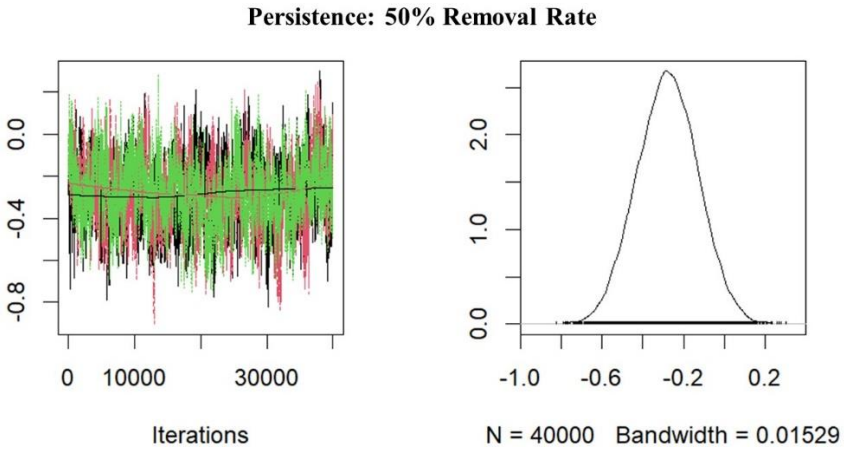


Figure D13. Gelman and Rubin diagnostic plots for the 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

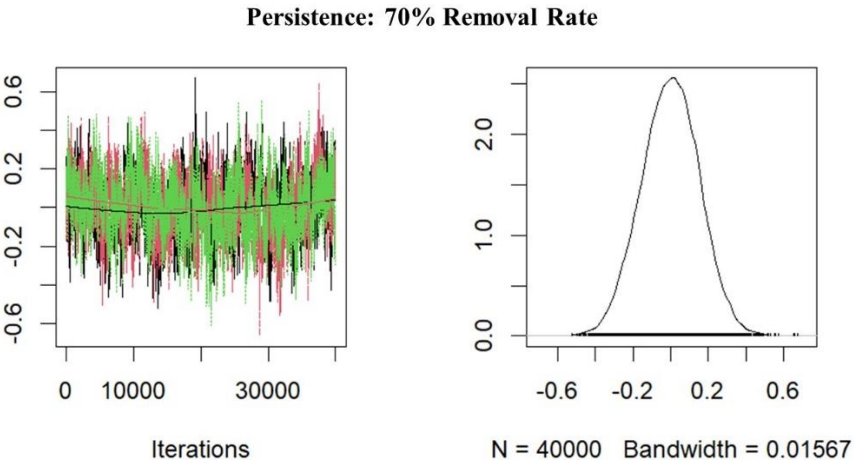


Figure D14. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

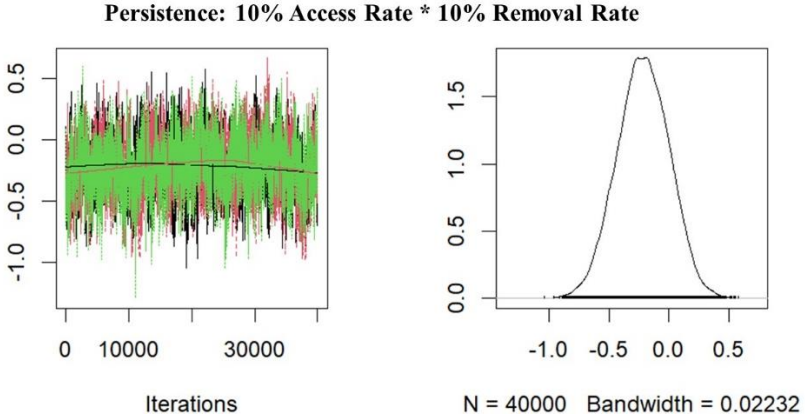


Figure D15. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

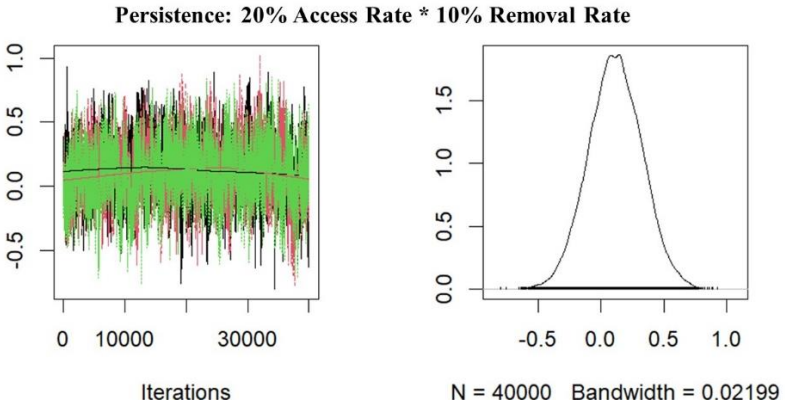


Figure D16. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

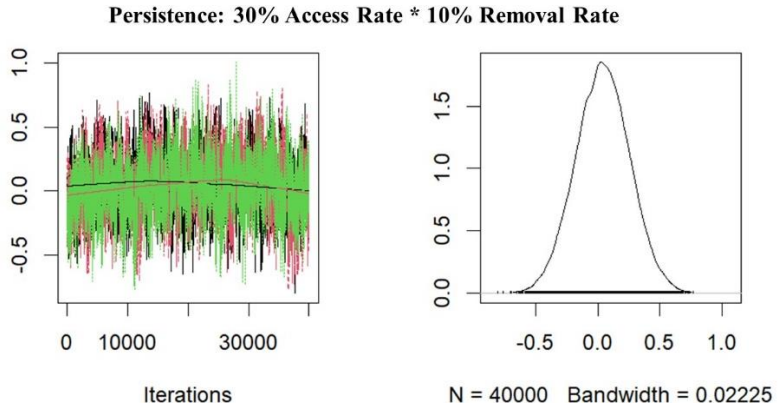


Figure D17. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

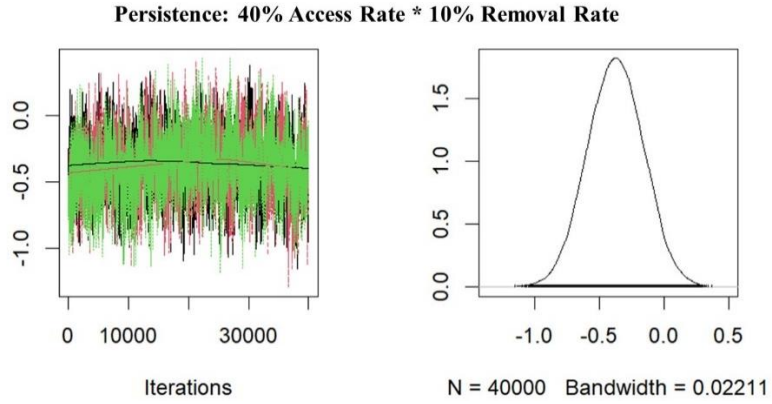


Figure D18. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

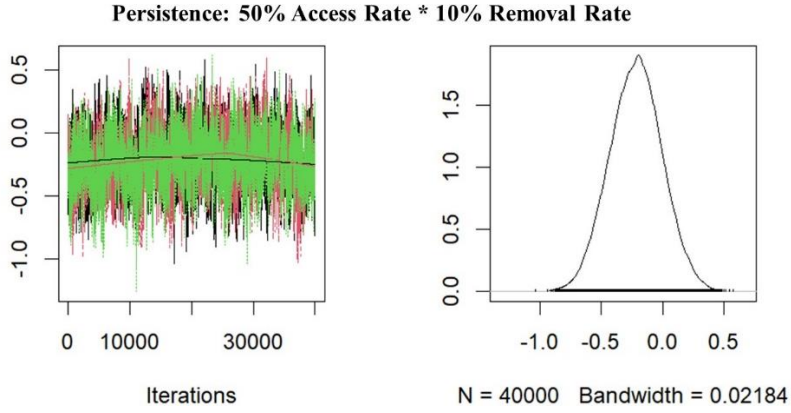


Figure D19. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

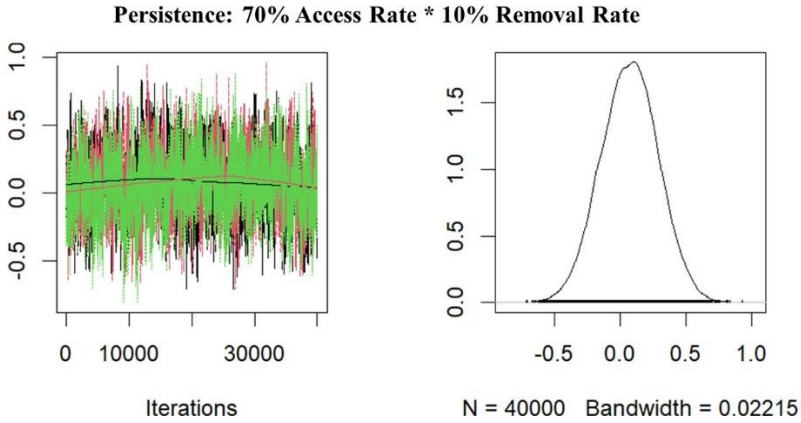


Figure D20. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

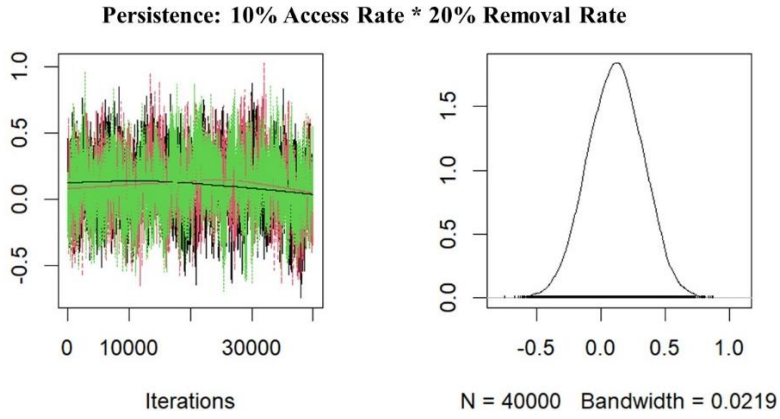


Figure D21. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

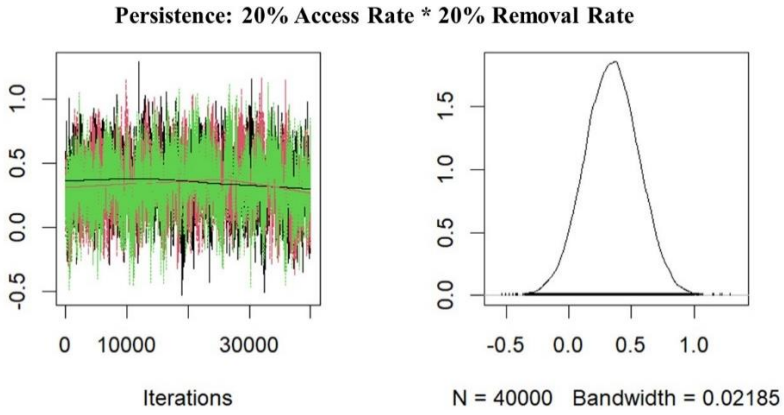


Figure D22 Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

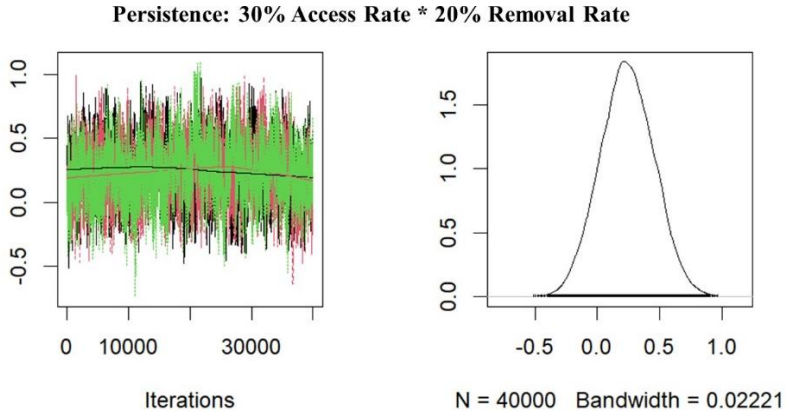


Figure D23. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

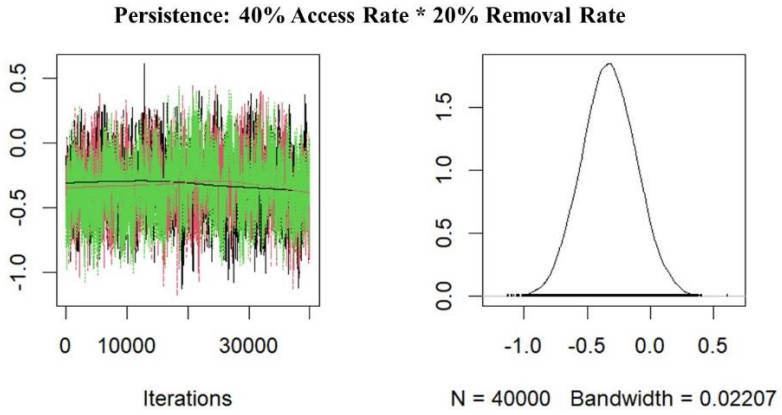


Figure D24. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

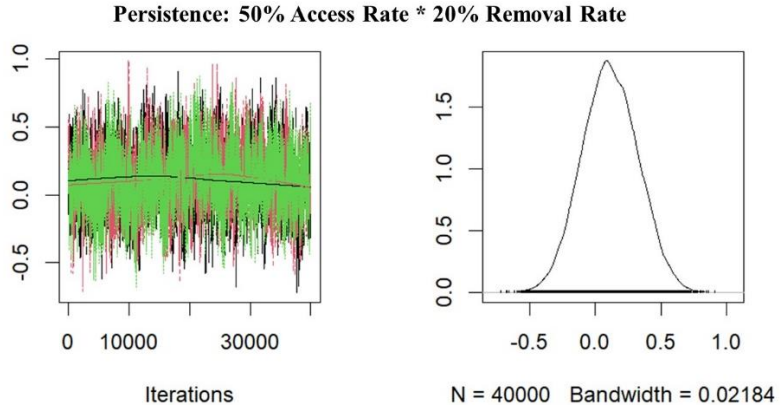


Figure D25. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 20% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

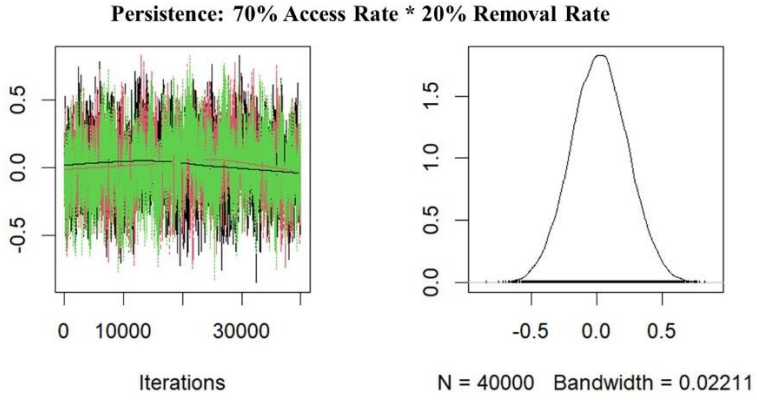


Figure D26. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 30% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

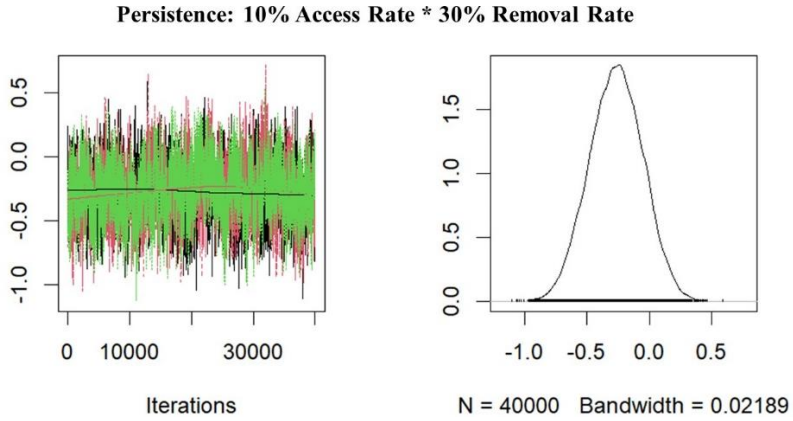


Figure D27. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

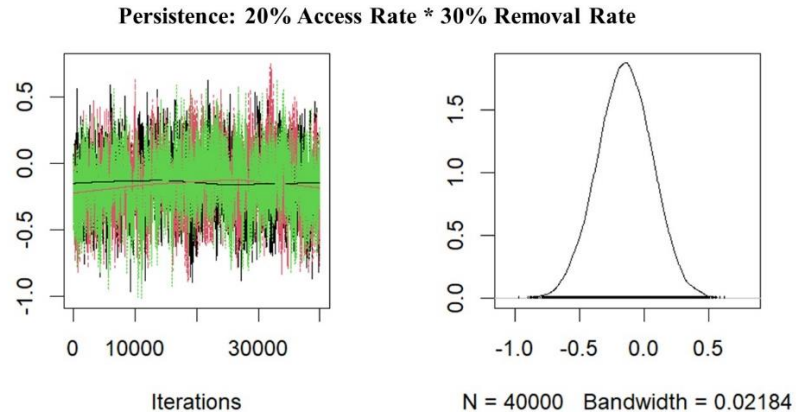


Figure D28. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

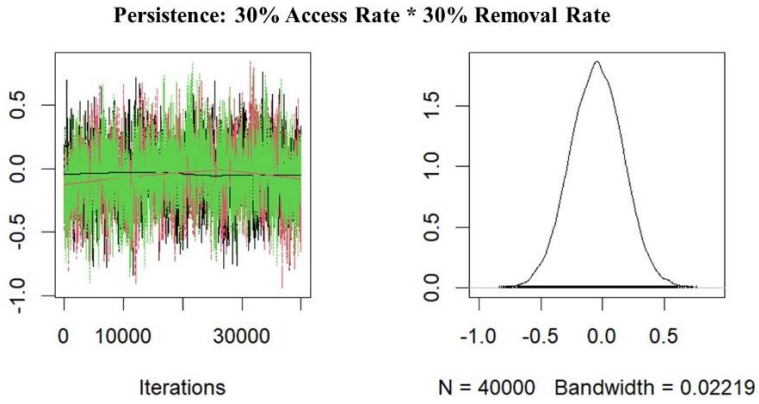


Figure D29. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 30% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

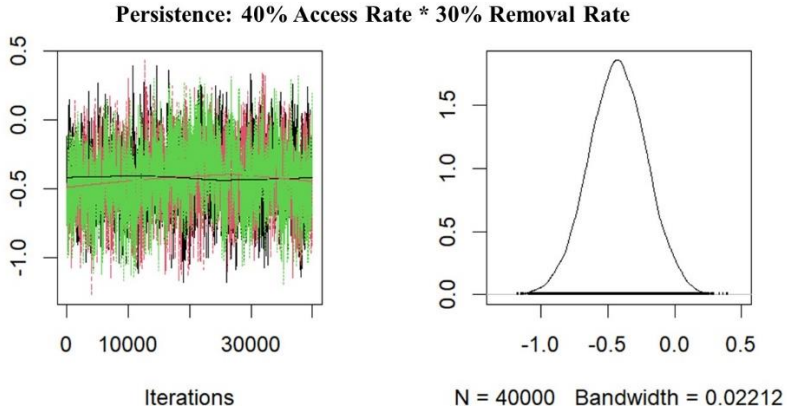


Figure D30. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 30% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

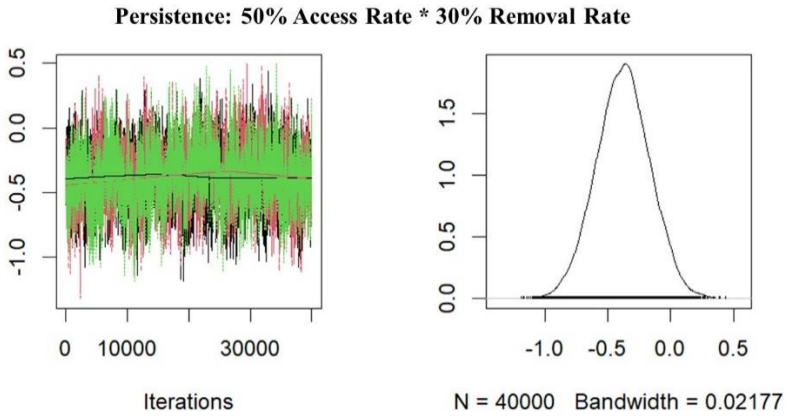


Figure D31. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 30% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

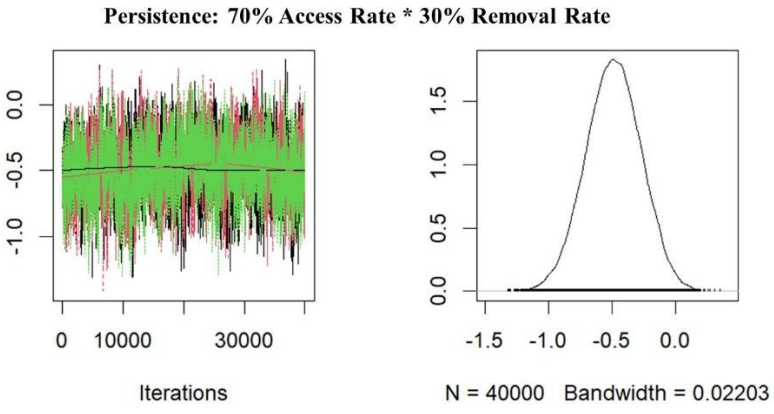


Figure D32. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 40% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

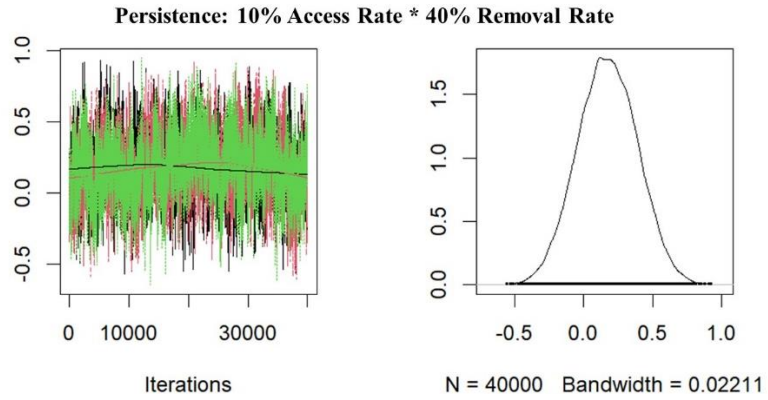


Figure D33. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 40% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

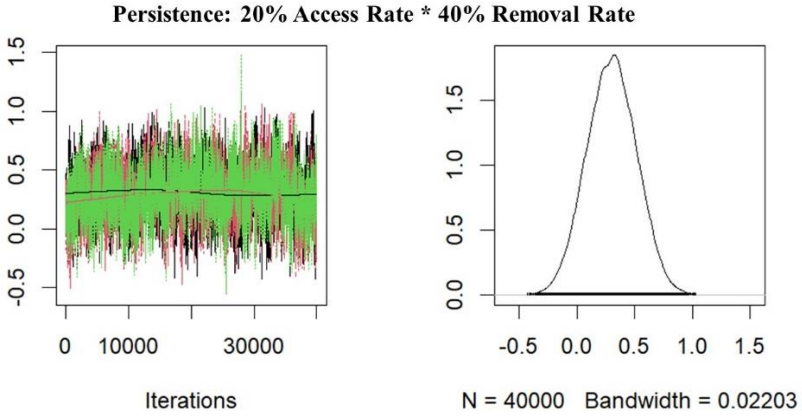


Figure D34. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 40% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

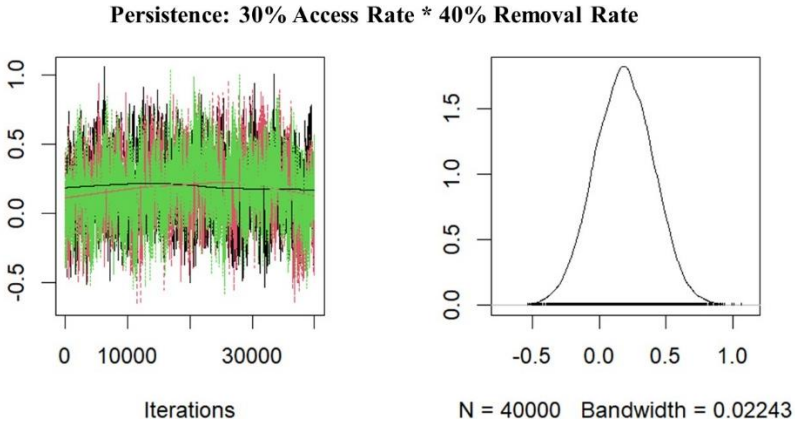


Figure D35. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

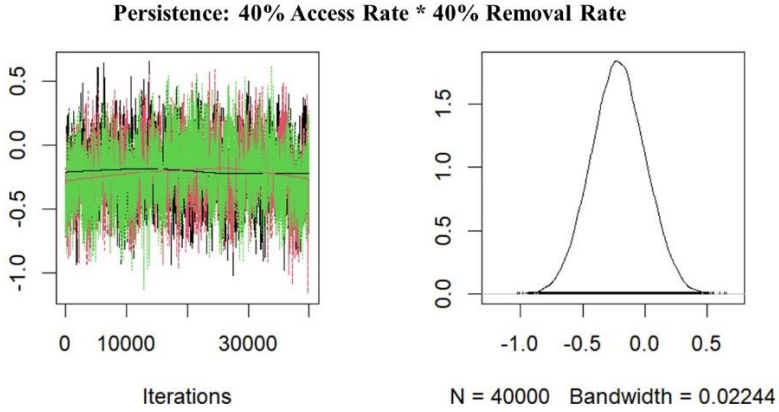


Figure D36. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 10% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

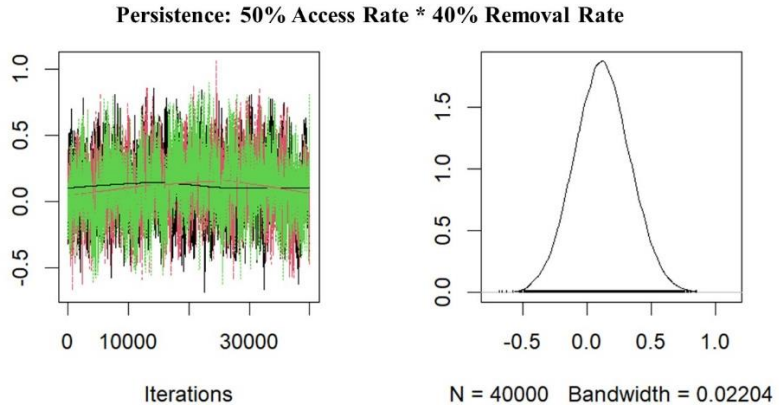


Figure D37. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 40% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

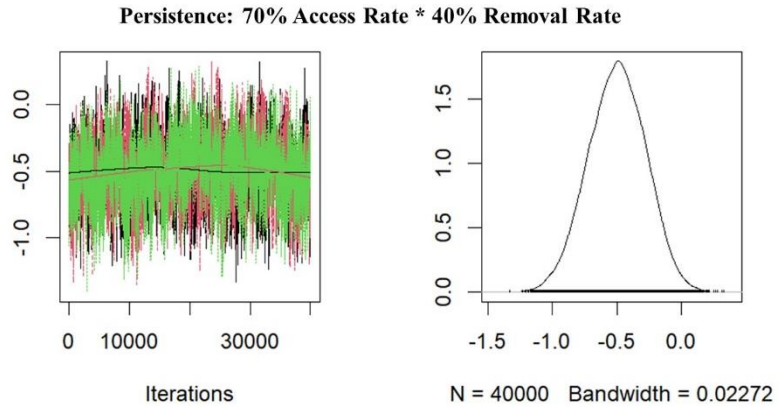


Figure D38. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

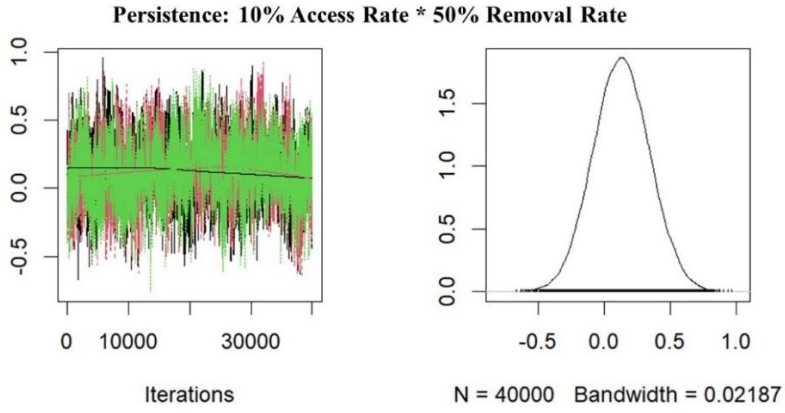


Figure D39. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

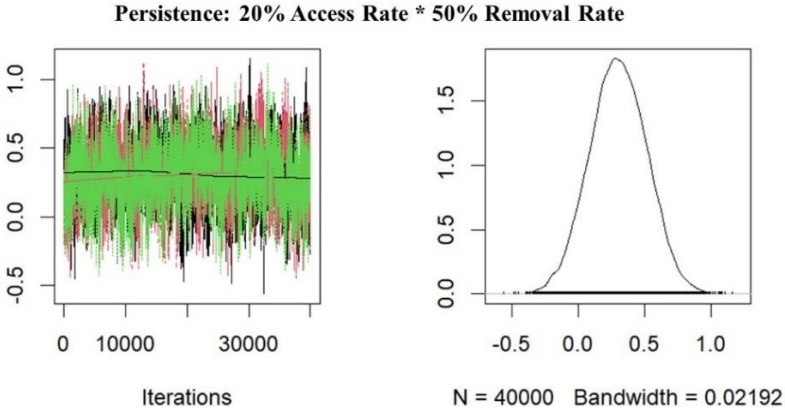


Figure D40. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

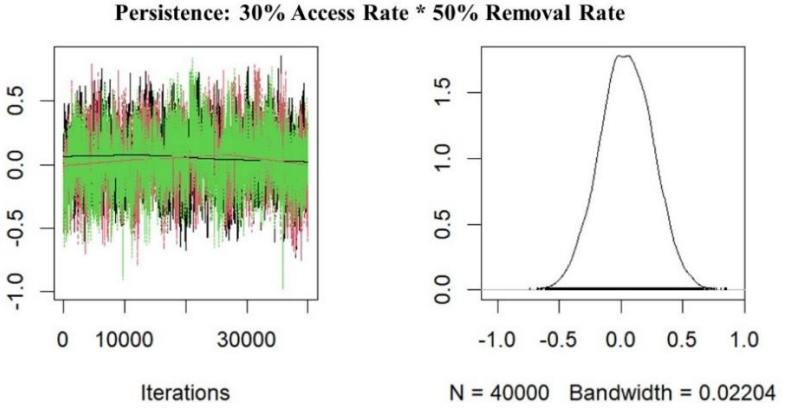


Figure D41. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

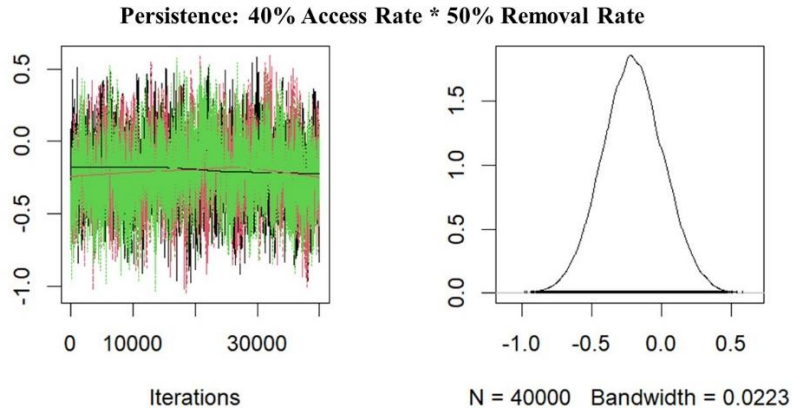


Figure D42. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

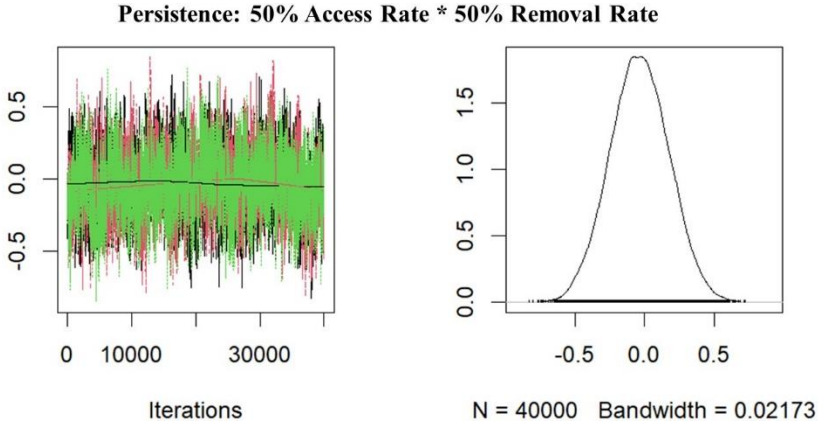


Figure D43. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 50% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

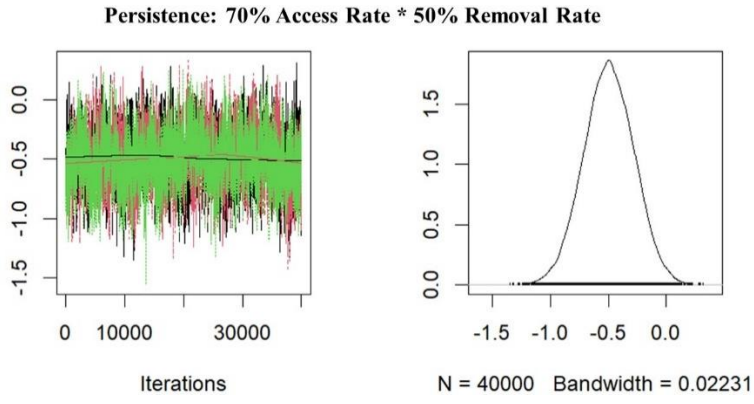


Figure D44. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

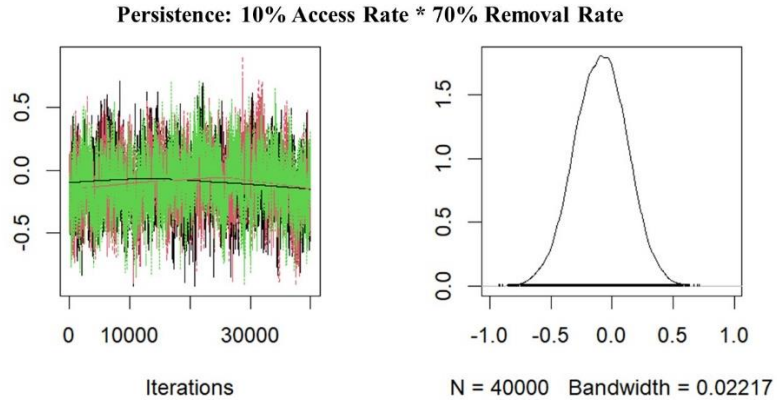


Figure D45. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

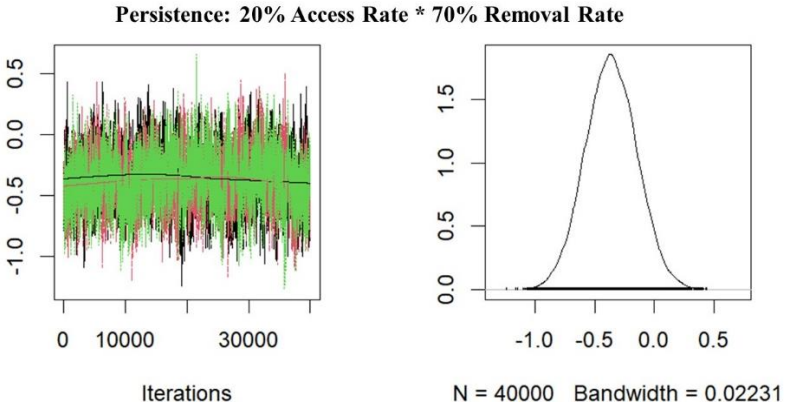


Figure D46. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

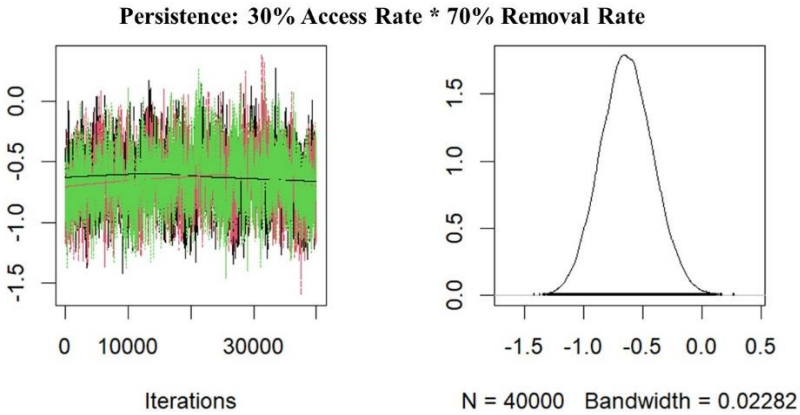


Figure D47. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

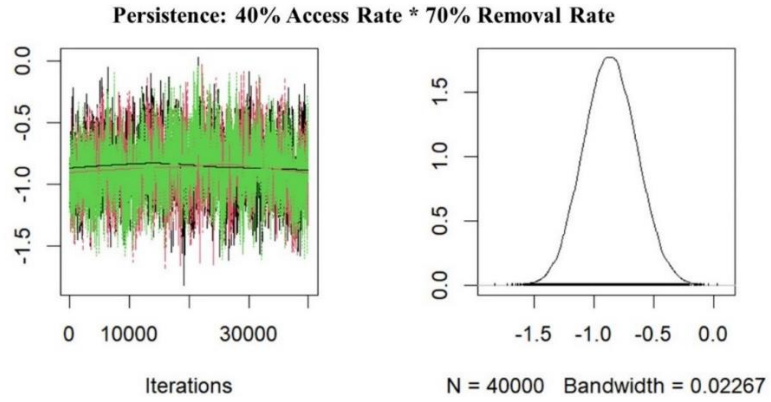


Figure D48. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

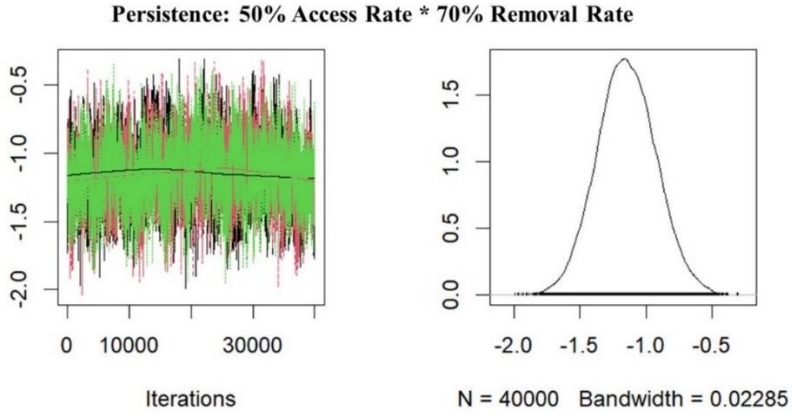


Figure D49. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 70% deer removal rate on the CWD persistence model outcome in the Bayesian hurdle model presented in Chapter 3.

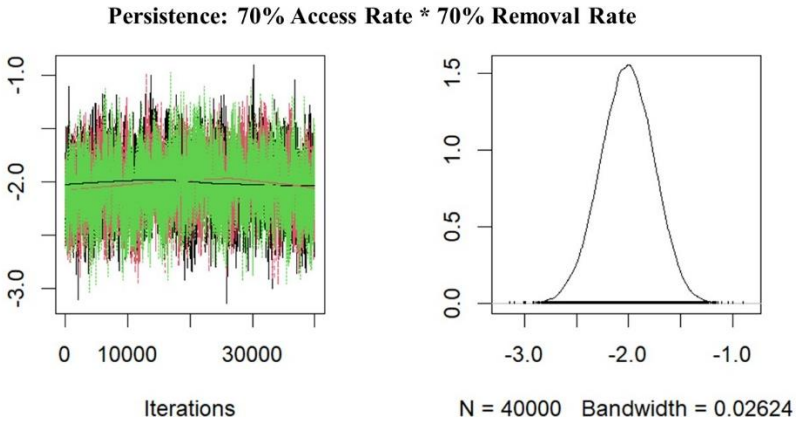


Figure D50. Gelman and Rubin diagnostic plots for the intercept on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

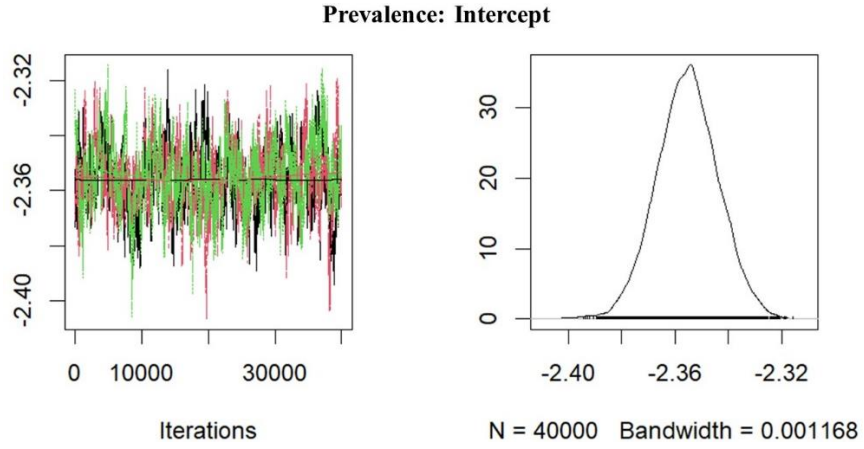


Figure D51. Gelman and Rubin diagnostic plots for the 10% land access rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

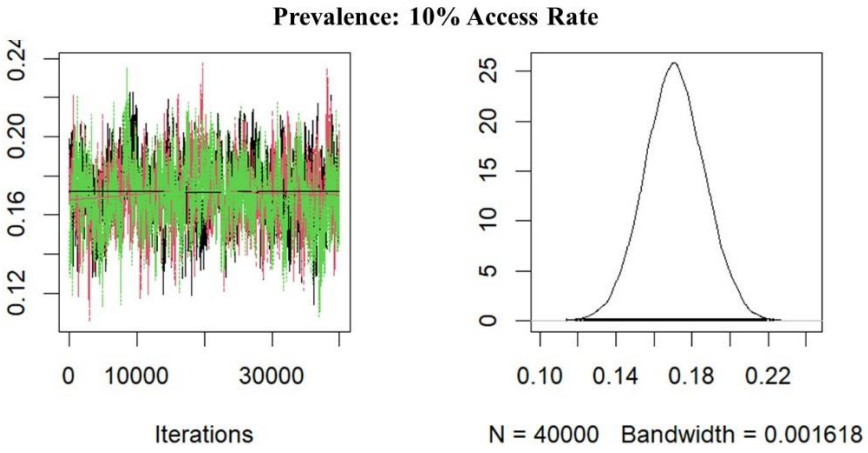


Figure D52. Gelman and Rubin diagnostic plots for the 20% land access rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

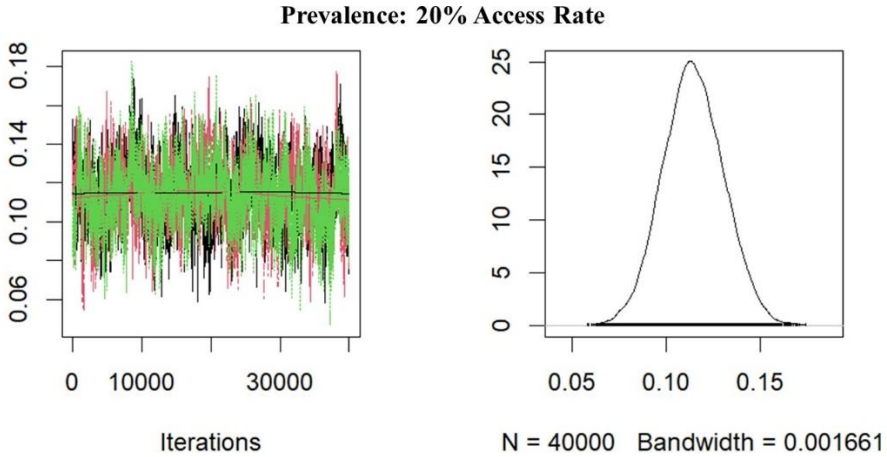


Figure D53. Gelman and Rubin diagnostic plots for the 30% land access rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

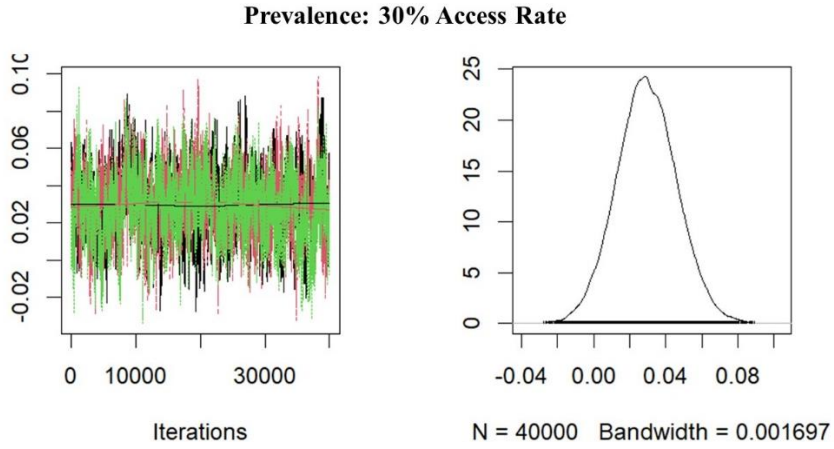


Figure D54. Gelman and Rubin diagnostic plots for the 40% land access rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

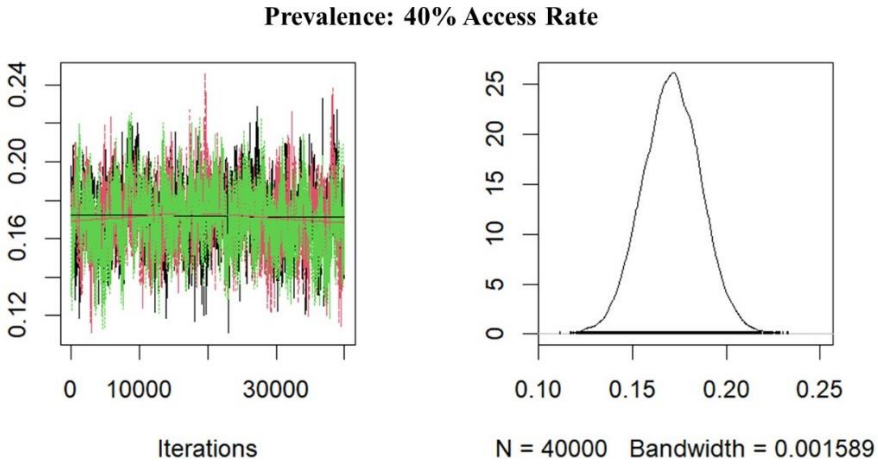


Figure D55. Gelman and Rubin diagnostic plots for the 50% land access rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

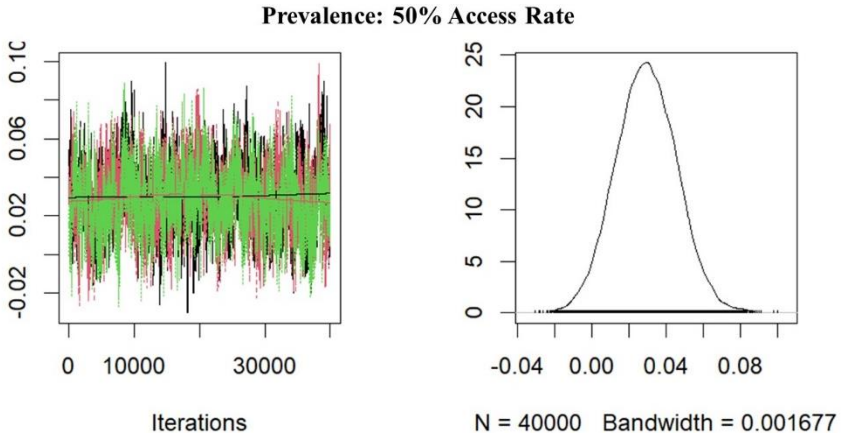


Figure D56. Gelman and Rubin diagnostic plots for the 70% land access rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

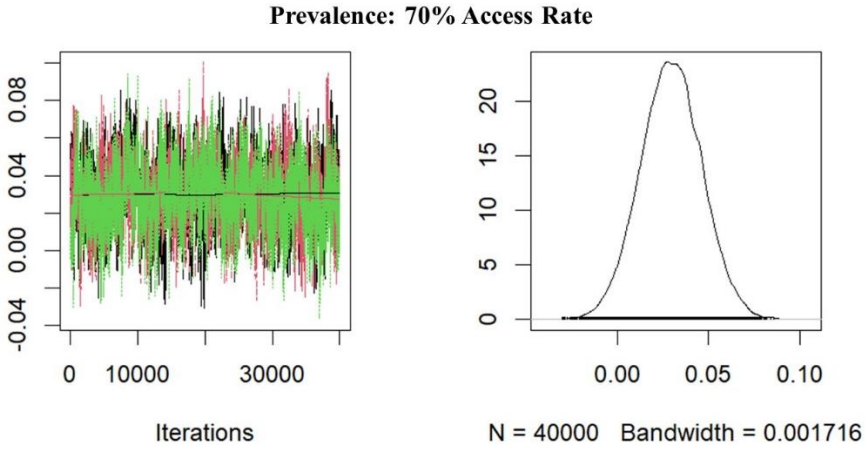


Figure D57. Gelman and Rubin diagnostic plots for the 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

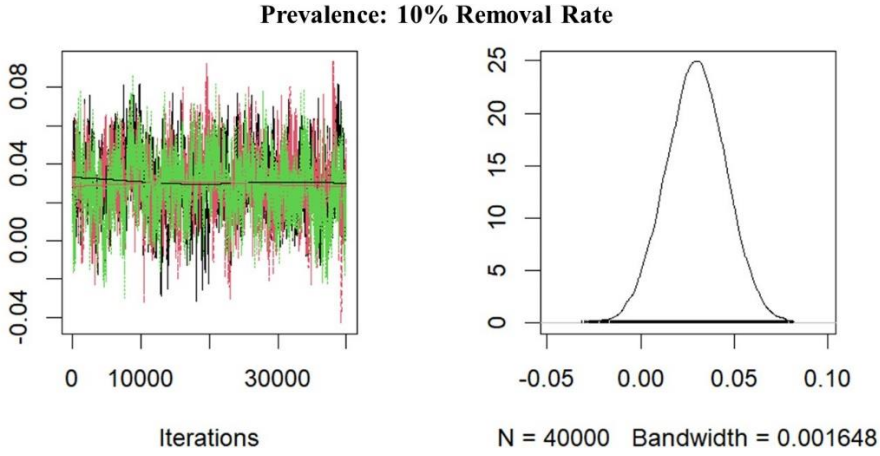


Figure D58. Gelman and Rubin diagnostic plots for the 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

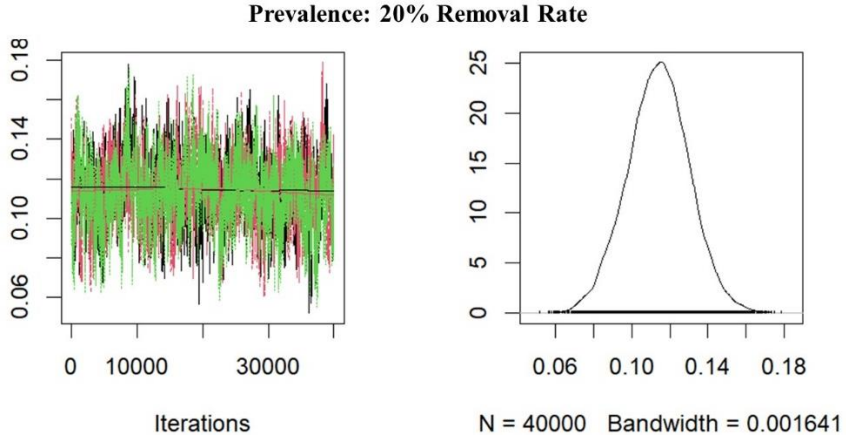


Figure D59. Gelman and Rubin diagnostic plots for the 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

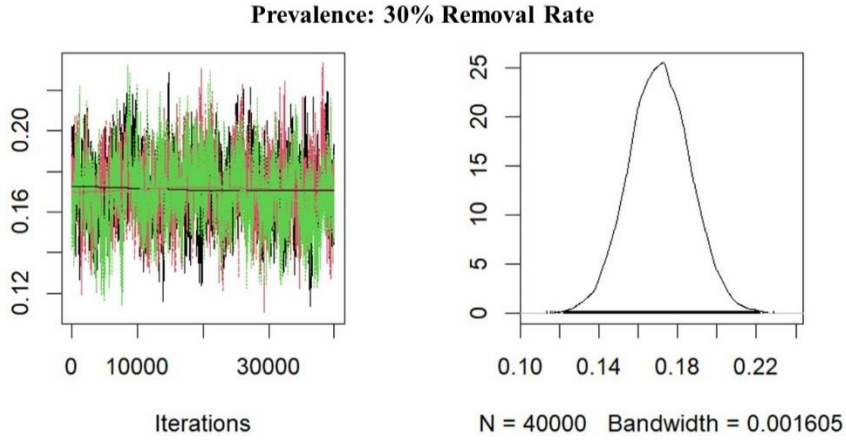


Figure D60. Gelman and Rubin diagnostic plots for the 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

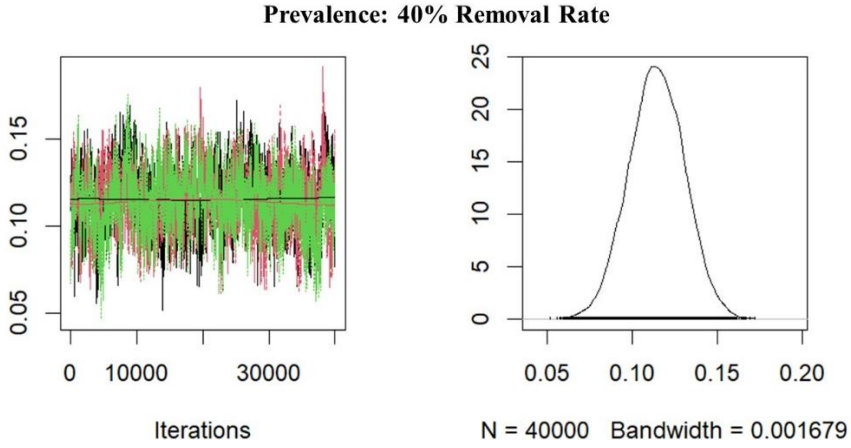


Figure D61. Gelman and Rubin diagnostic plots for the 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

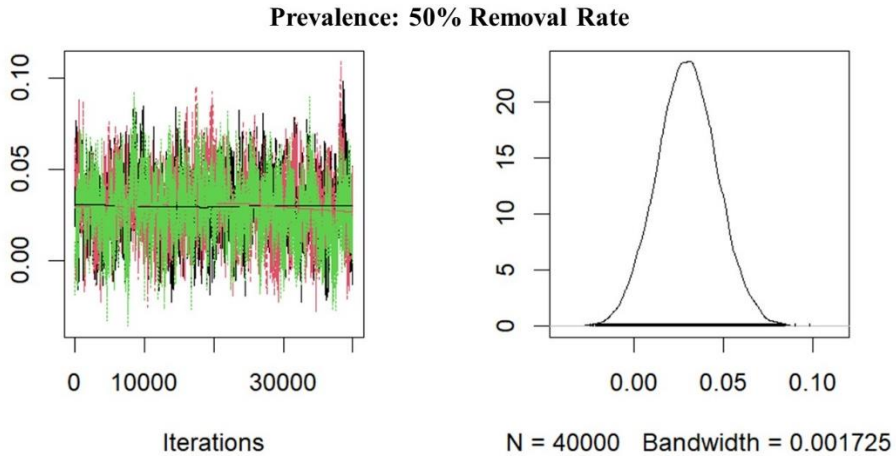


Figure D62. Gelman and Rubin diagnostic plots for the 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

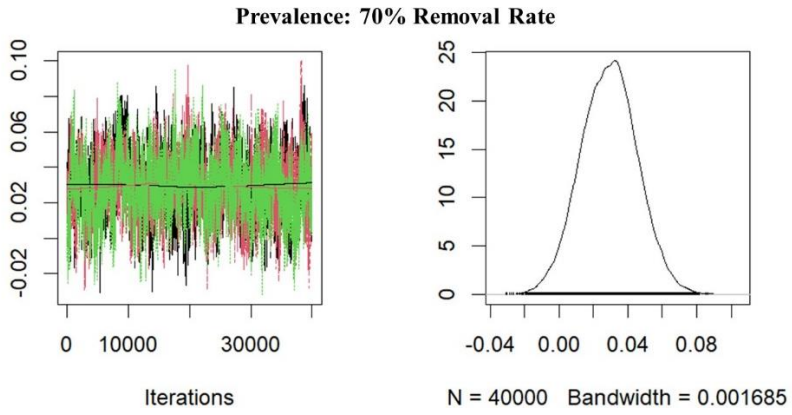


Figure D63. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

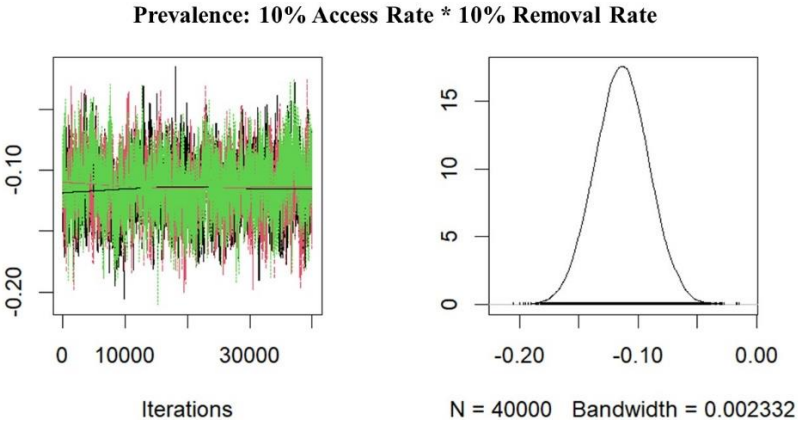


Figure D64. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

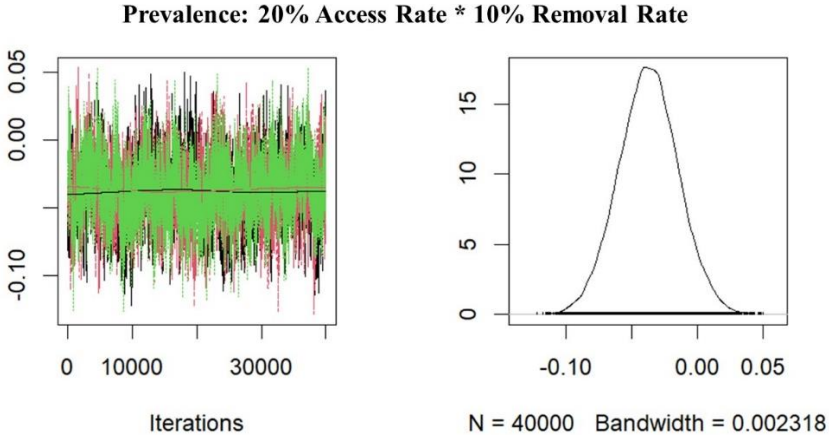


Figure D65. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

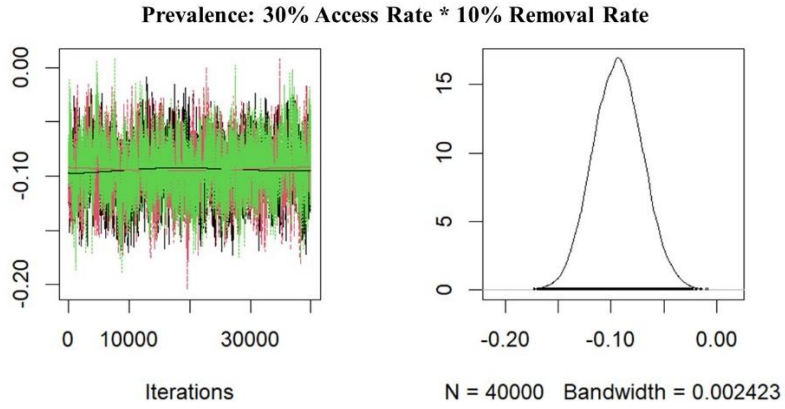


Figure D66. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

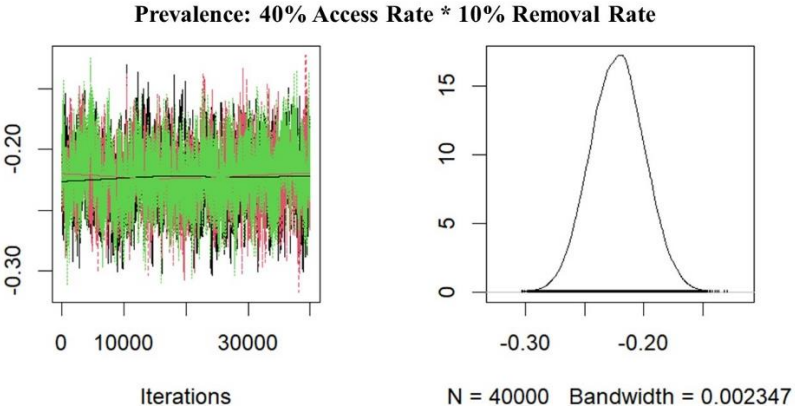


Figure D67. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

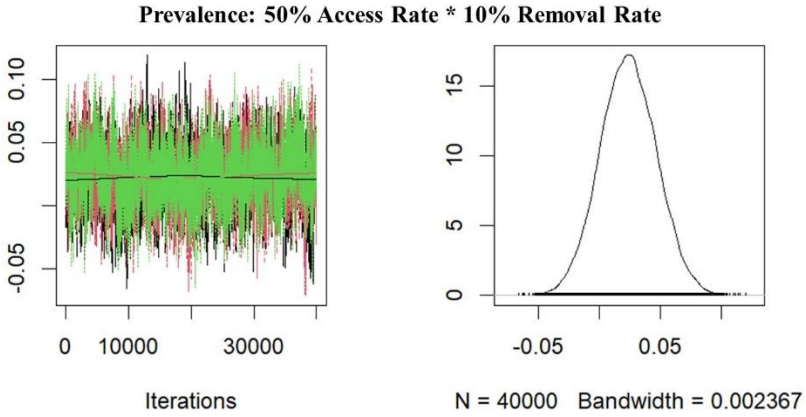


Figure D68. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 10% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

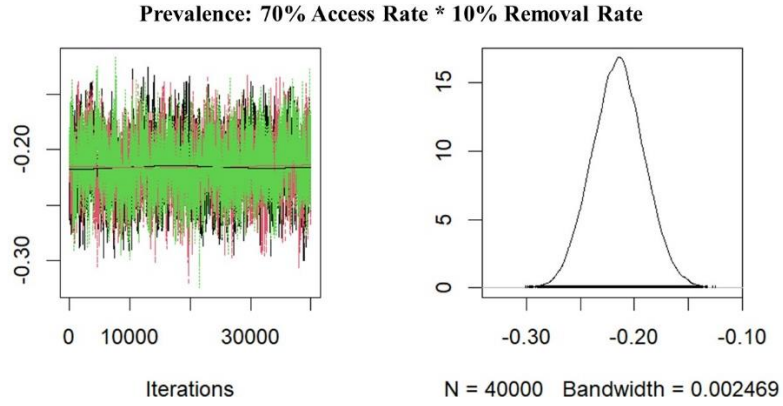


Figure D69. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

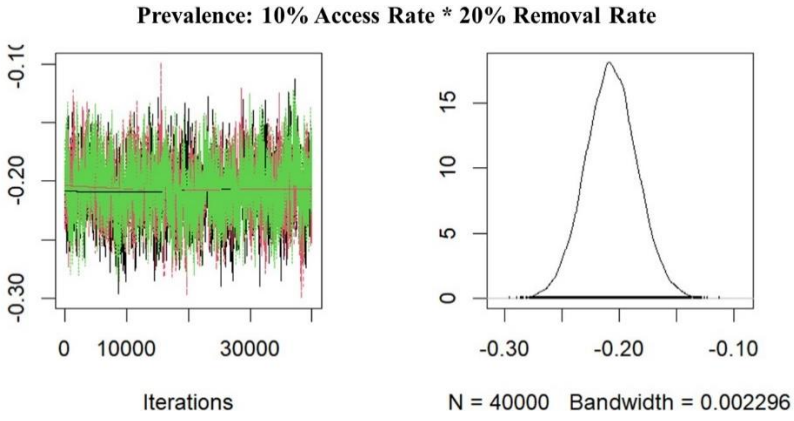


Figure D70. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

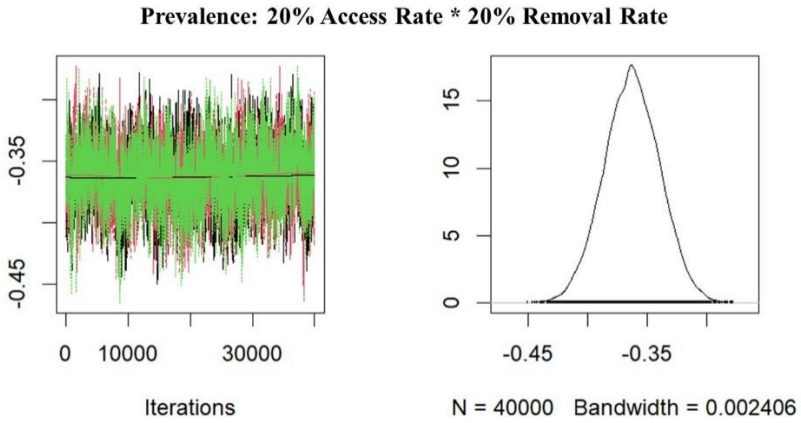


Figure D71. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

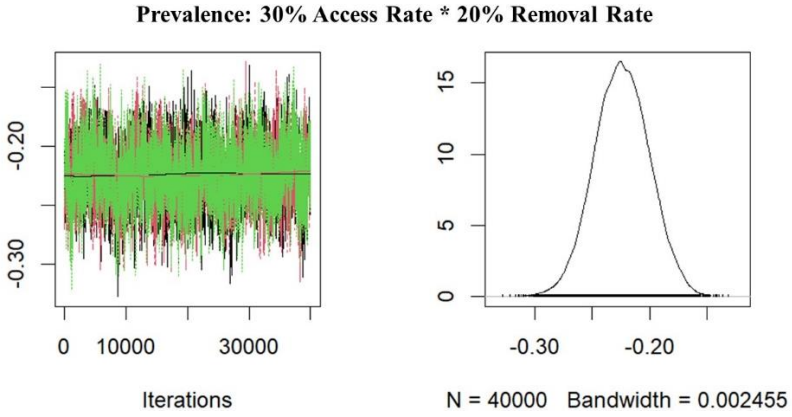


Figure D72. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

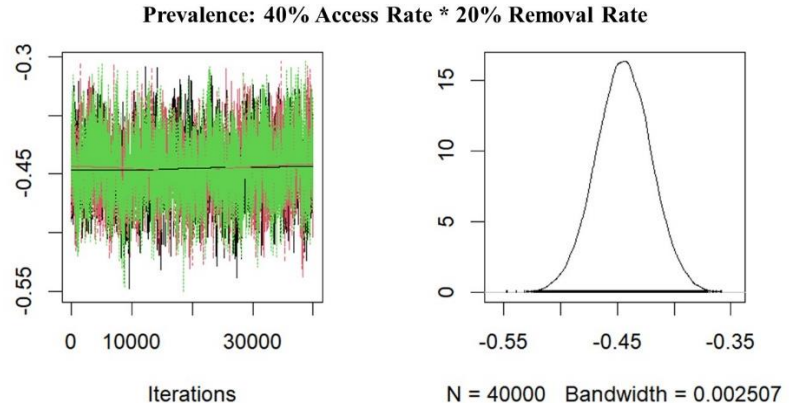


Figure D73. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

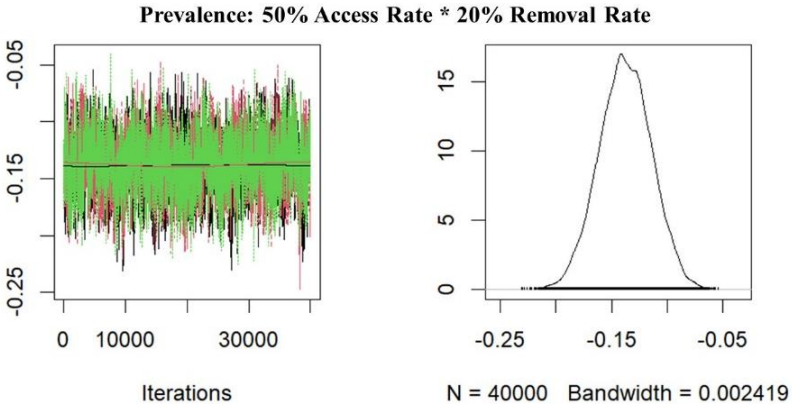


Figure D74. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 20% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

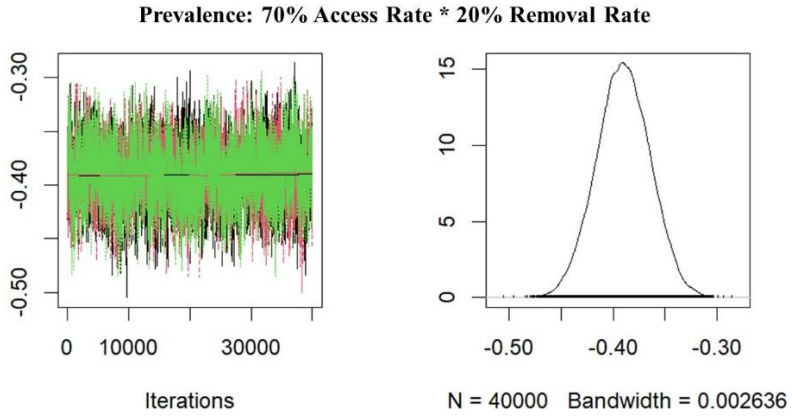


Figure D75. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

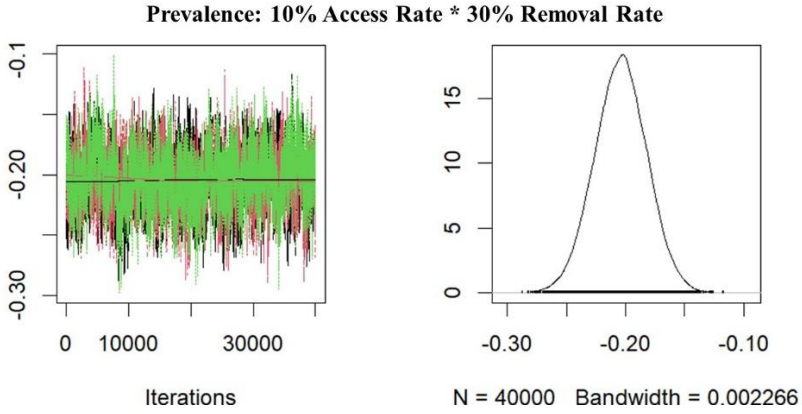


Figure D76. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

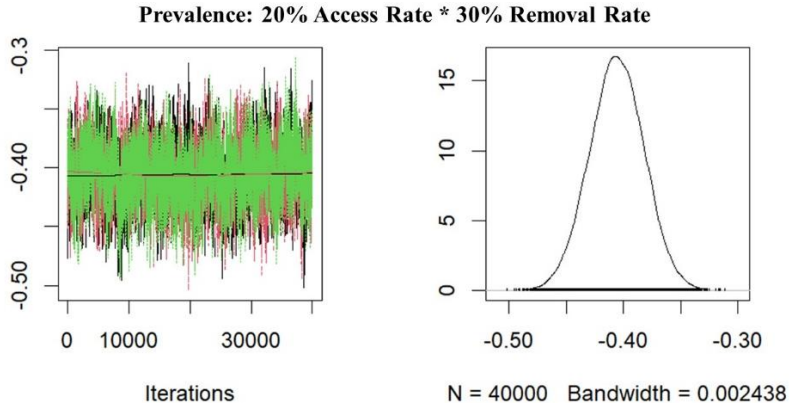


Figure D77. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

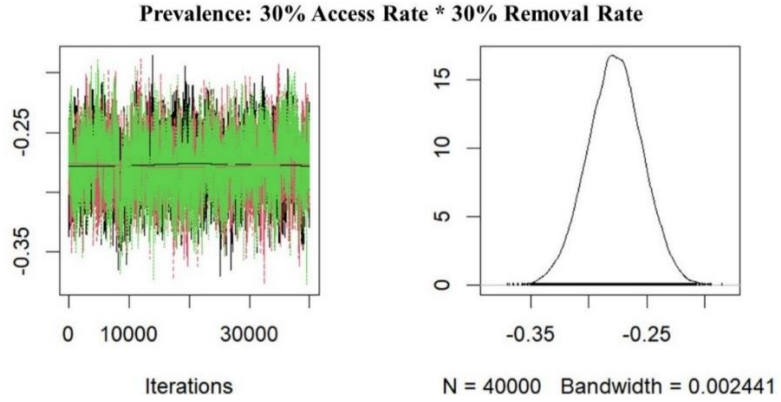


Figure D78. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

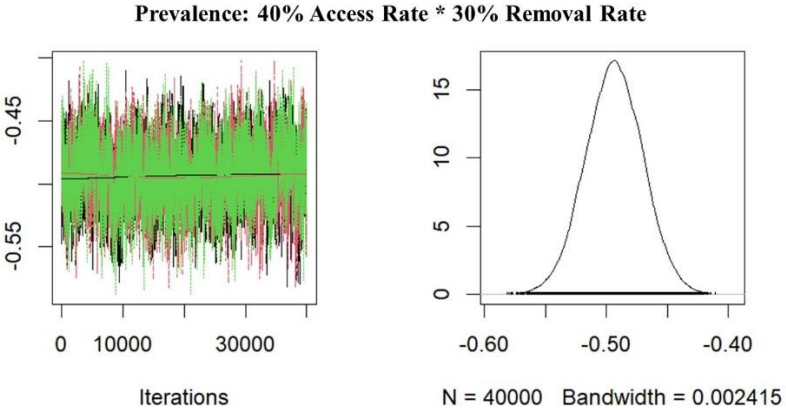


Figure D79. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

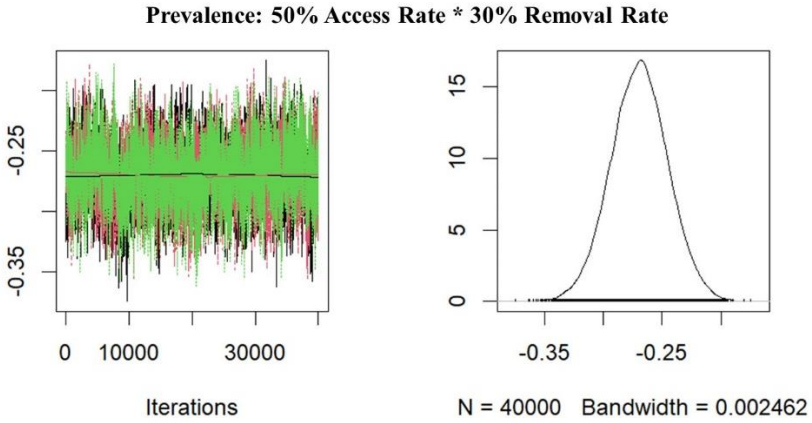


Figure D80. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 30% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

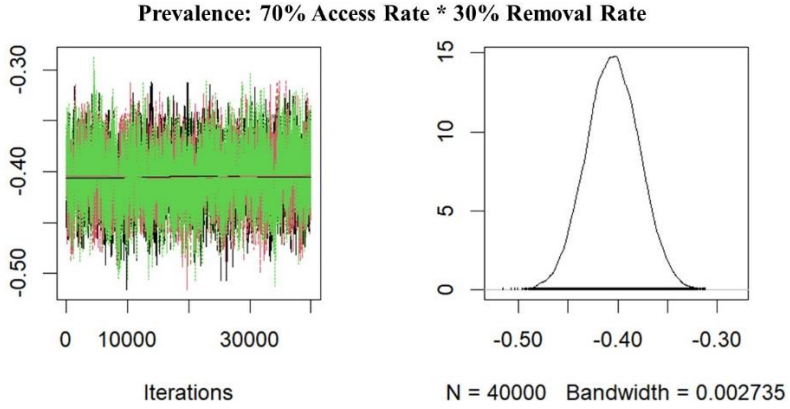


Figure D81. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

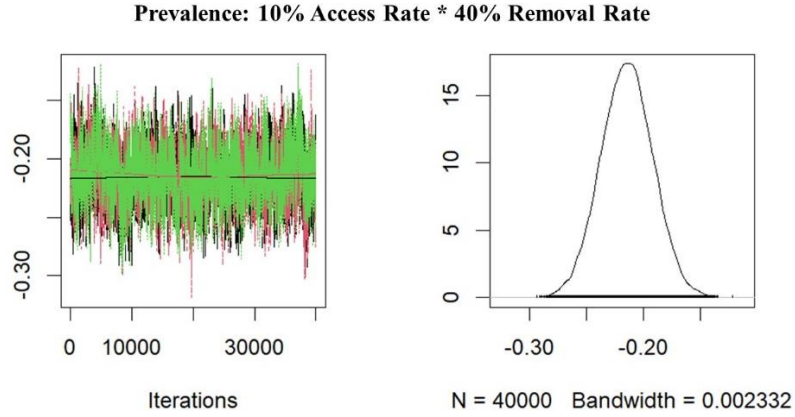


Figure D82. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

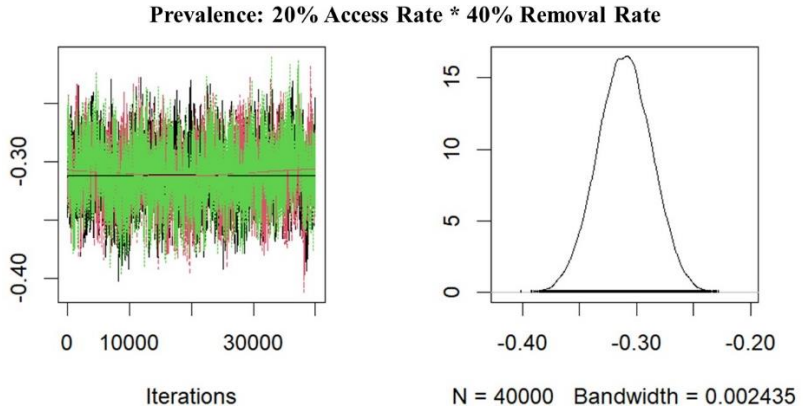


Figure D83. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

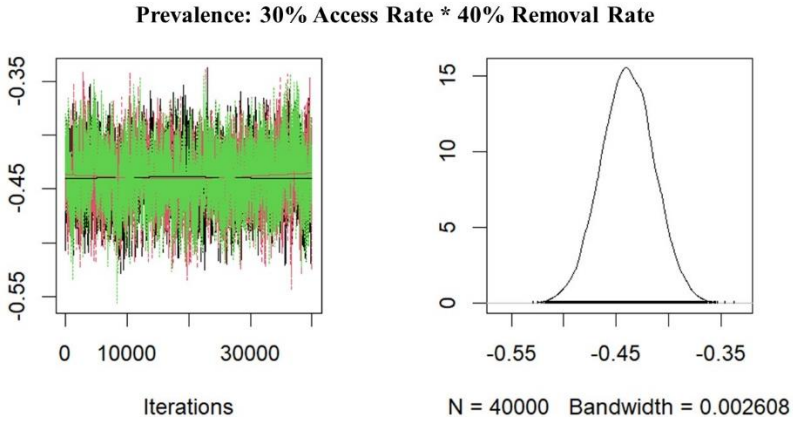


Figure D84. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

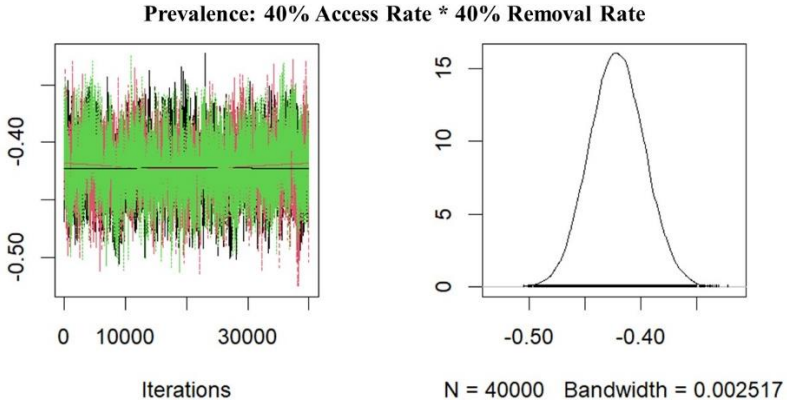


Figure D85. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

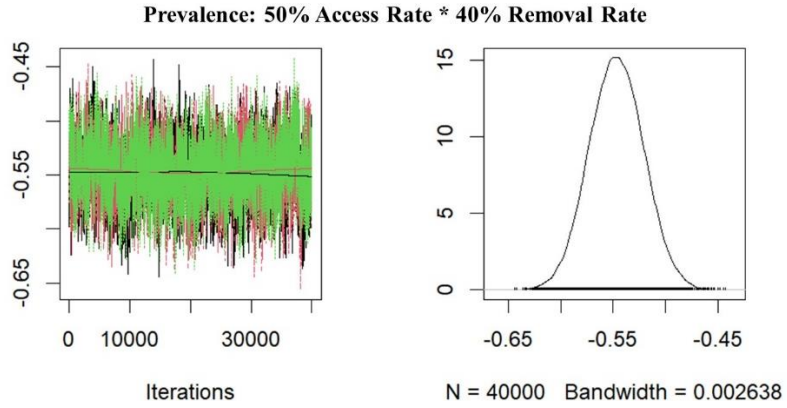


Figure D86. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 40% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

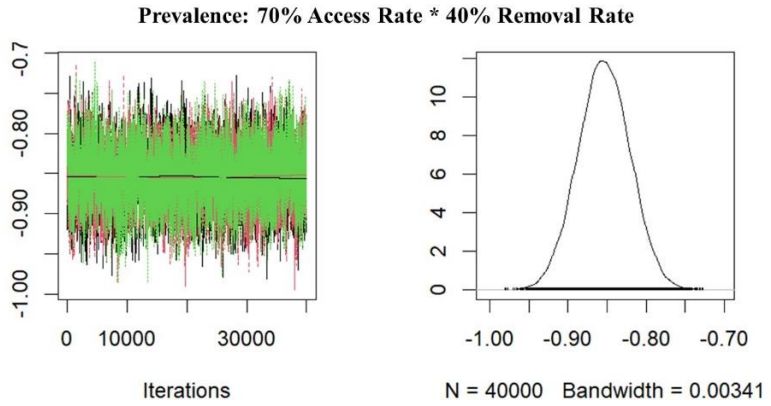


Figure D87. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

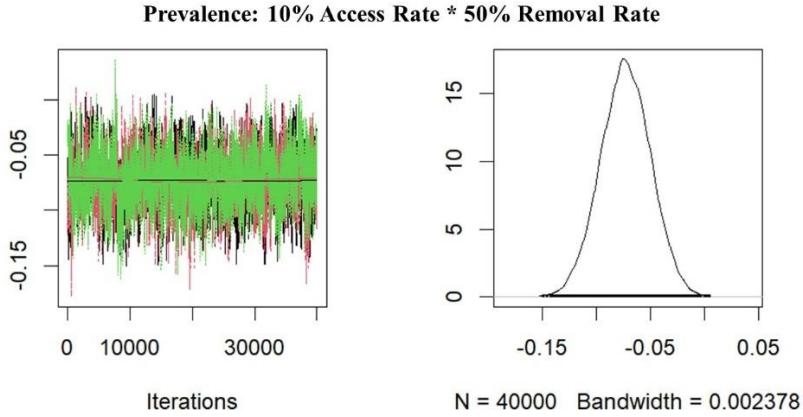


Figure D88. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

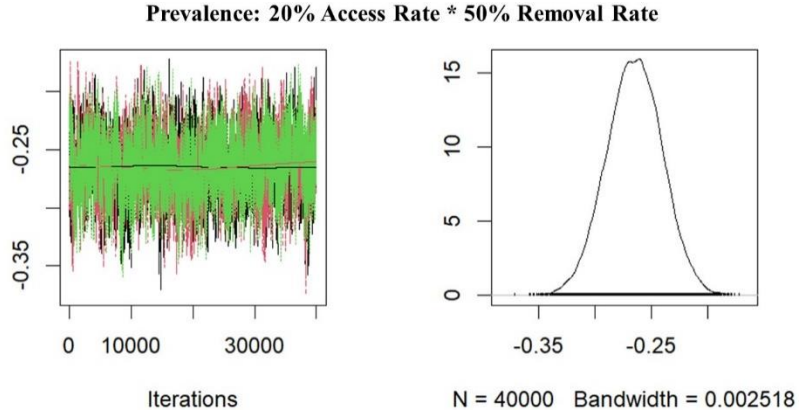


Figure D89. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

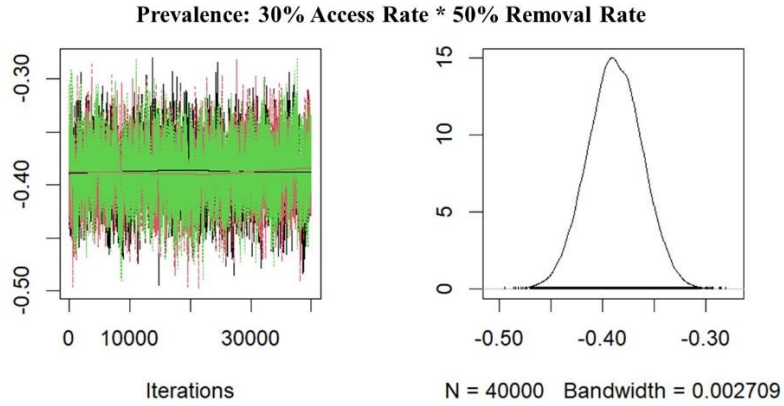


Figure D90. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

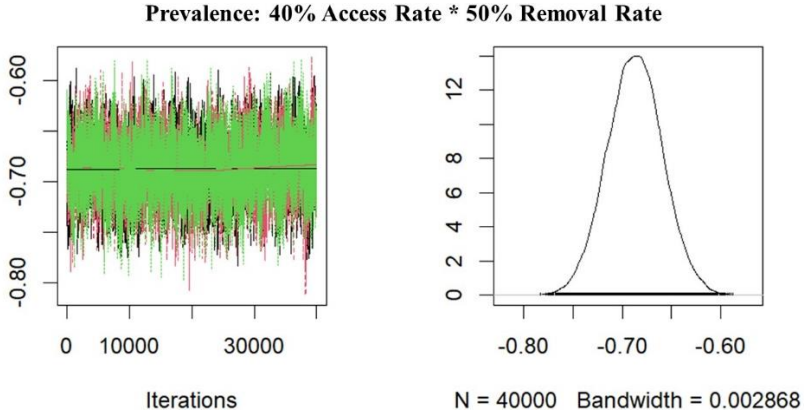


Figure D91. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

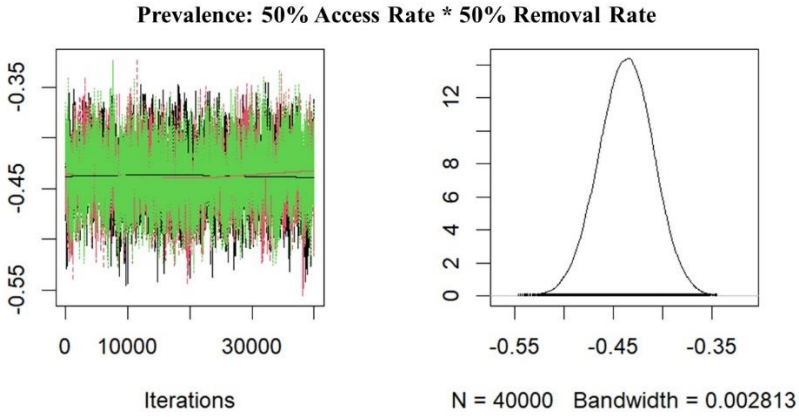


Figure D92. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 50% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

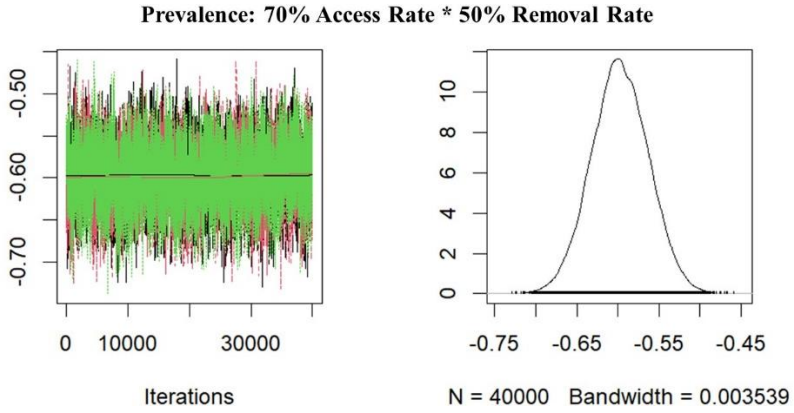


Figure D93. Gelman and Rubin diagnostic plots for the interaction between 10% land access rate and 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

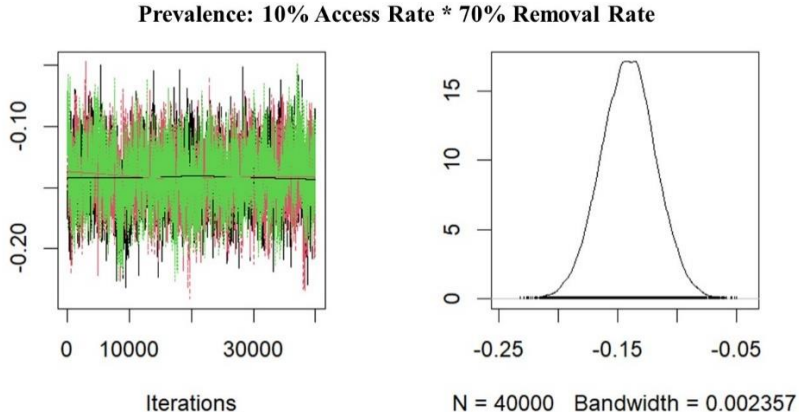


Figure D94. Gelman and Rubin diagnostic plots for the interaction between 20% land access rate and 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

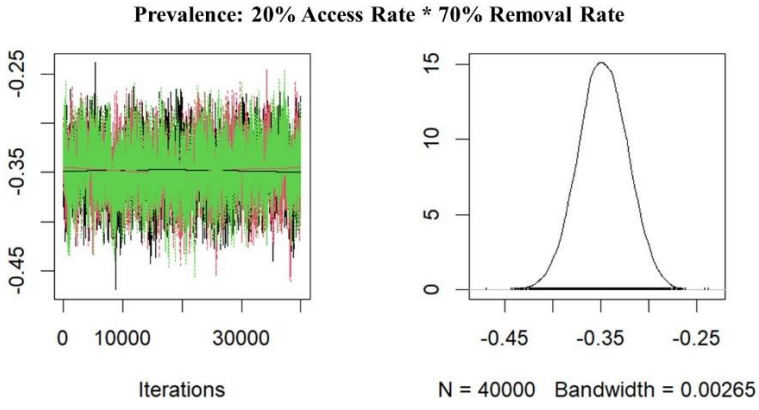


Figure D95. Gelman and Rubin diagnostic plots for the interaction between 30% land access rate and 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

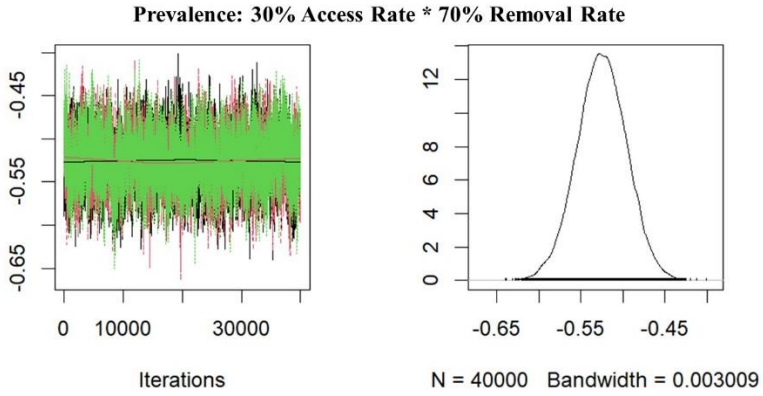


Figure D96. Gelman and Rubin diagnostic plots for the interaction between 40% land access rate and 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

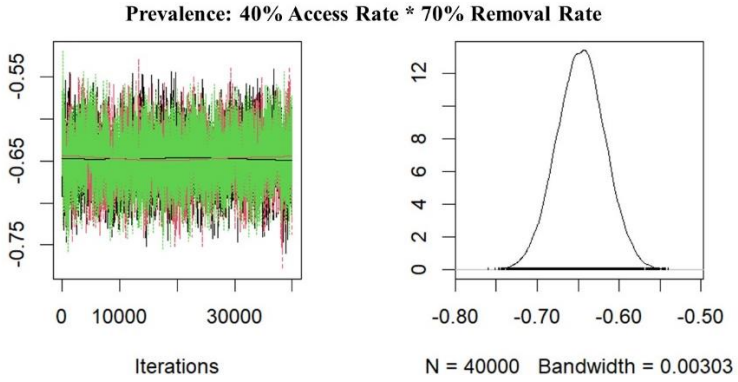


Figure D97. Gelman and Rubin diagnostic plots for the interaction between 50% land access rate and 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.

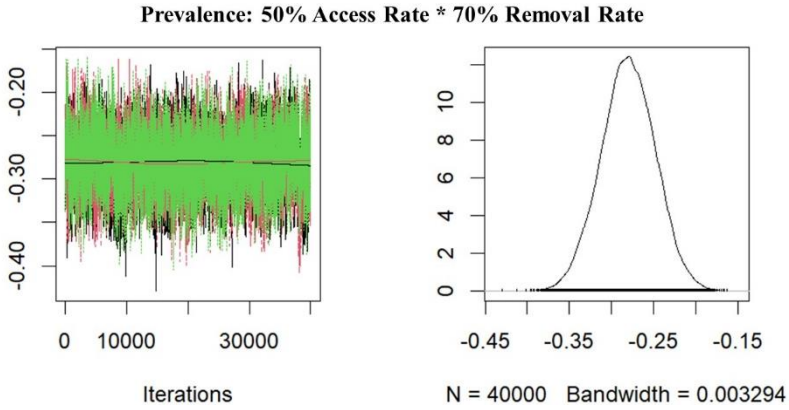


Figure D98. Gelman and Rubin diagnostic plots for the interaction between 70% land access rate and 70% deer removal rate on the number of CWD-infected deer model outcome in the Bayesian hurdle model presented in Chapter 3.