Agent Based Modeling: System Dynamics

Exploring Disease Impact on a Specialist Predator in a Predator-Prey Model Using ABM: Case Study of Isle Royale Wolves and Moose

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Abstract

This study investigates the impact of disease on the predator-prey dynamics between wolves (*Canis lupus*) and moose (*Alces alces*) on Isle Royale, utilizing Agent-Based Modeling (ABM) to simulate disease transmission scenarios. The introduction of canine parvovirus (CPV) was found to significantly reduce wolf populations, leading to decreased predation pressure on moose and a subsequent increase in their numbers. These findings enhance our understanding of how disease outbreaks can disrupt ecological balance, particularly in isolated ecosystems. While the research provides valuable insights into the long-term consequences of disease on predator-prey relationships, limitations such as reliance on historical data and simplified ecological interactions must be acknowledged. Overall, this study underscores the importance of integrating disease dynamics into ecological models and highlights the need for further research to inform conservation strategies in similar ecosystems.

Keywords: *Disease Dynamics, Predator-Prey Relationships, Canine Parvovirus, Agent-Based Modeling, Isle Royale*

1. Introduction

The predator-prey relationship between wolves (*Canis lupus*) and moose (*Alces alces*) on Isle Royale has become an iconic subject in ecology, offering a natural experiment for understanding the complexities of such dynamics in isolated environments. Since 1958, the Isle Royale Wolf-Moose Project has provided the longest continuous study of a predator-prey system globally, making it a cornerstone for ecological research (Peterson & Vucetich, 2016). Isle Royale, located in Lake Superior, is an ideal setting for such studies due to its isolation from the mainland, minimal human intervention, and the simplicity of its ecosystem. These unique conditions have allowed researchers to document fluctuations in wolf and moose populations over time, driven by factors such as food availability, climate variation, and disease outbreaks (Peterson et al., 1984).

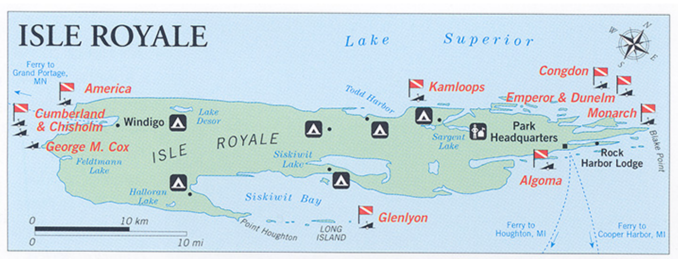

Figure 1. Map of Isle Royale National Park

One of the most significant challenges in predator-prey research lies in understanding the role of disease in shaping the population dynamics of specialist predators like wolves. While previous studies, such as those by Hudson et al. (2002) and Dobson and Hudson (1992), have highlighted the role of diseases in regulating wildlife populations, these studies primarily focused on prey species or generalist predators. The specific effects of diseases on specialist predators, especially in tightly coupled systems like that of Isle Royale, remain less understood. The introduction of canine parvovirus (CPV) to the Isle Royale wolf population in the 1980s is one of the most striking examples of how disease can disrupt predator-prey balance. CPV significantly reduced the wolf population, leading to an overabundance of moose, which in turn caused considerable damage to the island's vegetation (Peterson, 1995).

Despite the substantial short-term impacts of CPV on the wolf population, there remains a lack of comprehensive understanding of the long-term consequences of disease outbreaks on predator-prey dynamics. Longitudinal studies on Isle Royale have shown that diseases like CPV can alter not only predator behavior and population health but also the stability of the entire ecosystem. Yet, gaps in our understanding persist, particularly concerning how disease dynamics affect such specialist predator-prey systems' recovery and long-term resilience.

Our research seeks to address these knowledge gaps by focusing on the impact of disease on wolf populations and the effects on moose populations. Using Agent-Based Modeling (ABM), we simulate disease transmission scenarios to explore how outbreaks, such as those caused by CPV, influence the dynamics between wolves and moose. By examining the cascading effects of disease on predator behavior, pack structure, and population health, we aim to reveal how these changes in predator populations affect moose numbers and ecosystem balance over time.

Our findings indicate that the changes in disease transmission rate among the wolf population is leading to recurring disease outbreaks that cause significant population declines. These outbreaks reduce predation pressure on the moose population, leading to a substantial increase in moose numbers. Additionally, our research supports and validates empirical data collected from Isle Royale, reinforcing the observed impacts of disease on predator-prey dynamics. These results enhance our understanding of disease dynamics in specialist predator-prey systems and provide valuable insights for conservation efforts, particularly in isolated ecosystems such as Isle Royale.

2. Methodology

2.1. Overall Approach and Design of the Model

The model simulates interactions between two species, wolves and moose, on Isle Royale. The main focus is on understanding how disease dynamics in the wolf population impact the predator-prey relationship. The simulation is implemented using an Agent-Based Model (ABM) in NetLogo, where individual agents (wolves and moose) follow rules that govern their behavior, including movement, predation, reproduction, and disease transmission.

The code diagram (Figure 3) provides a visual representation of the model's structure, showing how different components interact. The diagram includes various rates and flows that dictate how populations change over time, such as birth rates, predation rates, and disease-related mortality.

2.2. Implementation Process

2.2.1. Agent Class Design

Moose:

- *Attributes*: Each moose agent has attributes such as age and energy levels.

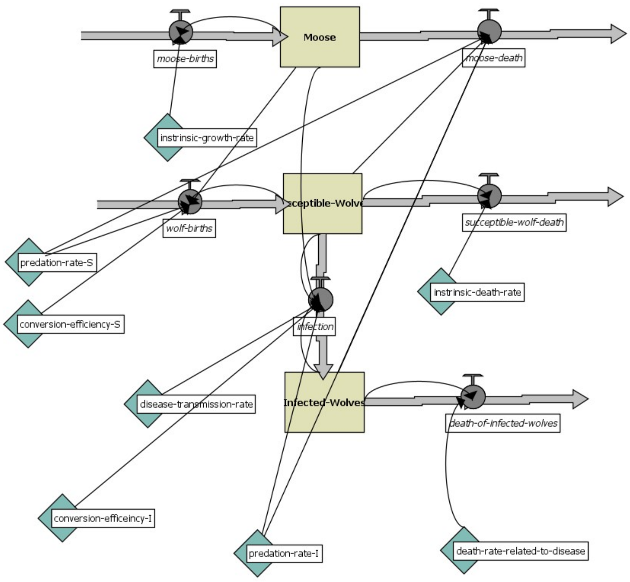

Figure 2. Code diagram illustrating the interactions between moose and wolves, with focus on birth rates, predation rates, and disease dynamics.

- *Processes*: Moose move randomly across the environment, reproduce based on their intrinsic growth rate, and die due to natural causes or predation by wolves. The code diagram shows the flow from "moose-births" to the "Moose" population and the outflow to "moose-death," indicating the balance between birth and death rates.

Wolves:

- *Attributes*: Wolves are divided into two categories: susceptible (healthy) and infected (diseased).
- *Processes*:
  - *Birth*: Wolf reproduction is shown in the diagram as "wolf-births," contributing to the "Susceptible-Wolves" population.
  - *Predation*: Wolves hunt moose, with the "predation-rate-S" affecting susceptible wolves and "predation-rate-I" affecting infected wolves. These rates determine the flow of energy to the wolves and the reduction in the moose population.
  - *Infection*: Susceptible wolves can become infected through a disease transmission process, represented by the "infection" flow between "Susceptible-Wolves" and "Infected-Wolves."
  - *Mortality*: Both susceptible and infected wolves have distinct death rates. The diagram indicates "susceptible-wolf-death" and "death-of-infected-wolves," with an additional death rate specific to the disease ("death-rate-related-to-disease").

2.2.2. Initialization

Population Initialization: The initial populations for both wolves and moose are set based on historical data, for example, 50 wolves and 800 moose. The agents are placed randomly within the simulated environment.

Parameter Initialization: Global parameters such as predation rates, birth rates, and disease transmission rates are initialized based on ecological studies. These parameters govern the flow rates and transitions shown in the diagram.

2.2.3. Process Implementation

Movement: Both wolves and moose move randomly across the environment, searching for food or prey.

Reproduction: Both species reproduce based on their intrinsic rates, which are influenced by age and energy levels. The diagram shows "intrinsic-growth-rate" and "moose-births" for moose, and "wolf-births" for wolves.

Predation: Wolves hunt moose according to their health status, with successful hunts increasing the wolves' energy levels. Predation rates differ for susceptible and infected wolves, as shown in the "predation-rate-S" and "predation-rate-I" in the diagram.

Disease Transmission: The disease spreads among wolves through contact, reducing their energy levels and lifespan. The "disease-transmission-rate" in the diagram connects "Susceptible-Wolves" to "Infected-Wolves," indicating the flow from healthy to diseased individuals.

2.3. Visualization Techniques and Sensitivity Analysis

2.3.1. Visualization

Population Dynamics: The model outputs time series graphs displaying the populations of wolves and moose over time, with separate lines for susceptible and infected wolves. This allows visualization of how disease impacts wolf population and predation rates.

2.3.2. Sensitivity Analysis

Parameter Variation: A sensitivity analysis is conducted by varying key parameters like predation rates, birth rates, and disease transmission rates. The results are analyzed to determine the robustness of the model outcomes.

Outcome Analysis: Different scenarios are tested to observe how changes in disease transmission or predation rates affect overall population dynamics, as depicted in the flows and interactions in the diagram.

2.4. Model Assumptions

The model focuses exclusively on the interactions between moose and wolves, specifically examining the dynamics between these two species. It simplifies the ecological context by assuming that the population of balsam fir, the primary food source for moose, remains constant throughout the study period. This assumption allows the model to concentrate on the predator-prey dynamics without the added complexity of fluctuating food resources. Additionally, the model presumes that the effects of climate change are negligible, thereby excluding any potential impact on the habitat or the populations of moose and wolves. These assumptions streamline the model, enabling a clearer analysis of the direct interactions between the predator (wolves) and prey (moose) populations.

3. Results and Discussion

In this section, we present the results derived from our comprehensive simulation models, which were meticulously constructed based on empirical studies of predator-prey dynamics and disease ecology. Our approach began with the careful collection and analysis of relevant empirical data to establish accurate initial conditions and parameter values for our models. With these data, we simulated the population dynamics of wolves and moose over a specified period, focusing on tracking changes in the numbers of susceptible and infected individuals within each species.

Our simulation efforts aimed to capture the complexities of real-world ecosystems, specifically examining how diseases such as Canine Parvovirus influence predator-prey interactions and overall population stability. To provide a thorough understanding of these dynamics, we implemented a range of sensitivity analyses. These analyses involved systematically varying key parameters, including disease transmission rates and the mortality rate due to disease, to observe how these changes impact the model's outcomes. By exploring different scenarios, we assessed the robustness of our model and its sensitivity to variations in disease dynamics.

It is important to note that due to the inherent limitations in the available data, our predictions cannot be exact. The lack of comprehensive data on certain aspects of the disease dynamics and ecological interactions means that while our model provides valuable insights and approximate predictions, it may not capture all nuances of real-world behavior. Nonetheless, the results offer a reasonable approximation and contribute meaningfully to our understanding of disease impacts on predator-prey systems. This detailed examination enhances our grasp of the specific case study of wolves and moose and underscores the importance of integrating disease dynamics into predator-prey models, despite the limitations of data constraints.

Parameter	Value
Intrinsic growth rate	0.048
Predation rate (Susceptible)	0.12
Conversion efficiency (Susceptible)	0.09
Disease transmission rate	0.99
Intrinsic death rate	0.45
Death rate related to disease	0.89
Predation rate (Infected)	0.000048
Conversion efficiency (Infected)	0.045
Infected Wolves	10
Moose	788
Susceptible Wolves	40

Table 1. Initial Model parameters and their values.

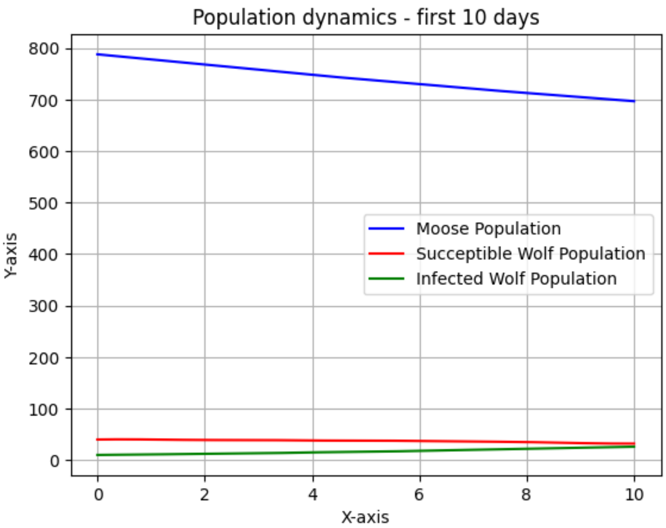

Figure 3. Simulation of first 10 days

The graph illustrates the population dynamics over the first 10 days for three groups: moose, susceptible wolves, and infected wolves. The moose population, represented by a blue line, shows a clear decreasing trend, starting from around 800 and gradually declining throughout the period. In contrast, the susceptible wolf population, shown by the red line, remains stable, hovering around 50 without significant change. Similarly, the infected wolf population, depicted by the green line, stays nearly flat near zero, indicating very little variation or a consistently low number of infected wolves over the same time frame.

Figure 4 indicates the current population counts for moose and wolves, showing that the moose population has decreased to 624, reflecting a decline from an earlier value (likely 800 as previously noted). Meanwhile, the population of susceptible wolves is critically low, with only one individual remaining susceptible to infection. In contrast, there are 59 infected wolves, suggesting that the infection has spread widely among the wolf population. This disparity high-

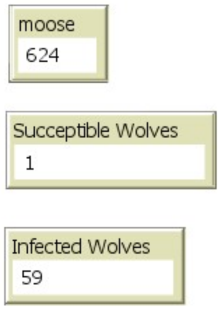

Figure 4. Population after 40 days

lights the ongoing decline in the moose population and the significant impact of infection within the wolf population.

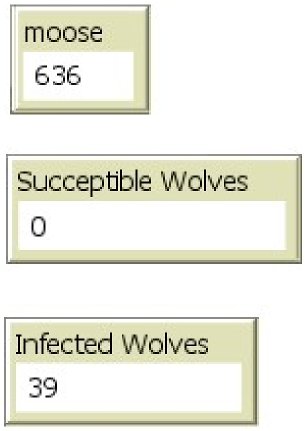

Figure 5. Population after 200 days

After 200 days (Figure 5), the population dynamics reveal that the moose population has increased slightly to 636. The number of susceptible wolves has dropped to zero, indicating that either all wolves have succumbed to the infection or the susceptible wolves have died out. The number of infected wolves has also decreased to 39, reflecting a significant decline from the 59 infected wolves observed at the 40-day mark. This suggests that the disease has caused deaths among the wolf population. As a result of the declining wolf population, the moose population appears to be growing, likely due to reduced predation pressure. The scenario reflects the detrimental impact of disease on the wolves, potentially leading to long-term ecological shifts in predator-prey dynamics.

The situation can be considered a disease outbreak within the wolf population. Initially, after 40 days, a significant portion of the wolves became infected, with 59 infected and only one susceptible wolf remaining. By 200 days, all susceptible wolves have either died or become infected, leaving the population with zero susceptible wolves and 39 infected individuals. This widespread and severe infection has caused a notable decline in the wolf population, indicating that the disease has spread rapidly and caused substantial mortality. As a result, the moose population has increased, likely due to reduced predation pressure, illustrating the outbreak's broader ecological consequences and its significant impact on predator-prey dynamics.

The sensitivity analysis table reveals how varying the disease transmission rates in a wolf population influences both the wolves and their prey, the moose. As the transmission rate decreases, a larger portion of the wolf population remains healthy, leading to more effective predation and a significant reduction in the moose population. Conversely, higher transmission rates result in a predominantly infected wolf population, which appears to reduce their predation efficiency,

Disease Transmission Rate	Susceptible Wolves	Infected Wolves	Moose
0.5	0	62	475
0.3	3	74	315
0.1	47	52	84

Table 2. Population dynamics under different disease transmission rates.

allowing the moose population to grow. This suggests that the disease transmission rate plays a critical role in the predator-prey dynamics, with lower rates maintaining a balanced ecosystem, while higher rates could lead to an imbalance, favoring the prey population due to the diminished hunting capability of the infected predators.

4. Conclussion

The study explored the dynamics of disease impact on the predator-prey relationship between wolves and moose on Isle Royale, using Agent-Based Modeling (ABM) to simulate various disease transmission scenarios. The main findings indicate that the introduction of canine parvovirus (CPV) significantly affects wolf populations, leading to a decrease in predation pressure on moose and consequently an increase in their numbers.

These results are crucial for understanding the original questions posed in the introduction regarding the long-term consequences of disease outbreaks on predator-prey dynamics. The findings enhance our comprehension of how diseases can alter not only predator behavior and health but also the overall stability of ecosystems, particularly in isolated environments like Isle Royale. By validating empirical data and demonstrating the cascading effects of disease on population dynamics, this research provides valuable insights for conservation efforts aimed at maintaining ecological balance.

However, the study has limitations that must be acknowledged. The reliance on historical data for initial population parameters may introduce uncertainties, as ecological dynamics can vary significantly over time. Additionally, the model simplifies complex interactions by assuming constant food resources and negligible climate impacts, which may not accurately reflect real-world conditions. The inherent challenges in capturing the full range of ecological interactions and disease dynamics also pose potential sources of error, limiting the generalizability of the findings.

In conclusion, this study underscores the significant role that disease plays in shaping predator-prey relationships and highlights the need for further research in this area. Future studies should aim to incorporate more comprehensive data on ecological interactions and consider the impacts of environmental changes, such as climate variability, on disease dynamics. By expanding the scope of research to include these factors, we can better understand the complexities of predator-prey systems and develop more effective conservation strategies.

5. References

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[2] Martcheva, M. (2015). An introduction to mathematical epidemiology (Vol. 61, pp. 9-31). New York: Springer.
[3] We have utilized ChatGPT (OpenAI, 2024) as a tool to assist with grammar and writing refinements.

6. Member Contributions

1. Tharuka Anthony - Methodology section
2. Danula perera - Results & Discussion section
3. Nimna Suharshani - Introduction Section, References Section
4. Sandali Hansika - Introduction Section, Conclusion Section

All members are equally contributed to create the NetLogo code.