

THE UNIVERSITY OF CALIFORNIA, LOS ANGELES  
MATH 42

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Predator/Prey Population Simulation Based on  
Precipitation, Temperature, and Cross-Special  
Transmitted Viral Infection

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# 1 Abstract

For our mathematical research experiment, we initially planned on concluding with an equation that would describe the realistic dynamic relationship between two species with a rampant cross-species transmissible virus. However, with the scope of the project and given deadline, we were only able to achieve possible connections between variables instead of a conclusive equation.

We started our research by wanting to simulate a realistic dynamic relationship between a predator-prey system and simulate cross-species viral infections. On a side note, this model can potentially be used to represent and observe results that could also be seen as two big topics of the year 2022: war and pandemic. For our data set, we referenced the Wolf-Elk data set provided by Yellowstone National Park. After the experiment, the simple implementation of differential equations and the Markov-chain method did not fit the real-life data. We achieved the intended Wolf-Elk model, and results with the Lotka Volterra equation modified through the Allee effect, but we could not quite simulate the reality of Yellowstone National Park and relate environmental factors to disease model. The parameters are not yet validated by the least-square method, leaving this project to be inconclusive yet still capable of further developments.

# Contents

<b>1</b>	<b>Abstract</b>	
<b>2</b>	<b>Problem</b>	<b>1</b>
2.1	Implementing Environmental Factors in Predator-Prey Model . . . . .	1
2.2	Idea of Implementing Cross-Special Virus in Predator-Prey Model . . . . .	1
2.3	Relationship Between Virus-Environment-Prey-Predator . . . . .	1
<b>3</b>	<b>Simplifications</b>	<b>2</b>
3.1	Assumptions . . . . .	3
<b>4</b>	<b>Development of Mathematical Model and Solution</b>	<b>4</b>
4.1	Developing from the Simple Predator-Prey Model . . . . .	4
4.2	Virus Infection and Death Rate . . . . .	5
4.3	Modifying the Population Differential Equation . . . . .	5
4.3.1	Herd Immunity Simplification . . . . .	5
4.3.2	Appending the Allee Effect . . . . .	6
4.3.3	New Model . . . . .	8
4.4	Inconclusive Results . . . . .	8
4.4.1	Inseparable Weather Factors . . . . .	9
4.4.2	Indeterminable Population Parameters . . . . .	9
4.5	Referring to Lotka-Volterra and Another Possible Equation . . . . .	9
4.6	Final Thoughts and Most Recently Modified Equation . . . . .	11
4.6.1	Summary of Derived Equations . . . . .	13
<b>5</b>	<b>Results</b>	<b>13</b>
<b>6</b>	<b>Improvements</b>	<b>20</b>
6.1	Limitations . . . . .	20
<b>7</b>	<b>Conclusions</b>	<b>21</b>
<b>8</b>	<b>Bibliography</b>	<b>22</b>

<b>9</b>	<b>Appendix</b>	<b>24</b>
9.1	R Code for Data Cleaning . . . . .	24
9.2	R Code for Environment plots . . . . .	25
9.3	R Code for Models . . . . .	25

## **2 Problem**

### **2.1 Implementing Environmental Factors in Predator-Prey Model**

A standard Predator vs Prey Model is too generic and simplified because it only takes into consideration growth rate and predation. Our initial plan was to formulate a realistic environment via an environment-affecting population model by using Markov chains to statistically affect the rates of changes in different age groups. The rates will then be placed in a Leslie Matrix to realistically simulate the growth of an initial population over time. This population will then be reduced via a viral infection.

### **2.2 Idea of Implementing Cross-Special Virus in Predator-Prey Model**

Since the cross-special disease is a rare case, we wanted to see how a lethal disease would have an effect on two populations if it transferred over from wolves to elk, and we would observe how it would affect the predator vs prey population. As expected, the hostile relationship will be a predation rate of wolves onto elk. This is an inductive situation, for there are rare cases for this event to occur. However, this simulation would showcase the behavior of this dire situation.

### **2.3 Relationship Between Virus-Environment-Prey-Predator**

We also wanted to dictate if environmental factors affect variable rates of growth in the age group population of both Elk and Wolf. More specifically, we wanted to see if temperature affects both virus and the population. We initially thought the temperature had an influence on the correlation between population growth and virus.

### 3 Simplifications

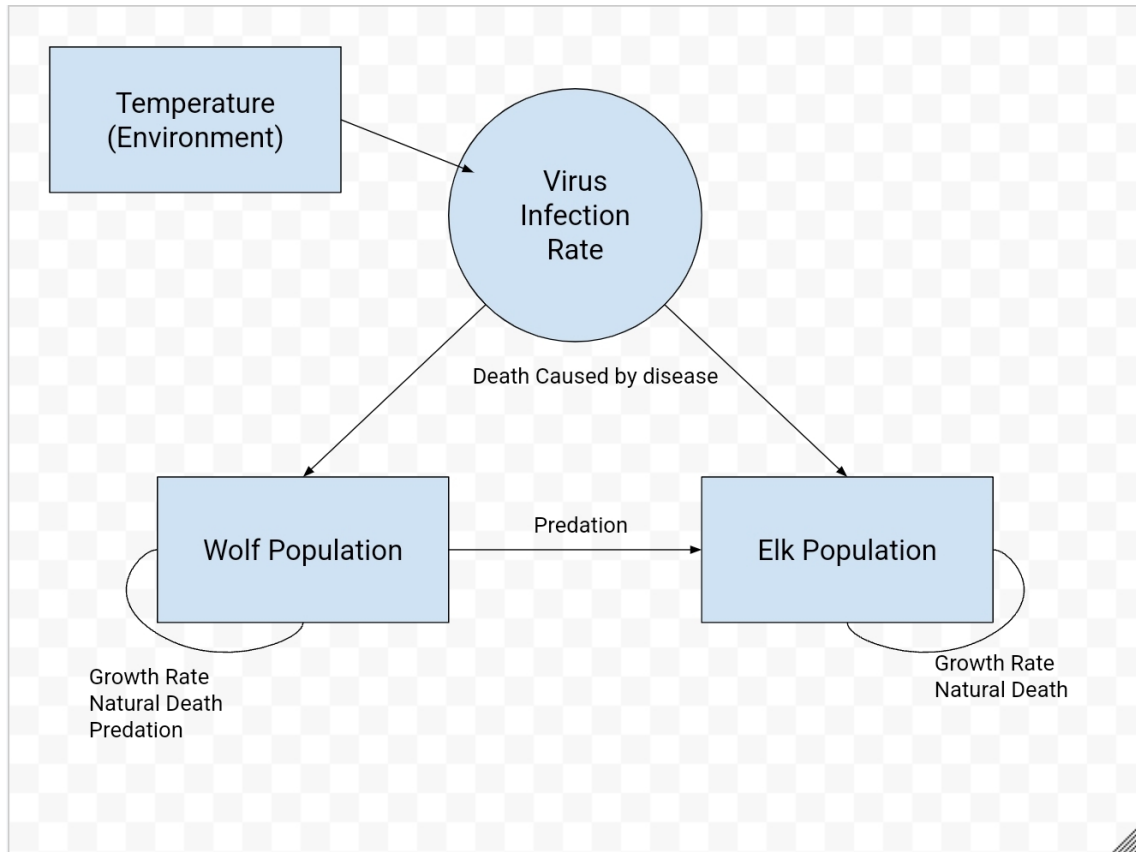


Figure 1: Simplification Design of the problem

We have divided our problem into two Dependent variables:

1. **Wolf Population**
2. **Elk Population**

With five Independent variables:

1. **Natural Growth Rate**
2. **Natural Death Rate**
3. **Temperature**
4. **Disease Infection Rate-Death Rate (proportional to each other)**
5. **Encounter Rate (Wolf-Wolf, Wolf-Elk predation)**

### 3.1 Assumptions

For our mathematical model, we will assume that:

- 1. Herd immunity has a negatively proportional decay (Refer to Section 4.3.1).**
  - 1a. In a limited time cycle, herd immunity can be represented by a constant decrease in efficiency of the effectiveness of the disease.
- 2. Elk and other wolves are the only victims of wolves.**
  - 2a. According to the data, wolves fight each other over food, resulting decrease in wolf population. Also, we only take elk as primary prey, while real-life data contains moose and bison as well.
- 3. The initial infection rate of a population is based on COVID-19's average infection rate per population.**
  - 3a. We are assuming that our disease is infectious to both species, and are interested to see the results when the disease is effective as COVID-19.
- 4. The carrying capacity is the maximum population we will see in our data set.**
  - 4a. Since we limited our environment to Yellow Stone National Park, we used the data through 1995-2021 to determine the maximum capacity of the certain environment.
- 5. The elk, prey, population can exponentially and infinitely grow.**
  - 5a. The elk live in an environment that provides an abundant source of food.
- 6. The death rate is constant with respect to the infected population.**
  - 6a. Since wild animals do not have vaccines, medications, and social distancing, we set death rate based on COVID-19 and Flu research.
- 7. Both wolves and elks are species capable of reproducing a strong Allee effect (Refer to Section 4.3.2).**
- 8. Weather can be decomposed into factors that can stochastically affect viral rates (Refer to Section 4.4.1).**

## 4 Development of Mathematical Model and Solution

### 4.1 Developing from the Simple Predator-Prey Model

The following models are derived from this source: [1]

The initial mathematical model reference was the basic predator-prey model:

$$\begin{aligned}\frac{dW}{dt} &= \alpha WE - \beta W \\ \frac{dE}{dt} &= \delta E - \gamma WE\end{aligned}$$

where  $\alpha WE$  is the growth from the predation of wolves on elk,  $\beta W$  is the natural death of wolves (Note: this includes all death sources such as intra-specific, inter-specific, human-sourced, and natural death),  $\delta E$  is the natural growth rate of elk (assuming elk have unlimited abundant resources), and  $\gamma WE$  is the death rate from wolf and elk encounters

In order to introduce complex variables into this simple predator-prey model, strongly correlated variables (such as intraspecific mortality rate, as it can be seen later in the paper) needed to be included in the formula (Refer to Figure 6). And so, we needed to decide on what formula to include in the model.

Based on the well-known SIR model, our initial equations for the growth rate of wolf and elk are the following:

$$\begin{aligned}\frac{dW}{dt} &= \alpha_1 \beta WE \left(1 - \frac{W}{K}\right) - \sigma \beta WE \\ \frac{dE}{dt} &= \alpha_2 E \left(1 - \frac{E}{K}\right) - \beta WE\end{aligned}$$

where:

- $\alpha_i$  are the growth rates of wolf and elk respectively
- The growth rate of wolves was expected to be based on a  $\beta$  (also known as the *encounter* rate) based on each wolf and elk
- On both differential equations, we included a logistic expression to limit the growth of each population



**Note:** the equations do not consider death rate yet

## 4.2 Virus Infection and Death Rate

With this basis of population growth, we can now add death rates to both populations that are based on the virus. Initially, we simplified the death rate to be linear, meaning:

$$D_W = r_1 I_W$$

$$D_E = r_2 I_E$$

The infection rate model that we developed was:

$$\begin{aligned}\frac{dI_W}{dt} &= \Phi_W(W - I_W)I_W\left(1 - \frac{t}{T_{h1}}\right) + (\Gamma_W\beta W E) \\ \frac{dI_E}{dt} &= \Phi_E(E - I_E)I_E\left(1 - \frac{t}{T_{h2}}\right) + (\Gamma_E\beta W E)\end{aligned}$$

where:

- $\Phi$  is the respective initial rate of infection for the disease
- $(W - I_W)$  is the representation for the uninfected wolves (following the format of the SIR model)
- $(1 - \frac{t}{T_{hi}})$  is the logistic function for the decaying effectiveness of the infection rate due to herd immunity

## 4.3 Modifying the Population Differential Equation

### 4.3.1 Herd Immunity Simplification

Due to research in [2], [3], and [4], herd immunity is a complicated variable to calculate. It includes many factors, such as surrounding species that are susceptible to the disease, the migration patterns of the susceptible and infected individuals, the innate treatment of how species treat their sick, and more. In short, more research can be done to develop a more efficient representation of herd immunity of a specific species.

Thus, because of this complex issue, we simplified herd immunity to decrement the efficiency of virus transmission by a constant factor. In other words, the infection rate will contain the following expression:

$$rSI(1 - \frac{t}{T_h})$$

where  $r$  is the initial infection rate applied to the encounters of the susceptible and infected.

This expression would decline the effectiveness of the virus' transmission.

#### 4.3.2 Appending the Allee Effect

Observing the pattern of the wolves and elk, it can be seen that the growth rate becomes abnormally high when the population count reaches a certain threshold. After reading certain research such as [2] and [4], the effect that is seen is called the *Allee Effect* (Refer to [5] and [6]).

In summary, the Allee effect is described as the magnitude of the sensitivity to how a species can "bounce" back from a depreciating population rate. In other words, if the population rate is going down, the species has a strong Allee effect if the population explodes in its proliferation rate. On the other hand, if the population has a weaker Allee effect, then the population would not grow effectively if the population numbers decrease.

Factors to determining the Allee effect include but are not limited to:

- feeding habits
- breeding patterns
- premature development/offspring nurturing behaviors
- hunting behaviors
- group/solo lifestyle behaviors

The Allee effect exists because some species with the previously listed criteria traits are able to group together at a higher density if the general population number goes below a certain number. By having species tending to higher densities when underpopulated, the birth rate can increase dramatically due to closer spatial living.

When a population is labeled to have a weak Allee effect, this means the species tends to drift to a lower density. This behavior makes it difficult for individuals to find suitable mates, decreasing the population's birth rate.

Based on the previously listed criteria, it can be assumed that both elk and wolves have a strong Allee effect.

This fact aids in creating the modified model because it allows to prevent the elk from growing too high and too low since it is an assumption that elk have an infinite source of food. In short, the Allee effect applies a lower and upper boundary to population growth. The strong Allee effect expression is of the following format:

$$\frac{dN}{dt} = rN(1 - \frac{N}{K})(\frac{N}{A} - 1) \quad (\text{strong})$$

$$\frac{dN}{dt} = \frac{r}{K}N^2(1 - \frac{N}{K}) \quad (\text{weak})$$

$$\frac{dN}{dt} = rN(1 - \frac{N}{K})(\frac{N - A}{K}) \quad (\text{flex})$$

where  $N$  is the population,  $K$  is the carrying capacity, and  $A$  is the threshold for the Allee effect (i.e. the lower bound).

### 4.3.3 New Model

After applying the herd immunity simplification and Allee effect, alongside other strongly correlated variables (i.e. intraspecific mortality), the following system of differential equations is describing the rate of change for the wolf and elk population:

$$\begin{aligned}\frac{dW}{dt} &= \alpha_1 \beta W E \left(1 - \frac{W}{200}\right) \left(\frac{W}{200} - \frac{1}{10}\right) - \sigma \gamma W^2 - D_W \\ \frac{dE}{dt} &= \alpha_2 E \left(1 - \frac{E}{16000}\right) \left(\frac{E}{16000} - \frac{39}{160}\right) - \beta W E - D_E\end{aligned}$$

where:

- $\alpha_i$  are the respective growth rates of wolves and elks (Note: wolf growth rate is based on the encounter rate of wolves and elk because kill rate has a correlation to birth rate)
- $\beta$  is the encounter/kill rate with respect to each wolf encountering an elk
- $\sigma$  is the probability of intraspecific death from each wolf-to-wolf encounter
- $\gamma$  is the encounter rate of a wolf to physically contact another wolf

We assumed that the carrying capacities and lower boundaries are close to the minimum and maximum data points in the wolf and elk population data sets. So, we let  $K_W = 200$ ,  $K_E = 16000$ ,  $A_W = 20$ ,  $A_E = 3900$

## 4.4 Inconclusive Results

The issues with this equation was that the resulting graph was not close to our given data set. Even through testing different parameters, we could not approach fitting this graph with a trial-and-error approach.

And so, we concluded that to solve for appropriate parameters in our given differential equations, we need to utilize a least squares method to solve for the best-fitting line.

#### **4.4.1 Inseparable Weather Factors**

In addition to the population graph being unable to reproduce accurate results, further research (Refer to [7] and [8]) showcases that there are strong correlations of weather variables to viral transmission. However, the research tells that there are too many interacting variables to isolate one variables sole affect on probability (Refer to Figure 7). It can be determined (through a separate side-project) on what that specific probability is for weather to stochastically affect viral transmission. But, for the scope of this research, there were too many variables to consider to conclude a "rate" or "single-value" for each weather variable on virus transmission.

#### **4.4.2 Indeterminable Population Parameters**

Moreover, referring to Section 4.4.1, the developed equations on the growth of the two populations contained many parameters that were difficult to solve for based on the given data. This was due to the fact that the data was too vague, lacked consistency, or did not show strong correlations to the desired parameter. Other methods could have been utilized to solve for the complicated differential equations. However, there was not enough time to learn the appropriate methods to solve for the multiple parameters based on the vague data set that was given.

### **4.5 Referring to Lotka-Volterra and Another Possible Equation**

In hopes of finding a more practical solution to our objective, we referred to the well-known model for predator-prey relations: the Lotka-Volterra model. The following system of equations is the general format of the model:

$$\begin{aligned}\frac{dx}{dt} &= \alpha x - \beta xy \\ \frac{dy}{dt} &= \delta xy - \gamma y\end{aligned}$$

The properties of the Lotka-Volterra model can be seen in Figures 5 and 4.

Based on this model, we could figure out how to apply the Allee effect and possible correlated variables (such as intraspecific rate) into our equation.

Based on the research of [9] and [10], we could utilize a ratio dependent model alongside an Allee effect to produce another equation that showcases possible relationships between elk and wolves, as follows:

$$\begin{aligned}\frac{dE}{dt} &= \alpha_1 \left(1 - \frac{E}{K}\right) E - \alpha_2 W E - \alpha_3 E^2 \\ \frac{dW}{dt} &= \sigma \alpha_2 W E - \alpha_4 \left(\frac{W}{E}\right)\end{aligned}$$

where:

- $\alpha_1$  is the elk growth rate with a logistic function appended for the sake of consideration of carrying capacity
- $\alpha_2$  is the predation rate which decreases the elk population
- $\alpha_3$  is the rate of natural birth rate decelerator
- $\sigma$  is the growth rate of wolf from the predation of elk
- $\alpha_4$  is the natural death rate of wolf (Note: wolves receive less food the more wolves exist in the population)

**Note:** deaths from a viral infection was not appended because most effort was designated toward developing a realistic population growth.

However, the pattern was still not accurate to the data set.

Another minor modification to the equation allowed us to derive the following system of equations:

$$\begin{aligned}\frac{dE}{dt} &= \alpha_1 E - \alpha_2 W E - \alpha_3 E^2 \\ \frac{dW}{dt} &= \sigma \alpha_2 W E - \alpha_4 W\end{aligned}$$

where the logistic expression has been removed for there existed a limiting function already (i.e.  $\alpha_3 E^2$ ), and the natural death on wolves has been changed to just depend solely on wolves.

#### 4.6 Final Thoughts and Most Recently Modified Equation

Our final resort to developing another equation that would showcase some connection between the two populations (excluding any viral transmission effects) would be the following system:

$$\begin{aligned}\frac{dE}{dt} &= \alpha_1 \left(1 - \frac{E}{19000}\right) E - \alpha_2 \beta W E \left(\frac{E}{3915} - 1\right) - D_E E \\ \frac{dW}{dt} &= \alpha_3 \beta W E \left(1 - \frac{W}{170}\right) - D_N W - \left(1 + \frac{W}{170}\right) \sigma W\end{aligned}$$

where:

- $\alpha_1$  is the growth rate of the elk
- $\alpha_2$  is the predation rate
- $D$  is the natural death rate (excluding any intraspecific rate)
- $\alpha_3$  is the birth rate of the wolves' predation on elk
- $\left(1 + \frac{W}{170}\right) \sigma W$  is the expression that tracks the growth of intraspecific mortality among the wolves based on population size

Once again, most of the parameters of these equations would be solved via any numerical method such as the least squared error process. However, due to the time restraint and the wide scope of this project, there was not enough allotted time to apply a solvable method.



#### 4.6.1 Summary of Derived Equations

$$rSI(1 - \frac{t}{T_h}) \quad (1)$$

$$D_W = r_1 I_W \quad (2)$$

$$D_E = r_2 I_E \quad (3)$$

$$\frac{dW}{dt} = \alpha_1 \beta W E (1 - \frac{W}{K}) - \sigma \beta W E \quad (4)$$

$$\frac{dE}{dt} = \alpha_2 E (1 - \frac{E}{K}) - \beta W E \quad (5)$$

$$\frac{dI_W}{dt} = \Phi_W (W - I_W) I_W (1 - \frac{t}{T_{h1}}) + (\Gamma_W \beta W E) \quad (6)$$

$$\frac{dI_E}{dt} = \Phi_E (E - I_E) I_E (1 - \frac{t}{T_{h2}}) + (\Gamma_E \beta W E) \quad (7)$$

$$\frac{dW}{dt} = \alpha_1 \beta W E (1 - \frac{W}{200}) (\frac{W}{200} - \frac{1}{10}) - \sigma \gamma W^2 - D_W \quad (8)$$

$$\frac{dE}{dt} = \alpha_2 E (1 - \frac{E}{16000}) (\frac{E}{16000} - \frac{39}{160}) - \beta W E - D_E \quad (9)$$

$$\frac{dE}{dt} = \alpha_1 (1 - \frac{E}{K}) E - \alpha_2 W E - \alpha_3 E^2 \quad (10)$$

$$\frac{dW}{dt} = \sigma \alpha_2 W E - \alpha_3 (\frac{W}{E}) \quad (11)$$

$$\frac{dE}{dt} = \alpha_1 E - \alpha_2 W E - \alpha_3 E^2 \quad (12)$$

$$\frac{dW}{dt} = \sigma \alpha_2 W E - \alpha_4 W \quad (13)$$

$$\frac{dE}{dt} = \alpha_1 (1 - \frac{E}{19000}) E - \alpha_2 \beta W E (\frac{E}{3915} - 1) - D_E E \quad (14)$$

$$\frac{dW}{dt} = \alpha_3 \beta W E (1 - \frac{W}{170}) - D_N W - (1 + \frac{W}{170}) \sigma W \quad (15)$$

$$(16)$$

## 5 Results

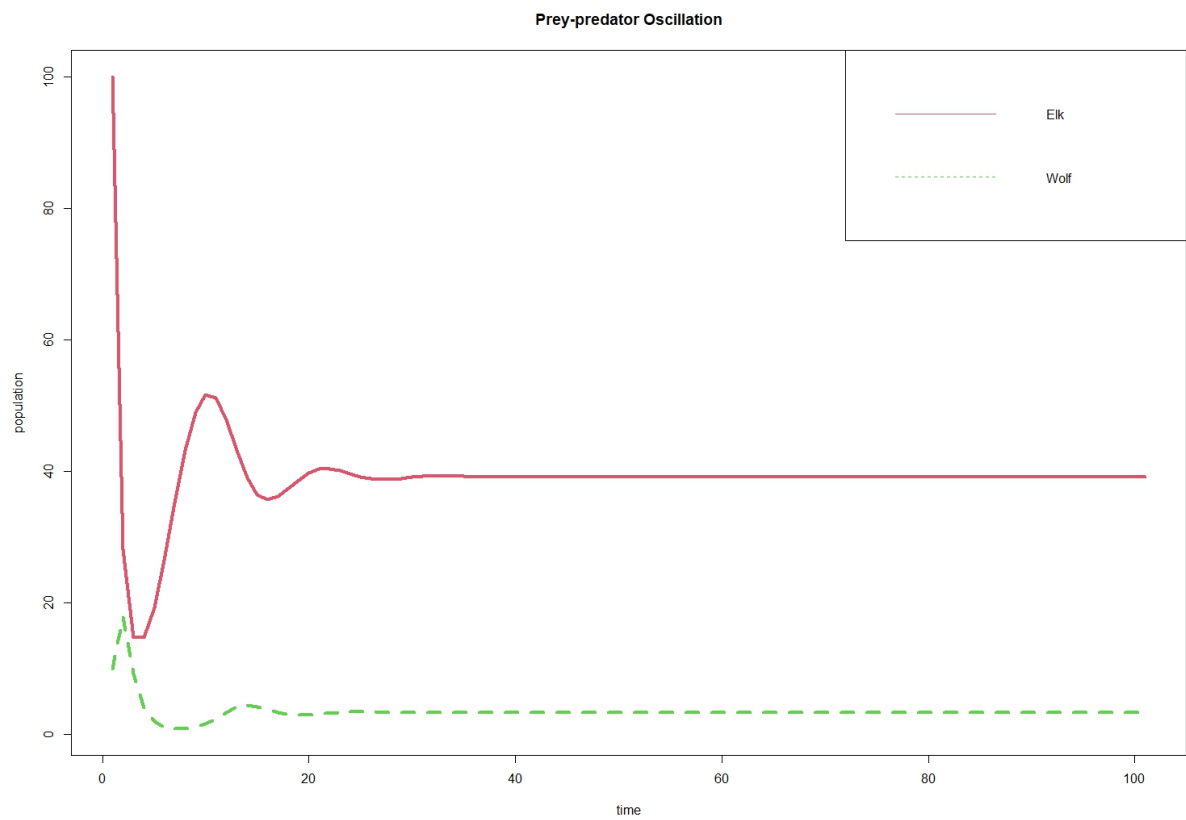


Figure 2: Describes the normal oscillation based on the simple Predator-Prey model initialized with random parameters.

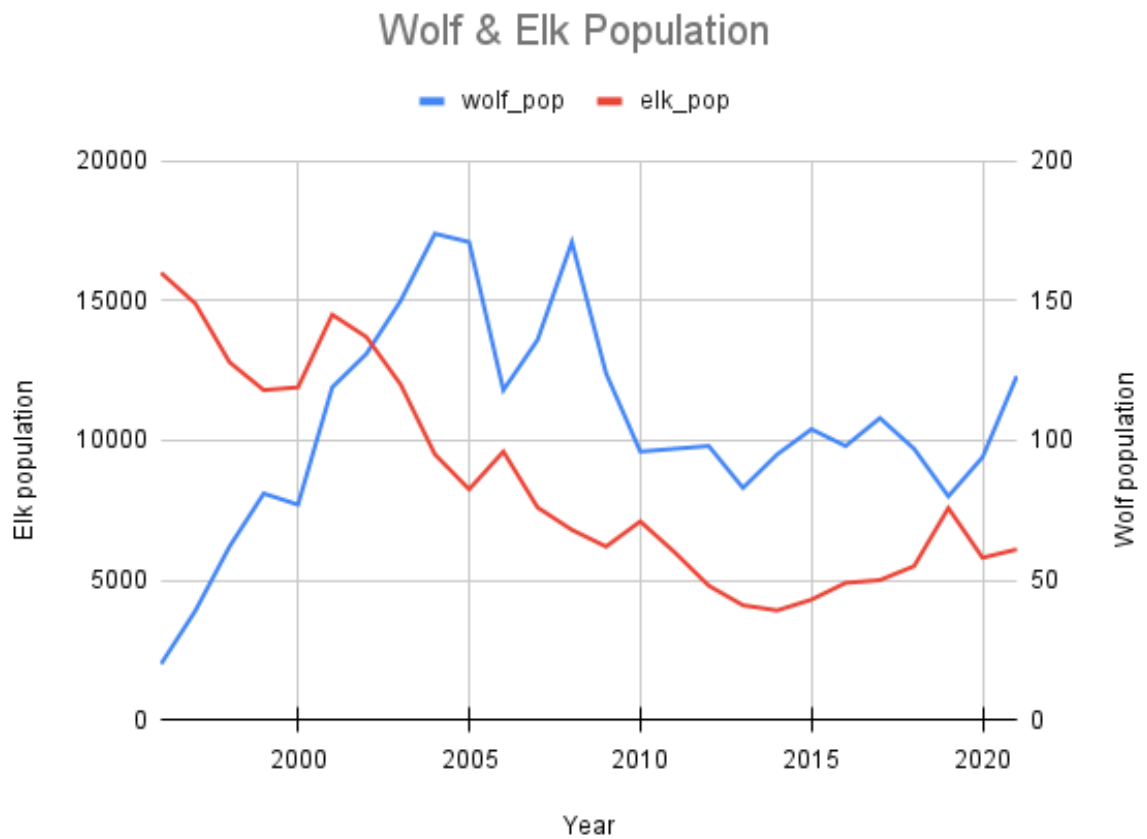


Figure 3: Displays the population of wolf and elk over time. Note: Take caution to refer to the correct respective y-axis for wolf/elk values.

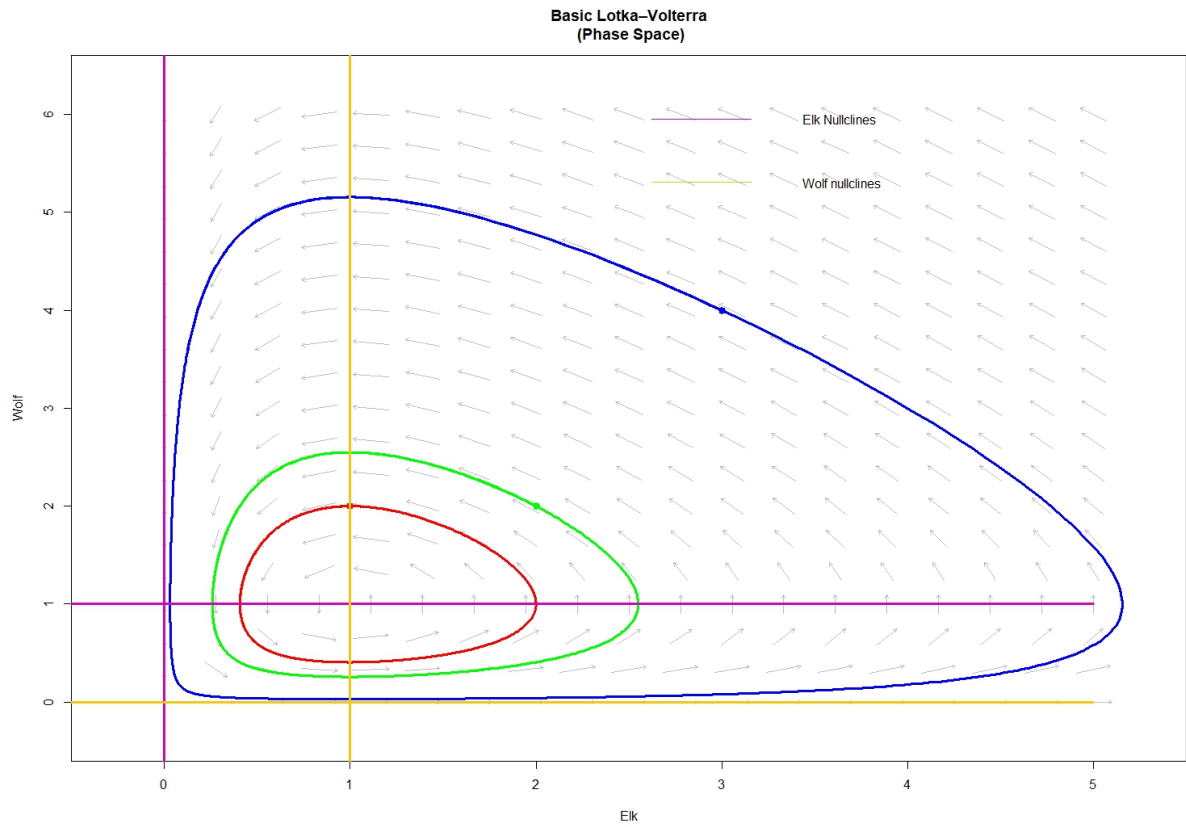


Figure 4: Displays the phase plane of the Lotka-Volterra predator-prey model, displaying how the population of both species changes over time; the different colored lines are different initial conditions.

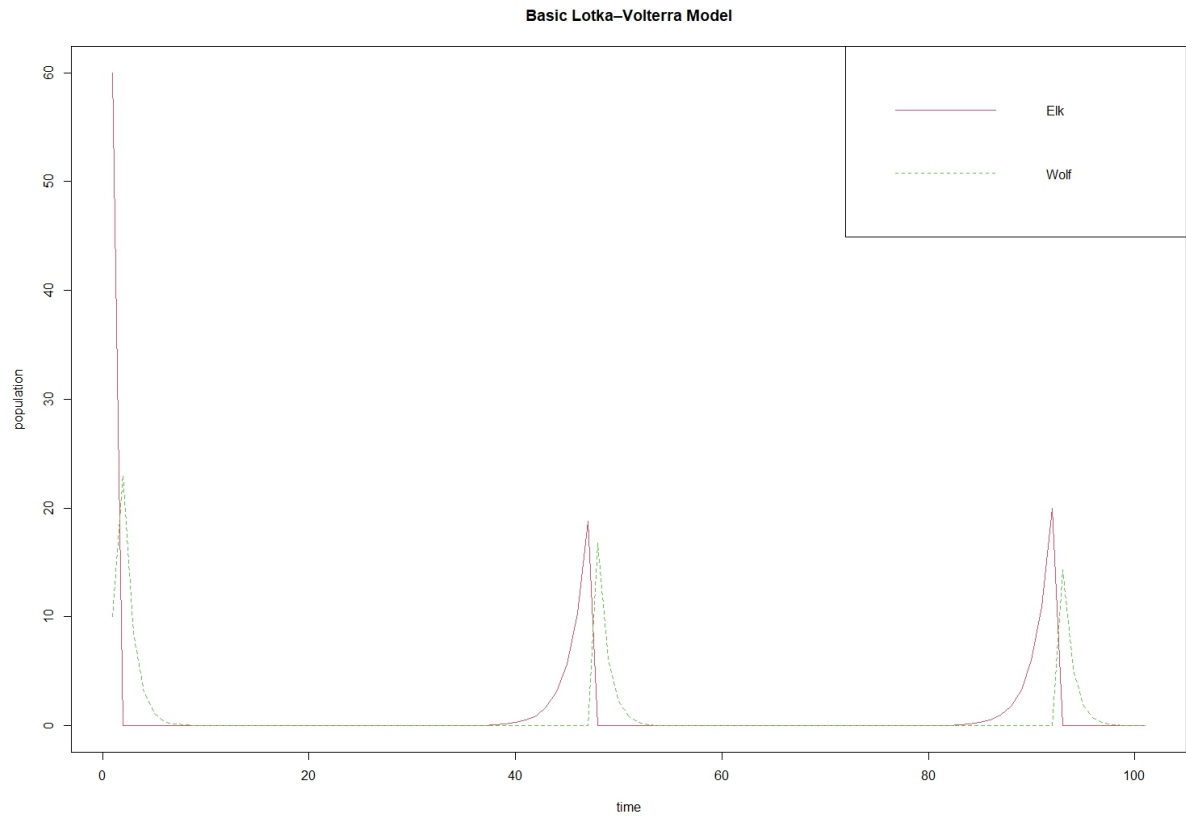


Figure 5: Displays the linear representation of prey and predator population values over time based on the Lotka-Volterra model; this model has been initialized with random parameters.

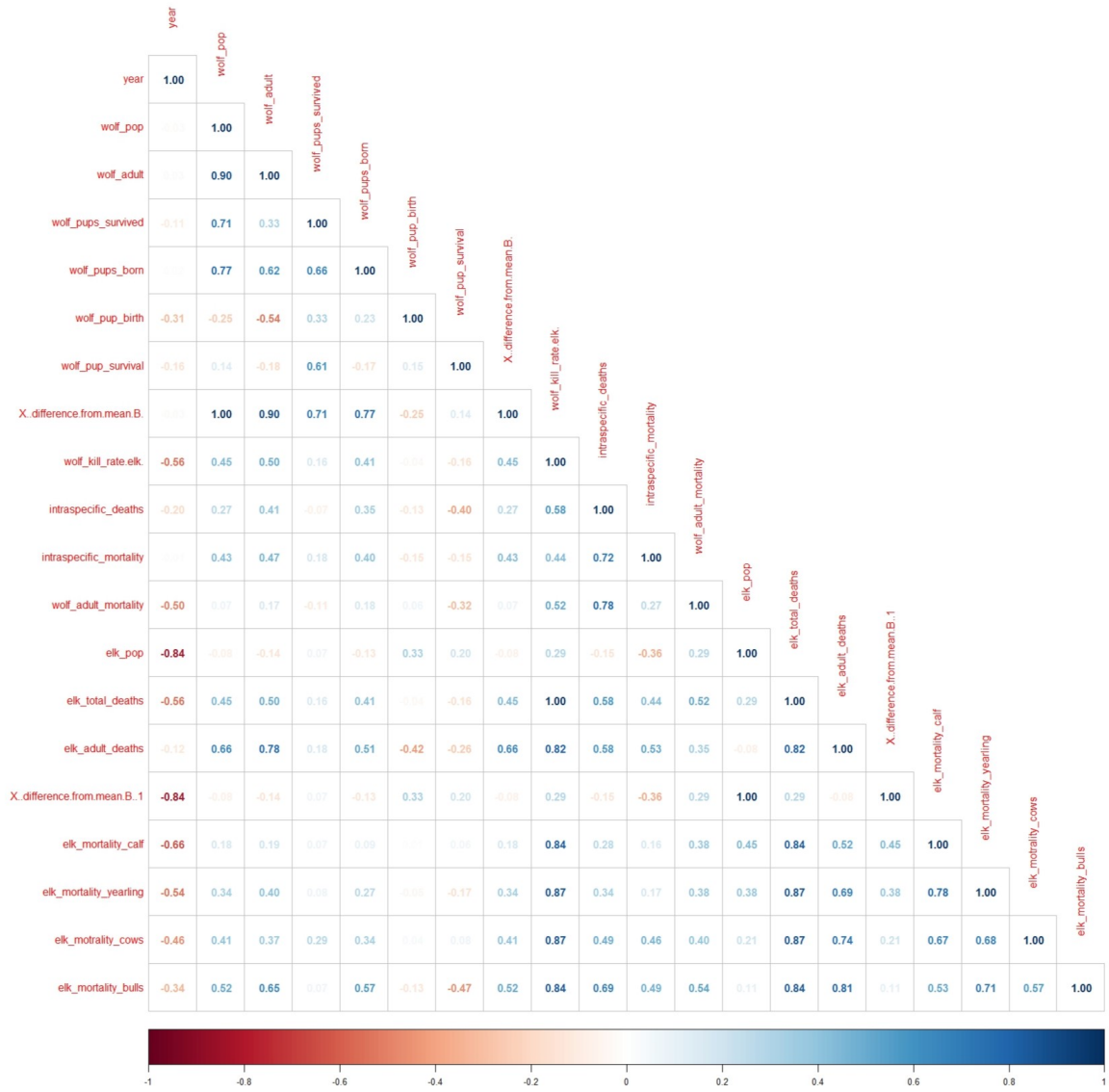


Figure 6: Displays the correlation coefficients of all data points that were collected from the wolf and elk data sets; the colors correspond to the strength of either negative or positive correlations.

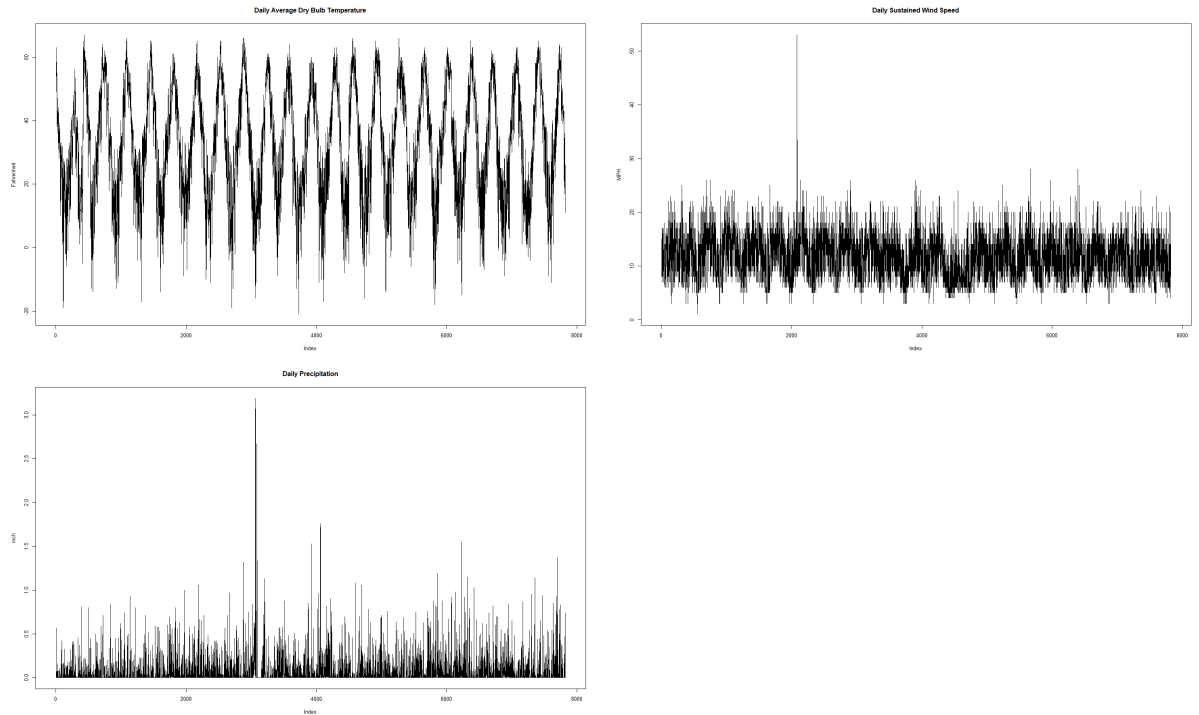


Figure 7: Showcases the visualization of the data from temperature, wind speed, and precipitation respectively.

## 6 Improvements

According to the data set we referenced, we noticed that the hunting rate of wolf on bison gradually increased due to a decrease in the elk population, so we have to take into consideration of other prey in the model.

Our current plan includes many changes to our initial model. First, we changed the Leslie matrix into an ordinary differential equation (ODE) for the entire population. Then, we wanted to determine specific parameters of our ODE and stochastically change the variables within the population differential equations with Markov chains. However, we found that there were too many factors affecting population change and the virus. So, we had to adjust and make the population variable independent of both the weather variable and the virus.

Although we have already made numerous revisions to our initial model, further research on the Lotka Volterra equation, the Allee effect, and the Least Squares Error Method would have improved the model greatly. We did not know how to correctly utilize the Lotka Volterra equation with the Allee effect, so more research would have increased our ability to accurately represent the data. In addition, if we had further research on the Least Squares Error Method, it would have been much easier to find parameters for the model's differential equations. Separately, it would have been an even stronger project if we were to have a subproject within this project to have a more varied and accurate representation of herd immunity.

### 6.1 Limitations

We initially planned on formulating a realistic environment via an environment-affecting population model by using Markov chains to stochastically affect the rates of change in different age groups. The age groups would then be placed into the Leslie matrix to be applied to the population vector. This data-driven model would have used the population data of wolves, elk, and possibly other prey within that allotted time. However, many things occurred that obscured our initial plan:

1. Too many factors were affecting the variables.
2. There was a lack of data for some of the variables such as the age group rates.
3. There was inconsistent data gathering as the time steps between each data set were different. For example, elk data were taken every 1-2 years and the wolf data was inconsistently taken every year.



## 7 Conclusions

We were unsuccessful in regards to making a practical analytical solution. We have an inconclusive result that we can develop and enhance with more time and practical error.

The final Lotka-Volterra with a modified Allee effect for both wolves and elks can be analyzed via phase-plane analysis. Doing so will conclude that the equations for both wolves and elk result in a pattern of a spiral sink. Meaning, the eigenvalues of the equations would be negative and have a complex conjugate.

However, for us to calculate the parameters of this equation, we would need to utilize the least squares error method or a differential equation method that we have yet to discover. And so, this experiment resulted in an inconclusive outcome.

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## 9 Appendix

### 9.1 R Code for Data Cleaning

```
# cleaning function
clean <- function (x) {
  x <- x[,-(3:21)]#1
  x <- x[,-(4:7)]#2
  x <- x[, -c(5,7,10)]#3
  x <- x[, -c(10,12,14)]#4
  x <- x[,-(12:95)]#5

  index <- numeric(1)
  for (i in seq_len(nrow(x))) {
    if (x[i,3] == "") {
      index <- c(index, i)
    }
  }
  x <- x[-c(index),]#6

  for (i in seq_len(ncol(x))) {
    x[[i]] <- gsub(pattern = "s", replacement = "", x[[i]]) #7
  }

  x<-x
}

# NOAA_one (2013-2022)
NOAA_one <- read.csv("NOAA_one.csv", fill = TRUE)
NOAA_one <- clean(NOAA_one)
write.csv(NOAA_one, "NOAA2013_2022.csv")

# NOAA_two (2003-2012)
```

```

NOAA_two <- read.csv("NOAA_two.csv", fill = TRUE)
NOAA_two <- clean(NOAA_two)
write.csv(NOAA_two, "NOAA2003_2012.csv")

# NOAA_three (1993-2002)
NOAA_three <- read.csv("NOAA_three.csv", fill = TRUE)
NOAA_three <- clean(NOAA_three)
write.csv(NOAA_three, "NOAA1993_2002.csv")

```

## 9.2 R Code for Environment plots

```

##### Environment #####
NOAA1993_2002 <- read.csv("NOAA1993_2002.csv", fill = TRUE)
NOAA2003_2012 <- read.csv("NOAA2003_2012.csv", fill = TRUE)
NOAA2013_2022 <- read.csv("NOAA2013_2022.csv", fill = TRUE)

# Reorganize Data
envdata <- rbind(NOAA1993_2002, NOAA2003_2012, NOAA2013_2022)

# Environment plots
par(mfrow = c(2,2))
plot(envdata$DailyAverageDryBulbTemperature, type = "l",
     main = "Daily Average Dry Bulb Temperature",
     ylab = "Fahrenheit")
plot(envdata$DailySustainedWindSpeed, type = "l",
     main = "Daily Sustained Wind Speed",
     ylab = "MPH")
plot(envdata$DailyPrecipitation, type = "l",
     main = "Daily Precipitation",
     ylab = "Inch")

```

## 9.3 R Code for Models

```

##### Prey_predator #####

```

```

pp_data <- read.csv("elk_wolf_data.csv")

#correlation plot 1
chart.Correlation(pp_data, pch = 20, cex.labels = 20)

#correlation plot 2
corr <- cor(pp_data, use = "complete.obs")
corrplot(corr, method = "number", type = 'lower', )

# 1.1 Basic Lotka{Volterra (linear)
rPred <- function(alpha, beta, delta, gamma){

  Pars <- c(alpha, beta, delta, gamma)
  ini_state <- c(x = 60, y = 10) #x is elk; y is wolf

  bas_lotka <- function (Time, State, Pars) {
    with(as.list(c(State, Pars)), {
      dx = alpha * x - beta * x* y
      dy = delta * x * y - gamma * y
      return(list(c(dx, dy)))
    })
  }

  Time <- seq(0, 100, by = 1) # Time Interval
  out <- as.data.frame(ode(func = bas_lotka, y = ini_state, parms = Pars, times = Time))

  par(mfrow = c(1,1))
  matplot(out[,-1], type = "l", xlab = "time", ylab = "population",
    col = c(2,3), lty = c(1,2),
    main = "Basic Lotka{Volterra Model")
  legend("topright", c("Elk", "Wolf"), lty = c(1,2), col = c(2,3), box.lwd = 0)

```

```

}

rPred(0.6,1.2,1, 1)

# 1.2 Basic Lotka{Volterra (Phase Space)
y0 <- matrix(c(1, 2, 2, 2, 3, 4), ncol = 2, nrow = 3, byrow = TRUE)
flowField(lotkaVolterra, xlim = c(0, 5), ylim = c(0, 6),
          parameters = c(1, 1, 1, 1), points = 19, add = FALSE,
          xlab = "Elk", ylab = "Wolf")
trajectory(lotkaVolterra, y0 = y0, tlim = c(0,10),
           parameters = c(1, 1, 1, 1), col = rainbow(3),
           system = "two.dim", lwd = 3, xlab = NULL, ylab = NULL)

# 2.1 Oscillation
rPred_osci <- function(a1, a2, a3, a4, sigma){

  Pars <- c(a1, a2, a3, a4, sigma)
  ini_state <- c(x = 100, y = 10) #x is elk; y is wolf

  pp_osci <- function (Time, State, Pars) {
    with(as.list(c(State, Pars)), {
      dx = a1 * x - a2 * (x^2) - a3 * x * y
      dy = sigma * a3 * x * y - a4 * y
      return(list(c(dx, dy)))
    })
  }

  Time <- seq(0, 100, by = 1) # Time Interval
  out <- as.data.frame(ode(func = pp_osci, y = ini_state, parms = Pars, times = Time))

  par(mfrow = c(1,1))

```

```

matplot(out[,-1], type = "l", xlab = "time", ylab = "population",
        col = c(2,3), lty = c(1,2), lwd = 4,
        main = "Prey-predator Oscillation")
legend("topright", c("Elk", "Wolf"), lty = c(1,2), col = c(2,3), box.lwd = 0)

}

rPred_osci(0.667, 0.01, 0.083, 1.3, 0.4)

# Final model
pp_final <- function(a1, a2, a3, a4, ratio, e_ndeath){

  Pars <- c(a1, a2, a3, a4, ratio, e_ndeath)
  ini_state <- c(x = 16000, y = 20) #x is elk; y is wolf

  pp_osci_cc <- function (Time, State, Pars) {
    with(as.list(c(State, Pars)), {
      dx = a1 * (1 - (x / 19000)) * x -
        (a2 * y) * (ratio * x) * ((x/3915) - 1) -
        e_ndeath * x
      #dx = elk growth - predation - natural death

      dy = (a3 * y) * (0.0004 * x) * (1-(y/170)) -
        0.067 * y -
        (1 + y/170)*0.223*y
      #dy = wolf growth - death other than intra - intra death
      return(list(c(dx, dy)))
    })
  }

  Time <- seq(0, 30, by = 1) # Time Interval

```



```

out <- as.data.frame(ode(func = pp_osci_cc, y = ini_state, parms = Pars, times = Time))

par(mfrow = c(1,1), mar = c(5, 4, 4, 4) + 0.3)
plot(out[, -c(1,3)], type = "l", xlab = "time", ylab = "Elk population",
      col = 2, lty = 1, lwd = 2,
      main = "Prey-predator (Carrying Capacity) ")
par(new = TRUE)
plot(out[, -c(1,2)], type = "l",
      col = 3, lty = 2, lwd = 2, axes = FALSE, xlab = "" , ylab = "")
axis(side = 4, at = pretty(range(out[, -c(1,2)])))
mtext("Wolf population", side = 4, line = 3)
legend("topright", c("Elk", "Wolf"), lty = c(1,2), col = c(2,3), bty = "n")

}

pp_final(0.045, 0.2, 0.5, 0.4, 0.0016, 0.032)

```