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Eco- Epidemiological Prey-Predator Model For Susceptible- Exposed-Infected-Recovered Species

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Abstract

Eco-epidemiological models are critical for understanding the intricate interplay between ecological and epidemiological processes in ecosystems. This study focuses on a prey-predator model that includes a prey species and the susceptible-exposed-infected-recovered (SEIR) epidemiological framework. Integrating these processes provides insight into how disease transmission affects population stability and predator-prey communications. We use mathematical and computational tools to analyze the model and find important thresholds and conditions that result in diverse environmental and epidemiological effects. Our findings emphasize the relevance of disease control and ecological conservation techniques in preserving ecosystem equilibrium.

Keywords:

Eco-epidemiological model, predator-prey dynamics, SEIR (Susceptible-Exposed-Infected-Recovered) model, disease spreading, population stability, mathematical modeling. Epidemiologic thresholds, Environmental protection, Lyapunov function.

Mathematics Subject Classification (MSC):

Epidemiology (92D30), Ecology (92D40), Stability (34D20), Dynamics in Biology (37N25), Medical Epidemiology (92C60), and Stability Theory (37C75).

1. Introduction

Eco-epidemiology, a subject that combines environmental and epidemiology, investigates how infectious illnesses interact with and are affected by the environment. Prey-predator models are important tools in ecological research, with a typical focus on the dynamics of predator populations and prey. However, adding epidemiological processes into these models introduces new complexity due to disease dynamics**.**

This study presents a model that combines prey-predator communications with a susceptible-exposed-infected-recovered (SEIR) framework to investigate these combined effects. In many ecosystems, prey species may encounter each other due to a variety of environmental and biological constraints, and when these species are susceptible to infectious diseases, the movement of the entire system can change significantly.
The SEIR model is a typical epidemiological framework that categorizes the population into four groups: susceptible (S), exposed (E), infected (I), and recovered. This model depicts how humans go through the stages of disease, from susceptibility to exposure, infection, and eventual recovery. Integrating this paradigm into prey-predator models enables a more accurate description of disease dynamics within environmental systems.

The aim of this process is to investigate the dynamics of a prey-predator model with limited prey species expansion, as well as the epidemiological structure of SEIR. We want to know how the transmission of disease affects population stability and predator-prey communications. Using mathematical and computational tools, we analyze the model to discover crucial thresholds and parameters that influence various ecological and epidemiological outcomes.

Our findings help to broaden our understanding of how disease affects ecological systems and provide suggestions for designing disease management techniques that also protect biodiversity. This study emphasizes the significance of incorporating both ecological and epidemiological elements when investigating natural populations and ecosystems.

2. MATHEMATICAL MODEL FORMATION

Consider using the susceptible-exposed-infected-recovered (SEIR) [17] paradigm to discuss the present pandemic. The SEIR model of disease Propagation [17] is predicated on three significant assumptions. At every given time t, the population (N) is classified into 4 classes: susceptible (S), exposed (E), infected (I), and recovered (R).[17]

So, we have

$$\mathrm{d}\,N(t) = \mathrm{d}S(t) + \mathrm{d}E(t) + \mathrm{d}I(t) + \mathrm{d}R(t)$$

$$\frac{dS}{dt} = \phi - \chi SI - \alpha S$$

$$\frac{dE}{dt} = \chi SI - (\alpha + m)E$$

$$\frac{dI}{dt} = \mathrm{mE} - (\alpha + \beta)I$$

$$\frac{dR}{dt} = \beta I - \alpha R \qquad \text{.....(2.1)}$$

With initial condition S (0) = S_0 >0, E (0) = E_0 >0, I (0) =I_0 >0, R(0) = R_0 >0 [18]

To formulate the mathematical model assumption, we make the following assumptions.

$\chi \longrightarrow$ effective contact rate

$\phi \longrightarrow$ Birth rate of Susceptible

$\alpha \longrightarrow$ mortality rate

m $\longrightarrow$ progression rate exposed to infected

$\beta \longrightarrow$ recovery rate

S$\longrightarrow$ Susceptible prey

E$\longrightarrow$ Exposed prey

I $\longrightarrow$ Infected prey

R$\longrightarrow$ Recovered predator

3. Positivity and boundedness of solutions

Theorem 1

All factors are non-negative for everyone t >0, the closed region [17]

$$\Omega = \left\{ (S, E, I, R) \epsilon R^4 : 0 < N < \frac{\phi}{\alpha} \right\}$$

Is positively invariant for the entire system (2.1) proving from the equation , we obtain [17]

$$\frac{dS}{dt} = \phi - \chi SI - \alpha S \geq -(\chi I + \alpha)S$$

We have

$$S(t) \geq S\,(0) expt\,(-\int_0^t (\chi I + \alpha) dp) > 0$$

Now

$$\frac{dE}{dt} = \chi SI - (\alpha + m)E \geq -(\alpha + m)E$$

We have

$$S(t) \geq S\,(0) expt\,(-\int_0^t (\chi I + \alpha) dp) > 0$$

Now

$$\frac{dE}{dt} = \chi SI - (\alpha + m)E \geq -(\alpha + m)E$$

We have

$$E(t) \geq E\,(0) expt\,(-\int_0^t (\alpha + m)E\, dp) > 0$$

Also,

$$\frac{dI}{dt} = \text{m E-}(\alpha + \beta)I \geq -(\alpha + \beta)I$$

We have

$$\text{I(t)} \geq I\ (0)expt\ (-\int_0^t (\alpha + \beta)Idp) > 0$$

Also,

$$\frac{dR}{dt} = \beta I - \alpha R$$

We have

$$\text{R(t)} \geq R\ (0)expt\ (-\int_0^t \alpha\ dp) > 0$$

Again

$$\frac{d(S + E + I + R)}{dt} = \phi - \alpha(S + E + I + R)$$

Therefore,

$$\frac{dN}{dt} = \phi - \alpha N \text{.....(2.2)}$$

If $\phi - \alpha N$ <0 then $\frac{dN}{dt} < 0$

Therefore, expression (2.2) is limited by $\frac{\phi}{\alpha}$

After that, we obtain S, E, I, and R as positive functional.[18]

Basic reproductive number, disease-free steady state, and pandemic steady state.[18]

The asymptomatic point of equilibrium is locally asymptotically [17] stable. R_0<1 the disease disappears. The disease-free equilibrium point is unstable when R_0>1, i.e. The disease spreads throughout the population, resulting in a pandemic. Given that the model under consideration is disease-free, equilibrium [18] at($\frac{\phi}{\alpha}$,0,0,0), The fundamental reproductive number can be determined analytically.[18]

The fundamental reproduction number (R_0) the particular model can be purchased from the.

Lead eigenvalue of the matrix,[18] where:

$$\text{F=}\begin{bmatrix} \frac{\chi\phi}{\alpha} & 0 \\ 0 & 0 \end{bmatrix} \text{ and V=} \begin{bmatrix} 0 & m + \alpha \\ \beta + \alpha & -m \end{bmatrix}$$

Therefore, the reproduction number

$$R_0 = \frac{m\chi\phi}{\alpha(m+\alpha)(\beta+\alpha)} \text{.....(2.3)}$$

4. Stability Analyses

To establish the equilibrium positions, put the right-hand side of the equation in the system (2.1) to zero.[17]

$$\frac{ds}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

.

A system has two equilibrium[19] points: $E_0 = \left(\frac{\phi}{\alpha}, 0, 0, 0\right)$ for disease-free equilibrium and $E_1 = (S^*, E^*, I^*, R^*)$ for the Particular pandemic point[17]

Here,

$$S^* = \frac{\phi - (\alpha+m)E}{\alpha}$$

$$E^* = \frac{\{\alpha(\alpha+m)(\alpha+\beta)\}(R_0-1)}{\chi m(\alpha+m)}$$

$$I^* = \frac{mE}{(\alpha+\beta)}$$

$$R^* = \frac{\phi mE}{\alpha(\alpha+\beta)}$$

Equation (2.3) gives R_0 In the case of an pandemic, E* Exists only at that point

R_0 is greater than one.

Theorem 1.0.0:

The free of illness equilibrium in the system is locally unsteady if $R_0>1$ and stable if $R_0<1$. [17]

Proof:

From the expression (2.1), that we consider.

$$\phi - \chi SI - \alpha S = F_1$$

$$\chi SI - (\alpha + m)E = F_2$$

$$m\,E - (\alpha + \beta)I = F_3$$

$$\beta I - \alpha R = F_4$$

The Jacobian matrix is

$$J = \begin{bmatrix} -\chi I - \alpha & 0 & -\chi S & 0 \\ \chi I & -(\alpha + m) & \chi S & 0 \\ 0 & m & -(\alpha + \beta) & 0 \\ 0 & 0 & 0 & -\alpha \end{bmatrix}$$

At equilibrium point $E_0 = \left(\frac{\phi}{\alpha}, 0, 0, 0\right)$ *the Jacobian matrix becomes*

$$J(E_0) = \begin{bmatrix} -\alpha & 0 & \frac{-\chi\phi}{\alpha} & 0 \\ 0 & -(\alpha+m) & \frac{\chi\phi}{\alpha} & 0 \\ 0 & m & -(\alpha+\beta) & 0 \\ 0 & 0 & 0 & -\alpha \end{bmatrix}$$

Therefore, its characteristic equation is

$$\begin{vmatrix} -\alpha & 0 & \frac{-\chi\phi}{\alpha} & 0 \\ 0 & -(\alpha+m) & \frac{\chi\phi}{\alpha} & 0 \\ 0 & m & -(\alpha+\beta) & 0 \\ 0 & 0 & 0 & -\alpha \end{vmatrix} = 0$$

The characteristic roots are $-\alpha, -\alpha, -(\alpha+\beta) and\ (\alpha+m)(R_0-1)$.

The typical roots include $-\alpha, -\alpha, -(\alpha+\beta), (\alpha+m)(R_0-1)$.
The first 3 roots are negative, while the last one is negative. if $R_0 < 1$ and positive if $R_0 > 1$.
Hence, the equilibrium point E0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.[17]

Theorem 2.0:

If $R_0>1$, The state of equilibrium[17] E_1 is local asymptotically steady

Proof:

Let evaluate the expression as follows.

$$\phi - \chi SI - \alpha S = F_1$$

$$\chi SI - (\alpha+m)E = F_2$$

$$\text{m E-}(\alpha+\beta)I = F_3$$

$$\beta I - \alpha R = F_4$$

The Jacobian matrix [17] is

$$J = \begin{bmatrix} -\chi I - \alpha & 0 & -\chi S & 0 \\ \chi I & -(\alpha+m) & \chi S & 0 \\ 0 & m & -(\alpha+\beta) & 0 \\ 0 & 0 & 0 & -\alpha \end{bmatrix}$$

At the point of equilibrium point $E_1 = (S^*, E^*, I^*, R^*)$ the Jacobian matrix becomes[17]

$$J(E_1) = \begin{bmatrix} -\chi I* - \alpha & 0 & -\chi S* & 0 \\ \chi I* & -(\alpha+m) & \chi S* & 0 \\ 0 & m & -(\alpha+\beta) & 0 \\ 0 & 0 & 0 & -\alpha \end{bmatrix}$$

Therefore, its characteristic equation is

$$\begin{vmatrix} -\chi I* -\alpha - x & 0 & -\chi S* & 0 \\ \chi I* & -(\alpha+m)-x & \chi S* & 0 \\ 0 & m & -(\alpha+\beta)-x & 0 \\ 0 & 0 & 0 & -\alpha - x \end{vmatrix} = 0$$

(or) $(-\alpha - x)(x^3 + ax^2 + bx + c) = 0$

Where,

a= $\chi I* + 3\alpha + m + \beta$

b= $(\chi I^* + \alpha)(2\alpha + m + \beta) + (\alpha + m)(\alpha + \beta)$

c= $(\chi I^* + \alpha) * \alpha + m)(\alpha + \beta) - \alpha\beta m S*$

use Routh Hurwitz criterion,[17] the system (2.1) is locally asymptotically steady if a >0, b>0, ab>c.

Thus E_1 is a local asymptotically steady point of equilibrium [17].

Theorem 3.0

The free of disease equilibrium of the system (2.1) is globally asymptotically [17] steady if R_0<1.

Proof:

Consider the following linear Lyapunov function:

L= B_1E+B_2I

Using Lyapunov function derivative (where a dot signifies differentiating regarding time) [17]

$$\dot{L} = B_1\dot{E} + B_2\dot{I}$$

Substitute the equation for $\dot{E}\ and\ \dot{I}\ for$ (2.1) *we have* [17]

$$\frac{dL}{dt} = B_1[\chi SI - (\alpha + m)E] + B_2[m\,E - (\alpha + \beta)I] \text{ ……..(2.4)}$$

A small change from equation (2.4) to reproduction number (2.3) yields

$B_1=\alpha m$,$B_2 = \alpha(\alpha + m)$ … … … … … . (2.5)

Substituting the equation of B_1, B_2 obtained from equation[17] (2.5) has:

$$\frac{dL}{dt} = \chi SI\, m - (\alpha + \beta)\alpha(\alpha + m)I \doteq I$$

$$[\,\chi SI\, m - (\alpha + \beta)\alpha(\alpha + m)] = I$$

$$[\frac{(\alpha+\beta)\alpha(\alpha+m)}{(\alpha+\beta)\alpha(\alpha+m)}] - 1$$

Since S= $\frac{\phi}{\alpha} \leq N$, $its\ follows\ that$ [17]

$$\frac{dL}{dt} \leq I[\ (\alpha+\beta)\alpha(\alpha+m)]$$

$$\left[\frac{\chi\phi m}{(\alpha+\beta)\alpha(\alpha+m)}\right] - 1$$

$$\left[\frac{\chi\phi m}{(\alpha+\beta)\alpha(\alpha+m)} - 1\right]$$

$$\frac{dL}{dt} \leq I[(\alpha+\beta)\alpha(\alpha+m)][R_0 - 1]$$

Hence if $R_0 < 1$, then $\frac{dL}{dt} < 0$. According to Lasalle's extension of Lyapunov's principle, if the disease-free equilibrium point is globally stable asymptotically.[17]

Theorem 4.0:

If R_0 is greater than one, then the pandemic equilibrium E_1 is globally stable[17] asymptotically .

Proof:

With model (2.1) and $R_0 > 1$, the pandemic equilibrium [17] E_1 model exists.

We look at the following non-linear Lyapunov function [17] of the Goh Volterra type:

V=(S-S*-log $\frac{S}{S*}$) + $(E - E^* - \log\frac{E}{E*}) + Q(I - I^* - log\ \frac{I}{I*})$

Using the Lyapunov derivative [17]

$$\dot{V} = \left(S - \frac{S^*\dot{s}}{s}\right) + \left(E - \frac{E^*\dot{E}}{E}\right) + \left(I - \frac{I^*\dot{I}}{I}\right) \ldots\ldots\ldots (2.6)$$

Substituting the values of S˙, E˙, and I˙[17] from (2.1) into (2.4) yields

$\dot{V} = (\phi - \chi SI - \alpha S - \frac{S^*(\phi-\chi SI-\alpha S)}{S}$+((χSI − (α + m)E − $\frac{E^*(\chi SI-(\alpha+m)E}{E}$ + Q((m E − (α + β)I − $I\frac{*(m\ E-(\alpha+\beta)I}{I}$))……..(2.7)

At the constant state form equation (2.1) we have:

$$\phi = \chi S^* I^* - \alpha s^* \ldots\ldots\ldots\ldots\ldots (2.8)$$

Substituting equations[17] (2.8) into (2.7)

$\dot{V} = (\chi s^* I^* - \alpha s^* - \frac{\chi SI-\alpha S-S^*(\chi S^* I^*-\alpha S^*-\chi SI-\alpha S)}{S}$ + ((χSI − (α + m)E − $\frac{E^*(\chi SI-(\alpha+m)E}{E}$) +

(m E-(α + β)I − I ∗ ($\frac{m\ E-(\alpha+\beta)I}{I}$))…….(2.9)

Further simplification gives:

$\dot{V} = \left(\chi s^* I^* - \alpha S^* - \alpha S - \frac{S^*(\chi S^* I^*-\alpha S^*-\chi SI-\alpha S)}{S}\right)$ + ((−((α + m)E − $\frac{E^*(\chi SI-(\alpha+m)E}{E}$) +Q(m E-(α + β)I − I ∗ ($\frac{m\ E-(\alpha+\beta)I}{I}$)) … … … … … … … … (2.10)

Gathering all infected classes[17] without a single star (*) from (2.10) and equating to zero:

$S^*\chi I - (\alpha + m)E) + Q(m\,E - (\alpha + m)I = 0$……(2.11)

A small change of steady state [17] from (2.1) and (2.9) resulted into

Q= $\frac{s^*\chi}{(\alpha+\beta)}$, $(\alpha + m) = \frac{I^*S^*\chi}{E^*}$, $K = \frac{(\alpha+\beta)I^*}{E^*}$……(2.12)

Substituting [17] the expression from (2.12) into (2.10) gives

$\dot{V} = (\chi s^* I^* + \alpha s^* - \alpha S - \frac{S^*(\chi S^* I^* + \alpha S^* - \alpha S)}{S} + ((\frac{E^*\chi SI}{E} + I^* S^* \chi) +$

$(\frac{-I^*S^*E\chi I^*}{IE^*} + \chi S^* I^*)$……(2.13)

Lastly, since the arithmetic mean[17] is higher than the geometric mean, we obtain

(2-$\frac{S}{S^*} - \frac{S^*}{S}$) ≤ 0

. (3-$\frac{S^*}{S} - \frac{I^*E}{IE^*} - \frac{SE^*I}{E}$) ≤ 0

Therefore, $\dot{V} \leq 0\ for\ R_0 > 1$.

Therefore, V is a Lyapunov function by Lasalle's Invariance principle, and the pandemic equilibrium $E_1\ is$ globally stable asymptotically .

5. Numerical stimulation

In this phase, we have completed the numerical answers, they are just as vital as the analytical findings, to verify them. We provide simulations of possible solutions to the system's nonlinear differential equation.

To begin, define the system parameters as ρ_1 =($\phi = 0.1, m = 0.02, \beta = 0.01, \alpha = 0.03, \chi = 0.05$. Then the beginning condition Satisfied S(0)= 0, E(0)=0, I(0) = 0, R(0)=0 is a susceptible prey population with a periodic point of one.

i) If we take the system parameter as ρ_1. Then the initial condition satisfied S(0)= 0, E(0)=0, I(0) = 1, R(0)=0 is the infected prey population (see figure 1).

ii) Assume the system's parameter is ρ_1. Then the starting condition satisfied S(0)= 0, E(0)=1, I(0)=0, and R(0)=0 is the exposed prey population (see figure 2)

iii) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)=1, E(0)=0, I(0)=0, R(0)=0 is the susceptible prey population (see figure 3)

iv) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0, E(0)=0, I(0) = 0, R(0)=1) is the recovered predator population (see figure 4)

v) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0, E(0)=0, I(0) = 0, R(0)=0.5) is the recovered predator population at periodic.(see figure 5)

vi) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0, E(0)=0, I(0) = 0.5, R(0)=0) is the infected prey population at periodic (see figure 6,7,8)

vii) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0, E(0)=0.5, I(0) = 0, R(0)=0) is exposed prey population at periodic (see figure 9)

viii) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0.5, E(0)=0, I(0) = 0, R(0)=0) is the Susceptible prey population at periodic (see figure 10)

ix) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 5, E(0)=0, I(0) = 5, R(0)=0)[19] is the susceptible and infected prey population (see figure 11)

x) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 5, E(0)=5, I(0) = 0, R(0)=0) [19]is the susceptible and exposed prey population (see figure 12)

xi) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 5, E(0)=5, I(0) = 0, R(0)=0)[19] is the susceptible prey and recovered predator population (see figure 13)

xii) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0, E(0)=5, I(0) = 0, R(0)=5)[19] is the Exposed prey and recovered predator population (see figure 14)

xiii) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 0, E(0)=0, I(0) = 2, R(0)=1)[19] is the Infected prey and recovered predator population (see figure 15)

xiv) Assume the system's parameter is ρ_1. The starting condition satisfied S(0)= 1, E(0)=0, I(0) = 0, R(0)=0.5)[19] are the susceptible prey and recovered predator population (see figure 16)

6. Conclusion

The SEIR (Susceptible, Exposed, Infectious, and Recovered) model is an important epidemiological tool for analyzing infectious disease dynamics. Here are some crucial considerations to consider when concluding from the SEIR model. The SEIR model efficiently reflects the evolution of infectious diseases by including an exposed stage, which is critical for infections that require an incubation period. This makes it more realistic than simpler models like the SIR (susceptible, infectious, recovered) model. The SEIR model, which simulates the transmission of an infection over time, aids in the prediction of future outbreaks and the impact of interventions. It offers vital insights into public health planning and response tactics. The paradigm emphasizes the value of early detection and intervention.

Model simulations show that measures like quarantine, vaccination, and social separation can have a significant impact on the course of an pandemic. The SEIR model accuracy is strongly dependent on parameter estimates such as transmission rate, incubation period, and recovery rate. Small adjustments to these parameters can provide different results, emphasizing the importance of exact data. While the SEIR model provides a solid framework, it assumes population homogeneity and constant parameters, which may not accurately reflect real-world complexities. Model extensions, such as age-structured or spatial models, can help to overcome these restrictions. The SEIR model is a crucial epidemiological tool that helps us understand infectious illness patterns. Its ability to model diverse scenarios and inform public health decisions makes it crucial, but ongoing refinement and correct parameter estimation are required for maximum performance.

7. Figures

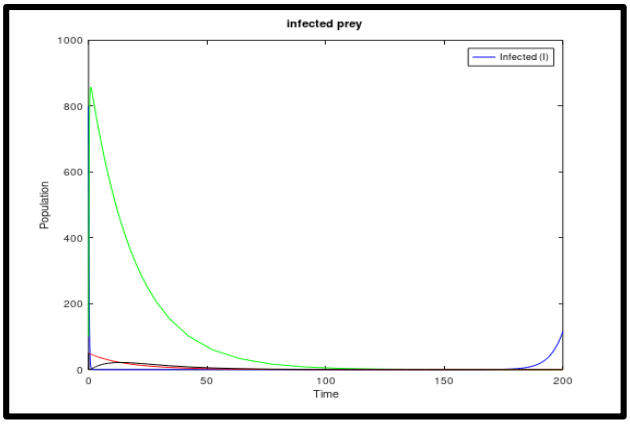

Figure 1 The Infected prey population

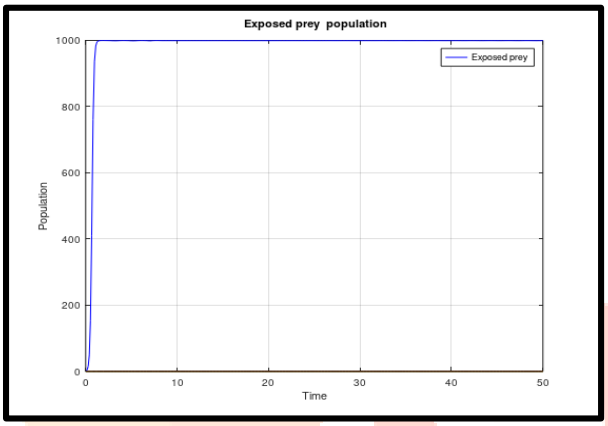

Figure 2 The Exposed prey population

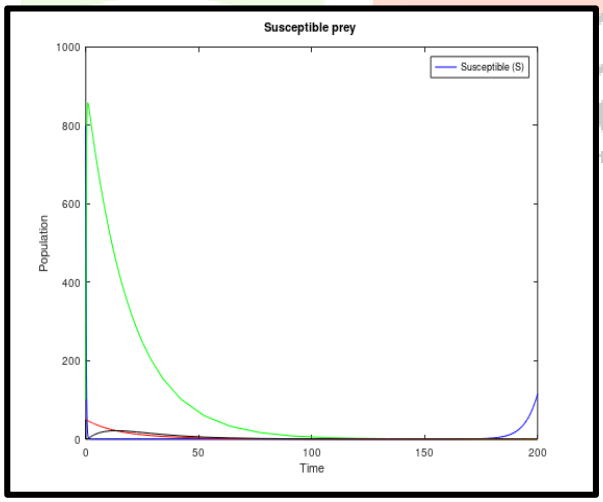

Figure 3 The susceptible prey population

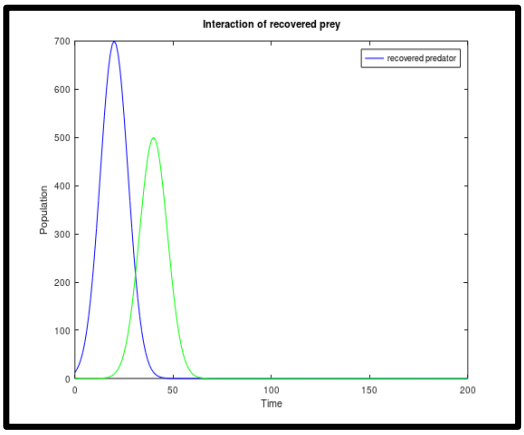

Figure 4 The Recovered predator population

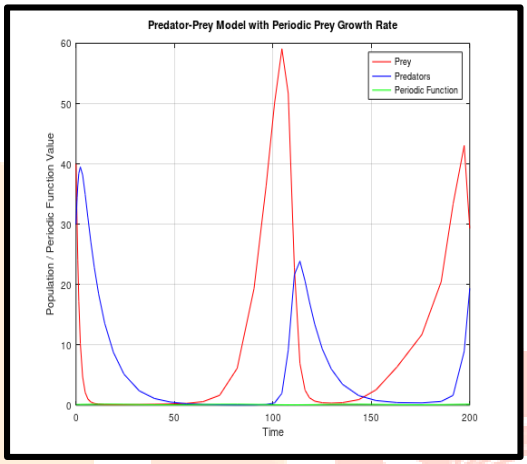

Figure 5 Infected prey at periodic

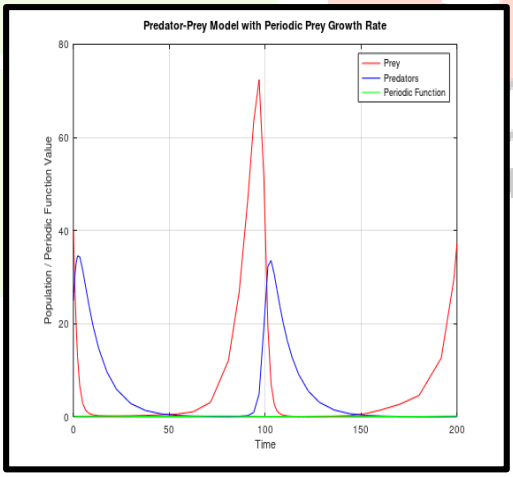

Figure 6 Exposed prey at periodic

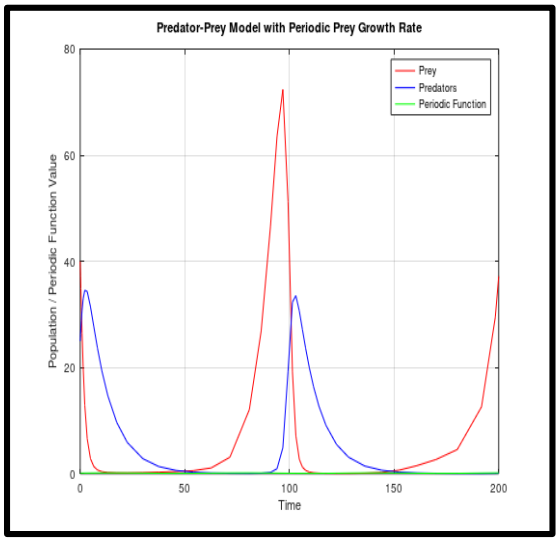

Figure 7 Susceptible prey at periodic

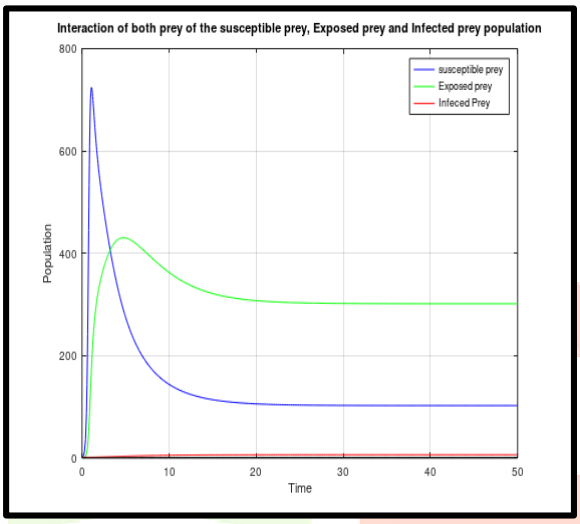

Figure 8 The communication of Susceptible prey and Infected Prey population

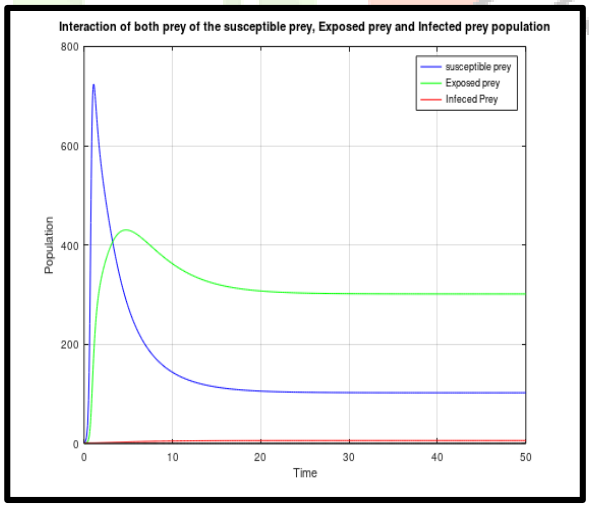

Figure 9 The communication of Susceptible prey, Exposed Prey and Infected Prey population

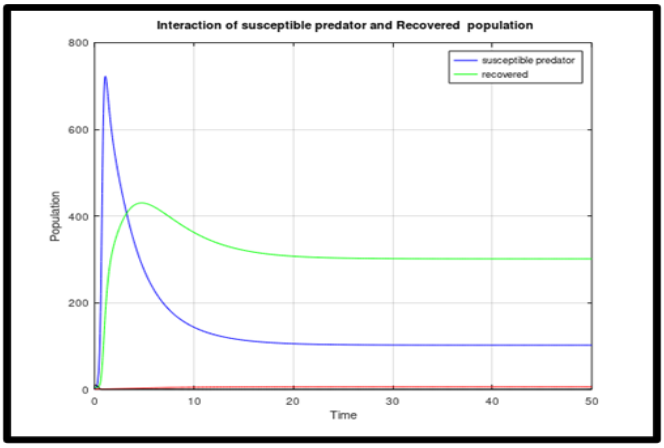

Figure 10 The communication of Susceptible predator and recovered Predator population

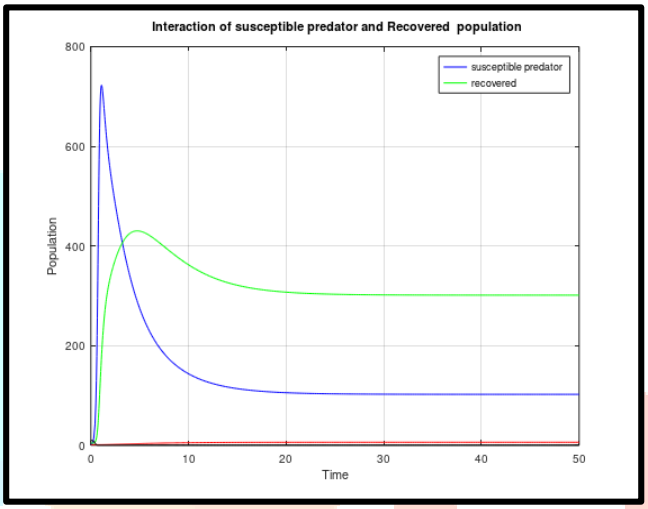

Figure 11 The communication of Susceptible prey and recovered predator population

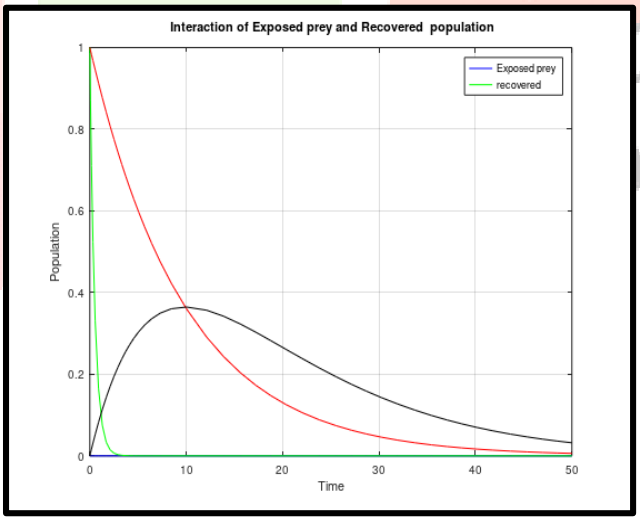

Figure 12 The communication of Exposed prey and recovered predator population

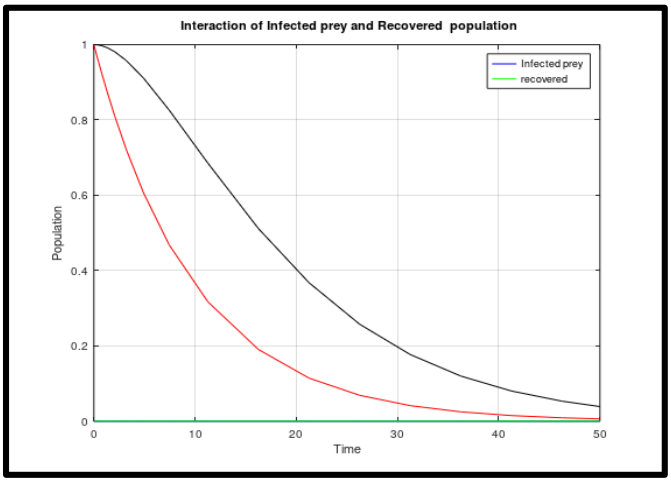

Figure 13 The communication of Infected prey and recovered predator population

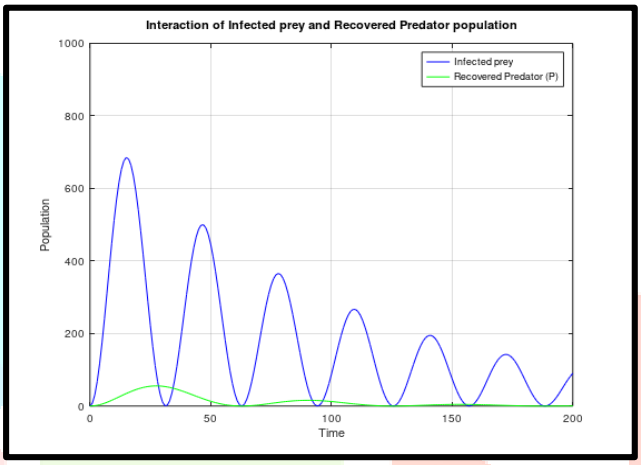

Figure 14 The communication of infected prey and recovered predator population

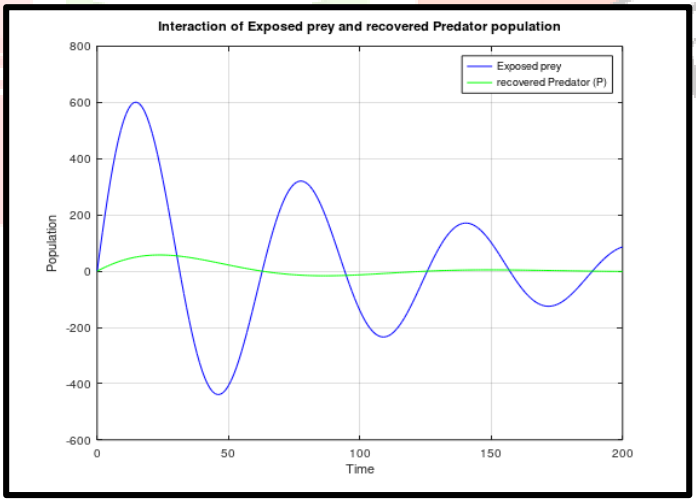

Figure 15 The communication of Exposed prey and recovered predator population

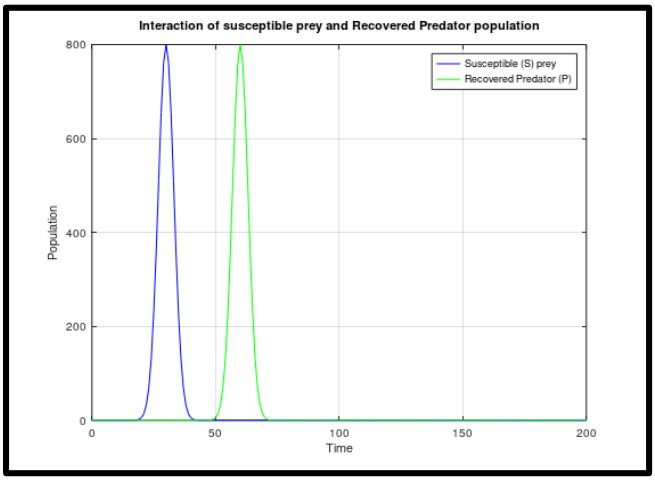

Figure 16 The communication of susceptible prey and recovered predator population

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9.Conflict of Interest

The author declares that there is no conflict of interest regarding the publication of this paper.

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