Mathematical Modeling of Aquatic Plant-Herbivore-Disease Dynamics with Stability Analysis and MATLAB Simulations Based on Herbivore Susceptibility Gene Knockout

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Abstract

This paper develops a mathematical model to describe the interaction among aquatic plants, herbivores, and plant disease, incorporating the effect of plant susceptibility to herbivory. Inspired by recent studies on herbivore susceptibility genes, this model uses a nonlinear system of differential equations to analyze population dynamics. Equilibrium points are identified and analyzed for local and global stability. Numerical simulations using MATLAB explore the impact of varying plant susceptibility on biomass, herbivore density, and disease spread. The results show that reducing susceptibility enhances plant stability, suppresses herbivore populations, and mitigates disease impacts.

1 Introduction

Aquatic plants play a vital role in ecosystems as primary producers. Herbivores feed on aquatic plants, while plant diseases spread and infect plants, leading to ecosystem imbalance. Recent genetic studies show that reducing plant susceptibility enhances resistance to herbivores and improves yield in crops like rice. Plots treated with insecticides that killed planthoppers saw an 18% increase in yield of a susceptible variety, while untreated plots saw a 25% increase[1].Every year, outbreaks of insect pests in paddy fields result in significant losses in the world's rice production; this problem is expected to be made worse by continuing climate change. Improved rice resistance only works against chewing or phloem-feeding insects, despite tremendous advancements in the screening, cloning, and introgression of insect resistance genes in rice germplasm into contemporary cultivars[2]. According to our findings, these lectin receptor kinase genes work in concert to produce long-lasting and broad-spectrum insect resistance as well as a resource for molecular breeding of rice cultivars resistant to insects[3]. Worldwide, planthoppers are a very damaging pest to crop output. Of all the rice pests, the brown planthopper (BPH) damages

rice crops the most severely worldwide. The best and most environmentally responsible way to safeguard the crop from BPH is to grow resistant cultivars. In rice breeding and production, over 19 BPH-resistant genes have been identified and utilised to varying degrees.The expression of Bph14 decreases the feeding, growth rate, and longevity of the BPH insects by activating the salicylic acid signalling pathway, causing callose deposition in phloem cells, and causing the synthesis of trypsin inhibitors following planthopper infection[4]. For more than two billion people, rice is a staple diet. In the majority of rice-growing locations in Asia, planthoppers such as BPH and WBPH coexist and significantly reduce yield by feeding on and spreading viruses that cause disease. Breeding resistant cultivars is a viable alternative to chemical pest management, which is costly and environmentally devastating. However, the majority of these initiatives target BPH with the hope that these types will also work well against WBPH[5]. Breeding varieties while taking into account the evolution of biotypes and shifting planthopper populations is the main problem facing plant breeders. Priorities for future research should focus on finding and transferring new resistance genes from various sources to expand the rice gene pool, finding durable gene combinations for marker-assisted pyramiding, and high-throughput screening of germplasm for field resistance to planthoppers[6]. In order to improve the long-term control of insect pests in this important crop, these continuing functional genomic research help to generate new insect-resistant rice varieties and shed light on the genetic underpinnings of interactions between insects and rice[7]. Insect-rice interactions at the transcriptome, proteome, metabolome, mitogenome, and metagenome levels are being investigated using a variety of molecular tools, particularly in relation to BPH and gall midge. These investigations are revealing novel strategies for managing insect pests as well as for comprehending the population genetics and phylogeography of rice pests[8]. Destructive diseases and phloem-feeding insect herbivores, especially the brown planthopper (BPH, Nilaparvata lugens), pose a serious threat to the production of rice, a staple crop in many parts of the world. The molecular basis of rice's resistance to BPH is still mostly unknown, even though numerous BPH resistance genes have been identified[9]. The Bph15 gene has been widely used in rice breeding because of its capacity to provide resistance to the brown planthopper (BPH; Nilaparvata lugens Stl). However, nothing is known about the molecular mechanism by which Bph15 confers resistance to BPH in rice[10]. According to the author, a superior resistance gene that developed long ago in an area where planthoppers are year-round residents may prove to be highly beneficial for managing agricultural insect pests[11]. We also go over the latest findings on biological control, trap cropping, and cultural management as ways to manage brown planthoppers. The development of environmentally friendly integrated management strategies for brown planthoppers is aided by these investigations[12]. One of the rice insects that causes significant damage in Asian countries is the brown planthopper (BPH), a monophagous migratory phloem-sucking bug (Nilaparvata lugens Stl). Over the past few years, this pest has wreaked havoc in some regions of India, Indonesia, China, Japan, Taiwan, Vietnam, and the Philippines due to high nitrogen and deliberate insecticide use along with rising temperatures[13]. Aquatic plants contribute significantly to the structure, function, and service delivery of aquatic ecosystems and fill a variety of ecological tasks. Research on aquatic plants is still booming because of their established significance in aquatic environments. The variety of nations and continents represented by conference attendees and authors of the papers in this special issue highlights both the numerous issues that this emerging field of study must deal with and the worldwide significance of aquatic plant research in the early twenty-first century[14].

1.1 Objective

This paper aims to:

1. Formulate a differential equation-based model for plant-herbivore-disease dynamics.
2. Identify equilibrium points of the system.
3. Conduct local and global stability analysis.
4. Simulate the system using MATLAB to study the effects of susceptibility factor on population dynamics.

2 Model Formulation

2.1 Model Variables

Let:

- $P(t)$: Healthy aquatic plant biomass at time t.
- $H(t)$: Herbivore population at time t.
- $I(t)$: Infected plant biomass at time t.

2.2 Parameters

- r: Growth rate of healthy plants.
- K: Carrying capacity of the plant population.
- α: Herbivore grazing rate.
- γ: Herbivore reproduction rate proportional to plant biomass.
- δ: Natural mortality rate of herbivores.
- β: Disease transmission rate (healthy to infected plants).
- η: Death rate of infected plants.
- S: Plant susceptibility factor ($0 \leq S \leq 1$).

2.3 Differential Equations

The system is modeled as follows:

$$\frac{dP}{dt} = rP\left(1 - \frac{P}{K}\right) - \alpha SHP - \beta PI, \tag{1}$$

$$\frac{dH}{dt} = \gamma SHP - \delta H, \tag{2}$$

$$\frac{dI}{dt} = \beta PI - \eta I. \tag{3}$$

2.4 Assumptions

1. Plant growth follows logistic dynamics.
2. Herbivore grazing and reproduction depend on plant biomass and susceptibility factor S.
3. Disease spreads through healthy and infected plant interactions.
4. Infected plants do not contribute to herbivore reproduction.

3 Boundedness

The system of equations is given as:

$$\begin{aligned}\frac{dP}{dt} &= rP\left(1-\frac{P}{K}\right)-\alpha SHP-\beta PI,\\ \frac{dH}{dt} &= \gamma SHP-\delta H,\\ \frac{dI}{dt} &= \beta PI-\eta I.\end{aligned}$$

To show that the system is bounded, we analyze the dynamics of the total populations.

- **Step 1: Prey Population Dynamics**

  For the prey population $P(t)$:

  $$\frac{dP}{dt} = rP\left(1-\frac{P}{K}\right)-\alpha SHP-\beta PI.$$

  Here: - The term $rP(1-\frac{P}{K})$ ensures that $P(t)$ is bounded above by the carrying capacity K. - The negative terms $-\alpha SHP$ and $-\beta PI$ further reduce $P(t)$, indicating that $P(t) \geq 0$ for all $t \geq 0$.

  Thus, $P(t)$ is bounded in the interval $[0, K]$.

- **Step 2: Predator Population Dynamics**

  For the predator population $H(t)$:

  $$\frac{dH}{dt} = \gamma SHP-\delta H.$$

  Factor out H:

  $$\frac{dH}{dt} = H\left(\gamma SP-\delta\right).$$

  - If $P(t) > \frac{\delta}{\gamma S}$, the predator population $H(t)$ grows. - If $P(t) \leq \frac{\delta}{\gamma S}$, the predator population $H(t)$ declines.

  Since $P(t)$ is bounded by K, there exists a maximum value of $H(t)$ determined by the interaction term γSP. Thus, $H(t)$ is bounded.

- **Step 3: Infection Population Dynamics**

  For the infection population $I(t)$:

  $$\frac{dI}{dt} = \beta PI - \eta I.$$

  Factor out I:

  $$\frac{dI}{dt} = I\left(\beta P - \eta\right).$$

  - If $P(t) > \frac{\eta}{\beta}$, the infected population $I(t)$ grows. - If $P(t) \leq \frac{\eta}{\beta}$, the infected population $I(t)$ declines.

  Since $P(t)$ is bounded, $I(t)$ will also be bounded by a maximum value determined by the interaction term βP.

- **Step 4: Total Boundedness**

  Define the total population $N(t) = P(t) + H(t) + I(t)$.

  Adding the equations:

  $$\frac{dN}{dt} = \frac{dP}{dt} + \frac{dH}{dt} + \frac{dI}{dt}.$$

  Substitute:

  $$\frac{dN}{dt} = \left[rP\left(1 - \frac{P}{K}\right) - \alpha SHP - \beta PI\right] + \left[\gamma SHP - \delta H\right] + \left[\beta PI - \eta I\right].$$

  After simplification:

  $$\frac{dN}{dt} = rP\left(1 - \frac{P}{K}\right) - \delta H - \eta I.$$

  Since $rP(1 - \frac{P}{K}) \leq rK/4$ (maximum value of logistic growth), and $-\delta H, -\eta I$ are negative terms, $\frac{dN}{dt}$ is bounded.

  Thus, the total population $N(t)$ is bounded.

- **Conclusion**

  Each of the populations $P(t)$, $H(t)$, and $I(t)$ is individually bounded, and therefore the system is globally bounded.

4 Equilibrium Points

To determine equilibrium points, we set $\frac{dP}{dt} = 0$, $\frac{dH}{dt} = 0$, and $\frac{dI}{dt} = 0$. The equilibrium points are:

1. **Trivial Equilibrium**: $(P^*, H^*, I^*) = (0, 0, 0)$.
2. **Herbivore-Free Equilibrium**: $(P^*, H^*, I^*) = (K, 0, 0)$.
3. **Disease-Free Equilibrium**:

   $$P^* = \frac{\delta}{\gamma S}, \quad H^* = \frac{r}{\alpha S}\left(1 - \frac{\delta}{\gamma SK}\right), \quad I^* = 0.$$

4. **Endemic Equilibrium**: Solving for non-zero P^*, H^*, I^* requires numerical solutions.

5 Local Stability

Jacobian Matrix

The Jacobian matrix J is defined as the matrix of partial derivatives of the system functions f_1, f_2, f_3 with respect to P, H, and I:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial P} & \frac{\partial f_1}{\partial H} & \frac{\partial f_1}{\partial I} \\ \frac{\partial f_2}{\partial P} & \frac{\partial f_2}{\partial H} & \frac{\partial f_2}{\partial I} \\ \frac{\partial f_3}{\partial P} & \frac{\partial f_3}{\partial H} & \frac{\partial f_3}{\partial I} \end{bmatrix}.$$

The system functions are given as:

$$\begin{aligned} f_1 &= rP\left(1 - \frac{P}{K}\right) - \alpha SHP - \beta PI, \\ f_2 &= \gamma SHP - \delta H, \\ f_3 &= \beta PI - \eta I. \end{aligned}$$

Substituting the partial derivatives, the Jacobian matrix becomes:

$$J = \begin{bmatrix} r\left(1 - \frac{2P}{K}\right) - \alpha SH - \beta I & -\alpha SP & -\beta P \\ \gamma SH & \gamma SP - \delta & 0 \\ \beta I & 0 & \beta P - \eta \end{bmatrix}.$$

Evaluation at Equilibrium Points

5.1 Equilibrium Point $E_0 = (0, 0, 0)$

Substitute $P = 0$, $H = 0$, $I = 0$:

$$J(E_0) = \begin{bmatrix} r & 0 & 0 \\ 0 & -\delta & 0 \\ 0 & 0 & -\eta \end{bmatrix}.$$

The eigenvalues are:

$$\lambda_1 = r, \quad \lambda_2 = -\delta, \quad \lambda_3 = -\eta.$$

Stability: Since $r > 0$, E_0 is unstable.

5.2 Equilibrium Point $E_1 = (K, 0, 0)$

Substitute $P = K$, $H = 0$, $I = 0$:

$$J(E_1) = \begin{bmatrix} -r & 0 & 0 \\ -\alpha SK & -\delta & 0 \\ 0 & 0 & -\eta \end{bmatrix}.$$

The eigenvalues are:

$$\lambda_1 = -r, \quad \lambda_2 = -\delta, \quad \lambda_3 = -\eta.$$

Stability: All eigenvalues are negative, so E_1 is locally stable.

5.3 Equilibrium Point $E_2 = (P^*, H^*, 0)$

Substitute $P^* = \frac{\delta}{\gamma S}$, $H^* > 0$, and $I = 0$: The Jacobian matrix is evaluated numerically due to complexity.

5.4 Equilibrium Point $E_3 = (P^*, H^*, I^*)$

This equilibrium requires solving for non-zero P^*, H^*, and I^*. The Jacobian can be evaluated numerically.

Eigenvalues and Stability

To determine stability:

- If all eigenvalues have negative real parts, the equilibrium point is locally stable.
- If any eigenvalue has a positive real part, the equilibrium point is unstable.

6 Global Stability

The system of equations is given as:

$$\begin{aligned}
\frac{dP}{dt} &= rP\left(1 - \frac{P}{K}\right) - \alpha SHP - \beta PI, \\
\frac{dH}{dt} &= \gamma SHP - \delta H, \\
\frac{dI}{dt} &= \beta PI - \eta I.
\end{aligned}$$

We aim to establish the global stability of the equilibrium point $E = (P^*, H^*, I^*)$ by constructing a Lyapunov function.

- **Step 1: Define a Lyapunov Function**

  Let us define a candidate Lyapunov function $V(P, H, I)$ as:

  $$V(P, H, I) = \frac{1}{2}(P - P^*)^2 + \frac{1}{2}(H - H^*)^2 + \frac{1}{2}(I - I^*)^2.$$

  This function is positive definite, i.e., $V(P, H, I) \geq 0$ and $V(P, H, I) = 0$ if and only if $(P, H, I) = (P^*, H^*, I^*)$.

- **Step 2: Compute the Time Derivative of V**

  The time derivative of $V(P, H, I)$ along the trajectories of the system is:

  $$\frac{dV}{dt} = \frac{\partial V}{\partial P}\frac{dP}{dt} + \frac{\partial V}{\partial H}\frac{dH}{dt} + \frac{\partial V}{\partial I}\frac{dI}{dt}.$$

  Substitute the derivatives:

  $$\begin{aligned}
  \frac{\partial V}{\partial P} &= P - P^*, \\
  \frac{\partial V}{\partial H} &= H - H^*, \\
  \frac{\partial V}{\partial I} &= I - I^*.
  \end{aligned}$$

Thus:

$$\frac{dV}{dt} = (P - P^*)\frac{dP}{dt} + (H - H^*)\frac{dH}{dt} + (I - I^*)\frac{dI}{dt}.$$

- **Step 3: Substitute the System Dynamics**

  Substitute the system equations into $\frac{dV}{dt}$:

$$\begin{aligned}\frac{dV}{dt} = (P - P^*)\left[rP\left(1 - \frac{P}{K}\right) - \alpha SHP - \beta PI\right] \\ + (H - H^*)\left[\gamma SHP - \delta H\right] \\ + (I - I^*)\left[\beta PI - \eta I\right].\end{aligned}$$

- **Step 4: Simplify and Analyze $\frac{dV}{dt}$**

  Expand each term and group similar components:

$$\begin{aligned}\frac{dV}{dt} = r(P - P^*)P\left(1 - \frac{P}{K}\right) - \alpha S(P - P^*)HP - \beta(P - P^*)PI \\ + \gamma S(H - H^*)HP - \delta(H - H^*)H \\ + \beta(I - I^*)PI - \eta(I - I^*)I.\end{aligned}$$

  Since P, H, I are bounded, let us verify that $\frac{dV}{dt} \leq 0$: 1. The term $r(P-P^*)P\left(1 - \frac{P}{K}\right)$ is negative for $P \neq P^*$ due to the logistic growth constraint. 2. The interaction terms $-\alpha S(P - P^*)HP$, $-\beta(P - P^*)PI$, and $-\delta(H - H^*)H$ are all negative or zero for all t. 3. The infected population terms $\beta(I - I^*)PI - \eta(I - I^*)I$ similarly reduce I toward I^*.

  Thus:

$$\frac{dV}{dt} \leq 0,$$

  and equality holds only when $(P, H, I) = (P^*, H^*, I^*)$.

Step 5: Conclusion

Since $V(P, H, I)$ is positive definite and $\frac{dV}{dt}$ is negative definite, the equilibrium point (P^*, H^*, I^*) is globally stable.

7 MATLAB Simulations

7.1 Graph for equilibrium point E_0 is obtained considering following para-metric values:

Parameters for the system

r = 0.5; K = 100; $\alpha = 0.01$; $\beta = 0.02$; $\gamma = 0.005$; $\delta = 0.1$; $\eta = 0.05$;

7.2 Graph for equilibrium point E_1 is obtained considering following para-metric values:

Parameters for the system
r = 0.5; K = 100; $\alpha = 0.01$; $\beta = 0.02$; $\gamma = 0.005$; $\delta = 0.1$; $\eta = 0.05$;

7.3 Graph for equilibrium point E_2 is obtained considering following para-metric values:

Parameters for the system
r = 0.5; K = 100; $\alpha = 0.01$; $\beta = 0.02$; $\gamma = 0.005$; $\delta = 0.1$; $\eta = 0.05$; S = 1.0;

7.4 Graph for equilibrium point E_3 is obtained considering following para-metric values:

Parameters for the system
r = 1; K = 100; $\alpha = 0.1$; $\gamma = 0.1$; $\delta = 0.05$; $\beta = 0.1$; $\eta = 0.1$; S = 0.8;

7.5 Graph for global stability is obtained considering following para-metric values:

Parameters for the system
r = 1; K = 100; $\alpha = 0.1$; $\gamma = 0.1$; $\delta = 0.05$; $\beta = 0.1$; η= 0.1; S = 0.8;

8 Results and Discussion

Simulations show that:

- **High Susceptibility** ($S \approx 1$): Herbivore populations rise rapidly, reducing plant biomass.
- **Low Susceptibility** ($S \approx 0.1$): Plant biomass stabilizes, herbivores decline, and disease spread slows.

9 Conclusion

In this study, we have developed a mathematical model to describe the dynamics of an aquatic ecosystem influenced by herbivory and plant diseases. The system incorporates three state variables: the healthy plant biomass (), herbivore population H(t) and infected plant biomass I(t), alongside key parameters representing ecological and biological processes. This paper presents a mathematical model to analyze aquatic plant-herbivore-disease dynamics. Reducing plant susceptibility enhances stability and minimizes herbivore and disease impacts. MATLAB simulations validate the model and provide insights into sustainable ecosystem management.

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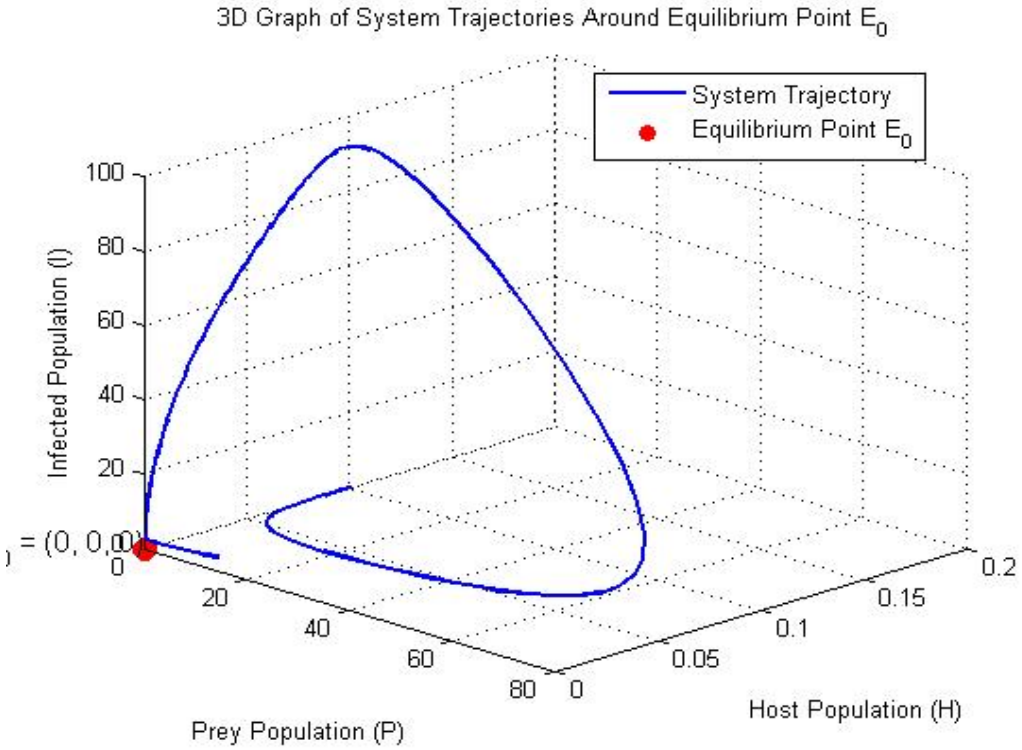

Figure 1: **Graph for** E_0

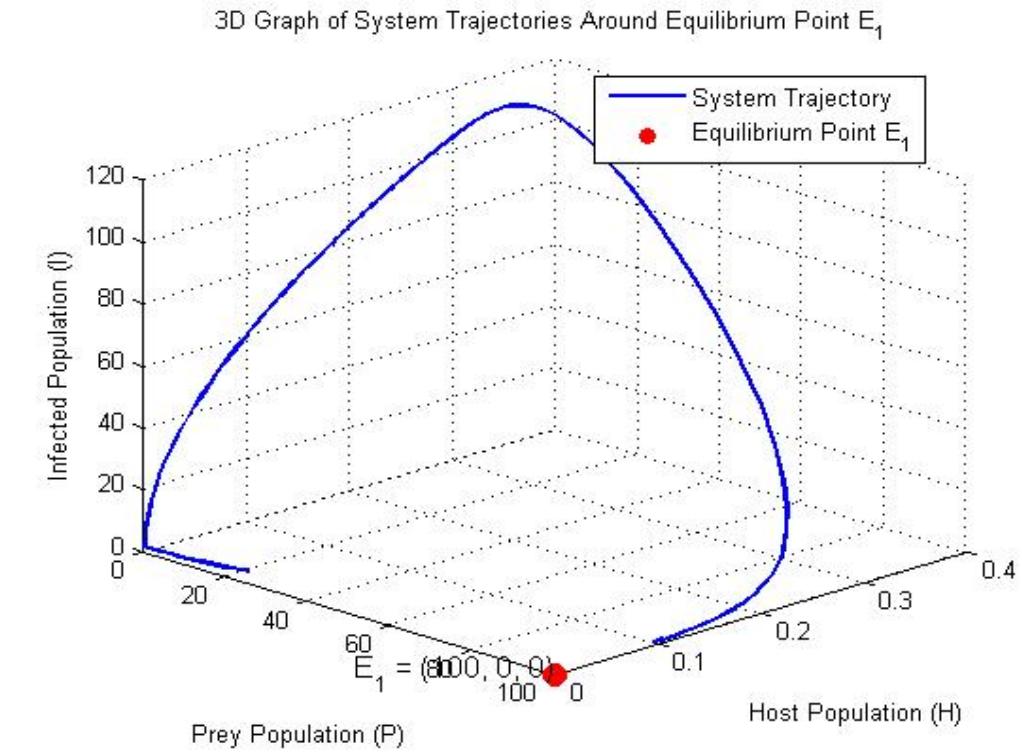

Figure 2: **Graph for** E_1

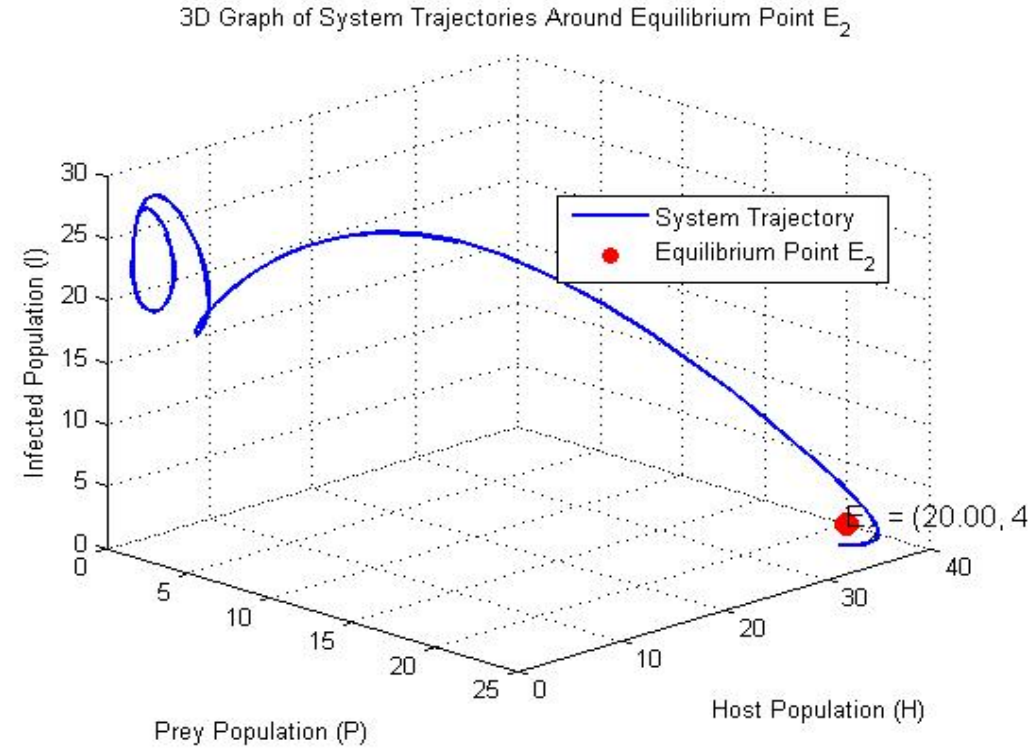

Figure 3: **Graph for** E_2

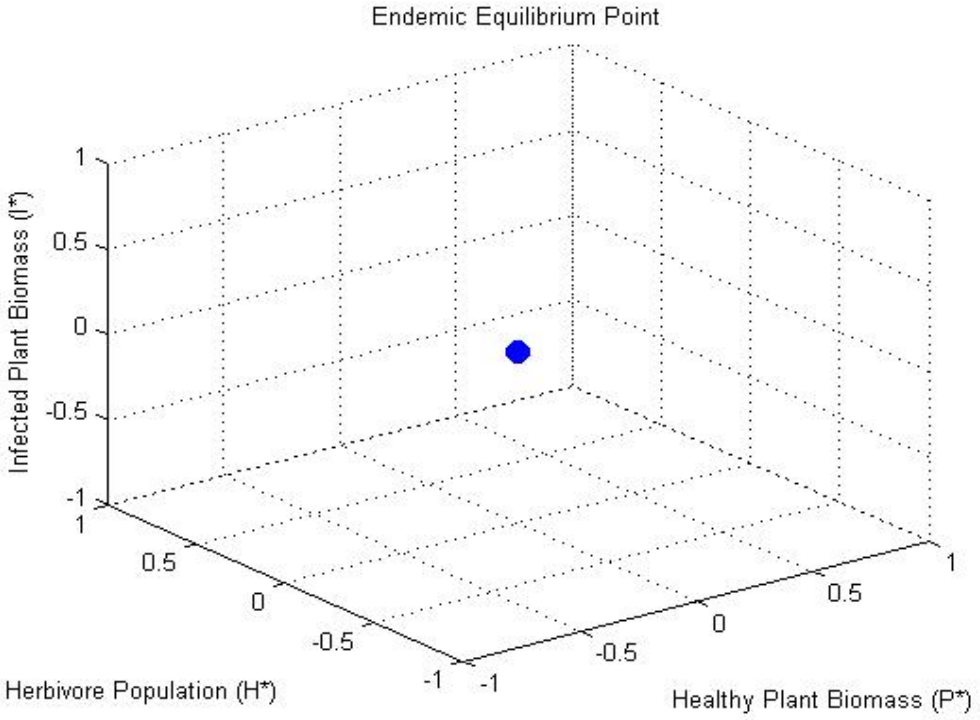

Figure 4: **Graph for** E_3

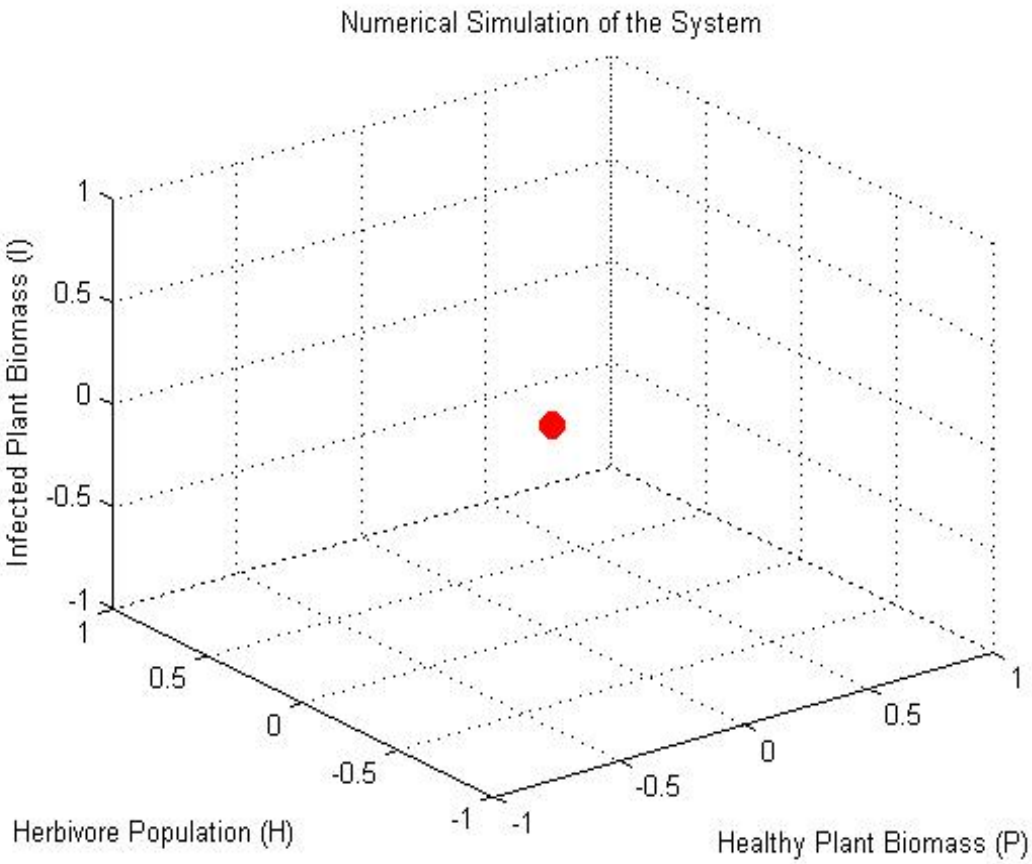

Figure 5: **graph for Global stability**