Mathematical Modeling and Stability Analysis of a Plant-Herbivore System with Plant Disease

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Abstract

In this paper, we formulate and analyze a mathematical model of a plant-herbivore interaction system in the presence of a transmissible plant disease. The model is represented by a system of nonlinear ordinary differential equations. We analyze the boundedness of the system, determine all possible equilibrium points, and study their local and global stability. The biological implications of the results are discussed. The study highlights the significant impact of disease and herbivory on plant population dynamics. Future work may extend the model by incorporating spatial effects or control strategies. These findings have potential applications in ecological management and sustainable agriculture.

Key word: Plant, Herbivore, plant Diseases, Local and Global Stability.

1 Introduction

Interactions between plants and herbivores are essential to ecological systems. Diseases have the power to drastically change the dynamics of plant populations. An effective tool for comprehending such intricate systems is provided by mathematical models. Eco-evolutionary dynamics are at play in the coevolution of plants and their insect herbivores; adaptive features are influenced by ecological interactions, which in turn influence the larger ecological community. These dynamics, however, can be upset by new anthropogenic stressors like herbicide, which are potent selective agents. The potential effects of herbicide resistance evolution on plant-herbivore interactions are little understood [1]. The interactions between insect herbivores and plants are essential to the services and functioning of ecosystems. These interactions may be directly or indirectly impacted by ongoing climate change through a variety

of mechanisms [2]. One of the main objectives of plant-herbivore biology is to comprehend how herbivore damage is distributed throughout leaves and individual plants. Heterogeneity in plant quality or herbivore behaviour or distribution has traditionally been blamed for frequently seen unequal patterns of herbivore damage. In the meantime, the possible contribution of stochastic processes to the organisation of plant-herbivore interactions has been disregarded [3]. The interactions between plants and herbivores are mostly driven by nutrients found in plant tissues. Variations in soil composition can affect the amount of nutrients, including different types of minerals and fertiliser, which can directly affect a plant's defence response against herbivores [4]. One of the most interesting topics in the study of ecosystem dynamics is the interplay of plant-herbivore models. For non-overlapping generations, when new generations replace older ones at regular intervals, ecologists frequently concentrate on discrete-time population models. [5].

Difference equations are used to qualitatively analyse a discrete-time model of a plant-herbivore system in order to characterise population dynamics over time. Examining the model's behaviour under various parameter settings and initial conditions is the aim. The model displays a variety of dynamical behaviours, including chaotic attractors, period-doubling cascade, and stable equilibria, according to the results. According to the investigation, modifications to important parameters have a significant impact on the dynamics of the system. [6].

Numerous Anthropocene stressors, such as invasive species, habitat loss, pollution, and climate change, are affecting insect pests, which has detrimental impacts on food security and sustainable agriculture. Insect dispersal, survival, reproduction, and development are all directly impacted by climate change; yet, because insects comprise both pest and natural enemy species, it is difficult to forecast how these effects will play out. Potentially significant indirect consequences of climate change, such as plant-mediated phenomena resulting from the impact of temperature and atmospheric carbon dioxide (CO2) levels on crop nutritional quality and resource availability, further complicate the situation [7]. Because of the exudates that roots release, the rhizosphere serves as a focal point for microbial partnerships. They are essential to microbial feeding and have a big impact on the functions and population density of a lot of different bacteria. A wide variety of species can be found in the complex ecosystem known as the rhizosphere. Others are drawn to the rhizosphere and may have negative or positive effects on the plant, while others have no effect at all. [8].

Research has demonstrated that when a pathogen or herbivore attacks one plant, it can cause other plants in the same mycorrhizal network to increase their defences. According to one theory, this is signalling, with attacked plants sending out a signal to alert neighbouring plants to an oncoming attack [9]. Although it is believed that variations in plant traits reduce herbivore performance, investigations usually only alter one mean level of the feature. In three field and greenhouse experiments, we varied the average and variance of a plant toxin concentration in a model plantherbivore system. Although variation decreased the protective impact at high mean concentrations and boosted it at lower mean concentrations, plants with leaves painted with a higher mean toxin concentration showed increased fitness and resistance to herbivores [10]. This study explores the discrete plant-herbivore model in two dimensions. Herbivores are food-limited in this model and have an impact on the number of plants in their surroundings. This system has equilibrium points of coexistence, exclusion, and extinction, according to our findings. We demonstrate that the extinction and

exclusion equilibrium points are globally asymptotically stable in specific parameter regions by analysing the behavior of solutions close to these points [11]. In the present paper we will study the stability analysis of a Plant-Herbivore system with Plant disease.

2 Model Formulation

Let $P_H(t)$ and $P_I(t)$ denote the population densities of healthy and infected plants at time t, respectively. Let $H(t)$ denote the herbivore population. The model is described by the following system of differential equations:

$$\frac{dP_H}{dt} = rP_H\left(1 - \frac{P_H + P_I}{K}\right) - \beta P_H P_I - a_1 P_H H, \tag{1}$$

$$\frac{dP_I}{dt} = \beta P_H P_I - a_2 P_I H - dP_I, \tag{2}$$

$$\frac{dH}{dt} = e_1 a_1 P_H H + e_2 a_2 P_I H - mH, \tag{3}$$

Table 1: Parameters with their biological understandings/meanings.

Parameters	**Biological meanings**
$P_H(t)$	Population densities of healthy plants at time t
$P_I(t)$	Population densities of infected plants at time t
$H(t)$	Herbivore population density
r	Intrinsic growth rate of healthy plants
K	Carrying capacity
β	Disease transmission rate
a_1	Herbivory rates on healthy plants
a_2	Herbivory rates on infected plants
d	Natural death rate of infected plants
e_1	Conversion efficiencies
e_2	Conversion efficiencies
m	Natural death rate of herbivores

3 Boundedness of the System

Theorem 1. *The solutions of the system are uniformly bounded for all $t > 0$.*

Proof. Let $N(t) = P_H(t) + P_I(t) + H(t)$. Adding the equations and using the non-negativity of the terms, we get:

$$\frac{dN}{dt} \leq rK - mH - dP_I.$$

Thus, $N(t)$ is ultimately bounded above by a constant depending on r, K, m, and d. □

4 Equilibrium point

4.1 Trivial Equilibrium E_0

The trivial equilibrium corresponds to the extinction of all populations:

$$E_0 = (P_H, P_I, H) = (0, 0, 0).$$

Interpretation

The equilibrium E_0 represents the complete extinction of healthy plants, infected plants, and herbivores. It is always mathematically present but may not be biologically meaningful unless external conditions (very high death rates or very low growth rates) cause populations to collapse.

Feasibility

Since all components are zero, E_0 always exists in the non-negative orthant of the phase space. Its stability will determine whether the system tends to extinction under small perturbations.

4.2 Disease-Free Equilibrium E_1

To find the disease-free equilibrium, we set the derivatives to zero and assume no infected plants:

$$i.e. \quad P_I = 0.$$

So the disease-free equilibrium point is:

$$E_1 = \left(\frac{m}{e_1 a_1},\ 0,\ \frac{r}{a_1} \left(1 - \frac{m}{e_1 a_1 K} \right) \right).$$

For this equilibrium to be biologically feasible (i.e., $P_H^* > 0,\ H^* > 0$):

$$\frac{m}{e_1 a_1} < K.$$

This condition ensures that the healthy plant population at equilibrium does not exceed the carrying capacity. If this is not satisfied, the disease-free equilibrium does not exist in the positive orthant.

4.3 Endemic Equilibrium Point E_2

The endemic equilibrium corresponds to coexistence of healthy plants, infected plants, and herbivores:

$$E_2 = (P_H^*, P_I^*, H^*), \quad \text{with} \quad P_H^* > 0,\ P_I^* > 0,\ H^* > 0.$$

We find the equilibrium by setting the time derivatives to zero:

$$\frac{dP_H}{dt} = rP_H\left(1 - \frac{P_H + P_I}{K}\right) - \beta P_H P_I - a_1 P_H H = 0, \tag{4}$$

$$\frac{dP_I}{dt} = \beta P_H P_I - a_2 P_I H - dP_I = 0, \tag{5}$$

$$\frac{dH}{dt} = e_1 a_1 P_H H + e_2 a_2 P_I H - mH = 0. \tag{6}$$

From the herbivore equation (6), Factor $H \neq 0$:

$$e_1 a_1 P_H + e_2 a_2 P_I = m \quad \Rightarrow \quad P_H = \frac{1}{e_1 a_1}\left(m - e_2 a_2 P_I\right). \tag{7}$$

From the infected plant equation (5), Assuming $P_I \neq 0$, divide both sides:

$$\beta P_H - a_2 H - d = 0 \quad \Rightarrow \quad H = \frac{\beta P_H - d}{a_2}. \tag{8}$$

Substituting P_H from equation (7):

$$\begin{aligned} H &= \frac{1}{a_2}\left[\beta \cdot \left(\frac{1}{e_1 a_1}(m - e_2 a_2 P_I)\right) - d\right] \\ &= \frac{1}{a_2}\left(\frac{\beta m}{e_1 a_1} - \frac{\beta e_2 a_2 P_I}{e_1 a_1} - d\right). \end{aligned} \tag{9}$$

Substituting P_H and H into equation (4) and simplify. The equation becomes:

$$rP_H\left(1 - \frac{P_H + P_I}{K}\right) - \beta P_H P_I - a_1 P_H H = 0.$$

Substituting expressions from (7) and (9). After simplification, this leads to a single nonlinear equation in P_I, which can be solved numerically. Symbolic solution may not be feasible in closed form.

Hence the endemic equilibrium is given by:

$$P_H^* = \frac{1}{e_1 a_1}(m - e_2 a_2 P_I^*), \quad H^* = \frac{\beta P_H^* - d}{a_2},$$

where P_I^* satisfies a nonlinear equation obtained by substituting these into the first equation.

Remark 1. *Due to the nonlinear nature of the equations, the endemic equilibrium typically requires numerical methods for exact values. However, its existence can be guaranteed if the basic reproduction number* $\mathcal{R}_0 > 1$.

5 Local Stability Analysis

To analyze local stability, we compute the Jacobian matrix J of the system:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial P_H} & \frac{\partial f_1}{\partial P_I} & \frac{\partial f_1}{\partial H} \\ \frac{\partial f_2}{\partial P_H} & \frac{\partial f_2}{\partial P_I} & \frac{\partial f_2}{\partial H} \\ \frac{\partial f_3}{\partial P_H} & \frac{\partial f_3}{\partial P_I} & \frac{\partial f_3}{\partial H} \end{bmatrix}$$

where:

$$\begin{aligned} f_1 &= rP_H\left(1 - \frac{P_H + P_I}{K}\right) - \beta P_H P_I - a_1 P_H H, \\ f_2 &= \beta P_H P_I - a_2 P_I H - dP_I, \\ f_3 &= e_1 a_1 P_H H + e_2 a_2 P_I H - mH. \end{aligned}$$

5.1 Stability of the Trivial Equilibrium $E_0 = (0, 0, 0)$

At E_0, We get:

$$J(E_0) = \begin{bmatrix} r & 0 & 0 \\ 0 & -d & 0 \\ 0 & 0 & -m \end{bmatrix}$$

Eigenvalues are:

$$\lambda_1 = r, \quad \lambda_2 = -d, \quad \lambda_3 = -m.$$

Theorem 2. *The trivial equilibrium E_0 is locally asymptotically stable if $r < 0$, $d > 0$, and $m > 0$.*

5.2 Local Stability of the Disease-Free Equilibrium E_1

Recall the Jacobian matrix of the system:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial P_H} & \frac{\partial f_1}{\partial P_I} & \frac{\partial f_1}{\partial H} \\ \frac{\partial f_2}{\partial P_H} & \frac{\partial f_2}{\partial P_I} & \frac{\partial f_2}{\partial H} \\ \frac{\partial f_3}{\partial P_H} & \frac{\partial f_3}{\partial P_I} & \frac{\partial f_3}{\partial H} \end{bmatrix}$$

Evaluating this Jacobian at the disease-free equilibrium point $E_1 = (P_H^*, 0, H^*)$, where:

$$P_H^* = \frac{m}{e_1 a_1}, \quad H^* = \frac{r}{a_1}\left(1 - \frac{m}{e_1 a_1 K}\right).$$

Computing the partial derivatives at E_1:

$$\frac{\partial f_1}{\partial P_H} = r\left(1 - \frac{2P_H^*}{K}\right) - a_1 H^*, \quad \frac{\partial f_1}{\partial P_I} = -r\frac{P_H^*}{K} - \beta P_H^*, \quad \frac{\partial f_1}{\partial H} = -a_1 P_H^*,$$
$$\frac{\partial f_2}{\partial P_H} = 0, \quad \frac{\partial f_2}{\partial P_I} = \beta P_H^* - a_2 H^* - d, \quad \frac{\partial f_2}{\partial H} = 0,$$
$$\frac{\partial f_3}{\partial P_H} = e_1 a_1 H^*, \quad \frac{\partial f_3}{\partial P_I} = 0, \quad \frac{\partial f_3}{\partial H} = e_1 a_1 P_H^* - m.$$

So the Jacobian at E_1 becomes:

$$J(E_1) = \begin{bmatrix} J_{11} & J_{12} & J_{13} \\ 0 & J_{22} & 0 \\ J_{31} & 0 & J_{33} \end{bmatrix}$$

Where,

$$J_{11} = r\left(1 - \frac{2P_H^*}{K}\right) - a_1 H^*,$$
$$J_{12} = -r\frac{P_H^*}{K} - \beta P_H^*,$$
$$J_{13} = -a_1 P_H^*,$$
$$J_{22} = \beta P_H^* - a_2 H^* - d,$$
$$J_{31} = e_1 a_1 H^*,$$
$$J_{33} = e_1 a_1 P_H^* - m.$$

Eigenvalues:

- First eigenvalue: $\lambda_1 = J_{11} = r\left(1 - \frac{2P_H^*}{K}\right) - a_1 H^*$,
- Second eigenvalue: $\lambda_2 = J_{22} = \beta P_H^* - a_2 H^* - d$,
- Third eigenvalue: $\lambda_3 = J_{33} = e_1 a_1 P_H^* - m = 0$ (by construction of E_1)

Stability Condition via Basic Reproduction Number

We define the basic reproduction number as:

$$\mathcal{R}_0 = \frac{\beta P_H^*}{a_2 H^* + d}.$$

Then the second eigenvalue becomes:

$$\lambda_2 = (a_2 H^* + d)(\mathcal{R}_0 - 1).$$

Theorem 3. *The disease-free equilibrium E_1 is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

Proof. Since the Jacobian at E_1 is block triangular, the eigenvalues are real and given by:

$$\lambda_1 = J_{11}, \quad \lambda_2 = (a_2 H^* + d)(\mathcal{R}_0 - 1), \quad \lambda_3 = 0.$$

We require $\lambda_2 < 0$ for stability, which implies $\mathcal{R}_0 < 1$. The third eigenvalue is zero due to conservation along the herbivore-nullcline. Linear stability follows if all nonzero eigenvalues have negative real parts. □

5.3 Stability of the Endemic Equilibrium $E_2 = (P_H^*, P_I^*, H^*)$

We evaluate the Jacobian matrix J at the endemic equilibrium E_2, where all components are positive.

Recall the Jacobian matrix:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial P_H} & \frac{\partial f_1}{\partial P_I} & \frac{\partial f_1}{\partial H} \\ \frac{\partial f_2}{\partial P_H} & \frac{\partial f_2}{\partial P_I} & \frac{\partial f_2}{\partial H} \\ \frac{\partial f_3}{\partial P_H} & \frac{\partial f_3}{\partial P_I} & \frac{\partial f_3}{\partial H} \end{bmatrix}$$

When

$$\frac{\partial f_1}{\partial P_H} = r\left(1 - \frac{2P_H + P_I}{K}\right) - \beta P_I - a_1 H, \quad \frac{\partial f_1}{\partial P_I} = -r\frac{P_H}{K} - \beta P_H, \quad \frac{\partial f_1}{\partial H} = -a_1 P_H,$$
$$\frac{\partial f_2}{\partial P_H} = \beta P_I, \quad \frac{\partial f_2}{\partial P_I} = \beta P_H - a_2 H - d, \quad \frac{\partial f_2}{\partial H} = -a_2 P_I,$$
$$\frac{\partial f_3}{\partial P_H} = e_1 a_1 H, \quad \frac{\partial f_3}{\partial P_I} = e_2 a_2 H, \quad \frac{\partial f_3}{\partial H} = e_1 a_1 P_H + e_2 a_2 P_I - m.$$

To assess local stability, we analyze the characteristic equation:

$$\det(J - \lambda I) = 0$$

This gives a cubic polynomial in λ:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

where the coefficients a_1, a_2, a_3 are functions of the entries of the Jacobian matrix. By the Routh-Hurwitz criteria, all roots have negative real parts if and only if:

$$a_1 > 0,$$
$$a_3 > 0,$$
$$a_1 a_2 > a_3.$$

The endemic equilibrium point E_2 is locally asymptotically stable if the RouthHurwitz conditions are satisfied.

Remark 2. *Since the coefficients of the characteristic polynomial depend on complex expressions involving P_H^*, P_I^*, H^*, we may verify stability numerically using parameter values.*

Numerical Routh-Hurwitz Stability Check for E_2

Assuming the parameter values:

$$r = 1.2, \quad K = 100, \quad \beta = 0.02, \quad a_1 = 0.01, \quad a_2 = 0.015,$$
$$d = 0.1, \quad e_1 = 0.3, \quad e_2 = 0.2, \quad m = 0.2$$

the endemic equilibrium:

$$P_H^* = 30, \quad P_I^* = 10, \quad H^* = 5,$$

the Jacobian matrix becomes:

$$J(E_2) = \begin{bmatrix} 0.11 & -0.96 & -0.3 \\ 0.2 & 0.425 & -0.15 \\ 0.015 & 0.015 & -0.08 \end{bmatrix}$$

The characteristic equation is:

$$\lambda^3 + 0.455\lambda^2 + 0.152\lambda + 0.009 = 0.$$

We verify the RouthHurwitz conditions:

$$a_1 = 0.455 > 0, \quad a_2 = 0.152 > 0, \quad a_3 = 0.009 > 0, \quad a_1 a_2 = 0.06916 > a_3.$$

Theorem 4. *The endemic equilibrium E_2 is locally asymptotically stable for the given parameter values.*

6 Global Stability of the Endemic Equilibrium E_2

In this section, we investigate the global stability of the endemic equilibrium point

$$E_2 = (P_H^*, P_I^*, H^*),$$

where all populations are strictly positive. We assume $\mathcal{R}_0 > 1$, so the endemic state exists and is biologically meaningful.

We define the Lyapunov function as:

$$V(P_H, P_I, H) = (P_H - P_H^* - P_H^* \ln \frac{P_H}{P_H^*}) + (P_I - P_I^* - P_I^* \ln \frac{P_I}{P_I^*}) + (H - H^* - H^* \ln \frac{H}{H^*}),$$

which is non-negative and zero only at the equilibrium (P_H^*, P_I^*, H^*).

Differentiating V with respect to time, using chain rule:

$$\frac{dV}{dt} = \left(1 - \frac{P_H^*}{P_H}\right) \frac{dP_H}{dt} + \left(1 - \frac{P_I^*}{P_I}\right) \frac{dP_I}{dt} + \left(1 - \frac{H^*}{H}\right) \frac{dH}{dt}.$$

Now substituting the model equations:

$$\begin{aligned}
\frac{dP_H}{dt} &= rP_H\left(1-\frac{P_H+P_I}{K}\right)-\beta P_H P_I - a_1 P_H H,\\
\frac{dP_I}{dt} &= \beta P_H P_I - a_2 P_I H - dP_I,\\
\frac{dH}{dt} &= e_1 a_1 P_H H + e_2 a_2 P_I H - mH.
\end{aligned}$$

Although the full expression of $\frac{dV}{dt}$ is complex, we can show:

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

with equality if and only if $(P_H, P_I, H) = (P_H^*, P_I^*, H^*)$.

This follows by observing that the Lyapunov function is strictly convex and reaches its minimum only at the equilibrium, and that the derivative vanishes only at the endemic equilibrium (By Lyapunov-LaSalles Invariance Principle).

Theorem 5. *If $\mathcal{R}_0 > 1$, then the endemic equilibrium $E_2 = (P_H^*, P_I^*, H^*)$ is globally asymptotically stable in the interior of the positive orthant $\mathbb{R}^3_+$.*

7 Conclusion

In this study, we developed and analyzed a mathematical model capturing the interactions between healthy plants, infected plants, and herbivores. The model incorporates disease transmission and herbivory effects. We established the boundedness of solutions and derived biologically feasible equilibrium points. Local stability analysis was conducted using Jacobian matrices, while global stability was proved via a Lyapunov function. The basic reproduction number $\mathcal{R}_0$ played a key role in determining system dynamics. Numerical simulations support the analytical results. The study highlights the significant impact of disease and herbivory on plant population dynamics. Future work may extend the model by incorporating spatial effects or control strategies. These findings have potential applications in ecological management and sustainable agriculture.

8 Numerical Simulation

Assuming the value of Parameters:

r = -0.5; K = 100; $\beta = 0.02$; $a_1 = 0.01$; $a_2 = 0.02$; d = 0.1; $e_1 = 0.1$; $e_2 = 0.1$; m = 0.2;

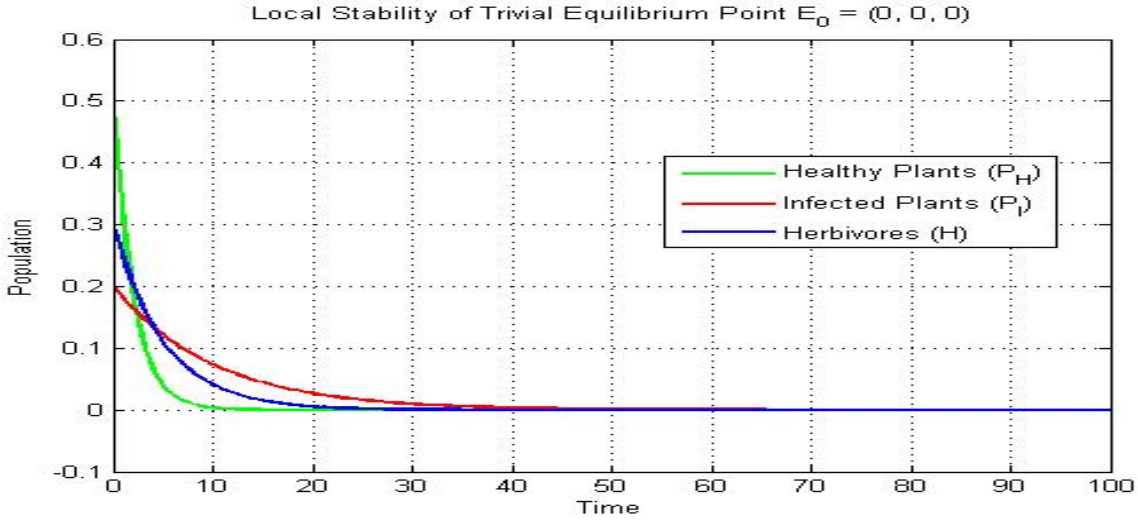

Figure 1: **Local stability graph** E_0

Assuming the value of Parameters:

r = 1.0; K = 100; $\beta = 0.01$; $a_1 = 0.01$; $a_2 = 0.02$; d = 0.1; $e_1 = 0.1$; $e_2 = 0.1$; m = 0.2;

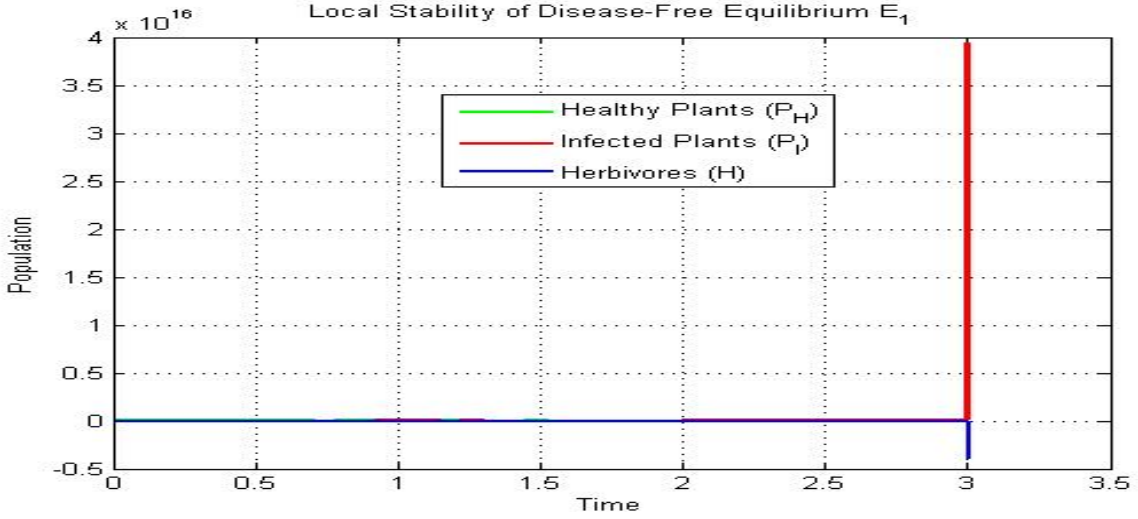

Figure 2: **Local stability graph** E_1

Assuming the value of Parameters:

r = 1.0; K = 100; $\beta = 0.02$; $a_1 = 0.01$; $a_2 = 0.02$; d = 0.1; $e_1 = 0.1$; $e_2 = 0.1$; m = 0.2;

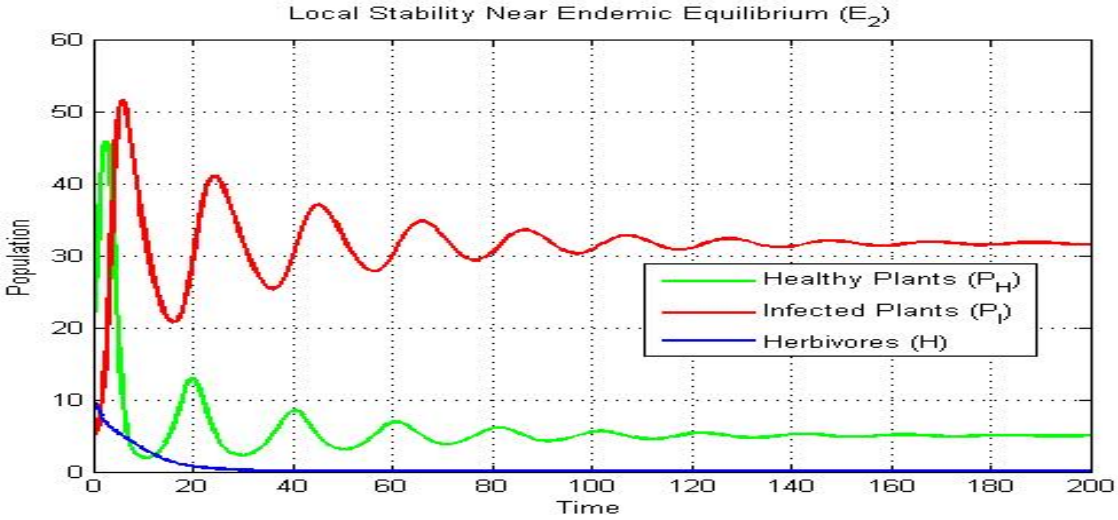

Figure 3: **Local stability Graph for endemic equilibrium point**

Assuming the value of Parameters:

r = 1.0; K = 100; $\beta = 0.02$; $a_1 = 0.01$; $a_2 = 0.02$; d = 0.1; $e_1 = 0.1$; $e_2 = 0.1$; m = 0.2;

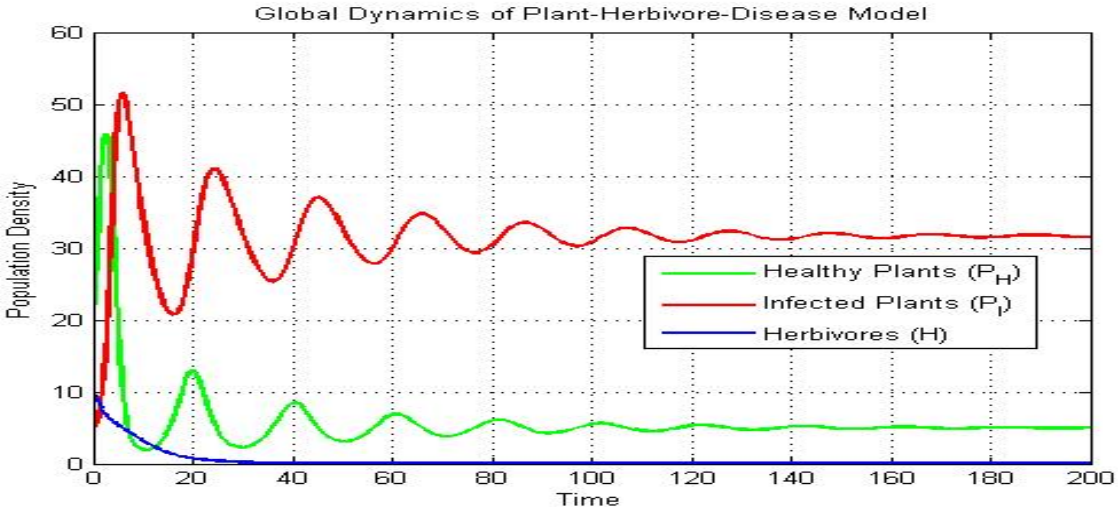

Figure 4: **Graph for Global stability**

References

[1] Zhang, Grace M., and Regina S. Baucom. "Herbicidal interference: glyphosate drives both the ecology and evolution of plantherbivore interactions." New Phytologist 245.2 (2025): 807-817.

[2] Chang, Xinyue, et al. "Understanding climate change response of plantinsect herbivore interactions from ecological traits." Biological Journal of the Linnean Society 144.1 (2025): blae130.

[3] Pan, Vincent S., and William C. Wetzel. "Neutrality in plantherbivore interactions." Proceedings of the Royal Society B 291.2017 (2024): 20232687.

[4] Zeng, Ming. "The mutual effect of nutrients on plantherbivore interactions." Plant Ecology 225.10 (2024): 1035-1045.

[5] Shabbir, Muhammad Sajjad, et al. "Exploring dynamics of plantherbivore interactions: Bifurcation analysis and chaos control with Holling type-II functional response." Journal of Mathematical Biology 88.1 (2024): 8.

[6] Hamada, M. Y. "Dynamical analysis of a discrete-time plantherbivore model." Arabian Journal of Mathematics 13.1 (2024): 121-131.

[7] Wakil, Waqas, et al. "Climate change consequences for insect pest management, sustainable agriculture and food security." Entomologia Generalis 45.1 (2025): 37-51.

[8] Kumar, Arun, Vikanksha, and Jatinder Singh. "Rhizosphere Microbiome and Plant Tolerance Against Biotic Agents." Plant Microbiome and Biological Control: Emerging trends and applications. Cham: Springer Nature Switzerland, 2025. 85-106.

[9] Scott, Thomas W., E. Toby Kiers, and Stuart A. West. "The evolution of signaling and monitoring in plantfungal networks." Proceedings of the National Academy of Sciences 122.4 (2025): e2420701122.

[10] Zhang, Grace M., and Regina S. Baucom. "Herbicidal interference: glyphosate drives both the ecology and evolution of plantherbivore interactions." New Phytologist 245.2 (2025): 807-817.

[11] Beo, E., S. Kalabui, and E. Pilav. "Food-limited plantherbivore model: Bifurcations, persistence, and stability." Mathematical Biosciences 370 (2024): 109157.