Contents lists available at Science-Gate

International Journal of Advanced and Applied Sciences

Journal homepage: http://www.science-gate.com/IJAAS.html

Stability and control of a plant epidemic model with pesticide intervention

Balajied Me Syrti *, Anuradha Devi

Department of Mathematics, The Assam Royal Global University, Guwahati-781035, India

ARTICLE INFO

Article history:
Received 31 August 2023
Received in revised form
1 January 2024
Accepted 26 January 2024

Keywords:
Plant epidemic model
Stability
Routh-Hurwitz criterion
Lyapunov function
Sensitivity analysis

ABSTRACT

This paper introduces a model for studying plant epidemics that applies pesticides to control disease spread among two types of plant populations: those that are susceptible and those that are already infected. The model uses non-linear ordinary differential equations and the Holling type II response function to depict how disease spreads based on the number of susceptible plants available. The model is carefully checked for biological accuracy, ensuring characteristics such as positivity and boundedness. It defines points of equilibrium where the numbers of susceptible and infected plants stabilize. The study looks at scenarios with no infected plants (disease-free equilibrium) and scenarios where the disease continues to exist within the plant population (endemic equilibrium). The basic reproduction number, R_0, is calculated to assess the system's stability. If R_0 is less than 1, the disease is unlikely to spread widely, and the system is likely to return to being disease-free, both locally and globally, over time. However, if R_0 is greater than 1, it indicates that the disease will persist in the population. This endemic state has also been shown to be stable both locally and globally. A sensitivity analysis helps identify key factors that affect disease spread and assists in forming strategies to manage the disease. Finally, numerical simulations are used to support the findings of the analysis.

1. Introduction

Mathematical models play a crucial role in understanding the impact of pesticides on various aspects of ecological and epidemiological systems. These models provide a quantitative framework to explore how the application of pesticides influences the dynamics of populations, disease spread, and the overall ecosystem (Arino et al., 2004). Researchers can use mathematical equations to understand the effects of pesticides on the interactions between susceptible and infective individuals, enabling them to simulate different scenarios and predict outcomes for various pesticide application strategies. For instance, in the context of disease control, mathematical models can help determine the optimal timing and dosage of pesticide application to minimize disease prevalence (Chowdhury et al., 2019). They can also assess the potential for pesticide resistance to develop in target populations over time (Schechtman et al., 2020). Furthermore, mathematical models can be used to study the unintended consequences of pesticide use. These include effects on non-target organisms, ecological disruptions, and shifts in the balance of predator-prey relationships. By integrating data on pesticide toxicity, decay rates, and ecological parameters, models can quantify the impact of pesticides on various components of the ecosystem (Anguelov et al., 2017). Overall, mathematical models are essential for comprehending the impact of pesticides on populations and ecosystems, ultimately enabling more informed and sustainable decision-making in pest and disease management.

When discussing the impact of pesticides on disease dynamics, the term "susceptible-infective" is usually associated with epidemiological models that study the spread of infectious diseases in populations (Brauer, 2005). In this context, "susceptible" refers to the plant populations that are susceptible to contracting the disease, while "infective" refers to populations that are infected and can transmit the disease to the susceptible plants. According to the World Health Organization (WHO), pesticides are chemicals used to control pests, including insects, fungi, and weeds, in agricultural and environmental settings. While pesticides are primarily designed to target pests, they can also have indirect effects on non-target organisms, including

* Corresponding Author.
Email Address: mbalajiedmesyrti@gmail.com (B. M. Syrti)
https://doi.org/10.21833/ijaas.2024.02.011
Corresponding author's ORCID profile:
https://orcid.org/0009-0006-1141-1413

beneficial insects and microbes. These indirect effects can have implications for disease dynamics in a population (Overton et al., 2021). Research on the impact of pesticides on disease dynamics often involves ecological and epidemiological models. These models can help scientists understand how pesticides may influence the prevalence and transmission of diseases in susceptible populations. Some studies focus on how pesticides affect the abundance or behavior of vector organisms (e.g., mosquitoes transmitting diseases like malaria or Zika virus) or how they alter the interactions between hosts and pathogens (Hilker and Schmitz, 2008; Kar, 2005; Pal and Samanta, 2010).

Employing pesticides within plant populations serves as a strategic approach to address various challenges and concerns related to plant health and agriculture. This practice involves the controlled application of chemical substances designed to manage pests, pathogens, and diseases that can adversely affect plant growth, yield, and overall health. It is important to note that while pesticides offer substantial benefits, their use requires careful consideration. Misuse or overreliance on pesticides can lead to unintended consequences, such as the development of pest resistance, environmental pollution, harm to non-target species, and health risks for humans. Integrated Pest Management (IPM) is a holistic approach that combines various strategies, including cultural practices, biological controls, and judicious pesticide use, to ensure effective and sustainable pest and disease management while minimizing negative impacts (Ofuoku et al., 2009).

The term "Eco" used here refers to an intricate ecosystem composed of a few interdependent components. The foundation of ecosystem modeling dates back to the early work of Malthus and Verhulst in 1798 (Malthus, 2023), who focused on single populations, while Lotka (1910) introduced the initial models for interacting populations. The term "Epidemic" pertains to diseases that can manifest within the ecosystem (Bacaër, 2011). As such, an eco-epidemic model concerns the portrayal of the inherent behavior of an ecosystem influenced by specific diseases. The first epidemic model was formulated by Kermack and McKendrick (1927) (Kermack and McKendrick, 1991). Research has indicated that infectious diseases frequently disrupt predator-prey communities (Hsieh and Hsiao, 2008; Shorbaji et al., 2017). However, Bairagi and Adak (2015) have demonstrated that the infection of predators can also introduce a stabilizing effect. Most of the existing eco-epidemiology models concentrate on diseases within the prey population (Pal, 2020; Purnomo et al., 2017; Shorbaji et al., 2017). Only a handful of models involve an infected predator population. Some of these later models may exhibit continuous oscillations that are not found in the non-infected environmental models considered (Hilker and Schmitz, 2008; Kar, 2005).

The use of pesticides is key in controlling the spread of diseases among plants and maintaining ecological systems. This paper focuses on how pesticides affect plant disease spread by studying a specific plant epidemic model. We analyze how stable the disease control is when pesticides are used. The results offer new and valuable information that can help policymakers and ecologists make informed decisions about disease management and ecological preservation.

The study of plant epidemic models often uses models similar to those for predator-prey interactions, where the role of prey or predators is played by plant populations facing an epidemic. In these models, plant populations are categorized into groups like susceptible and infected. Various control measures, such as medication and culling, are included in the models, applicable to diseases like bird and swine flu. However, this study introduces a unique focus on plant-specific epidemics, particularly in crops like tea and rice, and examines the effects of pesticide use, a topic not previously explored in research.

This research was motivated by diseases that affect particular crops. For example, Algal leaf spot disease, also known as green scurf, caused by the algae Cephaleuros Virescens, affects tea plantations in regions like Assam and Meghalaya, causing significant economic losses. This disease, spread through rain-dispersed spores, challenges over 200 plant species in warm, humid climates, severely impacting tea production.

The paper is structured as follows: Sections 2 and 3 develop a mathematical model for diseases in plant populations, including basic assumptions. Section 4 discusses the model's positivity and boundedness. Sections 5 and 6 analyze the stability of all possible equilibrium states of the model. Section 7 focuses on the sensitivity of the basic reproduction number. Section 8 uses numerical simulations to illustrate analytical results. Finally, Sections 9 and 10 present a detailed discussion of the results and conclusions. Advanced tools such as Mathematica, Matlab, and MatCont were used for the analytical and numerical work in this study.

1.1. Assumptions of plant epidemic model

The necessary assumptions for the proposed plant epidemic model are given as follows:

1. The group of plants within a population that are susceptible to the disease but not yet infected is referred to as the susceptible class. This portion of the population is represented as $S(t)$.
2. The group of plants that have the capacity to spread the disease to other plants within the population is referred to as the infected class. This proportion of the population is denoted as $I(t)$.
3. In the absence of disease, the plant population experiences logistic growth with carrying capacity $\frac{1}{b_1}$ and the natural growth rate a_1. As a result, the plant populations will increase at a rate $a_1 S(1 - b_1 S)$.

4. When a disease exists, the plant populations get categorized into two disjoint classes, which change with time t: the susceptible plants, labeled as $S(t)$, and the infected plants, labeled as $I(t)$. Consequently, at time t, the overall population can be expressed as $S(t) + I(t) = N(t)$.
5. The infection of susceptible plants occurs upon contact with infected plants, and this interaction is assumed to follow mass action kinetics characterized by the convolution rate β_1.
6. To achieve disease control, a quantity of pesticides, which is denoted by $P(t)$, is administered within the plant populations. This approach involves the application of pesticides to both the susceptible and infected plants in the model.
7. The interaction between susceptible and infected plants is modeled using a Holling type II functional response given by $\frac{\beta_1 SI}{1+\gamma_1 I}$. Similarly, the impact of pesticides on reducing the infection rate in plants is represented by another Holling Type II functional response, $\frac{\beta_2 IP}{1+\gamma_2 P}$.
8. All the model parameters are assumed to be non-negative.

2. Mathematical model

At any time t, the plant population is divided into two sub-populations, namely the susceptible and the infected populations, which are denoted by $S = S(t)$ and $I = I(t)$ respectively and $S(t) + I(t) = N(t)$ denotes the Total Biomass of the plant populations. Let $P = P(t)$ be the Pesticides used in the population considered. The transfer diagram of the model is depicted in Fig. 1, while Table 1 provides the notations and descriptions for the model parameters.

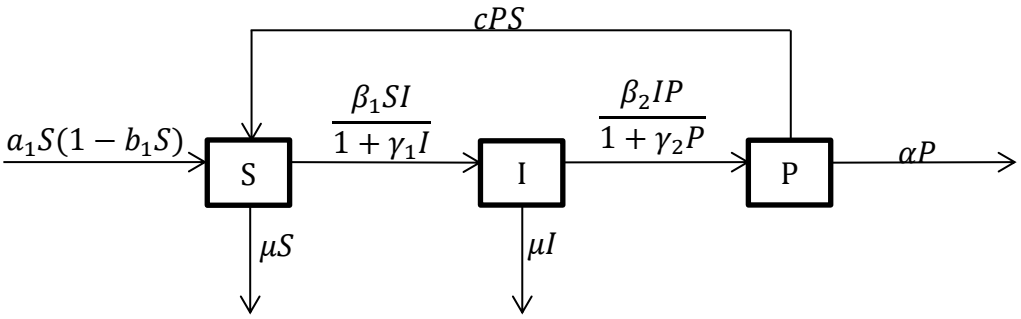

Fig. 1: Transfer diagram of the model (1)

Table 1: Notations and description of model parameters

Parameters	Definitions of parameters
a_1	The natural growth of susceptible plant population
$\frac{1}{b_1}$	The carrying capacity of plants
β_1	The contact rate of susceptible and infected plants
γ_1	The catching rate of disease by susceptible plants
c	The proportion of susceptible plants damaged by pesticides
μ	The natural death rate of plant populations
β_2	The contact rate of pesticides and infected plants
γ_2	The handling rate of infected plants by the use of pesticides
α	The rate at which pesticides are being used

From Fig. 1, the mathematical model will be governed by the following system of equations:

$$\begin{aligned}\frac{dS}{dt} &= a_1 S(1-b_1 S) - \frac{\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S \\ \frac{dI}{dt} &= \frac{\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P} \\ \frac{dP}{dt} &= -\alpha P. \end{aligned} \quad (1)$$

From the biological point of view, we are only interested in the dynamics of the system (Eq. 1) in the closed octant R_+^3. Thus, we consider the initial conditions:

$$S(0) \equiv S_0 > 0, I(0) \equiv I_0 > 0 \text{ and } P(0) \equiv P_0 > 0. \quad (2)$$

where, $\frac{dS}{dt}, \frac{dI}{dt}$ and $\frac{dP}{dt}$ represents the rates of change of the quantities $S(t), I(t)$ and $P(t)$ respectively.

2.1. Positivity and boundedness

To ensure the model under consideration is well-behaved and biologically valid, the paper examines certain essential properties such as positivity, which ensures that the populations of both susceptible and infected plants remain non-negative, and boundedness, which ensures that the populations do not grow indefinitely. The assertion of these properties is supported by Theorem 1 and Theorem 2, which provide assurance for the positivity and boundedness of the system in Eq. 1.

2.1.1. Positivity

Theorem 1: Let $S(0) > 0$, $I(0) > 0$, $P(0) > 0$. This implies all solutions of the system represented by Eq. 1 that start in R^3 remain positive at R_+^3 for all $t \geq 0$.

Proof: To prove the theorem, we use all the equations of the model (Eq. 1). Following a similar approach used by Hugo and Simanjilo (2019), we obtain the inequality expression from the 1st equation of model (Eq. 1) as follows:

$$\frac{dS}{dt} \leq a_1 S(1 - b_1 S),$$

which, when simplified, gives:

$$S \leq \frac{S(0)}{e^{-a_1 t}(1 - b_1 S(0)) + b_1 S(0)}.$$

Now, as $t \to \infty$, we obtain $0 < S \leq \frac{1}{b_1}$. Hence, the solution of system (1) is feasible in the region $\Omega = \{S, I, P\}$.

Similar proofs can be established using a similar approach for the remaining equations of the model.

Hence, the theorem.

2.1.2. Boundedness

Theorem 2: All solutions of system (Eq. 1) that start in ${R_+}^3$ are uniformly bounded.

Proof: Let $S(t), I(t), P(t))$ be any solution of the system (1). Since, $\frac{dS}{dt} \leq a_1 S(1 - b_1 S)$.

We have, $\limsup_{t \to \infty} S(t) \leq a_1$. Let $W = \frac{S}{1+a_1} + I + \frac{P}{\beta_2}$. Then,

$$\frac{dW}{dt} = \frac{a_1}{1+a_1} S(1 - b_1 S) - \mu I - \frac{\alpha}{\beta_2} P \leq \frac{a_1}{1+a_1} S - \mu I - \frac{\alpha}{\beta_2} P$$

$$\leq \frac{2a_1}{1+a_1} - \delta W,$$

where

$$\delta = min\{1, \mu, \alpha\}.$$

Therefore,

$$\frac{dW}{dt} + \delta W \leq \frac{2a_1}{1+a_1}.$$

Applying a theorem of Birkhoff and Rota (1982) on the above differential inequalities, we obtain:

$$0 \leq W(S, I, P) \leq \frac{2a_1}{(1+a_1)\delta} + \frac{W(S(0), I(0), P(0))}{e^{\delta t}}.$$

Now, as $t \to \infty$, we obtain

$$0 \leq W \leq \frac{2a_1}{(1+a_1)\delta}.$$

Thus, all the solutions of Eq. 1 lie in the region:

$$\Omega = \left\{(S, I, P): 0 \leq W \leq \frac{2a_1}{(1+a_1)\delta} + \eta \text{ for any } \eta > 0\right\}.$$

Hence, the theorem.

3. Equilibria

To determine the equilibrium points of the system of Eq. 1, we establish a state where the time derivatives of S, I, and P are all set to zero. This yields four equilibrium points in the coordinates (S^*, I^*, P^*), which are given as follows:

1. The trivial equilibrium $E_0(0,0,0)$ which exists only if $a_1 < \mu$.
2. The axial equilibrium $E_1\left(\frac{1}{b_1}, 0,0\right)$, where there are only susceptible plants, which always exist if $\frac{\beta_1}{\mu b_1} < 1$.
3. Disease-free equilibrium point $E_2\left(\frac{a_1 - \mu}{a_1 b_1}, 0,0\right)$. It is seen that the equilibrium E_1 consistently exists if and only if $a_1 > \mu$.

The basic reproduction number (R_0): The basic reproductive number, denoted as R_0 in the mathematical formulation of Eq. 1 set 1, is calculated through the application of the next-generation matrix method as outlined in the paper of Fantaye et al., (2022). R_0 characterizes the average quantity of secondary infections originating from a single infected plant in a population that is entirely susceptible. Now, let $x = (I, P, S)$. then the system of Eq. 1 can be rewritten as:

$$\frac{dx}{dt} = F(x) - V(x), \tag{3}$$

where,

$$F(x) = \begin{bmatrix} \frac{\beta_1 SI}{1+\gamma_1 I} \\ 0 \\ 0 \end{bmatrix}$$

and

$$V(x) = \begin{bmatrix} \mu I + \frac{\beta_2 IP}{1+\gamma_2 P} \\ \alpha P \\ \frac{\beta_1 SI}{1+\gamma_1 I} + cPS + \mu S - a_1 S(1 - b_1 S) \end{bmatrix}.$$

The Jacobian matrices of $F(x)$ and $V(x)$ are given by:

$$F(x) = \begin{bmatrix} \frac{\beta_1 S}{(1+\gamma_1 I)^2} & 0 & \frac{\beta_1 I}{1+\gamma_1 I} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$V(x) = \begin{bmatrix} \mu + \frac{\beta_2 P}{1+\gamma_2 P} & \frac{\beta_2 I}{(1+\gamma_1 P)^2} & 0 \\ 0 & \alpha & 0 \\ \frac{\beta_1 S}{(1+\gamma_1 I)^2} & cS & \frac{\beta_1 I}{1+\gamma_1 I} + cP + \mu - a_1 + 2a_1 b_1 S \end{bmatrix}.$$

At the disease-free equilibrium point $E_2\left(\frac{a_1 - \mu}{a_1 b_1}, 0,0\right)$, the Jacobian matrices of $F(x)$ and $V(x)$ become:

$$F(x) = \begin{bmatrix} \frac{\beta_1(a_1-\mu)}{a_1 b_1} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$V(x) = \begin{bmatrix} \mu & 0 & 0 \\ 0 & \alpha & 0 \\ \frac{\beta_1(a_1-\mu)}{a_1 b_1} & \frac{c\beta_1(a_1-\mu)}{a_1 b_1} & a_1-\mu \end{bmatrix}$$

Using the method of next-generation matrix, the basic reproduction number, R_0 is the spectral radius of FV^{-1} or the dominant eigenvalue of FV^{-1} and thus, the basic reproduction number R_0 is given by:

$$R_0 = \frac{\beta_1(a_1-\mu)}{\mu a_1 b_1}. \quad (4)$$

1. The disease endemic equilibrium point $E^*(S^*, I^*, P^*)$. By simple calculation, we get:

$$S^* = \frac{\mu(1+\gamma_1 I^*)}{\beta_1}, P^* = 0$$

and I^* are the roots of the following quadratic equation

$$\chi_1 I^{*2} + \chi_2 I^* + \chi_3 = 0 \quad (5)$$

where,

$$\chi_1 = a_1 b_1 \mu {\gamma_1}^2 > 0,$$
$$\chi_2 = a_1 b_1 \mu(2\gamma_1 + 1) - \beta_1(\beta_1 + \gamma_1(a_1 - \mu)),$$
$$\chi_3 = -\beta_1(a_1 - \mu).$$

Hence,

$$I^* = \frac{-[a_1 b_1 \mu(2\gamma_1+1) - \beta_1(\beta_1+\gamma_1(a_1-\mu))] \pm \sqrt{[a_1 b_1 \mu(2\gamma_1+1) - \beta_1(\beta_1+\gamma_1(a_1-\mu))]^2 + 4a_1 b_1 \beta_1 \mu {\gamma_1}^2 (a_1-\mu)}}{2a_1 b_1 \mu {\gamma_1}^2}.$$

4. Stability analysis

For the study of the stability properties, the Jacobian matrix J of the system (1) is reported as follows:

$$J = \begin{bmatrix} J_{11} & -\frac{\beta_1 S}{(1+\gamma_1 I)^2} & -cS \\ \frac{\beta_1 I}{1+\gamma_1 I} & J_{22} & -\frac{\beta_2 I}{(1+\gamma_2 P)^2} \\ 0 & 0 & -\alpha \end{bmatrix} \quad (6)$$

where,

$$J_{11} = a_1 - 2a_1 b_1 S - \frac{\beta_1 I}{1+\gamma_1 I} - cP - \mu$$
$$J_{22} = \frac{\beta_1 S}{(1+\gamma_1 I)^2} - \mu - \frac{\beta_2 P}{1+\gamma_2 P}.$$

4.1. Stability of trivial equilibrium point

Theorem 3: The trivial equilibrium point E_0 is stable if $a_1 < \mu$ and unstable otherwise.

Proof: The Jacobian matrix of E_0 is given by

$$J_{E_0} = \begin{bmatrix} a_1-\mu & 0 & 0 \\ 0 & -\mu & 0 \\ 0 & 0 & -\alpha \end{bmatrix}. \quad (7)$$

The values of the above matrix are

$$\lambda_1 = a_1 - \mu, \lambda_2 = -\mu, \lambda_3 = -\alpha.$$

The two eigenvalues λ_2, λ_3 are always negative. Then, for stability, we need to have $\lambda_1 < 0$ i.e., $a_1 < \mu$. Hence, the trivial equilibrium point E_0 is stable if $a_1 < \mu$.

Remark: $a_1 < \mu$ implies that the plant population's natural growth rate is lower than its natural death rate, a scenario that can arise in adverse conditions like forest fires, floods, or landslides. Typically, population models assume that the natural growth rate is higher than the death rate. While mathematically, the trivial equilibrium point is stable under these conditions, in reality, it's an unstable state.

4.2. Stability of axial equilibrium point

Theorem 4: The axial equilibrium point E_1 is stable if $\frac{\beta_1}{\mu b_1} < 1$ and unstable otherwise.

Proof: The Jacobian matrix of E_1 is given by

$$J_{E_1} = \begin{bmatrix} -a_1-\mu & \frac{-\beta_1}{b_1} & \frac{-c}{b_1} \\ 0 & \frac{\beta_1}{b_1}-\mu & 0 \\ 0 & 0 & -\alpha \end{bmatrix}. \quad (8)$$

The values of the above matrix are

$$\lambda_1 = -(a_1+\mu), \lambda_2 = \frac{\beta_1}{b_1} - \mu, \lambda_3 = -\alpha.$$

The two eigenvalues λ_1, λ_3 are always negative. Then, for the stability of the axial equilibrium point E_1, we must have $\lambda_2 < 0$ i.e., $\frac{\beta_1}{b_1} - \mu < 0 \Rightarrow \frac{\beta_1}{\mu b_1} < 1$. Hence, the axial equilibrium point E_1 is stable if $\frac{\beta_1}{\mu b_1} < 1$.

4.3. Local stability of the disease-free equilibrium

Theorem 5: The disease-free equilibrium $E_2\left(\frac{a_1-\mu}{a_1 b_1}, 0, 0\right)$ is locally asymptotically stable if $R_0 < 1$, where $R_0 = \frac{\beta_1(a_1-\mu)}{\mu a_1 b_1}$ is a threshold parameter.

Proof: The Jacobian matrix of E_2 is given by

$$J_{E_2} = \begin{bmatrix} -a_1+\mu & -\frac{\beta_1(a_1-\mu)}{a_1 b_1} & \frac{-c}{a_1 b_1}(a_1-\mu) \\ 0 & \frac{\beta_1(a_1-\mu)}{a_1 b_1}-\mu & 0 \\ 0 & 0 & -\alpha \end{bmatrix}. \quad (9)$$

There are three distinct eigenvalues of the matrix J_{E_2}. One is $\lambda_1 = -(a_1 - \mu) < 0$, the other is $\lambda_2 = -\alpha < 0$ and $\lambda_3 = \frac{\beta_1(a_1-\mu)}{a_1 b_1} - \mu$. Eigenvalues λ_1, λ_2 are always negative. It means that the stability of an equilibrium point E_2 depend upon the value

$\frac{\beta_1(a_1-\mu)}{a_1 b_1} - \mu.$

Now,

$\lambda_3 = \frac{\beta_1(a_1-\mu)}{a_1 b_1} - \mu = \mu\left(\frac{\beta_1(a_1-\mu)}{\mu a_1 b_1} - 1\right)$
$= \mu(R_0 - 1).$

Here, $\lambda_3 = \mu(R_0 - 1) < 0$ if $R_0 < 1$, which implies that all the eigenvalues are negative. Hence, the disease-free equilibrium point E_2 is node and asymptotically stable. It means that the infected population will vanish, and the disease will be eradicated in the plants population. On the other hand, if $R_0 > 1$, then the equilibrium point E_2 is saddle point and unstable.

4.4. Local stability of the endemic equilibrium

Theorem 6: Suppose that $R_0 > 1$, then the endemic equilibrium point E^* is locally asymptotically stable and unstable otherwise.

Proof: To establish the theorem, we utilize a parallel approach as described by Themairi et al. (2020). Let J_{E^*} denote the Jacobian matrix of E^*, then we have:

$$J_{E^*} = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ 0 & 0 & A_{33} \end{bmatrix} \quad (10)$$

where,

$A_{11} = a_1 - 2a_1 b_1 S^* - \frac{\beta_1 I^*}{1+\gamma_1 I^*} - \mu,\ A_{12} = -\frac{\beta_1 S^*}{(1+\gamma_1 I^*)^2},\ A_{13} = -cS^*,$
$A_{21} = \frac{\beta_1 I^*}{1+\gamma_1 I^*},\ A_{22} = \frac{\beta_1 S^*}{(1+\gamma_1 I^*)^2} - \mu,\ A_{23} = -\frac{\beta_2 I^*}{(1+\gamma_2 P^*)^2},\ A_{33} = -\alpha.$

The characteristic equation of the Jacobian matrix J_{E^*} is given by:

$$\varphi^3 + a_1\varphi^2 + a_2\varphi + a_3 = 0, \quad (11)$$

where,

$a_1 = -(A_{11} + A_{22} + A_{33}),$
$a_2 = A_{11}A_{22} + A_{11}A_{33} + A_{22}A_{33} - A_{12}A_{21},$
$a_3 = A_{12}A_{21}A_{33} - A_{11}A_{22}A_{33}.$

Hence,

$a_1 a_2 - a_3 = [-(A_{11} + A_{22} + A_{33})(A_{11}A_{22} + A_{11}A_{33} + A_{22}A_{33}) + A_{12}A_{21}(A_{11} + A_{22})] + A_{11}A_{22}A_{33}. \quad (12)$

Let $W_1 = A_{11}A_{22}A_{33}$. If $A_{11} < 0$, $A_{22} < 0$ and $A_{33} < 0$, then $a_1 > 0, a_3 > 0, W_1 < 0,$

and the first bracket in Eq. 12 is positive. Thus, if

$W_1 < [-(A_{11} + A_{22} + A_{33})(A_{11}A_{22} + A_{11}A_{33} + A_{22}A_{33}) + A_{12}A_{21}(A_{11} + A_{22})],$

then, by using the Routh-Hurwitz criterion, E^* is asymptotically stable.

4.5. Global stability of the disease-free equilibrium

Theorem 7: Suppose that $R_0 < 1$, then the disease-free equilibrium point E_2 is globally asymptotically stable.

Proof: To prove the global stability of the disease-free equilibrium point E_2, we will construct the following Lyapunov Function, which is given by:

$$J(S, I, P) = \frac{1}{2}[(S - S^0) + (I - I^0)]^2. \quad (13)$$

Clearly, $J(S, I, P) \geq 0$ at the disease-free equilibrium and equal to zero whenever $S = S^0$ and $I = I^0$. Then, the derivative of Eq. 13 with respect to time t becomes:

$$\frac{d}{dt}J(S, I, P) = [(S - S^0) + (I - I^0)]\left(\frac{dS}{dt} + \frac{dI}{dt}\right). \quad (14)$$

Substituting the values of $\left(\frac{dS}{dt}\right)$ and $\left(\frac{dI}{dt}\right)$ from the system of Eq. 1 in Eq. 14, we have

$\frac{d}{dt}J(S, I, P) = (S - S^0 + I - I^0)\left(a_1 S - a_1 b_1 S^2 - cPS - \mu S - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P}\right),$
$= -[(S - S^0) + (I - I^0)](V - U).$

Clearly, $\frac{d}{dt}J(S, I, P) \leq 0$ if and only if $V - U > 0$, where $V = a_1 S - a_1 b_1 S^2 - cPS - \mu S - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P}$ and $U = a_1 S$. Moreover, $\frac{d}{dt}J(S, I, P) = 0$ if and only if $S = S^0$ and $I = I^0$. Thus, by the invariance principle of LaSalle (1976), the disease-free equilibrium point E_2 is globally asymptotically stable.

4.6. Global stability of the endemic equilibrium

For examining the global asymptotic stability of the disease endemic equilibrium point E^*, the following model is used.

$a_1 S^* = a_1 b_1 S^{*2} + \frac{\beta_1 S^* I^*}{1+\gamma_1 I^*} + cP^* S^* + \mu S^*,$
$\mu I^* = \frac{\beta_1 S^* I^*}{1+\gamma_1 I^*} - \frac{\beta_2 I^* P^*}{1+\gamma_2 P^*},$
$\alpha P^* = 0.$

Theorem 8: If $R_0 > 1$, then the endemic equilibrium E^* of system (1) exhibits global asymptotic stability in the case where $c = 0$.

Proof: To prove the global stability of the endemic equilibrium E^*, we use the method proposed by

Fantaye and Birhanu (2022) and construct the following Lyapunov Function, which is given by:

$$G(t) = \left(S - S^* - S^* ln \frac{S}{S^*}\right) + \left(I - I^* - I^* ln \frac{I}{I^*}\right) + \left(P - P^* - P^* ln \frac{P}{P^*}\right), \quad (15)$$

after differentiating Eq. 15 with respect to time t, we have:

$$\frac{dG}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{P^*}{P}\right)\frac{dP}{dt}. \quad (16)$$

Now,

$$\left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} = \left(1 - \frac{S^*}{S}\right)\left[a_1 S(1 - b_1 S) - \frac{\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S\right]$$
$$= \left(1 - \frac{S^*}{S}\right)(a_1 S - a_1 S^*)$$
$$= a_1 S\left(1 - \frac{S^*}{S}\right)^2 \quad (17)$$
$$\left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} = \left(1 - \frac{I^*}{I}\right)\left[\frac{\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P}\right]$$
$$= \left(1 - \frac{I^*}{I}\right)[\mu I^* - \mu I]$$
$$= \left(1 - \frac{I^*}{I}\right)\left[-\mu I\left(1 - \frac{I^*}{I}\right)\right]$$
$$= -\mu I\left(1 - \frac{I^*}{I}\right)^2 \quad (18)$$
$$\left(1 - \frac{P^*}{P}\right)\frac{dP}{dt} = -\alpha P\left(1 - \frac{P^*}{P}\right). \quad (19)$$

When the outcomes of Eqs. 17-19 are substituted to Eq. 16, we obtain:

$$\frac{dG}{dt} = a_1 S\left(1 - \frac{S^*}{S}\right)^2 - \mu I\left(1 - \frac{I^*}{I}\right)^2 - \alpha P\left(1 - \frac{P^*}{P}\right),$$
$$= a_1 S - 2a_1 S^* + \frac{a_1 S^{*2}}{S} - \mu I + 2\mu I^* - \frac{I^{*2}}{I} - \alpha P + \alpha P^*,$$
$$= \left[a_1 S + 2\mu I^* + \alpha P^* + \frac{a_1 S^{*2}}{S}\right] - \left[\mu I + 2a_1 S^* + \alpha P + \frac{I^{*2}}{I}\right].$$

Here, $\frac{dG}{dt} \leq 0$ if $\left[a_1 S + 2\mu I^* + \alpha P^* + \frac{a_1 S^{*2}}{S}\right] \leq 0$. Therefore, using the invariance principle of LaSalle (1976), E^* is globally asymptotically stable whenever $R_0 > 1$.

5. Sensitivity analysis

Determining the most sensitive parameters requires knowing the relative importance of the various factors involved in its transmission. We compute the sensitivity index of R_0 for various parameters in the model. These indices indicate how important each parameter is for disease transmission (Rosa and Torres, 2018). The threshold parameter R_0 is a function of four parameters, namely; β_1, b_1, a_1 and μ. The normalized forward sensitivity index of R_0, which is differentiable with respect to a given parameter p, is defined by:

$$\gamma_p^{R_0} = \frac{\partial R_0}{\partial p}\frac{p}{R_0}. \quad (20)$$

The analytical expression for the sensitivity of R_0 can be easily calculated using the explicit formula (20) for each parameter included in it. The sensitivity index values for the parameter values in Table 1 are shown in Table 2. Note that the sensitivity index can depend on several system parameters, but it can also be constant regardless of the parameters. For example, $\gamma_{\beta_1}^{R_0} = +1$ and $\gamma_{b_1}^{R_0} = -1$, which means that increasing (decreasing) β_1, b_1 by a certain percentage will always increase (decrease) R_0 by the same percentage.

Table 2: Sensitivity index table

Parameters	Sensitivity index	Sensitivity index values
β_1	1	1
b_1	-1	-1
a_1	$\frac{\mu}{a_1 - \mu}$	0.11
μ	$\frac{a_1}{\mu - a_1}$	−1.11

From Table 2, we see that the most sensitive parameters are the contact rate of susceptible and infected plants β_1 and the natural death rate of plant populations μ.

The contact rate β_1 reflects how easily a susceptible plant can contract the disease when it comes into contact with an infected plant. A higher β_1 indicates that the disease can spread more rapidly within the population because there is a higher chance of transmission during contact. A lower β_1, on the other hand, implies a slower transmission rate, potentially leading to a more controlled or contained spread of the disease.

The natural death rate μ reflects the rate at which plants naturally die within the population, unrelated to the disease under investigation. A high μ indicates a high natural mortality rate. The sensitivity of this parameter implies that changes in the natural death rate have a significant impact on the dynamics of the plant population, which is essential to consider when studying the disease's effects over time.

6. Numerical analysis

The proposed plant epidemic model is analyzed numerically to observe the behavior of the spread of disease and the role of control measures in the decline of the disease. Numerical analysis is done on Matlab 2018a.

6.1. Parameters and initial conditions

$S(0) = 100$ (100% of plant population), $I(0) = 1$ (10% of plant population infected), $P(0) = 10$(proportion of pesticide used). $a_1 = 0.1$, $b_1 = 0.001$, $\beta_1 = 0.001$, $\gamma_1 = 0.001$, $c = 0.001$, $\mu = 0.06$, $\beta_2 = 0.02$, $\gamma_2 = 0.021$ and $\alpha = 0.02$.

7. Result and discussion

Using the specified parameters and initial conditions, we perform simulations of the model (1) until $t = 1200$. The results are illustrated in Figs. 2-8, considering scenarios both with and without control measures, where the application of pesticides is considered a form of control in the proposed plant epidemic model. Additionally, the stability of the model is depicted through the phase

portraits showcasing the relationship between susceptible and infected plant populations in both Fig. 6 and Fig. 8.

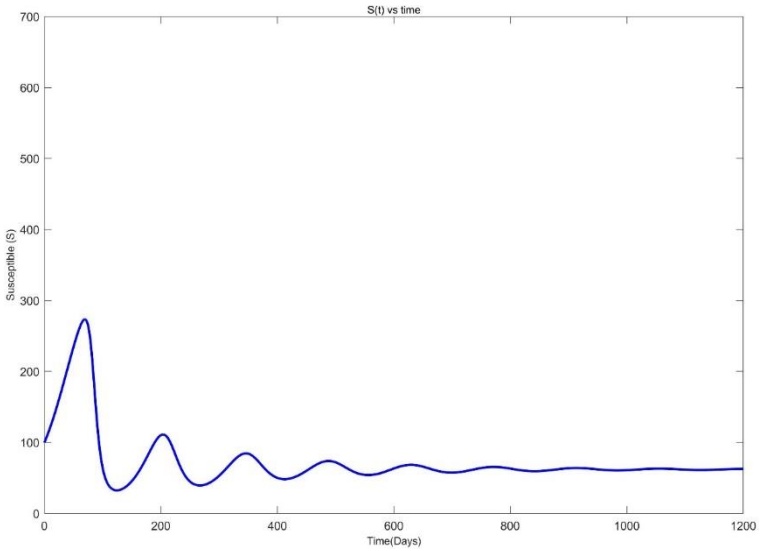

Fig. 2: $S(t)$ vs. time under the application of pesticide

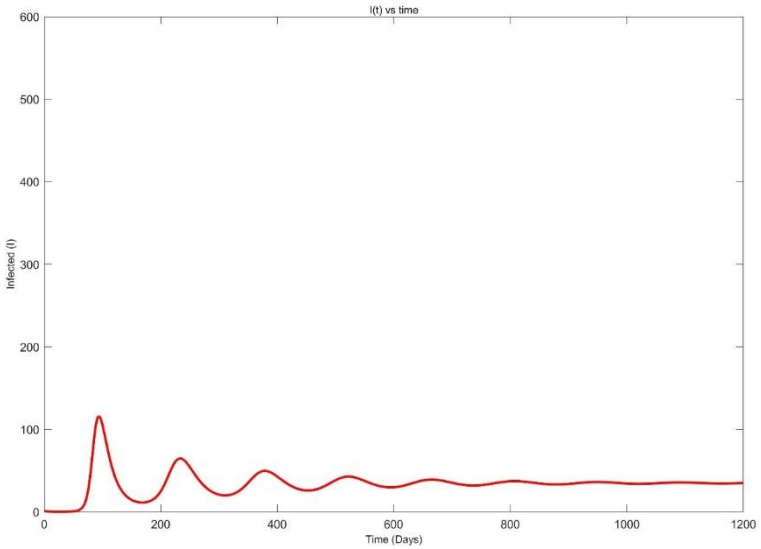

Fig. 3: $I(t)$ vs. time under the application of pesticide

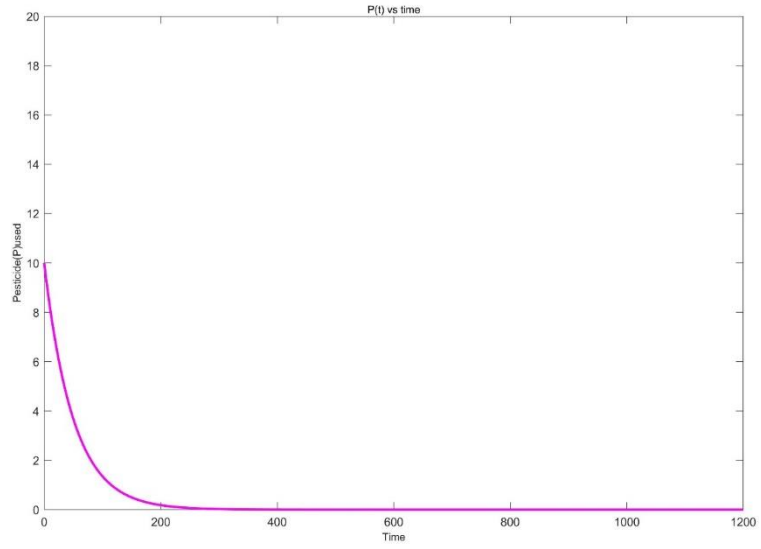

Fig. 4: Amount of pesticides $P(t)$ used vs. time

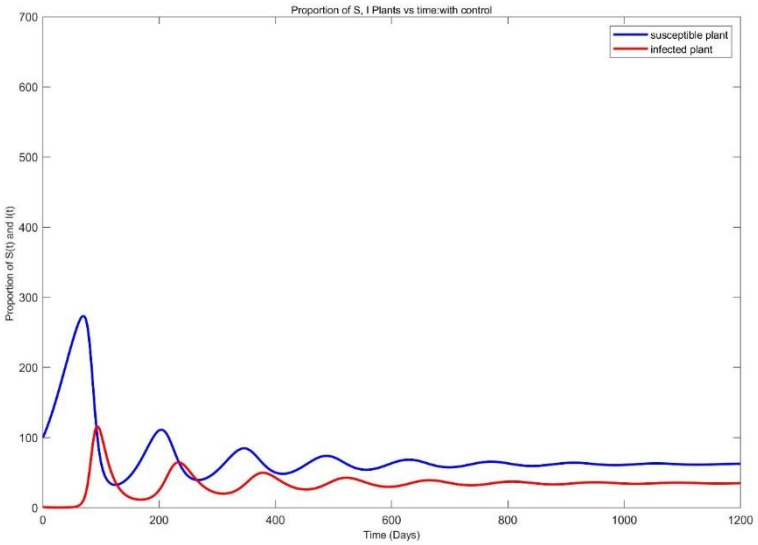

Fig. 5: $S(t)$ and $I(t)$ vs. time under the application of pesticide

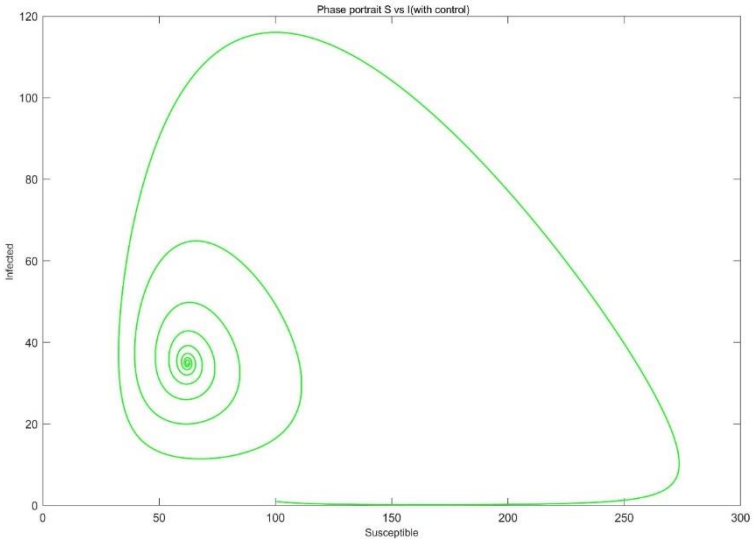

Fig. 6: Phase portrait of $S(t)$ vs. $I(t)$ under the application of pesticide

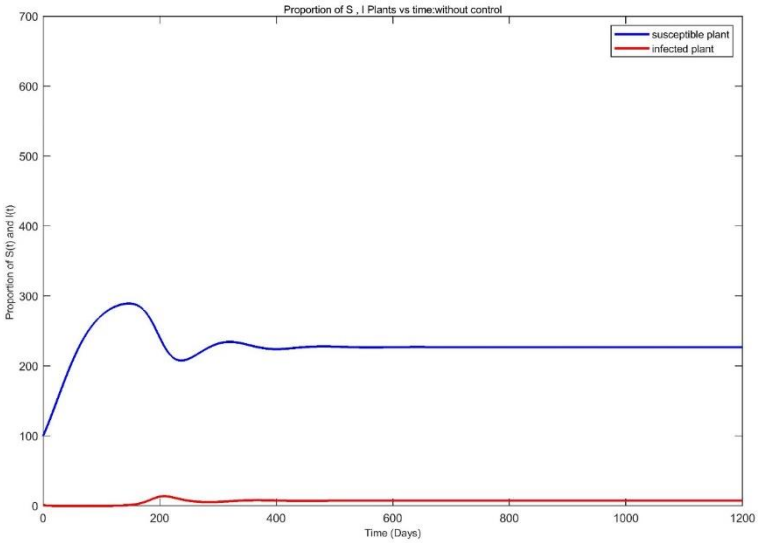

Fig. 7: $S(t)$ and $I(t)$ vs. time without the use of pesticide

In the absence of pesticide application, the susceptible plant population demonstrates resistance to decline, hindering its attainment of a stable equilibrium, as shown in Fig. 2. Meanwhile, the infected plant population experiences an initial rise, eventually adopting a linear pattern without achieving a stable state, as evident in Fig. 3. Notably, it is evident that the absence of control measures leads to a prolonged time frame required for both the susceptible and infected plants to reach a steady

condition. When pesticide is introduced, as depicted in Fig. 5, a decline in plant infections becomes apparent. This results in oscillations of both susceptible and infected plant populations. This phenomenon is illustrated in Fig. 3 and Fig. 5, where the number of infected plants significantly diminishes. However, despite an initial increase in the susceptible plant population, its numbers are also impacted by the pesticide's effects, causing a subsequent decrease. The conceptual framework of the proposed model suggests that the use of pesticides not only curbs infection but may also interfere with the normal growth of the plant population.

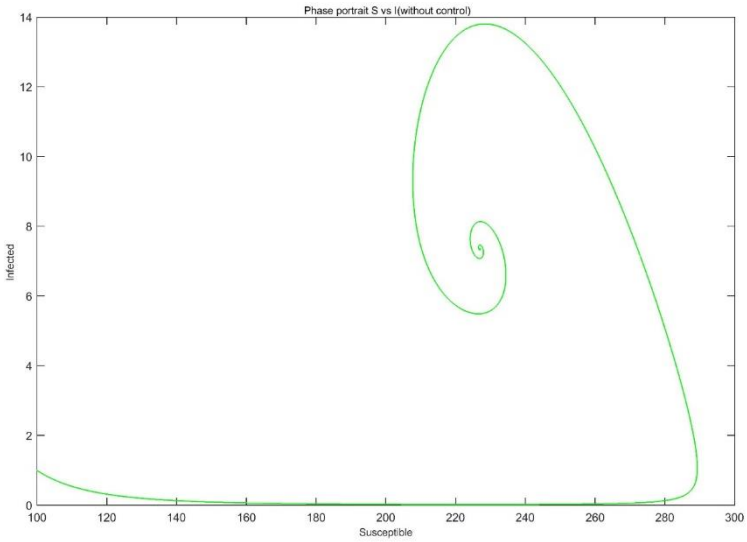

Fig. 8: Phase portrait of $S(t)$ vs. $I(t)$ without the use of pesticide

The phase portrait depicting the relationship between susceptible and infected plants unveils a state of instability in the absence of control measures, as represented in Fig. 8. Conversely when control measures are employed to mitigate the number of infected plants, the trajectory converges toward an equilibrium point, as exemplified in Fig. 6.

8. Conclusion

This paper introduces a detailed model designed to understand the dynamics of epidemics affecting plant populations. The main goal of the model is to explore how effective different control measures are at reducing the impact of these epidemics. Unlike animal or human populations where vaccination is commonly used to control epidemics, plant epidemics are typically managed through the use of pesticides. The application of pesticides, however, not only targets the infected plants but also affects those that are not infected, adding complexity to the situation. This complexity necessitates a sophisticated and nonlinear mathematical model to accurately represent plant epidemics. Within this model, the transmission of susceptibility to infection is assumed to follow a Holling Type II response, which is also used to model the reduction in infection due to pesticide application. Additionally, the model examines the effects of chemical control on the total biomass and the overall health of the plant population. The findings from our analysis are summarized as follows:

1. The positivity and boundedness of the solutions of the system are shown to hold, indicating the system is biologically valid and well-behaved.
2. The point of axial equilibrium, denoted as E_1, represents a scenario where exclusively susceptible plants are present. This equilibrium state exists consistently under the condition $\frac{\beta_1}{\mu b_1} < 1$. In this situation, the disease is not spreading rapidly, and the number of susceptible plants remains stable.
3. Through the utilization of the next-generation matrix technique, we have computed the basic reproduction number, denoted as R_0, which serves as a crucial threshold parameter and determines its value to be $R_0 = \frac{\beta_1(a_1-\mu)}{\mu a_1 b_1}$.
4. If R_0 is less than 1, it signifies the elimination of the infected plant population, resulting in the eradication of the disease from the plant community. On the contrary, if R_0 exceeds 1, there is a heightened probability of disease transmission among different plants within the population, potentially leading to a disease outbreak. A value of R_0 equal to 1 acts as a disease threshold, indicating the disease's sustained presence and stability, although the likelihood of a widespread outbreak or epidemic remains limited.
5. To analyze the stability properties of the system, we utilized and calculated the Jacobian matrix for the system of Eq. 1.
6. We have shown that the disease-free equilibrium E_2 is both locally and globally asymptotically stable in cases where R_0 is less than 1.
7. Utilizing the Routh-Hurwitz criteria, we have established the local asymptotic stability of the endemic equilibrium E^* within the system (1). Furthermore, through the consideration of a Lyapunov function, we have determined the global asymptotic stability of E^*.

8. The model's behavior remains stable near the disease-free and endemic equilibrium points, both on local and global scales. Both susceptible and infected plants display oscillatory behavior before settling into an equilibrium state over time. Without any control measures, it takes longer for both susceptible and infected plants to reach equilibrium. However, using pesticides to control the infection helps both types of plants achieve equilibrium more quickly.
9. To identify the most sensitive parameters, it is crucial to understand the relative significance of the multiple factors contributing to its transmission. Consequently, we have conducted calculations for the sensitivity index of R_0 concerning various parameters within the model. These indices provide insight into the individual significance of each parameter in the context of disease transmission. It has been determined that the most sensitive parameters in our model are the contact rate of susceptible and infected plants, denoted as β_1, and the natural death rate of plant populations, represented by μ. Identifying and understanding these sensitive parameters is crucial for making informed decisions and interventions in disease control and plant population management. It allows researchers and policymakers to develop targeted strategies that are more likely to be effective in controlling disease outbreaks, preserving plant populations, and maintaining ecosystem health.
10. In conclusion, numerical simulations have been conducted to validate and further support the analytical conclusions presented within the study.

The stability and control of plant epidemic models under pesticide application are complex and multifaceted issues. This research offers novel insights that bridge the gap between disease management and ecosystem preservation. By optimizing pesticide dosages, identifying disease hotspots, and understanding the nonlinear dynamics at play, we present a comprehensive framework to guide sustainable management strategies in the plant community. Our findings have far-reaching implications for the conservation of ecosystems and the protection of plant populations against infectious diseases.

Compliance with ethical standards

Conflict of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

References

Anguelov R, Dufourd C, and Dumont Y (2017). Mathematical model for pest-insect control using mating disruption and trapping. Applied Mathematical Modelling, 52(1): 437-457. https://doi.org/10.1016/j.apm.2017.07.060

Arino O, Abdllaoui AE, Mikram J, and Chattopadhyay J (2004). Infection in prey population may act as a biological control in ratio-dependent predator-prey models. Nonlinearity, 17(3): 1101-1116. https://doi.org/10.1088/0951-7715/17/3/018

Bacaër N (2011). Lotka, Volterra and the predator–prey system (1920–1926). In: Bacaër N (Ed.), A short history of mathematical population dynamics: 71–76. Springer, London, UK. https://doi.org/10.1007/978-0-85729-115-8_13

Bairagi N and Adak D (2015). Complex dynamics of a predator–prey–parasite system: An interplay among infection rate, predator's reproductive gain and preference. Ecological Complexity, 22: 1-12. https://doi.org/10.1016/j.ecocom.2015.01.002

Birkhoff G and Rota GC (1982). Ordinary differential equations. Ginn, Boston, USA.

Brauer F (2005). The Kermack–McKendrick epidemic model revisited. Mathematical Biosciences, 198(2):119-131. https://doi.org/10.1016/j.mbs.2005.07.006 **PMid:16135371**

Chowdhury J, Basir FA, Takeuchi Y, Roy PK, and Ghosh M (2019). A mathematical model for pest management in Jatropha curcas with integrated pesticides - An optimal control approach. Ecological Complexity, 37: 24-31. https://doi.org/10.1016/j.ecocom.2018.12.004

Fantaye AK and Birhanu ZK (2022). Mathematical model and analysis of corruption dynamics with optimal control. Journal of Applied Mathematics, 2022: 8073877. https://doi.org/10.1155/2022/8073877

Fantaye AK, Goshu MD, Zeleke BB, Gessesse AA, Endalew MF, and Birhanu ZK (2022). Mathematical model and stability analysis on the transmission dynamics of skin sores. Epidemiology and Infection, 150: e207. https://doi.org/10.1017/S0950268822001807 **PMid:36397272 PMCid:PMC9987028**

Hilker FM and Schmitz K (2008). Disease-induced stabilization of predator–prey oscillations. Journal of Theoretical Biology, 255(3): 299-306. https://doi.org/10.1016/j.jtbi.2008.08.018 **PMid:18801376**

Hsieh YH and Hsiao CK (2008). Predator-prey model with disease infection in both populations. Mathematical Medicine and Biology: A Journal of the IMA, 25(3):247-266. https://doi.org/10.1093/imammb/dqn017 **PMid:18701422 PMCid:PMC7108608**

Hugo A and Simanjilo E (2019). Analysis of an eco-epidemiological model under optimal control measures for infected prey. Applications and Applied Mathematics: An International Journal, 14(1): 117-138.

Kar TK (2005). Stability analysis of a prey-predator model incorporating a prey refuge. Communications in Nonlinear Science and Numerical Simulation, 10(6): 681–691. https://doi.org/10.1016/j.cnsns.2003.08.006

Kermack W and McKendrick A (1991). Contributions to the mathematical theory of epidemics – I. Bulletin of Mathematical Biology, 53(1–2): 33–55. https://doi.org/10.1016/S0092-8240(05)80040-0 **PMid:2059741**

Kermack WO and McKendrick AG (1927). A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, 115(772): 700-721. https://doi.org/10.1098/rspa.1927.0118

Lasalle JP (1976). The stability of dynamical systems. Society for Industrial and Applied Mathematics, Philadelphia, USA.

Lotka AJ (1910). Contribution to the theory of periodic reactions. The Journal of Physical Chemistry, 14(3): 271-274. https://doi.org/10.1021/j150111a004

Malthus T (2023). An essay on the principle of population. In: Hough P (Ed.), British Politics and the Environment in the Long Nineteenth Century: 77-84. Routledge, London, UK.

Ofuoku AU, Egho EO, and Enujeke EC (2009). Integrated pest management (IPM) adoption among farmers in central agro-ecological zone of delta state, Nigeria. Advances in Biological Research, 3(1-2): 29-33.

Overton K, Hoffmann AA, Reynolds OL, and Umina PA (2021). Toxicity of insecticides and Miticides to natural enemies in Australian grains. Insects, 12(2): 187. https://doi.org/10.3390/insects12020187 **PMid:33671702 PMCid:PMC7927080**

Pal AK (2020). Effect of fear on a modified Lesli-Gower predator-prey eco-epidemiological model with disease in predator. Journal of Applied Mathematics and Informatics, 38(5-6): 375-406.

Pal AK and Samanta GP (2010). Stability analysis of an eco-epidemiological model incorporating a prey refuge. Nonlinear Analysis: Modelling and Control, 15(4): 473-491. https://doi.org/10.15388/NA.15.4.14319

Purnomo AS, Darti I, and Suryanto A (2017). Dynamics of eco-epidemiological model with harvesting. AIP Conference Proceedings, AIP Publishing, Malang, Indonesia, 1913(1): 020018. https://doi.org/10.1063/1.5016652

Rosa S and Torres DFM (2018). Parameter estimation, sensitivity analysis and optimal control of a periodic epidemic model with application to HRSV in Florida. Statistics Optimization and Information Computing, 6(1): 139-149. https://doi.org/10.19139/soic.v6i1.472

Schechtman H, Valle D, and Souza MO (2020). From resistance to persistence: Insights of a mathematical model on the indiscriminate use of insecticide. PLOS Neglected Tropical Diseases, 14(11): e0008862. https://doi.org/10.1371/journal.pntd.0008862 **PMid:33206645 PMCid:PMC7723293**

Shorbaji FA, Roche B, Britton R, Andreou D, and Gozlan R (2017). Influence of predation on community resilience to disease. Journal of Animal Ecology, 86(5): 1147-1158. https://doi.org/10.1111/1365-2656.12722 **PMid:28758196**

Themairi AA and Alqudah MA (2020). Predator-prey model of Holling-type II with harvesting and predator in disease. Italian Journal of Pure and Applied Mathematics, 43: 744-753.