Chapter 4

Stability and control of a plant epidemic model with pesticide intervention

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4.1 Introduction

Plant epidemic models may help control diseases that spread throughout plant populations. These models have the potential to enhance agricultural productivity and ecological equilibrium, leading to a reduction in pesticide intervention rates, a reduction in agricultural expenses, and a decrease in disease development. Only two factors affect the effectiveness of these interventions: disease transmission dynamics and ecological interactions between plant populations. Our research areas include pesticide control and plant epidemics. Pesticide applications play a critical role in the ongoing battle against the spread of infectious diseases within plant populations and

the preservation of ecological systems. The application of pesticides plays a crucial role. Understanding the dynamics of plant epidemic models under the influence of pesticide intervention holds significant importance for the effective control of diseases and the management of ecosystems. This paper explores a novel area by examining the stability and control of a plant epidemic model in the presence of pesticide applications. The results of this study provide valuable and innovative insights that can assist policymakers and ecologists in making well-informed decisions pertaining to disease management and the preservation of ecological systems. Prey-predator models, in which an epidemic affect either the prey or the predators, serve as the primary basis for the research on plant epidemic models. In these models, the populations of prey or predators are divided into compartments, such as susceptible and infective. Additionally, these models allow for the implementation of a wide range of control variables, such as medication and removal through killing (e.g., bird flu, swine flu, etc.). However, the focus of this study is on the ecoepidemic model related to plant populations, specifically the epidemics that occur in plantations such as tea and rice. It is important to note that no previous research has considered the plant epidemic model under the application of pesticides, making this work a novel contribution to the field. By examining a model of a plant epidemic with pesticide intervention, this study offers a comprehensive analysis. To discover a long-term solution to agricultural diseases, the model measures disease stability and control. This study investigates ecological dynamics and agricultural life as part of a long-term plan to combat disease [83].

4.2 Assumptions of Plant Epidemic Model

The disease epidemic model that we are examining is constructed on a few essential assumptions that assist us in understanding the complex mechanisms that govern the propagation of diseases and the expansion of disease populations within plant ecosystems. A distinction is made between plants that are susceptible and plants that are infected. According to the conceptual terms, biological processes are responsible for slowing down the growth rate of plant populations that are both susceptible and infected. When planning the development of logistics, it is critical to consider population growth as well as resource availability.

According to this model, diseases are transmitted naturally between plants that are susceptible and plants that are unwell. This transmission mechanism is frequently represented by the contact rates between susceptible and the infection rates per contact. For the purpose of demonstrating this connection, the saturation effect is frequently used. The number of susceptible hosts has a negative impact on the rate at which the disease spreads. The Holling reaction rate of type II is utilised [87].

The plant epidemic rate plant disease model, which is supported by these assumptions, is utilised for the purpose of studying the dynamics of epidemic transmission and the usefulness of the model. It is important to exercise caution when utilising the results because the modifications may lead to a reduction in the model's ability to understand complex plant ecosystems [91]. For the purpose of avoiding these problems, one could continue to assume that plant populations mix equally and that the success of ecosystems is determined by slow or immediate ecosystem dynamics, while ignoring the geographical and temporal variation that exists within ecosystems [97].

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

i. 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 $S(t)$.

ii. 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 $I(t)$.

iii. 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 the rate $a_1 S(1 - b_1 S)$.

iv. When a disease exists, the plant populations get categorized into two disjoint classes which changes with time t: the susceptible plants, labelled as $S(t)$, and the infected plants, labelled as $I(t)$. Consequently, at any time t, the overall population can be expressed as $S(t) + I(t) = N(t)$.

v. 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.

vi. 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.

vii. The interaction between susceptible and infected plants is modelled 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, $\frac{\beta_2 IP}{1+\gamma_2 P}$.

viii. All the model parameters are assumed to be non-negative.

4.3 Mathematical Model

4.3.1 Mathematical model, including the differential equations

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 Figure 4.1, while Table 1 provides the notations and descriptions for the model parameters.

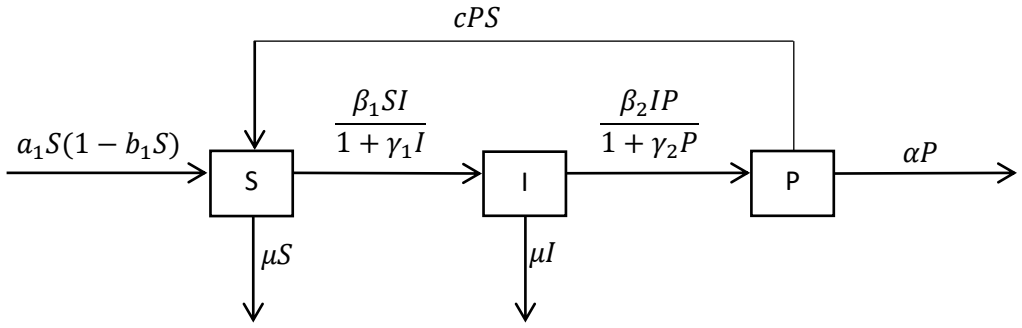

Figure 4.1: Transfer diagram of model (4.1)

Table 4.1: 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 damages 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 pesticide.
α	The rate at which pesticides is being used.

From Figure 4.1, it can be seen that the mathematical model will be governed based on the following system of equations:

$$\begin{aligned} \frac{dS}{dt} &= a_1 S(1 - b_1 S) - \frac{\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S, \\ \frac{dI}{dt} &= \frac{\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P}, \\ \frac{dP}{dt} &= -\alpha P. \end{aligned} \tag{4.1}$$

From the biological point of view, we are only interested in the dynamics of system (4.1) in the closed octant R_+^3. Thus, we consider the initial conditions $S(0) \equiv S_0 > 0, I(0) \equiv I_0 > 0$ and $P(0) \equiv P_0 > 0$.

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

Using differential equations to explain the connections between susceptible (S) and infected (I) plant groups and the application of pesticides (P), the mathematical model looks at the dynamics of disease spread within plant populations. This model integrates the natural growth rate of the susceptible population, denoted by a_1, and the carrying capacity, $1/b_1$, to describe logistic growth: $dS/dt = a_1S(1 - b_1S) - \beta_1SI/(1 + \gamma_1I) - cPS - \mu S$. In order to account for the effectiveness of disease spread as the size of the infected population grows, the equation includes the infection process, which is affected by the contact rate β_1 and changed by the Holling type II functional response.

The rate of infection in plants is expressed by the equation $dI/dt = \beta_1SI/(1 + \gamma_1I) - \mu I - \beta_2IP/(1 + \gamma_2P)$, which shows how plants go from being susceptible to being resistant to infection. This change is also influenced by pesticide application. The effectiveness of pesticides in preventing infection is taken into account in this formulation along with the average plant death rate (μ) for both susceptible and infected plants (β_2). The rate at which the pesticides are taken out of the system or break down is shown. Input $dP/dt = -\alpha P$ to set the dynamics of the pesticide.

Two model factors, $\gamma_1$and γ_2, show that the rates are not going in a straight line. The changing factors for disease growth and pesticide effectiveness are demonstrated by these measures. The model shows a normal growth rate (a_1) and a carrying capacity ($1/b_1$). It appears to be built on ecological concepts as a result. The term "common pesticide situation" (cPS) was chosen to provide a full picture of the ecological system and to highlight the potential harm that pesticides may cause to plants that are susceptible to them. This model provides a strong framework to evaluate the dynamics of plant diseases in response to the rate of pesticide intervention, which is important for identifying the most effective management methods.

4.3.2 Model's significance and how it simulates plant epidemic dynamics

The effects of pesticide intervention on plant epidemic dynamics were examined in this study using a mathematical model. To model how populations of susceptible and infected plants interact in an agricultural ecosystem, non-linear ordinary differential equations (ODEs) are used. By looking at the Holling type II functional reaction, we can see how closely the rates of disease spread are linked to the number of plants that are susceptible. It can be stated that the plant population is either susceptible (S) or infected (I) for the purposes of using the model. The rate of pesticide introduction is shown by the variable that can be controlled (P).

It is necessary to understand the difficulty of stopping disease spread within plant populations for this model to be effective in the long term. The release of new data has enabled a thorough analysis of how pesticide use influences the spread of epidemics in these populations. An analysis of the effects of pesticides in the model demonstrates how to keep plant populations stable while lowering epidemic rates. This framework is important because an excess of chemicals could prevent the disease from spreading, but at the expense of the ecological balance or the population of susceptible plants [98].

The rapid development rate of plants, the rapid propagation of diseases, and the exponential plant death rate brought on by pests are all taken into consideration in the mathematical formulation. This model accurately captures epidemic dynamics, making it useful for evaluating plant management effectiveness. Policymakers may use these dynamics to assist with decision-making on the timing and amount of pesticide application in order to reduce the frequency of diseases while maintaining ecological equilibrium and plant health.

To ensure that the analysis is based on solid facts, the study includes thorough information about pesticide introduction and the plant populations affected by and susceptible to these chemicals. This comprehensive approach guarantees a thorough understanding of their interactions. To prevent incurable plant diseases and reduce environmental effects, researchers investigate various pesticide intervention strategies, such as altering treatment rates or timing. By experimenting with these variables, they are hoping to discover the most efficient strategies for disease control while minimising environmental consequences, supporting sustainable farming practices that safeguard both plant health and the ecosystem.

4.4 Positivity and Boundedness

4.4.1 Positivity

Theorem 4.1: Let $S(0) > 0, I(0) > 0, P(0) > 0$. This implies all solutions of the system represented by (4.1) 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 (4.1). Following a similar approach used in [84], we obtain the inequality expression from the1st equation of model (4.1) as follows:

$\frac{dS}{dt} \leq a_1 S(1 - b_1 S)$, which, when simplified gives:

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

Now, as $t \to \infty$, we obtain $0 < S \leq \frac{1}{b_1}$. Hence, the solution of system (4.1) is feasible in the region $\Omega = \{S, l, P\}$.

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

Hence, the theorem stands proved.

4.4.2 Boundedness

Theorem 4.2: All solutions of system (4.1) that start in $R_+^{\ 3}$ are uniformly bounded.

Proof: Let $S(t), I(t), P(t))$ be any solution of the system (4.1). Since, $\frac{dS}{dt} \leq a_1 S(1 - b_1 S)$. We have,

$\lim_{t \to \infty} \sup S(t) \leq a_1$.

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 et al., [27] on the above differential inequalities, we obtain:

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

Now, as $t \to \infty$, we obtain $0 \leq W \leq \frac{2a_1}{(1+a_1)\delta}$.

Thus, all the solutions of (4.1) lie in the region: $\Omega = \left\{(S, 1, P): 0 \leq W \leq \frac{2a_1}{(1+a_1)\delta} + \eta \text{ for any } \eta > 0\right\}$.

Hence, the theorem stands proved.

Plant epidemic model results must be biologically accurate and helpful; hence, positivity and boundedness are essential. Due to biological restrictions, plant populations cannot have negative numbers, and positivity makes sure that both infected and susceptible plant populations are positive throughout the simulation. As population numbers grow, boundedness indicates that the environment has limited resources and a carrying capacity [115].

These characteristics are significant for a variety of reasons. The first phase, known as positivity, verifies the physical and biological consistency of the model's conclusions. Assumptions made by the model would be incorrect in the event that there were negative populations, which would be a violation of epidemiology and population dynamics. Boundedness maintains the model's claims by restricting the population's growth. Population growth may continue to rise, but not at a sustainable rate unless there are restrictions placed on it. This does not happen in the realm of ecological processes.

Real-life positivity and boundedness have an impact on the strategies that we expect to use to combat diseases. In the event that these parameters are not adhered to, the model may result in

unusual disease populations reporting unfavourable results. This could lead to incorrect conclusions regarding the spread of the disease and the success of the intervention. As long as the model's solutions remain positive and limited, researchers are able to accurately evaluate the effectiveness of disease management strategies and make intelligent decisions regarding disease outbreak control [117].

Population positivity and boundedness indicate long-term disease dynamics. Keeping these traits, a model shows disease populations move towards or near equilibrium. This indicates that disease dynamics and target populations match. Failure to maintain positivity and boundedness may suggest unbound disease dynamics. By using the correct intervention measures, the system may be rectified.

4.5 Equilibria

To determine the equilibrium points of the system of equations (4.1), 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:

(i) The trivial equilibrium $E_0(0,0,0)$ which exists only if $a_1 < \mu$.

(ii) The axial equilibrium $E_1\left(\frac{1}{b_1}, 0,0\right)$, where there are only susceptible plants, which always exist if $\frac{\beta_1}{\mu b_1} < 1$.

(iii) 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 equation set (4.1), is calculated through the application of the next-generation matrix method as outlined in the paper of Fantaye et al., (2022) [54]. 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 equation (4.1) can be rewritten as:

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

Where,

$$F(x) = \begin{bmatrix} \frac{\beta_1 SI}{1+\gamma_1 I} \\ 0 \\ 0 \end{bmatrix} \text{ and } V(x) = \begin{bmatrix} \mu I + \frac{\beta_2 IP}{1+\gamma_2 P} \\ \alpha P \\ \frac{\beta_1 SI}{1+\gamma_1 I} + cPS + \mu S - a_1 S(1 - b_1 S) \end{bmatrix}.$$

The Jacobian matrices of $F(x)$ and $V(x)$ is 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 $V(x)$ becomes:

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

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:

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

(iv) 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:

$$\chi_1 I^{*2} + \chi_2 I^* + \chi_3 = 0,$$

Where,

$$\chi_1 = a_1 b_1 \mu \gamma_1^2 > 0,$$

$$\chi_2 = a_1 b_1 \mu(2\gamma_1 + 1) - \beta_1\big(\beta_1 + \gamma_1(a_1 - \mu)\big),$$

$$\chi_3 = -\beta_1(a_1 - \mu).$$

Hence, $I^* = \frac{-[a_1 b_1 \mu(2\gamma_1+1) - \beta_1(\beta_1+\gamma_1(a_1-\mu))] \pm \sqrt{[a_1 b_1 \mu(2\gamma_1+1) - \beta_1(\beta_1+\gamma_1(a_1-\mu)]^2 + 4a_1 b_1 \beta_1 \mu \gamma_2^2 (a_1-\mu)}}{2a_1 b_1 \mu \gamma_1^2}$.

The two primary states of equilibrium that are depicted by the model are an equilibrium that is free of disease and an equilibrium that is endemic. Disease is unable to develop below a particular point due to the presence of certain factors. This creates a state of equilibrium free from disease. It appears that this population does not contain any infectious plants at the moment. When it comes to determining the stability of the system, this limit, which is represented by the basic reproduction number R_0, is an essential component. When R_0 goes below 1, the disease is no longer able to spread through the plant population. This indicates the absence of any diseases within the population. The plant population is completely sensitive to spread, but it is healthy in this equilibrium, which is why the system is naturally resistant to disease progression. This equilibrium is stable, as demonstrated by the mathematical foundations that support it; it is asymptotically stable both locally and globally, which suggests that any slight deviation from this state brought about by the introduction of the disease will cause the system to naturally return to its disease-free state over the course of time [130].

Once R_0 goes above 1, however, the endemic equilibrium state becomes significant. As a result, each affected plant typically infects more than one other plant with the disease. As a result, the disease persists in the plant population. This disease indicates a crucial phase in the formation of a stable plant population within the ecosystem. The model's term describes the number of susceptible and damaged plants. This equilibrium point provides insights into the long-term

dynamics of disease survival within plant populations, highlighting situations where the disease remains endemic despite intervention efforts [131].

The examination of the profound biological impacts of these disease processes provides a foundation for understanding the dynamics of plant diseases and the methods used to control them. A disease-free equilibrium is the ideal situation that disease prevention and control efforts strive to achieve. In this equilibrium, the plant population is completely free of any disease that could potentially affect it. On the other hand, when diseases become endemic within an ecosystem, it becomes more difficult to achieve a state of eco-equilibrium. The term "eco-equilibrium" refers to a state of equilibrium in which efforts are made to prevent the emergence of disease and measures are taken to control the spread of disease if it has already occurred. Under these circumstances, the disease continues to be present throughout the population, but it is possible to control it by implementing intervention strategies that are persistent and consistent. This continuous effort is essential in order to keep the disease at a manageable level, prevent it from becoming out of control, and work towards the goal of protecting the overall health of the plant population [134].

4.6 Stability Analysis

For the study of the properties of stability, the Jacobian matrix J of the system (4.1) is as follows:

$$J = \begin{bmatrix} J_{11} & \frac{-\beta_1 S}{(1+\gamma_1 I)^2} & -cS \\ \frac{\beta_1 I}{1+\gamma_1 I} & J_{22} & \frac{-\beta_2 I}{(1+\gamma_2 P)^2} \\ 0 & 0 & -\alpha \end{bmatrix} \tag{4.2}$$

Where,

$$J_{11} = a_1 - 2a_1 b_1 S - \frac{\beta_1 I}{1+\gamma_1 I} - cP - \mu,$$

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

4.6.1 Stability of trivial equilibrium point

Theorem 4.3: 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:

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

Eigenvalues of the above matrix are $\lambda_1 = a_1 - \mu_1,\ \lambda_2 = -\mu_1,\ \lambda_3 = -\alpha.$

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.

4.6.2 Stability of axial equilibrium point

Theorem 4.4: 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:

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

Eigenvalues 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 stability of the axial equilibrium point E_1, we must have $\lambda_2 < 0$ ie., $\frac{\beta_1}{b_1} - \mu < 0 \Rightarrow \frac{\beta_1}{\mu b_1} < 1$.

Hence, the axial equilibrium point E_1 is stable if $\frac{\beta_1}{\mu b_1} < 1$.

4.6.3 Local stability of the disease-free equilibrium

Theorem 4.5: 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:

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

There are three distinct eigenvalues of matrix J_{E_2}. One is $\lambda_1 = -(a_1 - \mu) < 0$, the other ones are given by $\lambda_2 = -\alpha < 0$ 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 be vanished and the disease will be eradicated in the plant population. On the other hand, if $R_0 > 1$, then the equilibrium point E_2 is saddle point and unstable.

4.6.4 Local stability of the endemic equilibrium

Theorem 4.6: Suppose that $R_0 > 1$, then the endemic equilibrium point E^* is locally asymptotically stable and unstable otherwise.

Proof: To establish the theorem, we utilise a parallel approach as described by Themairi et al, [182]. Let J_{E^*} denote the Jacobian matrix of E^*, then we have:

$$J_{E^*} = \begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ 0 & 0 & A_{33} \end{bmatrix} \tag{4.3}$$

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:

$$\varphi^3 + x_1\varphi^2 + x_2\varphi + x_3 = 0 \tag{4.4}$$

Where:

$$x_1 = -(A_{11} + A_{22} + A_{33}),$$

$$x_2 = A_{11}A_{22} + A_{11}A_{33} + A_{22}A_{33} - A_{12}A_{21},$$

$$x_3 = A_{12}A_{21}A_{33} - A_{11}A_{22}A_{33}.$$

Hence,

$$x_1x_2 - x_3 = [-(A_{11} + A_{22} + A_{33})(A_{11}A_{22} + A_{11}A_{33} + A_{22}A_{33}) + A_{12}A_{21}(A_{11} + A_{22})] + A_{11}A_{22}A_{33}. \tag{4.5}$$

Let $W_1 = A_{11}A_{22}A_{33}$. If $A_{11} < 0, A_{22} < 0$ and $A_{33} < 0$, then $x_1 > 0, x_3 > 0, w_1 < 0$, and the first bracket in (4.5) 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 Routh-Hurwitz criterion, E^* is asymptotically stable.

4.6.5 Global stability of the disease-free equilibrium

Theorem 4.7: 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:

$$J(S,I,P) = \frac{1}{2}[(S - S^0) + (I - I^0)]^2 \tag{4.6}$$

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 equation (4.6) with respect to time t becomes:

$$\frac{d}{dt}J(S,I,P) = [(S - S^0) + (I - I^0)]\left(\frac{dS}{dt} + \frac{dI}{dt}\right) \tag{4.7}$$

Substituting the values of $\left(\frac{dS}{dt}\right)$ and $\left(\frac{dI}{dt}\right)$ from the system of equation (4.1) in (4.7), 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, $\frac{d}{dr}J(S,I,P) = 0$ if and only if $S = S^0$ and $I = I^0$.

Thus, by the invariance principle of LaSalle [112], the disease-free equilibrium point E_2 is globally asymptotically stable.

4.6.6 Global Stability of the endemic equilibrium

In order to examine the global asymptotic stability for the disease endemic equilibrium point E^*, the following model is used.

$$a_1 S^* = a_1 b_1 S^{*2} + \frac{\beta_1 S^* I^*}{1+\gamma_1 I^*} + cP^* S^* + \mu S^*$$

$$I^* = \frac{\beta_1 S^* I^*}{1+\gamma_2 I^*} - \frac{\beta_2 I^* P^*}{1+\gamma_2 P^*}$$

$$\alpha P^* = 0.$$

Theorem 4.8: If $R_0 > 1$, then the endemic equilibrium E^* of system (4.1) exhibits global asymptotic stability in the case where $c = 0$.

Proof: To prove the global stability corresponding to the endemic equilibrium E^*, the method proposed by Rosa and Torres [153] can be used and followed by constructing the following Lyapunov Function:

$$G(t) = \left(S - S^* - S^* \ln \frac{S}{S^*}\right) + \left(I - I^* - I^* \ln \frac{I}{I^*}\right) + \left(P - P^* - P^* \ln \frac{P}{P^*}\right) \tag{4.8}$$

After differentiating equation (4.8) with respect to time t, we have:

$$\frac{dG}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{P^*}{P}\right)\frac{dP}{dt}. \tag{4.9}$$

Now,

$$\left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} = \left(1 - \frac{S^*}{S}\right)\left[a_1 S(1 - b_1 S) - \frac{\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S\right],$$

$$= \left(1 - \frac{S^*}{S}\right)(a_1 S - a_1 S^*),$$

$$= a_1 S\left(1 - \frac{S^*}{S}\right)^2. \tag{4.10}$$

$$\left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} = \left(1 - \frac{I^*}{I}\right)\left[\frac{\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P}\right],$$

$$= \left(1 - \frac{I^*}{I}\right)[\mu I^* - \mu I],$$

$$= \left(1 - \frac{I^*}{I}\right)\left[-\mu I\left(1 - \frac{I^*}{I}\right)\right],$$

$$= -\mu I\left(1 - \frac{I^*}{I}\right)^2. \tag{4.11}$$

$$\left(1 - \frac{P^*}{P}\right)\frac{dP}{dt} = -\alpha P\left(1 - \frac{P^*}{P}\right). \tag{4.12}$$

When the outcomes of equations (4.10) -(4.12) are substituted to equation (4.9), 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 [112], E^* is globally asymptotically stable whenever $R_0 > 1$.

The plant epidemic model's term stability analysis examines how the system behaves around the equilibrium points to determine whether they are stable or not. This could happen in two ways: the disease-free equilibrium (DFE) or the endemic equilibrium (EE). It is called the disease-free equilibrium (DFE) when there are no unhealthy plants. It is also called the endemic equilibrium (EE) when the disease stays in the population [135].

Before analysing the eigenvalues of the resulting Jacobian matrix, the stability analysis begins with the disease-free equilibrium and involves linearizing the system of nonlinear ordinary differential equations (ODEs) around the DFE point. The eigenvalues can provide information about the local stability of the equilibrium point. The DFE is locally asymptotically stable if all of its eigenvalues have real parts that are negative. With these small changes, the system will finally get back to a state where it is free of disease [136].

Mathematically, the stability criteria for a disease-free equilibrium are as follows:

If Re $(\lambda_i) < 0$ for all i, then the DFE is locally asymptotically stable.

In the same way, the stability of the endemic equilibrium is studied by making the system linear around the EE point and checking the Jacobian matrix's eigenvalues. Asymptotically, the EE is stable in its local area if all of its eigenvalues have negative real parts. The disease affects the population for a long time.

It's possible to state this as the need for stability in an endemic equilibrium:

Re $(\lambda_i) < 0$ for all i, then the EE is asymptotically stable in that area.

A global stability analysis is also carried out to find out if the term of the equilibrium points holds true for the whole system or just the area around them. From any point of view, we need to look at how the system works as a whole and see if it tends to move towards the equilibrium points.

The term basic reproduction number (R_0) is important for determining the stability of equilibrium points. The disease-free equilibrium remains stable almost until the end if R_0 is less than 1. This demonstrates that each unhealthy plant results in less than one secondary infection. When R_0 exceeds 1, the stable endemic equilibrium globally approaches near-stability. Now, the disease can spread through the population.

Researchers may evaluate the potential effectiveness of various disease control measures by using the results of the disease analysis. Making pesticides more widely accessible or fostering plant kinds that are resistant to them are two ways to limit pesticide transmission and preserve a disease-free condition within the population. If the disease-free equilibrium is stable, these measures may be used. This allows for the efficient containment of disease epidemics. A stable disease equilibrium rate, in contrast, indicates that the disease will most likely persist despite intervention attempts and that continuous management methods are required to mitigate its impacts.

Plant stability analysis helps with disease management strategy development by providing important information about the dynamics of disease transmission in plant populations. Researchers may assess the potential effectiveness of various interventions and make wise judgments to manage epidemics by looking at the stability of equilibrium points and the fundamental epidemic decision [138].

4.7 Sensitivity Analysis

Determining the most sensitive parameters requires knowing the relative importance of the various factors involved in its transmission. The sensitivity index of R_0 is computed for various

parameters in the model. These indices indicate how important each parameter is for disease transmission [153].

The threshold parameter R_0 is a function of four parameters, namely; β_1, b_1, a_1 and μ. The nomalized forward sensitivity index R_0, which is differentiable with respect to a given parameter p, is defined by:

$$\gamma_p^{R_0} = \frac{\partial R_0}{\partial p}\frac{p}{R_0}. \tag{4.13}$$

The analytical expression for the sensitivity of the basic reproduction number R_0 can be easily calculated using the explicit formula (4.13) for each parameter included in it. The sensitivity index values for the parameter values in Table 4.1 are shown in Table 4.2. 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 $\gamma_{b_1}^{\hat{R}_0} = -1$, this means that increasing (decreasing) β_1, b_1 by a certain percentage will always increase (decrease) R_0 by the same percentage.

Table 4. 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 4.2, 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.

Through sensitivity analysis, the plant epidemic model attempts to determine which factors significantly impact disease dynamics and plant development. As part of this analysis, the model factors will be altered. If significant model results, such as the basic reproduction number (R_0) or the locations of equilibrium, alter, this is what may be shown. Disease management techniques should give greater weight to sensitive factors if they are shown to significantly affect disease development and population dynamics [140].

The importance of ecological growth rate in developing disease dynamics is highlighted by the fact that it is sensitive to host ecological population factors. By modifying the ecosystem of animals that are susceptible and the human population, it is possible to substantially influence the rate at which diseases spread. In order to expand the number of the target population, interventions that change the environment or breed for genetic safety may be utilised alongside conventional disease management measures [142].

The disease sensitivity analysis, which also shows the importance of different disease factors, shapes the disease dynamics. Researchers may pick interventions that concentrate on the most crucial intervention by putting factors in order of sensitivity. Spending money on better disease tracking and early finding may be preferable to reducing pesticide use, for example, if it turns out that transfer rates are very sensitive.

Overall, the sensitivity analysis helps us figure out how the model's complex connections impact the spread of diseases and how to better handle them. This analysis, which identifies key factors and their relative importance, aids in the development of tailored intervention methods suitable for specific ecological and epidemiological conditions. Through informed decision-making guided by disease sensitivity analysis results, stakeholders can enhance resource sharing and boost the effectiveness of disease control efforts [149].

4.8 Numerical Analysis

The proposed plant epidemic model is analysed numerically to observe the behaviour of the spread of disease and the role of control measures in the decline of the disease. Numerical analysis is done on MATLAB 2018a. Computer simulations must validate the predictions of the plant epidemic model. This is especially true when it comes to how chemicals and pesticides

change disease dynamics in plant populations. To test how well the expected results of the model match up with actual data or claims made by well-known theories, researchers run these simulations. Researchers conduct these simulations to verify the accuracy and practicality of the model. The simulations look at a variety of factors, including the basic reproduction number (R_0), the effects of different factors, and the dynamics of plant populations that are susceptible to and unhealthy.

The present study utilised the model to investigate and analyse the various ways that pesticide application alters plant behaviour. The purpose of this study was to improve our understanding of the influence that these treatments have on the spread of diseases among plants by carefully studying such interventions. It was discovered through the use of this model that increasing the frequency of pesticide sprays as well as the amount of sprays can effectively reduce the rate at which a plant population is damaged. Furthermore, the findings of this model indicate that one way to improve the care that is provided to plants that have been afflicted by disease is to adjust the proportion of susceptible plants to diseased plants. Considering that there are fewer diseases spreading, the system is getting closer and closer to an equilibrium that is free of diseases [158].

4.8.1 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 $\alpha = 0.02$.

4.9 Numerical simulations

Using the specified parameters and initial conditions, we perform simulations of the model (4.1) until $t = 1200$. The results are illustrated in Figure 4.2 through Figure 4.8, considering scenarios both with and without control measures, where the application of pesticides is considered as 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 Figure 4.6 and Figure 4.8.

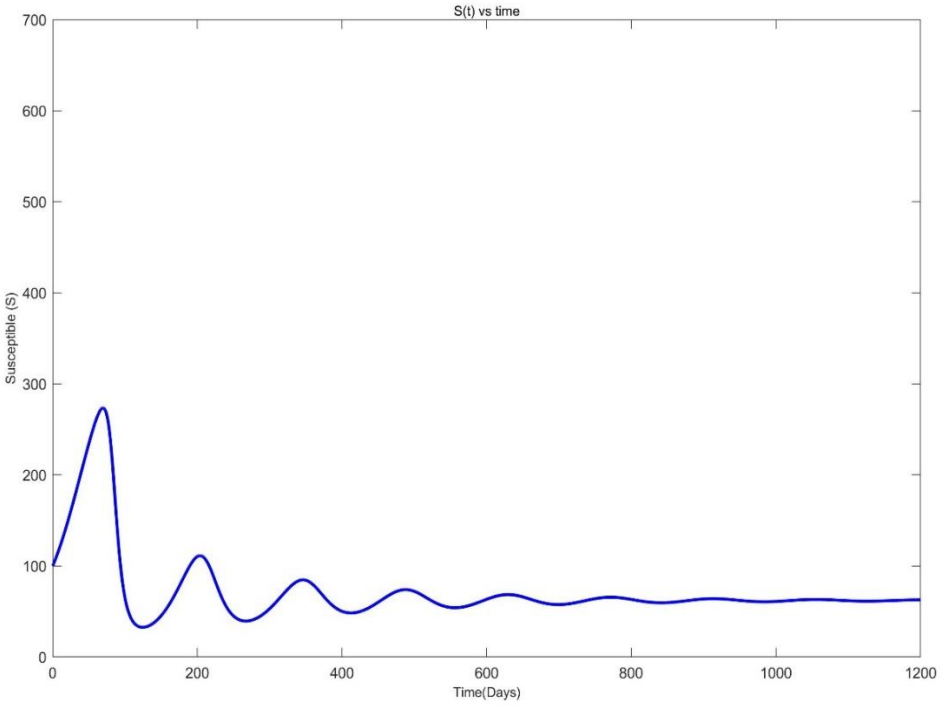

Figure 4.2: $S(t)$ vs Time under the application of Pesticide

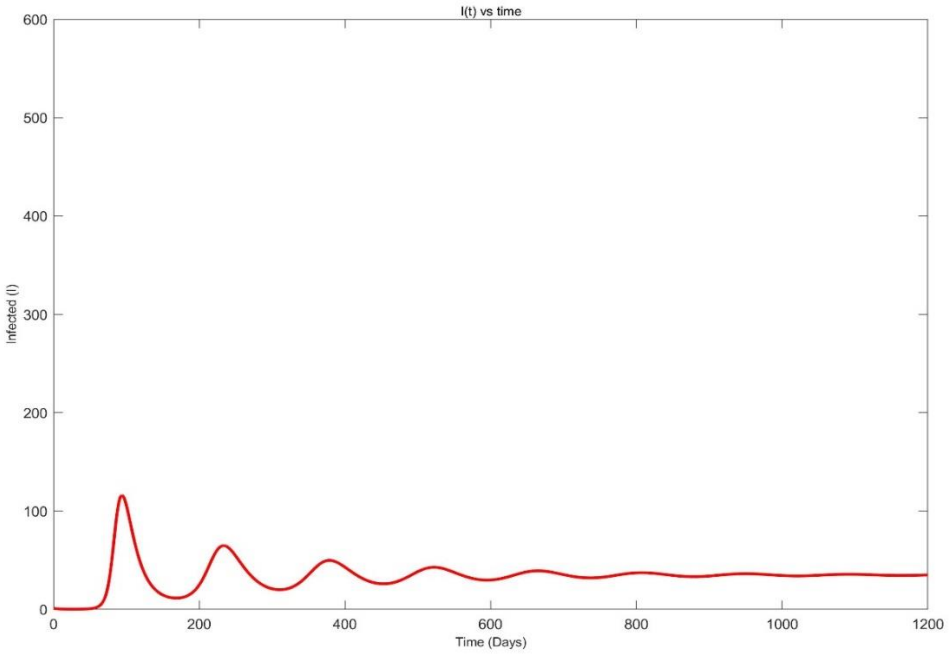

Figure 4.3: $I(t)$ vs Time under the application of Pesticide

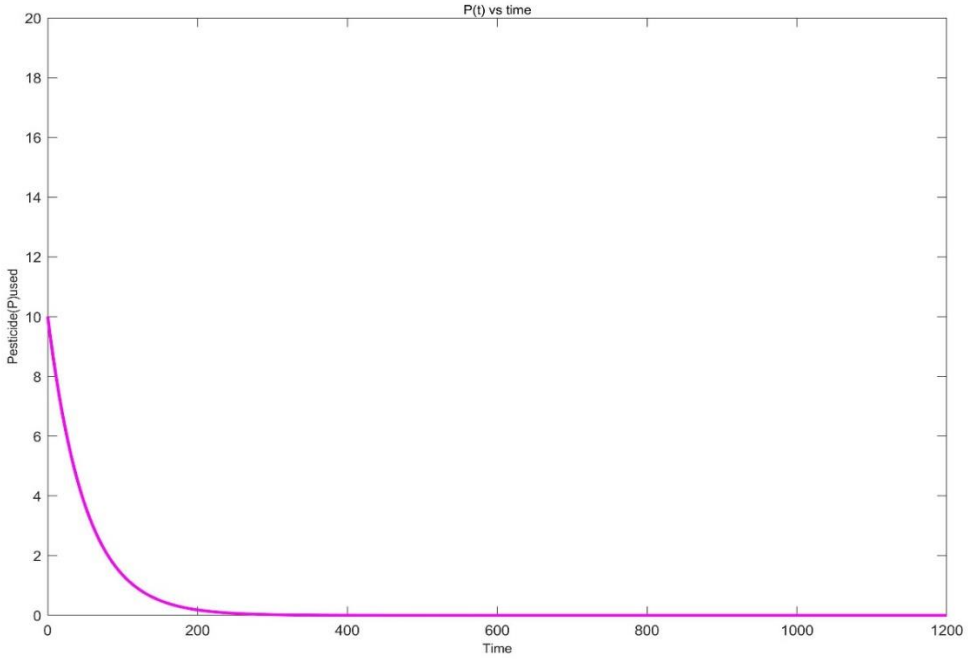

Figure 4.4: Amount of Pesticides $P(t)$ used vs Time

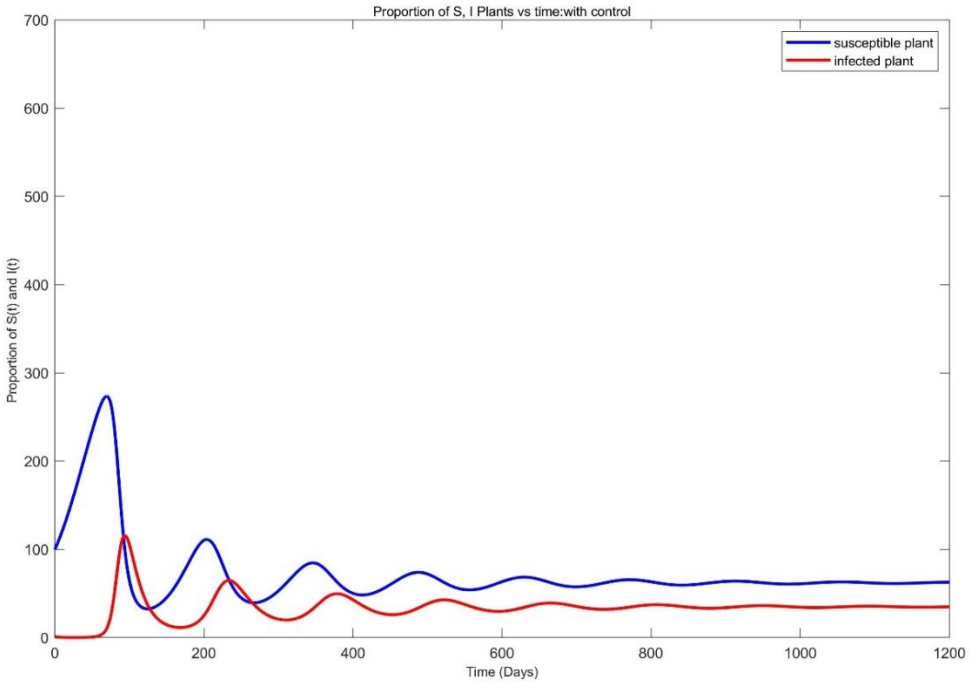

Figure 4.5: $S(t)$ and $I(t)$ vs Time under the application of Pesticide

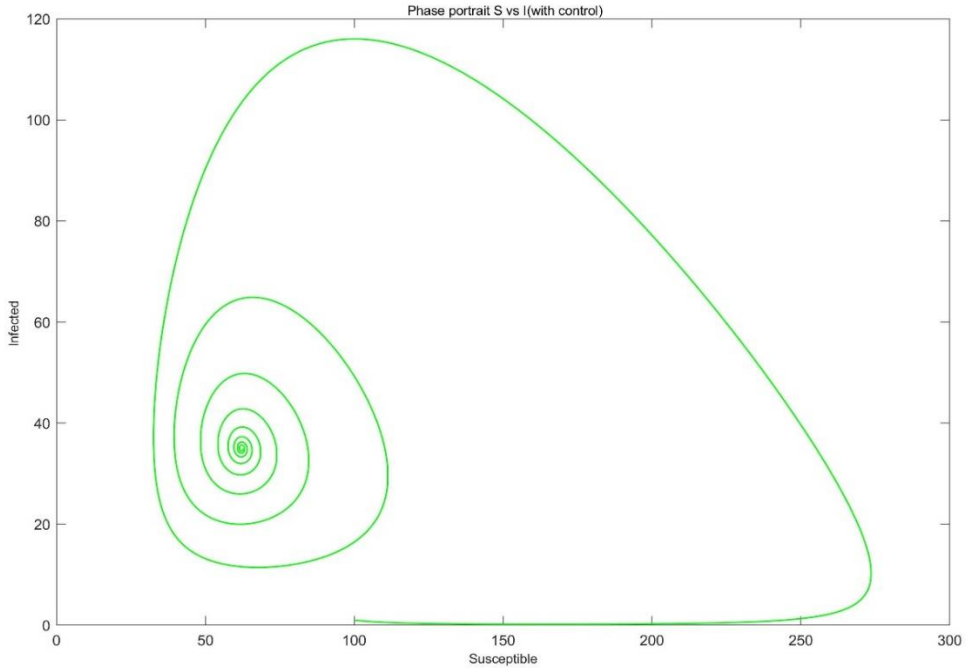

Figure 4.6: Phase portrait of $S(t)$ vs $I(t)$ under the application of Pesticide

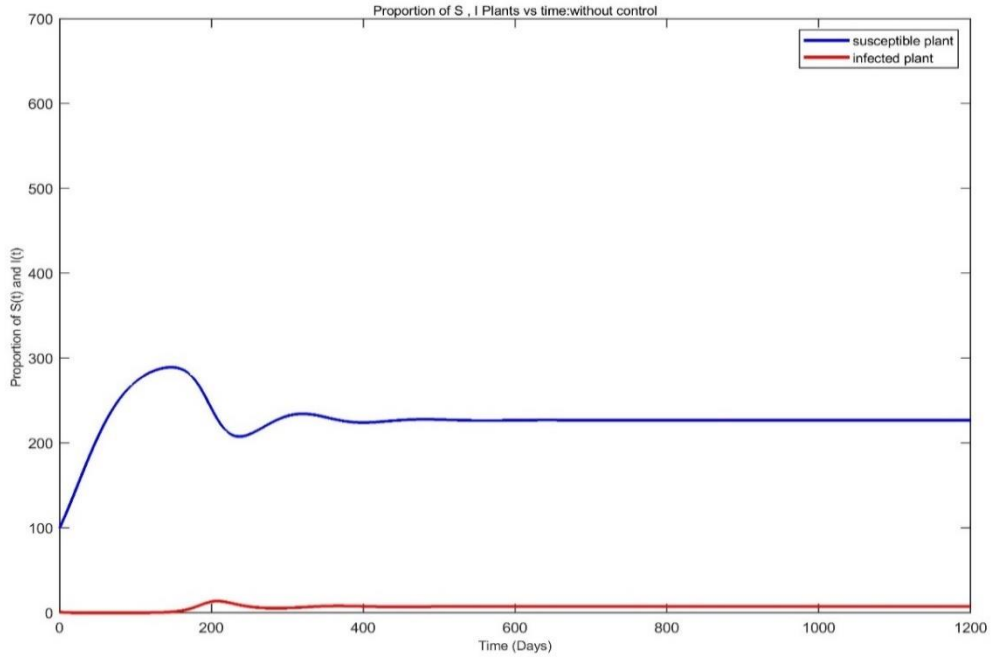

Figure 4.7: $S(t)$ and $I(t)$ vs Time without the use of Pesticide

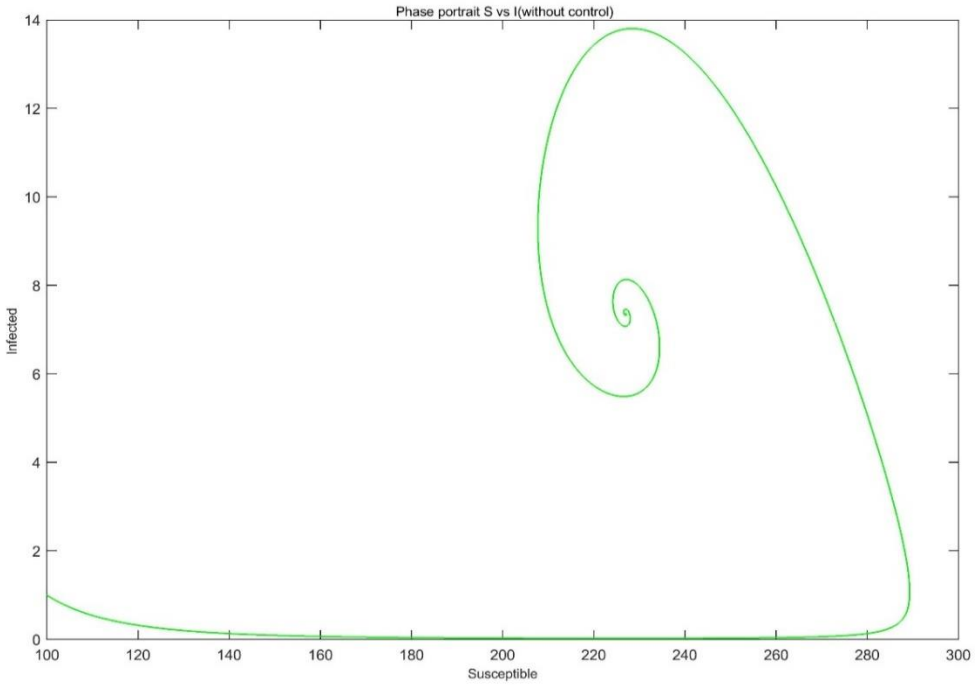

Figure 4.8: Phase portrait of $S(t)$ vs $I(t)$ without the use of Pesticide

In the absence of pesticide application, the susceptible plant population demonstrates resistance to decline, hindering its attainment of a stable equilibrium, as shown in Figure 4.2. Meanwhile, the infected plant population experiences an initial rise, eventually adopting a linear pattern without achieving a stable state, as evident in Figure 4.3. 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 pesticides are introduced, as depicted in Figure 4.5, a decline in plant infections becomes apparent. This results in oscillations in both susceptible and infected plant populations. This phenomenon is illustrated in Figures 4.3 and 4.5, 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. The phase portrait depicting the relationship between susceptible and infected plants reveals a state of instability in the absence of control measures, as represented in Figure 4.8. Conversely, when

control measures are employed to mitigate the number of infected plants, the trajectory converges towards an equilibrium point, as exemplified in Figure 4.6.

4.10 Results and Discussion

Through mathematical, stability, plant, and numerical studies, this study gives a complete understanding of the dynamics of plant epidemics with pesticide intervention.

Under different pesticide application conditions, the mathematical model shows how a plant population can reach an equilibrium state, either disease-free or endemic. The stability analysis shows that the system tends to return to a disease-free state when the basic reproduction number, R_0, is less than one. If the R_0 number is more than one, the disease is still in the population. The usual model for diseases in epidemiology says that this is what should happen [167].

The sensitivity analysis shows how important some factors are in controlling the disease and how well it is controlled. These include the contact rate between plants that are susceptible and plants that are infected (β_1) and the natural death rate of plant populations (μ). These results back up what other studies have found about how important transmission dynamics and natural population change rates are for effectively controlling plant diseases. The study, for example, agrees with the findings of other researchers who have examined the impact of contact rates on disease spread in agricultural settings, emphasizing the significance of targeted interventions.

4.11 Conclusion

This research was able to shed light on agricultural disease management tactics as a result of the development of a new plant model that incorporates the use of pesticides. By conducting in-depth tests such as pesticide and disease testing, the mathematical analysis allows for the identification of significant elements that influence the effectiveness of pesticides and the dynamics of their application. According to this model, the basic reproduction rate, also known as R_0, is one of the most important factors that determines whether plant populations will be able to avoid disease spread or become extinct. Summarising our analysis, the results can be outlined as follows:

I. The positivity and boundedness of solutions of the system are shown to hold indicating the system is biologically valid and well behaved.

II. 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.

III. Through the utilisation 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 determined its value to be $R_0 = \frac{\beta_1(a_1-\mu)}{\mu a_1 b_1}$.

IV. 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 act as a disease threshold, indicating the disease's sustained presence and stability, although the likelihood of a widespread outbreak or epidemic remains limited.

V. To analyse the stability properties of the system, we utilised and calculated the Jacobian matrix for the system of equation (4.1).

VI. It has been shown that the disease-free equilibrium (DFE) E_2 is both locally and globally asymptotically stable in cases where R_0 is less than 1.

VII. Utilising the Routh-Hurwitz criteria, we have established the local asymptotic stability of the endemic equilibrium E^* within the system (4.1). Furthermore, through the consideration of a Lyapunov function, we have determined the global asymptotic stability of E^*.

VIII. The behaviour of the model remains stable around the disease-free and endemic equilibria, both locally and globally. Both susceptible and infected plants exhibit oscillatory behaviour and eventually reach a state of equilibrium over time. In the absence of any control measures in the plant epidemic model, it requires a longer time for both the susceptible and infected plants to reach equilibrium. However, the application of pesticides to manage the infection accelerates the attainment of equilibrium for both the plants.

IX. To identify the most sensitive parameters, it is crucial to understand the relative significance of the multiple factors contributing to its transmission. Consequently, calculations for the sensitivity index of R_0 concerning various parameters within the model have been conducted. 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 populations 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.

X. 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 optimising pesticide dosages, identifying disease hotspots, and understanding the nonlinear dynamics at play, a comprehensive framework is presented to guide sustainable management strategies in the plant community. The findings have far-reaching implications for the conservation of ecosystems and the protection of plants population against infectious diseases and the results show the importance of weighing the pros and cons of drugs used to treat diseases. Attention to proper pesticide application is crucial, as the model indicates substantial potential reductions in disease rates, emphasizing the need to maintain ecological balance and promote plant health.