Chapter 5

Analysis of Stability, Sensitivity Index and Hopf Bifurcation of Eco-Epidemiological SIR Model under Pesticide Application

This article has been published under the title "***Analysis of Stability, Sensitivity Index and Hopf Bifurcation of Eco-Epidemiological SIR Model under Pesticide Application***" in the Communication in Biomathematical Sciences, **Vol. 6, No. 2 (2023), page no. 126-144.**

5

Analysis of Stability, Sensitivity Index and Hopf Bifurcation of Eco-Epidemiological SIR Model under Pesticide Application

5.1 Introduction

This study's logical SIR (susceptible-infected-recovered) plant mathematical model focuses on pesticide use as a way to reduce the number of diseases in the plant population. This ecosystem research was motivated by concerns about plant diseases and their serious damage to crops around the world. These diseases, often caused by pests, threaten public health, affect crop yields, and pose a food hazard. Agriculture plays an important role in protecting ecosystems and

sustaining people, especially in areas where food is important. Therefore, understanding and controlling plant diseases has become important for scientists. The primary goal of this study is to improve our understanding of disease transmission in crop populations and evaluate the effectiveness of pesticide use in controlling the disease [179].

The importance of studying eco-epidemiological models, particularly the SIR model under pesticide application, cannot be overstated. These theoretical frameworks provide valuable tools for testing and predicting the relationships between susceptible, infected, and recovered plant populations. By incorporating pesticide application into this model, researchers aim to evaluate how various disease control strategies might influence these interactions and overall disease prevalence. This research not only advances our understanding of plant disease dynamics but also informs practical strategies for managing and mitigating the impact of plant diseases on agriculture. Understanding these dynamics is essential for developing effective interventions that can protect crop yields, ensure food security, and maintain the health of ecosystems.

5.2 Background and Literature Review

5.2.1 Summary of previous research on plant diseases

Plant diseases, which pose a major threat to agricultural output, have long been the focus of intense study in order to understand their causes, effects, and disease control measures. Based on the type of pathogens involved, researchers divide these diseases into groups that can spread and groups that cannot. Infectious diseases, which are frequently caused by fungi, viruses, or bacteria, can range in severity from mild to severe and can cause plant population declines and even death. Non-infectious diseases, which are brought on by poor conditions in the environment, do not spread from one plant to another, making it easy to stop their spread [181].

The presence of plant diseases has caused a significant amount of devastation to the ecosystem. The Food and Agriculture Organisation (FAO) estimates that pests destroy approximately forty percent of the world's agricultural crops annually. This number demonstrates just how important it is to have effective methods to prevent diseases in order to ensure that more people have access to food. Experts recommend using pesticides as a common approach to address this problem. Between the middle of the 20th century and the present day, there has been a

significant increase in the production and application of pesticides. They have a significant role in combating diseases and insects, which is an important function. The high production rate highlights the crucial role of pesticides in modern day farming.

5.2.2 Mathematical modelling in biology and its applications

Through the application of mathematical models, it might be possible to comprehend numerous biological processes and how they connect with their environment. These models are used by biologists to forecast diseases, replicate biological processes, and develop efficient disease control measures. The mathematical application of disease dynamics in biology, especially the study of disease dynamics, provides insight into the procedures of spread, effects, and possible interventions for viral diseases influencing human and plant populations.

To stop the propagation of plant diseases, it is very important to keep the world's food supply free of pests. Plant disease can be brought on by germs like fungi, viruses, and bacteria that enter the environment. By spreading disease, insects and other pests can mess up watering systems. Numerous factors, such as how predisposed people are to the disease and the number of people who catch it, influence how infectious diseases start and spread. A mathematical framework called Susceptible-Infected-Recovered (SIR) helps researchers guess what will happen in the future with disease eco dynamics in different settings [184].

5.3 Model Formulation

5.3.1 Assumptions made for the SIR model formulation

Here, the SIR model is utilised to assess the impact of pesticide formulation on disease prevalence in plant populations. Plant populations naturally develop in disease-free settings due to fundamental changes in plant development and surroundings. Certain natural factors make it impossible for plant populations to grow. To form the mathematical model, the following assumptions are taken into consideration:

a) In the absence of disease, the plant population grows logistically with an intrinsic growth rate $r > 0$ and environmental carrying capacity $k > 0$.

b) In the presence of the disease, the plant population is divided into three compartments: the susceptible population $S(t)$, the infected population $I(t)$ and the recovered population $R(t)$. Therefore, for any time t, the total plant population is given by $S(t) + I(t) + R(t) = N(t)$. Then the growth rate of the susceptible plant population is given by $rS\left(1 - \frac{N}{k}\right)$ or $rS\left(1 - \frac{S+I+R}{k}\right)$.

c) The susceptible population becomes infected when they come into contact with infected populations. This contact process is assumed to follow the kinetics of simple mass action using $\beta > 0$ as the conversion factor.

d) Only the susceptible population $S(t)$ can reproduce and the death rate of plants due to pests is assumed to be $\mu > 0$. The natural mortality rate of plants is ignored from the incubation period to the death of the plants. However, the infected population I contributes with S to population growth towards the carrying capacity $k > 0$.

e) As a control measure, we assume that a general pesticide $P(t)$ is used to minimize diseases in the population. Due to the application of pesticides, plants within the infected compartment transition to the recovered compartment and eventually return to a susceptible compartment within a specific timeframe. Pesticides are used in both susceptible and infected populations, and it is assumed that the use of pesticides has negative impacts on both the susceptible and the infected populations. The negative impact of pesticides is ignored for the plant population in the recovered compartment, as they have already been exposed to the pathogen or pest and have developed immunity or resistance against it. Also, the recovered population eventually reverts to a susceptible state after a certain time. For instance, *Propiconazole* and *Tricyclazole* are two common fungicides primarily targeted at controlling fungal diseases like blast disease and dirty panicle disease in rice crops. They are not intended to harm non-infected rice plants [67]. Still, their residues and potential for phytotoxicity emphasize the importance of responsible and precise application, that can range from mild stress symptoms to severe damage and plant death.

f) The amount of pesticides used is just one of several factors that can influence the contact rate between plants populations and pesticides. Let the amount of pesticide used to be

$\alpha > 0$. The contact rate between susceptible plants and pesticides is assumed to be $\widetilde{d_1}(\alpha) > 0$. Similarly, the contact rate between infected plants and pesticides is assumed to be $\widetilde{d_2}(\alpha) > 0$. Here, we consider, $\widetilde{d_i}(\alpha) = d_i, i = 1,2$, where d_i are constants. Therefore, the term $-d_1SP$ represents the removal of plants from susceptible plant compartment due to the application of pesticides. Similarly, the contact rate between infected plants and pesticides is assumed to be $d_2 > 0$. The term $-d_2IP$ represents the removal of plants from infected plant compartment due to the application of pesticides.

g) The presence of plant infections can often lead to the application of pesticides. However, the decision to use pesticides depends on various factors, including the severity of the infection, the type of pathogen involved, the crop being grown, and the overall management practices employed by the farmer. When plants are infected by pathogens, it can lead to the development of plant diseases, which can impact the health and productivity of the crop. In some cases, farmers may choose to use pesticides to control the spread of the pathogens and mitigate the damage caused by the disease. Pesticides specifically formulated to target the pathogens causing the infection may be employed as a means to suppress or eliminate them. Therefore, we assumed that the infections in plants indirectly forces the farmers to apply pesticides. The term $\theta I,\ \theta > 0$ denotes the infective induced rate of pesticides.

h) The application of pesticides enhances the recovery rate of the infected plants. Let $g > 0$ be the recovery rate of the infected plants due to the application of pesticides.

i) Let $\nu > 0$ be the rate of infected plants which have recovered and returned to the susceptible class [4,118].

The model splits the whole plant population into three groups: the susceptible (S) group, the infected (I) group, and the recovered (R) group. This sorting helps us understand how disease dynamics work because it takes into account both the current level of ecosystem within the disease and the possibility of subsequent infection and exposure. As suggested in the above assumption, the susceptible population is assumed to become infected when it comes into contact with the infected population using a conversion factor to measure this interaction. With this reduction, it is simpler to mathematically model disease spread in the plant community [190].

5.3.2 Model equations and the interpretation of model parameters and variables

Table 5.1: Notation and definition of model variables

Variables	Definitions	Units
$S(t)$	Susceptible population	[Stems]
$I(t)$	Infected population	[Stems]
$P(t)$	Pesticides	[SI unit]
$R(t)$	Recovered population	[Stems]

Table 5.2: Notations and definition of model parameters

Parameters	Definitions of parameters	Units
r	Intrinsic growth rate of the plant population	$Per\ day$
k	Environmental carrying capacity	$Per\ sq.meter$
β	Contact rate between susceptible and infected plants	$Per\ day$
d_1	Contact rate between susceptible plants and pesticides	$Per\ day$
d_2	Contact rate between infected plants and pesticides	$Per\ day$
μ	Death rate of plants due to pests	$Per\ day$
v	Rate of infected plants which have recovered and returned to the susceptible class.	$Per\ day$
g	Recovery rate of infected plants	$Per\ day$
θ	Infective induce rate of pesticides	$Per\ day$
α	Amount of pesticides used	$Per\ day$

In accordance with the above assumptions and the descriptions of variables and parameters, the present model will be governed by the following system of equations:

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I+R}{k}\right) - \beta SI - d_1 SP - \mu S + \nu R,$$

$$\frac{dI}{dt} = \beta SI - (g + \mu)I - d_2 IP,$$

$$\frac{dP}{dt} = \theta I - \alpha P, \tag{5.1}$$

$$\frac{dR}{dt} = gI - (\mu + \nu)R.$$

With initial conditions:

$$S(0) \equiv S_0 > 0, I(0) \equiv I_0 > 0, P(0) \equiv P_0 > 0 \; and \; R(0) \equiv R_0 > 0 \tag{5.2}$$

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

5.4 Theoretical Analysis

5.4.1 Positivity and Boundedness.

Theorem 5.1 (Positivity): All solutions of the system represented by (5.1) with initial conditions (5.2) are positive for all $t \geq 0$.

Proof: Let $S(t), I(t), P(t), R(t)$ be the solutions of System (5.1) with initial conditions (5.2). Integrating both sides of the first equation of (5.1) from 0 to t, gives,

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I+R}{k}\right) - \beta SI - d_1 SP - \mu S + \geq -\left\{\beta SI + d_1 SP + \mu S - rS\left(1 - \frac{S+I+R}{k}\right)\right\}$$

or

$$\int_0^t \frac{dS}{S} \geq \int_0^t -\left\{\beta I + d_1 P + \mu - r\left(1 - \frac{S+I+R}{k}\right)\right\} dt$$

or

$$S(t) \geq S(0)\exp\left[\int_0^t -\left\{\beta I + d_1 P + \mu - r\left(1 - \frac{S+I+R}{k}\right)\right\}dt\right]$$

$\Longrightarrow S(t) > 0.$

From the second equation of System (5.1), we get,

$$I(t) \geq I(0)\exp\left[\int_0^t \{\beta SI - (g+\mu)I - d_2 IP\}dt\right]$$

$\Longrightarrow I(t) > 0.$

From the third equation of System (5.1), we get,

$$P(t) \geq P(0)\exp\left[\int_0^t -\alpha P dt\right]$$

$\Longrightarrow P(t) > 0.$

From the fourth equation of System (5.1), we get,

$$R(t) \geq R(0)\exp\left[\int_0^t \{-(\mu+\nu)R\}dt\right]$$

$\Longrightarrow R(t) > 0.$

Hence, the theorem stands proved.

Theorem 5.2 (Boundedness): All solutions of System (5.1) that start in R_+^4 are uniformly bounded in the solution set $\Omega = \left\{(S,I,P,R): 0 \leq S \leq \frac{rk}{4\mu}, 0 \leq I \leq \frac{rk}{4\mu}, 0 \leq R \leq \frac{rk}{4\mu}, 0 \leq P \leq \frac{rk\theta}{4\alpha\mu}, 0 \leq S+I+R \leq \frac{rk}{4\mu}\right\}$.

Proof: Let $S(t), I(t), P(t), R(t)$ be the solution of System (5.1).

Let $W = S + I + R$.

Then,

$\frac{dW}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = rS\left(1 - \frac{S}{k}\right) - \mu(S + I + R) - d_1SP - d_2IP - rS\left(\frac{I+R}{k}\right)$,

$\Longrightarrow \frac{dW}{dt} + \mu W \leq rS\left(1 - \frac{S}{k}\right)$.

Let $f(S) = rS\left(1 - \frac{S}{k}\right)$.

Therefore, $\frac{df}{dS} = r - \frac{2rS}{k}$ and $\frac{d^2f}{dS^2} = -\frac{2r}{k}$.

Now, $\frac{df}{dS} = 0 \Longrightarrow r - \frac{2rS}{k} = 0 \Longrightarrow S = \frac{k}{2}$.

Then, $\frac{d^2f}{dS^2} = -\frac{2r}{k} < 0$, which gives a maximum value for S.

Therefore, $\frac{dW}{dt} + \mu W \leq \frac{rk}{4} \Longrightarrow W \leq \frac{rk}{4\mu} + \left(W_0 - \frac{rk}{4\mu}\right)e^{-\mu t}$.

As $t \to \infty, e^{-\mu t} \to 0 \Longrightarrow W \to \frac{rk}{4\mu} \Longrightarrow W(t) \leq \frac{rk}{4\mu}$ and hence W is bounded.

Clearly $I(t)$ is bounded above by $\frac{rk}{4\mu}$. Therefore, the third equation of System (5.1) becomes

$\frac{dP}{dt} + \alpha P \leq \frac{rk\theta}{4\mu}$,

$\Longrightarrow P \leq \frac{rk\theta}{4\alpha\mu} + \left(P_0 - \frac{rk\theta}{4\alpha\mu}\right)e^{-\alpha t}$.

where P_0 is the initial amount of pesticide used.

As $t \to \infty, e^{-\alpha t} \to 0 \Longrightarrow P \to \frac{rk\theta}{4\alpha\mu} \Longrightarrow P(t) \leq \frac{rk\theta}{4\alpha\mu}$ and hence, P is bounded for any initial value and for all t. Therefore $S(t), I(t), P(t), \vec{R}(t)$ are uniformly bounded.

Note: From Theorem 5.2, it is clear that each population is bounded above. So, the total population $N(t)$ is also bounded above whenever time $t \to \infty$.

5.4.2 Existence and Uniqueness of Solution for the SIPR Model

In this section, uniqueness theorem and existence of System (5.1) is formulated. Following the method used by Samuel et al. [159], the proof of the following theorems is performed.

"The general first-order ODE is in the form:"

$$x' = f(t, x), x(t_0) = x_0. \tag{5.3}$$

The questions which can be asked as follows:

1. Under what conditions the solution of Equation (5.3) exists?
2. Under what conditions Equation (5.3) has a unique solution?

To answer the above question, we use the following theorem.

Theorem 5.3 (Uniqueness of Solution): Let D denote the region:

$$|t - t_0| \leq a, \|x - x_0\| \leq b, x = (x_1, x_2, x_3, \dots, x_n), x_0 = (x_{10}, x_{20}, x_{30}, \dots, x_{n0}). \tag{5.4}$$

Suppose the function $f(t, x)$ satisfies the Lipschitz condition:

$$\|f(t, x_1) - f(t, x_2)\| \leq M\|x_1 - x_2\|, \tag{5.5}$$

and whenever (t, x_1) and (t, x_2) belong to the region D and M represent a positive constant.

Then, ∃ a constant $\delta > 0$ such that there exists a unique continuous vector solution $x(t)$ of the system (5.3) in the interval $|t - t_0| < \delta$.

Remark 1: It is important to note that condition (5.5) is satisfied by the requirement that:

$$\frac{\partial f_i}{\partial x_j}, i, j = 1,2, \dots n,$$

is continuous and bounded in the region D.

Lemma 5.1: If $f(t,x)$ has continuous partial derivative $\frac{\partial f_i}{\partial x_j}$ on a bounded closed convex domain $\mathcal{R}$ (i.e., convex set of real numbers), where $\mathcal{R}$ is used to denotes real numbers, then it satisfies a Lipschitz condition in $\mathcal{R}$.

Our interest is in the domain: $1 \leq \epsilon \leq \mathcal{R}$. (5.6)

So, we look for a bounded solution of the form $0 < \mathcal{R} < \infty$. We now prove the following existence theorem.

Theorem 5.4. (Existence of solution): Let D denote the region defined in (5.4) such that (5.5) and (5.6) holds. Then, there exists a solution of the equations of System (5.1) which is bounded in the region D.

Proof: From System (5.11), we define the following:

$$f_1 = rS\left(1 - \frac{S+I+R}{k}\right) - \beta SI - d_1 SP - \mu S + vR, \quad (5.7)$$

$$f_2 = \beta SI - (g+\mu)I - d_2 IP, \quad (5.8)$$

$$f_3 = \theta I - \alpha P, \quad (5.9)$$

$$f_4 = gI - (\mu + v)R, \quad (5.10)$$

We show that $\frac{\partial f_i}{\partial x_j}, i,j = 1,2,\ldots,n$ are continuous and bounded. We consider the following partial derivatives for all the model equations:

From Equation (5.7):

$$\left|\frac{\partial f_1}{\partial S}\right| = \left|r\left(1 - \frac{2S+I+R}{k}\right) - \beta I - d_1 P - \mu\right| < \infty, \left|\frac{\partial f_1}{\partial I}\right| = \left|\frac{-(r+\beta k)S}{k}\right| < \infty,$$

$$\left|\frac{\partial f_1}{\partial P}\right| = |-d_1 S| < \infty, \left|\frac{\partial f_1}{\partial R}\right| = \left|\frac{-rS}{k} - v\right| < \infty.$$

From Equation (5.8):

$$\left|\frac{\partial f_2}{\partial S}\right| = |\beta I| < \infty, \left|\frac{\partial f_2}{\partial I}\right| = |\beta S - (g+\mu) - d_2 P| < \infty,$$

$\left|\frac{\partial f_2}{\partial P}\right| = |-d_2 I| < \infty, \left|\frac{\partial f_2}{\partial R}\right| = 0 < \infty.$

From Equation (5.9):

$\left|\frac{\partial f_3}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_3}{\partial I}\right| = |\theta| < \infty,$

$\left|\frac{\partial f_3}{\partial P}\right| = |-\alpha| < \infty, \left|\frac{\partial f_3}{\partial R}\right| = 0 < \infty.$

From Equation (5.10):

$\left|\frac{\partial f_4}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial I}\right| = |g| < \infty,$

$\left|\frac{\partial f_4}{\partial P}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial R}\right| = |-(\mu + \nu)| < \infty.$

We have clearly established that all these partial derivatives are continuous and bounded in D. Hence, by Theorem (4.2.1), there exists a unique solution of the system (5.1) in the region D.

Hence, the positivity (Theorem 5.1), boundedness (Theorem 5.2) and the uniqueness existence (Theorem 5.3) of System (5.1) implies that the model is biologically valid and well behaved.

The main aim of the theoretical analysis of the SIR model under pesticide application is to establish the model's mathematical stability and its implications for ecological and epidemiological dynamics. To ensure the boundedness and positivity of the answers, researchers must first ensure that the model remains biologically true over time. They prove that the system always gives positive answers, even if the starting point is positive.

5.4.3 Analysis of the equilibrium points, including the trivial, disease-free, and endemic equilibrium states

The trivial point, disease-free point, and endemic point are observed in equilibrium point analysis. The trivial equilibrium, when all populations are zero, is mathematically intriguing but not biologically interesting. Disease-free equilibrium (DFE) is attained when there are no diseases in the plant population. The DFE's stability, shown by the fundamental ecosystem term R_0, governs disease elimination circumstances. The disease is assumed to have died out when R_0 is smaller than one since the DFE is stable [194].

For finding the equilibrium points, we set the right-hand side of System (5.1) equals to zero as follows:

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I+R}{k}\right) - \beta SI - d_1 SP - \mu S + \nu R = 0,$$

$$\frac{dI}{dt} = \beta SI - (g+\mu)I - d_2 IP = 0,$$

$$\frac{dP}{dt} = \theta I - \alpha P = 0, \tag{5.11}$$

$$\frac{dR}{dt} = gI - (\mu+\nu)R = 0.$$

On solving the above equations, then three equilibrium points in the coordinate (S^*, I^*, P^*, R^*) are obtained and are given as follows:

(i) The trivial equilibrium point $T_0(0,0,0,0)$.

(ii) Disease-free equilibrium point $T_1\left(\frac{k(r-\mu)}{r}, 0,0,0\right)$. It is seen that the equilibrium point T_1 consistently exists if and only if $r > \mu$.

(iii) The disease-endemic equilibrium point $T_2(S^*, I^*, P^*, R^*)$ which is explicitly expressed in term of I^* as follows:

$S^* = \frac{1}{\alpha\beta}[d_2\theta I^* + \alpha(g+\mu)]$, $P^* = \frac{\theta}{\alpha}I^*$, $R^* = \frac{gI^*}{\mu+\nu}$ and I^* is a positive root of the following equation:

$$\Psi_1 (I^*)^2 + \Psi_2 I^* + \Psi_3 = 0 \tag{5.12}$$

Where:

$$\Psi_1 = \frac{\alpha d_2\theta(r+\beta) + d_1 d_2 k\theta^2}{\alpha^2\beta} + r\left(\frac{d_2\theta}{\alpha\beta}\right)^2 - \frac{\alpha\beta r g d_2\theta}{\mu+\nu},$$

$$\Psi_2 = \frac{\alpha\beta[\alpha(\alpha+\beta)(g+\mu) + k\mu\theta + kd_1\theta(g+\mu)] + \alpha d_2\theta[(r+1)(g+\mu) - \beta r]}{(\alpha\beta)^2} + \frac{\alpha\beta g[\alpha\beta k\nu - \alpha r(g+\mu)]}{\mu+\nu},$$

$$\Psi_3 = \frac{(g+\mu)[k\mu + \alpha^2\beta((g+\mu) - \beta k]}{\beta}.$$

Equation (5.12) implies,

$$I^{*2} + M_1 I^* + M_2 = 0, \tag{5.13}$$

where $M_1 = \Psi_2/\Psi_1, M_2 = \Psi_3/\Psi_1$.

From equation (5.13), $I^* > 0$ if one of the following conditions holds:

(a) $M_1 < 0, M_2 < 0$,

(b) $M_1 < 0, M_2 > 0$ and $M_1^2 - 4M_2 > 0$,

(c) $M_1 > 0, M_2 < 0$.

Real and positive solutions of I^* give $S^* > 0, P^* > 0, R^* > 0$. Due to the complexity of the model, it is difficult to determine the analytical solutions. So, we proceed our discussions using numerical techniques.

It also demonstrates the ecosystem factors that allow the disease to persist despite management efforts. This is endemic equilibrium. The ecosystem-plant coexistence of susceptible, infected, and recovered plants with long term pesticide usage is demonstrated by the stability of this equilibrium, which is affected by factors like the application of pesticide and plant resistance very early on.

5.4.4 Calculation and interpretation of the basic reproduction number (R_0) and its implications for disease control.

In this section, the basic reproduction number R_0 is determined. This can be characterised as the average number of secondary infections caused by typical cases of infection in the general population, which is vulnerable to everyone. R_0 is basically used to measure the potential for transmission of a disease.

Theorem 5.5: The basic reproduction number of the system (5.1) is given by $R_0 = \frac{\beta k(r-\mu)}{r(g+\mu)}$.

Proof: The Basic reproduction number R_0 is calculated with the help of the next generation matrix method which is given by $G = FV^{-1}$ [94], where F is the newly formed infection matrix, V is the transmitted infection matrix and V^{-1} is the inverse of V.

Then,

$$F_i = \begin{pmatrix} \beta SI \\ 0 \\ 0 \end{pmatrix},\ V_i = \begin{pmatrix} (d_2P + g + \mu)I \\ \alpha P - \theta I \\ R(\mu + \nu) - gI \end{pmatrix}, \text{ where } i = 1,2,3.$$

Therefore, we get:

$$F = \begin{pmatrix} \beta S & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},\ V = \begin{pmatrix} d_2P + g + \mu & d_2I & 0 \\ -\theta & \alpha & 0 \\ -g & 0 & \mu + \nu \end{pmatrix}.$$

At the disease-free equilibrium T_1, we have

$$F = \begin{pmatrix} \frac{\beta k(r-\mu)}{r} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} g+\mu & 0 & 0 \\ -\theta & \alpha & 0 \\ -g & 0 & \mu+\nu \end{pmatrix} \Rightarrow V^{-1} = \begin{pmatrix} \frac{1}{g+\mu} & 0 & 0 \\ \frac{\theta}{\alpha(g+\mu)} & \frac{1}{\alpha} & 0 \\ \frac{-g}{(g+\mu)(\mu+\nu)} & 0 & \frac{1}{\mu+\nu} \end{pmatrix}.$$

Hence, $G = FV^{-1} = \begin{pmatrix} \frac{\beta k(r-\mu)}{r(g+\mu)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$ and the basic reproduction number is the dominant eigenvalue of G which is given by: $R_0 = \frac{\beta k(r-\mu)}{r(g+\mu)}$. (5.14)

The theoretical application of the eco-epidemiological SIR model under pesticide applications is heavily dependent on calculating the basic reproduction number, denoted as R_0.

According to the findings of the studies, R_0 is the average number of secondary cases that can be caused by a single infected plant in a population that is entirely susceptible to the disease. It is the underlying parameter for this number that determines the likelihood that a disease would spread through a population of plants. The model expresses R_0 as $R_0 = \frac{\beta k(r-\mu)}{r(g+\mu)}$, where r growth rate of the plant population, μ the death rate due to pests, g the recovery rate due to pesticide

application, β the contact rate between susceptible and infected plants, and k the environmental carrying capacity.

Conducting an analysis of R_0 can be beneficial in gaining a better understanding of the dynamics of disease control and dissemination. The disease cannot continue to exist in the population if the value of R_0 is less than 1. This signifies the complete eradication of the disease from the population. Consequently, this indicates that the existing rates of pesticide application and plant healing are adequate to stop or at least reduce the progress of the disease. A value of R_0 greater than one, on the other hand, indicates that the disease has the potential to spread across the plant population. As a result, in order to reduce the infection rate or increase the rate at which infected plants heal, it is necessary to use control methods that are more active or targeted. $R_0 = 1$ functions as a disease threshold, which means that the disease continues to be active and stable, but the likelihood of an outbreak or epidemic of the disease is extremely low [30].

5.5 Stability Analysis

In order to study the stability properties, the general Jacobian matrix J of the system (5.1) is reported as follows:

$$J = \begin{bmatrix} J_{11} & \frac{-(r+\beta k)S}{k} & -d_1 S & \frac{kv-rS}{k} \\ \beta I & J_{22} & -d_2 I & 0 \\ 0 & \theta & J_{33} & 0 \\ 0 & g & 0 & J_{44} \end{bmatrix}, \tag{5.15}$$

Where:

$$J_{11} = r\left(1 - \frac{2S+I+R}{k}\right) - \beta I - d_1 P - \mu,$$
$$J_{22} = \beta S - (g+\mu) - d_2 P,$$
$$J_{33} = -\alpha,$$
$$J_{44} = -(\mu + v).$$

5.5.1 Stability of trivial equilibrium point

Theorem 5.6: The trivial equilibrium point $T_0(0,0,0,0)$ is stable if $r < \mu$ and unstable if $r > \mu$.

Proof: The Jacobian matrix of the system (5.1) at T_0 is given by:

$$J_{T_0} = \begin{bmatrix} r-\mu & 0 & 0 & v \\ 0 & -(g+\mu) & 0 & 0 \\ 0 & \theta & -\alpha & 0 \\ 0 & g & 0 & -(\mu+v) \end{bmatrix}.$$

The eigenvalues of the above matrix are given as follows:

$\lambda_1 = r-\mu, \lambda_2 = -(g+\mu), \lambda_3 = -\alpha$ and $\lambda_4 = -(\mu+v)$.

Clearly, $\lambda_2, \lambda_3, \lambda_4 < 0$. If $r-\mu < 0$, then $\lambda_1 < 0$ and the equilibrium T_0 is stable and unstable otherwise.

Hence, T_0 is stable if $r < \mu$ and unstable if $r > \mu$.

5.5.2 Local stability of the disease-free equilibrium

Theorem 5.7: The disease-free equilibrium $T_1\left(\frac{k(r-\mu)}{r}, 0,0,0\right)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The Jacobian matrix of the system (5.1) at T_1 is given by:

$$J_{T_1} = \begin{bmatrix} -(r-\mu) & \frac{-(r+\beta k)(r-\mu)}{r} & \frac{-d_1 k(r-\mu)}{r} & v-(r-\mu) \\ 0 & \frac{\beta k(r-\mu)}{r} - (g+\mu) & 0 & 0 \\ 0 & \theta & -\alpha & 0 \\ 0 & g & 0 & -(\mu+v) \end{bmatrix}. \quad (5.16)$$

Eigenvalues of the above matrix (5.16) are:

$\lambda_1 = -(r-\mu)$, $\lambda_2 = \frac{\beta k(r-\mu)}{r} - (g+\mu)$, $\lambda_3 = -\alpha$ and $\lambda_4 = -(\mu+v)$.

Clearly, $\lambda_1, \lambda_3, \lambda_4 < 0$.

Now, for the system (5.1) to be stable at T_1, we must have $\lambda_2 < 0$,

i.e., $\frac{\beta k(r-\mu)}{r} - (g+\mu) < 0$,

$$=> \frac{\beta k(r-\mu)}{r} < (g + \mu),$$

$$=> \frac{\beta k(r-\mu)}{r(g+\mu)} < 1 \ ,$$

$$=> R_0 < 1.$$

Thus, $\lambda_2 < 0$ if $R_0 < 1$, which implies all the eigenvalues of the characteristic equation (5.16) have a negative real part. Hence the equilibrium T_1 is locally asymptotically stable.

5.5.3 Local stability of the endemic equilibrium

Theorem 5.8: The endemic equilibrium $T_2(S^*, I^*, P^*, R^*)$ is locally asymptotically stable if the following condition holds [164]:

$$A_1 > 0,\ A_3 > 0, A_4 > 0 \text{ and } A_1A_2A_3 > A_3^2 + A_1^2A_4, \tag{5.17}$$

Where:

$$A_3 = r\left(\frac{2S^*+I^*+R^*}{k} - 1\right) - \beta(S^* - I^*) + (d_1+d_2)P^* + (3\mu + g + \alpha + v),$$

$$A_2 = G_1 + \alpha(\mu + v) - (\alpha + \mu + v)G_2 + \frac{\beta(r+\beta k)S^*I^*}{k},$$

$$A_1 = \alpha(\mu + v)G_3 + (\alpha + \mu + v)\left(G_1 + \frac{\beta(r+\beta k)S^*I^*}{k}\right) + d_1\beta\theta S^*I^* - d_2\theta I^* - \frac{g\beta(kv-rS^*)I^*}{k},$$

$$A_0 = \alpha(\mu + v)\left(G_1 + \frac{\beta(r+\beta k)S^*I^*}{k}\right) - (\mu + v)(d_2\theta I^* - d_1\beta\theta S^*I^*) - \frac{\alpha g\beta(kv-rS^*)I^*}{k}.$$

Here, $G_1 = (\beta S^* - d_2P^* - g - \mu)\left(r - \mu - \beta I^* - d_1P^* - \frac{(2S^*+I^*+R^*)r}{k}\right)$,

$$G_2 = \beta(S^* - I^*) + (r - g - 2\mu) - (d_1+d_2)P^* - \frac{(2S^*+I^*+R^*)r}{k},$$

$$G_3 = \frac{(2S^*+I^*+R^*)r}{k} + 2\mu + g + (d_1+d_2)P^* - \beta(S^* - I^*) - r.$$

Proof: The characteristic roots corresponding to the equilibrium T_2 are given by the equation:

$$\xi^4 + A_3\xi^3 + A_2\xi^2 + A_1\xi + A_0 = 0. \tag{5.18}$$

By Routh-Hurwitz criterion, the equation will have negative roots if

$$A_1 > 0,\ A_3 > 0, A_4 > 0 \text{ and } A_1A_2A_3 > A_3^2 + A_1^2A_4. \tag{5.19}$$

Hence T_2 is locally asymptotically stable if the above conditions are satisfied and unstable otherwise.

5.6 Sensitivity Analysis and Hopf Bifurcation

5.6.1 Sensitivity analysis performed on R_0 and the implications of the findings

The basic reproduction number R_0 is a function of five parameters β, k, r, μ, g. To understand the contribution of each of the parameters in the Reproduction number R_0 as given by (5.14), a sensitivity analysis [168] is being conducted which let us know how significant each parameter is to disease transmission.

Sensitivity index of the system is given as:

$$S_h^{R_0} = \frac{h}{R_0}\frac{\partial R_0}{\partial h}. \tag{5.20}$$

The sensitivity indices of the reproduction number with respect to β, k, r, μ, g are given by:

$$S_\beta^{R_0} = 1,\ S_k^{R_0} = 1,\ S_r^{R_0} = \frac{\mu}{r-\mu},\ S_\mu^{R_0} = \frac{-\mu(g+r)}{(r-\mu)(g+\mu)},\ S_g^{R_0} = \frac{-g}{g+\mu}.$$

The index table is shown in Table 5.3.

Table 5.3: Sensitivity index table.

Parameters	Sensitivity index	Sensitivity index values
β	1	$1/day$
k	1	$1/m^2$
r	$\frac{\mu}{r-\mu}$	$0.029/day$
μ	$\frac{-\mu(g+r)}{(r-\mu)(g+\mu)}$	$-0.938/day$
g	$\frac{-g}{g+\mu}$	$-0.091/day$

From Table 5.3, it can be seen that the sensitivity indices change in values with the change in values of parameters r, μ, and g except for β, k which has value 1, a constant value i.e., it is independent of any parameter. The sensitivity index $S_r^{R_0}$ is positive i.e., the value of R_0 increases as the value of r increase and the sensitivity indices $S_\mu^{R_0}$ and $S_g^{R_0}$ are negatives i.e., the value of R_0 decreases as the value of μ and g increases. The remaining sensitivity indices $S_\beta^{R_0}$ and $S_k^{R_0}$ are constants i.e., for any increase or decrease in values of β and k, the value of R_0 remain constant throughout. Figure 5.1 and 5.2 describes that the number of infected plants decreases with an increase in values of a specific parameter: μ and g.

The purpose of this research is to get an understanding of the manner in which critical components influence the propagation of disease throughout the plant population. This will be accomplished by doing a sensitivity analysis on the basic reproductive number, which is denoted by R_0. It is critical for the ecological system to be dependent on the calculation of sensitivity indices for various parameters. These parameters include the contact rate between susceptible and infected plants (β), the carrying capacity of the environment (k), the intrinsic growth rate of

the plant population (r), the death rate of plants due to pests (μ), and the recovery rate of infected plants (g). After taking this into consideration, the sensitivity parameter of the plant population is presented. When it comes to the process of disease transmission, it is possible to make use of sensitivity indices in order to ascertain which components are the most significant. The outcomes of these experiments illustrate what happens to R_0 when the values of the parameters are changed with respect to the model.

The findings demonstrate how the growth of plant populations contributes to the transmission of disease by showing that the value of R_0 increases as the natural growth rate (r) increases during the study. On the other hand, the value of R_0 decreases as both the death rate (μ) and the recovery rate (g) increase. This illustrates that the disease can be prevented from spreading by increasing the pace at which pests die or by increasing the rate at which plants recover themselves.

Furthermore, no changes have been made to the sensitivity measures for the contact rate (β) and the carrying capacity of the ecosystem (k). As a result, the changes in these factors do not directly impact R_0. In light of this, there is a pressing need for additional study to be conducted on the ways in which these factors influence the capacity of the disease to spread. This serves to demonstrate the intricate relationship that exists between the rate of disease transmission and the dynamics of plant populations.

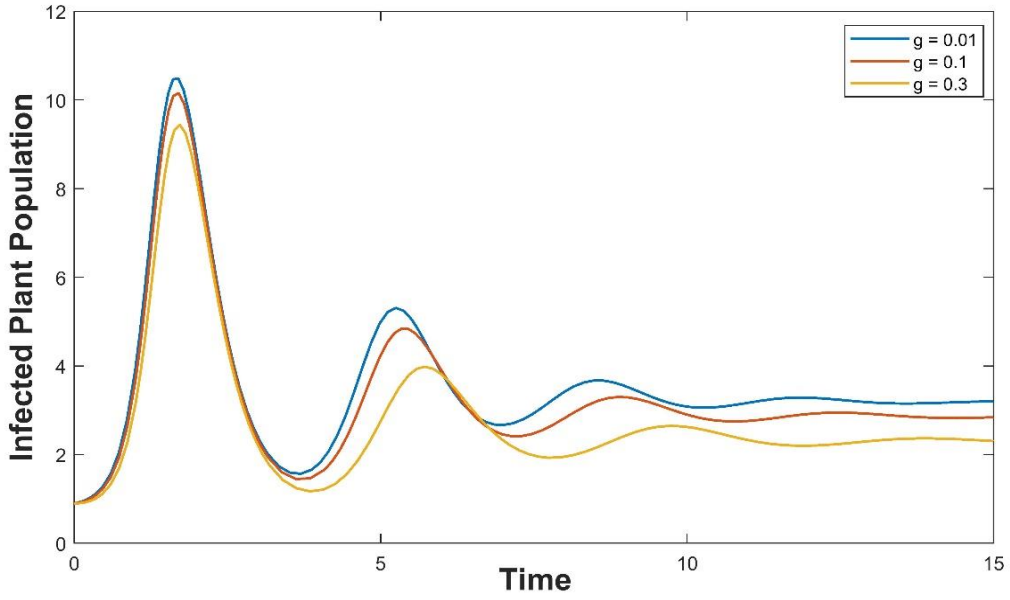

Figure 5.1: Impact of the variation of g in the number of infected plant population (difference not visible).

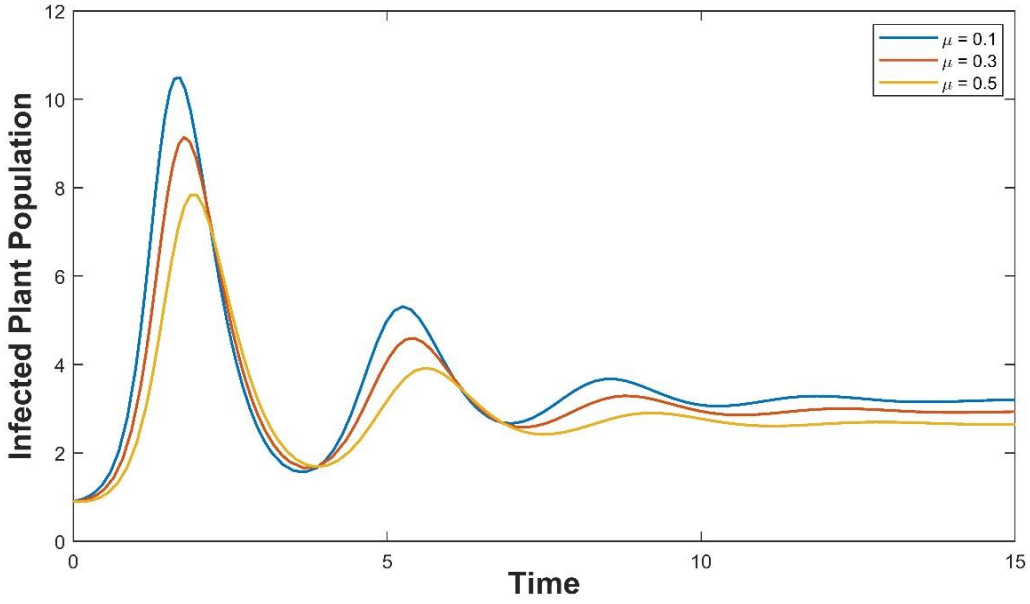

Figure 5.2: Impact of the variation of μ in the number of infected plant population.

5.7 Hopf bifurcation, its significance in the model, and the conditions

The investigation into Hopf bifurcation is another important aspect of the study. It aids in the identification of circumstances that result in periodic disease outbreaks by gathering data on conditions within the model. This can depend on a number of factors, including the application of pesticides and the values of contact rate (β) and recovery rate (g). Identifying stable dynamics that transition into oscillations at specific points is crucial for effectively managing and forecasting long-term diseases. This information is essential for implementing appropriate control and prediction measures.

In the mathematical theory of bifurcation, the term Hopf bifurcation refers to the local emergence or disappearance of periodic solutions or limit cycles (self-excited oscillations) from equilibrium when a parameter exceeds a critical value. This is the simplest bifurcation that does not involve only equilibria and belongs to what is sometimes called dynamic (rather than static) bifurcation theory. In differential equations, Hopf bifurcations usually occur when the complex conjugate pairs of eigenvalues of the linearized flow at a fixed point are purely imaginary. This means that the Hopf bifurcation can only occur in this two-dimensional or higher system. When a stable limit cycle surrounds an unstable equilibrium point, the bifurcation is called a supercritical Hopf bifurcation. If the limit cycle is unstable and surrounds a stable equilibrium point, then the bifurcation is called a subcritical Hopf bifurcation. A Hopf bifurcation is also known as a Poincar´e-Andronov-Hopf bifurcation and is named after Henri Poincar´e, Aleksandr Andronov, and Eberhard Hopf [111].

Researchers are particularly interested in Hopf bifurcation when analysing the dynamics of eco-epidemiological models. When a parameter reaches a critical value, the mathematical event leads to strange solutions or limit cycles from a steady state. Researchers use SIR models in conjunction with a pesticide model to study Hopf splits. It emphasises the significance of understanding disease transmission and disease control in plant population dynamics [196].

In this particular study, the infective induce rate of pesticides (θ) is considered to be one of the most crucial characteristics that are utilised to initiate the investigation of pesticides. This phenomenon, known as Hopf bifurcation, which occurs when the dynamics of a plant population undergo a transition from a steady state to random leaps that exceed a predetermined threshold value of θ, where θ^H represents the prescribed value. With the implementation of the

framework, the situation has significantly changed. This has altered our approach to dealing with plant diseases and preventing their significant spread. As a means of avoiding unanticipated effects on the dynamics of plant populations, it is imperative that the rate of pesticide application be carefully controlled [197].

The analysis also includes computer simulations to provide a more comprehensive illustration of the Hopf bifurcation rate of the model. This is performed in order to provide a more accurate representation of the model. The findings of the simulation provide evidence in support of the findings of this hypothesis by demonstrating how changing the infective trigger rate of the pesticides has an influence on the stability and behaviour of the system. This evidence is provided by the simulation's results.

According to Routh-Hurwitz theorem, the endemic equilibrium $T_2(S^*, I^*, P^*, R^*)$ is locally asymptotically stable if $A_1 > 0$, $A_3 > 0, A_4 > 0$ and $\Delta = A_1A_2A_3 - A_3^2 - A_1^2A_4 > 0$. Wei-Min Liu [119] introduced an equivalent condition for simple Hopf bifurcation without determining eigenvalues. According to the theorem by Liu, the endemic equilibrium T_2 undergoes a simple Hopf bifurcation if:

CH1: $A_1(\theta^H), A_2(\theta^H), A_3(\theta^H), A_4(\theta^H) > 0$ and $\Delta(\theta^H) = 0$.

CH2: $\frac{d\Delta(\theta^H)}{d\theta} \neq 0$.

Considering Δ as a function of θ, it is obtained that for the parameters in Table 4 with $d_1 = 0.5$, at $\theta = \theta^H \approx 0.805340$, $\Delta = 0$ (Figure 3). At the point $\theta = \theta^H \approx 0.805340$, $\frac{d\Delta(\theta^H)}{d\theta} \approx -0.572197 \neq 0$ (Figure 5.3). Also, $A_1 > 0, A_2 > 0$, $A_3 > 0, A_4 > 0$ at the point $\theta = \theta^H$ (Figure 5.4). Hence conditions **CH1**, **CH2** are satisfied and the disease-endemic equilibrium undergoes a simple Hopf bifurcation at $\theta = \theta^H$. At $\theta = \theta^H$, eigenvalues of the Jacobian matrix at the disease-endemic equilibrium are $-0.874414, -0.110232, \pm 1.05103\, i$, which also confirms the existence of Hopf bifurcation. In Figure 5.5, the phase portraits are drawn for $\theta = 0.78/day$ and $\theta = 0.84/day$, which clearly depicts the existence of limit cycles.

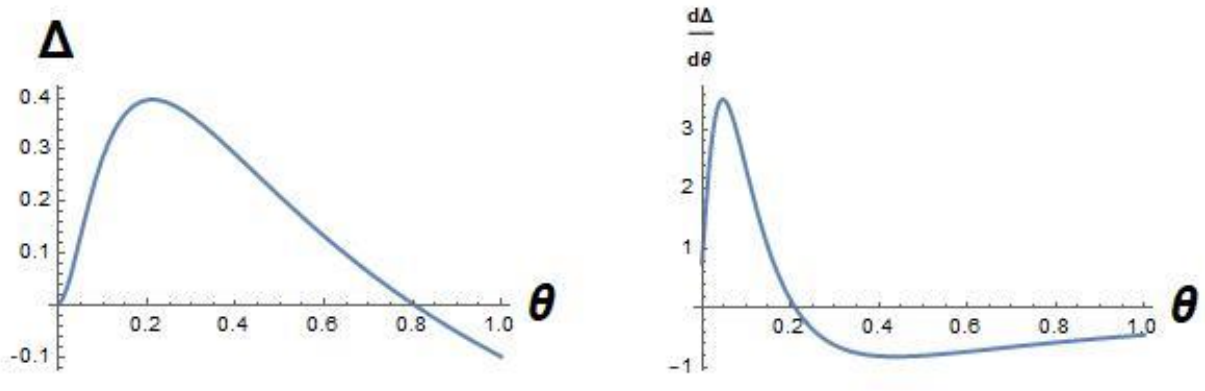

Figure 5.3: Plot of $\Delta = A_1A_2A_3 - A_3^2 - A_1^2A_4$ and $\frac{d\Delta}{d\theta}$ as functions of θ (Parameters are taken from Table 5.4).

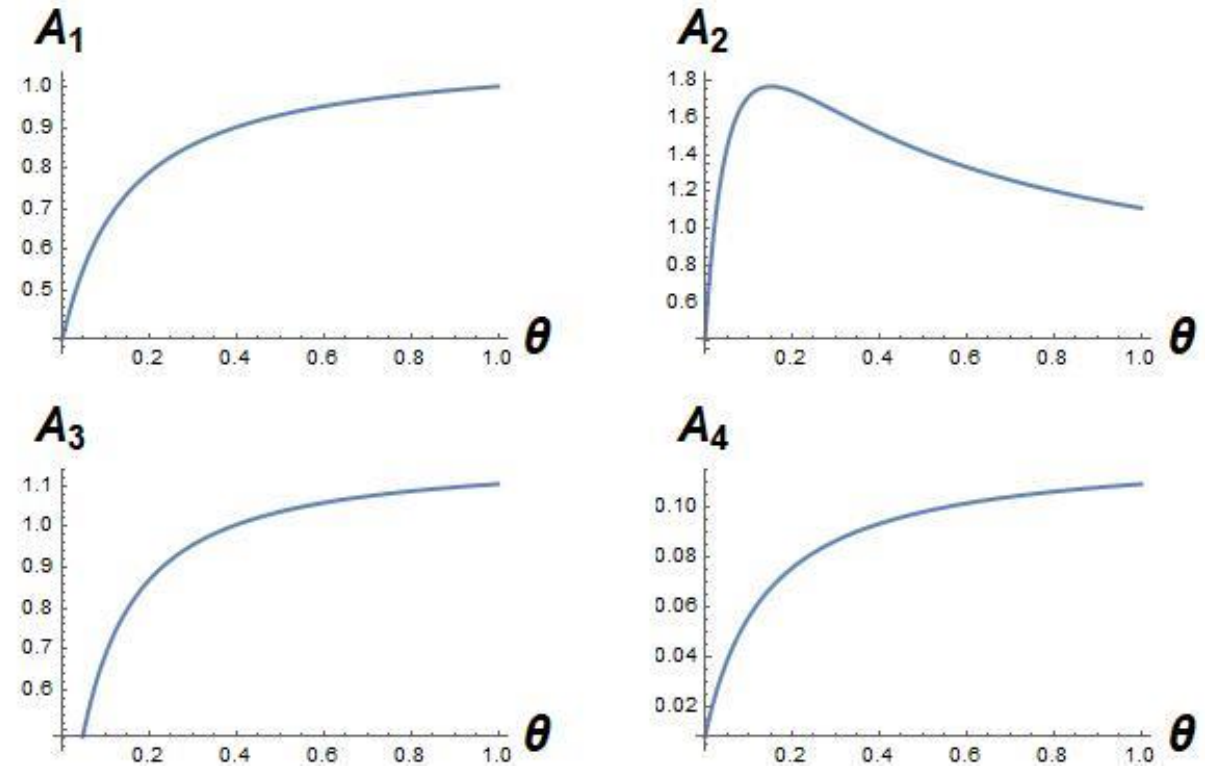

Figure 5.4: Plot of A_1, A_2, A_3, A_4 as functions of θ (Parameters are taken from Table 5.4).

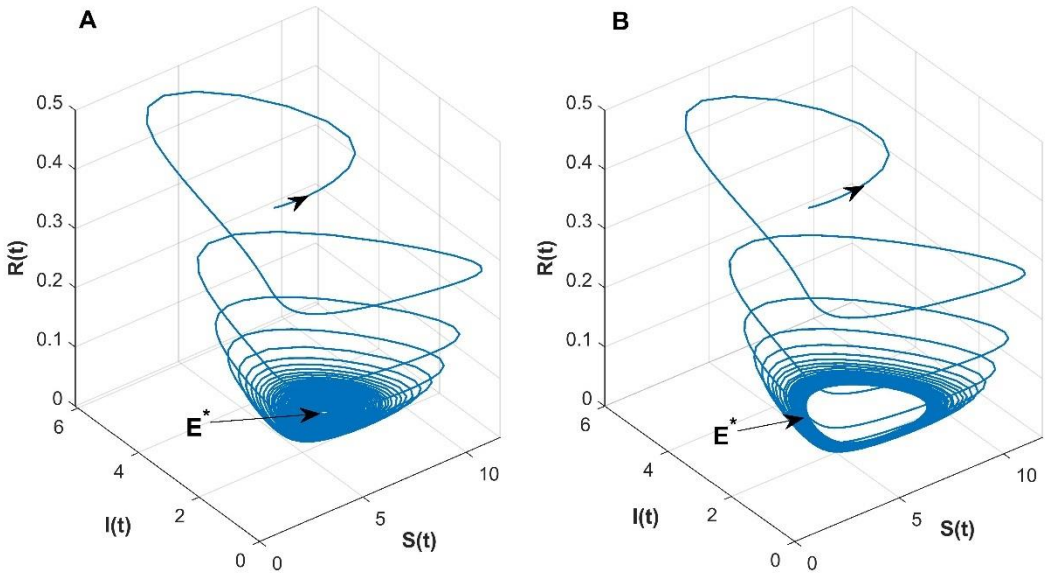

Figure 5.5: Phase portrait of the system (5.1) for $\theta = 0.78/day$ and $\theta = 0.84/day$ (other parameters are considered as mentioned in Table 5.4 with $d_1 = 0.5$).

5.8. Numerical Simulations

5.8.1 Numerical methods and software used for simulation

In this section, the proposed model is analysed numerically to observe the behaviour of the spread of disease and the role of control measures on the decline of the disease. Numerical analysis is done in MATLAB R2015a. This tool is renowned for its effectiveness in handling mathematical figures, graphics, and code. For numerical simulations, we set $S(0) = 2$, $I(0) = 0.9$, $R(0) = 0.5$ and $P(0) = 0.7$ and the estimated values of parameters are shown in Table 4. The original settings for this scenario were carefully chosen to resemble the real pesticide application and the presence of a disease in a certain percentage of the plant population. It is observed that the trajectories of the system (5.1), initiating from the mentioned initial points, approach to the disease endemic equilibrium $E^* = (8.3238, 3.1797, 7.9527, 0.2891)$ (Figure 5.6).

5.8.2 Presentation and interpretation of simulation results

Table 5.4: Parameter values used for Simulation

Variables	Definitions	Value
r	Intrinsic growth rate of the plant populations	$3.5/day$
k	Environmental carrying capacity	$25/m^2$
β	Contact rate between susceptible and infected plants	$0.3/day$
μ	Death rate of plants due to pest	$0.1/day$
v	Rate of infected plants which have recovered and returned to the susceptible class.	$0.01/day$
d_1	Contact rate between susceptible plants and pesticides	$0.1/day$
d_2	Contact rate between infected plants and pesticides	$0.3/day$
g	Recovery rate of infected plants	$0.01/day$
θ	Infective induce rate of pesticides	$0.5/day$
α	Amount of pesticides used	$0.2/day$

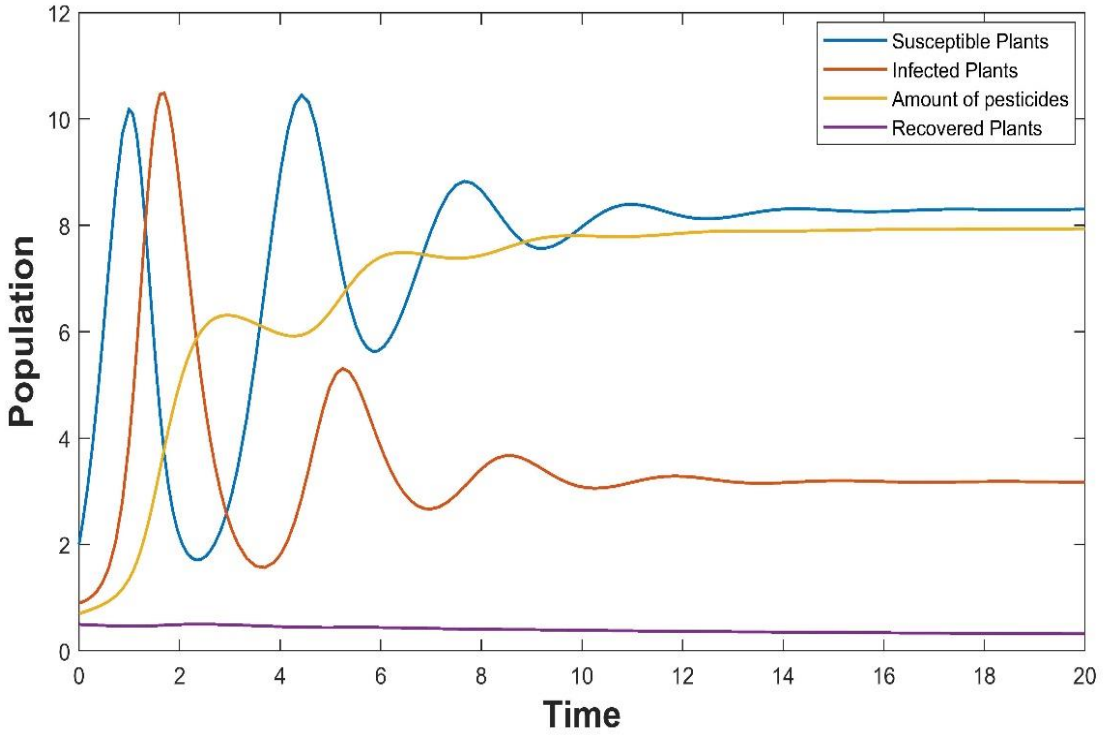

Figure 5.6: Time evolution of system (5.1) with the parameters mentioned in Table 5.4.

From Figure 5.6, it can be observed that initially the populations of the infected plants are dominant over the susceptible, but with an increase in the amount of pesticides use, the infected plants population decreases with increase in time. Both the plant populations, after a certain time, become stable with the equilibrium state E^*. For the same parameter set (Table 5.4) with $d_1 = 0.5/day\,, \theta = 0.5/day,$ the system (5.1) also has a disease endemic equilibrium $\overline{E^*} = (4.4438, 1.6229, 4.0786, 0.14832)$. Starting from the equilibrium $\overline{E^*}$, we plot the curve of equilibrium using θ as free parameter. The system (5.1) undergoes a supercritical Hopf bifurcation at $\theta^H = 0.805340/day$. The nature of the Hopf bifurcation is confirmed with the first Lyapunov coefficient, which is found to be $-2.453621 \times 10^{-03}$. Starting from the Hopf point θ^H, we plot the Hopf bifurcation curve varying parameters θ and α (Figure 5.7) which leads to the detection of Generalised-Hopf (denoted as GH) and Bogdanov-Takens bifurcations (denoted as BT) at $(\theta = 0.818986, \alpha = 0.000470)$ and $(\theta = 0.825201, \alpha = 0)$ respectively. Near the point GH along the curve, the endemic equilibrium displays varying characteristics, transitioning from a supercritical to a subcritical state. This Hopf curve separates the θ-α space into stable and unstable. In the unstable region, all the populations of system (5.1) start oscillating periodically, i.e., the populations of the susceptible and infected plants oscillate periodically. In Figure 5.8, the oscillating populations of the system (5.1) are represented for $\theta = 1/day$. It is seen that though the plant populations are oscillatory in nature, the susceptible populations are dominant over infected, i.e., the populations of the susceptible plants oscillate with a higher population than the infected.

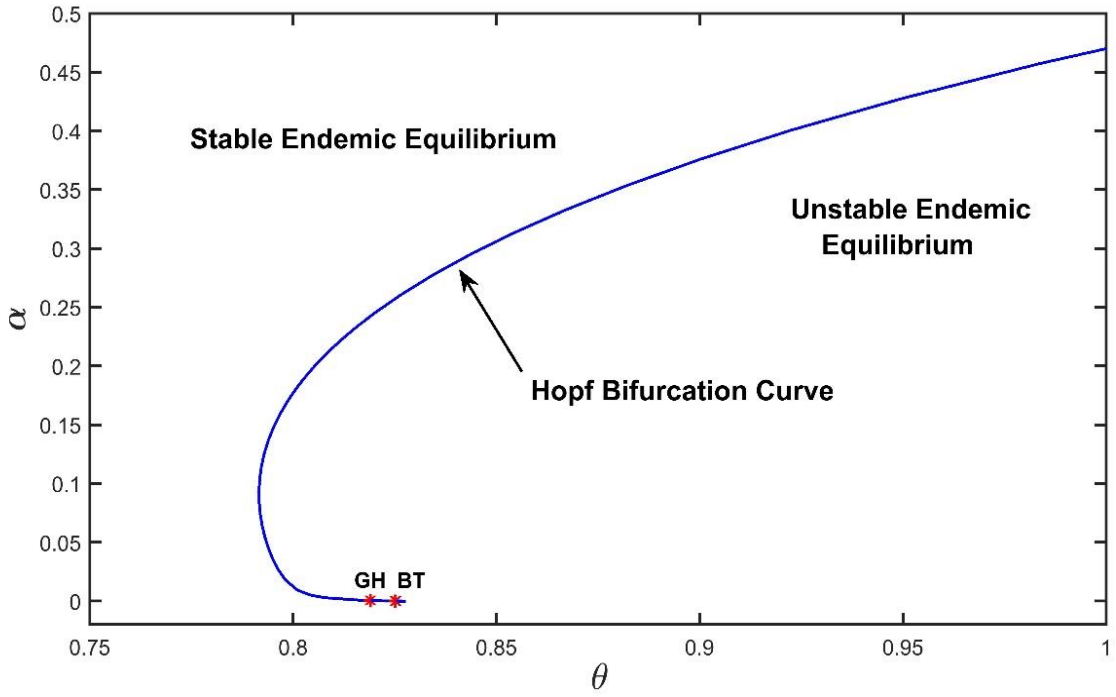

Figure 5.7: Two-dimensional projection of Hopf bifurcation curve with free parameter θ and α.

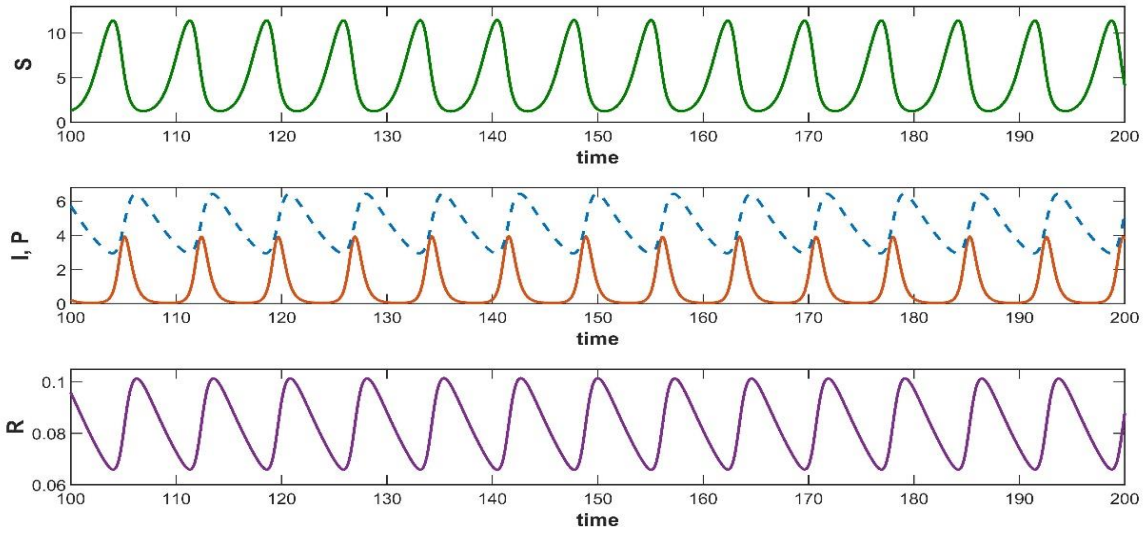

Figure 5.8: Time evolution of system (5.1) with the parameters mentioned in Table 5.4 and $d_1 = 0.5/day, \theta = 1/day$. The dotted line represents the amount of pesticides used.

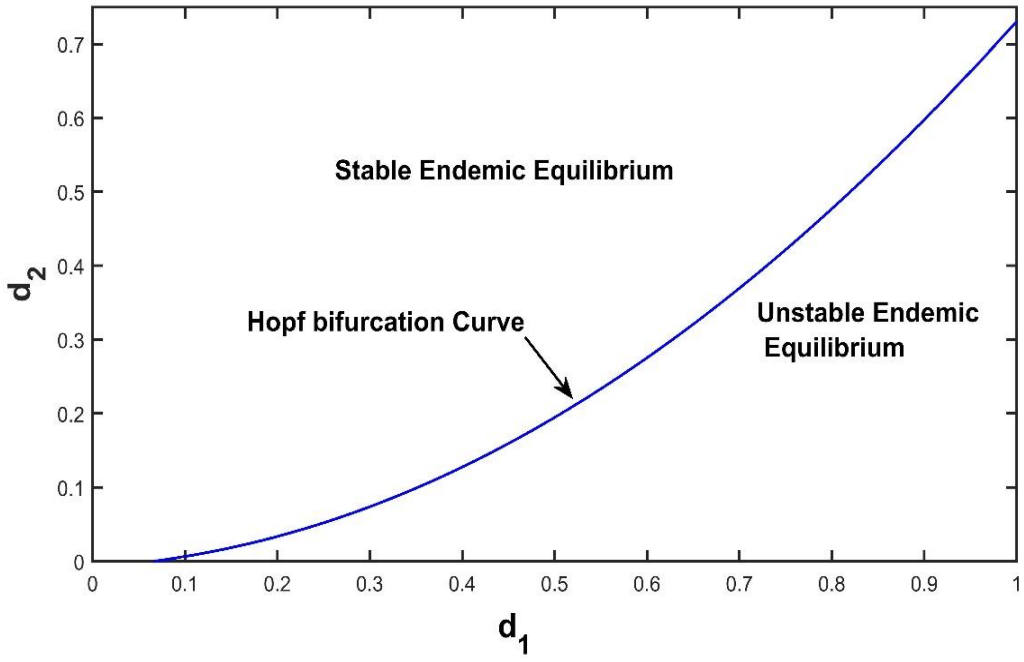

Figure 5.9: Two-dimensional projection of Hopf bifurcation curve with free parameter d_1 and d_2.

Again, starting from the equilibrium point $\bar{E}*$, we compute the curve of equilibria with free parameter d_1 which leads to a supercritical Hopf bifurcation at ${d_1}^H = 0.627463/day$, where the first Lyapunov coefficient is -2.227478×10^{-3}. From this point ${d_1}^H$, the two-dimensional projection of Hopf bifurcation curve is computed with free parameters d_1 and d_2 (Figure 5.9). Figure 5.9 represents a parametric region where the endemic equilibrium shows different stability. For the unstable region the endemic equilibrium shows periodic oscillatory behaviour. The eco system specifically supports coexistence among all populations for numbers above the stated range. In comparison, parameter values below the slope cause the populations of susceptible and infected plants to vary on a regular basis. This is a sign of a fragile eco system in which populations change over time and never reach a stable state [201].

It was also very clear what the numbers of the factors used in the simulations were. Important factors for the dynamics of the SIR model simulation were the natural growth rate of the plant population (r), the carrying capacity of the environment (k), the contact rate between susceptible and infected plants (β), and others. Because they have a direct effect on how the disease grows

and is managed in the model, these factors are very important for making sure that the simulation results are correct [198].

5.9 Conclusion and Future Directions

In this paper, a compartmental plant-pesticide model represented by a system of ordinary differential equations (ODEs) is proposed and analysed. The plant populations are divided into three compartments: the susceptible, the infected, and the recovered population. As a control measure, pesticides are applied to all the plants to reduce disease transmission from infected to susceptible plants. It is assumed that pesticides affect both the susceptible and infected populations. The necessary mathematical analysis for the biological validity of the proposed model were presented first. The boundedness theorem (Theorem 5.2) implies that each plant population is bounded above for $t \to \infty$. The total plant population $N(t)$ is also bounded above whenever $t \to \infty$, i.e., the system will not be collapsed due to population explosion. Uniqueness and the existence of solutions are one of the most important parts of mathematical modeling. In our model, unique solutions exist. If the solutions are not unique then there may exist two different equilibria, e.g., two different diseases endemic equilibrium. In that context, different initial populations may lead to different equilibrium states. In our study, we also determined a domain in which solutions of the system exist. Our proposed system has three feasible equilibrium points. The first is the trivial equilibrium point T_0, which always exists and is stable if $r < \mu$. If $r > \mu$ the equilibrium point T_0 becomes unstable resulting the appearance of the disease-free equilibrium (DFE) point T_1 and the endemic equilibrium point T_2. Using next generation matrix method, we determined the basic basic reproduction number R_0 of the infection. Sensitivity analysis was carried out to understand the relation between basic reproduction number R_0 and the associated parameters. Finally, a biologically plausible set of parameters was employed to conduct numerical simulations, aimed at comparing with analytical findings. Additionally, numerical simulations were used to generate Hopf bifurcation curves across various parameter spaces. Summarising our analysis, the results can be outlined as follows:

i. The disease-free equilibrium (DFE) is locally asymptotically stable whenever the basic reproduction number of the epidemic is less than unity. It signifies that the disease has been eradicated from the plant population. On the other hand, when the basic reproduction number exceeds unity, the DFE becomes unstable, indicating the presence of the disease in the plant population.

ii. The endemic equilibrium is found to be locally asymptotically stable under specific conditions which can be obtained utilising the Routh-Hurwitz Criteria. For the provided parameters, all the population coexists with an endemic equilibrium $E^* = (8.3238, 3.1797, 7.9527, 0.2891)$.

iii. The sensitivity indices of the basic reproduction number R_0 are determined and the impacts of associated parameters have been analysed. R_0 tend to change its value as the value of the associated parameter increases or decreases, and remain constant whenever the value of the associated parameters is constant. It is observed that the value of R_0 increases as the value of r increases and the value of R_0 decreases as the value of μ and g increases.

iv. It is observed that initially the population of infected plants predominates over the susceptible plants, but as the amount of pesticide increases, the infected plant population decreases over time (Figure 5.6). Both plant populations become stable after a certain period of time.

v. The inner dynamics of the system for varying the infective induce rate of pesticides was also discussed. It was found that the endemic equilibrium undergoes a supercritical Hopf bifurcation at $\theta = \theta^H \approx 0.805340$ i.e., above this critical parameter, all the population starts oscillating periodically and the equilibrium state becomes unstable.

vi. With free parameter d_1, the model leads to a supercritical Hopf bifurcation at ${d_1}^H = 0.627463/day$. A parametric region in parameters (d_1,d_2), where the endemic equilibrium shows different stabilities, is determined. For the parameters $d_1$and d_2, above the curve (Figure 5.9), all the populations coexist within the ecosystem, while for parameters below the curve all the populations will start oscillating periodically. Hence an unstable ecosystem can be observed where populations will fluctuate, never tending to a stable state.

Previous studies in the literature have examined eco-epidemic models focusing on either prey or predator populations, where they are divided into susceptible and infected categories. However, this research emphasizes the plant populations undergoing an epidemic with a disease and is partitioned into susceptible and infected. Furthermore, to mitigate the epidemic, the application of pesticides is implemented, resulting in the recovery of plant populations. Plant epidemics have been documented in various cultivated plants like tea and pineapples, leading to significant revenue losses [113]. Rice is the most important economic crop in India, China, East-Asia, South East Asia, Africa and Latin America catering to nutritional needs of 70% of the population in these countries [55], [109]. Rice diseases caused by fungi are considered the main constraint in rice production and cause both qualitative and quantitative losses. In particular, rice blast disease caused by Pyricularia oryzae (Magnaporthe grisea) has been reported as the most significant disease, resulting in yield losses of up to 50%. Dirty panicle disease or rice grain discoloration may be caused by many fungi, viz., Alternaria padwickii, Curvularia lunata, Fusarium moniliforme, and Bipolaris oryzae. Propiconazole and Tricyclazole are often applied in rice crops as a prevention measure for these fungal diseases. Although they are not intended to harm non-infected rice plants, their residues and the risk of phytotoxicity underscore the potential consequences, which can vary and lead to plant fatality [107]. This instance is a suitable illustration for the proposed model, and the conclusions drawn rely entirely on analytical results. Experimental validation will indicate any required modifications to underlying assumptions.

The work in this paper can be extended to review several important crop epidemics. Also, there is a scope for using optimal control theory to optimise the cost-effectiveness of the system [50], [147]. The objective will be to minimise the damage caused by the infected plant populations and the cost of application of pesticides as a control measure. Application of pesticides does not always give immediate recovery of the infected plants. There is a possible delay in the recovery process. Our studied model can be extended to a time-delay model using delay differential equations. Over the years, researchers have paid much attention to the studies of fractional order eco-epidemiological models as well [133], [43], [23]. This work can also be extended using fractional order derivatives. Furthermore, researchers with a keen interest can explore this model by examining contact rates between plants and pesticides, which are entirely dependent on the quantity of pesticides applied. This can be achieved through the utilization of functions that rely on the variable α.