Chapter 7

A Mathematical Analysis of Plant-Pesticide Interaction: Existence, Uniqueness, and Optimal Control

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7.1 Introduction

The study of plant epidemic models in connection with pesticide application is a critical component of agricultural research, particularly in relation to the preservation of crop fitness and the guarantee of food safety. If not effectively controlled, plant epidemics caused by various diseases like fungus, bacteria, and viruses can cause enormous losses in agricultural production. Pesticide application is one of the primary methods used in the management of these epidemics

[203]. Despite this, optimisation is essential when it comes to the use of pesticides, taking into account their effectiveness, costs, and environmental impacts.

Mathematical modelling is essential for understanding plant disease dynamics and the impact of pesticide use. These models are critical tools for predicting disease spread and formulating effective containment strategies [89]. By simulating different situations, they offer valuable insights into the influence of various factors on disease progression and the effectiveness of control strategies. A standard plant epidemic model includes the following basic components: an infection rate, which indicates the rate at which a disease can spread from infected to healthy plants; plant growth and mortality rates that affect overall population dynamics; and the consequences of pesticide application, which can limit virus transmission but also impact plant well-being [195]. Mathematical models incorporate these elements to help academics and practitioners predict disease outbreaks and optimise pesticide use. This ultimately contributes to the development of more sustainable and effective plant disease management strategies [42].

Uniqueness and the existence of solutions are crucial elements of these models. For a model to be credible and useful, it must produce a unique solution that stays valid over time. To ensure reliability, a significant amount of mathematics is required to ensure that, given specific starting points and parameters, the answers to the differential equations describing the epidemic's spread are unambiguous. Essentially, this means proving that the model equations have solutions that not only exist but are also unique for the given inputs. This process is necessary to ensure that the model can accurately predict the spread of the disease and help develop effective control strategies [159].

Another factor to consider is how pesticide applications can be efficiently managed. The optimal control theory provides us with a method for determining which approach to the application of pesticides will minimise the impact of an epidemic while taking into account factors like cost-effective constraints, environmental concerns, and other factors [76]. To put it another way, this process entails formulating an objective function that aims to decrease both the number of infected plants and the amount of pesticide used, and then identifying control variables that optimize this function.

In conclusion, using the uniqueness and existence theorems along with the best control strategies in plant epidemic models while pesticides are being applied gives us a complete way to deal with plant diseases. It provides a comprehensive comprehension of the most effective methods for utilising these chemicals, thereby ensuring that agricultural practices are sustainable and have minimal environmental impact. This field is not only profoundly mathematical but also highly relevant to the practical administration of agriculture.

7.2 Mathematical model

At any given time t, let $S(t)$ represent the susceptible population and $I(t)$ represent the infected population of plants. The total biomass of the plant populations, indicated by $N(t)$, is equal to the sum of $S(t)$ and $I(t)$, i.e., $S(t) + I(t) = N(t)$. Let $P(t)$ denote the amount of pesticides used in the population under consideration. Next, we present the following model:

$$\begin{aligned} \frac{dS}{dt} &= a_1 S(1 - b_1 S) - \frac{\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S \\ \frac{dI}{dt} &= \frac{\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P} \\ \frac{dP}{dt} &= -\alpha P \end{aligned} \tag{7.1}$$

Where:

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.

Biologically, our focus is strictly on the dynamics of system (7.1) within the closed octant R_+^3. Consequently, we take into account the initial conditions $S(0) \equiv S_0 > 0, I(0) \equiv I_0 > 0$ and $P(0) \equiv P_0 > 0$.

The model above represents plant population dynamics under pesticide application. The remaining assumptions are specified in **Chapter 4** of this Thesis.

7.3 Existence and uniqueness of solutions

This section presents the formulation of the existence and uniqueness theorem for the three equations of system (7.1) and the proof of the theorem. To formulate and demonstrate the theorem, the system of equations is approached as follows:

$$\left.\begin{array}{l} y_1' = f_1(t, y_1, y_2, \ldots\ldots, y_n),\ y_1(t_0) = y_{10} \\ y_2' = f_2(t, y_1, y_2, \ldots\ldots, y_n),\ y_2(t_0) = y_{20} \\ \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \\ \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \\ \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \\ y_n' = f_n(t, y_1, y_2, \ldots\ldots, y_n),\ y_n(t_0) = y_{n0} \end{array}\right\} \tag{7.2}$$

We may write equation (7.2) in the compact form as:

$$y' = f(t, y), \quad y(t_0) = y_0 \tag{7.3}$$

Theorem 7.1: [159]

Let D denote the region:

$$\left.\begin{array}{l} |t - t_0| \le a,\ \|y - y_0\| \le b,\ y = (y_1, y_2, \ldots\ldots, y_n) \\ y_0 = (y_{10}, y_{20}, \ldots, y_{n0}) \end{array}\right\} \tag{7.4}$$

and suppose that $f(t, y)$ satisfies the Lipschitz condition:

$$\|f(t, y_1) - f(t, y_2)\| \le K\|y_1 - y_2\| \tag{7.5}$$

whenever the pairs (t, y_1) and (t, y_2) belong to D, where K is a positive constant.

Then there exists a constant $\delta > 0$ such that there exists a unique continuous vector solution $y(t)$ of the system (7.3) in the interval $|t - t_0| < \delta$.

Remark: It is important to note that condition (7.5) is satisfied by the requirement that $\frac{\partial f_i}{\partial y_j}$, $i, j = 1,2, \ldots, n$ is continuous and bounded in D.

Proof: From (7.4), we have,

$$\left.\begin{array}{c} D = (S, I, P, t) \\ \text{Where} \\ |S - S_0| \leq c_1,\ |I - I_0| \leq c_2,\ |P - P_0| \leq c_3,\ |t - t_0| \leq c_4 \end{array}\right\} \tag{7.6}$$

From the system (7.1),

$$\text{Let } f_1 = a_1 S(1 - b_1 S) - \frac{\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S \tag{7.7}$$

$$f_2 = \frac{\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P} \tag{7.8}$$

$$f_3 = -\alpha P \tag{7.9}$$

By theorem (7.1), it is sufficient to show that $\left|\frac{\partial f_i}{\partial y_j}\right|$ are bounded in order to establish the existence and uniqueness of the system (7.1).

Consider the partial derivatives:

$$\frac{\partial f_1}{\partial S} = a_1 - 2a_1 b_1 S - \frac{\beta_1 I}{1+\gamma_1 I} - cP - \mu, \frac{\partial f_1}{\partial I} = -\frac{\beta_1 S}{(1+\gamma_1 I)^2}, \frac{\partial f_1}{\partial P} = -cS \tag{7.10}$$

$$\frac{\partial f_2}{\partial S} = \frac{\beta_1 I}{1+\gamma_1 I}, \frac{\partial f_2}{\partial I} = \frac{\beta_1 S}{(1+\gamma_1 I)^2} - \mu - \frac{\beta_2 P}{1+\gamma_2 P}, \frac{\partial f_2}{\partial P} = -\frac{\beta_2 I}{(1+\gamma_2 P)^2} \tag{7.11}$$

$$\frac{\partial f_3}{\partial S} = 0, \frac{\partial f_3}{\partial I} = 0, \frac{\partial f_3}{\partial P} = -\alpha \tag{7.12}$$

Now, by substituting (7.6) into (7.10) - (7.12), we have,

$$\left.\begin{aligned} \left|\frac{\partial f_1}{\partial S}\right| &= \left|a_1 - 2a_1b_1S - \frac{\beta_1 I}{1+\gamma_1 I} - cP - \mu\right| \\ &\leq \left|a_1 - 2a_1b_1(S_0 + c_1) - \frac{\beta_1(I_0+c_2)}{1+\gamma_1(I_0+c_2)} - c(P + c_3) - \mu\right| < \infty, \\ \left|\frac{\partial f_1}{\partial I}\right| = \left|\frac{-\beta_1 S}{(1+\gamma_1 I)^2}\right| &\leq \left|\frac{\beta_1(S_0+c_1)}{(1+\gamma_1(I_0+c_2))^2}\right| < \infty, \left|\frac{\partial f_1}{\partial P}\right| = |-cS| \leq |c(S_0 + c_1)| < \infty \end{aligned}\right\} \quad (7.13)$$

$$\left.\begin{aligned} \left|\frac{\partial f_2}{\partial S}\right| &= \left|\frac{\beta_1 I}{1+\gamma_1 I}\right| \leq \left|\frac{\beta_1(I_0+c_2)}{1+\gamma_1(I_0+c_2)}\right| < \infty, \\ \left|\frac{\partial f_2}{\partial I}\right| = \left|\frac{\beta_1 S}{(1+\gamma_1 I)^2} - \mu - \frac{\beta_2 P}{1+\gamma_2 P}\right| &\leq \left|\frac{\beta_1(S_0+c_1)}{(1+\gamma_1(I_0+c_2))^2} - \mu - \frac{\beta_2(P+c_3)}{1+\gamma_2(P+c_3)}\right| < \infty, \\ \left|\frac{\partial f_2}{\partial P}\right| = \left|-\frac{\beta_2 I}{(1+\gamma_2 P)^2}\right| &\leq \left|\frac{\beta_2(I_0+c_2)}{(1+\gamma_2(P+c_3))^2}\right| < \infty \end{aligned}\right\} \quad (7.14)$$

$$\left.\left|\frac{\partial f_3}{\partial S}\right| = 0, \left|\frac{\partial f_3}{\partial I}\right| = 0, \left|\frac{\partial f_3}{\partial P}\right| = |-\alpha| = |\alpha| < \infty\right\} \quad (7.15)$$

Clearly, equations (7.10) - (7.12) are bounded in D. Hence, by Theorem (7.1), there exists a unique solution of the system (7.1), i.e., there exists a unique solution of equations (7.10) - (7.12) that satisfies equation (7.6).

7.4 Application of Optimal Control to Plant epidemic system

The time-dependent control is introduced into the model equations (7.1) with the intention of preventing the spread of disease from one plant population to another and distinguishing the susceptible plant population from the affected plant population. In order to achieve optimal control over the effects of plant inoculation, the goal is to identify and comprehend the critical circumstances that are required. The primary model, which is referred to as system (7.1), is extended in order to integrate a dynamic control parameter, which is denoted by $u(t)$ [76]. This parameter represents the control measure that executes throughout the plant inoculation process. The purpose of this control measure is to reduce the risk of plant disease.

Consider the variable $u(t) \in U$ as a control variable on plant inoculation in order to limit the incidence of plant disease. When time-dependent control is incorporated into the updated model (7.1), the following equation is obtained:

$$\begin{aligned}
\frac{dS}{dt} &= a_1 S(1 - b_1 S) - \frac{(1-u)\beta_1 SI}{1+\gamma_1 I} - cPS - \mu S \\
\frac{dI}{dt} &= \frac{(1-u)\beta_1 SI}{1+\gamma_1 I} - \mu I - \frac{\beta_2 IP}{1+\gamma_2 P} \\
\frac{dP}{dt} &= -\alpha P
\end{aligned} \tag{7.16}$$

Here, $U = \{u | u(t)\ is\ measurable\ , 0 \leq u(t) \leq u_{max} < \infty, \mathrm{t} \in [0, t_f]\}$ represents an admissible control set. The objective functional J is defined within a feasible range of the control variable $u(t)$ applied over the finite time interval $(0, t_f)$ is given by:

$$J(u) = \min_u \int_0^{t_f} \left(BI(t) + \frac{1}{2} B_1 u^2(t) \right) dt \tag{7.17}$$

In the presented scenario, t_f represents the final time. B is a positive constant that represents the costs associated with infected plants, while B_1 serves as the relative cost weights for the control measure. Specifically, the objective function $J(u)$ aims to minimise both the overall number of infected plants and the cost of the control $u(t)$. Consequently, the aim is to identify an optimal control $u^*(t)$ that is capable of simultaneously achieving both of these objectives. Hence, the optimal controls $u^*(t)$ exist in such a way that:

$$J(u^*) = min\{J(u)\} \tag{7.18}$$

Here, the existence of an optimal control (7.18) for the system (7.16) will first be demonstrated. This is supported by the following Theorem 7.2.

Theorem 7.2: There exists an optimal control $u^*(t)$ such that $J(u^*(\mathrm{t})) = min\{J(u(t))\}$ subject to the control system (7.1) with initial conditions.

Proof: The integrand of the objective functional $BI(t) + \frac{1}{2}B_1u^2(t)$ is a convex function of the control variable $u(t)$. Both the control and state variables are greater than or equal to zero, and the system (7.1) satisfies Lipchitz criteria in relation to the state variables since the state solutions are bounded. Therefore, it can be concluded that there exists an optimal control [59].

In order to obtain the optimal solution, the initial step is to calculate the Lagrangian and Hamiltonian for the problem described in equations (7.16) to (7.17). The Lagrangian for this optimal control problem is given by:

$$L(I,u) = BI(t) + \frac{1}{2}B_1u^2(t).$$

The goal is to determine the minimum Lagrangian values. This is accomplished by defining the control problem's Hamiltonian as follows:

$$H(I,u,\lambda_S,\lambda_I,\lambda_P,t) = L(I,u) + \lambda_S(t)\frac{dS(t)}{dt} + \lambda_I(t)\frac{dI(t)}{dt} + \lambda_P(t)\frac{dP(t)}{dt} \tag{7.19}$$

Where λ_S, λ_I and λ_P are adjoint variables or co-state variables to be determined. The formulated model must conform to the necessary criteria outlined by Pontryagin's Maximum [76]. Pontryagin's Maximum Principle is commonly used to convert the system of equations (7.16) and (7.17) into a point-wise minimisation problem of the Hamiltonian H, taking into consideration the control variable $u(t)$.

Theorem 7.3: Let $S^*(t), I^*(t), P^*(t)$ represent the optimal state solutions corresponding to the optimal control variable $u^*(t)$ for the optimal control problem (7.16) – (7.17). This implies the existence of adjoint variables λ_S, λ_I and λ_P satisfying:

$$\frac{d\lambda_S}{dt} = \lambda_S[a_1(2b_1S - 1) + cP + \mu] + \frac{\beta_1(1-u)}{1+\gamma_1 I}(\lambda_S - \lambda_I)I,$$

$\frac{d\lambda_I}{dt} = \frac{\beta_1(1-u)}{(1+\gamma_1 I)^2}(\lambda_S - \lambda_I)S + \lambda_I\left(\frac{\beta_2 P}{1+\gamma_2 P} + \mu\right) - B,$

$\frac{d\lambda_P}{dt} = c\lambda_S S + \frac{\beta_2}{(1+\gamma_1 P)^2}\lambda_I I + \alpha\lambda_P,$

with transversality conditions $\lambda_S(t_f),\ \lambda_I(t_f),\ \lambda_P(t_f) = 0.$

Furthermore, the optimal control variable u^* minimises J over the region U and it is given by:

$$u^*(t) = max\left(min\left(\frac{\lambda_I - \lambda_S}{B_1}, u_{max}\right), 0\right).$$

Proof: To derive both the transversality conditions and the adjoint equations, the Hamiltonian (7.19) is utilised. The adjoint equations are derived through the application of Pontryagin's Maximum Principle.

$\frac{d\lambda_S}{dt} = -\frac{\partial H}{\partial S}$, $\frac{d\lambda_I}{dt} = -\frac{\partial H}{\partial I}$ and $\frac{d\lambda_P}{dt} = -\frac{\partial H}{\partial P}$ with $\lambda_S(t_f),\ \lambda_I(t_f),\ \lambda_P(t_f) = 0.$

The optimality of the control problem is determined by $\frac{\partial H}{\partial u} = 0$ on the interior of the control set.

Hence, we get, $B_1 u(t) + \frac{\beta_1 SI}{1+\gamma_1 I}\lambda_S(t) - \frac{\beta_1 SI}{1+\gamma_1 I}\lambda_I(t) = 0.$

This implies that $u(t) = \frac{\beta_1(\lambda_I - \lambda_S)SI}{B_1(1+\gamma_1 I)} (= u^*(t))$, say.

Using the property of control space, we obtain:

$u^*(t) = 0$ if $\frac{\beta_1(\lambda_I - \lambda_S)SI}{B_1(1+\gamma_1 I)} \leq 0,$

$u^*(t) = \frac{\beta_1(\lambda_I - \lambda_S)SI}{B_1(1+\gamma_1 I)}$ if $0 < \frac{\beta_1(\lambda_I - \lambda_S)SI}{B_1(1+\gamma_1 I)} < u_{max}$

$u^*(t) = u_{max}$ if $\frac{\beta_1(\lambda_I - \lambda_S)SI}{B_1(1+\gamma_1 I)} \geq u_{max}.$

So, the optimal control is characterized as $u^*(t) = max\left(min\left(\frac{\lambda_I - \lambda_S}{B_1}, u_{max}\right), 0\right)$. This represents the characterization of the optimal control.

7.5 Numerical Analysis

The proposed model for plant epidemics is studied through numerical methods to see the dynamics of disease spread and the role of optimal control measures in reducing diseases. This numerical analysis is carried out with the help of MATLAB 2018a. The initial conditions are set as $S(0) = 100, I(0) = 50, and\ P(0) = 10$, which represent the initial state for susceptible and infected plant populations and pesticides respectively described in this order. The provided starting values give a reference point for analysing how each population behaves over time as the disease progresses and with application of optimal control strategies. This method helps to understand which strategies are more effective and what the impact of these strategies on disease the control line, giving practical insight into the epidemic management system.

Table 7.1: Parameters and its Values

Parameter	Value
a_1	0.1
b_1	0.001
β_1	0.001
γ_1	0.001
c	0.001
μ	0.06
β_2	0.02
γ_2	0.021
α	0.02
B	1
B_1	0.1
$u(t)$	0.5
t_f	2000

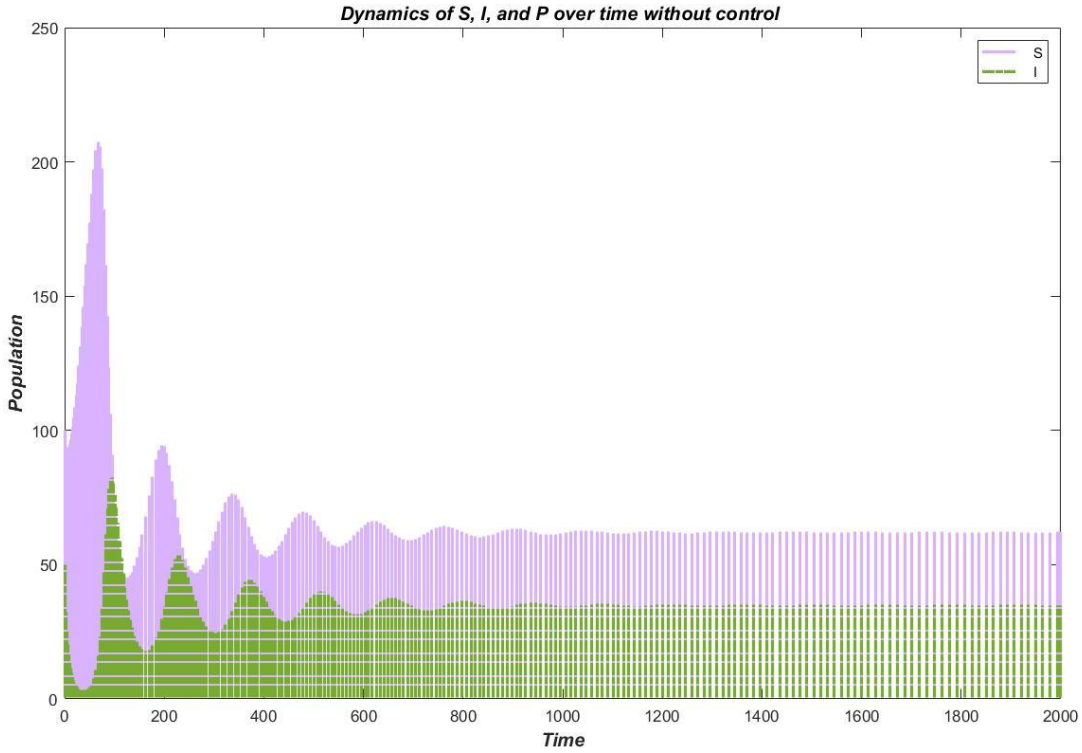

Figure 7.1: Dynamics of S and I over time without control $u(t)$

Figure 7.1 presents a narrative that shows the populations of susceptible and infected plants take much longer to reach equilibrium without the application of pesticide or at $u(t) = 0$. In the absence of these strategies, the infection can spread much more easily, resulting in larger fluctuations and slower stabilisation of plant populations. The drastic difference in this comparison demonstrates the importance of control strategies in managing disease dynamics and helping the system stabilise more quickly.

The infection that spreads without the application of $u(t)$ or pesticides creates longer periods of not only high infections but also more variability in the plant populations. This means that growth and infection dominate the natural processes, which causes slower population stabilisation and larger swings in plant numbers. This all shows how difficult managing plant diseases can be without human intervention, as the populations oscillated much more and took a longer time to reach equilibrium.

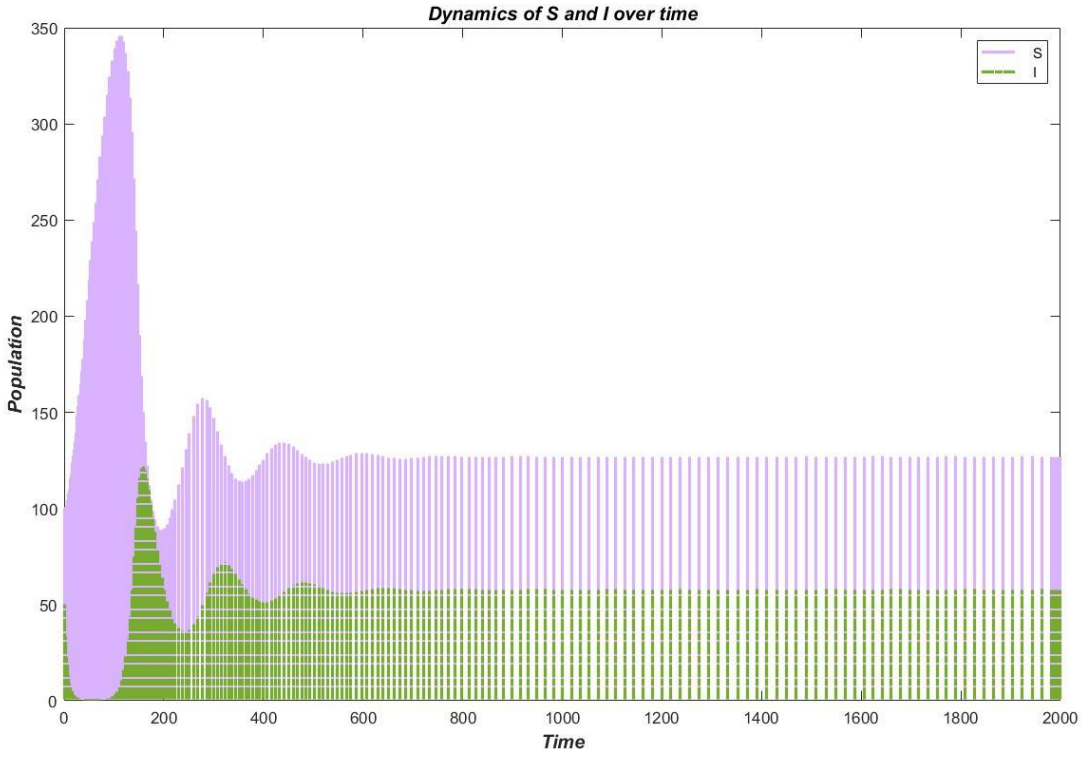

Figure 7.2: Dynamics of S and I over time with control $u(t)$

Figure 7.2 shows that the populations of susceptible plants (S) and infected plants (I) rapidly reach equilibrium values. The results show that setting the control value $u(t) = 0.5$ efficiently stabilises the system's dynamics. The controlled variable $u(t) = 0.5$ directly influences the system's behaviour, leading to a decrease in infections and accelerating system function. By reducing the rate of disease spread and its harmful effects on affected plants, this intervention creates a more controlled and predictable system. Implementing $u(t) = 0.5$ results in a significant decrease in the number of infected plants, thereby stabilising growth among susceptible plants. It is essential to implement this kind of control in order to keep the plant population stable and provide a more seamless transition towards equilibrium levels.

Pesticide use can help to balance the effects of the control variable $u(t) = 0.5$. Pesticides stabilise the population growth rate of susceptible plants by further reducing the number of unhealthy plants. Rapid outbreaks or extremely high infection rates require this type of response.

Pesticides have the ability to stabilise plant populations by reducing their erratic growth patterns. Pesticides act as an extra-stabilising component, preventing sudden shifts or variations in plant populations. Avoiding extreme fluctuations in plant populations makes this concept a reality. This demonstrates the pesticide's efficacy in controlling plant diseases and its contribution to maintaining a steady and predictable plant ecosystem balance. Using pesticides with the control variable $u(t) = 0.5$ is important for keeping plant populations healthy and stable over time because it lowers the rate of transmission and lessens the damage to diseased plants.

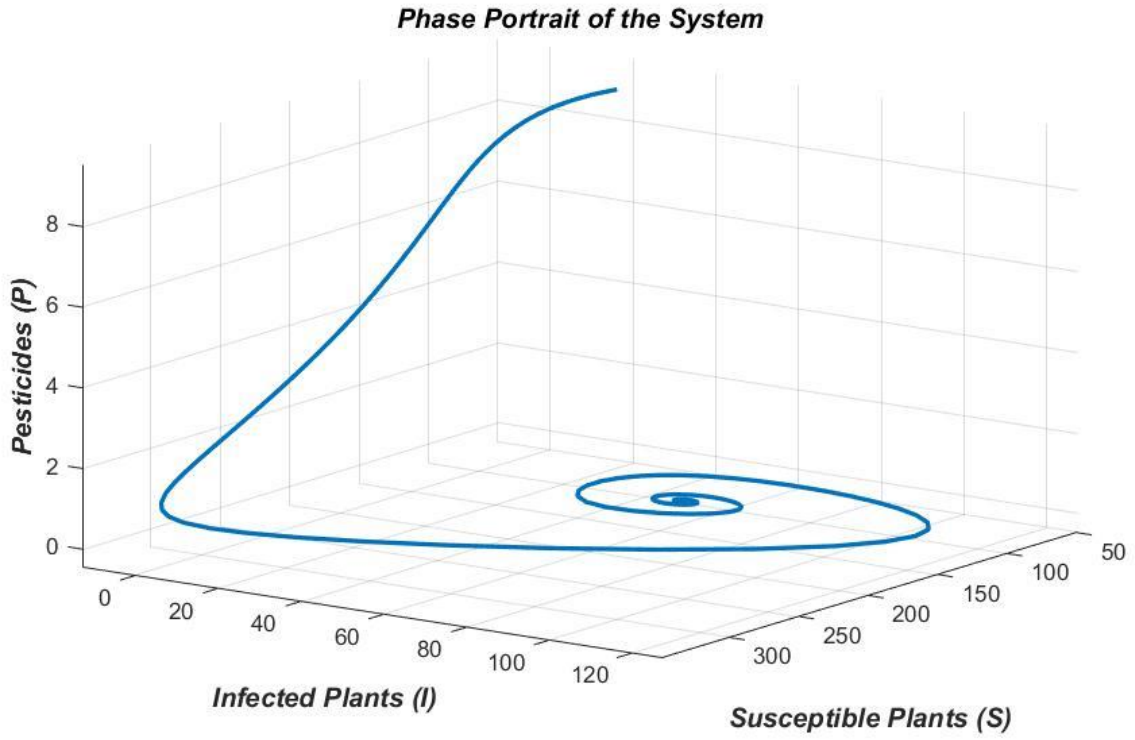

Figure 7.3: Phase Portrait of the System

The phase portrait in Figure 7.3 provides a dynamical view of the interaction between susceptible plants (S), infected plants (I), and pesticide levels (P) over time. The axes in this three-dimensional plot indicate the quantity of susceptible plants, infected plants, and pesticide level, and each point depicts the system's condition at that specific time.

The phase portrait shows that the system is progressively approaching a stable state where the populations of susceptible plants, infected plants, and pesticide levels stay constant. In the phase portrait, the trajectory spirals inward towards a central equilibrium point. The system may wobble at first with some oscillations, but these fluctuations eventually diminish and bring about stability, according to this spiralling pattern. The pesticide level (P) starts high but gradually decreases due to its natural degradation rate (α), which influences infection dynamics by reducing interactions between infected and susceptible plants and helping to control the spread of infection.

Initially, the populations of susceptible and infected plants oscillate more significantly, reflecting changes as the system adjusts to the pesticide's presence. Due to its growth rate and the initial decrease in infection, the susceptible plant population (S) initially grows, while the infected plant population (I) experiences comparable fluctuations as the infection progresses and the pesticide regulates it. Over time, the fluctuations decrease, resulting in stable populations of S, I, and P, which demonstrates the sustained effectiveness of the control strategy.

The inward spiralling trajectory provides a demonstration of the effectiveness of the control $u(t) = 0.5$ in stabilising the system. The pesticide treatment allows susceptible plants to recover and thrive, thereby reducing the number of affected plants. The system is stable under the provided control approach, as evidenced by this progression towards equilibrium. Over time, the system has smoothed out any initial disruptions, resulting in a predictable and steady state. Finally, the system establishes a balance in which the levels of susceptible plants, infected plants, and pesticides remain constant, implying that methods of sustainable farming may manage the infection without requiring ongoing, large-scale interventions. As a result, the phase portrait effectively demonstrates how the control method affects the interactions between susceptible and infected plants, as well as the amounts of pesticides, resulting in a system that is stable and manageable over time.

7.6 Conclusion

This study investigates the ways in which the use of pesticides impacts the transmission of plant diseases. In particular, it validates the existence of the model and guarantees that there is a unique solution to it. Another contribution to the study is the identification of optimal control strategies for efficient plant disease management. The research findings provide farmers with actionable guidance on how to use pesticides in a sustainable manner. It is vital for farmers to have this knowledge in order to assist them in achieving a balance between effective pest control, environmental preservation, and economic viability, which will ultimately lead to more sustainable farming practices.