Chapter 6

Optimizing Plant Epidemic Control: A Mathematical Model Integrating Susceptible Plants, Infectives, and Herbivores with Pesticide Intervention

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Optimizing Plant Epidemic Control: A Mathematical Model Integrating Susceptible and Infectives Plants, and Herbivores with Pesticide Intervention

6.1 Introduction

Research on infectious diseases is an important component of biomathematics, as it enables researchers to study the impact of different epidemiological factors on ecosystems. This branch

of mathematics has made significant advances over the years. Mathematical ecology and mathematical epidemiology are two distinct topics in biology and applied mathematics. Ecological epidemiological models, a significant field in biological mathematics, emerged as a result of numerous research attempts to integrate simultaneous research in ecology and epidemiological models. For many years, infectious diseases posed a serious threat to plant populations, which in turn endangered animal and human health as well as the environment [76]. Recently, mathematicians and ecologists have teamed up to study epidemiology and ecology from a mathematical perspective [139]. As a result, several mathematical model equations have emerged, which are now essential resources for studying plant disease populations and developing strategies to control them, including the use of pesticides and natural enemies [2]. In mathematical modelling, we first express a real-world problem as a formula and then solve it mathematically. Next, we translate the outcome into a real-world language [110]. Ecosystems, epidemiology, cancer, diabetes, HIV, and demographics are just a few of the many biological fields that have benefited from mathematical modelling in the life sciences [56, 104, 137].

Plants and herbivores interact in a natural environment that is dynamic and complex, the result of millions of years of evolution. In order to protect themselves from herbivores, plants utilise a wide variety of defence mechanisms in these interactions [204], including physical and chemical defences, mimicry, and indirect defences. These interactions are critical in ecosystem formation, have an impact on the evolutionary processes of herbivores and plants, and preserve ecological balance in the natural world. The numerous ways in which plants and herbivores interact are fundamental to the dynamics of ecosystems. Herbivorous animals, such as birds, mammals, and insects, obtain the majority of their nutrition from plant tissues. By ingesting plant material, herbivores engage in this direct interaction, which can result in a variety of consequences for the plants. A variety of defence mechanisms have evolved in plants to repel herbivores and safeguard themselves [122]. These barriers may be physical or chemical in nature. Certain plants, for instance, are capable of producing bitter-tasting or toxic compounds that discourage herbivores from consuming them. Certain plant species have evolved physical barriers such as thorns, spines, or tough leaves, which render them less attractive or more challenging for herbivores to ingest. Herbivores and plants engage in a dynamic and ever-changing interaction. This frequently results in a co-evolutionary "arms race," wherein herbivores evolve mechanisms

that overcome the defences that plants develop in response to their presence. The continuous nature of this process gives rise to an ever-evolving dynamic between the two. In addition, plants are capable of interacting with one another and other organisms to defend against herbivore attacks [5]. Herbivore-induced injury to a single plant can result in the emission of volatile organic compounds (VOCs), which function as chemical messengers for adjacent plants. By informing adjacent plants of potential herbivore threats, these volatile organic compounds (VOCs) enable them to activate their own defence mechanisms. Additionally, certain plant VOCs, such as parasitoid wasps and predatory insects, attract natural herbivore enemies [11]. These herbivore-controlling natural adversaries contribute to an intricate web of ecosystem interactions [7, 114].

Herbivores have both direct and indirect impacts on ecosystems, leading to significant consequences. The possibility of trophic cascades is an important indirect consequence. Modifications in herbivore populations can initiate a series of interconnected consequences across the entire ecosystem. When the number of predators decreases, the number of herbivores can increase without any restrictions, resulting in excessive grazing and a decrease in plant richness and variety [166]. Consequently, this has an impact on other species that rely on these plants for nutrition and shelter, triggering a sequence of ecological transformations. In contrast, the presence of predators can play a role in controlling herbivore numbers, thereby ensuring a stable equilibrium within the ecosystem [28]. Herbivores are also essential for the process of nutrient cycling in ecosystems. They actively participate in nutrient redistribution by ingesting plant material and excreting waste. They actively participate in the redistribution of nutrients. Herbivores absorb plant nutrients into their bodies, which they then excrete into the environment through their waste and urine [44]. The process of recycling nutrients can have an impact on the availability of nutrients in the soil, which in turn benefits the growth of nearby plants. Herbivores can sometimes impact the nitrogen levels in plant tissues, which in turn affects the quality of plants as a food source for higher trophic levels [106]. In addition, herbivores have the ability to establish mutually advantageous associations with plants [169]. For example, certain herbivores, such as bees and butterflies, play the role of pollinators for flowering plants. These insects unintentionally transport pollen between the flowers while searching for nectar or pollen, which helps plants reproduce. The plants reciprocate by offering the herbivores an important source of

nourishment. Furthermore, animals that consume fruit play a critical role in plant seed dispersal. These animals help to spread and colonize plant species by ingesting fruits and excreting seeds in various areas. These mutually beneficial partnerships emphasise the complex network of interactions between herbivores and plants in ecosystems.

Plants, herbivores, and pesticides engage in complex interactions that manifest in diverse ways. Pesticides are chemical compounds designed to manage or eradicate pests, such as herbivorous insects and other organisms that consume plants. Pesticides, when sprayed on plants, function as a means of protection. The application of the treatment might vary and include sprays, dusts, or systemic methods [58]. Herbivores, when consuming these treated plants, may ingest the pesticides, resulting in direct poisoning. This can inflict harm upon herbivores, resulting in a spectrum of consequences ranging from physical pain to mortality. Furthermore, pesticide residues may remain on plant surfaces or within plant tissues, exposing herbivores to these chemicals long after they have consumed them. These residues may have sublethal effects or gradually accumulate over time. An essential element of this connection is the possibility of unintended consequences. Pesticides can harm non-targeted organisms, including beneficial insects and wildlife that interact with herbivores. Collateral damage can disrupt ecosystems and have far-reaching ecological consequences. Over time, certain herbivorous pests may acquire resistance to pesticides, thus requiring the use of more potent or alternative chemicals. This resistance is a result of genetic alterations in the pest population. Furthermore, pesticides may occasionally affect the plants themselves. Exposure to pesticides can cause phytotoxicity when plants show visible damage or strain. This emphasises the significance of careful consideration when selecting and administering insecticides.

The ecological impacts of pesticides are a crucial factor to take into account in agricultural and pest management practices. Pesticides are chemical substances created to manage or eradicate pests, with the capacity to not only affect the intended pests but also the wider ecosystem. These chemicals can enter water bodies through runoff from fields, contaminating aquatic ecosystems. Pesticide residues present in water can have detrimental effects on aquatic organisms, such as fish and aquatic invertebrates, causing disturbances in the delicate equilibrium of these vulnerable ecosystems. In addition, pesticides have the ability to remain in the soil for long

periods of time, which can have an impact on soil quality and the well-being of species that were not the intended target. In response to these environmental concerns, the adoption of integrated pest management (IPM) tactics has gained significant popularity in the field of agriculture. Integrated Pest Management (IPM) is a comprehensive strategy that seeks to effectively control pests while reducing the environmental consequences of pesticide application. This method integrates a range of strategies, including biological control, cultural practices, and the cautious application of pesticides as a final option. Biological control refers to the use of natural predators, parasites, or pathogens to manage and control pest populations. Cultural practices include crop rotation, the deliberate selection of pest-resistant crop varieties, and habitat management to promote the presence of beneficial creatures that feed on pests. The core principle of Integrated Pest Management (IPM) is to employ pesticides solely when deemed essential, opting for the least harmful alternatives, thereby reducing their adverse effects on non-target organisms and the ecosystem [95].

6.2 Mathematical model formulation and description

In our mathematical model, we have taken into account three distinct populations, namely the plant populations and the herbivores population. The plant and herbivore populations have been further classified into two subpopulations and single population, respectively. These subpopulations include the susceptible plants, denoted by $X_1(t)$, and the infected plants, denoted by $X_2(t)$. The herbivore population is denoted by the variable $Y(t)$. Our model aims to analyse the dynamics of these populations and their interactions. By studying the behaviour of these populations, we can gain valuable insights into the ecological processes that govern their growth and survival. Pesticides represented by $Z(t)$ are utilised as a control mechanism in the model and are administered to both the susceptible and infected plants. The main goal of pesticide treatment is to reduce the occurrence of diseases among plant populations. Through the application of pesticides, we can protect our crops and guarantee their enduring health and productivity. In the absence of this regulatory mechanism, there is a significant danger of losing entire crops to the detrimental effects of diseases and pests. It is essential that we persist in the responsible and efficient use of pesticides to protect our food supply and promote sustainable agriculture. In the present study, a model is developed based on a series of assumptions. The model has been formulated on the basis of these assumptions:

A. At any given time t, the plant populations can be classified into two distinct categories, namely the susceptible plants denoted as $X_1(t)$ and the infected plants denoted as $X_2(t)$. The sum of these two categories represents the total biomass of the plant populations, which is expressed as $X_1(t) + X_2(t)$. Additionally, assuming that $N(t)$ represents the total biomass of the plant populations, it follows that $X_1(t) + X_2(t) = N(t)$. This mathematical relationship provides a useful framework for understanding the dynamics of plant populations and their susceptibility to infection.

B. In the realm of plant classifications, the population of susceptible plants, denoted as X_1, is subject to the logistic law of growth, characterized by an intrinsic growth rate denoted as r, and an environmental carrying capacity denoted as K. As a result, the alteration of biomass within the susceptible plant populations can be expressed as a differential equation in the following manner:

$$\frac{dX_1}{dt} = \mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right). \tag{6.1}$$

C. The spread of the disease among susceptible plants primarily occurs due to their direct interaction with infected plants, with the force of infection denoted as β. Herbivores represented by $Y(t)$, are a common threat to plants, consuming both susceptible and infected ones. However, infected plants are considerably more susceptible to herbivores due to their weakened state, making them easier prey. In contrast, predation of susceptible plants requires some handling time [99]. Consequently, we assume that herbivores prey on susceptible plants at a rate following a Holling type I functional response, expressed as aX_1Y with a representing the maximum capture rate. Additionally, herbivores target infected plants with a Holling type I functional response denoted as dX_2Y where d signifies the maximum capture rate. Furthermore, we take into account the application of pesticides, denoted as $Z(t)$, to both susceptible and infected plants. The application rates follow Holling type I responses, characterized by bX_1Z for susceptible plants and eX_2Z for infected plants. Here, b represents the contact rate between susceptible plants and pesticides, while e signifies the contact rate between infected plants and pesticides. Additionally, we consider that both susceptible and

infected plants experience a natural death rate denoted as c. As a result, we can separate the rate of change for susceptible and infected plants as follows:

$$\frac{dX_1}{dt} = \mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_1X_2 - aX_1Y - bX_1Z - cX_1, \qquad (6.2)$$

$$\frac{dX_2}{dt} = \beta X_1X_2 - dX_2Y - eX_2Z - cX_2. \qquad (6.3)$$

D. Pesticides can have diverse impacts on herbivorous animals that consume plants treated with these chemicals. These effects can be direct or indirect and vary depending on the specific pesticide, application method, and ecosystem. It is hypothesised that herbivores feeding on plants sprayed with pesticides can negatively impact other herbivores. This interaction between herbivores and pesticides can be classified as a Holling type I response occurring at a rate denoted by fYZ, with f denoting the degree to which herbivores engage with plants that have been subjected to pesticide application. This dynamic interaction can result in adverse outcomes for herbivores, potentially leading to mortality occurring at a rate denoted by g. Therefore, the rate of change for the herbivores population is as follows:

$$\frac{dY}{dt} = aX_1Y + dX_2Y - fYZ - gY. \qquad (6.4)$$

E. Pesticides are utilised in both susceptible and infected plant populations, with the understanding that their use has an impact on both groups. To implement disease control measures, we assume the application of a general pesticide denoted as $Z(t)$. This pesticide is employed to reduce disease levels in the plant populations at a rate described by the equation $-\rho Z + \delta$, where ρ represents the rate at which pesticides is being used and δ is the constant amount of pesticides used. Hence, the rate of change of pesticides is given as follows:

$$\frac{dZ}{dt} = -\rho Z + \delta. \qquad (6.5)$$

Taking into account all the aforementioned assumptions, we formulate our ultimate eco-epidemic system in the following manner:

$$\frac{dX_1}{dt} = \mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_1X_2 - aX_1Y - bX_1Z - cX_1$$
$$\frac{dX_2}{dt} = \beta X_1X_2 - dX_2Y - eX_2Z - cX_2$$
$$\frac{dY}{dt} = aX_1Y + dX_2Y - fYZ - gY$$
$$\frac{dZ}{dt} = -\rho Z + \delta. \tag{6.6}$$

subject to the initial conditions: $X_1(0) > 0$, $X_2(0) > 0$, $Y(0) > 0$ and $Z(0) > 0$. (6.7)

6.3 Preliminaries

In this section, the aim is to demonstrate the positivity, boundedness and Existence and Uniqueness of the model for the solutions of system (6.6).

6.3.1 Positivity of solutions

In order for system (6.6) to possess biological significance and be well-defined, it is necessary that any solutions originating from positive initial data will consistently maintain their positivity for all time intervals where $t > 0$. This crucial aspect will be established through the application of the following theorem.

Theorem 6.1: Let the parameters for model (6.6) be positive constants. A non-negative solution $(X_1(t), X_2(t), Y(t), Z(t))$ for model (6.6) exists for all states variables with positive initial conditions $(X_1(0) > 0, X_2(0) > 0, Y(0) > 0, Z(0) > 0)$ for all $t \geq 0$.

Proof: From the first equation of model (6.6), we have,

$$\frac{dX_1}{dt} = X_1\left[\mathrm{r}\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_2 - aY - bZ - c\right]. \tag{6.8}$$

Let $l(X_1, X_2, Y, Z) = \mathrm{r}\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_2 - aY - bZ$. Then the equation (6.8) becomes:

$$\frac{dX_1}{dt} = X_1[l(X_1, X_2, Y, Z) - c].$$

By separation of variable method, we have,

$$\frac{dX_1}{X_1} = [l(X_1, X_2, Y, Z) - c]dt.$$

Hence,

$$\ln X_1(t) = \int_0^t [l(X_1(\epsilon), X_2(\epsilon), Y(\epsilon), Z(\epsilon)) - c]d\epsilon + C_0$$

So that

$$X_1(t) = C_1 + e^{\int_0^t [l(X_1(\epsilon), X_2(\epsilon), Y(\epsilon), Z(\epsilon)) - c]d\epsilon} > 0 \ (\because \ C_1 = e^{C_0}). \tag{6.9}$$

We conclude that $X_1(t) > 0 \ \forall \ t \geq 0$. Next, we consider $X_2(t) \ \forall \ t \geq 0$. From the second equation of the model (6.6), we have,

$$\frac{dX_2}{dt} = \ \beta X_1 X_2 - dX_2 Y - eX_2 Z - \ cX_2 \geq -(dY + eZ + c)X_2.$$

The integration of the above inequality gives $X_2(t) \geq X_2(0)e^{\int_0^t [-(dY(s) + eZ(s) + c)]ds}$. Since $X_2(0) > 0$ from the initial condition, we conclude that $X_2(t) \geq X_2(0)e^{\int_0^t [-(dY(s) + eZ(s) + c)]ds} > 0 \ \forall \ t \geq 0$. Similarly, using the same argument, it can be shown that $Y(t) \geq Y(0)e^{\int_0^t [-(fZ(s) + g)]ds} > 0$ and $Z(t) \geq Z(0)e^{-\rho t} > 0$. Thus, the solutions of system (6.6) remain positive for all $t \geq 0$, meaning that the model is meaningful and well posed. This completes the proof of the theorem.

6.3.2 Boundedness of solutions

Theorem 6.2: All solutions of the system (6.6) which initiate in R_+^4 are uniformly bounded.

Proof: Define a function $W(t) = X_1(t) + X_2(t) + Y(t) + Z(t)$. Then, on taking the derivative with respect to time t,we get:

$$\frac{dW}{dt} = \frac{dX_1}{dt} + \frac{dX_2}{dt} + \frac{dY}{dt} + \frac{dZ}{dt}. \tag{6.10}$$

By substituting the model equations (6.6) in (6.10), one gets:

$$\frac{dW}{dt} \leq \mathrm{r}X_1\left(1 - \frac{X_1}{K}\right) - bX_1Z - cX_1 - eX_2Z - cX_2 - fYZ - gY - \rho Z + \delta.$$

or

$$\frac{dW}{dt} \leq \mathrm{r}X_1 - cX_2 - gY - \rho Z \leq (r+1)X_1 - (X_1 + cX_2 + gY + \rho Z) \leq \widehat{K}(r+1) - hW$$

Where, $\widehat{K} = max(X_1(0), K)$ and $h = min\{1 + c + g + \rho\}$.

The equation $\frac{dW}{dt} + hW \leq \widehat{K}(r+1)$ has a solution $W \leq \frac{\widehat{K}}{h}(r+1)(1 - e^{-ht})$.

As $t \to \infty$, we have $W \leq \frac{\widehat{K}}{h}(r+1)$, implying that the solution is bounded for $0 \leq W \leq \frac{\widehat{K}}{h}(r+1)$. Thus, all the solutions of the system (6.6) are confined in the region: $\Omega = \left\{(X_1, X_2, Y, Z) \in R_+^4 : W \leq \frac{\widehat{K}}{h}(r+1) + \omega\right\}$ for all $\omega > 0$ and $t \to \infty$.

6.3.3 Existence and Uniqueness of the system

In this section, the existence and uniqueness of the solutions of the eco-epidemic system (6.6) in the region $\Pi \times (0, T]$ are investigated.

Here, $\Pi = \{(X_1, X_2, Y, Z) \in \mathbb{R}^4 : max\{|X_1|, |X_2|, |Y|, |Z|\} \leq \varphi\}$, for sufficiently large φ.

Theorem 6.3: For each $S_0 = \left[X_{1(0)}, X_{2(0)}, Y_{(0)}, Z_{(0)}\right] \in \Pi$, there exists a unique solution $S(t) \in \Pi$ of the eco-epidemic system (6.6), which is defined for all $t \geq 0$.

Proof: Define a mapping $G(S) = \left(G_1(S), G_2(S), G_3(S), G_4(S)\right)$, in which:

$$\begin{aligned} G_1(S) &= \mathrm{r}X_1\left(1 - \frac{X_1 + X_2}{K}\right) - \beta X_1X_2 - aX_1Y - bX_1Z - cX_1, \\ G_2(S) &= \beta X_1X_2 - dX_2Y - eX_2Z - cX_2, \\ G_3(S) &= aX_1Y + dX_2Y - fYZ - gY, \\ G_4(S) &= -\rho Z + \delta. \end{aligned} \tag{6.11}$$

For each $S, \bar{S} \in \Pi$, it follows from (6.6) that:

$$\|G(S) - G(\bar{S})\| = |G_1(S) - G_1(\bar{S})| + |G_2(S) - G_2(\bar{S})| + |G_3(S) - G_3(\bar{S})| + |G_4(S) - G_4(\bar{S})|. \quad (6.12)$$

Now,

$$|G_1(S) - G_1(\bar{S})|$$
$$= \left|\mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_1X_2 - aX_1Y - bX_1Z - cX_1 - \mathrm{r}\overline{X_1}\left(1 - \frac{\overline{X_1}+\overline{X_2}}{K}\right) + \beta\overline{X_1}\,\overline{X_2} + a\overline{X_1}\,\bar{Y} + b\overline{X_1}\,\bar{Z} + c\overline{X_1}\right|,$$
$$= \left|r(X_1 - \overline{X_1}) - \frac{\mathrm{r}X_1(X_1+X_2)}{K} - \beta X_1X_2 - aX_1Y - bX_1Z - cX_1 + \frac{\mathrm{r}\overline{X_1}(\overline{X_1}+\overline{X_2})}{K} + \beta\overline{X_1}\,\overline{X_2} + a\overline{X_1}\,\bar{Y} + b\overline{X_1}\,\bar{Z} + c\overline{X_1}\right|,$$
$$\leq |r(X_1 - \overline{X_1})| + \frac{\mathrm{r}}{K}\left|X_1^2 - \overline{X_1}^2\right| + \left(\frac{r}{K} + \beta\right)|X_1X_2 - \overline{X_1}\,\overline{X_2}| + a|X_1Y - \overline{X_1}\,\bar{Y}| + b|X_1Z - \overline{X_1}\,\bar{Z}| + c|X_1 - \overline{X_1}|,$$
$$\leq r|(X_1 - \overline{X_1})| + \frac{2\mathrm{r}}{K}\varphi|X_1 - \overline{X_1}| + \left(\frac{r}{K} + \beta\right)\varphi|X_1 - \overline{X_1}\,| + \left(\frac{r}{K} + \beta\right)\varphi|X_2 - \overline{X_2}\,| + a\varphi|X_1 - \overline{X_1}\,| + a\varphi|Y - \bar{Y}\,| + b\varphi|X_1 - \overline{X_1}\,| + b\varphi|Z - \bar{Z}\,| + c|X_1 - \overline{X_1}|.$$

$$|G_2(S) - G_2(\bar{S})|$$
$$= |\beta X_1X_2 - dX_2Y - eX_2Z - cX_2 - \beta\overline{X_1}\,\overline{X_2} + d\overline{X_2}\bar{Y} + e\overline{X_2}\bar{Z} + c\overline{X_2}|,$$
$$\leq \beta\varphi|X_1 - \overline{X_1}| + \beta\varphi|X_2 - \overline{X_2}| + d\varphi|X_2 - \overline{X_2}\,| + d\varphi|Y - \bar{Y}\,| + e\varphi|X_2 - \overline{X_2}\,| + e\varphi|Z - \bar{Z}\,| + c|X_2 - \overline{X_2}\,|.$$

$$|G_3(S) - G_3(\bar{S})|$$
$$= |aX_1Y + dX_2Y - fYZ - gY - a\overline{X_1}\,\bar{Y} - d\overline{X_2}\bar{Y} + f\bar{Y}\,\bar{Z} + g\bar{Y}|,$$
$$\leq a\varphi|X_1 - \overline{X_1}\,| + a\varphi|Y - \bar{Y}\,| + d\varphi|X_2 - \overline{X_2}\,| + d\varphi|Y - \bar{Y}\,| + f\varphi|Y - \bar{Y}\,| + f\varphi|Z - \bar{Z}\,| + g|Y - \bar{Y}\,|.$$

$$|G_4(S) - G_4(\bar{S})| = |-\rho Z + \delta + \rho\bar{Z} - \delta| \leq \rho|Z - \bar{Z}|.$$

Then equation (6.12) becomes:

$$\|G(S) - G(\bar{S})\|$$

$$\leq r|(X_1 - \overline{X_1})| + \frac{2r}{K}\varphi|X_1 - \overline{X_1}| + \left(\frac{r}{K} + \beta\right)\varphi|X_1 - \overline{X_1}| + \left(\frac{r}{K} + \beta\right)\varphi|X_2 - \overline{X_2}| + a\varphi|X_1 - \overline{X_1}| + a\varphi|Y - \bar{Y}| + b\varphi|X_1 - \overline{X_1}| + b\varphi|Z - \bar{Z}| + c|X_1 - \overline{X_1}| + \beta\varphi|X_1 - \overline{X_1}| + \beta\varphi|X_2 - \overline{X_2}| + d\varphi|X_2 - \overline{X_2}| + d\varphi|Y - \bar{Y}| + e\varphi|X_2 - \overline{X_2}| + e\varphi|Z - \bar{Z}| + c|X_2 - \overline{X_2}| + a\varphi|X_1 - \overline{X_1}| + a\varphi|Y - \bar{Y}| + d\varphi|X_2 - \overline{X_2}| + d\varphi|Y - \bar{Y}| + f\varphi|Y - \bar{Y}| + f\varphi|Z - \bar{Z}| + g|Y - \bar{Y}| + \rho|Z - \bar{Z}|,$$

$$\leq \left\{r + \left(\frac{3r}{K} + 2\beta + 2a + b\right)\varphi + c\right\}|X_1 - \overline{X_1}| + \left\{\left(\frac{r}{K} + 2\beta + 2d + e\right)\varphi + c\right\}|X_2 - \overline{X_2}| + \{(2a + 2d + f)\varphi + g\}|Y - \bar{Y}| + \{(b + e + f)\varphi + \rho\}|Z - \bar{Z}|,$$

$$\leq H_0\|S - \bar{S}\|.$$

Where:

$$H_0 = max\left\{r + \left(\frac{3r}{K} + 2\beta + 2a + b\right)\varphi + c, \left(\frac{r}{K} + 2\beta + 2d + e\right)\varphi + c, (2a + 2d + f)\varphi + g, (b + e + f)\varphi + \rho\right\}.$$

Hence, $G(S)$ satisfies the Lipschitz condition with respect to S, which implies the existence and uniqueness of solution of the system (6.6).

6.4 Equilibria and Stability

The eco-epidemic model (6.6) has the following four equilibrium points:

I. The trivial equilibrium point $P_1 = \left(0,0,0,\frac{\delta}{\rho}\right)$, which always exists. From an ecological perspective, the concept of trivial equilibrium holds significance as it ensures that no population will face simultaneous extinction. This underscores the importance of maintaining a balance in the ecosystem, as it allows for the survival and sustainability of various plants and animal species over time.

II. The axial equilibrium point, denoted as $P_2 = \left(K,0,0,\frac{\delta}{\rho}\right)$, which represents a state in which the population of healthy plants not only sustains but also thrives and is always feasible.

III. The disease-free equilibrium point $P_3 = \left(\frac{f\delta+g\rho}{a\rho}, 0, \frac{Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)}{K\rho a^2}, \frac{\delta}{\rho}\right)$, represents a state in which plants are free from any disease. This state is of utmost importance in the

field of agriculture as it ensures the health and well-being of plant crops. Additionally, it creates an environment where herbivores can safely feed on plants without the risk of disease transmission, ensuring their well-being.

Now, we aim to determine the basic reproduction number R_0 of System (6.6) at the disease-free equilibrium point P_3. Let us introduce matrices F and V in the following manner:

$$F = \begin{bmatrix} \frac{\beta(f\delta+g\rho)}{a\rho} & 0 \\ 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \frac{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}{K\rho a^2} & 0 \\ 0 & 0 \end{bmatrix}.$$

Using the next generation matrix method developed by Van den Driessche and Watmough [47], the basic reproduction number, denoted as R_0, can be determined as the spectral radius of the next generation operator FV^{-1}. Hence, $R_0 = \sigma(FV^{-1}) = \frac{K\beta a(f\delta+g\rho)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}$. R_0 holds a pivotal role in the field of epidemiology and disease modelling, particularly when it comes to understanding the dynamics of infectious diseases within populations of susceptible individuals, such as plants, in this context. It serves as a fundamental concept for assessing the contagiousness and potential for disease transmission [46]. It can be defined as the average rate of infection within the susceptible plant population resulting from a multitude of secondary infections.

IV. The coexistence equilibrium point $P_4 = (X_1^*, X_2^*, Y^*, Z^*)$, where:

$$X_1^* = \frac{Ka(e\delta+c\rho)+Kdr\rho-Kd(b\delta+c\rho)-(K+r)(f\delta+g\rho)}{K\beta\rho a+rd\rho-a\rho(K+r)},$$

$$X_2^* = \frac{(f\delta+g\rho)(K\beta\rho a+rd\rho-a\rho(K+r))-a\rho\big(Ka(e\delta+c\rho)+Kdr\rho-Kd(b\delta+c\rho)-(K+r)(f\delta+g\rho)\big)}{d\rho(K\beta\rho a+rd\rho-a\rho(K+r))},$$

$$Y^* = \frac{\beta\rho\big(Ka(e\delta+c\rho)+Kdr\rho-Kd(b\delta+c\rho)-(K+r)(f\delta+g\rho)\big)-(c\rho+e\delta)(K\beta\rho a+rd\rho-a\rho(K+r))}{d\rho(K\beta\rho a+rd\rho-a\rho(K+r))},$$

$$Z^* = \frac{\delta}{\rho}.$$

The coexistence equilibrium between plants and herbivores represents a state in which plant species and herbivorous have developed strategies to coexist within an ecosystem, with neither group dominating the other. This equilibrium is maintained through a combination of factors,

including herbivore feeding preferences, plant defence mechanisms, and the influence of predators and resource availability, ultimately promoting biodiversity and ecosystem stability.

Now, the local stability of the system (6.6) is investigated. The Jacobian matrix J is given as follows:

$$J = \begin{bmatrix} J_{11} & \frac{-(\beta K + r)X_1}{K} & -aX_1 & -bX_1 \\ \beta X_2 & J_{22} & -dX_2 & -eX_2 \\ aY & dY & J_{33} & -fY \\ 0 & 0 & 0 & -\rho \end{bmatrix}, \tag{6.13}$$

Where, $J_{11} = r\left(1 - \frac{2X_1 + X_2}{K}\right) - \beta X_2 - aY - bZ - c,$

$J_{22} = \beta X_1 - dY - eZ - c,$

$J_{33} = aX_1 + dX_2 - fZ - g.$

The eigenvalues of the Jacobian matrix J evaluated at the trivial point $P_1\left(0,0,0,\frac{\delta}{\rho}\right)$ are given by $r - \left(c + \frac{b\delta}{\rho}\right), -\left(c + \frac{e\delta}{\rho}\right), -\left(g + \frac{f\delta}{\rho}\right)$ and $-\rho$. Consequently, when $r - \left(c + \frac{b\delta}{\rho}\right) > 0$ and for all parameter values, the equilibrium point P_1 is classified as a saddle point with three-dimensional stable manifolds and a one-dimensional unstable manifold. Conversely, when $r - \left(c + \frac{b\delta}{\rho}\right) < 0$, all eigenvalues are negative, indicating that P_1 is a stable equilibrium point.

Remark: In practical terms, the health and survival of a plant population hinge on a delicate balance between two critical factors: birth rate and natural death rate. The birth rate refers to the rate at which new plants are born or seeds are germinated, while the natural death rate represents the rate at which plants in the population naturally die off due to various factors such as aging, disease, predation, or adverse environmental conditions. When the birth rate of plants is less than its natural death rate, the plant population is at risk of decline and potential extinction, especially if adverse conditions persist. Even though mathematically the equilibrium point seems stable, it signifies a precarious situation for the population because it suggests that the population cannot sustain itself in the long run under these conditions. Therefore, it highlights the vulnerability of

the plant population to adverse environmental factors and underscores the necessity for conservation measures or strategies to secure its survival.

Theorem 6.4: If $\beta K < c + \frac{e\delta}{\rho}$, $aK < g + \frac{f\delta}{\rho}$, then $P_2\left(K, 0, 0, \frac{\delta}{\rho}\right)$ is locally asymptotically stable.

Proof: The Jacobian matrix $J(P_2)$ of the model (3) around P_1 is as follows:

$$J(P_2) = \begin{bmatrix} -\left(r + c + \frac{b\delta}{\rho}\right) & -(\beta K + r) & -aK & -bK \\ 0 & \beta K - \left(c + \frac{e\delta}{\rho}\right) & 0 & 0 \\ 0 & 0 & aK - \left(g + \frac{f\delta}{\rho}\right) & 0 \\ 0 & 0 & 0 & -\rho \end{bmatrix},$$

The eigenvalues of $J(P_2)$ consists of $-\left(r + c + \frac{b\delta}{\rho}\right)$, $\beta K - \left(c + \frac{e\delta}{\rho}\right)$, $aK - \left(g + \frac{f\delta}{\rho}\right)$ and $-\rho$. In order for P_2 to be stable, it is necessary that both $\beta K - \left(c + \frac{e\delta}{\rho}\right)$ and $aK - \left(g + \frac{f\delta}{\rho}\right)$ are less than zero. This can be simplified to the conditions $\beta K < c + \frac{e\delta}{\rho}$ and $aK < g + \frac{f\delta}{\rho}$. Consequently, we conclude that P_2 exhibits local stability when $\beta K < c + \frac{e\delta}{\rho}$ and $aK < g + \frac{f\delta}{\rho}$.

Theorem 6.5: The disease-free equilibrium point $P_3\left(\frac{f\delta+g\rho}{a\rho}, 0, \frac{Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)}{K\rho a^2}, \frac{\delta}{\rho}\right)$ is locally asymptotically stable whenever $R_0 < 1$ and is unstable when $R_0 > 1$.

Proof: The disease-free equilibrium point P_3 demonstrates local asymptotic stability when all the real parts of the eigenvalues (φ) are negative. Here, the Jacobian matrix corresponding to the system (6.6) centered around P_3 is as follows:

$$J(P_3) = \begin{bmatrix} \frac{-r(f\delta+g\rho)}{K\rho a} & \frac{-(K\beta+r)(f\delta+g\rho)}{K\rho a} & \frac{-(f\delta+g\rho)}{\rho} & \frac{-b(f\delta+g\rho)}{a\rho} \\ 0 & J_{22}(P_3) & 0 & 0 \\ \frac{Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)}{K\rho a} & J_{32}(P_3) & 0 & \frac{f[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2} \\ 0 & 0 & 0 & -\rho \end{bmatrix}.$$

Here,

$$J_{22}(P_3) = \frac{Ka[\beta(f\delta+g\rho)-a(e\delta+c\rho)]-d[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2},$$

$$J_{32}(P_3) = \frac{d[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2}.$$

The characteristic equation of the above Jacobian matrix $J(P_2)$ is given by:

$$(-\rho-\chi)\left(\frac{Ka[\beta(f\delta+g\rho)-a(e\delta+c\rho)]-d[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2}-\chi\right)(\chi^2+c_1\chi+c_2)=0.$$

We can see the eigenvalues of $J(P_3)$ are:

$$\chi_1 = -\rho < 0,\ \chi_2 = \frac{Ka[\beta(f\delta+g\rho)-a(e\delta+c\rho)]-d[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2}.$$

To achieve stability in the system, it is necessary that the value of χ_2 remains less than zero,

i.e., $\chi_2 < 0$,

$$\Rightarrow \frac{Ka[\beta(f\delta+g\rho)-a(e\delta+c\rho)]-d[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2} < 0,$$

$$\Rightarrow \frac{K\beta a(f\delta+g\rho)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)} < 1,$$

$$\Rightarrow \mathrm{R}_0 < 1.$$

Now, the other eigenvalues can be determined by solving the quadratic equation of degree 2 provided below:

$$c_1\chi^2 + c_2\chi + c_3 = 0 \tag{6.14}$$

with coefficients $c_1 = 1$, $c_2 = \frac{r(f\delta+g\rho)}{K\rho a}$, $c_3 = \frac{(f\delta+g\rho)\left(Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)\right)}{K\rho^2 a}$.

Therefore, $\chi_3 = \frac{-r(f\delta+g\rho)-\sqrt{\left(r(f\delta+g\rho)\right)^2+4Ka\left[(f\delta+g\rho)\left(Ka(c\rho+b\delta-r\rho)+r(f\delta+g\rho)\right)\right]}}{2K\rho a}$,

$$\chi_4 = \frac{-r(f\delta+g\rho)+\sqrt{\left(r(f\delta+g\rho)\right)^2+4Ka\left[(f\delta+g\rho)\left(Ka(c\rho+b\delta-r\rho)+r(f\delta+g\rho)\right)\right]}}{2K\rho a}.$$

The eigenvalues χ_3 and χ_4 have negative real parts. Hence, the disease-free equilibrium point P_3 is locally asymptotically stable whenever $\mathrm{R}_0 < 1$ and is unstable when $\mathrm{R}_0 > 1$.

Theorem 6.6: The coexistence equilibrium point $P_4(X_1^*, X_2^*, Y^*, Z^*)$ is locally asymptotically stable if [164]:

$$M_2 > 0, M_3 > 0, M_4 > 0, M_1M_2 - M_3 > 0 \text{ and } (M_1M_2 - M_3)M_3 - M_1^2M_4 > 0. \quad (6.15)$$

With

$$M_1 = -(T_{11} + T_{22} + T_{33}) + \rho,$$

$$M_2 = T_{11}T_{22} + T_{11}T_{33} + T_{22}T_{33} - \rho(T_{11} + T_{22} + T_{33}) + (a^2X_1^* + d^2X_2^*)Y^* + \frac{\beta(K\beta+r)X_1^*X_2^*}{K},$$

$$M_3 = \rho(T_{11}T_{22} + T_{11}T_{33} + T_{22}T_{33}) + d^2(\rho - T_{11})X_2^*Y^* + a^2(\rho - T_{22})X_1^*Y^* - T_{11}T_{22}T_{33} + \frac{[K\beta daY^* + (K\beta+r)(\beta\rho - \beta T_{33} - adY^*)]X_1^*X_2^*}{K},$$

$$M_4 = a\rho[\beta dX_2^* - aT_{22}]X_1^*Y^* - \rho T_{11}(T_{22}T_{33} + d^2X_2^*Y^*) - \frac{\rho(K\beta+r)(\beta T_{33} + adY^*)X_1^*X_2^*}{K}.$$

Here,

$$X_1^* = \frac{Ka(e\delta+c\rho) + Kdr\rho - Kd(b\delta+c\rho) - (K+r)(f\delta+g\rho)}{K\beta\rho a + rd\rho - a\rho(K+r)},$$

$$X_2^* = \frac{(f\delta+g\rho)(K\beta\rho a + rd\rho - a\rho(K+r)) - a\rho\big(Ka(e\delta+c\rho) + Kdr\rho - Kd(b\delta+c\rho) - (K+r)(f\delta+g\rho)\big)}{d\rho(K\beta\rho a + rd\rho - a\rho(K+r))},$$

$$Y^* = \frac{\beta\rho\big(Ka(e\delta+c\rho) + Kdr\rho - Kd(b\delta+c\rho) - (K+r)(f\delta+g\rho)\big) - (c\rho+e\delta)(K\beta\rho a + rd\rho - a\rho(K+r))}{d\rho(K\beta\rho a + rd\rho - a\rho(K+r))},$$

$$Z^* = \frac{\delta}{\rho}.$$

Proof: The computation of the Jacobian matrix $J(P_4)$ at the coexistence equilibrium point P_4 is performed in the following manner:

$$J(P_4) = J(X_1^*, X_2^*, Y^*, Z^*) = \begin{bmatrix} T_{11} & \frac{-(\beta K+r)X_1^*}{K} & -aX_1^* & -bX_1^* \\ \beta X_2^* & T_{22} & -dX_2^* & -eX_2^* \\ aY^* & dY^* & T_{33} & -fY^* \\ 0 & 0 & 0 & -\rho \end{bmatrix}. \quad (6.15)$$

Where:

$$T_{11} = r\left(1 - \frac{2X_1^* + X_2^*}{K}\right) - \beta X_2^* - aY^* - bZ^* - c,$$

$$T_{22} = \beta X_1^* - dY^* - eZ^* - c,$$

$$T_{33} = aX_1^* + dX_2^* - fZ^* - g.$$

The characteristic equation in θ for the Jacobian matrix $J(P_4)$ is given by:

$$|J(P_4) - \theta I| = \begin{vmatrix} T_{11} - \theta & \frac{-(\beta K + r)X_1^*}{K} & -aX_1^* & -bX_1^* \\ \beta X_2^* & T_{22} - \theta & -dX_2^* & -eX_2^* \\ aY^* & dY^* & T_{33} - \theta & -fY^* \\ 0 & 0 & 0 & -\rho - \theta \end{vmatrix} = 0. \quad (6.16)$$

Which gives,

$$\theta^4 + M_1\theta^3 + M_2\theta^2 + M_3\theta + M_4 = 0. \quad (6.17)$$

Recognising that $M_1 > 0$, then by applying the Routh–Hurwitz criterion in conjunction with the conditions outlined in (6.15), we conclude that the coexistence equilibrium P_4 of system (6.6) exhibits local asymptotic stability if $M_2 > 0$, $M_3 > 0$, $M_4 > 0$, $M_1M_2 - M_3 > 0$ and $(M_1M_2 - M_3)M_3 - M_1^2M_4 > 0$ [164]. Conversely, if any of these conditions are not satisfied, the coexistence equilibrium P_4 is determined to be unstable.

6.5 Permanence of the System

From a biological point of view, the permanence of a system is a crucial factor in ensuring the long-term survival of all populations within it, thereby preventing any of them from facing extinction. To achieve this uniform persistence, we have implemented the methodology described by Das et al. [38].

Definition 6.1: The system (6.6) is said to be permanent if $\exists\, N \geq n > 0$, such that for any solution of $(X_1(t), X_2(t), Y(t), Z(t))$ of system (6.6), $(X_1(0), X_2(0), Y(0), Z(0)) > 0$,

$$n \leq \lim_{t\to\infty} inf\big(X_1(t)\big) \leq \lim_{t\to\infty} sup\big(X_1(t)\big) \leq N,$$

$$n \leq \lim_{t\to\infty} inf\big(X_2(t)\big) \leq \lim_{t\to\infty} sup\big(X_2(t)\big) \leq N,$$

$$n \leq \lim_{t\to\infty} inf\big(Y(t)\big) \leq \lim_{t\to\infty} sup\big(Y(t)\big) \leq N,$$

$$n \leq \lim_{t\to\infty} inf\big(Z(t)\big) \leq \lim_{t\to\infty} sup\big(Z(t)\big) \leq N.$$

Definition 6.2: A population is said to be uniformly persistent if $\exists\ \delta > 0$, which is independent of $X_1(0)$ where $X_1(0) > 0$, such that:

$$\lim_{t\to\infty} inf\big(X_1(t)\big) > \delta.$$

Theorem 6.7: The system (6.6) is uniformly persistent if the following condition is satisfied:

$$\beta\widehat{X_1} - d\hat{Y} - \left(\frac{e\delta+c\rho}{\rho}\right) > 0 \text{ holds.} \tag{6.18}$$

Proof: We will prove this theorem by the method of Lyapunov average function. Let the average Lyapunov function for the system (6.6) be $\sigma(V) = X_1^{p_1}X_2^{q_1}Y^{r_1}Z^{s_1}$, where p_1, q_1, r_1, s_1 are positive constants. Clearly, $\sigma(V)$ is non-negative function defined in D of R_+^4,

where $R_+^4 = \{(X_1, X_2, Y, Z): X_1 > 0, X_2 > 0, Y > 0, Z > 0\}$.

Then, we have:

$$\Psi(V) = \frac{\dot{\sigma}(V)}{\sigma(V)} = p_1\frac{\dot{X_1}}{X_1} + q_1\frac{\dot{X_2}}{X_2} + r_1\frac{\dot{Y}}{Y} + s_1\frac{\dot{Z}}{Z},$$

$$\Psi(V) = p_1\left(\mathrm{r}\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_2 - aY - bZ - c\right) + q_1(\beta X_1 - dY - eZ - c) + r_1(aX_1 +$$

$$dX_2 - fZ - g) + s_1\left(-\rho + \frac{\delta}{Z}\right). \tag{6.19}$$

To establish the uniform persistence of the system, it is sufficient to show that $\Psi(V) > 0$ for a suitable selection of positive values for $p_1, q_1, r_1, s_1 > 0$:

$$\Psi(P_1) = p_1\left[r - \left(\frac{b\delta+c\rho}{\rho}\right)\right] - q_1\left(\frac{e\delta+c\rho}{\rho}\right) - r_1\left(\frac{f\delta+g\rho}{\rho}\right) > 0, \tag{6.20}$$

$$\Psi(P_2) = -p_1\left(\frac{b\delta+c\rho}{\rho}\right) + q_1\left[\beta K - \left(\frac{e\delta+c\rho}{\rho}\right)\right] + r_1\left[aK - \left(\frac{f\delta+g\rho}{\rho}\right)\right] > 0, \tag{6.21}$$

$$\Psi(P_3) = q_1\left[\beta\widehat{X_1} - d\hat{Y} - \left(\frac{e\delta+c\rho}{\rho}\right)\right] > 0. \tag{6.22}$$

We observed that, when increasing the value of p_1, while $(b\delta + c\rho) < \rho$ and $p_1[r\rho - (b\delta + c\rho)] > q_1(e\delta + c\rho) + r_1(f\delta + g\rho)$, then $\Psi(P_1)$ can be made positive. Thus, the inequality (6.20) holds. If $K(\beta q_1 + ar_1)\rho > p_1(b\delta + c\rho) + q_1(e\delta + c\rho) + r_1(f\delta + g\rho)$, then $\Psi(P_2)$ is positive. Thus, the inequality (6.21) holds. If the inequality in Equation (6.20) holds, then (6.22) is satisfied.

6.6 Sensitivity Analysis

We aim to understand the various factors contributing to disease transmission and prevalence. This knowledge can help us reduce plant infections and decrease herbivore populations' mortality caused by diseases. Sensitivity analysis provides insights into the significance of each parameter in disease transmission. This information is not only vital for designing experiments but also for incorporating data and simplifying complex nonlinear models [152]. Sensitivity analysis is a common tool for assessing how model predictions respond to variations in parameter values, as data collection and assumed parameter values often involve some degree of uncertainty. It helps identify parameters that strongly influence the basic reproduction number and should be the focus of intervention strategies. When a parameter undergoes a change, sensitivity indices enable us to quantify the relative impact on a variable [74]. By examining these indices, we can identify which parameters play a more critical role in disease transmission and prevalence.

Definition 6.3: The normalized forward sensitivity index of the basic reproduction number R_0, which is differentiable with respect specific parameter h, is precisely defined as follows:

$$\gamma_h^{R_0} = \frac{h}{R_0}\frac{\partial R_0}{\partial h}.$$

6.6.1 Sensitivity Analysis Results:

In this study, we used MATLAB to perform sensitivity analyses in order to investigate the dynamics of infectious disease transmission within the plant and herbivore populations. We implemented our eco-epidemiological model using computational approaches, specifically MATLAB's robust numerical solvers and visualisation tools, to study how parameter variations affect model outcomes. In this MATLAB implementation, we evaluated how changes in important factors, such as the infection rate (β), affected the basic reproduction number (R_0) and final state values of population variables. This computational technique enabled us to efficiently explore a wide range of situations while gaining vital insights into the underlying mechanisms that drive disease development.

Table 6.1: Parameters and its Values.

Parameters	Values
r	0.1
K	100
β	$0.01\ to\ 0.05\ (50\ values)$
a	0.02
b	0.01
c	0.1
d	0.01
e	0.02
f	0.01
g	0.01
ρ	0.05
δ	0.1

The simulation explores the dynamics of a population system governed by a set of parameters, including growth rates, carrying capacity, infection rates, and various birth and death rates. By varying the infection rate (β), the model examines how different levels of contagiousness influence the final state populations of X_1, X_2, Y, and Z. The results illustrate complex interdependencies among these populations, revealing how changes in infection rates affect the equilibrium states. Key parameters such as r, K and δ influence the system's stability and responses to external factors, highlighting the intricate ecological relationships embedded within the population dynamics. These findings provide insights into the system's resilience to infections and the effectiveness of control measures in managing population dynamics under varying environmental conditions.

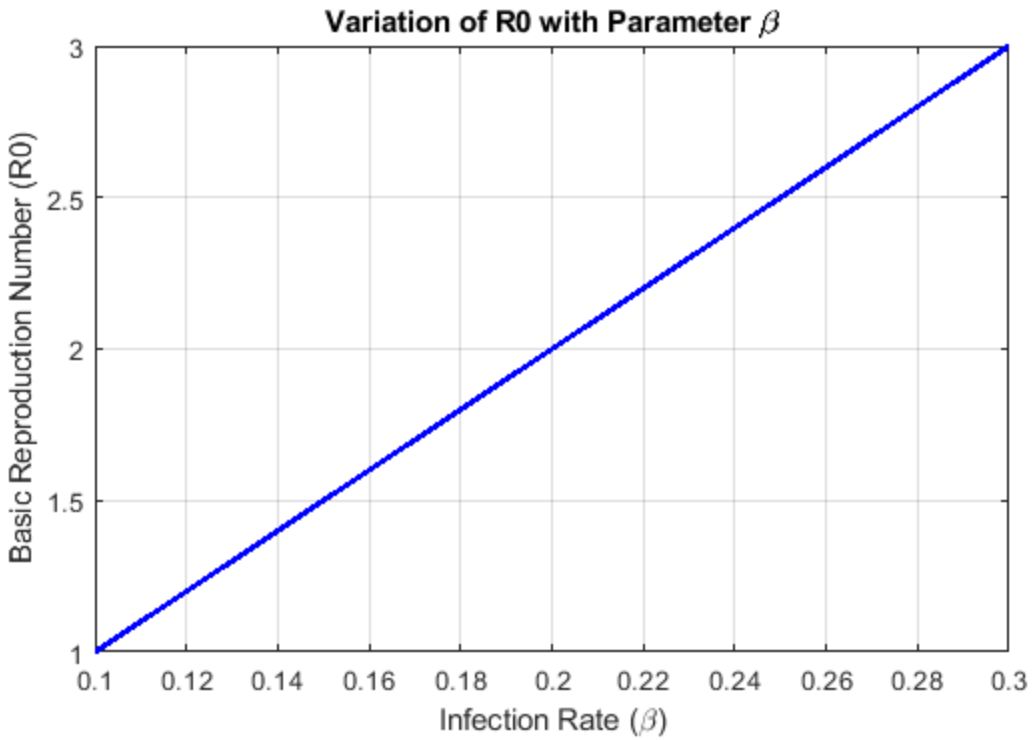

Figure 6.1: Variations in the Basic Reproduction Number (R_0) with Infection Rate (β)

Figure 6.1 depicting the variation of R_0 with parameter β offers critical insights into the dynamics of disease transmission. As the infection rate (β) increases, indicating a higher likelihood of populations becoming infected upon contact with infectious population, the basic reproduction number (R_0) also rises. This escalation signifies an elevated potential for disease spread throughout the population, with each infected ones, on average, infecting more susceptible populations. Conversely, a decrease in β leads to a reduction in R_0, suggesting a lower risk of disease dissemination. Understanding this relationship is important for devising effective control strategies, as interventions targeting the control of R_0 by moderating β can substantially mitigate disease transmission. Peaks and troughs in the graph denote points where R_0 reaches maximum and minimum values, respectively, representing distinct levels of disease transmissibility. This detailed analysis of R_0 variations elucidate the intricate interplay between the infection rate and the potential for disease propagation, guiding the formulation of targeted interventions to curb infectious disease outbreaks.

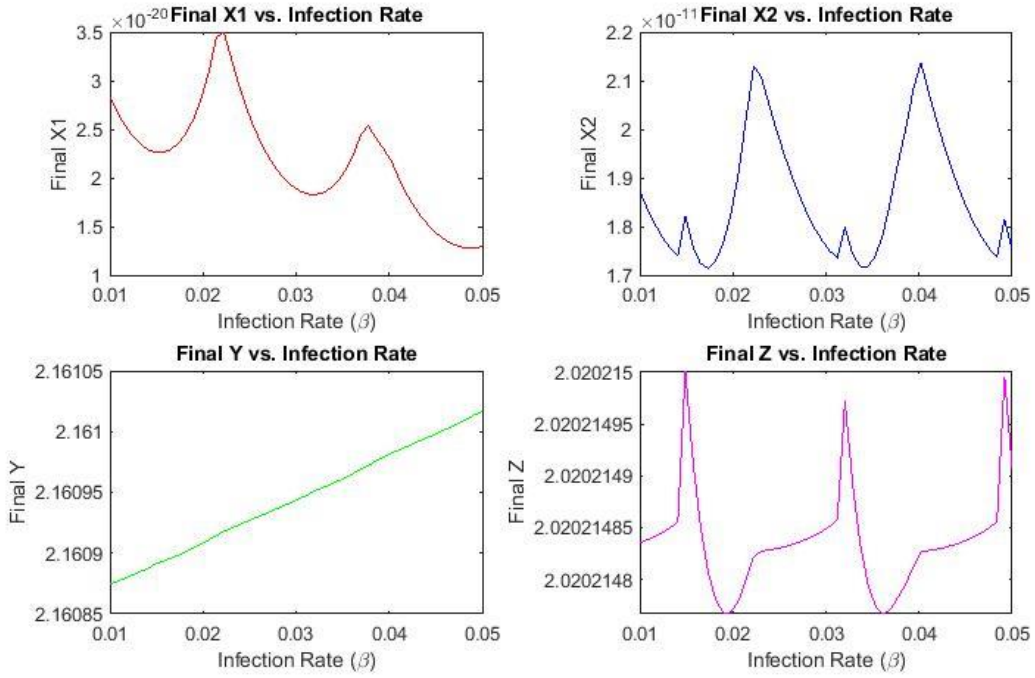

Figure 6.2: Final State Population Dynamics against Infection Rate (β)

Figure 6.2 illustrates the final state population dynamics of variables X_1, X_2, Y, and Z against the infection rate (β) in a system governed by the specified parameter values. As the infection rate (β) increases, there is a discernible trend in the final populations of the system's constituents. Notably, X_1 and X_2 populations exhibit a gradual decline, indicating a decrease in the population of the two interacting plant species over time. Conversely, Y population and pesticides Z show an upward trajectory, suggesting an increase in the population of the herbivores and pesticides, respectively. This dynamic interplay among the populations X_1,X_2,Y and Control measure Z reflects the intricate ecological relationships encoded within the system's dynamics, as influenced by the parameters $r = 0.1$, $K = 100$, $a = 0.02$, $b = 0.01$, $c = 0.1$, $d = 0.01$, $e = 0.02$, $f = 0.01$, $g = 0.01$, $\rho = 0.05$ and $\delta = 0.1$. These parameter values shape the behavior of the system, governing the growth, interaction, and equilibrium of the populations over time.

6.7 Analysis of Optimal Control

In this particular section of our study, we focus on the utilisation of Pontryagin's Maximum Principle, a mathematical framework used for solving optimal control problems [200]. The objective is to identify and understand the crucial conditions necessary for optimally controlling the impact of plant inoculation. The fundamental model, denoted as system (6.6), undergoes expansion to incorporate a dynamic control parameter known as $u(t)$. This parameter represents the control measure implemented during the process of plant inoculation, which aims to prevent the occurrence of plant diseases.

The introduced control measure, $u(t)$, plays a pivotal role in regulating the transmission rate from infected plants to susceptible ones. Moreover, it exerts influence over the populations of herbivores in the ecological system under consideration. The resulting model, shaped by the incorporation of this time-dependent control variable, captures the intricate dynamics involved in preventing plant diseases. Through the exploration and application of Pontryagin's Maximum Principle, our goal is to determine the optimal strategies and conditions for effectively managing the impact of plant inoculation within this complex ecological context. Consider $u(t) \in U$ to be a control variable on plant inoculation to reduce plant disease. Then, the resulting model is outlined below:

$$\begin{aligned}
\frac{dX_1}{dt} &= \mathrm{r}X_1\left(1-\frac{X_1+X_2}{K}\right)-(1-u)\beta X_1X_2-aX_1Y-bX_1Z-cX_1,\\
\frac{dX_2}{dt} &= (1-u)\beta X_1X_2-dX_2Y-eX_2Z-cX_2,\\
\frac{dY}{dt} &= aX_1Y+dX_2Y-fYZ-gY,\\
\frac{dZ}{dt} &= -\rho Z+\delta.
\end{aligned} \tag{6.23}$$

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

$$J^*(u)=\min_u\int_0^{t_f}\left(AX_1(t)+BX_2(t)+\frac{1}{2}B_1u^2(t)\right)dt, \tag{6.24}$$

In the given scenario, t_f represents the final time, A and B are positive constants and it denote the costs associated with susceptible and infected plants, respectively, and B_1 serves as relative cost weights for the control measure. The objective function $J^*(u)$ is formulated to minimize the number of infected plants while also minimizing the cost of the control $u(t)$. Therefore, we are in pursuit of an optimal control $u^*(t)$ that achieves these dual objectives. Consequently, the optimal controls $u^*(t)$ exist in such a way that:

$$J^*(u^*) = min\{J^*(u)\} \tag{6.25}$$

Here, we shall first show the existence of an optimal control (6.25) for the system (6.23). This is supported by the following theorem.

Theorem 6.8: There exists an optimal control $u^*(t)$ such that $J^*(u^*(\mathrm{t})) = min\{J^*(u(t))\}$ subject to the control system (6.6) with initial conditions.

Proof: The integrand of the objective functional $AX_1(t) + BX_2(t) + \frac{1}{2}B_1u^2(t)$ is a convex function of the control variable $u(t)$. Both the control and state variables are non-negative, and the system (6.6) satisfies Lipchitz conditions with respect to the state variables due to the boundedness of state solutions. As a result, we can conclude that there exists an optimal control [207].

To achieve an optimal solution, the initial step involves determining the Lagrangian and Hamiltonian for the given problem outlined in equations (6.23) to (6.24). The Lagrangian associated with this optimal control problem is given by:

$$L(X_1, X_2, u) = AX_1(t) + BX_2(t) + \frac{1}{2}B_1u^2(t). \tag{6.26}$$

We aim to obtain the minimum values of the Lagrangian. To achieve this, we define the Hamiltonian for the control problem as follows:

$$H\left(X_1, X_2, u, \lambda_{X_1}, \lambda_{X_2}, \lambda_Y, \lambda_Z, t\right) = L(X_1, X_2, u) + \lambda_{X_1}(t)\frac{dX_1(t)}{dt} + \lambda_{X_2}(t)\frac{dX_2(t)}{dt} + \lambda_Y(t)\frac{dY(t)}{dt} + \lambda_Z(t)\frac{dZ(t)}{dt}. \tag{6.27}$$

Where λ_{X_1}, λ_{X_2}, λ_Y and λ_Z are adjoint variables or co-state variables to be determined. The formulated model must satisfy the necessary conditions established by Pontryagin's Maximum [127]. The application of Pontryagin's Maximum Principle typically transforms the system of equations (6.23) and (6.24) into a point-wise minimization problem of the Hamiltonian H, with respect to the control variable $u(t)$.

Theorem 6.9: Let $X_1^*(t), X_2^*(t), Y^*(t), Z^*(t)$ represent the optimal state solutions corresponding to the optimal control variable $u^*(t)$ for the optimal control problem (6.23) – (6.24). This implies the existence of adjoint variables λ_{X_1}, λ_{X_2}, λ_Y and λ_Z satisfying:

$$\frac{d\lambda_{X_1}}{dt} = \lambda_{X_1}\left[r\left(\frac{2X_1+X_2}{K} - 1\right) + bZ + c\right] + \beta(1-u)\left(\lambda_{X_1} - \lambda_{X_2}\right)X_2 + a\left(\lambda_{X_1} - \lambda_Y\right)Y - A,$$

$$\frac{d\lambda_{X_1}}{dt} = \frac{rX_1}{K}\lambda_{X_1} + \beta(1-u)\left(\lambda_{X_1} - \lambda_{X_2}\right)X_1 + d\left(\lambda_{X_2} - \lambda_Y\right)Y + \lambda_{X_2}(eZ + c) - B,$$

$$\frac{d\lambda_Y}{dt} = a\left(\lambda_{X_1} - \lambda_Y\right)X_1 + d\left(\lambda_{X_2} - \lambda_Y\right)X_2 + \lambda_Y(fZ + g),$$

$$\frac{d\lambda_Z}{dt} = bX_1\lambda_{X_1} + eX_2\lambda_{X_2} + fY\lambda_Y + \rho\lambda_Z,$$

with transversality conditions $\lambda_{X_1}(t_f),\ \lambda_{X_2}(t_f),\ \lambda_Y(t_f),\ \lambda_Z(t_f) = 0$.

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

$$u^*(t) = max\left(min\left(\frac{\lambda_{X_2}-\lambda_{X_1}}{B_1}, u_{max}\right), 0\right).$$

Proof: To derive both the transversality conditions and the adjoint equations, we utilise Hamiltonian (6.27). The adjoint equations are derived through the application of Pontryagin's Maximum Principle.

$$\frac{d\lambda_{X_1}}{dt} = -\frac{\partial H}{\partial X_1}, \frac{d\lambda_{X_2}}{dt} = -\frac{\partial H}{\partial X_2}, \frac{d\lambda_Y}{dt} = -\frac{\partial H}{\partial Y} \text{ and } \frac{d\lambda_Z}{dt} = -\frac{\partial H}{\partial Z}$$

with $\lambda_{X_1}(t_f),\ \lambda_{X_2}(t_f),\ \lambda_Y(t_f),\ \lambda_Z(t_f) = 0$.

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 $B_1 u(t) + \lambda_{X_1} \beta X_1 X_2 - \lambda_{X_2} \beta X_1 X_2 = 0$.

This implies that $u(t) = \frac{\beta(\lambda_{X_2} - \lambda_{X_1}) X_1 X_2}{B_1} (= u^*(t))$, say.

Using the property of control space, we obtain:

$u^*(t) = 0$ if $\frac{\beta(\lambda_{X_2} - \lambda_{X_1}) X_1 X_2}{B_1} \leq 0$,

$u^*(t) = \frac{\beta(\lambda_{X_2} - \lambda_{X_1}) X_1 X_2}{B_1}$ if $0 < \frac{\beta(\lambda_{X_2} - \lambda_{X_1}) X_1 X_2}{B_1} < u_{max}$,

$u^*(t) = u_{max}$ if $\frac{\beta(\lambda_{X_2} - \lambda_{X_1}) X_1 X_2}{B_1} \geq u_{max}$.

So, the optimal control is characterized as $u^*(t) = max\left(min\left(\frac{\lambda_{X_2} - \lambda_{X_1}}{B_1}, u_{max}\right), 0\right)$. This represents the characterization of the optimal control.

6.7.1 Optimal Control Analysis Results:

Optimal control analysis is the use of mathematical frameworks such as Pontryagin's Maximum Principle in the derivation of dynamic system management techniques. MATLAB plays a crucial role in these tests, enabling the integration of theoretical concepts into numerical simulations. Plots and graphs are used to illustrate the numerical solutions of differential equations describing system dynamics and the derivation of optimal control techniques. This method allows for the investigation of system behaviour, the assessment of various control inputs, and the identification of the most effective solutions to complex control problems. In the end, MATLAB implementation helps to fully comprehend optimal control systems and supports informed decisions on control techniques and system optimization.

Table 6.2: Parameters and its Values

Parameters	Values
r	0.1
K	100
β	0.01
a	0.02
b	0.01
c	0.1
d	0.01
e	0.02
f	0.01
g	0.01
ρ	0.05
δ	0.1
A	1
B	1
B_1	1

The parameter values provided offer a comprehensive insight into the dynamics of the plant epidemic system (6.6) under consideration. With a growth rate (r) of 0.1 and a carrying capacity (K) of 100, the system's behaviour is being influenced by the interplay of birth and death rates of populations X_1 and X_2. Additionally, the rate at which pesticides is being used (ρ) and the constant amount of pesticides used (δ) of pesticides Z are crucial factors at 0.05 and 0.1, respectively. These parameters encapsulate the intricate relationships within the system, shaping its stability and persistence over time. Initial conditions ($X_1(0) = 50, X_2(0) = 50, Y(0) = 10, Z(0) = 5$) further describe the starting state of each population, offering a foundation for understanding its evolution.

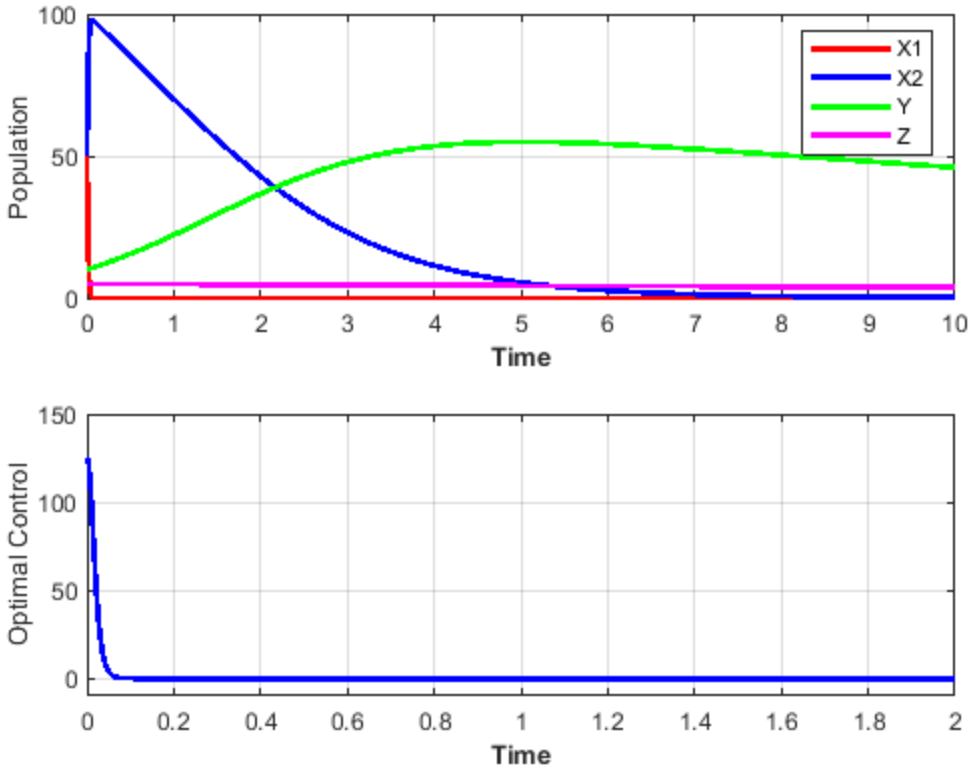

Figure 6.3: Dynamics of Ecological Variables and Optimal Control Profile

Figure 6.3 provides a comprehensive visualisation of the system's behavior and the corresponding optimal control strategy. The upper subplot depicts the trajectories of ecological variables (X_1, X_2, Y, Z) over time, showcasing their dynamic evolution based on the given differential equations and initial conditions. The lower subplot illustrates the optimal control profile, representing the control measure's magnitude applied to mitigate plant diseases. The trajectory of the optimal control reveals how the control strategy varies over time to minimise the number of infected plants while minimising the control cost, as dictated by the objective function. This visualisation allows for a holistic understanding of the system's dynamics and the effectiveness of the optimal control strategy in managing plant inoculation within the ecological context.

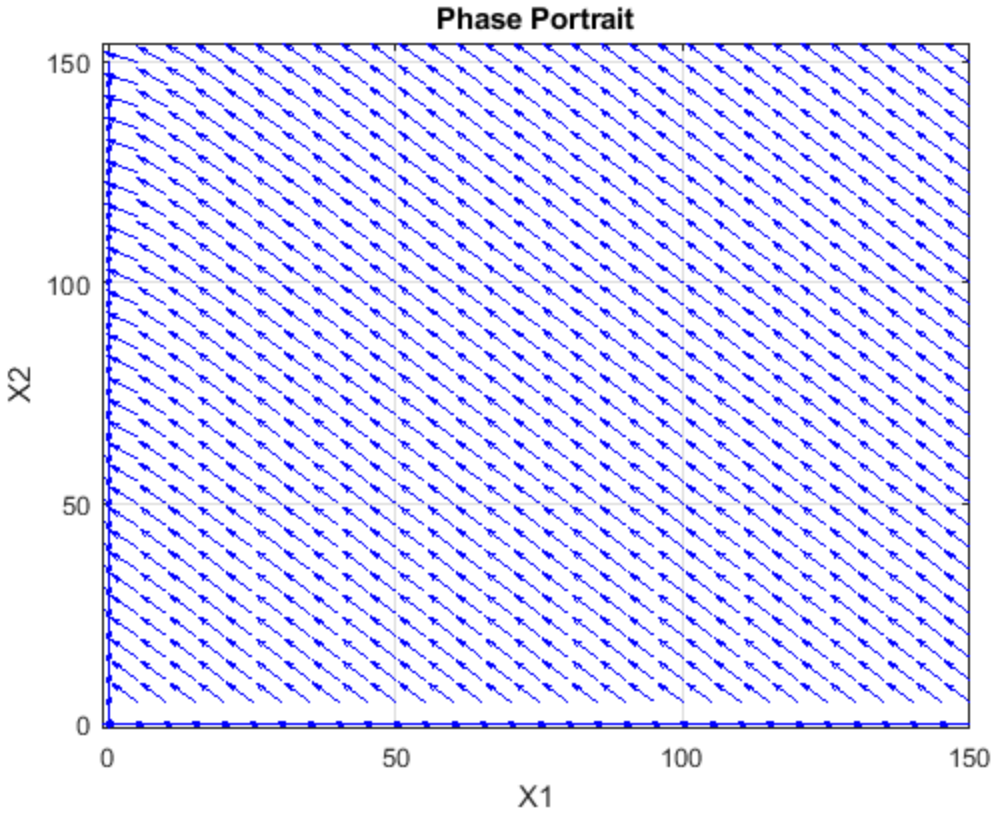

Figure 6.4: Phase Portrait: State Space Dynamics Visualisation

Figure 6.4 illustrates the system's dynamical behavior in its state space, depicting the trajectories of the system's state variables over time. Each curve represents a unique initial condition, demonstrating how the system evolves over time. Important insights into the stability, equilibrium points, and qualitative behavior of the system can be gained by analysing the shape and direction of these trajectories. In the phase portrait, stable equilibrium points correspond to attracting regions, whereas unstable equilibrium points are associated with repelling regions. Additionally, closed trajectories can identify limit cycles or other types of periodic behaviour. Overall, the phase portrait provides a comprehensive visual representation of the system's dynamics, aiding in the understanding of its behaviour and stability properties.

6.8. Discussion

Rigorous mathematical frameworks and computational approaches were employed in this study to analyze the dynamics of infectious disease transmission within ecological systems and identify optimal control measures for managing disease spread. The study aimed to comprehend the

intricate relationships among key factors influencing disease spread and population size changes. Additionally, effective strategies were developed to mitigate disease incidence.

6.8.1 Mathematical Analysis of Disease Dynamics:

The analysis began with the formulation of a comprehensive mathematical model (6.6) describing the dynamics of susceptible plants (X_1), infected plants (X_2), and the herbivores populations (Y), as well as the Pesticides (Z) as control measure. Leveraging techniques from differential equations and dynamical systems theory, we examined the stability and equilibrium points of the model, elucidating the conditions under which disease outbreaks occur and identifying critical thresholds for disease control. Through sensitivity analysis, we quantified the relative impact of each model parameter on disease transmission and prevalence. By calculating normalized forward sensitivity indices, we assessed the sensitivity of the basic reproduction number (R_0) to variations in parameters such as the infection rate (β). This analysis provided valuable insights into the factors driving disease dynamics and highlighted parameters that are key targets for intervention strategies.

6.8.2 Optimal Control Framework:

To devise optimal control strategies for managing disease spread, Pontryagin's Maximum Principle, a robust mathematical framework for solving optimal control problems, was employed. Our base model was extended to include a dynamic control parameter $u(t)$ representing the implementation of control measures, such as plant inoculation, aimed at reducing disease transmission. The optimal control problem aimed to minimise the number of infected plants while minimising the cost associated with the control measure $u(t)$. Through rigorous mathematical analysis, including the derivation of Hamiltonians and adjoint equations, we determined optimal control profiles that achieve these dual objectives. The characterisation of optimal controls ($u^*(t)$) was based on the interplay between state variables, adjoint variables, and maximum allowable control magnitudes.

6.8.3 Computational Implementation and Analysis:

Utilising computational techniques, particularly leveraging MATLAB's numerical solvers and visualization tools, the implemented mathematical models were used to explore the dynamics of disease and investigate optimal strategies for disease control. Through numerical solutions of differential equations and simulations of various control scenarios, valuable insights were gained into how different measures can effectively mitigate the spread of disease.

6.9 Conclusion

The present study utilised advanced mathematical modelling, computer analysis, and optimisation tools to investigate the dynamics of infectious diseases in ecological systems. By conducting thorough analysis of stability characteristics, equilibrium points, and sensitivity to parameter modifications, crucial thresholds have been discovered that determine the persistence and elimination of diseases. Additionally, we have identified the main factors that drive transmission and prevalence. By utilising Pontryagin's Maximum Principle, optimal control methods have been developed aiming to minimise the burden of disease while optimising control expenditures. These strategies provide practical insights into the effectiveness of various intervention measures. The integration of multiple disciplines has enhanced our comprehension of the patterns and changes in disease occurrence, providing essential direction for public health policy based on solid data and focused intervention tactics. These findings are extremely significant resources in the ongoing fight against infectious diseases, providing policymakers and public health professionals with the necessary tools and knowledge to protect human health and ecological integrity. This study used rigorous mathematical frameworks and computational approaches to analyse the dynamics of infectious disease transmission within ecological systems and to develop optimal control mechanisms for managing the spread of diseases. Our work aimed to understand the complex relationships between important factors that influence the spread of disease and changes in population size. Additionally, effective methods aimed at reducing the prevalence of the disease were developed.