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OPTIMISING PLANT EPIDEMIC CONTROL: A MATHEMATICAL MODEL INTEGRATING SUSCEPTIBLE AND INFECTIVES PLANTS, AND HERBIVORES WITH PESTICIDE INTERVENTION

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Abstract: This work presents and analyses a deterministic mathematical model incorporating herbivores and plant populations, with pesticide application as a control measure. There are three distinct populations under consideration: susceptible and infected plants, as well as herbivorous population. Our model's goal is to analyse the interactions and dynamics of these populations. By analysing their behaviour, we can gain insight into the ecological processes that regulate their growth and survival. The model administers pesticides as a control measure to both susceptible and infected plants. Furthermore, given that herbivores have the potential to consume pesticide-sprayed plants, we can observe an interaction between herbivores and pesticides. The primary goal of pesticide application is to mitigate disease transmission among plant populations. To verify the biological validity and precisely defined characteristics of the model, we assess the system's permanence and examine its positivity, boundedness, uniqueness, and existence of solutions. The determination of the infection's basic reproduction number (R_0) and observation of the disease-free equilibrium state reveal that it is locally asymptotically stable when R_0 is less than unity, but unstable otherwise. In addition, we conduct sensitivity analysis on the basic reproduction number and use Pontryagin's Minimum Principle to determine a necessary condition for the existence of optimal controls. Finally, we perform numerical simulations using the MATLAB software to compare the analytical results. In summation, the findings yielded by this analysis are innovative and substantial, thereby constituting a meaningful addition to the body of knowledge in theoretical

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1. INTRODUCTION

The study of infectious diseases is an essential aspect of biomathematics because it enables scientists to investigate the effects of various components in ecosystems. This area of mathematics has a long history of advancement. Mathematical ecology and mathematical epidemiology are two distinct subfields of applied mathematics and biology. Eco-epidemiological modelling is an important part of biomathematics, resulting from many experimental studies that examine ecological and epidemiological models simultaneously. Over the years, infectious diseases have become a major threat to breeders, thus harming the environment as well as animal and human health [7]. Recently, mathematicians and ecologists have joined forces to study epidemiology and ecology from a mathematical perspective [9]. Therefore, many mathematical model equations have emerged that are now important for the study of plant diseases and the development of control strategies, including the use of pesticides and natural enemies [8]. In mathematical modelling, we first introduce a real-world problem as a model, then solve it mathematically. We then translate the outcome into real-world language [17]. Ecology, epidemiology, cancer, diabetes, HIV, and demography are just a few of the many biological disciplines that utilise mathematical models in the life sciences [10, 11, 22].

Plants and herbivores interact in dynamic and complex natural environments that are the result of millions of years of evolution. To protect themselves from herbivores, plants use various defence strategies during this attack, including physical and chemical defence, aggression, and non-destructive defence [15]. These interactions are important in creating ecosystems, influencing the evolution of animals and plants, and maintaining the balance of the natural world. There are many ways that plants and herbivores interact in ecosystem dynamics. Herbivores, such as birds, mammals, and insects, obtain most of their nutrients from plant tissues. Herbivores participate in this direct interaction by eating plant material, and this can have many benefits for plants. Plants have developed many defence mechanisms to protect themselves against herbivores [16]. These problems can be physical or chemical. For example, some plants can produce bitter or toxic chemicals that prevent animals from eating them. Some plant species have modified defence mechanisms, such as thorns or tough leaves, making them less attractive or difficult for predators.

Herbivores and plants interact and are constantly changing. This often leads to a concerted "arms race" in which animals adapt mechanisms to overcome the defences created by plants in response to their presence. The continuation of this process leads to the evolution of both. In addition, plants are capable of interacting with one another and other organisms to defend against herbivore attacks [18]. 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 [12]. These herbivore-controlling natural adversaries contribute to an intricate web of ecosystem interactions [13, 14].

Herbivores, creatures that survive on plants, launch both direct and indirect impacts. This leads to significant results within ecosystems. The possibility of trophic cascades is an important indirect effect. Herbivore modifications initiate a chain of linked outcomes. These cascade across the entire ecosystem. If the predatory population decreases, herbivores tend to proliferate limitlessly. This leads to misery and overgrazing. The diversity and richness of plant life are on the decline. This scenario impacts additional species. Those who need these plants for sustenance and shelter bear the brunt. This triggers a series of ecological changes. When predators are present, we see a different picture [19]. When predators are around, their role becomes apparent. They control the number of herbivores. This action guarantees a stable balance within the ecosystem. This underlines the importance of a controlled flow in an ecosystem [20]. 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 [24]. The nutrient recycling process can impact the availability of nutrients in the soil, which in turn benefits the growth of surrounding plants. Herbivores can sometimes affect nitrogen levels in plant tissues, which in turn affects the quality of plants as food sources for higher trophic levels [21]. In addition, herbivores have the ability to establish mutually advantageous associations with plants [25]. 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 participate in complex interactions that manifest themselves in different ways. Pesticides are chemical compounds designed to control or eradicate pests such as herbivorous insects and other organisms that eat plants. Pesticides act as a means of protection when sprayed on plants. Treatment application is mutable. It could incorporate different methods. Preferably, either sprays, dust, or systemic applications [26]. When herbivores consume these treated plants, they effectively digest pesticides. As a result, there is direct poisoning. Such poisoning can cause harm to herbivores. Outcomes range from inherent physical pain to death. Additionally, there could be residues of pesticides on plants. These residues transfer to or remain in plant tissues. Herbivores are thus exposed to pesticides after ingesting them. These residues could lead to sublethal impacts. Alternatively, the accumulation might be gradual. The connection between plants, herbivores and the application of pesticides is crucial. This nexus introduces the likelihood of unexpected effects. Pesticides can endanger non-target organisms. This group includes beneficial insects and wildlife. Interaction between these creatures and herbivores is part of natural ecosystems. Interference with this has ripple effects on the environment. Collateral damage is a real possibility from pesticide applications. This can disturb an ecosystem with extensive consequences. Resilience in certain herbivorous pests to pesticides can develop. In such situations, resorting to more potent chemicals or different chemical treatments becomes necessary. This resistance is the result of genetic changes in the pest population. In addition, pesticides can occasionally affect the plants themselves. Exposure to pesticides can cause phytotoxicity if plants show visible damage or strain. This highlights the importance of careful consideration in insecticide selection and administration.

The ecological impacts of pesticides are integral to agricultural and pest management. Pesticides are chemical substances. They are designed to control or kill pests. With the ability to alter intended pests, they have more wide-ranging effects, also influencing the broader ecosystem. These chemical compounds may filter into water. This often happens due to runoff from fields poisoning aquatic ecosystems. Water with pesticide residues can harm aquatic organisms. They can cause havoc to fish and compose the delicate equilibrium of susceptible ecosystems. Furthermore, pesticides can linger in soil for extensive periods. This has the potential to disrupt soil quality. It can harm various species that were not the target of pesticides. As a counter to these environmental threats, adoption of integrated pest management or IPM methods has a strong base

in the agriculture sector. Integrated Pest Management, referred to as IPM, is an all-encompassing tactic. It aims to control pests. Moreover, it focuses on minimising the environmental impacts of applying pesticides. This technique intertwines a variety of measures, including biological control. Other useful strategies are also part of this technique. Cultural practices are another such strategy. It also includes the careful use of pesticides as the final choice. Biological control is about using natural means to control pests. These are natural predators, parasites or pathogens. Pathogens can work to manage and control pest populations. Cultural practices, however, include crop rotation. Other principles of cultural practice are the deliberate selection of pest-resistant crop types and habitat management. Habitat management specifically aims to bring beneficial creatures onto crops. These beneficial creatures feed on pests, reducing pest populations. Integrated pest management has a basic principle. This principle is to employ pesticides only when essential. The focus is always on using non-harmful alternatives. Using these alternatives reduces the negative impacts pesticides have on non-target organisms. It also benefits the ecosystem generally [27].

Our study presents a novel approach by including the herbivore population in a mathematical model of the dynamics of susceptible infected plants together with the application of pesticides as a control measure. This inclusion demonstrates the important role of herbivores in ecological systems, where their consumption of susceptible and infected plants can significantly influence the dynamics of disease transmission. Our goal in modelling these interactions is to show how the feeding behaviour of herbivores affects the spread of disease and the effectiveness of pesticide use. This integrated approach expands the traditional scope of epidemiological models and provides an understanding of the complex ecological relationships that govern the occurrence of plant diseases. In addition, our research deals with the interaction between pesticides and herbivores. This aspect is crucial for the control of plant diseases but has not yet been studied in depth. We are researching pesticides. They aim to control diseases in plants. However, they also have an impact on herbivore populations. These populations feed on plants that have been treated. The dual effect indicates a need for an integrated pest management plan. The plan should factor in both disease control and ecological sustainability. We quantify these interactions in our model. This, in turn, provides a basis for the improvement of more effective pest management practices. This would be done in an environmentally responsible manner. In these areas, we contribute to theoretical ecology and agricultural sustainability.

2. MATHEMATICAL MODEL FORMULATION AND DESCRIPTION

In our mathematical model, we consider three different groups: the plant group and the herbivores group. Plant and herbivore populations were divided into two subpopulations and one population, respectively. This subpopulation includes susceptible plants ($X_1(t)$) and infected plants ($X_2(t)$). The variable $Y(t)$ represents the herbivore population. Our goal is to determine the dynamics and interactions of these groups. By studying the behaviour of these populations, we can better understand the ecological processes that control their growth and survival. The model uses pesticides represented by $Z(t)$ as a control mechanism, administering them to both susceptible and infected plants. The primary goal of pesticide treatment is to reduce the incidence of diseases in plants. Pesticides can protect crops and increase their long-term health and productivity. In the absence of these management techniques, there is a risk of losing the entire crop due to the dangers posed by diseases and pests. We must insist on the responsible and sustainable use of pesticides to protect our food and promote permaculture. In this study, we create a model based on a series of considerations. We built this model based on the following 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:

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

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 [1]. 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:

$$\frac{dX_1}{dt} = \mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_1X_2 - aX_1Y - bX_1Z - cX_1, \quad (2)$$

$$\frac{dX_2}{dt} = \beta X_1X_2 - dX_2Y - eX_2Z - cX_2. \quad (3)$$

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:

$$\frac{dY}{dt} = aX_1Y + dX_2Y - fYZ - gY. \quad (4)$$

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:

$$\frac{dZ}{dt} = -\rho Z + \delta. \tag{5}$$

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

$$\begin{aligned}
\frac{dX_1}{dt} &= \mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_1 X_2 - aX_1 Y - bX_1 Z - cX_1 \\
\frac{dX_2}{dt} &= \beta X_1 X_2 - dX_2 Y - eX_2 Z - cX_2 \\
\frac{dY}{dt} &= aX_1 Y + dX_2 Y - fYZ - gY \\
\frac{dZ}{dt} &= -\rho Z + \delta.
\end{aligned} \tag{6}$$

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

3. PRELIMINARIES

In this section, we aim to demonstrate the positivity, boundedness and Existence and Uniqueness of the model for the solutions of system (6).

3.1. Positivity of Solutions

In order for system (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 1.

Theorem 1: Let the parameters for model (6) be positive constants. A non-negative solution $(X_1(t), X_2(t), Y(t), Z(t))$ for model (6) exists for all state 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), we have:

$$\frac{dX_1}{dt} = X_1\left[\mathrm{r}\left(1 - \frac{X_1+X_2}{K}\right) - \beta X_2 - aY - bZ - c\right]. \tag{7}$$

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 (7) becomes:

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

By separation of variable method, we have, $\frac{dX_1}{X_1} = [l(X_1, X_2, Y, Z) - c]dt$.

Integrating both sides, we get:

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

Exponentiating both sides, we obtain:

$$X_1(t) = C_1 e^{\int_0^t [l(X_1(\epsilon), X_2(\epsilon), Y(\epsilon), Z(\epsilon)) - c]d\epsilon}, \tag{8}$$

Where $C_1 = e^{C_0}$.

Since $C_1 > 0$, 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), 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 for $X_1(t)$ and $X_2(t)$, 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) remain positive for all $t \geq 0$, meaning that the model is meaningful and well posed. This completes the proof of the theorem.

Remark 1: Theorem 1 ensures that the populations of susceptible plants ($X_1(t)$), infected plants ($X_2(t)$), herbivores ($Y(t)$), and the amount of pesticides used ($Z(t)$) remain positive over time if they start with positive initial values. This is crucial because negative values would be biologically meaningless. Susceptible plants ($X_1(t)$) are influenced by environmental factors, competition, and herbivores, and will remain positive, ensuring they don't die out. Infected plants ($X_2(t)$), will also stay positive despite reductions by herbivores and pesticides. Herbivores ($Y(t)$), reliant on plant availability, will continue to exist, and the amount of pesticides ($Z(t)$), which naturally degrades over time, will always be positive. Thus, the model remains biologically significant and realistic over time.

3.2. Boundedness of Solutions

Theorem 2: All solutions of the system (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:

$$\frac{dW}{dt} = \frac{dX_1}{dt} + \frac{dX_2}{dt} + \frac{dY}{dt} + \frac{dZ}{dt}. \tag{9}$$

By substituting the model equations (6) in (9), we obtain:

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

Grouping the terms, we get:

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

Further simplifying, we get:

$$\frac{dW}{dt} \leq \widehat{K}(r+1) - hW,$$

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

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

As $t \to \infty$, we have:

$W(t) \leq \frac{\widehat{K}}{h}(r+1)$, implying that the solution is bounded within $0 \leq W \leq \frac{\widehat{K}}{h}(r+1)$.

Thus, all the solutions of the system (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 any $\omega > 0$ and $t \to \infty$.

Remark 2: Theorem 2 shows that the populations of susceptible plants ($X_1(t)$), infected plants ($X_2(t)$), herbivores ($Y(t)$), and the amount of pesticides used ($Z(t)$) will stay within certain limits over time. This is important because it keeps the model realistic and ensures it works in the real-world system. Susceptible plants ($X_1(t)$) grow and compete with infected plants, and are affected by herbivores and pesticides, but their numbers won't get too high. Infected plants ($X_2(t)$) are kept in check by natural decay, herbivores, and pesticides, so they won't spread out of control. Herbivores ($Y(t)$) will also stay in a stable range because they rely on the plants. The amount of pesticides ($Z(t)$) used will not build up endlessly. Overall, this means that all parts of the system will stay balanced and manageable.

3.3. Existence and Uniqueness of the System

In this section, we investigate the existence and uniqueness of the solutions of the eco-epidemic

system (6) in the region $\Pi \times (0, T]$.

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 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), 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:

$$\begin{aligned} G_1(S) &= \mathrm{r}X_1\left(1 - \tfrac{X_1+X_2}{K}\right) - \beta X_1 X_2 - aX_1 Y - bX_1 Z - cX_1, \\ G_2(S) &= \beta X_1 X_2 - dX_2 Y - eX_2 Z - cX_2, \\ G_3(S) &= aX_1 Y + dX_2 Y - fYZ - gY, \\ G_4(S) &= -\rho Z + \delta. \end{aligned} \tag{10}$$

For each $S, \bar{S} \in \Pi$, it follows from (6) that:

$$\|G(S) - G(\bar{S})\| = |G_1(S) - G_1(\bar{S})| + |G_2(S) - G_2(\bar{S})| + |G_3(S) - G_3(\bar{S})| + |G_4(S) - G_4(\bar{S})| \tag{11}$$

Now,

$|G_1(S) - G_1(\bar{S})|$

$= \left|\mathrm{r}X_1\left(1 - \tfrac{X_1+X_2}{K}\right) - \beta X_1 X_2 - aX_1 Y - bX_1 Z - cX_1 - \mathrm{r}\overline{X_1}\left(1 - \tfrac{\overline{X_1}+\overline{X_2}}{K}\right) + \beta\overline{X_1}\,\overline{X_2} + a\overline{X_1}\,\bar{Y} + b\overline{X_1}\,\bar{Z} + c\overline{X_1}\right|,$

$= \left|r(X_1 - \overline{X_1}) - \tfrac{\mathrm{r}X_1(X_1+X_2)}{K} - \beta X_1 X_2 - aX_1 Y - bX_1 Z - cX_1 + \tfrac{\mathrm{r}\overline{X_1}(\overline{X_1}+\overline{X_2})}{K} + \beta\overline{X_1}\,\overline{X_2} + a\overline{X_1}\,\bar{Y} + b\overline{X_1}\,\bar{Z} + c\overline{X_1}\right|,$

$\leq |r(X_1 - \overline{X_1})| + \tfrac{\mathrm{r}}{K}\left|X_1^2 - \overline{X_1}^2\right| + \left(\tfrac{r}{K} + \beta\right)|X_1 X_2 - \overline{X_1}\,\overline{X_2}| + a|X_1 Y - \overline{X_1}\,\bar{Y}| + b|X_1 Z - \overline{X_1}\,\bar{Z}| + c|X_1 - \overline{X_1}|,$

$\leq r|(X_1 - \overline{X_1})| + \tfrac{2\mathrm{r}}{K}\varphi|X_1 - \overline{X_1}| + \left(\tfrac{r}{K} + \beta\right)\varphi|X_1 - \overline{X_1}| + \left(\tfrac{r}{K} + \beta\right)\varphi|X_2 - \overline{X_2}| + a\varphi|X_1 - \overline{X_1}| + a\varphi|Y - \bar{Y}| + b\varphi|X_1 - \overline{X_1}| + b\varphi|Z - \bar{Z}| + c|X_1 - \overline{X_1}|.$

$|G_2(S) - G_2(\bar{S})|$

$= |\beta X_1 X_2 - dX_2 Y - eX_2 Z - cX_2 - \beta\overline{X_1}\,\overline{X_2} + d\overline{X_2}\bar{Y} + e\overline{X_2}\bar{Z} + c\overline{X_2}|,$

$\leq \beta\varphi|X_1 - \overline{X_1}| + \beta\varphi|X_2 - \overline{X_2}| + d\varphi|X_2 - \overline{X_2}| + d\varphi|Y - \bar{Y}| + e\varphi|X_2 - \overline{X_2}| + e\varphi|Z - \bar{Z}| + c|X_2 - \overline{X_2}|.$

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

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

Then equation (11) 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:
$H_0 = max\left\{r + \left(\frac{3r}{K} + 2\beta + 2a + b\right)\varphi + c, \left(\frac{r}{K} + 2\beta + 2d + e\right)\varphi + c, (2a + 2d + f)\varphi + g,\right.$
$\left.(b + e + f)\varphi + \rho\right\}$. Hence, $G(S)$ satisfies the Lipschitz condition with respect to S, which implies the existence and uniqueness of solution of the system (6).

Remark 3: Theorem 3 confirms that, within a realistic range of initial conditions, the eco-epidemic system (6) has a unique, continuous solution over time. From a biological perspective, this implies that we can reliably model the interactions between a defined number of susceptible and infected plants, herbivores, and pesticide levels, shaped by growth rates, infections, predation, and pesticide use. This unique solution makes sure that these populations follow the path set by the system's equations. This shows that the model can accurately predict how environmental factors and human actions affect plant diseases and the behaviour of herbivores in agriculture.

4. EQUILIBRIA AND STABILITY

The eco-epidemic model (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 highlights 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 $P_3 = \left(\widehat{X_1}, \widehat{X_2}, \widehat{Y}, \hat{Z}\right) = \left(\frac{f\delta+g\rho}{a\rho}, 0, \frac{Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)}{K\rho a^2}, \frac{\delta}{\rho}\right)$, which 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, thereby ensuring their well-being.

Now, we aim to determine the basic reproduction number R_0 of System (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 [2], the basic reproduction number, denoted as R_0, can be determined as the spectral radius of the next generation operator FV^{-1}.

Therefore, $R_0 = \sigma(FV^{-1}) = \frac{K\beta a(f\delta+g\rho)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}$.

R_0 plays an important role in 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 case. It is an important notion for determining contagiousness and disease transmission potential. It is defined as the average rate of infection within a susceptible plant population caused by a variety of secondary infections.

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

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

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

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

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

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) is investigated. The Jacobian matrix J is given as follows:

$$J = \begin{bmatrix} J_{11} & \frac{-(\beta K+r)X_1}{K} & -aX_1 & -bX_1 \\ \beta X_2 & J_{22} & -dX_2 & -eX_2 \\ aY & dY & J_{33} & -fY \\ 0 & 0 & 0 & -\rho \end{bmatrix} \tag{12}$$

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

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

$J_{33} = \beta a X_1 + dX_2 - fZ - g.$

The eigenvalues of the Jacobian matrix J evaluated at the trivial equilibrium point $P_1\left(0,0,0,\frac{\delta}{\rho}\right)$ for the system (6) 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.

Remarks 4: In practical terms, the health and survival of a plant population hinge on a delicate balance between two critical factors: It is a relationship between the birth rate, or, in other words, the biotic potential of the population, and the natural death rate. The birth rate is the rate at which new plants are being born or new seeds germinating to join the population, while the natural death

rate is the rate at which plants in the population die a natural death, say due to old age, disease, predation or unfavourable environmental conditions. Indeed, if the number of plants cannot replace itself, the plant population is threatened with decline and extinction when coupled with unfavourable circumstances. Nevertheless, despite the fact that we can get a stable equilibrium point mathematically, this point means a very dangerous position for the population because it indicates that the population has decreased significantly and cannot remain populated if people live in such conditions. Thus, it underlines the plant population's sensitivity to adverse environmental factors and stresses the need to protect the plant population by implementing specific conservation actions or paradigms.

Theorem 4: If $\beta K < c + \frac{e\delta}{\rho}$, $aK < g + \frac{f\delta}{\rho}$, then the axial equilibrium point $P_2\left(K, 0, 0, \frac{\delta}{\rho}\right)$ is locally asymptotically stable.

Proof: The Jacobian matrix $J(P_2)$ of the model (6) around P_2 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 $aK - \left(g + \frac{f\delta}{\rho}\right)$ must be 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 can conclude that the equilibrium point P_2 exhibits local stability when $\beta K < c + \frac{e\delta}{\rho}$ and $aK < g + \frac{f\delta}{\rho}$.

Theorem 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 $\mathrm{R}_0 < 1$ and is unstable when $\mathrm{R}_0 > 1$.

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) centered around P_3 is as follows:

$$J(P_3)=\begin{bmatrix} \frac{-r(f\delta+g\rho)}{K\rho a} & \frac{-(K\beta+r)(f\delta+g\rho)}{K\rho a} & \frac{-(f\delta+g\rho)}{\rho} & \frac{-b(f\delta+g\rho)}{a\rho} \\ 0 & J_{22}(P_3) & 0 & 0 \\ \frac{Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)}{K\rho a} & \frac{d[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2} & 0 & J_{34}(P_3) \\ 0 & 0 & 0 & -\rho \end{bmatrix}.$$

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

$J_{34}(P_3)=\frac{f[Ka(r\rho-c\rho-b\delta)-r(f\delta+g\rho)]}{K\rho a^2}$.

The characteristic equation of the above Jacobian matrix $J(P_3)$ 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:

$$c_1\chi^2+c_2\chi+c_3=0 \tag{13}$$

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

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

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

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: The coexistence equilibrium point $P_4(X_1^*,X_2^*,Y^*,Z^*)$ is locally asymptotically stable

if:

$$M_2 > 0,\ M_3 > 0,\ M_4 > 0,\ M_1M_2 - M_3 > 0 \text{ and } (M_1M_2 - M_3)M_3 - M_1^2M_4 > 0 \quad (14)$$

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:

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

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:

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

Which gives,

$$\theta^4 + M_1\theta^3 + M_2\theta^2 + M_3\theta + M_4 = 0. \quad (15)$$

Recognising that $M_1 > 0$, then by applying the Routh–Hurwitz criterion in conjunction with the conditions outlined in (14), we conclude that the coexistence equilibrium P_4 of system (6) exhibits local asymptotic stability if $M_2 > 0$, $M_3 > 0$, $M_4 > 0$, $M_1M_2 - M_3 > 0$ and $(M_1M_2 - M_3)M_3 - M_1^2M_4 > 0$. Conversely, if any of these conditions are not satisfied, the coexistence equilibrium P_4 is determined to be unstable.

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 in reference [3].

Definition 1: The system (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), $(X_1(0), X_2(0), Y(0), Z(0)) > 0$,

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

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

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

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

Definition 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 7: The system (6) is uniformly persistent if the following condition is satisfied:

$$\beta\widehat{X_1} - d\hat{Y} - \left(\frac{e\delta + c\rho}{\rho}\right) > 0 \text{ holds.} \quad (15)$$

Proof: We will prove this theorem by the method of Lyapunov average function. Let the average Lyapunov function for the system (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,

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

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

$$dX_2 - fZ - g) + s_1 \left(-\rho + \frac{\delta}{Z} \right). \quad (16)$$

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 $p_1, q_1, r_1, s_1 > 0$:

$$\Psi(P_1) = p_1 \left[r - \left(\frac{b\delta + c\rho}{\rho} \right) \right] - q_1 \left(\frac{e\delta + c\rho}{\rho} \right) - r_1 \left(\frac{f\delta + g\rho}{\rho} \right) > 0, \quad (17)$$

$$\Psi(P_2) = -p_1 \left(\frac{b\delta + c\rho}{\rho} \right) + q_1 \left[\beta K - \left(\frac{e\delta + c\rho}{\rho} \right) \right] + r_1 \left[aK - \left(\frac{f\delta + g\rho}{\rho} \right) \right] > 0, \quad (18)$$

$$\Psi(P_3) = q_1 \left[\beta \widehat{X_1} - d\hat{Y} - \left(\frac{e\delta + c\rho}{\rho} \right) \right] > 0. \quad (19)$$

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 (17) 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 (18) holds. If the inequality in Equation (15) holds, then (19) is satisfied.

6. SENSITIVITY ANALYSIS

We want to know what factors contribute to disease transmission and prevalence. This information can help us reduce herbivore mortality due to disease and lower plant infections. Sensitivity analysis provides insight into the importance of every factor in disease spread. Designing experiments depends not only on this knowledge, but also on the incorporation of data and the simplification of complex nonlinear models [4]. Since data collection and expected parameter values generally involve some degree of uncertainty, sensitivity analysis is a common instrument for evaluating how model predictions react to fluctuations in parameter values. It should be the main focus of intervention plans because it helps define criteria that have a significant impact on the basic reproduction number. When a parameter changes, sensitivity indices help us measure the relative influence of a variable [5]. Analysing these metrics helps us pinpoint the factors most important for disease spread and frequency.

Definition 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:

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

1. **Sensitivity of R_0 to K**:

$$\gamma_K^{R_0} = \frac{K}{R_0}\frac{\partial R_0}{\partial K} = \frac{dr(f\delta+g\rho)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

2. **Sensitivity of R_0 to β**:

$$\gamma_\beta^{R_0} = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta} = 1.$$

3. **Sensitivity of R_0 to a**:

$$\gamma_a^{R_0} = \frac{a}{R_0}\frac{\partial R_0}{\partial a} = \frac{-dr(f\delta+g\rho)-Ka^2(e\delta+c\rho)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

4. **Sensitivity of R_0 to d**:

$$\gamma_d^{R_0} = \frac{d}{R_0}\frac{\partial R_0}{\partial d} = \frac{d\left(-Ka(r\rho-c\rho-b\delta)+r(f\delta+g\rho)\right)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

5. **Sensitivity of R_0 to r**:

$$\gamma_r^{R_0} = \frac{r}{R_0}\frac{\partial R_0}{\partial r} = \frac{r\left(-Kda\rho+d(f\delta+g\rho)\right)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

6. **Sensitivity of R_0 to c**:

$$\gamma_c^{R_0} = \frac{c}{R_0}\frac{\partial R_0}{\partial c} = \frac{c\left(Kda\rho-Ka^2\rho\right)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

7. **Sensitivity of R_0 to b**:

$$\gamma_b^{R_0} = \frac{b}{R_0}\frac{\partial R_0}{\partial b} = \frac{Kbda\delta}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

8. **Sensitivity of R_0 to e**:

$$\gamma_e^{R_0} = \frac{e}{R_0}\frac{\partial R_0}{\partial e} = \frac{Kea^2\delta}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

9. **Sensitivity of R_0 to f**:

$$\gamma_f^{R_0} = \frac{f}{R_0}\frac{\partial R_0}{\partial f} = \frac{f(f\delta+g\rho)\left[Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)\right]+Kf\beta d\delta a(f\delta+g\rho)}{Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)}.$$

10. **Sensitivity of R_0 to g**:

$$\gamma_g^{R_0} = \frac{g}{R_0}\frac{\partial R_0}{\partial g} = \frac{g\rho^2(Kda-dr)^2}{(f\delta+g\rho)[Kda(r\rho-c\rho-b\delta)-dr(f\delta+g\rho)+Ka^2(e\delta+c\rho)]^2}.$$

11. **Sensitivity of R_0 to ρ**:

$$\gamma_\rho^{R_0} = \frac{\rho}{R_0}\frac{\partial R_0}{\partial \rho} = \frac{\rho(g-c\rho)}{(f\delta+g\rho)}.$$

12. **Sensitivity of R_0 to δ**:

$$\gamma_{\delta}^{R_0} = \frac{\delta}{R_0}\frac{\partial R_0}{\partial \delta} = \frac{\delta[-Kda(r\rho - c\rho - b\delta) + dr(f\delta + g\rho) - Ka^2(e\delta + c\rho)]}{(f\delta + g\rho)}.$$

The sensitivity indices of the basic reproduction number R_0 with respect to different parameters show that some parameters, like β, e, and ρ, have simple proportional relationships with R_0, while others, like a, d, r, c, b, f, and g, have more complex relationships. Parameters with high positive sensitivities, such as β, e, and g, should be prioritised in disease control strategies because they have a significant impact on R_0 and disease spread. Conversely, parameters with lower or negative sensitivities, like a, d, r, c, b, and f, may offer alternative avenues for intervention. This sensitivity analysis is critical for refining plant disease models, guiding public health responses, and forming policy decisions to effectively control and mitigate plant infectious disease transmission.

6.1 Sensitivity Analysis Results

In this study, we conducted sensitivity studies using MATLAB to analyse the dynamics of infectious disease transmission within plant communities. To investigate how parameter changes affect model outputs, we used our eco-epidemiological model using computational methods, more especially, MATLAB's strong numerical solvers and visualisation capabilities. We investigated in this MATLAB implementation how variations in significant parameters, including the infection rate (β), affected the basic reproduction number (R_0) and final state values of population variables. This computer method helped us to effectively investigate a broad spectrum of scenarios and obtain important understanding of the fundamental processes behind the emergence of diseases.

Table 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 focuses on a variety of factors, including growth rates, carrying capacity, infection rates, and other types of birth and death rates found in plant populations. The model indicates how degrees of contagiousness affect the steady values of X_1, X_2, Y, and Z through variations in the infection rate β. The results show how the change in β influences population equilibriums and reflects relationships between those populations. It is clear that the dynamics of the population layer entail numerous linked ecological balances since many important factors, including r, K, and δ, dictate the stability of the system as well as the reactivity of many elements of the environment. These revelations help one to appreciate the capacity of the system to resist infection and the effect of management strategies in altering population rates in an evolving environment.

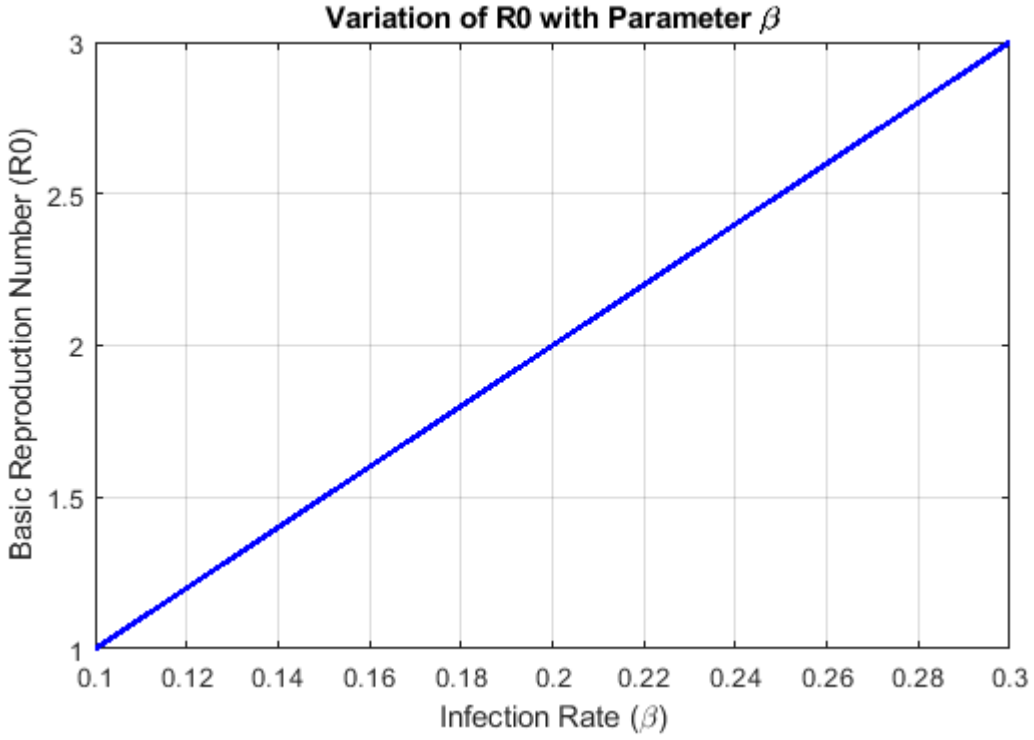

Figure 1: Variations in Basic Reproduction Number (R_0) with Infection Rate (β)

Figure 1 describes the variation of R_0 depending on the parameter β, thereby clarifying the capacity of disease spread. According to the model, increasing the infection rate per contact (β) leads to a greater reproduction number R_0, indicating that the plant population is more likely to become infected when they come into contact with an infectious population. This indicates a greater population danger; specifically, every one of these cases infects more of the easily susceptible group. Conversely, a decrease in β results in a drop in R_0, which denotes either minimal or no disease spreading. Developing a population's control strategies depends on

awareness of this link since efforts meant to lower the control parameter R_0 by affecting β significantly affect disease transmission. Having identified those three time-variant indices, graph peaks and troughs show particular quantities of R_0 as corresponding to varying degrees of disease transmissibility. These theories of the interactions between R_0 and the likelihood of the spread of disease offer a clear knowledge of the particular oscillations in the rate of infection and for the development of particular strategies to prevent the spread of infectious diseases.

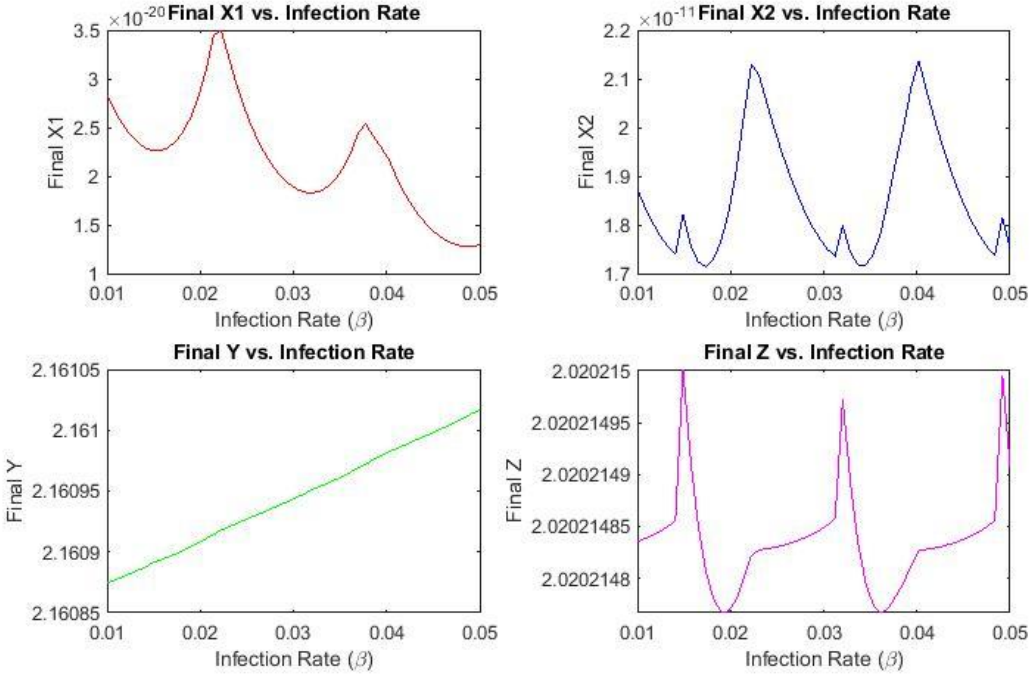

Figure 2: Final State Population Dynamics against Infection Rate (β)

Figure 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:

I. **Final X_1 vs. Infection Rate**: The population of susceptible plants (X_1) shows oscillatory behaviour with peaks and troughs, but there is a general downward trend as β increases.

II. **Final X_2 vs. Infection Rate**: The population of infected plants (X_2) also displays oscillatory behaviour, with several peaks, but there is a slight overall decrease as β increases.

III. **Final Y vs. Infection Rate**: The population of herbivores (Y) shows a steady increase as the infection rate (β) rises.

IV. **Final Z vs. Infection Rate**: The amount of pesticides used (Z) also demonstrates an oscillatory pattern, with distinct peaks, but the overall trend is a slight increase with increasing β.

These patterns show the intricate ecological connections maintained within the system's dynamics, governed by parameters such as $r = 0.1,\ K = 100,\ a = 0.02,\ b = 0.01,\ c = 0.1,\ d = 0.01,\ e = 0.02,\ f = 0.01,\ g = 0.01,\ \rho = 0.05$ and $\delta = 0.1$. These parameter values control the population's equilibrium, interaction, and expansion over time, therefore determining the system's behaviour.

7. ANALYSIS OF OPTIMAL CONTROL

In this section, we focus on using Pontryagin's Maximum Principle, a mathematical framework for solving optimum control issues [7]. The aim is to find out and understand the key factors required for best control of the effects of plant inoculation. We extend the fundamental model, system (6), to include $u(t)$, a dynamic control parameter. This parameter shows the control action carried out during the plant inoculation process, meant to stop the spread 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:

$$\begin{aligned}
\frac{dX_1}{dt} &= \mathrm{r}X_1\left(1 - \frac{X_1+X_2}{K}\right) - (1-u)\beta X_1 X_2 - aX_1 Y - bX_1 Z - cX_1,\\
\frac{dX_2}{dt} &= (1-u)\beta X_1 X_2 - dX_2 Y - eX_2 Z - cX_2, \qquad (20)\\
\frac{dY}{dt} &= aX_1 Y + dX_2 Y - fYZ - gY\\
\frac{dZ}{dt} &= -\rho Z + \delta.
\end{aligned}$$

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:

$$J^*(u) = \min_u \int_0^{t_f} \left(AX_1(t) + BX_2(t) + \frac{1}{2}B_1u^2(t)\right) dt \quad (21)$$

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 minimise the number of infected plants while also minimising 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:

$$J^*(u^*) = min\{J^*(u)\} \quad (22)$$

Here, we will first show the existence of an optimal control (22) for the system (20). This is supported by the following **Theorem 8**.

Theorem 8: There exists an optimal control $u^*(t)$ such that $J^*(u^*(\mathrm{t})) = min\{J^*(u(t))\}$ subject to the control system (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) 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 [6].

To achieve an optimal solution, the initial step involves determining the Lagrangian and Hamiltonian for the given problem outlined in equations (20) to (21). The Lagrangian associated with this optimal control problem is given by:

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

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

$$H\left(X_1, X_2, u, \lambda_{X_1}, \lambda_{X_2}, \lambda_Y, \lambda_Z, t\right) = L(X_1, X_2, u) + \lambda_{X_1}(t)\frac{dX_1(t)}{dt} + \lambda_{X_2}(t)\frac{dX_2(t)}{dt} + \lambda_Y(t)\frac{dY(t)}{dt} + \lambda_Z(t)\frac{dZ(t)}{dt} \quad (23)$$

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 [7].

The application of Pontryagin's Maximum Principle typically transforms the system of equations (20) and (21) into a point-wise minimisation problem of the Hamiltonian H, with respect to the control variable $u(t)$.

Theorem 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 (20) – (21). 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 (23). The adjoint equations are derived through the application of Pontryagin's Maximum Principle.

$\frac{d\lambda_{X_1}}{dt} = -\frac{\partial H}{\partial X_1}$, $\frac{d\lambda_{X_2}}{dt} = -\frac{\partial H}{\partial X_2}$, $\frac{d\lambda_Y}{dt} = -\frac{\partial H}{\partial Y}$ 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 $B_1u(t)+\lambda_{X_1}\beta X_1X_2-\lambda_{X_2}\beta X_1X_2=0$.

This implies that $u(t)=\frac{\beta(\lambda_{X_2}-\lambda_{X_1})X_1X_2}{B_1}(=u^*(t))$, say.

Using the property of control space, we obtain

$u^*(t)=0$ if $\frac{\beta(\lambda_{X_2}-\lambda_{X_1})X_1X_2}{B_1}\leq 0$,

$u^*(t)=\frac{\beta(\lambda_{X_2}-\lambda_{X_1})X_1X_2}{B_1}$ if $0<\frac{\beta(\lambda_{X_2}-\lambda_{X_1})X_1X_2}{B_1}<u_{max}$

$u^*(t)=u_{max}$ if $\frac{\beta(\lambda_{X_2}-\lambda_{X_1})X_1X_2}{B_1}\geq u_{max}$.

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.

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. We use plots and graphs to illustrate the numerical solution 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 optimisation.

Table 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) 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.

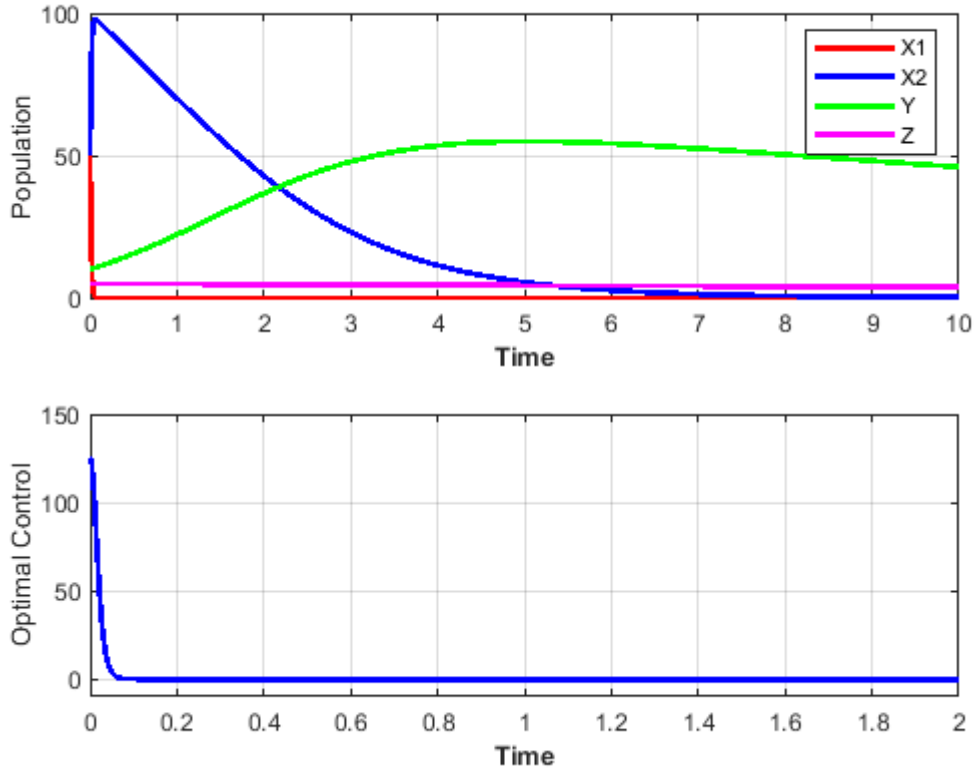

Figure 3: Dynamics of Ecological Variables and Optimal Control Profile

Figure 3 explains the dynamics of ecological variables and the optimal control strategy used to manage plant diseases within an ecological system.

1) **Ecological Variables Dynamics (Upper Subplot):**
   - $\boldsymbol{X_1(t)}$ **(Red line):** Represents the susceptible plant population. This population starts at a certain level and quickly decreases, indicating that these plants are being infected by the disease or impacted by pesticide application aimed at controlling the spread of the disease.

- $\boldsymbol{X_2(t)}$ **(Blue line):** Represents the infected plant population. Initially high, this population decreases over time, demonstrating the effectiveness of the pesticide in reducing the number of infected plants.
- $\boldsymbol{Y(t)}$ **(Green Line):** Represents the herbivores population. This population starts low, increases to a peak, and then gradually decreases. The herbivores initially benefit from the availability of plants (both susceptible and infected) but eventually decline as the plant populations diminish due to disease and pesticide use.
- $\boldsymbol{Z(t)}$ **(Pink Line):** Represents the amount of pesticides. This variable stays low throughout the period, suggesting a strategy of minimal yet effective pesticide use.

2) **Optimal Control Profile (Lower Subplot):**
    - **Optimal Control:** This refers to the level of pesticide application used as a control measure.
    - **Trajectory:** The optimal control begins at a high value, indicating an aggressive initial pesticide application to swiftly halt the disease's spread. It then quickly falls and settles at a lower value, implying a maintenance phase with modest pesticide applications to help control the disease.
    - **Biological Interpretation:** The initial application is important to prevent the disease from spreading rapidly. Once the disease is under control, maintaining a reduced degree of control allows for the management of any new infections without overloading resources. This, in turn, reduces the potential negative side effects of control measures, such as resistance development and environmental impact.

Figure 3 represents the dynamic relationship that exists between plant populations and the methods taken to control diseases. A sharp decrease in the number of diseased plants and the stabilization of control measures suggest that a sustainable management approach followed an effective first response to the disease. This contributes to a better understanding of how to implement and modify disease control strategies over time in order to achieve long-term ecological stability and plant population health. This helps to minimize the spread of the disease, as well as the negative effects that control measures have.

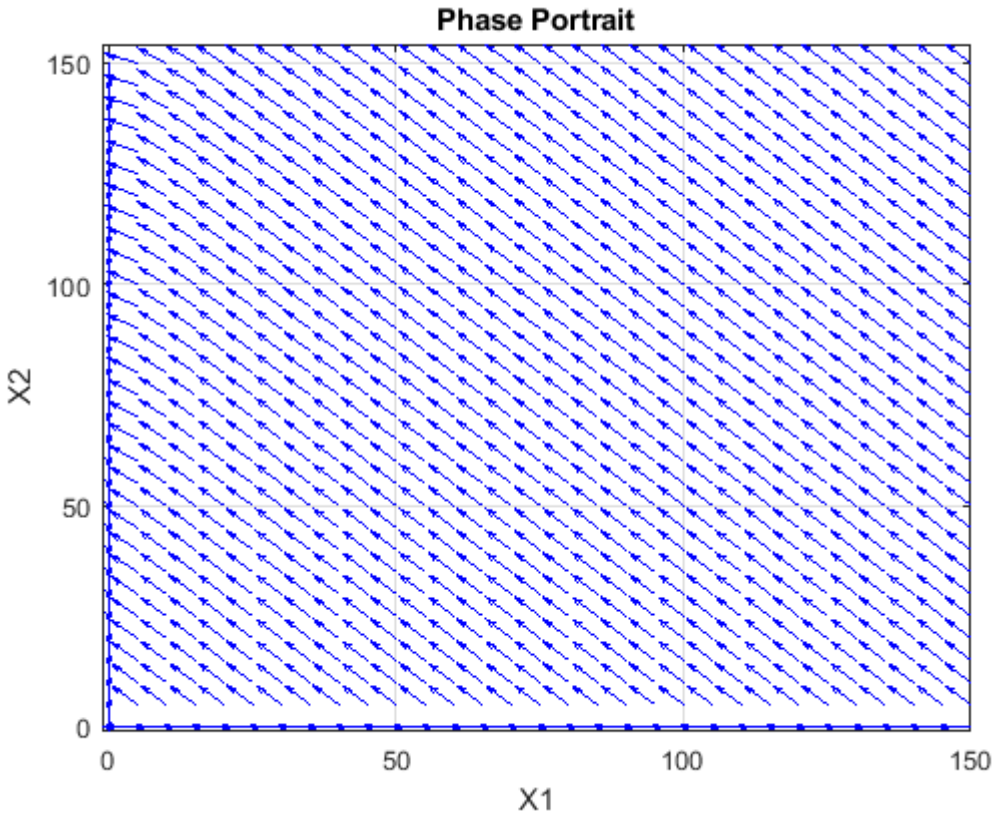

Figure 4: Phase Portrait: State Space Dynamics Visualisation

Figure 4, a phase portrait, depicts the state space dynamics of a system with susceptible plants ($X_1(t)$) and infected plants ($X_2(t)$). In this regard, the phase picture shows how, with respect to each other, the quantity of susceptible and infected plants changes with time. Every arrow in a phase portrait shows the direction and speed of change of the state variables of the system. The arrows in this diagram show the path the system will follow from any one starting point (X_1, X_2). The arrows' general direction points to how dynamically the populations of infected and susceptible plants interact. The arrows might show convergence to an equilibrium point, cyclical behaviour, or divergence, therefore indicating whether the populations stabilise, oscillate, or expand without limit.

The following are some of the most significant implications that may be drawn from the phase portrait:

- **Equilibrium Points:** These are the points where the arrows converge, which indicates steady-state solutions in which the populations of susceptible and infected plants remain constant.

- **Trajectories:** The paths that follow the arrows on the diagram indicate the various possible evolution trajectories of the system, starting with a variety of initial conditions.
- **Stability:** The stability of those points is shown by the direction and density of the arrows surrounding equilibrium points. Convergent arrows point to stable equilibrium; diverging arrows point to instability.

The phase portrait provides insight into the qualitative dynamics of plant infection without the need to explicitly solve the system's equations.

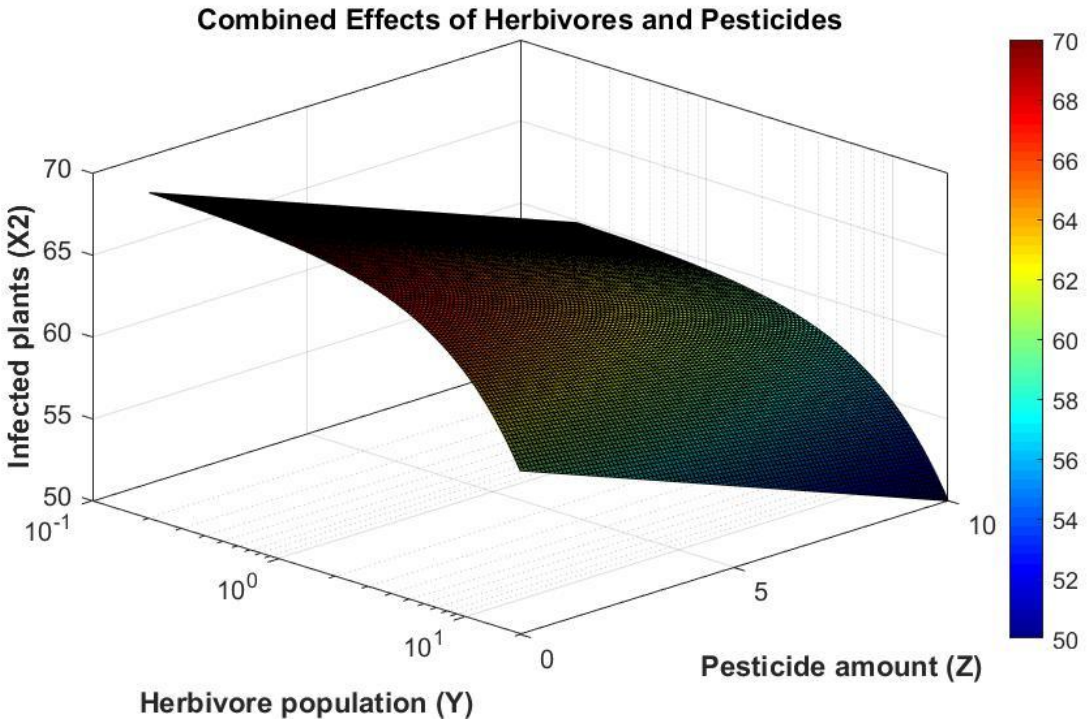

Figure 5: Combined Effects of Herbivores and Pesticides

Figure 5 illustrates the dynamic interactions between herbivores, pesticides, and plant infections in an ecosystem. Herbivores can get infected either through direct exposure to pesticides during spray applications or by feeding on infected plants ($X_2(t)$). Pesticides are introduced as a control measure to mitigate these infections. The effectiveness of pesticides ($Z(t)$) depends on factors like timing, dosage, and plant resilience. Adequate pesticide application can suppress pathogens, thereby reducing $X_2(t)$.

The figure demonstrates how these factors interact: areas with high $X_2(t)$ indicate scenarios where high herbivore populations ($Y(t)$) override the protective effects of pesticides ($Z(t)$), resulting in increased plant infections. Conversely, low $X_2(t)$ regions highlight effective pest management strategies, where either low herbivore populations, optimal pesticide use, or both contribute to minimizing plant infections.

Understanding these dynamics is important for sustainable agriculture and ecosystem management. It informs decisions on integrated pest management (IPM), where balancing pesticide use with natural controls is essential to minimize environmental impact while ensuring crop health. Insights from Figure 5 guide strategies for disease prevention and control, aiming to enhance plant health and productivity in agricultural environment.

8. DISCUSSION

We used rigorous mathematical frameworks and computational approaches in this work to analyse the dynamics of infectious disease transmission within ecological systems and determine the best control measures for managing disease spread. Our study aimed to understand the complex relationships between important factors that influence disease spread and population size changes. Furthermore, we sought to develop effective strategies to reduce the incidence of the disease.

8.1 Mathematical Analysis of Disease Dynamics

Our study began with the development of a complete mathematical model (6) that describes the dynamics of susceptible plants ($X_1(t)$), infected plants ($X_2(t)$), herbivore populations ($Y(t)$), and pesticides ($Z(t)$) as a control measure. Using methods from differential equations and dynamical systems theory, we investigated the stability and equilibrium points of the model, clarifying the conditions under which disease outbreaks arise and pointing out important thresholds for disease control. We measured, via sensitivity analysis, the proportionate influence of every model parameter on disease prevalence and spread. We evaluated the sensitivity of the basic reproduction number (R_0) to parameter fluctuations, including the infection rate (β), by computing normalized forward sensitivity indices. This study sheds important information on the elements influencing diseases progression and underlines aspects that are main targets for intervention programmes.

8.2 Optimal Control Framework

We used Pontryagin's Maximum Principle, a strong mathematical foundation for solving optimal control problems, to develop ideal control strategies for controlling disease spread. We expanded

our base model to include a dynamic control parameter ($u(t)$) reflecting the application of control strategies, such as plant inoculation, meant to lower disease spread. The optimal control problem sought to minimise the cost of the control measure ($u(t)$) and the number of infected plants. We obtained optimal control profiles that satisfy these dual objectives by thorough mathematical investigation including the formulation of Hamiltonians and adjoint equations. The interaction between state variables, adjoint variables, and maximum allowed control magnitudes defined the characterisations of optimal controls ($u^*(t)$).

8.3 Computational Implementation and Analysis

We developed our mathematical models by making use of computational techniques, including the numerical solvers and visualisation tools that are available in MATLAB. Our goal was to investigate the dynamics of the disease and determine the most effective methods of disease control. Through the process of numerically solving differential equations and modelling optimal control scenarios, we were able to get insights into the effectiveness of various control strategies in reducing the spread of disease.

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, we have discovered crucial thresholds 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, we have developed optimal control methods that aim 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, we aimed to develop effective methods to reduce the prevalence of the disease.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

REFERENCES

[1] T. Kar, A. Ghorai, S. Jana, Dynamics of Pest and Its Predator Model with Disease in the Pest and Optimal Use of Pesticide, J. Theor. Biol. 310 (2012), 187-198. https://doi.org/10.1016/j.jtbi.2012.06.032.

[2] P. van den Driessche, J. Watmough, Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission, Math. Biosci. 180 (2002), 29-48. https://doi.org/10.1016/s0025-5564(02)00108-6.

[3] K. Das, M.N. Srinivas, V. Madhusudanan, S. Pinelas, Mathematical Analysis of a Prey–predator System: An Adaptive Back-Stepping Control and Stochastic Approach, Math. Comput. Appl. 24 (2019), 22. https://doi.org/10.3390/mca24010022.

[4] H.S. Rodrigues, M.T.T. Monteiro, D.F.M. Torres, Sensitivity Analysis in a Dengue Epidemiological Model, Conf. Pap. Math. 2013 (2013), 721406. https://doi.org/10.1155/2013/721406.

[5] N.I. Hamdan, A. Kilicman, Sensitivity Analysis in a Dengue Fever Transmission Model: a Fractional Order System Approach, J. Phys.: Conf. Ser. 1366 (2019), 012048. https://doi.org/10.1088/1742-6596/1366/1/012048.

[6] W.H. Fleming, R. Rishel, Deterministic and Stochastic Optimal Control, Springer, New York, 1975. https://doi.org/10.1007/978-1-4612-6380-7.

[7] F.M. Hamelin, B. Bowen, P. Bernhard, V.A. Bokil, Optimal Control of Plant Disease Epidemics with Clean Seed Usage, Bull. Math. Biol. 83 (2021), 46. https://doi.org/10.1007/s11538-021-00872-w.

[8] S. Adhurya, F.A. Basir, S. Ray, Stage-structure Model for the Dynamics of Whitefly Transmitted Plant Viral Disease: An Optimal Control Approach, Comput. Appl. Math. 41 (2022), 154. https://doi.org/10.1007/s40314-022-01864-9.

[9] I.S. Onah, S.E. Aniaku, O.M. Ezugorie, Analysis and Optimal Control Measures of Diseases in Cassava Population, Optim. Control. Appl. Methods 43 (2022), 1450-1478. https://doi.org/10.1002/oca.2901.

[10] R.A. Khan, T. Hussain, M. Ozair, F. Tasneem, M. Faizan, Dynamical Features of Pine Wilt Disease Through Stability, Sensitivity and Optimal Control, Adv. Differ. Equ. 2021 (2021), 261. https://doi.org/10.1186/s13662-021-03411-y.

[11] L.N. Nkamba, T.T. Manga, Stability and Optimal Control of an Mathematical Model of Tuberculosis/AIDS Co-infection with Vaccination, in: New Ideas Concerning Science and Technology, Book Publisher International, 2021: pp. 39--56. https://doi.org/10.9734/bpi/nicst/v11/7665d.

[12] I.G. Ameen, D. Baleanu, H.M. Ali, Different Strategies to Confront Maize Streak Disease Based on Fractional Optimal Control Formulation, Chaos, Solitons Fractals 164 (2022), 112699. https://doi.org/10.1016/j.chaos.2022.112699.

[13] H.T. Alemneh, O.D. Makinde, D.M. Theuri, Optimal Control Model and Cost Effectiveness Analysis of Maize Streak Virus Pathogen Interaction with Pest Invasion in Maize Plant, Egypt. J. Basic Appl. Sci. 7 (2020), 180-193. https://doi.org/10.1080/2314808x.2020.1769303.

[14] O. Lazaar, M. Serhani, Stability and Optimal Control of a Prey–predator Model with Prey Refuge and Prey Infection, Int. J. Dyn. Control. 11 (2022), 1934-1951. https://doi.org/10.1007/s40435-022-01064-7.

[15] F. Zhang, Z. Qiu, A. Huang, X. Zhao, Optimal Control and Cost-Effectiveness Analysis of a Huanglongbing Model with Comprehensive Interventions, Appl. Math. Model. 90 (2021), 719-741. https://doi.org/10.1016/j.apm.2020.09.033.

[16] M.H.R. Luiz, L.T. Takahashi, R.C. Bassanezi, Optimal Control in Citrus Diseases, Comput. Appl. Math. 40 (2021), 191. https://doi.org/10.1007/s40314-021-01581-9.

[17] G.M. Kumar, M. Mullai, Deterministic and Stochastic Optimal Control Models for Plant Growth Using Locust Fertilizer, Model. Earth Syst. Environ. 9 (2022), 1891-1908. https://doi.org/10.1007/s40808-022-01596-z.

[18] F. Al Basir, S. Ray, Impact of Farming Awareness Based Roguing, Insecticide Spraying and Optimal Control on the Dynamics of Mosaic Disease, Ric. Mat. 69 (2020), 393-412. https://doi.org/10.1007/s11587-020-00522-8.

[19] J.A. Sharp, A.P. Browning, T. Mapder, C.M. Baker, K. Burrage, M.J. Simpson, Designing Combination Therapies Using Multiple Optimal Controls, J. Theor. Biol. 497 (2020), 110277. https://doi.org/10.1016/j.jtbi.2020.110277.

[20] A. Boonyaprapasorn, S. Kuntanapreeda, T. Sangpet, P.S. Ngiamsunthorn, E. Pengwang, Biological Pest Control Based on Tensor Product Transformation Method, Acta Polytech. Hung. 17 (2020), 25-40. https://doi.org/10.12700/aph.17.6.2020.6.2.

[21] T. Bakhtiar, I.R. Fitri, F. Hanum, A. Kusnanto, Mathematical Model of Pest Control Using Different Release Rates of Sterile Insects and Natural Enemies, Mathematics 10 (2022), 883. https://doi.org/10.3390/math10060883.

[22] B. Fatima, M. Yavuz, M.U. Rahman, F.S. Al-Duais, Modeling the Epidemic Trend of Middle Eastern Respiratory Syndrome Coronavirus with Optimal Control, Math. Biosci. Eng. 20 (2023), 11847-11874. https://doi.org/10.3934/mbe.2023527.

[23] W. Adel, A. Elsonbaty, A. Aldurayhim, A. El-Mesady, Investigating the Dynamics of a Novel Fractional-Order Monkeypox Epidemic Model with Optimal Control, Alex. Eng. J. 73 (2023), 519-542. https://doi.org/10.1016/j.aej.2023.04.051.

[24] C. Djuikem, A. Gabriel Yabo, F. Grognard, S. Touzeau, Mathematical Modelling and Optimal Control of the Seasonal Coffee Leaf Rust Propagation, IFAC-PapersOnLine 54 (2021), 193-198. https://doi.org/10.1016/j.ifacol.2021.08.497.

[25] P. Panja, Dynamics of a Crop, Pest and Predator Model in an Agricultural System, Results Control. Optim. 12 (2023), 100274. https://doi.org/10.1016/j.rico.2023.100274.

[26] I.R. Fitri, F. Hanum, A. Kusnanto, T. Bakhtiar, Optimal Pest Control Strategies with Cost-Effectiveness Analysis, Sci. World J. 2021 (2021), 6630193. https://doi.org/10.1155/2021/6630193.

[27] P. Kalra, M. Kaur, Mathematical Modeling on Impulsive Pest Control Strategy: A Review, Int. J. Emerg. Technol. Innov. Res. 6 (2019), 65-68.