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THE GLOBAL DYNAMICS AND OPTIMAL CONTROL OF A PLANT EPIDEMIC MODEL

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Abstract. This paper proposes and investigates a model for the spread of an infection into a plant population, considering the effects of both primary and secondary infections. We determine the basic reproduction number of the plant pathogens R_0 and prove that if $R_0 > 1$, then the positive equilibrium is globally stable, provided that several auxiliary inequalities, determined using the geometric approach of Li and Muldowney [23], hold. Also, we find a necessary condition for the existence of optimal controls by applying Pontryagin's Minimum Principle. Finally, a numerical example is given to illustrate the applicability of our analytical findings.

Keywords: plant epidemics; global stability; optimal control; Pontryagin's maximum principle.

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1. Introduction

The issue of food security has first come to public attention in the 1970s, when a widespread world food crisis broke out. As the continuous population growth and the global climate change put an ever increasing pressure on food production, food security concerns may remain prominent for at least the next fifty years [1]. With an estimated fourteen percent of world crops lost

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due to various plant diseases [2], detailed knowledge of plant epidemiology is vital from both an economic and humanitarian viewpoint. In particular, an ability to predict the spread of crop diseases and the results of deploying control strategies may help towards increasing crop yield and hence influence governmental policies.

To prevent crop diseases, farmers often resort to using chemicals [3]. However, pesticide abuse leads to unwanted consequences which include the resistance-driven evolution of pathogens and the accumulation of chemical residues into the human food chain and the environment. This is enough motivation for an increase in the legislative constraints on using pesticides [4]. There is also pressure on the use of pathogen-resistant crop varieties due to the time and cost of breeding resistance and to the ethical objections of certain consumers concerning genetic modification, as well as on the use of cultural controls, which are labor intensive and lead to reduced yield. Hence, experimentalists have become more interested in biological control [5], approach which relies on the deployment of pathogen antagonists to reduce disease spread.

Recently, certain deterministic, mechanistically based epidemiological models were proposed to study how to influence and control the spread of soil-borne plant pathogens. For example, Kleczkowski *et al*. investigated the interactions between *Rhizoctonia solani* and *Trichoderma viride* in [6, 7, 8], while Gubbins and Gilligan analyzed the dynamics between *Sporidesmium sclerotivorum*, and its host, *Sclerotinia minor*, in a disturbed or closed environment in [9, 10, 11, 12]. Furthermore, Bailey *et al*. studied the efficiency of the biological control of soil-borne diseases in a controlled environment [13]. In [15], Jeger *et al*. allowed tissue that is colonised by the antagonist to enter the removed class, which implies that the deployment of the antagonist leads to the removal of host tissue. In the model of Xu *et al.* [16], the colonised tissue becomes susceptible again instead, assumption which seems more biologically plausible. Gilligan analyzed in [14] the likely efficacy of control by investigating its effect on epidemiologically meaningful parameters which play important roles in pathogen invasion and persistence. Also, Cunniffe and Gilligan considered how microbial antagonists affect the spread of pathogens through a general plant population in [17].

However, the above-mentioned body of work did not target the estimation of optimal dynamic control regimes. In this paper, we extend an existing compartmental model for an interaction

between plant hosts and soil-borne pathogens [13] and use optimal control theory [18, 19, 20, 21, 22] to evaluate how the deployment of an antagonist affects key epidemiological quantities such as the rates of primary and secondary infection. We also consider the minimization of the size of infected plant population, of the amount of inoculum as well as of the total cost of biological control.

The remaining part of paper is organized as follows. In Section 2, we consider the local and global stability properties of the compartmental model. Section 3 is concerned with the corresponding optimal control problem. Section 4 includes a numerical example which validates our previous analysis. Finally, the basic outcomes of our analytical findings and their ecological interpretations are drawn in Section 5 on the basis of the theoretical and numerical frameworks developed in the previous sections.

2. The model and its dynamical analysis

2.1. The model

In this section, we establish local and global stability results for an epidemiological model of plant-pathogen-antagonist interaction within a deterministic environment, giving first an adimensalization of the model. We assume that the population of plant hosts is divided into two classes, susceptible (S) and infected (I). During the course of each season, the primary infection is triggered by the inoculum. The plants which are infected in the primary infection wave then become an additional source of infection as soon as the fungus spreads to susceptible plants, which starts the secondary infection wave. In this regard, we denote by $X = X(t)$ the density of external pathogen inoculum at time t. The antagonist agent variables, denoted by u_1 and u_2, have the potential to decrease the rates of primary infection and secondary infection, respectively. By the above considerations, we may introduce the model of concern in this paper in the

following form

$$\frac{dS}{dt} = \eta(K-(S+I)) - \left(\frac{\beta_p X}{1+u_1} + \frac{\beta_s I}{1+u_2}\right) S, \tag{1a}$$

$$\frac{dI}{dt} = \left(\frac{\beta_p X}{1+u_1} + \frac{\beta_s I}{1+u_2}\right) S - \mu I, \tag{1b}$$

$$\frac{dX}{dt} = \nu I - \gamma X. \tag{1c}$$

In the above model, the growth of the susceptible host class is linear, governed by the rate η and both susceptible and infective plants contribute towards the carrying capacity K. The quantities β_p, β_s are the per capita rate of the primary and secondary infection, respectively, μ denotes the removal rate of infected hosts, γ is the decay rate of external inoculum and ν is the release rate of inoculum by infectious hosts.

In order to simplify the dynamical analysis of the model, we now introduce the dimensionless variables and parameters $\hat{S} = SK^{-1}$, $\hat{I} = IK^{-1}$, $\hat{X} = \eta X \nu^{-1} K^{-1}$, $\hat{t} = \eta t$, $\hat{\beta}_p = \beta_p \nu K \eta^{-2}$, $\hat{\beta}_s = \beta_s K \eta^{-1}$, $\hat{\mu} = \mu\eta^{-1}$, $\hat{\gamma} = \gamma\eta^{-1}$. With these notations, the system (1a)–(1c) is transformed into the following adimensional one

$$\frac{d\hat{S}}{d\hat{t}} = 1-(\hat{S}+\hat{I}) - \left(\frac{\hat{\beta}_p \hat{X}}{1+u_1} + \frac{\hat{\beta}_s \hat{I}}{1+u_2}\right)\hat{S}, \tag{2a}$$

$$\frac{d\hat{I}}{d\hat{t}} = \left(\frac{\hat{\beta}_p \hat{X}}{1+u_1} + \frac{\hat{\beta}_s \hat{I}}{1+u_2}\right)\hat{S} - \hat{\mu}\hat{I}, \tag{2b}$$

$$\frac{d\hat{X}}{d\hat{t}} = \hat{I} - \hat{\gamma}\hat{X}. \tag{2c}$$

By denoting $\hat{N} = \hat{S} + \hat{I}$ and summing up the first two equations, it is seen that the system (2a)–(2c) is equivalent to the following one

$$\frac{d\hat{N}}{d\hat{t}} = 1 - \hat{N} - \hat{\mu}\hat{I}, \tag{3a}$$

$$\frac{d\hat{I}}{d\hat{t}} = \left(\frac{\hat{\beta}_p \hat{X}}{1+u_1} + \frac{\hat{\beta}_s \hat{I}}{1+u_2}\right)(\hat{N}-\hat{I}) - \hat{\mu}\hat{I}, \tag{3b}$$

$$\frac{d\hat{X}}{d\hat{t}} = \hat{I} - \hat{\gamma}\hat{X}. \tag{3c}$$

One notes that the system (2a)–(2c) has two nonnegative equilibria: the trivial equilibrium $E_1(1,0,0)$ and the positive equilibrium $E_2(\hat{S}^*, \hat{I}^*, \hat{X}^*)$. Biologically speaking, E_1 corresponds to

the ideal (from a grower's viewpoint) pathogen-free state, while E_2 corresponds to the stationary state of coexistence between pathogens and hosts. Here,

$$\hat{S}^* = \frac{\hat{\gamma}\hat{\mu}(1+u_1)(1+u_2)}{\hat{\beta}_p(1+u_2)+\hat{\gamma}\hat{\beta}_s(1+u_1)},$$
$$\hat{I}^* = \frac{1}{1+\hat{\mu}}\left(1-\frac{\hat{\gamma}\hat{\mu}(1+u_1)(1+u_2)}{\hat{\beta}_p(1+u_2)+\hat{\gamma}\hat{\beta}_s(1+u_1)}\right),$$
$$\hat{X}^* = \frac{1}{(1+\hat{\mu})\hat{\gamma}}\left(1-\frac{\hat{\gamma}\hat{\mu}(1+u_1)(1+u_2)}{\hat{\beta}_p(1+u_2)+\hat{\gamma}\hat{\beta}_s(1+u_1)}\right).$$

Let us observe that the basic reproductive number of plant pathogens R_0 is given by

$$R_0 \doteq \frac{1}{\hat{\mu}}\left(\frac{\hat{\beta}_p}{\hat{\gamma}(1+u_1)}+\frac{\hat{\beta}_s}{1+u_2}\right) = \frac{\hat{\beta}_p(1+u_2)+\hat{\gamma}\hat{\beta}_s(1+u_1)}{\hat{\gamma}\hat{\mu}(1+u_1)(1+u_2)}.$$

With this notation, the coordinates of the positive equilibrium are given by

$$\hat{S}^* = \frac{1}{R_0}, \quad \hat{I}^* = \frac{1}{1+\hat{\mu}}\left(1-\frac{1}{R_0}\right), \quad \hat{X}^* = \frac{1}{(1+\hat{\mu})\gamma}\left(1-\frac{1}{R_0}\right).$$

That is, while E_1 exists regardless of the value of the basic reproductive number R_0, E_2 exists if and only if $R_0 \geq 1$ and is indeed a positive equilibrium if and only if $R_0 > 1$.

2.2. The local stability of the equilibria

To discuss the local stability of the trivial equilibrium $E_1(1,0,0)$ of the system (2a)–(2c), we observe that the Jacobian matrix of the system (2a)–(2c) at E_1 is given by

$$J = \begin{pmatrix} -1 & -1-\frac{\hat{\beta}_s}{1+u_2} & -\frac{\hat{\beta}_p}{1+u_1} \\ 0 & \frac{\hat{\beta}_s}{1+u_2}-\hat{\mu} & \frac{\hat{\beta}_p}{1+u_1} \\ 0 & 1 & -\hat{\gamma} \end{pmatrix}, \tag{4}$$

the associated characteristic equation being

$$(\lambda+1)\left[\lambda^2+\left(\hat{\gamma}-\frac{\hat{\beta}_s}{1+u_2}+\hat{\mu}\right)\lambda+\hat{\mu}\hat{\gamma}-\frac{\hat{\gamma}\hat{\beta}_s}{1+u_2}-\frac{\hat{\beta}_p}{1+u_1}\right]=0.$$

Clearly, -1 is one of the eigenvalues. From the Routh-Hurwitz criterion, we know that the other two eigenvalues have negative real parts if and only if

$$\hat{\gamma}-\frac{\hat{\beta}_s}{1+u_2}+\hat{\mu}>0, \quad \hat{\mu}\hat{\gamma}-\frac{\hat{\gamma}\hat{\beta}_s}{1+u_2}-\frac{\hat{\beta}_p}{1+u_1}>0,$$

which altogether imply that

$$\frac{1}{\hat{\mu}}\left(\frac{\hat{\beta}_p}{\hat{\gamma}(1+u_1)}+\frac{\hat{\beta}_s}{1+u_2}\right)<1.$$

Consequently, a necessary condition for the local stability of E_1 is $R_0<1$, while the stability of E_1 remains undetermined if $R_0=1$. Having obtained these hints, we shall further investigate the stability of E_1 in the next subsection.

We now turn our attention to the stability of the positive equilibrium $E_2(\hat{S}^*,\hat{I}^*,\hat{X}^*)$. The Jacobian matrix of the system (2a)–(2c) at E_2 is given by

$$J=\begin{pmatrix} -1-A & -1-B & -C \\ A & B-\hat{\mu} & C \\ 0 & 1 & -\hat{\gamma} \end{pmatrix} \tag{5}$$

with

$$A=\frac{\hat{\mu}}{1+\hat{\mu}}(R_0-1),\quad B=\frac{\hat{\beta}_s}{R_0(1+u_2)},\quad C=\frac{\hat{\beta}_p}{R_0(1+u_1)}.$$

The associated characteristic equation can be expressed as

$$\lambda^3+a_1\lambda^2+a_2\lambda+a_3=0,$$

in which

$$\begin{aligned} a_1 &= A+1-B+\hat{\mu}+\hat{\gamma}=\frac{\hat{\mu}}{1+\hat{\mu}}(R_0-1)+\hat{\mu}-\frac{\hat{\beta}_s}{R_0(1+u_2)}+\hat{\gamma}+1,\\ a_2 &= \hat{\gamma}(A+1-B+\hat{\mu})+A\hat{\mu}-B+\hat{\mu}+A-C\\ &= \frac{\hat{\mu}\hat{\gamma}}{1+\hat{\gamma}}(R_0-1)+\hat{\mu}-\frac{\hat{\beta}_s}{R_0(1+u_2)}+\hat{\gamma}+\hat{\mu}(R_0-1),\\ a_3 &= A\hat{\mu}\hat{\gamma}-B\hat{\gamma}+\hat{\mu}\hat{\gamma}+A\hat{\gamma}-C=\hat{\gamma}\hat{\mu}(R_0-1). \end{aligned}$$

It then follows from the Routh-Hurwitz criterion that all three eigenvalues have negative real parts if and only if $a_3>0$, $a_1>0$, $a_1a_2-a_3>0$. Since $\hat{\mu}-\frac{\hat{\beta}_s}{R_0(1+u_2)}=\frac{\hat{\beta}_p}{R_0\hat{\gamma}(1+u_1)}$, it is obvious that $R_0>1$ is a sufficient rather than necessary condition for $a_1>0$ and $a_1a_2-a_3>0$, while it is both necessary and sufficient for $a_3>0$. From the above analysis, we then obtain the following result.

Theorem 2.1. *Suppose that $R_0 > 1$. Then the unique positive equilibrium E_2 of the system (2a)–(2c) is locally asymptotically stable.*

2.3. The global stability of the equilibria

From obvious biological considerations, we shall study the dynamics of the system (2a)–(2c) in the feasible domain

$$\Omega = \left\{ (\hat{S},\hat{I},\hat{X}) | \hat{S} > 0,\ \hat{I},\hat{X} \geq 0 \text{ and } \hat{S}+\hat{I} \leq 1, \hat{X} \leq \frac{1}{\hat{\gamma}} \right\}.$$

It can be easily verified that Ω is positively invariant with respect to the system (2a)–(2c). In the following we shall investigate the global stability of the trivial equilibrium E_1 in Ω.

Theorem 2.2. *Suppose that $R_0 \leq 1$. Then the trivial equilibrium E_1 of the system (2a)–(2c) is globally asymptotically stable in Ω.*

Proof. Let us consider the following Lyapunov functional

$$U_1(\hat{S},\hat{I},\hat{X}) = \int_1^{\hat{S}} \left(1 - \frac{1}{\tau}\right) d\tau + \hat{I} + \left(\hat{\mu} - \frac{\hat{\beta}_s}{1+u_2}\right)\hat{X},$$

and observe that $R_0 \leq 1$ implies that $\hat{\mu} > \frac{\hat{\beta}_s}{1+u_2}$. Computing the time derivative of U_1 along the solutions of the system (2a)–(2c), one finds that

$$\begin{aligned}
\frac{dU_1}{d\hat{t}} &= \left(1 - \frac{1}{\hat{S}}\right)\left[1 - (\hat{S}+\hat{I}) - \left(\frac{\hat{\beta}_p\hat{X}}{1+u_1} + \frac{\hat{\beta}_s\hat{I}}{1+u_2}\right)\hat{S}\right] + \left(\frac{\hat{\beta}_p\hat{X}}{1+u_1} + \frac{\hat{\beta}_s\hat{I}}{1+u_2}\right)\hat{S} - \hat{\mu}\hat{I} \\
&\quad + \left(\hat{\mu} - \frac{\hat{\beta}_s}{1+u_2}\right)(\hat{I} - \hat{\gamma}\hat{X}) \\
&= \left(1 - \frac{1}{\hat{S}}\right)\left[1 - (\hat{S}+\hat{I})\right] + \frac{\hat{\beta}_p\hat{X}}{1+u_1} - \hat{\mu}\hat{\gamma}\hat{X} + \frac{\hat{\beta}_s\hat{\gamma}\hat{X}}{1+u_2} \\
&= \left(1 - \frac{1}{\hat{S}}\right)[1 - (\hat{S}+\hat{I})] + \hat{\mu}\hat{\gamma}\hat{X}\left[\left(\frac{\hat{\beta}_p}{(1+u_1)\hat{\gamma}} + \frac{\hat{\beta}_s}{1+u_2}\right)\frac{1}{\hat{\mu}} - 1\right] \\
&= \left(1 - \frac{1}{\hat{S}}\right)\left[1 - (\hat{S}+\hat{I})\right] + \hat{\mu}\hat{\gamma}\hat{X}[R_0 - 1].
\end{aligned}$$

Since $(1 - \frac{1}{\hat{S}})[1 - (\hat{S}+\hat{I})] \leq 0$, we obtain that if $R_0 \leq 1$, then $\frac{dU_1}{d\hat{t}} \leq 0$, with equality if and only if $R_0 = 1$ and $\hat{S}+\hat{I} = 1$. The use of LaSalle's Invariance Theorem then concludes the proof.

To discuss the global stability of the unique positive equilibrium E_2, we shall again consider Ω as the feasible domain. We then start by indicating a brief outline of the geometrical approach introduced by Li and Muldowney in [23], which will be of use in what follows.

Let $x \mapsto f(x) \in \mathbb{R}^n$ be a C^1 function defined on an open set $D \subset \mathbb{R}^n$. Consider the system

$$x' = f(x) \tag{6}$$

and denote by $x(t,x_0)$ the solution of (6) with initial data x_0, that is, $x(0,x_0) = x_0$. We make the following two assumptions:

(H_1)**:** There exists a compact absorbing set $K \subset D$;

(H_2)**:** The system (6) has a unique equilibrium $\bar{x}$ in D.

The system (6) is said to be uniformly persistent if there exists a constant $c > 0$ such that for any solution $x(t,x_0)$ of (6) one has

$$\liminf_{t\to\infty} x_1(t) \geq c, \quad \liminf_{t\to\infty} x_2(t) \geq c, \quad \ldots, \quad \liminf_{t\to\infty} x_n(t) \geq c.$$

If the system (6) is uniformly persistent and D is bounded, then the system (6) admits a compact absorbing set.

The equilibrium $\bar{x}$ is said to be globally stable in D if it is locally stable and all trajectories in D are convergent to $\bar{x}$. For $n \geq 2$, by a Bendixson criterion we mean a condition satisfied by f which precludes the existence of non-constant periodic solutions of (6). It is known that the classical Bendixson's condition div $f(x) < 0$ for $n = 2$ is robust under C^1 local perturbations of f. For higher dimensional systems, the C^1 robustness properties are discussed in [23]. A point $x_0 \in D$ is said to be a wandering point for (6) if there exists a neighborhood U of x_0 and $T > 0$ such that $U \cap x(t,U)$ is empty for all $t > T$. Thus, for instance, all equilibria and limit points are non-wandering.

The following global stability principle is established in Li and Muldowney in [23] for autonomous systems in any finite dimension.

Theorem 2.3. *Suppose that (H_1) and (H_2) hold. Assume that (6) satisfied a Bendixson criterion that is robust under C^1 local perturbations of f at all non-equilibrium non-wandering points for (6). Then $\bar{x}$ is globally stable in D provided that it is stable.*

The following Bendixson criterion is also given in [23]. Let $N = \begin{pmatrix} n \\ 2 \end{pmatrix}$ and $x \mapsto P(x)$ be a $N \times N$ matrix-valued function that is C^1 for $x \in D$. Assume that $P^{-1}(x)$ exists and is continuous for $x \in K$, the compact absorbing set, and define

$$\bar{q}_2 = \limsup_{t\to\infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^t \mu(B(x(s,x_0)))ds, \tag{7}$$

where

$$B = P_f P^{-1} + P \frac{\partial f^{[2]}}{\partial x} P^{-1}. \tag{8}$$

In the above, the matrix P_f is obtained by replacing each entry of P by its derivative in the direction of f and $\mu(B)$ is the Lozinskiĭ measure of B with respect to a vector norm $|\cdot|$ in $\mathbb{R}^N$,

$$\mu(B) = \lim_{h\to 0^+} \frac{|I + hB| - 1}{h}.$$

Let us also recall that the second additive compound of a matrix $A = (a_{ij})_{1\le i,j\le 3}$ is

$$A^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}.$$

It is shown in [23] that, if D is simply connected, then the condition $\bar{q}_2 < 0$ rules out the presence of any orbit that gives rise to a simple closed rectifiable curve that is invariant, such as periodic orbits, homoclinic orbits, and heteroclinic cycles. Moreover, this condition is robust under C^1 local perturbations of f near any non-equilibrium point that is non-wandering. In particular, the following global-stability result is proved in Li and Muldowney [23].

Theorem 2.4. *Assume that D is simply connected and that the assumptions (H_1) and (H_2) hold. Then the unique positive equilibrium $\bar{x}$ of (6) is globally stable in D if $\bar{q}_2 < 0$.*

To prove the existence of a compact absorbing set K for the system (2a)–(2c), we start by proving the uniform persistence of (6).

Theorem 2.5. *If $R_0 > 1$, then (6) is persistent.*

Proof. First, we prove that E_1 cannot be the ω-limit point of any orbit starting in the interior of Ω. To this purpose, let us introduce

$$\alpha = \min\left\{R_0 - 1, \frac{\hat{\beta}_p}{\hat{\mu}\hat{\gamma}(1+u_1)}\right\}$$

take $\varepsilon \in (0, \alpha)$ and define

$$L(\hat{S}, \hat{I}, \hat{X}) = \hat{I} + \left(\frac{\hat{\beta}_p}{\hat{\gamma}(1+u_1)} - \varepsilon\hat{\mu}\right)\hat{X}.$$

Then $L \geq 0$ and the time derivative of L along the solutions of the system (2a)–(2c) is given by

$$\begin{aligned}
\frac{dL}{d\hat{t}} &= \left(\frac{\hat{\beta}_p\hat{X}}{1+u_1} + \frac{\hat{\beta}_s\hat{I}}{1+u_2}\right)\hat{S} - \hat{\mu}\hat{I} + \left(\frac{\hat{\beta}_p}{\hat{\gamma}(1+u_1)} - \varepsilon\hat{\mu}\right)(\hat{I} - \hat{\gamma}\hat{X}) \\
&= \hat{X}\left(\frac{\hat{\beta}_p}{1+u_1}(\hat{S}-1) + \varepsilon\hat{\mu}\hat{\gamma}\right) + \hat{\mu}\hat{I}\left[\left(\frac{\hat{\beta}_p}{\hat{\gamma}(1+u_1)} + \frac{\hat{\beta}_s\hat{S}}{1+u_2}\right)\cdot\frac{1}{\hat{\mu}} - (1+\varepsilon)\right] \\
&= \hat{X}\left(\varepsilon\hat{\mu}\hat{\gamma} - \frac{\hat{\beta}_p}{1+u_1}(1-\hat{S})\right) + \hat{\mu}\hat{I}\left(R_0 - (1+\varepsilon) - \frac{\hat{\beta}_s\hat{S}}{1+u_2}(1-S)\right).
\end{aligned}$$

Consequently, $\frac{dL}{d\hat{t}} > 0$ for S close enough to 1 and E_1 cannot be the ω-limit point of any orbit starting in the interior of Ω. Since the system (2a)–(2c) does not have other ω-limit points on the boundary of Ω, this implies that the system (2a)–(2c) is uniformly persistent. This completes the proof.

Since Ω is bounded, it is then seen that the system (2a)–(2c) admits a compact absorbing set K. In the following, from the above considerations, we shall obtain a sufficient condition for the global stability of the steady state E_2 in Ω.

Theorem 2.6. *If the following three conditions are satisfied*

(i): $R_0 > 1$;
(ii): $1 + \hat{\mu} > \frac{2\hat{\beta}_p}{1+u_1} + \frac{\hat{\beta}_s}{1+u_2}$;
(iii): $\frac{\hat{\mu}+\hat{\gamma}}{2} > 1 + \frac{\hat{\beta}_s}{1+u_2}$,

then the equilibrium E_2 of the system (2a)–(2c) is globally asymptotically stable in the interior of Ω.

Proof. Let J be the Jacobian matrix J of the system (2a)–(2c) at E_2. Its second additive compound matrix $J^{[2]}$ is as given below

$$J^{[2]} = \begin{pmatrix} -1-\frac{\hat{\beta}_p\hat{X}}{1+u_1}-\frac{\hat{\beta}_s\hat{I}}{1+u_2}+\frac{\hat{\beta}_s\hat{S}}{1+u_2}-\hat{\mu} & \frac{\hat{\beta}_p\hat{S}}{1+u_1} & \frac{\hat{\beta}_p\hat{S}}{1+u_1} \\ 1 & -1-\frac{\hat{\beta}_p\hat{X}}{1+u_1}-\frac{\hat{\beta}_s\hat{I}}{1+u_2}-\hat{\gamma} & -1-\frac{\hat{\beta}_s\hat{S}}{1+u_2} \\ 0 & \frac{\hat{\beta}_p\hat{X}}{1+u_1}+\frac{\hat{\beta}_s\hat{I}}{1+u_2} & \frac{\hat{\beta}_s\hat{S}}{1+u_2}-\hat{\mu}-\hat{\gamma} \end{pmatrix}.$$

Let us also define

$$P(\hat{S},\hat{I},\hat{X}) = \mathrm{diag}(\frac{\hat{S}}{\hat{I}},\frac{\hat{S}}{\hat{I}},\frac{\hat{S}}{\hat{I}}).$$

Then

$$P_f P^{-1} = \mathrm{diag}(\frac{\hat{S}'}{\hat{S}}-\frac{\hat{I}'}{\hat{I}},\frac{\hat{S}'}{\hat{S}}-\frac{\hat{I}'}{\hat{I}},\frac{\hat{S}'}{\hat{S}}-\frac{\hat{I}'}{\hat{I}})$$

and the matrix

$$B = P_f P^{-1} + PJ^{[2]}P^{-1}$$

can be written in block form

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

where

$$B_{11} = -1-\frac{\hat{\beta}_p\hat{X}}{1+u_1}-\frac{\hat{\beta}_s\hat{I}}{1+u_2}+\frac{\hat{\beta}_s\hat{S}}{1+u_2}-\hat{\mu}+\frac{\hat{S}'}{\hat{S}}-\frac{\hat{I}'}{\hat{I}}$$

$$B_{12} = \begin{pmatrix} \frac{\hat{\beta}_p\hat{S}}{1+u_1} & \frac{\hat{\beta}_p\hat{S}}{1+u_1} \end{pmatrix}$$

$$B_{21} = \begin{pmatrix} 1 \\ 0 \end{pmatrix}$$

$$B_{22} = \begin{pmatrix} -1-\frac{\hat{\beta}_p\hat{X}}{1+u_1}-\frac{\hat{\beta}_s\hat{I}}{1+u_2}-\hat{\gamma}+\frac{\hat{S}'}{\hat{S}}-\frac{\hat{I}'}{\hat{I}} & -1-\frac{\hat{\beta}_s\hat{S}}{1+u_2} \\ \frac{\hat{\beta}_p\hat{X}}{1+u_1}+\frac{\hat{\beta}_s\hat{I}}{1+u_2} & \frac{\hat{\beta}_s\hat{S}}{1+u_2}-\hat{\mu}-\hat{\gamma}+\frac{\hat{S}'}{\hat{S}}-\frac{\hat{I}'}{\hat{I}} \end{pmatrix},$$

Let us consider on $R^3 \cong R\begin{pmatrix} 3 \\ 2 \end{pmatrix}$ the norm given by

$$|(\mu,\nu,\omega)| = \max\{|\mu|,|\nu|+|\omega|\}.$$

We then estimate the Lozinskiĭ measure μ with respect to the the vector norm $|\cdot|$ as

$$\mu(B) \leq \sup\{g_1,g_2\},$$

where

$$g_1 = \mu_1(B_{11}) + \|B_{12}\|,\ g_2 = \|B_{21}\| + \mu_1(B_{22}).$$

Here, $\mu_1(B_{11})$, $\mu_1(B_{22})$ are the Lozinskiĭ measures of B_{11} and B_{22} with respect to the l_1 norm, and $\|B_{12}\|$, $\|B_{21}\|$ are matrix norms with respect to the l_1 vector norm. More specifically,

$$\mu_1(B_{11}) = -1 - \frac{\hat{\beta}_p \hat{X}}{1+u_1} - \frac{\hat{\beta}_s \hat{I}}{1+u_2} + \frac{\hat{\beta}_s \hat{S}}{1+u_2} - \hat{\mu} + \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}}$$

and

$$\|B_{12}\| = \frac{2\hat{\beta}_p \hat{S}}{1+u_1}, \quad \|B_{21}\| = 1.$$

To calculate $\mu_1(B_{22})$, add the absolute value of the off-diagonal elements to the diagonal one in each column of B_{22}, and then take the maximum of two sums. One obtains

$$\mu_1(B_{22}) = \max\big\{-1-\hat{\gamma}+\frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}}, 1 + \frac{2\hat{\beta}_s \hat{S}}{1+u_2} - \hat{\mu} - \hat{\gamma} + \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}}\big\},$$

and consequently

$$\begin{aligned} g_1 &= -1 - \frac{\hat{\beta}_p \hat{X}}{1+u_1} - \frac{\hat{\beta}_s \hat{I}}{1+u_2} + \frac{\hat{\beta}_s \hat{S}}{1+u_2} - \hat{\mu} + \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}} + \frac{2\hat{\beta}_p \hat{S}}{1+u_1} \\ &\le \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}} + \frac{2\hat{\beta}_p \hat{S}}{1+u_1} + \frac{\hat{\beta}_s \hat{S}}{1+u_2} - (1+\hat{\mu}) \\ g_2 &= 1 + \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}} - \hat{\gamma} + \max\big\{-1, 1 + \frac{2\hat{\beta}_s \hat{S}}{1+u_2} - \hat{\mu}\big\} \\ &= \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}} - \hat{\gamma} + \max\big\{0, 2 + \frac{2\hat{\beta}_s \hat{S}}{1+u_2} - \hat{\mu}\big\}. \end{aligned}$$

It then follows that

$$\mu(B) \le \frac{\hat{S}'}{\hat{S}} - \frac{\hat{I}'}{\hat{I}} - \bar{b},$$

where

$$\bar{b} = \min\big\{1 + \hat{\mu} - (\frac{2\hat{\beta}_p}{1+u_1} + \frac{\hat{\beta}_s}{1+u_2}), \hat{\mu} + \hat{\gamma} - 2(1 + \frac{\hat{\beta}_s}{1+u_2})\big\} > 0.$$

Along each solution $(\hat{S}, \hat{I}, \hat{X})$ of the system (2a)–(2c) with $\big(\hat{S}(0), \hat{I}(0), \hat{X}(0)\big) \in K$, where K is the compact absorbing set, we have

$$\frac{1}{t}\int_0^t \mu(B)ds = \frac{1}{t}\ln\frac{S(t)}{S(0)} - \frac{1}{t}\ln\frac{I(t)}{I(0)} - \bar{b},$$

which implies that $\bar{q_2} \le -\frac{\bar{b}}{2} < 0$. This completes the proof.

3. The optimal control problem

In this section, we discuss the optimal control of the system (2a)–(2c). In order to minimize the density of infected hosts and of the soil-borne inoculum, we then consider u_1 and u_2 as two control variables of the antagonist. Our cost functional, which we attempt to minimize, is

$$J[u_1,u_2]=\frac{1}{2}\int_0^T [c_1u_1^2+c_2u_2^2+\hat{I}^2+\hat{X}^2]d\hat{t},$$

where the parameters u_1 and u_2 are assumed to be positive and the final time T is fixed.

The associated Hamiltonian function is given by

$$\begin{aligned} H &= \frac{1}{2}(c_1u_1^2+c_2u_2^2+\hat{I}^2+\hat{X}^2)+\lambda_1[1-(\hat{S}+\hat{I})-(\frac{\hat{\beta}_p\hat{X}}{1+u_1}+\frac{\hat{\beta}_s\hat{I}}{1+u_2})\hat{S}] \\ &\quad +\lambda_2[(\frac{\hat{\beta}_p\hat{X}}{1+u_1}+\frac{\hat{\beta}_s\hat{I}}{1+u_2})\hat{S}-\hat{\mu}\hat{I}]+\lambda_3[\hat{I}-\hat{\gamma}\hat{X}]. \end{aligned} \tag{9}$$

In (9), $\lambda_i, i=1,2,3$ are the adjoint variables. By using Pontryagin's Minimum Principle, we obtain the following equations

$$H\Big(\big(\hat{S}(\hat{t}),\hat{I}(\hat{t}),\hat{X}(\hat{t})\big),u^*(\hat{t}),\lambda(\hat{t}),\hat{t}\Big)=\min_{u\in U}H\Big(\big(\hat{S}(\hat{t}),\hat{I}(\hat{t}),\hat{X}(\hat{t})\big),u(\hat{t}),\lambda(\hat{t}),\hat{t}\Big)$$

$$\begin{aligned} \frac{d\lambda_1}{d\hat{t}} &= -\frac{\partial H}{\partial \hat{S}} \\ \frac{d\lambda_2}{d\hat{t}} &= -\frac{\partial H}{\partial \hat{I}} \\ \frac{d\lambda_3}{d\hat{t}} &= -\frac{\partial H}{\partial \hat{X}}. \end{aligned}$$

We then deduce that

$$\begin{aligned} \frac{d\lambda_1}{d\hat{t}} &= \lambda_1+(\lambda_1-\lambda_2)(\frac{\hat{\beta}_p\hat{X}}{1+u_1}+\frac{\hat{\beta}_s\hat{I}}{1+u_2}) \\ \frac{d\lambda_2}{d\hat{t}} &= -\hat{I}+\lambda_1+(\lambda_1-\lambda_2)\frac{\hat{\beta}_s\hat{S}}{1+u_2}+\lambda_2\hat{\mu}-\lambda_3 \\ \frac{d\lambda_3}{d\hat{t}} &= -\hat{X}+(\lambda_1-\lambda_2)\frac{\hat{\beta}_p\hat{S}}{1+u_1}+\lambda_3\hat{\gamma}. \end{aligned}$$

From Pontryagin's Minimum Principle, we may find $\frac{\partial H}{\partial u_i}$ and solve for $u_i^*, i = 1, 2$, by setting the partial derivatives of H equal to zero. Thus, from

$$\frac{\partial H}{\partial u_1^*} = c_1 u_1^* + (\lambda_1 - \lambda_2)\frac{\hat{\beta}_p \hat{S}\hat{X}}{(1+u_1^*)^2} = 0,$$
$$\frac{\partial H}{\partial u_2^*} = c_2 u_2^* + (\lambda_1 - \lambda_2)\frac{\hat{\beta}_s \hat{S}\hat{I}}{(1+u_2^*)^2} = 0.$$

We obtain that

$$u_1^* = -\frac{2}{3} + \frac{[(8c_1 + 108B + 12\sqrt{3}\sqrt{B(4c_1+27B)})c_1^2]^{\frac{1}{3}}}{6c_1} + \frac{2c_1}{3[(8c_1 + 108B + 12\sqrt{3}\sqrt{B(4c_1+27B)})c_1^2]^{\frac{1}{3}}},$$
$$u_2^* = -\frac{2}{3} + \frac{[(8c_2 + 108C + 12\sqrt{3}\sqrt{C(4c_2+27C)})c_2^2]^{\frac{1}{3}}}{6c_2} + \frac{2c_2}{3[(8c_2 + 108C + 12\sqrt{3}\sqrt{C(4c_2+27C)})c_2^2]^{\frac{1}{3}}}$$

with

$$B = (\lambda_2 - \lambda_1)\hat{\beta}_p \hat{S}\hat{X} \quad C = (\lambda_2 - \lambda_1)\hat{\beta}_s \hat{S}\hat{I}.$$

Since the final values for the state variables are not pre-set, we use these conditions at the final time T

$$\lambda_i(T) = 0, i = 1, 2. \tag{10}$$

4. A numerical example

Our aim is to understand the effect of a combination between two control mechanisms, referring to both primary and secondary infections, upon the final outcome. As for the epidemiological and demographic parameters, we choose $\hat{\beta}_p = 0.5$, $\hat{\beta}_s = 0.375$, $\hat{\mu} = 0.25$ and $\hat{\gamma} = 0.8$ in [17]. The primary and secondary control costs are chosen as being $c_1 = 4$ and $c_2 = 1$, respectively. Also, the initial conditions for the state variables are given by $\hat{S}(0) = 0.15$, $\hat{I}(0) = 0.85$ and $\hat{X}(0) = 0.8$. Figure 1 illustrates the trajectories for the state variables under the optimal control values u_1^* and u_2^* shown in Figure 2. It is seen that the curves corresponding to the control mechanism u_1 and u_2, respectively, have a similar shape, due to similar infection functions

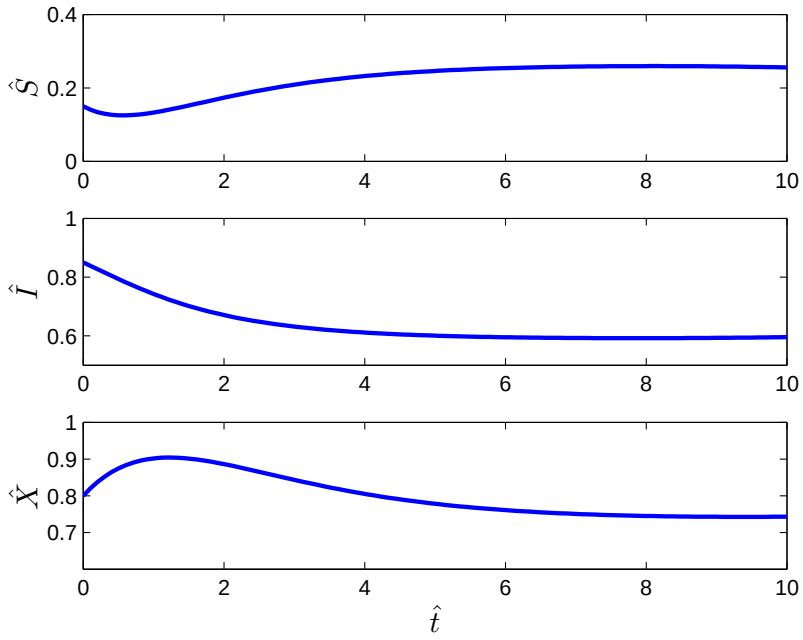

FIGURE 1. Optimal control trajectories for the state variables.

given in the system (2a)–(2c). In addition, it is obvious that the amount of u_1 is lower that the amount of u_2 due to the higher cost of the control mechanism for the primary infection.

4. Concluding remarks

The present paper attempts to formulate and study a differential model for the spread of a disease into a plant population assuming that the disease is subject to two control mechanisms, namely the control of the infection rate between susceptible hosts and soil-borne inoculum and the control of the infection rate between susceptible hosts and infected hosts.

We derive a basic reproduction number R_0 and find out that it determines the global dynamics of (2a)–(2c). If $R_0 > 1$, the unique plant endemic equilibrium E_2 is globally asymptotically stable in the interior of the feasible region provided that several auxiliary conditions are met, so that the plant disease persists at the endemic equilibrium level if it is initially present.

Also, we derive the optimal control strategies by using a quadratic functional J. Minimizing the cost, we obtain the optimal controls u_1^* and u_2^*, for which the size of the infected plant

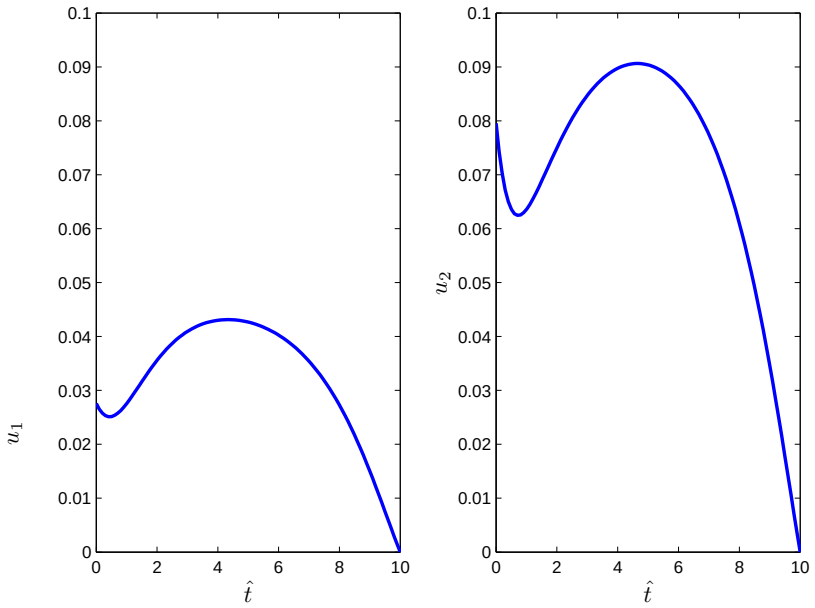

FIGURE 2. Optimal control trajectories for the control mechanisms.

population $\hat{I}$ and the amount of pathogen $\hat{X}$ are both minimized. Optimal control strategies corresponding to mathematical results show that the density of susceptible plants is incremented 2 times if we consider both primary and secondary infection control mechanisms. Hence, growers have a variety of options available for controlling plant disease epidemics, being able to diminish the reliance upon pesticide spraying.

Conflict of Interests

The authors declare that there is no conflict of interests.

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