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# Optimal strategies for biological control of soil-borne plant pathogens

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**Abstract:** In this paper we consider a mathematical model to assess the control strategies of the primary and the secondary infection of the plants, and then obtain the sufficient condition for the global asymptotic stability of the unique interior equilibrium point. Subsequently, we derive the optimal control strategies by using the functional $J$ in terms of quadratic forms and applying the Pontryagin's maximum principle. Finally, some numerical simulations about three control scenarios are assessed adjusting the control parameters are given to verify the mathematical conclusions and the corresponding biological significances are also discussed.

**Keywords:** soil-borne plant; local stability; global stability; Pontryagin maximum principle; optimal control

## 1 Introduction

Soil-borne pathogens belong to several different types: bacteria, fungi, viruses or nematodes [1]. And most plants are susceptible to some type of fungus [2]. In the case of soil-borne diseases, the pathogens can reside in the soil for long periods, and survive on plant residues or as resting organisms until root exudates reach them and allow them to grow. They then escape competition with other microorganisms by penetrating the roots. Most of them cannot be seen by the eye and go undetected, until the plant becomes ill. Plants infected by soil-borne pathogens suffer from root rots, stem, collar and crown rots, wilt diseases, stunting or seedling damping-off diseases. Hence, in both industrialized and developing countries, soil-borne pathogens can have devastating effects on field and greenhouse crops [3–5].

Plant disease is the result of a complex interaction between the host plant, the pathogen and the environmental conditions including those in the soil. Moreover, the plant-pathogen interactions have two special features which distinguish them from other epidemiological systems. First of all, there are two modes of transmission in the systems: (i) primary infection from contact between hosts and externally introduced inoculum. (ii) secondary infection from contact between susceptible and infected hosts in the current epidemic. Secondly, a response to simulation or inhibition in the production of susceptible tissue could be exhibited in many infected individuals [3].

Soil-borne pathogens are notoriously difficult to control. Commonly-used techniques for controlling plant diseases, including crop rotation and the application of fungicides have ecological, economic and environmental disadvantages. So experimentalists have become more interested in biological control [6], where an antagonist is used to reduce disease spread [7].

Recently, a number of authors have developed mathematical models of the specific host-pathogen-antagonist interaction, for instance, Kleczkowski, Bailey and Gilligan investigated the interactions between *Rhizoctonia solani* and *Trichoderma viride* in [8], and Gubbins and Gilligan analyzed the dynamics between *Sporidesmium sclerotivorum*, and its host, *Sclerotinia minor*, in a disturbed or closed environment in [9, 10]. Swinton and Anderson considered model frameworks for plant-pathogen interactions in [11]. In [3], Madden and van den Bosch assessed the threat of plant pathogens as biological weapons against annual crops. In 2008, van den Bosch *et al.* [12] investigated the basic reproduction number of plant pathogens by matrix approaches. Jeger *et al.* [13] proposed a model for biological control of foliar plant diseases. In 2011, Moore *et al.* [14] considered the spatiotemporal Model of plant virus transmission dynamics with seasonality and plant competition. Furthermore, Cunniffe and Gilligan considered how microbial antagonists affect the spread of pathogens through the plants in general in [7]. However, previous work has not targeted optimal biological control. In this work, by building mathematical models of biological control, we hope to answer the following two questions: (1) How can biological control be made more reliable? (2) How should biological control be deployed in order to minimize the values

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of the costs as well as the density of external pathogen inoculum and the infected plants? Here we extend an existing compartmental model of interaction between a plant host and a soil-borne pathogen [15] and use optimal control theory to evaluate the effectiveness of the application that antagonists block the primary infection and the secondary infection as well. We want to find the minimal effort necessary to reduce the infected plants and fungi inoculum, considering the cost of biological control application.

# 2 The basic model

Plants can only become infected by primary and secondary infections. Within each season primary infection is triggered by the primary inoculum. Plants infected by primary infection then become a source of infection as the fungus spreads to susceptible plants. For the sake of simplicity, we will make the following assumptions: (1) The population of plant hosts is divided into two classes, susceptible ($S$) and infected ($I$). There is no birth or death and the size of a plant population is fixed and denoted by $N$ over a given interval of time; (2) Plant disease with no latent period does not confer long–lasting immunity. Additionally we denote by $X$ the density of external pathogen inoculum at time $t$. The antagonist agents, denoted by $u_1$ and $u_2$, potentially decrease the rates of primary infection and secondary infection, respectively. The model is given by the following system of ODEs.

$$\begin{cases} \frac{dI}{dt} = (\frac{\beta_p X}{1+u_1(t)} + \frac{\beta_s I}{1+u_2(t)})(N-I) - \mu I, \\ \frac{dX}{dt} = \nu I - \gamma X. \end{cases} \tag{1}$$

Here all parameters in the model are constrained to be positive. $\beta_p$, $\beta_s$ are the per capita rate of the primary and secondly infection, respectively, $\mu$ denotes the removal rate of infected hosts, $\gamma$ is the decay rate of external inoculum, $\nu$ is the reproduction rate by releasing from infectious hosts. In order to fully characterize the dynamical behavior of the solutions and find the suitable control sets, we need further knowledge about the control mechanisms. In what follows, we consider fixed values for the control variables, that is, $u_1(t) \equiv u_1$, $u_2(t) \equiv u_2$. Then the system (1) has a trivial equilibrium $E_1(0,0)$ corresponding to the ideal inoculum-free state. The positive equilibrium $E_2(I^*, X^*)$ corresponds to the coexistence state of the plant disease and the plant hosts, in which $I^* = N(1-\frac{1}{R_0})$ and $X^* = \frac{\nu}{\gamma}N(1-\frac{1}{R_0})$, in which the basic reproductive number of the pathogen with the antagonist agents is

$$R_0 = \frac{1}{\mu}[\frac{N\beta_s}{1+u_2} + \frac{\nu N\beta_p}{\gamma(1+u_1)}]. \tag{2}$$

**Theorem 1** *For the system (1) the equilibrium point $E_1$ is globally asymptotic stability if $R_0 \leq 1$, the equilibrium point $E_2$ is globally asymptotically stable if $R_0 > 1$.*

**Proof.** To discuss the local stability of the model at two equilibrium points, we compute the the Jacobian matrix of the system (1) as follows

$$J = \begin{pmatrix} -\frac{X\beta_p}{1+u_1} + \frac{N\beta_s}{1+u_2} - \frac{2I\beta_s}{1+u_2} - \mu & \frac{N\beta_p}{1+u_1} - \frac{I\beta_p}{1+u_1} \\ \nu & -\gamma \end{pmatrix} \tag{3}$$

At the pathogen-free equilibrium point $E_1$, the eigenvalues $\lambda$ satisfies

$$\lambda^2 + (\gamma - \frac{N\beta_s}{1+u_2} + \mu)\lambda + \mu\gamma - \frac{\gamma N\beta_s}{1+u_2} - \frac{\nu N\beta_p}{1+u_1} = 0$$

According to the Routh-Hurwitz criteria, both two eigenvalues have negative real parts if and only if $\gamma - \frac{N\beta_s}{1+u_2} + \mu > 0$ and $\mu\gamma - \frac{\gamma N\beta_s}{1+u_2} - \frac{\nu N\beta_p}{1+u_1} > 0$. So if the equilibrium is locally asymptotically stable, the condition is reduced to $\mu - \frac{N\beta_s}{1+u_2} > \max\{-\gamma, \frac{\nu N\beta_p}{\gamma(1+u_1)}\}$. Since all parameters are positive, one notes that $\frac{1}{\mu}[\frac{N\beta_s}{1+u_2} + \frac{\nu N\beta_p}{\gamma(1+u_1)}] < 1$, which is equivalent to $R_0 < 1$. At pathogen-present equilibrium point $E_2$, the characteristic equation is given by

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

in which

$$a_1 = \gamma - \frac{N\beta_s}{1+u_2} + \mu + \frac{2\beta_s N}{1+u_2}(1-\frac{1}{R_0}) + \frac{\nu N\beta_p}{\gamma(1+u_1)}(1-\frac{1}{R_0}) = \gamma + \frac{\nu N\beta_p}{\gamma(1+u_1)} + \frac{\beta_s N}{1+u_2}(1-\frac{1}{R_0})$$

and

$$a_2 = -[\frac{\gamma N\beta_s}{1+u_2} - \gamma\mu - \frac{2\gamma\beta_s N}{1+u_2}(1-\frac{1}{R_0}) - \frac{\nu N\beta_p}{1+u_1}(1-\frac{1}{R_0}) + \frac{\nu N\beta_p}{1+u_1}\frac{1}{R_0}] = \mu\gamma(R_0-1).$$

According to the Routh-Hurwitz criteria, all eigenvalues have negative real parts if and only if $a_1 > 0$ and $a_2 > 0$. Moreover, it follows that that $R_0 > 1$ is a sufficient rather than necessary condition for $a_1 > 0$, and $R_0 > 1$ is a sufficient and necessary condition for $a_2 > 0$. Hence, the equilibrium point $E_2$ is locally asymptotically stable if $R_0 > 1$. In the following we will turn our attention to the global stability of the equilibrium points. First of all, we shall show the boundedness of the solutions of the system (1).

Since $\frac{dX}{dt} = \nu I - \gamma X < \nu N - \gamma X$, it follows that $0 < X < \frac{\nu N}{\gamma} + X(0)$ for $t \geq 0$. Also, one notes that

$$\begin{aligned} I' &< N[\frac{\beta_p(\frac{\nu N}{\gamma} + X(0))}{1+u_1} + \frac{\beta_s N}{1+u_2}] - [\frac{\beta_p(\frac{\nu N}{\gamma} + X(0))}{1+u_1} + \frac{\beta_s N}{1+u_2} + \mu]I, \end{aligned} \tag{4}$$

which implies $0 < I < \min\Big\{\frac{[\frac{\beta_p(\frac{\nu N}{\gamma}+X(0))}{1+u_1} + \frac{\beta_s N}{1+u_2}]}{[\frac{\beta_p(\frac{\nu N}{\gamma}+X(0))}{1+u_1} + \frac{\beta_s N}{1+u_2} + \mu]}N + I(0), N\Big\}$. Secondly, let us consider the following Lyapunov function $U_1(I,X) = I + \frac{NX\beta_p}{\gamma(1+u_1)}$. Now we compute the derivative of $U_1$ along the solutions of the system (1), one obtains that

$$\frac{dU_1}{dt} = I\mu(R_0-1) - \frac{XI\beta_p}{1+u_1} - \frac{I^2\beta_s}{1+u_2}$$

It follows from $R_0 < 1$ that $\frac{dU_1}{dt} < 0$, which implies the equilibrium point $E_1$ is globally asymptotically stable on $(0,\infty)^2$. Finally, let us consider the following Dulac function $h = I^\alpha X^\beta$, in which $\alpha$ and $\beta$ are undetermined coefficients. Let us denote by $f(I,X) = (\frac{\beta_p X}{1+u_1} + \frac{\beta_s I}{1+u_2})(N-I) - \mu I,\ g(I,X) = \nu I - \gamma X$. Then

$$\begin{aligned} D &= \frac{\partial h(I,X)f(I,X)}{\partial I} + \frac{\partial h(I,X)g(I,X)}{\partial X} \\ &= \alpha N\beta_p I^{\alpha-1}X^{\beta+1} - (\alpha+1)\beta_p I^\alpha X^{\beta+1} + (\alpha+1)\beta_s N I^\alpha X^\beta \\ &\quad -(\alpha+2)\beta_s I^{\alpha+1}X^\beta - (\alpha+1)\mu I^\alpha X^\beta + \beta\nu I^{\alpha+1}X^{\beta-1} \\ &\quad -(\beta+1)\gamma I^\alpha X^\beta \end{aligned} \tag{5}$$

We then need to find the appropriate $\alpha$ and $\beta$ such that $D$ is of constant sign on $(0,\infty)^2$. Assume that

$$[(\alpha+1)\beta_s N - (\alpha+1)\mu - (\beta+1)\gamma]I^\alpha X^\beta = 0.$$

We then get that

$$\frac{\alpha+1}{\beta+1} = \frac{\gamma}{\beta_s N - \mu}.$$

Without loss of generality, we assume that $\beta_s N - \mu > 0$. It makes sense that we can choose a suitable value of $\alpha$ such that

$$\frac{\alpha N}{I} - (\alpha+1) > 0.$$

Subsequently, we obtain that

$$\alpha N\beta_p I^{\alpha-1}X^{\beta+1} - (\alpha+1)\beta_p I^\alpha X^{\beta+1} = \beta_p I^\alpha X^{\beta+1}[\frac{\alpha N}{I} - (\alpha+1)] > 0.$$

Also, we choose a $\beta$ such that $\beta\nu - (\alpha+2)\beta_s X > 0$, which implies $\beta > \frac{\beta_s X}{\nu}[\frac{\gamma\beta+\gamma+\beta_s N-\mu}{\beta_s N-\mu}]$. One notes that

$$\beta > \frac{\gamma\beta_s X + \beta_s^2 XN - \mu\beta_s X}{\nu\beta_s N - \nu\mu - \gamma\beta_s X} \text{ for } N \text{ large enough.}$$

So

$$-(\alpha+2)\beta_s I^{\alpha+1}X^\beta + \beta\nu I^{\alpha+1}X^{\beta-1} = I^{\alpha+1}X^{\beta-1}[\beta\nu - (\alpha+2)\beta_s X] > 0. \tag{6}$$

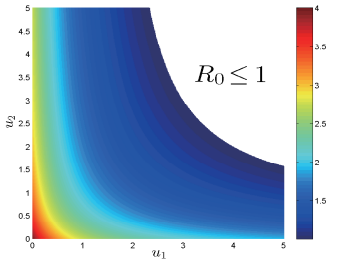


Figure 1: The relationship between $u_1$ and $u_2$ for $R_0 > 1$. The white region indicates $R_0 \leq 1$. Also, the colorbar indicates different values of $R_0$.

Hence we choose

$$(\alpha, \beta) \in \{(x, y) | x = \frac{\gamma(y+1)}{\beta_s N - \mu} + 1, y > \frac{\gamma\beta_s(\frac{\nu N}{\gamma} + X(0)) + \beta_s^2(\frac{\nu N}{\gamma} + X(0))N}{\nu\beta_s N - \nu\mu}\}.$$

Then, according to Dulac's criterion, we thereby construct a Dulac function $h = I^\alpha X^\beta$ to make $D > 0$. Hence, the system (1) has no closed orbit contained in the first quadrant together with the ultimate boundedness of the system (1), which indicates that the equilibrium point $E_2$ is globally asymptotically stable on $(0, \infty)^2$. The proof is completed. ■

**Remark 2** *In the real world, plants are grown in the area that pathogen inoculum is ubiquitous, which means that growers could not kill all pathogen inocula. According to Theorem 1, it is interesting to notice that, from the economic view of point, $R_0 > 1$ indicates ancillary constraints, that is, the amount of available antagonist might be limited as shown in Figure 1.*

# 3 The optimal control problem

In this section, we formulate the optimal control problem applied to the system (1). Our purpose is to minimize the density of infected hosts and pathogen inoculum. For this end we consider as control variables: (1) The primary infection control mechanism denoted by $u_1(t)$; (2) The secondary infection control mechanism denoted by $u_2(t)$. Hence, we consider the following performance index $J[u_1, u_2] = \frac{1}{2}\int_0^T [c_1 u_1^2 + c_2 u_2^2 + I^2 + X^2]dt$, where $c_1$ and $c_2$ are weight constants of prevention of primary and secondary contacts, respectively. The costs associated with prevention of the primary and secondary infections are described in the terms $c_1 u_1^2$ and $c_2 u_2^2$, respectively. In the control problem, we assume fixed final time, and free dynamical variables at this time. Also, a quadratic functional cost is used due to the reason that the performance index should be a nonlinear function. Here the optimization approach we adopt is based upon the Hamiltonian method [16–18]. To this end, we note that our control problem associated Hamiltonian function is given by

$$\begin{aligned} H &= \frac{1}{2}(c_1 u_1^2 + c_2 u_2^2 + I^2 + X^2) + \lambda_1[(\frac{\beta_p X}{1+u_1} + \frac{\beta_s I}{1+u_2})(N - I) - \mu I] + \lambda_2(\nu I - \gamma X) \\ &= \frac{1}{2}(c_1 u_1^2 + c_2 u_2^2 + I^2 + X^2) + \lambda_1[\frac{\beta_p N X}{1+u_1} - \frac{\beta_p I X}{1+u_1} + \frac{\beta_s N I}{1+u_2} - \frac{\beta_s I^2}{1+u_2} - \mu I] + \lambda_2(\nu I - \gamma X), \end{aligned} \tag{7}$$

in which $\lambda_i$, $i = 1, 2$ are the adjoint variables.

By the Pontryagin minimum principle, we have the following equations

$$\frac{d\lambda}{dt} = -\frac{\partial H}{\partial x} \tag{8}$$

$$H(x(t), u^*(t), \lambda(t), t) = \max_{u \in U} H(x(t), u(t), \lambda(t), t) \tag{9}$$

Additionally, the costate equations $\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial I} and \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial X}$. That is,

$$\begin{aligned}\frac{d\lambda_1}{dt} &= -I + \frac{\lambda_1\beta_p X}{1+u_1} - \frac{\lambda_1\beta_s N}{1+u_2} + \frac{2\lambda_1\beta_s I}{1+u_2} + \lambda_1\mu - \lambda_2\nu.\\ \frac{d\lambda_2}{dt} &= -X - \frac{\lambda_1\beta_p N}{1+u_1} + \frac{\lambda_1\beta_p I}{1+u_1} + \lambda_2\gamma.\end{aligned}$$

Also, the optimal variables $u_1^*$ and $u_2^*$ should satisfy

$$\frac{\partial H}{\partial u_1^*} = \frac{\partial H}{\partial u_2^*} = 0.$$

i.e.,

$$\frac{\partial H}{\partial u_1^*} = c_1 u_1^* - \frac{\lambda_1\beta_p NX}{(1+u_1^*)^2} + \frac{\lambda_1\beta_p IX}{(1+u_1^*)^2} = 0, \quad \frac{\partial H}{\partial u_2^*} = c_2 u_2^* - \frac{\lambda_1\beta_s NI}{(1+u_2^*)^2} + \frac{\lambda_1\beta_s I^2}{(1+u_2^*)^2} = 0.$$

Let $B = \lambda_1\beta_p X(N-I)$ and $C = \lambda_1\beta_s I(N-I)$. We then obtain

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

Finally, since there are not terminal values for the state variables in our problem, we give transversality conditions at the final time $T$ by

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

# 4 The optimality system

We use the optimality system to represent how the system behaves under the application of the controls that minimize $J$. It is composed of the state system, the adjoint system, the optimal control $u_1^*$ and $u_2^*$, the transversality conditions and the initial conditions, which are given by.

$$\frac{dI}{dt} = (\frac{\beta_p X}{1+u_1} + \frac{\beta_s I}{1+u_2})(N-I) - \mu I, \frac{dX}{dt} = \nu I - \gamma X$$

$$\frac{d\lambda_1}{dt} = -I + \frac{\lambda_1\beta_p X}{1+u_1} - \frac{\lambda_1\beta_s N}{1+u_2} + \frac{2\lambda_1\beta_s I}{1+u_2} + \lambda_1\mu - \lambda_2\nu$$

$$\frac{d\lambda_2}{dt} = -X - \frac{\lambda_1\beta_p N}{1+u_1} + \frac{\lambda_1\beta_p I}{1+u_1} + \lambda_2\gamma$$

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

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

$$\lambda_i(T) = 0,\ i = 1, 2, I(0) = I_0, X(0) = X_0.$$

The numerical method deals with a two-point boundary problem with the boundary conditions at $t = 0$ and $t = T$ by using the software package *bvp4c* of **Matlab**. To obtain a more numerically tractable, since the primary infection characteristic of plant pathogen is completely different the secondary transmission of disease, which allow us to introduce the following appropriate scales before numerical simulations. We introduce the new non-dimensional variables and parameters $\hat{I} = \frac{I}{N}$, $\hat{X} = \frac{\eta X}{\nu N}$, $\hat{t} = \eta t$, $\hat{\beta}_p = \frac{\beta_p \nu N}{\eta^2}$, $\hat{\beta}_s = \frac{\beta_s N}{\eta}$, $\hat{\mu} = \frac{\mu}{\eta}$ and $\hat{\gamma} = \frac{\gamma}{\eta}$, in which $\eta$ is a rescaling constant. Our aim is to understand the effect of two combinational mechanisms of control primary and secondary infections. 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 [7]. The initial conditions for the state variables are given by $I_0 = 0.85$ and $X_0 = 0.8$. On the other hand, considering that $R_0 \leq 1$ is probably too strong

a condition for controlling primary and secondary infection, we would like to extend the options for its containment by analyzing more closely the case $R_0 > 1$.

Figure 2 illustrates the optimal trajectories $u_1^*$ and $u_2^*$ with the primary control cost $c_1 = 1$ and the secondary control cost $c_2 = 1$, both showing approximately bell shaped curves. Moreover, the top of bell is almost constant plateau. Also, the curve corresponding to the infected hosts have the similar shape to the inverted graph of $u_1$(or $u_2$) as shown in Figure 2. Another remarking feature is that a slight increase in the density of external pathogen inoculum could be observed during the first days because infectious hosts initially release pathogen inoculum, and then the density of pathogen inoculum is keeping at almost constant rate. Subsequently, in Figure 2 we show the optimal controls $u_1^*$ and $u_2^*$ when the cost of the control mechanism for the secondary infection is incremented 10 times. It is interesting to notice that huge amount of the control variable $u_1$ must be applied during the first days, while the extremely expensive control mechanism for the secondary infection keeps the amount of $u_2$ in at a low level though a slight increase in $u_2$ initially. Interestingly, we observe an oscillatory pattern in both state variables. Let us discuss the oscillatory behavior of this case by analyzing the infected plant trajectory, which has a rapid decay following by an increase up to a plateau, and increases at final times. Finally, we consider that the primary infection control is a little bit more expensive than the secondary infection control. From Figure 2, it is seen that the curves corresponding to the state variables as well as control mechanisms have the similar shape to the graphs of $c_1 = c_2$. It is obvious that the amount of $u_1$ is lower that the amount of $u_2$.

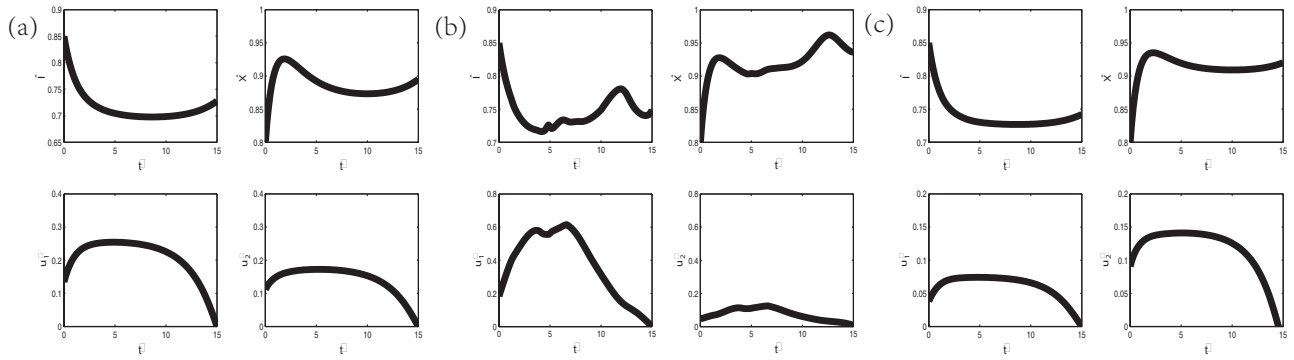


Figure 2: Optimal control trajectories for the state variables as well as control mechanisms. (a) $c_1 = c_2 = 1$; (b) $c_1 = 1.5$ and $c_2 = 10$; (c) $c_1 = 4$ and $c_2 = 1$.

# 5 Discussion

In this paper we analyze the global dynamics of the model and then derived the optimal control strategies by using the functional $J$ in terms of quadratic forms. Minimizing the cost we obtain the optimal controls $u_1^*$ and $u_2^*$, where $I$ and $X$ are both minimized. Additionally, we let the variables to be free at the final time. Then, we compare the dynamical trajectories under optimal controls, in order to assess the effects of relative cost parameters $c_1$ and $c_2$. Three control scenarios are assessed adjusting the control parameters. First, we numerically study the optimal control strategy with $c_1 = c_2$. Further, the secondary infection control cost is increased about 7 times. Finally, we analyze what happens if the primary infection control cost increases 4 times. One interesting observation is that oscillations of infected hosts and pathogen inoculum will appear depending on the huge difference between the primary control cost and the secondary one. All the cases shown in this work did not achieve the eradication of the soil-borne plant disease. However, optimal control strategies as mathematical results at least show that the amount of the primary infection control mechanism and the secondary one should be introduced at different costs with the purpose of minimizing the state variables as well as control variables. In a future work we will consider optimal control forecasted mathematically that is biologically and economically acceptable as feasible implementation in order to cease the soil-borne plant diseases.

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