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# ANALYTICAL SOLUTION OF INDUCED RESISTANCE TO CONTROL PLANT DISEASE DEVELOPMENT IN AGRICULTURAL SYSTEMS

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## ABSTRACT

A mathematical model to describe the effect of elicitor interaction with plants to manage the final disease has been developed. This model contains a non-linear term related to susceptible populations. The analytical expressions pertaining to the resistance, diseased and elicitor concentrations were reported for all potential practical values of  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$ . In this work, we report the theoretically evaluated non-steady state effectiveness factor for optimal strategy systems. These analytical results were found to be in good agreement with numerical results. Moreover, herein we employ homotopy perturbation method to solve non-linear reaction/ diffusion equations.

Key words: optimal strategy, non-linear equations, simulation, Diffusion, Induced resistance.

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## 1. INTRODUCTION

When constructing a mathematical model, there are many factors influencing the mechanism, such as natural biological processes, that can be realistically implemented into the model and that can mimic the dynamic behavior within the biological system. However, a decision must be made about what to include in the model and what to exclude. While it is true that large, detailed models are easier to sell to a biological audience since it is more biologically realistic- the level of complexity in such a detailed model poses many problems. For instance, with a large number of parameters, it is not possible to determine the accuracy of the parameter values, and a study of the parameter sensitivity is also a very difficult task. Recall that parameter values must be chosen carefully so that the model exhibits realistic behavior.

Although simple models contain the essential mechanism of the interested process, they can often provide unforeseen insight into the biological process. With simple models, it is easier to see

what mechanisms are driving the models behavior. Consequently, models can feed back directly into the biological understanding of a process. In this study, a mathematical model to describe the effect of elicitor interaction with plants to manage the final disease has been proposed and analyzed.

Previously, the efficacy of induced resistance to control plant diseases has typically been studied experimentally, whether in the field or in greenhouses. The building on the approach of jeger et al.and Xu et al. in previous modeling of biological control systems, the compartmental SIR model was applied for the potential of using a plant-defence elicitor to induce resistance to plant disease. Several assumptions on the biological background were made in order to simplify the biological process associated with IR. The developed model was generic and can be used for any combination of plant-elicitor-pathogen scenarios; which of course will have different parameters [1-2]. The new IR model has the potential to be implemented as a decision support tool for the management of plant diseases, which involves an assessment of the risk and economic cost critical for commercial operations [3].

To my knowledge no rigorous analytical solutions of mathematical model of induced resistance are developed. Such a model has not been proposed in any literature. The purpose of this communication is to derive approximate analytical expressions for the non-steady-state concentration for ainduced resistance to plant disease for all values of  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$  using Homotopy perturbation method.

### 2. Mathematical model

dt

The optimal control problem strategy is obtained by solving the optimal system, which consists of ordinary differential equations and its boundary conditions:

$$\frac{d\kappa}{dt} = (E - \gamma R)(1 - R - D); \tag{1}$$

$$\frac{dD}{dt} = \beta D(1 - R - D)(2)$$

$$\frac{dE}{dt} = -\theta E + s_0 c(3)$$

$$\frac{d\lambda_R}{dt} = \lambda_R (E + \gamma (1 - 2R - D)) + \lambda_D \beta D (4)$$

$$\frac{d\lambda_D}{dt} = \lambda_R (E - \gamma R) - \lambda_D \beta (1 - R - 2D) \tag{5}$$

$$\frac{d\lambda_{E}}{dt} = \lambda_{E}\theta - \lambda_{R}(1 - R - D)$$
(6)

where  $\gamma$  is the specific rate the resistant plant become susceptible,  $\beta$  is the specific rate at which disease spreads,  $\theta$  is the diffusion,  $s_0c$  is the elicitor effect. These equations are solved for the following initial conditions:

$$R(0) = R_i, D(0) = D_i; E(0) = 0; \lambda_R(0) = 0; \lambda_D(0) = 0; \lambda_E(0) = 0$$
(7)

where  $R_i$  is the proportion of the plant population that exhibits natural resistance at the initial time t = 0.

#### 3. Analytical expression of concentrations using HPM

Recently, many authors have applied the HPM to various problems and demonstrated the efficiency of the HPM for handling non-linear structures and solving various physics and engineering problems [4-9]. This method is a combination of homotopy in topology and classic perturbation techniques. The set of expressions presented in Eqs. (1) - (7) defines the initial value problem. The Homotopy perturbation method [10-15] is used to give the approximate analytical solutions of coupled non-linear reaction/diffusion Eqs. (1) to (6). The dimensionless reaction diffusion parameters  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$  are related to one another, since the bulk solution is at equilibrium in the non-steady state. Using HPM (see Appendix A), we can obtain the following solutions to the Eqs.(1) to (6).

$$\begin{split} & R(t) = R_{l}e^{-\gamma t} + \left[\frac{R_{l}^{2}}{2} - \frac{\gamma R_{l}D_{l}}{\beta - 2\gamma}\right]e^{-\gamma t} - \frac{s_{0}ct}{2\gamma} + \frac{s_{0}ctR_{l}e^{-\gamma t}}{2\gamma} - \frac{s_{0}ctD_{l}e^{\beta t}}{\beta - \gamma} - \frac{R_{l}^{2}e^{-\gamma t}}{2} + \frac{\gamma R_{l}D_{l}e^{(\beta - \gamma)t}}{\beta - 2\gamma} \right]e^{\beta t} + \frac{\beta D_{l}R_{l}}{\gamma}e^{(\beta - \gamma)t} - D_{l}^{2}e^{2\beta t} + D_{l}e^{\beta t}(9) \\ & E(t) = s_{0}ct - \theta s_{0}ct^{2} + \theta^{2}s_{0}ct^{3} \quad (10) \\ & \lambda_{R}(t) = \left[\frac{\beta D_{l}}{\gamma \beta + \beta - \gamma} - \frac{\beta D_{l}}{\beta - \gamma}\right]e^{\beta - \frac{\beta D_{l}e^{(\beta + \beta)t}}{\gamma \beta + \beta - \gamma}} - \frac{\beta D_{l}e^{(\beta + \beta)t}}{\gamma \beta + \beta - \gamma} + \frac{\beta D_{l}e^{\beta t}}{\beta - \gamma} - \frac{2R_{l}\beta D_{l}e^{\beta t}}{\gamma^{2} + \gamma \beta - \gamma^{2}} + \frac{\gamma \beta D_{l}^{2}e^{\beta t}}{\gamma \beta^{2} - \gamma \beta} + \frac{2R_{l}\beta D_{l}e^{\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - \frac{\gamma \beta D_{l}^{2}e^{\beta t}}{(\gamma \beta + \beta - \gamma)(\gamma \beta + 2\beta - \gamma)} + \frac{2R_{l}\beta D_{l}e^{\beta t}}{(\beta - \gamma)(\beta - 2\gamma)} + \frac{\gamma \beta D_{l}^{2}e^{\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - \frac{\gamma \beta D_{l}^{2}e^{\beta t}}{\beta - \gamma} + \frac{\gamma \beta D_{l}e^{-\beta t}}{\beta - \gamma} - \frac{\gamma \beta D_{l}e^{-\beta t}}{\beta - \gamma} - \frac{\gamma \beta D_{l}e^{-\beta t}}{(\gamma \beta + \beta - \gamma)(\gamma \beta + 2\beta - \gamma)} + \frac{\gamma \beta R_{l}D_{l}e^{-\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - \frac{\beta R_{l}e^{-\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - D_{l}e^{-\beta t}} + \frac{\beta D_{l}e^{-\beta t}}{\beta - \gamma} - \frac{\gamma \beta D_{l}e^{-\beta t}}{\beta - \gamma} - \frac{\gamma \beta D_{l}e^{-\beta t}}{(\gamma \beta + \beta - \gamma)(\gamma \beta + \beta - \gamma)(\gamma \beta + \beta - \gamma)} + \frac{\gamma \beta R_{l}D_{l}e^{-\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - \frac{\beta D_{l}e^{\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - D_{l}e^{-\beta t}} + \frac{\beta D_{l}e^{-\beta t}}{(\beta - \gamma)(2\beta - \gamma)} - \frac{\beta D_{l}e^{-\beta t}}{(\beta - \gamma)(2\beta - \gamma)} + \frac{\beta D_{l}R_{l}e^{\beta t}}{(\beta - \gamma)(2\beta - \eta)} - \frac{\beta D_{l}R_{l}e^{\beta t}}{(\beta - \gamma)(2\beta - \eta)} - \frac{\beta D_{l}R_{l}e^{\beta t}}{(\beta - \gamma)(2\beta - \eta)} + \frac{\beta D_{l}R_{l}e^{\beta t}}{(\beta - \gamma)(\gamma + \beta - \gamma)(\gamma \beta + \beta - \gamma)(\gamma \beta + \beta - \gamma)} + \frac{\beta D_{l}R_{l}e^{\beta t}}{(\beta - \gamma)(2\beta - \eta)} + \frac{\beta D_{l}R_{l}e^{\beta t}}{(\beta$$

$$B = -\frac{\beta D_{i} e^{\gamma t}}{\gamma - \theta} + \frac{\beta D_{i} e^{\gamma t}}{\gamma - \theta} + \frac{\beta D_{i} e^{(\gamma + \beta)t}}{\gamma - \theta} + \frac{\beta D_{i} e^{(\gamma + \beta)t}}{\gamma \beta + \beta - \eta} + \frac{\beta D_{i} e^{\beta t}}{\beta - \gamma} + \frac{\beta D_{i} e^{\beta t}}{\beta - \eta} + \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma - \theta)} + \frac{\beta D_{i} e^{(\gamma + \beta)t}}{(\gamma \beta + \beta - \gamma - \theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\gamma \beta + \beta - \gamma)(-\theta)} - \frac{\beta D_{i} R_{i}}{(\beta - \gamma)(-\theta)} - \frac{\beta D_{i}}{(\beta - \gamma)(-\theta)} - \frac{\beta D_$$

### 4. Discussion

In fig. 1 shows that, the elicitor application effect is determined by the elicitor applied daily until it reaches the target disease control. In fig. 2, the time series of induced resistance model for the treated plant. Fig. 3 shows the dimensionless non-steady-state concentration of resistance for the potential values of  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$  calculated using Eq. (8). From this figure, we can see that the

value of the concentration decreases when time (days) increases. The concentration of resistance decreases slowly and abruptly constant when  $t \ge 15$  and all values of  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$ . When, t = 0, the concentration of resistant plants drop then decreases and to reach the steady-state value.

Fig. 4.represents the dimensionless concentration of disease D(t) versus dimensionless time (days) for different values of dimensionless  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$ . From these figure, it is inferred that, the proportion of the diseased plant increases and then reaches its absolute steady state after 15 days. Fig. 5.represents the dimensionless concentration of elicitor E(t) versus dimensionless time (days) for different values of dimensionless  $\beta$ ,  $\gamma$ ,  $\theta$  and  $s_0c$ . From this figure, it is inferred that, the cumulative elicitor effect which corresponds to the elicitor application every day. Moreover, the initial time is zero, a large amount of elicitor has to be applied and then slowly decreased. Fig. 6-8 represents the dimensionless concentration as a function of time.







Fig. 2: Time series for the IR model for the treated plants.

# 5. Numerical simulation

The HPM provides an analytical solution in terms of an infinite power series. However, there is a practical need to evaluate this solution and to obtain numerical values from the infinite power series. The consequent series truncation and the practical procedure conducted to accomplish this task, together transforms the otherwise analytical results into an exact solution, which is evaluated to a finite degree of accuracy. In order to investigate the accuracy of the HPM solution with a finite number of terms, the system of differential equation were solved. To show the efficiency of the present method for our problem in comparison with the numerical solution (MATLAB program) we report our results graphically. The MATLAB program is also given in Appendix B.



Fig. 3:Plot of dimensionless concentration of resistance versus time t forfixed value of  $\beta$ ,  $\gamma$ , and  $s_0c$ . Concentration is calculated using the equation (8).



Fig. 4:Plot of dimensionless concentration of diseased versus time t forfixed value of  $\beta$ ,  $\gamma$ , and  $s_0c$ . Concentration is calculated using the equation (9).



Fig. 5: Plot of dimensionless concentration of elicitor versus time t forfixed value of  $\beta$ ,  $\gamma$ , and  $s_0c$ . Concentration is calculated using the equation (10).



Fig. 6: Plot of dimensionless concentration of  $\lambda_R(t)$  versus time t forfixed value of  $\beta$ ,  $\gamma$ , and  $s_0c$ . Concentration is calculated using the equation (11).



**Fig. 7**: Plot of dimensionless concentration of  $\lambda_D(t)$  versus time t forfixed value of  $\beta$ ,  $\gamma$ , and  $s_0c$ . Concentration is calculated using the equation (12).



Fig. 8: Plot of dimensionless concentration of  $\lambda_E(t)$  versus time t forfixed value of  $\beta$ ,  $\gamma$ , and  $s_0c$ . Concentration is calculated using the equation (13).

## 6. Conclusions

The time dependent non-linear reaction/diffusion equationsfor induced resistance mechanism using elicitor application have been formulated and solved using HPM. The primary result of this work is simple approximate calculation of dynamical concentration profiles for all values of potential parameters. We have presented the estimated parameters were then used to analyse the dynamical IR model based on theHomotopy perturbation method. This method can be easily extended to find the concentrations for all mechanism for various complex boundary conditions. The goal was to determine the best application might cause phytotoxicity to the plants. This daily application strategy is reasonable for plants that may be already infected. The results from

this numerical experiment gave the optimal application strategy, and thus will help farmers avoid unnecessary application.

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### Appendix A.

In this appendix we outline the basic idea of Homotopy Perturbation method [10-15]. This method has eliminated the limitations of the traditional perturbation methods. On the other hand it can take full advantage of the traditional perturbation techniques, so there has been a considerable deal of research in applying homotopy technique for solving various strongly nonlinear equations. To explain this method, let us consider the following function

$$A(u) - f(r) = 0, \quad r \in \Omega \tag{A1}$$

with the boundary conditions of

$$B(u, \frac{\partial \mathbf{u}}{\partial n}) = 0, \quad r \in \Gamma$$
(A2)

where A, B, f(r) and  $\Gamma$  denote a general differential operator, a boundary operator, a known analytical function and the boundary of the domain  $\Omega$ , respectively. Generally speaking, the operator A can be divided into a linear part L and a nonlinear part N. Eq.(A1) can therefore, be written as

$$L(u) + N(u) - f(r) = 0$$
 (A3)

By the Homotopy technique, we construct a Homotopy v(r, p):  $\Omega \times [0,1] \rightarrow R$  which satisfies

$$H(v, p) = (1 - p)[L(v) - L(u_0)] + p[A(v) - f(r)] = 0. \qquad p \in [0,1], r \in \Omega$$
 (A4) or

$$H(v, p) = L(v) - L(u_0) + pL(u_0) + p[N(v) - f(r)] = 0.$$
 (A5)

where  $p \in [0,1]$  is an embedding parameter, and  $u_0$  is an initial approximation of Eq. (A1), which satisfies the boundary conditions. Obviously, from Eqs. (A4) and (A5), we will have

$$H(v,0) = L(v) - L(u_0) = 0$$
(A6)

$$H(v,1) = A(v) - f(r) = 0.$$
 (A7)

when p = 0 Eq. (A4) or Eq. (A5) becomes a linear equation; when p = 1 it becomes a non-linear equation. So the changing process of p from zero to unity is just that of  $L(v) - L(u_0) = 0$  to A(v) - f(r) = 0. We can first use the embedding parameter p as a "small parameter", and assume that the solutions of Eqs. (A4) and (A5) can be written as a power series in p

$$v = v_0 + pv_1 + p^2 v_2 + \dots$$
(A8)

Setting p = 1 results in the approximate solution of Eq.(A1):

$$u = \lim_{p \to 1} v = v_0 + v_1 + v_2 + \dots$$
(A9)

The combination of the perturbation method and the Homotopy method is called the HPM.

# Appendix B

```
function main1
options= odeset('RelTol',1e-6,'Stats','on');
%initial conditions
Xo = [0.6118; 0.0168; 0; -4; 18; -11];
tspan = [0, 20];
xspan = [0,0.5];
tic
[t,X] = ode45(@TestFunction,tspan,Xo,options);
toc
figure
holdon
plot(t, X(:,6))
plot(t, X(:,6),'.')
legend('x1','x2', 'x3','x4','x5','x6')
ylabel('x')
xlabel('t')
return
function [dx_dt]= TestFunction(t,x)
b=0.7379;
r=0.2801;
o=0.05;
s=0.1;
dx_dt(1) = (x(3)-r^*x(1))^*(1-x(1)-x(2));
dx_dt(2) =b*x(2)*(1-x(1)-x(2));
dx_dt(3) = -o^*x(3)+s;
dx_dt(4) = x(4)^*(x(3)+r^*(1-2^*x(1)-x(2)))+x(5)^*b^*x(2);
dx_dt(5) = x(4)^*(x(3)-r^*x(1))-x(5)^*b^*(1-x(1)-2^*x(2));
```

dx\_dt(6) =x(6)\*o-x(4)\*(1-x(1)-x(2));

dx\_dt = dx\_dt';

return

# Appendix C.Nomenclature

R	Proportion of plant population able to express	
	resistance to infection.	dimensionless
D	Proportion of plant population being infected	
	and becoming diseased.	dimensionless
S	Proportion of plant population which is susceptible.	dimensionless
t	Time	[days]
$\alpha$	The specific rate at which untreated plants	
	lose their resistance due to the pathogen attack.	$[days^{-1}]$
β	The specific rate at which the disease spreads.	$[days^{-1}]$
$\gamma$	The specific rate the resistant plant becomes susceptible.	$[days^{-1}]$
e(t)	The effectiveness of the elicitor at a single application.	$[days^{-1}]$
k	Determines the effectiveness of the elicitor.	dimensionless
L	The time where the elicitor effectiveness is at the peak.	[days]
$t_p$	The induction time of the pathogen i.e. the time interval	
	between the elicitor application and the pathogen challenge.	[days]
$R_i$	The proportion of the plant population that exhibits	
	natural resistance at the initial time $t = 0$ .	dimensionless
$D_i$	The proportion of the plant population which becomes	
	infected immediately after the pathogen challenge.	dimensionless
a	The scaled dimensionless elicitor concentration.	dimensionless
r	The parameter determines the sub-linear	
	effect of elicitor concentration.	dimensionless
E(t)	The cumulative effectiveness of the elicitor at daily application.	[days]
c(t)	The continuous elicitor application.	[mass days <sup>-1</sup> ]