The Impact of Predators on Malicious Worms Propagation: a Theoretical Approach

Qingyi Zhu, Yi Jiang, Ye Zhang

Abstract—**Predators, a class of programs that can travel over Internet, and replicate and multiply themselves, are specifically designed to eliminate the computer viruses. To better understand the effects of predators on virus propagation, a model described the coevolution between viruses and predators is proposed. This model has one virus-free equilibrium and two potential viral equilibria. The existence and global stability of these equilibria are fully studied. Furthermore, the optimal solution to control virus propagation is obtained by using Pontryagin's minimum principle. And some numerical experiments are carried out to verify the theoretical results. From the obtained results it can be deduced that predators are quite effective in preventing the widely propagation of viruses with extremely high infection rate.**

Index Terms—**Computer virus, Predators, Epidemic model, Equilibrium, Global asymptotic stability.**

I. Introduction

WITH the popularity and maturity of Internet, computer viruses, as a type of malicious programs that can self-replicate and widely spread among different computers, mobile devices and sensors[1], have formed a great threat to human society. Every year, computer viruses cause multi-billion dollars economic loss, by destroying operating systems, wasting computing resources, tampering data, stealing valuable information, etc. Antivirus, as a kind of computer software used to prevent, detect and remove malwares, plays an important role in the campaign against worms. But no currently existing antivirus software is strong enough to deal with all computer viruses (especially new ones). And in fact, Cohen, who introduced the item "computer virus" firstly, proved theoretically that such antivirus does not exist [2]. As a result, computer security researchers are actively searching for new ways to prevent the widely distribution of viruses. In this context, the study of the macro view of computer viruses, which leads to some new insights and tools that may help society to cope better with the virus crisis, has received more and more attentions.

Following the suggestions of Cohen [2], Murray [4] and other pioneers in network security, the mathematical modeling approach in the epidemiology [3] has been widely exploited to study the propagation in the macro view of computer viruses, due to the intriguing analogies between computer viruses and their biological counterparts. Through this approach, varieties of virus propagation models, ranging from conventional models [5], [6], [7], [8], [9], [10], [11], [12], [13], [14], [15], to delayed models [16], [17], [18], [19], [20], [21], [22], to impulsive models [23], and to stochastic models [24], [25], [26], have been proposed.

Predators, as a class of benevolent, self-propagating programs with the ability to eliminate the malware from computer systems, have been regarded as holding significant promise as an alternative to the currently popular centralized patches distribution mechanism which suffers from distribution bottlenecks due to the limit in network bandwidth and the number of servers [27], [28], [29], [30], [31]. Most of previous work in this area is intended to develop a realistic anti-worms (predators) system [27], [28], [29], [31]. To our knowledge, no work on the theoretical study of the effects of predators on computers virus spread has been reported in the literature.

This paper is intended to examine the interaction between viruses and predators theoretically. Based on the assumptions that the computer network is fully-connected and that the predators have only temporary immunity, a propagation model of worms and predators is proposed. This model has one virus-free equilibrium and two potential endemic equilibria. The existence and global stability of these equilibria are studied, from which it is concluded that predators are quite effective in preventing the widely propagation of viruses with extremely high infection rate.

The remainder of this paper is arranged as follows: Section 2 formulates the new model. Section 3 calculates the potential equilibria of the model. Sections 4 and 5 examine the local and global stability of the equilibria, respectively. Section 6 formulates the optimal control problem of proposed model. Section 7 provides several numerical examples. This work is outlined in section 8.

II. Model formulation

For our purposes, a computer is referred to as $internal$ or $external$ according as it is connected to the Internet or not. All internal computers are divided into the following three classes.

- (C1) Susceptible internal computers (S-computers), i.e., uninfected internal computers that have no immunity;
- (C2) Infected internal computers (I-computers), i.e., infected internal computers;
- (C3) P-computers, i.e., uninfected internal computers with predators.

Let $S(t)$, $I(t)$, and $P(t)$ denote the numbers of S-, I-, and P-computers at time t, respectively; without ambiguity, they will be abbreviated as S, I, and P, respectively.

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To capture the coevolution behaviors of computer viruses and predators, the following assumptions are made.

(A1) All external computers are susceptible.
(A2) External computers are connected to the Internet at constant rate λ.
(A3) Internal computers are disconnected from the Internet at constant rate μ.
(A4) Every S-computer is infected by I-computers with probability $\beta I(t)$, where β is a positive constant.
(A5) Every I-computer could be disinfected by predators with probability $\sigma_1 P(t)$, where σ_1 is a positive constant. And this cured I-computer becomes S-computer (P-computer) with constant probability θ ($(1-\theta)$, respectively), where $0 < \theta < 1$.
(A6) Every I-computer is cured with constant probability σ_2 due to other disinfection measures.
(A7) Due to the invalidation of predators, every P-computer becomes S-computer with constant probability γ.

These assumptions can be presented schematically as Figure 1.

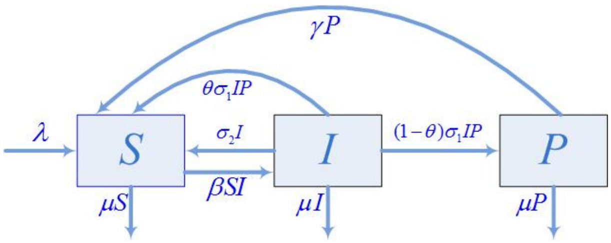

Fig. 1. The state transition diagram.

From this collection of assumptions, one can derive the following coevolution propagation model of computer viruses and predators:

$$\begin{cases} \dot{S} = \lambda - \beta SI + \theta\sigma_1 IP + \sigma_2 I + \gamma P - \mu S, \\ \dot{I} = \beta SI - \sigma_1 IP - (\sigma_2 + \mu) I, \\ \dot{P} = (1-\theta)\sigma_1 IP - (\gamma + \mu) P, \end{cases} \quad (1)$$

with initial condition $(S(0), I(0), P(0)) \in R^3_+$.

Let $N = N(t)$ denotes the total number of internal computers at time t. Then, $N(t) = S(t) + I(t) + P(t)$. Clearly,

$$\dot{N} = \lambda - \mu N.$$

Obviously, $N \to \frac{\lambda}{\mu}$ as $t \to +\infty$. Therefore, system (1) can be reduced as the following limiting system:

$$\begin{cases} \dot{I} = \beta(\frac{\lambda}{\mu} - I - P) I - \sigma_1 IP - (\sigma_2 + \mu) I, \\ \dot{P} = (1-\theta)\sigma_1 IP - (\gamma + \mu) P. \end{cases} \quad (2)$$

One can conclude that all solutions to system (2) approach the simply connected compact set

$$\Omega = \left\{ (I, P) \in R^2_+ : I + P \le \frac{\lambda}{\mu} \right\}, \quad (3)$$

which is positively invariant. And from here on, we denote the boundary of Ω as $\partial\Omega$ for convenience.

III. Equilibria

For system (2), direct calculations give three potential equilibria, which are listed below.

$E_1 = (I_1, P_1)$, where $I_1 = P_1 = 0$;
$E_2 = (I_2, P_2)$, where $I_2 = \frac{\beta\lambda - \mu(\mu+\sigma_2)}{\mu\beta}$ and $P_2 = 0$;
$E_3 = (I_3, P_3)$, where $I_3 = \frac{\gamma+\mu}{(1-\theta)\sigma_1}$ and $P_3 = \frac{\beta\lambda - \mu\beta I_3 - \mu(\mu+\sigma_2)}{\mu(\beta+\sigma_1)}$.

Clearly, E_1 is virus-free, whereas E_2 and E_3 are endemic. A careful checking gives

Theorem 1: Consider system (1).

(A1) There is a unique equilibrium, E_1, if $\beta \le \frac{\mu(\mu+\sigma_2)}{\lambda}$.
(A2) There are exactly two equilibria, E_1 and E_2, if $\beta > \frac{\mu(\mu+\sigma_2)}{\lambda}$ and $\beta(\lambda - \mu I_3) \le \mu(\mu + \sigma_2)$.
(A3) There are three equilibria, E_1, E_2 and E_3, if $\beta > \frac{\mu(\mu+\sigma_2)}{\lambda - \mu I_3}$ and $\frac{\gamma+\mu}{(1-\theta)\sigma_1} < \frac{\lambda}{\mu}$.

IV. Local stability analysis

This section examines the local stability of the three potential equilibria mentioned in the previous section. For the linearized system of system (1) evaluated at a potential equilibrium $\bar{E} = (\bar{I}, \bar{P})$, its corresponding characteristic equation is

$$\begin{vmatrix} \begin{matrix} \xi - \beta(\frac{\lambda}{\mu} - 2\bar{I} - \bar{P}) \\ +\sigma_1\bar{P} + \mu + \sigma_2 \end{matrix} & (\sigma_1 + \beta)\bar{I} \\ -(1-\theta)\sigma_1\bar{P} & \xi - (1-\theta)\sigma_1\bar{I} + \gamma + \mu \end{vmatrix} = 0. \quad (4)$$

Theorem 2: E_1 is locally asymptotically stable if $\beta < \frac{\mu(\mu+\sigma_2)}{\lambda}$.

Proof 1: For $\bar{E} = E_1$, equation (4) reduces to

$$\begin{vmatrix} \xi - \frac{\beta\lambda}{\mu} + \mu + \sigma_2 & 0 \\ 0 & \xi + \gamma + \mu \end{vmatrix} = 0,$$

whose roots are

$$\xi_1 = \frac{\beta\lambda}{\mu} - \mu - \sigma_2, \quad \xi_2 = -\gamma - \mu.$$

Clearly, both of these two roots are negative. The claimed result follows by the Lyapunov theorem [32].

Theorem 3: E_2 is locally asymptotically stable if $\beta > \frac{\mu(\mu+\sigma_2)}{\lambda}$ and $\beta(\lambda - \mu I_3) < \mu(\mu + \sigma_2)$.

Proof 2: For $\bar{E} = E_2$, equation (3) reduces to

$$\begin{vmatrix} \xi - \beta(\frac{\lambda}{\mu} - 2I_2) + \mu + \sigma_2 & (\sigma_1 + \beta)I_2 \\ 0 & \xi - (1-\theta)\sigma_1 I_2 + \gamma + \mu \end{vmatrix} = 0,$$

whose roots are

$$\xi_1 = \mu + \sigma_2 - \frac{\beta\lambda}{\mu}, \quad \xi_2 = \frac{(1-\theta)\sigma_1}{\mu\beta}[\beta(\lambda - \mu I_3) - \mu(\mu + \sigma_2)].$$

Clearly, both of these two roots are negative. The claimed result follows from the Lyapunov theorem [32].

Theorem 4: E_3 is locally asymptotically stable, if $\beta > \frac{\mu(\mu+\sigma_2)}{\lambda - \mu I_3}$ and $\frac{\gamma+\mu}{(1-\theta)\sigma_1} < \frac{\lambda}{\mu}$.

Proof 3: For $\bar{E} = E_3$, equation (3) reduces to

$$\begin{vmatrix} \begin{matrix} \xi - \beta(\frac{\lambda}{\mu} - 2I_3 - P_3) \\ +\sigma_1 P_3 + \mu + \sigma_2 \end{matrix} & (\sigma_1 + \beta)I_3 \\ -(1-\theta)\sigma_1 P_3 & \xi - (1-\theta)\sigma_1 I_3 + \gamma + \mu \end{vmatrix} = 0.$$

whose roots are

$$\xi_{1,2}=\frac{-\beta I_3\pm\sqrt{\beta^2I_3^2-(\sigma_1+\beta)(\mu+\gamma)}}{2}.$$

Clearly, both of these two roots are negative. The claimed result follows from the Lyapunov theorem [32].

V. Global stability analysis

This section deals wtih the global stability of the three potential equilibria. For that purpose, we begin with the following lemma.

Lemma 1: Consider system (2). There is no periodic solution within Ω.

Proof 4: Let

$$\begin{aligned} f_1(I,P) &= \beta(\frac{\lambda}{\mu}-I-P)I-\sigma_1 IP-(\sigma_2+\mu)I,\\ f_2(I,P) &= (1-\theta)\sigma_1 IP-(\gamma+\mu)P. \end{aligned}$$

Define $D(I,P)=\frac{1}{IP}$. Then,

$$\frac{\partial Df_1(I,P)}{\partial I}+\frac{\partial Df_2(I,P)}{\partial P}=-\frac{\beta}{I}<0.$$

By the Bendixson-Dulac criterion [32], system (2) has no periodic orbit in the interior of Ω.

Owing to the smoothness of the orbits of system (2), there is no periodic orbit that passes through any one of the three corners of Ω.

Now we investigate the situation that a periodic orbit passes through a non-corner point on $\partial\Omega$. That is to say, this orbit must be tangent to $\partial\Omega$ at that point. Here, suppose there is a orbit C that passes through a non-corner point $(\bar I,\bar P)$ on $\partial\Omega$, then the following three cases are considered:

Case 1: $\bar I=0$, $0<\bar P<\frac{\lambda}{\mu}$. Then $\frac{dI(t)}{dt}\mid_{(\bar I,\bar P)}=0$.

Case 2: $\bar P=0$, $0<\bar I<\frac{\lambda}{\mu}$. Then $\frac{dP(t)}{dt}\mid_{(\bar I,\bar P)}=0$.

Case 3: $\bar I+\bar P=\frac{\lambda}{\mu}$, $\bar I>0$ and $\bar P>0$. Then $\frac{d(I(t)+P(t))}{dt}\mid_{(\bar I,\bar P)}=-\theta\sigma_1 IP-(\sigma_2+\mu)I-(\gamma+\mu)P<0$.

Obviously, there is no periodic orbit falling in the set $\{(I,P)\in\Omega: I=0\}$ nor in the set $\{(I,P)\in\Omega: P=0\}$. And from Case 3, we know that there is no orbit can be target to that boundary of $\partial\Omega$. As a result, system (2) admits no periodic orbit in the whole Ω.

Here, we are ready to establish the main results of this paper.

Theorem 5: Consider system (2),

(a) E_1 is asymptotically stable with respect to Ω if $\beta<\frac{\mu(\mu+\sigma_2)}{\lambda}$.

(b) E_1 is attractive with respect to $\{(I,P)\in\Omega: I=0\}$, and E_2 is asymptotically stable with respect to $\{(I,P)\in\Omega: I\neq 0\}$, if $\beta>\frac{\mu(\mu+\sigma_2)}{\lambda}$ and $\beta(\lambda-\mu I_3)<\mu(\mu+\sigma_2)$.

(c) E_1 is attractive with respect to $\{(I,P)\in\Omega: I=0\}$, E_2 is attractive with respect to $\{(I,P)\in\Omega: P=0 \text{ and } I>0\}$ and E_3 is asymptotically stable with respect to $\{(I,P)\in\Omega: I\neq 0 \text{ and } P\neq 0\}$, if $\beta>\frac{\mu(\mu+\sigma_2)}{\lambda-\mu I_3}$ and $\frac{\gamma+\mu}{(1-\theta)\sigma_1}<\frac{\lambda}{\mu}$.

Proof 5: (a) The claimed result follows from the generalized Poincare-Bendixson theorem [32], Theorem 2, and Lemma 1.

(b) Let $I(0)=0$. Then $I(t)\equiv 0$. Plugging it into the second equation of system (2) and solving this equation, we get that $P(t)\to 0$. Clearly, the characteristic equation at E_1 has exactly one positive root $\xi_1=\frac{\beta\lambda}{\mu}-\mu-\sigma_2$ and one negative root $\xi_2=-\gamma-\mu$, if $\frac{\mu(\mu+\sigma_2)}{\lambda}<\beta$. And, the eigenvector with respect to ξ_1 is $\mathbf{v}_2=k(0,1)^T$, $k\in\mathbb{R}$. Hence, E_1 is attractive with respect to $\{(I,P)\in\Omega: I=0\}$, if $\beta>\frac{\mu(\mu+\sigma_2)}{\lambda}$. Moreover, E_2 is asymptotically stable with respect to $\{(I,P)\in\Omega: I\neq 0\}$, if $\beta>\frac{\mu(\mu+\sigma_2)}{\lambda}$ and $\beta(\lambda-\mu I_3)<\mu(\mu+\sigma_2)$ from theorem 1-3 and the generalized Poincare Bendixson theorem [32].

(c)Similarly, The claimed result follows by theorem 1-4 and the generalized Poincare Bendixson theorem [32].

VI. The optimal control model

To formulate the optimal control problem of system (1), a Lebesgue square integrable control function $u(t)$ is introduced, where $0\leqslant u(t)\leqslant u_{\max}$. Then the controlled state system can be rewritten as:

$$\begin{cases} \dot{S_L}=b+\gamma I+\delta S_H-f(I)S_L-\beta_1 S_L I-\mu S_L\\ \dot{S_H}=f(I)S_L-\delta S_H-\beta_2 S_H I-\mu S_H+u(t)I\\ \dot{I}=\beta_1 S_L I+\beta_2 S_H I-\gamma I-\mu I-u(t)I \end{cases} \tag{5}$$

Furthermore, we define following objective functional to minimize:

$$J=\int_0^T I+\frac{1}{2}wu^2(t)\mathrm{d}t, \tag{6}$$

where w is the weight index of control costs. Then, one can obtain the following corresponding Hamiltonian:

$$\begin{aligned} H=&\lambda_1(b+\gamma I+\delta S_H-f(I)S_L-\beta_1 S_L I-\mu S_L)\\ &+\lambda_2(f(I)S_L-\delta S_H-\beta_2 S_H I-\mu S_H+u(t)I)\\ &+\lambda_3(\beta_1 S_L I+\beta_2 S_H I-\gamma I-\mu I-u(t)I)+I+\frac{1}{2}wu^2(t). \end{aligned} \tag{7}$$

where $\lambda_i (i=1,2,3)$ are the adjoint variables. By directly calculation, we can obtain following results.

$$\begin{aligned} \dot{\lambda_1}=&-\frac{\partial H}{\partial S_L}\\ =&(f(I)+\beta_1 I+\mu)\lambda_1-f(I)\lambda_2-\beta_1 I\lambda_3,\\ \dot{\lambda_2}=&-\frac{\partial H}{\partial S_H}\\ =&-\delta\lambda_1+(\delta+\beta_2 I+\mu)\lambda_2-\beta_2 I\lambda_3,\\ \dot{\lambda_3}=&-\frac{\partial H}{\partial I}\\ =&-1-(\gamma-f'(I)S_L-\beta_1 S_L)\lambda_1\\ &-(f'(I)-\beta_2 S_H+u(t))\lambda_2\\ &-(\beta_1 S_L+\beta_2 S_H-\gamma-\mu-u(t))\lambda_3. \end{aligned}$$

By using the optimality condition, we obtain

$$\frac{\partial H}{\partial u(t)}=wu(t)+(\eta_2-\eta_3)I=0. \tag{8}$$

Hence, the optimality solution respect to system (5) is

$$u(t)=\min\{\max\{\frac{(\eta_3-\eta_2)I}{w},0\},u_{\max}\}. \tag{9}$$

VII. Numerical Examples and Discussions

Here, we illustrate the global stability of the three potential equilibria of system (1). In all simulations, the total number of nodes (computers) is fixed at 1000. For each example, we are going to run each test ten times and take the mean to eradicate any discrepancies.

Example 1: Consider system (2) with parameters $\lambda = 0.1$, $\mu = 0.1$, $\beta = 0.3$, $\sigma_1 = 0.3$, $\sigma_2 = 0.2$, $\gamma = 0.2$ and $\theta = 0.13$. The simulation results show in Figure 2, and Figure 3 shows the phase portrait and the time plot of numerical computing results.

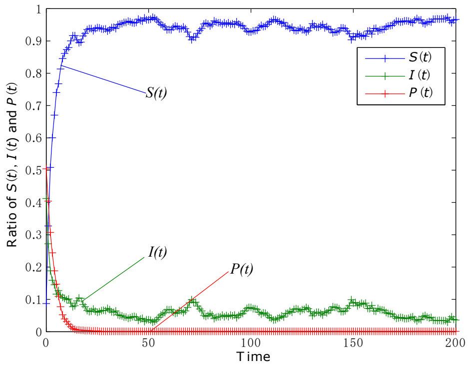

Fig. 2. The simulation results for the system given in Example 1.

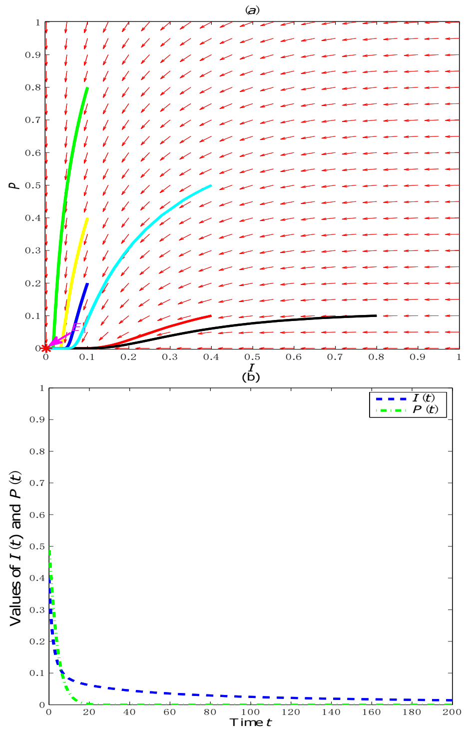

Fig. 3. The numerical computing results for the system given in Example 1.

Example 2: Consider system (2) with parameters $\lambda = 0.1$, $\mu = 0.1$, $\beta = 0.6$, $\sigma_1 = 0.4$, $\sigma_2 = 0.2$, $\gamma = 0.2$ and $\theta = 0.13$. The simulation results show in Figure 4, and Figure 5 shows the phase portrait and the time plot of numerical computing results.

Example 3: Consider system (2) with parameters $\lambda = 0.1$, $\mu = 0.1$, $\beta = 0.5$, $\sigma_1 = 0.8$, $\sigma_2 = 0.2$, $\gamma = 0.01$ and $\theta = 0.2$. The simulation results show in Figure 6, and Figure 7 shows the phase portrait and the time plot of numerical computing results.

Example 4: Consider the controlled system (5) with parameters $\lambda = 0.1$, $\mu = 0.1$, $\beta = 0.6$, $\sigma_1 = 0.4$, $\sigma_2 = 0.2$, $\gamma = 0.2$, $\theta = 0.13$, $u_{\max} = 0.1$, and $w = 10$. Figure 8 shows the comparison of the system with optimal control and without control. Obviously, the number of infected computers is significantly reduced by applying the optimal control strategy.

In Figure 9, the existence and stability of the three potential equilibria with respect to β is shown, and other parameters are given as $\lambda = 0.1$, $\mu = 0.1$, $\sigma_1 = 0.8$, $\sigma_2 = 0.2$, $\gamma = 0.01$ and $\theta = 0.2$. Obviously, if the infection rate β is quite small, the virus-free equilibrium is always asymptotically stable, which finally will lead to eradication of the viruses in the Internet. As the increase of β, the virus-free equilibrium loses its stability finally and the viral equilibrium E_2 which is asymptotically stable emerges. In these two cases, the predators accelerate the arrival of these steady-state of this system, although the state which the system will finally process to is independent of them. With the further increase of β, E_2 also loses its stability, and the coexist equilibrium E_3 which is asymptotically stable emerges. Note that I_3 is independent of β, that is to say, the infection density keeps constant as the increase of β.

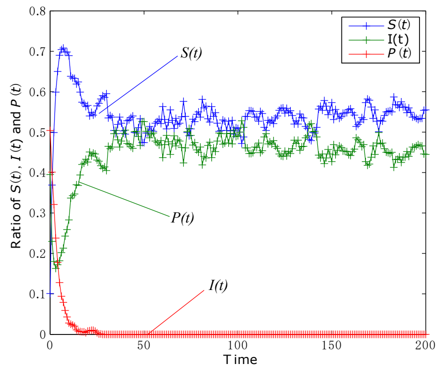

Fig. 4. The simulation results for the system given in Example 2.

Consequently, Predators hold significant effects to prevent the widely spread of computer viruses, especially those with extremely high infection rate. Moreover, from the expression of I_3, to reduce the density of infected computer further, more effective predators should be chosen.

VIII. Conclusions

In this paper, a propagation model of computer viruses and predators has been proposed. This model has one potential virus-free equilibrium and two potential endemic equilibria. The existence and global stability of these equilibria have been fully studied. An optimal control strategy is also applied to control the spread of computer virus. This work shows that the introduction of predator contributes to the inhibition of widely spread of computer viruses.

Our next work is to study the behavior of this model on complex networks.

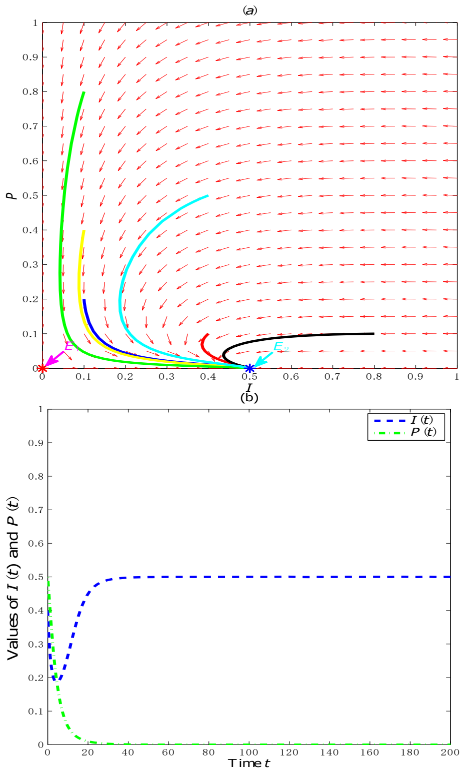

Fig. 5. The numerical computing results for the system given in Example 2.

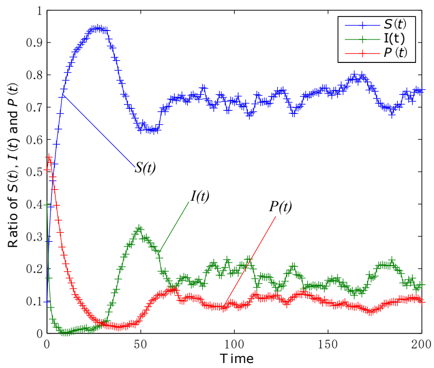

Fig. 6. The simulation results for the system given in Example 3.

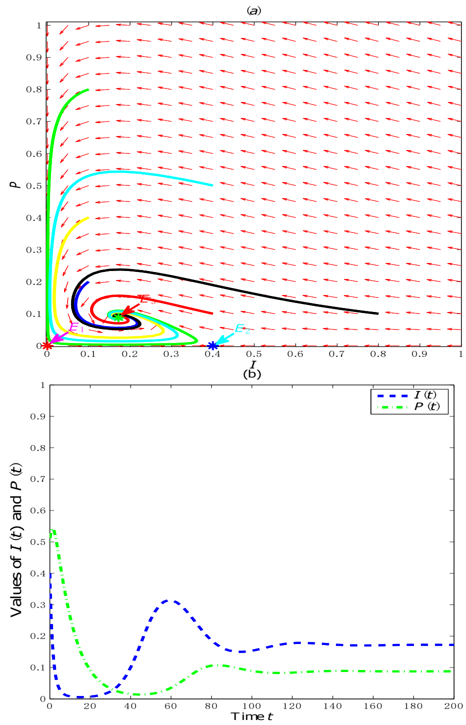

Fig. 7. The numerical computing results for the system given in Example 3.

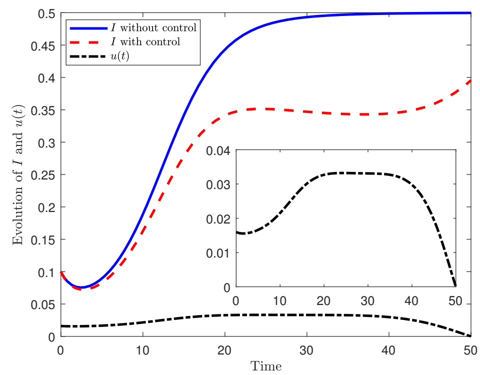

Fig. 8. The comparison of the system with optimal control and without control in Example 4.

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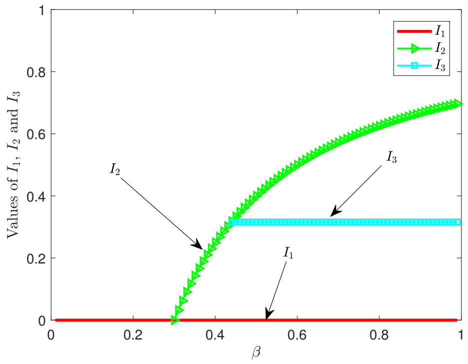

Fig. 9. The values of I_1, I_2 and I_3 with respect to β.

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