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OPTIMAL CONTROL ANALYSIS ON A NEW COMPUTER VIRUS MODEL WITH SATURATED INCIDENCE, VACCINATION, AND TREATMENTS

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Abstract: The mathematical model of computer virus infection with new saturated incidence, vaccination and nonlinear treatments is a new issue addressed in this study. This paper deals with comprehending how the two treatments, vaccination in susceptible classes (antivirus installation) and treatment in infected classes (reinstall operation system), affect the spread of the virus on the network. The investigation of the basic reproduction number and the best treatment control showed that combined treatments produced the best performance. Some numerical examples support the suggested results.

Keywords: computer virus model; saturated incidence; vaccination; anti virus installation; reinstall; control optimal.

2020 AMS Subject Classification: 12H20, 34H05.

1. INTRODUCTION

Mathematical modelling in epidemiology has been developed into a potent and vital tool to understand infectious computer network virus dynamics better and improve population infection

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management. The concept for developing these models comes from the similarity between biological viruses and computer viruses. A mathematical model that can predict any potential virus outbreak and successfully slow its spread constitutes a good model.

The epidemic model that is often used to study computer virus spread is the SIR model (see [3, 21, 25, 18]). Computers on the internet are divided into three categories: S or susceptible computers (those vulnerable to virus infection), I or infected computers, and R or recovered computers. Susceptible computers had a likelihood of getting tainted by infected computers. Users of contaminated computers became mindful of the infection and had a likelihood of changing their computers back into vulnerable ones by introducing an antivirus program or reinstalling the operating system. After receiving treatment, an infected computer becomes a recovered computer. Further research by adding delay time to the model. This delay may reflect the time it takes for a virus to spread from one device to another or the time it takes to detect and respond to an infection see [1, 13, 9, 22, 24, 21, 12, 14]. Another model is the stochastic model see [19, 3]. These studies understand the unpredictable nature of virus propagation through networks.

In the field of applied science, there have recently been numerous infectious disease models with pulse vaccination see [17] and [20]. The epidemic model introduces the concept of vaccination. Individuals who are vaccinated have immunity so they are not infected see [17] and [20]. In the model for spreading computer. viruses, the concept of vaccination has also been introduced with the addition of anti-virus which will change a susceptible computer into a recovered computer. The concept of impulsive vaccination is being employed to prevent the spread of viruses see [19, 3].

The incidence rate is also important factor to consider while analyzing the spread of infection. Depending on how the disease spreads on epidemic model the bi-linear incidence rate see [10] is the simplest. To include the behavioral change and crowding effect of infected individuals, the saturated incidence rate see [2] introduced

$$\frac{\beta I(t)}{1+\alpha I(t)}, \tag{1}$$

where β is transmission rate and α is the inhibitory coefficient. This incidence rate is an

increasing function of S as well as I , and by this incidence rate, the total growth of the infected population is less than the standard incidence. This type of infection shows that epidemic (taking appropriate preventive measures and awareness) and the rate of infection decreases as the inhibitory coefficient α increases.

The development of computers nowadays is increasingly sophisticated, including in protecting against viruses, then this research uses infection rates with non-linear saturation modification Eq. (1). We apply two concepts of saturation in infection and treatment patterns. The spread of computer viruses is due to an inhibitory coefficient which indicates that the computer has a built-in anti-virus. To describe the saturation phenomenon of the limited medical resources [17] recently presented a continuously differentiable treatment function (1). In addition, in the treatment of infected computers considering the limited resources the treatment of infected computers cannot be handled at all, this model uses the concept of non-linear saturation Eq. (1).

Moreover, we are conscious that controlling computer viruses through vaccination by anti virus installation and treatment by reinstalling the operating system is essential. In this research, we attempt to demonstrate how impulsive vaccination, non-linear treatments, and the new saturation effect restrict virus propagation over networks in light of the discussion above. Organization of the paper is as follows. We formulated the new model in section mathematical model. Furthermore, we discussed the boundedness of the solutions, the equilibria and basic reproduction number in the Boundedness of Solutions, Equilibria and the Basic Reproduction Number section. Moreover, the optimal control section describes the characterization of optimal control. In addition, the numerical simulations are given in the numerical simulations section, and the final section provides the conclusions and future research.

2. RESULTS AND DISCUSSION

2.1. Mathematical Model. We govern a mathematical model with a non-linear saturation effect and impart treatments. Let $S(t), I(t)$ and $R(t)$ are the number of susceptible, infected, recovered computers at time t , respectively. For our objectives, the following assumptions are

proposed.

1. Every computer is susceptible whenever it is accessed to the internet. And susceptible computers access the internet with constant rate $A > 0$.
2. Every computer leaves the network with constant rate $\theta > 0$ is the level of nodes whose lifetime has expired.
3. Every susceptible computer is infected, where $\beta > 0$ and $\alpha > 0$ are constants. We modify saturation rate Eq. (1). The rate of the infected nodes is

$$\frac{\beta I(t)}{(1+\alpha I(t))^2},$$

β is transmission rate from susceptible to infected and α is the inhibitory coefficient of infection. This means that the measure of the resilience of infected nodes comes from the influence of the behavior of susceptible nodes when the number of infected nodes increases. In addition, if there are enough infected nodes, the population will experience a decrease in the number of contacts per unit time. If many computers are infected, susceptible computers will have less contact with infected computers because computers are getting smarter nowadays.

4. γ is the rate at which nodes move from infected to recovered class because they have received treatment from the user (for example scanning with an antivirus). δ is the rate of nodes that leave the infected class because the nodes are infected, have been treated by the user but still cannot return to the computer network.
5. Furthermore, we give u_1 is vaccination means giving anti virus extra to the susceptible computers. While u_2 is the treatment to the infected computers. r is rate of treatments and b is limitation rate of treatment resources. We modify saturation rate of Eq.(1) for limitation resources, then u_2 cure rate is

$$\frac{ru_2(t)I(t)}{(1+bu_2(t)I((t))^2},$$

As a result, we purpose a new mathematical model as follows

$$(2)\qquad \begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = A - \dfrac{\beta S(t)I(t)}{\big(1+\alpha I(t)\big)^2} - \theta S(t) - u_1(t)S(t) \\ \dfrac{\mathrm{dI}(t)}{\mathrm{d}t} = \dfrac{\beta S(t)I(t)}{\big(1+\alpha I(t)\big)^2} - (\theta+\delta+\gamma)I(t) - \dfrac{ru_2(t)I(t)}{(1+bu_2(t)I((t))^2} \\ \dfrac{\mathrm{dR}(t)}{\mathrm{d}t} = \dfrac{ru_2(t)I(t)}{(1+bu_2(t)I((t))^2} + \gamma I(t) + u_1(t)S(t) - \theta R(t) \end{cases}$$

where $0 \le u_1(t), u_2(t) \le 1$. Furthermore, this model is a development of a [11]. Further, we assign the upper bound of system (1).

2.2. Boundedness of Solutions, the Equilibria and the Basic Reproduction Number. We indicate that the population is finite overall by denoting a limiting solution for the entire population. The bounded solutions, virus-free equilibrium points and their stability, and fundamental reproduction numbers are all described in the inner section. We denote the limit solution for the entire population and then imply population as a whole is finite. Now, $N(t)$ is the total populations, where $N(t) = S(t) + I(t) + R(t)$, as follows

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \frac{\mathrm{d}S(t)}{\mathrm{d}t} + \frac{\mathrm{dI}(t)}{\mathrm{d}t} + \frac{\mathrm{dR}(t)}{\mathrm{d}t},$$
$$= A - \theta\big(S(t)+I(t)+R(t)\big) - \delta I(t) = A - \theta N(t) - \delta I(t) \le A - \theta N(t)$$

For $t \to +\infty$, we have

$$\lim_{t\to+\infty} N(t) \le \frac{A}{\theta},$$

Moreover, we will determine the virus-free equilibrium point for system (2), while the endemic virus equilibrium point will be determined numerically in the next section. The virus-free equilibrium point will be obtained for $\bar{I} = 0$, then first equation of system (2) as follows.

$$A - \theta\bar{S} - u_1\bar{S} = 0 \to \bar{S} = \frac{A}{\theta + u_1}.$$

While the third equation of the system (2) becomes

$$u_1\bar{S} - \theta\bar{R} = 0 \to \bar{R} = \frac{u_1\bar{S}}{\theta}.$$

So, we get

$$\bar{R} = \frac{u_1\bar{S}}{\theta} = \frac{u_1\left(\frac{A}{\theta+u_1}\right)}{\theta} = \frac{Au_1}{\theta(\theta+u_1)}.$$

The virus-free equilibrium point is $(\bar{S},\bar{I},\bar{R}) = \left(\frac{A}{d+u_1}, 0, \frac{Au_1}{\theta(d+u_1)}\right)$.

Moreover, we analyze the stability of the linearized system around free equilibrium point through the Jacobian matrix as follows.

$$P = \begin{pmatrix} \beta S - \theta - u_1 & -\beta S & 0 \\ \beta S & \beta S - (\theta+\delta+\gamma) - ru_2 & 0 \\ u_1 & ru_2 + \gamma & -\theta \end{pmatrix}.$$

Local stability around the free equilibrium point is determined by examining the real part of eigen values matrix P as follows.

$$\begin{vmatrix} \beta S - \theta - u_1 - \lambda & -\beta S & 0 \\ \beta S & \beta S - (\theta+\delta+\gamma) - ru_2 - \lambda & 0 \\ u_1 & ru_2 + \gamma & -\theta - \lambda \end{vmatrix} = 0$$

$$(-\theta-\lambda)\begin{vmatrix} \beta S - \theta - u_1 - \lambda & -\beta S \\ \beta S & \beta S - (\theta+\delta+\gamma) - ru_2 - \lambda \end{vmatrix} = 0$$

$$(-\theta-\lambda)\big((\beta S - \theta - u_1 - \lambda)(\beta S - (\theta+\delta+\gamma) - ru_2 - \lambda) + (\beta S)^2\big) = 0$$

$$(-\theta-\lambda)(\lambda^2 + a_1\lambda + a_2) = 0, \quad (3)$$

where

$$a_1 = ru_2 + \theta + \delta + \gamma + \beta S + u_1 + \theta - 1$$

$$a_2 = 2(\beta S)^2 - \beta S(\delta+\theta+\gamma) - \beta S r u_2 - \beta S - \beta S\theta - \theta(\delta+\theta+\gamma) + \theta r u_2 - u_1\beta S + u_1(\delta+\theta+\gamma) + ru_1u_2,$$

Following equation (3), System (2) is asymptotic stable for

$$\lambda_1 = -\theta < 0.$$

By Routh Hurwitz table as follows

$a_0 = 1$	a_2
a_1	

System (2) will be asymptotic stable when the first column has the same sign. Because the value of $a_0 = 1 > 0$ (positive), so a_1 must be positive. In other words, we say

$$ru_2 + \theta + \delta + \gamma + \beta S + u_1 + \theta > 1.$$

Furthermore, we determine the basic reproduction number of system (2), which is a level of the secondary virus infection rate. R_0 is the largest eigenvalue of the next-generation matrix, where $R_0 = FV^{-1}$ is a representation matrix that adds infected classes, while V is a matrix that reduces

infected classes.

$$R_0 = FV^{-1} = \frac{\beta A}{(\theta + u_1)(\theta + \delta + \gamma + ru_2)}.$$

When $R_0 < 1$ indicates that the computer network will be free of viruses. However, when $R_0 > 1$, the computer network will be endemic. For a geometric interpretation regarding the spread of viruses on computer networks by considering the value of R_0 is shown in Figure 1. This means that there are infected nodes, then these two nodes transmit to other nodes, and so on.

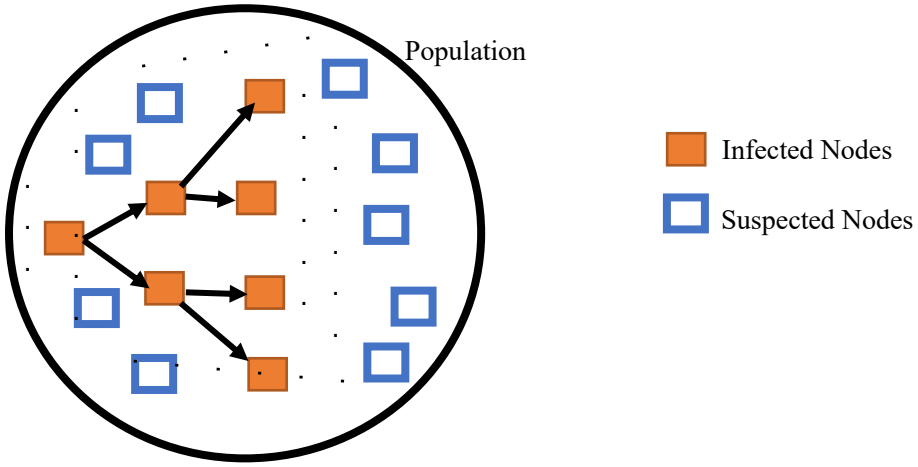

Figure 1. Basic Reproduction Number

2.3. Control Optimal Analyze. In this model, we have considered two controls, one control variable is used for vaccinating the susceptible populations (u_1 is extra anti virus installations) and other control variable is used for treatment efforts for infected individuals (u_2 is reinstalling operating system). We assume that both vaccination and treatment controls are the continuous functions of time t as they are applied according to the necessity. Our main objective is to minimize the total loss occurs due to the presence of infection and the cost due to vaccination of susceptible individuals and treatment of infected individuals. Thus, the strategy of the optimal control is to minimize the susceptible and infected individuals as well as the cost of implementing the two controls. Thus, we construct the objective functional to be minimized as follows:

$$J(u_1.u_2) = \int_0^T \left(B_1 S + B_2 I + \frac{B_3}{2}u_1^2 + \frac{B_4}{2}u_2^2\right) dt,$$

where $0 \le u_1, u_2 \le 1$, u_1, u_2 are Lebesgue Integrable with

$$U = \{(u_1(t), u_2(t) | 0 \le u_1, u_2 \le 1, t \in [0, T])\}.$$

The problem is to find optimal functions (u_1^*, u_2^*), such that

$$J = \min\{J(u_1, u_2) \text{ with } (u_1, u_2) \in U\}.$$

where the constants B_1 and B_2 are respectively the per capita loss due to presence of susceptible and infected population at any time instant. Also, the constants B_3 and B_4, respectively, represent the costs associated with vaccination (costs to anti virus extra installation) and treatment of infected individuals. The Lagrangian of the problem is given by

$$L = B_1 S + B_2 I + \frac{B_3}{2} u_1^2 + \frac{B_4}{2} u_2^2.$$

Furthermore, we govern the Hamiltonian H as follows

$$\begin{aligned} H(S, I, R, u_1, u_2, \lambda_S, \lambda_I, \lambda_R) &= B_1 S + B_2 I + \frac{B_3}{2} u_1^2 + \frac{B_4}{2} u_2^2 \\ &+\lambda_S \left(A - \frac{\beta S(t) I(t)}{\left(1 + \alpha I(t)\right)^2} - \theta S(t) - u_1(t) S(t) \right) \\ &+\lambda_I \left(\frac{\beta S(t) I(t)}{\left(1 + \alpha I(t)\right)^2} - (\theta + \delta + \gamma) I(t) - \frac{r u_2(t) I(t)}{\left(1 + b u_2(t) I(t)\right)^2} \right) \\ &+\lambda_R \left(\frac{r u_2(t) I(t)}{\left(1 + b u_2(t) I(t)\right)^2} + \gamma I(t) + u_1(t) S(t) - \theta R(t) \right). \end{aligned}$$

In order to determine the adjoint equations and transversally conditions, we use Pontryagins Maximum Principle refer to [17] and [20] which gives

$$\frac{\mathrm{d}\lambda_S(t)}{\mathrm{d}t} = -\frac{\partial H}{\partial S}, \quad \frac{\mathrm{d}\lambda_I(t)}{\mathrm{d}t} = -\frac{\partial H}{\partial I}, \quad \frac{\mathrm{d}\lambda_R(t)}{\mathrm{d}t} = -\frac{\partial H}{\partial R}.$$

We have as follows

$$\begin{cases} \dfrac{\mathrm{d}\lambda_S}{\mathrm{d}t} = -B_1 - \lambda_S \left(\dfrac{\beta I}{(1 + \alpha I)^2} + \delta + u_1 \right) - \lambda_I \left(\dfrac{\beta I}{(1 + \alpha I)^2} \right) - \lambda_R u_1 \\ \dfrac{\mathrm{d}\lambda_I}{\mathrm{d}t} = -B_2 - \lambda_S \left(\dfrac{\beta S (1 - (\alpha I)^2)}{(1 + \alpha I)^4} \right) \\ \quad +\lambda_I \left((\theta + \delta + \gamma) + \dfrac{r u_2 (1 - (b u_2 I)^2)}{(1 + b u_2 I)^4} - \dfrac{\beta S (1 - (\alpha I)^2)}{(1 + \alpha I)^4} \right) \\ \quad -\lambda_R \left(\dfrac{r u_2 (1 - (b u_2 I)^2)}{(1 + b u_2 I)^4} + \gamma \right) \\ \dfrac{\mathrm{d}\lambda_R}{\mathrm{d}t} = \theta \lambda_R \end{cases},$$

with the transversally conditions

$$\lambda_S(T) = \lambda_I(T) = \lambda_R(T) = 0.$$

Now, using the optimality conditions

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

So, we get

$$\frac{\partial H}{\partial u_1} = 0$$
$$B_3\overline{u_1} - S\lambda_S + S\lambda_R = 0$$
$$\overline{u_1} = \frac{S(\lambda_S + \lambda_R)}{B_3},$$

Furthermore, the optimum of u_1 as follows

$$u_1^* = \max\left\{0, \min\left\{\frac{S(\lambda_S + \lambda_R)}{B_3}, 1\right\}\right\}.$$

While

$$\frac{\partial H}{\partial u_2} = 0$$
$$B_4 u_2 + \frac{rI\lambda_I(bu_2I - 1)}{(1 + bu_2I)^3} + \frac{rI\lambda_R(1 - 3bu_2I)}{(1 + bu_2I)^3} = 0$$

(5) $$c_1u_2^4 + c_2u_2^3 + c_3u_2^2 + c_4u_2 + c_5 = 0,$$

With

$$c_1 = b^3B_4I^3 \qquad c_2 = 3B_4b^2I^2 \qquad c_3 = 3bB_4I$$
$$c_4 = B_4 + rbI^2\lambda_I - 3rbI^2\lambda_R \qquad c_5 = rI(\lambda_R - \lambda_I),$$

Moreover, by using [16], we determine u_2. Equation (5) divide by c_1, we have

(6) $$u_2^4 + f_1u_2^3 + f_2u_2^2 + f_3u_2 + f_4 = 0.$$

where $f_1 = \frac{c_2}{c_1}, f_2 = \frac{c_3}{c_1}, f_3 = \frac{c_4}{c_1}, f_4 = \frac{c_5}{c_1}$. For $u_2 = v_2 - \frac{f_1}{4}$, we have

(7) $$v_2^4 + g_1v_2^2 + g_2v_2 + g_3 = 0.$$

Where

$$g_1 = f_2 - \frac{3f_1^2}{8}, g_2 = f_2 + \frac{f_1^3}{8} - \frac{f_1f_2}{2}, g_3 = f_4 - \frac{3f_2^4}{256} + \frac{f_1^2f_2}{16} - \frac{f_1f_3}{4}.$$

Further more, we factorize equation (6) and transform to equation (7).

$$w_2^3 - f_2 w_2^2 + (f_1 f_3 - 4f_4)w_2 + (4f_2 f_4 - f_3^2 - f_1^2 f_4) = 0.$$

For w_{20} is real root of equation (6) , we have

$$\bar{u}_2^1 = -\frac{f_1}{4} + \frac{1}{2}(A + B)$$
$$\bar{u}_2^2 = -\frac{f_1}{4} + \frac{1}{2}(A - B)$$
$$\bar{u}_2^3 = -\frac{f_1}{4} - \frac{1}{2}(A - C)$$
$$\bar{u}_2^4 = -\frac{f_1}{4} - \frac{1}{2}(A + C),$$

where

$$A = \sqrt{\frac{1}{4}f_1^2 - f_2 + w_{20}}$$

$$B = \begin{cases} \sqrt{\frac{3}{4}f_1^2 - 2f_2 + 2\sqrt{w_{20}^2 - 4f_4}} & , \text{for } A = 0 \\ \sqrt{\frac{3}{4}f_1^2 - A^2 - 2f_2 + \frac{1}{4}(4f_1 f_2 - 8f_3 - f_1^3)A^{-1}} & , \text{for } A \neq 0 \end{cases}$$

$$C = \begin{cases} \sqrt{\frac{3}{4}f_1^2 - 2f_2 - 2\sqrt{w_{20}^2 - 4f_4}} & , \text{for } A = 0 \\ \sqrt{\frac{3}{4}f_1^2 - A^2 - 2f_2 - \frac{1}{4}(4f_1 f_2 - 8f_3 - f_1^3)A^{-1}} & , \text{for } A \neq 0, \end{cases}$$

For $\bar{u}_2$ is positive real root equation (6), we have

$$u_2^* = \max\left\{0, \min\{\bar{u}_2^k, 1\}\right\}, \qquad \text{for } k = 1,2,3,4. \tag{8}$$

Therefore, the optimal problem is minimum at controls u_1^* and u_2^* where

$u_1^* = \max\left\{0, \min\left\{\frac{S(\lambda_S + \lambda_R)}{B_3}, 1\right\}\right\}$, where $\bar{u}_2$ is positive real root equation (8). Here,(S^*, I^*, R^*) are respectively the optimum values of S, I, R and $(\lambda_1^*, \lambda_2^*, \lambda_3^*)$ is the solution of the system (2) with the condition (8).

3. SIMULATION

To establish the effectiveness of optimal control, we have employed a fourth order Runge–Kutta method for the state system (2) and system (4) and the transversely conditions. The simulation which we performed out by utilizing the parameters referred to [11] is given in Table 1 with the initial conditions both around the free equilibrium point (Figure 2) and infected equilibrium point (Figure 3,4,5). We vary the β, which indicates the virus's exposure level. Figure 2 shows a simulation of $R_0 < 1$. These results suggest that the optimal combination of the two treatments causes the network to be free from the spread of the virus with minimal costs.

The second case explains that the primary reproduction number is more than one at a time without control, so the infected equilibrium point is stable. Then there will be the continuous spread of the virus. Figure 3 implements the u_1 control, while Figure 4 implements the u_2 management other Figure 5 implements both u_1 and u_2 control strategies.

Figure 2 show the time series of the susceptible (S), infected (I) and recovered (R) individuals with optimum control both vaccination and treatment. From Fig. 2, we see that optimal controls due to vaccination and treatment are very effective for reducing the number of susceptible and infected individuals and so enhancing the number of recovered individuals significantly. In this paper, we have considered two controls, one is vaccination control u_1 and other is treatment control u_2.

Table 1. Parameters value

Notation	The parameter on free equilibrium point	Ref	The parameter on endemic equilibrium point	Ref
A	10	Assumption	100	[11]
β	0.001	Assumption	0.1	[11]
θ	0.004	[11]	0.004	[11]
δ	0.8	Assumption	0.02	[11]
γ	0.7	[11]	0.7	[11]
r	0.4	[11]	0.4	[11]
α	0.5	[11]	0.5	[11]
b	0.5	[11]	0.05	Assumption
u_1^*	0.19	Calculation	0.19	Calculation
u_2^*	0.2625	Calculation	0.2625	Calculation

Based on table (1) second column, we have a basic reproduction number $R_0 = 0.46$. This means that infected nodes will not infect other nodes then the virus will disappear from the population. Meanwhile, the virus-free equilibrium point is $(37.52, 0, 2462.34)$.

Furthermore, from the characteristic equation in Equation (3), obtained

$$(-0.004 - \lambda)(\lambda^2 + 0.8840\lambda + 0.3050) = 0$$

$$\lambda_1 = -0.004 \quad \text{or} \quad \lambda_2 = -0.442 + 0.331i \quad \text{or} \quad \lambda_3 = -0.442 - 0.331i.$$

Since real part of $\lambda_1, \lambda_2,$ dan λ_3 are negative, then the free equilibrium point is asymptotic stable.

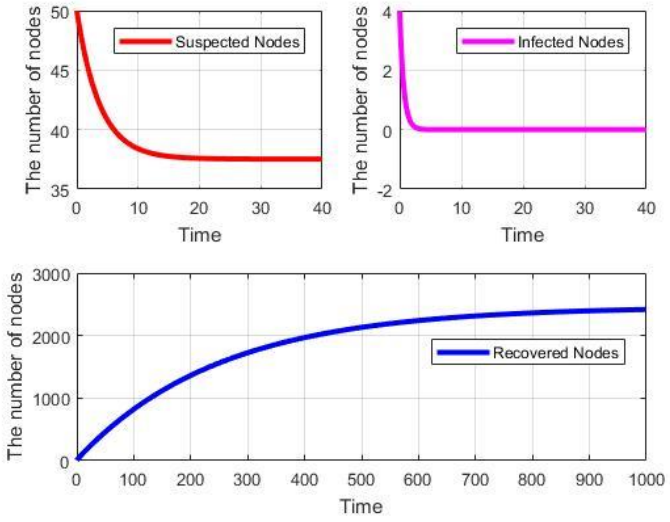

Figure 2. Simulation of free virus equilibrium point

The number of susceptible nodes to viruses over time continues to decrease towards to 37.52 nodes, following the analytical calculations that have been carried out. Meanwhile, the number of infected nodes continues to decline and converge to 0. This indicates that there are no virus-infected nodes in a computer network. In contrast, this situation differs from the number of nodes in the recovered class, which continues to increase, and then converges to 2462.34 nodes. Furthermore, the simulations carried out are under analytical calculations.

Figure 2 shows a scenario where the virus spreads uncontrolled; we examine the impact of the two controls and simulate the optimal control value determined by the calculations in the preceding section. The parameter value pertains to column three of table (x), where the basic reproduction number is 46.90. One afflicted node can infect as many as 46 to 47 more nodes. Additionally,

46 to 47 nodes will become infected by those 46 or 47 nodes. Furthermore, we calculate the eigenvalues to determine the stability of the linearized system using equation (3). The characteristic equation is as follows.

$$(-0.004 - \lambda)(\lambda^2 + 37.089\lambda + 2738.684) = 0$$

$$\lambda_1 = -0.004 \quad \text{or} \quad \lambda_2 = -18.7949 + 48.840i \quad \text{or} \quad \lambda_3 = 18.7949 - 48.840i.$$

The endemic virus equilibrium point is locally asymptotically stable because all real elements of $\lambda_1, \lambda_2,$ dan λ_3 are negative. Furthermore, the number of nodes from vulnerable, infected, and recovered classes over time will have a specific value. Moreover, we carry out three strategies to determine effectiveness of the control variables.

First Strategy. Installing IDS and IPS on a computer network, but when there is an infected computer, it is not uninstalled. Applying a vaccination strategy without curing the infected.

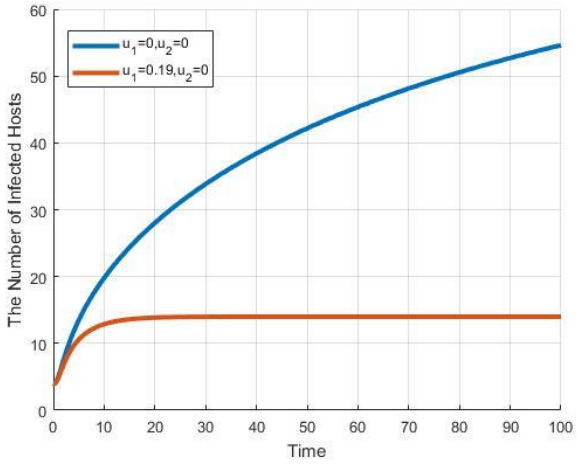

Figure 3. Simulation of infected populations with First Strategy

Figure 3 shows that installing IDS and IPS is effective enough to control computer network malware. Two plots of graphs show that without control $(u_1 = u_2 = 0)$ and with control $u_1 = 0.19$ dan $u_2 = 0$. The number of infected nodes experienced a significant increase when uncontrolled. However, in the controlled model, at the initial time, it increases and then stabilizes towards a particular value. The model with control more realistic conclusions because the number of infected nodes is quite a bit compared to the model without control.

Second Strategy. Not installing IDS and IPS on a computer network but reinstalling infected computers. Treating the infected but without vaccination strategy.

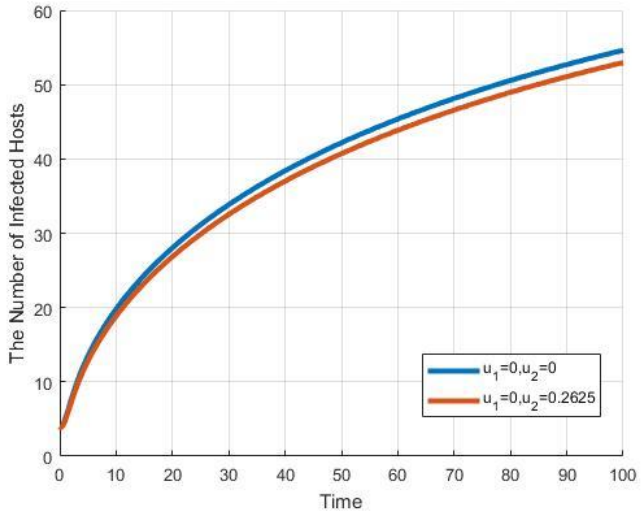

Figure 4. Simulation of Infected Populations with Second Strategy

Giving a control treatment of 0.2625, as shown in Figure 4, has little impact on the number of infected nodes. The number of nodes in both control and uncontrol has steadily expanded. Healthy computers revert to susceptibility and become infected.

Third Strategy. Install IDS and IPS on a computer network and reinstall an infected computer.

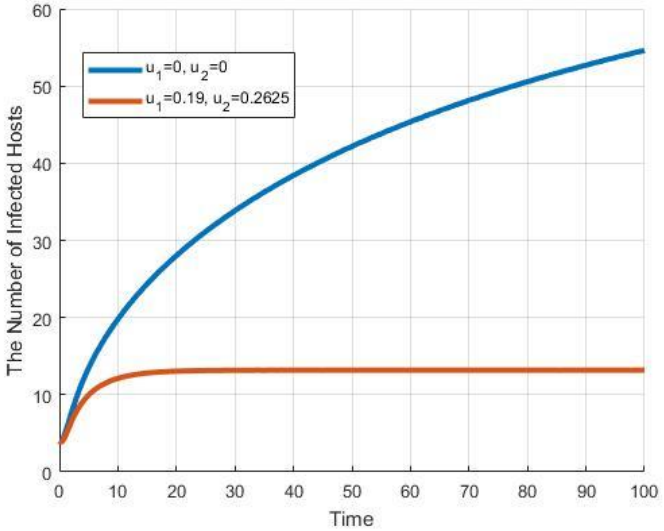

Figure 5. Simulation of infected populations with Strategi III

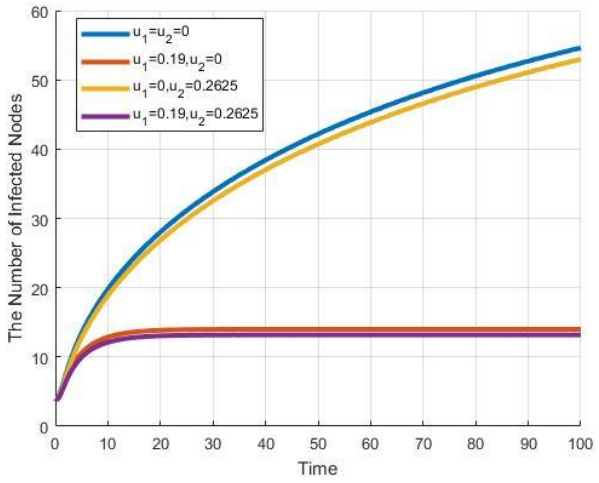

Figure 6. Simulation of infected populations with all strategy compared to uncontrol

Figure 5 shows the third strategy, installing IPS and IDS and reinstalling the infected computer $u_1 = 0.19, u_2 = 0.2625$, which did not significantly reduce the infected population compared to the first strategy (installing IPS and IDS, $u_1 = 0.19, u_2 = 0$) but was significantly different compared to uncontrol ($u_1 = 0, u_2 = 0$). This shows that vaccination is very important to reduce the infected population. Figure 6 supports that reinstalling the infected computer is not enough to prevent the virus from spreading in the network. The provision of antivirus plays an essential role in the spread of computer viruses in the network. Installing an antivirus accompanied by treating an infected computer can slightly reduce the spread of the virus compared to just installing an antivirus. This situation occurs in cases where the basic reproduction number is more than one, meaning that the infected equilibrium point is stable and the virus spreads. Although both antivirus and reinstalled infected computers, the virus still spread in reality. A non-linear model with vaccination and saturation can model this phenomenon. Installing an antivirus can significantly reduce infected computers, referring to the parameter data in Table 1.

5. CONCLUSIONS

This study applies the vaccination and special saturated function in modelling the infection of virus in computer network. We intend to emphasize that the virus in computer networks can be significantly decreased by vaccination. Curing an infected computer is not enough to get a healthy

computer network. So this model is suitable for reality. We apply optimal control to get a recommended treatment. We compare the first strategy, second strategy, and third strategy. Based on the analysis results, we conclude that installing the anti virus provides an effective treatment for a good computer network. Based on the results and discussion, we suggest developing a method that considers the delay on the computer network. We can consider implementing and developing the delay model because virus screening in the susceptible group by anti virus takes time. This filter determines whether the computer enters recovery or is infected. This time is mathematically the time delay, which in this study was still ignored.

AUTHOR CONTRIBUTIONS

Authors have read and approved the final version of the manuscript. The authors contributed equally to this work.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interest.

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