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Stability analysis of a worm propagation model with partial immunization

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ABSTRACT

Internet worm attacks the Internet infrastructure, reduces network security and causes economic losses. In order to effectively defend against worms, this paper proposes a novel epidemic SVEIR model with partial immunization. Using this SVEIR model, we obtain the basic reproduction number for determining whether the worm dies out completely. The global stability of worm-free equilibrium is proven using a Lyapunov function. By the use of Hurwitz criterion, the local stability of the unique endemic equilibrium is proven. The impact of different parameters of this model is studied. Simulation results show that the number of susceptible and infected hosts is consistent with theoretical analysis. The model provides a theoretical foundation for control and forecasting Internet worms.

KEYWORDS

Network security; Internet worm; Stability analysis; Endemic equilibrium; Partial immunization.

INTRODUCTION

Internet worms are malicious codes which can replicate themselves and propagate via Internet. With the ever increasing number of Internet applications and the emergence of new technologies, Internet worms have become a great threat to our work and daily life, caused tremendous economic losses. Especially, the advent of the Internet of things would make the threat increasingly serious. How to combat Internet worms effectively is an urgent issue confronted with defenders. Therefore, it is necessary to comprehend the long-term behaviour of worms and to propose effective strategies to defend against Internet worms.

Based on the similarity between a malicious worm and a biological virus, some mathematical models representing worm propagation were presented to depict and model the propagation of worms in the past decade years. In order to depict and defend against worms, researchers have presented some models [1-8] based on epidemiology. These models assume that exposed hosts can not infect other hosts. Actually, an infected host which is in latency can infect other hosts by means of some methods, e.g., vulnerability seeking. The previous models do not take this passive infectivity into consideration. Immunization is one of commonly used method for controlling the propagation process of worms. Some epidemic models with immunization have been proposed [9, 10]. However, these models all assumed that the vaccine hosts obtained the immunization fully. This is not consistent with the reality. In real networks, it is very difficult to obtain the immunization fully for the vaccine hosts. Thus, partial immunization should be a fungible and feasible method for eliminating worms, which have been used for predicting and controlling infectious diseases [11, 12]. This paper proposes a new worm attack model, referred to as SVEIR (susceptible-vaccinated-exposed-infectious-recovered) model, which is appropriate for measuring the effects of security countermeasures on worm propagation. Contrary to existing model, our model takes the partial immunization and two infection rates. Using the reproduction number, we derive global stabilities of the worm-free equilibrium and endemic equilibrium. Furthermore, simulation results show the effectiveness of our model. Finally, equilibrium points are confirmed by plots.

MATHEMATICAL MODEL FORMULATION

The total host population *N* is partitioned into five groups and any host can potential be in any of these groups at any time *t*: the susceptible, vaccinated, exposed, infectious, recovered, with sizes denoted by *S*, *V*, *E*, *I*, *R*, respectively. The total number of population *N* at time *t* is given by $N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$. The dynamical transfer of hosts is depicted in the following figure.

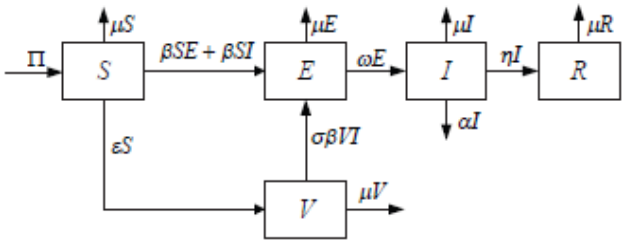

Figure 1 : Schematic diagram for the flow of worms

Fig. 1 shows the five states and state transition in *SVEIR*. Based on the compartment model presented in Fig. 1, the *SVEIR* model having infectious force in the exposed, infected period and partial immunization is described by the following system of differential equations:

$$\begin{cases} S'(t) = \Pi - \beta SE - \beta SI - (\mu + \varepsilon)S, \\ V'(t) = \varepsilon S - \sigma\beta VI - \mu V, \\ E'(t) = \beta SE + \beta SI + \sigma\beta VI - (\mu + \omega)E, \\ I'(t) = \omega E - (\mu + \alpha + \eta)I, \\ R'(t) = \eta I - \mu R, \end{cases} \quad (1)$$

where Π is a constant recruitment of susceptible hosts. Let β be the transmission rate of worm attack when susceptible hosts contact with infected ones. The positive parameter μ is the rate of natural death, α are non-negative constant and denote the rate of worm-caused death. The vaccinated hosts which contact infected ones before obtaining immunization have the infection probability with a transmission rate $\sigma\beta$ $(0 \le \sigma\beta \le 1)$. $\sigma = 0$ means that the vaccinated hosts obtain the full immunization, $\sigma = 1$ means that vaccine loses efficacy in work fully. Taking some real factors into account, we assume that the vaccinated hosts can obtain partial immunization, that is to say, $0 < \sigma < 1$. ε is the transfer rates between the susceptible and the vaccinated. ω is the rate at which exposed hosts become infectious, η is the recovered rate of infected hosts.

Since the state R does not appear explicitly in the first four equations in the system (1), the dynamics of (1) is the same as the following system:

$$\begin{cases} S'(t) = \Pi - \beta SE - \beta SI - (\mu + \varepsilon)S, \\ V'(t) = \varepsilon S - \sigma\beta VI - \mu V, \\ E'(t) = \beta SE + \beta SI + \sigma\beta VI - (\mu + \omega)E, \\ I'(t) = \omega E - (\mu + \alpha + \eta)I. \end{cases} \quad (2)$$

Summing the equations of the system (2), we obtain $N'(t) = \Pi - \mu N - \alpha I$.

Therefore, the total population N may vary with time t. In the absence of disease, the total population size $N(t)$ converges to the the equilibrium Π / μ. We thus study our system (2) in the following feasible region: $\Omega = \{(S,V,E,I) \in R_+^4 : S + V + E + I \le \Pi / \mu\}$, which is a positively invariant set of the model (2). We next consider the dynamic behaviour of the model (2) on Ω.

Firstly, we obtain the basic reproduction number of the model (2) by the method of next generation matrix [13]. It is easy to see that the model (2) always has a worm-free equilibrium, $P_0 = (\Pi/(\mu+\varepsilon), \varepsilon\Pi/(\mu(\mu+\varepsilon)),0,0)$.

Let $x = (E, I, V, S)$, then the model (2) can be written as

$$\frac{dx}{dt} = F(x) - V(x),$$

where

$$F(x) = \begin{pmatrix} \beta SE + \beta SI + \sigma\beta VI \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad V(x) = \begin{pmatrix} (\mu + \omega)E \\ (\mu + \alpha + \eta)I - \omega E \\ -\varepsilon S + \sigma\beta VI + \mu V \\ \beta SE + \beta SI + (\mu + \varepsilon)S - \Pi \end{pmatrix}.$$

Differentiating $F(x)$ and $V(x)$ with respect to E, I, V, S and evaluating at the worm-free equilibrium $P_0 = (\Pi/(\mu+\varepsilon), \varepsilon\Pi/(\mu(\mu+\varepsilon)),0,0)$, respectively, we have

$$DF(P_0) = \begin{pmatrix} \beta S_0 & \beta S_0 + \sigma\beta V_0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad DV(P_0) = \begin{pmatrix} \mu + \omega & 0 & 0 & 0 \\ -\omega & \mu + \alpha + \eta & 0 & 0 \\ 0 & \sigma\beta V_0 & 0 & -\varepsilon \\ \beta S_0 & \beta S_0 & 0 & \mu + \varepsilon \end{pmatrix},$$

Thus, the spectral radius of the next generation matrix FV^{-1} can be found as,

$$\rho(FV^{-1}) = \frac{\beta S_0(\mu + \alpha + \eta) + \omega(\beta S_0 + \sigma\beta V_0)}{(\mu + \omega)(\mu + \alpha + \eta)}.$$

According to Theorem 2 in [13], the basic reproduction number of model (2) is

$$R_0 = \frac{\beta S_0(\mu + \alpha + \eta) + \omega(\beta S_0 + \sigma\beta V_0)}{(\mu + \omega)(\mu + \alpha + \eta)} = \frac{\beta\Pi[(\mu + \alpha + \eta) + \omega(\beta S_0 + \sigma\beta V_0)]}{\mu(\mu + \omega)(\mu + \alpha + \eta)}. \quad (3)$$

The endemic equilibrium $P^*(S^*, V^*, E^*, I^*)$ of the model (2) is determined by the following equations

$$\begin{cases} \Pi - \beta SE - \beta SI - (\mu + \varepsilon)S = 0, \\ \varepsilon S - \sigma\beta VI - \mu V = 0, \\ \beta SE + \beta SI + \sigma\beta VI - (\mu + \omega)E = 0, \\ \omega E - (\mu + \alpha + \eta)I = 0, \\ \Pi - \mu N - \alpha I = 0. \end{cases} \quad (4)$$

By the direct computation, we obtain $S^* = \Pi/(\beta E^* + \beta I^* + \mu + \varepsilon)$, $V^* = \varepsilon S^*/(\sigma\beta I^* + \mu)$, $E^* = (\mu + \alpha + \eta)I^*/\omega$, $I^* = (\Pi - \mu N)/\alpha$. Once these constant parameters are given, S^*, V^*, E^*, I^* are all determined. Thus, $P^*(S^*, V^*, E^*, I^*)$ is the unique endemic equilibrium of the model (2).

THE GLOBAL STABILITY OF THE WORM-FREE EQUILIBIRUM

It is easily obtained that the model has a worm-free equilibrium given by $P_0 = (\Pi/(\mu+\varepsilon), \varepsilon\Pi/(\mu(\mu+\varepsilon)),0,0)$.

Lemma 1: When $R_0 < 1$, the worm-free equilibrium P_0 is locally asymptotically stable. When $R_0 > 1$, the worm-free equilibrium P_0 is an unstable saddle point.

Proof: The Jacobian matrices of the model (2) at P_0 is

$$J(P_0)=\begin{pmatrix} -(\mu+\varepsilon) & 0 & -\beta S_0 & -\beta S_0 \\ \varepsilon & -\mu & 0 & -\sigma\beta V_0 \\ 0 & 0 & \beta S_0-(\mu+\omega) & \beta S_0+\sigma\beta V_0 \\ 0 & 0 & \omega & -(\mu+\alpha+\eta) \end{pmatrix}.$$

It is easily obtained that $J(P_0)$ has two negative eigenvalues $\lambda_1=-\mu$, and $\lambda_2=-(\mu+\varepsilon)$, the other eigenvalues of $J(P_0)$ are determined by the following equation

$$\lambda^2+(\mu+\omega-\beta S_0+\mu+\alpha+\eta)\lambda+(\mu+\omega-\beta S_0)(\mu+\alpha+\eta)-\omega(\beta S_0+\sigma\beta V_0)=0. \quad (5)$$

When $R_0<1$, then $(\mu+\alpha+\eta)(\mu+\omega)-\beta S_0(\mu+\alpha+\eta)-\omega(\beta S_0+\sigma\beta V_0)>0$, two roots of Eq, (5) are negative. The four-dimensional model to be asymptotically stable is that $\lambda_i<0$, for $i=1,2,3,4$, which meets the sufficient condition of the stability theory [14]. When $R_0>1$, $(\mu+\alpha+\eta)(\mu+\omega)-\beta S_0(\mu+\alpha+\eta)-\omega(\beta S_0+\sigma\beta V_0)<0$, which means that $J(P_0)$ has a positive root and a negative root. Therefore, the worm-free equilibrium P_0 is an unstable saddle point. This completes the proof.

Lemma 2: When $R_0\le 1$, the worm-free equilibrium P_0 is globally asymptotically stable.

Proof: Consider the following Lyapunov function

$L=\omega E+(\mu+\omega)I$.

Its derivative along the solutions to the model (2) is

$$\begin{aligned} L' &= \omega(\beta SE+\beta SI+\sigma\beta VI-(\mu+\omega)E)-\omega(\mu+\omega)E-(\mu+\omega)(\mu+\alpha+\eta)I \\ &= [\beta S(\mu+\alpha+\eta)+\omega(\beta S+\sigma\beta V)-(\mu+\omega)(\mu+\alpha+\eta)]I \\ &= (\mu+\omega)(\mu+\alpha+\eta)\left(\frac{\beta S(\mu+\alpha+\eta)+\omega(\beta S+\sigma\beta V)}{(\mu+\omega)(\mu+\alpha+\eta)}-1\right)I \\ &= (\mu+\omega)(\mu+\alpha+\eta)(R_0-1)I \quad \le 0. \end{aligned}$$

Furthermore, $L'=0$ if and only if $I=0$ or $R_0=1$. Thus, the largest compact invariant set in $\{(S,V,E,I)\mid L'=0\}$ is the singleton $\{P_0\}$. When $R_0\le 1$, the global stability of P_0 follows from LaSalle's invariance principle [15]. LaSalle's invariance principle [15] implies that P_0 is globally asymptotically stable in Ω. When $R_0>1$, it follows from the fact $L'>0$ if $I>0$. This completes the proof.

THE LOCAL STABILITY OF THE ENDEMIC EQUILIBRIUM

Lemma 3: When $R_0>1$, the endemic equilibrium P^* is locally asymptotically stable.

Proof: The Jacobian matrix of the model (2) at $P^*(S^*,V^*,E^*,I^*)$ is

$$J(P^*)=\begin{pmatrix} -\beta E^*-\beta I^*-\mu-\varepsilon & 0 & -\beta S^* & -\beta S^* \\ \varepsilon & -\sigma\beta I^*-\mu & 0 & -\sigma\beta V^* \\ -\sigma\beta I^*-\mu & \sigma\beta I^* & \beta S^*-(\mu+\omega) & \beta S^*+\sigma\beta V^* \\ 0 & 0 & \omega & -\mu-\alpha-\eta \end{pmatrix}, \quad (6)$$

Therefore, the corresponding characteristic equation can be denoted as

$$\lambda^4+C_1\lambda^3+C_2\lambda^2+C_3\lambda+C_4=0, \quad (7)$$

where, $C_1=4\mu+\alpha+\eta+\varepsilon+\beta E^*+\beta I^*-\beta S^*+\sigma\beta I^*>4\mu+\alpha+\eta+\varepsilon+\sigma\beta I^*>0$,

$$C_2>(2\mu+\alpha+\eta+\omega)(2\mu+\varepsilon+\omega+\sigma\beta I^*)+(\mu+\sigma\beta I^*)(\mu+\varepsilon)>0,$$

$$\begin{aligned} C_3 &> (\mu+\omega)(2\mu+\varepsilon+\sigma\beta I^*)(\mu+\alpha+\eta)+(\mu+\sigma\beta I^*)(\mu+\varepsilon)(\mu+\alpha+\eta)+(\mu+\sigma\beta I^*)(\mu+\omega)(\mu+\varepsilon) \\ &> 0, \end{aligned}$$

$$C_4>(\mu+\alpha+\eta)(\mu+\sigma\beta I^*)(\mu+\omega)(\mu+\varepsilon)+\omega\sigma^2\beta^2V^*I^*(3\mu+\varepsilon+\omega+\sigma\beta I^*)>0,$$

By a direct calculation, we obtain that

$$H_1=C_1>0,\ H_2=C_1C_2-C_3>0,\ H_3=C_3H_2-C_1^2C_4>0,\ H_4=C_4H_3>0.$$

According to the theorem of Routh-Hurwitz, it follows that all the roots of the Eq. (7) have negative real parts. Therefore, the endemic equilibrium P^* is locally asymptotically stable. This completes the proof.

NUMERICAL SIMULATIONS

In this experiment, we choose the Conficker as basic behavior of a worm. The Conficker infected 1,400,000 hosts in 2009 [2], thus we select 1,400,000 hosts as the population size. According to the real conditions of the Conficker worm, its infection rate is $\beta = 1.7 \times 10^{-5}$ [2]. At the beginning, the number of susceptible, vaccinated, exposed, infectious, recovered hosts are $S(0) = 1{,}399{,}998$, $V(0) = 1$, $E(0) = 0$, $I(0) = 1$, $R(0) = 0$, respectively. Other parameters in these simulations are given as follows: $\Pi = 140$, $\mu = 0.0003$, $\omega = 0.2$, $\sigma = 0.4$, $\eta = 0.08$, $\varepsilon = 0.1$.

Using the parameters above, we can obtain $R_0 = 0.7548 < 1$. The worm will gradually disappear according to **Lemma** 2. Fig. 2 illustrates the number of susceptible and infected hosts when R_0 is 0.7548. From Fig. 2, we can clearly see that the tendency of the worm propagation is depressive, which is consistent with **Lemma** 2. Finally, the whole population, in the long term, is in a recovered state. In order to effectively defend against such worms, we must adopt feasible methods to adjust some related parameters, and then guarantee the basic reproduction number $R_0 < 1$.

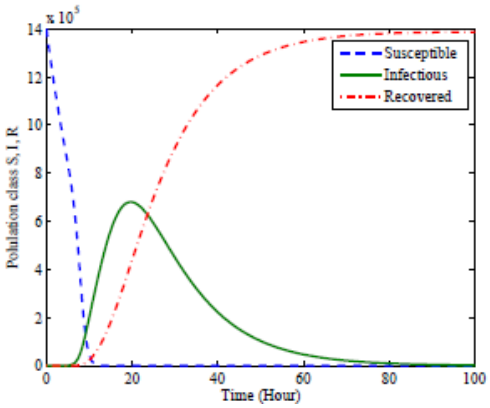

Figure 2 : Globally stable worm-free equilibrium

In the second experiment, when $\omega = 0.7$, we can obtain $R_0 = 4.3541 > 1$. Other parameters do not vary. We can see the results in Fig. 3. As can be seen from Fig. 3, the number of susceptible and infected hosts eventually become positive values between 0 and Π / μ. $S(t)$, $I(t)$ all approach their steady state, and the worm persists. This is fully consistent with the conclusions of **Lemma** 3. Furthermore, we can know that the unique endemic equilibrium is locally asymptotically stable.

With other parameters remaining the same, the partial immunization rate σ is set to different values each time in order to state that the number of infected hosts is affected by every different value of the partial immunization rate. Fig. 4 shows the effect of changing the partial immunization rate (which varies between 0.1 and 0.7) on worm propagations. From Fig. 4, we can see that a smaller partial immunization rate results in diminishing the worm propagation speed, more importantly, lowering the total number of infected hosts, and prolonging the time of reaching their propagation peaks. However, in real-world networks, it is very difficult to implement full immunization. Therefore, in order to eliminate worms as soon as possible, we require the support from all circles of society, which can guarantee to reach a smaller partial immunization rate σ.

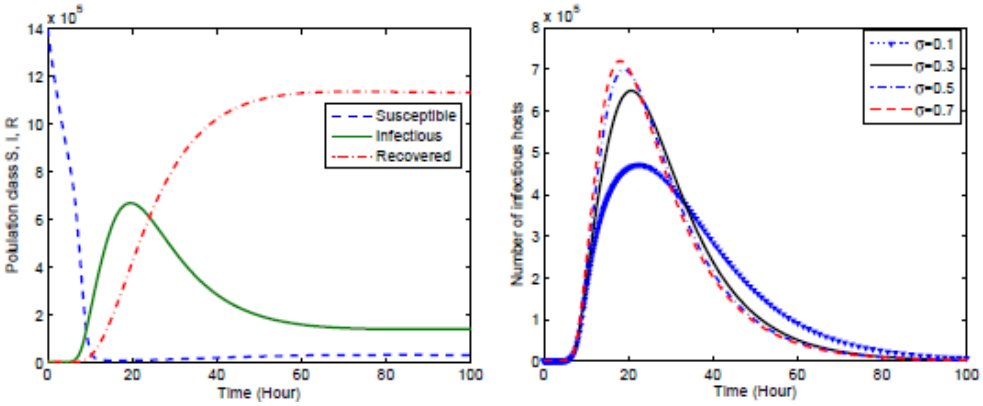

Figure 3 : Locally stable equilibrium equilibrium Figure 4: Effect of partial immunization rate

CONCLUSIONS

This paper presented a mathematical model describing the dynamical behaviour of an SVEIR epidemic model with partial immunization for Internet worms. Firstly, by the method of next generation matrix, we give the basic reproduction number determining whether the worm extinguishes. Secondly, the global stability of worm-free equilibrium and the local stability of endemic equilibrium have proved. When the basic reproduction number is less than and equal to one, the proposed model has only a worm-free equilibrium which is globally stable, it implies the worm dies out eventually; when the basic reproduction number is larger than one, our model has unique endemic equilibrium which is globally stable, it implies that the worm persists in the whole host population and tends to a steady state. Finally, some numerical examples are given to verify our conclusions. Our future work will verify the model by the use of NS2 (Network Simulation version 2) and expand this model proposed in this paper to scale-free networks.

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