An SVEIR Defending Model with Partial Immunization for Worms

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Abstract

Internet worms can propagate across networks horrendously, reduce network security remarkably, and cause economic losses heavily. How to quickly eliminate the Internet worms using partial immunization becomes a big issue for sustaining Internet infrastructure smoothly. This paper addresses this issue by presenting a novel worm attack model through incorporating a saturated incidence rate and a partial immunization rate, named *SVEIR* model. Using the basic reproduction number, we derive the global stability of the infection-free equilibrium and local stability of the unique endemic equilibrium. Numerical methods are employed to solve and simulate the developed system and also verify the proposed *SVEIR* model. Simulation results show that the partial immunization is highly effective for eliminating worms.

Keywords: Internet worm, partial immunization, propagation model, saturated incidence, stability

1 Introduction

Internet worms are malicious codes which can replicate themselves and propagate across the Internet. Code red worm, Slammer worm, Blaster worm, Witty worm, and Conficker worm are a few examples of Internet worms, which have caused heavy economic losses and tremendous social panic. Especially, with the advent and development of the Internet of Things (IoT), the threat of Internet worms will become increasingly serious for network security. Combating worms effectively is an urgent task confronted with defenders. Based on the similarity between a malicious worm and a biological virus, a few mathematical models representing worm propagation have been presented to depict the propagation of worms in the past decade years [4, 9, 13]. Appropriate mathematical models can provide a qualitative assessment for worms' attack. Many numerous models and tools are proposed to address the dynamic attacking behaviour of worms and effectively counterattack them in different conditions, e.g., time delay [15, 18], quarantine [19, 20], antivirus software [21], etc. All the previous models are based on the *SIR* classical epidemic model [7]. The *SIR* has some drawbacks because it assumes that a susceptible host becomes infectious immediately after contact with an infected one. Actually, many worms own an exposed period during which susceptible hosts are infected but not yet contagious. To overcome this drawback, a new model, named as *SEIR* model [1], is introduced. An exposed class is added into the *SEIR* model.

Immunization is one of the commonly used methods for controlling and eliminating worms propagation [5, 6]. However, these models assumed that the vaccine hosts obtained the immunization fully. This is not consistent with the reality. For worms' horrendous propagation speed, users or network administrators can not immunize the whole host population in real networks. Thus, partial immunization as a fungible and feasible method for eliminating worms has been used for predicting and controlling infectious diseases [2, 12]. In many worm propagation models [5, 14], bilinear infection rate βSI is used, where, S and I denote the number of susceptible hosts and infectious hosts, respectively. The saturated infection rate $\frac{\beta SI}{1+\eta I}$ was firstly introduced by Capasso and Seior [3], where $\frac{\beta I}{1+\eta I}$

tends to a saturation level when I becomes large, $\frac{1}{1+\eta I}$ measures the inhibition effect from the behavioral change of susceptible hosts when their number increases or from the crowding effect of the infected hosts. The saturated infection rate $\frac{\beta SI}{1+\eta I}$ is more reasonable than the linear rate βSI. This is due to the fact that it takes the behavioral change and the effect of the infected hosts into consideration.

In this paper, we propose an *SVEIR* model based on the *SEIR* model. Contrary to existing models, the proposed *SVEIR* model is armed with the partial immunization and saturated infection. This paper will argue that *SVEIR* model is appropriate for measuring the effects of security countermeasures on worm propagation. Using the basic reproduction number, we derive global stability of the infection-free equilibrium and local stability of the unique endemic equilibrium.

The rest of this paper is organized as follows. Section 2 formulates the extended *SVEIR* model, which takes two important factors: Partial immunization and a saturated incidence rate, and obtains the basic reproduction number. Section 3 proves the stabilities of the equilibria. Section 4 covers the numerical analysis and the simulations. Section 5 summarizes the paper with some future directions.

2 A Mathematical Formulation for SVEIR Model

This section will examine the *SVEIR* model with a mathematical formulation. The *SVEIR* model extends the classical *SEIR* model through incorporating a saturated incidence rate and a partial immunization rate. The total host population N is divided into five groups and a host at any time t can potentially be in one of the following groups: 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 dynamic transition of the hosts is shown in Figure 1.

In Figure 1, Π is the constant recruitment rate of the host population, μ is the natural death rate of the population, and α is the death rate for worm attack of infectious hosts. Let β be the transmission rate of worm attack when susceptible hosts contact with infected ones. p is the fraction of recruited hosts which are vaccinated. γ is the rate at which vaccine wanes. The emergence of this scenario is due to worm variants. η is the parameter measuring the inhibitory effect. $\frac{\beta SI}{1+\eta I}$ is the saturated infection rate. ω is the rate at which exposed hosts become infectious, and δ is the recovered rate of infected hosts. The vaccinated hosts which contact infected ones before obtaining immunization have the infection probability with a transmission rate $\sigma\beta$ ($0 \leq \sigma \leq 1$). $\sigma = 0$ means that the vaccinated hosts obtain the full immunization, which $\sigma = 1$ means that vaccine loses efficacy in work fully. Taking some real factors into account, we assume that the vaccinated hosts

can obtain partial immunization, i.e. $0 < \sigma < 1$.

Based on the above assumptions, the *SVEIR* worm propagation model with partial immunization in the host population is described by the following system of differential equations:

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

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

$$\begin{cases} S'(t) = (1-p)\Pi - \frac{\beta SI}{1+\eta I} - \mu S + \gamma V, \\ V'(t) = p\Pi - \sigma\beta VI - (\mu+\gamma)V, \\ E'(t) = \frac{\beta SI}{1+\eta I} + \sigma\beta VI - (\mu+\omega)E, \\ I'(t) = \omega E - (\mu+\alpha+\delta)I. \end{cases} \quad (2)$$

Summing equations in (2), we obtain $(S+V+E+I)' = \Pi - \mu(S+V+E+I) - (\alpha+\delta)I \leq \Pi - \mu(S+V+E+I)$. Then it follows that $\limsup_{t\to\infty}[S(t)+V(t)+E(t)+I(t)] \leq \Pi/\mu$, thus the set

$$\Omega = \{(S,V,E,I) \in \mathcal{R}^4 : S+V+E+I \leq \Pi/\mu\}$$

is positively invariant for (2). Therefore, we will study the global stability of (2) on the set Ω.

It is easily seen that the model (2) always has an infection-free equilibrium, $P_0 = (S_0, V_0, 0, 0, 0)$, where $S_0 = \frac{\Pi(\mu+\gamma-p\mu)}{\mu(\mu+\gamma)}$, $V_0 = \frac{p\Pi}{\mu+\gamma}$. Let $x = (E, I, V, S)^T$, then the Model (2) can be represented as

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

where

$$\mathcal{F}(x) = \begin{pmatrix} \frac{\beta SI}{1+\eta I} + \sigma\beta VI \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$\mathcal{V}(x) = \begin{pmatrix} (\mu+\omega)E \\ (\mu+\alpha+\delta)I - \omega E \\ \sigma\beta VI + (\mu+\gamma)V - p\Pi \\ \frac{\beta SI}{1+\eta I} + \mu S - (1-p)\Pi - \gamma V \end{pmatrix}$$

Differentiating $\mathcal{F}(x)$ and $\mathcal{V}(x)$ with respect to E, I, V, S and computing them at the infection-free equilibrium $P_0 = (\frac{\Pi(\mu+\gamma-p\mu)}{\mu(\mu+\gamma)}, \frac{p\Pi}{\mu+\gamma}, 0, 0)$, respectively, we obtain

$$D\mathcal{F}(P_0) = \begin{pmatrix} 0 & \beta S_0 + \sigma\beta V_0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$
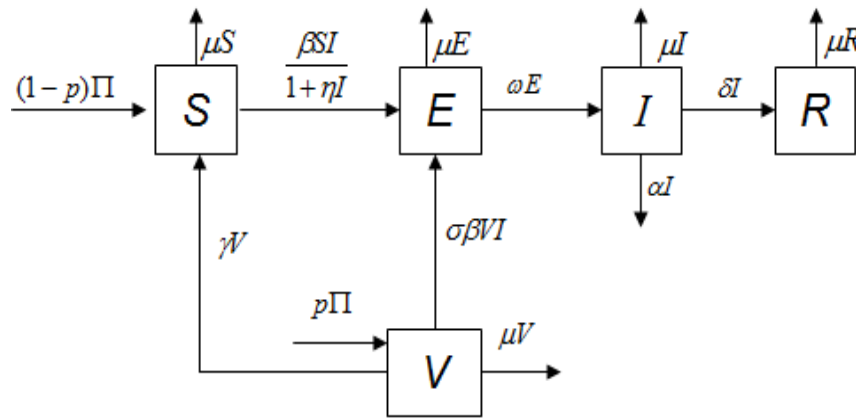
Figure 1: State transition diagram of the SVEIR model

$$DV(P_0) = \begin{pmatrix} \mu + \omega & 0 & 0 & 0 \\ -\omega & \mu + \alpha + \delta & 0 & 0 \\ 0 & \sigma\beta V_0 & \mu + \gamma & 0 \\ 0 & -\beta S_0 & -\gamma & \mu \end{pmatrix}$$

Thus, the spectral radius of the next generation matrix $\mathcal{F}\mathcal{V}^{-1}$ can be written as,

$$\rho(\mathcal{F}\mathcal{V}^{-1}) = \frac{\omega\beta(S_0 + \sigma V_0)}{(\mu + \omega)(\mu + \alpha + \delta)}.$$

According to Theorem 2 in [17], the basic reproduction number of the Model (2) is

$$\begin{aligned} R_0 &= \frac{\omega\beta(S_0 + \sigma V_0)}{(\mu + \omega)(\mu + \alpha + \delta)} \\ &= \frac{\omega\beta\Pi(\frac{\mu+\gamma-p\mu}{\mu(\mu+\gamma)} + \frac{\sigma p}{\mu+\gamma})}{(\mu + \omega)(\mu + \alpha + \delta)}. \end{aligned} \quad (3)$$

3 Stability Analysis for Equilibriums

The endemic equilibrium $P^*(S^*, V^*, E^*, I^*)$ of the Model (2) can be obtained by the following Equations (4)

$$\begin{cases} (1-p)\Pi - \frac{\beta SI}{1+\eta I} - \mu S + \gamma V = 0, \\ p\Pi - \sigma\beta VI - (\mu + \gamma)V = 0, \\ \frac{\beta SI}{1+\eta I} + \sigma\beta VI - (\mu + \omega)E = 0, \\ \omega E - (\mu + \alpha + \delta)I = 0. \end{cases} \quad (4)$$

From the fourth equation of the Model (4), we can obtain $E = \frac{(\mu+\alpha+\delta)I}{\omega}$. Substituting E into the third equation of the Model (4), we can obtain $V = \frac{(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta} - \frac{\beta S}{(1+\eta I)\sigma\beta}$. According to the second equation of the Model (4), we obtain $I = \frac{p\Pi-(\mu+\gamma)V}{\sigma\beta V}$.

Substituting V and I into the first equation of Model (4) with the foregoing obtained values, we obtain

the following equation

$$(1-p)\Pi - \frac{\beta S \frac{p\Pi-(\mu+\gamma)V}{\sigma\beta V}}{1+\eta I} - \mu S + \gamma A_1,$$

where, $A_1 = \frac{(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta} - \frac{\beta S}{(1+\eta I)\sigma\beta}$.

By a simple computation, we have

$$\Pi - \mu S - \frac{p\Pi(\mu+\omega)(\mu+\alpha+\delta)}{\frac{\omega\beta S}{1+\eta I} - (\mu+\omega)(\mu+\alpha+\delta)} + \frac{\mu S}{\sigma(1+\eta I)} + A_2 = 0,$$

where, $A_2 = \frac{\gamma(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta}$. Supposing

$$\begin{aligned} F(S) &= \Pi - \mu S - \frac{p\Pi(\mu+\omega)(\mu+\alpha+\delta)}{\frac{\omega\beta S}{1+\eta I} - (\mu+\omega)(\mu+\alpha+\delta)} \\ &+ \frac{\mu S}{\sigma(1+\eta I)} + \frac{\gamma(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta}. \end{aligned}$$

For $S = 0$, $F(0) = (1-p)\Pi + \frac{\gamma(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta}$. It is easily seen that $F(0) > 0$.

$$\begin{aligned} F'(S) &= -\mu - \frac{p\Pi\omega\beta(\mu+\omega)(\mu+\alpha+\delta)\frac{1}{1+\eta I}}{(\frac{\omega\beta S}{1+\eta I} - (\mu+\omega)(\mu+\alpha+\delta))^2} + \frac{\mu}{\sigma(1+\eta I)} \\ &< -\mu + \frac{p\Pi\omega\beta\frac{1}{1+\eta I}}{\frac{\omega\beta S}{1+\eta I} - (\mu+\omega)(\mu+\alpha+\delta)} + \frac{\mu}{\sigma(1+\eta I)} \\ &= -\mu + (\frac{\mu}{\sigma} - \frac{p\beta\Pi}{\frac{(\mu+\omega)(\mu+\alpha+\delta)}{\omega} - \frac{\beta S}{1+\eta I}})\frac{1}{1+\eta I} \\ &< 0. \end{aligned}$$

Therefore, the sign of $F'(S)$ is negative. On the other hand, if $R_0 > 1$, $\omega\beta(S_0 + \sigma V_0) = \omega\beta(S_0 + \frac{\sigma p\Pi}{\mu+\gamma}) > (\mu +$

$\omega)(\mu + \alpha + \delta)$.

$$
\begin{aligned}
F(S_0) &= \Pi - \mu S_0 - \frac{p\Pi(\mu+\omega)(\mu+\alpha+\delta)}{\frac{\omega\beta S_0}{1+\eta I} - (\mu+\omega)(\mu+\alpha+\delta)} \\
&\quad + \frac{\mu S_0}{\sigma(1+\eta I)} + \frac{\gamma(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta} \\
&< \Pi - \mu S_0 - \frac{(\mu+\gamma)(\mu+\omega)(\mu+\alpha+\delta)}{\omega\beta\sigma} \\
&\quad + \frac{\mu S_0}{\sigma(1+\eta I)} + \frac{\gamma(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta} \\
&< \Pi - \mu S_0 - \frac{\mu S_0}{\sigma(1+\eta I)} - \frac{\mu p\Pi}{\mu+\gamma} + \frac{\mu S_0}{\sigma(1+\eta I)} \\
&= \Pi - \mu S_0 - \frac{\mu p\Pi}{\mu+\gamma} \\
&= 0.
\end{aligned}
$$

If $S > S_0$, $F(S) < 0$. As a result, the equation $F(S) = 0$ only has a root S^* which always exists in $(0, S_0)$. When $R_0 \leq 1$, the System (2) only has an infection-free equilibrium $P_0(S_0, V_0, 0, 0)$. When $R_0 > 1$, the System (2) has the unique endemic equilibrium $P^*(S^*, V^*, E^*, I^*)$ except for the infection-free equilibrium P_0.

3.1 Infection-free Equilibrium and its Stability

It can be easily obtained that the Model (2) has an infection-free equilibrium given by $P_0 = (\frac{\Pi(\mu+\gamma-p\mu)}{\mu(\mu+\gamma)}, \frac{p\Pi}{\mu+\gamma}, 0, 0)$. The infection-free equilibrium corresponds to the model condition of non-worm breakout.

Proposition 1. *The infection-free equilibrium P_0 is locally asymptotically stable in the set Ω if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. According to $P_0 = (\frac{\Pi(\mu+\gamma-p\mu)}{\mu(\mu+\gamma)}, \frac{p\Pi}{\mu+\gamma}, 0, 0)$, the Jacobian matrix at the infection-free equilibrium P_0 of the Model (2) is

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

Therefore, the corresponding characteristic equation is described by

$$
\begin{aligned}
&(\lambda+\mu)(\lambda+\mu+\gamma) \\
&\quad \cdot [(\lambda+\mu+\omega)(\lambda+\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0)] \\
&= 0.
\end{aligned}
$$
(5)

From the characteristic Equation (5), we know that it always has two negative eigenvalues $\lambda_1 = -\mu$, and $\lambda_2 = -\mu-\gamma$. The other eigenvalues are decided by the following equation

$$(\lambda+\mu+\omega)(\lambda+\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0) = 0. \quad (6)$$

By the simple computation, Equation (6) is equal to

$$\lambda^2 + (2\mu+\omega+\alpha+\delta)\lambda + (\mu+\omega)(\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0) = 0. \quad (7)$$

If $R_0 < 1$, $(\mu+\omega)(\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0) > 0$, thus two roots of Equation (7) are negative. The infection-free equilibrium P_0 to be locally asymptotically stable is that $\lambda_i < 0$, for $i = 1, 2, 3, 4$, which meets the sufficient condition of the stability theory [16]. When $R_0 > 1$, $(\mu+\omega)(\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0) < 0$, which means that $J(P_0)$ has a positive root and a negative root. Therefore, the infection-free equilibrium P_0 is an unstable saddle point. This completes the proof. □

Proposition 2. *When $R_0 \leq 1$, the infection-free equilibrium P_0 is globally asymptotically stable in the set Ω.*

Proof. To prove the infection-free equilibrium P_0 is globally asymptotically stable, we construct the following Lyapunov function: $L(E, I) = \omega E + (\mu+\omega)I$.

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

$$
\begin{aligned}
L'(t) &= \omega E' + (\mu+\omega)I' \\
&= \frac{\omega\beta SI}{1+\eta I} + \omega\sigma\beta VI - \omega(\mu+\omega)E + (\mu+\omega)\omega E \\
&\quad - (\mu+\omega)(\mu+\alpha+\delta)I \\
&= \frac{\omega\beta SI}{1+\eta I} + \omega\sigma\beta VI - (\mu+\omega)(\mu+\alpha+\delta)I \\
&\leq (\omega\beta S + \omega\sigma\beta V - (\mu+\omega)(\mu+\alpha+\delta))I \\
&= \frac{\omega\beta(S_0+\sigma V_0)}{R_0}\left(\frac{R_0(S+\sigma V)}{S_0+\sigma V_0} - 1\right)I \\
&\leq 0.
\end{aligned}
$$

Furthermore, $L' = 0$ if and only if $I = 0$. Thus, the largest compact invariant set in $\{(S, V, E, I)|L' = 0\}$ is the singleton P_0. When $R_0 \leq 1$, the global stability of P_0 satisfies LaSalle's invariance principle [8]. LaSalle's invariance principle [8] hints that P_0 is globally asymptotically stable in the set Ω. This completes the proof. □

3.2 Endemic Equilibrium and Its Stability

From the aforementioned computation, we know that the Model (2) has the unique endemic equilibrium P^*. The endemic equilibrium P^* means that the worm does not die out when it appears. Finally, every class of the model reach its stable state. S^*, V^*, E^*, I^* and R^* are not equal to zero. Next, we investigate the local stability of the endemic equilibrium $P^* = (S^*, V^*, E^*, I^*)$.

Proposition 3. *When $R_0 > 1$, the endemic equilibrium P^* is locally asymptotically stable in the region Ω.*

Proof. The Jacobian matrix of (2) at the endemic equilibrium P^* is

$$J(P^*) = \begin{pmatrix} -B_1 & \gamma & 0 & -\frac{\beta S^*}{(1+\eta I^*)^2} \\ 0 & -B_2 & 0 & -\sigma\beta V^* \\ \frac{\beta I^*}{1+\eta I^*} & \sigma\beta I^* & -(\mu+\omega) & B_3 \\ 0 & 0 & \omega & -(\mu+\alpha+\delta) \end{pmatrix}$$

where,

$$\begin{aligned} B_1 &= \frac{\beta I^*}{1+\eta I^*} + \mu, \\ B_2 &= \sigma\beta I^* + (\mu+\gamma), \\ B_3 &= \frac{\beta S^*}{(1+\eta I^*)^2} + \sigma\beta V^*. \end{aligned}$$

Thus, the corresponding characteristic equation can be described as

$$\lambda^4 + C_1\lambda^3 + C_2\lambda^2 + C_3\lambda + C_4 = 0, \qquad (8)$$

where,

$$\begin{aligned} C_1 &= 4\mu + \alpha + \omega + \delta + \gamma + \sigma\beta I^* + \frac{\beta I^*}{1+\eta I^*} \\ &> 0, \\ C_2 &= (\mu+\omega)(\mu+\alpha+\delta) + B_2(2\mu+\omega+\alpha+\delta) \\ &\quad + B_1(\sigma\beta I^* + 3\mu + \gamma + \omega + \alpha + \delta) \\ &> 0, \\ C_3 &= B_2(\mu+\omega)(\mu+\alpha+\delta) + \beta\omega\mu\frac{S^*}{(1+\eta I^*)^2} \\ &\quad + B_1[(\mu+\omega)(\mu+\alpha+\delta) + B_2(2\mu+\omega+\alpha+\delta)] \\ &\geq (\mu+\omega)(\mu+\alpha+\delta)(\sigma\beta I^* + 2\mu + \gamma + \frac{\beta I^*}{1+\eta I^*}) \\ &\quad + B_1 B_2(2\mu+\omega+\alpha+\delta) \\ &> 0, \\ C_4 &= B_1 B_2(\mu+\alpha+\delta) + \gamma\omega\mu\sigma\beta V^* \\ &\quad + \beta\omega\mu\frac{S^*}{(1+\eta I^*)^2}B_2 \\ &\geq B_1 B_2(\mu+\omega)(\mu+\alpha+\delta) + \gamma\omega\mu\sigma\beta V^* \\ &> 0. \end{aligned}$$

Through a simple computation, we obtain that $H_1 = C_1 > 0$, $H_2 = C_1 C_2 - C_3 > 0$, $H_3 = C_3 H_2 - C_1^2 C_4 > 0$, $H_4 = C_4 H_3 > 0$.

According to the theorem of Routh-Hurwitz [10], we obtain that all the roots of the Equation (8) have negative real parts. As a result, the endemic equilibrium P^* is locally asymptotically stable. $\square$

4   Numerical Simulations

This section develops numerical experimental steps to analyze the stability of the proposed model and evaluates the effects of the implemented countermeasures. It is very difficult to use realistic parameters or real-world traffic
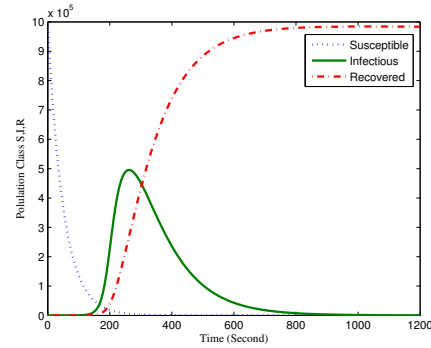

Figure 2: Globally asymptotically stable infection-free equilibrium

traces for our research, because many parameters used in previous models are assumed according to their hypothesis. To obtain the spread of worms in a large-scale network, $N = 1,000,000$ hosts are selected as the population size. According to the real Slammer worm, the average scan rate is $s = 4,000$ per second [11]. The infection rate of the Slammer worm is $\beta = s/2^{32} = 9.3 \times 10^{-7}$. We take proper values of Π and μ so that $\Pi/\mu = N$, implying that the total number of hosts remain unchanged. Therefore, we set $\Pi = 100$ and $\mu = 0.0001$. The partial immunization rate is set to $\sigma = 0.4$. The transition rate ω from E to I is 0.02. The transition rate δ from I to R is 0.01. At the beginning, the number of susceptible, vaccinated, exposed, infected, and recovered hosts are $S(0) = 999,985$, $V(0) = 10$, $E(0) = 0$, $I(0) = 5$, and $R(0) = 0$, respectively.

Other parameters in these simulations are given as follows: $\eta = 2$, $p = 0.2$, $\alpha = 0.0001$, and $\gamma = 0.05$. The results are based on the average of at least 10 simulation runs. Using the above parameters, we can obtain the basic reproduction number $R_0 = 0.908 < 1$. The worm will gradually die out according to Proposition 1 and 2. Figure 2 illustrates the number of susceptible, infected and recovered hosts when R_0 is 0.908, respectively. From Figure 2, we can clearly see that the tendency of the worm propagation is depressive, which is consistent with Proposition 1 and 2. Finally, all infected hosts vanish and the population, in the long term, is in a recovered state.

In the second experiment, we change some related parameters about R_0 to guarantee $R_0 > 1$. When $p = 0.4$ and $\delta = 0.003$, we have $R_0 = 9.847 > 1$. Other parameters remain unchanged. The results are shown in Figure 3. As can be seen from Figure 3, the number of susceptible, infected and recovered hosts eventually become positive values between 0 and Π/μ, which indicates that the worm does not disappear, if worms initially present. Finally, these three states reach their equilibrium points $P^*(38094, 83543, 835428)$. This is fully consistent with the conclusions of Proposition 3. The unique endemic equilibrium P^* is globally asymptotically stable.

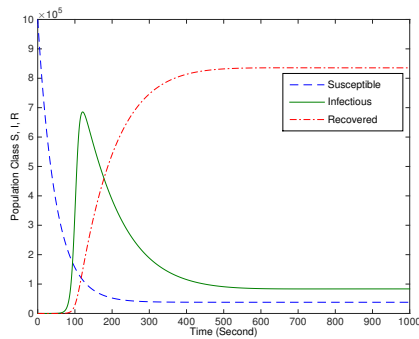To demonstrate that the effect of the partial immunization rate on the number of infected hosts, we set

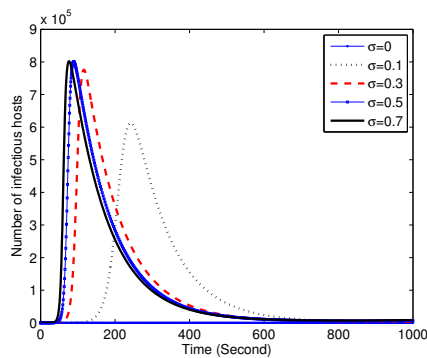Figure 3: Locally asymptotically stable endemic equilibrium

Figure 4: Effect of the partial immunization rate

the partial immunization rate σ to different values, with other parameters remaining the same. Figure 4 shows the effect of changing the partial immunization rate ($\sigma = 0, 0.1, 0.3, 0.5, 0.7$, respectively) on worm propagations. From Figure 4, no hosts are infected when $\sigma = 0$. $\sigma = 0$ means that all hosts gain full immunization. However, in real-world networks, it is very difficult to implement full immunization. As expected, a smaller partial immunization rate results in slowing down the worm propagation speed, more importantly, and decreasing the total number of infected hosts. Once the vaccine has been studied, computer users should immunize their computers as quickly as possible, which can guarantee to reach a smaller partial immunization rate σ.

5   Conclusion

This paper presented a novel dynamic *SVEIR* model with a saturated incidence rate and a partial immunization rate for the propagation of worms. More specifically, this paper investigated the global dynamic behavior of the *SVEIR* model, which is determined by the basic reproduction number. The theoretical analysis demonstrated that when the basic reproduction number is smaller than or equal to one, the *SVEIR* model has a infection-free equilibrium, and is globally asymptotically stable. That is to say, it implies that the worm dies out eventually.

When the basic reproduction number is larger than one, the *SVEIR* model has a unique endemic equilibrium which is locally stable. Moreover, it implies that worms are able to pervade across networks. The simulation results are consistent with theoretical analysis. Our proposed *SVEIR* model will be highly useful to analyze the availability and efficiency of partial immunization. The partial immunization will be efficient if the partial immunization rate is very small.

Acknowledgments

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