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Research paper

Analysis of SVEIR worm attack model with saturated incidence and partial immunization

WANG Fangwei[1,2], HUANG Wenyan[1], SHEN Yulong[2,3], WANG Changguang[1]

1. College of Information Technology, Hebei Normal University, Shijiazhuang 050024, China
2. Shannxi Key Laboratory of Network and System Security, Xidian University, Xi'an 710071, China
3. College of Computers, Xidian University, Xi'an 710071, China

Abstract: Internet worms can propagate across networks at terrifying speeds, reduce network security to a remarkable extent, and cause heavy economic losses. Thus, the rapid elimination of Internet worms using partial immunization becomes a significant matter for sustaining Internet infrastructure. This paper addresses this issue by presenting a novel worm susceptible-vaccinated-exposed-infectious-recovered model, named the SVEIR model. The SVEIR model extends the classical susceptible-exposed-infectious-recovered model (refer to SEIR model) through incorporating a saturated incidence rate and a partial immunization rate. The basic reproduction number in the SVEIR model is obtained. By virtue of the basic reproduction number, we prove the global stabilities of an infection-free equilibrium point and a unique endemic equilibrium point. Numerical methods are used to verify the proposed SVEIR model. Simulation results show that partial immunization is highly effective for eliminating worms, and the SVEIR model is viable for controlling and forecasting Internet worms.

Key words: Internet worm, attack model, stability, saturated incidence, partial immunization.

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1 Introduction

An Internet worm is a type of malicious code capable of duplicating itself and propagating across the Internet. The code red worm in 2001, Slammer worm in 2003, Blaster worm in 2003, Witty worm in 2004, and Conficker worm in 2008 are a few examples of Internet worms, which have caused heavy economic losses and tremendous social panic[1-3]. Network experts considered Internet worms the highest security risks on networks[4]. Especially, with the development of the IoT (Internet of Things), the threat of Internet worms can be expected to become increasingly serious for network security. Therefore, combating worms is an impending task for defenders. Based on the comparability between malicious worms and biological viruses, numerous mathematical models describing worm propagation have been proposed to study worms′ behaviors in the past decade[3,5-8]. Mathematical modeling is important in

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determining effective methods against Internet worms in different transmission settings and in quantifying the effects of defending methods. Many models and tools have been proposed to address the dynamic attacking behaviors of worms and effectively counter attacking them under different conditions, e.g., time delay[9], quarantine[10], and antivirus software[11]. All the above models are based on the SIR classical epidemic model[12]. The SIR model has some drawbacks because it assumes that a susceptible host becomes infectious immediately after contacting with an infected one. Actually, many worms have an exposed period during which susceptible hosts are infected but are not yet contagious. To overcome this drawback, a new model, named the SEIR model, was introduced. The SEIR model incorporates an exposed class[13]. Immunization is one of the commonly used methods for controlling and eliminating worms′ propagation[14-16]. However, all of the models assumed that the vaccine hosts obtained full immunization. This is not consistent with reality. Considering worms′ rapid propagation, users or network administrators cannot immunize the entire host population in real networks. In other words, it is very difficult to obtain full immunization for the vaccine hosts. Thus, partial immunization as a fungible and feasible method for eliminating worms has been used for predicting and controlling infectious diseases[17-19]. In many worm propagation models, the bilinear infection rate βSI[15,16]is used, where, S and I denote the number of susceptible and infectious hosts, respectively. The saturated infection rate $\beta SI/(1+\eta I)$ was firstly introduced by Capasso and Seior[20], where $\beta I/(1+\eta I)$ gravitates towards a saturation value when it becomes large, $1/(1+\eta I)$ measures the restrain effect of susceptible hosts on the infected hosts[21]. The saturated infection rate $\beta SI/(1+\eta I)$ is more rational than the linear rate βSI. Because it takes the effect of the infected hosts into consideration. The saturated infection rate has subsequently been used in many epidemic models[21-24]. This paper proposes a novel SVEIR model based on the SEIR model. Contrary to the existing models, the proposed SVEIR model is armed with partial immunization and saturated infection. Thus, it is a novel worm propagation model with partial immunization. This paper argues that the SVEIR model is appropriate for studying the effects of some security countermeasures on worm propagation. By virtue of the basic reproduction number, we prove the global stabilities of an infection-free equilibrium point and a unique endemic equilibrium. Based on these simulation results, we propose some effective countermeasures for eliminating worms.

This paper is organized as follows: In Section 2, we formulate the extended SVEIR model, which discusses two important factors, i.e., a partial immunization and a saturated incidence rate, and obtains the basic reproduction number. In Section 3, we resolve the global stability problems of the equilibriums. In Section 4, we provide the simulation results and propose some defending methods. Section 5 concludes the paper.

2 Mathematical formulation of the SVEIR model

The SVEIR model extends the classical SEIR model by incorporating a saturated incidence rate and a partial immunization rate. The host population N is divided into five classes and a host at any time t can potentially be in one of the following groups: susceptible, vaccinated, exposed, infectious, recovered, which are denoted by S, V, E, I, R, respectively. S is the class of susceptible hosts, V is the class of partially immunizing hosts, E is the class of exposed hosts, I is the class of infective hosts, and R is the class of recovered hosts. The host population N at time t is represented by $N(t)=S(t)+V(t)+E(t)+I(t)+R(t)$. The dynamic transition of the hosts is shown in the following figure.

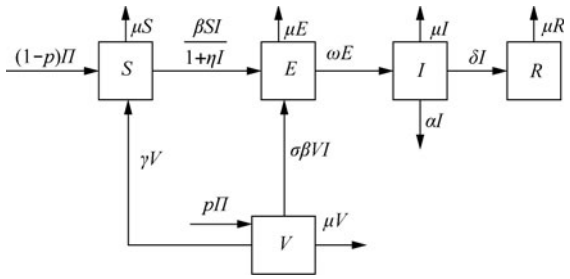

Figure 1 State transition diagram of the SVEIR model

In Fig.1, Π is the constant recruitment rate of the host population, μ denotes the natural death rate of the host population, and α denotes the death rate for a worm attack on infectious hosts. Let β be the transmission rate of a worm attack when susceptible hosts have contact with infected ones, p the fraction of recruited hosts that are vaccinated. Let γ be the rate at which the vaccine wanes. The emergence of this scenario is due to worm variants. Let η represent the parameter measuring the inhibitory effect. Let $\beta SI/(1+\eta I)$ be the saturated infection rate, ω the rate at which exposed hosts become infectious, and σ the recovered rate of infected hosts. Vaccinated hosts which contact infected ones before obtaining immunization, have an infection probability with a transmission rate $\sigma\beta$ ($0 \leqslant \sigma \leqslant 1$)[18,19]. $\sigma = 0$ means that the vaccinated hosts obtain the full immunization, while $\sigma = 1$ meaning that the vaccine loses full efficacy to work. Taking some real factors into account, we assume that the vaccinated hosts can obtain partial immunization, i.e. $0 < \sigma < 1$. According to the previous assumptions, the SVEIR worm propagation model with partial immunization can be described by the following system of differential equations:

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

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

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

Our proposed SVEIR model has two aspects that are different from existing models. At the beginning of worm propagation, there is no available vaccine to eliminate infected hosts. The worm propagates with a saturated infection rate $\beta SI/(1+\eta I)$, which is more reasonable than βSI. Once we detect a worm breakout using an intrusion detection system, we can study the related vaccine capable of defending against such a worm. Some newly arrived hosts are vaccinated through the vaccine, and no longer are infected. As a result, the model reduces the total number of vulnerable hosts and infected hosts.

When summing equations in model in Eq.(2), we have $(S+V+E+I)' \leqslant \Pi - \mu(S+V+E+I)$. Then the inequality satisfies that $\limsup_{t\to\infty}[S(t)+V(t)+E(t)+I(t)] \leqslant \Pi/\mu$, thus the set

$$\Omega = \{(C,V,E,I) \in \Re^4 : S+V+E+I \leqslant \Pi/\mu\}$$

is positively invariant for model in Eq.(2). As a result, we study the stability condition of the model in Eq.(2) on the set Ω.

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

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \mathcal{F}(x) - \mathcal{V}(x),$$

where

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

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

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

$$D\mathcal{F}(P_0)=\begin{pmatrix}0&\beta S_0+\sigma\beta V_0&0&0\\0&0&0&0\\0&0&0&0\\0&0&0&0\end{pmatrix},$$

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

Therefore, according to Theorem 2 in Ref.[25], the basic reproduction number of the model in Eq.(2), represented by R_0 is given by

$$R_0=\rho(\mathcal{F}\mathcal{V}^{-1})=\frac{\omega\beta\Pi\left(\dfrac{\mu+\gamma-p\mu}{\mu(\mu+\gamma)}+\dfrac{\sigma p}{\mu+\gamma}\right)}{(\mu+\omega)(\mu+\alpha+\delta)}. \quad (3)$$

3 Stability analysis for equilibriums in SVEIR

The endemic equilibrium $P^*(S^*, V^*, E^*, I^*)$ of the model in Eq.(2) can be obtained by the following equations:

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

When solving the model in Eq.(4), we have

$V=\dfrac{(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta}-\dfrac{BS}{(1+\eta I)\sigma\beta}$, $E=\dfrac{(\mu+\alpha+\delta)I}{\omega}$, $I=\dfrac{p\Pi-(\mu+\gamma)V}{\sigma\beta V}$. Substituting V and I into the first equation of model in Eq.(4) with the foregoing obtained values, we obtain the following equation

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

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

By a direct algebraic computation, we have

$$\Pi-\mu S-A_2+\mu A_4 S+A_3=0,$$

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

$$A_3=\frac{\gamma(\mu+\omega)(\mu+\alpha+\delta)}{\omega\sigma\beta}, A_4=\frac{1}{\sigma(1+\eta I)}.$$

Supposing

$$F(S)=\Pi-\mu S-A_2+\mu A_4 S+\gamma A_3.$$

For $S=0, F(0)=(1-p)\Pi+A_3$. It is easily seen that $F(0)>0$.

$$F'(S)=-\mu-\frac{p\Pi\omega\beta(\mu+\omega)(\mu+\alpha+\delta)\dfrac{1}{1+\eta I}}{\left(\dfrac{\omega\beta S}{1+\eta I}-(\mu+\omega)(\mu+\alpha+\delta)\right)^2}+\mu A_4$$

$$<-\mu+\frac{p\Pi\omega\beta\dfrac{1}{1+\eta I}}{\dfrac{\omega\beta S}{1+\eta I}-(\mu+\omega)(\mu+\alpha+\delta)}+\mu A_4$$

$$=-\mu+\left(\frac{\mu}{\sigma}-\frac{p\beta\Pi}{\dfrac{(\mu+\omega)(\mu+\alpha+\delta)}{\omega}-\dfrac{\beta S}{1+\eta I}}\right)\frac{1}{1+\eta I}<0.$$

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

$$F(S_0) = \Pi - \mu S_0 - (\mu+\gamma)A_3 + \mu A_4 S_0 + \gamma A_3$$
$$< \Pi - \mu S_0 - \frac{\mu S_0}{\sigma(1+\eta I)} - \frac{\mu p \Pi}{\mu+\gamma} + \frac{\mu S_0}{\sigma(1+\eta I)}$$
$$= \Pi - \mu S_0 - \frac{\mu p \Pi}{\mu+\gamma} = 0.$$

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 $R_0 \leqslant 1$, the system in Eq.(2) only has an infection-free equilibrium $P_0(S_0, V_0, 0, 0)$. When $R_0 > 1$, the system in Eq.(2) has the unique endemic equilibrium $P^*(S^*, V^*, E^*, I^*)$ except for the infection-free equilibrium P_0.

3.1 Stability of infection-free equilibrium

The model in Eq.(2) always has an infection-free equilibrium $P_0 = \left(\frac{\Pi(\mu+\gamma-p\mu)}{\mu(\mu+\gamma)}, \frac{p\Pi}{\mu+\gamma}, 0, 0\right)$. P_0 corresponds to the model condition of non-worm breakout. It is important to eliminate worms for defenders.

Proposition 1 For the model in Eq.(2), the infection-free equilibrium P_0 is locally asymptotically stable in the set Ω if $R_0 < 1$.

Proof 1 According to $P_0 = (S_0, V_0, 0, 0)$, the Jacobian matrix at P_0 of the model in Eq.(2) is

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

We easily obtain that it always has two negative eigenvalues $\lambda_1 = -\mu$, and $\lambda_2 = -\mu-\gamma$. The other eigenvalues are decided by the following equation

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

A simple computation is used to show that Eq. (5) is equal to

$$\lambda^2 + (2\mu+\omega+\alpha+\delta)\lambda + C = 0, \quad (6)$$

where, $C = (\mu+\omega)(\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0)$.

If $R_0 < 1$, $(\mu+\omega)(\mu+\alpha+\delta) - \omega(\beta S_0 + \sigma\beta V_0) > 0$, thus the two roots of Eq.(6) are negative. The locally asymptotically stable condition of P_0 is that $\lambda_i < 0$, for $i = 1, 2, 3, 4$, which meets the sufficient condition of the stability theory[26]. 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 both 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 For the model in Eq.(2), the infection-free equilibrium point P_0 is globally asymptotically stable if $R_0 \leqslant 1$.

Proof 2 To prove P_0 is globally asymptotically stable, we construct the following Lyapunov function: $L(E, I) = \omega E + (\mu+\omega) I$.

The derivation of $L(E, I)$ with respect to t gives

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

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 $R_0 \leqslant 1$, the global stability of P_0 satisfies LaSalle's invariance principle [27]. LaSalle's invariance principle[27] implies 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 in Eq.(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 reaches 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 3 The Jacobian matrix of Eq.(2) at the endemic equilibrium P^* is

$$J(P^*)=\begin{pmatrix} -D_1 & \gamma & 0 & -\dfrac{\beta S^*}{(1+\eta I^*)^2} \\ 0 & -D_2 & 0 & -\sigma\beta V^* \\ \dfrac{\beta I^*}{1+\eta I^*} & \sigma\beta I^* & -(\mu+\omega) & D_3 \\ 0 & 0 & \omega & -(\mu+\alpha+\delta) \end{pmatrix},$$

where, $D_1=\dfrac{\beta I^*}{1+\eta I^*}+\mu, D_2=\sigma\beta I^*+(\mu+\gamma),$

$D_3=\dfrac{\beta S^*}{(1+\eta I^*)^2}+\sigma\beta V^*$.

Thus, the corresponding characteristic equation can be described as

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

where,

$$C_1=4\mu+\alpha+\omega+\delta+\gamma+\sigma\beta I^*+\frac{\beta S^*}{1+\eta I^*}>0,$$

$$C_2=(\mu+\omega)(\mu+\alpha+\delta)+B_2(2\mu+\omega+\alpha+\delta)$$
$$+B_1(\sigma\beta I^*+3\mu+\gamma+\omega+\alpha+\delta)>0,$$

$$C_3=B_2(\mu+\omega)(\mu+\alpha+\delta)+\beta\omega\mu\frac{S^*}{(1+\eta I^*)^2}$$
$$+B_1[(\mu+\omega)(\mu+\alpha+\delta)+B_2(2\mu+\omega+\alpha+\delta)]$$
$$\geqslant(\mu+\omega)(\mu+\alpha+\delta)\left(\sigma\beta I^*+2\mu+\gamma+\frac{\beta I^*}{1+\eta I^*}\right)$$
$$+B_1B_2(2\mu+\omega+\alpha+\delta)>0,$$

$$C_4=B_1B_2(\mu+\alpha+\delta)+\gamma\omega\mu\sigma\beta V^*+\beta\omega\mu\frac{S^*}{(1+\eta I^*)^2}B_2$$
$$\geqslant B_1B_2(\mu+\omega)(\mu+\alpha+\delta)+\gamma\omega\mu\sigma\beta V^*>0.$$

Through a simple computation, we obtain that $H_1=C_1>0$, $H_2=C_1C_2-C_3>0$, $H_3=C_3H_2-C_1^2C_4>0$, $H_4=C_3H_3>0$.

According to the theorem of Routh-Hurwitz[28], if follows that the endemic equilibrium P^* is locally asymptotically stable. From the above discussion, we summarize the following conclusion.

In what follows, we use the geometrical approach[29] to study the stability of the endemic endemic equilibrium P^*.

Theorem 1 Consider the following systems[27]:

$$x'=f(x), x\in\Omega, \qquad (8)$$

and its corresponding periodic linear system

$$z'=\frac{\partial f^{[2]}}{\partial x}p(t)z(t), \qquad (9)$$

where, $\partial f^{[2]}/\partial x$ is the second additive compound matrix of $\partial f/\partial x$ and $\Theta=\{p(t):0\leqslant t\leqslant\omega\}$ is the period orbit of Eq.(8).

We make the following four assumptions:

1) there is a compact absorbing set $K\subset\Omega$ and a unique equilibrium $\overline{x}\in D$;

2) model in Eq.(8) satisfies the Poincare-Bendixson property;

3) Eq.(9) is asymptotically stable for each periodic solution $x=p(t)$ to Eq.(8) with $p(0)\in D$;

4) $(-1)^n det\dfrac{\partial f}{\partial x}(\overline{x})>0$,

Then, the unique equilibrium $\overline{x}$ of model in Eq.(8) is globally asymptotically stable in the set Ω.

Proposition 4 If $R_0>1$, the endemic positive equilibrium P^* is globally asymptotically stable in the set Ω.

Proof 4 If $R_0>1$, model in Eq.(2) is uniformly permanent, and the unique positive equilibrium P^* is locally asymptotically stable according to Proposition 3. Then the infection-free equilibrium point is unstable according to Proposition 1. Furthermore, there exists a compact absorbing set $K\subset\Omega$. Therefore, assumption Eq.(1) holds.

The Jacobian matrix of the model in Eq.(2) is denoted by

$$J(P)=\begin{pmatrix} -G_1-\mu & \gamma & 0 & -G_2 \\ 0 & -D_4 & 0 & -G_3 \\ G_1 & \sigma\beta I & 0-(\mu+\omega) & G_2+G_3 \\ 0 & 0 & \omega & -G_5 \end{pmatrix},$$

where, $G_1=\beta I/(1+\eta I)$, $G_2=\beta I/(1+\eta I)^2$, $G_3=\sigma\beta V$, $G_4=\sigma\beta I+(\mu+\gamma)$, $G_5=\mu+\alpha+\delta$.

We choose the matrix H with H = diag(−1, −1, 1, −1). It is easily proven that all off-diagonal elements of $HJ(P)H$ are not positive. Thus, the model in Eq.(2) is competitive. Therefore, assumption 2) holds.

Through an elementary row transformation of the matrix $J(P)$, we obtain

$$J(P)=\begin{pmatrix} -G_1-\mu & \gamma & 0 & -G_2 \\ 0 & -D_4 & 0 & -G_3 \\ -\mu & -\mu & -(\mu+\omega) & 0 \\ 0 & 0 & \omega & -G_5 \end{pmatrix}.$$

According to Proposition in Ref.[30], the second additive compound matrix $J^{[2]}(P)$ of $J(P)$ can be denoted by

$$J(P)=\begin{pmatrix} -E_1 & 0 & -\sigma\beta V & 0 & G_2 & 0 \\ -\mu & E_2 & 0 & \gamma & 0 & G_2 \\ 0 & \omega & E_3 & 0 & \gamma & 0 \\ \mu & 0 & 0 & E_4 & 0 & \sigma\beta V \\ 0 & 0 & 0 & \omega & E_5 & 0 \\ 0 & 0 & -\mu & 0 & -\mu & E_6 \end{pmatrix},$$

where,

$$E_1=-\left(\frac{\beta I}{1+\eta I}+2\mu+\sigma\beta I+\gamma\right), E_2=-\left(\frac{\beta I}{1+\eta I}+2\mu+\omega\right),$$
$$E_3=-\left(\frac{\beta I}{1+\eta I}+2\mu+\alpha+\sigma\right), E_4=-\left(\sigma\beta I+2\mu+\gamma+\omega\right),$$
$$E_5=-\left(\sigma\beta I+2\mu+\gamma+\alpha+\delta\right), E_6=-\left(2\mu+\omega+\alpha+\delta\right).$$

The second compound system of the model in Eq.(2) within a periodic solution is described by

$$\begin{cases} X'(t)=-E_1X+\sigma\beta VZ+G_2M, \\ Y'(t)=-\mu X-E_2Y+\gamma L-\beta_1G_2U, \\ Z'(t)=\omega Y-\left(\frac{\beta I}{1+\eta I}+2\mu+\alpha+\delta\right)Z+\gamma M, \\ L'(t)=\mu X+(\sigma\beta I+2\mu+\gamma+\omega)L+\sigma\beta VU, \\ M'(t)=\omega L+(\sigma\beta I+2\mu+\gamma+\alpha+\delta)M, \\ U'(t)=-\mu Z-\mu M-(2\mu+\omega+\alpha+\delta)U. \end{cases} \quad (10)$$

To verify that the system in Eq.(10) is asymptotically stable, we choose the following Lyapunov function:

$$V(X,Y,Z,L,M,U;S,V,E,I)$$
$$=\sup\left\{|X|+|Y|+|L|,\frac{E}{I}(|Z|+|M|+|U|)\right\}.$$

Using the uniform persistence, we know that there is a positive distance between the orbit of $P(t)=(S(t), V(t), E(t), I(t))$ and the boundary of Ω. As a result, there exists a constant c satisfying the following inequality:

$$V(X,Y,Z,L,M,U;S,V,E,I)$$
$$\geqslant c\sup\{|X|,|Y|,|Z|,|L|,|M|,|U|\},$$

for all $(X, Y, Z, L, M, U)\in\mathbb{R}^6$ and $(S, V, E, I)\in P(t)$.

Let $\phi=\{\gamma, \omega\}$. By direct computations, we obtain the following inequalities:

$$D_+|X(t)|\leqslant -E_1X+\frac{\beta S}{(1+\eta I)^2}M\leqslant -(2\mu+\phi)$$
$$+\frac{\beta S}{(1+\eta I)^2}(|M(t)|+|Z(t)|+U(t)|),$$
$$D_+|Y(t)|\leqslant -(2\mu+\phi)|Y(t)|+\gamma|L(t)|$$
$$+\frac{\beta S}{(1+\eta I)^2}(|M(t)|+|Z(t)|+U(t)|),$$
$$D_+|Z(t)|\leqslant \omega|Y(t)|-(2\mu+\alpha+\delta)|Z(t)|+\gamma|M(t)|,$$
$$D_+|L(t)|\leqslant \mu|X(t)|-(2\mu+\phi)|L(t)|+\sigma\beta V|U(t)|,$$
$$D_+|M(t)|\leqslant \omega|L(t)|-(2\mu+\alpha+\delta)|M(t)|-\gamma|M(t)|,$$
$$D_+|U(t)|\leqslant -(2\mu+\alpha+\delta)|U(t)|.$$

Therefore, we obtain

$$D_+(|X|+|Y|+|L|)\leqslant -(2\mu+\phi)(|X|+|Y|+|L|)$$
$$+\frac{E}{I}\left(\frac{\beta SI}{E(1+\eta I)^2}+\sigma\beta V\frac{I}{E}\right)(|Z|+|M|+|U|),$$
$$D_+(|Z|+|M|+|U|)\leqslant \omega(|X|+|Y|+|L|)$$
$$-(2\mu+\alpha+\delta)(|Z|+|M|+|U|).$$

Then,

$$D_+\frac{E}{I}(|Z|+|M|+|U|)\leqslant \omega\frac{E}{I}(|X|+|Y|+|L|)$$
$$+\left(\frac{E'}{E}-\frac{I'}{I}-2\mu-\alpha-\delta\right)\frac{E}{I}(|L|+|M|+|U|).$$

From the pervious formula, we have

$$D_+|V(t)|\leqslant \max\{g_1(t),g_2(t)\}V(t),$$

where,

$$g_1(t) = -2\mu - \phi + \beta S \frac{I}{E(1+\eta I)^2} + \sigma\beta V \frac{I}{E},$$

$$g_2(t) = \omega \frac{E}{I} + \frac{E'}{E} - \frac{I'}{I} - (2\mu + \alpha + \delta).$$

From the model (2), we can obtain

$$\frac{E'}{E} = \frac{\beta SI}{(1+\eta I)^2} + \sigma\beta V \frac{I}{E} - (\mu + \omega),$$

$$\frac{I'}{I} = \omega \frac{E}{I} - (\mu + \alpha + \delta).$$

Therefore,

$$g_1(t) = \frac{E'}{E} - \mu - (\mu - \phi), g_2(t) = \frac{E'}{E} - \mu.$$

If $\gamma \geqslant \omega$, then $\omega - \phi = \omega - \omega = 0$. If $\gamma < \omega$, then $\omega - \phi = \omega - \gamma > 0$. Under the two cases, we always have $\int_0^{\varsigma} \sup\{g_1(t), g_2(t)\}dt \leqslant \ln E(t)|_0^{\varsigma} - \mu\varsigma = -\mu\varsigma < 0$ which implies that $(X(t), Y(t), Z(t), L(t), M(t), U(t)) \to 0$, as $t \to \infty$. Therefore, the second compound system in Eq.(2) is asymptotically stable. This verifies the assumption 3).

Let $J(P^*)$ be the Jacobian matrix of the model in Eq.(2) at P^*, we obtain

$$J(P^*) = \begin{vmatrix} -D_1 & \gamma & 0 & -\dfrac{\beta S^*}{(1+\eta I^*)^2} \\ 0 & -D_2 & 0 & \sigma\beta V^* \\ -\mu & -\mu & -D_5 & 0 \\ 0 & 0 & \omega & -D_4 \end{vmatrix}$$

$$= \omega \begin{vmatrix} -D_1 & \gamma & -\dfrac{\beta S^*}{(1+\eta I^*)^2} \\ 0 & -D_2 & \sigma\beta V^* \\ \mu & \mu & 0 \end{vmatrix}$$

$$+ (\mu + \alpha + \delta)(\mu + \omega) \begin{vmatrix} -D_1 & \gamma \\ 0 & -D_2 \end{vmatrix}$$

$$= \mu\omega \left[\sigma\beta V^* (D_1 + \gamma) + \frac{\beta S^*}{(1+\eta I^*)^2} D_2 \right]$$

$$+ (\mu + \alpha + \delta)(\mu + \omega) D_1 D_2 > 0.$$

Therefore, $(-1)^6 det(J(P^*)) > 0$. This verifies the assumption 4).

We verify all the assumptions of Theorem 1. Therefore, P^* is globally asymptotically stable in Ω.

3.3 Worm epidemic control

Proposition 2 indicates that the combination efforts (represented by the formulation of R_0) can eliminate worm prevalence in the real networks. Under the SVEIR propagation model, we investigate how to control the infection-free equilibrium in network administration.

Corollary 1 In order to eliminate worms, the partial immunization rate σ should satisfy

$$\sigma \leqslant \frac{(\mu + \omega)(\mu + \alpha + \delta)(\mu + \gamma)}{p\omega\beta\Pi} - \frac{\mu + \gamma - p\mu}{p\mu}. \quad (11)$$

Proof 5 Using both Eq.(3) and Proposition 2, this corollary holds.

4 Numerical simulations

This section develops numerical experimental steps to examine our model and evaluates the effect of the implemented methods. It is difficult to adopt realistic parameters or real network traffic for our study, because many parameters in previous models are assumed according to their hypothesis. We choose the total host population N = 1 000 000. The average scan rate of the Slammer worm is s = 4 000 per second[2]. 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 remains unchanged. Therefore, we set Π = 100 and μ=0.000 1. δ = 0.4, ω = 0.02, γ= 0.01, $S(0)$ = 999, 985, $V(0)$ = 10, $E(0)$ = 0, $I(0)$ = 5, $R(0)$ = 0, η = 2, p = 0.2, α= 0.000 1, σ = 0.05. Using the above parameters, we can obtain the basic reproduction number R_0 = 0.908 < 1. The worm should gradually be eliminated according to Proposition 1 and 2. Fig.2 illustrates the change trend of susceptible, infected and recovered

hosts when R_0 is 0.908, respectively. From Fig.2, it is clearly seen that the worm propagation is depressive, which is consistent with an analysis of the theory. Finally, all the infected hosts vanish, and reach a recovered state.

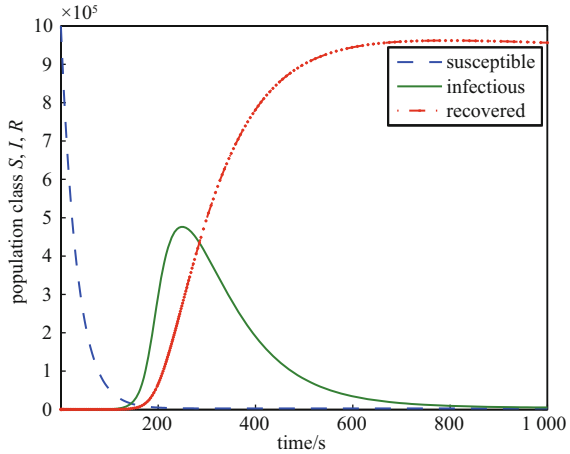

Figure 2 Globally stable infection-free equilibrium

In the second experiment, we change some related parameters about to guarantee $R_0 > 1$. When $p = 0.4$ and $\delta = 0.003$, we have $R_0 = 9.847 > 1$. The other parameters remain unchanged. The simulation results are shown in Fig.3, where it can be seen that the number of susceptible, infected and recovered hosts eventually maintain positive values between 0 and Π/μ, which indicates that the worm does not vanish, if worms are initially present. Finally, these three states reach their equilibrium points P^*= (38 094, 83 543, 83 528), which is consistent with Proposition 3 and 4. The unique endemic equilibrium P^* is globally asymptotically stable if the basic reproduction number is larger than unity.

To demonstrate that the effect of the partial immunization rate on infected hosts, we set the partial immunization rate σ to different values. The others parameters remain the same. Fig.4 shows that the effect of changing the partial immunization rate on worm propagations when $\sigma = 0$, 0.1, 0.3, 0.5, 0.7, respectively. Fig.4 shows that no hosts are infected when $\sigma = 0$, which 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. The partial immunization rate σ is related to many factors, such as the performance of network security devices, professional knowledge of the network administrator, and the security consciousness of users. As a result, in order to remove worms as soon as possible, we require the support from all circles of society. Once the vaccine has been studied, computer users should

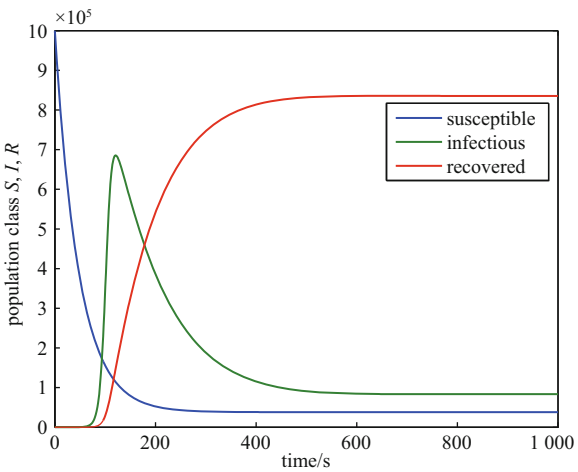

Figure 3 Globally stable endemic equilibrium

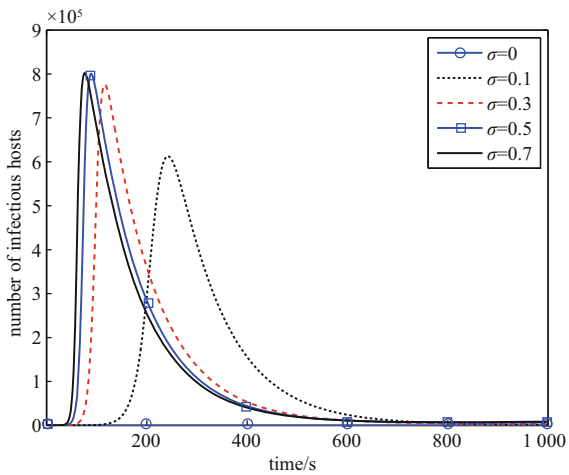

Figure 4 Effect of the partial immunization rate

immunize their computers in a very short time, which would guarantee them reaching a smaller partial immunization rate σ.

5 Conclusion and future work

This paper presented a novel dynamic SVEIR model for worms. The SVEIR model extends the classical SEIR model by incorporating a saturated incidence rate and a partial immunization rate. More specifically, this study investigated the global dynamic behavior of the SVEIR model by using the Lyapunov function and a geometric approach. The theoretical analysis demonstrated that when the basic reproduction number is smaller than or equal to one, the SVEIR model has an infection-free equilibrium, and is globally asymptotically stable. When the basic reproduction number is larger than one, the SVEIR model has a unique endemic equilibrium and is globally stable. The simulation results are consistent with theoretical analysis. Our proposed SVEIR model is expected to be highly useful to analyze the availability and efficiency of partial immunization, which becomes efficient if the partial immunization rate is very small. This is very helpful to eliminate worms as soon as possible.

In future, we plan to examine how to eliminate worms, quickly prevent their propagation across networks after detecting the worms attack, and expand the proposed SVEIR model to scale-free networks.

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About the authors

WANG Fangwei was born in 1976. He received his B.S. degree in 2000 from College of Mathematics & Information Sciences, Hebei Normal University, his M.S. degree in 2003 from College of Computer Science and Software, Hebei University of Technology, his Ph.D. degree in 2009 from College of Computer at Xidian University. Currently he is an associate professor at Hebei Normal University, Shijiazhuang, China. His research interests include: network and information security, sensor networks. (Email: fw wang@hebtu.edu.cn)

SHEN Yulong was born in 1978. He received the B.E., M.E., and Ph.D. degrees in Computer Science from Xidian University, China, in 2002, 2005, and 2008, respectively. Currently he is professor at Xidian University, Xi'an, China. His research interests include wireless network security and optimization, wireless network information theory, service computing, distributed network storage and security. (Email: ylshen@mail.xidian.edu.cn)

HUANG Wenyan was born in 1978. She received her M.S. degree in 2003 from College of Computer Science and Software, Hebei University of Technology, China. Currently she is a lecture at Hebei Normal University, Shijiazhuang, China. Her research interests include: network and information security. (Email: huangwy@hebtu.edu.cn)

WANG Changguang [corresponding author] was born in 1971. He received his M.S. degree in 1996 from School of Physical Science and Technology, Sichuan University, and his Ph.D. degree in 2009 from College of Computer at Xidian University. Currently he is a professor at Hebei Normal University, Shijiazhuang, China. His research interests include network and information security. (Email: wangcg@hebtu.edu.cn)