Study of Virus Propagation Model Under the Cloud

Tongrang Fan[1], Yanjing Li[1], Feng Gao[1]

1School of Information Science and Technology, Shijiazhuang Tiedao University, Shijiazhuang, 050043, China
Fantr2009@126.com, 532465444 @qq.com, f.gao@live.com

Abstract. The impacts of the changes about the number of the nodes with different states by the external factors are not considered in the traditional model. So, they are not suitable for the cloud network which is dynamic. A virus propagation model of computer which applies to the cloud environment, HSIR propagation model, is proposed in this paper. It takes the dynamic changes of the cloud environment, the implementation of the immunization strategy and the time difference between the virus spread and the implementation of the immunization strategy into account. In addition, the balance point of the HSIR model is found and the stability of the balance point is proved by the mathematical theory. So, the HSIR virus propagation model can describe the process of the virus propagation in the cloud environment more realistic.

Keywords: cloud network, HSIR propagation model, balance point, stability

1 Introduction

Computer viruses have many similarities with the biological viruses, so the propagation model of the computer is established by using the propagation model of the biology[1-3]. BK Mishra and Navnit Jha[4] propose the dynamic model of the virus propagation, and gives the estimate of the time evolution of the infected nodes. Then they discuss the behaviors of the nodes with different states by using the SEIQR model. The SEIRS model is proposed by BK Mishra and DK Saini[5]. The model describes the propagation of the worm, studies its disease-free equilibrium and explains the stability of the simulation results based on the threshold parameters. BK Mishra and SK Pandey[6,7] analysis the fuzzy transmission of the worms in the computer network and propose the SIRS model of fuzzy transmission. The impact of the virus for the immune response of the computer network is studied by paper [8] by using the nonlinear mathematical model. They find that the immune system response changes with the concentration of the virus. The characteristics of the transmission dynamic of computer virus are proposed by Z Dezso[9], Newman and Forrest[10]. The behaviors of the transmission dynamic of network are analyzed by using the SIR and SIS model by Grassberger[11].

The impacts of the changes about the number of the nodes with different states by the external factors are not considered in the above models. Cloud environment which

is a typical dynamic environment is accessed by lots of nodes at any time. The number of nodes in the cloud environment is changing all the time. Therefore the above models are all not suitable for the cloud network. A virus propagation model of computer which applies to the cloud environment is proposed in this paper. The dynamic changes of the cloud environment, the implementation of the immunization strategy and the time difference between the virus spread and the implementation of the immunization strategy are all take into account. So, it can describe the process of the virus propagation in the cloud environment more realistic.

2 HSIR model

The paper only analyzes the propagation process of one virus in order to facilitate the research. If only one virus is considered, then the nodes in the network can be divided into four states: *H* represents the healthy nodes which are not immune, this kind of nodes are healthy nodes and their neighbors are not infected, if their neighbors become infected nodes then the nodes become susceptible nodes; *S* represents the susceptible nodes, this kind of nodes are healthy nodes, but there are infected nodes in their neighbors; *I* represents the infected nodes; *R* represents the immune nodes, this kind of node are immune, never to be infected.

The relevant definitions and hypotheses of the model are as follows:

Definition 1: Immunization rate

The nodes which are not infected (include the healthy nodes and the susceptible nodes) and the infected nodes in the network will change into immune nodes by a certain probability respectively after injected the immunization strategy at a unit time. The probability is called immunization rate and the immunization rate of the infected nodes and the susceptible nodes and the healthy nodes is γ and ω and δ respectively;

Definition 2: Immune delay

There is a delay between the virus outbreak and the immune. This delay is called immune delay, represented by μ.

Hypothesis 1: There is only virus in the network at the initial time, i.e. no immunization strategy; all the nodes can be infected at that time; the number of healthy nodes $H_0 = N$, the susceptible nodes $S_0 = 0$, the infected nodes $I_0 = 0$ and the immune nodes $R_0 = 0$.

Hypothesis 2: The node is infected instantaneously. The time which virus propagate from one node to the other one is fixed, called a unit time.

Hypothesis 3: The cloud network studied in this paper is the network of logic-level.

Hypothesis 4: The virus is always in active state. The infected node infects its neighbors by the probability β.

The state transition of this model is shown in fig. 1. *A* represents the nodes added into the cloud at a unit time. $\beta C(N)$ represents the contact rate, in which *C(N)* is the contact number that the infected nodes contact to the susceptible nodes. $\beta C(N)SI < k > / N$ represents the probability that the healthy node change into the

susceptible node at a unit time. $< k >$ represents the average degree of the nodes in the network. α represents the probability that the susceptible node change into the infected node. σ represents the rate of the nodes exited from the cloud. d represents the rate of the dead nodes.

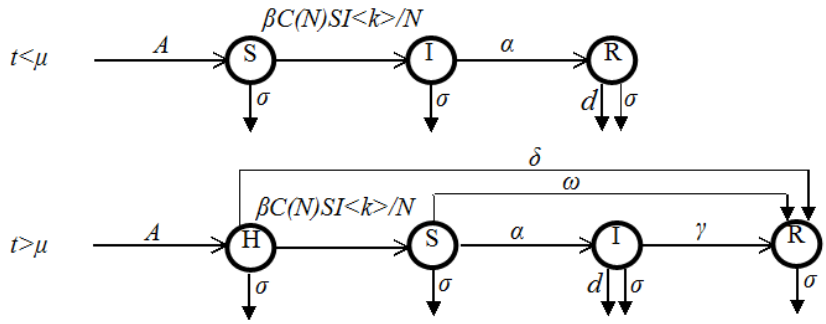

Fig. 1. The state transition of HSIR virus propagation model

Make $\sigma dt = d\tau$, The dynamic differential equation of the HSIR propagation after $t > \mu$ is as follows:

$$\begin{cases} \dfrac{dH(t)}{d\tau} = \dfrac{A}{\sigma} - \beta_1 H(t) S(t) I(t) - \delta_1 H(t) - H(t) \\ \dfrac{dS(t)}{d\tau} = \beta_1 H(t) S(t) I(t) - P_1 S(t) \\ \dfrac{dI(t)}{d\tau} = \alpha_1 S(t) - P_2 I(t) \\ \dfrac{dR(t)}{d\tau} = \gamma_1 I(t) + \omega_1 S(t) + \delta_1 H(t) - R(t) \end{cases} . \qquad (2\text{-}1)$$

In which, $H(t)$ represents the number of healthy nodes which are not immune at time t; $S(t)$ represents the number of susceptible nodes at time t; $I(t)$ represents the number of infected nodes at time t; $R(t)$ represents the number of the immune nodes at time t; and $\beta_1 = \dfrac{\beta C(N) < k >}{\sigma N}$, $\delta_1 = \dfrac{\delta}{\sigma}$, $\omega_1 = \dfrac{\omega}{\sigma}$, $\alpha_1 = \dfrac{\alpha}{\sigma}$, $d_1 = \dfrac{d}{\sigma}$, $\gamma_1 = \dfrac{\gamma}{\sigma}$, $P_1 = \omega_1 + \alpha_1 + 1$, $P_2 = \gamma_1 + d_1 + 1$. The total number of the nodes in the network is represented by the differential equation

$$\frac{dN(t)}{dt} = \frac{dH(t)}{dt} + \frac{dS(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} = \frac{A}{\sigma} - N - d_1 I(t).$$

By using the variable *N* instead of the variable *H*, it has:

$$\begin{cases} \dfrac{dS(t)}{d\tau} = \beta_1 H(t) S(t) I(t) - P_1 S(t) \\ \dfrac{dI(t)}{d\tau} = \alpha_1 S(t) - P_2 I(t) \\ \dfrac{dR(t)}{d\tau} = \gamma_1 I(t) + \omega_1 S(t) + \delta_1 H(t) - R(t) \\ \dfrac{dN(t)}{d\tau} = \dfrac{A}{\sigma} - N(t) - d_1 I(t) \end{cases} \tag{2-2}$$

System (2-2) is equivalent to system (2-1). The system (2-1) can be comprehended by studying the system (2-2). From the biology angle, the system (2-2) can be studied in $M = \{(S, I, R, N) \in R_+^4 : 0 \le S + I + R \le N \le \dfrac{A}{\sigma}\}$ .Considering the practical significance of the system (2-2), various initial values are in M, so *M* is the maximum positive invariant set of the system (2-2).

3 Balance Point and The Proof of it's Stability

The balance point of the model is found and its stability is proved in the following content. If the right sides of the four differential equations in system (2-1) and system (2-2) are equal to 0, then we have:

$$\begin{cases} H^* = \dfrac{A\alpha_1\sigma d_1^{\,2}}{\beta_1 P_2 (A-\sigma N)^2 + (\delta_1 + 1)\alpha_1 \sigma d_1^{\,2}} \\ S^* = \dfrac{P_2 (A - \sigma N)}{\sigma \alpha_1 d_1} \\ I^* = \dfrac{A - \sigma N}{\sigma d_1} \\ R^* = \gamma_1 I^* + \omega_1 S^* + \delta_1 H^* \end{cases} \tag{2-3}$$

And,

$$\frac{P_2}{\alpha_1 \sigma d_1}\left(\frac{A\alpha_1\beta_1 d_1 (A-\sigma N)}{\beta_1 P_2 (A-\sigma N)^2 + (\delta_1+1)\alpha_1 \sigma d_1^{\,2}} - P_1\right)(A - \sigma N) = 0 . \tag{2-4}$$

It is known from (2-3) and (2-4) that the system (2-2) have disease-free

equilibrium, that is $N^* = \frac{A}{\sigma}$, then the disease-free equilibrium is

$$P^0 = (0, 0, \frac{A\delta_1}{\sigma(\delta_1 + 1)}, \frac{A}{\sigma}).$$

Assume $F(N) = \frac{P_2}{\alpha_1 \sigma d_1}\left(\frac{A\alpha_1\beta_1 d_1(A-\sigma N)}{\beta_1 P_2 (A-\sigma N)^2 + (\delta_1+1)\alpha_1 \sigma d_1^{\ 2}} - P_1\right)$, there are:

$$F\left(\frac{A}{\sigma}\right) = -\frac{P_1 P_2}{\alpha_1 \sigma d_1} < 0,$$

$$F(0) = \frac{P_1 P_2}{\alpha_1 \sigma d_1}\left(\frac{A\alpha_1\beta_1 d_1(A-\sigma N)}{P_1\left[\beta_1 P_2 (A-\sigma N)^2 + (\delta_1+1)\alpha_1 \sigma d_1^{\ 2}\right]} - 1\right) = \frac{P_1 P_2}{\alpha_1 \sigma d_1}(R_0 - 1).$$

$R_0 = \frac{A\alpha_1\beta_1 d_1(A-\sigma N)}{P_1\left[\beta_1 P_2 (A-\sigma N)^2 + (\delta_1+1)\alpha_1 \sigma d_1^{\ 2}\right]}$ is the threshold of the system (2-2). $F(N)$ is monotonic decreasing when $R_0 > 1$, moreover $F(0) > 0$, $F\left(\frac{A}{\sigma}\right) < 0$,so $F(N) = 0$ has a unique positive root N^* in the interval $(0, \frac{A}{\sigma})$. Thus the system (2-2) has a unique endemic equilibrium $P^*(S^*, I^*, R^*, N^*)$, the values of S^*, I^*, R^* are determined by (2-3).

Theorem 1: The disease-free equilibrium P^0 is global asymptotic stable in *M* when $R_0 \le 1$. P^0 is not stable when $R_0 > 1$.

Proof: take the Lyapunov function $V = \alpha_1 S + P_1 I$, then

$$\begin{aligned} V'|_{system(2-1)} &= \beta_1\alpha_1 H(t) S(t) I(t) - \alpha_1 P_1 S(t) + \alpha_1 P_1 S(t) - P_1 P_2 I(t) \\ &= P_1 P_2 (R_0 - 1) I(t) \end{aligned}$$

So, it has $V' \le 0$ when $R_0 \le 1$. In addition, $V' = 0$ only when $I(t) = 0$ or $R_0 = 1$. Making $D = \{(S, I, R, N) \mid V' = 0\}$, then the maximum positive invariant set of *D* is a set of single point $\{P^0\}$. Therefore it is known from the Lyapunov Stability Theory that when $R_0 \le 1$, the disease-free equilibrium P^0 is global asymptotic stable.

When $R_0 > 1$, it has $V^{'} > 0$, so P^0 is not stable.

Theorem 2: System (2-2) has a unique endemic equilibrium P^* and it is asymptotically stable when $R_0 > 1$.

Proof: the Jacobian matrix $J(P^*)$ of system (2-2) at $P^* = \left(S^*, I^*, R^*, N^*\right)$ is:

$$\begin{pmatrix} \beta_1 H^* I^* - P_1 & \beta_1 H^* S^* & 0 & 0 \\ \alpha_1 & -P_2 & 0 & 0 \\ \omega_1 & \gamma_1 & -1 & 0 \\ 0 & -d_1 & 0 & -1 \end{pmatrix}$$

Its characteristic equation is $\lambda E - J(P^*) = 0$, in which E is unit matrix, so the characteristic equation can be reduced to:

$$(\lambda+1)^2[(\lambda + P_1 - \beta_1 H^* I^*)(\lambda + P_2) - \alpha_1 \beta_1 H^* S^*] = 0 .$$

Define $\alpha_1 = P_1 + P_2 - \beta_1 H^* I^*$, $\alpha_2 = P_2 \times \left(P_1 - \beta_1 H^* I^*\right) - \alpha_1 \beta_1 H^* S^*$, then the above equation can be expressed as:

$$\left(\lambda+1\right)^2 \left(\lambda^2 + \alpha_1 \lambda + \alpha_2\right) = 0$$

Because:

$$P_1 - \beta_1 H^* I^* - \alpha_1 \beta_1 H^* S^* = P_1 + \beta_1 H^* I^* \left(\gamma_1 + d_1\right) > 0$$

Then:

$$\alpha_1 = P_1 + P_2 - \beta_1 H^* I^* > 0 ;$$

$$\alpha_2 = \left(\gamma_1 + d_1\right)\left(P_1 - \beta_1 H^* I^*\right) + P_1 - \beta_1 H^* I^* - \alpha_1 \beta_1 H^* S^* > 0 .$$

So:

$$\begin{vmatrix} a_1 & a_0 \\ a_3 & a_2 \end{vmatrix} = a_1 a_2 > 0 .$$

It is known from the Hurwitz criterion that the endemic equilibrium P^* is asymptotically stable.

Therefore R_0 is the immune threshold of the HSIR propagation model. The system is stable in disease-free equilibrium when $R_0 \le 1$, at this time the disease disappear.

The system is stable in endemic equilibrium when $R_0 > 1$, at this time the disease still exists.

4 Summary

The impacts of the changes about the number of the nodes with different states by the external factors are not considered in the traditional models. So, they are all passive propagation model. The cloud environment is large, distributed and dynamic. So the traditional models are not suitable for the cloud environment. A virus propagation model of computer which applies to the cloud environment, HSIR propagation model, is proposed in this paper. It takes the dynamic changes of the cloud environment, the implementation of the immunization strategy and the time difference between the virus spread and the implementation of the immunization strategy into account. In addition, the balance point of the HSIR model is found and the stability of the balance point is proved by the mathematical theory. And the threshold of the virus propagation in the HSIR model is found too.

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