

ANALYSIS OF A MATHEMATICAL MODEL FOR WORM VIRUS PROPAGATION

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In recent years, the two-factor model has been improved in many papers. In this paper, we consider two new factors and build a new consider SIQR model for Internet worm virus propagation. By using theory of differential equations, the dynamical property of the model is analyzed and the regularity of Internet worm virus propagation is gained. In the end, the numerical simulation is presented.

1. Introduction

With the popularity of the network, the state of information security is subjected to unprecedented threats. Almost all countries have taken measures to protect their own information security. Recently, computer worms remain a significant threat to networks. Computer viruses have caused millions of dollars of damage and untold aggravation. It is a focus for the scholars to discuss how worms spread, and how we can monitor and defend against the propagation of worms effectively. Many people have studied the computer virus [1-3] by using differential theory. To understand the characteristics of the spread of Code Red worm, Zoo C C Builds a Two-factor model [4]. But this model has not considered two things: (1) Because some users can reinstall their computer system for some especial reasons, the removed hosts can become the susceptible hosts. (2) Lacking the network knowledge, some computer users have not known how can protect their computers against worms. But if many users around them have taken measures to protect their hosts before being infected, they can study from them. So the number of the hosts which is removed from to is not only related to the number of and but also related to the number of. We will consider the two probabilities in this paper and build an improved Two-factor model. By using the theory of differential equations, the stability of equilibrium point is analyzed and the regularity of

internet worm virus propagation is gained. By using MATLAB, the numerical simulation is presented.

2. Mathematical Modeling

The early stage of the outbreak of the worm, every system in the Internet is susceptible to the outbreak if they possess exploitable vulnerability, and network worms propagates at a startling speed. We let $S(t)$ is the number of susceptible hosts at time t , $I(t)$ is the number of infected hosts at time t and β is the initial infection rate. So the differential equation model is

$$\begin{cases} \frac{dI}{dt} = \beta I \\ S + I = N \end{cases} \quad (1)$$

Theorem 1. The system (1) has a unique globally asymptotically stable equilibrium $(0, N)$.

From Theorem 1, we can get that $I(t)$ must satisfy $\lim_{t \rightarrow \infty} I(t) = 0$. But in fact hardly any worm can infect all the hosts. Thus the system (1) can not fit the worm propagation in the later. In order to describe the behavior of worm propagation in the later, we give some appropriate assumptions as follows:

(1) $R(t)$ is the number of removed hosts from infected hosts at time t , and $Q(t)$ is the number of removed hosts from susceptible hosts at time t .

(2) Let $\mu > 0, k > 0, \lambda > 0$ denote removal rate from $S(t)$ to $Q(t)$, from $Q(t)$ and $R(t)$ to $S(t)$ and from $I(t)$ to $R(t)$ respectively.

(3) The exponent $\theta > 1$ is used to adjust the infection rate sensitivity to the number of infectious hosts.

(4) The number of the hosts which is removed from $S(t)$ to $Q(t)$ is not only related to the number of $I(t)$ and $R(t)$ but also related to the number of $Q(t)$. Because the more the hosts have taken action and their computers have been immune from the worm, the more the rest hosts can get the technique to protect their computers. In the same time this can build up user's confidence to immune their computers. So the number of the hosts which are removed from $S(t)$ to $Q(t)$ can be $\mu S(I + R + Q)$

Based on the above hypotheses, we now propose the following differential equation model:

$$\begin{cases} \frac{dS}{dt} = -\beta \left(1 - \frac{I}{N}\right)^\theta SI - \mu S(I + R + Q) + k(R + Q) \\ \frac{dI}{dt} = \beta \left(1 - \frac{I}{N}\right)^\theta SI - \lambda I \\ \frac{dR}{dt} = \lambda I - kR \\ \frac{dQ}{dt} = \mu S(I + R + Q) - kQ \end{cases} \quad (2)$$

Theorem 2 Given system (2),

(1) If $R_1 \leq 1$, the equilibrium $P_0(N, 0, 0, 0)$ of system (2) is globally asymptotically stable.

(2) If $R_1 > 1$, the equilibrium $P_0(N, 0, 0, 0)$ of system is unstable, and there exists positive equilibrium $P_1(S^*, I^*, R^*, Q^*)$ of system (2), which is local asymptotically stable provided that $\beta\theta N > (\theta + 1)(k + \lambda)$. On this basis, the equilibrium $P_1(S^*, I^*, R^*, Q^*)$ is global asymptotically stable if it satisfies $\mu N < k$. Where $R_1 = \beta N / \lambda$, I^* is a unique positive solution of the equation

$$\mu\lambda N - k\beta\lambda = (k\beta N - k\beta I - \lambda\beta I)\beta\left(1 - \frac{I}{N}\right)^\theta + \frac{\mu\lambda^2}{\beta\left(1 - \frac{I}{N}\right)^\theta}$$

Proof: It is obvious that there always exists equilibrium $P_0(N, 0, 0, 0)$ of system (2), and there may be a possible equilibriums $P_1(S^*, I^*, R^*, Q^*)$ of system (2) in the non-negative cone $T = \{(S, I, R, Q) \in \mathbb{R}_+^4 : S + I + R + Q \leq N\}$

with $R^* = \frac{\lambda}{k}I^*$ and $S^* = \frac{\lambda}{\beta\left(1 - \frac{I^*}{N}\right)^\theta}$. Let

$$F(I) = \mu\lambda N - k\beta\lambda,$$

$$G(I) = (k\beta N - k\beta I - \lambda\beta I)\beta\left(1 - \frac{I}{N}\right)^\theta + \frac{\mu\lambda^2}{\beta\left(1 - \frac{I}{N}\right)^\theta},$$

we can get that

$$G'(I) = \beta\left(1 - \frac{I}{N}\right)^{\theta-1} \left[\frac{(\theta+1)I\beta}{N}(k+\lambda) - (\theta k + k + \lambda)\beta \right]$$

$$G(0) = k\beta^2 N + \frac{\mu\lambda^2}{\beta} \text{ and } G(N) = +\infty. \text{ If } R_1 > 1,$$

together with $\mu\lambda N - k\beta\lambda > k\beta^2 N + \frac{\mu\lambda^2}{\beta}$, leads to

$F(0) > G(0)$, consequently, there is a unique positive point of intersection in the curves $F(I)$ and $G(I)$, which implies that the equation

$$\mu\lambda N - k\beta\lambda = (k\beta N - k\beta I - \lambda\beta I)\beta\left(1 - \frac{I}{N}\right)^\theta + \frac{\mu\lambda^2}{\beta\left(1 - \frac{I}{N}\right)^\theta} \text{ has unique}$$

positive solution. So we can get equilibrium $P_1(S^*, I^*, R^*, Q^*)$.

(I) Let $E = R + Q$, then $S = N - I - E$, the system (2) can be reduced to

$$\begin{cases} \frac{dI}{dt} = \beta \left(1 - \frac{I}{N}\right)^\theta (N - I - E)I - \lambda I, \\ \frac{dE}{dt} = \mu(N - I - E)(I + E) + \lambda I - kE. \end{cases} \quad (3)$$

Constructing Lyapunov functional $V(I, R, Q) = I$ and calculating the derivative of V along the system (3), we can obtain

$$\begin{aligned} \left. \frac{dV}{dt} \right|_{(3)} &= [\beta\left(1 - \frac{I}{N}\right)^\theta S - \lambda]I \leq [\beta\left(1 - \frac{I}{N}\right)^\theta (N - I) - \lambda]I \\ &\leq [\beta N - \lambda - \beta I]I \leq -\beta I^2 \leq 0 \end{aligned}$$

Hence we can

get $T = \{(I, R, Q) \in D \mid V'(t) = 0\} = \{I = 0\}$. Thus the

equilibrium P_0 is globally asymptotically stable by the LaSalle invariant principle and the theory of the limitation.

(II) The Jacobi matrix of the system (3) about $(0, 0)$ is given by

$$J_0 = \begin{pmatrix} \beta N - \lambda & 0 \\ \mu N + \lambda & \mu N - k \end{pmatrix}.$$

Then the characteristic equation of liberalized equations corresponding to system read $(r + \lambda - \beta N)(r + k - \mu N) = 0$. If $R_1 > 1$, the characteristic root $r = \beta N - \lambda > 0$, equilibrium $(0, 0)$ is not stable.

(III) The Jacobi Matrix of the system (3) about $P_1(I^*, E^*)$ is given by

$$J_1 = \begin{pmatrix} -a_1 - a_2 & -a_2 \\ a_3 + \lambda & a_3 - k \end{pmatrix}$$

Where

$$a_1 = \frac{\lambda \theta I^*}{N - I^*},$$

$$a_2 = \frac{\lambda I^*}{N - I^* - E^*},$$

$$a_3 = \mu(N - 2I^* - 2E^*).$$

If $\beta \theta N > (\theta + 1)(k + \lambda)$, we can get

$$p = a_1 + a_2 - a_3 + k > 0,$$

$$q = |J_1| = ka_1 + ka_2 - a_3 + \theta a_2 + \lambda a_2 > 0$$

Thus the equilibrium P_1 is locally asymptotically stable. Let $D(I, E) = \frac{1}{I}$. Note that $\frac{\partial(PD)}{\partial I} + \frac{\partial(QD)}{\partial E} < 0$ dues to $\mu N < k$. The Bendixson-Dulac criterion holds and thus the equilibrium (I^*, E^*) of system (3) is globally asymptotically stable.

3. Simulation

(1) Let $N = 500000, I_0 = 100, \beta = 0.00002$, $\theta = 2, k = 0.2, \mu = 0.0000003, \lambda = 0.2$, where $R_1 > 1$. We can see the result in Fig. 1..

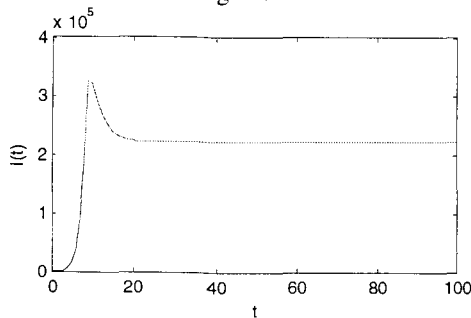


Fig.1. the number of infected when $R_1 > 1$

From our recorded data in Fig.1, we know that the worms have spread freely at the early stages of a worm incident, so the worm was spreading very fast. When the users have woken up to the harm of the worm and effective countermeasures have been taken, the number of infected hosts reduces. Due to $R_1 > 1$, the number of infected hosts $I(t)$ tends to a certain value.

(2) Let $N = 500000, I_0 = 100, \beta = 0.00002$, $\theta = 2, k = 0.2, \mu = 0.0003, \lambda = 0.2$, and we have $R_1 < 1$. The result of numerical simulation is presented in Fig. 2.

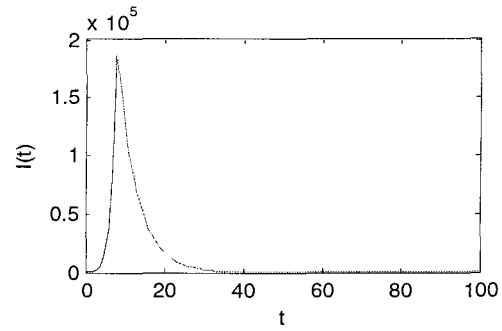


Fig.2. the number of infected when $R_1 < 1$

From Fig.2, we can see that the worm will eventually disappear if $R_1 < 1$, This is in line with the conclusions of Theorem 2.

(3) Let $N = 500000, I_0 = 100, \beta = 0.000002$, $\theta = 2, k = 0.2, \mu = 0.0000003, \lambda = 0.2$, let us see the result in Fig. 3.

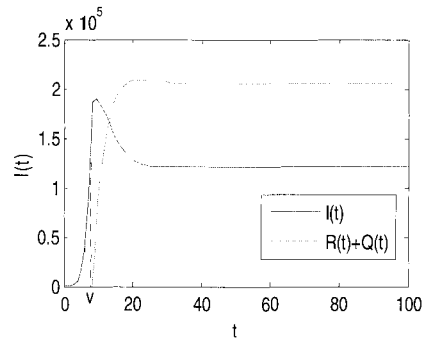


Fig.3. Influence of the number of infected hosts when β is Changed

(4) Let $N = 500000, I_0 = 100, \beta = 0.000002$, $\theta = 2, k = 0.6, \mu = 0.0000003, \lambda = 0.5$, let us see the result in Fig. 4.

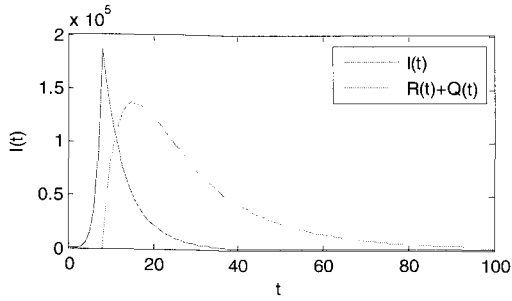


Fig.4. $I(t)$ and $E(t)$ when $R_1 < 1$

We describe $I(t)$ and $E(t)$ when $R_1 > 1$ and $R_1 < 1$ respectively in figure 3 and 4, and simulation results illustrates the theorem is correct.

4. Conclusion

In this paper, we build an improved Two-Factor model. By analyzing the model, we get the conditions for the disappearance of the worm. We can find that if the sample population N is certain, the disappearance of the worm is closely related to β and λ . According to Theorem 2, it is necessary to take some effective measures to reduce β and to increase λ . Thus we can provide some theoretical basis to prevention and treatment of worms.

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