

## Research Article

# Two Quarantine Models on the Attack of Malicious Objects in Computer Network

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SEIQR (Susceptible, Exposed, Infectious, Quarantined, and Recovered) models for the transmission of malicious objects with simple mass action incidence and standard incidence rate in computer network are formulated. Threshold, equilibrium, and their stability are discussed for the simple mass action incidence and standard incidence rate. Global stability and asymptotic stability of endemic equilibrium for simple mass action incidence have been shown. With the help of Poincare Bendixson Property, asymptotic stability of endemic equilibrium for standard incidence rate has been shown. Numerical methods have been used to solve and simulate the system of differential equations. The effect of quarantine on recovered nodes is analyzed. We have also analyzed the behavior of the susceptible, exposed, infected, quarantine, and recovered nodes in the computer network.

## 1. Introduction

It is a well-known fact that cyber world brought massive changes in the society. But nowadays cyber world is being threatened by the attack of malicious objects. Electronic mails and use of secondary devices are the major sources for the transmission of malicious objects in the computer network these days [1]. In accordance with their propagating behavior and characteristic, malicious objects spread in different way to each other. To curb the spread and impact of these malicious objects, it is important to study about their feature propagating methods, means, and limitation. Isolation may also be a very important and easy way to curb the transmission of these malicious objects. The word quarantine has evolved, meaning a forced isolation or stoppage of interactions with others. In biological world, quarantine has been adopted to reduce the transmission of human diseases, such as Leprosy, Plague, and Smallpox. Same concept has been adopted in the cyber world; the most infected nodes are isolated from the computer network till they get recovered. Anderson

and May [2, 3] discussed the spreading nature of biological viruses, parasite, and so forth. leading to infectious diseases in human population through several epidemic models. The action of malicious objects throughout a network can be studied by using epidemiological models for disease propagation [4–9]. Richard and Mark [10] proposed an improved SEI (Susceptible-Exposed-Infected) model to simulate virus propagation. However, they do not show the length of latency and take into account the impact of antivirus software. Mishra and Saini [11, 12] presented a SEIRS model with latent and temporary immune periods to overcome limitation, which can reveal common worm propagation. Feng and Thieme [13–15] considered very general endemic models that include SEIQR model, with arbitrarily distributed periods of infection including quarantine and with a general form for the incidence term that includes the three forms. Wa and Feng [16] showed that an epidemic approximation near threshold number ( $R_q = 1$ ) can have a homoclinic bifurcation, so that some perturbation of the original model might also have a homoclinic bifurcation. Several authors studied the global stability of several epidemiological models [17–24]. Wang et al. studied the robustness of filtering on nonlinearities in packet losses, sensors, and so forth, [25–30].

In the SEIQR models for infection that confers immunity, susceptible nodes go to latent period, that is, nodes become infected but not infectious called exposed nodes, thereafter some nodes go to infectious class. Some infected nodes remain in the infected class while they are infectious and then move to the recovered class after the run of antimalicious software. Other most infected nodes are transferred into the quarantine class while they are infectious and then move to the recovered class after their recovery. The models here have a variable total population size, because they have recruitment into the susceptible class by inclusion of some new nodes and they have both crashing of nodes due to reason other than the attack of malicious codes and crashing of nodes due to the attack of malicious codes. We have developed two models and have taken simple mass action incidence and standard incidence rate, because standard incidence rate is more realistic than the simple mass action incidence [31].

## 2. Model 1: Mathematical Formulation for the SEIQR Model with Simple Mass Action Incidence

Let  $S(t)$  be the number of susceptible at time  $t$ ,  $E(t)$  be the number of exposed,  $I(t)$  be the number of infected nodes,  $Q(t)$  be the number of quarantined nodes,  $R(t)$  be the recovered nodes after the run of antimalicious software, and  $N(t)$  be the total population size in time  $t$ . The schematic diagram for the flow of malicious objects is depicted in Figure 1.

As per our assumption, we have the following system of equations:

$$\begin{aligned}
 \frac{dS}{dt} &= A - \beta SI - dS, \\
 \frac{dE}{dt} &= \beta SI - (\gamma + d)E, \\
 \frac{dI}{dt} &= \gamma E - (d + \alpha_1 + \delta + \sigma - \eta)I, \\
 \frac{dQ}{dt} &= \delta I - (d + \alpha_2 + \varepsilon)Q, \\
 \frac{dR}{dt} &= \varepsilon Q + \sigma I - dR,
 \end{aligned} \tag{2.1}$$

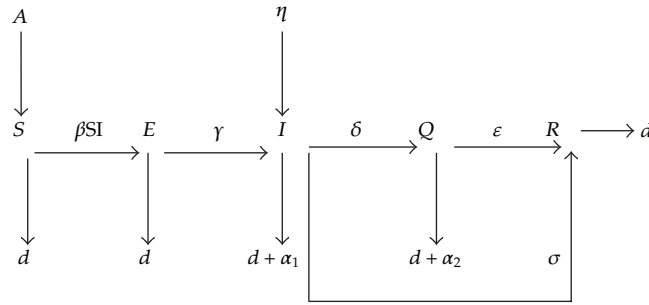


Figure 1: Schematic diagram for the flow of malicious objects in computer network.

where  $A$  is the recruitment rate of susceptible nodes,  $d$  is the per capita natural mortality rate,  $\alpha_1$  the death rate in infective compartment due to malicious objects, and  $\alpha_2$  the death rate in quarantine class due to malicious objects. The per capita contact rate  $\beta$  is average number of effective contacts with other nodes per unit time,  $\gamma_2$  is the rate constant which leaves the exposed compartment for infective class,  $\delta$  is the rate constant which leaves the infective class for quarantine class,  $\sigma$  is the rate by which the nodes go from quarantine class into recovered class, and  $\eta$  is the rate of vertical transmission into the infective class.

**Lemma 2.1.** Consider the following two systems:

$$\frac{dx}{dt} = f(t, x), \quad \frac{dy}{dt} = g(y), \tag{2.2}$$

where  $x, y$  belong to  $R^n$ ,  $f$  and  $g$  are continuous function which satisfy a local Lipschitz condition in any compact set  $X$  which belongs to  $R^n$ , and  $f(t, x) \rightarrow g(x)$  as  $t$  tends to infinity so that the second system is the limit system. Let  $\varphi(t, t_0, x_0)$  and  $\psi(t, t_0, y_0)$  be the solutions of these systems, respectively. Suppose that  $e \in X$  is a locally asymptotically stable equilibrium of the limit system and its attractive region is

$$W(e) = \{y \in X / \psi(t, t_0, x_0) \rightarrow e, t \rightarrow +\infty\}. \tag{2.3}$$

Let  $W_\phi$  be the omega limit set of  $\varphi(t, t_0, x_0)$ . If  $W_\phi \cap W(e) = \phi$ , then  $\lim_{t \rightarrow \infty} \varphi(t, t_0, x_0) = 0$ .

*Lipschitz's condition*

If for some function  $F(y)$ , the following condition is satisfied:

$$|F(y_2) - F(y_1)| \leq K|y_2 - y_1|. \tag{2.4}$$

where  $y_1$  and  $y_2$  are any points in the domain and  $K$  is a constant, this condition is called the Lipschitz's condition.

Now, the total population size  $N(t)$  satisfies the equation

$$\frac{dN}{dt} = A - dN - \alpha_1 I - \alpha_2 Q. \quad (2.5)$$

When  $t \rightarrow \infty$ , then from the above equation  $N \rightarrow A/d$ .

Let us define the solution region by  $D = \{(S, E, I, Q, R) / S \geq 0; E \geq 0; I \geq 0, Q \geq 0; R \geq 0; S + E + I + Q + R \leq A/D\}$ .

The system (2.1) always has the malicious oject-free equilibrium  $P_0 = (A/d, 0, 0, 0, 0)$ .

Here the quarantine reproduction number is

$$R_q = \frac{\beta(A/d)}{(\gamma + d)(d + \alpha_1 + \delta + \sigma - \eta)}. \quad (2.6)$$

If  $R_q > 1$ , then  $D$  also contains a unique positive, endemic equilibrium  $P^* = (S^*, E^*, I^*, Q^*, R^*)$ .

Now from (2.1), on simplification, we have

$$\begin{aligned} S^* &= \frac{A/d}{\gamma R_q}, & I^* &= (R_q \gamma - 1) \frac{d}{\beta}, \\ E^* &= \frac{A(R_q \gamma - 1)}{\gamma(\gamma + d)R_q}, & R^* &= \frac{1}{d} \left\{ \frac{\varepsilon \delta (\gamma R_q - 1)d}{\beta(d + \alpha_2 + \varepsilon)} + \frac{\sigma d (\gamma R_q - 1)}{\beta} \right\}, & Q^* &= \frac{(\gamma R_q - 1)d\delta}{\beta(d + \alpha_2 + \varepsilon)}. \end{aligned} \quad (2.7)$$

We have  $N^* = S^* + E^* + I^* + Q^* + R^*$ , Hence,

$$N^* = \frac{A/d}{\gamma R_q} + \frac{A(\gamma R_q - 1)}{\gamma(\gamma + d)R_q} + \frac{d}{\beta} (R_q \gamma - 1) + \frac{1}{d} \left\{ \frac{\varepsilon \delta (R_q \gamma - 1)d}{\beta(d + \alpha_2 + \varepsilon)} + \frac{\sigma d (\gamma R_q - 1)}{\beta} \right\}. \quad (2.8)$$

**Theorem 2.2.** Consider the system (2.1). If  $R_q < 1$ , then solution set  $D = \{(S, E, I, Q, R) / S \geq 0; E \geq 0; I \geq 0, Q \geq 0; R \geq 0; S + E + I + Q + R \leq A/D\}$  is locally asymptotically stable for disease-free equilibrium  $P_0$ . If  $R_q > 1$ , then the region  $D - \{(S, E, I, Q, R) / I = 0\}$ , is an asymptotically stable region for the endemic equilibrium  $P^*$ .

*Proof.* For local stability, Jacobian of system (2.1) at equilibrium  $P_0$  is

$$J_{p_0} = \begin{bmatrix} -d & 0 & -\beta(A/d) & 0 & 0 \\ 0 & -(\gamma + d) & \beta(A/d) & 0 & 0 \\ 0 & \gamma & -(d + \alpha_1 + \delta + \sigma - \eta) & 0 & 0 \\ 0 & \gamma & \delta & -(d + \alpha_2 + \varepsilon) & 0 \\ 0 & 0 & \sigma & \varepsilon & -d \end{bmatrix}. \quad (2.9)$$

The eigenvalues of  $J_{p_0}$  are  $-d; -(\gamma + d); -(d + \alpha_1 + \delta + \sigma - \eta); -(d + \alpha_2 + \varepsilon); -d$ . Since all the roots are real and negative, system is locally asymptotically stable.

In order to prove the global stability when  $R_q \leq 1$ , consider the Liapunov function  $V = I$ . Liapunov derivative  $dV/dt = \gamma E - I(d + \alpha_1 + \sigma + \delta - \eta)$ :

$$\begin{aligned}\frac{dV}{dt} &= \gamma(\gamma R_q - 1) \frac{d}{\beta} (d + \alpha_1 + \delta + \sigma - \eta), \\ \frac{dV}{dt} &= \gamma\{\gamma R_q - 1\} \frac{d}{\beta} - (d + \alpha_1 + \delta + \sigma - \eta)I \leq 0, \quad (\text{since } R_q \gamma - 1 \leq 0).\end{aligned}\tag{2.10}$$

As we know the Liapunov Lasalle theorem [17] implies that solutions in  $D$  approach the largest positively invariant subset of the set where  $dV/dt = 0$ , which is the set where  $I = 0$ .

In this set,

$$\begin{aligned}\frac{dQ}{dt} &= -(d + \alpha_2 + \varepsilon)Q, \\ \frac{dS}{dt} &= A - dS.\end{aligned}\tag{2.11}$$

We have

$$Q = \frac{1}{e^{(d+\alpha_2+\varepsilon)t}}\tag{2.12}$$

when

$$t \rightarrow \infty,\tag{2.13}$$

then

$$\begin{aligned}Q &\rightarrow 0, \\ S &\rightarrow \frac{A}{d}.\end{aligned}\tag{2.14}$$

We have from (2.1),

$$\frac{dR}{dt} = \varepsilon Q + \sigma I - dR;\tag{2.15}$$

this implies

$$R = e^{-t}.\tag{2.16}$$

Thus,

$$R \longrightarrow 0 \quad (2.17)$$

when

$$t \longrightarrow \infty. \quad (2.18)$$

Thus, all solutions in the set  $I = 0$  go to the disease, free equilibrium  $P_0$ . By Lemma 2.1, the system is globally asymptotically stable, when  $R_q < 1$ .

From the fourth equation in system (2.1), we can solve to obtain

$$Q(t) = \frac{\left\{ Q_0 + \int_{t_0}^t \delta I(\tau) e^{(d+\alpha_2+\varepsilon)(\tau-t_0)} d\tau \right\}}{e^{(\varepsilon+d+\alpha_2)(t-t_0)}}. \quad (2.19)$$

Now,  $\lim_{t \rightarrow \infty} Q(t) = \lim_{t \rightarrow \infty} (\delta I(t)) / (\varepsilon + d + \alpha_2)$ . This implies  $Q^* = \delta I^* / (\varepsilon + d + \alpha_2)$ ;

The similarly, solving for  $R$  by using, fifth equation in (2.1), we obtain

$$\lim_{t \rightarrow \infty} R(t) = \lim_{t \rightarrow \infty} \frac{\varepsilon Q(t) + \sigma I(t)}{d}, \quad \implies R^* = \frac{\sigma I^* + \varepsilon Q^*}{d}. \quad (2.20)$$

An application of Lemma 2.1 shows that the endemic equilibrium  $P^*$  of model (2.1) is globally asymptotically stable in the region  $D - \{(S, E, I, Q, R) / I = 0\}$ .  $\square$

### 3. Model 2: The SEIQR Model with the Standard Incidence Rate

The flow chart for the SEIQR model will be the same as depicted in Figure 1, but instead of simple mass action incidence  $\beta SI$ , we take standard incidence rate  $\beta SI/N$ , where  $N = S + E + I + Q + R$ .

The system of differential equations for this model is

$$\begin{aligned} \frac{dS}{dt} &= A - \frac{\beta SI}{N} - dS, \\ \frac{dE}{dt} &= \frac{\beta SI}{N} - (\gamma + d)E, \\ \frac{dI}{dt} &= \gamma E - (d + \alpha_1 + \delta + \sigma - \eta)I, \\ \frac{dQ}{dt} &= \delta I - (d + \alpha_2 + \varepsilon)Q, \\ \frac{dR}{dt} &= \varepsilon Q + \sigma I - dR, \end{aligned} \quad (3.1)$$

where the parameters are the same as in the previous model,

$$\frac{dN}{dt} = A - dN - \alpha_1 I - \alpha_2 Q. \quad (3.2)$$

When  $t \rightarrow \infty$  then  $N \rightarrow A/d$ . When there are malicious objects free equilibrium  $P_0 = (A/d, 0, 0, 0, 0)$ . For this model basic reproduction number is

$$R_q = \frac{\beta}{(\gamma + d)(d + \alpha_1 + \delta + \sigma - \eta)}. \quad (3.3)$$

If  $R_q > 1$ , then  $D$  also contains a unique positive, endemic equilibrium  $P^* = (S^*, E^*, I^*, Q^*, R^*)$  where

$$\begin{aligned} S^* &= [1 / \{ -(\gamma + d)(d + \alpha_2 + \varepsilon)\beta + \gamma R_q(\gamma + d)\beta(d + \alpha_2 + \varepsilon) + d\beta(d + \alpha_2 + \varepsilon) \\ &\quad + \gamma R_q(\gamma + d)(\alpha_2 + \varepsilon + d) + \delta \gamma R_q d(\gamma + d) + \varepsilon \gamma R_q(\gamma + d) \\ &\quad + \sigma \gamma R_q(\gamma + d)(d + \alpha_2 + \varepsilon) \}] \\ &\quad \times [\beta A d(d + \alpha_2 + \varepsilon) + \gamma R_q d(\gamma + d)(d + \alpha_2 + \varepsilon) \\ &\quad + \delta \gamma R_q A(\gamma + d)d + \varepsilon \delta \gamma R_q A(\gamma + d) + \sigma \gamma R_q A(\gamma + d)(d + \alpha_2 + \varepsilon)], \\ E^* &= \frac{1}{(\gamma + d)}(A - dS^*), \\ I^* &= \frac{\gamma R_q(A - dS^*)}{\beta}, \quad Q^* = \frac{\delta}{(d + \alpha_2 + \varepsilon)} \times \frac{\gamma R_q(A - dS^*)}{\beta}, \\ R^* &= \frac{\varepsilon \delta \gamma R_q(A - dS^*)}{\beta d(d + \alpha_2 + \varepsilon)} + \frac{\sigma \gamma R_q(A - dS^*)}{\beta d}. \end{aligned} \quad (3.4)$$

**Theorem 3.1.** Consider the system (3.1). If  $R_q < 1$ , then solution set  $D = \{(S, E, I, Q, R) / S \geq 0; E \geq 0; I \geq 0; Q \geq 0; R \geq 0; S + E + I + Q + R \leq A/D\}$  is locally asymptotic stable for disease-free equilibrium  $P_0$ . If  $R_q > 1$ , then the region  $D - \{(S, E, I, Q, R) / I = 0\}$  is an asymptotically stable region for the endemic equilibrium  $P^*$ .

*Proof.* For the local stability, Jacobian of the system (3.1) at equilibrium  $P_0$  is

$$J_{P_0} = \begin{bmatrix} -d & 0 & -\beta \frac{(A/D)}{N} & 0 & 0 \\ 0 & -(d + \gamma) & \beta \left( \frac{A/D}{N} \right) & 0 & 0 \\ 0 & \gamma & -(d + \alpha_1 + \delta + \sigma - \eta) & 0 & 0 \\ 0 & 0 & \delta & -(d + \alpha_2 + \varepsilon) & 0 \\ 0 & 0 & \sigma & \varepsilon & -d \end{bmatrix}. \quad (3.5)$$

**Table 1:** Parametric values used in simulating the models.

Parameters	Simple mass action	Standard incidence rate
$N$	10,000	10,000
$S(0)$	9900	9900
$E(0)$	100	100
$I(0)$	0	0
$Q(0)$	0	0
$R(0)$	0	0
$\beta$	0.1	0.01
$\gamma$	0.09	0.09
$\delta$	0.01	0.09
$\varepsilon$	0.07	0.07
$\sigma$	0.09	0.03
$A$	0.01	0.01
$d$	0.01	0.01
$\alpha_1$	0.1	0.03
$\alpha_2$	0.1	0.04
$\eta$	0.01	0.03

The eigenvalues of  $J_{P_0}$  are  $-d$ ;  $-(d + \gamma)$ ;  $-(d + \alpha_1 + \delta + \sigma - \eta)$ ;  $(d + \alpha_2 + \varepsilon)$ ;  $-d$ . Since all the roots are real and negative, system is locally asymptotically stable.

With a view to prove the global stability when  $R_q \leq 1$ , we use the Liapunov function by putting  $V = I$ , we get the same equation as we have found in the model 1; therefore, the proof will be analogous with the proofs in the previous section.

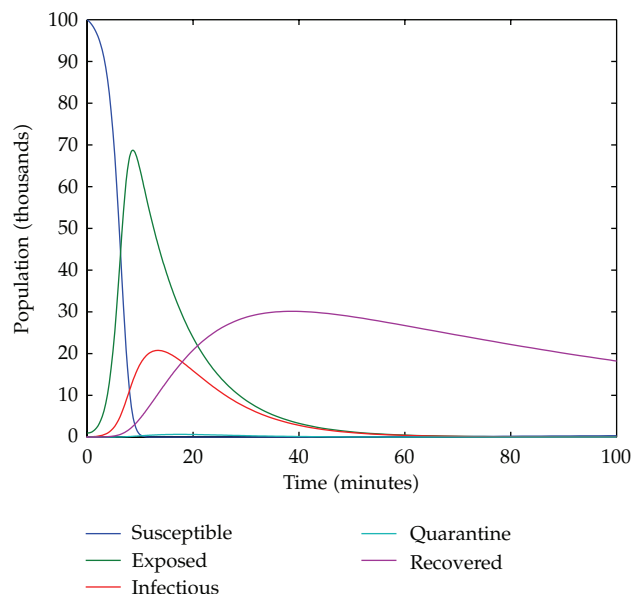
In order to prove the global stability when  $R_q > 1$  and  $\alpha_1 = \alpha_2 = 0$ , first we get  $dN/dt = A - Nd$ , this implies  $N \rightarrow A/d$  when  $t$  tends to infinity.

The limit system for (3.1) is

$$\begin{aligned}
 \frac{dS}{dt} &= A - \frac{d\beta SI}{A} - dS, \\
 \frac{dE}{dt} &= \frac{d\beta SI}{A} - (\gamma + d)E, \\
 \frac{dI}{dt} &= \gamma E - (d + \alpha_1 + \delta + \sigma - \eta), \\
 \frac{dQ}{dt} &= \delta I - (d + \alpha_2 + \varepsilon)Q, \\
 \frac{dR}{dt} &= \varepsilon Q + \sigma I - dR,
 \end{aligned} \tag{3.6}$$

where  $N = A/d$ . The first three equations are independent of  $Q$  and  $R$ . In the three dimensional  $S E I$  first octant region with  $S + E + I \leq A/d$ , the equilibrium  $(0, 0, 0)$  is saddle, that is, attractive along  $I = 0$  and has a repulsive direction into the region. The other equilibrium  $(S^*, I^*)$  in the region is locally asymptotically stable.





**Figure 2:** Time series of susceptible population  $S(t)$ ,  $E(t)$ ,  $I(t)$ ,  $Q(t)$ , and  $R(t)$  of the system (2.1).

Using Dulac's criteria with multiplier  $1/I$ , we have

$$\begin{aligned} \frac{\partial}{\partial S} \left[ \frac{A}{I} - \frac{d\beta S}{A} - \frac{dS}{I} \right] + \frac{\partial}{\partial E} \left[ \frac{d\beta S}{A} - \frac{E}{I}(\gamma + d) \right] + \frac{\partial}{\partial I} \left[ \frac{\gamma E}{I} - (d + \alpha_1 + \delta + \sigma - \eta) \right] \\ = -\frac{d\beta}{A} - \frac{d}{I} - (\gamma + d) \frac{1}{I} - \gamma \frac{E}{I^2} < 0, \end{aligned} \quad (3.7)$$

so that there are no periodic solutions in the region. Thus, by the Poincare-Bendixson theory, all solutions starting in the first octant region with  $I > 0$  and  $S + E + I \leq A/d$  approach  $(S^*, E^*, I^*)$  as  $t$  tends infinity.

In this case, the differential equation for  $Q$  has the limiting equation

$$\frac{dQ}{dt} = \delta I - (d + \varepsilon + \alpha_2)Q, \quad (3.8)$$

so that  $Q$  tends to  $Q^*$  by Lemma 2.1.

Similarly, the differential equation for  $R$  has the limiting equation

$$\frac{dR}{dt} = \varepsilon Q^* + \sigma I^* - dR, \quad (3.9)$$

so that  $R$  tends to  $R^*$  by Lemma 2.1. Thus  $P^*$  is a globally asymptotically stable equilibrium for the limit system (3.6). Hence, by Lemma 2.1, all solutions starting in the region  $D - \{(S, E, I, Q, R) / I = 0\}$  of the system (3.1) approach the endemic equilibrium  $P^*$  as  $t$  tends to infinity.

□

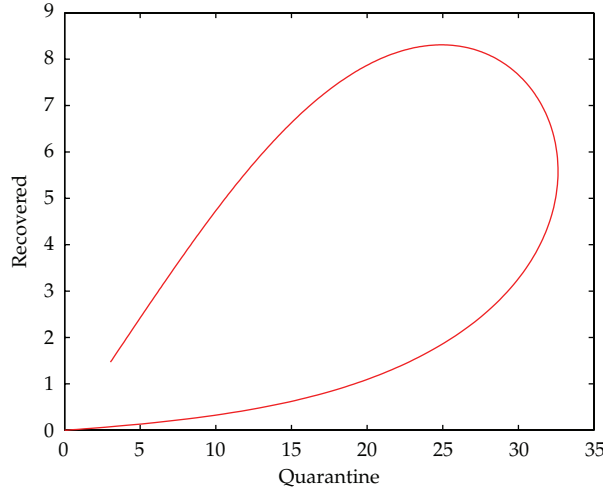


Figure 3: Effect of quarantine  $Q$  on recovered nodes  $R$ .

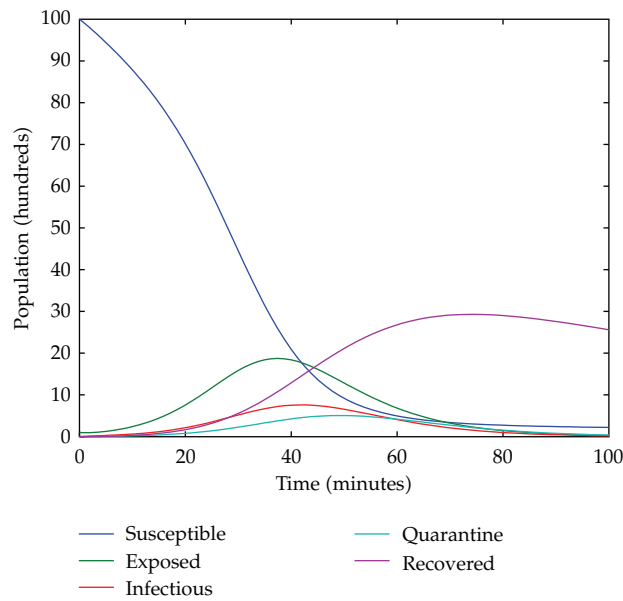


Figure 4: Time series of susceptible population  $S(t)$ ,  $E(t)$ ,  $I(t)$ ,  $Q(t)$ , and  $R(t)$  of the system (3.1).

#### 4. Conclusion

Inspired by the biological compartmental epidemic model, we made an attempt to develop two SEIQR models, one using simple mass action incidence and the other using standard incidence rate. Vertical transmission has been included into infectious compartment. Runge Kutta Fehlberg fourth-fifth order method is used to solve and simulate the system (2.1) and (3.1) by using parametrical values mentioned in Table 1. The model has a constant recruitment of the nodes and exponential natural and infection-related death (crashing) of the nodes. Global stability of the unique endemic equilibrium for the epidemic model has been established. We observe that the behavior of the Susceptible, Exposed, Infected, and

Quarantined nodes with respect to time is asymptotically stable, which is depicted in Figures 2 and 4. The effect of  $Q$  on  $R$  is depicted in Figure 3. When the nodes are highly infected by different kinds of malicious objects, quarantine is one of the remedy. We run antimalicious software of latest signature against quarantined nodes, and these nodes are kept under observation. The more we quarantine the most infected nodes, the more the recovery is; the lesser we quarantine, the lesser the recovery is. Also at a very specific short interval of time, the recovery of the nodes is constant when the quarantine node decreases. These can be observed in Figure 3. Simulation result agrees with the real life situation. The basic reproduction number  $R_q$  is obtained and has been identified as a threshold parameter. If  $R_q \leq 1$ , the disease-free equilibrium  $D$  is globally stable in the feasible region and the disease always dies out. If  $R_q > 1$ , a unique endemic equilibrium  $P^*$  exists and is globally stable in the interior of the feasible region, and once the disease appears, it eventually persists at the unique endemic equilibrium level. Lyapunov function is used to prove the global stability of  $D$  when  $R_q \leq 1$ . In our model, the number of contacts is influenced by the size of the quarantine class  $Q$ . The quarantine process is an alternative method for reducing the average infectious period by isolating some infectives, so that they do not transmit the malicious objects in the computer network. We have observed that both the effective infectious period  $1/(d + \alpha_1 + \delta + \eta)(\gamma + d)$  and  $R_q$  decrease as the quarantine rate  $\delta$  increases.

The analysis of quarantine reproduction number  $R_q$  by Feng and Thieme [14, 15] and Hethcote et al. [31] agrees with our model. Feng and Thieme [14, 15] in their SIQR model observed that the quarantine reproduction number was independent of the mean residence time in the quarantine class  $Q$ . Hethcote et al. [31] also had their same observation regarding the independence of the mean residence time in the  $Q$  class for all of their endemic models. We also have the same observation for our SEIQRS model. The mean residence time in the class  $Q$  for our model SEIQRS is  $1/\varepsilon$ . The expression for the threshold does not involve the parameter  $\varepsilon$ . This comes from our assumption that the nodes in the quarantine class  $Q$  do not infect other nodes and nodes are not infectious when they move out of the quarantine class. The quarantine reproduction number  $R_q$ , depends on parameter  $\delta$ . For example, if  $\delta = n\sigma, n \in \mathbb{Z}^+$ , then transfer out of infectious class  $I$  to quarantine class  $Q$  is  $n$  times as frequent as transfer to the removed class  $R$ . A positive rate constant  $\delta$  to transfer out of infectious class  $I$  by quarantine does decrease the quarantine reproduction number  $R_q$ , so that it is less than its value without quarantine. Hence the use of quarantine to control a disease not only decreases the endemic infective class size when  $R_q$  remains above 1, but also makes it easier to obtain  $R_q \leq 1$  leading to disease extinction.

The future work will involve in taking time delay constraints in various compartments which may lead to more interesting result.

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## References

- [1] M. E. J. Newman, S. Forrest, and J. Balthrop, "Email networks and the spread of computer viruses," *Physical Review E*, vol. 66, no. 3, Article ID 035101, 4 pages, 2002.
- [2] R. M. Anderson and R. M. May, *Infection Disease of Humans, Dynamics and Control*, Oxford University Press, Oxford, UK, 1992.

- [3] R. M. Anderson and R. M. May, "Population biology of infectious diseases," *Nature*, vol. 180, pp. 361–367, 1999.
- [4] S. Forrest, S. Hofmeyr, A. Somayaji, and T. Longstaff, "Self-nonsel self discrimination in a computer," in *Proceedings of the IEEE Symposium on Research in Security and Privacy*, pp. 202–212, May 1994.
- [5] E. Gelenbe, "Keeping viruses under control," in *Proceedings of the 20th International Symposium on Computer and Information Sciences (ISCIS '05)*, vol. 3733 of *Lecture Notes in Computer Science*, pp. 304–311, Springer, October 2005.
- [6] E. Gelenbe, "Dealing with software viruses: a biological paradigm," *Information Security Technical Report*, vol. 12, no. 4, pp. 242–250, 2007.
- [7] E. Gelenbe, V. Kaptan, and Y. Wang, "Biological metaphors for agent behavior," in *Computer Science*, pp. 667–675, Springer-Verlag, 2004.
- [8] J. R. C. Piqueira and F. B. Cesar, "Dynamical models for computer viruses propagation," *Mathematical Problems in Engineering*, vol. 2008, Article ID 940526, 11 pages, 2008.
- [9] J. R. C. Piqueira, B. F. Navarro, and L. H. A. Monteiro, "Epidemiological models applied to virus in computer networks," *Journal of Computer Science*, vol. 1, no. 1, pp. 31–34, 2005.
- [10] W. T. Richard and J. C. Mark, "Modeling virus propagation in peer-to-peer networks," in *Proceedings of the 5th International Conference on Information, Communications and Signal Processing*, pp. 981–985, December 2005.
- [11] B. K. Mishra and D. K. Saini, "SEIRS epidemic model with delay for transmission of malicious objects in computer network," *Applied Mathematics and Computation*, vol. 188, no. 2, pp. 1476–1482, 2007.
- [12] B. K. Mishra and D. K. Saini, "Mathematical models on computer viruses," *Applied Mathematics and Computation*, vol. 187, no. 2, pp. 929–936, 2007.
- [13] Z. Feng and H. R. Thieme, "Recurrent outbreaks of childhood diseases revisited: the impact of isolation," *Mathematical Biosciences*, vol. 128, no. 1-2, pp. 93–130, 1995.
- [14] Z. Feng and H. R. Thieme, "Endemic models with arbitrarily distributed periods of infection I: general theory," *SIAM Journal on Applied Mathematics*, vol. 61, no. 3, pp. 803–833, 2000.
- [15] Z. Feng and H. R. Thieme, "Endemic models with arbitrarily distributed periods of infection II: fast disease dynamics and permanent recovery," *SIAM Journal on Applied Mathematics*, vol. 61, no. 3, pp. 983–1012, 2000.
- [16] L. I. Wu and Z. Feng, "Homoclinic bifurcation in an SIQR model for childhood diseases," *Journal of Differential Equations*, vol. 168, no. 1, pp. 150–167, 2000.
- [17] K. L. Cooke and P. van den Driessche, "Analysis of an SEIRS epidemic model with two delays," *Journal of Mathematical Biology*, vol. 35, no. 2, pp. 240–260, 1996.
- [18] M. Y. Li, J. R. Graef, L. C. Wang, and J. Karsai, "Global dynamics of a SEIR model with varying total population size," *Mathematical Biosciences*, vol. 160, no. 2, pp. 191–213, 1999.
- [19] D. Greenhalgh, "Hopf bifurcation in epidemic models with a latent period and nonpermanent immunity," *Mathematical and Computer Modelling*, vol. 25, no. 2, pp. 85–107, 1997.
- [20] J. K. Hale, *Ordinary Differential Equations*, Krieger, Basel, Switzerland, 2nd edition, 1980.
- [21] H. W. Hethcote, H. W. Stech, and P. van dan Driessche, "Periodicity and stability in epidemic models: a survey," in *Differential Equations and Applications in Ecology, Epidemics and Population Problems*, K. L. Cook, Ed., pp. 65–85, Academic Press, New York, NY, USA, 1981.
- [22] M. Y. Li and J. S. Muldowney, "Global stability for the SEIR model in epidemiology," *Mathematical Biosciences*, vol. 125, no. 2, pp. 155–164, 1995.
- [23] M. Y. Li and L. Wang, "Global stability in some SEIR epidemic models," in *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory*, C. Chavez, S. Blower, P. van den Driessche, D. Kirschner, and A. Yakhdu, Eds., vol. 126, pp. 295–311, Springer, New York, NY, USA, 2002.
- [24] Y. Michel, H. Smith, and L. Wang, "Global dynamics of an seir epidemic model with vertical transmission," *SIAM Journal on Applied Mathematics*, vol. 62, no. 1, pp. 58–69, 2001.
- [25] H. Dong, Z. Wang, and H. Gao, "Observer-based  $H_\infty$  control for systems with repeated scalar nonlinearities and multiple packet losses," *International Journal of Robust and Nonlinear Control*, vol. 20, no. 12, pp. 1363–1378, 2010.
- [26] Z. Wang, D. W. C. Ho, H. Dong, and H. Gao, "Robust  $H_\infty$  finite-horizon control for a class of stochastic nonlinear time-varying systems subject to sensor and actuator saturations," *IEEE Transactions on Automatic Control*, vol. 55, no. 7, Article ID 5441037, pp. 1716–1722, 2010.
- [27] B. Shen, Z. Wang, and Y. S. Hung, "Distributed  $H_\infty$ -consensus filtering in sensor networks with multiple missing measurements: the finite-horizon case," *Automatica*, vol. 46, no. 10, pp. 1682–1688, 2010.

- [28] B. Shen, Z. Wang, H. Shu, and G. Wei, "Robust  $H_\infty$  finite-horizon filtering with randomly occurred nonlinearities and quantization effects," *Automatica*, vol. 46, no. 11, pp. 1743–1751, 2010.
- [29] B. Shen, Z. Wang, and X. Liu, "Bounded  $H$ -infinity synchronization and state estimation for discrete time-varying stochastic complex networks over a finite-horizon," *IEEE Transactions on Neural Networks*, vol. 22, no. 1, pp. 145–157, 2011.
- [30] H. Dong, Z. Wang, D. W. C. Ho, and H. Gao, "Variance-constrained  $H_\infty$  filtering for a class of nonlinear time-varying systems with multiple missing measurements: the finite-horizon case," *IEEE Transactions on Signal Processing*, vol. 58, no. 5, Article ID 5410142, pp. 2534–2543, 2010.
- [31] H. Hethcote, M. Zhen, and L. Shengbing, "Effects of quarantine in six endemic models for infectious diseases," *Mathematical Biosciences*, vol. 180, pp. 141–160, 2002.



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