IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

Effect of Quarantine On The Transmission Dynamics Of Malicious Attacks In Network With VerticalTransmission

1 DR. VIVEK SINGH 2 DR. ASHOK KUMAR

1 Assistant Professor 2 Assistant Professor

Department of Mathematic RTC Institute of Technology, Ranchi, India

ABSTRACT

An attempt is made to analyze a susceptible- exposed-infectious-quarantine –vaccinated and susceptible (SEIQVS) e- epidemic model for the attack of malicious objects through vertical transmission in networks. Temporary immunity (vaccination) and very specifically quarantine plays an important role in controlling the attack is incorporated in this model. The basic reproduction number is found, under certain conditions on the incidence rate and treatment function. It is shown that the model exhibits two equilibria, namely, the disease-free equilibrium and the endemic equilibrium. Equilibrium analysis is presented and it is found that in each case the equilibrium points are locally asymptotically stable under certain conditions. The stability of the equilibrium is established by using the Routh-Hurwitz criteria.

Keywords: cyber attack; R_0 computer network; vertical transmission; quarantine; vaccination; local asymptotic stability.

1. INTRODUCTION:

Mathematical models which describes the dynamics of infectious disease have recently played a crucial role in the disease control in epidemiological aspect. Many authors have proposed various kinds of epidemic models to understand the mechanism of disease transmission [1,2,3,4] .

Based on the infectivity between a worm and a biological virus, some epidemic models representing worm propagations were presented to depict the propagation of worms, e.g., *SIR* model [5], *SIRS* model [6], [7], *SIQ* model [8], *SEIR* model [9], *SEIRS* model [10], [11], *SEIQV* model [12], *SEIQRS* model [13], which assume that infected hosts in which the worm resides arein an exposed state can not infect other hosts. Actually, an infected host which is in latency can infect other hosts by means of some methods, e.g., vulnerability seeking. All the previous models do not take this passive infectivity into consideration. Recently, Yang et al. proposed some models [14], [15], [16], by taking into account the fact that a host immediately possesses infectivity once it is infected. These models, however, all make an assumption that exposed hosts and infected hosts have the same infectivity [17].

2. THE MATHEMATICAL MODEL AND FORMULATION:

\The schematic description of our model is in following figure:

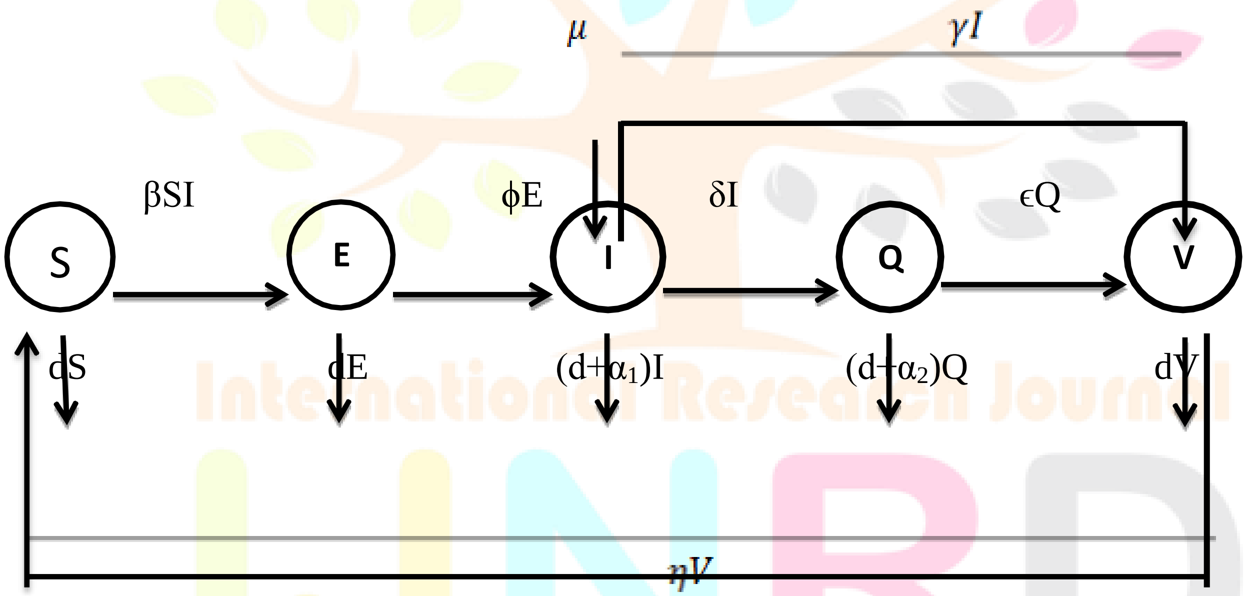

Fig 10

The transfer diagram leads to the following system of differential equations:

$$\left.\begin{aligned}
\frac{dS}{dt} &= A - \beta SI - dS + \eta V \\
\frac{dE}{dt} &= \beta SI - (d + \phi)E \\
\frac{dI}{dt} &= \phi E - (d + \alpha_1 + \gamma + \delta - \mu)I \\
\frac{dQ}{dt} &= \delta I - (d + \alpha_2 + \varepsilon)Q \\
\frac{dV}{dt} &= \varepsilon Q + \gamma I - (d + \eta)V
\end{aligned}\right\} \quad (1)$$

N(t)= S(t)+E(t)+I(t)+Q(t)+V(t)

TABLE 1: NOMENCLATURE

(1)

SYMBOL	DESCRIPTION	A
	Constant A is the recruitment rate of susceptible class	
S	Number of nudes in the susceptible class	
E	Number of nudes which are exposed	
I	Number of nudes which are infectious	
Q	Number of individuals which are quarantined	
V	Number of nudes with vaccination	
β	Infectivity contact rate	
γ	Rate of infectious to vaccination class	
δ	Rate of infectious class to quarantine class	
ε	Rate of quarantine class to vaccinated class	
η	Susceptible after vaccination	
ϕ	Rate of exposed class to infectious class Vertical	
μ	transmission in the infectious class	
d	Natural death rate	

α_1	Death due to malicious objects in Infected Class
α_2	Death due to malicious objects in Quarantine Class

3. Stability Analysis:

For the equilibrium points the above differential equation should be equated to zeroi.e

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dQ}{dt} = \frac{dV}{dt} = 0$$

We have five equilibrium points are given by $P_0 = (A, 0,0,0,0)$ is the disease freeequilibrium points of the system (1) and the unique endemic equilibrium points

$$P^* = (S^*, E^*, I^*, Q^*, V^*), where$$

$$S^* = \frac{(d + \alpha_1 + \gamma + \delta - \mu)}{\beta}$$

$$E^* = \frac{(d + \alpha_1 + \gamma + \delta - \mu)(d + \phi)}{\delta\phi(\phi - 1)}$$

$$I^* = \frac{(d + \phi)}{\delta\phi(\phi - 1)}$$

$$Q^* = \frac{(d + \phi)}{(d + \alpha_2 + \varepsilon)(d + \phi)}$$

$$V^* = \frac{\frac{(d+\phi)}{(\phi-1)}\left\{\frac{\varepsilon}{(d+\alpha_2+\varepsilon)} + \frac{\gamma}{\delta}\right\}}{(d + \eta)}$$

The reproduction number given by

$$R_0 = \frac{\phi}{((d + \alpha_1 + \gamma + \delta) - \mu)}$$

Theorem. The disease free equilibrium of the system is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: We consider the following equations

$$F_1 = A - \beta SI - dS + \eta V$$

$$F_2 = \beta SI - (d + \phi)E$$

$$F_3 = \phi E - (d + \alpha_1 + \gamma + \delta - \mu)I$$

$$F_4 = \delta I - (d + \alpha_2 + \varepsilon)Q$$

$$F_5 = \varepsilon Q + \gamma I - (d + \eta)V$$

The variation matrix of the system (1) is given by

$$J = \begin{bmatrix} -\beta I - d & 0 & \beta S & 0 & 0 \\ \beta I & -(d+\phi) & \beta S & 0 & 0 \\ 0 & \phi & -(d+\alpha_1+\gamma+\delta)+\mu & 0 & 0 \\ 0 & 0 & \delta & -(d+\alpha_2+\varepsilon) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta) \end{bmatrix}$$

At the equilibrium point $P_0 = (A, 0,0,0,0)$ the Jacobean matrix becomes

$$J = \begin{bmatrix} -d & 0 & \beta A & 0 & 0 \\ 0 & -(d+\phi) & \beta A & 0 & 0 \\ 0 & \phi & \mu-(d+\alpha_1+\gamma+\delta) & 0 & 0 \\ 0 & 0 & \delta & -(d+\alpha_2+\varepsilon) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta) \end{bmatrix}$$

The characteristic equation $|J - \lambda I| = 0$,can be written as

$$J = \begin{vmatrix} -d-\lambda & 0 & \beta A & 0 & 0 \\ 0 & -(d+\phi)-\lambda & \beta A & 0 & 0 \\ 0 & \phi & \mu-(d+\alpha_1+\gamma+\delta)-\lambda & 0 & 0 \\ 0 & 0 & \delta & -(d+\alpha_2+\varepsilon)-\lambda & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta)-\lambda \end{vmatrix} = 0$$

$$J = \begin{vmatrix} -(\lambda+d) & 0 & \beta A & 0 & 0 \\ 0 & -(\lambda+d+\phi) & \beta A & 0 & 0 \\ 0 & \phi & \mu-(d+\alpha_1+\gamma+\delta)-\lambda & 0 & 0 \\ 0 & 0 & \delta & -(\lambda+d+\alpha_2+\varepsilon) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(\lambda+d+\eta) \end{vmatrix} = 0$$

$$(\lambda + d)\,(\lambda + d + \phi)(\lambda + d + \alpha_2 + \varepsilon)(\lambda + d + \eta) = 0$$

It is evident that four Eigen values are negative and one Eigen value is negative if $\mu - (d + \alpha_1 + \gamma + \delta) < 1$ or $\frac{\phi}{(d+\alpha_1+\gamma+\delta)-\mu} < 1$

i.e $R_0 < 1$

Therefore, all the Eigen value of the characteristic equation are negative. Hence the equilibrium point P_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem. If $R_0 > 1$, the endemic equilibrium P^* is locally asymptotically stable.

Proof: we consider the following equations

$$F_1 = A - \beta SI - dS + \eta V$$

$$F_2 = \beta SI - (d + \phi)E$$

$$F_3 = \phi E - (d + \alpha_1 + \gamma + \delta + \mu)I$$

$$F_4 = \delta I - (d + \alpha_2 + \varepsilon)Q$$

$$F_5 = \varepsilon Q + \gamma I - (d + \eta)V$$

The variation matrix of the system (1) is given by

$$J = \begin{bmatrix} -\beta I - d & 0 & \beta S & 0 & 0 \\ \beta I & -(d+\phi) & \beta S & 0 & 0 \\ 0 & \phi & \mu - (d + a_1 + \gamma + \delta) & 0 & 0 \\ 0 & 0 & \delta & -(d + \alpha_2 + \varepsilon) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta) \end{bmatrix}$$

At the endemic equilibrium point $P^* = S^*, E^*, I^*, Q^*, V^*$

$$J^* = \begin{bmatrix} -\beta I^* - d & 0 & \beta S^* & 0 & 0 \\ \beta I^* & -(d+\phi) & \beta S^* & 0 & 0 \\ 0 & \phi & \mu - (d + a_1 + \gamma + \delta) & 0 & 0 \\ 0 & 0 & \delta & -(d + \alpha_2 + \varepsilon) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta) \end{bmatrix}$$

Let $J_1 = \beta I^*$

$J_2 = \beta S^*$

Then,

$$J^* = \begin{bmatrix} -J_1 - d & 0 & J_2 & 0 & 0 \\ J_1 & -(d+\phi) & J_2 & 0 & 0 \\ 0 & \phi & \mu - (d + a_1 + \gamma + \delta) & 0 & 0 \\ 0 & 0 & \delta & -(d + \alpha_2 + \varepsilon) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta) \end{bmatrix}$$

The characteristic equation $|J^* - \lambda I| = 0$ can be written as

$$\begin{vmatrix} -(J_1 + d + \lambda) & 0 & J_2 & 0 & 0 \\ J_1 & -(d+\phi+\lambda) & J_2 & 0 & 0 \\ 0 & \phi & \mu - (d + a_1 + \gamma + \delta) & 0 & 0 \\ 0 & 0 & \delta & -(d + \alpha_2 + \varepsilon + \lambda) & 0 \\ 0 & 0 & \gamma & \varepsilon & -(d+\eta+\lambda) \end{vmatrix} = 0$$

//

$$(J_1 + d + \lambda)\ (d + \phi + \lambda)(d + \alpha_2 + \varepsilon + \lambda)(d + \eta + \lambda)$$

Routh-Hurwirtz criteria method solve to find fifth eigen value

Hence all four Eigen values are negative.

So $R_0 < 1$, Hence the equilibrium point P^* is locally asymptotically stable if $R_0 > 1$ unstable

4. NUMERICAL SIMULATIONS:

In order to verify the theoretical predictions of the model, the numerical simulations of the model (1) are carriedout using the following set of estimated parameter values:

STABILITY OF DISEASE-FREE STATE:

$\alpha = 0.04, d = 2.20, \phi = 0.8\ and\ \beta = 0.8$ the exposed/quarantine reproduction No are

$P_0(\beta,0,0,0,0) = P_0(0.8,0,0,0,0)\ and\ R_0 = 0.26315789 < 1.$ Fig.2 shows that S(t) goes to its steady state, while E(t),I(t),Q(t) and V(t) goes to zero with respect to time. Hence the diseasedies out.

CONCLUSION:

In this paper, we have analyzed SEIQVS model with general/natural incident rate and we observed that quarantine reproduction number plays an important role to control the disease, when

$R_0 < 1$, disease free equilibrium of the system is locally stable and if $R_0 > 1$,the endemic equilibrium is locally asymptotically stable.

By constructing a suitable linearization analysis.it is found that in each case the equilibrium points are locally asymptotically stable. Numerical simulations are alsopresented to illustrate our main results.

REFERENCES:

[1]. Diekmann O.and Kretzschmar M.(1991). Patterns in the effects of infectious diseases onpopulation growth, *J. Math. Biol.*, **29**,(PP. 539-570)

http://dx.doi.org/10.1007/bf00164051

[2] Pathak S., Maiti A. and Samanta G.P.(2010) Rich dynamics of an SIR epidemic model, *NonlinearAnalysis: Modelling and Control*, **15** (PP. 71-81)

[3] Zhang X. and Jia J.(2013) Stability of a SIR epidemic model with Information Variable and Limited Medical Resources, *International Journal of Research and Reviews in Applied Sciences*, **16** (PP. 91-103)

[4] Mishra B.K. and Pandey S.K. (2010) "Fuzzy epidemic model for the transmission of worms in computer network, "*Nonlinear Analysis: Real World Applications*,vol.11, no. 5 (PP. 4335-4341)

[5] Ren J., Yang X., Yang L. and F. Yang (2012) "A delayed computer virus propagation model and its dynamics,"*Chaos Solitons Fractals*, vol. 45, no. 1(PP.74-79).

[6] Mishra B.K. and Pandey S.K. (2011) "Dynamic model of worms with vertical transmission in computer network," *AppliedMathematics and Computation*, vol. 217, no. 21, (PP. 8438-8445)

[7] Toutonji O.A., Yoo S.M. and Park M. (2012) "Stability analysis of VEISV propagation modeling for network worm attack," *Applied Mathematical Modelling*, vol. 36, no. 6 (PP. 2751-2761)

[8] Mishra B.K. and Jha N. (2010) "SEIQRS model for thetransmission of malicious objects in computer network,"*Applied Mathematical Modelling*, vol. 34, no. 3 (PP. 710-715)

[9] Yang L., Yang X., Zhu Q. and L. Wen, (2013) "A computer virus model with graded cure rates," *Nonlinear Analysis: RealWorld Applications*, vol. 14, no. 1(PP. 414-422)

[10] Yang M., Zhang Z., Li Q. and Zhang G. (2012) "An SLBRS model with vertical transmission of Computer virus over the Internet," *Discrete Dynamics in Natureand Society*, vol. 2012, Article ID 925648(PP. 1-15)

[11] Wang F., Yang F., Zhang Y. and Ma J. (2014)"Stability Analysis of a SEIQRS Model with Graded Infection Rates for Internet Worms",JOURNAL OF COMPUTERS, VOL. 9, NO.10

[12] Wang F., Zhang Y., Wang C., Ma J. and Moon S.J. (2010) "Stability analysis of a SEIQV epidemic model for rapid spreading worms," *Computers and Security*, vol. 29, no. 4 (PP. 410-418)