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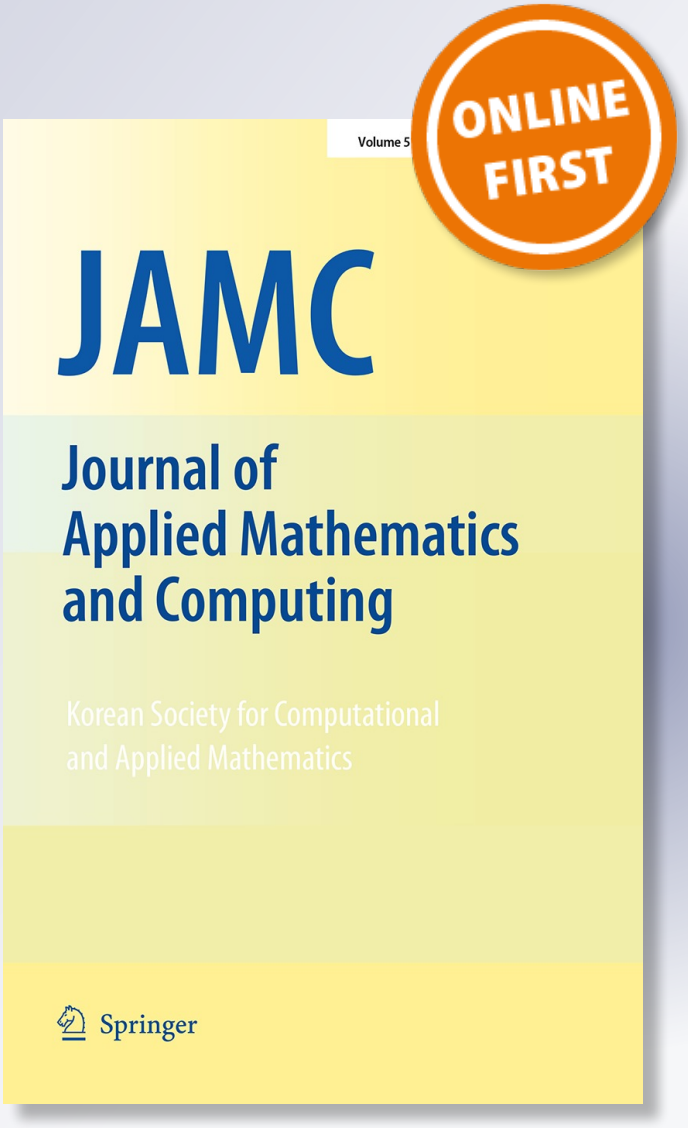

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ORIGINAL RESEARCH

Modeling the virus dynamics in computer network with SVEIR model and nonlinear incident rate

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Abstract In this paper, an e-epidemic Susceptible–Vaccinated–Exposed–Infectious–Recovered (*SVEIR*) model is formulated for the treatment of infective nodes considering the development of acquired immunity in recovered nodes. We have employed Holling type II functional response as the treatment function. Stability analysis for virus-free as well as interior/endemic equilibria is performed. It is observed that the existence of unique interior equilibrium depends on the basic reproduction number R_0 as well as on the treatment rate. Numerical simulations are performed to support analytical findings. We have analyzed the behavior of the susceptible, exposed and infected nodes in the computer network with real parameter values with time series and phase plane analysis.

Keywords Computer network model · Non-linear incidence rate · Viruses · Stability · Holling type II functional response

Mathematics Subject Classification 34D23 · 65C20 · 92D30

1 Introduction

Since the revolution of personal computers in 1980s, the computer networks are growing with rapid pace. The development of computer networks has made the spread and storage of information easy. Nowadays computer networks are the main mode of data

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communication. The wide range of applications of internet has made it a part of our day-to-day life. With the evolution of computer networks, the vulnerability of computers connected to networks to the virus attack has also increased. A computer virus is a malicious program that can propagate by attaching itself to some computer program or data file. A virus attached to a file can become active only when the user open or run the host file. It spreads from one computer to another when the infected host file is transferred from one computer to other through email or other media. Virus has ability to self-replicate and can insert copies of itself into other computer programs or file, when executed. Due to this property, virus spreads very rapidly through the network and can cause severe damage. A computer virus may corrupt system programs and files, damage the boot sector or even erase all the data stored in the hard disk. To protect the computer from any possible threat of virus attack, antivirus software is installed in it. Anti-virus software is a program which is designed to detect and remove viruses from the computer system. It usually detects virus by using a list of virus signature definitions which is updated in a regular manner. The system can lose the protection once the antivirus protection definitions are not updated for a long time.

The spread of computer virus in a computer network can be seen analogous to the spread of an infectious disease in a population [20]. The susceptible and infective individuals in the spread of epidemic diseases correspond to non-infected and infected computer nodes in the dynamics of virus infected computer network. Like a disease virus, which usually require some time, called incubation period, to show its symptoms or affect other individuals, computer virus also remains undetected for a long time and can't spread until the infected file is run by the user. The antivirus programs can be seen analogous to the treatment and vaccination programs. Just as in the case of disease, immunization of all the computers in a large network is not economically viable. It is always desirable to find out the threshold values which determine the possible strategies for eradication of virus from the network. As the spread of virus in computer network is akin to the spread of disease in population, epidemiological modeling approach can be used to gain a profound understanding of the effect of immunization and treatment of nodes through antivirus programs on the spread of virus in the network.

In the recent years, a plenty of investigations have been conducted to study the dynamics of spread of virus in a computer network by using the epidemiological approach ([11–13,19,21,22,26,31,32,36], and references therein). In these studies, e-epidemic models analogous to *SIS*, *SIR*, *SIER*, *SEIQRS* (S-susceptible class, E-exposed class, I-infective class, Q-quarantined class, R-removed class) epidemic model are proposed to study the virus dynamics in the computer network. In particular, Jia and Li [8] investigated the global analysis of an *SVEIR* epidemic model with nonlinear incidence rate and the vaccinated individuals have partial immunity. Wang et al. [30] proposed a novel e-epidemic *SVEIR* model with partial immunization to defend against worm. Peng and Mou [22] has proposed a *SEIR* e-epidemic model to study the propagation of computer virus in a network by considering the exposed class of nodes. Mishra and Saini [19] have applied *SEIRS* model with time delay to study transmission of malicious objects in a computer network. Keshri and Mishra [14] studied a *SEIR* e-epidemic model with two delays and describe the transmission dynamics of malicious signals in wireless sensor network. Misra et al. [20]

have studied the interplay between malware and anti-malware in computer network. Some e-epidemic models have been proposed to study the effect of antivirus softwares [25,26] and vaccination [4,5] on the spread of computer viruses in a network.

The choices of the treatment function and the transmission process are very important factors for the modelling of epidemic and e-epidemic systems. In order to model the transmission process, several authors employed different incidence functions [9]. The dynamics of the model system heavily depends on the choice of these functions. Gan et al. [4,5] have considered the different incidence rates $\beta SI/f(I)$ and βSI respectively and the linear treatment rate αI in both of his work. Zhang and Suo [33], Zhou and Fan [34] and Dubey et al. [3] have modified the treatment function as $h(I) = \frac{\beta I}{I+a}, I \geq 0, \beta, a > 0$. We have considered the same treatment function in our work. Due to the effect of treatment, every infected node in the internet becomes recovered with probability $\beta > 0$. In the beginning, the treatment rate is slow due to the less effectiveness of anti-virus. Then, the treatment rate will increase slowly and attains its peak and finally settles down at its saturation value with the improved and effective antivirus and skillful techniques etc. The e-epidemicity of virus is closely related to the stability of the equilibria of the model. Many researcher have taken the different incidence rates like bilinear incidence rate (βSI) [5,6,15,18], standard incidence rate ($\beta SI/N$) [17], nonlinear incidence rate $\beta SI/f(I)$ [5]. Dubey et al. [2], and Kaddar [9,10] defined modified saturated incidence rate as $f(S, I) = \frac{\alpha SI}{(1+\beta S+\gamma I)}$ (Beddington-DeAngelis type), where $\alpha, \beta, \gamma > 0$. It is interesting to note that for different values of β, γ, we can derive different types of nonlinear incidence rates. Piqueira et al. [23] investigated the use of classical epidemiological transmission for studying computer virus propagation. In our work, we have taken a specific type of incidence rate as $f(S, I) = \frac{\alpha SI}{(S+I+c)}$ [1,28].

In the present paper, we propose a novel *SEIRV* e-epidemic model of the virus propagation in a network by considering Holling type II functional response as treatment function. We have considered the nonlinear incidence rate of infection in the network. The paper is organized as follows: In Sect. 2, we formulate the mathematical model governing the dynamics of the problem. In Sect. 3, we present the existence of equilibria and derive the expression for the basic reproduction number. The local stability analysis, persistence of virus and global stability analysis of the equilibria are performed in Sect. 4. In Sect. 5, numerical simulations are carried out to verify the analytical findings taking relevant values of the parameters and using software environments MATLAB and Mathematica. Finally in Sect. 6, the paper is concluded.

2 Formulation of the mathematical model for virus propagation

Let $N(t)$ represents the total number of nodes present in a computer network at time t. The nodes are divided into five subclasses; susceptible nodes (S), vaccinated nodes (V), exposed nodes (E), infectious nodes (I) and recovered nodes (R). Thus,

$$N(t) = S(t) + V(t) + E(t) + I(t) + R(t).$$

While formulation of the model, we have made following assumptions:

(i) The new nodes are added to the network with a constant rate and all the new nodes are susceptible.

(ii) The virus propagates into network when an infected file is transferred from an infectious node to the susceptible node. We have considered the nonlinear incidence rate of infection of the form $f(S, I) = \frac{\alpha SI}{S+I+c}$ [1,28], where α denotes the rate of transfer of virus from infectious node to the susceptible node and c is a half saturation constant for saturated nodes S. This signifies the fact that the number of contacts a particular node carrying the virus can have with other nodes reaches some finite maximum value due to the spatial distribution of nodes and or limitation of time [1,28].

(iii) Susceptible nodes move to exposed class upon contracting infection and after some time (i.e., latency period), they become infectious and move to infectious class.

(iv) The infectious nodes are assumed to leave the infectious class as a result of crashing rate of the node due to hardware or software problem and attack of viruses as well as recovery of infected nodes. The infectious nodes are also assumed [3] to leave the infectious class due to treatment at a rate $h(I)$, where

$$h(I) = \frac{\beta I}{I + a}, I \geq 0, \beta, a > 0.$$

It is easy to see that the treatment rate $h(I)$ is a continuously differentiable increasing function of I and $h(0) = 0, h'(I) > 0$ and $\lim_{I\to\infty} h(I) = \beta$, where β is the maximal treatment capacity of the network.

(v) After recovery the nodes become immunized and hence they are no longer susceptible to it. This happens because they acquired immune response which leads to the development of immunological memory and therefore nodes cannot get infected from the same virus again and does not enter into the susceptible class.

(vi) It is assumed that a fraction of susceptible nodes get vaccinated and join the vaccinated class. A fraction of vaccinated nodes may lose their immunity against virus attack and rejoin the susceptible class.

Keeping the above assumptions in mind, the mathematical model can be governed by the following system of ordinary differential equations:

$$\left.\begin{aligned}
\frac{dS}{dt} &= A - \delta_0 S - \frac{\alpha SI}{S + I + c} + \eta V - \mu S,\\
\frac{dE}{dt} &= \frac{\alpha SI}{S + I + c} - (\delta_0 + \delta_1)E,\\
\frac{dI}{dt} &= \delta_1 E - (\delta_0 + \delta_2 + \delta_3)I - \frac{\beta I}{I + a},\\
\frac{dR}{dt} &= \delta_2 I - \delta_0 R + \frac{\beta I}{I + a},\\
\frac{dV}{dt} &= \mu S - (\delta_0 + \eta)V,
\end{aligned}\right\} \tag{1}$$

with initial conditions: $S(0) = S_0 > 0, E(0) = E_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0, V(0) = V_0 \geq 0$. All the parameters in model system (1) are positive and are defined in the Table 1. The schematic diagram for the flow of viruses in computer network is presented in Fig. 1.

3 Existence analysis of equilibria and basic reproduction number

In this section, we discuss the existence of the virus-free equilibrium and the endemic equilibrium of the model system (1). Since removed class R does not have any effect on the dynamics of S, V, E and I class, we shall study the following reduced system:

$$\left.\begin{aligned} \frac{dS}{dt} &= A - \delta_0 S - \frac{\alpha SI}{S+I+c} + \eta V - \mu S, \\ \frac{dE}{dt} &= \frac{\alpha SI}{S+I+c} - (\delta_0 + \delta_1)E, \\ \frac{dI}{dt} &= \delta_1 E - (\delta_0 + \delta_2 + \delta_3)I - \frac{\beta I}{I+a}, \\ \frac{dV}{dt} &= \mu S - (\delta_0 + \eta)V, \end{aligned}\right\} \tag{2}$$

Table 1 Definition of variables and parameters

Parameter	Description	Unit
S	Susceptible node	In number
E	Exposed node	In number
I	Infectious node	In number
R	Recovered node	In number
V	Vaccinated node	In number
A	Recruitment rate of new nodes	Day^{-1}
α	Contact rate or the rate of transfer of virus from an infectious node to the susceptible node	Day^{-1}
η	Rate at which the vaccinated nodes lose their immunity and join the susceptible class	Day^{-1}
β	Maximal treatment capacity of a network	Day^{-1}
δ_0	Natural crashing rate of nodes all classes	Day^{-1}
δ_1	Rate at which infected node become infectious	Day^{-1}
δ_2	Recovery rate of infected nodes	Day^{-1}
δ_3	Virus induced crashing rate	Day^{-1}
a	Half saturation constant for infected node I	In number
c	Half saturation constant for susceptible node S	In number
μ	Vaccination rate coefficient	Day^{-1}

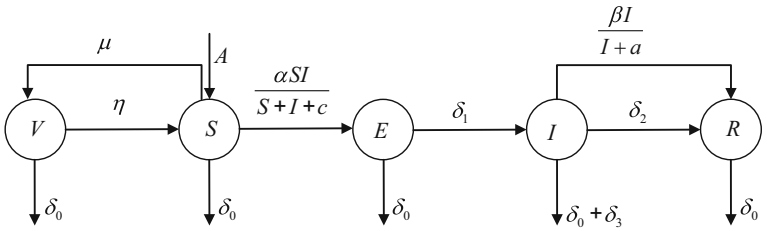

Fig. 1 Schematic diagram for the flow of viruses in computer network

with initial conditions: $S(0) = S_0 > 0, E(0) = E_0 \geq 0, I(0) = I_0 \geq 0, V(0) = V_0 \geq 0$, where all parameters are positive.

The dynamical properties of R are completely determined once we know those of S, E, I and V i.e., all the property of model system (1) can be drawn from those of system (2). Hence, we shall focus our study on the model system (2).

We observe that total number of nodes N satisfies the equation

$$\frac{dN}{dt} = A - \delta_0 N - \delta_2 I - \delta_3 I - \frac{\beta I}{I+a} \leq A - \delta_0 N, \text{ then } \lim_{t\to\infty} \sup N(t) \to \frac{A}{\delta_0}.$$

Therefore, the feasible region $\Omega = \left\{(S, E, I, V) \in \Re_+^4 : S + E + I + V \leq \frac{A}{\delta_0}\right\}$, is a positively invariant region for the model system (2) and is obviously well-posed in Ω. Hence, we consider only solutions with initial conditions inside the region Ω, in which the usual existence, uniqueness of solutions and continuation results hold. The model system (2) always has the virus-free equilibrium (*VFE*) $P_0\left(S^0, 0, 0, V^0\right)$, where

$$S^0 = \left(\frac{\delta_0 + \eta}{\delta_0 + \eta + \mu}\right) N^0, V^0 = \left(\frac{\mu}{\delta_0 + \eta + \mu}\right) N^0, N^0 = \frac{A}{\delta_0}, \tag{3}$$

represents the level of susceptible, vaccinated and total number of nodes respectively in the absence of infection.

To consider the existence and uniqueness of endemic equilibrium $P^*(S^*, E^*, I^*, V^*)$, we firstly study the basic reproduction number R_0 of model system (2) according to the next generation matrix.

The basic reproduction number, R_0 is defined as the expected numbers of secondary infections produced by one infected computer nodes in completely susceptible nodes. We calculate the basic reproduction number. Let $x = (E, I)$. Then from the model system (2), it follows:

$$\frac{dx}{dt} = f - \upsilon,$$

where

$$f = \begin{bmatrix} \frac{\alpha SI}{S+I+c} \\ 0 \end{bmatrix} \text{ and } \upsilon = \begin{bmatrix} (\delta_0 + \delta_1)E \\ -\delta_1 E + \left(\delta_0 + \delta_2 + \delta_3 + \frac{\beta}{I+a}\right) I \end{bmatrix}.$$

We obtain

$$F = \text{Jacobian of } f \text{ at } VFE = \begin{bmatrix} 0 & \frac{\alpha S^0}{S^0+c} \\ 0 & 0 \end{bmatrix},$$

$$\text{and } M = \text{Jacobian of } \upsilon \text{ at } VFE = \begin{bmatrix} \delta_0+\delta_1 & 0 \\ -\delta_1 & \delta_0+\delta_2+\delta_3+\frac{\beta}{a} \end{bmatrix}.$$

Hence, next generation matrix for the system is

$$K = FM^{-1} = \begin{bmatrix} \frac{S^0\alpha\delta_1}{(S^0+c)(\delta_0+\delta_1)\left(\frac{\beta}{a}+\delta_0+\delta_2+\delta_3\right)} & \frac{S^0\alpha}{(S^0+c)\left(\frac{\beta}{a}+\delta_0+\delta_2+\delta_3\right)} \\ 0 & 0 \end{bmatrix}.$$

Again, the spectral radius R_0 of the matrix $K = FM^{-1}$, is the basic reproduction number of the model, i.e., $R_0 = \rho(FM^{-1})$, hence

$$\begin{aligned} R_0 &= \frac{aS^0\alpha\delta_1}{(S^0+c)(\delta_0+\delta_1)(\beta+a(\delta_0+\delta_2+\delta_3))} \\ &= \frac{aA\alpha(\eta+\delta_0)\delta_1}{(A\eta+\delta_0(A+c(\eta+\mu)+c\delta_0))(\delta_0+\delta_1)(\beta+a(\delta_0+\delta_2+\delta_3))}. \end{aligned} \tag{4}$$

Further, the model system (2) also has an interior equilibrium called endemic equilibrium given by $P^* = (S^*, E^*, I^*, V^*)$. The solution is

$$\begin{aligned} S^* &= \frac{(\eta+\delta_0)\left(A-\frac{(\delta_0+\delta_1)}{\delta_1}\left(\frac{\beta}{a+I^*}+\delta_0+\delta_2+\delta_3\right)I^*\right)}{\delta_0(\mu+\eta+\delta_0)}, \\ E^* &= \left(\delta_0+\delta_2+\delta_3+\frac{\beta}{I^*+a}\right)\frac{I^*}{\delta_1}, \\ V^* &= \frac{\mu\left(A-\frac{(\delta_0+\delta_1)}{\delta_1}\left(\frac{\beta}{a+I^*}+\delta_0+\delta_2+\delta_3\right)I^*\right)}{\delta_0(\mu+\eta+\delta_0)}. \end{aligned}$$

The value I^* is the positive real root of

$$A_0I^3 + 3A_1I^2 + 3A_2I + A_3 = 0, \tag{5}$$

with

$$\begin{aligned} A_0 &= pq\left(-pqS^0+\left(A+S^0\alpha\right)\delta_1\right) \\ &= pq\left(pq(R_0-1)S^0+A\delta_1+R_0\left(\frac{\beta}{a}(c+S^0)+cp\right)q\right), \\ 3A_1 &= -2pq^2S^0(ap+\beta)+q(Ap(c+S^0)+2ap(A+S^0\alpha) \\ &\quad +(A+S^0\alpha)\beta)\delta_1-AS^0\alpha\delta_1^2, \end{aligned}$$

$$3A_2 = -q^2S^0(ap+\beta)^2 + \delta_1(apq(2A(c+S^0) + a(A+S^0\alpha)) + q(A(a+c+S^0) + aS^0\alpha)\beta - 2aAS^0\alpha\delta_1),$$

$$A_3 = aA\delta_1(q(c+S^0)(ap+\beta) - aS^0\alpha\delta_1) = -aA\delta_1 q(c+S^0)(ap+\beta)(R_0-1).$$

Also

$$p = \delta_0 + \delta_2 + \delta_3, q = \delta_0 + \delta_1.$$

We observe that A_0 is positive and $A_3 < 0$ if $R_0 > 1$. Therefore, by Descarte's rule of signs, Eq. (5) has at least one positive real root. Further, the condition $G^2 + 4H^3 > 0$, where $G = A_0^2A_3 - 3A_0A_1A_2 + 2A_1^3$, $H = A_0A_2 - A_1^2$, implies that this equation has only one real root. Both the conditions together imply that Eq. (5) has a unique positive root and by using Cardan's method, we obtain the root as $\frac{1}{A_0}\left(p - \frac{H}{p} - A_1\right)$, where p denotes the real value of $\left[\frac{1}{2}\left(-G + \sqrt{G^2+4H^3}\right)^{\frac{1}{3}}\right]$.

Hence, the nontrivial equilibrium $P^* = (S^*, E^*, I^*, V^*)$ exists in the Int. $\Re_+^4$, if and only if there is a positive solution to the Eq. (5). It is also clear that the endemic equilibrium point exists if $R_0 > 1$.

4 Stability analysis of the model system

In this section, we will discuss the stability of the virus-free equilibrium, persistence of virus and the stability of the endemic equilibrium of the model system (2).

4.1 Linear stability analysis

In this section, we investigate the linear stability of both the equilibria.

Theorem 4.1 *The virus-free equilibrium (VFE) P_0 is*

(i) locally asymptotically stable, if $R_0 < 1$,
(ii) unstable, if $R_0 > 1$.

Proof The Jacobian matrix at *VFE* is given by

$$J_0 = \begin{bmatrix} -(\delta_0+\mu) & 0 & -\frac{\alpha S^0}{S^0+c} & \eta \\ 0 & -(\delta_0+\delta_1) & \frac{\alpha S^0}{S^0+c} & 0 \\ 0 & \delta_1 & -\left(\delta_0+\delta_2+\delta_3+\frac{\beta}{a}\right) & 0 \\ \mu & 0 & 0 & -(\delta_0+\eta) \end{bmatrix}.$$

The characteristic equation of J_0 is given by

$$(\lambda+\eta+\mu+\delta_0)(\lambda+\delta_0)\left[\lambda^2+\lambda\left(\frac{\beta}{a}+2\delta_0+\delta_1+\delta_2+\delta_3\right)\right.$$
$$\left.+(1-R_0)(\delta_0+\delta_1)\left(\frac{\beta}{a}+\delta_0+\delta_2+\delta_3\right)\right]=0. \tag{6}$$

The two roots of the above equation are $-(\eta+\mu+\delta_0)$ and $-\delta_0$, which are clearly negative; however the other two roots are given by a quadratic equation, which are either negative or with negative real part if $R_0<1$. Thus, for $R_0<1$, all the four eigenvalues of the Jacobian matrix J_0 are either negative or with negative real part. For $R_0>1$, one root of Eq. (6) will be positive and so in this case J_0 has three negative and one positive eigenvalues. □

Hence, *VFE* is locally asymptotically stable, if $R_0<1$ and unstable, if $R_0>1$.

Theorem 4.2 *If $R_0>1$, then the endemic equilibrium point P^* exists and is locally asymptotically stable if the following conditions hold*

(i) $\eta<\frac{(a_{33}+a_{44})(a_{33}+q)(a_{44}+q)+a_{11}^2(a_{33}+a_{44}+q)+a_{11}(a_{33}+a_{44}+q)^2}{(a_{11}+a_{44})\mu}$ *and* $0<a_{13}<\frac{(a_{33}+a_{44})(a_{33}+q)(a_{44}+q)+a_{11}^2(a_{33}+a_{44}+q)-a_{44}\eta\mu+a_{11}((a_{33}+a_{44}+q)^2-\eta\mu)}{(a_{21}+a_{33}+q)\delta_1}$,

(ii) $0<h\le\frac{z}{a_{33}a_{44}+(a_{33}+a_{44})q}$ *and* $a_{11}>\frac{z(a_{44}hq+a_{33}h(a_{44}+q)-z-h\eta\mu-a_{13}h\delta_1)}{h(a_{33}a_{44}hq-(a_{33}+a_{44}+q)z)}$,

(iii) $z>0$ *if* $a_{21}\ge a_{44}+\frac{-a_{33}a_{44}q+(a_{33}+q)\eta\mu}{a_{13}\delta_1}$.

where all the parameters $a_{11}, a_{13}, a_{21}, a_{33}, a_{44}, p$ and q are defined in Eq. (7). *The values of h and z are given in* Eq. (8) *and* (9).

Proof The Jacobian matrix at endemic equilibrium point is given by

$$J_{P^*}=\begin{bmatrix}-(\delta_0+\mu)-\frac{\alpha I^*(c+I^*)}{(S^*+I^*+c)^2} & 0 & -\frac{\alpha S^*(c+S^*)}{(S^*+I^*+c)^2} & \eta\\ \frac{\alpha I^*(c+I^*)}{(S^*+I^*+c)^2} & -(\delta_0+\delta_1) & \frac{\alpha S^*(c+S^*)}{(S^*+I^*+c)^2} & 0\\ 0 & \delta_1 & -(\delta_0+\delta_2+\delta_3)-\frac{a\beta}{(a+I^*)^2} & 0\\ \mu & 0 & 0 & -(\delta_0+\eta)\end{bmatrix}$$
$$=\begin{bmatrix}-a_{11} & 0 & -a_{13} & \eta\\ -a_{21} & -q & a_{13} & 0\\ 0 & \delta_1 & -a_{33} & 0\\ \mu & 0 & 0 & -a_{44}\end{bmatrix}.$$

here

$$\left.\begin{aligned}&a_{11}=(\delta_0+\mu)+\tfrac{(I^*+c)\alpha I^*}{(S^*+I^*+c)^2}, a_{13}=\tfrac{(S^*+c)\alpha S^*}{(S^*+I^*+c)^2}, a_{21}=\tfrac{(I^*+c)\alpha I^*}{(S^*+I^*+c)^2},\\ &a_{33}=\delta_0+\delta_2+\delta_3+\tfrac{a\beta}{(I^*+a)^2}, a_{44}=\delta_0+\eta, p=\delta_0+\delta_2+\delta_3, q=\delta_0+\delta_1.\end{aligned}\right\} \tag{7}$$

The characteristic equation of J_{P^*} is $P(\lambda)=\lambda^4+Q_1\lambda^3+Q_2\lambda^2+Q_3\lambda+Q_4=0$, where

$$Q_1=(a_{11}+a_{33}+a_{44}+q)>0,$$

$$
\begin{aligned}
Q_2 &= a_{33}a_{44} + (a_{33} + a_{44})q + a_{11}(a_{33} + a_{44} + q) - \eta\mu - a_{13}\delta_1,\\
Q_3 &= a_{11}a_{33}a_{44} + a_{33}a_{44}q + a_{11}(a_{33} + a_{44})q - (a_{33} + q)\eta\mu\\
&\quad - a_{13}(a_{11} - a_{21} + a_{44})\delta_1,\\
Q_4 &= a_{11}a_{33}a_{44}q > 0.
\end{aligned}
$$

An application of Routh-Hurwitz criteria gives $\mathrm{Re}(\lambda) < 0$ if and only if

$$
\begin{aligned}
H_1 = Q_1 &= \frac{I^*(c + I^*)\alpha}{(S^* + I^* + c)^2} + \frac{a\beta}{(a + I^*)^2} + \eta + \mu + 4\delta_0 + \delta_1 + \delta_2 + \delta_3 > 0,\\
H_2 &= Q_1Q_2 - Q_3\\
&= (a_{33} + a_{44})(a_{33} + q)(a_{44} + q) + a_{11}^2(a_{33} + a_{44} + q)\\
&\quad - a_{44}\eta\mu + a_{11}((a_{33} + a_{44} + q)^2 - \eta\mu) - (a_{13}a_{21} + a_{23}(a_{33} + q))\delta_1 > 0,\\
&\text{if } \eta < \frac{(a_{33} + a_{44})(a_{33} + q)(a_{44} + q) + a_{11}^2(a_{33} + a_{44} + q) + a_{11}(a_{33} + a_{44} + q)^2}{(a_{11} + a_{44})\mu}\\
&\text{and } a_{13} < \frac{(a_{33} + a_{44})(a_{33} + q)(a_{44} + q) + a_{11}^2(a_{33} + a_{44} + q) - a_{44}\eta\mu + a_{11}((a_{33} + a_{44} + q)^2 - \eta\mu)}{(a_{21} + a_{33} + q)\delta_1}.
\end{aligned}
$$

$$
\begin{aligned}
H_3 &= \begin{vmatrix} Q_1 & Q_3 & 0\\ 1 & Q_2 & Q_4\\ 0 & Q_1 & Q_3 \end{vmatrix} = -Q_3^2 + Q_1Q_2Q_3 - Q_1^2Q_4 = Q_3H_2 - Q_1^2Q_4\\
&= -a_{11}a_{33}a_{44}q(a_{11} + a_{33} + a_{44} + q)^2 + (a_{11} + a_{33} + a_{44} + q)(a_{33}a_{44}\\
&\quad + (a_{33} + a_{44})q + a_{11}(a_{33} + a_{44} + q) - \eta\mu - a_{13}\delta_1)(a_{11}a_{33}a_{44}\\
&\quad + a_{33}a_{44}q + a_{11}(a_{33} + a_{44})q - (a_{33} + q)\eta\mu - a_{13}(a_{11} - a_{21} + a_{44})\delta_1)\\
&\quad - (a_{11}a_{33}a_{44} + a_{33}a_{44}q + a_{11}(a_{33} + a_{44})q - (a_{33} + q)\eta\mu\\
&\quad - a_{13}(a_{11} - a_{21} + a_{44})\delta_1)^2 > 0,\\
&\text{if } 0 < h \le \frac{z}{a_{33}a_{44} + (a_{33} + a_{44})q} \text{ and}\\
&a_{11} > \frac{z(a_{44}hq + a_{33}h(a_{44} + q) - z - h\eta\mu - a_{13}h\delta_1)}{h(a_{33}a_{44}hq - (a_{33} + a_{44} + q)z)}
\end{aligned}
$$

where

$$
h = (a_{11} + a_{33} + a_{44} + q), \tag{8}
$$

and

$$
\begin{aligned}
z &= (a_{11}a_{33}a_{44} + a_{33}a_{44}q + a_{11}(a_{33} + a_{44})q - (a_{33} + q)\eta\mu\\
&\quad - a_{13}(a_{11} - a_{21} + a_{44})\delta_1). \qquad (9)\\
&z > 0 \text{ if } a_{21} \ge a_{44} + \frac{-a_{33}a_{44}q + (a_{33} + q)\eta\mu}{a_{13}\delta_1}.\\
H_4 &= Q_4H_3 > 0 \text{ (Since } Q_4 > 0,\ H_3 > 0).
\end{aligned}
$$

Thus, the endemic equilibrium point P^* is locally asymptotically stable. □

4.2 Global stability analysis of virus-free equilibrium

In this section, we consider the global stability of virus-free equilibrium point.

Theorem 4.3 *If* $R_0 < 1 - \frac{\beta A}{(a\delta_0+A)(a(\delta_0+\delta_2+\delta_3)+\beta)}$, *then the virus-free equilibrium* P_0 *of model* (2) *is globally stable.*

Proof From the above discussion of Theorem 4.1, we have obtained that the unique virus-free-equilibrium P_0 of the reduced system (2) is locally asymptotically stable as $R_0 < 1$.

From the first equation of model (2), we obtain

$$\begin{aligned}
\frac{dS}{dt} &= A - \delta_0 S - \frac{\alpha SI}{S+I+c} + \eta V - \mu S \\
&\le A - \delta_0 S - \mu S + \eta(N - S - E - I) \\
&\le A + \eta N - \delta_0 S - \mu S - \eta S \\
&\le A + \eta \frac{A}{\delta_0} - (\delta_0 + \mu + \eta)S, \\
\frac{dS}{dt} &\le \frac{A}{\delta_0}(\delta_0 + \eta) - (\delta_0 + \mu + \eta)S.
\end{aligned}$$

Let $\frac{dX}{dt} = \frac{A}{\delta_0}(\delta_0 + \eta) - (\delta_0 + \mu + \eta)X$.

A solution of the equation $\frac{dX}{dt} = \frac{A}{\delta_0}(\delta_0 + \eta) - (\delta_0 + \mu + \eta)X$ is an upper solution of $S(t)$. That is, $X(t) \ge S(t)$for all $t \ge 0$.

Noting that, $X(t) \to \frac{(\delta_0+\eta)}{(\delta_0+\mu+\eta)}\frac{A}{\delta_0}$ as $t \to \infty$, it follows that for a given $\in_1 > 0$, there is a t_0, such that $S(t) \le X(t) \le \frac{(\delta_0+\eta)}{(\delta_0+\mu+\eta)}\frac{A}{\delta_0} + \in_1$, for $t \ge t_0$.

Let $\in_1 \to 0$, we have

$$S(t) \le \frac{(\delta_0 + \eta)}{(\delta_0 + \mu + \eta)}\frac{A}{\delta_0}. \tag{10}$$

Consider the Lyapunov function $L = \delta_1 E + (\delta_0 + \delta_1)I$, thus

$$\begin{aligned}
L' &= \delta_1 E' + (\delta_0 + \delta_1)I' \\
&= \delta_1 \left(\frac{\alpha SI}{S+I+c} - (\delta_0 + \delta_1)E\right) + (\delta_0 + \delta_1)\left(\delta_1 E - (\delta_0 + \delta_2 + \delta_3)I - \frac{\beta I}{I+a}\right), \\
&= \delta_1 \frac{\alpha SI}{S+I+c} - (\delta_0 + \delta_1)(\delta_0 + \delta_2 + \delta_3)I - \frac{\beta I}{I+a}(\delta_0 + \delta_1), \\
&= \left(\frac{\alpha\delta_1 S}{S+I+c} - (\delta_0 + \delta_1)(\delta_0 + \delta_2 + \delta_3) + \left(\frac{\beta}{a} - \frac{\beta}{a} - \frac{\beta}{I+a}\right)(\delta_0 + \delta_1)\right) I, \\
&= \left(\frac{\alpha\delta_1 S}{S+I+c} - (\delta_0 + \delta_1)\left(\delta_0 + \delta_2 + \delta_3 + \frac{\beta}{a}\right) + \left(\frac{\beta I}{a(I+a)}\right)(\delta_0 + \delta_1)\right) I, \\
&\le \left(\frac{\alpha\delta_1 S}{S+c} - (\delta_0 + \delta_1)\left(\delta_0 + \delta_2 + \delta_3 + \frac{\beta}{a}\right) + \frac{\beta I}{a(I+a)}(\delta_0 + \delta_1)\right) I,
\end{aligned}$$

$$
\begin{aligned}
&\leq (\delta_0+\delta_1)\left(\delta_0+\delta_2+\delta_3+\frac{\beta}{a}\right)\\
&\cdot\left(\frac{\alpha\delta_1\frac{(\delta_0+\eta)}{(\delta_0+\mu+\eta)}\frac{A}{\delta_0}}{\left(\frac{(\delta_0+\eta)}{(\delta_0+\mu+\eta)}\frac{A}{\delta_0}+c\right)(\delta_0+\delta_1)(\delta_0+\delta_2+\delta_3+\frac{\beta}{a})}\right.\\
&\left.-1+\frac{\beta I}{a(I+a)(\delta_0+\delta_2+\delta_3+\frac{\beta}{a})}\right)I, \{\text{from 10}\}\\
&=(\delta_0+\delta_1)\left(\delta_0+\delta_2+\delta_3+\frac{\beta}{a}\right)\left(R_0-1+\frac{\beta I}{(I+a)(a(\delta_0+\delta_2+\delta_3)+\beta)}\right)I,\\
&\leq (\delta_0+\delta_1)\left(\delta_0+\delta_2+\delta_3+\frac{\beta}{a}\right)\left(R_0-1+\frac{\beta\frac{A}{\delta_0}}{(\frac{A}{\delta_0}+a)(a(\delta_0+\delta_2+\delta_3)+\beta)}\right)I,\\
&\leq (\delta_0+\delta_1)\left(\delta_0+\delta_2+\delta_3+\frac{\beta}{a}\right)\left(R_0-1+\frac{\beta A}{(A+a\delta_0)(a(\delta_0+\delta_2+\delta_3)+\beta)}\right)I.
\end{aligned}
$$

Furthermore, $L'=0$ iff $I=0$. Therefore, the largest compact invariant set in $\{(S,E,I,V)\in\Omega : L'=0\}$ is the singleton P_0. By the LaSalle's invariance principle theorem [7], the virus-free equilibrium P_0 is globally stable if $R_0<1-\frac{\beta A}{(A+a\delta_0)(a(\delta_0+\delta_2+\delta_3)+\beta)}$. □

4.3 Persistence of virus

In this section, we shall apply the techniques given in [24,27] to study the persistence of virus. Rebelo et al. [24] have investigated the persistence in a large class of system in a seasonally forces epidemiological models. Here, we present the persistence results for e-epidemiological system.

Theorem 4.4 *If $R_0>1$, model system (2) is uniformly permanent.*

Proof Let $X=\{(S,E,I,V):S,E,I,V\geq 0\}$ be a metric space and $\Phi_t(S_0,E_0,I_0,V_0)$ be the solution semi-flow of model system (2) with $S(0)=S_0$, $E(0)=E_0$, $I(0)=I_0$, $V(0)=V_0$. It is easy to prove that Φ is continuous. In the Sect. 3, we have shown that $\Omega=\left\{(S,E,I,V):S,E,I,V\leq\frac{A}{\delta_0}\right\}$ is a closed and positively invariant set of X, and the metric space is a compact one. Thus, there exists a compact set N, in which all solutions of model system (2) initiated from Ω ultimately enter and remain in it forever. Let $w(y)$ be the w-limit set of the solution of system (2) starting from Ω. We need to show the following set holds $N_\infty=\cup w(y)_{y\in Y}$, $Y=\{x_0\in\partial\Omega : x(t,x_0)\in\partial\Omega,\forall t>0\}$.

There always exists the unique virus-free equilibrium $P_0(S^0,0,0,V^0)$ on the boundary of Ω, from the previous proof we know that, P_0 is unstable as $R_0>1$, P_0 is unique largest invariant subset on the boundary of Ω, and P_0 is covering of Ω, which is isolated and acyclic. Thus, $N_\infty=\{P_0\}$. Since the closed positive octant is positively invariant for model system (2), it follows that

$$\lim_{t\to+\infty}\sup d(x(t,x_0),P_0)=\lim_{t\to+\infty}\inf d(x(t,x_0),P_0)=0.$$

To show model system (2) is uniformly permanent, according to Lemma 4 of [35], we only need to verify $W^+(P_0)\cap \mathrm{int}(\Omega)=\phi$, where $W^+(P_0)$ denotes the stable manifold of P_0. If $R_0>1$, P_0 is unstable. In particular, the Jacobian matrix of system (2) has one eigenvalue with positive real part, which denotes as λ_+, and three eigenvalues with negative real part, which are respectively, denote as λ_-, $-(\delta_0+\delta_1)$ and $-(\delta_0+\delta_2+\delta_3+\beta/a)$. ($\lambda_-$ may be equal to $-(\delta_0+\delta_1)$ or $-(\delta_0+\delta_2+\delta_3+\beta/a)$). We shall proceed by determining the location of $E(P_0)$ (the stable eigenspace of P_0). Then the eigenvector associated to λ_- is $(0,n_1,n_2,0)^T$ as $\lambda_-\neq-(\delta_0+\delta_1)$ and $\lambda_-\neq-(\delta_0+\delta_2+\delta_3+\beta/a)$, where n_1, n_2 satisfy the eigenvector equation

$$\begin{pmatrix} -(\delta_0+\delta_1) & \dfrac{\alpha\frac{(\delta_0+\eta)}{(\delta_0+\mu+\eta)}\frac{A}{\delta_0}}{\frac{(\delta_0+\eta)}{(\delta_0+\mu+\eta)}\frac{A}{\delta_0}+c} \\ \delta_1 & -\left(\delta_0+\delta_2+\delta_3+\frac{\beta}{a}\right)\end{pmatrix}\begin{pmatrix}n_1\\n_2\end{pmatrix}=\lambda_-\begin{pmatrix}n_1\\n_2\end{pmatrix}. \tag{11}$$

In the rest of the proof, if we show that in both cases

(1) $\lambda_-=-(\delta_0+\omega)$ or $\lambda_-=-\delta_0$,
(2) $\lambda_-\neq-(\delta_0+\omega)$ or $\lambda_-\neq-\delta_0$,

The vector $(n_1,n_2)^T\notin\Re^2_+$, then the proof of Theorem 4.4 is complete. □

In fact, by the definition of an irreducible matrix, the matrix in (11) is an irreducible Metzler matrix, denoted by M. Thus, $M+NI_{2\times2}$ is a nonnegative irreducible matrix, where, N is a sufficiently large positive constant, $I_{2\times2}$ is the identity matrix. Thus, the conditions of the Perron–Frobenius theorem in [29] are satisfied. By the Perron–Frobenius theorem, we know that M possesses the dominant eigenvalue λ_+. But the Perron–Frobenius theorem also implies that every eigenvector does not belong to the closed positive octant since it is not associated with the dominant eigenvalue. This means that $(n_1,n_2)^T\notin\Re^2_+$. Therefore, $E^+(P_0)\cap\mathrm{int}(\Omega)=\phi$. Hence, $W^+(P_0)\cap\mathrm{int}(\Omega)=\phi$ is verified. Thus, if $R_0>1$, model system (2) is uniformly permanent.

4.4 Global stability of the endemic equilibrium

In this section, we apply the geometrical approach [16] to investigate the global stability of the endemic equilibrium P^* in the feasible region Ω.

Lemma 1 [16] *Consider the differential equation*

$$x'=f(x), \tag{12}$$

and its corresponding periodic linear system

$$z'=\frac{\partial f^{[2]}}{\partial x}(p(t))z(t), \tag{13}$$

where $\frac{\partial f^{[2]}}{\partial x}$ *is the second additive compound matrix of* $\frac{\partial f}{\partial x}$ *and* $\Theta = \{p(t) : 0 \leq t \leq w\}$ *is the periodic orbit of* (12).

Now, we make the following assumptions

(i) There is a compact absorbing set $K \subset D$ and a unique equilibrium $\bar{x} \in D$.
(ii) Model system (12) satisfies the $Poincare - Bendixson$ property.
(iii) System (13) is asymptotically stable for each periodic solution $x = p(t)$ to (12) with $p(0) \in D$, and
(iv) $(-1)^n \det\left(\frac{\partial f}{\partial x}(\bar{x})\right) > 0$.

Then, the unique equilibrium $\bar{x}$ of the model system (12) is globally asymptotically stable in D.

Theorem 4.5 *If* $R_0 > 1$, *the unique positive equilibrium* P^* *of the model system (2) is globally asymptotically stable in* Ω.

Proof We only need to prove that four assumptions of Lemma 1 hold.

If $R_0 > 1$, model system (2) is uniformly permanent, and the unique positive equilibrium P^* of the model system (2) is locally asymptotically stable in Ω. Therefore, there is a compact absorbing set $K \subset \Omega$. Assumption (i) holds.

The Jacobian matrix of the model (2) is as follow

$$J_{P^*} = \begin{pmatrix} -(\delta_0+\mu) - \frac{(I^*+c)\alpha I^*}{(S^*+I^*+c)^2} & 0 & -\frac{(S^*+c)\alpha S^*}{(S^*+I^*+c)^2} & \eta \\ \frac{(I^*+c)\alpha I^*}{(S^*+I^*+c)^2} & -(\delta_0+\delta_1) & \frac{(S^*+c)\alpha S^*}{(S^*+I^*+c)^2} & 0 \\ 0 & \delta_1 & -(\delta_0+\delta_2+\delta_3) - \frac{a\beta}{(I^*+a)^2} & 0 \\ \mu & 0 & 0 & -(\delta_0+\eta) \end{pmatrix}.$$

Choosing the matrix H as $H = diag(-1, -1, 1, -1)$, it is easy to prove that HJH has non-positive off-diagonal elements, so we can see that model system (2) is competitive. This verifies the assumption (ii).

For our convenience we denote $S^* = S$, $E^* = E$, $I^* = I$ and $V^* = V$. The second additive compound matrix $J^{[2]}_{P^*}$ of the matrix J is

$$J^{[2]}_{P^*} = \begin{pmatrix} \Phi_1 & \frac{(S+c)\alpha S}{(S+I+c)^2} & 0 & \frac{(S+c)\alpha S}{(S+I+c)^2} & -\eta & 0 \\ \delta_1 & \Phi_2 & 0 & 0 & 0 & -\eta \\ 0 & 0 & \Phi_3 & 0 & 0 & -\frac{(S+c)\alpha S}{(S+I+c)^2} \\ 0 & \frac{(I+c)\alpha I}{(S+I+c)^2} & 0 & \Phi_4 & 0 & 0 \\ -\mu & 0 & 0 & 0 & \Phi_5 & \frac{(S+c)\alpha S}{(S+I+c)^2} \\ 0 & -\mu & \frac{(I+c)\alpha I}{(S+I+c)^2} & 0 & \delta_1 & \Phi_6 \end{pmatrix},$$

where

$$\Phi_1 = -\left(2\delta_0 + \delta_1 + \mu + \frac{(I+c)\alpha I}{(S+I+c)^2}\right), \qquad \Phi_4 = -\left(2\delta_0 + \delta_1 + \delta_2 + \delta_3 + \frac{a\beta}{(I+a)^2}\right),$$
$$\Phi_2 = -\left(2\delta_0 + \delta_2 + \delta_3 + \mu + \frac{(I+c)\alpha I}{(S+I+c)^2} + \frac{a\beta}{(I+a)^2}\right), \qquad \Phi_5 = -(2\delta_0 + \delta_1 + \eta),$$
$$\Phi_3 = -\left(2\delta_0 + \mu + \eta + \frac{(I+c)\alpha I}{(S+I+c)^2}\right), \qquad \Phi_6 = -\left(2\delta_0 + \delta_2 + \delta_3 + \eta + \frac{a\beta}{(I+a)^2}\right).$$

We have the second compound system of the model system (2) in a periodic solution

$$\left.\begin{aligned}
\frac{dX}{dt} &= -\left(\mu + 2\delta_0 + \delta_1 + \frac{\alpha(I+c)I}{(S+I+c)^2}\right)X + \frac{\alpha(S+c)S}{(S+I+c)^2}Y + \frac{\alpha(S+c)S}{(S+I+c)^2}L - \eta M,\\
\frac{dY}{dt} &= \delta_1 X - \left(\frac{\alpha(I+c)I}{(S+I+c)^2} + \frac{a\beta}{(I+a)^2} + \mu + 2\delta_0 + \delta_2 + \delta_3\right)Y - \eta N,\\
\frac{dZ}{dt} &= -\frac{\alpha(I+c)I}{(S+I+c)^2}Z - (\eta + \mu + 2\delta_0)L - \frac{\alpha(S+c)S}{(S+I+c)^2}N,\\
\frac{dL}{dt} &= \frac{\alpha(I+c)I}{(S+I+c)^2}Y - \left(\frac{a\beta}{(I+a)^2} + 2\delta_0 + \delta_1 + \delta_2 + \delta_3\right)L,\\
\frac{dM}{dt} &= -\mu X + \frac{\alpha(I+c)I}{(S+I+c)^2}Z - (\eta + 2\delta_0 + \delta_1)M + \frac{\alpha(S+c)S}{(S+I+c)^2}N,\\
\frac{dN}{dt} &= -\mu Y + \delta_1 M - \left(\frac{a\beta}{(I+a)^2} + \eta + 2\delta_0 + \delta_2 + \delta_3\right)N.
\end{aligned}\right\} \quad (14)$$

Next, we prove that system (14) is asymptotically stable. We can choose Lyapunov function as

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

By the uniform persistence, we obtain that the orbit of $P(t) = (S(t), E(t), I(t), V(t))$ remains a positive distance from the boundary of Ω, therefore we can know there exists a constant $c_1 > 0$, such that

$$V(X, Y, Z, L, M, N; S, E, I, V) \geq c_1 \sup\{|X|, |Y|, |Z|, |L|, |M|, |N|\},$$
$$\text{for all } (X, Y, Z, L, M, N) \in \Re^6 \text{and } (S, E, I, V) \in P(t).$$

Now, calculations lead to the following differential inequalities. Noting that,

$$\begin{aligned}
D_+(|X| + |Z| + |M|) &= -\left(\frac{\alpha(I+a)I}{(S+I+c)^2} + 2\mu + 2\delta_0 + \delta_1\right)|X|\\
&\quad + \frac{\alpha(S+a)S}{(S+I+c)^2}|Y| - (\eta + \mu + 2\delta_0)|Z|\\
&\quad + \frac{\alpha(S+a)S}{(S+I+c)^2}|L| - (2\eta + 2\delta_0 + \delta_1)|M|\\
&\leq -(2\delta_0 + \delta_1)|X| - (2\delta_0 + \delta_1)|Z| - (2\delta_0 + \delta_1)|M|\\
&\quad + \frac{\alpha(S+a)S}{(S+I+c)^2}(|Y| + |L|) \text{ [either } \eta > \delta_1 \text{ or } \mu > \delta_1]\\
&= -(2\delta_0 + \delta_1)(|X| + |Z| + |M|)\\
&\quad + \left(\frac{\alpha S}{S+I+c} - \frac{\alpha SI}{(S+I+c)^2}\right)(|Y| + |L|)\\
&\leq -(2\delta_0 + \delta_1)(|X| + |Z| + |M|) + \frac{\alpha S}{S+I+c}(|Y| + |L|)\\
&\leq -(2\delta_0+\delta_1)(|X|+|Z|+|M|)+ \frac{\alpha S}{S+I+c}(|Y|+|L|+|N|)\\
&= -(2\delta_0+\delta_1)(|X|+|Z|+|M|)+\frac{\alpha SI}{(S+I+c)E}\frac{E}{I}(|Y|+|L|+|N|).
\end{aligned}$$

$$
\begin{aligned}
D_+\left(|Y|+|L|+|N|\right) &= \delta_1|X| - \left(\frac{a\beta}{(a+I)^2}+2\mu+2\delta_0+\delta_2+\delta_3\right)|Y| + \delta_1|M| \\
&\quad - \left(\frac{a\beta}{(I+a)^2}+2\delta_0+\delta_1+\delta_2+\delta_3\right)|L| \\
&\quad - \left(\frac{a\beta}{(I+a)^2}+2\eta+2\delta_0+\delta_2+\delta_3\right)|N| \\
&\leq \delta_1\left(|X|+|M|\right) - \left(\frac{a\beta}{(I+a)^2}+2\delta_0+\delta_2+\delta_3\right) \\
&\quad \left(|Y|+|L|+|N|\right) \\
&= \delta_1\left(|X|+|M|\right) - \left(\frac{\beta}{I+a}-\frac{\beta I}{(I+a)^2}+2\delta_0+\delta_2+\delta_3\right) \\
&\quad \left(|Y|+|L|+|N|\right) \\
&= \delta_1\left(|X|+|M|\right) - \left(\frac{\beta}{I+a}+2\delta_0+\delta_2+\delta_3\right) \\
&\quad \left(|Y|+|L|+|N|\right) + \frac{\beta I}{(I+a)^2}\left(|Y|+|L|+|N|\right) \\
&\leq \delta_1\left(|X|+|Z|+|M|\right) - \left(\frac{\beta}{I+a}+2\delta_0+\delta_2+\delta_3\right) \\
&\quad \left(|Y|+|L|+|N|\right) + I\beta\left(|Y|+|L|+|N|\right)
\end{aligned}
$$

$$
\begin{aligned}
&D_+\frac{E}{I}\left(|Y|+|L|+|N|\right) \\
&= \frac{E}{I}D_+\left(|Y|+|L|+|N|\right) + \left(\frac{E'}{I}-\frac{EI'}{I^2}\right)\left(|Y|+|L|+|N|\right) \\
&= \frac{E}{I}D_+\left(|Y|+|L|+|N|\right) + \left(\frac{E'}{E}-\frac{I'}{I}\right)\frac{E}{I}\left(|Y|+|L|+|N|\right) \\
&= \frac{E}{I}\left(\delta_1\left(|X|+|Z|+|M|\right) - \left(\frac{\beta}{I+a}+2\delta_0+\delta_2+\delta_3-\beta I\right)\left(|Y|+|L|+|N|\right)\right) \\
&\quad + \left(\frac{E'}{E}-\frac{I'}{I}\right)\frac{E}{I}\left(|Y|+|L|+|N|\right) \\
&= \frac{E}{I}\delta_1\left(|X|+|Z|+|M|\right) + \left(\frac{E'}{E}-\frac{I'}{I}-\left(\frac{\beta}{I+a}+2\delta_0+\delta_2+\delta_3-\beta I\right)\right) \\
&\quad \frac{E}{I}\left(|Y|+|L|+|N|\right).
\end{aligned}
$$

From the previous formula, we lead to

$$
D_+|V(t)| \leq \max\left\{g_1(t), g_2(t)\right\}V(t),
$$

where

$$
g_1(t) = -(2\delta_0+\delta_1) + \frac{\alpha S}{(S+I+c)}\frac{I}{E},
$$

$$g_2(t) = \delta_1 \frac{E}{I} + \frac{E'}{E} - \frac{I'}{I} - \left(\frac{\beta}{I+a} + 2\delta_0 + \delta_2 + \delta_3 - \beta I \right).$$

From the model (2), we obtain

$$\frac{E'}{E} = \frac{\alpha S}{S+I+c}\frac{I}{E} - \delta_0 - \delta_1, \frac{I'}{I} = \delta_1 \frac{E}{I} - \left(\delta_0 + \delta_2 + \delta_3 + \frac{\beta}{I+a} \right).$$

Therefore,

$$g_1(t) = \frac{E'}{E} - \Delta_1, \quad g_2(t) = \frac{E'}{E} - \Delta_2. \quad \text{where } \Delta_1 = \delta_0 \text{ and } \Delta_2 = \delta_0 - \beta I.$$

Then,

$$\int_0^w \max\{g_1(t), g_2(t)\} dt = \ln E(t)|_0^w - w\Delta = -w\Delta. \quad \text{where } \Delta = \max(\Delta_1, \Delta_2)$$

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

which implies that $(X(t), Y(t), Z(t), L(t), M(t), N(t)) \to 0$, as $t \to \infty$. As a result, the second compound system (14) is asymptotically stable. □

This verifies the assumption (iii) of Lemma 1.

Let $J(P^*)$ be the Jacobian matrix of the model system (2) at P^*, we have

$$\det(J(P^*)) = \begin{vmatrix} -\left(\delta_0 + \mu + \frac{\alpha(I+c)I}{(S+I+c)^2}\right) & 0 & -\frac{\alpha(S+c)S}{(S+I+c)^2} & \eta \\ \frac{\alpha(I+c)I}{(S+I+c)^2} & -(\delta_0+\delta_1) & \frac{\alpha(S+c)S}{(S+I+c)^2} & 0 \\ 0 & \delta_1 & -\left(\delta_0+\delta_2+\delta_3+\frac{a\beta}{(I+a)^2}\right) & 0 \\ \mu & 0 & 0 & -(\delta_0+\eta) \end{vmatrix}$$

$$\begin{aligned} &= (\eta+\delta_0)\left(\left(\frac{\alpha(I+c)I}{(S+I+c)^2} + \mu + \delta_0\right)(\delta_0+\delta_1)\right. \\ &\quad \left.\left(\frac{a\beta}{(I+a)^2} + \delta_0 + \delta_2 + \delta_3\right) - \frac{\alpha(S+c)S(\mu+\delta_0)\delta_1}{(S+I+c)^2}\right) \\ &\quad + \eta\mu\left(\frac{\delta_1\alpha(S+c)S}{(S+I+c)^2} - (\delta_0+\delta_1)\left(\frac{a\beta}{(I+a)^2} + \delta_0 + \delta_2 + \delta_3\right)\right) \\ &= \left((\delta_0+\eta)\left(\frac{\alpha(I+c)I}{(S+I+c)^2} + \delta_0\right) + \mu\delta_0\right)(\delta_0+\delta_1)\left(\frac{a\beta}{(I+a)^2} + \delta_0 + \delta_2 + \delta_3\right) \\ &\quad - \frac{\delta_1\alpha(S+c)S}{(S+I+c)^2}(\mu+\eta+\delta_0)\delta_0 > 0. \end{aligned}$$

Thus, $(-1)^6 \det(J(P^*)) > 0$. The assumption (iv) holds.

This verifies all assumptions of Lemma 1, thus P^* is globally asymptotically stable in Ω.

5 Numerical simulations

To check the feasibility of our analysis regarding the existence of interior equilibrium and the corresponding stability conditions and illustrate the dynamical behaviour of the system, we have conducted some numerical computation using MATLAB 7.5. Figs. 2 and 3 shows the variations in susceptible, exposed, infected and vaccinated nodes with time for $R_0 < 1$ and $R_0 > 1$, respectively. From Fig. 2, it can be observed

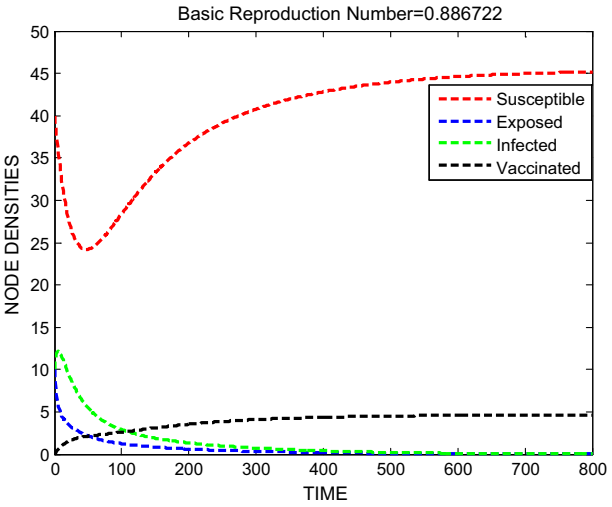

Fig. 2 Time series plot of model system (2) when $R_0 < 1$. $A = 1, \delta_0 = 0.02, \mu = 0.003, \alpha = 0.1, c = 0.01, \eta = 0.009, \delta_1 = 0.2, \delta_2 = 0.025, \delta_3 = 0.05, \beta = 0.003, a = 0.4$, Initial condition (40, 10, 10, 0)

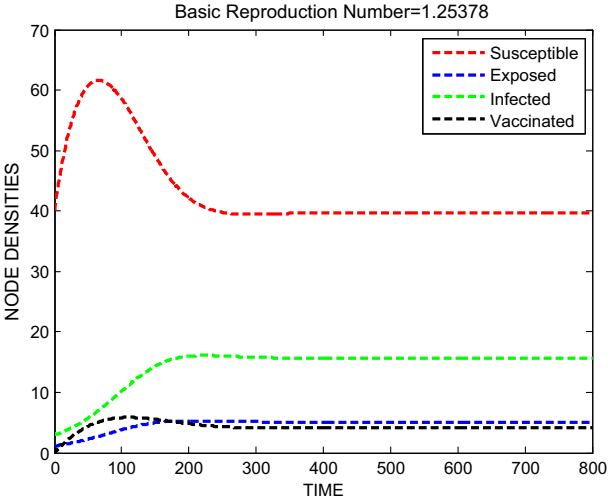

Fig. 3 Time series plot of model system (2) when $R_0 > 1\, A = 2, \delta_0 = 0.02, \mu = 0.003, \alpha = 0.1, c = 0.01, \eta = 0.009, \delta_1 = 0.2, \delta_2 = 0.025, \delta_3 = 0.02, \beta = 0.003, a = 0.4$, Initial condition (40, 1, 3, 0)

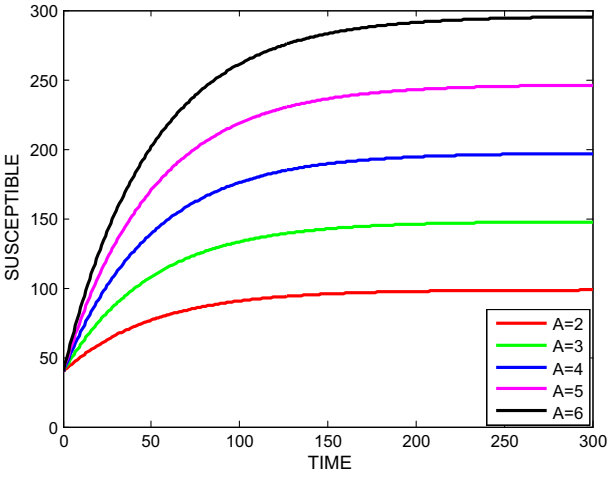

Fig. 4 Time series plot of susceptible class for different values of A. $A = 2, 3, 4, 5, 6$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.07$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.025$, $\delta_3 = 0.03$, $a = 0.4$, $\beta = 0.003$, Initial condition (40, 0, 0, 0)

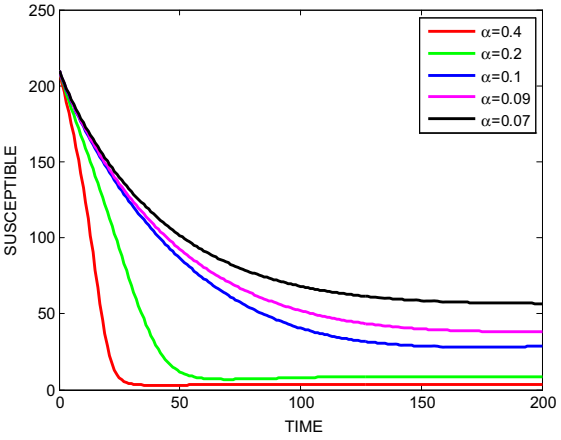

Fig. 5 Time series plot of susceptible class for different values of α. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.4, 0.2, 0.1, 0.09, 0.07$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.025$, $\delta_3 = 0.03$, $a = 0.4$, $\beta = 0.003$, Initial condition (210, 0, 10, 0)

that when $R_0 < 1$, the number of infected and exposed nodes decreases with time and finally becomes zero. This shows that when $R_0 < 1$, system settles to the virus-free equilibrium. Fig. 3 shows that when $R_0 > 1$, for a particular set of parameter values, all the variables settle down to a positive state and the virus is established in the system. In Fig. 4, the variations in number of susceptible nodes with respect to time for different values of recruitment rate 'A' are drawn. This figure shows that as the recruitment rate increases, the equilibrium number of susceptible nodes increases. Figs. 5, 6 and 7 are drawn to show the dynamical behavior of susceptible, infected and exposed nodes with respect to time for different values of α respectively. It is apparent

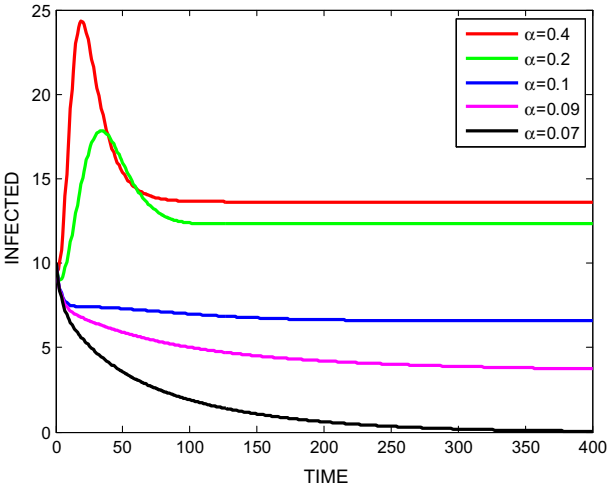

Fig. 6 Time series plot of infected class for different values of α. $A = 1.2, \delta_0 = 0.02, \mu = 0.003, \alpha = 0.4, 0.2, 0.1, 0.09, 0.07, c = 0.01, \eta = 0.2, \delta_1 = 0.2, \delta_2 = 0.025, \delta_3 = 0.03, \beta = 0.003, a = 0.4$, Initial (40, 0, 10, 0)

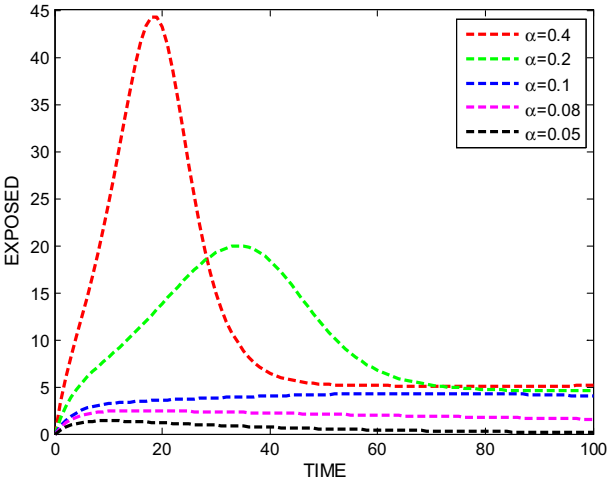

Fig. 7 Time series plot of exposed class for different values of α. $A = 1.2, \delta_0 = 0.02, \mu = 0.003, \alpha = 0.4, 0.2, 0.1, 0.08, 0.05, c = 0.01, \eta = 0.2, \delta_1 = 0.2, \delta_2 = 0.025, \delta_3 = 0.03, a = 0.4, \beta = 0.003$, Initial condition (210, 0, 10, 0)

from these figures that as the value of α increases, the number of susceptible nodes decreases while the number of infected and exposed nodes increases. Figs. 8, 9, 10 and 11 are the time series plot of susceptible and infected nodes for different values of δ_2 and δ_3. As the value of δ_2 and δ_3 increases, the number of susceptible nodes increases and the number of infected node decreases. Fig. 12 represents the dynamical behavior of the model in the SI-plane for different values of α, the rate at which virus transfer from infected to susceptible nodes. The equilibrium points are $(S, I) = (50, 15)$ and

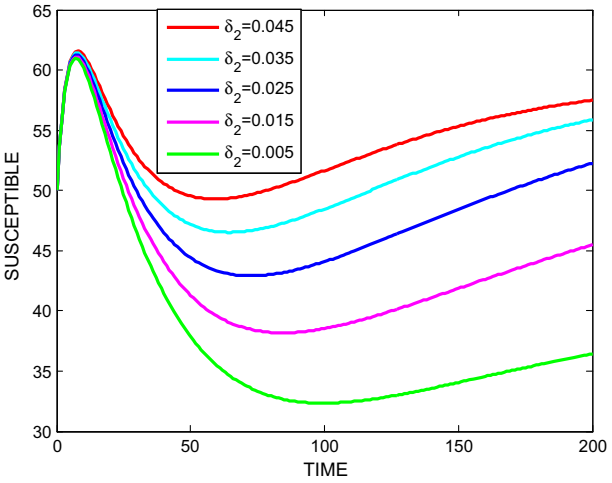

Fig. 8 Time series plot of susceptible class for different values of δ_2. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.07$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.045, 0.035, 0.025, 0.015, 0.005$, $\delta_3 = 0.03$, $\beta = 0.003$, $a = 0.4$, Initial condition (50, 10, 15, 25)

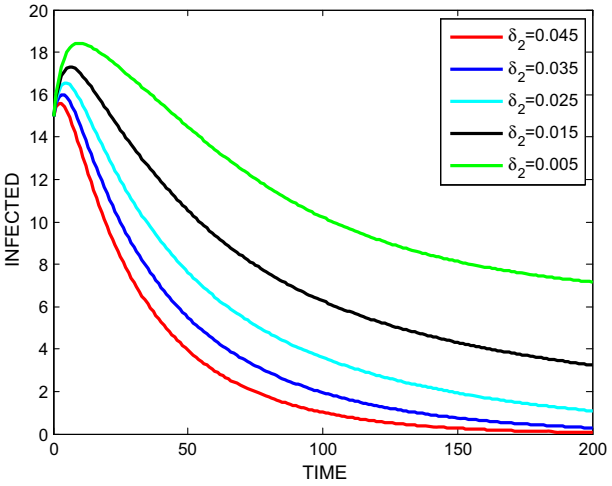

Fig. 9 Time series plot of infected class for different values of δ_2. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.07$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.045, 0.035, 0.025, 0.015, 0.005$, $\delta_3 = 0.03$, $\beta = 0.003$, $a = 0.4$, Initial condition (50, 10, 15, 25)

(59, 0) which shows that as we decrease the contact rates between susceptible and infected nodes, the number of susceptible nodes increases and for $\alpha = 0.05$, system becomes protected and no nodes is infected i.e., it is virus free case. Also from Fig. 13, we observe that as we decrease the contact rate α, the number of susceptible nodes increases from $(S, E) = (50, 0)$ to (59, 0) and we obtain the virus free system.

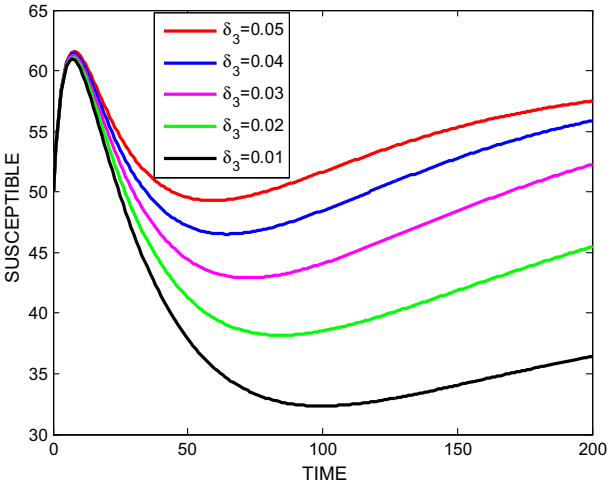

Fig. 10 Time series plot of susceptible class for different values of δ_3. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.07$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.025$, $\delta_3 = 0.05, 0.04, 0.03, 0.02, 0.01$, $\beta = 0.003$, $a = 0.4$, Initial condition (50, 10, 15, 25)

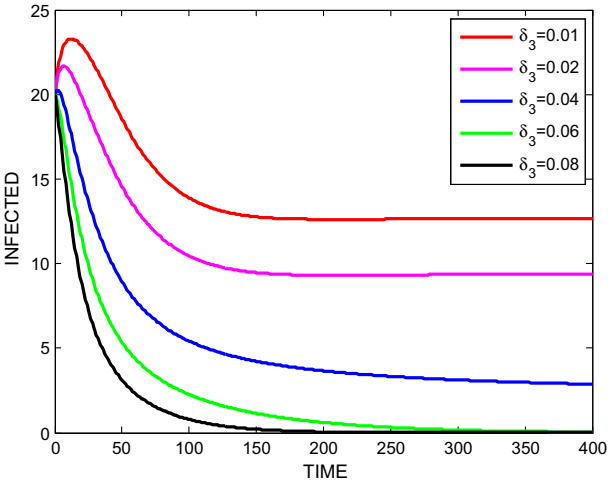

Fig. 11 Time series plot of infected class for different values of δ_3. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.1$, $c = 0.01$, $\eta = 0.009$, $\delta_1 = 0.2$, $\delta_2 = 0.025$, $\delta_3 = 0.01, 0.02, 0.04, 0.06, 0.08$, $\beta = 0.003$, $a = 0.4$, Initial condition (40, 10, 20, 25)

6 Discussions and conclusions

Recently, Kalbhor et al. [11] presented hidden markov models as a compelling option for malware identification and virus analysis. The transmission of viruses can be through horizontal and vertical transmission. Mishra and Pandey [17] developed an e-epidemic *SEIS-V* model for the transmission of worm using vertical transmission rate. In this paper, a dynamic e-epidemic *SVEIR* model has been proposed for the

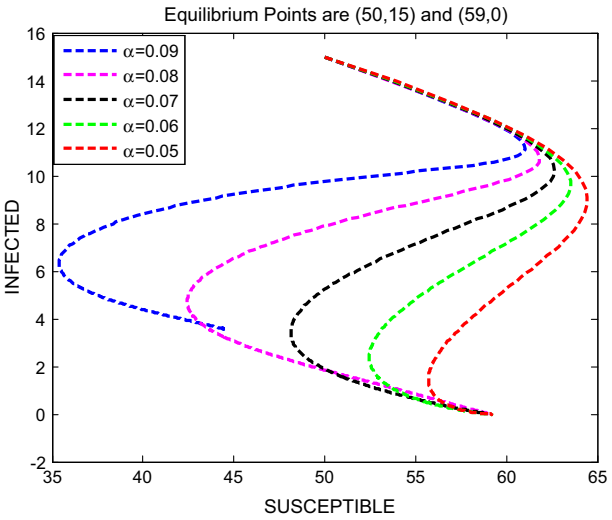

Fig. 12 SI-Phase plot with respect to time for different values of α. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.09, 0.08, 0.07, 0.06, 0.05$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.025$, $\delta_3 = 0.03$, $a = 0.4$, $\beta = 0.003$, Initial condition (50, 0, 15, 25)

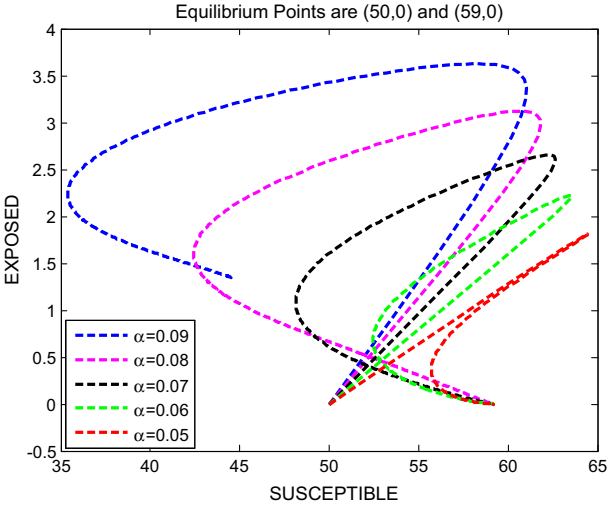

Fig. 13 SE-Phase plot with respect to time for different values of α. $A = 1.2$, $\delta_0 = 0.02$, $\mu = 0.003$, $\alpha = 0.09, 0.08, 0.07, 0.06, 0.05$, $c = 0.01$, $\eta = 0.2$, $\delta_1 = 0.2$, $\delta_2 = 0.025$, $\delta_3 = 0.03$, $a = 0.4$, $\beta = 0.003$, Initial condition (50, 0, 15, 25)

transmission of viruses using horizontal transmission in the computer network. We assume that the viruses possess a non-negligible latent period and infected nodes will stay in the latent period before they become infectious. We investigate the global dynamics behaviour of the reduced model system (2). For designed model system (1), we obtain the basic reproduction number $R_0 = 0.886722$. We have discussed the characteristic of basic reproduction number and established that if

$R_0 < \left(1 - \frac{\beta A}{(A+a\delta_0)(a(\delta_0+\delta_2+\delta_3)+\beta)}\right) = 0.92741$, the virus-free equilibrium is globally stable in the feasible region and the viruses fade out from the network, where as if $R_0 > 1$, the virus-free equilibrium is unstable. By analysis of the model system (2) and simulation studies, it has been investigated that the time of anti-virus technique and user observations are essential factors to controlling the virus transmission. By decreasing the contact rate α, we can increase the number of susceptible nodes. We also conclude that in modelling of communicable computer virus, the incidence rate (the rate of new infections) is considered to play a vital role in ensuring that the model can give a reasonable qualitative description of the virus dynamics.

Appendix

The matrix $J^{[2]}$ is the second additive compound matrix of the Jacobian, which for $n = 4$ is defined as

$$J^{[2]} = \begin{pmatrix} a_{11}+a_{22} & a_{23} & a_{24} & -a_{13} & -a_{14} & 0 \\ a_{32} & a_{11}+a_{33} & a_{34} & a_{12} & 0 & -a_{14} \\ a_{42} & a_{43} & a_{11}+a_{44} & 0 & a_{12} & a_{13} \\ -a_{31} & a_{21} & 0 & a_{22}+a_{33} & a_{34} & -a_{24} \\ -a_{41} & 0 & a_{21} & a_{43} & a_{22}+a_{44} & a_{23} \\ 0 & -a_{41} & a_{31} & -a_{42} & a_{32} & a_{33}+a_{44} \end{pmatrix}.$$

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