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On the local and global stability of an sirs epidemic model with logistic growth and information intervention

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Abstract: In this study, we investigate an susceptible-infected-recovered-susceptible (SIRS) epidemic model with logistic growth and information intervention. Firstly, the basic reproduction number R_0 is defined and the main results are given in terms of local stability. Then, sufficient conditions for the global stability of endemic equilibrium are obtained. Finally, some numerical simulations are given to validate our theoretical conclusions.

Key words: SIRS (susceptible-infected-recovered-susceptible) epidemic model, global stability, Lyapunov function, Volterra–Lyapunov stability

1. Introduction

Epidemiology is the scientific field that plays a decisive role in health-related states or events in specified populations [14]. In recent years, the establishment and analysis of mathematical models play a critical role in epidemiology. In [3, 6, 9, 11], a variety of epidemic models have been demonstrated and investigated extensively. With the investigation of epidemic diseases, it was noted that the behaviors of individuals changed according to the information they obtained about the epidemic. Behavior change resulting from this information has been found beneficial as it reduces the power of the epidemic [1, 2, 8, 13]. Information on the course of the epidemic can be disseminated through the media and social activities, so it can be used in the disease outbreak as an external intervention [9]. Therefore, researchers are increasingly interested in studying the impact of factors influencing behavior on the spread of infectious diseases [2, 8, 13]. Basically, two approaches have been used in the literature to include the impact of knowledge in a mathematical model. One of these approaches is the acceptance of a correction in the incidence rate [4, 7, 13], the other is the introduction of a new class of conscious individuals [5, 9, 13].

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In [9], the authors proposed the following SIRS epidemic model with information intervention:

$$\frac{dS(t)}{dt} = \Lambda - \beta S(t)I(t) - \rho S(t) + \delta R(t) - \mu m Z(t)S(t) \tag{1.1}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - (\rho + \nu + \gamma)I(t) \tag{1.2}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - (\rho + \delta)R(t) + \mu m Z(t)S(t) \tag{1.3}$$

$$\frac{dZ(t)}{dt} = \frac{\alpha_0 I(t)}{1 + \beta_0 I(t)} - \alpha_1 Z(t) \tag{1.4}$$

where $S(t), I(t)$, and $R(t)$ represent the densities of the susceptible, infectious, and recovery at time t , respectively. Also, $Z(t)$ denotes the density of information in the population. The parameter Λ denotes the birth rate of susceptible population, β indicates the rate of disease transmission from susceptible population to infective population. ρ is the natural mortality rate and δ is the rate at which total immunity disappears. m and $0 \leq \mu \leq 1$ represents the information interaction rate and response intensity, respectively. ν is the death rate related to the disease; γ is the recovery rate of the infected population. α_0 is the information growth rate and β_0 is the saturation constant; α_1 is the natural decay rate of information.

In this study, we suppose that the susceptible population in a country has logistic growth. Because in many realistic problems, the assumption that the susceptible population has logistical growth may be more appropriate for a relatively long-term disease or a disease with a high mortality rate. Thus we present the following SIRS epidemic model:

$$\frac{dS(t)}{dt} = rS(t)\left(1 - \frac{S(t)}{K}\right) - \frac{\beta S(t)I(t)}{1 + aI(t)} + \delta R(t) - \mu m Z(t)S(t), \tag{1.5}$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t)}{1 + aI(t)} - (\rho + \nu + \gamma)I(t), \tag{1.6}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - (\rho + \delta)R(t) + \mu m Z(t)S(t), \tag{1.7}$$

$$\frac{dZ(t)}{dt} = \frac{\alpha_0 I(t)}{1 + \beta_0 I(t)} - \alpha_1 Z(t). \tag{1.8}$$

Here r is the endogenous growth rate of susceptible population; K is the carrying capacity of the country that ignores the infected and recovered persons and a is the saturation constant. It is assumed in this paper all parameters are positive constants.

The rest of this paper is organized as follows. In Section 2, we show that all solutions of model (1.5)-(1.8) are non-negative and bounded in the non-negative cone of $\mathbb{R}^4$. In Section 3, we investigate the existence of equilibria and the local dynamics of equilibria. In Section 4, we concentrate on the analysis of global stability. In Section 5, we give some numerical examples to validate our theoretical conclusions.

2. Positivity and boundedness

Theorem 2.1 *All solutions* $(S(t), I(t), R(t), Z(t))$ *of model (1.5)-(1.8) with initial conditions* $S(0) \geq 0, I(0) \geq 0, R(0) \geq 0, Z(0) \geq 0$ *are non-negative and bounded for all* $t \geq 0$.

Proof From the model system (1.5)-(1.8), we have

$$\frac{dS}{dt}|_{S=0} = \delta R, \quad \frac{dI}{dt}|_{I=0} = 0, \quad \frac{dR}{dt}|_{R=0} = \gamma I + \mu m Z S, \quad \frac{dZ}{dt}|_{Z=0} = \frac{\alpha_0 I}{1+\beta_0 I}.$$

Note that all above ratios are non-negative on bounding planes of the non-negative cone of $\mathbb{R}^4$. So, if we start from the inside of this cone, we shall always stay in this cone in the direction of vector field is inward on all the bounding planes. As a result, all the solutions of the model (1.5)-(1.8) are non-negative [9].

Moreover from Eqs. (1.5)-(1.7), we take $V = S + I + R$ gives the differential equation given below:

$$\begin{aligned} \frac{dV}{dt} &= rS\left(1 - \frac{S}{K}\right) - (\rho + \nu)I - \rho R \\ &\leq rS\left(1 - \frac{S}{K}\right) - \rho(I + R), \end{aligned}$$

then there exists $\Lambda > 0$ and $\eta > 0$ such that

$$\frac{dV}{dt} \leq \Lambda - \eta(S + I + R) = \Lambda - \eta V.$$

This gives $\limsup_{t\to\infty} V \leq \frac{\Lambda}{\eta}$. Consequently, all solutions S, I and R are bounded by $\frac{\Lambda}{\eta}$. By using the bound of I, we obtain $\limsup_{t\to\infty} Z \leq \frac{\alpha_0\Lambda}{\alpha_1(\eta+\beta_0\Lambda)}$. Hence, we get the following positively invariant bounded set:

$$\Gamma = \{(S, I, R, Z) \in \mathbb{R}^4_+ | S + I + R \leq \frac{\Lambda}{\eta}, \ Z \leq \frac{\alpha_0\Lambda}{\alpha_1(\eta + \beta_0\Lambda)}, \ S \geq 0, \ I \geq 0, \ R \geq 0, \ Z \geq 0\}$$

for the model (1.5)-(1.8). This proves that the solutions of Eqs. (1.5)-(1.8) are bounded. □

3. Equilibria and local dynamics

In this section, we investigate the local stability analysis for the model (1.5)-(1.8). A significant threshold basic reproduction number R_0, which is the average number of secondary cases produced by an infected individual entering a population of susceptible individuals determines the stability of equilibrium points of a model. We obtain the basic reproduction number R_0 for the model (1.5)-(1.8) as follows:

$$R_0 = \frac{K\beta}{\rho + \nu + \gamma}. \tag{3.1}$$

The model (1.5)-(1.8) has following equilibria:

(i) the disease-free equilibrium $E^0 = (K, 0, 0, 0)$ and

(ii) the endemic equilibrium $E^* = (S^*, I^*, R^*, Z^*)$ which exists whenever $R_0 > 1$. Here,

$$\begin{aligned} S^* &= \frac{(\rho + \nu + \gamma)(1 + aI^*)}{\beta}, \\ R^* &= \frac{1}{\rho + \delta}\left(\gamma I^* + \mu m \frac{\alpha_0(\rho + \nu + \gamma)(1 + aI^*)I^*}{\alpha_1\beta(1 + \beta_0 I^*)}\right), \\ Z^* &= \frac{\alpha_0 I^*}{\alpha_1(1 + \beta_0 I^*)} \end{aligned}$$

and I^* is the positive root of the equation below

$$f(I) = A_1 I^3 + B_1 I^2 + C_1 I + D_1 = 0$$

where

$$\begin{aligned}
A_1 &= \frac{r\alpha_1 a^2 \beta_0 (\rho+\nu+\gamma)^2}{K\beta}, \\
B_1 &= \frac{r\alpha_1 (\rho+\nu+\gamma)^2 (a^2 + a\beta_0)}{K\beta} + \alpha_1 \beta \beta_0 (\rho+\nu) + \frac{\rho\gamma\alpha_1\beta\beta_0}{\rho+\delta} - r\alpha_1 a\beta_0(\rho+\nu+\gamma), \\
C_1 &= \frac{r\alpha_1 (\rho+\nu+\gamma)^2 (2a_0 + \beta_0)}{K\beta} + \alpha_1 \beta (\rho+\nu) + \frac{\rho\gamma\alpha_1\beta}{\rho+\delta} - r\alpha_1(\rho+\nu+\gamma)(a+\beta_0), \\
D_1 &= \frac{r\alpha_1 (\rho+\nu+\gamma)^2}{K\beta} - r\alpha_1(\rho+\nu+\gamma).
\end{aligned}$$

Note that $A_1 > 0$. If $R_0 > 1$, then $D_1 < 0$ and so the Eqs. (1.5)-(1.8) has at least one positive equilibrium. Further, if $B_1 > 0$, the model Eqs. (1.5)-(1.8) has a unique positive equilibrium.

The variational matrix corresponding to the model Eqs. (1.5)-(1.8) is given as:

$$J = \begin{pmatrix}
r - \frac{2rS}{K} - \frac{\beta I}{1+aI} - \mu m Z & -\frac{\beta S}{(1+aI)^2} & \delta & -\mu m S \\
\frac{\beta I}{1+aI} & \frac{\beta S}{(1+aI)^2} - (\rho+\nu+\gamma) & 0 & 0 \\
\mu m Z & \gamma & -(\rho+\delta) & \mu m S \\
0 & \frac{\alpha_0}{(1+\beta_0 I)^2} & 0 & -\alpha_1
\end{pmatrix}.$$

We now get the local stability of equilibrium points using the variational matrix J obtained above.

Theorem 3.1 *(i) The disease free equilibrium E^0 of the Eqs. (1.5)-(1.8) is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.*
(ii) If $R_0 > 1$ then the Eqs. (1.5)-(1.8) has a unique infected equilibrium E^* and in this instance E^* locally asymptotically stable provided the following conditions are satisfied:
$A_2 > 0, D_2 > 0, A_2 B_2 > C_2$ and $A_2(B_2 C_2 - A_2 D_2) > C_2^2$.

Proof (i) The Jacobian matrix corresponding to $E^0 = (K, 0, 0, 0)$ of Eqs. (1.5)-(1.8) is as follows

$$J^0 = \begin{pmatrix}
-r & -\beta K & \delta & -\mu m K \\
0 & \beta K - (\rho+\nu+\gamma) & 0 & 0 \\
0 & \gamma & -(\rho+\delta) & \mu m K \\
0 & \alpha_0 & 0 & -\alpha_1
\end{pmatrix}.$$

The eigenvalues of J^0 are

$$\lambda_1 = -r, \;\; \lambda_2 = -(\rho+\delta), \;\; \lambda_3 = -\alpha_1, \;\; \lambda_4 = (\rho+\nu+\gamma)(R_0 - 1).$$

It is clear that $\lambda_1, \lambda_2, \lambda_3 < 0$. Note that if $R_0 < 1$, $\lambda_4 < 0$ and so the disease-free equilibrium E^0 is locally asymptotically stable. Conversely, if $R_0 > 1$, $\lambda_4 > 0$ and so E^0 is unstable.

(ii) Let J^* be the Jacobian matrix corresponding to $E^* = (S^*, I^*, R^*, Z^*)$ of Eqs. (1.5)-(1.8). The characteristic equation of J^* as follows

$$\lambda^4 + A_2\lambda^3 + B_2\lambda^2 + C_2\lambda + D_2 = 0,$$

where

$$\begin{aligned}
A_2 &= \alpha_1 + \rho + \delta + \mu m Z^* + \frac{\beta I^*}{1+aI^*} + (\rho+\nu+\gamma)\left(1 - \frac{1}{1+aI^*}\right) + r\left(\frac{2S^*}{K} - 1\right), \\
B_2 &= \alpha_1(\rho+\nu+\gamma)\left(1 - \frac{1}{1+aI^*}\right) + \rho\mu m Z^* + \frac{\beta(\rho+\delta)I^*}{1+aI^*} + (\rho+\delta)r\left(\frac{2S^*}{K} - 1\right) \\
&+ \left(\alpha_1 + (\rho+\nu+\gamma)\left(1 - \frac{1}{1+aI^*}\right)\right)\left(\mu m Z^* + \frac{\beta I^*}{1+aI^*} + \rho + \delta + r\left(\frac{2S^*}{K} - 1\right)\right) \\
&+ \frac{\beta(\rho+\nu+\gamma)}{(1+aI^*)^2}, \\
C_2 &= \left(\alpha_1 + (\rho+\nu+\gamma)\left(1 - \frac{1}{1+aI^*}\right)\right)\left(\rho\mu m Z^* + \frac{\beta(\rho+\delta)I^*}{1+aI^*} + (\rho+\delta)r\left(\frac{2S^*}{K} - 1\right)\right) \\
&+ \alpha_1(\rho+\nu+\gamma)\left(1 - \frac{1}{1+aI^*}\right)\left(\mu m Z^* + \frac{\beta I^*}{1+aI^*} + \rho + \delta + r\left(\frac{2S^*}{K} - 1\right)\right) \\
&+ \frac{\beta(\rho+\alpha_1+\delta)(\rho+\nu+\gamma)I^*}{(1+aI^*)^2} + \frac{\alpha_0\mu m(\rho+\nu+\gamma)I^*}{(1+\beta_0 I^*)^2}, \\
D_2 &= \alpha_1(\rho+\nu+\gamma)\left(1 - \frac{1}{1+aI^*}\right)\left(\rho\mu m Z^* + \frac{\beta(\rho+\delta)I^*}{1+aI^*} + (\rho+\delta)r\left(\frac{2S^*}{K} - 1\right)\right) \\
&+ \frac{\alpha_0\mu m\rho(\rho+\nu+\gamma)I^*}{(1+\beta_0 I^*)^2}.
\end{aligned}$$

If $A_2 > 0, D_2 > 0, A_2B_2 > C_2$ and $A_2(B_2C_2 - A_2D_2) > C_2^2$ then by Routh–Hurwitz criterion, characteristic equation of J^* has the roots which are negative or with negative real parts. Hence, we finalize that E^* is locally asymptotically stable for $R_0 > 1$ supplied $A_2 > 0, D_2 > 0, A_2B_2 > C_2$ and $A_2(B_2C_2 - A_2D_2) > C_2^2$. □

4. Global stability analysis

In this section, we have obtained the sufficient conditions for global stability of E^0 and E^*.

Theorem 4.1 *The disease-free equilibrium $E^0 = (K, 0, 0, 0)$ of Eqs. (1.5)-(1.8) is globally asymptotically stable provided that the following condition holds:*

$$\mu m K < \alpha_1, \quad \beta K + \alpha_0 < \rho + \nu. \tag{4.1}$$

Proof Consider the following Lyapunov function

$$V_0(S, I, R, Z) = \left(S - S_0 - S_0 ln\frac{S}{S_0}\right) + I + R + Z. \tag{4.2}$$

Clearly V_0 is a positive definite function. If we differentiate V_0 with respect to t, we get

$$\begin{aligned}
\frac{dV_0}{dt} &= \frac{S-S_0}{S}\frac{dS}{dt}+\frac{dI}{dt}+\frac{dR}{dt}+\frac{dZ}{dt} \\
&= -\frac{r}{K}(S-S_0)^2-\left(\frac{S_0}{S}\delta+\rho\right)R+(\mu mK-\alpha_1)Z \\
&+ \left(\frac{\beta K}{1+aI}+\frac{\alpha_0}{1+\beta_0 I}-(\rho+\nu)\right)I \\
&\leq -\frac{r}{K}(S-S_0)^2-\left(\frac{S_0}{S}\delta+\rho\right)R+(\mu mK-\alpha_1)Z+(\beta K+\alpha_0-(\rho+\nu))I.
\end{aligned}$$

Obviously, if $\mu mK<\alpha_1$ and $\beta K+\alpha_0<\rho+\nu$, then $\frac{dV_0}{dt}\leq 0$. Therefore, E^0 is globally asymptotically stable, i.e. the disease fades out. Hence, the proof is completed. □

Theorem 4.2 *If $R_0>1$, then the infected equilibrium $E^*=(S^*,I^*,R^*,Z^*)$ is globally asymptotically stable.*

Proof We apply the method of Lyapunov functions combined with the theory of Volterra–Lyapunov stable matrices to prove the global asymptotic stability of E^*. For this, we determine the Lyapunov function as follows:

$$V^*=2w_1\left(S-S^*-ln\frac{S}{S^*}\right)+w_2(I-I^*)^2+w_3(R-R^*)^2+w_4(Z-Z^*)^2, \tag{4.3}$$

where w_1,w_2,w_3,w_4 are positive constants. If we calculate the time derivative of V^* along the trajectories of Eqs. (1.5)-(1.8), we get

$$\begin{aligned}
\frac{dV^*}{dt} &= 2w_1\left(1-\frac{S^*}{S}\right)\frac{dS}{dt}+2w_2(I-I^*)\frac{dI}{dt}+2w_3(R-R^*)\frac{dR}{dt}+2w_4(Z-Z^*)\frac{dZ}{dt} \\
&= 2w_1(S-S^*)\left(r\left(1-\frac{S}{K}\right)-\frac{\beta I}{1+aI}+\frac{\delta R}{S}-\mu mZ\right) \\
&+ 2w_2(I-I^*)\left(\frac{\beta SI}{1+aI}-(\rho+\nu+\gamma)I\right)+2w_3\left(\gamma I-(\rho+\delta)R+\mu mZS\right) \\
&+ 2w_4(Z-Z^*)\left(\frac{\alpha_0 I}{1+\beta_0 I}-\alpha_1 Z\right) \\
&= -2w_1\left(\frac{r}{K}+\frac{\delta R}{SS^*}\right)(S-S^*)^2+2w_1\left(\frac{a\beta I^*}{(1+aI)(1+aI^*)}-\frac{\beta}{1+aI}\right)(S-S^*)(I-I^*) \\
&+ 2w_1\frac{\delta}{S^*}(S-S^*)(R-R^*)-2w_1\mu m(S-S^*)(Z-Z^*)+2w_2\frac{\beta I}{1+aI}(I-I^*)(S-S^*) \\
&- 2w_2\left(\rho+\nu+\gamma-\frac{\beta S^*}{(1+aI)(1+aI^*)}\right)(I-I^*)^2+2w_3\gamma(R-R^*)(I-I^*) \\
&- 2w_3(\rho+\delta)(R-R^*)^2+2w_3\mu mS(R-R^*)(Z-Z^*)-2w_3\mu mZ^*(R-R^*)(S-S^*) \\
&- 2w_4\alpha_1(Z-Z^*)^2+2w_4\frac{\alpha_0}{1+\beta_0 I}(Z-Z^*)(I-I^*)-2w_4\frac{\alpha_0\beta_0 I^*}{(1+\beta_0 I)(1+\beta_0 I^*)}(Z-Z^*)(I-I^*) \\
&= Y(WA+A^TW^T)Y^T,
\end{aligned}$$

where $Y=(S-S^*, I-I^*, R-R^*, Z-Z^*)$, $W=diag(w_1,w_2,w_3,w_4)$ and

$$A=\begin{pmatrix} -\frac{r}{K}-\frac{\delta R}{SS^*} & -\frac{\beta}{(1+aI)(1+aI^*)} & \frac{\delta}{S^*} & -\mu m \\ \frac{\beta I}{1+aI} & \frac{\beta S^*}{(1+aI)(1+aI^*)}-(\rho+\nu+\gamma) & 0 & 0 \\ -\mu m Z^* & 0 & -(\rho+\delta) & \mu m S \\ 0 & \frac{\alpha_0}{1+\beta_0 I}-\frac{\alpha_0\beta_0 I^*}{(1+\beta_0 I)(1+\beta_0 I^*)} & 0 & -\alpha_1 \end{pmatrix}.$$

Now, to prove the global stability of E^*, we investigate that A is Volterra–Lyapunov stable. For this purpose, we show that matrices $\tilde{A}$ and $\tilde{A^{-1}}$ are Volterra–Lyapunov stable.

Step I:

$$D=-\tilde{A}=\begin{pmatrix} \frac{r}{K}+\frac{\delta R}{SS^*} & \frac{\beta}{(1+aI)(1+aI^*)} & -\frac{\delta}{S^*} \\ -\frac{\beta I}{1+aI} & \rho+\nu+\gamma-\frac{\beta S^*}{(1+aI)(1+aI^*)} & 0 \\ \mu m Z^* & 0 & \rho+\delta \end{pmatrix}.$$

$$-\tilde{D}=\begin{pmatrix} -\frac{r}{K}-\frac{\delta R}{SS^*} & -\frac{\beta}{(1+aI)(1+aI^*)} \\ \frac{\beta I}{1+aI} & \frac{\beta S^*}{(1+aI)(1+aI^*)}-(\rho+\nu+\gamma) \end{pmatrix}.$$

Clearly, $-\frac{r}{K}-\frac{\delta R}{SS^*}<0$. Remember that $S^*=\frac{(\rho+\nu+\gamma)(1+aI^*)}{\beta}$, then $\frac{\beta S^*}{(1+aI)(1+aI^*)}-(\rho+\nu+\gamma)=(\rho+\nu+\gamma)\left(\frac{1}{1+aI}-1\right)<0$. Hence

$$det(-\tilde{D})=(\rho+\nu+\gamma)\left(-\frac{r}{K}-\frac{\delta R}{SS^*}\right)\left(\frac{1}{1+aI}-1\right)+\frac{\beta^2 I}{(1+aI)^2(1+aI^*)}>0 \tag{4.4}$$

Based on the theory of Volterra–Lyapunov stable matrices [12], $-\tilde{D}$ is Volterra–Lyapunov stable.

Moreover, we obtain

$$-\tilde{D^{-1}}=\frac{1}{det(-\tilde{D})}\begin{pmatrix} (\rho+\nu+\gamma)\left(\frac{1}{1+aI}-1\right) & \frac{\beta}{(1+aI)(1+aI^*)} \\ -\frac{\beta I}{1+aI} & -\frac{r}{K}-\frac{\delta R}{SS^*} \end{pmatrix}.$$

It is clear that $det(-\tilde{D^{-1}})>0$. Thus, from the theory of Volterra–Lyapunov stable matrices [12], $-\tilde{D^{-1}}$ is Volterra–Lyapunov stable.

Therefore, $D=-\tilde{A}$ is diagonally stable, and so $\tilde{A}$ is Volterra-Lyapunov stable.

Step II: In a similar manner as above, we can show that $\tilde{A^{-1}}$ is Volterra–Lyapunov stable. We can obtain $E=-\tilde{A^{-1}}$,

$$E=\frac{1}{det(-\tilde{A})}\begin{pmatrix} e_{11} & e_{12} & e_{13} \\ e_{21} & e_{22} & e_{23} \\ e_{31} & e_{32} & e_{33} \end{pmatrix}.$$

where

$e_{11}=(\rho+\delta)(\rho+\nu+\gamma)\left(1-\frac{1}{1+aI}\right)$,

$$e_{12} = \frac{\beta(\rho+\delta)}{(1+aI)(1+aI^*)},$$
$$e_{13} = (\rho+\nu+\gamma)\frac{\delta}{S^*}\left(1-\frac{1}{1+aI}\right),$$
$$e_{21} = -(\rho+\delta)\frac{\beta I}{(1+aI)},$$
$$e_{22} = -\left[\left(\frac{r}{K}+\frac{\delta R}{SS^*}\right)(\rho+\delta)+\frac{\delta\mu m Z^*}{S^*}\right],$$
$$e_{23} = \frac{\beta\delta I}{S^*(1+aI)},$$
$$e_{31} = (\rho+\nu+\gamma)\left(1-\frac{1}{1+aI}\right)\mu m Z^*,$$
$$e_{32} = \frac{\beta m Z^*}{(1+aI)(1+aI^*)},$$
$$e_{33} = \left(\frac{r}{K}+\frac{\delta R}{SS^*}\right)(\rho+\nu+\gamma)\left(1-\frac{1}{1+aI}\right)+\frac{\beta^2 I}{(1+aI)^2(1+aI^*)}.$$

$$-\tilde{E} = \frac{1}{det(-\tilde{A})}\begin{pmatrix} (\rho+\delta)(\rho+\nu+\gamma)\left(\frac{1}{1+aI}-1\right) & \frac{\beta(\rho+\delta)}{(1+aI)(1+aI^*)} \\ -(\rho+\delta)\frac{\beta I}{(1+aI)} & -\left[\left(\frac{r}{K}+\frac{\delta R}{SS^*}\right)(\rho+\delta)+\frac{\delta\mu m Z^*}{S^*}\right] \end{pmatrix}.$$

It is clear that $det(-\tilde{E}) > 0$, and $-\tilde{E}$ is Volterra-Lyapunov stable.

$$-\tilde{E^{-1}} = \frac{1}{det(-\tilde{A})det(-\tilde{E})}\begin{pmatrix} -\left[\left(\frac{r}{K}+\frac{\delta R}{SS^*}\right)(\rho+\delta)+\frac{\delta\mu m Z^*}{S^*}\right] & -\frac{\beta(\rho+\delta)}{(1+aI)(1+aI^*)} \\ (\rho+\delta)\frac{\beta I}{(1+aI)} & (\rho+\delta)(\rho+\nu+\gamma)\left(\frac{1}{1+aI}-1\right) \end{pmatrix}.$$

From the theory of Volterra–Lyapunov stable matrices [12], it is easy to observe that $-\tilde{E^{-1}}$ is Volterra–Lyapunov stable. Therefore, A^{-1} is Volterra-Lyapunov stable.

Finally, $\frac{dV^*}{dt} < 0$, and by LaSalle's invariance principle [10], E^* is globally asymptotically stable in the interior of Γ provided that $R_0 > 1$. □

5. Numerical results

We now give some numerical simulations to confirm the global stability of the model investigated in Section 4. In this section, we set the hypothetical initial values as $(S(0), I(0), R(0), Z(0)) = (40, 10, 1, 10)$. We also take the parameter values that we have determined hypothetically as in Table . All the numerical simulations are done in MATLAB (MathWorks, Inc., Natick, MA, USA).

Using the parameters in Table , we obtain the basic reproduction number $R_0 = 9.0909 > 1$ and the unique infected equilibrium $E^* = (S^* = 4.7380, I^* = 9.6014, R^* = 46.8938, Z^* = 0.6487)$. Figure 1 and Figure 2 show the global stability of the infected equilibrium E^* when $R_0 > 1$. This means that when $R_0 > 1$, the disease becomes endemic. Finally, we can say that our numerical results validate our theoretical conclusions.

Table . Parameter values.

Parameter	*Description*	*Value*
r	Endogenous growth rate	0.5 $person\ day^{-1}$
K	Carrying capacity	40 $person\ day^{-1}$
a	Saturation constant of treatment	0.008
δ	Loss of immunity rate	0.001 day^{-1}
μ	Response intensity	0.008 day^{-1}
m	Information interaction rate	1 day^{-1}
β	Disease transmission rate	0.05 $person^{-1}day^{-1}$
ρ	Natural mortality rate	0.02 day^{-1}
ν	Death rate	0.1 day^{-1}
γ	Recovery rate	0.1 day^{-1}
α_0	Information growth rate	0.05
β_0	Saturation constant of information	0.05
α_1	Natural decay rate of information	0.5

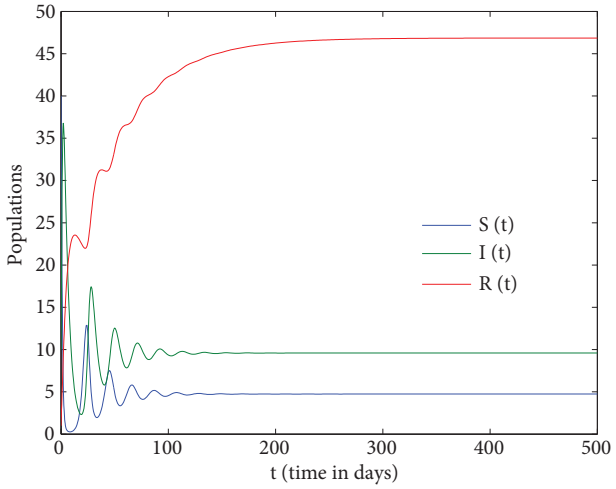

Figure 1. When $R_0 > 1$, solutions have reached the equilibrium point E^*.

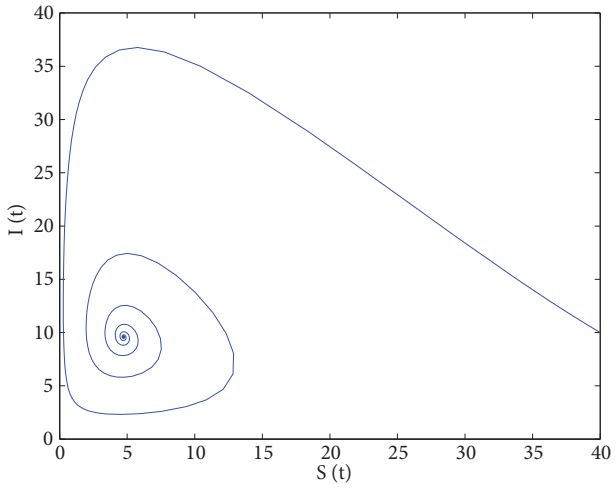

Figure 2. When $R_0 > 1$, E^* is globally stable.

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