Stability Analysis of SEIS Infectious Disease Model with Saturation Incidence

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Abstract

The SEIS epidemic model with saturation incidence as discussed, and the basic regeneration number R_0 was proposed. Methods: Qualitative and stability theory of differential equations and numerical simulation. The basic regeneration number R_0 was proposed, and the system is globally asymptotically stable at the equilibrium point. The basic regeneration number $R_0 \le 1$, the system had disease-free equilibrium point D_0, and the disease gradually died out. $R_0 > 1$ the model had a unique endemic equilibrium point D^* and the disease kept spreading and evolving into local endemic disease.

Keywords*: the basic regeneration number, equilibrium point, stability*

I. Background

The sudden attack of novel Coronavirus at the end of 2019 has brought immeasurable impact to China and the whole world. The popularity of many infectious diseases to human existence in history brought great harm, threat to humans. It not only brings pain to human beings, physically and mentally a certain negative impact to the people. Therefore, the current human desperately need to infectious diseases epidemic rule, transmission path, and through analyzing effective control measures to prevent [1-4].

After entering the 21st century, as people use mathematical model to explore the epidemic analysis, significant progress has been made in the prevention and control of infectious diseases. Capasso and other collaborators for a few kinds of nonlinear incidence of cholera virus dynamics model to carry on the deep research [5]. Then, a large number of scholars, based on the research results of different forms of nonlinear incidence analysis on inquiry, for example with actual more saturated incidence rate of joint, etc. Capasso and Serio firstly introduced the saturated incidence rate, with the increase of the number of infected people, Incidence gradually saturated. When this infectious disease into susceptible populations, βI measure the abilities of infections, $\frac{1}{1+mI}$ measuring inhibition, this inhibition reflects the susceptible population increase in the number of the influence of their behavior change and infected period individual crowding effect, using the appropriate parameter to prevent exposure to unboundedness.

Inspired by literature [6-7], this paper constructed an infectious disease model of SEIS with saturation incidence, and studied the stability of its basic regeneration number, disease-free equilibrium point and endemic equilibrium point.

II. Introduction to the Stability Analysis

2.1 Establishment of the model

The total population was divided into latent E(t), susceptible S(t) and infected I(t), and the epidemic transmission system was established in strict accordance with the concept of "warehouse" model, as shown in the figure 1 below:

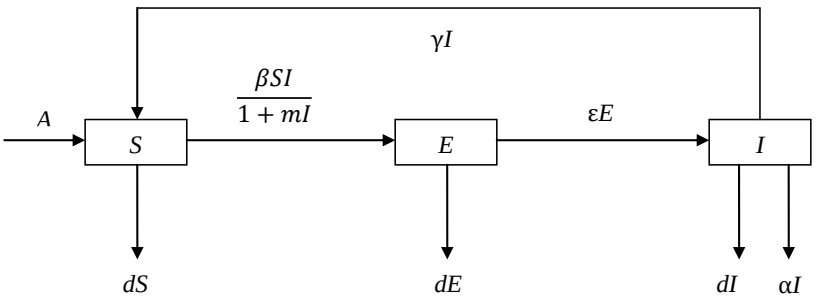

Fig 1: Block diagram of SEIS

According to the above plan, SEIS infectious disease model with saturation incidence

$$\begin{cases} \dfrac{dS}{dt} = A - dS - \dfrac{\beta SI}{1+mI} + \gamma I \\ \dfrac{dE}{dt} = \dfrac{\beta SI}{1+mI} - dE - \varepsilon E \\ \dfrac{dI}{dt} = \varepsilon E - \alpha I - dI - \gamma I \end{cases} \tag{1}$$

$N(t) = S(t) + E(t) + I(t)$,and S = S (t), E = E (t), I = I (t) represent the three types of the total amount of population, N = N (t) on behalf of the total population in t time, and A is population of input rate, $d(d>0)$ natural population mortality, β for individuals with transmission capacity, $\varepsilon(\varepsilon>0)$ said by the conversion rate of sleeper to litters, $\alpha(\alpha>0)$ for mortality due to illness, $\gamma(\gamma>0)$ for sick recovery rate, $m \in (0,1)$ the infected person after recovery do not have immunity. Then the system (1) of the three equations together: $\dfrac{d(S+E+I)}{dt} = \dfrac{dN}{dt} = A - dN - \alpha I \le A - dN$

According to the practical significance of the biological model, system (1) is considered in the set $\Omega = \left\{ (S+E+I) \in R_+^3; 0 < S+E+I \le \dfrac{A}{d} \right\}$.

Therefore, the maximum positive invariant set of system (1) is Ω [8].

2.2 Existence of equilibrium point and basic regeneration number

Theorem 1 At that time $R_0 > 1$, the system (1) has only disease-free equilibrium point $D^*(S^*, E^*, I^*)$ inside Ω; At that time $R_0 \le 1$, in system (1), there is a unique endemic equilibrium point $D_0\left(\dfrac{A}{d}, 0, 0\right)$ in addition to the disease-free equilibrium. The endemic equilibrium of system (1) is the system of equations

$$\begin{cases} A - dS - \dfrac{\beta SI}{1+mI} + \gamma I = 0 \\ \dfrac{\beta SI}{1+mI} - dE - \varepsilon E = 0 \\ \varepsilon E - \alpha I - dI - \gamma I = 0 \end{cases}$$

The positive solution on the set Ω can be solved as follows: $E^* = \frac{\alpha+d+\gamma}{\varepsilon} I^*, S^* = \frac{(\alpha+d+\gamma)(d+\varepsilon)}{\varepsilon\beta}(1+mI), I^* = \frac{d(\alpha+d+\gamma)(d+\varepsilon)-\varepsilon\beta A}{\varepsilon\beta\gamma-(\alpha+d+\gamma)(d+\varepsilon)(dm-\beta)}$

The stability of the equilibrium point is determined based on whether the basic regeneration number is greater than one. Therefore, in order to obtain the basic regeneration number R_0, the generation matrix of the second generation is considered: $F_1 = \begin{bmatrix} \frac{\beta SI}{1+mI} \\ 0 \end{bmatrix}, V_1 = \begin{bmatrix} dE+\varepsilon E \\ (\alpha+d+\gamma)-\varepsilon E \end{bmatrix}$, $F = \begin{bmatrix} \frac{\beta A}{d} & 0 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} 0 & d+\varepsilon \\ \alpha+d+\gamma & -\varepsilon \end{bmatrix}$

the $FV^{-1} = \begin{bmatrix} \frac{\varepsilon\beta A}{d(\alpha+d+\gamma)(d+\varepsilon)} & 0 \\ 0 & 0 \end{bmatrix}$.

The basic regeneration number R_0 can be obtained from the spectral radius of the matrix FV^{-1}, $R_0 = \rho(FV^{-1}) = \frac{\varepsilon\beta A}{d(\alpha+d+\gamma)(d+\varepsilon)}$.

Note: $b = d+\varepsilon, c = \alpha+d+\gamma$, so $R_0 = \frac{\varepsilon\beta A}{bcd}$.

2.3 Local and global stability of disease-free equilibrium points

Theorem 2 For the system (1), when $R_0 \le 1$, the disease-free equilibrium point $D_0\left(\frac{A}{d},0,0\right)$ is locally stable within the set Ω, and conversely, when $R_0 > 1$, it is $D_0\left(\frac{A}{d},0,0\right)$ unstable.

Proved: When $R_0 \le 1$, and $\frac{\varepsilon\beta A}{d} \le bc$, the Jacobian matrix of system (1) at the disease-free equilibrium point D_0 is: $J_{D_0} = \begin{bmatrix} -d & 0 & \frac{\beta A}{d}+\gamma \\ 0 & -b & \frac{\beta A}{d} \\ 0 & \varepsilon & -c \end{bmatrix}$

Therefore, the characteristic equation [9] at the disease-free equilibrium point D_0 is $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$, where $a_1 = b+c+d > 0, a_2 = bc - \frac{\varepsilon\beta A}{d} + d(b+c), a_3 = bcd - \varepsilon\beta A,$

$a_1a_2 - a_3 = (b+c+d)\left[bc - \frac{\varepsilon\beta A}{d} + d(b+c)\right] - (bcd - \varepsilon\beta A) \ge (b+c)\left[d(b+c)+d^2\right] > 0$.

Therefore, according to Routh-Hurwitz criterion, the disease-free equilibrium point $D_0\left(\frac{A}{d},0,0\right)$ is asymptotically stable locally on the set Ω, while on the contrary, it is unstable at that time $R_0 > 1$.

Theorem 3 For system (1), then $R_0 \le 1$, the disease-free equilibrium point $D_0\left(\frac{A}{d},0,0\right)$ is globally asymptotically stable within the set Ω.

Proved: Construct Liapunov function, then

$$V(S,E,I)=\varepsilon E+bIV'(S,E,I)=\varepsilon E'+bI'=I\frac{\varepsilon\beta A(R_0-1)-\varepsilon\beta AmI}{dR_0(1+mI)}\le 0,$$

when $I=0$, therefore, $V'(S,E,I)=0$.

According to Lassalle invariant set principle, the disease-free equilibrium point $D_0\left(\frac{A}{d},0,0\right)$ is globally asymptotically stable within the set Ω at that time $R_0 \le 1$.

2.4 Stability analysis of endemic equilibrium point

Theorem 4 About system (1), at that time $R_0>1$, the endemic disease equilibrium point $D^*\left(S^*,E^*,I^*\right)$ is locally asymptotically in Ω.

Proved: When $R_0>1$, Jacobian matrix of system (1) at that time D^* is: $J_{D^*}=\begin{bmatrix} -\frac{\beta I^*}{1+mI^*}-d & 0 & \frac{\beta S}{\left(1+mI^*\right)^2} \\ \frac{\beta I^*}{1+mI^*} & -b & 0 \\ 0 & \varepsilon & -c \end{bmatrix}$

Then the characteristic equation is: $\lambda^3+A_1\lambda^2+A_2\lambda+A_3=0$, where

$$A_1=b+c+d+\frac{\beta I^*}{1+mI^*}>0, A_2=\left(\frac{\beta I^*}{1+mI^*}+d\right)(b+c)+bc>0, A_3=bc\left(\frac{\beta I^*}{1+mI^*}+d\right)+\frac{\varepsilon\beta^2 SI}{\left(1+mI^*\right)^3}>0,$$

$$A_1A_2-A_3=\left(\frac{\beta I^*}{1+mI^*}+d\right)(b+c)^2+bc(b+c)+\left(\frac{\beta I^*}{1+mI^*}+d\right)^2(b+c)+\frac{\varepsilon\beta^2 SI}{\left(1+mI^*\right)^3}>0.$$

Therefore, according to Routh-Hurwitz criterion [10], the equilibrium point $D^*\left(S^*,E^*,I^*\right)$ of endemic disease was asymptotically stable Ω at that time $R_0>1$.

Theorem4.2 At that time $R_0>1$, the endemic disease equilibrium point $D^*\left(S^*,E^*,I^*\right)$ was globally asymptotically stable Ω.

Proved: The subsystem composed of the first two equations of the system (1) is considered:

$$\begin{cases} P(t)=A-dS-\frac{\beta SI}{1+mI}+\gamma I \\ Q(t)=\frac{\beta SI}{1+mI}-dE-\varepsilon E \end{cases} \tag{2}$$

Take the Dulac function $B(I)=\frac{1+mI}{I}$,

then there is $\frac{\partial(BP)}{\partial S}+\frac{\partial(BQ)}{\partial E}=-d\cdot\frac{1+mI}{I}-\beta-(d+\varepsilon)\cdot\frac{1+mI}{I}<0$

According to Bendixon-Dulac judgment, the equilibrium point $D^*(S^*,E^*,I^*)$ of endemic disease is globally asymptotic and stable Ω.

III. Example Verification

In system (1), if the parameters $A=0.35, d=0.7, \beta=0.01, \gamma=0.4, \varepsilon=0.24, \alpha=0.13, m\in(0,1)$ then $R_0\le 1$, the system is stable and there is no trend of the development of infectious diseases.

Initial values: $S(0)=0.79, E(0)=0.25, I(0)=0.01$.

Data simulation is shown in Figure 2 and Figure 3:

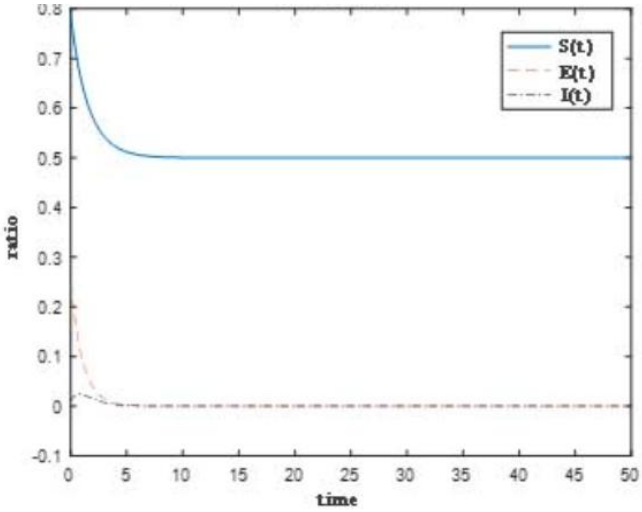

Fig 2: The trend of change of the variable

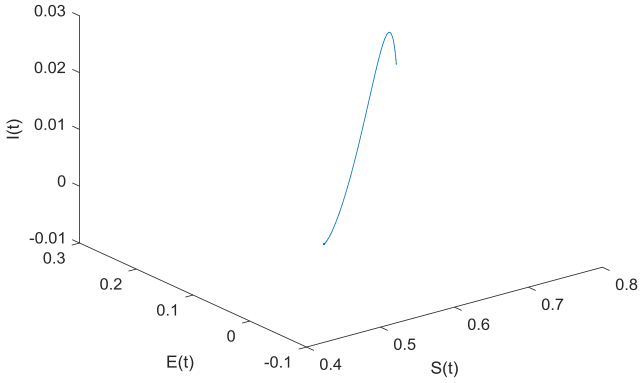

Fig 3: Stability of equilibrium

In system (1), the parameters $A=0.74, d=0.09, \beta=0.87, \gamma=0.9, \varepsilon=0.25, \alpha=0.01, m\in(0,1)$, when $R_0>1$ the system is unstable and the infectious disease is in the stage of transmission.

Data simulation is shown in Figure 4 and Figure 5:

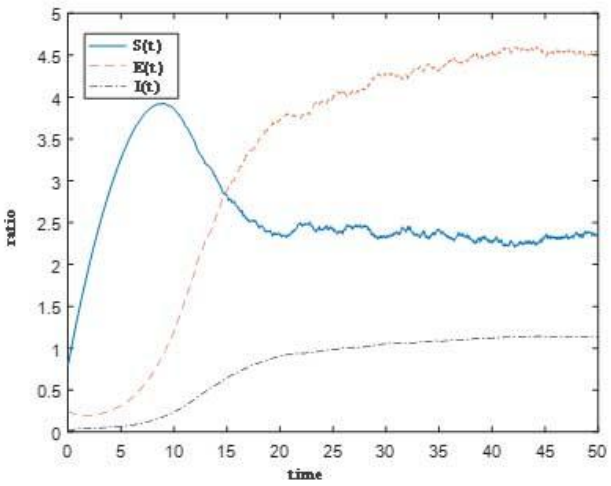

Fig 4: The trend of change of the variable

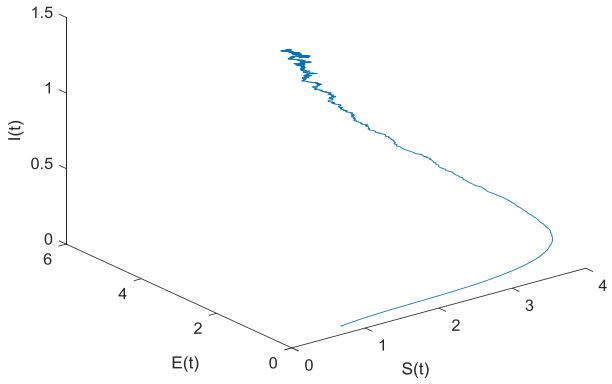

Fig 5: Stability of equilibrium

IV. Conclusions

Exist in the study to a class of saturated incidence SEIS epidemic model as the object of study, and the system of the basic reproductive number, the stability of the disease-free equilibrium and the endemic equilibrium is discussed, whether we can get R_0 less than 1 decided to infectious disease epidemic status. At that time $R_0\le 1$, the system is globally asymptotically stable in the disease-free equilibrium, that die of infectious diseases; In this case $R_0>1$, the equilibrium point of endemic diseases is globally progressive and stable, and infectious diseases will gradually become endemic diseases and their spread scope will continue to expand. Smaller R_0 is more conducive to the control and elimination of infectious diseases, $R_0=\dfrac{\varepsilon\beta A}{d(\alpha+d+\gamma)(d+\varepsilon)}$ which can be

minimized $\varepsilon\beta A$ and maximized $d(\alpha+d+\gamma)(d+\varepsilon)$ from availability. Specific measures to be taken include:

(1) Timely and effective treatment should be given to patients, enlargement γ.

(2) Reduce the constant A, and conduct reasonable control and screening of people entering the epidemic area.

Thus, the use of mathematical models help deeper understanding the dynamics of infectious diseases. Therefore, in combination with characteristics of the formation reason of disease, population growth and the development of the disease within populations, propagation law, and many other factors, build up step by step can be fully embody the mathematical model of dynamic characteristics of infectious diseases, relies on a model of dynamic form of quantitative and qualitative analysis to research, fully present the evolution of the disease process, its popular patterns revealed, for its change trend forecast, deep study of epidemic factors and reasons, find out the optimal strategy of prevention and control. From the theoretical level to the relevant subjects to develop effective prevention and treatment programs to pave the way.

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