เสถียรภาพกำกับวงกว้างของแบบจำลอง เอส ไอ อาร์ ที่มีอัตราการติดเชื้อแบบไม่เชิงเส้น

GLOBAL STABILITY OF SIR MODEL WITH NONLINEAR INCIDENCE RATE

สลิลทิพย์ แดงกองโค

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บทคัดย่อ

ในงานวิจัยนี้ เราได้ขยายการศึกษาแบบจำลองการแพร่ระบาดของเชื้อไวรัสแบบ เอส ไอ อาร์ ที่มีอัตราการติดเชื้อแบบไม่เชิงเส้นโดยสังข์สุวรรณ และแดงกองโค ในปี ค.ศ. 2015 โดยศึกษาเงื่อนไขของพารามิเตอร์ของแบบจำลองที่ทำให้จุดสมดุลแบบไม่มีเชื้อไวรัสมีเสถียรภาพกำกับวงกว้าง สิ่งที่ได้รับจากงานวิจัยนี้คือสามารถหาแนวทางในการป้องกันและควบคุมโรคระบาดได้ โดยการควบคุมค่าพารามิเตอร์ของอัตราการติดเชื้อไวรัสและอัตราการหายจากการติดเชื้อไวรัส ให้สอดคล้องกับเงื่อนไข $R_0 < 1$ เมื่อ R_0 คือค่าสืบพันธุ์พื้นฐาน

คำสำคัญ: แบบจำลอง เอส ไอ อาร์ จุดสมดุล อัตราการติดเชื้อแบบไม่เชิงเส้น เสถียรภาพกำกับวงกว้าง

Abstract

In this paper, we have extended the study of SIR model with nonlinear incidence rate of Sungsuwan & Daengkongkho in 2015 by considering the sufficient conditions for the model's parameters that will make the disease-free equilibrium point to be global stable. It was found that prevention and control of epidemic condition by controlling the parameters of the infection rate and the recovery rate that is $R_0 < 1$ where R_0 is basic reproduction number.

Keywords: SIR model, Equilibrium point, Nonlinear incidence rate, Global stability

Introduction

There are many germs living around us which cause diseases. The severity of diseases can be observed from the spread out of germs and immunity of the patients such as Cholera, Malaria, Dengue fever etc. This means that the more infectious germs combining with weaker immunity in the patients, the more severity of the epidemic. Therefore, mathematical model is an appropriate approach using in this study to investigate the behavior of epidemic model in order to offer a suggestion one how to control the disease. For many years, earlier researchers used simple models to study infection behavior, which the classical SIR model was established by Kermack & McKendrick 1927 (McCluskey, 2010). This model is based on the assumptions that the total population size does not change, no new population enters the system, none of population dies and recovered population has permanent immunity that can not be infected again.

Incidence rate also plays an important role in the modeling of epidemical dynamics. It has been suggested by several authors (Xu & Ma, 2009; McCluskey, 2010; Dumrongpokaphan et al., 2014; Sungsuwan & Daengkongkho, 2015) that the disease transmission process may have a nonlinear incidence rate. In many epidemic models, the bilinear incidence rate βSI and the standard incidence rate $\frac{\beta SI}{N}$ are frequently used. The bilinear incidence rate is based on the law of mass action. This contact law is suitable for communicable diseases such as influenza etc., but not for sexually transmitted diseases. It has been pointed out that for standard incidence rate, it may be good approximation if the number of available partners is large enough and everybody could not make more contacts than its practically feasible. After studying the cholera epidemic spread in Bari in 1973, by Capasso and Serio (Capasso & Serio, 1978) introduced a saturation level when I was large, i.e. $g(I)=\frac{\beta I}{1+\omega I}$, where βI measures the infection force of the disease and $\frac{1}{1+\omega I}$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate βSI because it includes the behavioral

change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters.

In this paper, we extend the study of SIR model with nonlinear incidence rate by Sungsuwan and Daengkongkho (Sungsuwan & Daengkongkho, 2015) as follows :

$$\left.\begin{aligned}\frac{dS(t)}{dt} &= C-\sigma_1 S(t)-\frac{\beta S(t)I(t)}{1+\alpha I(t)},\\ \frac{dI(t)}{dt} &= \frac{\beta S(t)I(t)}{1+\alpha I(t)}-(\sigma_2+\gamma)I(t),\\ \frac{dR(t)}{dt} &= \gamma I(t)-\sigma_3 R(t)\end{aligned}\right\}. \quad (1)$$

where $S(t)$ is the number of individuals susceptible to the disease at time t,

$I(t)$ is the number of infective member at time t,

$R(t)$ is the number of member, who have been removed from the possibility of infection through full immunity at time t,

C is a parameter which is a positive constant representing the birth rate and immigration rate of the population,

$\sigma_1, \sigma_2, \sigma_3$ are positive constants representing the natural death rate of the population which is assumed that $\sigma_1 \le \min\{\sigma_2, \sigma_3\}$,

β is the average number of contacts per infective per day,

$\frac{1}{1+\alpha I}$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals,

γ is a parameter which is a positive constant representing the recovery rate of infective individuals.

The global stability of the disease-free equilibrium point of such model is considered. That is, if the disease-free equilibrium point is stable, then the disease will not affect the population in the system.

Preliminaries

Definition 1 (Rothman & Greenland, 1998) **Basic reproduction number** R_0 is used to measure the transmission potential of a disease. It is thought of as the number of

secondary infections produced by a typical case of an infection in a population that is totally susceptible.

Definition 2 (Barnett & Cameron, 1985)

Considering the system described by

$$\dot{x} = f\left(t, x\right), \tag{2}$$

where as before $x\left(t\right)$ is the state vector and f is a vector having components $f_i\left(x_1, x_2, \ldots, x_n, t\right), i = 1, 2, \ldots, n$. We shall assume that the f_i are continuous and satisfy standard conditions, such as having continuous first partial derivatives so that the solution of (2) exist and is unique for given initial conditions. If $f\left(c, t\right) = 0$ for all t, where c is some constant vector, then it follows at once from (2) that if $x\left(t_0\right) = c$ then $x\left(t\right) = c$, all $t \geq t_0$. Thus solutions starting at c remain there, and c is said to be an **equilibrium point**.

Definition 3 (Barnett & Cameron, 1985) The equilibrium point $x = 0$ is said to be

5.1 **Stable** if for any positive scalar ε there exists a positive scalar δ such that $\left\|x\left(t_0\right)\right\|_e < \delta$ implies $\left\|x\left(t\right)\right\|_e < \varepsilon,\ t \geq t_0$, which $\left\|.\right\|_e$ is Euclidean norm.

5.2 **Asymptotically stable** if it is stable and if in addition $x\left(t\right) \to 0$ as $t \to \infty$.

Proposition 4 [**Standard comparison principle**(Robinson & Sierzega, 2018)]

Let $y\left(t\right) \in \mathrm{R}, t \in \left[0, T\right]$ be the unique solution of the differential equation

$$\dot{y} = f\left(y\right)$$

and let $x\left(t\right)$ and $z\left(t\right)$ satisfy the differential inequalities

$$\dot{x} \leq f\left(x\right) \text{ and } \dot{z} \geq f\left(z\right) \text{ for } t \in \left[0, T\right]$$

with $x\left(0\right) \leq y\left(0\right) \leq z\left(0\right)$. Then $x\left(t\right) \leq y\left(t\right) \leq z\left(t\right)$ on $\left[0, T\right]$.

Results

We rewrite the system (1) into the form

$$\left.\begin{aligned}\frac{dS}{dt} &= C-\sigma_1 S-\frac{\beta SI}{1+\alpha I},\\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha I}-(\sigma_2+\gamma)I,\\ \frac{dR}{dt} &= \gamma I-\sigma_3 R\end{aligned}\right\} \quad (3)$$

where $S=S(t), I=I(t)$ and $R=R(t)$.

The equilibrium points

Firstly, we study the equilibrium points of the system (3). These equilibrium points are determined analytically by setting $\dot{S}=\dot{I}=\dot{R}=0$. In (Sungsuwan & Daengkongkho, 2015), we obtained the equilibrium points of the system (3) when we set $\dot{S}=\dot{I}=\dot{R}=0$. Our result is the disease-free equilibrium point $E_0=(S_0,I_0,R_0)=\left(\frac{C}{\sigma_1},0,0\right)$ and an endemic equilibrium point $E_+=(S_+,I_+,R_+)$, where

$$S_+=\frac{C\alpha+\sigma_2+\gamma}{\beta+\alpha\sigma_1},$$

$$I_+=\frac{\beta C-\sigma_1(\sigma_2+\gamma)}{(\sigma_2+\gamma)(\alpha\sigma_1+\beta)},$$

$$R_+=\frac{\gamma}{\sigma_3}\frac{\beta C-\sigma_1(\sigma_2+\gamma)}{(\sigma_2+\gamma)(\alpha\sigma_1+\beta)}$$

and the basic reproduction number $R_0=\frac{C\beta}{\sigma_1(\sigma_2+\gamma)}$.

Stability Analysis

In what follows, In (Sungsuwan & Daengkongkho, 2015), we considered the local stability of the system (3), which is the following theorem.

Theorem 5 If $R_0<1$, then the disease-free equilibrium point E_0 is locally asymptotically stable.

Theorem 6 If $R_0>1$, then the endemic equilibrium point E_+ is locally asymptotically stable.

Now, we will study the global stability of disease-free equilibrium point. Firstly, we consider the following equation

$$\dot{u}(t)=\frac{au(t)}{1+\alpha u(t)}-cu(t);\ u(t)\geq 0 \text{ and } t\in[0,T] \tag{4}$$

where a,α and c are positive constants. By (4) always has a trivial equilibrium $u=0$.

The following lemma, we will present that the trivial equilibrium $u=0$ is globally asymptotically stable.

Lemma 7 If $a<c$, then the equilibrium $u=0$ of (4) is globally asymptotically stable.

Proof Assume that $a<c$. From (4), we have

$$\begin{aligned}\dot{u}(t)&\leq au(t)-cu(t)\\&=(a-c)u(t).\end{aligned}$$

Consider the following auxiliary equation

$$\dot{v}(t)=(a-c)v(t), \tag{5}$$

which $v(t)\geq 0$; $t\in[0,T]$.

Define

$$V(t)=\frac{1}{2}v^2(t).$$

Calculating the derivative of $V(t)$ along solution of equation (5), it follows that

$$\frac{d}{dt}V(t)=(a-c)v^2(t).$$

Hence, if $a<c$, then $\lim_{t\to+\infty}v(t)=0$. By standard comparison principle, it follows that if $a<c$, then $\lim_{t\to+\infty}u(t)=0$. □

Theorem 8 If $R_0<1$, then the disease-free equilibrium point E_0 is globally asymptotically stable.

Proof Let (S,I,R) be any solution of system (3). If $R_0<1$, then

$$\frac{\beta C}{\sigma_1}<\sigma_2+\gamma.$$

By Archimedian property, we may choose $\varepsilon>0$ sufficiently small and by inequality $\frac{\sigma_2+\gamma}{\beta}-\frac{C}{\sigma_1}>0$, this implies that

$$\varepsilon<\frac{\sigma_2+\gamma}{\beta}-\frac{C}{\sigma_1}.$$

Thus

$$\beta\left(\frac{C}{\sigma_1}+\varepsilon\right)<\sigma_2+\gamma. \tag{6}$$

We derive from the first equation of system (3), we have

$$\dot{S}\le C-\sigma_1 S.$$

By standard comparison principle, it follows that

$$\limsup_{t\to+\infty} S\le\frac{C}{\sigma_1}. \tag{7}$$

Hence, for $\varepsilon>0$ sufficiently small satisfying inequality (6), there is a $T_1>0$ such that if $t\ge T_1$, then $S\le\frac{C}{\sigma_1}+\varepsilon$.

For $\varepsilon>0$ sufficiently small satisfying inequality (6) and by inequality $S\le\frac{C}{\sigma_1}+\varepsilon$, we derive from the second equation of system (3) that, for $t>T_1$,

$$\dot{I}\le\frac{\beta\left(\frac{C}{\sigma_1}+\varepsilon\right)I}{1+\alpha I}-\left(\sigma_2+\gamma\right)I.$$

Next, we will consider the following auxiliary equation

$$\dot{u}=\frac{\beta\left(\frac{C}{\sigma_1}+\varepsilon\right)u}{1+\alpha u}-\left(\sigma_2+\gamma\right)u.$$

From inequality (6) and in the proof of Lemma 7, it follows that

$$\lim_{t\to+\infty} u=0.$$

By standard comparison principle, it follows that

$$\lim_{t\to+\infty} I=0,$$

that is,

$$\limsup_{t\to+\infty} I=0.$$

Hence, for $\varepsilon>0$ sufficiently small satisfying inequality (6), there is a $T_2>T_1$ such that if $t>T_2$, then $I\le\varepsilon$.

For $\varepsilon>0$ sufficiently small satisfying inequality (6) and by inequality $I\le\varepsilon$, we derive from the third equation of system (3) that for $t>T_2$,

$$\dot{R}\le\gamma\varepsilon-\sigma_3 R.$$

By standard comparison principle again and $\varepsilon > 0$ sufficiently small, it follows that

$$\lim_{t\to+\infty} R = 0,$$

that is,

$$\limsup_{t\to+\infty} R = 0.$$

For $\varepsilon > 0$ sufficiently small satisfying inequality (6) and by inequality $I \le \varepsilon$, we have

$$\frac{\beta SI}{1+\alpha I} \le \frac{\beta S\varepsilon}{1+\alpha\varepsilon}.$$

We derive from the first equation of system (3) that, for $t > T_2$,

$$\dot{S} \ge C - \sigma_1 S - \frac{\beta S\varepsilon}{1+\alpha\varepsilon}.$$

By standard comparison principle and $\varepsilon > 0$ sufficiently small, it follows that

$$\begin{aligned}\liminf_{t\to+\infty} S &\ge \frac{C}{\sigma_1 + \dfrac{\beta\varepsilon}{1+\alpha\varepsilon}} \\ &= \frac{C(1+\alpha\varepsilon)}{\sigma_1 + (\sigma_1\alpha+\beta)\varepsilon}.\end{aligned}$$

Letting $\varepsilon \to 0$, we obtain

$$\liminf_{t\to+\infty} S \ge \frac{C}{\sigma_1}. \tag{8}$$

From (7) and (8), we obtain that

$$\lim_{t\to+\infty} S = \frac{C}{\sigma_1}.$$

We conclude that if $R_0 < 1$ then E_0 is globally asymptotically stable. □

Conclusion

The author studied the global stability of a SIR model with a nonlinear incidence rate. The disease-free equilibrium point will be global asymptotically stable if $R_0 < 1$, which means that the disease will be eradicated in a period of time. The result of this paper found that the global stability of the disease-free equilibrium point was controlled by the basic reproduction number R_0. We known that the ways to control the spread of the disease is to decrease the incidence rate β and to increase the recovery rate γ for $R_0 < 1$. That is, if we developed the vaccines, anti-virus or the

medical profession then the recovery γ will been increasing. Therefore, we could protect and control the spread of disease by controlling $R_0 < 1$.

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