ANALYSIS ON STABILITY OF AN AUTONOMOUS DYNAMICS SYSTEM FOR SARS EPIDEMIC *

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Abstract: An extended dynamic model for SARS epidemic was deduced on the basis of the K-M infection model with taking the density constraint of susceptible population and the cure and death rates of patients into consideration. It is shown that the infection-free equilibrium is the global asymptotic stability under given conditions, and endemic equilibrium is not the asymptotic stability. It comes to the conclusion that the epidemic system is the permanent persistence existence under appropriate conditions.

Key words: infection model; SARS epidemic; equilibrium point; asymptotic stability

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Introduction

Severe acute respiratory syndrome (SARS) spreads most rapidly through the 23 areas and countries in the world since the first SARS case was reported in Guangdong in November, 2002 and reached its climax in April-May 2003. Although the period of the epidemic was over now, it was not clear about SARS origin, the mechanism of transmission, and the regularity of SARS emerging except that SARS as a novel coronavirus was known. The studies of SARS were made by a variety of ways and in many fields in order that SARS would be well known and effective measures of its prevention and treatment would be found as early as possible.

Two lectures on the dynamic model of SARS were published in Science Express of USA on May 23, 2003[1,2], in which the epidemiologic data from Hong Kong and Singapore were analyzed and epidemiologic parameters were estimated and predicted by both statistical and simulating methods. The result of study proved that the isolation of the people who were contacted by the patients would stop and prevent SARS from transmitting and extending. Wang Duo and Zhao Xiaofei found that the change and development of epidemic situation would be nearly described with Kermarck-Mckendric model after they made a verifiable

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analysis and prediction of epidemic situation of SARS on the basis of the original K-M
dynamic model\textsuperscript{[3]}. The model was too simple to take the birth rate and density constraint of
susceptible population and the cure and death rates of patients into consideration. We build a
more general dynamic model of SARS including all the factors and make a further
mathematical analysis on the stability of the system for SARS epidemic on the basis of the
qualitative theory.

The structure of the paper is arranged as follows. In Section 1, an autonomous
dynamics system for SARS epidemic is built. In Second 2, the nonnegativity and
boundedness of solution of the dynamic system are discussed. Both local stability of the
equilibria and global asymptotic stability of the system are separately discussed in Sections 3
and 4. Finally, we study the uniform permanence of the system in Section 5.

For easy discussion, the assumption is made as follows:

A1 During the prevalence of SARS we have three populations: the susceptible whose
total population density is denoted by $S$; the isolating whose total population density is
denoted by $E$; the patient with the symptom of SARS whose total population density is
denoted by $I$, where $S, E, I$ are all the function of time $t$.

A2 We assume that the susceptible population density grows according to a logistic
equation with carrying intrinsic growth rate $a$ and the density confining coefficient $b$.

A3 If the susceptible have a touch with the patient of SARS, then they can be
separated and sent to do the medical observation. We assume that the contact rate is $k$ of the
susceptible with the patient of SARS.

A4 Under the medical observation we assume that the confirmed diagnosis rate is $\lambda$.
Thus the $1/\lambda = T$ is the delitescence of SARS.

A5 The death rate of SARS is $d$ and the cure rate is $\mu$.

Based on the above assumption, we have the extended K-M\textsuperscript{[4]} differential model,
namely,

\begin{align}
\frac{dS}{dt} &= (a - bS - kI)S, \\
\frac{dE}{dt} &= kSI - \lambda E, \\
\frac{dI}{dt} &= -dI - \mu I + \lambda E,
\end{align}

where $a, b, k, \lambda, d, \mu$ are all positive constants.

We further assume that initiate conditions of the system (1) are

\begin{align}
S_0 = S(0) > 0, \quad E_0 = E(0) \geq 0, \quad I_0 = I(0) \geq 0. \quad (2)
\end{align}

1 Non-Negativity and Boundedness of Solutions

1.1 Equilibrium point of system

Let the right side of the system (1) be equal to zero. We obtain the equation set and
solve it. We have the extinction equilibrium point $E_0 = (0,0,0)$ and infection-free
equilibrium point $E_f = (a/b, 0, 0)$ of the system (1). There is a unique endemic equilibrium
point $E_+ = (S^*, E^*, I^*)$ for $R_0 = ak/(b(d + \mu)) > 1$, where
\[
\begin{align*}
S^* &= \frac{d + \mu}{k}, \\
E^* &= \frac{(d + \mu)(ak - b(d + \mu))}{\lambda k^2}, \\
I^* &= \frac{ak - b(d + \mu)}{k^2}.
\end{align*}
\] (3)

From Eq. (3) we know that the condition of existing the positive equilibrium point is \(R_0 > 1\). For \(R_0 = 1\), we have \(E_* = E_f\).

1.2 Positive invariability

Lemma 1 All solutions of the system (1) satisfying the initiate conditions (2) are positive.

Proof From the first equation of the system (1), namely, \(\frac{dS}{S} = (a - bS - kI)dt\), we obtain

\[S(t) = S(0)e^{\int_0^t (a - bS - kI)dt} > 0, \text{ for all } t.\]

If there is a solution of the system (1) satisfying that \(E(t), I(t)\) are non-positive, then let \(t_0\) denote the first point in the direction of number axis satisfying that \(E(t)\) or \(I(t)\) is equal to zero. When \(E(t)\) is equal to zero, from the second equation of the system (1), we have \(E'(t_0) = k(t_0)I(t_0) > 0\). For the point \(t\) of infinitely near the point \(t_0\) and \(t < t_0\), there is \(E(t) < 0\). It is in contradiction with the \(t_0\)'s definition. Similarly, if \(I(t)\) is equal to zero, from the third equation of the system (1), we also have the contradiction with the \(t_0\)'s definition. Hence all solutions of the system (1) satisfying the initiate conditions (2) are positive.

Let us denote \(R^3_{t_0} = \{ (S,E,I) : S \geq 0, E \geq 0, I \geq 0 \}\). According to the Lemma 1, we only discuss the properties of the system (1) in \(R^3_{t_0}\).

Lemma 2 \(R^3_{t_0}\) is the positive invariable set of the system (1).

Proof For any solution of the system (1), that is, \(S(t), E(t), I(t)\) there is

\[
\left| \frac{dS}{dt} \right|_{S=0} = 0, \quad \left| \frac{dE}{dt} \right|_{E=0} = kSI \geq 0, \quad S \geq 0, \quad I \geq 0,
\]

\[
\left| \frac{dI}{dt} \right|_{I=0} = \lambda E \geq 0, \quad \lambda, E \geq 0.
\]

This proves the Lemma 2.

1.3 Dissipativity of system

Proposition 1 The system (1) is the dissipative system, namely, the solutions of the system (1) for the all initiate conditions are the boundedness in the end.

Proof Let \((S(t), E(t), I(t))\) denote the solution of the system (1) satisfying the initiate conditions (2), then \(S, E, I \geq 0\) for any \(t > 0\). From the first equation of the system (1) we can have \(\lim_{t \to \infty} S(t) \leq a/b\). In brief, we introduce the notation \(Z(t) = S(t) + E(t) + \frac{1}{\lambda} I(t)\), thus

\[
\frac{dZ(t)}{dt} = aS(t) - bS^2(t) - \lambda E(t) - \frac{d + \mu}{\lambda} I(t) + E(t)
\]
< (a + 1)S(t) - S(t) - (\lambda - 1)E(t) - \frac{d + \mu}{\lambda}I(t)
\\
\leq \frac{a(a + 1)}{b} \delta Z(t),
\]
\]
where \( \delta = \min(1, \lambda - 1, (d + \mu)/\lambda) \). Applying the comparison theorem, we have
\]
\[ Z(t) \leq \frac{a(a + 1)}{b\lambda} + \left( Z(0) - \frac{a(a + 1)}{b\lambda} \right) e^{-\delta t}
\\
\leq \frac{a(a + 1)}{b\lambda} (t \to \infty).
\]

According to the non-negativity of solution in the system (1) we know that Proposition 1 holds.

In the following we define
\]
\[ \Omega = \left\{ (S, E, I) : S + E + I \leq M, \quad S, E, I \geq 0, M = \frac{a(a + 1)}{b\lambda} > 0 \right\}. \quad (4)
\]

Applying Proposition 1 we know that \( \Omega \) is the bounded set of the system (1) in the end.

2 Local Stability of Equilibrium Point

The Jacbian matrix of the system (1) is
\]
\]
\[ J(S, E, I) = \begin{pmatrix}
  a - 2bS - kI & 0 & -kS \\
  kI & -\lambda & kS \\
  0 & \lambda & -(d + \mu)
\end{pmatrix}. \quad (5)
\]

At the extinction equilibrium point \( E_0 \) the eigenequation is \((\lambda - a)(\lambda + \lambda)(\Lambda + d + \mu) = 0\). We can obtain a positive eigenvalue \( \Lambda_1 = a > 0 \) of the system (1). Thus \( E_0 = (0,0,0) \) is an instable saddle point.

Now we turn to consider the infection-free equilibrium \( E_f = (a/b, 0, 0) \). From Eq. (5) we have the \( E_f \)'s characteristic equation
\]
\[ (\Lambda + a)[\Lambda^2 + (\lambda + d + \mu)\Lambda + \lambda(d + \mu) - \lambda ka/b] = 0. \quad (6)
\]

Obviously, Eq. (6) has an eigenvalue \( \Lambda_1 = -a < 0 \). The other eigenvalues are the roots of the binomial equation
\]
\[ \Lambda^2 + (\lambda + d + \mu)\Lambda + \lambda(d + \mu) - \lambda ka/b = 0, \quad (7)
\]

that is, \( \Lambda = -\left(\lambda + d + \mu\right) \pm \sqrt{(\lambda + d + \mu)^2 - 4[\lambda(d + \mu) - \lambda ka/b]} \).

Hence Eq. (7) has two negative real roots for \( R_0 = ak/(b(d + \mu)) < 1 \) which implies that \( E_f = (a/b, 0, 0) \) is the stable equilibrium point. Equation (7) has a negative root and a zero root for \( R_0 = ak/(b(d + \mu)) = 1 \) which implies that \( E_f = (a/b, 0, 0) \) is the quasi-stable equilibrium point. Equation (7) has a positive real roots for \( R_0 = ak/(b(d + \mu)) > 1 \) which implies that \( E_f = (a/b, 0, 0) \) is the instable equilibrium point.

Based on the analysis above we come to Theorem 1.

**Theorem 1** The extinction equilibrium point \( E_0 = (0,0,0) \) of the system (1) is an instable saddle point. The infection-free equilibrium \( E_f = (a/b, 0, 0) \) of the system (1) for \( R_0 < 1 \) is the local asymptotically stable equilibrium point. At \( R_0 > 1 \), \( E_f \) is unstable.

Let us turn to analyze the local stability of the endemic disease equilibrium point
\]
\[ E_\ast = (S^\ast, E^\ast, I^\ast) = \left( \frac{d + \mu}{k}, \frac{(d + \mu)(ak - b(d + \mu))}{\lambda k^2}, \frac{ak - b(d + \mu)}{k^2} \right).
\]
From Eq. (5) we know that $E^+$'s Jacobian matrix is
\[
J(E^+) = \begin{pmatrix}
a - 2bS^* - kI^* & 0 & -kS^* \\
kI^* & -\lambda & kS^* \\
0 & \lambda & -d - \mu
\end{pmatrix}.
\]
The corresponding eigen-equation is
\[
\begin{align*}
\begin{vmatrix}
A - \lambda & \mu & kS^* \\
\mu & A - \lambda & \lambda d + \mu \\
kS^* & \lambda d + \mu & A - \lambda
\end{vmatrix} &= 0,
\end{align*}
\]
where
\[
A = a - 2bS^* - kI^*.
\]
Let us replace $S^*$, $I^*$ by Eq. (3) to the above equation, we have
\[
\begin{align*}
A(A + b(d + \mu)/k)(a + A + d + \mu) = 0. 
\end{align*}
\]
Thus Eq. (8) has a zero root, namely, the positive equilibrium point of the system (1) is unstable. We can obtain Theorem 2.

\textbf{Theorem 2} There is the unique endemic equilibrium point $E^+ (S^*, E^*, I^*)$ of the system (1) for $R_0 = ak/(b(d + \mu)) > 1$, and $E^+ (S^*, E^*, I^*)$ is critically stable but not asymptotically stable.

3 Global Asymptotic Stability

By Theorem 1, we know that the infection-free equilibrium $E_f = (a/b, 0, 0)$ of the system (1) for $R_0 < 1$ is the local asymptotically stable equilibrium point and the positive equilibrium point does not exist. We also know that the infection-free equilibrium $E_f = (a/b, 0, 0)$ of the system (1) for $R_0 > 1$ is unstable and the positive equilibrium point exists. There is $E^+ (S^*, E^*, I^*) \rightarrow E_f(a/b, 0, 0)$ for $R_0 \rightarrow 1$. In Theorem 3 we discuss the global asymptotic stability of $E_f = (a/b, 0, 0)$.

\textbf{Theorem 3} If $R_0 \leq 1$, then the infection-free equilibrium $E_f = (a/b, 0, 0)$ of the system (1) is globally asymptotically stable in $R_3^+$.

\textbf{Proof} Here we still use $\Omega$ of Eq. (4) as the subset in $R_3^0$. Let $\bar{R}_3^0$ denote $\{(S, E, I) \in R_3^0 \mid S > 0\}$. Consider Liapunov function $V: \bar{R}_3^0 \rightarrow R, V(S, E, I) = (S - (a/b)\ln S) + E + I$. Calculating the differential of the $V(S, E, I)$ at the direction of the system (1)’s solution, we obtain
\[
V' \big|_{(1)} = S' - \frac{a}{b} \frac{1}{S} S' + E' + I' = \left(1 - \frac{a}{b} \frac{1}{S}\right)(a - bS - kI)S + kSI - \lambda E - (d + \mu)I + \lambda E
\]
\[
= -b \left(S - \frac{a}{b}\right)^2 - \left(d + \mu - k \frac{a}{b}\right)I.
\]
From Eq. (9) we can know that $-b(S - a/b)^2$ is negative. If $R_0 < 1$, then $(d + \mu - ka/b)I$ is also negative. Hence $V'(t) \big| \_{(1)} < 0$ is correct. If $R_0 = 1$, then $V'(t) \big| \_{(1)} = 0$, namely, $V'(t) \big| \_{(1)}$ is always negative and $V'(t) \big| \_{(1)} = 0$ if and only if $S = a/b, E = 0$. Because of $N = \{(S, E, I) \mid V(S, E, I) \big| \_{(1)} = 0\} = \{(I, E, I) \mid S = a/b, E \geq 0, I = 0\} \cap \Omega$, the biggest invariant set of $N$ is $E_f = (a/b, 0, 0)$. According to the Liapunov-Lasalle theorem, $E_f = (a/b, 0, 0)$ is global asymptotically stable in $R_3^+$.  

4 Persistence Existence of System

For convenience, we introduce the definition as follows.
Definition 1 (Strong uniform repellence) The equilibrium point of system. If there is \( \lim d(X, \hat{E}) > 0 \) for any solution \( X \) of system in domain \( \Omega \), then \( \hat{E} \) is called the strong uniform repellence in \( \Omega \).

Definition 2 (Strong uniform permanent existence) If there is \( \min \{ \liminf X(t) \} > 0 \) for any solution \( X \) of system, then the system is called the strong uniform permanent existence.

On the basis of Definitions 1 and 2, we verify system (1) is strong uniform permanent.

Lemma 3 If \( R_0 > 1 \), then it is impossible to find \( (S, E, I) \in \Omega^2 (\Omega^2 \) is the interior of \( \Omega \)) satisfying \( \lim (S(t), E(t), I(t)) = \left( \frac{a}{b}, 0, 0 \right) \).

Proof Constructing the function \( V(E, I) = \omega_1 E + \omega_2 I, \omega_i \in \mathbb{R}_+, i = 1, 2 \), we obviously obtain that \( V(E, I) \) is the positive definite in \( \Omega^2 \). Let \( I_\varepsilon \) denote the \( \varepsilon \)-neighborhood of \( E_\varepsilon = \left( \frac{a}{b}, 0, 0 \right) \) in \( \Omega \), then

\[
V'(E, I) \mid_{(1)} = \omega_1 kSI - \omega_1 \lambda E - \omega_2 (d + \mu) I + \omega_2 \lambda E \\
= (\omega_1 kS - \omega_2 (d + \mu)) I + (\omega_2 - \omega_1) \lambda E \\
\geq (\omega_1 k(\frac{a}{b} - \varepsilon) - \omega_2 (d + \mu)) I + (\omega_2 - \omega_1) \lambda E. \tag{10}
\]

If both coefficients \( (\omega_1 k(\frac{a}{b} - \varepsilon) - \omega_2 (d + \mu)) \) and \( (\omega_2 - \omega_1) \) of Eq. (10) are positive, namely,

\[
\omega_2 > \omega_1, \quad \omega_1 > \frac{d + \mu}{k(\frac{a}{b} - \varepsilon)}, \tag{11}
\]

then \( \omega_2 > \frac{d + \mu}{k(\frac{a}{b} - \varepsilon)} \) and \( (d + \mu)/(k(\frac{a}{b} - \varepsilon)) < 1 \), or \( b(d + \mu)/(ak) < 1 - (b/a) \varepsilon \). Hence it is easy to testify that if \( (d + \mu)/k < a/b - \varepsilon \) (here the positive equilibrium point \( S^* \) exists and \( S^* < a/b \)), then for any \( \varepsilon \in (0, a/b - S^*) \), \( b(d + \mu)/(ak) < 1 - (b/a) \varepsilon \) holds true.

As long as we correctly select \( \omega_1, \omega_2 \), Eq. (11) holds. Thus there is \( \delta > 0 \) satisfying \( V'(E, I) \mid_{(1)} > \delta V(E, I) \) in \( I_\varepsilon \). \( V(E, I) \to +\infty \) holds for \( t \to +\infty \). If there is a solution of the system (1) satisfying \( \lim (S(t), E(t), I(t)) = (a/b, 0, 0) \) for \( T > 0 \) and \( t > T \), then \( (S(t), E(t), I(t)) \in I_\varepsilon \). This is in contradiction to \( V(E, I) \to +\infty \). This proves Lemma 3.

Theorem 4 The infection-free equilibrium \( E_f = (a/b, 0, 0) \) in \( \Omega^0 \) for \( R_0 > 1 \) is the strong uniform repellence.

Proof Here we make use of the norm definition \( \| (S, E, I) \| = \| S \| + \| E \| + \| I \| \).

By Lemma 3 we can choose \( \varepsilon \in (0, a/b - S^*) \) satisfying \( (S, E, I) \in \Omega^0 \) for \( R_0 > 1 \). Thus there is \( t_0 \geq 0 \) satisfying

\[
\| (S(t), E(t), I(t)) - (a/b, 0, 0) \| \geq 1 \| S(t) - a/b \| > \varepsilon, \quad \text{for } t \geq t_0,
\]

which implied that there is \( \varepsilon > 0 \) satisfying \( \liminf_{t \to +\infty} d((S, E, I), E_f) > \varepsilon \), where

\[
d((S, E, I), E_f) = \inf_{t > 0} \| (S, E, I) - (a/b, 0, 0) \|.
\]

By Definition 1, Theorem 4 holds.

Applying Definition 2 and Theorem 4 we are able to deduce the most important Theorem 5.

Theorem 5 The system (1) is the strong uniform permanent existence for \( R_0 > 1 \), namely, there is \( \min \{ \liminf S(t), \liminf E(t), \liminf I(t) \} \geq \varepsilon \) for \( \varepsilon > 0 \) and any solution \( (S(t), E(t), I(t)) \) of the system (1).
On the basis of Theorem 5 and Proposition 1, namely, the solution of the system is bounded, we can know that system (1) is the permanent persistence existence\(^4\).

5 Results

Because we have got little knowledge about SARS and it was supposed that many accidental factors would contribute to the outbreak of SARS, we shall make a further study of SARS from many parts of its pathogeny, pathology and epidemic. We have drawn two conclusions from our mathematical model that for the system (1) the infection-free equilibrium is global asymptotically stable for \( R_0 \leq 1 \) and strong uniform permanently existent for \( R_0 > 1 \). That is to say that the system (1) is a controllable and stable system for \( R_0 \leq 1 \) and it is permanently existent for \( R_0 > 1 \) (namely, the infection will become an endemic).

The stability and persistence of the system entirely depended on the threshold \( R_0 \). According to expression \( R_0 = \frac{ak}{b(d + \mu)} \), we knew that \( R_0 \) was decided by parameters \( k, d, \mu, a \) and \( b \). For some regions, the intrinsic growth rate \( a \) and death rate \( d \) and the density confined coefficient \( b \) can be thought as constants, but \( k, \mu \) changed greatly during some period. For example, if \( k \) decreases and \( \mu \) increases, then \( R_0 \) will become smaller. The system (1) will become stable. Similarly, whereas \( k \) increases and \( \mu \) decreases, \( R_0 \) will become bigger. The system (1) will become instable and permanently existent. Thus we can foresee that the SARS will be eliminated if the contact rate \( k \) is rigorously controlled and the cure rate \( \mu \) is improved. The result coincides with the real situation.

The model, however, may be simple and limited. For example, the rates of exposure, death and cure were assumed to be constants, but these parameters, in fact, were related to the particular distribution of the population. So the rate of the exposure to SARS varied among the people in the different area. These factors were lost or simplified in consideration of mathematical discussion. However, our study has shown that the model is useful for the description of changing condition of the SARS. A lot of work will be done to build an effective and exact model.

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