Research Article

Persistence of an SEIR Model with Immigration Dependent on the Prevalence of Infection

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1. Introduction

Mathematical models have been used to predict the spread of infectious diseases of humans and animals since the pioneering work of Anderson and May [1]. Many diseases such as tuberculosis and chronic hepatitis have the longer exposed period; thus, in some common researches, a population is divided into four classes: susceptible, exposed, infective, and recovered. In many studies on epidemic models, the goal is to understand the key factors affecting disease transmission [2–5], and this often includes determining a threshold condition for the persistence and extinction of the disease.

Many diseases such as influenza, measles, and sexually transmitted diseases are easily spread between regions (such as countries and cities) due to travel. This population dispersal is an important aspect to consider when studying the spread of a disease [6–8]. We will investigate a disease transmission model with population immigration from other regions to the one considered.

In many models, it is assumed that, in the absence of infection, the growth rate of population is given by \( N' = A - \mu N \), where \( A \) is thought to be the input rate of population. Here, we consider \( A \) as the sum of two parts, \( A_1 \) and \( A_2 \), where \( A_1 \) is the birth rate of the population and \( A_2 \) is the immigration rate from other regions. Since the spread of the infection usually affects the immigration to the region, then we will introduce the effect into an SEIR epidemic model and consider this persistence and extinction of the disease in this paper.
2. Model

In this paper, we consider an SEIR epidemic model with immigration:

\[ S' = \mu_1 B_1 + \frac{\mu_1 B_2}{1 + mI} - \mu_1 S - \beta SI, \]
\[ E' = \beta SI - (\mu_1 + \epsilon)E, \]
\[ I' = \epsilon E - (\mu_1 + \alpha + \gamma)I, \]
\[ R' = \gamma I - \mu_1 R. \]

(2.1)

Here, \( S = S(t), E = E(t), I = I(t), \) and \( R = R(t) \) represent the numbers of susceptible, exposed, infectious, and recovery individuals at time \( t \), respectively. \( \mu_1 B_1 \) is the input rate; \( \mu_1 B_2/(1 + mI) \) is the immigration rate from other regions (such as countries or cities); it depends on the number of infectious individuals in the region considered, where \( \mu_1 B_2 \) is the immigration rate in the absence of disease and \( m \) reflects the effect of infection on immigration from other regions; \( \mu_1 \) is the per capita natural death rate; \( \beta \) is the transmission coefficient of infection; \( \epsilon \) is the transfer rate from the exposed compartment to the infectious one; \( \alpha \) is the per capita disease-induced death rate.

From model (2.1) we have

\[ (S + E + I + R)' = \mu_1 B_1 + \frac{\mu_1 B_2}{1 + mI} - \mu_1 (S + E + I + R) - \alpha I \]
\[ \leq \mu_1 [(B_1 + B_2) - (S + E + I + R)]. \]

(2.2)

It follows that \( \limsup_{t \to \infty} (S + E + I + R) \leq B_1 + B_2 \), then system (2.1) is bounded.

Since the variable \( R \) does not appear explicitly in the first three equations in system (2.1), then we need only to consider the dynamics of a subsystem consisting of the first three equations in system (2.1). For this subsystem, making the following variable transformations:

\[ S = \frac{\beta}{(\epsilon m^2) \cdot \overline{S}}, \quad E = \frac{\beta}{(\epsilon m^2) \cdot \overline{E}}, \quad I = \frac{1}{m} \cdot \overline{I}, \quad t = \frac{m}{\overline{\beta}} \cdot \overline{t}, \]

(2.3)

and removing the bar in \( \overline{S}, \overline{E}, \overline{I} \), then we obtain the simplified system

\[ S' = \mu A_1 + \frac{\mu A_2}{1 + I} - \mu S - SI, \]
\[ E' = SI - b_1 E, \]
\[ I' = E - b_2 I, \]

(2.4)

where \( \mu = \mu_1 m/\beta, A_1 = \epsilon m^2 B_1/\beta, A_2 = \epsilon m^2 B_2/\beta, b_1 = (\mu_1 + \epsilon)m/\beta, \) and \( b_2 = (\mu_1 + \alpha + \gamma)m/\beta. \)
3. The Existence and Local Stability of Equilibria

It is obvious that system (2.4) always has the disease-free equilibrium \( E_0(A_1 + A_2, 0, 0) \). Its endemic equilibrium \( E^*(S^*, E^*, I^*) \) is determined by the following equations:

\[
\begin{align*}
\mu A_1 + \frac{\mu A_2}{1 + I} - \mu S - SI &= 0, \\
SI - b_1 I &= 0, \\
E - b_2 I &= 0.
\end{align*}
\]

From the last two equations in (3.1), we have \( S = b_1 b_2 \) and \( E = b_2 I \) for \( I \neq 0 \). Substituting \( S = b_1 b_2 \) into the first equation in (3.1) gives

\[
\mu \left( A_1 + \frac{A_2}{1 + I} \right) = b_1 b_2 (\mu + I),
\]

then \( I^* \) is the positive root of (3.2).

According to the monotonicity of functions at the two sides of (3.2), we know that (3.2) has a unique positive root if \( (A_1 + A_2)/(b_1 b_2) > 1 \) and no positive roots if \( (A_1 + A_2)/(b_1 b_2) \leq 1 \). Therefore, with respect to the existence of equilibria of system (2.4), we have the following theorem.

**Theorem 3.1.** Denote that \( R_0 = (A_1 + A_2)/(b_1 b_2) \). When \( R_0 \leq 1 \), system (2.4) has only the disease-free equilibrium \( E_0(A_1 + A_2, 0, 0) \) on the set \( \Omega \); when \( R_0 > 1 \), besides the disease-free equilibrium \( E_0 \), system (2.4) also has a unique endemic equilibrium \( E^*(S^*, E^*, I^*) \), where \( S^* = b_1 b_2, E^* = b_2 I^* \), and \( I^* \) is determined by (3.2).

With respect to the local stability of equilibria \( E_0 \) and \( E^* \) of system (2.4), we have the following theorem.

**Theorem 3.2.** The disease-free equilibrium \( E_0 \) is locally asymptotically stable as \( R_0 < 1 \) and unstable as \( R_0 > 1 \). The endemic equilibrium \( E^* \) is locally asymptotically stable as it exists.

**Proof.** (i) From the Jacobian matrix of system (2.4) at the disease-free equilibrium \( E_0 \), it is easy to know that the disease-free equilibrium \( E_0 \) is locally asymptotically stable as \( R_0 < 1 \) and unstable as \( R_0 > 1 \).
For the Jacobian matrix of system (2.4) at the endemic equilibrium $E^*$, the characteristic equation is given by $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$, where $a_1 = \mu + I^* + b_1 + b_2$, $a_2 = (\mu + I^*)(b_1 + b_2)$, and $a_3 = I^*[\mu A_2/(1 + I^*)^2 + b_1 b_2]$, then

$$a_1a_2 - a_3 = (\mu + I^* + b_1 + b_2)(\mu + I^*)(b_1 + b_2) - I^* \left[ \frac{\mu A_2}{(1 + I^*)^2} + b_1 b_2 \right]. \tag{3.3}$$

Notice that (3.2) can be rewritten as

$$(\mu + I)(1 + I) = \frac{\mu [(A_1 + A_2) + A_1 I]}{(b_1 b_2)},$$

$$\frac{\mu A_2}{(1 + I)} = b_1 b_2 I + \mu (b_1 b_2 - A_1). \tag{3.4}$$

Using (3.4) gives

$$(1 + I^*)(a_1a_2 - a_3) = \left[ \frac{\mu A_1(b_1 + b_2)}{b_1 b_2} - 2b_1 b_2 \right]I^2 + \frac{\mu (A_1 + A_2)(b_1 + b_2)(\mu + b_1 + b_2)}{b_1 b_2}
\left[\mu (b_1 + b_2) \left( (A_1 + A_2) + A_1 (\mu + b_1 + b_2) \right) - (1 + \mu) b_1 b_2 + \mu A_1 \right] I^*
\overset{\Delta}{=} f_1(I^*). \tag{3.5}$$

On the other hand, (3.2) can become

$$f_2(I) \overset{\Delta}{=} b_1 b_2 I^2 + [b_1 b_2 (1 + \mu) - \mu A_1] I + \mu [b_1 b_2 - (A_1 + A_2)] = 0, \tag{3.6}$$

then

$$f_1(I^*) + 2f_2(I^*) = c_1 I^2 + c_2 I^* + c_3 \mu > 0, \tag{3.7}$$

where $c_1 = \mu A_1(b_1 + b_2)/(b_1 b_2)$, $c_2 = b_1 b_2 (1 + \mu) + \mu [(b_1 + b_2) (A_1 + A_2 + \mu A_1) + A_1 (b_1^2 + b_2^2 + b_1 b_2)]/(b_1 b_2)$, and $c_3 = (A_1 + A_2) [\mu (b_1 + b_2) + b_1^2 + b_2^2] / (b_1 b_2) + 2b_1 b_2$. It follows from $f_2(I^*) = 0$ that $f_1(I^*) > 0$, that is, $a_1 a_2 - a_3 > 0$. Therefore, it follows from Hurwitz criterion that the endemic equilibrium $E^*$ is locally asymptotically stable.

\[\square\]

4. The Extinction and Persistence of Infection

In this section, we will consider the ultimate state of infection; that is, the disease will be whether extinct or persistent ultimately.
When \( R_0 < 1 \), define function \( V_1 = \rho E + I \), where \( \rho \in (1/b_1, b_2/(A_1 + A_2)) \), then the derivative of \( V_1 \) with respect to \( t \) along the solution of (2.4) on the set \( \Omega \) is given by

\[
V_1' = (1 - \rho b_1)E + \left[ \rho (A_1 + A_2) - b_2 \right] I.
\]  

(4.1)

It follows from \( \rho \in (1/b_1, b_2/(A_1 + A_2)) \) that \( 1 - \rho b_1 < 0 \) and \( \rho (A_1 + A_2) - b_2 < 0 \), then there exists a positive number \( \sigma \) such that \( 1 - \rho b_1 < \sigma \rho (R_0 - 1) \) and \( \rho (A_1 + A_2) - b_2 < \sigma (R_0 - 1) \). Therefore, from (4.1) we have

\[
V_1' \leq \sigma (R_0 - 1) V_1, \quad V_1(t) \leq V_1(0) \exp[\sigma (R_0 - 1) t],
\]

where \( V_1(0) = \rho E(0) + I(0) \), therefore, \( \lim_{t \to \infty} V_1(t) = 0 \) for \( R_0 < 1 \); that is, \( \lim_{t \to \infty} E(t) = \lim_{t \to \infty} I(t) = 0 \) as \( R_0 < 1 \). It implies that the disease will be extinct ultimately when \( R_0 < 1 \).

In order to discuss the persistence of the disease, we first introduce some definitions and lemmas.

Assume that \( X \) is a locally compact metric space with metric \( d \), and let \( F \) be a closed subset of \( X \) with the boundary \( \partial F \) and the interior \( int F \). Let \( \pi \) be a semidynamical system defined on \( F \).

We say that \( \pi \) is persistent if, for all \( u \in \text{int} \, F \), \( \lim \inf_{t \to \infty} d(\pi(u, t), \partial F) > 0 \) and that \( \pi \) is uniformly persistent if there is \( \xi > 0 \) such that, for all \( u \in \text{int} \, F \), \( \lim \inf_{t \to \infty} d(\pi(u, t), \partial F) > \xi \).

In [3], Fonda gives a result about persistence in terms of repellers. A subset \( \Sigma \) of \( F \) is said to be a uniform repeller if there is an \( \eta > 0 \) such that, for each \( u \in F \setminus \Sigma \), \( \lim \inf_{t \to \infty} d(\pi(u, t), \Sigma) > \eta \). A semiflow on a closed subset \( F \) of a locally compact metric space is uniformly persistent if the boundary of \( F \) is repelling in a suitable strong sense [9]. The result by Fonda is as follows.

**Lemma 4.1.** Let \( \Sigma \) be a compact subset of \( X \) such that \( X \setminus \Sigma \) is positively invariant. A necessary and sufficient condition for \( \Sigma \) to be a uniform repeller is that there exists a neighborhood \( U \) of \( \Sigma \) and a continuous function \( P : X \to \mathbb{R} \), satisfying

1. \( P(u) = 0 \) if and only if \( u \in \Sigma \),
2. for all \( u \in U \setminus \Sigma \) there is a \( T_u > 0 \) such that \( P(\pi(u, T_u)) > P(u) \).

For any \( u_0 = (S_0, E_0, I_0) \in \Omega \), there is a unique solution \( \pi(u_0, t) = (S, E, I)(t; u_0) \) of system (2.4), which is defined in \( R_+ \) and satisfies \( \pi(u_0, 0) = (S_0, E_0, I_0) \). Since \( \Omega \) is a positively invariant set of system (2.4), then \( \pi(u_0, t) \in \Omega \) for \( t \in R_+ \) and is a semidynamical system in \( \Omega \).

In the following, we will prove that, when \( R_0 > 1 \), \( \Sigma = \{(S, E, I) \in \Sigma : I = 0\} \) is a uniform repeller, which implies that the semidynamical system \( \pi \) is uniformly persistent.

Obviously, \( I(t) > 0 \) for \( t > 0 \) if \( I(0) > 0 \), then \( \Omega \setminus \Sigma \) is invariant to (2.4). Again the set \( \Sigma \) is a compact subset of \( \Omega \).

Let \( P : \Omega \to \mathbb{R} \) be defined by \( P(S, E, I) = I \), and let \( \mathcal{U} = \{(S, E, I) \in \Omega : P(S, E, I) < \eta_1\} \), where \( \eta_1 > 0 \) is small enough so that

\[
\frac{\mu}{\mu + 2\rho \eta_1} \left( A_1 + \frac{A_2}{1 + \eta_1} \right) > b_1 b_2.
\]

(4.2)

Since \( R_0 > 1 \) is equivalent to \( A_1 + A_2 > b_1 b_2 \), then there exists a positive number \( \eta_1 \) such small that inequality (4.2) holds.
Assume that there is \( \overline{u} \in U \) (\( \overline{u} = (\overline{S}, \overline{E}, \overline{I}) \)) such that for each \( t > 0 \) we have \( P(\pi(\overline{u}, t)) < P(\overline{u}) < \eta_1 \), which implies that \( I(t; \overline{u}) < \eta_1 \) for \( t > 0 \). From the first equation in system (2.4) we have

\[
S' \geq \mu \left( A_1 + \frac{A_2}{1 + \eta_1} \right) - (\mu + \eta_1) S, \quad (4.3)
\]

then

\[
\liminf_{t \to \infty} S(t; \overline{u}) \geq \frac{\mu}{\mu + \eta_1} \left( A_1 + \frac{A_2}{1 + \eta_1} \right). \quad (4.4)
\]

So there is a sufficiently large number \( T > 0 \) such that \( S(t; \overline{u}) > \mu [A_1 + A_2/(1 + \eta_1)] / (\mu + 2\eta_1) \) for \( t \geq T \).

Define the auxiliary function \( V_2(t) = (1 - \eta_2) E(t) + b_1 I(t) \), where \( \eta_2 (0 < \eta_2 < 1) \) is a sufficiently small constant so that \( \mu (1 - \eta_2) / (\mu + 2\eta_1) \cdot [A_1 + A_2/(1 + \eta_1)] > b_1 b_2 \). Direct calculation gives the derivative of \( V_2(t) \) along with \( \pi(\overline{u}, t) \) as follows:

\[
V'_2 = b_1 \eta_2 E + [(1 - \eta_2) S - b_1 b_2] I. \quad (4.5)
\]

Then, for \( t \geq T \), we have

\[
V'_2 \geq b_1 \eta_2 E + \left[ \frac{\mu (1 - \eta_2)}{\mu + 2\eta_1} \left( A_1 + \frac{A_2}{1 + \eta_1} \right) - b_1 b_2 \right] I > \sigma V_2, \quad (4.6)
\]

where

\[
\sigma = \min \left\{ \frac{b_1 \eta_2}{1 - \eta_2}, \frac{1}{b_1} \left[ \frac{\mu (1 - \eta_2)}{\mu + 2\eta_1} \left( A_1 + \frac{A_2}{1 + \eta_1} \right) - b_1 b_2 \right] \right\} > 0, \quad (4.7)
\]

therefore, \( \lim_{t \to \infty} V_2(t) = +\infty \).

On the other hand, the boundedness of the solution of (2.1) implies that of \( V_2(t) \) on the set \( \Omega \). It implies that the assumption above is not true. Therefore, the above proof shows that, for each \( u \in \Omega \setminus \Sigma \) with \( u \) belonging to a suitably small neighborhood of \( \Sigma \), there is some \( T_u \) such that \( P(\pi(u, T_u)) > P(u) \). Therefore, it follows from Lemma 4.1 that \( \Sigma = \{(S, E, I) \in \Sigma : I = 0\} \) is a uniform repeller when \( R_0 > 1 \); that is, the infection is uniformly persistent. So we have the following theorem.

**Theorem 4.2.** For system (2.4), the infection will be extinct when \( R_0 < 1 \) and persistent when \( R_0 > 1 \).
5. Conclusion and Discussion

In Sections 3 and 4, for system (2.4) we investigated the qualitative behavior and obtained the threshold $R_0$ determining the persistence of infection. Corresponding to the original model (2.1), the basic reproduction number is $R_0 = (\beta_1 + \beta_2)e / [(\mu_1 + \epsilon)(\mu_1 + \alpha + \gamma)]$. According to the results in Sections 3 and 4, model (2.1) only has the disease-free equilibrium which is globally stable when $R_0 < 1$; it implies that the disease is extinct ultimately; when $R_0 > 1$, model (2.1) has a unique endemic equilibrium which is locally asymptotically stable and the disease persists in the population. Since the expression of $R_0$ here is independent of the parameter $m$, then this shows that this parameter has no effect on the persistence of disease, but it can affect the strength of spread of disease according to Theorem 3.1.

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References
