Global Stability of A Tuberculosis Model with Vertical Transmission

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Abstract

This paper considers an SEIT epidemic model that incorporates proportion recruitment and with vertical transmission. By means of Lyapunov function and LaSalle’s invariant set theorem, we proved the global asymptotical stable results of the disease-free equilibrium and the endemic equilibrium with the help of numerical simulations.

1. Introduction

Tuberculosis (TB) is caused by Mycobacterium tuberculosis. The disease is most commonly transmitted from a person suffering from infectious (active)TB to other persons by infected droplets created when the person with active TB coughs or sneezes. Among generally healthy persons, infection with TB is highly likely to be asymptomatic. Data from a variety of sources suggest that the life time risk of developing clinically evident TB after being infected is approximately 10, with 90% likelihood of the infection remaining latent. Mycobacterium tuberculosis is the infectious agent primarily responsible for TB in humans. TB remains a major cause of disability and death in many areas of the world, see [2], especially among groups with a high prevalence of HIV or those living in crowded conditions in [1]. Effective treatment is available for both latent and infectious individuals. If untreated, about half of TB patients die within five years of becoming infective. Several ordinary differential equation models for TB have been formulated and studied; (see [5]-[11]). The common models include classes of susceptible, exposed, infective and Treated individuals, and hence are known as SEIT models. Most TB models assume that all immigrants and newborns are susceptible with the disease transmitted horizontally according to standard or mass action incidence. Despite its sociological and historical importance, the study of the spread of TB using statistical and mathematical models has not received enough attention. In fact we have observed only an extremely limited use of mathematical models in the study of the transmission dynamics of TB in human populations (personal communication with Blower during the meeting of Mathematical Modeling of TB in 1995).

2. Model formulation and equilibrium discussion

The established model of infectious diseases focuses on the transmitting features of TB, the host population is partitioned into four compartments, the susceptible, exposed(latent), infectious and Treated individuals, with sizes denoted by S,E,I,T, respectively. The host total population N = S + E + I + T. Typically after the initial infection, a host stays in a latent period before becoming infectious. The common TB models are not consider proportion recruitment and vertical transmission. The SEIT model of the paper is described by the following system of differential equations

\[
\begin{align*}
S' &= bN - \beta cS \frac{I}{N} - pbE - qbI - \mu S \\
E' &= \beta cS \frac{I}{N} - (\mu + k + r_1)E + pbE + qbI \\
I' &= kE - (\mu + d + r_2)I \\
T' &= r_1 E + r_2 I - \mu T
\end{align*}
\]

where the positive parameters \( \mu \) is the per-capita natural death rate; exponential birth with rate constant \( b \); \( \beta \) is the probabilities that susceptible become infected by one infectious individual per contact per unit of time; \( c \) is the per-capita contact rate; \( d \) is the per-capita disease induced death rate; The parameter \( k \) is the rate at which an individual leaves the latent class by becoming infectious; \( r_1 \) and \( r_2 \) denote the treatment rates for latent and infectious individuals, respectively. we assume that a fraction \( p \) and a fraction \( q \) of the offspring from the exposed and the infectious classes, respectively, are born into the exposed class \( E \). Consequently, the birth flux into the exposed class is given by \( pbE + qbI \). Thus the total population size \( N \) implies

\[ N' = (b - \mu)N - dI \]
Let \( s = \frac{s}{N}, e = \frac{e}{N}, i = \frac{i}{N}, t = \frac{t}{N} \) denote the fractions of the classes \( S, E, I, T \) in the population, respectively. It is easy to verify that \( s, e, i, t \) satisfy the system of differential equations

\[
\begin{align*}
s' &= b(1-s) - \beta csi - pbe - qbi + dis \\
 e' &= \beta csi - (b+k+r_1)e + pbe + qbi + die \\
 i' &= ke - (b+d+r_2)i + di^2 \\
 t' &= r_1e + r_2i - bt + dit 
\end{align*}
\]

subject to the restriction \( s + e + i + t = 1 \). Observe that the variable \( t \) does not appear in the first three equations of (2.2). This allows us to attack (2.2) by studying the subsystem

\[
\begin{align*}
s' &= b(1-s) - \beta csi - pbe - qbi + dis \\
 e' &= \beta csi - (b+k+r_1)e + pbe + qbi + die \\
 i' &= ke - (b+d+r_2)i + di^2 
\end{align*}
\]

(2.2)

From biological considerations, we study (2.3) in the closed set

\[ A = \{(s,e,i) \in R^3_+ : 0 \leq s + e + i \leq 1 \} \]

where \( R^3_+ \) denotes the non-negative cone of \( R^3 \) including its lower dimensional faces. It can be verified that \( A \) is positively invariant with respect to (2.3).

System (2.3) has two possible equilibria in \( A \): the disease-free equilibrium \( P_0 = (1,0,0) \) and an endemic equilibrium \( P^* = (s^*, e^*, i^*) \), where

\[ \sigma = \frac{\beta ck}{(b+k+r_1-pb)(b+r_2+d) - qbk} \]

is the modified contact number of the system (2.3).

**Theorem 2.1.** If \( \sigma \leq 1 \), the disease-free equilibrium \( P_0 \) of the system (2.3) is globally asymptotically stable in \( A \). It is unstable if \( \sigma > 1 \), and the solutions of (2.3) starting sufficiently close to \( P_0 \) in \( A \) move away from \( P_0 \) except those starting on the invariant s-axis approach \( P_0 \) along this axis.

The following lemma will be used in the proof of Theorem 2.1.

**Lemma 2.1.** Let \( \Delta = \{(x,y) \in R^2_+ : 0 \leq x + y \leq 1 \} \) and

\[ h(x,y) = (a_1 - b_1)x + (c_1 - b_2)y + b_1. \]

Then, for any positive constants \( a_1, b_1 \) and \( c_1 \),

\[ \max_{(x,y) \in \Delta} h(x,y) = \max\{a_1, b_1, c_1\}. \]

**proof.** The affinity of \( h \) implies that its maximum in the closed set \( \Delta \) is achieved at the extremal points of the boundary \( \partial \Delta \). The proof is now a straightforward evaluation of \( h \) on the three vertices of the triangular set \( \Delta \).

**proof of Theorem 2.1.** Set \( L = ke + (k+b+r_1-pb)i \). Then

\[
L' = ke' + (k+b+r_1-pb)i' = k\beta cs - k(b+k+r_1)e + kpbe + kqbi + kdie \\
+ (k+b+r_1-pb)(ke - (b+p+2d) + di^2) \\
= k\beta cskqbi + kdie - (k+b+r_1-pb)(b+r_2+d)i \\
+ d(k+b+r_1-pb)i^2 \\
= i[k\beta cs + kqb + kde - (k+b+r_1-pb)(b+r_2+d)] \\
+ d(k+b+r_1-pb)i \\
\leq i[k\beta c(1-e-i) + kde + d(k+b+r_1-pb)i] \\
+ kqb - (k+b+r_1-pb)(b+r_2+d)] \\
= i[h(e,i) + kqb - (k+b+r_1-pb)(b+r_2+d)] \\
\]

(2.4)

where \( h(e,i) = (kd-k\beta c)e + [d(k+b+r_1-pb) - k\beta c]i \).

Applying Lemma 2.1 to \( h(e,i) \) leads to

\[ L' \leq \max\{kd, k\beta c, d(k+b+r_1-pb)\} \\
-(k+b+r_1-pb)(b+r_2+d) + kqb \]

\[ \leq 0 \quad \text{if} \quad \sigma \leq 1 \]

if \( L' = 0 \) and \( i \neq 0 \), then

\[ \max\{kd, k\beta c, d(k+b+r_1-pb)\} \\
= kqb - (k+b+r_1-pb)(b+r_2+d) \]

and (2.4) becomes an equality. Thus, \( L' = 0 \) only if either (1) \( i = 0 \), or (2) \( \sigma = 1 \) and \( s + e + i = 1 \). The maximum invariant set in \( \{(s,e,i) \in A : L' = 0\} \) is the singleton \( P_0 \). The global stability of \( P_0 \) when \( \sigma \leq 1 \) follows from LaSalle’s Invariance Principle ([12], Chapter 2, Theorem 6.4).

If \( \sigma > 1 \), then \( L' > 0 \) for \( s \) sufficiently close to 1 except when \( e = i = 0 \). Solutions starting sufficiently close to \( P_0 \) leave a neighborhood of \( P_0 \) except those on the invariant s-axis, on which (2.3) reduces to \( s' = b - bs \) and thus \( s(t) \to 1 \), as \( t \to \infty \). This establishes the theorem.

Theorem 2.1 completely determines the global dynamics of (2.3) in \( A \) for the case \( \sigma \leq 1 \). Its epidemiological implication is that the infected fraction (the sum of the latent and the infectious fractions) of the population vanishes in time so the disease dies out. In the rest of this section, we show that the disease persists when \( \sigma > 1 \). We say the disease is endemic if the infected fraction of the population persists above a certain positive level for sufficiently large time. The endemicity of disease can be well captured and analyzed through the notion of uniform persistence. System (2.3) is said to be uniformly persistent (see [14-16]) if there exists a constant \( 0 < \xi < 1 \) such that any solution \( (s(t), e(t), i(t)) \) with \( (s(0), e(0), i(0)) \in A \) satisfies

\[ \min\{ \lim_{t \to \infty} \inf s(t), \lim_{t \to \infty} \inf e(t), \lim_{t \to \infty} \inf i(t) \} \geq \xi. \] (2.5)

The disease is endemic if (2.3) is uniformly persistent. In this case, both the infective and the latent fractions persist.
above a certain positive level. Weaker notions of persistence have been defined and used in the literature of population dynamics (see [16]). One may choose to define endemicity of the disease using one of the weaker notions of persistence. However, as the following result shows, persistence of (2.3) in any reasonable sense is equivalent to the uniform persistence defined above.

**Proposition 2.3.** System (2.3) is uniformly persistent in \( \mathring{A} \) if and only if \( \sigma > 1 \).

**Proof.** The necessity of \( \sigma > 1 \) follows from Theorem 2.1 and the fact that the asymptotic stability of \( P_0 \) precludes any kind of persistence. The sufficiency of the condition \( \sigma > 1 \) follows from a uniform persistence result, Theorem 4.3, in [17]. To demonstrate that (2.3) satisfies all the conditions of Theorem 4.3 in [17] when \( \sigma > 1 \), choose \( X = R^3 \) and \( E = A \). The maximal invariant set \( N \) on the boundary \( \partial A \) is the singleton \{ \( P_0 \) \} and is isolated. Thus, the hypothesis (H) of [17] holds for (2.3). The proposition is proved by observing that, in the setting of (2.3), the necessary and sufficient condition for uniform persistence in Theorem 4.3 of [17] is equivalent to \( P_0 \) being unstable.

**Theorem 2.3.** If \( \sigma \geq 1 \), the endemic equilibrium \( P^* \) of the system (2.3) is local asymptotically stable in \( A \). We assume that \( b > d \).

**proof.**

\[
J(P^*) = \begin{pmatrix}
-b - \beta c s^* + d i^* & -p b & \Phi_1 \\
\beta c s^* & -(b + k + r_1) + p b + d i^* & \Phi_2 \\
0 & k & \Phi_3
\end{pmatrix}
\]

\[\Phi_1 = \beta c s^* - q b + d s^* \]
\[\Phi_2 = \beta c s^* + q b + d e^* \]
\[\Phi_3 = -(b + r_2 + d) + 2 d i^* \]

Its characteristic equation is \( \text{det}(\lambda I - J(P^*)) = 0 \), where \( I \) is the unit matrix and

\[
s^* = \frac{(b + k + r_1)e^* - p b e^* - q b i^* - d i^* e^*}{\beta c i^*},
\]

\[
e^* = \frac{(b + r_2 + d)i^* - d i^2}{k}.
\]

So the characteristic equation become to

\[
\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0,
\]

where \( a_i > 0, i = 1, 2, 3 \). then, we use Descartes Rule of Signs to conclude that Eq. (2.7) has (i) no positive root and either (ii) one negative root and two complex roots with negative real part or (iii) three negative roots, we know that the endemic equilibrium \( P^* \) of the system (2.3) is local asymptotically stable in \( A \).

### 3. Numerical results

![Graph](image-url)
Fig. 1. In Figure (a), we show the case report with the outcome of the system (2.3) when the modified contact number $\sigma < 1$. The parameters are chosen as $b = 0.71$, $\beta = 13$, $c = 1$, $d = 0.143$, $k = 0.6$, $r_1 = 2$, $r_2 = 2$, $p = 0.1$ and $q = 0.3$. $\sigma = 0.9988$. It suggests the disease-free equilibrium $P_0$ of the system (2.3) is globally asymptotically stable in $A$ when the $\sigma < 1$. In Figure (b)-(c), we show the case report with the outcome of the system (2.3) when the modified contact number $\sigma > 1$. $b = 0.3$, $\beta = 13$, $c = 1$, $d = 0.143$, $k = 0.6$, $r_1 = 2$, $r_2 = 2$, $p = 0.1$ and $q = 0.3$. $\sigma = 1.3354$. It suggests the disease-free equilibrium $P_0$ of the system (2.3) is globally asymptotically stable in $A$ when the $\sigma < 1$.

References


