A Discrete Tuberculosis Model with Two Different Infectious Compartments

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***Abstract*—A discrete tuberculosis model with different infectious compartments is formulated and studied. The basic reproduction number, R_0 of the model is defined. The global stability of the disease-free equilibrium is proved if $R_0 < 1$. The existence of endemic equilibrium and the persistence of TB is established if $R_0 > 1$. The main attention is paid on the influence of the infectivity proportion on the case number. The numerical simulations show that the infection cases increase as p increase.**

Keywords-Discrete epidemic model, Smear-positive, Smear-negative, Tuberculosis,

I. INTRODUCTION

Tuberculosis (TB) is a chronic bacterial infection, and it causes more deaths worldwide than any other infectious diseases. TB is a great threat to public heath. Someone in the world is newly infected with TB bacilli every second. Overall, one-third of the world's population is currently infected with the TB bacillus[1]. There were 9.3 million new TB cases in 2007, and 1.8 million people died from TB in 2007. Although TB annual incidence rates have peaked globally in 2003-2004, the total number of deaths and cases keep increasing due to population growth[2], especially in Asia and Africa[12].

TB infection remains a serious public health challenge in China. Much attention has been paid to TB control in recently years. There were four large scale national sampling survey of TB epidemiology in China. The forth survey was carried in 2000. The sampling population in that survey was 365,097. The standardized prevalence of active tuberculosis was 367/100,000. The annual reduction rates between 1979 and 2000 for active tuberculosis was 4.5%[3].

The pulmonary TB patients are classified into smear-negative pulmonary TB and smear-positive pulmonary TB, according to their test results, respectively. The smear-positive pulmonary TB case is more infectious than smear-negative pulmonary TB case. Smear-positive pulmonary TB patients are the main therapeutic in China though smear-negative pulmonary TB accounts for about 50%-70% of pulmonary TB patients. Researches have shown that 20.8%-64% of smear-negative pulmonary TB patients will become smear-positive pulmonary TB patients within one to five years, if they do not get treatment, becoming new infectious source[10]. Therefore, smear-negative pulmonary TB patients should be considered in TB control.

Discrete models are the good choice for the description of the epidemic process. Most reported infection or illness incidence data are discrete in nature (e.g., daily or weekly illness rates), and discrete-time models are appropriate to analyze such data in a natural fashion[9]. For these reasons, there is increasing interest in discrete-time models for describing epidemics [4,5,7,8]. Zhou and Cao had discussed discrete-time *SEIR* models with application to TB infection[12]. We improve those models by further dividing the infectious group into smear-negative compartment, $I_1(t)$, and smear-positive compartment, $I_2(t)$, and assume that the smear-negative patients may become smear-positive ones.

The paper is organized as follows. The *SEIR* TB transmission model is formulated, and the basic reproductive number of the model is defined in section 2. The dynamics of model is studied in section 3. Conclusion and discussion are presented in the last section.

II. THE MODEL AND THE BASIC REPRODUCTION NUMBER

Let us consider TB transmission in a population. Following the framework of the *SEIR* epidemic model, we group the population into four epidemiological compartments: the susceptibles, the exposed, the infectious, and the treated. The infectious is divided into the smear-negative compartment, with less infectivity, and smear-positive compartment, with more infectivity. Let $S(t)$, $E(t)$, $I_1(t)$, $I_2(t)$, and $R(t)$ denote the number of individuals in the susceptible, exposed, sputum smear-negative, sputum smear-positive and treated compartments at time t, respectively. The discrete TB model with two infectious compartments is

$$\begin{aligned}
S(t+1) &= S(t) + \Lambda - (\beta_1 I_1(t) + \beta_2 I_2(t))S(t) - \mu S(t),\\
E(t+1) &= E(t) + (\beta_1 I_1(t) + \beta_2 I_2(t))S(t) - (\mu+\alpha)E(t),\\
I_1(t+1) &= I_1(t) + p\alpha E(t) - (\mu+\gamma+k)I_1(t),\\
I_2(t+1) &= I_2(t) + (1-p)\alpha E(t) + kI_1(t) - (\mu+\gamma)I_2(t),\\
R(t+1) &= R(t) + \gamma(I_1(t) + I_2(t)) - \mu R(t),
\end{aligned} \tag{1}$$

where Λ is the birth rate, μ is the death rate, β_i is transmission rate of infectious individuals $I_i(t)(i=1,2)$, α is the transfer rate from exposed group to infectious groups, k is the

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transfer rate from smear-negative $I_1(t)$ to smear-positive $I_2(t)$, γ is the treated rate., p is fraction of active tuberculosis into smear-negative infection $I_1(t)$.

The natural requirement of epidemiological interpretation is that all parameters are positive, $0<\mu+\gamma+k<1, 0<p<1$, $\mu+\alpha<1, \beta_1<\beta_2$, and $\mu+(\beta_1 I_1(t)+\beta_2 I_2(t))<1$. A sufficient condition for last inequality is that $\mu+\frac{\Lambda}{\mu}(\beta_1+\beta_2)<1$.

Those conditions ensure that the solution of (1) is nonnegative if it starts from a nonnegative point.

Let $N(t)=S(t)+E(t)+I_1(t)+I_2(t)+R(t)$, denote the total number of the population at time t, and $N(t)$ satisfies

$$N(t)=\Lambda+(1-\mu)N(t-1)=\Lambda\frac{1-(1-\mu)^t}{\mu}+(1-\mu)^t N(0) \quad (2)$$

The fact $\mu>0$ and Eq.(2) imply that $\lim_{t\to\infty} N(t)=\Lambda/\mu$. $N^*=\Lambda/\mu$ is unique equilibrium of (2), and it is globally asymptotically stable.

The definition of $N(t)$ and the last equation of (2) imply that the domain

$$\Omega:=\{(S,E,I_1,I_2,R)\in R_+^5 : S+E+I_1+I_2+R\le \Lambda/\mu\} \quad (3)$$

is a compact, positively invariant set of (1). Ω is a global compact attractor of (1) since it attracts all positive orbits with initial value $(S^0,E^0,I_1^0,I_2^0,R)\in R_+^5$.

Following the procedure of defining the basic reproduction number[6], we rearrange equations in (1) to have

$$\tilde{x}(t+1)=\tilde{F}(\tilde{x}(t))+\tilde{T}(\tilde{x}(t)) \quad (4)$$

Let $\tilde{x}=(E,I_1,I_2,S,R)^T$, we have

$$\tilde{F}(\tilde{x})=\begin{pmatrix}(\beta_1 I_1(t)+\beta_2 I_2(t))S(t)\\ p\alpha E(t)\\ (1-p)\alpha E(t)\\ 0\\ 0\end{pmatrix}, \text{ and}$$

$$\tilde{T}(\tilde{x})=\begin{pmatrix}(1-\mu-\alpha)E(t)\\ (1-\mu-\gamma-k)I_1(t)\\ kI_1(t)+(1-\mu-\gamma)I_2(t)\\ \Lambda+(1-\mu-\beta_1 I_1(t)-\beta_2 I_2(t))S(t)\\ \gamma(I_1(t)+I_2(t))+(1-\mu)R(t)\end{pmatrix}.$$

Corresponding to the unique disease-free equilibrium, $p^0=(\Lambda/\mu,0,0,0,0)$ of model (1), (4) has a equilibrium $\tilde{x}^0=(0,0,0,\Lambda/\mu,0)$. The linearization at the disease-free equilibrium $\tilde{x}^0$ yields that

$$F=\begin{pmatrix}0 & \Lambda\beta_1/\mu & \Lambda\beta_2/\mu\\ p\alpha & 0 & 0\\ (1-p)\alpha & 0 & 0\end{pmatrix}, \quad C=\begin{pmatrix}1-\mu & 0\\ 0 & 1-\mu\end{pmatrix},$$

and $T=\begin{pmatrix}1-\mu-\alpha & 0 & 0\\ 0 & 1-\mu-\gamma-k & 0\\ 0 & k & 1-\mu-\gamma\end{pmatrix}$. The calculation yields that

$$F(I-T)^{-1}=\begin{pmatrix}0 & \frac{\Lambda\beta_1}{\mu(\mu+\gamma+k)}+\frac{\Lambda\beta_2 k}{\mu(\mu+\gamma)(\mu+\gamma+k)} & \frac{\Lambda\beta_2}{\mu(\mu+\gamma)}\\ p\alpha/(\mu+\alpha) & 0 & 0\\ (1-p)\alpha/(\mu+\alpha) & 0 & 0\end{pmatrix},$$

where I is the identical matrix of 3×3. (1), or the equivalent model (4), satisfies those assumptions in [6]. Thus, the basic reproduction number R_0 of (1) is given by

$$R=\rho(F(I-T)^{-1})=\sqrt{\frac{\Lambda}{\mu}\frac{\alpha(\beta_1 p(\mu+\gamma)+\beta_2(1-p)(\mu+\gamma)+\beta_2 k)}{(\mu+\alpha)(\mu+\gamma)(\mu+\gamma+k)}}$$

The expression of the derivative of R_0 with respect to β_1

$$\frac{\partial R_0}{\partial\beta_1}=\left(\frac{\Lambda\alpha(\beta_1 p(\mu+\gamma)+\beta_2(1-p)(\mu+\gamma)+\beta_2 k)}{\mu(\mu+\alpha)(\mu+\gamma)(\mu+\gamma+k)}\right)^{-1/2}\frac{\Lambda p\alpha}{2\mu(\mu+\alpha)(\mu+\gamma+k)}$$

implies that $\partial R_0/\partial\beta_1>0$. Similarly, $\partial R_0/\partial\beta_2>0$. R_0 is an increasing function of $\beta_i (i=1,2)$. It is important to reduce the transmission rate in controlling TB. In addition, by numerical simulation, we show the relationship of the number of infection and the parameter p, taking $\mu=0.000563$, $\Lambda=0.001016$, $\gamma=1/6$, $k=0.01178$, , $\alpha=0.00025$, and $\beta_2=0.09$. We assume that the total number of population is 10000. According to the proportion of different compartments in population from the result in the forth survey in 2000, we chose the initial value $S(1)=6293$, $E(1)=3597$, $I_1(1)=3$, $I_2(1)=2$, and $R(1)=105$. Unit time interval is one month. $R_0=0.47<1$ as $p=0.4$, and $R_0=0.44<1$ as $p=0.7$. The numerical simulation shows that a interesting phenomenon(see Fig.1), that the number of infection increase s as p increases. This phenomenon suggests that we should also pay more attention to treat smear-negative pulmonary TB patients in the future.

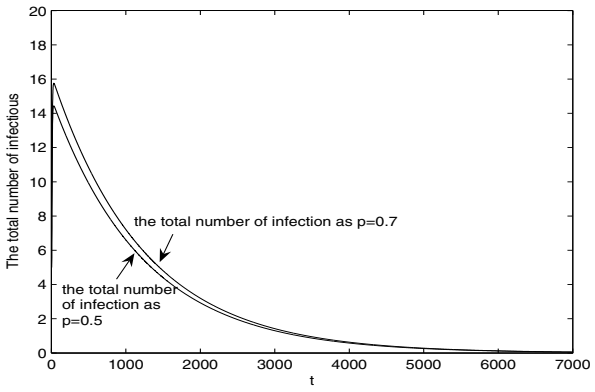

Figure 1. The number of infection for different p.

III. THE DYNAMICS OF THE MODEL

We discuss the stability of the disease-free equilibrium, the existence of endemic equilibrium of (1), and the persistence of TB in population.

Theorem 1 the disease-free equilibrium p^0 of (1) is globally asymptotically stable if $R_0 < 1$, and the disease-free equilibrium p^0 is unstable if $R_0 > 1$.

Proof. By use of Theorem 2.1 in [6], we know that p^0 is locally asymptotically stable if $R_0 < 1$. and unstable if $R_0 > 1$.

We focus on the global dynamics of the disease-free equilibrium p^0 of (1). $S_1(t+1) = \Lambda + (1-\mu)S_1(t)$ has a globally attractive solution $S_* = \Lambda/\mu$ in R_+. For any $\xi > 0$, there exists a $T > 0$, such that

$$S(t) < \xi + \Lambda/\mu, t > T.$$

From the fact that $R_0 = \rho(F(I-T)^{-1}) < 1$, we can choose ξ small enough, such that $\rho(F_\xi(I-T)^{-1}) < 1$,

where $F_\xi = \begin{pmatrix} 0 & \Lambda\beta_1/\mu + \beta_1\xi & \Lambda\beta_2/\mu + \beta_2\xi \\ p\alpha & 0 & 0 \\ (1-p)\alpha & 0 & 0 \end{pmatrix}$.

The linearized system

$$\begin{pmatrix} \tilde{E}(t+1) \\ \tilde{I}_1(t+1) \\ \tilde{I}_2(t+1) \end{pmatrix} = \begin{pmatrix} 1-\mu-\alpha & \frac{\Lambda\beta_1}{\mu} + \beta_1\xi & \frac{\Lambda\beta_2}{\mu} + \beta_2\xi \\ p\alpha & 1-\mu-\gamma-k & 0 \\ (1-p)\alpha & k & 1-\mu-\gamma \end{pmatrix} \begin{pmatrix} \tilde{E}(t) \\ \tilde{I}_1(t) \\ \tilde{I}_2(t) \end{pmatrix}$$

leads to $\begin{pmatrix} \tilde{E}(t+1) \\ \tilde{I}_1(t+1) \\ \tilde{I}_2(t+1) \end{pmatrix} = (F_\xi + T) \begin{pmatrix} \tilde{E}(t) \\ \tilde{I}_1(t) \\ \tilde{I}_2(t) \end{pmatrix} = (F_\xi + T)^{t+1} \begin{pmatrix} \tilde{E}(0) \\ \tilde{I}_1(0) \\ \tilde{I}_2(0) \end{pmatrix}$.

From those equalities and the fact $\rho(F_\xi(I-T)^{-1}) < 1$ follow that $\lim_{t\to\infty} \tilde{E}(t) = \lim_{t\to\infty} \tilde{I}_1(t) = \lim_{t\to\infty} \tilde{I}_2(t) = 0$ .The comparison theorem implies that $\lim_{t\to\infty} E(t) = \lim_{t\to\infty} I_1(t) = \lim_{t\to\infty} I_2(t) = 0$. For any $\xi > 0$, there exists a $T > 0$, such that $0 < I_i(t) < \xi$ ($i = 1, 2$) for all $t > T$ .From the first equation of (1), we have

$$\begin{aligned} &S(t+T+1) = \Lambda + (1-\mu-\beta_1 I_1(t+T) - \beta_2 I_2(t+T))S(t+T) \\ &\geq \Lambda + (1-\mu-\beta_1\xi-\beta_2\xi)S(t+T) \\ &= \Lambda[1+(1-\mu-\beta_1\xi-\beta_2\xi)] + (1-\mu-\beta_1\xi-\beta_2\xi)* \\ &[1-\mu-\beta_1 I_1(t+T-1) - \beta_2 I_2(t+T-1)]S(t+T-1) \\ &\geq \Lambda[1+(1-\mu-\beta_1\xi-\beta_2\xi)] + (1-\mu-\beta_1\xi-\beta_2\xi)^2 S(t+T-1) \\ &\geq \cdots \geq \Lambda + \sum_{i=0}^{t} (1-\mu-\beta_1\xi-\beta_2\xi)^i + (1-\mu-\beta_1\xi-\beta_2\xi)^{t+1} S(T) \\ &= \Lambda \frac{1-(1-\mu-\beta_1\xi-\beta_2\xi)^{t+1}}{\mu+\beta_1\xi+\beta_2\xi} + (1-\mu-\beta_1\xi-\beta_2\xi)^{t+1} S(T) \end{aligned} \quad (5)$$

Taking t tend to infinity and ξ tend to zero, we can obtain $S(t)$ approaches Λ/μ since $S(t) \leq \Lambda/\mu$ and (5). Further $\lim_{t\to\infty} R(t) = \lim_{t\to\infty}(N(t) - S(t) - E(t) - I_1(t) - I_2(t)) = 0$. The disease-free equilibrium p^0 is globally asymptotically stable. □

When $R_0 > 1$, model (1) has unique positive equilibrium $p^* = (S^*, E^*, I_1^*, I_2^*, R^*)$ with $S^* = \frac{(\mu+\alpha)E^*}{\beta_1 I_1^* + \beta_2 I_2^*}$, $I_1^* = \frac{p\alpha E^*}{\mu+\gamma+k}$,

$I_2^* = \frac{(1-p)\alpha E^* + kI_1^*}{\mu+\gamma}$, and $R^* = \frac{\gamma(I_1^* + I_2^*)}{\mu}$, where

$$E^* = \frac{\Lambda}{\mu+\alpha} - \frac{\mu^2(\mu+\gamma)(\mu+\gamma+k)}{\Lambda\alpha(\beta_1 p(\mu+\gamma) + \beta_2(1-p)(\mu+\gamma) + \beta_2 k)}.$$

We can not get better theoretical result on the stability of the endemic equilibrium due to the higher dimension of model (1) and the complex expression of the endemic equilibrium. The numerical simulation gives the hint that the endemic equilibrium may be globally asymptotically stable as $R_0 > 1$. After taking $\Lambda = 1/1000$, $\mu = 1/2000$, $\beta_1 = 0.4$, $\beta_2 = 0.9$, $\alpha = 0.00025$, $k = 0.01178$, $\gamma = 1/6$, and $p = 0.7$, we get that $R_0 = 1.51172 > 1$ .We selected two different sets of initial values, and unit time interval is one month. The simulation shows that the endemic equilibrium of (1) may be globally asymptotically stable(see Fig 2).

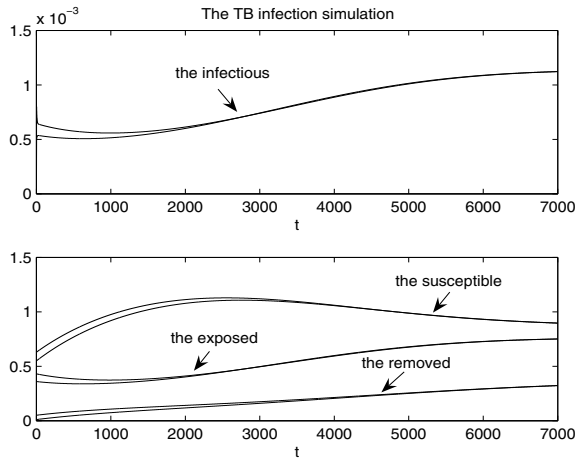

Figure 2. The globally stable of positive equilibrium of (1)

Next, we discuss the persistence of model (1).

Theorem 2 System (1) is uniformly persistent if $R_0 > 1$.

Proof. We define $X = \{(S, E, I_1, I_2, R) \in R_+^5\}$,

$X_0 = \{(S, E, I_1, I_2, R) \in R_+^5, E > 0, I_1 > 0, I_2 > 0\}$,

$\partial X_0 = X \setminus X_0$. Let $x(t) = (S(t), E(t), I_1(t), I_2(t), R(t))^T$, and

$f(x) = (f(x)_1, f_2(x), f_3(x), f_4(x), f_5(x))^T$ with

$f_1(x(t)) = S(t+1)$, $f_2(x(t)) = E(t+1)$, $f_3(x(t)) = I_1(t+1)$,

$f_4(x(t)) = I_2(t+1)$, and $f_5(x(t)) = R(t+1)$. Clearly,

$f(X_0) \subset X_0$. (3) implies that Ω is a global attractor of f . Set

$$M_\partial = \left\{(S^0, E^0, I_1^0, I_2^0, R^0) \in \partial X_0 : f^n(x) \in \partial X_0, \forall n \geq 0\right\} ,$$

where $x(t) = (S(t), E(t), I_1(t), I_2(t), R(t))^T$ with initial value $(S^0, E^0, I_1^0, I_2^0, R)^T$. We claim that

$$M_\partial = \{(S, 0, 0, 0, R), S > 0, R \geq 0\} .$$

In fact, for $\forall (S^0, E^0, I_1^0, I_2^0, R^0) \in \partial X_0$ with $E^0 > 0$, $I_i^0 > 0$, $(i = 1, 2)$, if $I_i^0 > 0 (i = 1, 2)$, $\mu + (\beta_1 I_1(t) + \beta_2 I_2(t)) < 1$ implies that $S(t) > 0$ for $\forall t > 0$. Further, $E(t) > 0$, $I_1(t) > 0$ and $I_2(t) > 0$ for $\forall t > 0$ with $E^0 > 0$. If $E^0 > 0$, $I_1^0 > 0$ and $I_2^0 > 0$, it is clear that $E(t) > 0$, $I_1(t) > 0$ and $I_2(t) > 0$ for $\forall t > 0$. Therefore, we have, $f^n(x(t)) \notin \partial X_0$ for $\forall n > 0$. Clearly, there is exactly one fixed point $p^0 = (\Lambda/\mu, 0, 0, 0, 0)$ in M_∂ . It follows that $A_\partial = \Omega \bigcap M_\partial = M_\partial$ and A_∂ has Morse decomposition $M_1 = p^0$.

Now, let us prove that $\limsup_{t \to \infty} E(t) = U > 0$. Otherwise, $U = 0$, and for any given number ε (ε is sufficiently small), there will exist a large positive integer K such that $E(t) < \varepsilon$ for all $t > \varepsilon$. Denote $B = \max\left\{(1-\mu-\gamma-k)^{K+1}, (1-\mu-\gamma)^K\right\}$, where we can chose K large enough such that $0 < B < \varepsilon$. Then we have

$$\begin{aligned}
&I_1(t+2K+1) = p\alpha E(t+2K) + (1-\mu-\gamma-k) I_1(t+2K) \\
&\leq p\alpha\varepsilon + (1-\mu-\gamma-k) I_1(t+2K) \\
&= p\alpha\varepsilon + (1-\mu-\gamma-k)[p\alpha E(t+2K-1) + (1-\mu-\gamma-k) I_1(t+2K-1)] \\
&\leq p\alpha\varepsilon[1 + (1-\mu-\gamma-k)] + (1-\mu-\gamma-k)^2 I_1(t+2K-1) \\
&\leq \cdots \leq p\alpha\varepsilon \sum_{i=0}^{K} (1-\mu-\gamma-k)^i + (1-\mu-\gamma-k)^{K+1} I_1(t+K) \\
&= p\alpha\varepsilon\{[1-(1-\mu-\gamma-k)^{K+1}]/(\mu+\gamma+k)\} + (1-\mu-\gamma-k)^{K+1} I_1(t+K) \\
&\leq p\alpha\varepsilon/(\mu+\gamma+k) + \varepsilon\Lambda/\mu .
\end{aligned} \tag{6}$$

Further, we have

$$\begin{aligned}
&I_2(t+3K+1) = (1-p)\alpha E(t+3K) + k I_1(t+3K) + (1-\mu-\gamma) I_2(t+3K) \\
&\leq (1-p)\alpha\varepsilon + k[p\alpha\varepsilon/(\mu+\gamma+k) + \varepsilon\Lambda/\mu] + (1-\mu-\gamma) I_2(t+3K) \\
&= (1-p)\alpha\varepsilon + k[p\alpha\varepsilon/(\mu+\gamma+k) + \varepsilon\Lambda/\mu] + (1-\mu-\gamma)(1-p)\alpha E(t+3K-1) \\
&+ (1-\mu-\gamma)[k I_1(t+3K-1) + (1-\mu-\gamma) I_2(t+3K-1)] \\
&\leq \cdots \leq \{(1-p)\alpha\varepsilon + k[p\alpha\varepsilon/(\mu+\gamma+k) + \varepsilon\Lambda/\mu]\} \\
&\sum_{i=0}^{K-1} (1-\mu-\gamma)^i + (1-\mu-\gamma)^K I_2(t+2K+1) \\
&= \{(1-p)\alpha\varepsilon + k[p\alpha\varepsilon/(\mu+\gamma+k) + \varepsilon\Lambda/\mu]\} \\
&\{[1-(1-\mu-\gamma)^K]/(\mu+\gamma)\} + (1-\mu-\gamma)^K I_2(t+2K+1) \\
&\leq \{(1-p)\alpha\varepsilon + k[p\alpha\varepsilon/(\mu+\gamma+k) + \varepsilon\Lambda/\mu]\}/(\mu+\gamma) + \varepsilon\Lambda/\mu .
\end{aligned} \tag{7}$$

The above discussions hint us, for any given positive number ε (ε is sufficiently small), there will exist a large positive integer K such that $E(t) < \varepsilon$, $I_1(t) < \dfrac{p\alpha\varepsilon}{\mu+\gamma+k} + \dfrac{\Lambda\varepsilon}{\mu}$, and

$$I_2(t) < \frac{(1-p)(\mu+\gamma+k)\mu\alpha\varepsilon + k(\mu p\alpha\varepsilon + \varepsilon\Lambda(\mu+\gamma+k))}{\mu(\mu+\gamma)(\mu+\gamma+k)} + \varepsilon\frac{\Lambda}{\mu}$$

for all $t > 3K+1$ .Subsequently, $\lim_{t\to\infty} E(t) = \lim_{t\to\infty} I_1(t) = \lim_{t\to\infty} I_2(t) = 0$.

On the other hand, Let

$$L(E(t), I_1(t), I_2(t)) = \alpha E(t) + (\mu+\alpha)(I_1(t) + I_2(t)) .$$

For a sufficiently small ε and $t > 4K+1$, we have

$$\begin{aligned}
&L(E(t+4K+2), I_1(t+4K+2), I_2(t+4K+2)) \\
&- L(E(t+4K+1), I_1(t+4K+1), I_2(t+4K+1)) \\
&= (\mu+\alpha)(\mu+\gamma+k) I_1(t+4K+1)\left(\frac{\alpha\beta_1}{(\mu+\alpha)(\mu+\gamma+k)} S(t+4K+1) - 1\right) \\
&+ (\mu+\alpha)(\mu+\gamma) I_2(t+4K+1)\left(\frac{\alpha\beta_2}{(\mu+\alpha)(\mu+\gamma)} S(t+4K+1) - 1\right).
\end{aligned} \tag{8}$$

Using the similar procedure as that in inequality (5) , we can have

$$S(t+4K+1) \geq \Lambda \frac{1-(1-\mu-\beta_1 m_1-\beta_2 m_2)^{K+1}}{\mu+\beta_1 m_1+\beta_2 m_2} \quad \text{with}$$

$$m_2 = \frac{(1-p)(\mu+\gamma+k)\mu\alpha\varepsilon + k(\mu p\alpha\varepsilon + \varepsilon\Lambda(\mu+\gamma+k))}{\mu(\mu+\gamma)(\mu+\gamma+k)} + \varepsilon\frac{\Lambda}{\mu} \quad ,$$

and $m_1 = \frac{p\alpha\varepsilon}{\mu+\gamma+k} + \frac{\Lambda\varepsilon}{\mu}$. Therefore, we can choose ε small enough and K large enough such that , for all positive integer t , $\frac{\alpha\beta_2}{(\mu+\alpha)(\mu+\gamma)} S(t+4K+1) \geq 1$ .The LaSalle principle implies that $L(E(t), I_1(t), I_2(t))$ will keep increase for $t > 4K+2$, and $E(t)$ can not less than ε for all $t > 4K+2$. This contradiction implies $\limsup_{t\to\infty} E(t) = U > 0$. Similarly, we have $\limsup_{t\to\infty} I_i(t) = U_i > 0 (i = 1, 2).$ Let $\sigma = \min\{U, U_1, U_2\}$, we have

$$\limsup_{t\to\infty} d((S(t), E(t), I_1(t), I_2(t), R(t)), p^0) \geq \sigma \qquad (9)$$

which implies that p^0 is isolated invariant set in $X = R_+^5$ and $W^s(p^0) \bigcap X_0 = \varnothing$. Conditions (a) and (b) hold and Theorem 1.3.1[11] conforms the persistence of (1) for $R_0 > 1$. □

IV. CONLUSION AND DISCUSSION

We improve the discrete TB model in [12] by introducing two different infectious compartments. The model can have a better description of the infectivity difference between smear-negative individuals and smear-positive patients. Model (1) is more realistic to predict TB spread.

The basic reproductive number plays an important role in the dynamics of the model, it is usually defined to be the spectrum of the next generation operator. We give the calculation formula of the basic reproduction number R_0 of model (1). It is proved that $R_0 = 1$ is a threshold value of the disease persistence: the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$, the disease will die out, while there exists an endemic equilibrium if $R_0 > 1$, and the disease will keep persistent in the population. The numerical simulation gives us a hint that the endemic equilibrium may be globally asymptotically stable. Unfortunately, the stability of the endemic equilibrium remains a great challenge. In fact, even for the simple discrete *SEIR* epidemic model, the global stability of the endemic equilibrium keeps being an open problem.

We investigated the influence of model parameters on TB case number. TB case number will increase with β_1 and β_2 since $\partial R_0 / \partial \beta_1 > 0$ and $\partial R_0 / \partial \beta_2 > 0$. After an exposed individual becomes active, he enters the smear-negative compartment at the rate p , and the smear-positive compartment at the rate $1-p$. The intuition leads us to guess that the TB case number will decrease when p increases since $\beta_1 < \beta_2$. However, the numerical simulation shows that TB case number increases when p increasing, which is a interesting phenomenon. Although the transmission rate of smear-negative pulmonary TB is lower than that of smear-positive pulmonary TB, some individuals in the smear-negative compartment will enter the smear-positive compartment, eventually, since they do not get treatment. Those individuals, who enter the smear-positive compartment，will produce new infection in smear-negative and smear positive compartments. Considering the fact that the number of smear-negative pulmonary TB cases are twice of the smear-positive pulmonary TB cases, It is better to have timely diagnosis and effective treatment to smear-negative pulmonary TB patients, and to let they transferred to the recovery compartment, directly. The better treatment for both smear-negative and smear-positive TB patients is an effective way to control TB infection.

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