Mathematical modeling and analysis with various parameters, for infection dynamics of Tuberculosis

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Abstract. Mathematical model is needed to study the epidemiology of tuberculosis. Here we have proposed a model that is more realistic. We are exhibiting a theoretical framework for getting the control and eradication methodologies to minimize the number of infectious tuberculosis cases in the community. For this purpose, the model population has been compartmentalized and the consequential model equations have been solved analytically. Numerical Simulation has been given to validate the results obtained by the theoretical approach. The effect of latent periods on the epidemics of tuberculosis with respect to population density has been studied. The equilibrium points of the model are calculated and their stability is established by using the ‘Basic Reproduction number’. It is observed that when basic reproduction number is less or equal to unity, the disease-free equilibrium point (DEF) is globally asymptotically stable, while when it is greater to unity, the endemic equilibrium point (EE) is globally asymptotically stable i.e., illness will persist in the population and epidemic will turn out to be endemic. Also, it is obtained that compactness of people decides the infection rate of tuberculosis i.e. the risk of instability of disease free equilibrium increases as the population density increases.

Keywords: Tuberculosis, Equilibrium points, Basic Reproduction Number, Stability analysis, Lyapunov function, Lasalle invariance principle.

1. Introduction
Diseases are a serious issue of present society. Among them, infectious epidemic diseases are the main threat, which result in a great calamity of the human population. Such type of infectious epidemic diseases can be restricted and reduced from society by modeling them mathematically [28]. Tuberculosis is one of them which is leading infectious killer of the population caused by pathogen \textit{Mycobacterium tuberculosis} and the second most common infectious disease worldwide. Mycobacterium tuberculosis infection spreads in the population through inhaling the tiny droplets from the cough or sneeze of a person suffering from active tuberculosis. It majorly affects the lungs called Pulmonary Tuberculosis but can also affect the other parts of the body called Extra Pulmonary Tuberculosis [29]. One-third of the world's population is currently infected with Mycobacterium tuberculosis [1-2, 30]. A large number of tuberculosis patients prompted the World Health Organization (WHO) in 1993 to declare TB a “Global Emergency” [30, 14]. The mathematical model is required to understand the transmission dynamics of tuberculosis so that the plan can be developed to restrict it. Currently near about 10 million peoples are estimated to have tuberculosis disease in 2017. Two thirds among them were in eight countries: India (27%), China (9%), Indonesia (8%), Philippines (6%), Pakistan (5%), Nigeria (4%), Bangladesh (4%) and South Africa (3%), According to “Global Tuberculosis Report 2018” by WHO [29]. Current TB treatment known as Directly Observed Treatment Short-course (DOTS) comprises of an underlying period of treatment with 4 first-line drugs for example, Isoniazid (INH), Rifampin (RIF), Pyrazinamide (PZA), Ethambutol (EMB) for 2 months day by day, trailed by treatment with INH and RIF for another 4 months [18]. The treatment may fail due to intolerance to one or more first - line drugs, then Second-line drugs like Para-Amino-Salicylate...
(PAS), kanamycin etc. can be used but they are either toxic with side effects or not much effective [3]. Although TB is curable provided the early diagnosis is made but the process takes too much time (6 - 24 months). Some researchers believed that the reason behind the increasing number of new cases of Tuberculosis may be the consequence of a huge pool of latent tuberculosis infection [5, 6, 7]. About 1.7 billion individuals of the world’s population are assessed to have a latent TB infection and thus in this manner are at danger of transmitting TB disease their lifetime [29]. However, extensive efforts have been made for eradicating the disease but the numbers of new TB cases are still increasing. For complete eradication of tuberculosis has needed attention to latent infection of tuberculosis [8-9]. Various researchers have put up many mathematical models after the outbreak of tuberculosis notably among them are Frost (1937), Louden, et al, (1954), Ferebee (1970), Anderson, et al., (1988) , Anderson and May (1991), Blower, et al (1995, 1996), Earnson (1994), Emason, et al. (1996), Ssenoga (1998) and Adetunde (2008) to mention few. Blower, et al (1996) discussed control and eradication strategies of tuberculosis by utilizing a simple mathematical model under which they gave a hypothetical structure for designing effective control strategies of TB. For knowing the epidemic behavior of TB, many researchers have explored the internal mechanism for transmission of TB with a latent period [10, 11, 14]. Ziv and co-employee assessed the procedure of therapy for the early latent period [12]. In reality, the latent period of TB has uncertainty, however, we can assure that if an individual goes to dynamic case rapidly, would not have a long latent period i.e. an individual who has a long latent period, would transmit the disease slowly in comparison to short latent individual. The manuscript contains 5 sections, in next section, we have formulated the mathematical model, in section (3) assumptions of the model are discussed, in section (4) equilibrium points and their stability is analyzed by deriving the Basic Reproduction Number (R₀), section (5) contains a numerical example to validate the analytical results, finally section (6) which contains the conclusion part.

2. Model Formulation
The proposed model is developed from the idea of the basic SEIR compartment model. Here we have classified the total population (N) into five compartments: the Susceptible (S), the short latent period (E₁) which consists of a population which has high risk to become infectious by TB, the long latent period (E₂), which has low risk to become infectious by TB, the infective (I), and the recovered compartment (R). We considered that an individual joins the infective class if and only if he is in contact with an infectious person. The flow of model is depicted in figure 1.

![Figure 1. Compartmental flow diagram, showing the dynamics of tuberculosis.](image)

Used model terminology and parameters are as follows.

λ: Constant recruitment rate to susceptible class.
\( \mu \): The per capita natural death rate (excluding TB induced)
\( d \): The mortality rate induced by tuberculosis.
\( \beta_1 \): The rate at which susceptible individual will join the short latent class (\( E_1 \))
\( \beta_2 \): The rate at which susceptible individual will join the long latent class (\( E_2 \))
\( k_1 \): The rate of progression to active tuberculosis from \( E_1 \) class.
\( k_2 \): The rate of progression to active tuberculosis from \( E_2 \) class.
\( P_1 \): The progression rate of the short latent period class (\( E_1 \)) to recovered class (\( R \)).
\( P_2 \): The progression rate of the long latent period class (\( E_2 \)) to recovered class (\( R \)).
\( \gamma \): The rate at which infective class joins the recovered class or recovery rate.
\( c \): The per capita contact rate.
\( A \): Total inhabited area.

3. Assumptions of the model

The following assumptions have been considered for model building:
- All people in the system are probably going to be infected in case of contact.
- All new born and immigrants are treated as uninfected.
- The population has fixed area size.
- The each compartment has equal death rate ‘\( \mu \)’ except tuberculosis induced death rate ‘\( d \)’.
- Some fraction of latently infected persons may directly join the recoverd class without joining the infectious class.
- Once recovered from treatment an individual will not join any other compartment again.
- The population size is \( N = S(t) + E_1(t) + E_2(t) + I(t) + R(t) \) at any time ‘\( t \)’.
- It is assumed that all the parameters and variables are non negative.

The proposed model consist a system of five nonlinear ordinary differential equations:

\[
\begin{align*}
\frac{dS}{dt} &= \lambda - \mu S - (\beta_1 + \beta_2)c \frac{S}{A} \frac{I}{A} \\
\frac{dE_1}{dt} &= \beta_1 c \frac{S}{A} - \mu - k_1 + P_1 E_1 \\
\frac{dE_2}{dt} &= \beta_2 c \frac{S}{A} - (\mu + k_2 + P_2) E_2 \\
\frac{dI}{dt} &= (k_1 E_1 + k_2 E_2) - (\mu + d + \gamma) I \\
\frac{dR}{dt} &= (\gamma I + P_1 E_1 + P_2 E_2) - \mu R
\end{align*}
\]

For simplicity we can reduce the above system of differential equations by choosing

\( s = \frac{S}{A}, e_1 = \frac{E_1}{A}, e_2 = \frac{E_2}{A}, i = \frac{I}{A}, r = \frac{R}{A} \) and \( \pi = \frac{\lambda}{A} \) and then substituting these in our model differential equations (1), it is reduced to

\[
\frac{ds}{dt} = \frac{1}{A} \frac{dS}{dt} = \frac{1}{A} \left( \lambda - \mu S - (\beta_1 + \beta_2)c \frac{S}{A} \frac{I}{A} \right) = \frac{\lambda}{A} - \mu \frac{S}{A} - \left( (\beta_1 + \beta_2)c \frac{S}{A} \frac{I}{A} \right) = \pi - \mu s - (\beta_1 + \beta_2)csi
\]

Similarly we get that

\[
\begin{align*}
\frac{de_1}{dt} &= \beta_1 csi - (\mu + k_1 + P_1) e_1 \\
\frac{de_2}{dt} &= \beta_2 csi - (\mu + k_2 + P_2) e_2
\end{align*}
\]
With the initial conditions as $s(0) \geq 0, e_1(0) \geq 0, e_2(0) \geq 0, i(0) \geq 0, r(0) \geq 0$ now also $n = s(t) + e_1(t) + e_2(t) + i(t) + r(t)$, since first four equations are free from ‘r’, so it is sufficient to take the first four equations into consideration

\[
\frac{ds}{dt} = \pi - \mu s - (\beta_1 + \beta_2)cs \quad (2)
\]

\[
\frac{de_1}{dt} = \beta_1 csi - (\mu + k_1 + P_1)e_1
\]

\[
\frac{de_2}{dt} = \beta_2 csi - (\mu + k_2 + P_2)e_2
\]

\[
\frac{di}{dt} = (k_1 e_1 + k_2 e_2) - (\mu + d + \gamma)i
\]

By considering it as a real world phenomenon we can examine the system in domain

\[\Omega = \left\{ (s, e_1, e_2, i) \in \mathbb{R}^4, 0 \leq s(t) + e_1(t) + e_2(t) + i(t) \leq \frac{\pi}{\mu} \right\} \]

The set ‘$\Omega$’ is positively invariant with respect to system (2) which is equivalent to system equation (1).

4. Analysis of the model

4.1. The existence of equilibrium of model

For determining the existence of equilibrium points of the model equation (2), we have

\[
\frac{ds}{dt} = \frac{de_1}{dt} = \frac{de_2}{dt} = \frac{di}{dt} = 0
\]

The governing equations of the shown model (Fig.1) have two non-negative equilibrium points namely

4.1.1. Disease Free Equilibrium.

Let $E_0(\pi/\mu, 0, 0, 0)$ is known as disease-free equilibrium point for the studied system equation (2). For the disease-free equilibrium point, we have $e_1 = e_2 = i = 0$ and we define $s_0 = \frac{\pi}{\mu} = \frac{\lambda}{\mu}$. The following conditions must be noted for the existence of susceptible class ‘s’

(I) The susceptible cannot have a non-trivial equilibrium i.e. $\lambda \neq 0$, and susceptible population goes to extinction when $\lambda = 0$.

(II) The susceptible will have an equilibrium value when there is recruitment, i.e. when $\lambda > 0$.

Expected population in the disease free equilibrium state

The disease can be annihilated from the population when there are no more infective and latent people, i.e. $i = e_1 = e_2 = 0$. So the total population size is reduced to $n = s(t) + r(t)$, and the remaining differential equations are

\[
\frac{ds}{dt} = \pi - \mu s, \quad \frac{dr}{dt} = -\mu r,
\]

After solving these we get that $s(t) = \frac{\pi}{\mu} + c_1 e^{-\mu t}, \quad r = r_0 e^{-\mu t}$ respectively. Let initially $s(0) = s_0$ and $r(0) = r_0$ then $s(t) = \frac{\pi}{\mu} + \left( s_0 - \frac{\pi}{\mu} \right) e^{-\mu t}$ & $r = r_0 e^{-\mu t}$ then as $t \to \infty$, $s(t) \to \pi/\mu$ & $r(t) \to 0$ respectively. Hence the population will contain only the susceptible class.

4.1.2. Endemic Equilibrium.

Let $E'(s', e_1', e_2', i')$ be the endemic equilibrium point for the system equation (2) of studied model (Fig.1) where $(s', e_1', e_2', i') \neq 0$, the corresponding derived values at which can be given by following.
4.2. Stability of the Equilibrium states

This section includes investigation on the stability of both the equilibrium points, infection free equilibrium point \( E_0 \), which contains susceptible as the only non zero compartment and also the endemic point \( E^* \) whose all compartments are taken to be non-negative [27]. For studying the stability of equilibrium states, we determined the following threshold parameter ‘\( R_0 \)’ by using the ‘next generation matrix’ approach

\[
R_0 = \frac{c\lambda}{\mu A (\mu + d + \gamma)} \sum_{i=1}^{i=2} \frac{\beta_i k_i}{(\mu + k_i + P_i)}
\]

We define \( R_0 \) as the expected secondary number of cases induced by one case during his/her entire infectious period in all populations [14, 17]. It is also known as the ‘basic reproduction number’ [26]. It can be shown that when \( R_0 \leq 1 \), the infection free equilibrium \( E_0 \) is globally asymptotically stable, so the population will be disease free [14, 24], and when \( R_0 > 1 \) the endemic equilibrium \( E^* \) is globally asymptotically stable i.e., the disease will remain in the population.

**Theorem (1)**. If \( R_0 \leq 1 \), the disease-free equilibrium state \( E_0 \) is comprehensively asymptotically stable in \( \Omega \).

**Proof.** For showing the global stability of disease free state \( E_0 \), we determined the Lyapunov function ‘\( L \)’ as follows

\[
L = k_1(\mu + k_2 + P_2)e_1 + k_2(\mu + k_1 + P_1)e_2 + (\mu + k_1 + P_1)(\mu + k_2 + P_2)i
\]

Then the time derivative of \( L \) is

\[
\frac{dL}{dt} = k_1(\mu + k_2 + P_2)(\beta_1 csi - (\mu + k_1 + P_1)e_1) + k_2(\mu + k_1 + P_1)(\beta_2 csi - (\mu + k_2 + P_2)e_2)
\]

\[
+ (\mu + k_1 + P_1)(\mu + k_2 + P_2)((k_1e_1 + k_2e_2)(\mu + d + \gamma))(\mu + d + \gamma)(R_0 - 1)i
\]

So, \( \frac{dL}{dt} \leq 0 \) when \( R_0 \leq 1 \) furthermore \( \frac{dL}{dt} = 0 \) holds if and only if \( R_0 = 1 \) or \( i(t) = 0 \)

Hence the greatest invariant set contained in the set \( \Omega \) for which \( \frac{dL}{dt} = 0 \) is unique “disease free equilibrium point” \( E_0 \), so by applying LaSalle Invariance principle [24, 25], it is clear that disease free equilibrium point is globally asymptotically stable in \( \Omega \).

**Theorem (2)**. If \( R_0 > 1 \), then the endemic equilibrium state \( E^*(s^*, e_1^*, e_2^*, i^*) \) is globally asymptotically stable in \( \Omega^* \).

**Proof.** Here we have used the similar approach [19, 20, 21, and 23] which is mostly used to define the stability of complex epidemiological compartmental models i.e., by constructing the Lyapunov function ‘\( L \)’ as follows

\[
L: \{(s, e_1, e_2, i) \in \Omega^* \} \rightarrow R \text{ Where } \Omega^* = \{(s, e_1, e_2, i) \in R^*_4 ; s(t), e_1(t), e_2(t), i(t) > 0\}
\]

\[
L = W_1(s - s^*\ln \left( \frac{s}{s^*} \right)) + W_2(e_1 - e_1^*\ln \left( \frac{e_1}{e_1^*} \right)) + W_3(e_2 - e_2^*\ln \left( \frac{e_2}{e_2^*} \right)) + W_4(i - i^*\ln \left( \frac{i}{i^*} \right))
\]

Where \( W_1, W_2, W_3, W_4 \) are non negative constants in \( \Omega \). Now taking the time derivative of Lyapunov function \( L \) we get that

\[
s^* = \frac{\pi}{\mu R_0}, \quad e_1^* = \frac{\beta_1 \pi}{(\beta_1 + \beta_2)(\mu + k_1 + P_1)} \left(1 - \frac{1}{R_0} \right),
\]

\[
e_2^* = \frac{\beta_2 \pi}{(\beta_1 + \beta_2)(\mu + k_2 + P_2)} \left(1 - \frac{1}{R_0} \right), \quad i^* = \frac{(R_0 - 1)\mu}{c(\beta_1 + \beta_2)}
\]
At an endemic equilibrium point we have that

\[\pi = \mu s^* + (\beta_1 + \beta_2)cs^*i^*\]

\[(\mu + k_1 + P_1) = \frac{\beta_1 cs^*i^*}{e_1}\]

\[(\mu + k_2 + P_2) = \frac{\beta_2 cs^*i^*}{e_2}\]

\[(\mu + d + \gamma) = \frac{k_1 e_1 + k_2 e_2}{i^*}\]

Substituting these values in above equation we have that

\[\frac{dL}{dt} = \frac{W_1(s - s^*)}{s} \left(\mu s^* + (\beta_1 + \beta_2)cs^*i^* - \mu s - (\beta_1 + \beta_2)cs i + W_2(e_1 - e_1)\left(\frac{\beta_1 cs i}{e_1} - \frac{\beta_1 cs^*i^*}{e_1}\right)\right)\]

\[+ W_3(e_2 - e_2)\left(\frac{\beta_2 cs i}{e_2} - \frac{\beta_2 cs^*i^*}{e_2}\right) + W_4(i - i^*)\left(\frac{k_1 e_1 + k_2 e_2}{i} - \frac{k_1 e_1^* + k_2 e_2^*}{i^*}\right)\]

The function \(M(s, e_1, e_2, i)\) is non positive by following the approach used in [21, 22, 23], that is \(M \leq 0\) for every \(s, e_1, e_2, i > 0\) thus \(\frac{dt}{dt} \leq 0\) and \(\frac{dt}{dt} = 0\). When \(s = s^*, e_1 = e_1^*, e_2 = e_2^*, i = i^*\).

Hence the greatest compact invariant set in domain for which \(\frac{dt}{dt} = 0\) is the singleton \(\{E^*\}\), which is endemic equilibrium point. So, by using LaSalle invariance principal [24, 25] we can conclude that \(\{E^*\}\) is globally asymptotically stable in \(\Omega^*\), if \(R_0 > 1\).

5. Numerical Analysis and Result Discussions

We have discussed the endemic point \(\{E^*\}\) of the model by presenting the numerical example. Following Castillo-Chavez Zhilan (1997), Feng et al.(2001), Castillo-chavez and song (2002), Song et al. (2002), A. Sematimaba et al.(2005), Jia and Li et.al. (2007) and Adetunde (2008), we calculated the values of parameters from [13] as, \(\lambda = 3.805, \mu = 0.0185, \beta_1 = \beta_2 = 2, \gamma = 2, d = 0.365, c = 2, R^* = 3000, I^* = 90, S^* = 5000, E_1^* = E_2^* = 2500\) and \(A = 200/300/400\) square meter. The results are depicted graphically in figure 2-5.

Now we divided our model into four different classes on the basis of values of \(k_1, k_2\) and \(P_1, P_2\) Then found the variations in graphs with respect to population density.

- Case 1: when \(k_1 = k_2\) and \(P_1 = P_2\)
- Case 2: when \(k_1 > k_2\) and \(P_1 > P_2\)
- Case 3: when \(k_1 = k_2\) and \(P_1 > P_2\)
- Case 4: when \(k_1 > k_2\) and \(P_1 \geq P_2\)

**Case 1:** when \(k_1 = k_2\) and \(P_1 = P_2\)

In this case we have taken that when \(k_1,\) (the rate at which short latent population joins the infected class) is equal to \(k_2,\) (the rate at which long latent population joins the infected class) and also The progression rate of the short latent period to recovered class (\(P_1\)) is equal to progression rate of the long latent period to recovered class (\(P_2\)). I.e. when \(k_1 = k_2 = 0.2\) and \(P_1 = P_2 = 2\)
In figure 2 (a), the graphical representation of the susceptible class with time in respect to the inhabited area is shown. When $k_1 = k_2$ and $P_1 = P_2$, we detected increment in susceptible population as an increment in area is done with respect to time which may be due to population size and the number of contacts per individual.

Figure 2 (b) and figure 2 (c) demonstrate the impact of the inhabited area $A$, on Short & long latent population with time. It is seen that, by increasing the area inhabited by latent population with time, latent population is decreasing. This is because of the fact that increasing the area size reduces the rate
of contact of susceptible with infectious people. So, the Impact of latent period population \( (E_1 \text{ and } E_2) \) with respect to population density can be shown clearly through these figures.

From figure 2 (d) it is observed that the infectious individual diminishes irrespective of the area size. A high peak has been identified in small area size this may be appearing due to excessive crowd, which furthermore prompted the higher rate of infection so, huge number of susceptible joins the infected class and becomes stationary later.

Figure 2 (e) shows the effect of inhabited area on the Recovered population with time. As the area increases, Recovered population decreases which may be due to the low congestion in the inhabited area.

Now we have taken different cases by varying the values of \( k_1, k_2 \) and \( P_1, P_2 \) and compare the variation in graphs of \( E_1 \) and \( E_2 \) with respect to the size of the area occupied.

**Case 2: when \( k_1 > k_2 \) and \( P_1 = P_2 \)**

Here we have taken a case into account that when \( k_1 = 0.2, k_2 = 0.1, P_1 = P_2 = 2 \) and saw the variations between the graphs of \( E_1 \) and \( E_2 \) on different area size i.e. \( A = 200/300/400/1400 \) square meter.

![Figure 3 (a) When A = 200 square meter](image1)

![Figure 3 (b) When A = 300 square meter](image2)

![Figure 3(c), When A = 400 square meter](image3)

![Figure 3(d), When A= 1400 square meter](image4)

We observed that with respect to time the emission rate of the people in Short Latent class \( (E_1) \) is more than the people in Long Latent Class \( (E_2) \), which can be seen from the graphs 3(a), 3(b), 3(c) and 3(d) while all the remaining compartments having the same graphical representation as we have discussed in former case.
**Case 3:** when $k_1 = k_2$ and $P_1 > P_2$

i.e. $k_1 = k_2 = 0.2$, $P_1 = 2$, $P_2 = 1.5$

Similarly when we have taken this case then also we are getting the same interpretation as in previous cases and there was not much difference between the graphs of $E_1$ and $E_2$ with respect to area size. So, we are not discussing much.

![Figure 4](attachment:figure4.png)

Figure 4 (a) When $A = 200$ square meter  
Figure 4(b) When $A = 300$ square meter

![Figure 4](attachment:figure4.png)

Figure 4 (c) When $A = 400$ square meter  
Figure 4(d) When $A = 1400$ square meter

**Case 4:** when $k_1 > k_2$ and $P_1 > P_2$

i.e. $k_1 = 0.2$, $k_2 = 0.1$, $P_1 = 2$, $P_2 = 1.5$

In this case also we have not seen any behavior difference between the graphs of $E_1$ and $E_2$ then the former cases. So, we are not discussing it as we have discussed it in earlier case.

![Figure 5](attachment:figure5.png)

Figure 5 (a) When $A = 200$ square meter  
Figure 5 (b) When $A = 300$ square meter
Conditions for minimization of disease

For minimization of tuberculosis incidence in a Community, the population sizes of Latent classes and infectious class must be decreasing in nature, for which we have following three, conditions that
\[
\frac{dE_1}{dt} < 0, \quad \frac{dE_2}{dt} < 0, \quad \frac{dl}{dt} < 0
\]

Now applying values from Equation (1) in these conditions we have that
\[
\frac{dE_1}{dt} = \beta_1 c \frac{S}{A} l - (\mu + k_1 + p_1)E_1 < 0
\]
\[
\frac{dE_2}{dt} = \beta_2 c \frac{S}{A} l - (\mu + k_2 + p_2)E_2 < 0
\]
\[
\frac{dl}{dt} = (k_1 E_1 + k_2 E_2) - (\mu + d + \gamma)l < 0
\]

Now we get that
\[
A > \frac{(k_1 E_1 + k_2 E_2)\beta_1 c S}{(\mu + d + \gamma)(\mu + k_1 + p_1)E_1}
\]
\[
A > \frac{(k_1 E_1 + k_2 E_2)\beta_2 c S}{(\mu + d + \gamma)(\mu + k_2 + p_2)E_2}
\]

Now combining these two we concluded that the disease can be minimized from the population if and only if
\[
A > \min \left\{ \frac{(k_1 E_1 + k_2 E_2)\beta_1 c S}{(\mu + d + \gamma)(\mu + k_1 + p_1)E_1}, \frac{(k_1 E_1 + k_2 E_2)\beta_2 c S}{(\mu + d + \gamma)(\mu + k_2 + p_2)E_2} \right\}
\]

6. Conclusion

In the present study, we have done mathematical modeling for the Infection dynamics of Tuberculosis. In which the consequence of population density on the transmission of tuberculosis epidemic is discussed. It is noticeable that the population density determines the rate of contact with another person, which directly has an impact on the infection rate of airborne disease tuberculosis. It is perceived that overcrowding is one of the important factors in increasing tuberculosis incidence. For the qualitative analysis, the equilibrium points of the shown model are obtained and their stabilities were analyzed. By calculating the threshold parameter $R_0$ we investigated the conditions of existence and stability of the population equilibrium points of the model. Using stability theory the Basic Reproduction number $R_0$ is derived and it is observed that when $R_0 \leq 1$, the disease-free equilibrium point is globally asymptotically stable in $\Omega$ that is the infected person on average will affect less than or equal to one individual in its entire life as an infectious person, so the disease will be eradicated from the population. While when $R_0 > 1$, the endemic equilibrium point is globally asymptotically stable in $\Omega$ that is the
infected individual will affect more than one person in its entire life as an infectious person. So the infected population will survive in the total population. Numerical simulation of the model attached here which validates the results obtained analytically, both highlights the dependence of population density on the tuberculosis incidence. Impact of latent periods (E₁ and E₂) with respect to population density is demonstrated clearly through numerical simulation and it is perceived that the emission rate of the epidemic from Latent Classes (Short/Long) to Infection Class declined as population density decreases or occupied area size increases. Conclusively, the increment in population density will increase the risk of instability of disease-free equilibrium. These results demonstrate that it is conceivable to minimize or remove tuberculosis in any population. This can be done by decreasing the overcrowding or increasing the area size inhabited by the population.

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