Two-Level Handling Model of Tuberculosis

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Abstract. Tuberculosis (TB) is an infectious disease caused by the Mycobacterium tuberculosis which most commonly attacks the lungs. In 2017, ten million people contracted TB and 1.6 million people died of TB. It is estimated that one million children suffering from TB and 230,000 children die due to TB, including children who die both TB and HIV. Global estimation show that there are ten million TB cases which are equivalent to 133 cases per hundred thousand population numbers, the country with the highest case is India by 27%, China by 9% and Indonesia by 8%. Some research related to the mathematical model of the spread of TB, for example the S-E-I/L model by Soyoung, the S-L-I/J-T model by Zhang, and the S-L-I-T model by Trauer. In this research proposal we will reconstruct a two-level model of the spread of tuberculosis infection, which means that this tuberculosis infection attacks two populations simultaneously, namely adult and individual children. The model used is the S-E-I-R/S-E-I-R model. The first individual who is attacked, in this case is an adult individual, then infects the individual child. Later, we obtain the disease free equilibrium which is \((\frac{\alpha N_a}{\mu}, 0, 0, 0, \frac{\alpha N_c}{\mu}, 0, 0, 0)\).

1. Introduction
Tuberculosis (TB) is an infectious disease caused by the bacterium Mycobacterium tuberculosis which most often attacks the lungs. In 2017, ten million people contracted TB and 1.6 million died from TB (including three hundred thousand people who suffered from TB along with HIV). Also in 2017, there are an estimated one million children who suffer from TB and 230,000 children die from TB, including children who have died from TB and HIV. [1].

TB, a chronic infectious disease caused by Mycobacterium tuberculosis, has become a major public health problem. One-third of the world’s population is infected with this bacterium, reported by WHO that three million people die every year (World Health Organization, 2018a). Although almost 95% of TB infections occur in developing countries, the emergence of HIV and drug resistance to TB bacteria have changed the dynamics of infection worldwide. [2]. Other factors that cause the TB epidemic are the elimination of TB control programs, such as poor drug consumption, poverty, low rates of early TB detection, HIV infection, diabetes and immigration. [3].

In 2017, global estimates show that there are ten million TB cases (around nine to eleven million) which are equivalent to 133 cases (range 120-148 cases) per hundred thousand population. Of these cases, the highest prevalence was found in the Southeast Asian region by 44%, Africa by 25%, West Pacific by 18% and several other cases in the Mediterranean region by 7.7%, North and South America by 2.7% and cases that were the least was found in the European region, which was 2.7%. Whereas the countries with the highest cases were India sequentially by 27%, China by 9% and Indonesia by 8% [1].
Children in the first five years of age are vulnerable to fatal and serious TB infection, even more so without the BCG vaccine. Accurate data regarding the incidence and prevalence of TB are not available because of the difficulty of TB diagnosis in children. However, the few available data sources indicate that the prevalence of TB in children aged 0-14 years is estimated to be 0.3% of cases found based on radiological examinations, and 0.15% of bacterial cases [4].

Research related to the mathematical model of TB spread, for example the mathematical model by Kim So-Young discusses the spread of TB and intervention strategies for TB mitigation using the S-E-I/L model, where the latent compartment in the study is divided into two, namely latent with high risk (E) and low risk (I) [5]. However, there was no recovered compartment in the model used by Kim. This was later refined by Jinhui Zhang. The model used by Zhang is similar to the model used by Kim, but there is a recovered or treatment compartment, so the model used is S-L-I/J-T. In Zhang’s study, there was only one latent compartment, but the infection compartment was divided into two, namely infected and treated in hospital (J) and infected but not treated in hospital (I) [6]. While the model used by Trauer is similar to the model used by Young. In the infection compartment, there is only compartment I, so the model used is S-L-I-T [7]. In this paper we would like to construct a S-E-I-R/S-E-I-R model.

2. Mathematical Model
Let subscripts \(a\) denote the adult population, and \(c\) denote the children. Also, let \(S, E, I\) and \(R\) be the Susceptible, Exposed, Infectious, and Recovered compartment respectively. The TB started both in either adult or children. The adult population is \(N_a\) while children population is \(N_c\). The ratio of change in susceptible population is denoted by \(\alpha\). The natural death is denoted by \(\mu\). While \(\beta, \lambda, \gamma, \text{and} \frac{I}{N}\) is the ratio of TB infection, ratio of adult population, population contact and the percentage of infected population and the whole population, respectively. The death ratio due to TB is \(d\), the recovery ratio of TB is \(r\), and \(k\) is the infection ratio of TB infection. We assumed that there is no reinfection in this case and the propagation of TB is slow. We can construct a compartmental model as follow:

![Diagram of the SBSE model](image)

**Figure 1.** The compartment diagram of the TB

The infection begins in both adult and children population. The change ratio of susceptible plays an important role in determining the susceptible population in both adult and children. In this paper, we assume that both ratio in adult and children are the same, then the cross-infection occurs. The infected adult infect the susceptible children and vice versa, and contribute a ratio of either \(\frac{I_a}{N_a}\) or \(\frac{I_c}{N_c}\). We assume that the bacteria propagates slowly, the susceptible compartment the susceptible will transfer to exposed compartment. The exposed population
has a $k$ variable which donates the infection ratio, differs in adult and children population. We obtain the mathematical model:

\[
\begin{align*}
\frac{dS_a}{dt} &= \alpha N_a + \beta \gamma S_a \frac{I_c}{N_a} - \mu_a S_a \\
\frac{dE_a}{dt} &= \beta \gamma S_a \frac{I_c}{N_a} - \mu_a E_a - k_a E_a \\
\frac{dI_a}{dt} &= k_a E_a - (\mu_a + d) I_a - r_a I_a \\
\frac{dR_a}{dt} &= r_a I_a - \mu_a R_a \\
\frac{dS_c}{dt} &= \alpha N_c + \beta \gamma S_c \frac{I_a}{N_c} - \mu_c S_c \\
\frac{dE_c}{dt} &= \beta \gamma S_c \frac{I_a}{N_c} - \mu_c E_c - k_c E_c \\
\frac{dI_c}{dt} &= k_c E_c - (\mu_c + d) I_c - r I_c \\
\frac{dR_c}{dt} &= r I_c - \mu_c R_c
\end{align*}
\]

By setting the right-hand side of equations 1 to zero and solving them, we obtained to equilibria, that is the disease-free equilibrium:

\[T_1 = \left( \frac{\alpha N_a}{\mu}, 0, 0, 0, \frac{\alpha N_c}{\mu}, 0, 0, 0 \right)\]

It is not easy to obtain the endemic equilibrium due to the complexity of the equation, however, we can still obtain and show the endemic equilibrium numerically. The endemic equilibrium:

\[T_2 = \left( S_a^*, E_a^*, I_a^*, R_a^*, S_c^*, E_c^*, I_c^*, R_c^* \right) \]

where:

\[
\begin{align*}
S_a^* &= \frac{\left( \gamma \alpha \beta k_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + \mu_c N_c (\mu_c + k_a) (\mu_c + d + r_c) (\mu_a + d + r_a) (\mu_a + k_a) \right)}{\left( \mu_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + N_c \alpha \beta \gamma k_c \right)} \\
E_a^* &= \frac{1}{k_a (\mu_a + k_a) \beta \gamma (\mu_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + N_c \alpha \beta \gamma k_c)} \\
I_a^* &= \frac{1}{\beta \gamma (\mu_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + N_c \alpha \beta \gamma k_c) (\mu_a + k_a) (\mu_a + d + r_a)} \\
R_a^* &= \frac{1}{\beta \gamma (\mu_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + N_c \alpha \beta \gamma k_c) (\mu_a + k_a) \mu_a} \\
S_c^* &= \frac{\left( \mu_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + N_c \alpha \beta \gamma k_c \right) (\mu_a + k_a) (\mu_a + d + r_a) N_c}{k_c \beta \gamma \left( \gamma \alpha \beta k_a N_a + \mu_c N_c (\mu_a + k_a) (\mu_a + d + r_a) \right)} \\
E_c^* &= \frac{1}{k_c \beta \gamma \left( \gamma \alpha \beta k_a (\mu_c + k_c) N_a + \mu_c N_c (\mu_a + k_a) (\mu_a + d + k_a) (\mu_a + k_c) \right)} \\
I_c^* &= \frac{1}{\left( \gamma \alpha \beta k_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + \mu_c N_c (\mu_a + d + r_c) \right) (\mu_a + d + r_a) (\mu_a + k_a) \gamma \beta} \\
R_c^* &= \frac{1}{\left( \gamma \alpha \beta k_a (\mu_c + k_c) (\mu_c + d + r_c) N_a + \mu_c N_c (\mu_a + d + r_a) (\mu_a + k_a) \right) \gamma \beta k_c}
\end{align*}
\]
3. Conclusion
In this paper, a two-level handling of TB model has been constructed. We can obtain two equilibrium point, i.e., the disease-free equilibrium point and the endemic equilibrium point. We can easily obtain the disease-free equilibrium but the endemic equilibrium is not easy to obtain analytically. By setting some values to the parameters, we can obtain the equilibria numerically.

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