A Modelling Technique for Controlling the Spread of Tuberculosis

O. P. Ogundile, S. O. Edeki, and S. O. Adewale

***Abstract*—** In this paper, a new modelling technique is considered for the study of tuberculosis and its spread. The model serves as a means of controlling the spread of tuberculosis. The mathematical analysis of the model equations is carried out to investigate the transmission dynamics of the disease. Also, solutions on how to reduce the spread of the disease in the community is proposed, and some simulations are performed to determine the consequence of the effective contact rate for Tuberculosis infection. Three hypothetical cases are considered and it is discovered that as the effective contact rate increases, the contact rate also increases. Results are illustrated graphically with the aid of MATLAB mathematical software.

***Keywords*—** Tuberculosis, epidemic, modelling, DOTS, DFE, reproduction number.

I. Introduction

TUBERCULOSIS (TB) is a bacterial disease which attacks some part of the human body such as lungs, kidneys, bones, lymph nodes, and brain. This disease is caused by a known *Mycobacterium tuberculosis*, that looks like a rod-shape bacterium. Some of the symptoms are in the form of cough, chest pain, shortness of breath, loss of appetite, weight loss, fever, chills, and fatigue.

TB majorly can be communicated from person to person via droplets. When an infected person coughs, sneezes, or talks, saliva or mucus are released into the air, which can be inhaled by another person.

The behaviour of precise mathematical models can be interpreted using mathematical methods and computer simulations.

Several researchers have worked on the analysis and modelling of TB epidemic. Some of these research works are theoretical and [1-3] used the approach of mathematics in analyzing it.

TB transmission model with Directly Observed Therapy Short-course (DOTS) was discussed in [1]. This model enables the identification and treatment of the people displaying signs and uses standard incidence function for the infection rate. The research shows that in the absence of re-infection, the model has a globally asymptotic stable Disease free equilibrium (DFE) whenever $R_d < 1$. A notable medical contribution in TB control was the introduction of antibiotics which resulted in significant decrease in mortality. As a consequence of this development, TB-infected people can be effectively treated using multiple drugs via the DOTS strategy. However, if not strictly complied to or administered wrongly, such therapy may lead to the evolution and development of multi-drug resistant TB (MDR-TB). A deterministic model for TB transmission dynamics, in the presence of DOTS is presented and rigorously analyzed.

Similarly, in 2010 a mathematical model of tuberculosis epidemics [2] was used to study the strengths and limitations of using homogeneous mixing and heterogeneous mixing epidemic models to explore the context of the transmission dynamics of tuberculosis.

In [3], dealt with TB that spread through one –strain and two strains models. They proved that if the basic reproduction ratio $R_o \geq 1$, then the DFE is globally asymptotically stable on the nonnegative orthant and if $R_o > 1$ an endemic equilibrium exists and is globally asymptotically stable. Olwaseun *et al.*, [4] worked on the synergistic interaction between HIV and Tuberculosis using a deterministic model which brings in many of the essential biological and epidemiological features of the two diseases.

An extensive review on the relevant literature review has revealed that while much research has been done on tuberculosis, adequate research on the spread of tuberculosis is yet to be done. Though TB is a disease with vaccines readily available, it is still a disease that claims lots of lives. Several people have worked on the mathematical modelling of the spread of tuberculosis and have come to different conclusions.

II. Formulation of the Model

Following [1, 2], we have the following TB model below which shall be used to study the transmission dynamics of tuberculosis.

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O. P. Ogundile (opeyemiogundile@yahoo.com) is with the Department of Mathematics, Covenant University, Ota, Nigeria.

S. O. Edeki (soedeki@yahoo.com)is with the Department of Mathematics, Covenant University, Ota, Nigeria.

S. O. Adewale (soadewale@lautech.edu.ng) is with the Department of Mathematics, Ladoke Akintola University of technology, Ogbomoso, Nigeria.

$$\begin{cases} \dfrac{dS}{dt} = \pi - \lambda S - \mu S, \\ \dfrac{dE}{dt} = \xi\lambda S - (\kappa + \mu)E, \\ \dfrac{dT_u}{dt} = \begin{pmatrix} (1-\xi)\lambda S + (1-\omega)\kappa E \\ (+\gamma_u + \sigma_u + \mu + \delta_u)T_u \end{pmatrix}, \\ \dfrac{dT_d}{dt} = \begin{pmatrix} \omega E + \gamma_u + T_u \\ -(\sigma_u + \tau + \mu + \delta_d)T_d \end{pmatrix}, \\ \dfrac{dF}{dt} = \tau T_d - (\sigma_f + \mu + \delta_f)F, \\ \dfrac{dR}{dt} = \sigma_u T_u + \sigma_d T_d + \sigma_f F - \mu R. \end{cases} \quad (1)$$

where,

$$\lambda = \frac{\beta(Tu + \eta dT_d + \eta fF)}{N}$$

For the disease equilibrium, we will let:

$$N = S + E + T_u + T_d + F + R. \quad (2)$$

In summary, the TB transmission dynamics model is illustrated with a model flow chart in fig. (1). While the corresponding parameters and variables are described in Table (1).

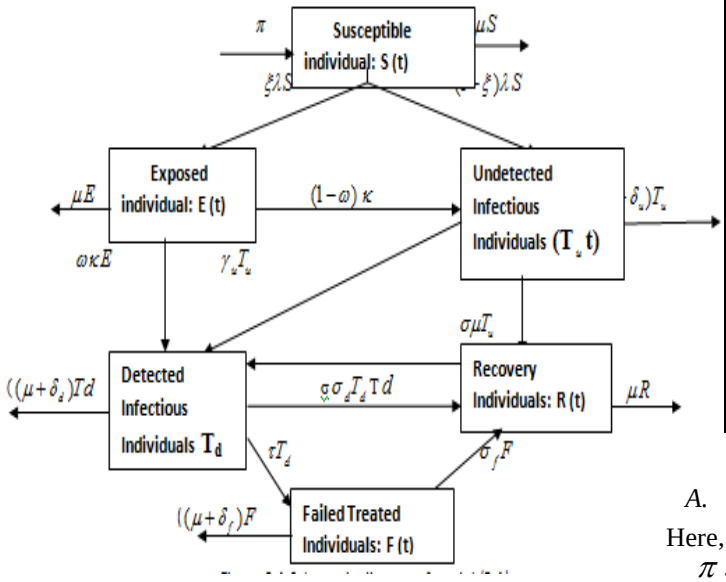

Figure 1 Schematic diagram of model (1)

The essential features of the model are that it:

i. allows disease transmission by individuals in the undetected (T_u), detected (T_d) and failed treatment F classes;

ii. allows for the endogenous re-activation of vulnerable individuals (at the rate κ); and

iii. allows for the possibility of treatment failure (at the rate τ).

The model extends to the models in many of the aforementioned studies [1, 5- 9] by including a separate compartment (F) for treated individuals who failed treatment. Furthermore, it extends the studies in [9, 10], which based on using mass action incidence and without exogenous re-infection (it also extends the work of Okuonghae [11] by using standard incidence).

Table 1: Parameter values

Parameter	Nominal Value (per year)	References
π	2000 (per100000 Population)	[4]
μ	0.2	Assumed
β	2,4,6	[12]
ξ	0.001, 0.7, 1.7	Assumed
κ	0.5	[4]
ω	0.75	[4]
$\sigma_u, \sigma_d, \sigma_f$	0.3,0.3,0.3	Assumed
γ_u	0.2	[13, 14]
τ	0.9	[12]
δ_u, δ_d	0.3, 0.3,0.3	[4]
δ_f	0.3	Assumed
η_d, η_f	0.001,0.8	Assumed

A. Parameters description

Here, the parameters are described as follows:

π : Rate of recruitment into the population setting

M : Per capita natural mortality rate

β : Effective rate of contact for TB infection

η_d, η_f : Modification parameters for detection and failure respectively.

$\sigma_u, \sigma_d, \sigma_f$: Recovery rate for individuals in T_u; T_d; and F classes.

ξ: Fraction of newly-infected individuals who are slow progressors.

κ : Endogenous factor rate for re-activation regarding exposed individuals.

ω: Portion of exposed individuals who are being detected.

γ_u : Discovered rate for un-detected infectious individuals.

τ : Treatment failure rate regarding detected infectious individuals.

$\delta_u, \delta_d, \delta_f : T_u, T_d$, and F classes induced mortality rate.

III. ANALYSIS OF THE MODEL

Theorem 1. [1]: Let D be the set associated with the models ((1) and (2)) be defined as :

$$D = \left\{ \left(S, E, T_u, T_d, F, R\right) \varepsilon \ \Re^6{}_+ : N \le \frac{\pi}{\mu} \right\}.$$

Then D is positively-invariant and attracting with respect to the model (1) and (2).

Proof: Let D be a feasible region in biological form as earlier defined. Then, the addition of all the model dynamics yields the abtainment of the total population rate of change.

$$\frac{dN}{dt} = \tau - \mu N - \delta_u T_u - \delta_d T_d - \delta_f F .$$

It follows that $\frac{dN}{dt} < 0$ whenever $N > \frac{\pi}{\mu}$, furthermore, since $\frac{dN}{dt} \le \pi - \mu N,$ it is clear that $N(t) \le \frac{\pi}{\mu}$. Hence, all corresponding solutions of the defined model based on the initial conditions as containd in D hold for all positive values of the time parameter. Thus, the region D is attracting and positively invariant. In the region D, the model can be referred to as being epidemiologically and well-posed mathematically [16].

A. Disease-free Equilibrium Model (DFEM)

This model posesses a Disease-free equilibrium (DFE), this can be obtained by equating the right-hand sides of the equations of the model to zero, given by

$$\varepsilon_0 = (S^*, E^*, T^*{}_u, T^*{}_d, F^*, R^*) = \left(\frac{\pi}{\mu}, 0,\ 0,\ 0,\ 0,\ 0 \right).$$

a

The DFE stability termed ε_0, is analyzed by the application of the (NG (next generation) method (see [17]). Thus, a matrix P (non-negative matrix) in relation to the new infection terms, and a non-singular M-matrix Q (of the remaining transforms) are given, respectively as:

$$P = \begin{pmatrix} \kappa & \xi\beta & \xi\beta\eta d & \xi\beta\eta d & 0 \\ 0 & (1-\xi)\beta & (1-\xi)\beta\eta d & (1-\xi)\beta\eta d & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$Q = \begin{pmatrix} \kappa 1 & 0 & 0 & 0 & 0 \\ -(1-\omega)k & k2 & 0 & 0 & 0 \\ -wk & -\gamma_u & k3 & 0 & 0 \\ 0 & 0 & -\tau & k4 & 0 \\ 0 & -\sigma u & -\sigma d & -\sigma f & \mu \end{pmatrix}$$

where, $k1 = \kappa + \mu,\ k2 = \gamma_u + \sigma_u + \mu + \delta_u,$

$k3 = \sigma_d + \tau + \mu + \delta_d$ and $k4 = (\sigma_f + \mu + \delta f)$. The associated reproduction number, denoted by R_d, is given by $R_d = \rho(PV^{-1})$, where ρ denotes the spectral radius (dominant Eigen value in magnitude) of the next generation matrix PV^{-1}.

It follows that

$$Rd = \frac{\beta}{k1k2k3k4}(A1 + A2 + A3)$$

with,

$$A1 = (1-\zeta)[K1K2(K3 + \eta d\gamma_u) + K1\eta f \tau\gamma_u],$$
$$A2 = \xi K(1-\omega)[K4(K3 + \eta d\gamma_u) + \eta f \tau\gamma_u],$$
$$A3 = \xi K\omega K2(\eta dK4 + \eta f \tau).$$

B. Numerical Solution

In this section, the model equations are solved numerically while three hypothetical cases are considered to investigate the effective rate of the transmission dynamics of tuberculosis. We make reference to Fig. 4.1 through Fig. 4.4.

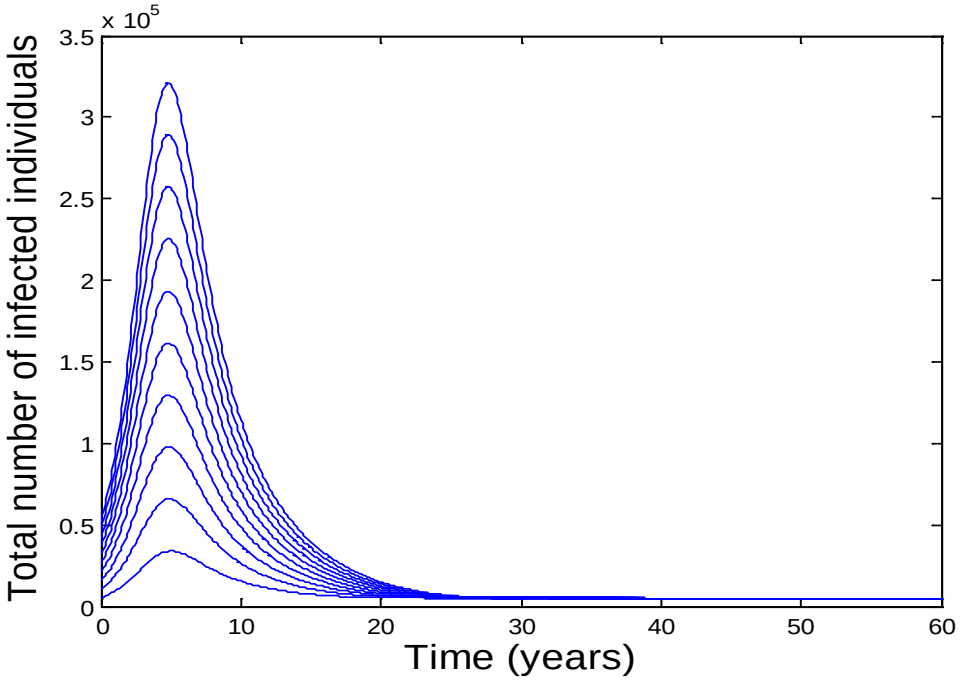

Figure 4.1: Shows the simulation result at $\beta = 2$.
Reference to Table 1. for values of other parameter

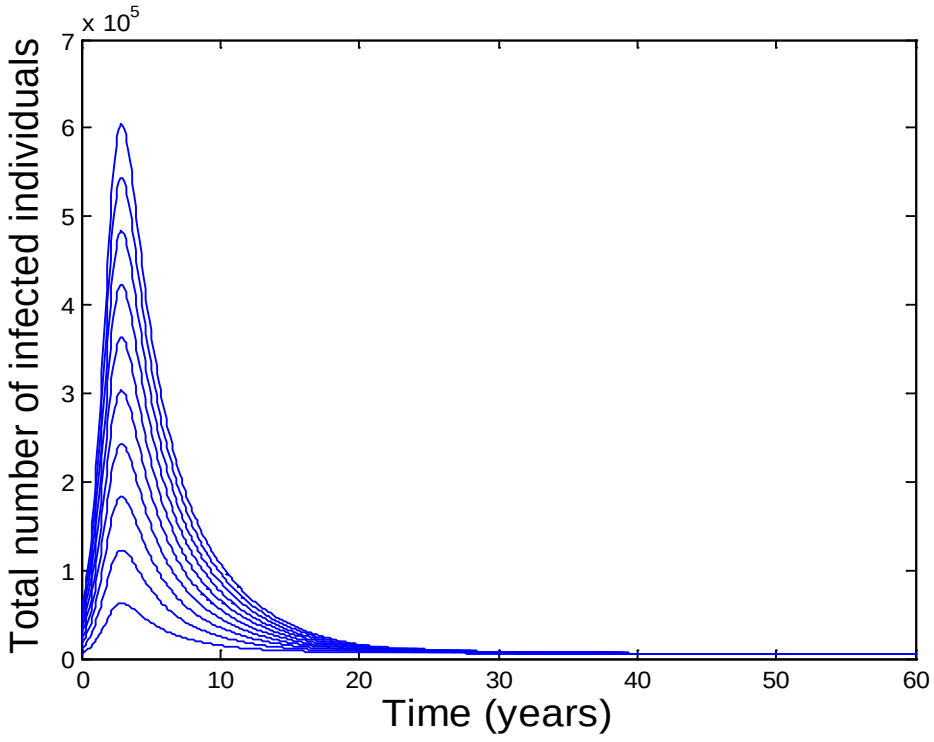

Figure 4.2: Shows the simulation result at $\beta = 4$.
Reference to Table 1. for values of other parameter

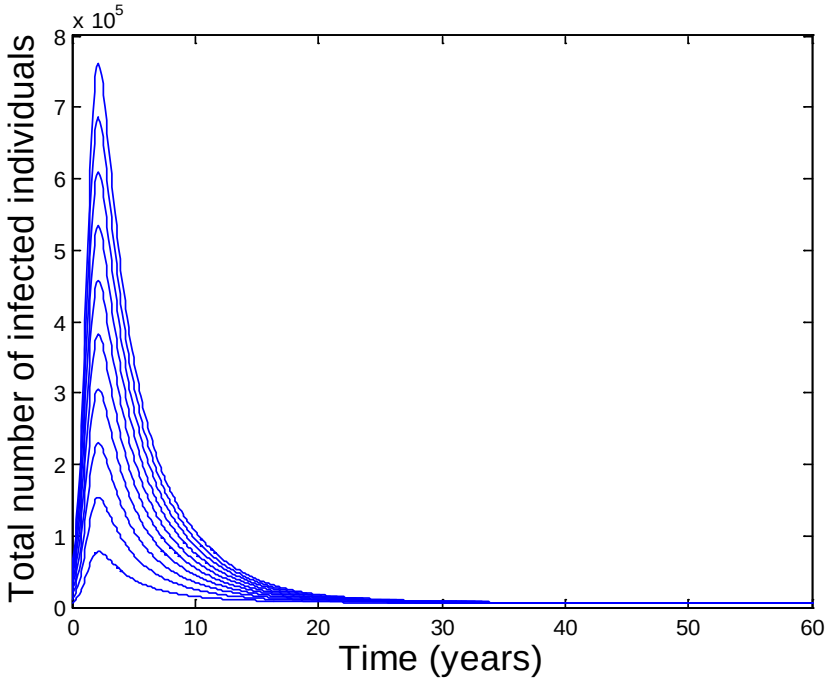

Figure 4.3: Shows the simulation result at $\beta = 6$.
Reference to Table 1. for values of other parameter

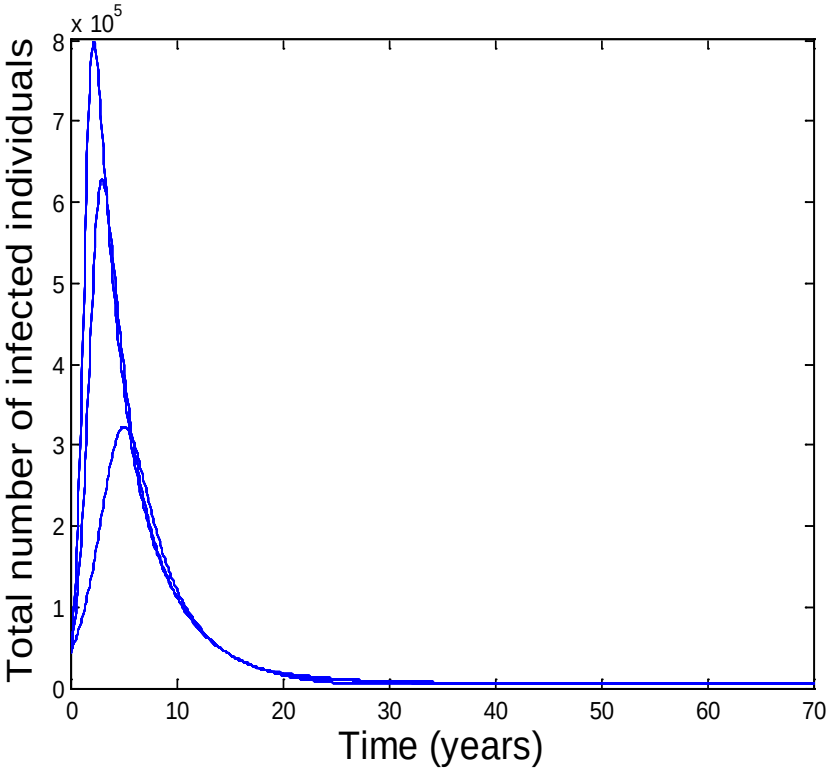

FIG 4.4

Fig 4.4: Shows the simulation result at $\beta = 2, 4, 6$.
Reference to Table 1. for values of other parameter

I. Discussion of results

A compartmental mathematical model to study the transmission dynamics of Tuberculosis was formulated. We also provided solution on how to reduce the spread of the disease in the community using three hypothetical cases and a simulation was also done to get the report of the effective contact rate for the tuberculosis invectives. In Fig4.1, the increase of infected numer of individuals is shown and the number in total for new TB infected persons being a function of time based on the various value of $\beta = 2$. In Fig4.2, the increase of infected numer of individuals is shown and the number in total for new TB infected persons being a function of time based on the various value $\beta = 4$. In Fig4.3, the increase of infected numer of individuals is shown and the number in total for new TB infected persons being a function of time based on the various value of $\beta = 6$. While Fig4.4 describe the comparison between the three at beta equal $2, 4, \text{and } 6$. Reference to [18, 19] for modelling approaches.

A. *Concluding Remarks*

In conclusion, the model analysis showed that when there is no disease in the environment, the disease free equilibrium is stable. Also, as the effective contact rate increases (β), the total number of infective people also increases. That shows that once there is large contact with infected people the disease may take a while to die in the community.

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