



Transmission dynamic of Tuberculosis in two dissimilar groups through pathogens: A SIRS model

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Abstract

Tuberculosis is a communicable disease which spreads in the human population through pathogens. Coughing by the infective individual generate large number of droplets. In this paper, a SIRS mathematical model is proposed to study the transmission of Tuberculosis by droplet infection in two dissimilar groups, considering the economic status of the individuals. The basic reproduction number R_0 from the model has been derived for the study of disease dynamics. It has been shown that the disease free equilibrium point is stable if $R_0 < 1$ and unstable if $R_0 > 1$. It has been also shown that the unique endemic equilibrium point exists when $R_0 > 1$. It may be concluded that if $R_0 < 1$ then the disease will not spread and if $R_0 > 1$ then the disease will be endemic in the population. We have also concluded from the analysis of the model that Tuberculosis can be controlled by reducing the rate at which an infective individual produces pathogens. The analytical results are supported by the relevant graphs.

Keywords

SIRS model, Equilibrium points, Basic Reproduction Number, Stability Analysis.

AMS Subject Classification

34D20, 34D23, 37C75, 49Q12, 90C31, 93C15, 93D05.

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1. Introduction

A Tubercle bacterium (Mycobacterium Tuberculosis) is the principle component of chronic infectious disease like Tu-

berculosis. Tuberculosis (TB) is a bacterial disease with Mycobacterium Tuberculi (MTB) as its primary causative agent. Other Mycobacteria such as Mycobacterium beviies, Mycobacterium africanum, Mycobacterium canetti and Mycobacterium microti are also its causes [1]. It is one of the largest cause of death from an infectious agent in developing countries [2, 3]. Infectious diseases have a profound effect on human populations, including their evolution and cultural development. Despite significant advances in medical science, infectious diseases continue to impact human populations in many parts of the world.

Tuberculosis is spread mainly by droplets of infectious case. Coughing by infective individual generates the large number of droplets [4] and mathematical models have been studied in this regard for the spread of infectious disease [4, 5]. It can also spread through use of an infected persons unsterilised eating utensils and in rare cases a pregnant woman with active TB can infect her foetus (vertical transmission) [6, 7]. Transmission can only occur from people with active TB but not latent TB. This transmission from one person to another depends upon the number of infectious droplets expelled by a carrier, the effectiveness of ventilation, duration

of the exposure and virulence of the MTB strain. The chain of transmission can therefore be broken by isolating patients with active disease and starting effective anti-tuberculosis therapy [7–15].

At present, about 95% of the estimated 8 million new cases of TB occurring each year are in developing countries, where 80% occur among people between the ages of 15–59 years [7]. In sub-Saharan Africa and also in developing countries, TB is the leading cause of mortality, it accounts for an estimated 2 million deaths which accounts for a quarter of avoidable adult deaths [7]. It is known that factors such as endogenous reactivation, emergence of multi-drug resistant TB, and increase in HIV incidence in the recent years call for improved control strategies for TB.

Tuberculosis is one of India’s major public health problems. According to WHO estimates, India has the world’s largest Tuberculosis epidemic. Many research studies have shown the effects and concerns revolving around TDR-TB, In India social and economic positions are still in progression. In Zarir Udhwadia’s report originated from the Hinduja Hospital in Mumbai, India explicitly discusses the drug-resistant effects and results. Approximately 30% of individuals contacted with an active-TB patient become infected and around 10% of infected individuals develop active-TB.[16–19].

In this paper, population is divided into two groups (first group of individuals belonging to economically lower strata and second group of individuals belonging to economically upper strata). Hence the two subpopulations can vary for this TB model which is based on the SIRS model. The basic Reproduction number R_0 is being calculated for determining the existence and stability of disease free equilibrium point and endemic equilibrium point.

It is assumed that the existence of the disease depends on two controlled measures one is rate at which an infective individual produces pathogens (η_1 and η_2) and rate of transmission from pathogens to susceptible humans (β_1 and β_2).

2. Formulation of Model

We consider underlying human population (N) which is divided into two dissimilar groups, one is economically lower strata (N_1) and second is economically upper strata (N_2). Since in the period of illness, most of the infective of higher strata group keep them isolated and because of their higher income status they are able to live without work for the period of illness, but most of the people belonging to lower income group have to go for work even in the illness period, so the production of droplets by higher strata group is less than lower strata group. Tuberculosis infection is more or less uniformly distributed in urban, semi urban and rural areas. Thus the vast majority of cases are to be found in rural and semi urban areas, where more than 80% of the country’s population lives. In urban areas, Tuberculosis is found more frequently in slum dwellers and lower socio-economic groups than in well-to-do groups. i.e. both groups are dissimilar in nature [20].

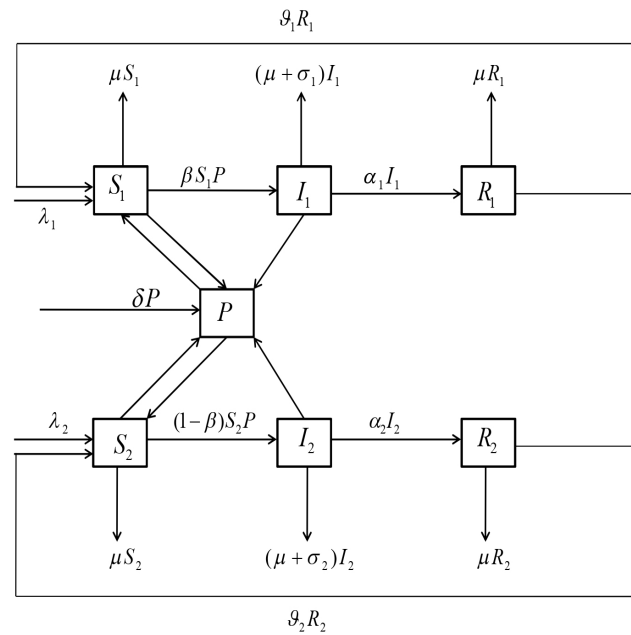


Figure 1. Schematic diagram of the model

Suppose

$$N = N_1 + N_2$$

Further lower strata population N_1 is divided into three classes: Susceptible (S_1), Infected (I_1) and Recovered (R_1) belonging to lower strata group. Similarly upper strata population N_2 is divided into three classes: Susceptible (S_2), Infected (I_2) and Recovered (R_2) belonging to upper strata group.

P is pathogens class which is produced by droplets of infected individuals [4].

In the formulation of this model; λ_1, λ_2 are rate of recruitment of human individuals S_1 and S_2 respectively. The susceptible population S_1 and S_2 decrease due to the infection following contact with pathogens at a constant rate β_1 and β_2 , respectively and by natural death rate μ .

When S_1 and S_2 come in contact with pathogens P , infected population I_1 and I_2 are increased by terms $\beta_1 S_1 P$ and $\beta_2 S_2 P$, respectively and there is decrease in I_1 and I_2 due to disease induced death with the rate of σ_1 and σ_2 , respectively and infected becomes recovered with the rate of $\alpha_1 I_1$ and $\alpha_2 I_2$, respectively, which generate the class R_1 and R_2 class respectively, recovered people become susceptible at the rate of ϑ_1 and ϑ_2 respectively.

We have discussed earlier that infection spread through pathogens produced by droplets from infected persons instead of directed contact of individuals, therefore it is assumed in the model that, the pathogens population P has been generated through infected individual I_1 and I_2 at a constant produce rates η_1 and η_2 , respectively where ($\eta_1 > \eta_2$) [21] and these pathogens are diminished due to natural death at the rate of δ .

A mathematical model for transmission of Tuberculosis by pathogens in two dissimilar groups has been framed with



the help of following system of non linear ordinary differential equations;

$$\frac{dS_1}{dt} = \lambda_1 - \beta_1 S_1 P - \mu S_1 + \vartheta_1 R_1 \tag{2.1}$$

$$\frac{dI_1}{dt} = \beta_1 S_1 P - (\alpha_1 + \mu + \sigma_1) I_1 \tag{2.2}$$

$$\frac{dR_1}{dt} = \alpha_1 I_1 - \mu R_1 - \vartheta_1 R_1 \tag{2.3}$$

$$\frac{dS_2}{dt} = \lambda_2 - \beta_2 S_2 P - \mu S_2 + \vartheta_2 R_2 \tag{2.4}$$

$$\frac{dI_2}{dt} = \beta_2 S_2 P - (\alpha_2 + \mu + \sigma_2) I_2 \tag{2.5}$$

$$\frac{dR_2}{dt} = \alpha_2 I_2 - \mu R_2 - \vartheta_2 R_2 \tag{2.6}$$

$$\frac{dP}{dt} = \eta_1 I_1 + \eta_2 I_2 - \delta P \tag{2.7}$$

With initial conditions

$S_i(0) = S_{i0} > 0, I_i(0) = I_{i0} > 0, R_i(0) = R_{i0} > 0$, for $i = 1, 2$

$$P(0) = P_0 > 0. \tag{2.8}$$

where

λ_i = Recruitment rate of human individuals.

β_i = Transmission rate of infection from pathogens to susceptibles humans.

μ = Natural death rate for human individuals.

ϑ_i = Rate at which recovered individuals become susceptibles.

α_i = Recovery rate.

σ_i = Disease induced death rate.

η_i = Rate at which an infective individual produces pathogens.

δ = Natural death rate for pathogens.

Now $N_1 = S_1 + I_1 + R_1$ and $N_2 = S_2 + I_2 + R_2$ are total population sizes of lower and upper strata groups respectively.

Differentiating N_1 w. r. to t , we have;

$$\frac{dN_1}{dt} = \lambda_1 - \mu N_1 - \sigma_1 I_1 \tag{2.9}$$

$$\frac{dN_1}{dt} \leq \lambda_1 - \mu N_1$$

$$\frac{dN_1}{dt} + \mu N_1 \leq \lambda_1$$

On solving, we get;

$$\lim_{t \rightarrow \infty} N_1 \leq \frac{\lambda_1}{\mu}$$

And again differentiating N_2 w. r. to t , we have;

$$\frac{dN_2}{dt} = \lambda_2 - \mu N_2 - \sigma_2 I_2 \tag{2.10}$$

$$\frac{dN_2}{dt} \leq \lambda_2 - \mu N_2$$

$$\frac{dN_2}{dt} + \mu N_2 \leq \lambda_2$$

On solving, we get;

$$\lim_{t \rightarrow \infty} N_2 \leq \frac{\lambda_2}{\mu}.$$

Further differentiating P w. r. to t , we have;

$$\frac{dP}{dt} = \eta_1 I_1 + \eta_2 I_2 - \delta P \tag{2.11}$$

$$\frac{dP}{dt} \leq \frac{\eta_1 \lambda_1 + \eta_2 \lambda_2}{\mu} - \delta P$$

$$\frac{dP}{dt} + \delta P \leq \frac{\eta_1 \lambda_1 + \eta_2 \lambda_2}{\mu}$$

On solving, we get;

$$\lim_{t \rightarrow \infty} P \leq \frac{\eta_1 \lambda_1 + \eta_2 \lambda_2}{\mu}$$

Thus all the solutions of the model will lie in the region.

$$\tau = \{ (\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2, \bar{P}) : \bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2, \bar{P} \geq 0 : (\bar{S}_1 + \bar{I}_1 + \bar{R}_1) \leq \frac{\lambda_1}{\mu}, (\bar{S}_2 + \bar{I}_2 + \bar{R}_2) \leq \frac{\lambda_2}{\mu} \text{ and } \bar{P} = \frac{\eta_1 \lambda_1 + \eta_2 \lambda_2}{\mu \delta} \}$$

and clearly τ is a compact positively invariant region in R_7^+ .

3. Existence and Stability Analysis of Equilibrium Points

3.1 Existence of the disease free equilibrium point and basic reproduction number

The model has a disease free equilibrium point $E_0 = (\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2, \bar{P})$ in the region τ .

For this we put

$$\frac{dS_1}{dt} = \frac{dI_1}{dt} = \frac{dR_1}{dt} = \frac{dS_2}{dt} = \frac{dI_2}{dt} = \frac{dR_2}{dt} = \frac{dP}{dt} = 0$$

and $I_1 = R_1 = I_2 = R_2 = P = 0$.

Hence the disease free equilibrium point is

$$E_0 = (\bar{S}_1, \bar{I}_1, \bar{R}_1, \bar{S}_2, \bar{I}_2, \bar{R}_2, \bar{P}) = \left(\frac{\lambda_1}{\mu}, 0, 0, \frac{\lambda_2}{\mu}, 0, 0, 0 \right).$$

Now, we define basic reproduction number R_0 as the number of secondary infections that one infectious individual would create over the duration of the infectious period.

We use the next generation matrix approach described in [22,23] to determine the basic reproduction number. Further,



We consider the equations (2.2), (2.5) and (2.7).

$$f_1(I_1, I_2, P) = \beta S_1 P,$$

$$f_2(I_1, I_2, P) = (1 - \beta)S_2 P \text{ and}$$

$$f_3(I_1, I_2, P) = 0$$

$$V_1(I_1, I_2, P) = (\alpha_1 + \mu + \sigma_1)I_1,$$

$$V_2(I_1, I_2, P) = (\alpha_2 + \mu + \sigma_2)I_2 \text{ and}$$

$$V_3(I_1, I_2, P) = \delta P - \eta_1 I_1 - \eta_2 I_2$$

Now,

$$f = \begin{bmatrix} \frac{\partial f_1}{\partial I_1} & \frac{\partial f_1}{\partial I_2} & \frac{\partial f_1}{\partial P} \\ \frac{\partial f_2}{\partial I_1} & \frac{\partial f_2}{\partial I_2} & \frac{\partial f_2}{\partial P} \\ \frac{\partial f_3}{\partial I_1} & \frac{\partial f_3}{\partial I_2} & \frac{\partial f_3}{\partial P} \end{bmatrix} = \begin{bmatrix} 0 & 0 & \beta_1 \bar{S}_1 \\ 0 & 0 & \beta_2 \bar{S}_2 \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \frac{\partial V_1}{\partial I_1} & \frac{\partial V_1}{\partial I_2} & \frac{\partial V_1}{\partial P} \\ \frac{\partial V_2}{\partial I_1} & \frac{\partial V_2}{\partial I_2} & \frac{\partial V_2}{\partial P} \\ \frac{\partial V_3}{\partial I_1} & \frac{\partial V_3}{\partial I_2} & \frac{\partial V_3}{\partial P} \end{bmatrix} = \begin{bmatrix} (\alpha_1 + \mu + \sigma_1) & 0 & 0 \\ 0 & (\alpha_2 + \mu + \sigma_2) & 0 \\ -\eta_1 & -\eta_2 & \delta \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{(\alpha_1 + \mu + \sigma_1)} & 0 & 0 \\ 0 & \frac{1}{(\alpha_2 + \mu + \sigma_2)} & 0 \\ \frac{\eta_1}{\delta(\alpha_1 + \mu + \sigma_1)} & \frac{\eta_2}{\delta(\alpha_2 + \mu + \sigma_2)} & \frac{1}{\delta} \end{bmatrix}$$

$$fV^{-1} = \begin{bmatrix} \frac{\beta_1 \bar{S}_1 \eta_1}{\delta(\alpha_1 + \mu + \sigma_1)} & \frac{\beta_1 \bar{S}_1 \eta_2}{\delta(\alpha_2 + \mu + \sigma_2)} & \frac{\beta_1 \bar{S}_1}{\delta} \\ \frac{\beta_2 \bar{S}_2 \eta_1}{\delta(\alpha_1 + \mu + \sigma_1)} & \frac{\beta_2 \bar{S}_2 \eta_2}{\delta(\alpha_2 + \mu + \sigma_2)} & \frac{\beta_2 \bar{S}_2}{\delta} \\ 0 & 0 & 0 \end{bmatrix}$$

The characteristic equation of fV^{-1} is as follows:

$$\lambda^2 \left(\lambda - \frac{\beta_1 \bar{S}_1 \eta_1 (\alpha_2 + \mu + \sigma_2) + \beta_2 \bar{S}_2 \eta_2 (\alpha_1 + \mu + \sigma_1)}{\delta(\alpha_1 + \mu + \sigma_1)(\alpha_2 + \mu + \sigma_2)} \right) = 0$$

The dominant eigen value of fV^{-1} is as follows;

$$\frac{\beta_1 \bar{S}_1 \eta_1 (\alpha_2 + \mu + \sigma_2) + \beta_2 \bar{S}_2 \eta_2 (\alpha_1 + \mu + \sigma_1)}{\delta(\alpha_1 + \mu + \sigma_1)(\alpha_2 + \mu + \sigma_2)}$$

Substituting the value of \bar{S}_1 and \bar{S}_2 in above

Therefore the basic reproduction number of above model is given by;

$$R_0 = \frac{\beta_1 \lambda_1 \eta_1 (\alpha_2 + \mu + \sigma_2) + \beta_2 \lambda_2 \eta_2 (\alpha_1 + \mu + \sigma_1)}{\delta \mu (\alpha_1 + \mu + \sigma_1)(\alpha_2 + \mu + \sigma_2)} \quad (3.1)$$

3.2 Stability analysis of the disease free equilibrium point

The variational matrix of the system (2.1-2.7) around disease free equilibrium point E_0 is given by;

$$J_0 = \begin{bmatrix} -\mu & 0 & \vartheta_1 & 0 \\ 0 & -(\alpha_1 + \mu + \sigma_1) & 0 & 0 \\ 0 & \alpha_1 & -(\mu + \vartheta_1) & 0 \\ 0 & 0 & 0 & -\mu \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \eta_1 & 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} 0 & 0 & -\frac{\beta_1 \lambda_1}{\mu} \\ 0 & 0 & \frac{\beta_1 \lambda_1}{\mu} \\ 0 & 0 & 0 \\ 0 & \vartheta_2 & -\frac{\beta_2 \lambda_2}{\mu} \\ -(\alpha_2 + \mu + \sigma_2) & 0 & \frac{\beta_2 \lambda_2}{\mu} \\ \alpha_2 & -(\mu + \vartheta_2) & 0 \\ \eta_2 & 0 & -\delta \end{bmatrix}$$

The characteristic equation of J_0 is as follows:

$$(\mu + \lambda)^2 (\mu + \vartheta_1 + \lambda) (\mu + \vartheta_2 + \lambda)$$

$$(\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3) = 0 \quad (3.2)$$

Clearly four roots of equation (3.2) are negative and remaining three characteristics roots are obtained by solving the following equation:

$$(\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3) = 0$$

where

$$a_1 = b_1 + b_2 + \delta, \quad a_2 = b_1 b_2 + \delta(b_1 + b_2) - (c_1 + c_2),$$

$$a_3 = \delta b_1 b_2 - (c_1 b_2 + c_2 b_1) \quad b_1 = (\alpha_1 + \mu + \sigma_1),$$

$$b_2 = (\alpha_2 + \mu + \sigma_2), \quad c_1 = \frac{\beta_1 \lambda_1}{\mu}, \quad c_2 = \frac{\beta_2 \lambda_2}{\mu}$$

From equation (3.2) we can easily write

$$a_1 = b_1 + b_2 + \delta,$$

$$a_2 = b_1 b_2 + \frac{c_2 b_1}{b_2} + \frac{c_1 b_2}{b_1} + \delta(b_1 + b_2) (1 - R_0),$$

$$a_3 = \delta b_1 b_2 (1 - R_0)$$

Now

$$a_1 a_2 - a_3 = (b_1 + b_2 + \delta) \left(b_1 b_2 + \frac{c_2 b_1}{b_2} + \frac{c_1 b_2}{b_1} \right)$$

$$+ \delta (1 - R_0) (b_1^2 + b_2^2 + \delta (b_1 + b_2) + b_1 b_2).$$

We can easily see that

$$a_1, a_2, a_3 > 0 \text{ if } R_0 < 1,$$

$$a_1 a_2 - a_3 > 0 \text{ if } R_0 < 1$$

We can easily conclude from Hurwitz's theorem that the characteristic equation (3.2) will have negative real roots or negative real parts if the roots are complex under the condition $R_0 < 1$.

Thus we find that the disease free equilibrium point E_0 is locally stable if $R_0 < 1$. However, the disease free equilibrium point E_0 is unstable if $R_0 > 1$.

Theorem 1 The disease free equilibrium point E_0 is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$.

3.3 Existence of the endemic equilibrium point

The endemic equilibrium point is the steady state solution when the disease persists in the population. The endemic equilibrium point, $E_1 = (\hat{S}_1, \hat{I}_1, \hat{R}_1, \hat{S}_2, \hat{I}_2, \hat{R}_2, \hat{P})$ in the region τ is obtained by putting $\frac{dS_1}{dt} = \frac{dI_1}{dt} = \frac{dR_1}{dt} = \frac{dS_2}{dt} = \frac{dI_2}{dt} = \frac{dR_2}{dt} = \frac{dP}{dt} = 0$,

On solving we have;

$$\hat{R}_1 = \left(\frac{\alpha_1}{\mu + \vartheta_1} \right) \hat{I}_1,$$

$$\hat{R}_2 = \left(\frac{\alpha_2}{\mu + \vartheta_2} \right) \hat{I}_2,$$



$$\hat{P} = \left(\frac{\eta_1 \hat{I}_1 + \eta_2 \hat{I}_2}{\delta} \right), \quad \frac{dy_5}{dt} = \beta_2(y_7 \hat{S}_2 + y_4 y_7 + y_4 \hat{P}) - (\alpha_2 + \mu + \sigma_2)y_5 \quad (3.7)$$

$$\hat{S}_1 = \left(\frac{\delta(\alpha_1 + \mu + \sigma_1)}{\beta_1(\eta_1 \hat{I}_1 + \eta_2 \hat{I}_2)} \right) \hat{I}_1,$$

$$\hat{S}_2 = \left(\frac{\delta(\alpha_2 + \mu + \sigma_2)}{\beta_2(\eta_1 \hat{I}_1 + \eta_2 \hat{I}_2)} \right) \hat{I}_2, \quad \frac{dy_6}{dt} = \alpha_2 y_5 - (\mu + \vartheta_2)y_6 \quad (3.8)$$

$$\hat{I}_2 = \frac{1}{\eta_2} \left(\frac{B}{\lambda_1 - (A - C)\hat{I}_1} - \eta_1 \right) \hat{I}_1 \quad \frac{dy_7}{dt} = \eta_1 y_2 + \eta_2 y_5 - \delta y_7 \quad (3.9)$$

Substituting these values in (2.2) and we get

$$f(\hat{I}_1) = D_0 (\hat{I}_1)^2 + D_1 (\hat{I}_1) + D_2 = 0$$

where

$$D_0 = \frac{\eta_1}{\eta_2} (A - C) [B_1(A - C) - B(A_1 - C_1)],$$

$$D_1 = - \left(\frac{BB_1(A-C)}{\eta_2} (R_0 - 1) + \frac{B^2(A_1-C_1)}{\eta_2} + \frac{\eta_1 \lambda_1}{\eta_2} [B_1(A - C) - B(A_1 - C_1)] \right)$$

$$D_2 = \frac{BB_1 \lambda_1}{\eta_2} (R_0 - 1),$$

$$A = (\alpha_1 + \mu + \sigma_1), B = \frac{\mu \delta (\alpha_1 + \mu + \sigma_1)}{\beta_1}, C = \frac{\alpha_1 \vartheta_1}{\mu + \vartheta_1},$$

$$A_1 = (\alpha_2 + \mu + \sigma_2), B_1 = \frac{\mu \delta (\alpha_2 + \mu + \sigma_2)}{\beta_2}, C_1 = \frac{\alpha_2 \vartheta_2}{\mu + \vartheta_2},$$

$$R_0 = \frac{\beta_1 \lambda_1 \eta_1 (\alpha_2 + \mu + \sigma_2) + \beta_2 \lambda_2 \eta_2 (\alpha_1 + \mu + \sigma_1)}{\delta (\alpha_1 + \mu + \sigma_1) (\alpha_2 + \mu + \sigma_2)}$$

$$D_0 = +ve \text{ if } A > C \text{ and } B_1(A - C) > B(A_1 - C_1),$$

$$D_1 = -ve \text{ if } A > C, A_1 > C_1, R_0 > 1 \text{ and } B_1(A - C) > B(A_1 - C_1)$$

$$D_2 = +ve \text{ if } R_0 > 1,$$

if $A > C, A_1 > C_1, R_0 > 1$ and $B_1(A - C) > B(A_1 - C_1)$ then $D_0 > 0, D_1 < 0$ and $D_2 > 0$. Therefore by descart's rule of sign, equation has two positive real roots.

Theorem 2 The system (2.1-2.7) has a unique endemic equilibrium whenever $R_0 > 1$ and no positive endemic equilibrium when $R_0 < 1$.

3.4 Stability analysis of the endemic equilibrium point

Applying the transforms,

$$S_1 = \hat{S}_1 + y_1, I_1 = \hat{I}_1 + y_2, R_1 = \hat{R}_1 + y_3, S_2 = \hat{S}_2 + y_4, I_2 = \hat{I}_2 + y_5, R_2 = \hat{R}_2 + y_6 \text{ and } P = \hat{P} + y_7 \text{ on model system (2.1-2.7)}$$

We have;

$$\frac{dy_1}{dt} = -\beta_1(y_7 \hat{S}_1 + y_1 y_7 + y_1 \hat{P}) - \mu y_1 + \vartheta_1 y_3 \quad (3.3)$$

$$\frac{dy_2}{dt} = \beta_1(y_7 \hat{S}_1 + y_1 y_7 + y_1 \hat{P}) - (\alpha_1 + \mu + \sigma_1)y_2 \quad (3.4)$$

$$\frac{dy_3}{dt} = \alpha_1 y_2 - (\mu + \vartheta_1)y_3 \quad (3.5)$$

$$\frac{dy_4}{dt} = -\beta_2(y_7 \hat{S}_2 + y_4 y_7 + y_4 \hat{P}) - \mu y_4 + \vartheta_2 y_6 \quad (3.6)$$

Linearizing the above system, we get the following linear system of differential equations;

$$\frac{dy_1}{dt} = -\beta_1(y_7 \hat{S}_1 + y_1 \hat{P}) - \mu y_1 + \vartheta_1 y_3 \quad (3.10)$$

$$\frac{dy_2}{dt} = \beta_1(y_7 \hat{S}_1 + y_1 \hat{P}) - (\alpha_1 + \mu + \sigma_1)y_2 \quad (3.11)$$

$$\frac{dy_3}{dt} = \alpha_1 y_2 - (\mu + \vartheta_1)y_3 \quad (3.12)$$

$$\frac{dy_4}{dt} = -\beta_2(y_7 \hat{S}_2 + y_4 \hat{P}) - \mu y_4 + \vartheta_2 y_6 \quad (3.13)$$

$$\frac{dy_5}{dt} = \beta_2(y_7 \hat{S}_2 + y_4 \hat{P}) - (\alpha_2 + \mu + \sigma_2)y_5 \quad (3.14)$$

$$\frac{dy_6}{dt} = \alpha_2 y_5 - (\mu + \vartheta_2)y_6 \quad (3.15)$$

$$\frac{dy_7}{dt} = \eta_1 y_2 + \eta_2 y_5 - \delta y_7 \quad (3.16)$$

Consider a positive definite function U as follows;

$$U = \frac{1}{2} (H_1 y_1^2 + H_2 y_2^2 + H_3 y_3^2 + H_4 y_4^2 + H_5 y_5^2 + H_6 y_6^2 + H_7 y_7^2)$$

Differentiating U w. r. to t and using linear system (3.10-3.16) in $\frac{dU}{dt}$ we get;

$$\frac{dU}{dt} = H_1 y_1 (-\beta_1(y_7 \hat{S}_1 + y_1 \hat{P}) - \mu y_1 + \vartheta_1 y_3)$$

$$+ H_2 y_2 (\beta_1(y_7 \hat{S}_1 + y_1 \hat{P}) - (\alpha_1 + \mu + \sigma_1)y_2)$$

$$+ H_3 y_3 (\alpha_1 y_2 - (\mu + \vartheta_1)y_3)$$

$$+ H_4 y_4 (-\beta_2(y_7 \hat{S}_2 + y_4 \hat{P}) - \mu y_4 + \vartheta_2 y_6)$$

$$+ H_5 y_5 (\beta_2(y_7 \hat{S}_2 + y_4 \hat{P}) - (\alpha_2 + \mu + \sigma_2)y_5)$$



$$+H_6y_6(\alpha_2y_5 - (\mu + \vartheta_2)y_6) + H_7y_7(\eta_1y_2 + \eta_2y_5 - \delta y_7) \tag{3.17}$$

On arranging the terms of (3.17) we get;

$$\begin{aligned} \frac{dU}{dt} = & -b_{11}y_1^2 + b_{13}y_1y_3 + b_{17}y_1y_7 + b_{12}y_1y_2 + b_{27}y_2y_7 - b_{22}y_2^2 \\ & + b_{23}y_2y_3 - b_{33}y_3^2 - b_{44}y_4^2 + b_{46}y_4y_6 + b_{47}y_4y_7 + b_{45}y_4y_5 \\ & + b_{57}y_5y_7 - b_{55}y_5^2 + b_{56}y_5y_6 - b_{66}y_6^2 - b_{77}y_7^2 \end{aligned}$$

where

$$\begin{aligned} b_{11} = & (\beta_1\hat{P} + \mu)H_1, \quad b_{13} = \vartheta_1H_1, \quad b_{17} = -\beta_1\hat{S}_1H_1, \\ b_{12} = & \beta_1\hat{P}H_2, \quad b_{27} = \beta_1\hat{S}_1H_2 + \eta_1H_7, \quad b_{22} = (\alpha_1 + \mu + \sigma_1)H_2, \\ b_{23} = & \alpha_1H_3, \quad b_{33} = (\mu + \vartheta_1)H_3, \quad b_{44} = (\beta_2\hat{P} + \mu)H_4, \\ b_{46} = & \vartheta_2H_4, \quad b_{47} = -\beta_2\hat{S}_2H_4, \quad b_{45} = \beta_2\hat{P}H_5, \\ b_{57} = & \beta_2\hat{S}_2H_5 + \eta_2H_7, \quad b_{55} = (\alpha_2 + \mu + \sigma_2)H_5, \\ b_{56} = & \alpha_2H_6, \quad b_{66} = (\mu + \vartheta_2)H_6, \quad b_{77} = \delta H_7. \end{aligned}$$

Further rearranging the terms of $\frac{dU}{dt}$ we get;

$$\begin{aligned} \frac{dU}{dt} = & -\left(\frac{b_{11}}{3}y_1^2 - b_{13}y_1y_3 + \frac{b_{33}}{2}y_3^2\right) \\ & + \left(\frac{b_{11}}{3}y_1^2 - b_{17}y_1y_7 + \frac{b_{77}}{4}y_7^2\right) + \left(\frac{b_{22}}{3}y_2^2 - b_{27}y_2y_7 + \frac{b_{77}}{4}y_7^2\right) \\ & + \left(\frac{b_{22}}{3}y_2^2 - b_{23}y_2y_3 + \frac{b_{33}}{2}y_3^2\right) + \left(\frac{b_{44}}{3}y_4^2 - b_{46}y_4y_6 + \frac{b_{66}}{2}y_6^2\right) \\ & + \left(\frac{b_{44}}{3}y_4^2 - b_{47}y_4y_7 + \frac{b_{77}}{4}y_7^2\right) + \left(\frac{b_{44}}{3}y_4^2 - b_{45}y_4y_5 + \frac{b_{55}}{3}y_5^2\right) \\ & + \left(\frac{b_{55}}{3}y_5^2 - b_{57}y_5y_7 + \frac{b_{77}}{4}y_7^2\right) + \left(\frac{b_{11}}{3}y_1^2 - b_{12}y_1y_2 + \frac{b_{22}}{3}y_2^2\right) \\ & + \left(\frac{b_{55}}{3}y_5^2 - b_{56}y_5y_6 + \frac{b_{66}}{2}y_6^2\right) \tag{3.18} \end{aligned}$$

Using the Sylvester criteria on the right hand side of (3.18), it can be shown that $\frac{dU}{dt}$ is negative define if the following conditions are being satisfied;

$$\begin{aligned} \frac{(\beta_1\hat{P} + \mu)(\mu + \vartheta_1)H_1H_3}{3} & > \frac{(\vartheta_1H_1)^2}{2}, \\ \frac{(\beta_1\hat{P} + \mu)\delta H_1H_7}{3} & > (\beta_1\hat{S}_1H_1)^2, \\ \frac{(\beta_1\hat{P} + \mu)(\alpha_1 + \mu + \sigma_1)H_1H_2}{9} & > \frac{(\beta_1\hat{P}H_2)^2}{4}, \\ \frac{(\alpha_1 + \mu + \sigma_1)\delta H_2H_7}{3} & > (\beta_1\hat{S}_1H_2 + \eta_1H_7)^2, \tag{3.19} \\ \frac{(\alpha_1 + \mu + \sigma_1)(\mu + \vartheta_1)H_2H_3}{3} & > (\alpha_1H_3)^2, \end{aligned}$$

Table 1. parameter values used in simulations

$\lambda_1=25/\text{day},$	$\lambda_2=20/\text{day},$	$\beta_1=0.0003/\text{day},$
$\beta_2=0.00028/\text{day},$	$\alpha_1=0.07/\text{day},$	$\alpha_2=0.09/\text{day},$
$\sigma_1=0.045/\text{day},$	$\sigma_2=0.040/\text{day},$	$\vartheta_1 =0.09/\text{day},$
$\vartheta_2=0.085/\text{day},$	$\delta=0.09/\text{day},$	$\mu=0.015/\text{day}.$

$$\begin{aligned} \frac{(\beta_2\hat{P} + \mu)(\mu + \vartheta_2)H_4H_6}{3} & > \frac{(\vartheta_2H_4)^2}{2}, \\ \frac{(\beta_2\hat{P} + \mu)\delta H_4H_7}{3} & > (\beta_2\hat{S}_2H_4)^2, \\ \frac{(\beta_2\hat{P} + \mu)(\alpha_2 + \mu + \sigma_2)H_4H_5}{9} & > \frac{(\beta_2\hat{P}H_5)^2}{4}, \\ \frac{(\alpha_2 + \mu + \sigma_2)\delta H_5H_7}{3} & > (\beta_2\hat{S}_2H_5 + \eta_2H_7)^2, \\ \frac{(\alpha_2 + \mu + \sigma_2)(\mu + \vartheta_2)H_5H_6}{3} & > \frac{(\alpha_2H_6)^2}{2} \end{aligned}$$

Hence from the lyapunov’s theorem it may be concluded the equilibrium point E_1 is globally stable under the conditions given in system (3.19).

Theorem 3 The endemic equilibrium point E_1 is globally stable if condition (3.19) holds otherwise unstable.

4. Graphs and Conclusion

In this paper, SIRS model is analysed for the transmission dynamics of Tuberculosis by pathogens infection in two dissimilar groups. It has been shown that there exists a feasible region where the model is well defined and where all (disease free and endemic) equilibrium points can be obtained. Basic reproduction number R_0 has also been derived. It has been shown that disease free equilibrium point is stable if $R_0 < 1$ and unstable if $R_0 > 1$. It has also been shown that an unique endemic equilibrium point (Positive) exists only when $R_0 > 1$.

Finally, with the help of numerical values of parameter given in Table 1 graphs has been plotted. Figure 2 is plotted between infective versus t for η_1 and η_2 for which R_0 is less than one which indicates disease free state as all trajectories for infectious moves towards origin. Figure 3, 4 and 5 are plotted between infective versus t for those values of η_1 and η_2 for which R_0 is more than one which indicates endemic state as all trajectories for infectious does not move towards origin. In Figure 6 graph between I_1 versus t has been shown from reflecting the effect of η_1, η_2 on R_0 and I_1 similarly, in Figure 7 graph between I_2 versus t has been show for reflecting the effect of η_1, η_2 on R_0 and I_2 .

It can be observed from the figures that rates η_1 and η_2 decreased by treatment of infected individuals. Eventually Tuberculosis will be controlled.

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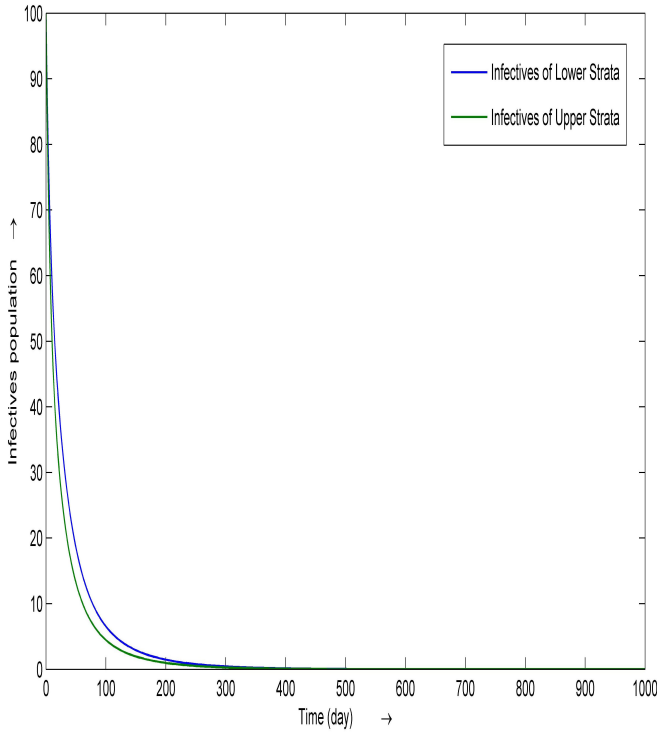


Figure 2. Graph between I_1 and I_2 versus t for $\eta_1 = 0.012$ and $\eta_2 = 0.01$ which corresponds to $R_0 = 0.7989$

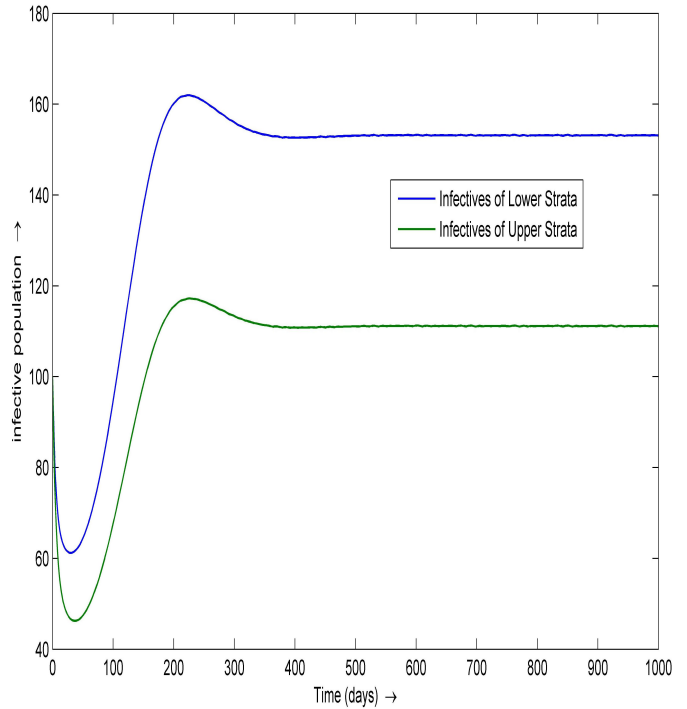


Figure 4. Graph between I_1 and I_2 versus t for $\eta_1 = 0.025$ and $\eta_2 = 0.022$ which corresponds to $R_0 = 1.6978$

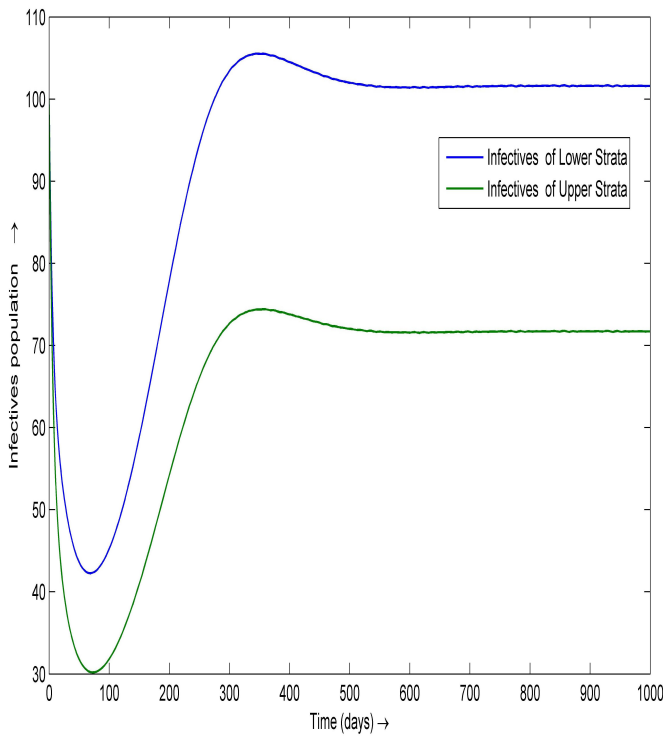


Figure 3. Graph between I_1 and I_2 versus t for $\eta_1 = 0.02$ and $\eta_2 = 0.018$ which corresponds to $R_0 = 1.3696$

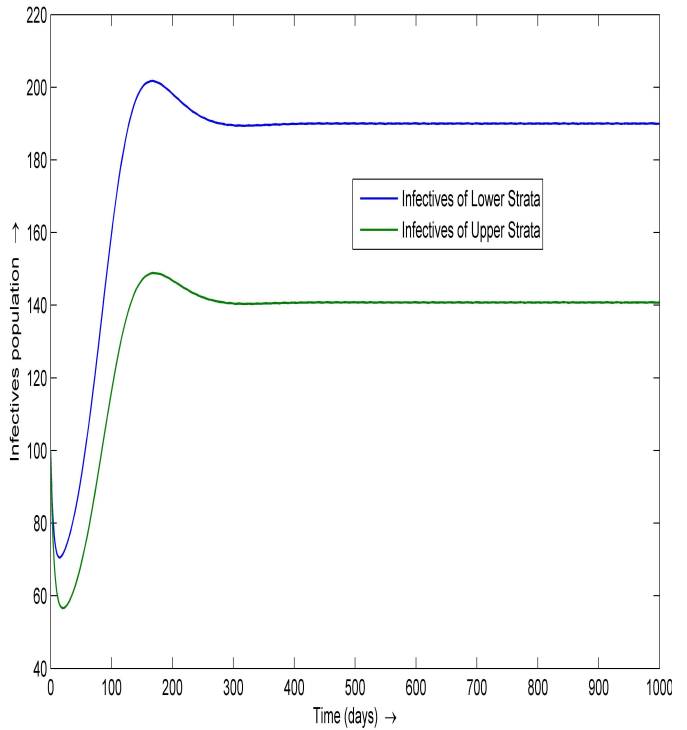


Figure 5. Graph between I_1 and I_2 versus t for $\eta_1 = 0.03$ and $\eta_2 = 0.027$ which corresponds to $R_0 = 2.0545$



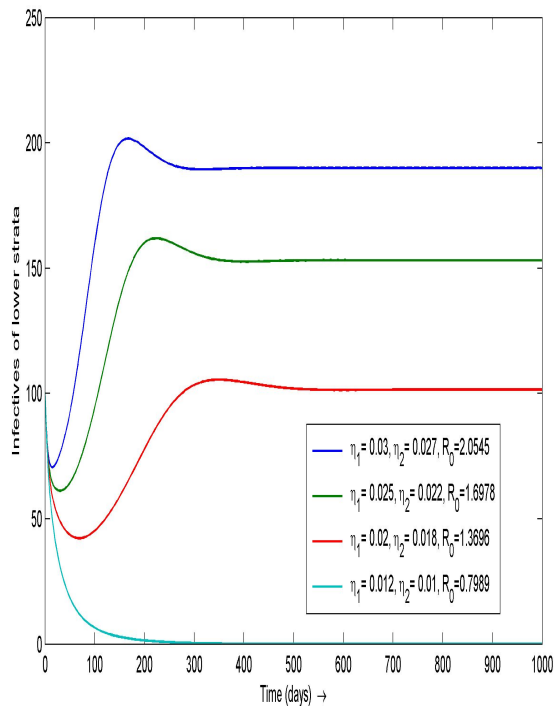


Figure 6. Variation in the infectives of lower strata group for different values of η_1, η_2

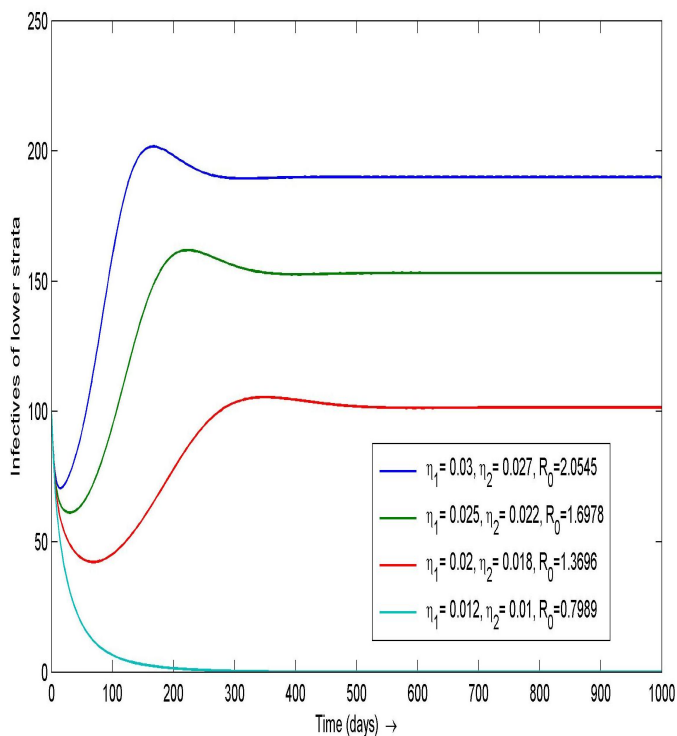


Figure 7. Variation in the infectives of upper strata group for different values of η_1, η_2

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