Modelling of HIV-TB Co-infection Transmission Dynamics

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Abstract In this paper, we have formulated a model for HIV-TB co-infection using differential equations in order to understand the dynamics of disease spread. The model is analysed for all the parameters responsible for the disease spread in order to find the most sensitive parameters out of all. Steady state conditions are derived. A threshold parameter $R_0$ is defined and is shown that the disease will spread only if its value exceeds 1. Numerical simulation is done for the model using MATLAB which shows the population dynamics in different compartments.

Keywords: mathematical modelling, differential equations, numerical simulation, HIV/AIDS, tuberculosis


1. Introduction

Tuberculosis is the most common HIV-related opportunistic infection in world, and caring for patients with both diseases is a major public health challenge. India has about 1.8 million new cases of tuberculosis annually, accounting for a fifth of new cases in the world - a greater number than in any other country. Patients with latent Mycobacterium tuberculosis infection are at higher risk for progression into active TB if they are co-infected with HIV. In recent decades, the dramatic spread of the HIV epidemic in sub-Saharan Africa has resulted in notification rates of TB increasing up to 10 - times. The incidence of TB is also increasing in other high HIV prevalence countries where the population with HIV infection and TB overlap. This is the underlying factor that suggests that TB control will not make much head way in HIV prevalent settings unless HIV control is also achieved.

The related spread of two or more infections has always been a cause of concern for human beings. HIV-TB co-infection is the largest cause among them. Tuberculosis (TB) is the most common opportunistic disease affecting HIV positive people and the leading cause of death in patients with AIDS.

As per the World Health Organisation (WHO) estimation, one third of the world’s population is infected with Mycobacterium Tuberculosis. All over the world, approximately 15% of TB patients have HIV co-infection. HIV patients are at higher risk for catching primary TB infection as well as reactivation of latent TB infection. HIV infection primarily affects those components of host immune system responsible for cell mediated immunity. These cells help us fight against various infections. Once they are destroyed our body’s resistance to fight infections goes down. Thus if a person with latent TB infection catches HIV infection, then the host’s immunity reduces resulting in active tuberculosis. Moreover the infection is poorly contained following reactivation, resulting in widespread dissemination causing extra-pulmonary disease.

The interaction between HIV and tuberculosis in patients who are co-infected is bidirectional. HIV infection accelerates the activation of tuberculosis and tuberculosis accelerates the HIV infection developing into AIDS. Also, the relative risk of death and development of other opportunistic infections is higher in HIV-TB co-infected patients as compared to those having only one disease out of two.

Research work is already in progress for understanding the dynamics of this co-epidemic. Krischner modelled the CD4+ cell counts and viral load in the case of two infections together [1]. Morris formed basic models and derived steady states for them [2]. Pawlowski et al reviewed the available literature in order to highlight immunological events responsible for developing the one infection in the presence of the other [3]. Escombe et al analysed how infectiousness of a co-infected person differs from the one having only TB [4]. Sharomi et al formulated the mathematical model and also included the treatment factor in it [5]. Baur et al examined that how the latent TB patient moves to active TB class if it catches HIV-1 infection too [7]. Shah et al developed a model for tuberculosis to explain the dynamics of disease taking pulmonary and extra-pulmonary TB separately [8]. Shah et al also developed a model to predict future trends of HIV/AIDS [9].

2. Mathematical Model

The entire population is divided into twelve compartments which are (1) Susceptible (S) (i.e. no
disease), (2) Latent TB – No HIV(E1), (3) Latent TB with HIV(E2), (4) Active TB – No HIV(I1), (5) Active TB with HIV (I2), (6) No TB but HIV (H), (7) TB treated – No HIV (T1), (8) TB treated with HIV (T2), (9) AIDS – No TB (A1), (10) AIDS with Latent TB (A2), (11) AIDS with Active TB (A3), (12) AIDS Treated TB (A4).

Here, it is assumed that a person when moves any of AIDS class, does not spread disease any more. The population dynamics among these compartments is shown in Figure 1:

![Figure 1. Flow of Population in Various compartments](image)

The state variables and the parameters used in model formulation are as follows:

- **S**: Number of susceptible (i.e. no disease)
- **E1**: Number of persons with latent TB infection and no HIV
- **E2**: Number of persons with latent TB infection and HIV positive
- **H**: Number of persons with HIV infection and no TB
- **I1**: Number of persons with active TB infection and no HIV
- **I2**: Number of persons with active TB infection and HIV positive
- **T1**: Number of persons with treated TB and no HIV
- **T2**: Number of persons with treated TB and HIV positive
- **A1**: Number of persons with AIDS and no TB
- **A2**: Number of persons with AIDS with latent TB infection
- **A3**: Number of persons with AIDS with active TB infection
- **A4**: Number of persons with AIDS with treated TB infection
- **B**: Recruitment rate in susceptible class
- **μ**: Natural death rate
- **δA**: AIDS induced death rate
- **δT**: Tuberculosis induced death rate
- **β1**: Probability of transmission of TB infection from an infective to a susceptible per contact per unit time
- **β2**: Probability of transmission of HIV infection from an infective to a susceptible per contact per unit time
- **c1**: Number of contacts made by a person from active TB class
- **c2**: Number of contacts made by a person having only HIV and no TB
- **c3**: Number of contacts made by a person having HIV and TB both
- **α**: Rate with which all type of infective develop AIDS.
- **τ1**: Rate of progression of individuals from the latent TB class to the active class who have only LTB and no HIV.
- **τ2**: Rate of progression of individuals from the LT class to the active TB class who have LTB and HIV/AIDS both.
- **γ1**: treatment rate of latent TB individuals
- **γ2**: treatment rate of infectious (active TB) individuals.

The model equations are given below:

\[
\frac{dS}{dt} = B - \frac{\beta_1 S}{N}(c_1 I_1 + c_4 I_2 + c_4 A_3) - \frac{\beta_2 S}{N}(c_3 H + c_4 E_2 + c_4 T_2) - \mu S
\]

\[
\frac{dE_1}{dt} = \frac{\beta_1 S}{N}(c_1 I_1 + c_4 I_2 + c_4 A_3) + \frac{\beta_3 I_1}{N}(c_1 I_1 + c_4 I_2 + c_4 A_3) - \frac{\beta_1 E_1}{N}(c_3 H + c_4 T_2) - (\mu + \alpha_1 + \gamma_1) E_1
\]

\[
\frac{dH}{dt} = \frac{\beta_1 H}{N}(c_1 I_1 + c_4 I_2 + c_4 A_3) - \frac{\beta_1 H}{N}(c_1 I_1 + c_4 I_2 + c_4 A_3) - (\mu + \alpha_2 + \nu_2 + \gamma_1) E_2
\]

\[
\frac{dI_1}{dt} = \nu_1 E_1 - \frac{\beta_1 I_1}{N}(c_3 H + c_4 T_2 + c_4 E_2 + c_4 I_2) - (\mu + \alpha_3 + \nu_2 + \gamma_1) I_1
\]

\[
\frac{dI_2}{dt} = \nu_2 E_2 + \frac{\beta_1 I_1}{N}(c_3 H + c_4 T_2 + c_4 E_2 + c_4 I_2) - (\mu + \alpha_4 + \nu_2 + \gamma_1) I_2
\]

\[
\frac{dT_1}{dt} = \gamma_1 E_1 + \gamma_2 I_1 - \frac{\beta_1 T_1}{N}(c_1 I_1 + c_4 I_2 + c_4 A_3) - \frac{\beta_1 T_1}{N}(c_3 H + c_4 E_2 + c_4 T_2) - \mu T_1 - \gamma_1 T_1
\]

\[
\frac{dT_2}{dt} = \gamma_1 E_2 + \gamma_2 I_2 - (\mu + \alpha_5) T_2
\]
\[
\frac{dA_1}{dt} = \alpha_1 H - \frac{\beta A_1}{N} (c_1 I_1 + c_4 I_2 + c_3 A_3) - (\mu + \delta_A) A_1
\]
\[
\frac{dA_2}{dt} = \alpha_2 E_2 + \frac{\beta A_1}{N} (c_1 I_1 + c_4 I_2 + c_3 A_3)
- (\mu + \delta_A + \nu_2 + \gamma_1) A_2
\]
\[
\frac{dA_3}{dt} = \alpha_3 I_2 + \nu_2 A_2 - (\mu + \delta_A + \delta_T + \gamma_2) A_3
\]
\[
\frac{dA_4}{dt} = \gamma_1 A_2 + \gamma_2 A_3 + \alpha_2 T_2 - (\mu + \delta_A) A_4
\]
We name this above set of twelve equations as system (1).

With
\[
N = S + E_1 + E_2 + H + I_1 + I_2 + T_1 + T_2 + A_1 + A_2 + A_3 + A_4
\]
\[
\Rightarrow \frac{dN}{dt} = B - \mu N - \delta_A (A_1 + A_2 + A_3 + A_4) - \delta_T (I_1 + I_2 + A_3)
\]
\[
\Rightarrow \frac{dN}{dt} \leq B - \mu N \Rightarrow \lim_{t \to \infty} \text{sup} N \leq \frac{B}{\mu}
\]
So, the feasible region for the system is
\[
\Lambda = \left\{ \left( E_1, E_2, H, I_1, I_2, T_1, T_2, A_1, A_2, A_3, A_4 \right) : \right. \\
(S + E_1 + E_2 + H + I_1 + I_2 + T_1 + T_2 + A_1 + A_2 + A_3 + A_4) \leq \frac{B}{\mu}, S > 0, E_1 \geq 0,
I_1 > 0, H > 0, E_2 \geq 0, I_2 \geq 0, T_1 \geq 0, T_2 \geq 0,
A_1 \geq 0, A_2 \geq 0, A_3 \geq 0, A_4 \geq 0
\}
\]
Let \( E \left( \bar{S}, E_1, E_2, \bar{H}, T_1, T_2, \bar{A_1}, \bar{A_2}, \bar{A_3}, \bar{A_4} \right) \) be the equilibrium point of the model given above.

Since, the recruitment term B can never be zero and population cannot vanish, therefore there is no trivial equilibrium point like
\[
E \left( \bar{S}, E_1, E_2, \bar{H}, T_1, T_2, \bar{A_1}, \bar{A_2}, \bar{A_3}, \bar{A_4} \right) = (0, 0, 0, 0, 0, 0, 0, 0, 0).
\]
So, let
\[
E \left( \bar{S}, E_1, E_2, \bar{H}, \bar{T_1}, \bar{T_2}, \bar{A_1}, \bar{A_2}, \bar{A_3}, \bar{A_4} \right) = (\bar{S}, 0, 0, 0, 0, 0, 0, 0, 0, 0).
\]
Then system of equations at this point gives
\[
\bar{S} = \frac{B}{\mu}
\]
So, we can see that there is a disease free equilibrium at
\[
E_0 \left( \bar{S}, E_1, E_2, \bar{H}, \bar{T_1}, \bar{T_2}, \bar{A_1}, \bar{A_2}, \bar{A_3}, \bar{A_4} \right) = \left( \frac{B}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0, 0 \right).
\]
Let
\[
X' = E \left( E_1, E_2, H, I_1, I_2, T_1, T_2, A_1, A_2, A_3, A_4, S \right)^T.
\]
Therefore,
\[
X' = \frac{dX}{dt} = \mathcal{F} (X) \cdot \mathcal{V} (X)
\]
where \( \mathcal{F} (X) \) and \( \mathcal{V} (X) \) are column matrices given by
\[
\mathcal{F} (X) = \begin{bmatrix}
\frac{\beta S}{N} (c_1 I_1 + c_4 I_2 + c_3 A_3) \\
\frac{\beta T_1}{N} (c_1 I_1 + c_4 I_2 + c_3 A_3) \\
\frac{\beta S}{N} (c_3 H + c_4 E_2 + c_4 T_2) \\
\frac{\beta I_1}{N} (c_3 H + c_4 T_2 + c_4 E_2 + c_4 I_2) \\
0 \\
\frac{\beta I_1}{N} (c_3 H + c_4 T_2 + c_4 E_2 + c_4 I_2) \\
0 \\
0 \\
0 \\
\end{bmatrix}
\]
and
\[
\mathcal{V} (X) = \begin{bmatrix}
\frac{\beta S}{N} (c_1 I_1 + c_4 I_2 + c_3 A_3) + (\mu + \alpha_1) H - (\mu + \delta_A + \nu_2 + \gamma_1) E_1 \\
+ (\mu + \alpha_1 + \nu_2 + \gamma_1) I_1 \\
+ (\mu + \delta_T + \gamma_2) I_2 \\
\gamma_1 E_1 - \gamma_2 I_1 + \beta T_1 (c_1 I_1 + c_4 I_2 + c_3 A_3) \\
-\gamma_1 E_1 + \gamma_2 I_1 + \beta T_1 (c_1 I_1 + c_4 I_2 + c_3 A_3) \\
\frac{\beta S}{N} (c_3 H + c_4 E_2 + c_4 T_2 + \mu T_1) \\
-\gamma_1 E_1 + \gamma_2 I_1 + \beta T_1 (c_1 I_1 + c_4 I_2 + c_3 A_3) \\
-\alpha_1 H + \beta T_1 (c_1 I_1 + c_4 I_2 + c_3 A_3) + (\mu + \delta_A) A_1 \\
-\alpha_2 E_2 + (\mu + \delta_A + \nu_2 + \gamma_1) A_2 \\
-\alpha_2 I_2 + \nu_2 A_2 + (\mu + \delta_A + \delta_T + \gamma_2) A_2 \\
-\gamma_1 A_2 + \gamma_2 A_2 - \alpha_2 T_2 + (\mu + \delta_A) A_4 \\
-B + \frac{\beta S}{N} (c_1 I_1 + c_4 I_2 + c_3 A_3) \\
+ \frac{\beta S}{N} (c_3 H + c_4 E_2 + c_4 T_2 + \mu S) \\
\end{bmatrix}
\]
The derivatives \( D \mathcal{F} (E_0) \) and \( D \mathcal{V} (E_0) \) at disease free equilibrium point \( E_0 \), are partitioned as
$D\mathcal{F}(E_0) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}$ and $D\mathcal{V}(E_0) = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix}$

where $F$ and $V$ are $10 \times 10$ matrices given by

$$F = \begin{bmatrix}
0 & 0 & 0 & \frac{\beta_1 c_1 B}{\mu N} & \frac{\beta_2 c_1 B}{\mu N} & 0 & 0 & 0 & 0 & 0 \\
0 & \frac{\beta_2 c_1 B}{\mu N} & \frac{\beta_2 c_1 B}{\mu N} & 0 & 0 & \frac{\beta_2 c_1 B}{\mu N} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\end{bmatrix}
$$

and

$$V = \begin{bmatrix}
(\mu + v_1 + \gamma_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & (\mu + a_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & (\mu + a_2 + v_2 + \gamma_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
-\gamma_1 & 0 & 0 & (\mu + \delta_1 + \gamma_2) & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -\gamma_2 & 0 & (\mu + \delta_1 + \gamma_2) & 0 & (\mu + a_1) & 0 & 0 & 0 \\
0 & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\end{bmatrix}
$$

Therefore, basic reproduction number $R_0 = \rho(FV^{-1})$

$= \text{spectral radius of } FV^{-1}$

$\Rightarrow R_0 = \max \left( \frac{\beta_1 c_1 v_1 B}{(\mu + \delta_1 + \gamma_1)(\mu + a_1) \mu N}, \frac{\beta_2 c_1 B}{(\mu + \delta_1 + \gamma_1)(\mu + a_1) \mu N} \right)$

$= \frac{\beta_1 c_1 v_1 B}{(\mu + \delta_1 + \gamma_2)(\mu + \gamma_1 + \gamma_1) \mu N}$

$$J = \begin{bmatrix}
-\mu & 0 & \frac{-\beta c_1 B}{\mu N} & \frac{-\beta c_1 B}{\mu N} & \frac{-\beta c_1 B}{\mu N} & 0 & \frac{-\beta c_1 B}{\mu N} & 0 & 0 & 0 \\
0 & -(\mu + v_1 + \gamma_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -(\mu + a_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -(\mu + a_1 + v_2 + \gamma_1) & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & v_1 & 0 & 0 & 0 & -\mu & 0 & 0 & 0 & 0 \\
0 & 0 & v_2 & 0 & 0 & -\mu & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_1) & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_2) & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_2) & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_2) & 0 \\
\end{bmatrix}
$$

The disease free equilibrium is stable if all the eigenvalues of the Jacobian matrix of the system under consideration have negative real parts.

Here, trace $(J)$ is clearly negative.

Now for det $(J)$, on solving we reach the relation

$$\det(J) = \mu^2 \left( \frac{\beta c_1 v_1 B}{\mu N} \right)^2 \left( \mu + a_1 \right) \left( \mu + a_2 \right)$$

$$\times \left( \mu + a_3 + v_2 + \gamma_1 \right) \left( \mu + a_3 + \delta_1 + \gamma_2 \right)$$

$$\times \left( \mu + a_4 + v_3 + \gamma_1 \right) \left( \mu + a_4 + \delta_1 + \gamma_2 \right)$$

$$\times \left[ \left( \mu + \delta_1 + \gamma_2 \right) \left( \mu + v_1 + \gamma_1 \right) \left( \frac{\beta c_1 v_1 B}{\mu N} \right) \right]$$

3. Stability of Disease Free Equilibrium

The Jacobian of model equations at the disease free equilibrium point can be written as

$$J = \begin{bmatrix}
-\mu & 0 & \frac{-\beta c_1 B}{\mu N} & \frac{-\beta c_1 B}{\mu N} & \frac{-\beta c_1 B}{\mu N} & 0 & \frac{-\beta c_1 B}{\mu N} & 0 & 0 & 0 \\
0 & -(\mu + v_1 + \gamma_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -(\mu + a_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -(\mu + a_1 + v_2 + \gamma_1) & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & v_1 & 0 & 0 & 0 & -\mu & 0 & 0 & 0 & 0 \\
0 & 0 & v_2 & 0 & 0 & -\mu & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_1) & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_2) & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_2) & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \delta_1 + \gamma_2) & 0 \\
\end{bmatrix}
$$
For \( \det(J) > 0 \), we have

\[
\mu^2 \left( \mu + \delta_1 \right)^2 \left( \mu + \alpha_1 \right) \left( \mu + \alpha_2 \right) \left( \mu + \alpha_2 + v_2 + \gamma_1 \right) \\
\times \left( \mu + \alpha_2 + \delta_T + \gamma_2 \right) \left( \mu + \delta_A + v_2 + \gamma_1 \right) \left( \mu + \delta_A + \delta_T + \gamma_2 \right) \\
\times \left[ \left( \mu + \delta_T + \gamma_2 \right) \left( \mu + v_1 + \gamma_1 \right) - \frac{\beta_1 \gamma_1 B}{\mu N} \right] > 0
\]

or

\[
\left[ \left( \mu + \delta_T + \gamma_2 \right) \left( \mu + v_1 + \gamma_1 \right) - \frac{\beta_1 \gamma_1 B}{\mu N} \right] > 0
\]

\[
\Rightarrow \frac{\beta_1 \gamma_1 B}{\left( \mu + \delta_T + \gamma_2 \right) \left( \mu + v_1 + \gamma_1 \right) \mu N} < 1 \Rightarrow R_0 < 1
\]

This shows that the disease free equilibrium is locally asymptotically stable if \( R_0 < 1 \) otherwise unstable.

4. Sensitivity Analysis

Sensitivity indices of \( R_0 \) to all the different parameters tell us that how crucial each parameter is to the disease spread. This helps us choose the right parameter(s) responsible for making the scenario endemic.

The values of different parameters used in model formulation are given in Table 1, [8,9,10,11,12].

[For formula refer A.1.]

We calculate the sensitivity indices for those parameters on which the value of basic reproduction depends. The results are given in Table 2.

These results show that the total population size, new recruitments as susceptible, contact rate with tuberculosis patients and probability of transmission of TB have a constant effect on the scenario. The most important parameters reflected here and need to be addressed are rate of progression of LTB to ATB and their treatment rates. So, if we try to control tuberculosis cases and treat the active TB cases more promptly then we can reduce the level of intensity of this lethal co-epidemic.

<table>
<thead>
<tr>
<th>Table 1. Values of Different Parameters used in Model</th>
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<td>Parameter</td>
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</tr>
<tr>
<td>B</td>
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<td>( \mu )</td>
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<table>
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<tr>
<th>Table 2. Sensitivity Indices of ( R_0 ) to the Parameters</th>
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<td>Parameter</td>
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<td>( N )</td>
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<td>( \beta_1 )</td>
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5. Numerical Simulation

This technique help us foresee the future trends of an epidemic and thus help us be ready for future or take safety measures today for making a better future. Here, we take a sample population of 35000. The results of simulation in different compartments are shown in following Figure 2-Figure 5:

![Figure 2. Simulation of Susceptible, LTB, HIV and LTB with HIV](image-url)
Here, we formulated a mathematical model using ordinary differential equations for HIV-TB co-infection scenario. We divided the entire population into twelve compartments. Then a relation for basic reproduction number $R_0$ is established. Steady state conditions are derived which show that the disease free equilibrium is locally asymptotically stable only if $R_0 < 1$. Sensitivity analysis results tell us that we need to work more rigorously in order to control this co-epidemic. Numerical simulation is done using MATLAB. Figure 5 shows the trends of population in different compartments in next 40 years.
Acknowledgement

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Appendix

A.1 The normalised forward sensitivity index of a variable, u, that depends continuously on a parameter, p, is defined as

$$ \gamma_p^u = \frac{\partial u}{\partial p} \cdot \frac{p}{u} $$

References