The Impact of Post-Exposure Prophylaxis (PEP) On the Sub-Model of the Transmission Dynamics of HIV/TB Co-Infection in A HIV Endemic Area, Where both HIV and Tb Are Receiving Other Treatments.

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Abstract: A sub-model for the transmission dynamics of HIV/TB co-infection with controls in a HIV endemic area was formulated using differential equations. The impact of post-exposure prophylaxis was considered given that both HIV and TB patients are receiving other treatment. The parameters responsible for the diseases spread were analyzedin other to find the most sensitive of them all. The effective basic reproduction number, R_0 of the systems was obtained and shown that the disease will spread only if $R_0 > 1$ and would die off with time, if $R_0 < 1$. Numerical Simulations carried out using MATLAB on the model showedthat with the judicious and increased use of post-exposure prophylaxis, the disease will phase out rapidly in the population.
Key words: Post-exposure prophylaxis, basic reproduction number, endemic equilibrium point, sensitivity index.

I. Introduction

Acquired immune deficiency syndrome (AIDS) is caused by Human Immune-deficiency virus (HIV), and has caused major public health and socio-economic challenges globally. Since the beginning of the epidemics in 1980s, more than 70 million people have been effected and it has claimed about 35 million lives.

Globally, 36.9 million people are said to be living with HIV as at 2017. An estimated 0.8% of Adults aged 15-49 years worldwide are living with HIV, although the burden of the epidemics continues to vary between countries and region. World Health Organization (WHO) maintains that African region remains the most severely affected with 4.1% of the Adults living with HIV, accounting for merely two-thirds of the people living with HIV worldwide. There is no known cure or vaccine for AIDS. However antiretroviral (ART) treatment improves the health of the infected individuals, pro-longs the life, and subsequently reduces the risk of the infectivity of victim. According to Silva; *et al* (2015), with the increased use of ATR, the number of AIDS death has declined with around 1.6 million AIDS death in 2012 down from 2.3 million in 2005. Though HIV exposed individual's duration is too short (72 hours), if effectively treated with post-exposure prophylaxis one may not be infected with HIV (AIDS gov.(2015)).

Our focus is to investigate the impact of this post-exposure prophylaxis on the sub-model of HIV/TB co-infection dynamics where both diseases are receiving other treatments.

II. Mathematical Model Formulation

We have the entire population divided into six compartments. The susceptible (S), HIV exposed class (E_H), infectious HIVclass that are undergoing treatment (I_{TH}), infectious HIV/ active TB with only HIV on treatment (I_{TC}), infectious HIV/ Latent TB with HIV on treatment (I_{TE}) and infectious HIV/TB with both on treatment (I_{TCT})

Since our focus is on the HIV endemic area, it is assumed that one first gets infected with HIV, thereby compromising the immune system, before being co-infected with TB. The drugs used for both diseases are

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assumed to be effective and that the latent TB, active TB infected individuals that are receiving treatment can no longer transmit the disease. HIV infectious individuals do not progress to AIDS because of ART drugs.

Mycobacterial tuberculosis is the cause of most occurrence of tuberculosis (TB) and is usually acquired via airborne from someone who has active TB. TB is the most common opportunistic disease affecting people that have had their immune system compromised.

The schematic diagram for this sub-model which is derived from the general flow diagram (Asogwa, *et al*; 2019) is given as:
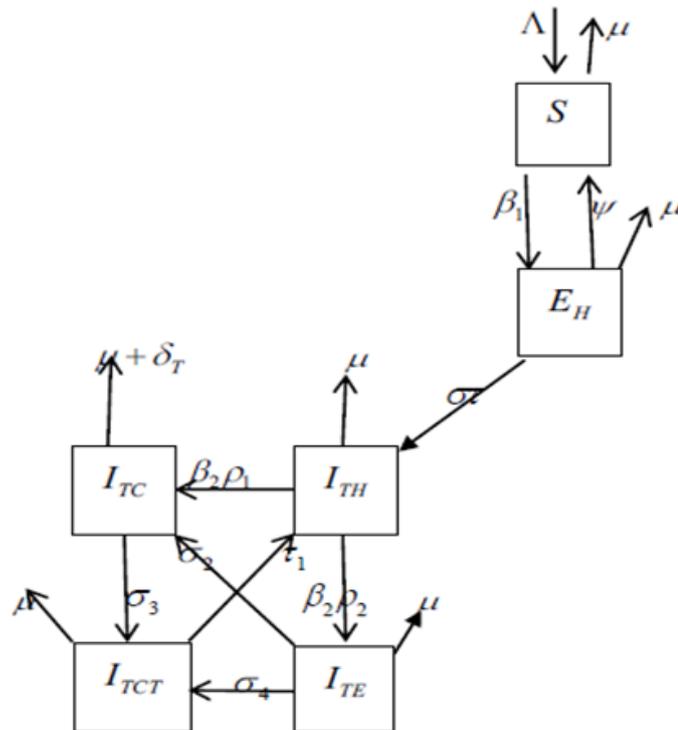
Figure 2.1: *The schematic diagram of the transmission dynamics of HIV/TB co-infection sub-model with both diseases on treatment*

2.0 Model Variables Of The HIV/TB Co-Infection

S :   Susceptible individuals

E_H :   Individuals who are exposed to HIV. Those infected but are not yet infectious, that is within 72 hours of being exposed to the infection.

I_{TH} :   Individuals who are infectious of HIV and undergoing treatment.

I_{TE} :   Individuals being treated of HIV and are latently infected with TB.

I_{TC} :   Individuals being treated of infectious HIV and now infected with active TB.

I_{TCT} :   HIV infectious individuals with TB where both are being treated.

2.1   OTHER PARAMETERS USED IN THE MODEL

Λ **:**   The number of individuals that enter into the susceptible class either by birth or migration.

β_1 :   The rate at which susceptible individuals that had contact with infective HIV individuals become infected with HIV.

β_2 :   The rate at which individuals with HIV infection that had contact with active TB individuals become infected with TB.

ψ **:**   The rate of the recovery of exposed HIV individuals due to post-exposure prophylaxis, back to the susceptible class.

τ :   The rate at which individuals exposed to HIV become infectious of HIV.

σ :   The proportion of infectiveHIV individualsthat progress to HIV infective classon treatment (I_{TH}).

τ_1 :   The rate at which individuals with TB and HIV, treated of TB, fully recover from the TB disease.

n_1, n_2, n_3, n_4 :   Different infectivity rates of HIV classes, while m_2 is the infectivity rate of active TB class.

μ :   Natural mortality /death rate.

δ_T :   TB-induced mortality/death rate.

ρ_1 :   Proportion of infected HIV individuals being treated of HIV that are also being co-infected with active TB.

$\rho_2 = 1 - \rho_1$:   The proportion of individuals infected and being treated of HIV that are also being co-infected with latent TB.

σ_2 :   The rate of progression of TB from latent TB with infective HIV being treated to active TB with infective HIV being treated.

σ_3 :   The rate at which active TB with HIV being treated class also starts receiving TB treatment.

σ_4 :   The rate at which individual that is being treated of HIV with latent TB starts receiving TB treatment.
The following model was then built with the help of the diagram;

2.2   MODEL EQUATIONS ON HIV/TB CO-INFECTION WITH AND WITHOUT HIV TREATMENT
. Thus the sub-model for the six compartments are given as below:

$$\frac{dS}{dt} = \Lambda + \psi E_H - (\beta_1 + \mu)S \qquad\qquad 2.2a$$

$$\frac{dE_H}{dt} = \beta_1 S - (\psi + \sigma\tau + \mu)E_H \qquad\qquad 2.2b$$

$$\frac{dI_{TH}}{dt} = \sigma\tau E_H + \tau_1 I_{TCT} - ((\rho_1 + \rho_2)\beta_2 + \mu)I_{TH} \;,$$

$$= \sigma\tau E_H + \tau_1 I_{TCT} - (\beta_2 + \mu)I_{TH} \qquad \text{since } \rho_1 + \rho_2 = 1 \qquad 2.2c$$

$$\frac{dI_{TC}}{dt} = \beta_2 \rho_1 I_{TH} + \sigma_2 I_{TE} - (\sigma_3 + \mu + \delta_T)I_{TC} \qquad\qquad 2.2d$$

$$\frac{dI_{TE}}{dt} = \beta_2 \rho_2 I_{TH} - (\sigma_2 + \sigma_4 + \mu)I_{TE} \qquad 2.2e$$

$$\frac{dI_{TCT}}{dt} = \sigma_3 I_{TC} + \sigma_4 I_{TE} - (\tau_1 + \mu)I_{TCT} \qquad\qquad 2.2f$$

where β_1, the rate of infectivity of HIV is defined as

$$\beta_1 = \frac{k_H (n_1 I_{TH} + n_2 I_{TCT} + n_3 I_{TE} + n_4 I_{TC})}{N_1}$$

since the sub-groups $I_{TH}, I_{TCT}, I_{TE}, I_{TC}$ are the HIV infectious classes while $\dfrac{K_H}{N_1}$ Is the force of infection.

β_2, the rate of infectivity of TB is defined as $\beta_2 = \dfrac{k_T (m_2 I_{TC})}{N_1}$.

We assume that TB individuals on TB drugs cannot transmit the disease any more. Thus only I_{TC} individuals are the ones in the sub-groups that can transmit the TB infection to I_{TH}, (the susceptible class for TB). $\dfrac{K_T}{N_1}$ is the force of infection. For this sub-model, $N_1 = S + E_H + I_{TH} + I_{TE} + I_{TCT} + I_{TC}$. Thus,

$\Omega_1 = \{(S, E_H, I_{TH}, I_{TE}, I_{TCT}, I_{TC}) \in \Re_+^6 \}$ is the feasible region for the sub model and it is positively-invariant.

2.3 EXISTENCE AND STABILITY ANALYSIS OF DISEASE FREE EQUILIBRIUM STATE (DFE), ε_1

Let $\varepsilon_1(\overset{*}{S}, \overset{*}{E_H}, \overset{*}{I_{TH}}, \overset{*}{I_{TC}}, \overset{*}{I_{TE}}, \overset{*}{T_{TCT}})$ be the equilibrium point of the model, 2.2(a-f) above. Since the

recruitment term, Λ can never vanish, that is $N_1 \neq 0$, there exist no trivial equilibrium point, like

$\varepsilon_1(\overset{*}{S}, \overset{*}{E_H}, \overset{*}{I_{TH}}, \overset{*}{I_{TC}}, \overset{*}{I_{TE}}, \overset{*}{T_{TCT}}) = (0,0,0,0,0,0)$;

So let $\varepsilon_1(\overset{*}{S}, \overset{*}{E_H}, \overset{*}{I_{TH}}, \overset{*}{I_{TC}}, \overset{*}{I_{TE}}, \overset{*}{T_{TCT}}) = (S,0,0,0,0,0)$

Theorem 2.1

A disease free equilibrium state for model exist at the point

$\varepsilon_1(\overset{*}{S}, \overset{*}{E_H}, \overset{*}{I_{TH}}, \overset{*}{I_{TC}}, \overset{*}{I_{TE}}, \overset{*}{T_{TCT}}) = (\dfrac{\Lambda}{\mu}, 0,0,0,0,0)$

Proof:

At equilibrium state, the rate of change is equal to zero. That is,

Let

$(S, E_H, I_{TH}, I_{TE}, I_{TC}, I_{TCT}) = (\overset{*}{S}, \overset{*}{E_H}, \overset{*}{I_{TH}}, \overset{*}{I_{TC}}, \overset{*}{I_{TE}}, \overset{*}{T_{TCT}})$ at equilibrium state. Thus we have

from the system

$\dfrac{d \overset{*}{S}}{dt} = \Lambda + \psi \overset{*}{E_H} - (\beta_1 + \mu) \overset{*}{S} = 0$

$\Rightarrow \Lambda - \mu \overset{*}{S} = 0$ since $\psi, \beta_1 = 0$ (disease free)

Therefore, $\overset{*}{S} = \dfrac{\Lambda}{\mu}$

$\dfrac{d \overset{*}{E_H}}{dt} = \beta_1 \overset{*}{S} - (\psi + \sigma\tau + \mu) \overset{*}{E_H} = 0$

$\Rightarrow -\mu \overset{*}{E_H} = 0$ since $\beta_1 = \psi = \tau = 0$ (no infection)

In the same manner, we have the disease free equilibrium point as

$\varepsilon_1(\overset{*}{S}, \overset{*}{E_H}, \overset{*}{I_{TH}}, \overset{*}{I_{TC}}, \overset{*}{I_{TE}}, \overset{*}{T_{TCT}}) = (\dfrac{\Lambda}{\mu}, 0,0,0,0,0)$

2.4 THE STABILITY ANALYSIS OF THE DISEASE FREE EQUILIBRIUM OF THIS SUB-MODEL

The disease free equilibrium, ε_1, is stable if all the eigenvalues of the Jacobian matrix of the system have negative real parts. Let the system at the disease free equilibrium point be represented by the following:

$g_1 = \Lambda + \psi E_H - (\beta_1 + \mu)S = 0$

$g_2 = \beta_1 S - (\psi + \sigma\tau + \mu)E_H = 0$

$g_3 = \sigma\tau E_H + \tau_1 I_{TCT} - (\beta_2 + \mu)I_{TH} = 0$

$g_4 = \beta_2 \rho_1 I_{TH} + \sigma_2 I_{TE} - (\sigma_3 + \mu + \delta_T)I_{TC} = 0$

$g_5 = \beta_2 \rho_2 I_{TH} - (\sigma_2 + \sigma_4 + \mu)I_{TE} = 0$

$$g_6 = \sigma_3 I_{TC} + \sigma_4 I_{TE} - (\tau_1 + \mu) I_{TCT} = 0$$

Note that,

$$\beta_1 S = \frac{k_H (n_1 I_{TH} + n_2 I_{TCT} + n_3 I_{TE} + n_4 I_{TC}) \Lambda}{\mu N_1} \quad \text{since,} \quad S = \frac{\Lambda}{\mu} \quad \text{and}$$

$$\beta_2 I_{TH} = \frac{k_T (m_2 I_{TC}) I_{TH}}{N_1} = 0 \quad \text{at the disease free equilibrium point.}$$

We linearize the system of equation to get the Jacobian matrix, $J(\varepsilon_1)$. Taking λ^* as the eigenvalue, Let

$$J(\varepsilon_1) = \begin{vmatrix}
\frac{\partial g_1}{\partial S} - \lambda^* & \frac{\partial g_1}{\partial E_H} & \frac{\partial g_1}{\partial I_{TH}} & \frac{\partial g_1}{\partial I_{TC}} & \frac{\partial g_1}{\partial I_{TE}} & \frac{\partial g_1}{\partial I_{TCT}} \\
\frac{\partial g_2}{\partial S} & \frac{\partial g_2}{\partial E_H} - \lambda^* & \frac{\partial g_2}{\partial I_{TH}} & \frac{\partial g_2}{\partial I_{TC}} & \frac{\partial g_2}{\partial I_{TE}} & \frac{\partial g_2}{\partial I_{TCT}} \\
\frac{\partial g_3}{\partial S} & \frac{\partial g_3}{\partial E_H} & \frac{\partial g_3}{\partial I_{TH}} - \lambda^* & \frac{\partial g_3}{\partial I_{TC}} & \frac{\partial g_3}{\partial I_{TE}} & \frac{\partial g_3}{\partial I_{TCT}} \\
\frac{\partial g_4}{\partial S} & \frac{\partial g_4}{\partial E_H} & \frac{\partial g_4}{\partial I_{TH}} & \frac{\partial g_4}{\partial I_{TC}} - \lambda^* & \frac{\partial g_4}{\partial I_{TE}} & \frac{\partial g_4}{\partial I_{TCT}} \\
\frac{\partial g_5}{\partial S} & \frac{\partial g_5}{\partial E_H} & \frac{\partial g_5}{\partial I_{TH}} & \frac{\partial g_5}{\partial I_{TC}} & \frac{\partial g_5}{\partial I_{TE}} - \lambda^* & \frac{\partial g_5}{\partial I_{TCT}} \\
\frac{\partial g_6}{\partial S} & \frac{\partial g_6}{\partial E_H} & \frac{\partial g_6}{\partial I_{TH}} & \frac{\partial g_6}{\partial I_{TC}} & \frac{\partial g_6}{\partial I_{TE}} & \frac{\partial g_6}{\partial I_{TCT}} - \lambda^*
\end{vmatrix} = 0$$

Therefore, we have:

$$J(\varepsilon_1) = \begin{vmatrix}
-\mu - \lambda^* & 0 & \frac{-n_1 k_H}{N_1} & \frac{-n_4 k_H}{N_1} & \frac{-n_3 k_H}{N_1} & \frac{-n_2 k_H}{N_1} \\
0 & -\left(\begin{matrix}\psi + \sigma\tau \\ + \mu\end{matrix}\right) - \lambda^* & \frac{n_1 k_H}{N_1} & \frac{n_4 k_H}{N_1} & \frac{n_3 k_H}{N_1} & \frac{n_2 k_H}{N_1} \\
0 & \sigma\tau & -\mu - \lambda^* & 0 & 0 & \tau_1 \\
0 & 0 & 0 & -\left(\begin{matrix}\sigma_3 + \mu \\ + \delta_T\end{matrix}\right) - \lambda^* & \sigma_2 & 0 \\
0 & 0 & 0 & 0 & -\left(\begin{matrix}\sigma_2 + \sigma_4 \\ + \mu\end{matrix}\right) - \lambda^* & 0 \\
0 & 0 & 0 & \sigma_3 & \sigma_4 & -(\tau_1 + \mu) - \lambda^*
\end{vmatrix} = 0$$

Simplifying we have;

$$\left(-\mu - \lambda^* \right)\left(\left(-\left(\begin{matrix}\psi + \sigma\tau \\ + \mu\end{matrix}\right) - \lambda^* \right) \begin{vmatrix} -\mu - \lambda^* & 0 & 0 & \tau_1 \\ 0 & -\left(\begin{matrix}\sigma_3 + \mu \\ + \delta_T\end{matrix}\right) - \lambda^* & \sigma_2 & 0 \\ 0 & 0 & -\left(\begin{matrix}\sigma_3 + \sigma_4 \\ + \mu\end{matrix}\right) - \lambda^* & 0 \\ 0 & \sigma_4 & \sigma_4 & -(\tau_1 + \mu) - \lambda^* \end{vmatrix} - \sigma\tau \begin{vmatrix} \frac{n_1 k_H}{N_1} & \frac{n_4 k_H}{N_1} & \frac{n_3 k_H}{N_1} & \frac{n_2 k_H}{N_1} \\ 0 & -\left(\begin{matrix}\sigma_3 + \mu \\ + \delta_T\end{matrix}\right) - \lambda^* & \sigma_2 & 0 \\ 0 & 0 & -\left(\begin{matrix}\sigma_3 + \sigma_4 \\ + \mu\end{matrix}\right) - \lambda^* & 0 \\ 0 & \sigma_3 & \sigma_4 & -(\tau_1 + \mu) - \lambda^* \end{vmatrix} \right) = 0$$

$$\left(-\mu-\lambda^*\right)\left(\left(\left(-\binom{\psi+\sigma\tau}{+\mu}-\lambda^*\right)\left(-\mu-\lambda^*\right)\left(-\left(\tau_1+\mu\right)-\lambda_*\right)^*\left(-\binom{\sigma_3+\mu}{+\delta_T}-\lambda^*\right)\left(-\binom{\sigma_3+\sigma_4}{+\mu}-\lambda^*\right)-\sigma\tau\left(\frac{n_1k_H}{N_1}\right)\left(-\left(\tau_1+\mu\right)-\lambda\right)^*\left(-\binom{\sigma_3+\mu}{+\delta_T}-\lambda^*\right)\left(-\binom{\sigma_3+\sigma_4}{+\mu}-\lambda^*\right)\right)=0$$

$$\left(-\mu-\lambda^*\right)\left(-\left(\tau_1+\mu\right)-\lambda^*\right)\left(-\binom{\sigma_3+\mu}{+\delta_T}-\lambda^*\right)\left(-\binom{\sigma_3+\sigma_4}{+\mu}-\lambda^*\right)\left(\left(-\binom{\psi+\sigma\tau}{+\mu}-\lambda^*\right)\left(-\mu-\lambda^*\right)-\sigma\tau\left(\frac{n_1k_H}{N_1}\right)\right)=0$$

Thus;

$$\left(-\mu-\lambda^*\right)=0 \quad \Rightarrow \lambda^*=-\mu \qquad\qquad\qquad ...(i)$$

$$\left[-\left(\tau_1+\mu\right)-\lambda^*\right]=0 \quad \Rightarrow \lambda^*=-\left(\tau_1+\mu\right) \qquad\qquad ...(ii)$$

$$\left[-\binom{\sigma_3+\mu}{+\delta_T}-\lambda^*\right]=0 \quad \Rightarrow \lambda^*=-\left(\sigma_3+\mu+\delta_T\right) \qquad\qquad ...(iii)$$

$$\left[-\binom{\sigma_3+\sigma_4}{+\mu}-\lambda^*\right]=0 \quad \Rightarrow \lambda^*=-\left(\sigma_3+\sigma_4+\mu\right) \qquad\qquad ...(iv)$$

$$\left(\left(-\binom{\psi+\sigma\tau}{+\mu}-\lambda^*\right)\left(-\mu-\lambda^*\right)-\sigma\tau\left(\frac{n_1k_H}{N_1}\right)\right)=0 \qquad ...(v)$$

From (v), we have;

$$\left(\mu\binom{\psi+\sigma\tau}{+\mu}+\mu\lambda^*+\lambda^*\binom{\psi+\sigma\tau}{+\mu}+\lambda^{*2}-\sigma\tau\left(\frac{n_1k_H}{N_1}\right)\right)=0$$

$$\lambda^{*2}+\lambda^*\left(\mu+\binom{\psi+\sigma\tau}{+\mu}\right)+\mu\binom{\psi+\sigma\tau}{+\mu}-\sigma\tau\left(\frac{n_1k_H}{N_1}\right)=0$$

$$\lambda^*=\frac{-\left(\mu+\binom{\psi+\sigma\tau}{+\mu}\right)\pm\sqrt{\left(\mu+\binom{\psi+\sigma\tau}{+\mu}\right)^2-4\left(\mu\binom{\psi+\sigma\tau}{+\mu}-\sigma\tau\left(\frac{n_1k_H}{N_1}\right)\right)}}{2}$$

Clearly,

Real parts of the eigenvalues of the Jacobian matrix $J(\varepsilon_1)$ are all negatives. Thus, the disease free equilibrium of the system is asymptotically stable.

III.   Computation Of The Effective Basic Reproduction Number, R_{01}

We also use the next generation operator approach described by Diekmann and Heesterbeek (2000) and subsequently analyzed by Van den Driessche and Watmough (2002). Using this technique, we obtain the effective basic reproduction number, R_{01}, of the system (2.2a-f) which is the spectra radius, (ρ), of the next generation matrix, FV^{-1}, that is $R_{01}=\rho(FV^{-1})$. Both F and V are obtained from the Jacobian matrix of linearized system about DFE. F is the matrix of the new infection terms and V, the matrix of the transition terms. Note that the matrices F and V are formed from the coefficients of the infected classes $(E_H, I_{TH}, I_{TC}, I_{TE}, I_{TCT})$. The spectral radius here means the maximum eigenvalues of FV^{-1}.

Let $X'=\varepsilon(\overset{*}{E_H},\overset{*}{I_{TH}},\overset{*}{I_{TC}},\overset{*}{I_{TE}},\overset{*}{T_{TCT}},\overset{*}{S})^T$

Therefore $\quad X' = \dfrac{dX}{dt} = F(x) - V(x)$

Where $F(x)\, and\, V(x)$ are column matrix given as follows;

$$F(x) = \begin{bmatrix} \dfrac{K_H S}{N_1}(n_1 I_{TH} + n_2 I_{TCT} + n_3 I_{TE} + n_4 I_{TC}) \\ 0 \\ \dfrac{K_T \rho_1 I_{TH}}{N_1}(m_2 I_{TC}) \\ \dfrac{K_T \rho_2 I_{TH}}{N_1}(m_2 I_{TC}) \\ 0 \\ 0 \end{bmatrix} \quad \text{and}$$

$$V(x) = \begin{bmatrix} (\psi + \tau\sigma + \mu)E_H \\ -\sigma \mathcal{E}_H - \tau_1 I_{TCT} + (\rho_1 + \rho_2)\dfrac{K_T}{N_1}(m_2 I_{TC})I_{TH} + \mu I_{TH} \\ -\sigma_2 I_{TE} + (\sigma_3 + \mu + \delta_T)I_{TC} \\ (\sigma_2 + \sigma_4 + \mu)I_{TE} \\ -\sigma_3 I_{TC} - \sigma_4 I_{TE} + (\tau_1 + \mu)I_{TCT} \\ -\Lambda - \psi E_H + \dfrac{K_H}{N_1}(n_1 I_{TH} + n_2 I_{TCT} + n_3 I_{TE} + n_4 I_{TC})S \end{bmatrix}$$

The sixth row is a disease free row and thus will not be considered in the computation of the reproductive number, R_{01}.

The derivatives $DF(\varepsilon_0)$ and $DV(\varepsilon_0)$ at disease free equilibrium, DFE, point, ε_0 are partitioned as

$$DF(\varepsilon_0) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad DV(\varepsilon_0) = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix}$$

Where $F\, and\, V$ are 5x5 matrices given by

$$F = \begin{bmatrix} \dfrac{dF_1}{dE_H} & \dfrac{dF_1}{dI_{TH}} & \dfrac{dF_1}{dI_{TC}} & \dfrac{dF_1}{dI_{TE}} & \dfrac{dF_1}{dI_{TCT}} \\ \dfrac{dF_2}{dE_H} & \dfrac{dF_2}{dI_{TH}} & \dfrac{dF_2}{dI_{TC}} & \dfrac{dF_2}{dI_{TE}} & \dfrac{dF_2}{dI_{TCT}} \\ \dfrac{dF_3}{dE_H} & \dfrac{dF_3}{dI_{TH}} & \dfrac{dF_3}{dI_{TC}} & \dfrac{dF_3}{dI_{TE}} & \dfrac{dF_3}{dI_{TCT}} \\ \dfrac{dF_4}{dE_H} & \dfrac{dF_4}{dI_{TH}} & \dfrac{dF_4}{dI_{TC}} & \dfrac{dF_4}{dI_{TE}} & \dfrac{dF_4}{dI_{TCT}} \\ \dfrac{dF_5}{dE_H} & \dfrac{dF_5}{dI_{TH}} & \dfrac{dF_5}{dI_{TC}} & \dfrac{dF_5}{dI_{TE}} & \dfrac{dF_5}{dI_{TCT}} \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \dfrac{dV_1}{dE_H} & \dfrac{dV_1}{dI_{TH}} & \dfrac{dV_1}{dI_{TC}} & \dfrac{dV_1}{dI_{TE}} & \dfrac{dV_1}{dI_{TCT}} \\ \dfrac{dV_2}{dE_H} & \dfrac{dV_2}{dI_{TH}} & \dfrac{dV_2}{dI_{TC}} & \dfrac{dV_2}{dI_{TE}} & \dfrac{dV_2}{dI_{TCT}} \\ \dfrac{dV_3}{dE_H} & \dfrac{dV_3}{dI_{TH}} & \dfrac{dV_3}{dI_{TC}} & \dfrac{dV_3}{dI_{TE}} & \dfrac{dV_3}{dI_{TCT}} \\ \dfrac{dV_4}{dE_H} & \dfrac{dV_4}{dI_{TH}} & \dfrac{dV_4}{dI_{TC}} & \dfrac{dV_4}{dI_{TE}} & \dfrac{dV_4}{dI_{TCT}} \\ \dfrac{dV_5}{dE_H} & \dfrac{dV_5}{dI_{TH}} & \dfrac{dV_5}{dI_{TC}} & \dfrac{dV_5}{dI_{TE}} & \dfrac{dV_5}{dI_{TCT}} \end{bmatrix}$$

The linear stability of ε_0 can then be established using the next generation operator method on the system.

At disease free equilibrium, $S = \dfrac{\Lambda}{\mu}$, Thus

$$\beta_1 S = \frac{k_H(n_1 I_{TH} + n_2 I_{TCT} + n_3 I_{TE} + n_4 I_{TC})S}{N_1} = \frac{k_H \Lambda(n_1 I_{TH} + n_2 I_{TCT} + n_3 I_{TE} + n_4 I_{TC})}{\mu N_1}$$

which is the rate of infectivity of HIV (β_1), while the rate of infectivity of TB, (β_2), at disease free

equilibrium point has $\quad (I_{TH}) = 0$ since the group already has HIV infection.

Thus we have that at disease free equilibrium point

$$\beta_2 I_{TH} = \frac{k_T(m_2 I_{TC})I_{TH}}{N_1} = 0$$

Simplifying, we have

$$F = \begin{bmatrix} 0 & \dfrac{n_1 K_H \Lambda}{N_1 \mu} & \dfrac{n_4 K_H \Lambda}{N_1 \mu} & \dfrac{n_3 K_H \Lambda}{N_1 \mu} & \dfrac{n_2 K_H \Lambda}{N_1 \mu} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and}$$

$$V = \begin{bmatrix} \psi + \mu + \tau\sigma & 0 & 0 & 0 & 0 \\ -\tau\sigma & \mu & 0 & 0 & -\tau_1 \\ 0 & 0 & \sigma_3 + \mu + \delta_T & -\sigma_2 & 0 \\ 0 & 0 & 0 & \sigma_2 + \sigma_4 + \mu & 0 \\ 0 & 0 & -\sigma_3 & -\sigma_4 & \tau_1 + \mu \end{bmatrix}$$

With matlab the highest the eigenvalue of $\quad FV^{-1}$ (spectral radius of FV^{-1}, gives

$$\frac{-n_1 K_H \Lambda(-\tau\sigma)}{\mu N_1} \cdot \frac{1}{\mu(\psi + \mu + \tau\sigma)} = \frac{n_1 \tau\sigma K_H \Lambda}{\mu^2 N_1(\psi + \mu + \tau\sigma)}$$

Thus the reproduction number, R_{01}, for this sub-model is

$$R_{01} = \frac{n_1 \tau\sigma K_H \Lambda}{\mu^2 N_1(\psi + \mu + \tau\sigma)}$$

2.6   SENSITIVITY INDICES OF THE PARAMETERS OF THIS SUBMODEL

The sensitivity index of the modeled parameters with respect to the basic reproduction number, R_{01} helps one to get insight on the appropriate intervention strategies to prevent and control the spread of the disease described in the model. Thus, we determine the sensitivity index of each of the parameters used in this sub-model, using the normalized forward sensitivity index of the basic reproduction number R_{01}, with respect to the

parameter values, given as $r\dfrac{R_{01}}{p} = \dfrac{\partial R_{01}}{\partial p} \times \dfrac{p}{R_{01}}$; where p is the parameter of the model. Calculating the

sensitivity index and substituting with the values of the parameter as obtained in table 1:

***Table 1:** Values of different parameters in the model.*

Parameters	Nominal values	References
Λ, K_H, μ, δ_T	2000, 0.45, 0.02, 0.24	Shar *et al* (2014)
K_T, τ	0.76, 0.86	Tebeje, *et al*(2010)
ψ	0.123	Calculated (Appendix A)

$\sigma, 1-\sigma$	0.48, 0.52	Assumed
$\tau_1, \sigma_2,$	0.5, 0.03,	Castilo-chavez C.,*etal*(2004)
$n_1, n_2, \cdots, n_4$	0.002, 0.001, 0.003, 0.002	Assumed
$\rho_1, \rho_2, \sigma_3, \sigma_4$	0.3, 0.7, 0.3, 0.35	Assumed
N_1	35,000	Assumed

we have the sensitive index of the parameters as in table 2 below:

Table 2: *Signs of sensitivity index of* R_{01}

S/N	Parameters	Sensitivity index
1.	K_H	+1
2.	Λ	+1
3.	τ	$+0.257$
4.	μ	-2.036
5.	σ	$+0.257$
6.	ψ	-0.221
7.	N_1	-1
8.	n_1	+1

Thus if we increase (decrease) any of the parameters, $K_H, \Lambda, \sigma, n_1 \text{ and } \tau$; then R_{01} will increase (decrease). Also if we increase (decrease) any of $\psi, N_1 \text{ and } \mu,$; then R_{01} will decrease (increase). Sensitive index of other parameters that did not appear in R_{01} are zero.

2.7 EXISTENCE OF AN ENDEMIC EQUILIBRIUM POINT OR STATE

From our assumption that one is first infected with HIV, lowering his immune system before he is infected with TB, we note that the control of HIV will lead to the control of TB. The endemic equilibrium point or state is where the disease cannot be totally eradicated but remains in the population. Let ε_H be the endemic equilibrium point. Hence, for HIV and TB to persist in the population, ε_H must not be equal to zero at the equilibrium point. That is at least one of infected groups, $E_H^{**}, I_{TH}^{**}, I_{TC}^{**}, I_{TE}^{**}, \text{ and } I_{TCT}^{**}$ is none zero. To calculate the endemic equilibrium point, we have $\varepsilon_H = \left(S^{**}, E_H^{**}, I_{TH}^{**}, I_{TC}^{**}, I_{TE}^{**}, I_{TCT}^{**} \right) \neq 0$.

Note that $\beta_1^{**} = \dfrac{k_H (n_1 I_{TH}^{**} + n_2 I_{TCT}^{**} + n_3 I_{TE}^{**} + n_4 I_{TC}^{**}) S^{**}}{N_1}$, for HIV infection

and $\beta_2^{**} = \dfrac{k_T (m_2 I_{TC}^{**}) I_{TH}^{**}}{N_1}$ for TB infection.

Let, $(\psi + \tau\sigma + \mu) = A$, $(\sigma_3 + \mu + \delta_T) = B$, $(\sigma_2 + \sigma_4 + \mu) = C$ and $(\tau_1 + \mu) = D$

Thus, setting the right hand side of the modeled equations, 2.2 (a-f), to zero (noting that $\beta_1 = \beta_1^{**}$ and $\beta_2 = \beta_2^{**}$) at the endemic equilibrium point, and solving simultaneously gives

$$S^{**} = \frac{\Lambda + \psi E_H^{**}}{\beta_1 + \mu} \qquad \qquad \ldots(i)$$

$$E_H^{**} = \frac{\beta_1 S^{**}}{A} \qquad \qquad \ldots(ii)$$

Substituting for E_H^{**} in (i) gives

$$S^{**} = \frac{\Lambda + \psi \dfrac{\beta_1 S^{**}}{A}}{\beta_1 + \mu}$$

$$S^{**}(\beta_1 + \mu) = \frac{A\Lambda + \psi \beta_1 S^{**}}{A}$$

$$S^{**}(A(\beta_1 + \mu) - \psi \beta_1) = A\Lambda$$

Therefore $S^{**} = \dfrac{A\Lambda}{(A(\beta_1 + \mu) - \psi \beta_1)}$ (2.7a)

Also from (ii),

$$E_H^{**} = \frac{\beta_1 \dfrac{A\Lambda}{(A(\beta_1 + \mu) - \psi \beta_1)}}{A} = \frac{\beta_1 \Lambda}{(A(\beta_1 + \mu) - \psi \beta_1)} \qquad (2.7b)$$

$$I_{TH}^{**} = \frac{\sigma\tau\, E_H^{**} + \tau_1\, I_{TCT}^{**}}{\beta_2 + \mu}$$

$$= (\sigma\tau\left[\frac{\beta_1 \Lambda}{A(\beta_1 + \mu) - \psi \beta_1}\right] + \tau_1\, I_{TCT}^{**})(\frac{1}{(\beta_2 + \mu)})$$

$$= \frac{\sigma\tau\beta_1 \Lambda + \tau_1\left(A(\beta_1 + \mu) - \psi \beta_1\right) I_{TCT}^{**}}{\left(A(\beta_1 + \mu) - \psi \beta_1\right)(\beta_2 + \mu)} \quad \text{(iii)}$$

$$I_{TC}^{**} = \frac{\beta_2 \rho_1 I_{TH}^{**} - \sigma_2 I_{TE}^{**}}{B} \qquad \qquad \text{(iv)}$$

$$I_{TE}^{**} = \frac{\beta_2 \rho_2 I_{TH}^{**}}{C} \quad \text{(v)}$$

$$I_{TCT}^{**} = \frac{\sigma_3 I_{TC}^{**} + \sigma_4 I_{TE}^{**}}{D} \quad \text{(vi)}$$

Substituting for I_{TCT}^{**} in (iii) gives

$$I_{TH}^{**} = \frac{\sigma\tau\beta_1 \Lambda + \tau_1\left(A(\beta_1 + \mu) - \psi \beta_1\right)\left(\dfrac{\sigma_3 I_{TC}^{**} + \sigma_4 I_{TE}^{**}}{D}\right)}{\left(A(\beta_1 + \mu) - \psi \beta_1\right)(\beta_2 + \mu)}$$

$$= \frac{D\sigma\tau\beta_1 \Lambda + \tau_1\left(A(\beta_1 + \mu) - \psi \beta_1\right)(\sigma_3 I_{TC}^{**} + \sigma_4 I_{TE}^{**})}{D\left(A(\beta_1 + \mu) - \psi \beta_1\right)(\beta_2 + \mu)}$$

Substituting for (iv), we have

$$I_{TH}^{**} = \frac{D\sigma\tau\beta_1\Lambda + \tau_1(A(\beta_1+\mu)-\psi\beta_1)(\sigma_3\left(\frac{\beta_2\rho_1 I_{TH}^{**} - \sigma_2 I_{TE}^{**}}{B}\right) + \sigma_4 I_{TE}^{**})}{D(A(\beta_1+\mu)-\psi\beta_1)(\beta_2+\mu)}$$

$$= \frac{BD\sigma\tau\beta_1\Lambda + \tau_1(A(\beta_1+\mu)-\psi\beta_1)(\sigma_3\left(\beta_2\rho_1 I_{TH}^{**} - \sigma_2 I_{TE}^{**}\right) + B\sigma_4 I_{TE}^{**})}{BD(A(\beta_1+\mu)-\psi\beta_1)(\beta_2+\mu)}$$

Substituting for I_{TE}^{**} , (v), to have

$$I_{TH}^{**} = \frac{BD\sigma\tau\beta_1\Lambda + \tau_1(A(\beta_1+\mu)-\psi\beta_1)(\sigma_3\left(\beta_2\rho_1 I_{TH}^{**} - \sigma_2(\frac{\beta_2\rho_2 I_{TH}^{**}}{C})\right) + B\sigma_4\left(\frac{\beta_2\rho_2 I_{TH}^{**}}{C}\right))}{BD(A(\beta_1+\mu)-\psi\beta_1)(\beta_2+\mu)}$$

$$= \frac{CBD\sigma\tau\beta_1\Lambda + \tau_1(A(\beta_1+\mu)-\psi\beta_1)\left(\sigma_3\left(C\beta_2\rho_1 I_{TH}^{**} - \sigma_2\beta_2\rho_2 I_{TH}^{**}\right) + B\sigma_4\beta_2\rho_2 I_{TH}^{**}\right)}{CBD(A(\beta_1+\mu)-\psi\beta_1)(\beta_2+\mu)}$$

$$(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2)) = CBD\sigma\tau\beta_1\Lambda$$

Therefore $I_{TH}^{**} = \dfrac{CBD\sigma\tau\beta_1\Lambda}{(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2))}$

(2.7c)

$$I_{TC}^{**} = \frac{\beta_2\rho_1 I_{TH}^{**} - \sigma_2 I_{TE}^{**}}{B}$$

$$= \frac{\beta_2\rho_1\left[\dfrac{CBD\sigma\tau\beta_1\Lambda}{(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2))}\right] - \sigma_2 I_{TE}^{**}}{B}$$

But, $I_{TE}^{**} = \dfrac{\beta_2\rho_2 I_{TH}^{**}}{C}$

$$= \frac{\beta_2\rho_2}{C}\left[\frac{CBD\sigma\tau\beta_1\Lambda}{(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2))}\right]$$

(2.7d)

Therefore,

$$I_{TC}^{**} = \frac{\beta_2\rho_1}{B}\left[\frac{CBD\sigma\tau\beta_1\Lambda}{(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2))}\right]$$

$$- \frac{\sigma_2\beta_2\rho_2}{BC}\left[\frac{CBD\sigma\tau\beta_1\Lambda}{(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2))}\right]$$

$$= \left(\frac{\beta_2\rho_1}{B} - \frac{\sigma_2\beta_2\rho_2}{BC}\right)\left[\frac{CBD\sigma\tau\beta_1\Lambda}{(A(\beta_1+\mu)-\psi\beta_1)(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1 - \sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2))}\right]$$

(2.7e)

$$I_{TCT}^{**} = \frac{\sigma_3 I_{TC}^{**} + \sigma_4 I_{TE}^{**}}{D}$$

$$= \left(\left(\frac{\sigma_3\beta_2\rho_1}{DB} - \frac{\sigma_3\sigma_2\beta_2\rho_2}{DBC}\right) + \frac{\sigma_4\beta_2\rho_2}{DC}\right)\left[\frac{CBD\sigma\tau\beta_1\Lambda}{\left(A(\beta_1+\mu)-\psi\beta_1\right)\left(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1-\sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2)\right)}\right]$$

(2.7f)

Using equations 2.7(a - f) in the expression of β_1, for $\beta_1 = \beta_1^{**}$, shows that none zero (endemic) equilibrium of the model satisfies;

$$\beta_1^{**} = \frac{\dfrac{K_H}{N_1}\left[n_1 + n_2\left(\dfrac{\sigma_3\beta_2\rho_1}{DB} - \dfrac{\sigma_3\sigma_2\beta_2\rho_2}{DBC} + \dfrac{\sigma_4\beta_2\rho_2}{DC}\right) + n_3\dfrac{\beta_2\rho_2}{C} + n_4\left(\dfrac{\beta_2\rho_1}{B} - \dfrac{\sigma_2\beta_2\rho_2}{BC}\right)\right]CBD\sigma\tau\beta_1\Lambda}{\left(A(\beta_1+\mu)-\psi\beta_1\right)\left(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1-\sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2)\right)}$$

$$A(\beta_1+\mu)-\psi\beta_1 = \frac{K_H\left[CBDn_1 + n_2\beta_2\left(C\sigma_3\rho_1 - \sigma_3\sigma_2\rho_2 + B\sigma_4\rho_2\right) + n_3BD\beta_2\rho_2 + n_4\beta_2D(C\rho_1-\sigma_2\rho_2)\right]CBD\sigma\tau\Lambda}{N_1CBD\left(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1-\sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2)\right)}$$

Expanding the left-hand side of the equation and substituting for the value of A, the coefficient of β_1, gives

$$A(\beta_1+\mu)-\psi\beta_1 = \beta_1(\psi+\sigma\tau+\mu)+A\mu-\psi\beta_1 = \beta_1(\sigma\tau+\mu)+A\mu$$

Thus,

$$\beta_1(\sigma\tau+\mu)+A\mu = \frac{\sigma\tau\Lambda K_H\left[n_1CBD + n_2\beta_2\left(C\sigma_3\rho_1 - \sigma_3\sigma_2\rho_2 + B\sigma_4\rho_2\right) + n_3BD\beta_2\rho_2 + n_4\beta_2D(C\rho_1-\sigma_2\rho_2)\right]}{N_1\left(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1-\sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2)\right)}$$

$$\beta_1 = \frac{\sigma\tau\Lambda K_H\left[n_1CBD + n_2\beta_2\left(C\sigma_3\rho_1 - \sigma_3\sigma_2\rho_2 + B\sigma_4\rho_2\right) + n_3BD\beta_2\rho_2 + n_4\beta_2D(C\rho_1-\sigma_2\rho_2)\right]}{N_1(\sigma\tau+\mu)\left(CBD(\beta_2+\mu)-\tau_1(\sigma_3(C\beta_2\rho_1-\sigma_2\beta_2\rho_2)+B\sigma_4\beta_2\rho_2)\right)} - \frac{A\mu}{(\sigma\tau+\mu)}$$

(2.7g)

Hence, to find the condition for the existence of an equilibrium for which HIV only is endemic in the sub-population, we let $\beta_2 = \beta_2^{**} = 0$ in equation (2.7g) so that the rate of infectivity of HIV, (β_1), becomes;

$$\beta_1^{**} = \frac{\sigma\tau\Lambda K_H(n_1CBD)}{N_1(\sigma\tau+\mu)(CBD)\mu} - \frac{A\mu}{\sigma\tau+\mu}$$

$$= \frac{n_1\sigma\tau\Lambda K_H}{N_1(\sigma\tau+\mu)\mu} - \frac{A\mu}{\sigma\tau+\mu}$$

(2.7h)

Multiply both the numerator and the denominator of the first part of the fraction, (4.39h), by $A\mu$, substitute for the value of A at the denominator, to have;

$$\beta_1^{**} = \frac{-A\mu(1-R_0)}{\sigma\tau+\mu} \qquad\qquad (2.71)$$

The force of infection at the steady-state β_1^{**} is positive, only if $R_0 > 1$. We have proved the following result:

Lemma 5

The sub model system 2.2 has a unique endemic equilibrium whenever $R_0 > 1$.

To find the condition for the existence of an equilibrium for which TB only, is endemic in the sub population, we have:

$$\overset{**}{\beta_2} = \frac{k_T(m_2 \overset{**}{I_{TC}}) \overset{**}{I_{TH}}}{N_1} = \frac{K_T}{N_1}\left(m_2\left(\frac{\beta_2\rho_1}{B} - \frac{\sigma_2\beta_2\rho_2}{BC}\right)\right)\left[\frac{CBD\sigma\tau\beta_1\Lambda}{\left(A(\beta_1+\mu)-\psi\beta_1\right)\left(CBD(\beta_2+\mu)-\tau_1\left(\sigma_3\left(C\beta_2\rho_1-\sigma_2\beta_2\rho_2\right)+B\sigma_4\beta_2\rho_2\right)\right)}\right]$$

$$\overset{**}{\beta_2} = \frac{K_T}{N_1}\left(m_2\left(\frac{\beta_2\rho_1}{B} - \frac{\sigma_2\beta_2\rho_2}{BC}\right)\right)\left[\frac{CBD\sigma\tau\beta_1\Lambda}{\left(A(\beta_1+\mu)-\psi\beta_1\right)\left(CBD(\beta_2+\mu)-\tau_1\left(\sigma_3\left(C\beta_2\rho_1-\sigma_2\beta_2\rho_2\right)+B\sigma_4\beta_2\rho_2\right)\right)}\right]$$

$$\left(ACBD(\beta_1+\mu)-\psi\beta_1\right)\beta_2+CBD\left(A(\beta_1+\mu)-\psi\beta_1\right)\mu-\tau_1\beta_2\left(A(\beta_1+\mu)-\psi\beta_1\right)\left(\sigma_3\left(C\rho_1-\sigma_2\rho_2\right)+B\sigma_4\rho_2\right)=\frac{K_T}{N_1}\left(m_2\left(\frac{\rho_1}{B}-\frac{\sigma_2\rho_2}{BC}\right)\right)CBD\sigma\tau\beta_1\Lambda$$

$$\overset{**}{\beta_2}=\frac{K_T}{N_1}\left(m_2\left(\frac{\rho_1}{B}-\frac{\sigma_2\rho_2}{BC}\right)\right)\frac{CBD\sigma\tau\beta_1\Lambda-CBD\left(A(\beta_1+\mu)-\psi\beta_1\right)\mu}{\left(ACBD(\beta_1+\mu)-\psi\beta_1\right)-\tau_1\left(A(\beta_1+\mu)-\psi\beta_1\right)\left(\sigma_3\left(C\rho_1-\sigma_2\rho_2\right)+B\sigma_4\rho_2\right)}$$

(2.72)

It can be clearly seen that if we let $\overset{**}{\beta_1} = 0,$ which is the infectivity rate of HIV, $\overset{**}{\beta_2}$, the infectivity of TB will be zero. This agrees with our assumption that TB infection depends on HIV infection.

V.   NUMERICAL RESULTS AND DISCUSSION

In this section we used MATLAB to simulate the model for HIV/TB co-infection in a HIV endemic area. This will enable us see the population dynamics of each class of the HIV/TB co-infection. We also vary the values of the control parameters ψ and see the effect on both the Susceptible, the Exposed and infective HIV compartments, taking a sample population of 35,000. The results are as shown below:
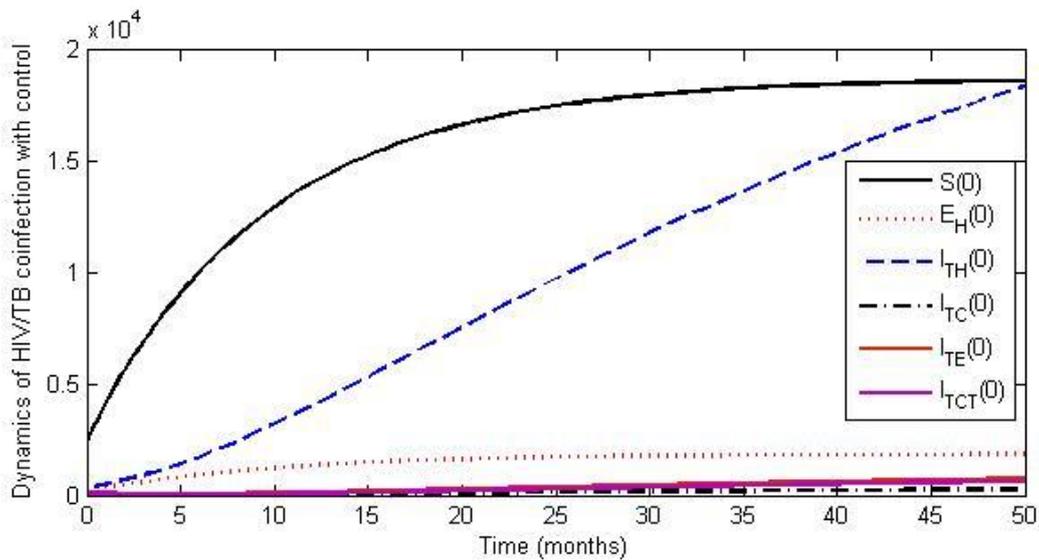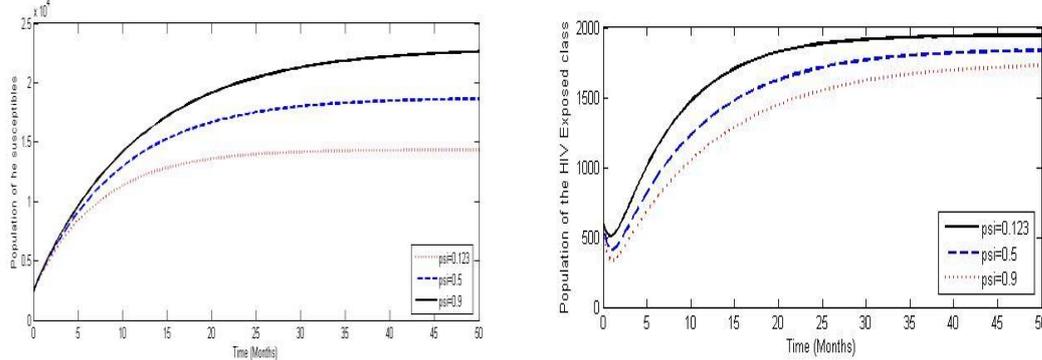

***Figure 5.1****: A graph showing the dynamics of HIV/TB co-infection in the population.*

5.1 The effect of an increased efficacy of post-exposure prophylaxis on both the susceptible and the HIV exposed population

Figure 5.2: *(a) Impact of psi (post- exposure prophylaxis effect) on the susceptible class (b) Impact of psi (post- exposure prophylaxis effect) on the HIV Exposed class*

From figure 5.2 above; as the efficacy of psiincreases, the number of the susceptible individuals in the population increases while the number of the exposed individuals in the population decreases. For instance, reading from the 50th month, the population of the susceptible class rose from 14,342 to 18645 while the number of Exposed HIV individuals decreased from 1948 to 1837because of the increased efficacy rate of the post exposure prophylaxis from 0.123 to 0.5.

5.2 The effect of an increased efficacy of post-exposure prophylaxis on infective HIV population
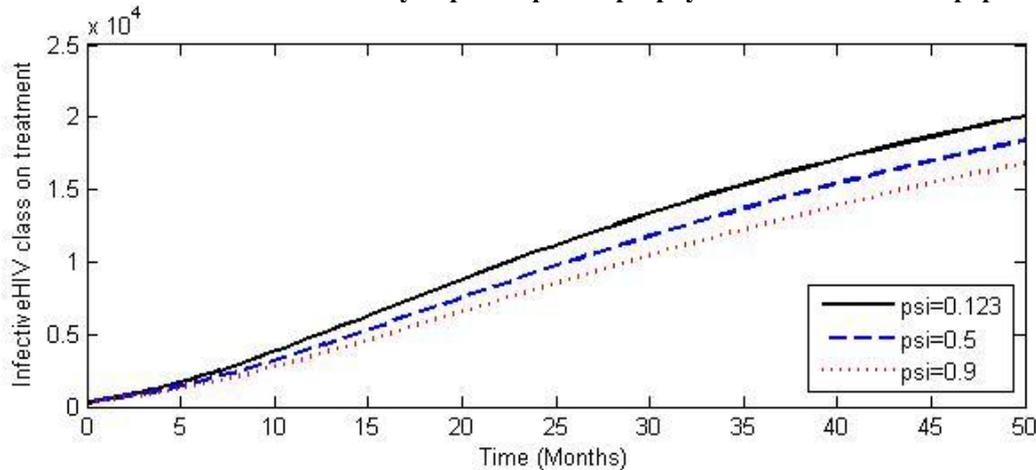
Figure 5.3: *impact of psi (post- exposure prophylaxis effect) on the Infective HIV class on treatment*
Also, from figure 5.3 above; as the efficacy of psiincreases, the number of the infective HIV population decreases. Thus, reading the graph from the 50th month, the population of the infective HIV class reduced from 20,094 to 18, 387 because of the increased efficacy rate of the post exposure prophylaxis from 0.123 to 0.5.

VI. RESULTS AND CONCLUSION

From the sensitive analysis of the parameters we noted that to control the infection, one should aim at minimizing the values of the HIV contact rate (K_H), the rate of influx of people in the susceptible class (Λ), the rate of progression of HIV infected individuals from the exposed class to infective HIV class (τ), The proportion of HIV infective individuals that enter infective HIV class on treatment (σ), and the infectivityrate of HIV class (n_1) and then maximize the efficacy rate of post exposure prophylaxis (ψ), death rate of infective HIV individuals (μ) and the number of individuals in the entire population.

We can maximize the efficacy of post exposure prophylaxis by noting the parameters that are involved from appendix A. These include raising the awareness of post-exposure prophylaxis (PEP) which is currently 20% in these HIV endemic areas. Increasing the people's knowledge of PEP and 72 hours HIV exposure duration, when it must be administered, for the HIV exposed patient to recover from the infection. Minimizing the HIV exposure rate through the encouragement of abstinence, use of condoms and preventive measures.

APPENDIX A
NUMERICAL CACULATION OF THE RATE OF RECOVERY OF HIV EXPOSED INDIVIDUALS DUE TO THE USE OF POST EXPOSURE PROPHYLAXIS

The rate of recovery of HIV exposed individuals due to the use of post-exposure prophylaxis is calculated based on the following findings:

According to Bosena Tebeje, et al (2010), finding in the HIV endemic areas of Ethiopia shows that;

The rate of HIV exposure is approximated to be 76%

The rate of awareness of post-exposure prophylaxis (PEP) is 20%

Percentage rate of PEP success when fully administered is 81% and above.

With these data, we calculate that the rate of recovery of the HIV exposed individuals, ψ as;

ψ =rate of HIV exposure (76%) x rate of PEP awareness (20%) x rate of PEP success (81%)

$= 0.123$

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