# Mathematical Modelling of Tuberculosis as an Opportunistic Respiratory Co-Infection in HIV/AIDS in the Presence of Protection

**Joyce K. Nthiiri**

Department of Mathematics
Masinde Muliro University of Science and Technology
P.O. Box 190-50100 Kakamega, Kenya

**George O. Lawi**

Department of Mathematics
Masinde Muliro University of Science and Technology
P.O. Box 190-50100 Kakamega, Kenya

**Alfred Manyonge**

Department of Pure and Applied Mathematics
Maseno University, Private Bag Maseno, Kenya

## Abstract

Tuberculosis is a common opportunistic infection that cause death in HIV/AIDS patients. Due to the high cost of treatment, protection against infection may be preferable in scarce resource settings. In this paper, we consider a deterministic model incorporating protection from infection for both tuberculosis(TB) disease and HIV/AIDS. Two cases are considered, namely, the case of maximum protection against TB and the case of maximum protection against HIV/AIDS. In both cases, an endemic state is shown to exist provided that

the reproduction number is greater than unity. By use of a suitable Lyapunov function, the endemic states are shown to be globally asymptotically stable. Numerical simulations indicate that enhanced protection against a disease lowers new incidences of the disease, hence low disease prevalence rates. Therefore, public awareness campaign efforts on protective measures should be enhanced.

**Mathematics Subject Classification:** 35E21

**Keywords:** Reproduction number, Protection, Tuberculosis, HIV/AIDS

# 1   Introduction

Human immunodeficiency virus (HIV), whose discovery dates back in 1981, is a virus that overtime weakens the body's immunity [23]. Immunity system is the body's main defence against the threat of invasion by pathogenic organisms such as bacteria and viruses[9]. HIV destroys the CD4 cells making the body unable to fight other infections and at this level, HIV leads to Acquired immunodeficiency syndrome (AIDS). With a compromised immune system, the body is at risk of opportunistic infections (OIs)[15]. These opportunistic infections include respiratory infections,sexually transmitted diseases e.g syphilis, gonorrhoea among others.

Initially, intervention for HIV/AIDS was aimed at preventing new infections through creating awareness and advocating for change of behaviour. Services of People Living with HIV/AIDS (PLWHA) include availability of antiretroviral therapy(ARV's) as a protective measure, which help to improve the quality of life of the HIV infected individual hence reducing the morbidity and mortality related to HIV/AIDS and restores the immune function by maximal suppression of viral replication[17]. The challenge of scarce economic resources is a threat to sustained access to ARV's. Furthermore, lack of adherence to treatment schedule is a challenge among HIV/AIDS patients. The search for cure and/or vaccine for HIV/AIDS is ongoing. Prevention against infection seems to be the viable alternative at the moment. Prevention against HIV/AIDS may include abstinence, being faithful, use of condoms, male circumcision among others.The search of HIV/AIDS vaccine has yielded no results[16], thus effective programs to reduce HIV transmission are still needed[10]. Lately, public heath campaigns against TB have focused on protection measures

Respiratory infections such as tuberculosis (TB) account for a significant portion of illnesses among HIV/AIDS patients, since they take advantage of the weakened immune system. The mortality and morbidity associated with these illnesses is

high in populations affected by poverty, social unrest and lack of proper health infrastructures, such as witnessed in the developing world[21]. TB is an airborne bacterial disease in human lungs caused by Mycobacterium tuberculosis (Mtb)[1]. It is a slow dynamics disease[11] and is mostly transmitted through the air when persons with pulmonary TB cough. The risk factors of TB infection are generally prolonged close interactions with infected individuals and immunosuppression such as in HIV/AIDS[22]. The incubation period for TB is 2 to 10 weeks. Most TB infections in children and adolescents are asymptomatic[7] . When infection causes disease, symptoms include; chronic cough, weight loss, fever, growth delay, night sweats and chills. Infection is often diagnosed by a positive TB skin test result. In 2003, the estimated number of new TB cases was 8.8 million with 1.7 million deaths worldwide[8].

TB is the leading cause of death among people infected with HIV/AIDS[15]. According to WHO estimates about 33% of HIV patients worldwide are co-infected with TB and about half of HIV infected persons are expected to acquire TB[22]. Unfortunately three quarters of all dually infected people live in sub-Saharan Africa [20]. HIV infected individuals with latent TB are more likely to progress to active TB faster than those who are sero-negative for HIV[19]. TB treatment involves a 4-drug regimen for a minimum of six months with multiple antibiotics and regimens shorter than six months duration are not recommended by WHO[6]. Due to this long period of treatment, in some cases, patients do not adhere to the treatment schedule and this leads to treatment failure and or drug resistance to TB. For instance in 2007, among all HIV/AIDS infected persons, 91.3% received and completed TB treatment whereas in 2012, 73.6% received and completed treatment[12]. This shows a decline in the level of adherence to treatment. Lately, public heath campaigns against TB have focused on protective measures. Thus, in the phase of diminishing donor support for health programmes in the developing countries, protection against infection is an option worth exploring.

## 2   Model Formulation

We formulate a model in which total human population at any time $t$, denoted by $N$ is subdivided it into classes, $S(t)$ the class of individuals susceptible to both TB and HIV/AIDS infection, $P_T$, individuals who are protected against TB, This protection is lost at the rate $\alpha_1$. The class $L_T$ consists of individuals who are asymptomatically infected with TB infection. This infection occurs at the rate $\lambda_T$. In the absence of treatment, TB symptoms are developed at the rate $\tau_1$. The class $I_T$ comprises of individuals symptomatically infected with TB. Treatment for TB is assumed to be successful and is done at the rate $\varepsilon$. Thus the class $T_T$ consists of individuals who

have recovered from TB infection. Mortality occurs among active TB patients at the rate $\delta_T$, while natural death is assumed to occur in all classes at the rate $\mu$.

The class $P_H$ consist of individuals who are protected against HIV/AIDS infection. This protection may be lost due to risky behaviour at the rate $\alpha_2$. Since the modes of transmission for the two diseases are different we do not assume simultaneous infection of an individual with the two diseases. Furthermore, for purposes of simplicity we do not assume dual protection for TB and HIV/AIDS. The class $I_H$ is made up of individuals who are asymptomatically infected with HIV/AIDS. This infection occurs at the rate $\lambda_H$. In the absence of intervention (therapy), individuals develop symptoms of HIV/AIDS and progress from the class $I_H$ to the class $I_A$ at the rate $\tau_2$. Mortality occurs among HIV/AIDS patients at the rate $\delta_A$.

Individuals in the class $I_H$ can acquire TB at the rate $\upsilon_1\lambda_T$ and progress to the class $I_{HL}$, where $\upsilon_1$ is a modification parameter. This group of individuals develop TB symptoms at the rate $\tau_3$, where $\tau_3 > \tau_1$, and progress to the class $I_{HT}$. Individuals in the class $I_A$ can also acquire TB at the rate $\upsilon_2\lambda_T$, where $\upsilon_2 > \upsilon_1$ and progress to the class $I_{AL}$. This group of individuals develop TB symptoms at the rate $\tau_4$, where $\tau_4 > \tau_3$, and progress to the class $I_{AT}$. In the absence of intervention (therapy), individuals develop symptoms of HIV/AIDS and progress from the class $I_{HT}$ to the class $I_{AT}$ at the rate $\upsilon_3\tau_2$, where $\upsilon_3$ is a modification. Upon effective TB treatment, treated individuals in the class $I_{AT}$ will move back to the class $I_A$. Mortality occurs due to the dual infection of HIV/AIDS and TB at the rate $\delta_{AT}$.

Individuals in the class $I_L$ can acquire HIV/AIDS at a rate $\kappa_1\lambda_H$ and progress to the class $I_{LH}$ Individuals in the class $I_T$ can acquire HIV/AIDS at a rate $\kappa_2\lambda_H$ and progress to the class $I_{TH}$. $\kappa_1$ and $\kappa_1$ are modification parameters and $\kappa_2 > \kappa_1$.

The total population is defined as

$$N = S(t) + P_T(t) + P_H(t) + I_H(t) + I_{HL}(t) + I_L(t) + I_T(t) + T_T(t)$$
$$+ I_{HT}(t) + I_A(t) + I_{AL}(t) + I_{AT}(t) \tag{1}$$

We define the rate at which individuals acquire TB as

$$\lambda_T = \frac{\theta\eta[\phi_1 I_T + \phi_2 I_{TH} + \phi_3 I_{AT}]}{N} \tag{2}$$

where $\theta$ is the probability that one will acquire TB upon contact with TB infected individuals and $\eta$ is the contact rate with TB infected individuals while and $\phi_3 > \phi_2 > \phi_1$ are modification parameters.

The rate at which individual acquire HIV/AIDS is defined as

$$\lambda_H = \frac{\pi C[I_H + \gamma[I_{LH} + \sigma I_{TH}] + \omega[I_A + \nu_1[I_{AL} + \nu_2 I_{AT}]]]}{N} \tag{3}$$

where $\pi$ is the probability that susceptible individuals acquire HIV upon effective contact with an HIV infected individual and C is the effective contact rate with HIV/AIDS infected individuals. This effective contact rate may include, sexual intercourse with an infected individual, blood transfusion with infected blood, sharing sharp objects with HIV/AIDS infected individuals and vertical transmission from mother to child during birth and through breastfeeding. while $\gamma$, $\sigma$, $\omega$, $\nu$ are modification parameters.

HIV/AIDS prevalence is defined by

$$\frac{[I_H + \gamma[I_{LH} + \sigma I_{TH}] + \omega[I_A + \nu_1[I_{AL} + \nu_2 I_{AT}]]]}{N} \tag{4}$$

It is assumed that an individual who is asymptomatic infected with HIV/AIDS and has latent TB is more infectious of HIV/AIDS than one asymptomatically infected with HIV/AIDS. The same assumption is made for an individual actively infected with TB and is asymptomatic with HIV/AIDS. Similarly AIDS individuals are more infectious than individuals asymptomatic with HIV due to high viral load[13].

The main aim of this work is to study the effect of protection for TB and HIV/AIDS in the co-infection model. Protection against these two infections involves limiting exposure to risk factors that can lead to infection. The risk factors for HIV involves; having unprotected anal or vaginal sex, having another sexually transmitted infection such as syphilis, herpes, chlamydia, gonorrhoea and bacterial vaginosis; sharing contaminated needles, syringes and other injecting equipment and drug solutions when injecting drugs; receiving unsafe injections, blood transfusions, medical procedures that involve unsterile cutting or piercing; and experiencing accidental needle stick injuries [4]. Persons at high risk for developing TB disease fall into two categories; persons who have been recently infected with TB bacteria and persons with medical conditions that weaken the immune system.

Let $\iota_T$ and $\iota_H$ denote the probability of success of protection against TB and HIV/AIDS respectively. The modified forces of infection $\lambda_T^p$ and $\lambda_H^p$ become

$$\lambda_T^p = \frac{\theta\eta(1 - \iota_T)[\phi_1 I_T + \phi_2 I_{TH} + \phi_3 I_{AT}]}{N} \tag{5}$$

and

$$\lambda_H^p = \frac{\pi C(1 - \iota_H)[I_H + \gamma[I_{LH} + \sigma I_{TH}] + \omega[I_A + \nu_1[I_{AL} + \nu_2 I_{AT}]]]}{N} \tag{6}$$
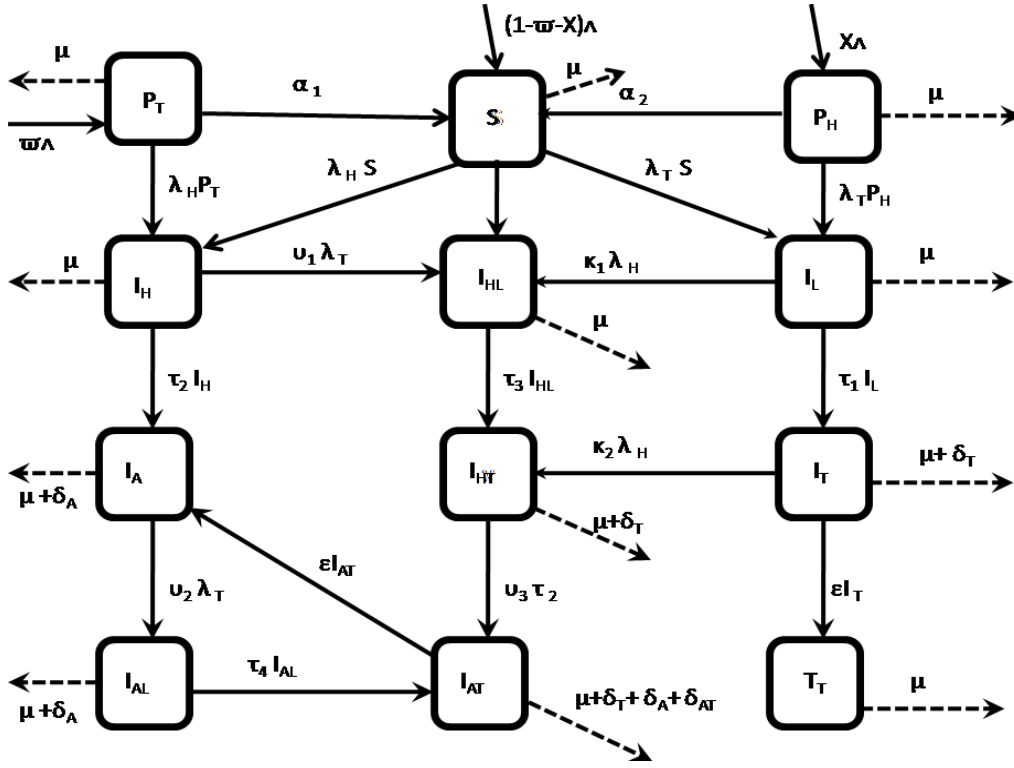
**Figure 1:** *The flow diagram*

The system of differential equations from this flow diagram are:

$$
\begin{aligned}
\frac{dS}{dt} &= (1 - \varpi - \chi)\Lambda + \alpha_1 P_T + \alpha_2 P_H \\
&\quad -(\mu + \lambda_H + \lambda_T)S \\
\frac{dP_T}{dt} &= \varpi\Lambda - (\mu + \alpha_1 + \lambda_H)P_T \\
\frac{dP_H}{dt} &= \chi\Lambda - (\alpha_2 + \mu + \lambda_T +)P_H \\
\frac{dI_H}{dt} &= \lambda_H P_T + \lambda_H S - (v_1\lambda_T + \mu + \tau_2)I_H \\
\frac{dI_A}{dt} &= \tau_2 I_H + \varepsilon I_{AT} - (\mu + \delta_A + v_2\lambda_T)I_A \\
\frac{dI_L}{dt} &= \lambda_T P_H + \lambda_T S - (\mu + \kappa_1\lambda_H + \tau_1)I_L \\
\frac{dI_T}{dt} &= \tau_1 I_L - (\mu + \delta_T + \varepsilon + \kappa_2\lambda_H)I_T \\
\frac{dT_T}{dt} &= \varepsilon I_T - \mu T_T
\end{aligned}
\tag{7}
$$

$$\frac{dI_{HL}}{dt} = \upsilon_1 \lambda_T I_H + \kappa_1 \lambda_H I_L - (\mu + \tau_3) I_{HL}$$

$$\frac{dI_{HT}}{dt} = \kappa_2 \lambda_H I_T + \tau_3 I_{HL} - (\mu + \delta_T + \upsilon_3 \tau_2) I_{HT}$$

$$\frac{dI_{AL}}{dt} = \upsilon_2 \lambda_T I_A - (\mu + \delta_A + \tau_4) I_{AL}$$

$$\frac{dI_{AT}}{dt} = \upsilon_3 \tau_2 I_{HT} + \tau_4 I_{AL} - (\mu + \delta_T + \delta_A + \delta_{AT} + \varepsilon) I_{AT}$$

where $\varpi\Lambda$ is the constant recruitment rate into the class of individuals protected against TB, $\chi\Lambda$ is the constant recruitment rate into the class of individuals protected against HIV and $(1 - \varpi - \chi)\Lambda$ is the constant recruitment rate into the class of susceptible individuals to both TB and HIV virus. Every individual is assumed to be susceptible to both infections. It is assumed that there is no simultaneous infection with HIV/AIDS and TB because of the different transmission routes. It is also assumed that when one is successfully treated against TB recovery does not not confer permanent immunity against TB infection.

# 3   Model Analysis

Based on the fact that the model deals with human population, all the state variables and parameters are assumed to be non-negative $\forall t \geq 0$. Thus we study the model equation (7)in the feasible region defined by $\Gamma$ , where

$$\{S(t), P_H(t), P_T(t), I_H(t), I_{HL}(t), I_L(t), I_T(t), T_T(t),$$
$$I_{TH}(t), I_A(t), I_{AL}(t), I_{AT}(t)\} \in \Gamma \ \subset \mathbb{R}_+^{12}. \tag{8}$$

Furthermore we can show that the solutions are bounded.

## 3.1   The Case of Maximum Protection against TB, $\iota_T = 1$

Suppose that there are no new TB infections and the only infection in the population is that of HIV/AIDS. This can be achieved by increasing protection and by reducing the contact rate$\eta$ with a TB infected individual to low levels such that the number of contacts is not sufficient for disease transmission and may be considered as $\eta = 0$. Thus equation (7) will be reduced to

$$\frac{dS}{dt} = (1 - \chi - \varpi)\Lambda + \alpha_2 P_H - (\mu + \lambda_H)S$$

$$\frac{dP_H}{dt} = \chi\Lambda - (\alpha_2 + \mu)P_H \tag{9}$$

$$\frac{dP_T}{dt} = \varpi\Lambda - (\mu + \lambda_H)P_T$$

$$\frac{dI_H}{dt} = \lambda_H(S + P_T) - (\mu + \tau_2)I_H$$

$$\frac{dI_A}{dt} = \tau_2 I_H - (\mu + \delta_A)I_A$$

The force of infection for equation (9)is defined as

$$\lambda_H^h = \frac{\pi C(1 - \iota_H)(I_H + \omega I_A)}{N} \tag{10}$$

with the basic reproduction number

$$R_H = \frac{\beta C(1 - \iota_H)(\delta_A + \mu + \omega\tau_2)(\mu(1 - \chi) + \alpha_2)}{(\delta_A + \mu)(\mu + \tau_2)(\mu + \alpha_2)}. \tag{11}$$

An endemic state $I_H^* > 0$ where

$$I_H^* = \frac{\Lambda(R_H - 1)(\delta_A + \mu)}{(C\beta)(1 - \iota_H)(\delta_A + \mu + \omega\tau_2) - \delta_A\tau_2} \tag{12}$$

exists provided $R_H > 1$ with $\beta C < \beta C \iota_H + \tau_2$ since $(1 - \iota_H) > 0$

## 3.2   Local Stability of the Endemic equilibrium

The first four equations in equation (9) do not contain the class $I_A$, and so we can analyze the reduced system

$$\frac{dS}{dt} = (1 - \chi - \varpi)\Lambda + \alpha_2 P_H - (\mu + \lambda_H^h)S$$

$$\frac{dP_H}{dt} = \chi\Lambda - (\alpha_2 + \mu)P_H \tag{13}$$

$$\frac{dP_T}{dt} = \varpi\Lambda - (\mu + \lambda_H^h)P_T$$

$$\frac{dI_H}{dt} = \lambda_H^h(S + P_T) - (\mu + \tau_2)I_H$$

The Jacobian matrix of equation (13) at $E^*(S^*, P_H^*, P_T^* I_H^*)$ is given by

$$\frac{\partial f}{\partial x}(E^*) = \begin{pmatrix} -(\mu + \lambda_H^h) & \alpha_2 & 0 & \frac{-\beta C(1-\iota_H)S^*}{N} \\ 0 & -(\alpha_2 + \mu) & 0 & 0 \\ 0 & 0 & -(\mu + \lambda_H^h) & \frac{-\beta C(1-\iota_H)P_T^*}{N} \\ \lambda_H^h & 0 & \lambda_H^h & -(\mu + \tau_2) \end{pmatrix} \tag{14}$$

Clearly $-(\mu + \alpha_2)$ is an eigenvalue of Equation (14). The other eigenvalues are obtained from the reduced matrix

$$\frac{\partial f}{\partial x}(E_1^*) = \begin{pmatrix} -(\mu + \lambda_H^h) & 0 & \frac{-\beta C(1-\iota_H)S^*}{N} \\ 0 & -(\mu + \lambda_H^h) & \frac{-\beta C(1-\iota_H)P_T^*}{N} \\ \lambda_H^h & \lambda_H^h & -(\mu + \tau_2) \end{pmatrix} \tag{15}$$

An important creterion by Routh-Hurwitz gives the necessary and sufficient conditions for all the roots of the characteristic polynomial (with real coefficients) to lie in the left half of the complex plane. In other words, all the roots of the polynomial are negative or have negative real roots iff the determinants of all Hurwitz matrices are positive [24].

Clearly, the trace of equation (15) is negative and the determinant of equation (15) is given by

$$det(\frac{\partial f}{\partial x}(E)^*) = [\frac{I_H^*(1 - \iota_H)\eta\theta}{N} + \mu][\frac{I_H^*(1 - \iota_H)(S^* - P_T^*)\eta\theta}{N^2}$$
$$(I_H^*(\mu + \tau) - (1 - \iota_H)\eta\theta + \mu + \tau) - \mu(\mu + \tau)] \tag{16}$$

$det(\frac{\partial f}{\partial x}(E)^*) > 0$ provided that

$$I_H^*(\mu + \tau) \geq ((1 - \iota_H)\eta\theta) + \mu + \tau \tag{17}$$

and

$$[\frac{I_H^*(1 - \iota_H)(S^* - P_T^*)\eta\theta}{N^2}(I_H^*(\mu + \tau) - (1 - \iota_H)\eta\theta + \mu + \tau) \geq (\mu(\mu + \tau)) \tag{18}$$

Thus, by Routh-Hurwitz creteria, the endemic state $E^*(S^*, P_H^*, P_T^*I_H^*)$ is locally asymptotically stable provided that inequality (17)and inequality (18) holds.

## 3.3 Global stability of the endemic equilibrium

We obtain the global stability by means of Lyapunov's direct method and LaSalle's invariance principle[3]. For equation (9), consider the Lyapunov function

$$L : (S, P_H, P_T, I_H, I_A) \in \Gamma \subset \mathbb{R}_+^5 : S, P_H, P_T, I_H, I_A > 0 \tag{19}$$

where

$$L : (S, P_H, P_T, I_H, I_A) = \lambda_H^h(S - S^* - S^* \log \frac{S}{S^*}) + \lambda_H^h(P_H - P_H^* - P_H^* \log \frac{P_H}{P_H^*}) +$$

$$\lambda_H^h(P_T - P_T^* - P_T^* \log \frac{P_T}{P_T^*}) + \lambda_H^h(I_H - I_H^* - I_H^* \log \frac{I_H}{I_H^*}) +$$

$$\lambda_H^h(I_A - I_A^* - I_A^* \log \frac{I_A}{I_A^*})$$

(20)

$L$ is $C^1$ in the interior of $\Gamma$. $E^*$ is the global minimum of $L$ on $\Gamma$ and $L : (S, P_H, P_T, I_H, I_A) = 0$. The time derivative of $L$ is given by

$$\frac{dL}{dt} = \dot{L} = \lambda_H^h(1 - \frac{S^*}{S})\frac{dS}{dt} + \lambda_H^h(1 - \frac{P_H^*}{P_H})\frac{dP_H}{dt} + \lambda_H^h(1 - \frac{P_T^*}{P_T})\frac{dP_T}{dt} +$$

$$\lambda_H^h(1 - \frac{I_H^*}{I_H})\frac{dI_H}{dt} + \lambda_H^h(1 - \frac{I_A^*}{I_A})\frac{dI_A}{dt}$$

$$= -\lambda_H^h(\frac{S - S^*}{S})[(\mu + \lambda_H^h)(S - S^*) + \alpha_2(P_H - P_H^*)]$$

$$-\lambda_H^h(\frac{P_H - P_H^*}{P_H})[(\alpha_2 + \mu)(P_H - P_H^*)] - \lambda_H^h(\frac{P_T - P_T^*}{P_T})[(\mu + \lambda_H^h)(P_T - P_T^*)]$$

$$-\lambda_H^h(\frac{I_H - I_H^*}{I_H})[(\mu + \tau_2)(I_H - I_H^*)] - \lambda_H^h(\frac{I_A - I_A^*}{I_A})[(\mu + \delta_A)(I_A - I_A^*)]$$

(21)

hence $\dot{L} \leq 0$ is negative . We see that $\dot{L} = 0$ at $E^*$. Thus $\dot{L}$ is negative definite and the largest compact invariant set in $\{S, P_H, P_T, I_H, I_A\} \in \Gamma : \dot{L} = 0$ is the Singlet on $E^*$, where $E^*$ is the endemic equilibrium. Thus $E^*$ is globally asymptotically stable in the interior of $\Gamma$.

## 3.4   Case of maximum protection against HIV/AIDS, $\iota_H = 1$

Assuming that there is no new HIV/AIDS infections in the TB and HIV/AIDS co-infection model, the resulting model will be

$$\begin{aligned}
\frac{dS}{dt} &= (1 - \varpi - \chi)\Lambda + \alpha_1 P_T - (\mu + \lambda_T)S \\
\frac{dP_T}{dt} &= \varpi\Lambda - (\mu + \alpha_1)P_T \\
\frac{dP_H}{dt} &= \chi\Lambda - (\mu + \lambda_T)P_H \\
\frac{dI_L}{dt} &= \lambda_T(S + P_H) - (\mu + \tau_1)I_L
\end{aligned}$$

(22)

$$\frac{dI_T}{dt} = \tau_1 I_L - (\mu + \delta_T + \varepsilon)I_T$$

$$\frac{dT_T}{dt} = \varepsilon I_T - \mu T_T$$

The force of infection for equation (22) is

$$\lambda_T^t = \frac{\theta\eta(1 - \iota_T)I_T}{N} \tag{23}$$

and the basic reproduction number is given by

$$R_T = \frac{\theta\eta(1 - \iota_T)(\mu(1 - \varpi) + \alpha_1)}{(\delta_T + \mu + \varepsilon)(\mu + \tau_1)(\mu + \alpha_1)} \tag{24}$$

An endemic state $I_L^* > 0$ where

$$I_L^* = \frac{\Lambda(\delta_A + \varepsilon + \mu)(R_T - 1)}{(\eta\theta\tau_1)(1 - \iota_T)} \tag{25}$$

exist provided that $R_T > 1$ with $(1 - \iota_T) > 0$.

## 3.5   Local Stability of the Endemic equilibrium

Since the first five equations of equation (22) are independent of $T_T$, we analyze the reduced system

$$\frac{dS}{dt} = (1 - \varpi - \chi)\Lambda + \alpha_1 P_T - (\mu + \lambda_T^t)S \tag{26}$$

$$\frac{dP_T}{dt} = \varpi\Lambda - (\mu + \alpha_1)P_T$$

$$\frac{dP_H}{dt} = \chi\Lambda - (\mu + \lambda_T^t)P_H$$

$$\frac{dI_L}{dt} = \lambda_T^t(S + P_H) - (\mu + \tau_1)I_L$$

$$\frac{dI_T}{dt} = \tau_1 I_L - (\mu + \delta_T + \varepsilon)I_T$$

The Jacobian of equation(26) at the endemic state $E^*(S^*, P_H^*, P_T^*, I_L^*, I_T^*)$ is given by

$$J(E^*) = \begin{pmatrix} -(\mu + \lambda_T^t) & \alpha_1 & 0 & 0 & \frac{-\theta\eta S^*(1-\iota_T)}{N} \\ 0 & -(\mu + \alpha_1) & 0 & 0 & 0 \\ 0 & 0 & -(\mu + \lambda_T^t) & 0 & \frac{-\theta\eta P_H^*(1-\iota_T)}{N} \\ \lambda_T^t & 0 & \lambda_T^t & -(\mu + \tau_1) & 0 \\ 0 & 0 & 0 & \tau_1 & -(\mu + \delta_T + \varepsilon) \end{pmatrix} \tag{27}$$

Since $-(\mu + \alpha_1)$ is an eigenvalue of Equation (27). Next we consider the reduced matrix

$$J(E_1^*) = \begin{pmatrix} -(\mu + \lambda_T^t) & 0 & 0 & \frac{-\theta\eta S^*(1-\iota_T)}{N} \\ 0 & -(\mu + \lambda_T^t) & 0 & \frac{-\theta\eta P_H^*(1-\iota_T)}{N} \\ \lambda_T^t & \lambda_T^t & -(\mu + \tau_1) & 0 \\ 0 & 0 & \tau_1 & -(\mu + \delta_T + \varepsilon) \end{pmatrix} \qquad (28)$$

The trace of equation (28) is negative and the determinant is given by

$$det J(E^*) = \frac{1}{N}\{[I_T^*(1 - \iota_T)\theta\eta + N\mu]$$
$$(I_T^*)^2(1 + P_H^*)(1 - \iota_T)^3\theta^3\eta^3 + (N)^2[(I_T^*)^2(1 - \iota_T)\theta\eta + N\mu](\mu + \delta_T + \varepsilon)(\mu + \tau_1)\} \qquad (29)$$

Which is positive since $(1 - \iota_T) > 0$. Thus, by Routh-Hurwitz criteria, the endemic state $E^*(S^*, P_H^*, P_T^*, I_L^*, I_T^*)$ is locally asymptotically stable.

## 3.6    Global Stability of the Endemic equilibrium

Consider the non-linear Lyapunov function

$$L_e : (S, P_H, P_T, I_L, I_T, T_T) \in \Gamma \subset \mathbb{R}_+^6 : S, P_H, P_T, I_L, I_T, T_T > 0 \qquad (30)$$

defined as

$$L_e : (S, P_H, P_T, I_L, I_T, T_T) = \lambda_T^t(S - S^* - S^* \log \frac{S}{S^*})$$
$$+\lambda_T^t(P_H - P_H^* - P_H^* \log \frac{P_H}{P_H^*}) + \lambda_T^t(P_T - P_T^* - P_T^* \log \frac{P_T}{P_T^*}) + \lambda_T^t(I_L - I_L^* - I_L^* \log \frac{I_L}{I_L^*}) +$$
$$\lambda_T^t(I_T - I_T^* - I_T^* \log \frac{I_T}{I_T^*}) + \lambda_T^t(T_T - T_T^* - T_T^* \log \frac{T_T}{T_T^*}) \qquad (31)$$

where $L_e$ is $C^1$ in the interior of $\Gamma$. $E^*$ is the global minimum of $L_e$ on $\Gamma$ and $L_e : (S, P_H, P_T, I_L, I_T, T_T) = 0$. The time derivative of $L_e$ is given by

$$\frac{dL_e}{dt} = \dot{L}_e = \lambda_T^t(1 - \frac{S^*}{S})\frac{dS}{dt} + \lambda_T^t(1 - \frac{P_H^*}{P_H})\frac{dP_H}{dt} + \lambda_T^t(1 - \frac{P_T^*}{P_T})\frac{dP_T}{dt} +$$

$$\lambda_T^t(1 - \frac{I_L^*}{I_L})\frac{dI_L}{dt} + \lambda_T^t(1 - \frac{I_T^*}{I_T})\frac{dI_T}{dt} + \lambda_T^t(1 - \frac{T_T^*}{T_T})\frac{dT_T}{dt}$$

$$= -\lambda_T^t(\frac{S - S^*}{S})[(\mu + \lambda_T^t)(S - S^*) + \alpha_1(P_T - P_T^*)]$$

$$-\lambda_T^t(\frac{P_T - P_T^*}{P_T})[(\alpha_1 + \mu)(P_T - P_T^*)] - \lambda_T^t(\frac{P_H - P_H^*}{P_H})[(\mu + \lambda_T^t)(P_H - P_H^*)]$$

$$-\lambda_T^t(\frac{I_L - I_L^*}{I_L})[(\mu + \tau_1)(I_L - I_L^*)]$$

$$-\lambda_T^t(\frac{I_T - I_T^*}{I_T})[(\mu + \delta_T + \varepsilon)(I_T - I_T^*)] - \lambda_T^t(\frac{T_T - T_T^*}{T_T})[\mu(T_T - T_T^*)]$$

$$(32)$$

hence $\dot{L}_e < 0$. We see that $\dot{L}_e = 0$ iff $S = S^*, P_H = P_H^*, P_T - P_T^*, I_L = I_L^*, I_T = I_T^*$ and $T_T = T_T^*$. Thus the largest compact invariant set in $\{S, P_H, P_T, I_L, I_T, T_T\} \in \Gamma : \dot{L}_e = 0$ is the Singlet on $E^*$, where $E^*$ is the endemic equilibrium. Thus $E^*$ is globally asymptotically stable in the interior of $\Gamma$.

## 3.7  NUMERICAL SIMULATIONS

Numerical simulations are carried out to graphically illustrate the effect of protection on the dynamics of infection.

**Table 3.7.1:** *Parameter Values*

| Parameter description | Symbol | Value | Source |
|---|---|---|---|
| Natural death rate | $\mu$ | $7.0 \times 10^{-3} days^{-1}$ | [5] |
| Recruitment rate | $\Lambda$ | $8.7 \times 10^{-3} days^{-1}$ | [5] |
| Rate of recruitment into HIV protected class | $\chi$ | $6.7 \times 10^{-3}$ | Estimated |
| Rate of recruitment into TB protected class | $\varpi$ | $1.2 \times 10^{-3}$ | Estimated |
| Loss of protection against HIV/AIDS | $\alpha_2$ | $1.0 \times 10^{-4}$ | Estimated |
| Death due to HIV/AIDS | $\delta_A$ | $2.3 \times 10^{-4} days^{-1}$ | [14] |
| Rate of progression to AIDS stage | $\tau_2$ | $1.25 \times 10^{-1} days^{-1}$ | [18] |
| Probability of acquiring HIV/AIDS | $\pi$ | $1.1 \times 10^{-10} days^{-1}$ | [2] |
| Protection against HIV/AIDS | $\iota_H$ | $9.0 \times 10^{-1}$ | Estimated |
| Contact rate with HIV/AIDS infectives | $C$ | $8.0 \times 10^{-2}$ | Estimated |
| Loss of protection against TB | $\alpha_1$ | $0.1 \times 10^{-3}$ | Estimated |
| Death due to TB | $\delta_T$ | $3.95 \times 10^{-1} days^{-1}$ | [15] |
| Rate of progression to symptomatic TB | $\tau_1$ | $5.0 \times 10^{-1} days^{-1}$ | [12] |
| Probability of acquiring TB | $\theta$ | $1.1 \times 10^{-4}$ | Estimated |
| Protection against TB | $\iota_T$ | $9.0 \times 10^{-1}$ | Estimated |
| Contact rate with TB infectives | $\eta$ | $2.0 \times 10^{-4}$ | Estimated |

*3.7.1 The effect of varying the protection term on HIV/AIDS infections*

Numerical simulations were carried out to investigate the effect of protection on HIV/AIDS and TB prevalence. The following graphs were obtained for a given set on initial conditions and parameter values in table 3.7.1.
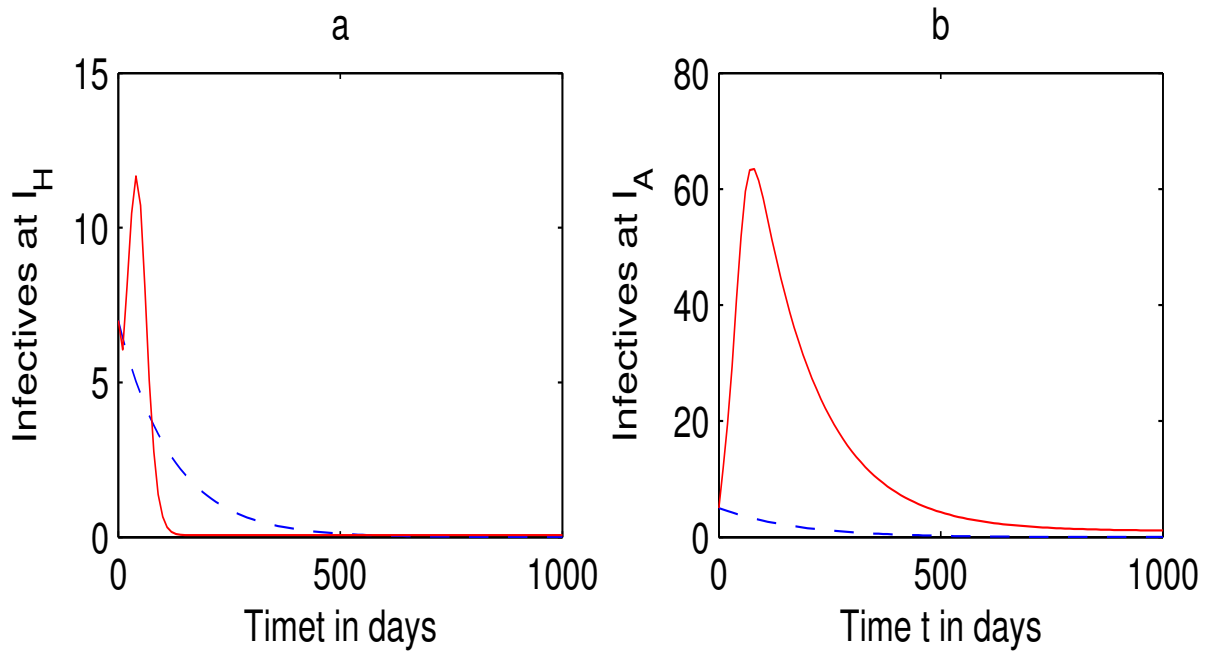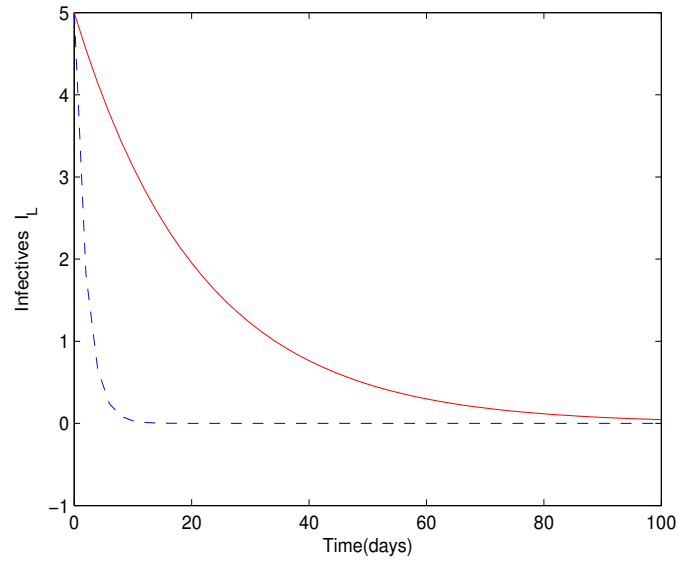


**Figure 1:***Simulation of model showing the evolution of HIV/AIDS against time; red Continuous line:* $\pi = 1.1 \times 10^{-2}, \iota_H = 1.0 \times 10^{-4}$ *blue dotted line* $\pi = 1.1 \times 10^{-10}, \iota_H = 9.0 \times 10^{-1}$
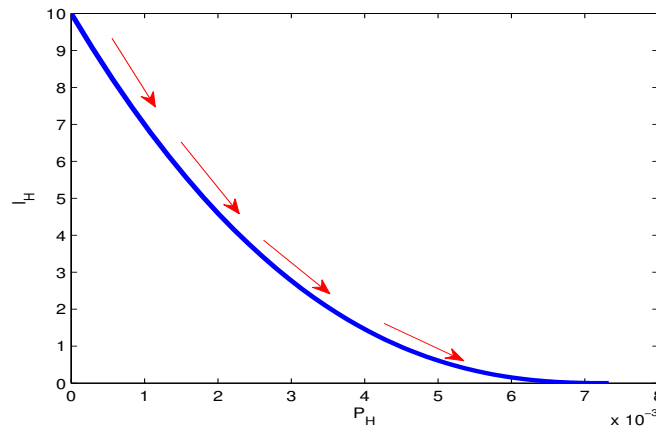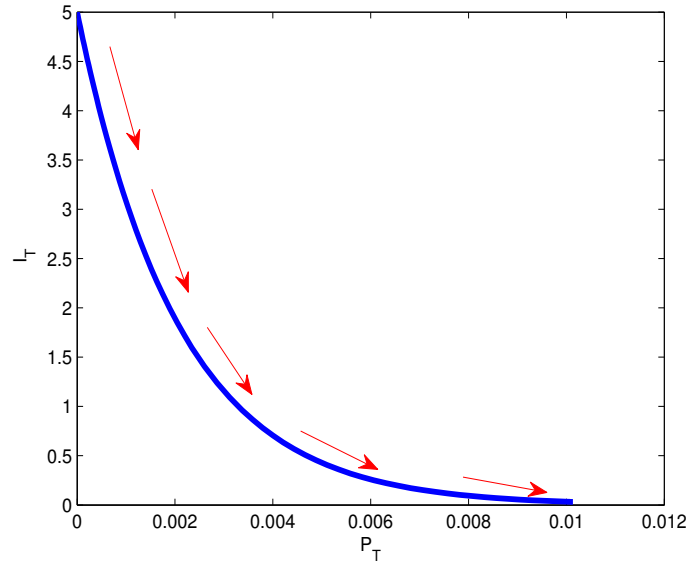


**Figure 2:***The graph of $I_H$ against $P_H$*

*3.7.2 The effect of varying the protection term on TB infection*



**Figure 3:** *evolution of TB against time*
time; red Continuous line: $\theta = 1.1 \times 10^{-2}, \iota_T = 4.0 \times 10^{-1}$ blue dotted line $\theta = 1.1 \times 10^{-4}, \iota_T = 9.0 \times 10^{-1}$



**Figure 4:** *The graph $I_T$ against $P_T$*

# 4  Discussion

From Figure 1(a), we see that , with a low protection rate the probability of infection is high and therefore the number of infectives $I_H$ rises sharply in a short span before drastically dropping. This sharp drop may be attributed to the depletion of susceptibles or the susceptibles embracing protective measures. From the same figure a very high protection rate with a low probability of transmission results in reduced disease prevalence. The time evolution of $I_A$, is lower than that of $I_H$ as depicted by Figure 1(b) since it takes time to progress from $I_H$ to $I_A$ as the body's immunity tries to fight the HIV virus. From Figure 3, with a high rate of protection, and a low probability of disease transmission, the diseases prevalence for TB also goes down. On the other hand, a low protection rate leads to a high disease prevalence.

From Figure 2, we observe that the number of $I_H$ infections reduces with increased protection. Similarly, figure 4 shows that the number of $I_T$ infectives reduces with increased protection. This is consistent with reality.

In order to reduce the number of new HIV/AIDS and TB infections, and reduce the impact of HIV/AIDS on individual, families and communities, there is need to employ strategies such as increasing the public awareness drive to behaviour change and encourage openness, increasing access to voluntary HIV testing and counselling, promoting increased condom use to reduce the spread of HIV infection, improving access to antiretroviral drugs (ARV's) for people living with AIDS, practising proper hygiene in the case of TB and avoiding clouded places. These strategies will help in reducing the economic burden that are borne by a country in giving care and treating the infected individuals. As evidence from these results, it is indeed true that prevention is better than cure.

# 5  Conclusion

In this work, we formulated a model for the co-infecion of HIV/AIDS with TB incorporating protection. To investigate the effect of protection, two cases were considered. That is, case of maximum protection against HIV/AIDS and the case of maximum protection against TB. The existence of the endemic equilibrium for the two cases was established and the stability of the same was analysed. In both cases the endemic equilibrium is found to be globally asymptotically stable. From the numerical simulations, we observe that protection against a disease has the effect of reducing the disease prevalence.

Since protection has been used in a general sense, further research may be car-

ried out to analyze the contribution of specific protective measures in the overall reduction of disease prevalence rates.

# References

[1] B. Song C. Castillo-Chavavez, J.P. Aparicio, Tuberculosis models with fast and slow dynamics: the role of close and casual contacts, *Mathematical Biosciences*, **180** (2002), 187-205. http://dx.doi.org/10.1016/s0025-5564(02)00112-8

[2] C.P. Bhunu, W. Garira and G. Magombedze, Mathematical analysis of a two strain HIV/AIDS model with antiretroviral treatment, *Acta Biotheoretica*, **57** (2009), 361 - 381. http://dx.doi.org/10.1007/s10441-009-9080-2

[3] C. V. De Leon, Constructions of Lyapunov functions for classics SIS, SIR and SIRS epidemic model with variable population size, *Foro-Red-Mat: Revista electronica de contenido matematico*, **26** (2009), 1 - 12.

[4] Centers for Disease Control and Prevention, Mobidity and Mortality weekly report, Nov. 7, **61** (2012).

[5] Central Intelligence Agency (C. I. A) World Factbook, (2014).

[6] D. Bhowmik, Chiranjib, R.M. Chandira, B. Jayakar, K.P.S. Kurmar, Recent trends of drug used treatment of tuberculosis, *Journal of Chemical and Pharmaceutical Research*, **1** (2009), 113 - 133.

[7] Division of disease control, North Dakota, Department of health, 2007.

[8] E.L. Coebett, C.J. Watt, N. Walker, M. Dermot, G.W. Brian, C.R. Mario and D. Cristopher, The growing burden of tuberculosis global trends and interactions with the HIV epidemic, *Arch. Inter. Med.*, **163** (2003), 1009 - 1021. http://dx.doi.org/10.1001/archinte.163.9.1009

[9] F.N. Britton, *Essential Mathematical Biology*, Springer-Verlag London Limited, 2003. http://dx.doi.org/10.1007/978-1-4471-0049-2

[10] J.A. Salomon, D.R. Hogan, J. Stover, K.A. Stanecki and N. Walker, P. D. Ghys, B. Schwartlander, Integrating HIV prevention and treatment: From slogans to impact, *PLOS Med.*, **2** (2005), 50 - 55. http://dx.doi.org/10.1371/journal.pmed.0020016

[11] J.P. Aparicio and C. Castillo-Chavez, Mathematical modeling of tuberculosis epidemics, *Mathematical Biosciences and Engineering*, **6** (2009), 209 - 237. http://dx.doi.org/10.3934/mbe.2009.6.209

[12] Kenya AIDS Indicator Survey 2012 (KAIS), Final Report.

[13] L.J. Abu-Raddad, P. Patnaik and J.G. Kublin, Dual infection with Hiv and malaria fuels the spread of both diseases in sub-saharan Africa, *Science*, **314** (2006), 1603 - 1606. http://dx.doi.org/10.1126/science.1132338

[14] L.M. Arriola and J.M. Hyman, Being sensitive to Uncertainity, Computing Science and Engineering, **9** (2007), 10 - 20. http://dx.doi.org/10.1109/mcse.2007.27

[15] L.W. Roeger, Z. Feng and C. Castillo-Chavez, Modeling TB and HIV co-infections, *Mathematical Biosciences and Engineering*, **6** (2009), 815 - 837. http://dx.doi.org/10.3934/mbe.2009.6.815

[16] M. Harrington, J. Syed, R. Camp, R. Jeffreys and T. Swan, What's in the pipeline: New HIV drugs, vaccines, microbicides, HCV and TB treatments in clinical trials, Treatment Action Group, New York, 2005.

[17] Ministry of Health, Government of Kenya (2001), Guidelines to Antiretroviral drug therapy in Kenya.

[18] N. Malunguza, S. Mushayabasa, C. Chiyaka, Z. Mukandavire, Modelling the effects of condom use and antiretroviral therapy in controlling HIV/AIDS among heterosexuals, homosexuals and bi sexuals, *Compt. Math. Methods in Med.*, **11** (2010), 201-222. http://dx.doi.org/10.1080/17486700903325167

[19] P. Sonnenberg, J. Murray, J.R. Glynn, S. Shearer, K. Bupe, G.F. Peter, HIV-1 and recurrence, relapse, and reinfection of tuberculosis after cure: a cohort study in South African mineworkers, *The Lancet*, **358** (2001), 1687 - 1693. http://dx.doi.org/10.1016/s0140-6736(01)06712-5

[20] S.K. Sharma, A. Mohan and T. Kadhiravan, HIV-TB co-infection: Epidemiology, diagnosis and management, *Indian J. Med. Res.*, **121** (2005), 550 - 567.

[21] S. Russell, The economic burden of illness for households in developing countries: a review of studies focusing on malaria, tuberculosis, and human immunodeficiency virus/acquired immunodeficiency syndrome, *Am. J. Trop. Med. Hyg.*, **71** (Suppl 2) (2004), 147 - 155.

[22] Strategic Frame Work to Decrease the Burden of TB/HIV, World Health Organization, Geneva, Switzerland, March, 2002.

[23] UNAIDS/WHO, Report on the global AIDS epidemic: Executive sum- mary, UNAIDS and WHO, Geneva (2006).

[24] http://matworld.wolfran.com/Routh-HurwitzTheorem.html