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The Impact of HIV/AIDS Treatment and Counseling on the Prevalence of Tuberculosis and Malaria Co Infections

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Abstract

HIV/AIDS remains one of the leading causes of death in the world with its effects most devastating in Sub Saharan Africa due to its dual infection with opportunistic infections especially malaria and tuberculosis.

An investigation of the prevalence level of Tuberculosis (TB) and malaria in the absence of any intervention strategy (treatment and counseling) for HIV/AIDS individuals when R_M and R_T is less than unity showed that the prevalence level of the two diseases rise with time. This unexpected rise in prevalence when $R_M < 1$ and $R_T < 1$ is due to the presence of HIV/AIDS. Administration of treatment and counseling for

the HIV/AIDS individuals at this lower levels of the reproduction numbers of TB and malaria, reduces the prevalence levels of TB and malaria. The study further observed that when the reproduction number of TB and malaria is greater than unity, then counseling and treatment for the HIV/AIDS individuals is more effective in reducing the prevalence level of TB and malaria in the population both at the initial stages of the administration of the strategies and in the long run.

Keywords: HIV/AIDS - rotavirus malaria, equilibria, stability, bifurcation, sensitivity, counseling, treatment.

1 Background Information

Research at the interface of mathematics and biology is increasing, and virtually any advance in disease dynamics today requires a sophisticated mathematical approach in order to map out the parameters necessary for control and containment of epidemic outbreaks.
Infectious diseases, alongside cardiovascular diseases and cancer, have been the main threat to human health. Acute and chronic respiratory diseases, especially pulmonary tuberculosis, malaria and HIV/AIDS are responsible for a large portion of mortality especially in developing countries [8].
The normal $CD4^+$ cell counts in a healthy HIV negative adult vary from 500 cells/μl (500 cells per mm^3 of blood) to 1500 cells/μl [8]. Audu *et al.*[2] investigated the possible impact of co infections of tuberculosis and malaria on the $CD4^+$ cell counts of HIV/AIDS patients and established the following: The healthy control group recorded a median $CD4^+$ cell counts of 789 cells/μl; subjects infected with HIV/AIDS only recorded a median $CD4^+$ cell counts of 386 cells/μl; subjects co infected with HIV/AIDS and TB recorded a median $CD4^+$ cell counts of 268 cells/μl; subjects co infected with HIV/AIDS and malaria recorded a median $CD4^+$ cell counts of 211 cells/μl and those co infected with HIV/AIDS, malaria and TB recorded the lowest median $CD4^+$ cell counts of 182 cells/μl.
Motivated by this finding, a deterministic model exploring the joint dynamics of the simultaneous co infections of HIV/AIDS, TB and malaria incorporating treatment and counseling for the HIV/AIDS infected individuals is formulated, analysed and simulated.

2 HIV/AIDS, TB and Malaria Co Infections Model

HIV/AIDS, TB and malaria co infections model is presented by the system of ordinary differential equations 2.0.1 and analysed using the Wolfram Research Mathematica software.

$$
\begin{aligned}
\frac{dS_H(t)}{dt} &= \Lambda_H + r_m I_M(t) + r_t I_T(t) - \lambda_{ah} S_H(t) \\
&\quad -\lambda_{mh} S_H(t) - \lambda_{th} S_H(t) - d_n S_H(t) \qquad (2.0.1)\\
\frac{dI_M(t)}{dt} &= \lambda_{mh} S_H(t) + r_t I_{MT}(t) - r_m I_M(t) - e_m^h \lambda_{ah} I_M(t) \\
&\quad -\lambda_{th} I_M(t) - d_n I_M(t) - d_m I_M(t). \\
\frac{dI_H(t)}{dt} &= \lambda_{ah} S_H(t) + r_m I_{HM}(t) + r_t I_{HT}(t) - (1-\alpha) p I_H(t) \\
&\quad -e_h^m \lambda_{mh} I_H(t) - e_h^t \lambda_{th} I_H(t) - d_n I_H(t) + \alpha I_A(t). \\
\frac{dI_A(t)}{dt} &= (1-\alpha) p I_H(t) + r_m I_{AM}(t) + r_t I_{AT}(t) - e_a^m \lambda_{mh} I_A(t) \\
&\quad -e_a^t \lambda_{th} I_A(t) - d_a I_A(t) - d_n I_A(t) - \alpha I_A(t) \\
\frac{dI_T(t)}{dt} &= \lambda_{th} S_H(t) + r_m I_{MT}(t) - e_t^h \lambda_{ah} I_T(t) - \lambda_{mh} I_T(t) \\
&\quad -d_n I_T(t) - d_t I_T(t) - r_t I_T(t) \\
\frac{dI_{HM}(t)}{dt} &= e_h^m \lambda_{mh} I_H(t) + e_m^h \lambda_{ah} I_M(t) + r_t I_{HMT}(t) - r_m I_{HM}(t) \\
&\quad -e_{hm}^t \lambda_{th} I_{HM}(t) + \alpha I_{AM}(t) \\
&\quad -d_m I_{HM}(t) - (1-\alpha)\theta_2 p I_{HM}(t) - d_n I_{HM}(t) \\
\frac{dI_{AM}(t)}{dt} &= (1-\alpha)\theta_2 p I_{HM}(t) + e_a^m \lambda_{mh} I_A(t) - r_m I_{AM}(t) \\
&\quad -d_m I_{AM}(t) - \alpha I_{AM}(t) + r_t I_{AMT}(t) - e_{am}^t \lambda_{th} I_{AM}(t) \\
&\quad -d_n I_{AM}(t) - d_a I_{AM}(t) - d_{am} I_{AM}(t). \\
\frac{dI_{MT}(t)}{dt} &= \lambda_{th} I_M(t) + \lambda_{mh} I_T(t) - r_m I_{MT}(t) - e_{mt}^h \lambda_{ah} I_{MT}(t) - r_t I_{MT}(t) \\
&\quad -d_m I_{MT}(t) - d_n I_{MT}(t) - d_t I_{MT}(t) - d_{mt} I_{MT}. \\
\frac{dI_{HT}(t)}{dt} &= e_t^h \lambda_{ah} I_T(t) + r_m I_{HMT}(t) + e_h^t \lambda_{th} I_H(t) - e_{ht}^m \lambda_{mh} I_{HT}(t) \\
&\quad -(1-\alpha)\theta_1 p I_{HT}(t) - d_n I_{HT}(t) - d_t I_{HT}(t) - r_t I_{HT}(t) + \alpha I_{AT}(t) \\
\frac{dI_{AT}(t)}{dt} &= e_a^t \lambda_{th} I_A(t) + r_m I_{AMT}(t) + (1-\alpha)\theta_1 p I_{HT}(t) - \alpha I_{AT}(t) \\
&\quad -e_{at}^m \lambda_{mh} I_{AT}(t) - d_n I_{AT}(t) - d_a I_{AT}(t) - d_t I_{AT}(t) \\
&\quad -r_t I_{AT}(t) - d_{at} I_{AT}. \\
\frac{dI_{HMT}(t)}{dt} &= e_{ht}^m \lambda_{mh} I_{HT}(t) + e_{hm}^t \lambda_{th} I_{HM}(t) + e_{mt}^h \lambda_{ah} I_{MT}(t) + \alpha I_{AMT}(t) \\
&\quad -r_m I_{HMT}(t) - d_m I_{HMT}(t) - d_n I_{HMT}(t) \\
&\quad -(1-\alpha)\theta_3 p I_{HMT}(t) - d_t I_{HMT}(t) - r_t I_{HMT}(t) - d_{mt} I_{HMT}
\end{aligned}
$$

$$\begin{aligned}
\frac{dI_{AMT}(t)}{dt} &= e_{at}^{m}\lambda_{mh}I_{AT}(t) + e_{am}^{t}\lambda_{th}I_{AM}(t) + (1-\alpha)\theta_3 pI_{HMT}(t) \\
&\quad -r_m I_{AMT}(t) - d_m I_{AMT}(t) - d_a I_{AMT}(t) - \alpha I_{AMT}(t) \\
&\quad -d_n I_{AMT}(t) - d_t I_{AMT}(t) - r_t I_{AMT}(t) - d_{amt} I_{AMT} \\
\frac{dS_V(t)}{dt} &= \Lambda_V - \lambda_{mv}S_V(t) - d_v S_V(t) \\
\frac{dI_V(t)}{dt} &= \lambda_{mv}S_V(t) - d_v I_V(t).
\end{aligned}$$

The rate of change of the total human population with time is given by: $\frac{dN_H}{dt} = \Lambda_H - d_n N_H - (I_M + I_{HM}(t) + I_{AM} + I_{MT} + I_{HMT} + I_{AMT})d_m\ (I_T + I_{MT} + I_{HT} + I_{AT} + I_{HMT} + I_{AMT})d_t - (I_A + I_{AM} + I_{AT} + I_{AMT})d_a - d_{am}I_{AM} - d_{mt}(I_{MT} + I_{HMT}) - d_{at}I_{AT} - d_{amt}I_{AMT}$

2.1 Positivity of Solutions

The model system 2.0.1 describes living populations therefore the associated state variables are non-negative for all time $t \geq 0$. The solutions of this model with positive initial data therefore remain positive for all time $t \geq 0$.

Lemma 2.1. *Let the initial data set be* $\{(S_H(0), S_V(0) > 0), (I_M(0), I_H(0), I_A(0), I_T(0), I_{HM}(0), I_{AM}(0), I_{MT}(0), I_{HT}(0), I_{AT}(0), I_{HMT}(0), I_{AMT}(0), I_V(0))\} \in \Psi$. *Then the solution set* $\{(S_H, S_V, I_M, I_H, I_A, I_T\ I_{HM}, I_{AM}, I_{MT}, I_{HT}, I_{AT}, I_{HMT}, I_{AMT}, I_V\}(t)$ *is positive for all time* $t > 0$.

Proof. Consider the first equation of 2.0.1 at time t

$$\frac{dS_H}{dt} = \Lambda_H + r_m I_M + r_t I_T - \lambda_{ah}S_H - \lambda_{mh}S_H - \lambda_{th}S_H - d_n S_H$$

then

$$\frac{dS_H}{dt} \geq -(\lambda_{ah} + \lambda_{mh} + \lambda_{th} + d_n)S_H$$

$$\int \frac{dS_H}{S_H} \geq -\int(\lambda_{ah} + \lambda_{mh} + \lambda_{th} + d_n)d(t)$$

$$S_H(t) \geq S_H(0)e^{-\int(\lambda_{ah}+\lambda_{mh}+\lambda_{th}+d_n)d(t)} \geq 0$$

From the second equation of 2.0.1 at time t

$$\frac{dI_M}{dt} = \lambda_{mh}S_H + r_t I_{TM} - r_m I_M - e_m^a \lambda_{ah} I_M - \lambda_{th} I_M - d_n I_M - d_m I_M.$$

then

$$\frac{dI_M}{dt} \geq -(r_m + e_m^a\lambda_{ah} + \lambda_{th} + d_n + d_m)I_M.$$

$$\frac{dI_M}{I_M} \geq -\int(r_m + e_m^a\lambda_{ah} + \lambda_{th} + d_n + d_m)dt.$$

$$I_M(t) \geq I_M(0)e^{-\int(r_m+e_m^a\lambda_{ah}+\lambda_{th}+d_n+d_m)dt} \geq 0.$$

We can proceed in a similar manner and show that all the state variables are positive for all time t. □

2.2 Boundedness of Solutions

The feasible solutions of the model 2.0.1 are uniformly bounded in a proper subset $\Psi = \Psi_H \times \Psi_V$, where $\Psi_H = \{(S_H, I_M, I_H, I_A, I_T, I_{MH}, I_{MA}, I_{MT}, I_{HT}, I_{TA}, I_{MHT}, I_{MAT}) : N(t) \leq \frac{\Lambda_H}{d_n}\}$ and $\Psi_V = \{(S_V, I_V) : N_V \leq \frac{\Lambda_V}{d_v}\}$. This implies that all the solutions in the boundary of Ψ enter the interior of Ψ eventually.

Lemma 2.2. *The solutions of the model 2.0.1 are uniformly bounded in a proper subset* $\Psi = \Psi_H \times \Psi_V$

Proof. Let $\{(S_H, I_M, I_H, I_A, I_T\, I_{HM}, I_{AM}, I_{MT}, I_{HT}, I_{AT}, I_{HMT}, I_{AMT}\}(t) \in \mathbb{R}_+^{12}$, be any solution with non-negative initial conditions. The rate of change of the total human population with time is given by:

$$\frac{dN_H}{dt} = \Lambda_H - d_nN_H - (I_M + I_{HM}(t) + I_{AM} + I_{MT} + I_{HMT} + I_{AMT})d_m \text{ -}$$

$$(I_T + I_{MT} + I_{HT} + I_{AT} + I_{HMT} + I_{AMT})d_t - (I_A + I_{AM} + I_{AT} + I_{AMT})d_a$$

$$-d_{am}I_{AM} - d_{mt}(I_{MT} + I_{HMT}) - d_{at}I_{AT} - d_{amt}I_{AMT}$$

The model system 2.0.1 has a varying human population size since $\frac{dN_H}{dt} \neq 0$ and therefore a trivial equilibrium is not feasible. Whenever $N_H > \frac{\Lambda_H}{d_n}$, then $\frac{dN_H}{dt} < 0$. Since $\frac{dN_H}{dt}$ is bounded by $\Lambda_H - d_nN_H$, a standard comparison theorem by (Birkoff and Rota, 1989) shows that $0 \leq N_H(t) \leq N_H(0)e^{-d_n} + \frac{\Lambda_H}{d_n}(1-e^{-d_n})$, where $N_H(0)$ represents the value of $N_H(t)$ evaluated at the initial values of the respective variables. Thus as $t \to \infty$, we have, $0 \leq N_H(t) \leq \frac{\Lambda_H}{d_n}$. In particular, $N_H(t) \leq \frac{\Lambda_H}{d_n}$, if $N_0 \leq \frac{\Lambda_H}{d_n}$. This shows that N_H is bounded and all the feasible solutions of the human only component of model 2.0.1 starting in the region Ψ_H approach, enter or stay in the region Ψ_H.
Similarly let $\{(S_V, I_V\}(t) \in \mathbb{R}_+^2$, be any solution with non-negative initial conditions. The rate of change of the total vector population with time is given by:$\frac{dN_V}{dt} = \Lambda_V - (S_V(t) - I_V(t))d_v$. $\frac{dN_V}{dt} \neq 0$ and therefore a trivial equilibrium is not feasible. Whenever $N_V > \frac{\Lambda_V}{d_v}$, then $\frac{dN_V}{dt} < 0$. Since $\frac{dN_V}{dt}$ is bounded by $\Lambda_V - d_vN_V$, a standard comparison theorem by Birkoff and

Rota (1989), shows that $0 \leq N_V(t) \leq N_V(0)e^{-d_v} + \frac{\Lambda_V}{d_v}(1 - e^{-d_v})$, where $N_V(0)$ represents the value of $N_V(t)$ evaluated at the initial values of the respective variables. Thus as $t \to \infty$, $0 \leq N_V(t) \leq \frac{\Lambda_V}{d_v}$. In particular, $N_V(t) \leq \frac{\Lambda_V}{d_v}$, if $N_0 \leq \frac{\Lambda_V}{d_v}$. This shows that N_V is bounded and all the feasible solutions of the vector only component of model 2.0.1 starting in the region Ψ_V approach, enter or stay in the region Ψ_V. $\square$.

2.3 Disease-Free Equilibrium Point of the Model

In the absence of infection by all the diseases, the model 2.0.1, has a steady-state solution called the DFE given by $\mathcal{E}_0^{hmt} = (S_H, I_M, I_H, I_A, I_T, I_{HM}, I_{AM}, I_{MT}, I_{HT}, I_{AT}, I_{HMT}, I_{AMT}, S_V, I_V) = (\frac{\Lambda_H}{d_n}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_V}{d_v}, 0)$. Define $\mathcal{F}_i$ as the rate of appearance of new infections in the class or compartment i and $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$, where $\mathcal{V}_i^-$ is the rate of transfer of individuals out of compartment i, and $\mathcal{V}_i^+$ is the rate of transfer of individuals into compartment i by all other means. The Jacobian of $\mathcal{F}_i$ and $\mathcal{V}_i$ at the disease-free equilibrium is given by:

$$F = \begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_1\beta_m \\
0 & a_1 & 0 & 0 & a_1 & 0 & 0 & a_1 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \beta_t c_2 & 0 & 0 & \beta_t c_2 & \beta_t c_2 & \beta_t c_2 & \beta_t c_2 & \beta_t c_2 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\alpha_1\beta_v & 0 & 0 & 0 & \alpha_1\beta_v & \alpha_1\beta_v & \alpha_1\beta_v & 0 & 0 & \alpha_1\beta_v & \alpha_1\beta_v & 0
\end{pmatrix}$$

where: $a_1 = \beta_a(1 - \delta)c_1$

$$V = \begin{pmatrix} u_1 & 0 & 0 & 0 & 0 & 0 & -r_t & 0 & 0 & 0 & 0 & 0 \\ 0 & u_2 & -\alpha & 0 & -r_m & 0 & 0 & -r_t & 0 & 0 & 0 & 0 \\ 0 & z_1 & u_3 & 0 & 0 & -r_m & 0 & 0 & -r_t & 0 & 0 & 0 \\ 0 & 0 & 0 & u_4 & 0 & 0 & -r_m & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & u_5 & -\alpha & 0 & 0 & 0 & -r_t & 0 & 0 \\ 0 & 0 & 0 & 0 & z_2 & u_6 & 0 & 0 & 0 & 0 & -r_t & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & u7 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & u_8 & -\alpha & -r_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & z_3 & u_9 & 0 & -r_m & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & u_{10} & -\alpha & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & z_4 & u_{11} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & d_v \end{pmatrix}$$

where $z_1 = -(1-\alpha)p,\ z_2 = -(1-\alpha)\theta_2 p,\ z_3 = -(1-\alpha)\theta_1 p,\ z_4 = -(1-\alpha)\theta_3 p, u_1 = r_m + d_n + d_m,\ u_2 = (1-\alpha)p + d_n,\ u_3 = \alpha + d_a + d_n,\ u_4 = d_n + d_t + r_t,\ u_5 = r_m + d_m + (1-\alpha)\theta_2 p + d_n,\ u_6 = r_m + d_m + \alpha + d_n + d_a + d_{am},\ u_7 = r_m + r_t + d_m + d_n + d_t + d_{mt},\ u_8 = (1-\alpha)\theta_1 p + d_n + d_t + r_t,\ u_9 = \alpha + d_n + d_{at} + d_t + r_t,\ u_{10} = r_m + d_m + d_n + (1-\alpha)\theta_3 p + d_t + r_t + d_{mt},\ u_{11} = r_m + d_m + d_a + \alpha + d_n + d_t + r_t + d_{amt}$

The basic reproduction number $R_0 = R_{HMT}$ is the spectral radius of the matrix FV^{-1} and is given by $R_{HMT} = \max\{R_M, R_H, R_T\}$.

Where:

$$R_M = \frac{\alpha_1 \sqrt{\beta_m \beta_v}}{\sqrt{d_m d_v + d_n d_v + d_v r_m}} \tag{2.3.1}$$

$$R_T = \frac{\beta_t c_2}{d_n + d_t + r_t} \tag{2.3.2}$$

$$R_H = \frac{c_1(1-\delta)(\alpha + d_a + d_n)\beta_a}{(\alpha d_n + d_a d_n + d_n^2 + d_a p - \alpha d_a p + d_n p - \alpha d_n p)} \tag{2.3.3}$$

Lemma 2.3. *The DFE of the HIV/AIDS,TB and malaria model is locally asymptotically stable (LAS) if $R_{HMT} < 1$, and unstable otherwise.*

Proof. Follows from Theorem 2 by Van and Watmough (2002).

2.4 Parameter values for the HIV/AIDS malaria model

Symbol	Parameter	Value (day^{-1})	Source
Λ_H	Recruitment rate of humans	4.38356×10^4	Kenya demographics profile (2014)
d_n	Natural death rate of humans	4.56630×10^{-5}	Kenya demographics profile (2014)
d_a	HIV/AIDS-induced death rate	1.09589×10^{-3}	WHO report (2014)
p	Progresion rate from HIV to AIDS (untreated)	2.73972×10^{-3}	Baryama, F. and Mugisha, T. (2007)
α	Proportion of the HIV/AIDS victims treated	1.64384×10^{-3}	Kenya NACC report (2014)
β_a	Transmission probability of HIV/AIDS	0.019	Baryama, F. and Mugisha, T. (2007)
c_1	Per capita number of sexual contacts	2.46575×10^{-2}	Kenya NACC report (2014)
δ	Effectiveness of counseling	Variable	
r_m	Proportion of malaria victims treated	1.86301×10^{-3}	WHO report (2013)
d_m	Death rate due to malaria	0.000345	Chitnis *et al* (2006)
α_1	Mosquito biting rate	0.125	Lawi *et al* (2011)
β_m	Transmission probability of malaria in humans	0.8333	Lawi *et al* (2011)
β_v	Transmission probability of malaria in vectors	(0 - 1)	Chiyaka and Dube (2007)
e_{at}^m	Increased susceptibility to malaria due to AIDS and TB co infections	10	Estimated
e_m^h	Reduced susceptibility to malaria due to reduced sexual activity	0.005	Estimated
Λ_V	Recruitment rate of vectors	6	Chiyaka and Dube (2007)
d_v	Death rate of mosquitoes	0.1429	Lawi *et al* (2011)
θ_1	Increased Progresion rate from HIV to AIDS due to TB	1.5	Estimated
θ_2	Increased Progresion rate from HIV to AIDS due to malaria	2	Estimated

Symbol	Parameter	Value (day^{-1})	Source
θ_3	Progresion rate from HIV to AIDS due to TB and malaria	3	Estimated
d_{am}	Death rate due to AIDS and malaria	0.0005175	Baryama, F. and Mugisha, T. (2007)
d_{at}	Death rate due to AIDS and tuberculosis	0.0016438356	WHO report (2013)
β_t	Transmission probability HIV of TB in humans	0.027	Juan and Castillo (2009)
c_2	contact rate of susceptible humans with TB infectives	15	Juan and Castillo (2009)
r_t	Proportion of TB victims treated	0.6	WHO report (2013)
d_{amt}	Death rate due to AIDS, malaria and TB	0.00069	Estimated
e_h^t	Increased susceptibility to TB due to AIDS infection	2.0	Estimated
e_a^t	Increased susceptibility to malaria due to HIV	6	Oluwaseun *et al* (2008)

Lemma 2.3 is illustrated numerically in figure 2.1 using the set of parameter values in table 2.4 with $R_H = 0.51$, $R_T = 0.69$ and $R_M = 0.50$ The figure shows the graph of the total infected population ($I_M + I_H + I_A + I_T + I_{HM} + I_{AM} + I_{MT} + I_{HT} + I_{AT} + I_{HMT} + I_{AMT}$) against time in days at different initial conditions.

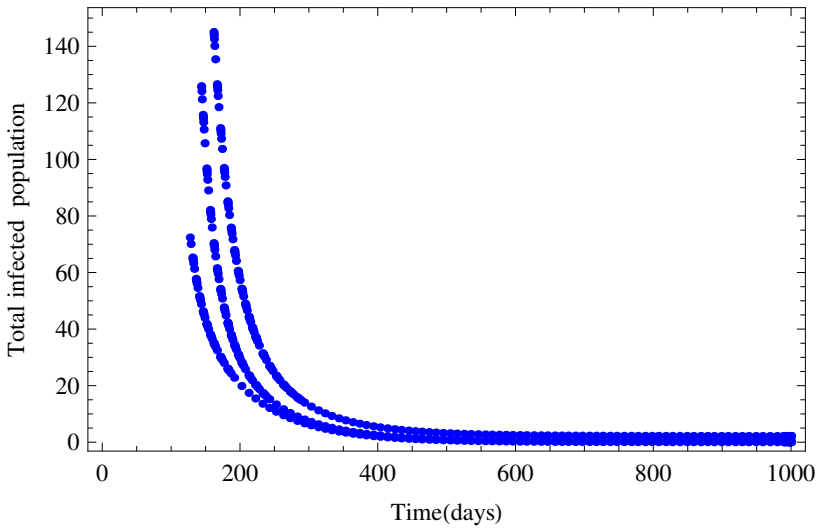


Figure 2.1: Local DFE of the Co Infections Model (R_T= 0.69, $R_H = 0.5$, and $R_M = 0.50$)

2.5 Sensitivity Analysis of Treatment and Counseling

To investigate the potential impact of counseling and treatment on disease progression, sensitivity analysis of the reproduction numbers with respect to counseling and treatment is carried out. The sensitivity index of R_H with respect to δ is given by:

$$R_H^{\delta} = -\frac{\delta}{1-\delta} \tag{2.5.1}$$

The negative sign in equation 2.5.1 indicates that there is an expected decline in the rate of new HIV/AIDS infections when counseling is scaled up.
Similarly, the sensitivity index of R_H with respect to α is given by:

$$R_H^{\alpha} = \frac{\alpha A_1\{-\frac{\beta_a c_1 A_2 A_3(1-\delta)}{A_1^2} + \frac{\beta_a c_1(1-\delta)}{A_1}\}}{\beta_a c_1 A_2(1-\delta)} \tag{2.5.2}$$

$A_1 = \alpha d_n + d_a d_n + d_n^2 + d_a p - \alpha d_a p + d_n p - \alpha d_n p$
$A_2 = \alpha + d_a + d_n$
$A_3 = d_n - d_a p - d_n p$
As shown in section 2.5 the sensitivity index of R_H with respect to treatment is positive indicating that an increase in the proportions of those treated leads to an increase in new HIV cases. The sensitivity index of R_M with respect to r_m is given by:

$$R_M^{r_m} = -\frac{d_v r_m}{2(d_m d_v + d_n d_v + d_v r_m)} \tag{2.5.3}$$

.
Similarly, the sensitivity index of R_T with respect to r_t is given by:

$$R_T^{r_t} = -\frac{r_t}{d_n + d_t + r_t} \tag{2.5.4}$$

The negative sign in equations 2.5.3 and 2.5.4 indicates that there is an expected decline in the rate of new malaria and TB cases when treatment is scaled up.

3 The Impact of HIV/AIDS Treatment and Counseling on the Prevalence Level of TB and Malaria.

In this section an assessment of the role and impact of HIV/AIDS treatment and counseling on the prevalence level of TB and malaria is simulated numerically using the parameter values in section 2.4 unless otherwise stated.

3.1 The Role of HIV/AIDS Treatment and Counseling on the Prevalence Level of TB

The impact of HIV/AIDS treatment and counseling on the prevalence level of TB in the presence of malaria is investigated numerically considering the reproduction numbers of the diseases. The parameter values in section 2.4 yields R_T, the reproduction number of TB as 0.549774. Figure 3.1a shows the numerical simulation of the TB prevalence against time in years in the absence of HIV/AIDS. The simulation shows that in the absence of HIV/AIDS, the TB disease would die out.

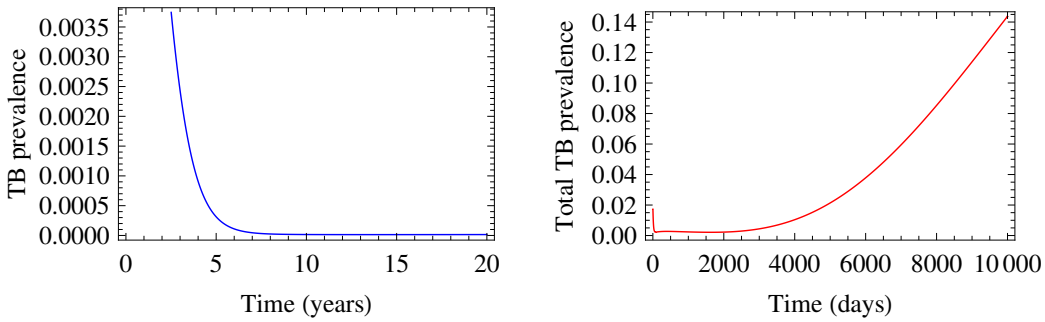


Figure 3.1 : TB Prevalence in the Absence and Presence of HIV/AIDS Respectively.

Figure 3.1b shows the graph of $(I_T + I_{AT} + I_{MT} + I_{AMT} + I_{HT} + I_{HMT}/N_H)$ against time in days indicating that the TB disease continues to persist in the presence of HIV/AIDS, though the reproduction number is less than unity.

Figure 3.1 shows that the TB disease cannot establish itself in a host population of the susceptibles when the reproduction number is less than unity. However in the presence of HIV/AIDS, the TB disease continues to persist even though the reproduction number is less than unity. This reveals the negative impact of the syndemic interactions between HIV/AIDS and TB.

When the reproduction number of TB is greater than unity, then counseling and treatment for the HIV/AIDS individuals is very effective in reducing the prevalence level of TB in the population both at the initial stages of the administration of the strategies and in the long run as illustrated in figure 3.2 where $R_T = 1.2828$.

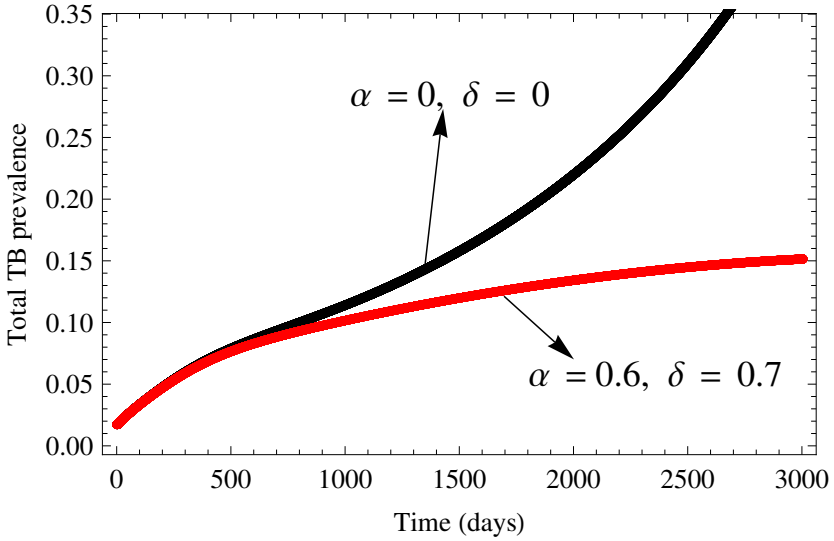


Figure 3.2: TB Prevalence in the Presence of HIV/AIDS when $R_T > 1$.

3.2 The Role of HIV/AIDS Treatment and Counseling on the Prevalence Level of Malaria

The parameter values in section 2.4 gives the reproduction number of malaria to be greater than one ($R_M = 1.53103$). Numerical simulations of malaria prevalence with time without treatment and counseling for the HIV/AIDS infected individuals yields a positive gradient indicating that the prevalence increases with time when $R_M > 1$ as shown in figure 3.3a. However when treatment and counseling for the HIV/AIDS individuals is applied, the prevalence level of malaria increases but at a slower rate as shown in figure 3.3b. This implies that attempts to control malaria from the population must also include treatment and counseling for the HIV/AIDS individuals.

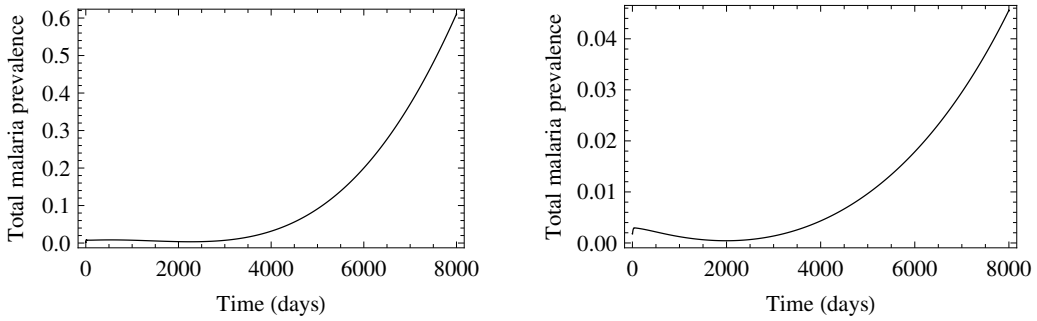


Figure 3.3: Malaria Prevalence in the Absence and Presence of HIV/AIDS Treatment and Counseling.

3.3 The Role of HIV/AIDS Treatment and Counseling on the Prevalence Level of TB and Malaria

In this section the prevalence level of TB and malaria is investigated both in the presence and absence of treatment and counseling for the HIV/AIDS individuals. Starting from lower levels of the reproduction numbers when R_M and R_T is less than one ($R_T = 0.549774$ and $R_M = 0.627724$), the graph of TB and malaria prevalence $(I_M + I_T + I_{MT} + I_{HM} + I_{HT} + I_{AT} + I_{AM} + I_{AMT} + I_{HMT})/(N_H)$ against time is expected to yield a negative gradient (fall). However the opposite happens as shown in figure 3.4.

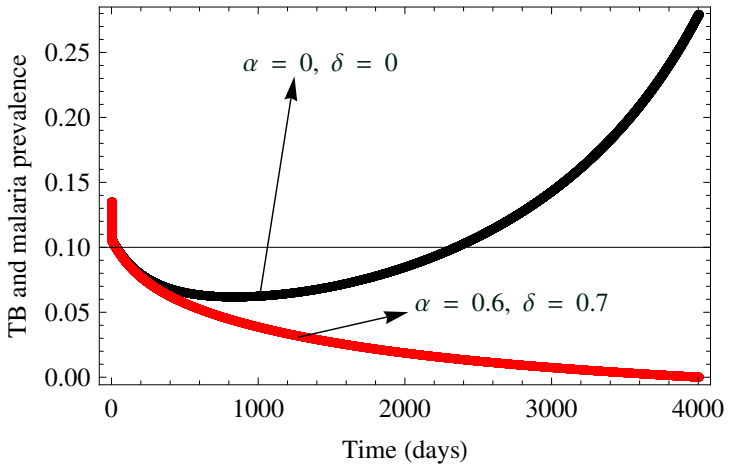
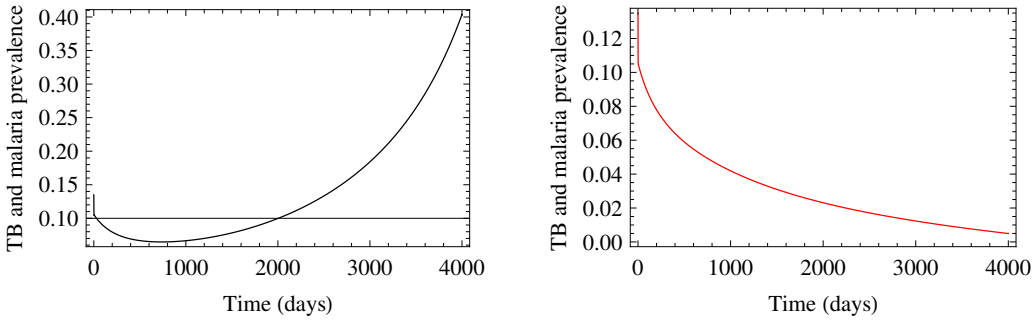


Figure 3.4: The Role of HIV/AIDS Treatment and Counseling on the Prevalence Level of TB and Malaria.

This figure shows that even at lower levels of the reproduction numbers ($R_M < 1$ and $R_T < 1$), in the absence of HIV/AIDS counseling and treatment ($\alpha = 0, \delta = 0$), the prevalence level of the two diseases rise with time. This un-

expected rise in prevalence when $R_M < 1$ and $R_T < 1$ is due to the presence of HIV/AIDS. However in the application of treatment and counseling ($\alpha = 0.6, \delta = 0.7$) for the HIV/AIDS individuals, the prevalence level of the two diseases reduces with time.
Using the parameter values in section 2.4 and adjusting the value of c_2, to give a higher level of R_T (when $R_T = 1.83258$ and $R_M = 1.53103$), the graph of malaria and TB prevalence with time is shown in figure 3.5. Figure 3.5a shows that in the absence of HIV/AIDS treatment and counseling ($\alpha = 0, \delta = 0$), the malaria and TB prevalence would rise as expected, however in the application of treatment and counseling ($\alpha = 0.6, \delta = 0.7$), the prevalence falls (figure 3.5b). Therefore treatment and counseling for the HIV/AIDS individuals is effective in controlling TB and malaria even at higher levels of R_M and R_T both at the initial stages of the application of counseling and treatment and in the long run.



(a) (b)

Figure 3.5: TB and Malaria Prevalence in the Absence and Presence of HIV/AIDS Counseling and Treatment.

3.4 The Threshold Parameters

To approximate the threshold levels of HIV/AIDS counseling that could eliminate HIV/AIDS, TB, malaria and the co infections from the community, numerical simulations of the total prevalence against time in years is conducted. The simulations show that the threshold levels of HIV/AIDS counseling is about 65 percent using the parameter values in section 2.4 as shown in figure 3.6. In figure 3.6a the value of counseling is 70 percent ($\delta = 0.7$), which eliminates the co infections whereas in figure 3.6b, the value of δ is varied from zero percent to 70 percent.

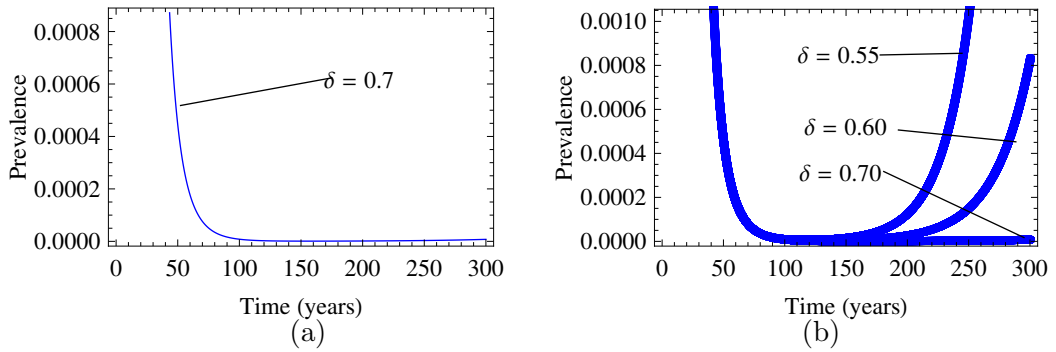


Figure 3.6: The Threshold Level of HIV/AIDS Treatment and Counseling in the Presence of Malaria and TB Treatment.

4 Conclusion.

Treatment and counseling for the HIV/AIDS individuals reduces the prevalence level of both TB and malaria in the community whereas treatment of TB and malaria has insignificant effect on the prevalence level of HIV/AIDS. The HIV/AIDS - TB and malaria model showed that in the absence of HIV/AIDS with the reproduction numbers of TB and malaria less than unity, TB and malaria would die out, however in the presence of HIV/AIDS, TB and malaria continues to persist though their reproduction numbers is less than unity.

An investigation of the prevalence level of TB and malaria in the absence of any intervention strategy (treatment and counseling) but in the presence of HIV/AIDS when R_M and R_T is less than unity showed that the prevalence level of the two diseases rise with time. This unexpected rise in prevalence when $R_M < 1$ and $R_T < 1$ is due to the presence of HIV/AIDS which shows the negative impact of the syndemic interactions between HIV/AIDS TB and malaria. Administration of treatment and counseling for the HIV/AIDS individuals at this lower levels of the reproduction numbers of TB and malaria, reduces the prevalence levels of TB and malaria.

When the reproduction number of TB and malaria is greater than unity then counseling and treatment for the HIV/AIDS individuals is very effective in reducing the prevalence level of TB and malaria in the population both at the initial stages of the administration of the strategies and in the long run. This implies that attempts to control malaria and TB from the population must also include treatment and counseling for the HIV/AIDS individuals.

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