Transactions of the Nigerian Association of Mathematical Physics
Volume 5,(September and November, 2017), pp285 –292

Population dynamics of a mathematical model for TB-Dengue co-infection

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Abstract

This work provides mathematical analysis of a mathematical model describing the co-infection of Tuberculosis and Dengue in a population where both diseases are endemic. Sub-models describing the dynamics of both diseases separately are analysed and results from the numerical simulations of the complete model, showing the impact of key parameters on the co-endemicity of both diseases, are discussed.

1. INTRODUTION

Tuberculosis (TB) is an airborne disease. *Mycobacterium tuberculosis* , the bacterial agent that causes TB, droplets are released into the air during coughing or sneezing by infectious individuals [1]. According to the World Health Organization (WHO), about 9 million persons were infected with TB in 2013, with about 1.5 million deaths reported [2]. On the average, TB incidence fell to about 1.5% per year, between 2000 and 2013 [2]. More than half of the approximately 9 million individuals infected in 2013 were in South-East Asia and Western Pacific. One quarter of these individuals are in the Africa Region, accounting for the highest rates of TB cases and deaths relative to population [2].

A TB vaccine called BCG (Bacillus of Chalmette and Guerin) has been available for many decades. Made of a live, weakened strain of mycobacterium Bovis (a cousin of mycobacterium tuberculosis), it remains the only vaccine available against tuberculosis till date [3]. The BCG vaccine is cheap. In Nigeria, the BCG vaccine is usually given to children when they are born as part of the vaccination program by the Federal Government [4]. The vaccine is essential for children who have a negative tuberculin test and who are continuously exposed and cannot be separated from adult who are untreated or ineffectively treated for TB [3].

Dengue is a viral, vector borne disease, spread by the Aedes Aegypti mosquito [5]. It was estimated that about 50 million infections occur annually in over 100 countries [6]. There is no specific treatment for curing dengue patients [5]. Hospital treatment, in general, is given as supportive care which includes bed rest and analgesics [5].

Dengue virus is one of the most difficult arboviruses to isolate [5]. There are four serotypes of the dengue virus; Den-1, Den-2, Den-3, Den-4, and each of the serotypes has numerous virus strains [5]. Infection with one dengue serotypes may provide long life immunity to that serotype, but there is no complete cross-protective immunity to other serotype [7]. Identification of the primary target cells of dengue viruses' replication in the infected human body has proven to be extremely difficult [5].

There are 22 Tuberculosis (TB) high burden countries worldwide, and together they account for about 80% of the world's tuberculosis (TB) infection [8]. India accounts for over 20% of the world's multi-drugs resistant tuberculosis (MDR-TB) cases [8]. Dengue fever risk is present throughout India, including most metropolitan cities and towns [8]. The purpose of this work is to investigate the population dynamics of TB-Dengue coinfection in the presence of treatment for both diseases, taking into cognisance the public health burden both diseases can have on the governmental public health plans.

2.0 Model Formulation

Let $N_H(t)$ and $N_V(t)$ denote the total number of humans and vectors at time t, respectively. The model sub-divides these populations into a number of mutually-exclusive compartments, as given below.

The total population of human and vectors is divided into the following mutually exclusive epidemiological classes, namely, susceptible humans ($S_H(t)$), humans with latent TB ($E_T(t)$), humans with active TB ($I_T(t)$), humans treated of active TB ($T_T(t)$), humans with latent dengue ($E_1(t)$), humans with dengue ($I_I(t)$), humans treated of dengue ($R_1(t)$), susceptible vectors ($S_V(t)$), vectors at the latent stage of dengue ($E_V(t)$), vectors infectious with dengue ($I_V(t)$), humans with latent TB and latent dengue ($E_2(t)$), humans with latent TB and infectious dengue ($E_3(t)$), humans with active TB and latent dengue ($E_4(t)$), and humans with active TB and dengue ($I_2(t)$). Hence, we have that,

$$N_H(t) = S_H(t) + E_T(t) + I_T(t) + T_T(t) + E_1(t) + I_1(t) + R_1(t) + E_2(t) + E_3(t) + E_4(t) + I_2(t)$$

and

$$N_V(t) = S_V(t) + E_V(t) + I_V(t)$$

Susceptible humans are recruited at a rate Λ_H while the susceptible vectors are recruited at a rate Λ_V. Susceptible humans contract TB at a rate

$$\lambda_T = \frac{\beta_T \left(I_T + \eta_{T1} E_4 + \eta_{T2} I_2\right)}{N_H},$$

$$\lambda_{DV} = \frac{\beta_{VH} \left(\eta_v E_v + I_v\right)}{N_H},$$

where $\eta_v < 1$ accounts for the relative infectiousness of vectors with latent dengue (E_V) compared to vectors in the I_V class. Susceptible vectors acquire dengue infection from infected humans at a rate

$$= \frac{\beta_{HV}(\eta_A E_1 + \eta_B I_1 + \eta_C E_2 + \eta_D E_3 + \eta_E E_4 + \eta_F I_2)}{N_H}.$$

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The modification parameters η_B, η_C, η_D, and η_F account for the relative infectiousness of those in the I_1, E_2, E_3 and I_2 classes compared to those in the E_1 and E_4 classes, where $\eta_A = \eta_E < 1$.

2.1 Derivation of Model Equations

Individuals in the E_T, E_2 and E_3 classes can be exogenously re-infected at the rate $\sigma_1\lambda_T, \sigma_2\lambda_T$ and $\sigma_3\lambda_T$, respectively, where σ_1, σ_2 and σ_3 are modification parameters. A fraction P_{T1} $(0 \le P_{T1} \le 1)$ of susceptible and treated individual's progress faster to the I_T class while a fraction $(1-P_{T2})$ $(0 \le P_{T2} \le 1)$ of those treated for dengue progress faster to the I_T class.

Also, a fraction $(1-P_{D1})$ $(0 \le P_{D1} \le 1)$ of individuals in the E_1 class progress faster to the E_4 class and $(1-P_{D2})$ $(0 \le P_{D2} \le 1)$ of those in the I_1 class progress faster to the I_2 class.

Active TB is treated at a rate r_1, r_2 and r_3 for those in the classes I_T, E_4 and I_2 classes, respectively, while dengue is treated at a rate τ_1, τ_2 and τ_3 for those in I_1, E_3 and I_2 classes respectively. Singly infected individuals with latent TB progress to active TB at a rate k_1, while dually infected individuals in the E_2 class progress to the E_4 class at the rate k_2. Individuals in the E_3 class progress to the I_2 class at the rate k_3. Singly infected individuals with latent dengue progress to active dengue at a rate γ_1 while dually infected individuals in the E_2 class progress to the E_3 class at the rate γ_2. Infected individuals in the E_4 class progress to the I_2 class at a rate γ_3.

Natural death in humans occurs at a rate μ_H in the classes S_H, E_T, I_T, T_T, E_1, I_1, R_1, E_2, E_3, E_4 *and* I_2 while those in the I_T, E_4 and I_2 classes undergo an additional TB induced death at the rates d_{T1}, d_{T2} and d_{T3}, respectively. Individuals in the I_1, E_3 and I_2 classes undergo an additional dengue induced death, at rates δ_{D1}, δ_{D2} and δ_{D3}, respectively. Treated individuals have a relative difference in susceptibility to TB after a previous infection compared to wholly susceptible individuals (with $\varepsilon \ge 0$ being the modification parameter accounting for this relative difference in susceptibility). Natural vector death occurs, at a rate μ_V, in the classes S_V, E_V *and* I_V, while the vectors in the I_V class undergoes additional dengue induced death, at a rate δ_{HV}, although this is negligible as infected vectors are not deemed to be affected by dengue. Exposed vectors progress to the infectious stage at the rate γ_V.

The above assumptions result in the following system of nonlinear ordinary differential equations:

$$
\begin{aligned}
\dot{S}_H &= \Lambda_H - \lambda_T S_H - \mu_H S_H - \lambda_{DV} S_H,\\
\dot{E}_T &= (1-P_{T1})\lambda_T S_H + (1-P_{T1})\epsilon\lambda_T T_T - \sigma_1\lambda_T E_T - (\mu_H + k_1)E_T - \lambda_{DV}E_T + P_{T2}\lambda_T R_1 + \tau_1 E_2,\\
\dot{I}_T &= P_{T1}\lambda_T S_H + P_{T1}\epsilon\lambda_T T_T - (\mu_H + d_{T1} + r_1)I_T + \sigma_1\lambda_T E_T - \lambda_{DV}I_T + (1-P_{T2})\lambda_T R_1 + \tau_3 I_2 + k_1 E_T,\\
\dot{T}_T &= r_1 I_T - \epsilon\lambda_T T_T - \mu_H T_T - \lambda_{DV}T_T,\\
\dot{E}_1 &= \lambda_{DV}S_H + \lambda_{DV}T_T - (\gamma_1 + \mu_H)E_1 - \lambda_T E_1 + r_2 E_4,\\
\dot{I}_1 &= \gamma_1 E_1 - (\tau_1 + \mu_H + \delta_{D1})I_1 - \lambda_T I_1 + r_3 I_2,\\
\dot{R}_1 &= \tau_1 I_1 - \mu_H R_1 - \lambda_T R_1, \qquad (1)\\
\dot{S}_V &= \Lambda_V - \lambda_{DH}S_V - \mu_V S_V,\\
\dot{E}_V &= \lambda_{DH}S_V - (\gamma_V + \mu_V)E_V,\\
\dot{I}_V &= \gamma_V E_V - (\mu_V + \delta_{HV})I_V,\\
\dot{E}_2 &= \lambda_{DV}E_T + P_{D1}\lambda_T E_1 - (\gamma_2 + k_2 + \mu_H)E_2 - \sigma_2\lambda_T E_2,\\
\dot{E}_3 &= \gamma_2 E_2 + P_{D2}\lambda_T I_1 - (k_3 + \tau_2 + \delta_{D2} + \mu_H)E_3 - \sigma_3\lambda_T E_3,\\
\dot{E}_4 &= (1-P_{D1})\lambda_T E_1 + \lambda_{DV}I_T + k_2 E_2 - (d_{T2} + r_2 + \gamma_3 + \mu_H)E_4 + \sigma_2\lambda_T E_2,\\
\dot{I}_2 &= (1-P_{D2})\lambda_T I_1 - (\tau_3 + r_3 + \delta_{D3} + d_{T3} + \mu_H)I_2 + k_3 E_3 + \gamma_3 E_4 + \sigma_3\lambda_T E_3.
\end{aligned}
$$

Table 1 gives the description of the state variables of the model (1) and Table 2 gives the description of the parameters (and their baseline values) of the model (1).

Table 1: Description of the state variables of the model (1)

Variable	Description
S_H	Susceptible human population
E_T	Human population with TB in latent stage (TB only)
I_T	Human population with TB in active stage (TB only)
T_T	Human population treated of TB (TB only)
E_1	Human population with dengue in latent stage (Dengue only)
I_1	Human population with dengue (Dengue only)
R_1	Human population treated of dengue (Dengue only)
S_V	Susceptible vectors population
E_V	Exposed vectors
I_V	Infectious vectors
E_2	Dually infected humans with latent TB and latent dengue
E_3	Dually infected humans with dengue and latent TB
E_4	Dually infected human with active TB and latent dengue
I_2	Dually infected human with active TB and dengue

Table 2: Description of Parameters of the Model (1)

Parameter	Description	Values	Unit	Reference
Λ_H, Λ_V	Recruitment rate into the population of susceptible humans,vectors respectively.	500,10000000	Year^{-1}	[9]
μ_H, μ_v	Natural death for humans, vectors respectively.	0.02041,36.5	Year^{-1} Year^{-1}	[4]
β_T	Effective contact rate for TB.	10	Year^{-1}	Assumed
β_{VH}	Effective contact rate for dengue from vectors to humans	5	Year^{-1}	[9]

β_{HV}	Effective contact rate for dengue from humans to vectors	4	Year^{-1}	[9]
P_{T2}	Fraction of newly infected humans with latent TB	0.9	Year^{-1}	[4]
P_{T1}	Fraction of newly infected humans with active TB	0.3	Year^{-1}	[4]
τ_1, τ_2, τ_3	Dengue treatment rate for I_1,E_3,I_2.	2.5,1.5,2	Ind^{-1} Year^{-1}	[9]
r_1, r_2, r_3	TB treatment rate for $I_T E_4, I_2$.	3,2.5,2.4	Ind^{-1} Year^{-1}	[9]
k_1, k_2, k_3	Progression rate to active. TB	0.02,0.02,0.025	Year^{-1}	[4]
$\gamma_1, \gamma_2 \cdot \gamma_3$	Progression rate to active dengue (humans)	0.3254,0.6462, 0.3265	Year^{-1}	[9].
γ_V	Progression rate to active dengue (vectors).	0.03	Year^{-1}	[9]
$d_{T1}, d_{T2}, d_{T3},/$ $\delta_{D1}, \delta_{D2}, \delta_{D3}$	Disease induced death TB/Dengue (humans)	0.365,0.365,0.375/ 0.365,0.365, 0.365	Year^{-1} Year^{-1}	[4]
δ_{HV}	Disease induced death dengue (vectors)	*0*	Year^{-1}	[9]
k_V	Progression rate to active dengue (vectors)	0.05	Year^{-1}	[9].
$\eta_{T1}, \eta_{T2}, \eta_V,$ $\eta_A, \eta_B, \eta_C,$ η_D, η_E, η_F	Modification parameters for E_4, I_2, E_v, E_1, I_1, E_2, E_3, E_4, I_2.	0.4,1.2,0.5,0.6,1,0. 6,1.1,1,1.5,	Year^{-1}	[4]
σ_1, σ_2, σ_3	Modification parameter for exogenous re-infection.	0.5,0.5,0.7	Year^{-1}	[9]
$\in$	Modification parameter	1.67	Year^{-1}	Assumed
P_{D1}	Fraction of newly infected active TB cases with latent dengue	0.6	Year^{-1}	[9]
P_{D2}	Fraction of newly infected active TB cases with active dengue	0.5	Year^{-1}	[9]

3.0 Analysis of sub-models

Before analyzing the complete model (1), it is instructive to gain insight into the dynamical features of the TB-only model and the dengue- only model.

3.1 TB-only model

The TB only model is derived in (1) by setting $E_1 = R_1 = S_V = E_V = I_V = E_2 = E_3 = E_4 = I_2 = 0$. Hence we have

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \lambda_T S_H - \mu_H S_H, \\
\frac{dE_T}{dt} &= (1-P_{T1})\lambda_T S_H + (1-P_{T1}) \in \lambda_T T_T - \sigma_1 \lambda_T E_T - (\mu_H + k_1) E_T, \\
\frac{dI_T}{dt} &= P_{T1}\lambda_T S_H + P_{T1} \in \lambda_T T_T - (\mu_H + d_{T1} + r_1) I_T + \sigma_1 \lambda_T E_T + k_1 E_T, \\
\frac{dT_T}{dt} &= r_1 I_T - \in \lambda_T T_T - \mu_H T_T,
\end{aligned} \tag{2}$$

where $\lambda_T = \frac{\beta_T I_T}{N_H}$ and $N_H = S_H + E_T + I_T + T_T$.

Consider the region $D_1 = \{(S_H, E_T, I_T, T_T) \in \mathbb{R}_+^4 : N_H \leq \frac{\Lambda_H}{\mu_H}\}$. It can be shown that the set D_1 is positively invariant and a global attractor of all positive solution of the system (2). We claim the following.

Lemma 1 *The region D_1 is positively invariant for the system (2).*

Proof: The rate of change of the total population is give as

$\dot{N}_H(t) = \dot{S}_H + \dot{E}_T + \dot{I}_T + \dot{T}_T = \Lambda_H - \mu_H(S_H + E_T + I_T + T_T) - d_{T1} I_T$

$\dot{N}_H(t) = \Lambda_H - \mu_H N_H - d_{T1} I_T$.

Since the right-hand side of the equation above is bounded by $\Lambda_H - \mu_H N_H$, standard comparison theorem [10] can be used to show that

$N_H \leq N_H(0) e^{-\mu_H t} + \frac{\Lambda_H}{\mu_H}[1 - e^{-\mu_H t}]$.

If $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$ then $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$. Thus, D_1 is a positively invariant set under the flow described in (2). Hence, no solution path leaves through and boundary of D_1. In this region, the model (2) is said to be well posed mathematically and epidemiologically [11].

We now prove the positivity of solutions of the model (2). We claim the following.

Lemma 2. *Let the initial data for the model (2) be $S_H(t) > 0$, $E_T(t) > 0, I_T(t) > 0$, and $T_T(t) > 0$ then the solution $S_H(t)$, $E_T(t)$,$I_T(t)$, and $T_T(t)$with positive initial data will remain positive for all time $t > 0$.*

Proof: Let $t_1 = sup\{t > 0 : S_H(t) > 0, E_T(t) > 0, I_T(t) > 0, T_T(t) > 0\} > 0$. The first equation in (2) is given by

$\dot{S}_H = \Lambda_H - (\lambda_T + \mu_H) S_H$,

which, when solved, leads to

$S_H(t_1) = S_H(0) exp\left\{-\mu_H t_1 - \int_0^{t_1} \lambda_T(\tau) d(\tau)\right\} + \left[exp\left\{-\mu_H t_1 - \int_0^{t_1} \lambda_T(\tau) d(\tau)\right\}\right] \int_0^{t_1} \Lambda_H \left[exp\{\mu_H y + \int_0^y \lambda_T(\tau) d(\tau)\}\right] dy > 0.$

Hence, S_H is positive for all time, t.

Similarly, we can show that $E_T(t) > 0, I_T(t) > 0$, *and* $T_T(t) > 0$ for all time, t.

3.1.1 Local Stability of Disease-Free Equilibrium (DFE) of the TB-only model

The model (2) has a disease-free equilibrium obtained by setting the right hand side of the model to zero, and this is given by

$\xi_1 = (S_H^*, E_T^*, I_T^*, T_T^*) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0\right)$.

The linear stability of ξ_1 is established using the next generation operator method on the system (2) [12]. Using the notation in [12], the matrices F and V, for the new infection terms and the remaining transfer terms respectively, are given by

$F = \begin{pmatrix} 0 & (1-P_{T1})\beta_T \\ 0 & P_{T1}\beta_T \end{pmatrix}$, and $V = \begin{pmatrix} g_1 & 0 \\ -k_1 & g_2 \end{pmatrix}$.

It follows that the effective reproduction number of the model (2), denoted by R_T, is given by $R_T = \rho(FV^{-1}) = \dfrac{\beta_T\left(g_1 P_{T1} + k_1(1-P_{T1})\right)}{g_1 g_2}$,

where $\rho(FV^{-1})$ is the spectral radius of the matrix FV^{-1}.The next result follows from Theorem 2 in [12].

Lemma 3 *The DFE, ξ_1, of the model (2) is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.*

The threshold quantity, R_T, is the effective reproduction number for the TB sub-model. It represents the average number of secondary TB infections generated by a typical infected individual in a completely susceptible population where treatment for TB is available. Epidemiologically speaking, Lemma 3 implies that TB can be eliminated from the population when $R_T < 1$ if the initial sizes of the sub-population of the sub-model are in the basin of attraction of ξ_1. Hence, a small influx of TB-infected individuals into the community will not generate large TB outbreaks, and the disease will die out with time.

3.1.2 Existence and Local Stability of Endemic Equilibrium Point (EEP) of the TB-Only Model.

Let the EEP of model (2) be denoted by $\xi_{(1,T)} = (S_H^{**},\ E_T^{**},\ I_T^{**},\ T_T^{**})$. The equations in (2) are solved in terms of the force of infection, at steady state, and the components of the EEP are given as

$$S_H^{**} = \frac{\Lambda_H}{\lambda_T^{**} + \mu_H},$$

$$E_T^{**} = \frac{(1-P_{T1})\lambda_T^{**}\Lambda_H\left(\in \lambda_T^{**} + \mu_H\right)\left(d_{T1} + r_1 + \mu_H\right)}{\left(\lambda_T^{**} \mu_H\right)\left(d_{T1}\left(\in \lambda_T^{**} \mu_H\right)\left(k_1 + \sigma_1\lambda_T^{**} \mu_H\right) + \mu_H\left(k_1\left(r_1 + \in \lambda_T^{**} \mu_H\right) + \left(\in \lambda_T^{**} + \mu_H\right)\right.\right.} \\ \left.\left(\sigma_1\lambda_T^{**} + \mu_H\right) + r_1\left(\left(\sigma_1 + (1-P_{T1})\in\right)\lambda_T^{**} + \mu_H\right)\right),$$

$$I_T^{**} = \frac{\lambda_T^{**}\Lambda_H\left(\in \lambda_T^{**} + \mu_H\right)\left(k_1 + \sigma_1\lambda_T^{**} + P_{T1}\mu_H\right)}{\left(\lambda_T^{**} + \mu_H\right)\left(d_{T1}\left(\in \lambda_T^{**} + \mu_H\right)\left(k_1 + \sigma_1\lambda_T^{**} + \mu_H\right) + \mu_H\left(k_1\left(r_1 + \in \lambda_T^{**} + \mu_H\right) + \right.\right.} \\ \left.\left.\left(\in \lambda_T^{**} + \mu_H\right)\left(\sigma_1\lambda_T^{**} + \mu_H\right) + r_1\left(\left(\sigma_1 + \in (1-P_{T1})\right)\lambda_T^{**} + \mu_H\right)\right)\right)$$

$$T_T^{**} = \frac{\gamma_1\lambda_T^{**}\Lambda_H\left(k_1 + \sigma_1\lambda_T^{**} + P_{T1}\mu_H\right)}{\left(\lambda_T^{**} + \mu_H\right)\left(d_{T1}\left(\in \lambda_T^{**}\mu_H\right)\left(k_1 + \sigma_1\lambda_T^{**} + \mu_H\right) + \mu_H\right)} \\ \left(k_1\left(r_1 + \in \lambda_T^{**} + \mu_H\right) + \left(\in \lambda_T^{**} + \mu_H\right)\left(\sigma_1\lambda_T^{**} + \mu_H\right) + r_1\left(\left(\sigma_1 + (1-P_{T1})\in\right)\lambda_T^{**} + \mu_H\right)\right).$$

Since $N_H^{**} = S_H^{**} + E_T^{**} + I_T^{**} + T_T^{**}$, we then have

$$N_H^{**} = \frac{\begin{array}{c}(1-P_{T1})\lambda_T^{**}\Lambda_H\left(d_{T1} + r_1 + \mu_H\right)\left(\in \lambda_T^{**} + \mu_H\right) + \lambda_T^{**}\Lambda_H\left(\in \lambda_T^{**} + \mu_H\right) \\ \left(k_1 + \sigma_1\lambda_T^{**} + \mu_H P_{T1}\right) + \gamma_1\lambda_T^{**}\Lambda_H\left(k_1 + \sigma_1\lambda_T^{**} + \mu_H P_{T1}\right) + \Lambda_H \\ \left[\left(\mu_H \in \lambda_T^{**} + {\mu_H}^2 + \in \lambda_T^{**} d_{T1} + r_1\mu_H + d_{T1}\mu_H\right)\right.\end{array}}{\left(\mu_H + \lambda_T^{**}\right)\left[\left(\mu_H \in \lambda_T^{**} + {\mu_H}^2 + \in \lambda_T^{**} d_{T1} + r_1\mu_H + \mu_H d_{T1}\right)\right.} \\ \left.\left(\sigma_1\lambda_T^{**} + \mu_H + k_1\right) + (1-P_{T1}) \in \lambda_T^{**} r_1\mu_H\right]$$

Now, since $\lambda_T^{**} = \frac{\beta_T I_T^{**}}{N_H^{**}}$ (at the endemic equilibrium point), substituting N_H^{**} and I_T^{**} into λ_T^{**} gives the following polynomial (in terms of λ_T^{**}):

$$A_1\lambda_T^{**3} + A_2\lambda_T^{**2} + A_3\lambda_T^{**} + A_4 = 0,$$

where

$$A_1 = \in \sigma_1 > 0,$$

$$A_2 = (1-P_{T1}) \in (d_{T1} + r_1 + \mu_H) + k_1 \in + P_{T1}\mu_H \in + \mu_H\sigma_1 + \sigma_1 r_1 + r_1\sigma_1\mu_H + \sigma_1 \in d_{T1} - \beta_T\sigma_1 \in,$$

$$A_3 = (1-P_{T1})\mu_H(d_{T1} + r_1 + \mu_H) + k_1\mu_H + P_{T1}{\mu_H}^2 + r_1k_1 + P_{T1}\mu_H r_1 + {\mu_H}^2\sigma_1 + \sigma_1 r_1\mu_H \\ + \sigma_1 d_{T1}\mu_H + {\mu_H}^2 \in + \in d_{T1}\mu_H + k_1\mu_H \in + k_1 \in d_{T1} + (1-P_{T1}) \in r_1\mu_H - \beta_T \in k_1 - \beta_T P_{T1}\mu_H \in - \sigma_1\mu_H\beta_T,$$

and

$$A_4 = \mu_H(d_{T1} + r_1 + \mu_H)(\mu_H + k_1)[1 - R_T].$$

Since $A_1 > 0$, the number of positive roots of the polynomial (which then determines the number of endemic equilibria of the model (2)) is determined by the signs of the coefficients A_2, A_3 and A_4. Hence, we claim the following.

Lemma 4 The number of endemic equilibria of model (2) is summarized as follows:

If $A_4 > 0 \Leftrightarrow R_T < 1$, then there are two endemic equilibria if A_2 and A_3 are of opposite signs or they are both negative

If $A_4 > 0 \Leftrightarrow R_T < 1$ and A_2 and A_3 are both positive, then there are no endemic equilibrium for this case.

If $A_4 < 0 \Leftrightarrow R_T > 1$, then a unique endemic equilibrium is possible.

Item (1) shows the possibility of a backward bifurcation in model (2), whereby there exist an endemic equilibrium coexisting with the DFE when $R_T < 1$ In this case, the reproduction number becomes only a necessary (but not sufficient) condition for disease eradication. In particular, if $\sigma_1 = \epsilon = 0$, we observe that the third degree polynomial reduces to a linear equation, and for this case, there is no endemic equilibrium when $R_T < 1$. However, there is a unique endemic equilibrium when $R_T > 1$.

3.1.3 Global Stability of the DFE

Considering the model (2) where there is no exogenous reinfection and there are no reinfection of treated individuals i.e., when $\sigma_1 = \epsilon = 0$. We claim the following

Theorem 1: *The DFE of the system (2), with $\sigma_1 = \epsilon = 0$, is globally asymptotically stable in D_1 whenever $R_T < 1$.*

Proof: Consider the following linear Lyapunov function:

$V = k_1E_T + (\mu_H + k_1)I_T$.

Clearly, $V>0$ except at the DFE. Differentiating V with respect to time, we have

$\dot{V} = k_1\dot{E}_T + (\mu_H + k_1)\dot{I}_T$

Substituting the expression for $\dot{E}$ and $\dot{I}_T$ into $\dot{V}$ yields

$\dot{V} = \frac{\beta_T I_T S_H}{N_H}[k_1(1-P_{T1}) + g_1 P_{T1}] - I_T[g_1 g_2]$

On D_1 , $S_H \le N_H \le \frac{\Lambda_H}{\mu_H}$. $Hence, \frac{S_H}{N_H} \le 1$, so that we now have

$\dot{V} \le I_T g_1 g_2 \left[\frac{(g_1 P_{T1} + k_1(1-P_{T1}))\beta_T}{g_1 g_2} - 1\right]$,

$\dot{V} \le g_1 g_2 I_T[R_T - 1]$, with the equality only at the DFE.

For $R_T \le 1$, we have that $\dot{V} \le 0$. Therefore, V is a Lyapunov function in D_1 and it follows from the LaSalle's Invariance Principle [13]that every solution to the equation in (2) with $\sigma_1 = \epsilon = 0$, and initial conditions in D_1, converges to ξ_1 as t→∞. This means that $(E_T(t), I_T(t), T_T(t)) \longrightarrow (0,0,0)$ as $t \longrightarrow \infty$. Substituting $E_T = I_T = T_T = 0$ into the system (2) gives$(t) S_H \to \frac{\Lambda_H}{\mu_H}$ $as\ t \to \infty$, so that $(S_H, E_T, I_T, T_T) = (\frac{\Lambda_H}{\mu_H}, 0, 0, 0)$ $as\ t \to \infty\ for\ R_T \le 1$. Hence, the DFE ξ_1 is GAS in D_I for $\sigma_1 = \epsilon = 0$.

The epidemiological significance of this is that in the absence of the exogenous reinfection and the case where there are no reinfection of TB treated individuals, tuberculosis can be eliminated from the population if $R_T < 1$, regardless of the initial conditions.

3.1.4 Global Stability of EEP of TB-only model.

Consider a special case of model (2) where $\epsilon = \sigma_1 = d_{T1} = 0$ i.e., there are no incidences of exogenous reinfection, there are no reinfection of treated individuals and no cases of disease-induced death. Let

$D_0 = \{(S_H, E_T, I_T, T_T) \in D_1 : E_T = I_T = T_T = 0\}$

be the stable manifold of the DFE (ξ_1). We claim the following.

Theorem 2.*The unique endemic equilibrium $\xi_{(1,T)}$ of the model (3.2), with $\epsilon = \sigma_1 = d_{T1} = 0$, is GAS in $D_1 \backslash D_0$ whenever $R_T > 1$.*

Proof: Consider the model (2) with $\epsilon = \sigma_1 = d_{T1} = 0$ and $R_T > 1$, so that the associated unique endemic equilibrium exists. Also, consider the following non-linear Lyapunov function (of the Goh-Volterra type) :

$F = S_H - S_H^{**} - S_H^{**} \ln \frac{S_H}{S_H^{**}} + \left(E_T - E_T^{**} - E_T^{**} \ln \frac{E_T}{E_T^{**}}\right) + \frac{\hat{\beta}_T S_H^{**}}{\mu_H + r_1}\left(I_T - I_T^{**} - I_T^{**} \ln \frac{I_T}{I_T^{**}}\right)$.

Taking the derivative of F yields

$\dot{F} = \dot{S}_H - \frac{S_H^{**}}{S_H}\dot{S}_H + \left(\dot{E}_T - \frac{E_T^{**}}{E_H}\dot{E}_T\right) + \frac{\hat{\beta}_T S_H^{**}}{\mu_H + r_1}\left(\dot{I}_T - \frac{I_T^{**}}{I_T}\dot{I}_T\right)$.

It should be noted that setting $d_{T1} = 0$ in (2) results in $N_H(t) \to \frac{\Lambda_H}{\mu_H}$ as $t \to \infty$. Let $N_H(t) = \frac{\Lambda_H}{\mu_H}$. Also, let $\hat{\beta}_T = \frac{\mu_H \beta_T}{\Lambda_H}$ so that $\lambda_T = \hat{\beta}_T I_T$. Substituting the expressions on the right hand side of model (2) into $\dot{F}$ gives

$\dot{F} = \Lambda_H - \lambda_T S_H - \mu_H S_H - \frac{S_H^{**}}{S_H}\Lambda_H + \lambda_T S_H^{**} + \mu_H S_H^{**} + \lambda_T S_H$

$-(\mu_H + k_1)E_T - \frac{\hat{\beta}_T I_T S_H E_T^{**}}{E_T} + (\mu_H + k_1)E_T^{**} + \frac{\hat{\beta}_T S_H^{**}}{\mu_H + r_1}k_1 E_T - \frac{\hat{\beta}_T S_H^{**}}{\mu_H + r_1}(\mu_H + r_1)I_T$

$-\frac{\hat{\beta}_T S_H^{**}}{\mu_H + r_1}\frac{k_1 E_T I_T^{**}}{I_T} + \frac{\hat{\beta}_T S_H^{**}}{\mu_H + r_1}(\mu_H + r_1)I_T^{**}$.

It can be shown from (2) that at steady state,

$\Lambda_H = \mu_H S_H^{**} + \hat{\beta}_T I_T^{**} S_H^{**}$,

$\mu_H + k_1 = \frac{\hat{\beta}_T I_T^{**} S_H^{**}}{E_T^{**}}, \mu_H + r_1 = \frac{K_1 E_T^{**}}{I_T^{**}}$.

Using the above relations, and after several algebraic calculations, we have that

$\dot{F} = \mu_H S_H^{**}\left(2 - \frac{S_H}{S_H^{**}} - \frac{S_H^{**}}{S_H}\right) + \hat{\beta}_T S_H^{**} I_T^{**}\left(3 - \frac{S_H E_T^{**} I_T}{E_T S_H^{**} I_T^{**}} - \frac{E_T I_T^{**}}{E_T^{**} I_T}\right)$

Since the arithmetic mean exceeds the geometric mean, the following inequalities hold:

$\left(2 - \frac{S_H}{S_H^{**}} - \frac{S_H^{**}}{S_H}\right) \le 0, \left(3 - \frac{S_H E_T^{**} I_T}{E_T S_H^{**} I_T^{**}} - \frac{E_T I_T^{**}}{E_T^{**} I_T}\right) \le 0$.

Thus, we have that $\dot{F} \le 0\ for\ R_T > 1$. Since the relevant variables in the equations for S_H, E_T, I_T and T_T are at the endemic steady state, it follows that these can be substituted into the equations for S_H, E_T, I_T and T_T so that

$(S(t), E_T(t), I_T(t), T_T(t)) \to (S_H^{**}, E_T^{**}, I_T^{**}, T_T^{**})$ $as\ t \to \infty$.

Hence, F is a Lyapunov function in $D_1 \backslash D_0$

3.2 Dengue-only model

The dengue only model is derived from system (1) by setting $E_T = I_T = T_T = E_2 = E_3 = E_4 = I_2 = 0$. This leads to the following sub-model:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \mu_H S_H - \lambda_{DV} S_H, \\
\frac{dE_1}{dt} &= \lambda_{DV} S_H - (\gamma_1 + \mu_H)E_1, \\
\frac{dI_1}{dt} &= \gamma_1 E_1 - (\tau_1 + \mu_H + \delta_{D1})I_1, \\
\frac{dR_1}{dt} &= \tau_1 I_1 - \mu_H R_1, \\
\frac{dSv}{dt} &= \Lambda_V - \lambda_{DH} S_V - \mu_V S_V, \\
\frac{dE_V}{dt} &= \lambda_{DH} S_V - (\gamma_V + \mu_V)E_V, \\
\frac{dI_V}{dt} &= \gamma_V E_V - (\mu_V + \delta_{HV})I_V,
\end{aligned} \qquad (3)$$

with

$\lambda_{DV} = \frac{\beta_{VH}(\eta_V E_V + I_V)}{N_H}$, $\lambda_{DH} = \frac{\beta_{HV}(\eta_A E_1 + \eta_B I_1 + \eta_C E_2 + \eta_D E_3 + \eta_E E_4 + \eta_F I_2)}{N_H}$, $N_H = S_H + E_1 + I_1 + R_1$ and $N_V = S_V + E_V + I_V$.

Consider the region $D_2 = \left\{(S_H, E_1, I_1, R_1, S_V, E_V, I_V) \epsilon \mathbb{R}_+^7 : N_H \leq \frac{\Lambda_H}{\mu_H}, N_V \leq \frac{\Lambda_V}{\mu_V}\right\}$. Using the approaches used in Section 3.1, it can be shown that the set D_2 is positively invariant and an attractor of all positive solution of the system (3). Hence, we claim the following

Lemma 5.*The region D_2 is positively invariant for the system (3).*

Lemma 6. *Let the initial data for the model (3) be $S_H(t) > 0$, $E_1(t) > 0, I_1(t) > 0, R_1(t) > 0, S_V(t) > 0, E_V(t)$ and $I_V(t) > 0$ then the solution $S_H(t)$, $E_1(t)$,$I_1(t)$, $R_1(t), S_V(t), E_V(t)$, and $I_V(t)$with positive initial data will remain positive for all time $t > 0$.*

3.2.1 Local Stability of Disease-Free Equilibrium (DFE) of the Dengue-only Model

The model (3) has a disease-free equilibrium, obtained by setting the right hand side of the model to zero, given by

$\xi_2 = \left(S_H^*, E_1^*, I_1^*, R_1^*, S_V^*, E_V^*, I_V^*\right) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0\right)$

The stability of ξ_2 is established using the next generation operator method on the system (3) [12]. Following the procedure, as implemented in Section 3.1.1, we have that the effective reproduction number of the model (3) is given by

$R_D = \sqrt{\frac{\Lambda_V \beta_{HV} \beta_{VH} \mu_H (g_4 \eta_A + \gamma_1 \eta_B)(\gamma_V + g_6 \eta_V)}{\Lambda_H g_3 g_4 g_5 g_6 \mu_V}}$,

where, $g_3 = \mu_H + \gamma_1, g_4 = \tau_1 + \mu_H + \delta_{D1}, g_5 = \gamma_V + \mu_V, g_6 = \mu_V + \delta_{HV}$. The next result follows from Theorem 2 in [12].

Lemma 7. *The DFE of the system (3) is locally asymptotically stable if $R_D < 1$ and unstable if $R_D > 1$.*

The threshold quantity R_D is the effective or control reproduction number for the Dengue only sub-model. The implication of Lemma 7 is that Dengue can be eliminated from the population when $R_D < 1$ if the initial sizes of the subpopulations of the sub-model are in the region of attraction of ξ_2.

3.3.2. Existence of Endemic Equilibrium Point (EEP) of Dengue-only model

Let the EEP of model (3) be denoted by $\xi_{(1,D)} = (S_H^{**}, E_1^{**}, I_1^{**}, R_1^{**}, S_V^{**}, E_V^{**}, I_V^{**})$. The equations in (3) are solved in terms of the force of infection at steady state and they are given as

$S_H^{**} = \frac{\Lambda_H}{\mu_H + \lambda_{DV}^{**}}$,

$E_1^{**} = \frac{\Lambda_H \lambda_{DV}^{**}}{(\mu_H + \lambda_{DV}^{**})(\gamma_1 + \mu_H)}$,

$I_1^{**} = \frac{\gamma_1 \lambda_{DV}^{**} \Lambda_H}{(\mu_H + \lambda_{DV}^{**})(\gamma_1 + \mu_H)(\tau_1 + \mu_H + \delta_{D1})}$,

$R_1^{**} = \frac{\tau_1 \gamma_1 \lambda_{DV}^{**} \Lambda_H}{(\mu_H + \lambda_{DV}^{**})(\gamma_1 + \mu_H)(\tau_1 + \mu_H + \delta_{D1})\mu_H}$,

$S_V^{**} = \frac{\Lambda_V}{(\mu_V + \lambda_{DH}^{**})}$,

$E_V^{**} = \frac{\lambda_{DH}^{**} \Lambda_V}{(\mu_V + \lambda_{DH}^{**})(\gamma_V + \mu_V)}$,

$I_V^{**} = \frac{\gamma_V \lambda_{DH}^{**} \Lambda_V}{(\mu_V + \lambda_{DH}^{**})(\gamma_V + \mu_V)(\mu_V + \delta_{HV})}$,

$N_H^{**} = \frac{\Lambda_H \mu_H (\gamma_1 + \mu_H)(\tau_1 + \mu_H + \delta_{D1}) + \lambda_{DV}^{**} \Lambda_H (\tau_1 + \mu_H + \delta_{D1})\mu_H + \gamma_1 \lambda_{DV}^{**} \Lambda_H \mu_H + \tau_1 \gamma_1 \lambda_{DV}^{**} \Lambda_H}{\mu_H (\mu_H + \lambda_{DV}^{**})(\gamma_1 + \mu_H)(\tau_1 + \mu_H + \gamma + \delta_{D1})}$.

Now,

$\lambda_{DH}^{**} = \frac{\beta_{HV}(\eta_A E_1^{**} + \eta_B I_1^{**})}{N_H^{**}}$ and $\lambda_{DV}^{**} = \frac{\beta_{VH}(\eta_V E_V^{**} + I_V^{**})}{N_H^{**}}$ (at steady state). Substituting the expressions for E_1^{**}, I_1^{**}, N_H^{**} and E_V^{**}, I_V^{**}, N_H^{**} into λ_{DH}^{**} and λ_{DV}^{**} leads to

$\lambda_{DH}^{**} = \frac{\beta_{HV} \mu_H (g_4 \eta_A + \gamma_1 \eta_B) \lambda_{DV}^{**}}{\mu_H g_3 g_4 + (\mu_H g_4 + \gamma_1 \mu_H + \tau_1 \gamma_1) \lambda_{DV}^{**}}$

and

$\lambda_{DV}^{**} = \frac{\beta_{VH} \Lambda_V \mu_H (\eta_V g_6 + \gamma_V) g_3 g_4 (\mu_H + \lambda_{DV}^{**}) \lambda_{DH}^{**}}{\Lambda_H \mu_H g_3 g_4 g_5 g_6 \mu_V + (\Lambda_H \mu_H g_4 g_5 g_6 \mu_H + (\Lambda_H \gamma_1 \mu_H + \tau_1 \gamma_1 \Lambda_H) g_5 g_6 \mu_V) \lambda_{DV}^{**} + (\Lambda_H \mu_H g_3 g_4 g_5 g_6 + (\Lambda_H \mu_H g_4 g_5 g_6 + (\Lambda_H \gamma_1 \mu_H + \tau_1 \gamma_1 \Lambda_H) g_5 g_6) \lambda_{DV}^{**}) \lambda_{DH}^{**}}$.

Substituting the expression for λ_{DH}^{**} into λ_{DV}^{**}, we now have the following polynomial in λ_{DV}^{**}:

$A_1 \lambda_{Dv}^{**2} + A_2 \lambda_{Dv}^{**} + A_3 = 0$,

where

$A_1 = (\mu_H g_4 + \gamma_1 \mu_H + \tau_1 \gamma_1) \Lambda_H \mu_H^2 g_4 g_5 g_6 + g_5 g_6 \mu_v (\mu_H g_4 + \gamma_1 \mu_H + \tau_1 \gamma_1)(\Lambda_H \gamma_1 \mu_H + \tau_1 \gamma_1 \Lambda_H) + \beta_{Hv} \mu_H^2 (g_4 \eta_A + \gamma_1 \eta_B) \Lambda_H g_4 g_5 g_6 + \beta_{Hv} \mu_H (g_4 \eta_A + \gamma_1 \eta_B) g_5 g_6 (\Lambda_H \gamma_1 \mu_H + \tau_1 \gamma_1 \Lambda_H) > 0$

$A_2 = (\mu_H g_4 + \gamma_1 \mu_H + \tau_1 \gamma_1) \Lambda_H g_3 g_4 g_5 g_6 \mu_v + \Lambda_H \mu_H^3 g_3 g_4^2 g_5 g_6 + \mu_H g_3 g_4 g_5 g_6 \mu_v (\Lambda_H \gamma_1 \mu_H + \tau_1 \gamma_1 \Lambda_H) + \beta_{Hv} \mu_H^2 (g_4 \eta_A + \gamma_1 \eta_B) \Lambda_H g_3 g_4 g_5 g_6 - \beta_{Hv} \beta_{vH} \Lambda_H \mu_H^2 g_3 g_4 (\eta_v g_6 + \gamma_v)(g_4 \eta_A + \gamma_1 \eta_B)$,

$A_3 = \Lambda_H \mu_H g_3^2 g_4^2 g_5 g_6 \mu_v \left[1 - (R_D)^2\right]$

where $g_3 = \mu_H + \gamma_1, g_4 = \tau_1 + \mu_H + \delta_{D1}, g_5 = \gamma_1 + \mu_v$, and $g_6 = \mu_v + \delta_{Hv}$.

As we can see, $A_1 > 0$. Hence, the number of positive roots of the polynomial will depend on the signs of A_2 and A_3. Therefore, we claim the following.

Theorem 3The number of endemic equilibria of model (2) is summarized as follows:

1. If $A_3 > 0 \Leftrightarrow R_D < 1$, then there are two endemic equilibria if and only if $A_2 < 0$.
2. If $A_3 > 0 \Leftrightarrow R_D < 1$ and $A_2 > 0$, then there are no endemic equilibria in this case
3. If $A_3 < 0 \Leftrightarrow R_D > 1$, then regardless of the sign of A_2, the model (3) will have a unique endemic equilibrium.

4.0 Analysis of complete model (1)

Consider the region

$D = \{(S_H, E_T, I_T, T_T, E_1, I_1, R_1 S_V, E_V, I_V, E_2, E_3, E_4, I_2) \epsilon \mathbb{R}_+^{14} : N_H \leq \frac{\Lambda_H}{\mu_H}, N_V \leq \frac{\Lambda_V}{\mu_V}\}$.

Using the approaches in Section 3.1, it can be shown that the set D is positively invariant and an attractor of all positive solution of the system (1). Hence, we claim the following.

Lemma 8. *The region D is positively invariant for the system (1).*

Lemma 9. *Let the initial data for the model (1) be* $S_H(t) > 0, E_T(t) > 0, I_T(t) > 0, T_T(t) > 0, E_1(t) > 0, I_1(t) > 0, R_1(t) > 0, S_V(t) > 0, E_V(t) > 0, I_V(t) > 0, E_2(t) > 0, E_3(t) > 0, E_4(t) > 0, and\ I_2(t) > 0$ *then the solution* $S_H(t), E_T(t), I_T(t), T_T(t), E_1(t), I_1(t), R_1(t), S_V(t), E_V(t), I_V(t), E_2(t), E_3(t), E_4(t), and\ I_2(t)$ *with positive initial data will remain positive for all time t > 0.*

4.1 Local Stability of disease-free equilibrium (DFE) of TB-Dengue Model

The model (1) has a disease-free equilibrium obtained by setting the right hand side of the model to zero given by

$\xi_3 = (S_H^*, E_T^*, I_T^*, T_T^*, E_1^*, I_1^*, R_1^*, S_V^*, E_V^*, I_V^*, E_2^*, E_3^*, E_4^*, I_2^*) = (\frac{\Lambda_H}{\mu_H}, 0,0,0,0,0,0, \frac{\Lambda_V}{\mu_V}, 0,0,0,0,0,0)$.

The linear stability of ξ_3 is established using the next generation operator method on the system (1) [12]. Following a similar procedure in Section 3.1.1, the effective reproduction number of the TB-Dengue model (1) is obtained as $R_C = \max\{R_T, R_D\}$, where $R_T = \frac{\beta_T (g_1 P_{T1} + k_1(1 - P_{T1}))}{g_1 g_2}$,

$R_D = \sqrt{\frac{\Lambda_v \beta_{Hv} \beta_{vH} \mu_H (g_4 \eta_A + \gamma_1 \eta_B)(\gamma_v + g_6 \eta_v)}{\Lambda_H g_3 g_4 g_5 g_6 \mu_v}}$, and

$g_1 = \mu_H + k_1, g_2 = \mu_H + d_{T1} + r_1, g_3 = \gamma_1 + \mu_H . g_4 = \tau_1 + \mu_H + \delta_{D1}, g_5 = \gamma_V + \mu_V, g_6 = \mu_V + \delta_{HV}$,

$g_7 = \gamma_2 + k_2 + \mu_H, g_8 = k_3 + \tau_2 + \delta_{D2} + \mu_H, g_9 = d_{T2} + r_2 + \gamma_3 + \mu_H, g_{10} = \tau_3 + r_3 + \delta_{D3} + d_{T3} + \mu_H$,

$N_H^* = \frac{\Lambda_H}{\mu_H}$, and $S_V^* = \frac{\Lambda_V}{\mu_V}$.

The control reproduction number, associated with the *DFE* (ξ_3) of the model (1), denoted by R_C. The following result follows from Theorem 2 in [12].

Lemma 10. *The DFE,* ξ_3 *of the model (1) is locally asymptotically stable (LAS) if* $R_C < 1$ *and unstable if* $R_C > 1$.

5.0 Numerical Simulations

Model (1) is now simulated, using the parameter estimates in Table 2 to gain insight into some of its quantitative features.

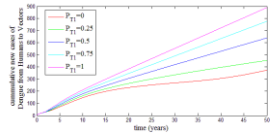

Figure 3: Numerical simulation of model (1) showing cumulative new cases of dengue from human to vectors when we vary $\boldsymbol{P_{T1}}$.

Figure 3 shows the cummulative new cases of dengue from human to vectors, when we vary the fraction of newly infectedhumans with active TB (fast progression)P_{T1}. We observe that the cummulative new cases from human to vectors is increasing significantly as the fraction P_{T1} increases.

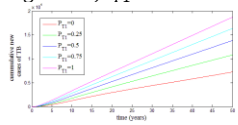

Figure 4: Numerical simulation of model (1) showing the cumulative new TB cases, with varied values of P_{T1}.

Figure 4 shows that the cummulative number of new TB cases increases as the fraction P_{T1} increase, as expected.

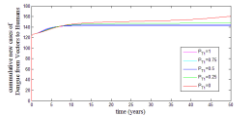

Figure 5: Numerical simulation of model (1) showing the cumulative new cases of dengue from vectors to human while varying P_{T1}.

In Figure 5, we observe that the cummulative number of new cases of dengue from vectors to humans was marginal as we vary P_{T1} compared to when we had the cases where the dengue was spread from humans to vectors.

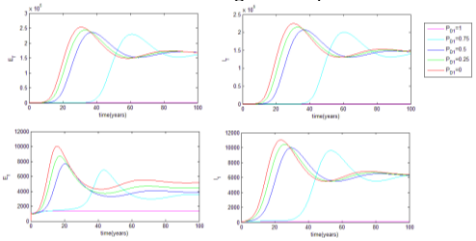

Figure 6: Numerical simulation of (1) depicting the$\boldsymbol{E_T}$,$\boldsymbol{I_T}$,$\boldsymbol{E_1}$ and $\boldsymbol{I_1}$classes while varying $\boldsymbol{P_{D1}}$.

Figure 6 is showing the number of individuals in the E_T,I_T, E_1 and I_1 classes as we varyP_{D1}., the fraction of individuals with dengue and in the latent stage of TB. Observe that there is a marginal change in the number of infected individuals in all four classes as P_{D1} increases, in the long run.

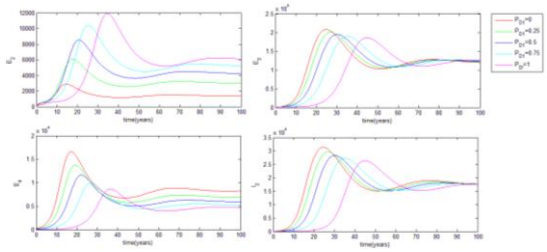

Figure 7: Numerical simulation of model (1) showing the dually infected classes $\mathbf{E_2}$,$\mathbf{I_3}$, $\mathbf{E_4}$ and $\mathbf{I_2}$while varying $\mathbf{P_{D1}}$.

Figure 7 is showing the number of individuals in the dually infected classes E_2, E_3, E_4 and I_2 classes while varyingP_{D1}. Observe that that there is no significant change in the number of individuals in the $E_3$and I_2 classes, in the long run, compared to persons in the $E_2$and E_4 classes, as we increase the value ofP_{D1}.

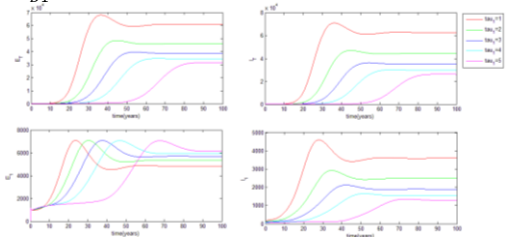

Figure 8: Numerical simulation of (1) depicting the number of individuals in the $\boldsymbol{E_T}$,$\boldsymbol{I_T}$, $\boldsymbol{E_1}$ and $\boldsymbol{I_1}$classes while varying $\boldsymbol{\tau_1}$.

In figure 8, we observe that the number of infected individuals in the E_T,I_T, E_1 and I_1 classes was decreasing with increase in the treatment rate for dengue(τ_1), except in theE_1 class (as this could be to the fact that there are now more susceptibles available for reinfection). Figure 9 is depicting the number of individuals in the singly infected classesE_T,I_T, E_1 and I_1 classes as we vary r_1, the treatment rate for TB. As expected, increases in r_1 results in a corresponding decrease in the number of infected individuals in the classes shown therein.

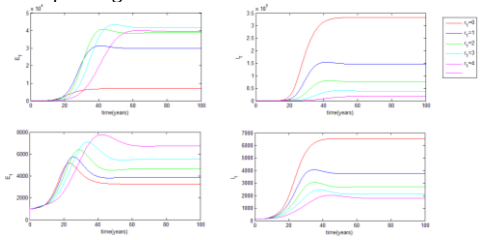

Figure 9: Numerical simulation of model (1) showing the number of infected individuals in the $\boldsymbol{E_T}$,$\boldsymbol{I_T}$, $\boldsymbol{E_1}$ and $\boldsymbol{I_1}$ classes while varying $\boldsymbol{r_1}$.

6.0 Discussions

In this work, a mathematical model for the population dynamics of TB and Dengue coinfection (where treatment is available for both diseases) is proposed and analyzed. The mathematical analysis herein shows that the disease free equilibrium (DFE) of the TB-only model is globally asymptotically stable when there are insignificant levels of exogenous reinfection and reinfection of treated individuals. Also, it was shown that the endemic equilibrium point (EEP) of the TB-only model also seen to be globally asymptotically stable when $R_T > 1$ and when there are insignificant levels of exogenous reinfection, reinfection of treated individuals and disease-induced death in humans. Furthermore, the analyses showed that the dengue-only model has a unique endemic equilibrium point whenever the associated reproduction number is greater than unity.

Numerical simulations of the model show that effective treatment for either Tb or dengue will result in a reduction in the disease burden of either diseases or its coinfection. Also, the simulations reveal that the fraction of fast progressions to active TB can impact on the number of new cases of dengue (on both the vectors and humans alike).

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