



The Co-Dynamics of Malaria and Tuberculosis with Optimal Control Strategies

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Abstract. Malaria and Tuberculosis are both the severe and causing death diseases in the world. The occurrence of TB and malaria as a coinfection is also an alarming threat to the human. Therefore, we consider a mathematical model of the dynamics of malaria and tuberculosis coinfection and explore its theoretical results. We formulate the model and obtain their basic properties. We show that at the disease free case each model is locally asymptotically stable, when the basic reproduction number less than unity. Further, we analyze the phenomenon of backward bifurcation for coinfection model. For the sub models, we present the local stability for the disease free case whenever the basic reproduction number less than 1. Further, an optimal control problem is presented to investigate the dynamics of malaria and tuberculosis coinfection. The numerical results with different scenarios are presented. The mathematical model with and without control problem are solved numerically using the Runge-Kutta backward and forward scheme of order four.

1. Introduction

Infectious diseases are modeled effectively through mathematics are considerably effective to address their disease mechanism well. Mathematical models can best describes the disease status, whether, it can be controlled or not. The micro-organisms, pathogens such as viruses, bacteria, fungi or parasites are the causes of infectious diseases. It may cause by a direct mode or an indirect mode or person to person, with different route transmission. Malaria and tuberculosis (TB) are considered the major public health problems throughout the world. An estimate shows the approximately 1200 children die each day due to malaria while 3800 children die due to TB each day, throughout the world [14, 15]. The recent advancement in medical sciences, these diseases are still an alarming for the public health. The most prevalent bacterial disease in the humans is the TB which is caused by the Mycobacterium tuberculosis. A report shows that this disease places the second position amongst the common infectious diseases in the world [22]. Mostly the TB, infects the lungs. Besides this, it can affects the circulatory, urinary and central nervous system, and also the skin, bones and joints. The transmission of this disease among human is occurred by droplets, those having infections in throat and lungs of active respiratory disease [15]. A people with active TB,

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the transmission takes place. The symptoms include, chest pains, coughing, weight loss, weakness and fever etc. [15]. This disease is treated with the available antibiotics, a course of six to nine months. The TB can be minimized by the isolation of patients who have the TB disease and an immediate start of the anti-tuberculosis therapy. A source shows that globally, the new cases of TB decreases whereas the cases increases in Sub-Saharan Africa [15].

Malaria is one of the vector borne disease that caused by the bites of mosquito. The plasmodium which is a parasite is the source of causing malaria. The parasites increases in the human liver and results to infect the red blood cells [14]. Vomiting, headache, and fever are the common symptoms of malaria. Delay in treatment can cause severe life threatening, by disrupting the blood supply to the vital organs [14]. In human population the exposure of pathogens is rare and in Africa, the infections with more than one shows a wide public health threat [9]. Malaria and TB, HIV-AIDS, are considered the three major global health problem, which causes, mortality, morbidity, human suffering and negative socio-economic impact [22]. The above mentioned diseases are endemic in populations. Literature show on the dynamics of malaria TB and related topics have been considered and discussed [8, 16, 19, 24, 25]. For example, in [16], an author investigates the presence of malaria infection with TB person. In [18], the author investigated and obtained the results. He observed that in adults the malaria increase significantly the incidence of reactivating latent TB. Mathematical modeling of coinfection is the interning research area now-a-days to the researchers and biologists. For example, in [20], a mathematical model on the dynamics of HIV and TB is formulated and discussed. In [10], HIV-malaria coinfection model is formulated and the results are investigated and discussed. In [13], the HIV-AIDS and cryptosporidiosis coinfection model is formulated with different control strategies.

Motivated by the above studies, we formulate a mathematical model on TB and malaria coinfection. The literature on each disease and on their coinfection has been discussed in detailed above. The rest of the work present in this paper is categorized is as follows: Model formulation of TB and malaria and their codynamics is presented in section 2. The only TB model is formulated in section 3 while in section 4 the dynamics of the only malaria model is presented. The TB malaria coinfection model is discussed in section 5. In section 6, the optimal control model of the coinfection is formulated and presented the results associated. Numerical results with brief discussion is presented in section 7 and in section 8, we finalized the work by conclusion.

2. Model Formulation of TB and Malaria Coinfection

The total human population, $N(t)$, subdivided into, susceptible individuals $S(t)$, individuals exposed to malaria only $E_m(t)$, individuals infected with malaria only $I_m(t)$, individuals recovered from TB and malaria both are R , individuals exposed to TB only $E_{tb}(t)$, individuals infected from TB only $I_{tb}(t)$, individuals infected from TB only treated T_{tb} and the individuals dually infected from TB and Malaria I_{mt} . So, the total population of humans individuals is,

$$N = S + E_m + I_m + R + E_{tb} + I_{tb} + T_{tb} + I_{mt}.$$

The population of vector $N_v(t)$ is subdivided into three mutually exclusive classes, $S_v(t)$ -susceptible, E_v -exposed and $I_v(t)$ -infected vector. So,

$$N_v = S_v + E_v + I_v.$$

The above discussions leads to the following system of nonlinear differential equations:

$$\left\{ \begin{aligned} \frac{d}{dt}S &= \Lambda - \lambda_{tb}S - \lambda_mS - dS, \\ \frac{d}{dt}E_m &= \lambda_mS - (\tau_m + d)E_m, \\ \frac{d}{dt}I_m &= \tau_mE_m - (d + d_m + \gamma_m)I_m - \lambda_{tb}I_m, \\ \frac{d}{dt}R &= \gamma_mI_m + \alpha_{tb}T_{tb} + \psi_{mt}I_{mt} - dR, \\ \frac{d}{dt}S_v &= \Lambda_v - \lambda_vS_v - d_vS_v, \\ \frac{d}{dt}E_v &= \lambda_vS_v - (d_v + \tau_v)E_v, \\ \frac{d}{dt}I_v &= \tau_vE_v - d_vI_v, \\ \frac{d}{dt}E_{tb} &= \lambda_{tb}S - (d + \epsilon_{tb})E_{tb} + (1 - \eta_{tb})\delta_{tb}T_{tb}, \\ \frac{d}{dt}I_{tb} &= \epsilon_{tb}E + \eta_{tb}\delta_{tb}T_{tb} - (d + \gamma_{tb} + \sigma_{tb})I_{tb} - \lambda_mI_{tb}, \\ \frac{d}{dt}T_{tb} &= \gamma_{tb}I_{tb} - (d + \delta_{tb} + \sigma_{tbt} + \alpha_{tb})T_{tb}, \\ \frac{d}{dt}I_{mt} &= \lambda_{tb}I_m + \lambda_mI_{tb} - (\epsilon + d + \psi_{mt})I_{mt}, \end{aligned} \right. \tag{1}$$

where $\lambda_{tb} = \frac{\beta_{tb}\sigma_{tb}I_{tb}}{N_{tb}}$, $\lambda_m = \frac{\beta_m\sigma_mI_v}{N}$ and $\lambda_v = \frac{\beta_v\sigma_vI_m}{N}$. The population of susceptible individuals is recruited by the rate Λ . The natural death rate of human and vector is respectively shown by d and d_v . The humans individuals exposed to malaria are infected at a rate of τ_m , d_m is the death rate of infected individuals due to malaria and the rate of recovery from malaria is shown by γ_m . The population of vector is recruited by Λ_v . The exposed vector becomes infected at a rate of τ_v . The parameters β_m shows the contact with malaria in humans while β_v with contacts of malaria in mosquitoes. The force of infections λ_m and λ_v respectively represent human contact with infected mosquito and mosquito contact with infected individuals. The per rate biting of mosquitos (females) is given by σ_m . The rate of progression of infected individuals due to TB to the infected class is shown by ϵ_{tb} , the death rate due to TB is given by σ_{tbt} . At a rate of γ_{tb} the infected individuals are treated. The individuals due to TB are recovered at a rate α_{tb} . The treated individuals enter to either latent class due to the remainder of Mycobacterium tuberculosis or infective class I_{tb} due to the failure of treatment at the rate δ_{tb} . The parameter η_{tb} measures the treatment failure. The parameter λ_{tb} represents force of infection with active TB individuals while the infection is transmitted at a rate σ_{tb} that shows its probability. The dually infected individuals dies from the coinfection is given by ϵ . The parameter β_{tb} shows the contact rate for TB while the recovery rate is ψ_{mt} for dual infected people. In Table 1, we shown the variables and the definitions of the parameters.

Solution positivity

Lemma 2.1. Consider the data initial be $\{(S, S_v)(0) > 0, (E_m, I_m, R, E_v, I_v, E_{tb}, I_{tb}, T_{tb}, I_{mt})(0) \geq 0\} \in \Pi$. Then, the solution set $\{(S, E_m, I_m, R, S_v, E_v, I_v, E_{tb}, I_{tb}, T_{tb}, I_{mt})(t)\}$ of the coinfection model (1) will remain positive for every $t > 0$.

Proof. It follows from the first equation of the coinfection model (1), we have

$$\begin{aligned} \frac{d}{dt}S &= \Lambda - \lambda_{tb}S - \lambda_mS - dS, \\ &\geq -(\lambda_{tb} + \lambda_m + d)S \end{aligned} \tag{2}$$

Taking integration of the equation (2) with respect to t , we obtain

$$S(t) \geq S(0)e^{-\int (\lambda_{tb} + \lambda_m + d)dt} \geq 0, \text{ as } \lambda_{tb} + \lambda_m + d > 0.$$

for the initial data, $S(0) > 0$, then we have $S(t) > 0$, for the rest of the variables of the coinfection model (1), are positive for all the initial data positive remains positive for all time $t > 0$. \square

Variable	Description
S	Population of susceptible individuals
E_m	Individuals exposed to malaria
I_m	Infection of people with malaria only
R	Recovery from malaria, TB and its dual infection
S_v	Susceptible vector
E_v	Exposed vector
I_v	Infected vector
E_{tb}	Individuals exposed to TB only
I_{tb}	Individuals infected from TB only
T_{tb}	Treatment of infected Individuals with TB only
I_{mt}	Individuals infected with both TB and malaria
Parameter	Description
Λ	Recruited rate of susceptible individuals
d, d_v	Natural death rate of human and vector
τ_m	Humans individuals exposed to malaria infection rate
$d_m, \sigma_{tbt}, \epsilon$	Disease death rate of humans due to malaria, due to TB, dual infection
$\gamma_m, \alpha_{tb}, \psi_{mt}$	Rate of recovery from malaria infected only, TB infected only and dual infection
Λ_v	Recruitment rate of vector population
τ_v	Rate of flow from exposed vector to infected vector
ϵ_{tb}	Rate of flow from exposed TB to infected TB
$\beta_{tb}, \beta_m, \beta_v$	Contacts rate
γ_{tb}	Treatment rate of TB infected individuals
δ_{tb}, η_{tb}	Rate of flow of TB, treatment failure
$\lambda_{tb}, \lambda_m, \lambda_v$	Force of infection
σ_m, σ_{tb}	Modification parameters for

Table 1: Definitions of the model variables and parameters.

2.1. Invariant regions

It is obvious that the coinfection model (1) consists of humans and vector populations so all the variables are positive and non-negative for every time $t > 0$.

Lemma 2.2. *The region $\Omega = \Omega_h \times \Omega_v$, contains the solutions of the coinfection model (1).*

Proof. First, we show that all the feasible solutions are uniformly bounded in the set Ω . The coinfection model (1) has two parts, the human population N and the vector population N_v . Consider $\{(S, E_m, I_m, R, E_{tb}, I_{tb}, T_{tb}, I_{mt}) \in R_+^8\}$ be any solution of the coinfection model with nonnegative initial condition, then, $N' < \Lambda - dN$, and we have the solution $0 \leq N \leq \frac{\Lambda}{d}$ when $t \rightarrow \infty$. So, it can be seen that all the feasible solutions of the coinfection model (1) remains in the region

$$\Omega_h = \{(S, E_m, I_m, R, E_{tb}, I_{tb}, T_{tb}, I_{mt}) : N \leq \frac{\Lambda}{d}\}. \tag{3}$$

Similar result can be show for vector population,

$$\Omega_v = \{(S_v, E_v, I_v) : N_v \leq \frac{\Lambda_v}{d_v}\}. \tag{4}$$

Thus, it follows from equations (3) and (4), that all the feasible solutions of the coinfection model (1) will remains in

$$\Omega = \Omega_h \times \Omega_v. \tag{5}$$

Thus, Ω is the feasible region for the coinfection model (1), and is positively invariant, bounded and the existence and uniqueness and the continuations results hold. Further, it is well-posed epidemiologically and mathematically and is sufficient to study the dynamics of the coinfection model (1) under the region Ω . \square

Next, we study each submodel in detail.

3. Only TB model

The aims of this section is to investigate the dynamics of the Only TB model. The only TB model given by (6), can be obtained easily by setting $E_m = I_m = S_v = E_v = I_v = I_{mt} = 0$. So, we have

$$\begin{aligned} \frac{d}{dt}S &= \Lambda - \lambda_{tb}S - dS, \\ \frac{d}{dt}E_{tb} &= \lambda_{tb}S - (d + \epsilon_{tb})E_{tb} + (1 - \eta_{tb})\delta_{tb}T_{tb}, \\ \frac{d}{dt}I_{tb} &= \epsilon_{tb}E_{tb} + \eta_{tb}\delta_{tb}T_{tb} - (d + \gamma_{tb} + \sigma_{tb})I_{tb}, \\ \frac{d}{dt}T_{tb} &= \gamma_{tb}I_{tb} - (d + \delta_{tb} + \sigma_{tb} + \alpha_{tb})T_{tb}, \\ \frac{d}{dt}R &= \alpha_{tb}T_{tb} - dR, \end{aligned} \tag{6}$$

where $\lambda_{tb} = \frac{\beta_{tb}I_{tb}}{N}$, and $N = S + E_{tb} + I_{tb} + T_{tb} + R$. The biological feasible region for the only TB model is $\Omega_T = \{(S, E_{tb}, I_{tb}, T_{tb}, R) \in \mathbb{R}_+^5 : (S, E_{tb}, I_{tb}, T_{tb}, R) \leq \frac{\Lambda}{d}\}$ which is positively invariant and is sufficient to consider the dynamics of the only Tb model (6) in the region Ω_T .

3.1. Local stability analysis disease free equilibrium (DFE) of the only TB model

The DFE of the only TB model is

$$P_{tb}^0 = (S, E_{tb}, I_{tb}, T_{tb}, R) = \left(\frac{\Lambda}{d}, 0, 0, 0, 0\right),$$

and their basic reproduction number \mathcal{R}_0^{TB} is obtained by using the method [21].

$$F = \begin{pmatrix} 0 & \beta_{tb}\sigma_{tb} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} d + \epsilon_{tb} & 0 & -\delta_{tb}(1 - \eta_{tb}) \\ -\epsilon_{tb} & d + \gamma_{tb} + \sigma_{tb} & -\delta_{tb}\eta_{tb} \\ 0 & -\gamma_{tb} & d + \alpha_{tb} + \delta_{tb} + \sigma_{tb} \end{pmatrix}$$

and the required basic reproduction number for only TB model is

$$\begin{aligned} \mathcal{R}_0^{TB} &= \frac{\beta_{tb}\sigma_{tb}\epsilon_{tb}}{(d + \epsilon_{tb})(d + \gamma_{tb} + \sigma_{tb})} + \frac{\gamma_{tb}\delta_{tb}\eta_{tb}}{(d + \gamma_{tb} + \sigma_{tb})(d + \alpha_{tb} + \delta_{tb} + \sigma_{tb})} \\ &\quad + \frac{\gamma_{tb}\delta_{tb}(1 - \eta_{tb})\epsilon_{tb}}{(d + \epsilon_1)(d + \gamma_{tb} + \sigma_{tb})(d + \alpha_{tb} + \delta_{tb} + \sigma_{tb})}, \\ &= \mathcal{R}_1^{TB} + \mathcal{R}_2^{TB} + \mathcal{R}_3^{TB}. \end{aligned}$$

Further, we have the local asymptotical stability result for the only TB model (6) in the following theorem.

Theorem 3.1. *The only TB model (6) at the DFE P_{tb}^0 is locally asymptotically stable if $\mathcal{R}_0^{TB} < 1$.*

3.2. Endemic equilibria of the only TB model

The endemic equilibrium of the only TB model (6) denoted by E_1^{TB} and is given below,

$$\begin{cases} S^* = \frac{\Lambda}{d + \lambda_{tb}^*}, \\ E_{tb}^* = \frac{\lambda_{tb}^* S^* + T_{tb}^* \delta_{tb} (1 - \eta_{tb})}{d + \epsilon_{tb}}, \\ T_{tb}^* = \frac{I_{tb}^* \gamma_{tb}}{d + \alpha_{tb} + \delta_{tb} + \sigma_{tb}}, \\ R^* = \frac{T_{tb}^* \alpha_{tb}}{d}, \end{cases} \tag{7}$$

Plugging the expression (7) in the second equation of the only TB model (6), we obtain

$$\Phi_1 I_{tb}^* + \Phi_2 = 0,$$

where

$$\begin{aligned} \Phi_1 &= \epsilon_{tb} ((1 - \beta_{tb}) \sigma_{tb} (d + \alpha_{tb} + \delta_{tb} + \sigma_{ibt}) + \gamma_{tb} \sigma_{ibt}) (\gamma_{tb} (d \delta_{tb} (1 - \eta_{tb}) + (d + \epsilon_{tb}) (d + \sigma_{ibt}))) \\ &\quad + \epsilon_{tb} ((1 - \beta_{tb}) \sigma_{tb} (d + \alpha_{tb} + \delta_{tb} + \sigma_{ibt}) + \gamma_{tb} \sigma_{ibt}) \\ &\quad (\alpha_{tb} (d + \epsilon_{tb}) (d + \gamma_{tb} + \sigma_{tb}) + (d + \sigma_{tb}) (d + \epsilon_{tb}) (d + \delta_{tb} + \sigma_{ibt})), \\ \Phi_2 &= (\Lambda \epsilon_{tb} (d + \epsilon_{tb}) (d + \gamma_{tb} + \sigma_{tb}) (d + \alpha_{tb} + \delta_{tb} + \sigma_{ibt})^2) (1 - \mathcal{R}_0^{TB}). \end{aligned}$$

The coefficients Φ_i for $i = 1, 2$ are positive and therefore the endemic equilibrium of the only TB model exists for $\mathcal{R}_0^{TB} > 1$.

4. Malaria model

The only malaria model (8) given by the following can be obtained by setting $E_{tb} = I_{tb} = T_{tb} = I_{mt} = 0$, we have

$$\begin{aligned} \frac{d}{dt} S &= \Lambda - \lambda_m S - dS, \\ \frac{d}{dt} E_m &= \lambda_m S - (\tau_m + d) E_m, \\ \frac{d}{dt} I_m &= \tau_m E_m - (d + d_m + \gamma_m) I_m, \\ \frac{d}{dt} R &= \gamma_m I_m - dR, \\ \frac{d}{dt} S_v &= \Lambda_v - \lambda_v S_v - d_v S_v, \\ \frac{d}{dt} E_v &= \lambda_v S_v - (d_v + \tau_v) E_v, \\ \frac{d}{dt} I_v &= \tau_v E_v - d_v I_v, \end{aligned} \tag{8}$$

where $\lambda_m = \frac{\beta_m \sigma_m I_v}{N}$, $\lambda_v = \frac{\beta_v \sigma_m I_m}{N}$, $N = S + E_m + I_m + R$ and $N_v = S_v + E_v + I_v$. The feasible region for the only malaria model (8) is

$$\Omega = \left\{ (S, E_m, I_m, R, S_v, E_v, I_v) : N \leq \frac{\Lambda}{d}, N_v \leq \frac{\Lambda_v}{d_v} \right\}.$$

We show further that Ω is positive invariant and will be sufficient to consider the dynamics of Ω :

$$\begin{aligned} N' &= S' + E_m' + I_m' + R', \\ &= \Lambda - dN - d_m I_m, \end{aligned} \tag{9}$$

and

$$\begin{aligned} N_v' &= S_v' + E_v' + I_v', \\ &= \Lambda_v - d_v N_v. \end{aligned} \tag{10}$$

The right hand sides of both the equations (9) and (10) are bounded by $\Lambda - dN$ and $\Lambda_v - d_v N_v$ respectively. It follows that $N'(t) < 0$ if $N(t) > \Lambda/d$ and $N_v'(t) < 0$ if $N_v(t) > \Lambda_v/d_v$. Further, we have by using the standard comparison theorem [6],

$$N(t) \leq \frac{\Lambda}{d} + \left(N(0) - \frac{\Lambda}{d} \right) e^{-dt},$$

$$N_v(t) \leq \frac{\Lambda_v}{d_v} + \left(N_v(0) - \frac{\Lambda_v}{d_v}\right)e^{-d_v t}.$$

In particular if

$$N(t) \leq \frac{\Lambda}{d}, \text{ if } N(0) \leq \frac{\Lambda}{d}$$

and

$$N_v(t) \leq \frac{\Lambda_v}{d_v}, \text{ if } N_v(0) \leq \frac{\Lambda_v}{d_v},$$

which shows that Ω is positive invariant. It is attracting also because $N(0) \geq \frac{\Lambda}{d}$ and $N_v(0) \geq \frac{\Lambda_v}{d_v}$ and then the solution enters Ω in finite time or $N_v(t) \rightarrow \frac{\Lambda_v}{d_v}$ and $N(t) \rightarrow \frac{\Lambda}{d}$ asymptotically and the rest of the infected variables E_m, I_m, E_v and I_v tend to zero.

4.1. Only malaria model basic properties

The disease free equilibrium of the only malaria model (8) denoted by E_0^M and is given by

$$E_0^M = (S^0, 0, 0, 0, S_v^0, 0, 0) = \left(\frac{\Lambda}{d}, 0, 0, 0, \frac{\Lambda_v}{d_v}, 0, 0\right).$$

The basic reproduction in epidemic models plays a vital role and useful for the model to shows the nature of the disease spread and control. For the only malaria model (8), we compute the basic reproduction number in the following by using the next generation matrix method [21],

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_m \sigma_m \\ 0 & 0 & 0 & 0 \\ 0 & \frac{d\beta_v \Lambda_v \sigma_m}{\Lambda d_v} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} d + \tau_m & 0 & 0 & 0 \\ -\tau_m & d + d_m + \gamma_m & 0 & 0 \\ 0 & 0 & d_v + \tau_v & 0 \\ 0 & 0 & -\tau_v & d_v \end{pmatrix}$$

and we have the basic reproduction number \mathcal{R}_0^M , given by

$$\mathcal{R}_0^M = \sqrt{\frac{d\beta_m \sigma_m^2 \tau_m \beta_v \Lambda_v \tau_v}{\Lambda d_v^2 (d_m + d + \gamma_m) (d + \tau_m) (d_v + \tau_v)}}.$$

Theorem 4.1. *The only malaria model (8) at the disease free case E_0^M is locally asymptotically stable if $\mathcal{R}_0^M < 1$.*

5. TB-Malaria coinfection model analysis

The present section describes the dynamics of the TB and malaria coinfection model (1). Initially, we present first the basic properties of the model.

The disease free equilibrium of the coinfection model (1), denoted by E_0^c and is given by

$$E_0^c = (S^0, 0, 0, 0, S_v^0, 0, 0, 0, 0, 0) = \left(\frac{\Lambda}{d}, 0, 0, 0, \frac{\Lambda_v}{d_v}, 0, 0, 0, 0, 0\right).$$

and the basic reproduction number

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_m \sigma_m & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{d\beta_v \Lambda_v \sigma_m}{\Lambda d_v} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_{tb} \sigma_{tb} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} Q_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\tau_m & Q_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & Q_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\tau_v & d_v & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & Q_4 & 0 & -\delta_{tb}(1 - \eta_{tb}) & 0 \\ 0 & 0 & 0 & 0 & -\epsilon_{tb} & Q_5 & -\delta_{tb}\eta_{tb} & 0 \\ 0 & 0 & 0 & 0 & 0 & -\gamma_{tb} & Q_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & Q_7 \end{pmatrix}.$$

The required basic reproduction of the coinfection model (1) is given by

$$\mathcal{R}_0 = \max\{\mathcal{R}_0^M, \mathcal{R}_0^{TB}\} = \left\{ \sqrt{\frac{d\beta_m\sigma_m^2\tau_m\beta_v\Lambda_v\tau_v}{\Lambda Q_1 Q_2 Q_3 d_v^2}}, \frac{Q_6\beta_{tb}\sigma_{tb}\epsilon_{tb} + Q_4\gamma_{tb}\delta_{tb}\eta_{tb} + \gamma_{tb}\delta_{tb}(1 - \eta_{tb})\epsilon_{tb}}{Q_4 Q_5 Q_6} \right\}.$$

Next, we show that the TB-malaria coinfection model is locally asymptotically stable if $\mathcal{R}_0 < 1$. The following result is established.

Theorem 5.1. *The TB-malaria coinfection model (1) is locally asymptotically stable when $\mathcal{R}_0 < 1$.*

Proof. At the disease free case E_0^c , we have the Jacobian matrix in the following:

$$J = \begin{pmatrix} -d & 0 & 0 & 0 & 0 & 0 & -\beta_m\sigma_m & 0 & -\beta_{tb}\sigma_{tb} & 0 & 0 \\ 0 & -Q_1 & 0 & 0 & 0 & 0 & \beta_m\sigma_m & 0 & 0 & 0 & 0 \\ 0 & \tau_m & -Q_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_m & -d & 0 & 0 & 0 & 0 & 0 & \alpha_{tb} & \psi_{mt} \\ 0 & 0 & -\frac{d\beta_v\Lambda_v\sigma_m}{\Lambda d_v} & 0 & -d_v & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{d\beta_v\Lambda_v\sigma_m}{\Lambda d_v} & 0 & 0 & -Q_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_v & -d_v & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -Q_4 & \beta_{tb}\sigma_{tb} & \delta_{tb}(1 - \eta_{tb}) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \epsilon_{tb} & -Q_5 & \delta_{tb}\eta_{tb} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{tb} & -Q_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -Q_7 \end{pmatrix}$$

where $Q_1 = d + \tau_m$, $Q_2 = d_m + d + \gamma_m$, $Q_3 = d_v + \tau_v$, $Q_4 = d + \epsilon_{tb}$, $Q_5 = d + \gamma_{tb} + \sigma_{tb}$, $Q_6 = d + \alpha_{tb} + \delta_{tb} + \sigma_{tb}$ and $Q_7 = d + \psi_{mt} + \epsilon$. The eigenvalues $-d, -d, -d, -d_v$ are negative for the remaining we have:

$$\lambda^7 + \Phi_1\lambda^6 + \Phi_2\lambda^5 + \Phi_3\lambda^4 + \Phi_4\lambda^3 + \Phi_5\lambda^2 + \Phi_6\lambda\Phi_7 = 0, \tag{11}$$

where

$$\begin{aligned} \Phi_1 &= d_v + Q_1 + Q_2 + Q_3 + Q_4 + Q_5 + Q_6, \\ \Phi_2 &= Q_2Q_3 + Q_2Q_4 + Q_3Q_4 + Q_2Q_5 + Q_3Q_5 + (Q_2 + Q_3 + Q_4)Q_6 \\ &\quad + Q_1(Q_2 + Q_3 + Q_4 + Q_5 + Q_6) + (Q_1 + Q_2 + Q_3 + Q_4 + Q_5 + Q_6)d_v \\ &\quad + Q_4Q_5(1 - \mathcal{R}_1^{TB}) + Q_5Q_6(1 - \mathcal{R}_2^{TB}), \\ \Phi_3 &= (Q_3(Q_4 + Q_5) + (Q_3 + Q_4)Q_6 + Q_2(Q_3 + Q_4 + Q_5 + Q_6))d_v \\ &\quad + Q_1(Q_2 + Q_3 + Q_4 + Q_5 + Q_6)d_v \\ &\quad + Q_2Q_3(Q_4 + Q_5) + (Q_3Q_4 + Q_2(Q_3 + Q_4))Q_6 \end{aligned}$$

$$\begin{aligned}
 &+Q_1 (Q_4Q_6 + Q_3 (Q_4 + Q_5 + Q_6) + Q_2 (Q_3 + Q_4 + Q_5 + Q_6)) \\
 &+ (Q_4Q_5(1 - \mathcal{R}_1^{TB}) + Q_6Q_5(1 - \mathcal{R}_2^{TB})) (d_v + Q_1 + Q_2 + Q_3) \\
 &+ Q_4Q_5Q_6(1 - \mathcal{R}_0^{TB}), \\
 \Phi_4 = & Q_4Q_5Q_6[d_v + Q_1 + (Q_2 + Q_3)](1 - \mathcal{R}_0^{TB}) + Q_1Q_2Q_3d_v(1 - (\mathcal{R}_0^M)^2) \\
 &(Q_4 + Q_5 + Q_6) ((Q_2Q_3 + Q_1 (Q_2 + Q_3)) d_v + Q_1Q_2Q_3) \\
 &+ ((1 - \mathcal{R}_1^{TB})Q_4Q_5 + (1 - \mathcal{R}_2^{TB})Q_6Q_5 + Q_4Q_6) \times \\
 &((Q_1 + Q_2 + Q_3) d_v + Q_2Q_3 + Q_1 (Q_2 + Q_3)), \\
 \Phi_5 = & ((Q_2Q_3 + Q_1 (Q_2 + Q_3)) d_v + Q_1Q_3) (Q_4Q_5(1 - \mathcal{R}_1^{TB}) + Q_5Q_6(1 - \mathcal{R}_2^{TB}) + Q_4Q_6) \\
 &+ Q_1Q_2Q_3 (Q_4 + Q_5 + Q_6) d_v(1 - (\mathcal{R}_0^M)^2) \\
 &+ Q_1Q_4^2Q_5^2Q_6^2d_v [Q_2 (d_v + Q_1) + Q_3 (d_v + Q_1 + Q_2)] (1 - \mathcal{R}_0^{TB}), \\
 \Phi_6 = & Q_4Q_5Q_6[(Q_2Q_3 + Q_1 (Q_2 + Q_3)) d_v + Q_1Q_2Q_3](1 - \mathcal{R}_0^{TB}) \\
 &+ [Q_4Q_5(1 - \mathcal{R}_1^{TB}) + Q_5Q_6(1 - \mathcal{R}_2^{TB}) + Q_4Q_6] (1 - (\mathcal{R}_0^M)^2), \\
 c_7 = & Q_1Q_2Q_3Q_4Q_5Q_6d_v(1 - (\mathcal{R}_0^M)^2)(1 - \mathcal{R}_0^{TB}).
 \end{aligned}$$

The coefficients Φ_i for $i = 1, 2, \dots, 7$ given in equation (11) are positive whenever the basic reproduction number less than unity. The positivity of the coefficients Φ_i for $i = 1, 2, \dots, 7$ ensures that the conditions given in [17] for the model (1) could be satisfy easily and it will have all the eigenvalues with negative real parts. Therefore, it is to be concluded that the model (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$. \square

Further, we discuss the phenomenon of backward bifurcation for the TB and Malaria coinfection model. To show this results we use the central manifold theory [2]. We the result:

5.1. Backward bifurcation for TB malaria coinfection model

We show the existence of backward bifurcation for the TB and malaria coinfection model (1). Apply the centre manifold theory to the model (1) and taking $\mathcal{R}_0^{TB} = 1$ and $\mathcal{R}_0^M = 1$ if and only if

$$\beta_m = \beta_m^* = \frac{\Lambda Q_1 Q_2 Q_3 d_v^2}{d \sigma_m^2 \tau_m \beta_v \Lambda_v \tau_v},$$

and

$$\beta_{tb} = \beta_{tb}^* = \frac{Q_4 (Q_5 Q_6 - \gamma_{tb} \delta_{tb} \eta_{tb}) - \gamma_{tb} \delta_{tb} (1 - \eta_{tb}) \epsilon_{tb}}{Q_6 \sigma_{tb} \epsilon_{tb}}.$$

Further, we change the variables and the model (1) and replace with the new set of variables, $y_1 = S$, $y_2 = E_m$, $y_3 = I_m$, $y_4 = R$, $y_5 = S_v$, $y_6 = E_v$, $y_7 = I_v$, $y_8 = E_{tb}$, $y_9 = I_{tb}$, $Y_{10} = T_{tb}$ and $y_{11} = I_{mt}$ with $N = y_1 + y_2 + y_3 + y_4 + y_8 + y_9 + y_{10} + y_{11}$ and $N_v = y_5 + y_6 + y_7$. Using the vector notation $\vec{y} = (y_1, \dots, y_{11})$

and express the model (1) in the form $y' = F\vec{y}$, where $F = f_1, \dots, F_{11}$, shown in the following:

$$\left\{ \begin{aligned} \frac{d}{dt}y_1 &= \Lambda - \lambda_{tb}y_1 - \lambda_my_1 - dy_1, \\ \frac{d}{dt}y_2 &= \lambda_my_1 - (\tau_m + d)y_2, \\ \frac{d}{dt}y_3 &= \tau_my_2 - (d + d_m + \gamma_m)y_3 - \lambda_{tb}y_3, \\ \frac{d}{dt}y_4 &= \gamma_my_3 + \alpha_{tb}y_{10} + \psi_{mt}y_{11} - dy_4, \\ \frac{d}{dt}y_5 &= \Lambda_v - \lambda_vy_5 - d_vy_5, \\ \frac{d}{dt}y_6 &= \lambda_vy_5 - (d_v + \tau_v)y_6, \\ \frac{d}{dt}y_7 &= \tau_vy_6 - d_vy_7, \\ \frac{d}{dt}y_8 &= \lambda_{tb}y_1 - (d + \epsilon_{tb})y_8 + (1 - \eta_{tb})\delta_{tb}y_{10}, \\ \frac{d}{dt}y_9 &= \epsilon_{tb}y_8 + \eta_{tb}\delta_{tb}y_{10} - (d + \gamma_{tb} + \sigma_{tb})y_9 - \lambda_my_9, \\ \frac{d}{dt}y_{10} &= \gamma_{tb}y_{tb} - (d + \delta_{tb} + \sigma_{tb} + \alpha_{tb})y_{10}, \\ \frac{d}{dt}y_{11} &= \lambda_{tb}y_3 + \lambda_my_9 - (\epsilon + d + \psi_{mt})y_{11}, \end{aligned} \right. \tag{12}$$

where $\lambda_{tb} = \frac{\beta_{tb}\sigma_{tb}y_9}{N}$, $\lambda_m = \frac{\beta_m\sigma_my_7}{N}$ and $\lambda_v = \frac{\beta_v\sigma_vy_3}{N}$.
 Computing the Jacobian matrix of the model (12) at E_0^c , we have

$$G = \begin{pmatrix} -d & 0 & 0 & 0 & 0 & 0 & -J_1 & 0 & -J_3 & 0 & 0 \\ 0 & -Q_1 & 0 & 0 & 0 & 0 & J_1 & 0 & 0 & 0 & 0 \\ 0 & \tau_m & -Q_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_m & -d & 0 & 0 & 0 & 0 & 0 & \alpha_{tb} & \psi_{mt} \\ 0 & 0 & -J_2 & 0 & -d_v & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_2 & 0 & 0 & -Q_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_v & -d_v & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -Q_4 & J_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \epsilon_{tb} & -Q_5 & \delta_{tb}\eta_{tb} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{tb} & -Q_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -Q_7 \end{pmatrix}$$

where $J_1 = \frac{\Lambda d_v^2 Q_1 Q_2 Q_3}{d \beta_v \Lambda_v \sigma_m \tau_m \tau_v}$, $J_2 = \frac{d \beta_v \Lambda_v \sigma_m}{\Lambda d_v}$, $J_3 = \frac{Q_4(Q_5 Q_6 - \gamma_{tb} \delta_{tb} \eta_{tb}) - \gamma_{tb} \delta_{tb} \epsilon_{tb} (1 - \eta_{tb})}{Q_6 \epsilon_{tb}}$.

It can be seen that the Jacobian matrix G has the simple zero eigenvalues and the rest have the negative real parts, this ensures, that we can apply the centre manifold theory to model of TB and malaria. To proceed further, we need to obtain the right and left eigenvectors associated to the matrix G respectively, we obtain,

$$\begin{aligned} w_1 &= -\frac{Q_1 Q_2 w_3}{d \tau_m} - \frac{w_9 (Q_4 (Q_5 Q_6 - \gamma_{tb} \delta_{tb} \eta_{tb}) - \gamma_{tb} \delta_{tb} (1 - \eta_{tb}) \epsilon_{tb})}{d Q_6 \epsilon_{tb}}, w_2 = \frac{Q_2 w_3}{\tau_m}, \\ w_3 &= w_3 > 0, w_4 = \frac{w_3 \gamma_m}{d} + \frac{w_9 \alpha_{tb} \gamma_{tb}}{d Q_6}, w_5 = -\frac{d w_3 \sigma_m \beta_v \Lambda_v}{\Lambda d_v^2}, w_6 = \frac{d w_3 \sigma_m \beta_v \Lambda_v}{\Lambda Q_3 d_v}, \\ w_7 &= \frac{d w_3 \sigma_m \beta_v \Lambda_v \tau_v}{\Lambda Q_3 d_v^2}, w_8 = -\frac{w_9 (\gamma_{tb} \delta_{tb} \eta_{tb} - Q_5 Q_6)}{Q_6 \epsilon_{tb}}, w_{10} = \frac{w_9 \gamma_{tb}}{Q_6}, w_{11} = 0, w_9 = w_0 > 0, \end{aligned}$$

and

$$v_1 = v_4 = v_5 = v_{11} = 0, v_2 = v_2 > 0, v_8 = v_8 > 0, v_3 = \frac{Q_1 v_2}{\tau_m}, v_6 = \frac{\Lambda Q_1 Q_2 v_2 d_v}{d \sigma_m \tau_m \beta_v \Lambda_v},$$

$$v_7 = \frac{\Lambda Q_1 Q_2 Q_3 v_2 d_v}{d \sigma_m \tau_m \beta_v \Lambda_v \tau_v}, v_9 = \frac{Q_4 v_8}{\epsilon_{tb}}, v_{10} = -\frac{v_8 \delta_{tb} (-Q_4 \eta_{tb} + \eta_{tb} \epsilon_{tb} - \epsilon_{tb})}{Q_6 \epsilon_{tb}}.$$

Further step is the computations of the values of a and b . We follow [2], and obtain the value of a and b after rigorous computations, we have

$$\begin{aligned} a = & -\frac{2}{\Lambda^2 Q_3 Q_6 d_v^2 \tau_m^2 \epsilon_{tb}} \left[d^2 Q_6 w_3 \beta_m \sigma_m^2 \tau_m \beta_v \Lambda_v \tau_v (w_9 \tau_m (Q_5 v_2 + Q_4 v_8) + Q_2 v_2 w_3 \epsilon_{tb}) \right. \\ & + d v_2 w_3 \beta_m \sigma_m^2 \tau_m^2 \beta_v \Lambda_v \tau_v (Q_6 \epsilon_{tb} (w_3 (d + \gamma_m) + d w_9) + w_9 \gamma_{tb} (\epsilon_{tb} (d + \alpha_{tb}) - d \delta_{tb} \eta_{tb})) \\ & + \Lambda Q_3 w_9 d_v^2 \tau_m (w_3 (d Q_6 (Q_1 v_2 + Q_2 v_8) \beta_{tb} \sigma_{tb} \epsilon_{tb} + Q_1 Q_2 v_2 \gamma_{tb} \delta_{tb} \eta_{tb} (Q_4 - \epsilon_{tb}))) \\ & + \Lambda Q_3 v_8 w_9 d_v^2 \tau_m^2 \beta_{tb} \sigma_{tb} (Q_6 \epsilon_{tb} (w_3 (d + \gamma_m) + d w_9) + d Q_5 Q_6 w_9 + w_9 \gamma_{tb} (\epsilon_{tb} (d + \alpha_{tb}) - d \delta_{tb} \eta_{tb})) \\ & + \Lambda Q_1 Q_2^2 Q_3 Q_6 v_2 w_3^2 (d - Q_1) d_v^2 \epsilon_{tb} + \Lambda Q_1 Q_2 Q_3 v_2 w_3 d_v^2 \tau_m \\ & \left. + \Lambda Q_1 Q_2 Q_3 v_2 w_3 d_v^2 \tau_m (Q_5 Q_6 w_9 (d - Q_4) + w_9 \gamma_{tb} (\epsilon_{tb} (d + \alpha_{tb} + \delta_{tb}) - d \delta_{tb} \eta_{tb})) \right] \end{aligned}$$

and

$$b = v_8 w_9 \sigma_{tb} > 0.$$

It is obvious that $b > 0$ and the value of a can determined the backward bifurcation in the coinfection model (1) if $a > 0$.

6. Optimal control problem

The aims of this section to formulate an optimal control problem for the coinfection of TB and Malaria. We use five controls u_i for $i = 1, 2, \dots, 5$ to minimize the coinfection in the model (13). In the given optimal control problem the control u_1 represents human mosquitos elimination by LLITNs (Long-Lasting Insecticide-Treated Nets). The control variable u_2 represents the treatment efforts used for malaria infected individuals. The control variable u_3 represents IRS(indoor residual spraying) which increase the death rate in mosquitos. The control variables u_4 and u_5 respectively represent the prevention and treatment efforts for TB infected individuals and the efforts of treatment for infected TB individuals. The parameters c_1, κ, c_2 and c_3 respectively represent, mosquitoes death rate by LLITNs, death by IRS, recovery with treatment of malaria infection of malaria-TB individuals, and recovery by the treatment of TB infection of malaria-TB individuals. Keeping in mind the above assumptions we formulate the following optimal control problem:

$$\left\{ \begin{aligned} \frac{d}{dt} S &= \Lambda - (1 - u_4) \lambda_{tb} S - (1 - u_1) \lambda_m S - dS, \\ \frac{d}{dt} E_m &= (1 - u_1) \lambda_m S - (\tau_m + d) E_m, \\ \frac{d}{dt} I_m &= \tau_m E_m - (d + d_m + u_2 \gamma_m) I_m - (1 - u_4) \lambda_{tb} I_m, \\ \frac{d}{dt} R &= u_2 \gamma_m I_m + \alpha_{tb} T_{tb} + (\psi_{mt} + c_2 u_2 + c_3 u_3) I_{mt} - dR, \\ \frac{d}{dt} S_v &= \Lambda_v - \lambda_v S_v - (d_v + \kappa u_3 + c_1 u_1) S_v, \\ \frac{d}{dt} E_v &= \lambda_v S_v - (d_v + \tau_v + \kappa u_3 + c_1 u_1) E_v, \\ \frac{d}{dt} I_v &= \tau_v E_v - (d_v + \kappa u_3 + c_1 u_1) I_v, \\ \frac{d}{dt} E_{tb} &= (1 - u_4) \lambda_{tb} S - (d + \epsilon_{tb}) E_{tb} + (1 - \eta_{tb}) \delta_{tb} T_{tb}, \\ \frac{d}{dt} I_{tb} &= \epsilon_{tb} E + \eta_{tb} \delta_{tb} T_{tb} - (d + u_5 \gamma_{tb} + \sigma_{tb}) I_{tb} - (1 - u_1) \lambda_m I_{tb}, \\ \frac{d}{dt} T_{tb} &= u_5 \gamma_{tb} I_{tb} - (d + \delta_{tb} + \sigma_{tb} + \alpha_{tb}) T_{tb}, \\ \frac{d}{dt} I_{mt} &= (1 - u_4) \lambda_{tb} I_m + (1 - u_1) \lambda_m I_{tb} - (\epsilon + d + \psi_{mt} + c_2 u_2 + c_3 u_3) I_{mt}. \end{aligned} \right. \tag{13}$$

where $\lambda_{tb} = \frac{\beta_{tb}\sigma_{tb}I_{tb}}{N}$, $\lambda_m = \frac{\beta_m\sigma_m I_v}{N}$ and $\lambda_v = \frac{\beta_v\sigma_v I_m}{N}$. The objective functional for the optimal control problem is defined by

$$\Delta(u_1, u_2, u_3, u_4, u_5) = \int_0^T [P_1 E_m + P_2 I_m + P_3 N_v + P_4 E_{tb} + P_5 I_{tb} + P_6 I_{mt} + \frac{1}{2}(A_1 u_1^2 + A_2 u_2^2 + A_3 u_3^2 + A_4 u_4^2 + A_5 u_5^2)] dt, \tag{14}$$

subject to the nonlinear system of coinfection model (13) with appropriate initial conditions. In the objective functional (14), the weight constants P_i for $i = 1, \dots, 6$ are used for state variables while A_i for $i = 1, 2, \dots, 5$ are used for the control variables. The **cost function** for P_1 is used for exposed only malaria, P_2 is for infected only malaria, P_3 for vector population, P_4 for exposed only TB, P_5 for infected only TB, and P_6 is for the dually infected individuals. The quadratic form is used for the controls, $\frac{1}{2}A_1 u_1^2$, $\frac{1}{2}A_2 u_2^2$, $\frac{1}{2}A_3 u_3^2$, $\frac{1}{2}A_4 u_4^2$, and $\frac{1}{2}A_5 u_5^2$ show the expenditure on LLITNs, malaria treatment, IRS, treatment and prevention for TB, and treatment of infected TB individuals. Hence using optimal controls u_i^* for $i = 1, 2, \dots, 5$ such that

$$\Delta(u_i^*) = \min_{u_i \in \Theta} \Delta(u_i),$$

where $\Theta = \{u = (u_1, u_2, u_3, u_4, u_5) \mid u_i(t) \text{ is lebesgue measurable, } u_i(t) \in [0, 1] \text{ for all } t \in [0, T], \text{ where } i = 1, 2, \dots, 5.\}$ is the control set associated to the coinfection control model (13). Further, we define the Lagrangian L and Hamiltonian H for the optimal control problem (13), given by

$$L = P_1 E_m + P_2 I_m + P_3 N_v + P_4 E_{tb} + P_5 I_{tb} + P_6 I_{mt} + \frac{1}{2}(A_1 u_1^2 + A_2 u_2^2 + A_3 u_3^2 + A_4 u_4^2 + A_5 u_5^2), \tag{15}$$

and

$$\begin{aligned} H = & L + \lambda_1[\Lambda - (1 - u_4)\lambda_{tb}S - (1 - u_1)\lambda_m S - dS] + \lambda_2[(1 - u_1)\lambda_m S - (\tau_m + d)E_m] \\ & + \lambda_3[\tau_m E_m - (d + d_m + u_2\gamma_m)I_m - (1 - u_4)\lambda_{tb}I_m] \\ & + \lambda_4[u_2\gamma_m I_m + \alpha_{tb}T_{tb} + (\psi_{mt} + c_2 u_2 + c_3 u_3)I_{mt} - dR] \\ & + \lambda_5[\Lambda_v - \lambda_v S_v - (d_v + \kappa u_3 + c_1 u_1)S_v] \\ & + \lambda_6[\lambda_v S_v - (d_v + \tau_v + \kappa u_3 + c_1 u_1)E_v] \\ & + \lambda_7[\tau_v E_v - (d_v + \kappa u_3 + c_1 u_1)I_v] \\ & + \lambda_8[(1 - u_4)\lambda_{tb}S - (d + \epsilon_{tb})E_{tb} + (1 - \eta_{tb})\delta_{tb}T_{tb}] \\ & + \lambda_9[\epsilon_{tb}E + \eta_{tb}\delta_{tb}T_{tb} - (d + u_5\gamma_{tb} + \sigma_{tb})I_{tb} - (1 - u_1)\lambda_m I_{tb}] \\ & + \lambda_{10}[u_5\gamma_{tb}I_{tb} - (d + \delta_{tb} + \sigma_{tb} + \alpha_{tb})T_{tb}] \\ & + \lambda_{11}[(1 - u_4)\lambda_{tb}I_m + (1 - u_1)\lambda_m I_{tb} - (\epsilon + d + \psi_{mt} + c_2 u_2 + c_3 u_3)I_{mt}]. \end{aligned} \tag{16}$$

This leads to the following statement.

Theorem 6.1. *The optimal control coinfection model (13) with appropriate initial conditions then there exists an optimal control $u^* = u_i^* \in \Theta$ for $i = 1, 2, \dots, 5$ such that*

$$\Delta(u_i^*) = \min_{u_i \in \Theta} \Delta(u_i).$$

The Lipschitz property of the control model with respect to the model variables are satisfied by the Theorem 6.1. So, then there exists some positive numbers ω_1 and ω_2 together with ν such that

$$\Delta(u_i) \geq \omega_1(|u_1|^2 + |u_2|^2 + |u_3|^2 + |u_4|^2 + |u_5|^2)^{\nu/2} - \omega_2.$$

This proves the optimal control problem existence. Next, we obtain the adjoint equations and the optimal control characterizations. We have

Theorem 6.2. *The adjoint variables λ_i for $i = 1, \dots, 11$ exists for the optimal controls u_i for $i = 1, 2, \dots, 5$ with optimal control model solutions $(S^*, E_m^*, I_m^*, R^*, S_v^*, E_v^*, I_v^*, E_{tb}^*, I_{tb}^*, T_{tb}^*, I_m^*)$ satisfying:*

$$\begin{aligned} \frac{d\lambda_1}{dt} &= (\lambda_1 - \lambda_8)(1 - u_4)\lambda_{tb}^* \frac{(N^* - S^*)}{N^*} + (\lambda_1 - \lambda_2)(1 - u_1)\lambda_m^* \frac{(N^* - S^*)}{N^*} \\ &\quad + (\lambda_{11} - \lambda_3)(1 - u_4)\lambda_{tb}^* \frac{I_m^*}{N^*} + (\lambda_{11} - \lambda_9)(1 - u_1)\lambda_m^* \frac{I_{tb}^*}{N^*} + d\lambda_1 \\ &\quad + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*}, \\ \frac{d\lambda_2}{dt} &= (\lambda_8 - \lambda_1)(1 - u_4)\lambda_{tb}^* \frac{S^*}{N^*} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_{11} - \lambda_3)(1 - u_4)\lambda_{tb}^* \frac{I_m^*}{N^*} \\ &\quad + (\lambda_{11} - \lambda_9)(1 - u_1)\lambda_m^* \frac{I_{tb}^*}{N^*} + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*} + (\lambda_2 - \lambda_3)\tau_m + d\lambda_2 - P_1, \\ \frac{d\lambda_3}{dt} &= (\lambda_8 - \lambda_1)(1 - u_4)\lambda_{tb}^* \frac{S^*}{N^*} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_5 - \lambda_6)S_v^*\beta_v\sigma_m \frac{(N^* - I_m^*)}{N^{*2}} \\ &\quad + (\lambda_3 - \lambda_{11})(1 - u_4)\lambda_{tb}^* \frac{(N^* - I_m^*)}{N^*} + (\lambda_{11} - \lambda_9)(1 - u_1)\lambda_m^* \frac{I_{tb}^*}{N^*} + (\lambda_3 - \lambda_4)u_2\gamma_m \\ &\quad + (d + d_m)\lambda_3 - P_2, \\ \frac{d\lambda_4}{dt} &= (\lambda_8 - \lambda_1)(1 - u_4)\lambda_{tb}^* \frac{S^*}{N^*} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_{11} - \lambda_3)(1 - u_4)\lambda_{tb}^* \frac{I_m^*}{N^*} \\ &\quad + (\lambda_{11} - \lambda_9)(1 - u_1)\lambda_m^* \frac{I_{tb}^*}{N^*} + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*} + d\lambda_4, \\ \frac{d\lambda_5}{dt} &= (\lambda_5 - \lambda_6)\lambda_v^* + \lambda_5(d_v + \kappa u_3 + c_1 u_1) - P_3, \\ \frac{d\lambda_6}{dt} &= (\lambda_6 - \lambda_7)\tau_v + \lambda_6(d_v + \kappa u_3 + c_1 u_3) - P_3, \\ \frac{d\lambda_7}{dt} &= (\lambda_1 - \lambda_2)(1 - u_1)\beta_m\sigma_m \frac{S^*}{N^*} + (\lambda_9 - \lambda_{11})(1 - u_1)\beta_m\sigma_m \frac{I_{tb}^*}{N^*} + (d_v + \kappa u_3 + c_1 u_1)\lambda_7 - P_3, \\ \frac{d\lambda_8}{dt} &= (\lambda_8 - \lambda_1)(1 - u_4)\lambda_{tb}^* \frac{S^*}{N^*} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*} \\ &\quad + (\lambda_{11} - \lambda_3)(1 - u_4)\lambda_{tb}^* \frac{I_m^*}{N^*} + (\lambda_{11} - \lambda_9)(1 - u_1)\lambda_m^* \frac{I_{tb}^*}{N^*} + (\lambda_8 - \lambda_9)\epsilon_{tb} + d\lambda_8 - P_4, \\ \frac{d\lambda_9}{dt} &= (\lambda_1 - \lambda_8)(1 - u_4)S^*\beta_{tb}\delta_{tb} \frac{(N^* - I_{tb}^*)}{N^{*2}} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*} \\ &\quad + (\lambda_3 - \lambda_{11})(1 - u_4)\beta_{tb}\delta_{tb}I_m^* \frac{(N^* - I_{tb}^*)}{N^{*2}} + (\lambda_9 - \lambda_{11})(1 - u_1)\lambda_m^* \frac{(N^* - I_{tb}^*)}{N^*} + \\ &\quad (\lambda_9 - \lambda_{10})u_5\gamma_{tb} + (d + \sigma_{tb})\lambda_9 - P_5, \\ \frac{d\lambda_{10}}{dt} &= (\lambda_8 - \lambda_1)(1 - u_4)\lambda_{tb}^* \frac{S^*}{N^*} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*} \end{aligned}$$

$$\begin{aligned}
 & +(\lambda_{11} - \lambda_3)(1 - u_4)\lambda_{tb}^* \frac{I_m^*}{N^*} + (\lambda_{11} - \lambda_9)\lambda_m^* \frac{I_{tb}^*}{N^*} + (\lambda_{10} - \lambda_4)\alpha_{tb} \\
 & + \lambda_{10}(\sigma_{tb} + d) + (\lambda_8 - \lambda_9)\eta_{tb}\delta_{tb} + \delta_{tb}(\lambda_{10} - \lambda_8), \\
 \frac{d\lambda_{11}}{dt} = & (\lambda_8 - \lambda_1)(1 - u_4)\lambda_{tb}^* \frac{S^*}{N^*} + (\lambda_2 - \lambda_1)(1 - u_1)\lambda_m^* \frac{S^*}{N^*} + (\lambda_{11} - \lambda_3)\lambda_{tb}^* \frac{I_m^*}{N^*}(1 - u_4) \\
 & + (\lambda_{11} - \lambda_9)(1 - u_1)\lambda_m^* \frac{I_{tb}^*}{N^*} + (\lambda_6 - \lambda_5)\lambda_v^* \frac{S_v^*}{N^*} + (c_2u_2 + c_3u_3)(\lambda_{11} - \lambda_4) \\
 & \psi_{mt}(\lambda_{11} - \lambda_4) + (d + \varepsilon)\lambda_{11} - P_6,
 \end{aligned} \tag{17}$$

with the transversality conditions $\lambda_i(T) = 0$ where $i = 1, 2, 3, \dots, 11$. Furthermore, the optimality condition:

$$\begin{aligned}
 u_1^* &= \max\left\{\min\left(\frac{(\lambda_2 - \lambda_1)\lambda_m^* S^* + (\lambda_{11} - \lambda_9)\lambda_m^* I_{tb}^* + c_1(\lambda_5 S_v^* + \lambda_6 E_v^* + \lambda_7 I_v^*)}{A_1}, 1\right), 0\right\} \\
 u_2^* &= \max\left\{\min\left(\frac{(\lambda_3 - \lambda_4)\gamma_m I_m^* + (\lambda_{11} - \lambda_4)c_2 I_{mt}^*}{A_2}, 1\right), 0\right\}, \\
 u_3^* &= \max\left\{\min\left(\frac{(\lambda_{11} - \lambda_4)c_3 I_{mt}^* + (\lambda_5 S_v^* + \lambda_6 E_v^* + \lambda_7 I_v^*)\kappa}{A_3}, 1\right), 0\right\}, \\
 u_4^* &= \max\left\{\min\left(\frac{(\lambda_8 - \lambda_1)\lambda_{tb}^* S^* + (\lambda_{11} - \lambda_3)\lambda_{tb}^* I_m^*}{A_4}, 1\right), 0\right\}, \\
 u_5^* &= \max\left\{\min\left(\frac{(\lambda_9 - \lambda_{10})\gamma_{tb} I_{tb}^*}{A_5}, 1\right), 0\right\},
 \end{aligned} \tag{18}$$

is satisfied by the optimal control u_i^* for $i = 1, 2, \dots, 5$ that minimizes Δ over Θ .

Proof. The equations that govern the adjoint equations are utilized to obtain the system of adjoint equations. Differentiating the Hamiltonian system, H , with time giving the system of adjoint equations (17). Further, solving $\frac{\partial H}{\partial u_i} = 0$ for $i = 1, 2, \dots, 5$ on the interior of the control set and obtain

$$\begin{aligned}
 u_1^* &= \max\left\{\min\left(\frac{(\lambda_2 - \lambda_1)\lambda_m^* S^* + (\lambda_{11} - \lambda_9)\lambda_m^* I_{tb}^* + c_1(\lambda_5 S_v^* + \lambda_6 E_v^* + \lambda_7 I_v^*)}{A_1}, 1\right), 0\right\} \\
 u_2^* &= \max\left\{\min\left(\frac{(\lambda_3 - \lambda_4)\gamma_m I_m^* + (\lambda_{11} - \lambda_4)c_2 I_{mt}^*}{A_2}, 1\right), 0\right\}, \\
 u_3^* &= \max\left\{\min\left(\frac{(\lambda_{11} - \lambda_4)c_3 I_{mt}^* + (\lambda_5 S_v^* + \lambda_6 E_v^* + \lambda_7 I_v^*)\kappa}{A_3}, 1\right), 0\right\}, \\
 u_4^* &= \max\left\{\min\left(\frac{(\lambda_8 - \lambda_1)\lambda_{tb}^* S^* + (\lambda_{11} - \lambda_3)\lambda_{tb}^* I_m^*}{A_4}, 1\right), 0\right\}, \\
 u_5^* &= \max\left\{\min\left(\frac{(\lambda_9 - \lambda_{10})\gamma_{tb} I_{tb}^*}{A_5}, 1\right), 0\right\}.
 \end{aligned} \tag{19}$$

□

7. Numerical results

Here, we consider the numerical solution of the optimal control problem (13) and system without control (2). The fourth order Runge-Kutta backward scheme is used to perform the simulations. In the optimal control model we used five controls variables for the disease of TB and malaria coinfection control and each control is defined in detailed in previous section. In this simulations, the parameters considered are given in Table 2. The authors parameters that is the weight constants used in the objective functional are

given as $A_1 = 200, A_2 = 100, A_3 = 100, A_4 = 100, A_5 = 100, P_1 = 100, P_2 = 200, P_3 = 100, P_4 = 100, P_5 = 100$ and $P_6 = 100$. The optimal control system together with adjoint equations and the optimal control characterizations with comparison to the system without control is numerically solved and the corresponding graphical results are presented in Figure 1 to 12. In the numerical simulation the time level is chosen in days and keep it upto 100. We perform different simulations based on the controls selection strategies and the respective graphical results for each strategy is provided. It is to be noted that these strategies selected for possible eliminations of the disease in the community. To explain each strategy in detail, we provide the following explanations.

Parameters	Description	value	Ref
Λ	Recruited rate of susceptible individuals	100 day^{-1}	[12]
d	Natural death rate of human	0.00004 day^{-1}	[23]
d_v	Natural death rate of vector	0.1429 day^{-1}	[3]
τ_m	Humans individuals exposed to malaria infection rate	1/17 day^{-1}	[1]
d_m	Disease death rate due to malaria	0.05 day^{-1}	[5]
σ_{ibt}	Disease death rate due to TB	0.01	[20]
γ_m	Recovery from malaria	0.0005	[4]
Λ_v	Recruitment rate of vector population	1000 day^{-1}	[12]
τ_v	Rate of flow from exposed vector to infected vector	1/18 day^{-1}	[1]
ϵ_{itb}	Rate of flow from exposed TB to infected TB	0.03	[20]
β_m	Contacts rate	0.8333	[3]
β_v	Contacts rate	0.09	[1]
σ_m	Biting rate of mosquito	0.2	[1]
σ_{itb}	Modification parameter	0.03 per day	[8]
κ	death rate of mosquitoes due to using of IRS	0.01 per day	[11]
c_1	death rate of mosquitoes due to using LLITNs	0.05 per day	[11]
γ_{itb}	Treatment rate of TB infected individuals	0.1	[7]
c_2	recovery rate due to malaria treatment of malaria-TB individuals	0.25 per day	[11]
c_3	recovery rate due to TB treatment of malaria-TB individuals	0.25 per day	[11]
α_{itb}	Recovery from TB	0.3968	[11]
β_{itb}	Contacts rate	0.05 per day	[8]
ψ_{mt}	Recovery from dual infection	source	Assumed
ϵ	Disease death rate due to dual infection	source	Assumed
δ_{itb}	Rate of flow of TB	1.1996	Assumed
η_{itb}	Treatment failure	0.15	Assumed

Table 2: Parameters values used in TB-Malaria coinfection simulation (13).

7.1. Strategy 1: ($u_3 = u_4 = u_5 \neq 0$)

In this strategy, we set the controls variables $u_1 = u_2 = 0$ and $u_3 = u_4 = u_5 \neq 0$ and perform the simulations by optimizing the objective functional Δ and obtain the graphical results given by Figure 1 and 2. Here in this strategy the control variable u_3 represents IRS which is used to increase the death rate in mosquitos while the control variables u_4 and u_5 is respectively used to represent the prevention and treatment efforts for TB infected individuals and the efforts of treatment for infected TB individuals. The graphical results obtained through this strategy demonstrate that the population of susceptible vector, exposed vector, infected vector and coinfeeced individuals decreases while the individuals exposed only with malaria, individuals only infected with malaria and individuals only infected with TB is decreases sharply and the there is no effect on the exposed individuals due to only TB. This strategy is helpful for the elimination of dual infections and the infections in vector populations while not much suitable for other infected compartments.

7.2. Strategy 2: ($u_1 = u_4 = u_5 \neq 0$)

This strategy is performed by using the controls variables $u_2 = u_3 = 0$ and $u_1 = u_4 = u_5 \neq 0$ and optimize the objective functional Δ to obtain the numerical results given in Figure 3 and 4. Using of the control u_1 , that is the human mosquitos elimination by LLITNs, and the controls variables u_4 and u_5 is respectively used to represent the prevention and treatment efforts for TB infected individuals and the efforts of treatment for

infected TB individuals, one can see that this strategy is helpful for the individuals exposed and infected with malaria only, individuals exposed and infected only with TB and the dually infected individuals. In comparison to the strategy 1, one can see that the infected individuals due to TB and malaria are decreases much while the decrease in the vector population occur at day 20 till day 100. The most important control in this strategy is the u_1 (the human mosquitos elimination by LLITNs), which greatly contributing in this strategy by eliminating the infections in the infected compartments.

7.3. Strategy 3: ($u_1 = u_3 = u_5 \neq 0$)

In this proposed strategy, we set the controls $u_2 = u_4 = 0$ and make active the controls $u_1 = u_3 = u_5 \neq 0$ and optimize the objective functional Δ . The graphical results obtained through this simulations is presented in Figure 5 and 6. Using of the control u_1 , that is the human mosquitos elimination by LLITNs, the control variable u_3 represents IRS which increase the death rate in mosquitos and the control u_5 the efforts of treatment for infected TB only individuals and performed the simulations. One can see that this strategy is also very helpful for the eliminations of individuals exposed and infected only with TB and malaria and the coinfecting individuals. In comparison to the strategy 2, there is a little decrease in individuals of malaria exposed only, exposed vector, infected vector, exposed individuals with TB only and the individuals infected only with TB while the rest have the same effect as previously strategy 2. This strategy could be useful for the elimination of infection in vector, individuals exposed and infected with TB and malaria and the coinfecting individuals.

7.4. Strategy 4: ($u_1 = u_2 = u_5 \neq 0$)

In the given strategy, we use $u_3 = u_4 = 0$ and $u_1 = u_2 = u_5 \neq 0$ and performed the experiment by optimizing the objective functional Δ . The graphical results associated to this strategy is depicted in Figure 7 and 8. Using the control u_1 which represents human mosquitos elimination by LLITNs, the control variable u_2 which represents the treatment efforts used for malaria infected individuals and the control u_5 which is the efforts of treatment for infected TB individuals to minimize the infections. One can see that this strategy is also helpful for the infection minimizing in the individuals infected with malaria only, TB only, vector population and the individuals dually infected. In comparison to the previous strategy 3, we can see that the number of exposed and infected with malaria decreases and goes to steady state after day 60 and day 80 respectively, which was not observed in the previous strategies. Similarly, the individuals infected with TB only goes to steady states after day 55. The rest of the results are also good for the minimizing infection in the infected and exposed classes.

7.5. Strategy 5: ($u_2 = u_3 = u_4 \neq 0$)

In this proposed strategy, we make the controls $u_1 = u_5 = 0$ and $u_2 = u_3 = u_4 \neq 0$ and performed the simulation by optimizing the objective functional Δ . The graphical results obtained from this strategy is given in Figure 9 and 10. Using The control variable u_2 which represents the treatment efforts used for malaria infected individuals, the control variable u_3 represents IRS which increase the death rate in mosquitos and the control u_4 which represents the prevention and treatment efforts for TB infected individuals to perform the experiment for infection elimination. One can see that the individuals exposed only with malaria very sharply decreases, the population of susceptible vector increases rapidly after day 70, the individuals infected only with TB decreases little and goes to steady state after day 60 and there is no change in the individuals exposed only with TB. The others exposed and infected compartments are decreases little and some good decreases in the dual infections. In comparison to the strategies discussed above this is not a useful strategy for minimizing infection.

7.6. Strategy 6: ($u_1 = u_2 = u_3 = u_4 = u_5 \neq 0$)

Every strategy has its own importance for the disease eliminations, but we have observed especially in strategy 1 and 5 that in some compartments of the infected and exposed individuals there occur a increase and some may posses no change, but we always in search to find such suitable strategy in which the infection in each strategy is the minimum, but this not the cases we presented above. Now, by using all the controls

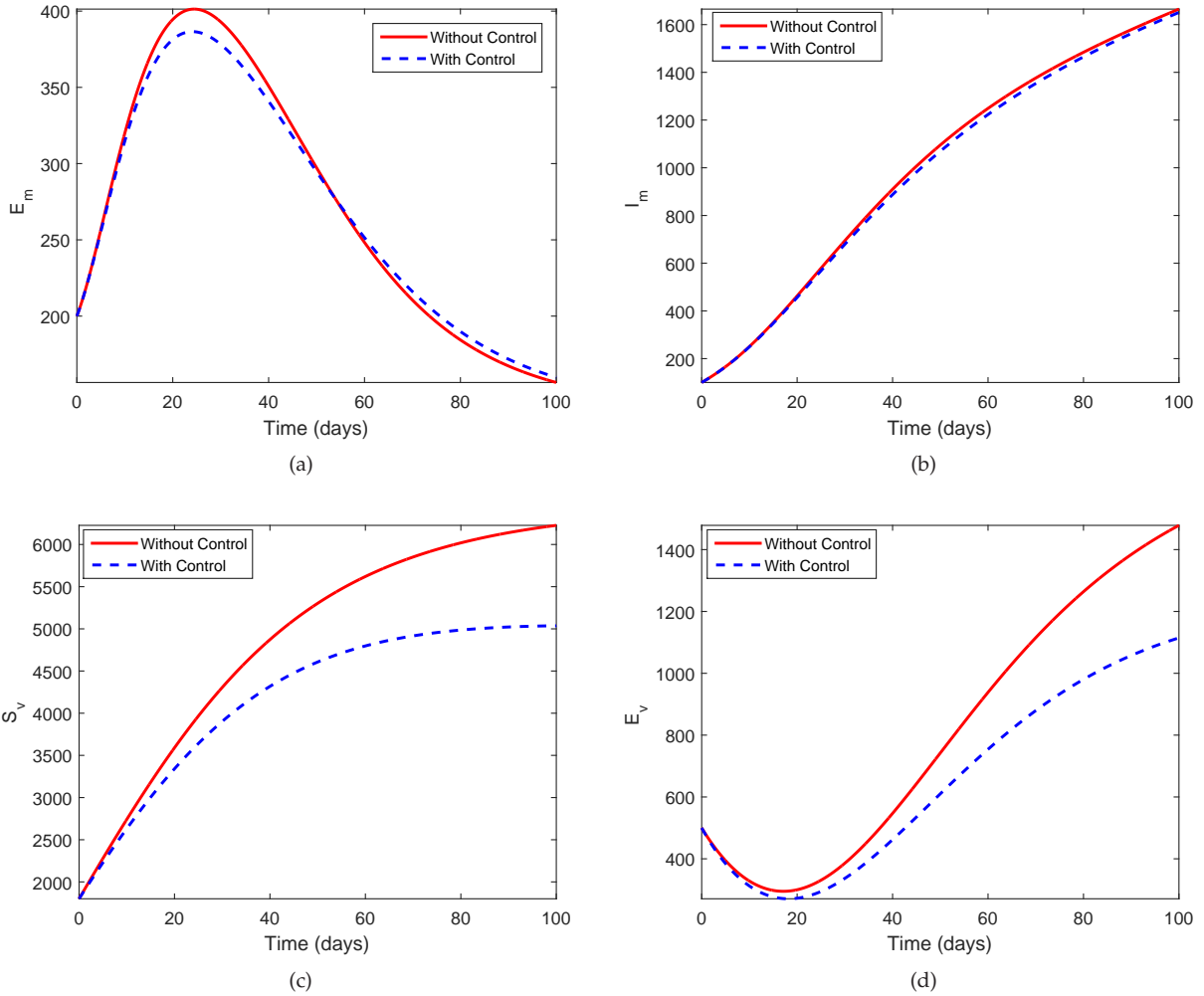


Figure 1: The graphical results for the strategy 1.

active and performing the experiments, one can see that the infection in each class of exposed and infected with TB and malaria only and the dual infection decreases efficiently, see Figure 11 and 12. The use of human mosquitos elimination by LLITNs, the treatment efforts used for malaria infected individuals, increase the death rate in mosquitos by IRS and the prevention and treatment efforts for TB infected individuals and the efforts of treatment for infected TB individuals is the best possible controls for elimination of individuals exposed and infected with TB and malaria only and the dually infected individuals. If we compare the results of strategy 6, with the previous strategies, then, we can say that this strategy is more suitable for the coinfection of TB and malaria.

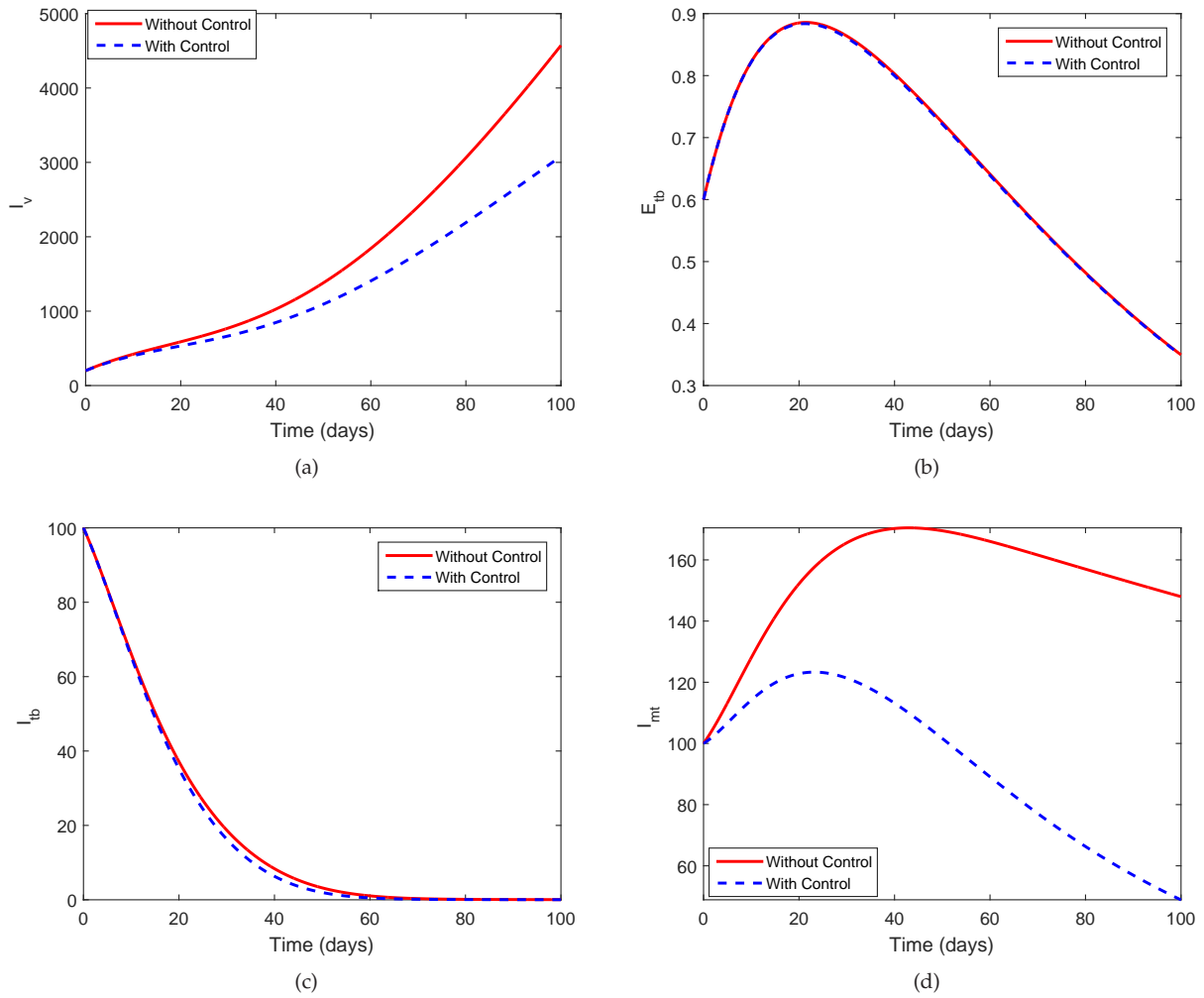


Figure 2: The graphical results for the strategy 1.

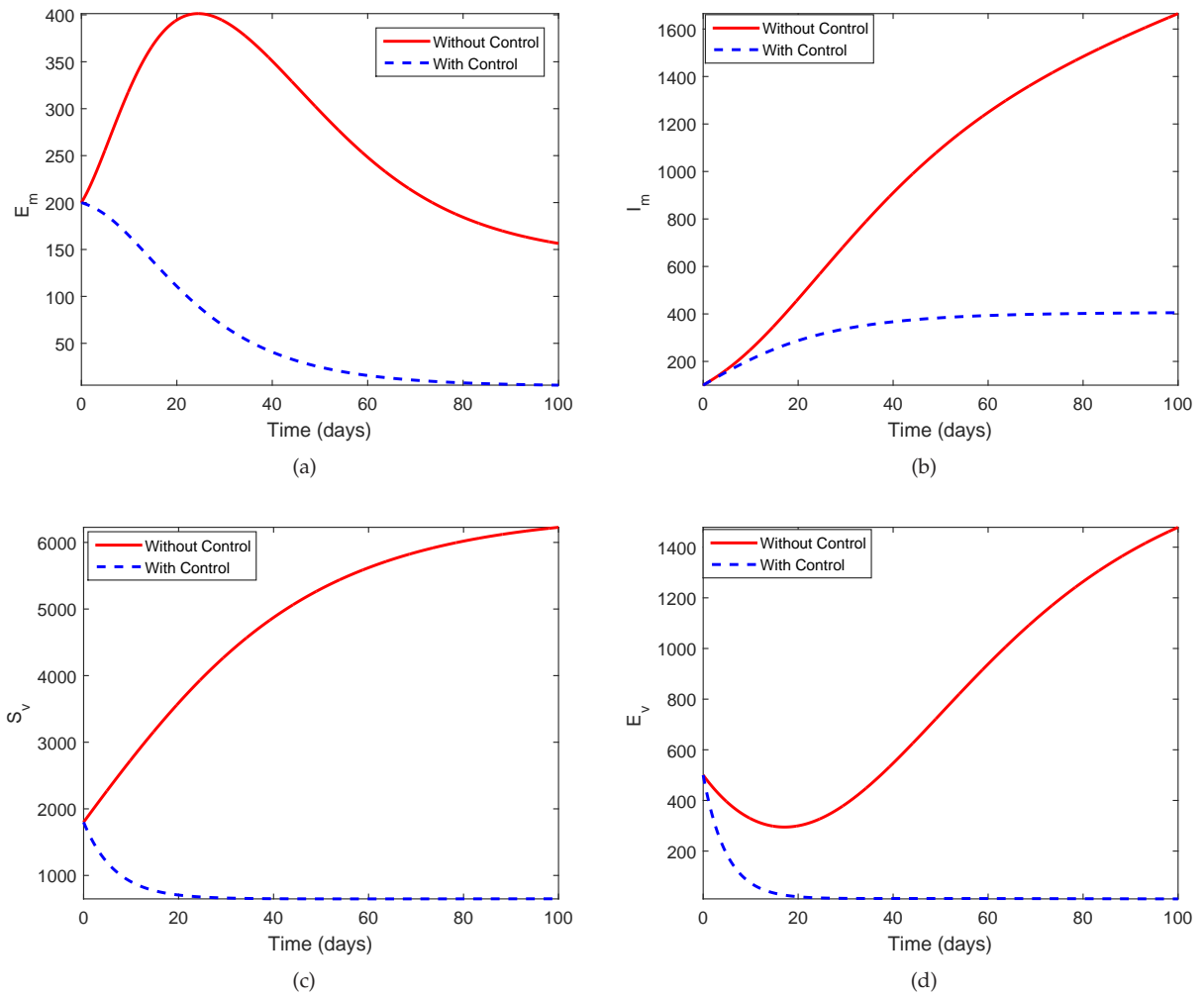


Figure 3: The graphical results for the strategy 2.

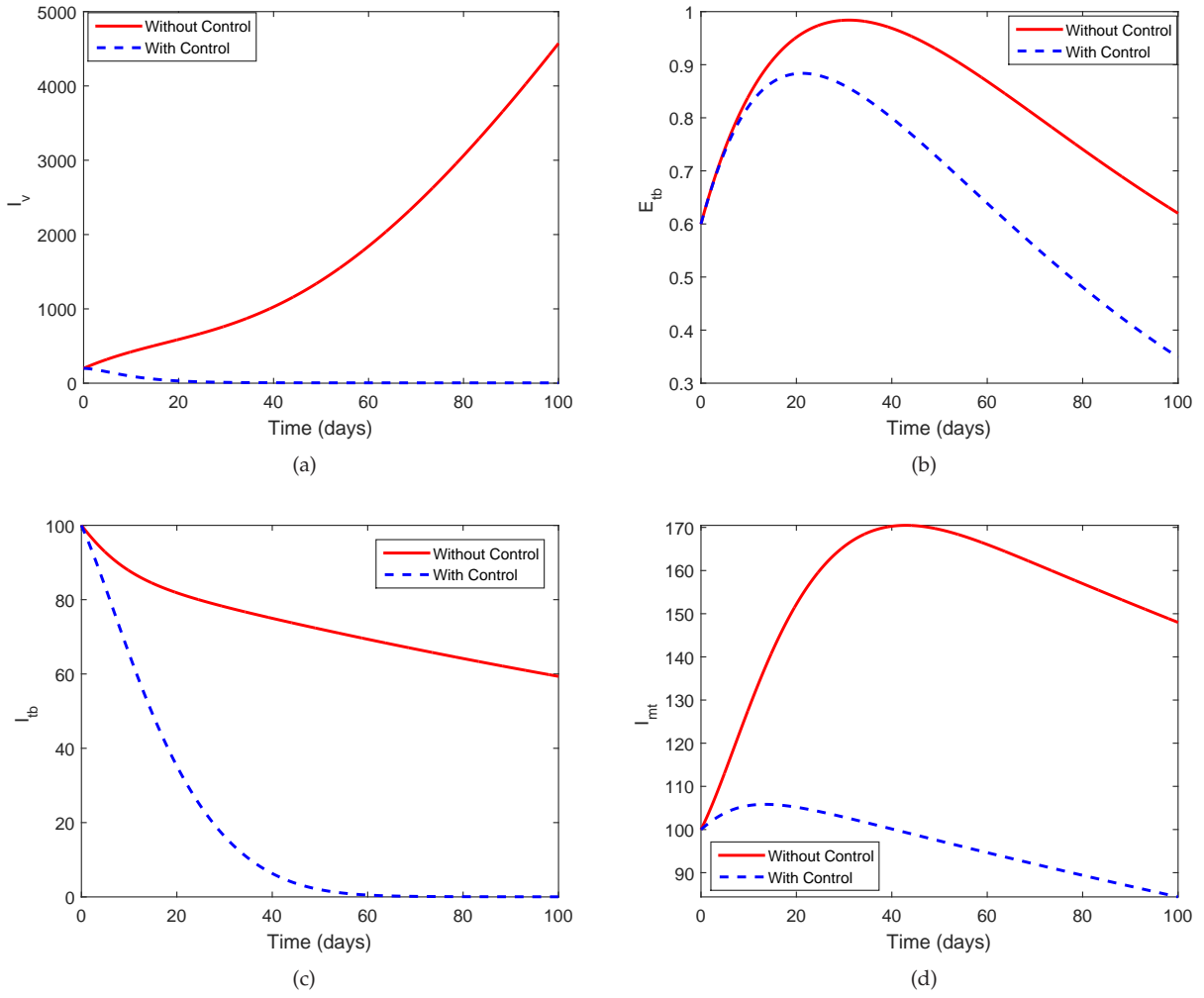


Figure 4: The graphical results for the strategy 2.

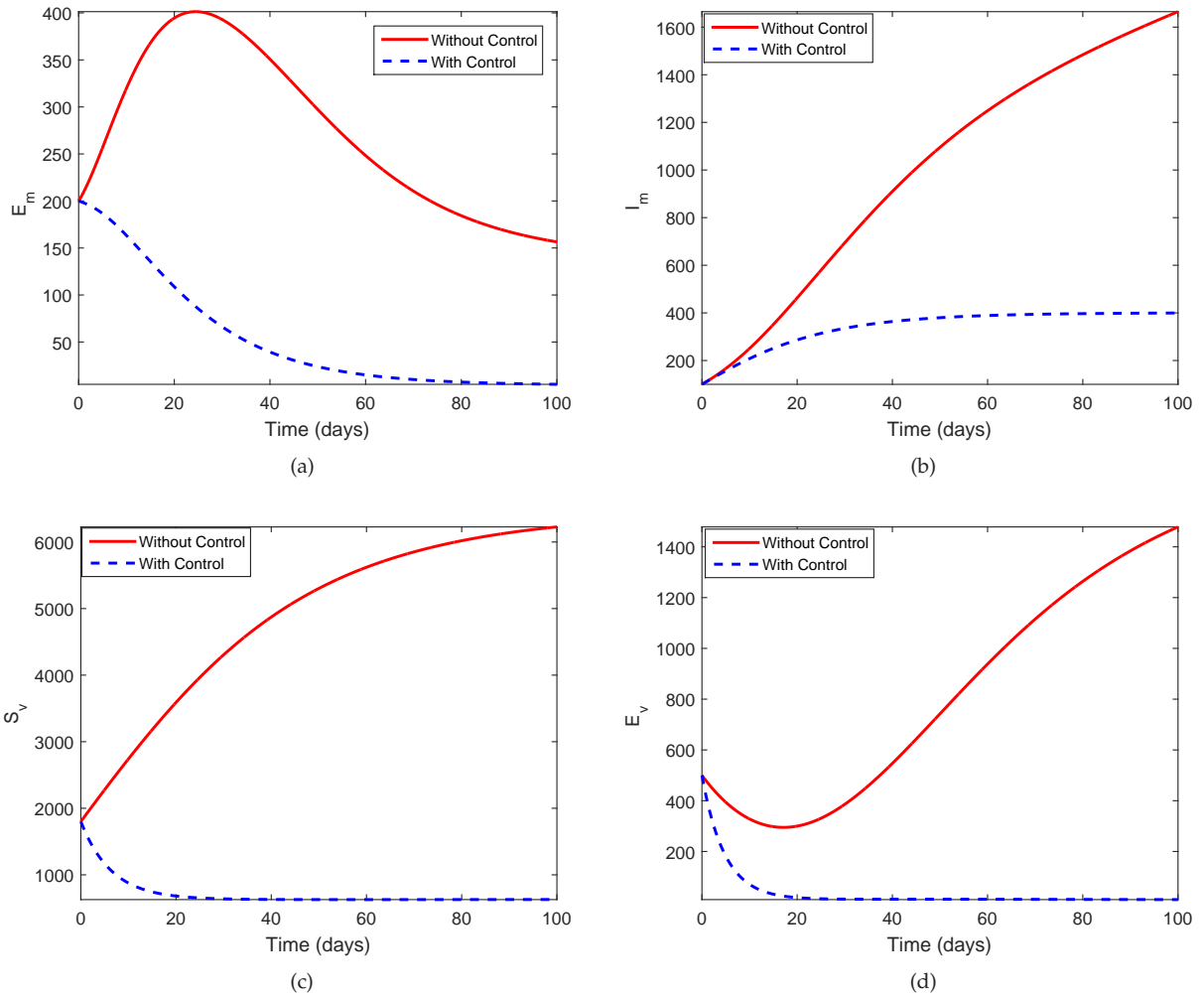


Figure 5: The graphical results for the strategy 3.

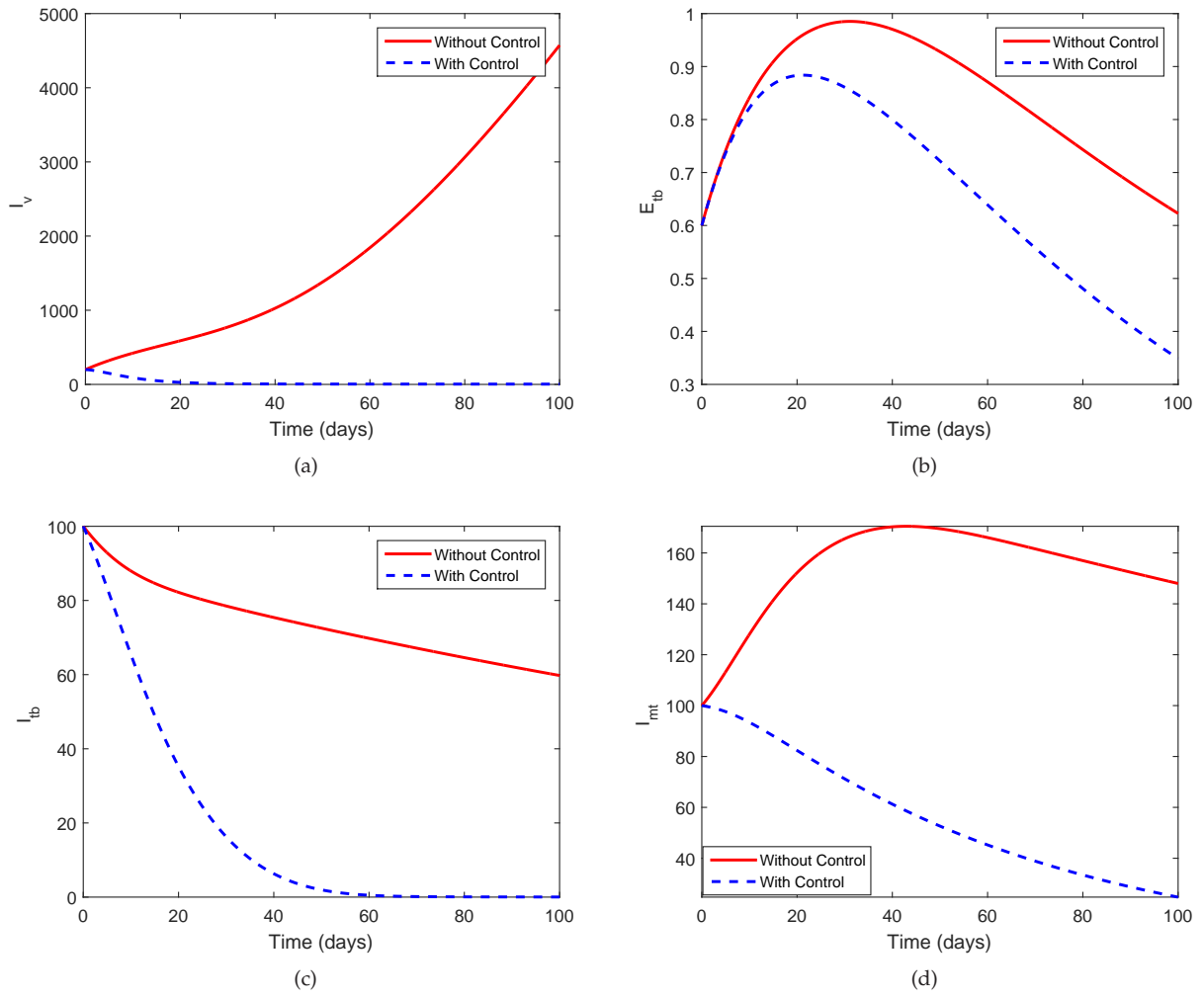


Figure 6: The graphical results for the strategy 3.

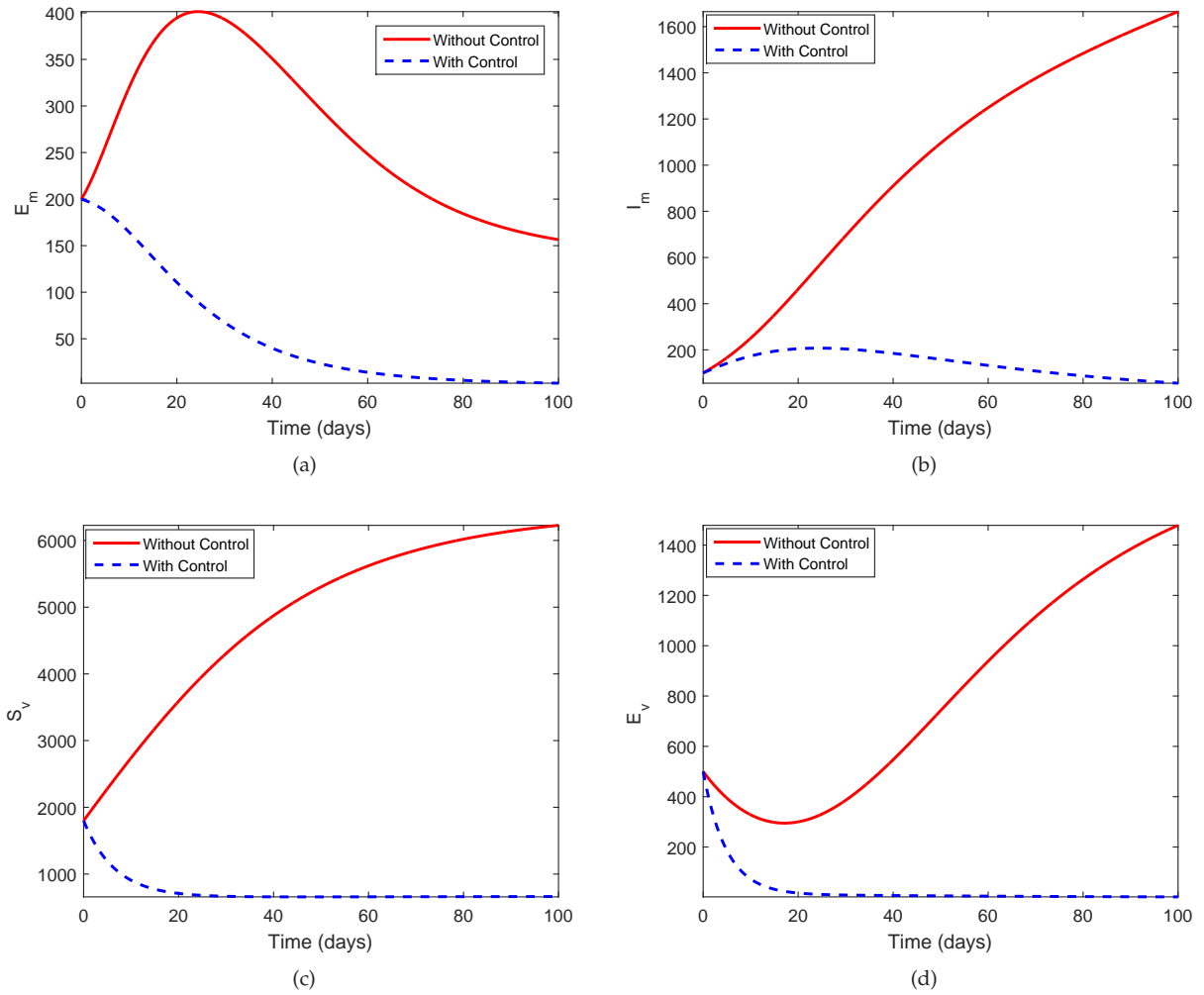


Figure 7: The graphical results for the strategy 4.

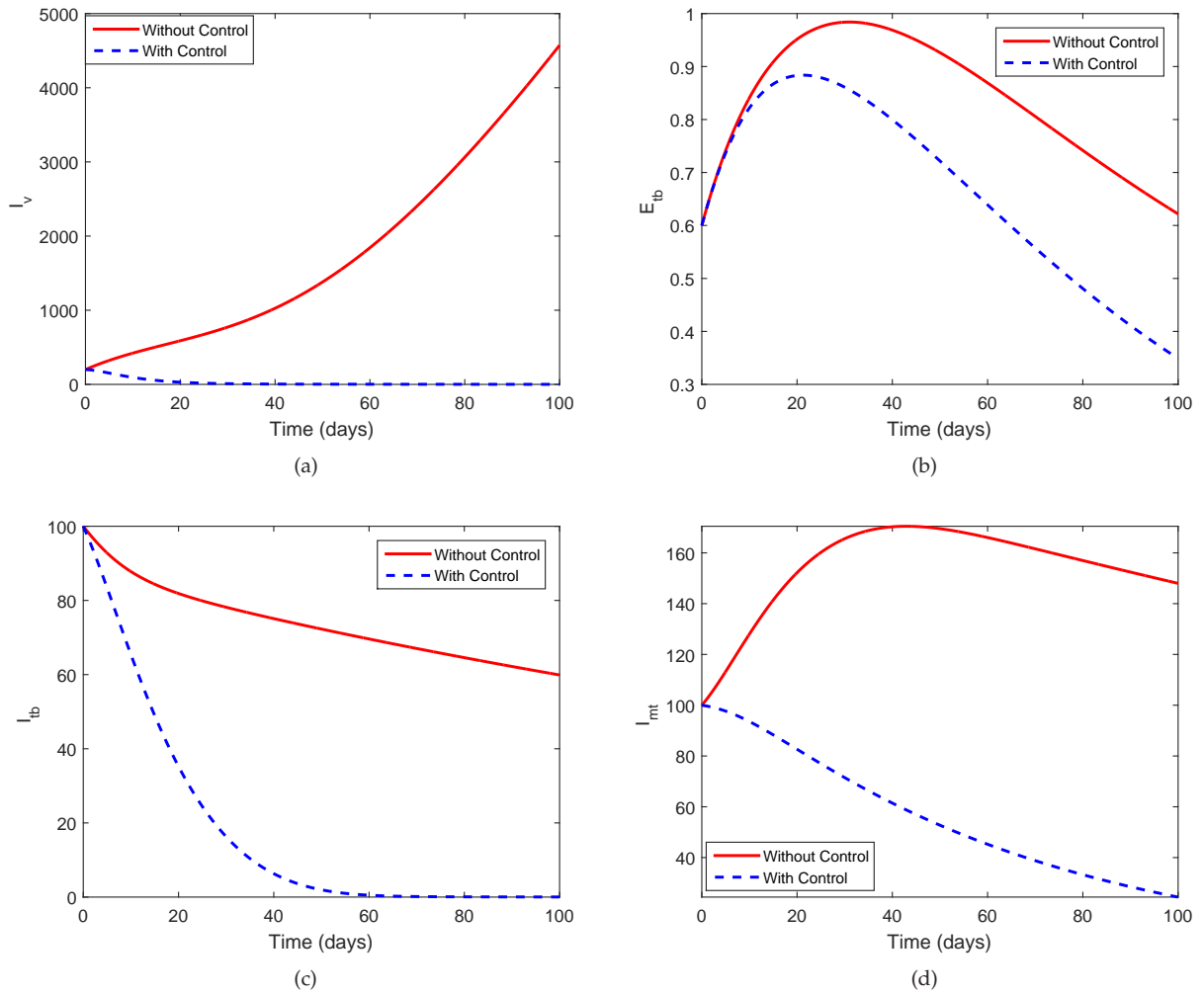


Figure 8: The graphical results for the strategy 4.

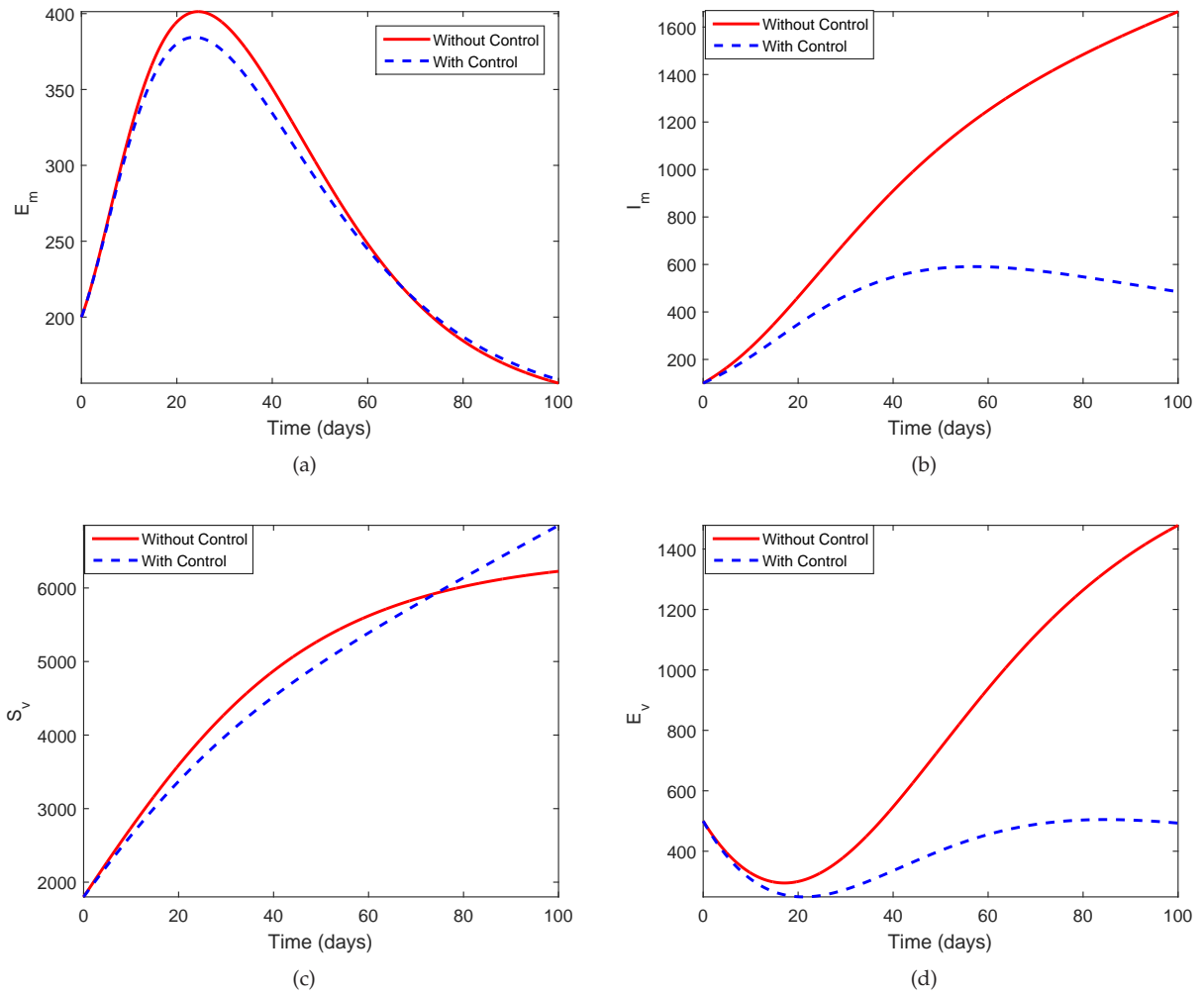


Figure 9: The graphical results for the strategy 5.

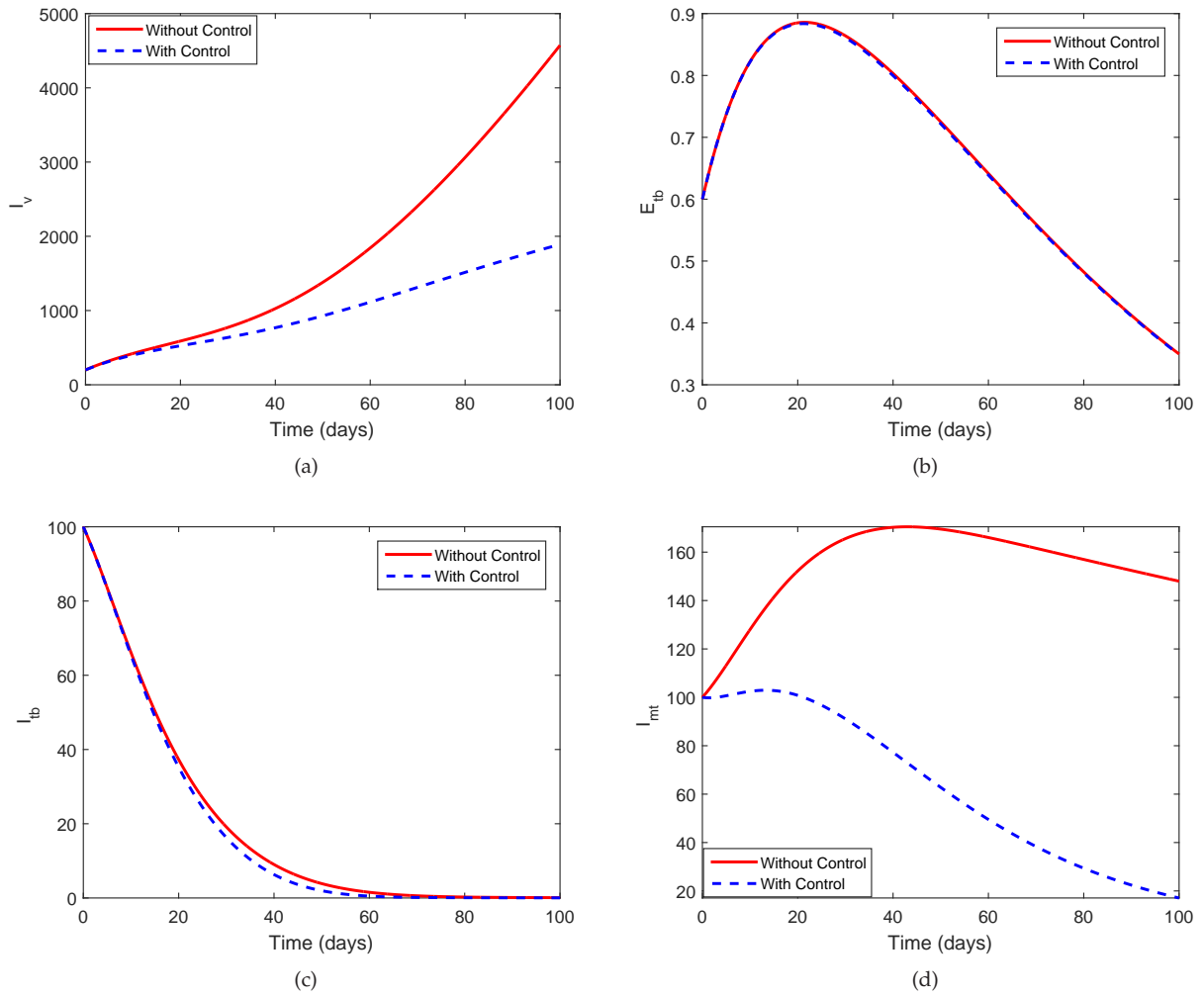


Figure 10: The graphical results for the strategy 5.

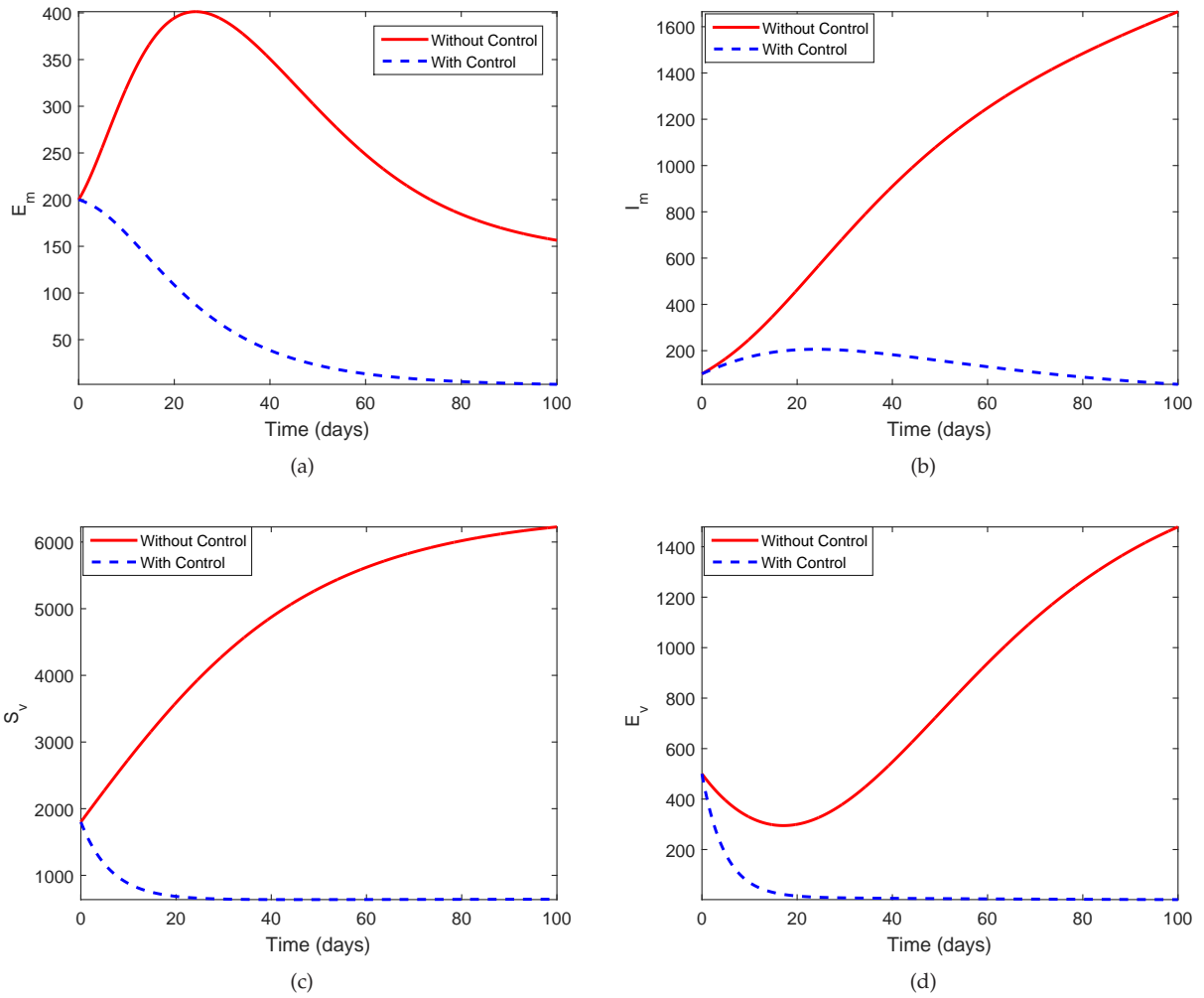


Figure 11: The graphical results for the strategy 6.

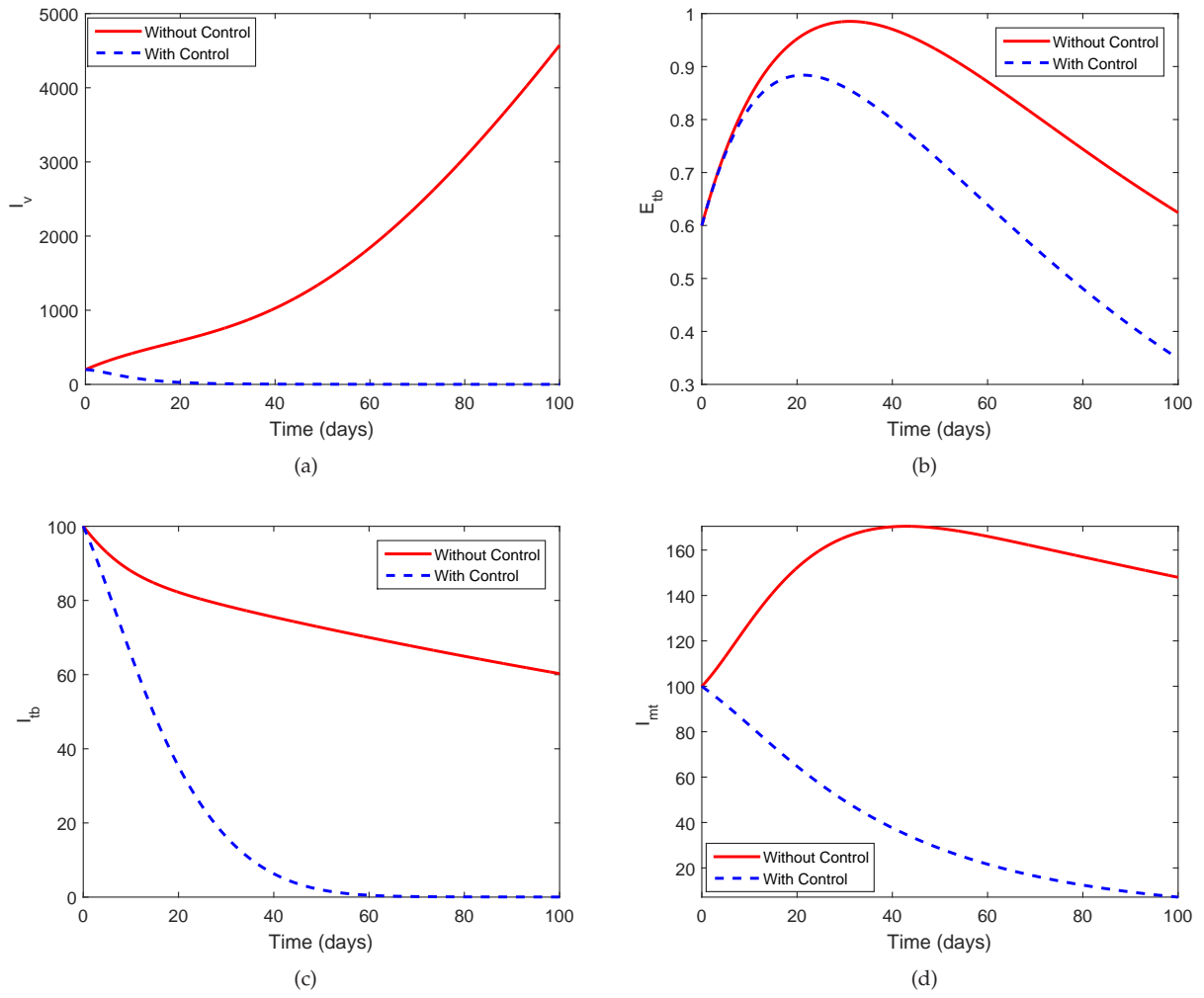


Figure 12: The graphical results for the strategy 6.

8. Conclusion

In the present paper we investigated the coinfection dynamics of the TB and malaria. Both the diseases are severe and causing deaths in the population. The occurring of both the diseases infection in an individual may cause severe infections and its spread in the community is alarming. Therefore, we deeply studied this issue and proposed a model and investigated each model in detailed. The TB infection only model is studied and obtained its basic mathematical results. The TB model at the disease free case is locally asymptotically stable when the basic reproduction number is less than unity. Further, the malaria only model is obtained and discussed their stability analysis. The malaria only model is stable locally asymptotically when the basic reproduction number less than unity. Further, we discussed that the coinfection model is stable locally asymptotically when the basic reproduction number is less than unity. The existence of the bifurcation analysis is studied for the coinfection model and concluded that the model may have a backward bifurcation if the condition given is fulfilled. The model is further used to formulate the optimal control characterization. Five different controls were chosen to optimize the objective functional and obtain the adjoint equations and optimal control characterizations. The chosen controls were, human mosquitos elimination by LLITNs, the treatment efforts used for malaria infected individuals, increase the death rate in mosquitos by IRS, the prevention and treatment efforts for TB infected individuals and the efforts of treatment for infected TB individuals. We performed different control strategies by selecting a set of control variables. Every strategy is performed and the graphical results were discussed and also compared with the previous strategies. Some of the strategies were found not suitable for disease elimination, but some were found suitable for individuals infection elimination in TB only, with malaria only and coinfecting individuals. In all of these there is no good strategies in 1-5, which provided useful results for elimination of infection, then, we finally utilized all the controls and obtained reasonable results and concluded that the strategy 6 is the useful strategy for infection of TB and malaria and their coinfection. This is the pioneer work to explore the dynamics of the TB and malaria and their coinfection and may lead to useful results for public health department and other health authorities.

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Conflict of interests

The authors declare that no competing interests exists regarding the publication of this work.

References

- [1] Kbenesh Blayneh, Yanzhao Cao, and Hee-Dae Kwon. Optimal control of vector-borne diseases: treatment and prevention. *Discrete and Continuous Dynamical Systems B*, 11(3):587–611, 2009.
- [2] Carlos Castillo-Chavez and Baojun Song. Dynamical models of tuberculosis and their applications. *Mathematical biosciences and engineering*, 1(2):361–404, 2004.
- [3] Nakul Chitnis, Jim M Cushing, and JM Hyman. Bifurcation analysis of a mathematical model for malaria transmission. *SIAM Journal on Applied Mathematics*, 67(1):24–45, 2006.
- [4] C Chiyaka, Jean M Tchuente, W Garira, and S Dube. A mathematical analysis of the effects of control strategies on the transmission dynamics of malaria. *Applied Mathematics and Computation*, 195(2):641–662, 2008.
- [5] Senelani D Hove-Musekwa et al. Determining effective spraying periods to control malaria via indoor residual spraying in sub-saharan africa. *Advances in Decision Sciences*, 2008, 2008.
- [6] Vangipuram Lakshmikantham, Srinivasa Leela, and Anatoly A Martynuk. *Stability analysis of nonlinear systems*. Springer, 1989.
- [7] Abhishek Mallela, Suzanne Lenhart, and Naveen K Vaidya. Hiv–tb co-infection treatment: Modeling and optimal control theory perspectives. *Journal of Computational and Applied Mathematics*, 307:143–161, 2016.
- [8] Expeditho Mtisi, Herieth Rwezaura, and Jean Michel Tchuente. A mathematical analysis of malaria and tuberculosis co-dynamics. *Discrete & Continuous Dynamical Systems-B*, 12(4):827–864, 2009.

- [9] Ann-Kristin Mueller, Jochen Behrends, Kristine Hagens, Jacqueline Mahlo, Ulrich E Schaible, and Bianca E Schneider. Natural transmission of *Plasmodium berghei* exacerbates chronic tuberculosis in an experimental co-infection model. *PLoS One*, 7(10):e48110, 2012.
- [10] Zindoga Mukandavire, Abba B Gumel, Winston Garira, and Jean Michel Tchuente. Mathematical analysis of a model for hiv-malaria co-infection. 2009.
- [11] Peter Mpasho Mwamtobe, Simphiwe Mpumelelo Simelane, Shirley Abelman, and Jean Michel Tchuente. Optimal control of intervention strategies in malaria-tuberculosis co-infection with relapse. *International Journal of Biomathematics*, 11(02):1850017, 2018.
- [12] Kazeem O Okosun, Rachid Ouifki, and Nizar Marcus. Optimal control analysis of a malaria disease transmission model that includes treatment and vaccination with waning immunity. *Biosystems*, 106(2-3):136–145, 2011.
- [13] KO Okosun, MA Khan, E Bonyah, and ST Ogunlade. On the dynamics of hiv-aids and cryptosporidiosis. *The European Physical Journal Plus*, 132(8):363, 2017.
- [14] World Health Organization. *World malaria report 2015*. World Health Organization, 2016.
- [15] World Health Organization et al. *Global tuberculosis report 2016*. 2016.
- [16] Kathleen R Page, Anne E Jedlicka, Benjamin Fakheri, Gregory S Noland, Anup K Kesavan, Alan L Scott, Nirbhay Kumar, and Yukari C Manabe. Mycobacterium-induced potentiation of type 1 immune responses and protection against malaria are host specific. *Infection and immunity*, 73(12):8369–8380, 2005.
- [17] Evelyn C Pielou et al. An introduction to mathematical ecology. *An introduction to mathematical ecology*, 1969.
- [18] L Renia and SM Potter. Co-infection of malaria with hiv: an immunological perspective. *Parasite immunology*, 28(11):589–595, 2006.
- [19] Cherise P Scott, Nirbhay Kumar, William R Bishai, and Yukari C Manabe. Modulation of mycobacterium tuberculosis infection by *Plasmodium* in the murine model. *The American journal of tropical medicine and hygiene*, 70(2):144–148, 2004.
- [20] Oluwaseun Sharomi, C Podder, A Gumel, and Baojun Song. Mathematical analysis of the transmission dynamics of hiv/tb coinfection in the presence of treatment. *Mathematical Biosciences and Engineering*, 5(1):145, 2008.
- [21] Pauline Van den Driessche and James Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences*, 180(1-2):29–48, 2002.
- [22] Marco Vitoria, Reuben Granich, Charles F Gilks, Christian Gunneberg, Mehran Hosseini, Wilson Were, Mario Raviglione, and Kevin M De Cock. The global fight against hiv/aids, tuberculosis, and malariacurrent status and future perspectives. *American journal of clinical pathology*, 131(6):844–848, 2009.
- [23] Hyun M Yang. A mathematical model for malaria transmission relating global warming and local socioeconomic conditions. *Revista de saude publica*, 35:224–231, 2001.
- [24] A. Duro, V. Piccione, M.A. Ragusa, V. Veneziano, New Environmentally Sensitive Patch Index - ESPI - for MEDALUS protocol, AIP Conference Proceedings, 1637, 305–312, 2014.
- [25] A. Cuspilici, P. Monforte, M.A. Ragusa, Study of Saharan dust influence on PM10 measures in Sicily from 2013 to 2015, *Ecological Indicators*, 76, 297–303, 2017.