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Research Article

Modeling the Effects of Helminth Infection on the Transmission Dynamics of Mycobacterium tuberculosis under Optimal Control Strategies

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A deterministic mathematical model for the transmission and control of cointeraction of helminths and tuberculosis is presented, to examine the impact of helminth on tuberculosis and the effect of control strategies. The equilibrium point is established, and the effective reproduction number is computed. The disease-free equilibrium point is confirmed to be asymptotically stable whenever the effective reproduction number is less than the unit. The analysis of the effective reproduction number indicates that an increase in the helminth cases increases the tuberculosis cases, suggesting that the control of helminth infection has a positive impact on controlling the dynamics of tuberculosis. The possibility of bifurcation is investigated using the Center Manifold Theorem. Sensitivity analysis is performed to determine the effect of every parameter on the spread of the two diseases. The model is extended to incorporate control measures, and Pontryagin’s Maximum Principle is applied to derive the necessary conditions for optimal control. The optimal control problem is solved numerically by the iterative scheme by considering vaccination of infants for Mtb, treatment of individuals with active tuberculosis, mass drug administration with regular antihelminthic drugs, and sanitation control strategies. The results show that a combination of educational campaign, treatment of individuals with active tuberculosis, mass drug administration, and sanitation is the most effective strategy to control helminth-Mtb coinfection. Thus, to effectively control the helminth-Mtb coinfection, we suggest to public health stakeholders to apply intervention strategies that are aimed at controlling helminth infection and the combination of vaccination of infants and treatment of individuals with active tuberculosis.

1. Introduction

Soil-transmitted helminth infections are common infections that affect poor communities of the world. The species that infect people are the roundworm (Ascaris lumbricoides), the whipworm (Trichuris trichiura), and the hookworm (Necator americanus). These parasites reside in the intestine and release eggs to contaminate the soil [1]. Approximately 1.5 billion people are infected with soil-transmitted helminths worldwide which are widely distributed in tropical and sub-tropical areas, with the greatest numbers occurring in sub-Saharan Africa, America, China, and East Asia [1]. On the other hand, tuberculosis (TB) is an infectious disease that is caused by the bacillus Mycobacterium tuberculosis (Mtb). Approximately 5-10% of the estimated 1.7 billion people infected with Mtb develop active TB during their lifetime.
[2]. The study by Watts et al. [3] suggests that infection by *trichuriasis* reduces the chance of latent tuberculosis (LTB) or blunts tuberculin skin tests (TST). Moreover, coinfection of helminth and Mtb has been a public health issue in developing countries [4]. The study by Dias et al. [5] suggests that intestinal helminth infection has a harmful effect to contain the Mtb infection and contributes to the development of active TB for coinfected individuals. Furthermore, coinfection with helminths increases the susceptibility to Mtb [6].

The usefulness of mathematical modeling for understanding and assessing the dynamics of infectious diseases cannot be underrated and has been used over the years to investigate the optimal mechanisms for intervention strategies in controlling the spread of infectious diseases. The transmission and dynamics of Mtb with other diseases have been studied by many researchers [7–9]. Bhunu et al. [7] developed an HIV/AIDS and TB coinfection model that considers the treatment of exposed and active TB individuals and therapy for AIDS. They found that the treatment of exposed and active forms of TB results in stuck commencement of the AIDS stage of HIV infection. Fatmawati and Tasman [8] proposed an optimal control model for the transmission of TB-HIV coinfection. They found that the best and effective strategy to lower the TB-HIV infection is to apply a combination of anti-TB and ARV treatment. Okosun [9] discussed the synergy between malaria and schistosomiasis in the presence of treatment. Their study revealed that an effective control of malaria could be achieved if efficient treatment and prevention of schistosomiasis are implemented. There is a need for modeling coinfection of infectious diseases in areas with neglected tropical diseases (NTD) which include helminth infections, Mtb, and malaria to list a few [10], due to their geographical overlap. To the best of our understanding, no study has been carried out to consider the helminth-Mtb coinfection with the use of optimal control theory.

In this paper, our emphasis is to propose a compartmental system for the codynamics of helminths and Mtb diseases and to explore the effect of helminths on Mtb and vice versa. The model is extended to incorporate four time-dependent controls, namely, educational campaign to sensitize the parents to take their babies to the Bacille Calmette-Guerin (BCG) vaccine clinic; treatment of individuals with active TB; treatment of individuals infected with helminth parasites through mass drug administration (MDA); and sanitation to lessen the ingestion rate of parasites from the polluted environment.

## 2. Materials and Methods

### 2.1. Model Formulation

The helminth-Mtb infection model is formulated under the following model assumptions:

(i) The susceptible individuals cannot simultaneously get infected by both diseases but rather get infected with one disease and later by the other disease

(ii) The population is homogeneously mixed

(iii) The incidence rate for helminth infection follows the logistic model while Mtb is assumed to be frequency-dependent

(iv) An individual gets helminth infection after sufficient contact with the polluted environment

(v) Due to the fact that Mtb infection is more severe than helminth infection, we assume that coinfect ed individuals seek TB treatment rather than helminth infection

(vi) The Bacille Calmette-Guerin (BCG) vaccine does not confer a total immunity

The model is formulated by considering two populations, namely, the human population and the parasite population \( M \) that are present in the contaminated environment. The human population is subdivided into nine classes, namely, susceptible \( S \), the vaccinated against Mtb \( V_T \), those infected with Mtb but do not show clinical symptoms of TB \( E_T \), those with active TB \( I_T \), TB-recovered individuals \( R_T \), those infected by helminths \( I_H \), those who have recovered from helminths \( R_H \), and those who are dually infected by helminths and Mtb \( I_{HT} \). Thus, the total human population is given by \( N = S + V_T + E_T + I_T + I_H + I_{HT} + R_T + R_H + R_{HT} \). The susceptible population increases by recruitment through birth at the rate \((1 - \eta)bn\) and by those who lose their temporary immunity against the helminths and Mtb at rates \( \gamma_1 \) and \( \gamma_2 \), respectively. The susceptible individuals get infected with Mtb upon sufficient contact with an infectious individual with the force of infection \( \lambda_T = (\beta_T(I_T + \sigma I_{HT}))/N \), where \( \beta_T \) is the transmission rate and \( \sigma > 1 \) is the modification parameter for coinfected individuals that models infectivity for susceptible individuals. The susceptible individuals also get infected by helminths after ingestion of eggs/larvae or skin penetration by infective larvae with the force of infection \( \lambda_H = \beta_HM/K + M \), where \( \beta_H \) is the ingestion rate of parasitic worms and \( K \) is the density of parasites in the soil. The helminth-infected individuals increase due to susceptible individuals being infected with helminths, Mtb-vaccinated individuals with helminths, and coinfected individuals who recover from Mtb at the rate \( \tau_2 \). Furthermore, due to the fact that the BCG vaccine does not grant total protection [11], then individuals exposed to Mtb increase due to Mtb-vaccinated individuals losing their protection against Mtb and getting infected at the rate \( \rho\lambda_T \), with \( \rho \in (0, 1) \) such that \( 1 - \rho \) is the efficacy of the vaccine.

The chronic infection by helminths alters the chance of getting infected and the progression of coinfected parasites (virus or bacteria) [12]; as a result, individuals infested with helminths are at higher risk of developing tuberculosis [13]. Therefore, individuals coinfected with helminth and Mtb are increased by helminth-infected individuals being infected with Mtb at the force of infection \( g\lambda_T \) where \( g > 1 \) is the modification parameter that models increased susceptibility to Mtb, and Mtb-exposed
individuals getting infected by helminths at a force of infection $\lambda_H$. In addition, the number of individuals with active TB increases as individuals progressed from the exposed class at the rate and coinfections recovered from helminth infection at the rate $r_T$ and decreases due to natural recovery at the rate $e_i$. The parasite populations in the environment as they are released by the helminth-infected individuals at the rate $e_i$ and coinfectd individuals at the rate $e_2$ and are cleared from the environment at a rate $\mu_M$. The description above is illustrated in Figure 1, and the governing system of nonlinear differential equations is shown in model (1). The model parameters are described in Table 1.

$$\frac{dS}{dt} = (1-\eta)bN + y_1 R_H + y_2 R_T - (\mu + \lambda_H + \lambda_I)S,$$

$$\frac{dV_T}{dt} = \eta bN - (\mu + \rho \lambda_T + \lambda_I)V_T,$$

$$\frac{dE_T}{dt} = \lambda_T S + \rho \lambda_T V_T + \lambda_I R_T - (\mu + k + \lambda_H)E_T,$$

$$\frac{dI_T}{dt} = kE_T + \tau_I I_{HT} - (\mu + d_T + \tau_2 + \lambda_I)I_T,$$

$$\frac{dH_T}{dt} = \lambda_H S + \lambda_H V_T - \lambda_I R_T - (\mu + d_H + \tau_1 + \epsilon_1)I_T,$$

$$\frac{dI_{HT}}{dt} = g\lambda_T I_T + \lambda_H (E_T + I_T) - (\mu + d_{HT} + \tau_1 + \tau_2 + \epsilon_2)I_{HT},$$

$$\frac{dR_T}{dt} = \tau_T I_T - (\lambda_H + \mu + \gamma_I)R_T,$$

$$\frac{R_H}{dt} = \tau_I i_T - (\lambda_H + \mu + \gamma_I)R_H,$$

$$\frac{dM}{dt} = e_i I_H + e_2 I_{HT} - \mu_M M, \quad (1)$$

subject to condition $s + v_T + e_T + i_T + i_H + i_{HT} + r_T + r_H = 1$.

2.2. Disease-Free Equilibrium (DFE). The DFE point of system (4) is obtained by setting the right-hand sides of the equations to zero with $c_T = 0, i_T = 0, i_H = 0, r_T = 0, r_H = 0, r_{HT} = 0$, and $m = 0$ to obtain the DFE $\mathcal{E}_0 = (1-\eta, \eta, 0, 0, 0, 0, 0, 0, 0, 0)$.

2.3. Effective Reproduction Number. The principles of the next-generation matrix [22] are used to obtain the effective reproduction number.

$$R_T = \frac{\beta_T k (1 + \eta (\rho - 1))}{(b + k)(b + d_T + \tau_2)}, \quad (5)$$

$$R_H = \frac{\beta_H \epsilon_1}{\mu_M (b + d_H + e_2 + \tau_1)}, \quad (6)$$

corresponding to the reproduction numbers for the Mtb and helminth infection transmission models obtained from the positive eigenvalues of the next-generation matrix, respectively.
2.4. Local Stability of the Disease-Free Equilibrium

**Theorem 1.** The disease-free equilibrium point is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if either $\mathcal{R}_T > 1$ or $\mathcal{R}_H > 1$.

**Proof.** We first obtain the Jacobian matrix at the disease-free equilibrium $E_0$ in order to prove this theorem. The Jacobian matrix is given by

$$J_{E_0} =
\begin{bmatrix}
-b & 0 & 0 & l_4 & -l_1 & -l_1 & y_1 & y_1 & -\beta_H s_0 \\
0 & -b & 0 & (\sigma \beta_T - d_T) s_0 & -l_1 & -l_1 & 0 & 0 & -\beta_H v_{T0} \\
0 & 0 & -(b + k) & \beta_T s_0 + \mu s_0 & -l_3 & 0 & 0 & 0 & 0 \\
0 & 0 & k & -l_2 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -l_2 & 0 & 0 & 0 & 0 \\
0 & 0 & \tau_2 & 0 & 0 & -l_2 & 0 & 0 & 0 \\
0 & 0 & 0 & \tau_1 & 0 & 0 & -l_4 & 0 & 0 \\
0 & 0 & 0 & \tau_1 & 0 & 0 & -l_3 & 0 & 0 \\
0 & 0 & 0 & \epsilon_1 & \epsilon_2 & 0 & 0 & -\mu_M & 0 \\
\end{bmatrix}
$$

where

$$l_0 = (d_T - \beta_T) s_0, \\
l_1 = (d_H + \epsilon_1) s_0, \\
l_2 = (\sigma \beta_T - (d_{HT} + \epsilon_2)) s_0, \\
l_3 = (d_{HT} + \epsilon_1) v_{T0}, \\
l_4 = (\rho \sigma \beta_T - (d_{HT} + \epsilon_2)) v_{T0}, \\
l_5 = (b + d_T + \tau_2), \\
l_6 = (b + d_H + \tau_1), \\
l_7 = (b + \tau_1 + \tau_2 + \epsilon_2 + d_{HT}), \\
l_8 = (b + y_2), \\
l_9 = (b + y_1).
$$

Now, the characteristic polynomial of the Jacobian matrix $J_{E_0}$ is given by

![Compartmental diagram for the helminth and Mycobacterium tuberculosis coinfection. The dotted lines indicate the interaction of individuals with the polluted environment.](image-url)
Table 1: Description of the model parameters.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Definition</th>
<th>Estimated value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>$b$</td>
<td>Natural birth rate</td>
<td>1/18250 day$^{-1}$</td>
<td>[14]</td>
</tr>
<tr>
<td>$\beta_T$</td>
<td>Transmission rate for Mtb</td>
<td>0.42 day$^{-1}$</td>
<td>[15]</td>
</tr>
<tr>
<td>$\mu$</td>
<td>Natural death rate</td>
<td>$1/(60 \times 365)$ day$^{-1}$</td>
<td>[16]</td>
</tr>
<tr>
<td>$\gamma_1$</td>
<td>Loss of temporary immunity for helminth-recovered individuals</td>
<td>$1/70$ day$^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\beta_H$</td>
<td>Ingestion rate of parasitic worms</td>
<td>1 parasite day$^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\rho$</td>
<td>Vaccine wane rate</td>
<td>0.7 dimensionless</td>
<td>[17]</td>
</tr>
<tr>
<td>$\tau_1$</td>
<td>Natural recovery rate for helminth-infected individuals</td>
<td>1/28 day$^{-1}$</td>
<td>[18]</td>
</tr>
<tr>
<td>$d_H$</td>
<td>Helminth disease-induced death rate</td>
<td>35/1000 day$^{-1}$</td>
<td>[14]</td>
</tr>
<tr>
<td>$\gamma_2$</td>
<td>Loss of temporary immunity for Mtb-recovered individuals</td>
<td>0.3 day$^{-1}$</td>
<td>[19]</td>
</tr>
<tr>
<td>$\tau_2$</td>
<td>Natural recovery rate for Mtb-infected individuals</td>
<td>0.2/365 day$^{-1}$</td>
<td>[20]</td>
</tr>
<tr>
<td>$d_T$</td>
<td>Mtb disease-induced death rate</td>
<td>0.08 day$^{-1}$</td>
<td>[17]</td>
</tr>
<tr>
<td>$d_HT$</td>
<td>Helminth-Mtb disease-induced death rate</td>
<td>0.08 day$^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$k$</td>
<td>Progression to active TB</td>
<td>0.00013/365 day$^{-1}$</td>
<td>[21]</td>
</tr>
<tr>
<td>$\mu_M$</td>
<td>Clearance rate of parasitic worms</td>
<td>13/37500 day$^{-1}$</td>
<td>[14]</td>
</tr>
<tr>
<td>$\epsilon_1$</td>
<td>Shading rate for helminth-infected individuals</td>
<td>0.09 day$^{-1}$</td>
<td>[18]</td>
</tr>
<tr>
<td>$K$</td>
<td>Number of parasites in the environment</td>
<td>$10^5$ parasites</td>
<td>[18]</td>
</tr>
<tr>
<td>$\epsilon_2$</td>
<td>Shading rate for coinfected individuals</td>
<td>0.1 day$^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$g$</td>
<td>Modification parameter</td>
<td>1.12 dimensionless</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>Modification parameter</td>
<td>1.5 dimensionless</td>
<td>Assumed</td>
</tr>
</tbody>
</table>

Thus, the DFE point $\mathcal{E}_0$ is locally asymptotically stable whenever $\mathcal{R}_T < 1$ and unstable if either $\mathcal{R}_T > 1$ or $\mathcal{R}_H > 1$.

2.5. Global Stability of the Disease-Free Equilibrium. We investigate the global asymptotic stability of the disease-free equilibrium of model (9) by using the theory applied by Chavez et al. [24]. System (9) is written as

$$
\frac{dX}{dt} = F(X, Y),
$$

$$
\frac{dY}{dt} = G(X, Y), G(X, 0) = 0,
$$

where $X = (s, v_T, r_T, r_H) \in \mathbb{R}^4$ represents the number of uninfected individuals and $Y = (\epsilon_T, i_T, i_H, i_{HT}, m) \in \mathbb{R}^5$ represents the number of infected individuals including the latent and infectious individuals.

The DFE is given by $\mathcal{E}_0 = (X_0^*, 0)$, where $X_0^* = (1 - \eta, \eta)$. The conditions in equation (12) must be satisfied to guarantee local asymptotic stability:

$$
\lambda_1^2 + (2b + d_H + \epsilon_1 + \mu_M + \tau_1)\lambda + (b + d_H + \epsilon_1 + \mu_M + \tau_1) = 0,
$$

$$
\lambda_2 = -(b + d_H + \epsilon_1 + \mu_M + \tau_1) + \sqrt{\left[(b + d_H + \epsilon_1 + \mu_M + \tau_1)^2 - 4(b + d_H + \epsilon_1 + \mu_M + \tau_1)(\lambda + \mu_M + \tau_1)\right]}.
$$

Clearly, from equation (9), the eigenvalues of $J_{\mathcal{E}_0}$ are as follows: $\lambda_1 = -b < 0$, $\lambda_2 = -(b + \gamma_1) < 0$, $\lambda_3 = -(b + \gamma_2) < 0$, $\lambda_4 = -(b + d_H + \tau_1 + \tau_2 + \epsilon_2) < 0$, and

$$
\lambda_5 = -(b + d_H + \tau_1 + \tau_2 + \epsilon_2) + \sqrt{\left[\left(-(b + d_H + \tau_1 + \tau_2 + \epsilon_2)\right)^2 - 4\left(-(b + d_H + \tau_1 + \tau_2 + \epsilon_2)\right)(\lambda + \mu_M + \tau_1)\right]} = 0.
$$

Applying Routh-Hurwitz conditions [23] on equation (10), it has strictly negative real roots if $\mathcal{R}_T < 1$ and $\mathcal{R}_H < 1$.\[\text{computational and mathematical methods in medicine}\]
\[ \frac{dX}{dt} = F(X^*, 0), \quad X^* \text{ is globally asymptotically stable (g.a.s)}, \]
\[ G(X, Y) = BY - \bar{G}(X, Y), \quad \text{where } \bar{G}(X, Y) \geq 0 \text{ for } (X, Y) \in \Sigma, \]
(12)
and \( B = D_x \bar{G}(X^*, 0) \) is M-matrix (that is, the off-diagonal elements of matrix \( B \) are nonnegative); and \( \Sigma \) is the region where the model makes biological sense. If the equations in (4) satisfy the conditions in (12), then the following theorem holds.

**Theorem 2.** The fixed point \( \mathcal{E}_o \) is a globally asymptotically stable equilibrium of system (4) given that \( \mathcal{R}_{HT} < 1 \) and that the conditions in (12) are fulfilled.

\[ \begin{pmatrix}
\bar{g}_1(X, Y) \\
\bar{g}_2(X, Y) \\
\bar{g}_3(X, Y) \\
\bar{g}_4(X, Y) \\
\bar{g}_5(X, Y)
\end{pmatrix} = 
\begin{pmatrix}
\chi_1 \lambda_T^* \left( 1 + \frac{s + \rho \nu y}{\sigma} \right) - \lambda_T^* r_H + \lambda_T^* e_T - d_T^* e_T - \chi_2^* h e_T - \chi_3^* s h e_T \\
\lambda_T^* r_H - d_T^* r_H - \chi_2^* h e_T - \chi_3^* s h e_T \\
\beta_H m \left( 1 - \frac{s + \nu y + \tau_T}{1 + m} \right) + \lambda_T^* r_H - d_T^* r_H - \chi_2^* h e_T - \chi_3^* s h e_T \\
-\lambda_T^* r_H - \lambda_T^* e_T + i_T - d_T^* e_T - \chi_2^* h e_T - \chi_3^* s h e_T \\
0
\end{pmatrix},
\]
(14)
where \( \chi_1 = (1 + \eta (\rho - 1)) \), \( \chi_2^* = d_T^* + e_T \), \( \chi_3^* = d_{HT} + e_T \), and \( \Gamma = b + \tau_1 + \tau_2 + \varepsilon_2 + d_{HT} \). From equation (14), we observe that \( \bar{g}_4(X, Y) < 0 \) implying that the second condition in (12) is not fulfilled, so \( \mathcal{E}_o \) may not be globally asymptotically stable. This suggests the existence of multiple equilibria.

**2.6. The Effect of Helminth Infection on Mtb and Vice Versa.**
We analyze the effect of helminth infection on Mtb infection and vice versa, by first expressing \( \mathcal{R}_H \) in terms of \( \mathcal{R}_T \). We solve for \( b \) from the expression of \( \mathcal{R}_T \) in equation (5) to get

\[ b = -\theta_1 \mathcal{R}_T + \sqrt{\theta_2 \mathcal{R}_T^2 + \theta_3 \mathcal{R}_T}, \]
(15)

\[ \text{Proof.} \text{ System (4) as written in equation (12) can be rewritten in a reduced form:} \]
\[ \frac{dX}{dt} \bigg|_{Y=0} = \begin{pmatrix} (1 - \eta) b - bs \\ \eta b - b \nu_T \\ 0 \end{pmatrix}. \]
(13)

Solving equation one in (13) gives \( s(t) = (1 - \eta) + (s_0 - (1 - \eta)) e^{-\lambda t} \) implying that \( s(t) \to 1 - \eta \) as \( t \to \infty \). Similarly, solving equation two in (13) gives \( \nu_T(t) = \eta + (\nu_{T0} - \eta) e^{-\lambda t} \) implying that \( \nu_T(t) \to \eta \) as \( t \to 0 \). Therefore, the first condition in (12) is satisfied.

Let
\[ \mathcal{R}_H = \frac{\beta_T^e \varepsilon_1}{\mu M \left( \left(-\theta_1 R_T + \sqrt{\theta_2 R_T^2 + \theta_3 R_T} \right)/2R_T \right) + d_H + \varepsilon_1 + \tau_1}. \]
(16)

Differentiating equation (16) partially with respect to \( \mathcal{R}_T \) gives
\[ \frac{\partial \mathcal{R}_H}{\partial \mathcal{R}_T} = \frac{\theta_3 \beta_T^e \varepsilon_1 \mathcal{R}_T}{\mu M \sqrt{\mathcal{R}_T (\theta_2 R_T + \theta_3) \left( 2 \mathcal{R}_T (d_H + \varepsilon_1 + \tau_1) - \theta_1 \mathcal{R}_T + \sqrt{\mathcal{R}_T (\theta_2 R_T + \theta_3)} \right)^2}}. \]
(17)
Now, whenever the right-hand side of equation (17) is strictly positive, an increase in the Mtb incidence in the society results in an increase in the incidence of helminth infection in the society.

If RHS of equation (17) is equal to zero, it means that Mtb incidences have no influence on the spread of helminth infection. Also, expressing $b$ in terms of $R_H$ from the expression of $R_H$ in equation (6), we get

$$b = \frac{\beta_H e_1 - \theta_6 R_H}{\mu_M R_H},$$

where $\theta_6 = \mu_M (d_1 + \tau_1)$. Then, expressing $R_T$ in terms of $R_H$, we have

$$R_T = \frac{\beta_H k(1 + \eta (\rho - 1)) \mu_M^2 \beta_1^2 R_H^2}{(\beta_H e_1 + (\mu_M k - \theta_6) R_H) (\beta_H e_1 + (\mu_M (d_1 + \tau_2) - \theta_6) R_H)}.$$  \hfill (19)

Differentiating $R_T$ with respect to $R_H$, we get

$$\frac{\partial R_T}{\partial R_H} = \frac{k \epsilon_1 R_H \mu_M R_H^3 (1 + \eta (\rho - 1)) [R_H (\mu_M (d_1 + k + \tau_2) - 2 \theta_6) + 2 \beta_H e_1]}{(\epsilon_1 R_H (k \mu_M - \theta_6) + \beta_H R_H e_1)^2 (\mu_M (d_1 + \tau_2 - \theta_6) + \beta_H R_H e_1)^2}.$$ \hfill (20)

Whenever $\mu_M (d_1 + k + \tau_2) \geq 2 \theta_6$, then $\partial R_T/\partial R_H$ is strictly positive. This indicates that helminth infection incidence results in an increase in Mtb infection incidence in the society. Thus, helminth infection enhances Mtb infection in the society.

2.7. Existence of Backward Bifurcation. To determine the local asymptotic stability of the endemic equilibrium, we apply the Center Manifold Theorem developed by [25]. To use the theory, we make the subsequent change of variables: let $x_1 = s$, $x_2 = v_T$, $x_3 = e_T$, $x_4 = i_T$, $x_5 = i_H$, $x_6 = i_H$, $x_7 = r_T$, $x_8 = r_H$, and $x_9 = m$. Then, model system (4) is often written within the following form:

$$\frac{dX}{dt} = F(x) = (f_1(x), f_2(x), f_3(x), f_4(x), f_5(x), f_6(x), f_7(x), f_8(x), f_9(x)),$$

where

$$\begin{align*}
\frac{dx_1}{dt} &= f_1(x) = (1 - \eta) b + \gamma_1 x_4 + \gamma_2 x_5 - (b + \beta_T x_4 + \sigma x_6) x_1 - d_T x_4 - (d_H + e_1) x_5 - (d_H + e_2) x_6, \\
\frac{dx_2}{dt} &= f_2(x) = \eta b - \left(b + \rho \beta_T (x_4 + \sigma x_6) + \frac{\beta_H x_9}{1 + x_9}\right) x_2 - d_T x_4 - (d_H + e_1) x_5 - (d_H + e_2) x_6, \\
\frac{dx_3}{dt} &= f_3(x) = \beta_T (x_4 + \sigma x_6) (x_1 + \rho x_2 + x_6) - \left(b + k + \frac{\beta_H x_9}{1 + x_9}\right) x_3 - d_T x_4 - (d_H + e_1) x_5 - (d_H + e_2) x_6. \\
\end{align*}$$

where $f_0 = (d_T - \beta_T) s_0$, $f_1 = (d_H + e_1) s_0$, $f_2 = (\sigma x_4 + (d_H + e_1)) s_0$, $f_3 = (d_H + e_1) H_0$, $f_4 = (\rho x_2 + (d_T - \beta_T) x_6) s_0$. So the Jacobian matrix of system (22) at the disease-free equilibrium ($\mathcal{E}_0$) is given by

$$\begin{align*}
J_0 &= (b + d_T + \tau_2), \\
J_1 &= (b + d_H + \tau_1), \\
J_2 &= (b + \tau_1 + \tau_2 + e_2 + d_H + \tau_2), \\
J_3 &= (b + \gamma_1). \\
\end{align*}$$
Recall that $R_{HT} = \max \{ R_{H}, R_{T} \}$. If we consider $R_{HT} = 1$ (that is, $R_{H} < R_{T} = 1$), let $\beta_T = \beta_T^*$ be a bifurcation parameter. Then, $R_T = 1$ gives

$$\beta_T = \beta_T^* = \frac{(b + k)(b + d_T + \tau_2)}{k(1 + \eta(\rho - 1))}.$$  \hfill (24)

The Jacobian $J_{\varepsilon_o}$ has a simple zero eigenvalue whose related right eigenvectors are denoted by $\mathbf{w} = [w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9]^T$, where $w_1 = ((f_T(d_T - \beta_T^*)v_0 + \gamma_2 v_2)/b_J T) w_5 + ((\mu_M d_T + \beta_T^*)/b_M J T) w_5$, $w_5 = ((\rho \beta_T^* - d_T) v_0) b_J T + ((\mu_M d_T + \beta_T^*)/b_M J T) w_5$, $w_3 = \beta_T^* (\rho v_T T) w_7/(b + k)$, $w_4 = w_5 > 0$, $w_5 = w_3 > 0$, $w_6 = 0$, $w_7 = (\tau_2, w_5)/b_J T$, $w_8 = (\tau_1, w_5)/b_J T$, and $w_9 = (\varepsilon_1, w_5)/b_M J T$.

The left eigenvectors of $J_{\varepsilon_o}$ associated with the simple eigenvalues are denoted by $\nu = [\nu_1, \nu_2, \nu_3, \nu_4, \nu_5, \nu_6, \nu_7, \nu_8, \nu_9]^T$, where $\nu_1 = \nu_2 = \nu_3 = \nu_5 = 0$, $\nu_4 = \nu_5 > 0$, $\nu_3 = k\nu_4/(b + k)$, $\nu_4 = \nu_5 > 0$, $\nu_5 = \nu_5 > 0$, $\nu_6 = ((k\beta_T^* \sigma (\rho v_T T) + \tau_1 (b + k))/b_J T (b + k)) \nu_4 + ((\tau_2 \mu_M + \varepsilon_2 \rho \beta_T^*)/b_M J T) \nu_5$, and $\nu_7 = (\beta_T^*)/b_M J T$.

The coefficients $\alpha$ and $\beta$ that decide the local dynamics of the endemic equilibrium point are defined in equations (25) and (26):

$$a = \sum_{k=1}^{n} \nu_k w_j \frac{\partial^2 f_k}{\partial x_j \partial x_k} (\varepsilon_0, \beta_T^*).$$ \hfill (25)

$$b = \sum_{k=1}^{n} \nu_k w_j \frac{\partial^2 f_k}{\partial \beta_T^* \partial x_j} (\varepsilon_0, \beta_T^*).$$ \hfill (26)

From system (22), the nonzero partial derivatives of $F$ related to $a$ at DFE are given by
\[
\frac{\partial^2 f_3}{\partial x_4 \partial x_2} = \rho \sigma \beta_T,
\]
\[
\frac{\partial^2 f_3}{\partial x_4 \partial x_4} = \beta_T,
\]
\[
\frac{\partial^2 f_3}{\partial x_6 \partial x_6} = \sigma \beta_T,
\]
\[
\frac{\partial^2 f_t}{\partial x_4^2} = d_T,
\]
\[
\frac{\partial^2 f_4}{\partial x_5 \partial x_4} = d_{II} + \epsilon_1,
\]
\[
\frac{\partial^2 f_5}{\partial x_6 \partial x_4} = d_{II} + \epsilon_2,
\]
\[
\frac{\partial^2 f_4}{\partial x_5 \partial x_4} = d_{II} + \epsilon_2 - g \beta_T^*,
\]
\[
\frac{\partial^2 f_5}{\partial x_4 \partial x_1} = \beta_T,
\]
\[
\frac{\partial^2 f_5}{\partial x_5 \partial x_2} = \beta_T,
\]
\[
\frac{\partial^2 f_5}{\partial x_6 \partial x_7} = \beta_T,
\]
\[
\frac{\partial^2 f_6}{\partial x_4 \partial x_4} = d_T,
\]
\[
\frac{\partial^2 f_6}{\partial x_5 \partial x_6} = d_{II} + \epsilon_1 + g \beta_T^*,
\]
\[
\frac{\partial^2 f_6}{\partial x_6^2} = d_{II} + \epsilon_2,
\]
\[
\frac{\partial^2 f_6}{\partial x_4 \partial x_5} = g \beta_T^*,
\]
\[
\frac{\partial^2 f_6}{\partial x_6 \partial x_5} = \beta_T,
\]
\[
\frac{\partial^2 f_6}{\partial x_5 \partial x_4} = \beta_T,
\]
\[
\frac{\partial^2 f_6}{\partial x_6 \partial x_7} = -\beta_T^*.
\]

Therefore, equation (25) becomes

\[
a = \left( \frac{a_0 w_2}{b(b+k)\tau_8} + \frac{a_1 w_4 w_5}{b J_M J_0 (b+k)} \right) v_3 + \left( d_T w_4 + \frac{\mu_M (d_{II} + \epsilon_1 - \beta_T^* \epsilon_1)}{\mu_M} w_4 \right) v_4 + \left( \frac{a_2 w_4 w_5}{b J_M^2} + \frac{a_4 w_4 w_5}{b J_M^2} \right) v_5 + \frac{a_4 w_4 w_5}{\mu_M (b+k)} v_6,
\]

where

\[
a_0 = \begin{align*}
&b d_T \beta_T^* J_8 (s_0 + \rho v_{T_0}) + J_8 \rho \beta_T^* \nu_{T_0} (b+k) (\rho \beta_T^* - d_T) + (J_8 (d_T - \beta_T^* s_0 + \gamma_2 \epsilon_2) \sigma \beta_T^*, \\
&+ J_8 (d_T - \beta_T^* s_0 + \gamma_2 \epsilon_2) \sigma \beta_T^*, \\
&+ J_8 (d_T - \beta_T^* s_0 + \gamma_2 \epsilon_2) \sigma \beta_T^*, \\
&+ J_8 (d_T - \beta_T^* s_0 + \gamma_2 \epsilon_2) \sigma \beta_T^*,
\end{align*}
\]

To determine the sign of \( b \), we find the subsequent non-vanishing partial derivatives of \( F \).


3. Sensitivity Analysis

In this section, we perform a sensitivity analysis of the effective reproduction number subject to each parameter in order to determine the impact of every parameter on the effective reproduction number. Therefore, we compute the forward sensitivity index of the effective reproduction number with respect to the parameters using the approach by Chitnis et al. [26]. Since \( R_{1111} = \max \left\{ R_H, R_T \right\} \), then the sensitivity indices for \( R_H \) and \( R_T \) are computed. The normalized forward sensitivity index of a variable \( P \) with respect to parameter \( r \) is defined as \( \zeta_r = (r/P)(\partial P/\partial r) \). For instance, the sensitivity index of \( R_T \) with respect to \( \beta_T \) is given by \( \delta_{\beta_T} = (\beta_T/R_T)(\partial R_T/\partial \beta_T) = 1 \), and other indices are evaluated using parameter values in Table 1. The remaining indices are given in Tables 2 and 3.

\[
\begin{align*}
\frac{\partial^2 f_1}{\partial x_1 \partial \beta_T} &= -s_0, \\
\frac{\partial^2 f_1}{\partial x_0 \partial \beta_T} &= -\sigma s_0, \\
\frac{\partial^2 f_1}{\partial x_0 \partial \beta_T} &= s_0 + \rho v_{TB}, \\
\frac{\partial^2 f_1}{\partial x_4 \partial \beta_T} &= \sigma (s_0 + \rho v_{TB}).
\end{align*}
\]

Lemma 3. Suppose that \( b > 0 \). Then, we have the following:

(i) System (4) undergoes forward bifurcation if the coefficient \( a \) is positive

(ii) System (4) will exhibit transcritical bifurcation if the coefficient \( a \) is negative

4. The Optimal Control Problem

In this section, the extension of model (1) is formed by including four time-dependent controls so as to decide the optimal strategy for controlling the two diseases. The controls are defined as follows:

(i) Educational campaign \( u_1(t) \) that sensitizes the parents to vaccinate more infants. Therefore, the recruitment for the Mtb-vaccinated individuals changes to \( \eta N(1 + u_1(t)) \) meaning that when this control is 100% implemented, then the number of vaccinated babies doubles

(ii) Treatment \( \alpha_1 u_2(t) \) of individuals with active TB, where \( \alpha_1 \) is the drug efficacy use for Mtb infection. Therefore, the Mtb recovery rate changes to \( \tau_2 + \alpha_1 u_2(t) \)

(iii) Deworming at a rate \( \alpha_2 u_3(t) \) for the helminth-infected individuals and coinfected individuals, where \( \alpha_2 \) is the drug efficacy use for helminth infection. Therefore, the helminth recovery rate changes to \( \tau_1 + \alpha_2 u_3(t) \)

(iv) Sanitation and proper hygiene \( u_4 \) that reduces the ingestion rate of parasites. Therefore, the force of infection for the helminth infection is changed to \( \lambda_1 = (1 - u_4)\beta_T M/(K + M) \)

From Tables 2 and 3, the most positive parameter indices are \( \beta_T, k \), and \( \beta_H \) while the most negative parameter indices are \( b, d_T, \) and \( \mu_M \). This implies that when parameters \( \beta_T, k \), and \( \beta_H \) are increased, and while the remaining are kept constant, the endemity of the disease is increased, while when the parameter \( \mu_M \) is increased, the endemity of disease is decreased. Increasing the natural birth rate and Mtb mortality rate does not make biological sense that the disease would be contained. Thus, we suggest the application of optimal control to sensitive parameters such as \( \beta_H, \beta_T, \) and \( \mu_M \) to study how effectively we can contain the helminth-Mtb infection.
After incorporating the controls \( u_1(t), u_2(t), u_3(t), \) and \( u_4(t) \) in the Mtb-helminth infection model (4), then the optimal control model becomes

\[
\begin{align*}
\frac{dS}{dt} &= (1 - \eta) b N + \gamma_1 R_H + \gamma_2 R_T - (\mu + \lambda_T + (1 - u_4) \lambda_H) S, \\
\frac{dV_T}{dt} &= \eta b (1 + u_1) N - (\mu + \rho \lambda_T + (1 - u_4) \lambda_H) V_T, \\
\frac{dE_T}{dt} &= \lambda_T S + \rho \lambda_T V_T + \lambda_T R_H - (\mu + k + (1 - u_4) \lambda_H) E_T, \\
\frac{dH}{dt} &= k E_T + (\tau_1 + \alpha_2 u_3) I_{HT} - (\mu + d_H + \tau_2 + (1 - u_4) \lambda_H) H - (\tau_2 + \alpha_1 u_2) I_T, \\
\frac{dH_{HT}}{dt} &= (1 - u_4) \lambda_H S + (1 - u_4) \lambda_H V_T + (1 - u_4) \lambda_H R_T + \tau_2 I_{HT} - g (\lambda_T + \mu + d_H + \epsilon_1) I_H - (\tau_1 + \alpha_2 u_3) I_{HT}, \\
\frac{dU}{dt} &= (\tau_1 + \alpha_1 u_1) I_T - (\tau_1 + \alpha_2 u_3) I_{HT}, \\
\frac{dR_T}{dt} &= (\tau_2 + \alpha_1 u_2) I_T - (\tau_2 + \alpha_1 u_2) I_T, \\
\frac{dM}{dt} &= \epsilon_1 I_H + \epsilon_2 H_{HT} - \mu M M.
\end{align*}
\] (31)

We employ Pontryagin’s Maximum Principle to figure out the required conditions for the optimal control of helminth-Mtb coinfecion. The control set \( U \) is Lebesgue measurable and is defined as \( U = \{u_1(t), u_2(t), u_3(t), u_4(t) : 0 \leq u_i(t) \leq 1, 0 < t < t_f\} \). Then, the objective is to minimize individuals with active TB, individuals infected with helminth parasites, and coinfected individuals while keeping the cost low. For this problem, we consider the objective functional defined by

\[ J = \int_{t_0}^{t_f} \left\{ C_1 I_T + C_2 I_H + C_3 I_{HT} + \frac{1}{2} \sum_{i=1}^{4} w_i u_i^2 \right\} dt, \] (32)

where \( C_1 I_T, C_2 I_H, C_3 I_{HT} \) are the costs related to active tuberculosis individuals, individuals infected with helminths, and coinfected individuals, respectively. The expression \((1/2)w_i u_i^2\) is related to the background costs with relative cost weights \( w_i \) for every control measure. The quadratic cost is tailored as utilized in other models with controls (see [16, 27]). The particular value of the weights requires intensive data processing; hence, we elect estimate values for theoretical purposes.

The aim is to minimize the objective functional \( J \) as defined in (32) subject to model (31). Therefore, we seek to get the optimal controls \( u_1^*, u_2^*, u_3^*, \) and \( u_4^* \) such that

\[ J(u_1^*, u_2^*, u_3^*, u_4^*) = \min_{u_1, u_2, u_3, u_4 \in U} J(u_1, u_2, u_3, u_4), \] (33)

where \( U = \{(u_1, u_2, u_3, u_4) \) and therefore the controls \( u_1, u_2, u_3, \) and \( u_4 \) are measurable with \( 0 \leq u_1 \leq 1, 0 \leq u_2 \leq 1, 0 \leq u_3 \leq 1, \) and \( 0 \leq u_4 \leq 1 \) for \( t \in [t_0, t_f]\). \]

\textbf{Theorem 4.} Consider the control problem with the system of equations in (31). There exist \( u^* = (u_1^*, u_2^*, u_3^*, u_4^*) \in U \) such that

\[ \min_{u_1, u_2, u_3, u_4 \in U} J(u_1, u_2, u_3, u_4) = J(u_1^*, u_2^*, u_3^*, u_4^*). \] (34)

The necessary conditions that an optimal solution must satisfy are derived from Pontryagin’s Maximum Principle [28]. The principle converts equations (31) and (32) into the problem of minimizing the pointwise Hamiltonian \( H \), with reference to \( u_1, u_2, u_3, \) and \( u_4 \). The Hamiltonian is given by
where  are the costate variables or the adjoint variables. Now, applying Pontryagin’s Maximum Principle [28], the problem has a solution following the results of the optimal control problem in [29].

**Theorem 5.** Given the optimal control , for or the solutions, the state equations (31) and (32) that minimize subject to (30) and (33), there exist adjoint variables  and  and satisfying

\[
\frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial \xi_i}
\]

where  with transversality conditions  and  for 0, and the subsequent characterization holds on the interior of the control set  if

\[
\begin{align*}
\mu^*_1 &= \max \left\{ \frac{\lambda_{11}}{w_1}, \frac{\lambda_{12}}{w_2}, \lambda_{13}, \lambda_{14}, \lambda_{15} \right\}, \\
\mu^*_2 &= \max \left\{ \frac{\lambda_{21}}{w_1}, \frac{\lambda_{22}}{w_2}, \lambda_{23}, \lambda_{24}, \lambda_{25} \right\}, \\
\mu^*_3 &= \max \left\{ \frac{\lambda_{31}}{w_1}, \frac{\lambda_{32}}{w_2}, \frac{\lambda_{33}}{w_3}, \frac{\lambda_{34}}{w_4}, \lambda_{35} \right\}, \\
\mu^*_4 &= \max \left\{ \frac{\lambda_{41}}{w_1}, \frac{\lambda_{42}}{w_2}, \frac{\lambda_{43}}{w_3}, \frac{\lambda_{44}}{w_4}, \lambda_{45} \right\}, \\
\mu^*_5 &= \max \left\{ \frac{\lambda_{51}}{w_1}, \frac{\lambda_{52}}{w_2}, \frac{\lambda_{53}}{w_3}, \frac{\lambda_{54}}{w_4}, \frac{\lambda_{55}}{\lambda_{56}} \right\}.
\end{align*}
\]

**Proof.** Fleming and Rishel [23] provide the existence of an optimal control model (31) and the costate variables (36) due to the boundedness of state equations and the Lipschitz structure of the ordinary differential equations. Therefore, applying the necessary conditions from Pontryagin’s Maximum Principle, we obtain the following system for costate variables:

\[
\begin{align*}
\frac{d\lambda_1}{dt} &= -(\lambda_{11} - \lambda_{12}) - \lambda_{13} - \lambda_{14} + \lambda_{15} \\
\frac{d\lambda_2}{dt} &= -(\lambda_{21} - \lambda_{22}) - \lambda_{23} - \lambda_{24} + \lambda_{25} \\
\frac{d\lambda_3}{dt} &= -(\lambda_{31} - \lambda_{32}) - \lambda_{33} - \lambda_{34} + \lambda_{35} \\
\frac{d\lambda_4}{dt} &= -(\lambda_{41} - \lambda_{42}) - \lambda_{43} - \lambda_{44} + \lambda_{45} \\
\frac{d\lambda_5}{dt} &= -(\lambda_{51} - \lambda_{52}) - \lambda_{53} - \lambda_{54} + \lambda_{55} + \lambda_{56}.
\end{align*}
\]
To get the controls, we solve the equations \( \partial H / \partial u_i = 0 \) at \( u_i^* \) for \( i = 1, \ldots, 4 \) and obtained the following:

\[
\begin{align*}
  u_1^* &= \frac{-\eta b N \lambda_{V_1}}{w_1}, \\
  u_2^* &= \frac{\alpha I_T (\lambda_{I_t} - \lambda_{R_t})}{w_2}, \\
  u_3^* &= \frac{\alpha I_H (\lambda_{I_{ht}} - \lambda_{I_t})}{w_3}, \\
  u_4^* &= \frac{\lambda_{I_{ht}} (S + V_T + R_T) + \lambda_{I_{ht}} (E_T + I_T)}{w_4} - \lambda_{I_T} (S + V_T + R_T).
\end{align*}
\]

Then, we write by standard control arguments involving the bounds on the controls as

\[
\begin{align*}
  u_1^* &= \begin{cases} 
  \Theta_1, & \text{if } 0 < \Theta_1 < 1, \\
  0, & \text{if } \Theta_1 \leq 0, \\
  1, & \text{if } \Theta_1 \geq 1,
  \end{cases} \\
  u_2^* &= \begin{cases} 
  \Theta_2, & \text{if } 0 < \Theta_2 < 1, \\
  0, & \text{if } \Theta_2 \leq 0, \\
  1, & \text{if } \Theta_2 \geq 1,
  \end{cases} \\
  u_3^* &= \begin{cases} 
  \Theta_3, & \text{if } 0 < \Theta_3 < 1, \\
  0, & \text{if } \Theta_3 \leq 0, \\
  1, & \text{if } \Theta_3 \geq 1,
  \end{cases} \\
  u_4^* &= \begin{cases} 
  \Theta_4, & \text{if } 0 < \Theta_4 < 1, \\
  0, & \text{if } \Theta_4 \leq 0, \\
  1, & \text{if } \Theta_4 \geq 1,
  \end{cases}
\end{align*}
\]

(40)

where

\[
\begin{align*}
  \Theta_1 &= -\frac{\eta b N \lambda_{V_1}}{w_1}, \\
  \Theta_2 &= \frac{\alpha I_T (\lambda_{I_t} - \lambda_{R_t})}{w_2}, \\
  \Theta_3 &= \frac{\alpha I_H (\lambda_{I_{ht}} - \lambda_{I_t})}{w_3}, \\
  \Theta_4 &= \frac{\lambda_{I_{ht}} (S + V_T + R_T) + \lambda_{I_{ht}} (E_T + I_T)}{w_4} - \lambda_{I_T} (S + V_T + R_T).
\end{align*}
\]

5. Numerical Simulation

In this section, different optimal control strategies are used to investigate numerically their effect on the spread of helminth and Mtb coinfection. The state system (31), the adjoint system (38), and therefore the characterization in equation (37) are solved numerically using an iterative scheme so as to attain the optimal control. Using the initial conditions for the state system and the transversality conditions for the adjoint system with the initial guess of the controls, we solve the state system forward in time and the adjoint system backward in time using the fourth-order Runge-Kutta scheme with the current solutions of the state system. Then, the controls are updated using a convex combination of the controls and the values from characterization (37). The solution of the state system and the adjoint system is repeated until the present iteration is close to the previous iteration [30].

In numerical simulation, the weights are assumed to depend on the cost of investment and importance of the controls. For example, the cost associated with control \( u_2 \) requires huge investment that includes clinical examination...
of Mtb cases and procurement and transportation of the drugs. The cost associated with control \( u_2 \) requires procurement and transportation of anthelmintic drugs and monitoring of the distribution of drugs. We choose the weights as \( C_1 = 5822, C_2 = 0.89, C_3 = 5822.89, w_1 = 20, w_2 = 40, w_3 = 30, w_4 = 10 \) and the initial state variables as \( S = 1500, V_T = 2000, E_T = 200, I_T = 150, I_{HT} = 100, I_{MT} = 80, R_T = 5, R_H = 5, \) and \( M = 200 \). The efficacy parameters are \( \alpha_1 = 0.75 \) and \( \alpha_2 = 0.8 \), and the remaining parameter values are given in Table 1.

5.1. Control with Educational Campaign \((u_1)\) and Treatment of Individuals with Active TB \((u_2)\). The educational campaign control \((u_1)\) and treatment of individuals with active TB control \((u_2)\) are used to optimize the objective functional \( J \) while the other controls \((u_3)\) and \((u_4)\) related to helminths were set to zero. We observed that in Figure 2(b) the number of individuals with active TB was controlled but starts to increase again until the end of the intervention period. This may be related to our previous analysis that helminth infection enhances Mtb infection. In this strategy, helminth infection is not controlled; Figures 2(c) and 2(d) indicate that there is slight significance in the case with controls and without controls for helminth-infected individuals and coinfecting individuals, respectively. Figure 2(e) indicates that the parasite population is not controlled with this strategy while Figure 2(f) indicates that the educational campaign control should be seized after 158 days while treatment of individuals with active TB control should be kept maximum in the entire period of intervention.

5.2. Control with Mass Drug Administration \((u_3)\) and Sanitation \((u_4)\). The mass drug administration (MDA) control \((u_3)\) and sanitation control \((u_4)\) were used to optimize the objective functional \( J \). Again, we observe that Figure 3(a) indicates the increase in vaccinated individuals. Also, Figure 3(b) indicates that the number of people with active TB increases rapidly and is eventually controlled after 55 days. An equivalent scenario is observed in Figures 3(c) and 3(d) where the helminth-infected individuals and coinfecting individuals were effectively controlled. Figure 3(e) suggests that the parasite population is substantially controlled at the end of the intervention period. This strategy seems to be effective in controlling the two diseases from the community. Figure 3(f) indicates that MDA and sanitation controls should be kept maximum throughout the intervention period.

5.3. Control with Educational Campaign \((u_1)\) and Sanitation \((u_4)\). The educational campaign control \((u_1)\) and sanitation control \((u_4)\) are employed to optimize the objective functional \( J \). This strategy has the same profiles as the strategy with mass drug administration and sanitation controls but takes a long time to control the two diseases. Figure 4(f) indicates that the educational campaign must be stopped after 30 days whereas sanitation control must be kept maximum in the entire period of intervention.

5.4. Control with Treatment of Individuals with Active TB \((u_2)\) and MDA \((u_3)\). The treatment of individuals with active TB control \((u_2)\) and MDA control \((u_3)\) are used to optimize the objective functional \( J \) while the other controls \((u_1)\) and \((u_4)\) are set to zero. Figure 5(a) indicates a slight increase in vaccinated individuals compared to the other strategies (Sections 5.1, 5.2, and 5.3). Figures 5(b) and 5(d) indicate that the treatment of individuals with active TB and coinfecting individuals is effectively controlled; while Figure 5(c) indicates a decrease in the helminth-infected individuals but a rise at the final intervention period. This is due to the absence of preventive measures in this strategy. Figure 5(f) indicates that both controls should be kept maximum until the final intervention period.

5.5. Control with Educational Campaign \((u_1)\), Treatment of Mtb-Infected Individuals \((u_2)\), MDA \((u_3)\), and Sanitation \((u_4)\). With this strategy, all four controls are employed to optimize the objective functional \( J \). We observed that Figure 6(a) indicates that the number of vaccinated individuals increases more at the end of the intervention period. Figures 6(b)–6(d) indicate that individuals with active TB, helminth-infected individuals, and coinfecting individuals are all effectively and efficiently controlled at the end of the intervention period. Figure 6(e) depicts that the parasite population is decreased to a number that cannot harm the human population. This strategy indicates that optimal educational campaign, treatment of individuals with active TB, MDA, and sanitation would effectively control the helminth-Mtb infection at the end of the intervention period. Figure 6(f) suggests that the educational campaign should be stopped after 115 days whereas the remaining controls should be maintained maximum for the whole intervention period.

6. Discussions and Conclusions

In this paper, we described and formulated a deterministic model for the transmission dynamics and control of helminth-Mtb coinfection. The effective reproduction number was computed, and we explored the steadiness of the disease-free equilibrium point. We again established the existence of backward or forward bifurcation using the Center Manifold Theorem. Then, we investigated the impact of the two diseases on each other and found that helminth infection increases Mtb infection. Also, we modulated the helminth-Mtb model by incorporating four control measures: educational campaign to sensitize the parents to take babies to the BCG vaccine clinic, treatment of individuals with active TB, mass drug administration for helminth and coinfecting individuals, and sanitation to reduce the intake rate of parasites. The optimal control was analyzed using Pontryagin’s Maximum Principle [28], by first finding the Hamiltonian, the costate variables, the characterization of the controls, and the optimality system.

Then, we solved numerically the optimality system by the iterative scheme using the Forward-Backward Sweep Method (FBSM) with the combination of the following control measures:
(i) By applying a combination of educational campaign and treatment of individuals with active TB

(ii) By applying a combination of mass drug administration (MDA) and sanitation

(iii) By applying a combination of educational campaign and sanitation

(iv) By applying a combination of treatment of individuals with active TB and mass drug administration

Figure 2: Simulations of the helminth-Mtb coinfection model with the effect of educational campaign and treatment of Mtb-infected individuals.
By applying a combination of educational campaign, treatment of individuals with active TB, mass drug administration, and sanitation, the control strategies which focus on TB only while helminths are not controlled would not lead to the efficient and effective control of TB or helminth infection. Moreover,

\[
\begin{align*}
    u_1 &= 0, u_2 = 0, u_3 \neq 0, u_4 \neq 0, \\
    u_i &= 0, \ i = 1,2,3,4
\end{align*}
\]

The control strategies which focus on TB only while helminths are not controlled would not lead to the efficient and effective control of TB or helminth infection. Moreover,
control strategies that focus on helminths only while TB is not controlled would control both TB and helminth infections. This is linked to the increased susceptibility of Mtb for individuals infested with helminths. However, control strategies that include all control measures would effectively control the helminth-Mtb
coinfection at the end of the intervention period. Thus, we suggest to the public health stakeholders that in order to control the helminth-Mtb coinfection, the intervention strategies that target controlling helminth infection and vaccination of babies with BCG after birth and treatment of individuals with active coinfection have to be further improved.

Figure 5: Simulations of the helminth-Mtb coinfection model with the effect of treatment of Mtb-infected individuals and MDA.
tuberculosis should be emphasized. One of the limitations of this study is the number of compartments involved that hinders some analytical analyses; however, numerical simulation shows the convergence.

**Data Availability**

The data are available inside the manuscript, and there is no restriction on the availability of the data.
Conflicts of Interest
The authors declare that they have no contesting interests regarding the publication of this article.

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Supplementary Materials
The supplementary material related to this article comprises the MATLAB codes used for numerical simulations. (Supplementary Materials)

References


