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Global Dynamics and Optimal Control Analysis of Tuberculosis Transmission Model With Incomplete Treatment

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Abstract. A mathematical model of tuberculosis (TB) with incomplete treatment is proposed. Two classes of treated individuals including treated latent and treated active TB are considered and both classes may reenter due to an incomplete treatment. Our model therefore consists of six classes which are susceptible, latent, active TB, treated latent, treated active TB, and recovered individuals. The model solutions are proved to be nonnegative and bounded. We calculate two equilibrium points (disease-free and endemic) and their stability conditions are analyzed locally and globally. The basic reproduction number is computed and it is obtained that when it is less than a unity, a global asymptotic stability is observed for disease-free equilibrium point. And when it is greater than a unity, an endemic equilibrium point exists and stable under some certain conditions. Furthermore, a model is extended to include optimal control problem by considering four control variables which are preventive control, the screening and put under treatment control, the treatment effort for active TB and the campaign to make sure patients obtain complete treatment. Numerical simulations are performed and the results show that single control could reduce patients with TB infection for some certain amount, however, a combination of four controls give the most promising in reducing TB patients and increasing recovered ones. These results can benefit in providing information for public health authorities to take further action.

1. Introduction

Tuberculosis (TB) is an airborne bacterial infectious disease which has been a leading cause of death globally for centuries. It is caused by Mycobacterium tuberculosis that most often affects the lung. Tuberculosis is present in all age groups and in all countries around the world. According to World Health Organization (WHO), in 2024 a total of 1.23 million people died from tuberculosis [26]. People with tuberculosis infection and disease are treated with special antibiotics, commonly are rifampicin, isoniazid, pyrazinamide and ethambutol. Stopping the treatment early

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or not following medical advice is a serious issue and is called incomplete treatment. Incomplete treatment can lead to several health complications including disease relapse which often more severely, treatment failure, and the development of drug-resistant TB which results in higher risk of death. Therefore, completion of TB treatment is highly important.

Mathematical model has been used as a tool to study the dynamic of various infectious diseases including tuberculosis. Here are some examples of tuberculosis models proposed by many researchers considering different aspects in the past decades. Tuberculosis model is first developed in 1962 by Waaler et al. [27], In 2011, Liu and Zhang proposed a TB model studying effects of vaccination and treatment on TB spread [15]. Yang and the team presented a TB model includes the impact of seasonality on the TB transmission in 2016 [32]. In the same year, Ronoh and the team proposed a model of tuberculosis with drug resistance effects [22]. In 2018, Gupta and the team developed a model with drug resistance to both the first and the second line of treatment [9]. In 2021, Otoo and the team proposed a TB model with drug resistance to the first-line treatment and leaky vaccination [19]. A TB model with the public health education and hospital treatment impact was proposed by Sulayman and Abdullah in 2022 [23]. A year later, Yavuz and the team presented a TB model under consciousness effect [33]. In 2025, Ochieng proposed a TB model considering a reinfection [18].

Further, with the importance of complete treatment, some TB models have been proposed to study an incomplete treatment effect on TB dynamics. In 2012, Yang and the team developed two TB models incorporate self-cure and incomplete treatment which leads to treatment failure [31]. The first model includes one latent compartment, whereas the second model includes two latent compartments. Three years later, Zhang and Feng developed a TB model with isolation and incomplete treatment [34]. In 2017, Xu and the team established a TB model with age of infection and incomplete treatment [29]. In 2020, Ullah and the team proposed a TB model with incomplete treatment [24]. They studied the impact of effective contact rate, treatment rate, and incomplete treatment versus efficient treatment. Recently, Wang and Wang established a stochastic model for TB with incomplete treatment [28].

Optimal control problems have been applied into the infectious disease models to seek potential strategy to control the spread of disease. Here are some optimal control models for tuberculosis transmission, the work by Gao and Huang [8], Yang et al. [30], Kereyu and Demie [7], Alfiniyah et al. [2], Kang et al. [6], Ochieng [18], and Mulugeta et al. [16], To the authors' knowledge, there has been only one optimal control model for TB transmission with incomplete treatment. This work was proposed by Abimbade and the team in 2020 [1]. They added three control variables into the model consisting of public awareness campaign to promote good personal hygiene, maximum treatment effort and effort to prevent incomplete treatment.

In this study, we therefore propose a deterministic model for tuberculosis transmission dynamics with incomplete treatment. We include two classes of treated individuals, i.e., treated individuals of latent and of active TB individuals and incorporate an incomplete treatment for both classes

of population. We then extend the model to optimal control model to find potential measures to reduce tuberculosis transmission.

The structure of the paper is as follows. The description of how model is formulated with all variables and parameters' definitions are presented in Section 2. Section 3 includes analysis of the model i.e., verification of positivity and boundedness of solutions, equilibrium points and their stability analysis, and basic reproduction number and its sensitivity analysis. Optimal control problem is applied into a model and is presented in Section 4, whereas its numerical simulation is demonstrated in Section 5 with discussion. Finally, some conclusions are stated in Section 6.

2. Model formulation

A deterministic model describing tuberculosis transmission with incomplete treatment is proposed. According to the work by Yavuz and the team [33], two classes of treated individuals were taken into account and they are treated individuals of latent and active TB. We therefore modified the work of Ullah and the team [24] by adding these two classes into the model and incorporate an incomplete treatment for both classes of population, i.e., incomplete treatment of latent individuals and incomplete treatment of active TB individuals. Our model then comprises of six classes of population which are susceptible (S), latent (L), active TB (I), treated latent (T_L), treated active TB (T_I), and recovered (R) individuals. The diagram of our proposed model is shown in Figure 1.

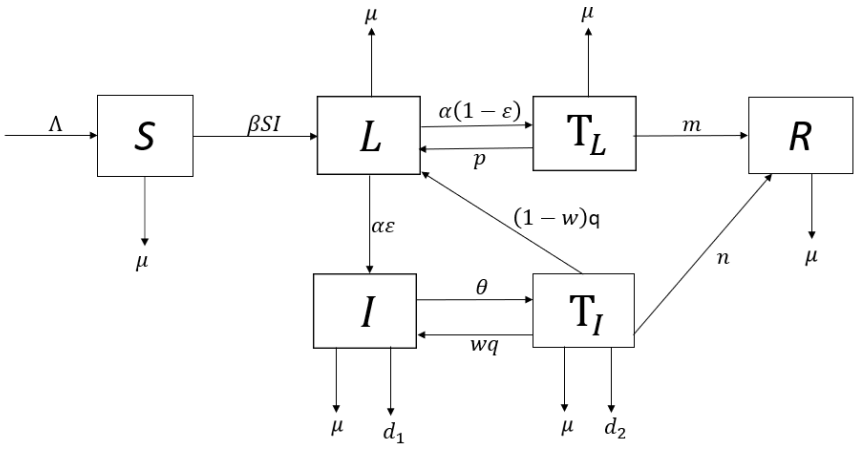


Figure 1. Flow diagram of our proposed model.

Here, susceptible individuals are recruited at a rate Λ. They get infected by TB at a transmission rate β, then become latent, giving an infection term as βSI. All individuals die at a natural death rate μ. Latent individuals leave their stage at a rate α to active TB class with a fraction ϵ, and to treated latent class with a fraction $1-\epsilon$. Active individuals are treated at a rate θ. Latent individuals reenter latent class due to incomplete treatment at a rate p, whereas treated active TB

individuals leave their class due to incomplete treatment at a rate q to active TB class with a fraction w, and to latent class with a fraction $1-w$. Treated latent and treated active TB are recovered at a rate m and n, respectively. Active TB, and treated active TB die due to the TB infection at a rate d_1, and d_2, respectively.

The system of equations describing above model is as follows.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - \mu S,\\
\frac{dL}{dt} &= \beta SI + pT_L + (1-w)qT_I - \alpha\epsilon L - \mu L - \alpha(1-\epsilon)L,\\
\frac{dI}{dt} &= \alpha\epsilon L + wqT_I - \mu I - d_1 I - \theta I,\\
\frac{dT_L}{dt} &= \alpha(1-\epsilon)L - pT_L - mT_L - \mu T_L,\\
\frac{dT_I}{dt} &= \theta I - (1-w)qT_I - wqT_I - d_2 T_I - \mu T_I - nT_L,\\
\frac{dR}{dt} &= mT_L + nT_I - \mu R,
\end{aligned} \tag{2.1}$$

with initial conditions: $S(0) \geq 0,\ L(0) \geq 0,\ I(0) \geq 0,\ T_L(0) \geq 0,\ T_I(0) \geq 0,\ R(0) \geq 0.$

3. Model analysis

3.1. Positivity and Boundary of solution.

Theorem 3.1. *With nonnegative initial conditions, all solutions of system* (2.1) *remain nonnegative and bounded for all* $t > 0$ *in the region* Ω*, where*

$$\Omega = \left\{(S, L, I, T_L, T_I, R) \in \mathbb{R}^6_+ : S + L + I + T_L + T_I + R \leq \frac{\Lambda}{\mu}\right\}.$$

Proof. First, we clarify that all solutions of (2.1) are nonnegative. For any nonnegative initial conditions, consider the following,

$$\begin{aligned}
\frac{dS}{dt}\Big|_{S=0} &= \Lambda \geq 0,\\
\frac{dL}{dt}\Big|_{L=0} &= \beta SI + pT_L + (1-w)qT_I \geq 0,\\
\frac{dI}{dt}\Big|_{I=0} &= \alpha\epsilon L + wqT_I \geq 0,\\
\frac{dT_L}{dt}|_{T_L=0} &= \alpha(1-\epsilon)L \geq 0,\\
\frac{dT_I}{dt}\Big|_{T_I=0} &= \theta I \geq 0,\\
\frac{dR}{dt}\Big|_{R=0} &= mT_L + nT_I \geq 0.
\end{aligned}$$

Therefore, by functional differential equations theory, the positivity of all solutions initiating in $\mathbb{R}^6_+$ is guaranteed for all $t > 0$.

Next, boundedness of solution of (2.1) is verified. Let the total number of population be $N = S + L + I + T_L + T_I + R$.

Then,

$$\begin{aligned}\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dL}{dt} + \frac{dI}{dt} + \frac{dT_L}{dt} + \frac{dT_L}{dt} + \frac{dR}{dt}\\ &= \Lambda - \beta SI - \mu S + \beta SI + pT_L + (1-w)qT_I - \alpha\epsilon L - \mu L - \alpha(1-\epsilon)L\\ &+ \alpha\epsilon L + wqT_I - \mu - d_1 I - \theta I + \alpha(1-\epsilon)L - pT_L - mT_L - \mu T_L\\ &+ \theta I - (1-w)qT_I - wqT_I - d_2 T_I - \mu T_I - nT_I + mT_L + nT_I - \mu R\\ &= \Lambda - \mu N - d_1 I - d_2 T_I. \end{aligned} \tag{3.1}$$

Therefore,

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{3.2}$$

We next use integrating factor method to solve (3.2), we obtain

$$N \leq \frac{\Lambda}{\mu} + C_1 e^{-\mu t}, \tag{3.3}$$

where C_1 is constant.

By taking $t \to \infty$, then $N \to \frac{\Lambda}{\mu}$, implying that $0 \leq N \leq \frac{\Lambda}{\mu}$.

Thus, the considered region for this model is

$$\Omega = \left\{(S, L, I, T_L, T_I, R) \in \mathbb{R}^6_+ : S + L + I + T_L + T_I + R \leq \frac{\Lambda}{\mu}\right\}.$$

All solutions of system (2.1) are bounded and enter the region Ω. Hence, Ω is a positively invariant. That is every solution of this model remains there for all $t > 0$. This completes the proof.

3.2. Equilibrium points.

Two equilibrium points are calculated and they are

(1) Disease-free equilibrium point E_0 is $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right)$.

(2) Endemic equilibrium point $E_1 = (S^*, L^*, I^*, T_L^*, T_I^*, R^*)$, where

$$S^* = \frac{\Lambda}{\beta I^* + \mu}, L^* = \frac{\frac{\beta\Lambda I^*}{\beta I^* + \mu} + \frac{(1-w)q\theta I^*}{q + d_2 + \mu + n}}{\alpha + \mu - \frac{p\alpha(1-\epsilon)}{p+m+\mu}}, I^* = \frac{\alpha\epsilon\beta\Lambda(q + d_2 + \mu + n) + \alpha\epsilon(1-w)q\theta\beta - A\mu}{A\beta - \alpha\epsilon(1-w)q\theta\beta},$$

$$T_L^* = \frac{\alpha(1-\epsilon)L^*}{p + m + \mu}, T_I^* = \frac{\theta I^*}{q + d_2 + \mu + n}, R^* = \frac{mT_L^* + nT_I^*}{\mu},$$

where $A = ((\mu + d_1 + \theta)(q + d_2 + \mu + n) - wq\theta)(\alpha + \mu - \frac{p\alpha(1-\epsilon)}{p+m+\mu})$.

Here, E_1 exists when $\alpha + \mu > \frac{p\alpha(1-\epsilon)}{p+m+\mu}$, $\alpha\epsilon\beta\Lambda > \mu(\alpha+\mu)(\mu + d_1 + \theta)$, and $A > \alpha\epsilon(1-w)q\theta$.

3.3. Basic reproduction number **(R_0).**

The basic reproduction number (R_0) is a transmission potential measurement which is defined as the mean number of secondary cases produced by a typical infective individual. It is calculated by using next generation method of [25]. We start by writing the equation $\frac{dL}{dt}$ and $\frac{dI}{dt}$ in the form $\mathcal{F} - \mathcal{V}$, where $\mathcal{F}$ is the matrix of the rate of appearance of new infections, and $\mathcal{V}$ is a transferring matrix.
Thus, for our model we have

$$\mathcal{F} = \begin{bmatrix} \beta SI \\ \\ 0 \end{bmatrix} \text{ and } \quad \mathcal{V} = \begin{bmatrix} -pT_L - (1-w)qT_I + \alpha L + \mu L \\ \\ -\alpha\epsilon L - wqT_I + \mu I + d_1 I + \theta I \end{bmatrix}.$$

The Jacobian matrices of above matrices are

$$F = \begin{bmatrix} 0 & \beta S \\ \\ 0 & 0 \end{bmatrix} \text{ and } \quad V = \begin{bmatrix} \alpha + \mu & 0 \\ \\ -\alpha\epsilon & \mu + d_1 + \theta \end{bmatrix}.$$

Substitute $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right)$ in F and V above, we have

$$F = \begin{bmatrix} 0 & \frac{\beta\Lambda}{\mu} \\ \\ 0 & 0 \end{bmatrix} \text{ and } \quad V = \begin{bmatrix} \alpha + \mu & 0 \\ \\ -\alpha\epsilon & \mu + d_1 + \theta \end{bmatrix}.$$

Next calculate V^{-1}, we have

$$V^{-1} = \frac{1}{(\alpha+\mu)(\mu+d_1+\theta)} \times \begin{bmatrix} \mu + d_1 + \theta & 0 \\ \\ \alpha\epsilon & \alpha + \mu \end{bmatrix}.$$

Thus, the next generation matrix is

$$FV^{-1} = \begin{bmatrix} \frac{\alpha\epsilon\beta\Lambda}{\mu(\alpha+\mu)(\mu+d_1+\theta)} & \frac{\beta\Lambda(\alpha+\mu)}{\mu(\alpha+\mu)(\mu+d_1+\theta)} \\ \\ 0 & 0 \end{bmatrix}.$$

Since R_0 is the leading eigenvalue of matrix FV^{-1}, therefore we then obtain the basic reproduction number (R_0) as

$$R_0 = \frac{\alpha\epsilon\beta\Lambda}{\mu(\alpha+\mu)(\mu+d_1+\theta)}.$$

3.4. Local stability analysis.

To analyze local stability of equilibrium point, we use the linearization method. We first calalate for jacobian matrix of each equilibrium point, then check the condition for being locally stable, i.e., all eigenvalues have negative real parts.
Jacobian matrix of the system (2.1) is

$$J(S,L,I,T_L,T_I,R)=\begin{bmatrix} -\beta I-\mu & 0 & -\beta S & 0 & 0 & 0\\ \beta I & -\alpha-\mu & \beta S & p & (1-w)q & 0\\ 0 & \alpha\epsilon & -\mu-d_1-\theta & 0 & wq & 0\\ 0 & \alpha(1-\epsilon) & 0 & -p-m-\mu & 0 & 0\\ 0 & 0 & \theta & 0 & -q-d_2-\mu-n & 0\\ 0 & 0 & 0 & m & n & -\mu \end{bmatrix}. \tag{3.4}$$

3.4.1. *Local stability of the disease free equilibrium point.*

Theorem 3.2. *The disease-free equilibrium point (E_0) is locally asymptotically stable if $R_0<1$, and if it satisfies Routh-Hurwitz criteria, otherwise it is unstable.*

Proof. The Jacobian matrix of the system (2.1) at E_0 is

$$J\left(\frac{\Lambda}{\mu},0,0,0,0,0\right)=\begin{bmatrix} -\mu & 0 & \frac{-\beta\Lambda}{\mu} & 0 & 0 & 0\\ 0 & -\alpha-\mu & \frac{\beta\Lambda}{\mu} & p & (1-w)q & 0\\ 0 & \alpha\epsilon & -\mu-d_1-\theta & 0 & wq & 0\\ 0 & \alpha(1-\epsilon) & 0 & -p-m-\mu & 0 & 0\\ 0 & 0 & \theta & 0 & -q-d_2-\mu-n & 0\\ 0 & 0 & 0 & m & n & -\mu \end{bmatrix}. \tag{3.5}$$

Then, consider $\det(J(E_0)-\lambda I)=0$, we have

$$(-\mu-\lambda)(-\mu-\lambda)\begin{vmatrix} -\alpha-\mu-\lambda & \frac{\beta\Lambda}{\mu} & p & (1-w)q\\ \alpha\epsilon & -\mu-d_1-\theta-\lambda & 0 & wq\\ \alpha(1-\epsilon) & 0 & -p-m-\mu-\lambda & 0\\ 0 & \theta & \mu\ 0 & -q-d_2-\mu-n-\lambda \end{vmatrix}=0. \tag{3.6}$$

Thus, the first two eigenvalues obtained are $\lambda_1 = -\mu < 0$, and $\lambda_2 = -\mu < 0$.
Next, consider the rest of characteristic equation, we have $\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$, where

$$
\begin{aligned}
a_1 &= \alpha + 4\mu + d_1 + \theta + p + m + q + d_2 + n > 0,\\
a_2 &= -p\alpha(1-\epsilon) - \theta wq + (\alpha+\mu)(\mu + d_1 + \theta)\Big(1 - R_0\Big) + (p+m+\mu)(q + d_2 + \mu + n)\\
&\quad + (p + m + 2\mu + q + d_2 + n)(\alpha + 2\mu + d_1 + \theta)\\
a_3 &= -p\alpha(1-\epsilon)(d_1 + \theta + q + d_2 + n + 2\mu) - \theta wq(p + m + \mu) - \theta wq(\alpha + \mu)\\
&\quad + (p + m + 2\mu + q + d_2 + n)(\alpha + \mu)(\mu + d_1 + \theta) - (p + m + 2\mu + q + d_2 + n)\frac{\alpha\epsilon\beta\Lambda}{\mu}\\
&\quad + (p + m + \mu)(q + d_2 + \mu + n)(\alpha + 2\mu + d_1 + \theta) - \theta\alpha\epsilon(1-w)q\\
a_4 &= -p\alpha(1-\epsilon)(\mu + d_1 + \theta)(q + d_2 + \mu + n) + p\alpha\theta(1-\epsilon)wq - \theta wq(p + m + \mu)(\alpha + \mu)\\
&\quad - \theta\alpha\epsilon(p + m + \mu)(1-w)q + (p + m + \mu)(q + d_2 + \mu + n)(\alpha + \mu)(\mu + d_1 + \theta)\\
&\quad - (p + m + \mu)(q + d_2 + \mu + n)\frac{\alpha\epsilon\beta\Lambda}{\mu}\\
&= -p\alpha(1-\epsilon)(\mu + d_1 + \theta)(q + d_2 + \mu + n) + p\alpha\theta(1-\epsilon)wq - \theta wq(p + m + \mu)(\alpha + \mu)\\
&\quad - \theta\alpha\epsilon(p + m + \mu)(1-w)q + (p + m + \mu)(q + d_2 + \mu + n)(\alpha + \mu)(\mu + d_1 + \theta)(1 - R_0).
\end{aligned}
$$

Hence, by the Routh-Hurwitz Criterion, the disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$ and if it satisfies $a_3 > 0, a_4 > 0$ and $a_1a_2a_3 > a_3^2 + a_1^2a_4$. This completes the proof. □

3.4.2. *Local stability of the endemic equilibrium point.*

Theorem 3.3. *When $R_0 > 1$, the endemic equilibrium point (E_1) is locally stable if it satisfies the Routh-Hurwitz criteria.*

Proof. The Jacobian matrix of the system (2.1) at E_1 is

$$
J(E_1) = \begin{bmatrix}
-\beta I^* - \mu & 0 & -\beta S^* & 0 & 0 & 0\\
\beta I^* & -\alpha - \mu & \beta S^* & p & (1-w)q & 0\\
0 & \alpha\epsilon & -\mu - d_1 - \theta & 0 & wq & 0\\
0 & \alpha(1-\epsilon) & 0 & -p - m - \mu & 0 & 0\\
0 & 0 & \theta & 0 & -q - d_2 - \mu - n & 0\\
0 & 0 & 0 & m & n & -\mu
\end{bmatrix}. \tag{3.7}
$$

Consider, $\det(J(E_1) - \lambda I) = 0$, we have

$$(-\mu-\lambda)\begin{vmatrix} -\beta I^*-\mu-\lambda & 0 & -\beta S^* & 0 & 0 \\ \beta I^* & -\alpha-\mu-\lambda & \beta S^* & p & (1-w)q \\ 0 & \alpha\epsilon & -\mu-d_1-\theta-\lambda & 0 & wq \\ 0 & \alpha(1-\epsilon) & 0 & -p-m-\mu-\lambda & 0 \\ 0 & 0 & \theta & 0 & -q-d_2-\mu-n-\lambda \end{vmatrix} = 0. \quad (3.8)$$

Thus, the first eigenvalue is $\lambda_1 = -\mu < 0$.
Next, consider the rest of characteristic equation, we have $\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0$, where

$$\begin{aligned}
a_1 &= B_4 + B_6 + B_1, \\
a_2 &= B_1B_6 + B_1B_4 - p\alpha(1-\epsilon) - wq\theta + B_7 - \alpha\epsilon\beta S^* + B_4B_6 + B_5, \\
a_3 &= \beta^2 I^*\alpha\epsilon S^* + B_5B_6 + B_4B_7 - B_4\alpha\epsilon\beta S^* - \alpha\epsilon\theta(1-w)q - \theta wq(\alpha+\mu) - \theta wqB_8 - p\alpha(1-\epsilon)B_4 \\
&\quad + B_1B_5 + B_1B_4B_6 + B_1B_7 - B_1\alpha\epsilon\beta S^* - B_1\theta wq - B_1p\alpha(1-\epsilon), \\
a_4 &= -p\alpha(1-\epsilon)B_1B_2 - B_1\theta wqB_8 - B_1\theta wq(\alpha+\mu) - \theta\alpha\epsilon B_1(1-w)q - B_1B_4\alpha\epsilon\beta S^* + B_1B_4B_7 \\
&\quad + B_1B_5B_6 - p\alpha(1-\epsilon)B_3 + p\alpha(-\epsilon)\theta wq - \theta wq(\alpha+\mu)B_8 - \theta\alpha\epsilon B_8(1-w)q \\
&\quad + B_5B_7 - B_5\alpha\epsilon\beta S^* + \beta^2 I^*\alpha\epsilon S^*B_9 + beta^2 I^*\alpha\epsilon S^*B_8, \\
a_5 &= -p\alpha B_1B_3 + p\alpha(1-\epsilon)B_1\theta wq - \theta wqB_1(\alpha+\mu)B_8 - \theta\alpha\epsilon B_1B_8(1-w)q + \beta^2 I^*\alpha\epsilon S^*B_8B_9 \\
&\quad + B_1B_5B_7 - B_1B_5\alpha\epsilon\beta S^*.
\end{aligned}$$

Here,

$$\begin{aligned}
&B_1 = (\beta I^* + \mu), B_2 = (d_1 + \theta + q + d_2 + 2\mu + n), B_3 = (\mu + d_1 + \theta)(q + d_2 + \mu + n), \\
&B_4 = (p + m + q + d_2 + 2\mu + n), B_5 = (p + m + \mu)(q + d_2 + \mu + n), B_6 = (\alpha + 2\mu + d_1 + \theta), \\
&B_7 = (\alpha + \mu)(\mu + d_1 + \theta), B_8 = (p + m + \mu), B_9 = (q + d_2 + \mu + n).
\end{aligned}$$

Hence, the endemic equilibrium point is locally asymptotically stable corresponding to the Routh- Hurwitz stability criteria if $a_1a_2a_3 > a_3^2 + a_1a_4$ and $(a_1a_4 - a_5)(a_1a_2a_3 - a_3^2 - a_1^2)a_4 > a_5(a_1a_2 - a_3)^2 + a_1a_5^2$. This complets the proof. □

3.5. Global stability analysis**.**

3.5.1. *Global stability of the disease free equilibrium point.*

Theorem 3.4 *The disease-free equilibrium point* (E_0) *is globally asymptotically stable if* $R_0 < 1$.

Proof. We use a method of Castillo-Chaves et al., 2002 [4] and their concept is given below.
Consider a model system in the form

$$\begin{aligned} \frac{dX_1}{dt} &= F(X_1, X_2), \\ \frac{dX_2}{dt} &= G(X_1, X_2), \quad G(X_1, 0) = 0 \end{aligned} \tag{3.9}$$

where $X_1 \in \mathbb{R}^m$ denotes (its components) the number of uninfected individuals and $X_2 \in \mathbb{R}^m$ denotes (its components) the number of infected individuals including latent, infectious, etc; $X_0 = (X_1^*)$ denotes the disease-free equilibrium of the system. Assume the conditions (H1) and (H2) below:

(H1) For $\frac{dX_1}{dt} = F(X_1, 0), X_1^*$ is globally asymptotically stable,
(H2) $G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2)$, all elements in $\hat{G}(X_1, X_2)$ are nonnegative for $(X_1, X_2) \in \Omega$

where the Jacobian $A = \frac{\partial G}{\partial X_2}(X_1^*, 0)$ is an M-matrix (the off diagonal elements of A are nonnegative) and Ω is the region where the model makes biological sense.

Then the DFE $X_0 = (X_1^*, 0)$ is globally asymptotically stable provided that $R_0 < 1$.

For our model, we first show that the conditions (H1) and (H2) hold when $R_0 < 1$. In system (2.1), we let $X_1 = (S, R)$, $X_2 = (L, I, T_L, T_I)$ and $X_1^* = \left(\frac{\Lambda}{\mu}, 0\right)$.
Then, we have

$$\frac{dX_1}{dt} = F(X_1, X_2) = \begin{bmatrix} \Lambda - \beta SI - \mu S \\ \\ mT_L + nT_I - \mu R \end{bmatrix}. \tag{3.10}$$

And,

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{bmatrix} \Lambda - \mu S \\ \\ -\mu R \end{bmatrix}. \tag{3.11}$$

By integration, we have

$$S = \frac{\Lambda}{\mu} + C_2 e^{-\mu t}. \tag{3.12}$$

Consider when $t \to \infty$, then $S(t) \to \frac{\Lambda}{\mu} = S_0$.
And,

$$R(t) = R(0)e^{-\mu t}. \tag{3.13}$$

Consider when $t \to \infty$, then $R(t) \to 0 = R_0$.
This show that (H1) holds.
Thus, $X_1^* = \left(\frac{\Lambda}{\mu}, 0\right)$ is globally asymptotically stable equilibrium point for the reduced system

$\frac{dX_1}{dt} = F(X_1, 0)$.
Next, consider

$$\frac{dX_2}{dt} = G(X_1, X_2) = \begin{bmatrix} \beta SI + pT_L + (1-w)qT_I - (\alpha+\mu)L \\ \alpha\epsilon L + wqT_I - (\mu + d_1 + \theta)I \\ \alpha(1-\epsilon)L - (p+m+\mu)T_L \\ \theta I - (q + d_2 + \mu + n)T_I \end{bmatrix}. \tag{3.14}$$

We have

$$\frac{dX_2}{dt} = G(X_1, 0) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}. \tag{3.15}$$

Then,

$$\frac{\partial G}{\partial X_2}(X_1^*, 0) = \begin{bmatrix} -(\alpha+\mu) & \frac{\beta\Lambda}{\mu} & p & (1-w)q \\ \alpha\epsilon & -(\mu + d_1 + \theta) & 0 & wq \\ \alpha(1-\epsilon) & 0 & -(p+m+\mu) & 0 \\ 0 & \theta & 0 & -(q + d_2 + \mu + n) \end{bmatrix} = A. \tag{3.16}$$

This is an M-matrix with non-negatives off diagonal elements.
Next, consider
$\widehat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$.

$$\widehat{G}(X_1, X_2) = \begin{bmatrix} \beta I\left(\frac{\Lambda}{\mu} - S\right) \\ 0 \\ 0 \\ 0 \end{bmatrix}. \tag{3.17}$$

Since $S \leq \frac{\Lambda}{\mu}$, then

$$\widehat{G}(X_1, X_2) \geq 0.$$

This shows that (H2) holds.

We can conclude that the disease-free equilibrium point is globally asymptotically stable under these extreme circumstances provided that $R_0 < 1$. □

3.5.2. *Global stability of the endemic equilibrium point.*

The global stability of the endemic equilibrium point is analyzed by using the geometric approach of Li and Muldowney [13, 14]. The concept of the geometric approach of Li and Muldowney is briefly explained below.

Consider the autonomous dynamical system

$$\dot{x} = f(x), \tag{3.18}$$

where $f : \Omega \to \mathbb{R}^n, \Omega \subset \mathbb{R}^n$ open set and $f \in C^1(\Omega)$.

The following assumptions are made:

(H1) Ω is simply connected.

(H2) there is a compact absorbing set $\Gamma \subset \Omega$.

(H3) $\bar{x}$ is the only equilibrium point of (3.18) in Ω.

Here is the result due to Li and Muldowney.

Theorem 3.5 Under the assumptions (H1), (H2) and (H3), the unique equilibrium point $\bar{x}$ of (3.18) is globally asymptotically stable int Ω provided the quantity $\bar{q}_2 < 0$, where

$$\bar{q}_2 = \limsup_{t\to\infty} \sup_{x_0 \in E} \frac{1}{t} \int_0^t v(B(x(s, x_0)))ds.$$

The matrix B is defined as $B = Q_f Q^{-1} + QJ^{[2]}Q^{-1}$, where Q_f is obtained by replacing the entry q_{ij} of Q by its derivative in the direction of solution of f and $J^{[2]}$ is the second additive compound matrix of Jacobian J of the system (3.18).

Further, the $v(B)$ is the *Lozinskiĭ* measure with respect to a vector norm $\|\cdot\|$ in $\mathbb{R}^n$. The following Lemma and Theorem are performed to check the global stability condition of the endemic equilibrium point.

Lemma 3.6 When $R_0 > 1$, the system (2.1) is uniformly persistent in int Ω.

Proof. From Theorem 3.4, we obtain that E_0 is stable when $R_0 < 1$, and when $R_0 > 1$ it is unstable. We conclude that when $R_0 > 1$ the system is uniformly persistent in the interior of Ω i.e., there exists a constant $w > 0$ such that

$$\liminf_{t\to\infty} S(t) > w, \liminf_{t\to\infty} L(t) > w, \liminf_{t\to\infty} I(t) > w, \liminf_{t\to\infty} T_L(t) > w, \liminf_{t\to\infty} T_I(t) > w,$$

$$\liminf_{t\to\infty} R(t) > w,$$

provided $(S(0), L(0), I(0), T_L(0), T_I(0), R(0)) \in \Omega$. This complete the proof.

The uniformly persistence together with boundedness of Ω is equivalent to the existence of a compact set, which is absorbing for our model in the interior of Ω.

Hence, the assumption ***(H1)*** and ***(H2)*** hold.

Theorem 3.7. *The endemic equilibrium point (E_1) is globally asymptotically stable in int (Ω) when $R_0 > 1$ and when $\bar{b} > 0$ ($\bar{b}$ is defined in the proof).*

Proof. From above, we obtain that all assumptions ***(H1) - (H3)*** hold.
First, we consider (S, L, I, T_L) by considering the Jacobian matrix of the first four equations of the system (2.1) as follows:

$$J(S,L,I,T_L) = \begin{bmatrix} -(\beta I+\mu) & 0 & -\beta S & 0 \\ \beta I & -(\alpha+\mu) & \beta S & p \\ 0 & \alpha\epsilon & -(\mu+d_1+\theta) & 0 \\ 0 & \alpha(1-\epsilon) & 0 & -(p+m+\mu) \end{bmatrix}. \tag{3.19}$$

Next, we let

$$M_{11} = \beta I + \mu, M_{22} = \alpha + \mu, M_{33} = \mu + d_1 + \theta \text{ and } M_{44} = p + m + \mu.$$

Then, the second additive compound matrix of (3.19) is

$$J^{[2]} = \begin{bmatrix} -(M_{11}+M_{22}) & \beta S & p & \beta S & 0 & 0 \\ \alpha\epsilon & -(M_{11}+M_{33}) & 0 & 0 & 0 & 0 \\ \alpha(1-\epsilon) & 0 & -(M_{11}+M_{44}) & 0 & 0 & -\beta S \\ 0 & \beta I & 0 & -(M_{22}+M_{33}) & 0 & -p \\ 0 & 0 & \beta I & 0 & -(M_{22}+M_{44}) & \beta S \\ 0 & 0 & 0 & -\alpha(1-\epsilon) & \alpha\epsilon & -(M_{33}+M_{44}) \end{bmatrix}. \tag{3.20}$$

We set the matrix function Q by

$$Q(S,L,I,T_L) = \operatorname{diag}\left\{1,1,1,1,\frac{I}{T_L},\frac{I}{T_L}\right\}. \tag{3.21}$$

Thus,

$$Q_f = \begin{bmatrix} 0&0&0&0&0&0 \\ 0&0&0&0&0&0 \\ 0&0&0&0&0&0 \\ 0&0&0&0&0&0 \\ 0&0&0&0&\frac{T_L I' - I T_L'}{T_L^2}&0 \\ 0&0&0&0&0&\frac{T_L I' - I T_L'}{T_L^2} \end{bmatrix} \text{ and } Q^{-1} = \begin{bmatrix} 1&0&0&0&0&0 \\ 0&1&0&0&0&0 \\ 0&0&1&0&0&0 \\ 0&0&0&1&0&0 \\ 0&0&0&0&\frac{T_L}{I}&0 \\ 0&0&0&0&0&\frac{T_L}{I} \end{bmatrix}. \tag{3.22}$$

Then, we obtain

$$Q_f Q^{-1} = \operatorname{diag}\left\{0,0,0,0,\frac{I'}{I}-\frac{T_L'}{T_L},\frac{I'}{I}-\frac{T_L'}{T_L}\right\}. \tag{3.23}$$

Next, we compute $QJ^{[2]}Q^{-1}$, we have

$$QJ^{[2]}Q^{-1} = \begin{bmatrix} -(M_{11}+M_{22}) & \beta S & p & \beta S & 0 & 0 \\ \alpha\epsilon & -(M_{11}+M_{33}) & 0 & 0 & 0 & 0 \\ \alpha(1-\epsilon) & 0 & -(M_{11}+M_{44}) & 0 & 0 & -\frac{\beta S T_L}{I} \\ 0 & \beta I & 0 & -(M_{22}+M_{33}) & 0 & -\frac{pT_L}{I} \\ 0 & 0 & \frac{\beta I^2}{T_L} & 0 & -(M_{22}+M_{44}) & \beta S \\ 0 & 0 & 0 & -\frac{\alpha(1-\epsilon)I}{T_L} & \alpha\epsilon & -(M_{33}+M_{44}) \end{bmatrix}. \quad (3.24)$$

Since by Theorem 3.5, $B = Q_f Q^{-1} + QJ^{[2]}Q^{-1}$, thus

$$B = \begin{bmatrix} -(M_{11}+M_{22}) & \beta S & p & \beta S & 0 & 0 \\ \alpha\epsilon & -(M_{11}+M_{33}) & 0 & 0 & 0 & 0 \\ \alpha(1-\epsilon) & 0 & -(M_{11}+M_{44}) & 0 & 0 & -\frac{\beta S T_L}{I} \\ 0 & \beta I & 0 & -(M_{22}+M_{33}) & 0 & -\frac{pT_L}{I} \\ 0 & 0 & \frac{\beta I^2}{T_L} & 0 & -(M_{22}+M_{44}) & \beta S \\ 0 & 0 & 0 & -\frac{\alpha(1-\epsilon)I}{T_L} & \alpha\epsilon & -(M_{33}+M_{44}) \end{bmatrix}. \quad (3.25)$$

We then write matrix $B = Q_f Q^{-1} + QJ^{[2]}Q^{-1}$ in a block form:

$$B = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix},$$

where

$$B_{11} = \Big[-(M_{11} + M_{22}) \Big],\ B_{12} = \begin{bmatrix} \beta S & p & \beta S & 0 & 0 \end{bmatrix},$$

$$B_{21} = \begin{bmatrix} \alpha\epsilon \\ \alpha(1-\epsilon) \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

$$B_{22} = \begin{bmatrix} -(M_{11}+M_{33}) & 0 & 0 & 0 & 0 \\ 0 & -(M_{11}+M_{44}) & 0 & 0 & -\frac{\beta S T_L}{I} \\ \beta I & 0 & -(M_{22}+M_{33}) & 0 & -\frac{pT_L}{I} \\ 0 & \frac{\beta I^2}{T_L} & 0 & -(M_{22}+M_{44}) + \left(\frac{I'}{I} - \frac{T_L'}{T_L}\right) & \beta S \\ 0 & 0 & -\frac{\alpha(1-\epsilon)I}{T_L} & \alpha\epsilon & -(M_{33}+M_{44}) + \left(\frac{I'}{I} - \frac{T_L'}{T_L}\right) \end{bmatrix}.$$

The *Lozinskiĭ* measure of matrix B is defined as $v(B) \leqslant \max\{g_1, g_2\}$, where

$$g_1 = v(B_{11}) + \|B_{12}\|, \text{ and } g_2 = \|B_{21}\| + v(B_{22}).$$

By calculation, then

$$v(B_{11}) = -(M_{11} + M_{22}), \|B_{12}\| = \max\{\beta S, p\}, \text{ and } \|B_{21}\| = \alpha$$

where $v(B_{22})$ is to be determined.
Thus,

$$g_1 = v(B_{11}) + \|B_{12}\| = -(M_{11} + M_{22}) + \max\{\beta S, p\}.$$
$$g_2 = \|B_{21}\| + v(B_{22}) = \alpha + v(B_{22}).$$

Then, we partition matrix B_{22} as

$$B_{22} = F = \begin{bmatrix} F_{11} & F_{12} \\ F_{21} & F_{22} \end{bmatrix},$$

where

$$F_{11} = \Big[-(M_{11} + M_{33}) \Big],\ F_{12} = \begin{bmatrix} 0 & 0 & 0 & 0 \end{bmatrix},$$

$$F_{21} = \begin{bmatrix} 0 \\ \beta I \\ 0 \\ 0 \end{bmatrix},$$

$$F_{22} = \begin{bmatrix} -(M_{11}+M_{44}) & 0 & 0 & -\frac{\beta S T_L}{I} \\ 0 & -(M_{22}+M_{33}) & 0 & -\frac{pT_L}{I} \\ \frac{\beta I^2}{T_L} & 0 & -(M_{22}+M_{44})+\left(\frac{I'}{I}-\frac{T_L'}{T_L}\right) & \beta S \\ 0 & -\frac{\alpha(1-\epsilon)I}{T_L} & \alpha\epsilon & -(M_{33}+M_{44})+\left(\frac{I'}{I}-\frac{T_L'}{T_L}\right) \end{bmatrix}.$$

As above, we define the *Lozinskiĭ* measure of matrix F as $v(F) \leqslant \max\{g_3, g_4\}$, where $g_3 = v(F_{11}) + \|F_{12}\|$ and $g_4 = \|F_{21}\| + v(F_{22})$.
Then,

$$v(F_{11}) = -(M_{11}+M_{33}), \|F_{12}\| = 0, \text{ and } \|F_{21}\| = \beta I,$$

where $v(F_{22})$ is to be determined.
Therefore, we have

$$g_3 = v(F_{11}) + \|F_{12}\| = -(M_{11}+M_{33}) + 0,$$
$$g_4 = \|F_{21}\| + v(F_{22}) = \beta I + v(F_{22}).$$

And, the matrix F_{22} is partitioned as

$$F_{22} = G = \begin{bmatrix} G_{11} & G_{12} \\ G_{21} & G_{22} \end{bmatrix},$$

where

$$G_{11} = \Big[-(M_{11}+M_{44}) \Big],\ G_{12} = \begin{bmatrix} 0 & 0 & -\frac{\beta S T_L}{I} \end{bmatrix},$$

$$G_{21} = \begin{bmatrix} 0 \\ \frac{\beta I^2}{T_L} \\ 0 \end{bmatrix},$$

$$G_{22} = \begin{bmatrix} -(M_{22}+M_{33}) & 0 & -\frac{pT_L}{I} \\ 0 & -(M_{22}+M_{44}) + \left(\frac{I'}{I} - \frac{T_L'}{T_L}\right) & \beta S \\ -\frac{\alpha(1-\epsilon)I}{T_L} & \alpha\epsilon & -(M_{33}+M_{44}) + \left(\frac{I'}{I} - \frac{T_L'}{T_L}\right) \end{bmatrix}.$$

Now, the *Lozinskiĭ* measure of matrix G is defined as $v(G) \leq \max\{g_5, g_6\}$,
where $g_5 = v(G_{11}) + \|G_{12}\|$ and $g_6 = \|G_{21}\| + v(G_{22})$.
Then,

$$v(G_{11}) = -(M_{11}+M_{44}), \|G_{12}\| = \frac{\beta S T_L}{I}, \text{ and } \|G_{21}\| = \frac{\beta I^2}{T_L},$$

and $v(G_{22})$ is to be determined.
Thus, we have

$$g_5 = v(G_{11}) + \|G_{12}\| = -(M_{11}+M_{44}) + \frac{\beta S T_L}{I},$$
$$g_6 = \|G_{21}\| + v(G_{22}) = \frac{\beta I^2}{T_L} + v(G_{22}).$$

The matrix G_{22} is then partitioned as

$$G_{22} = H = \begin{bmatrix} H_{11} & H_{12} \\ H_{21} & H_{22}, \end{bmatrix}$$

where

$$H_{11} = \Big[-(M_{22}+M_{33}) \Big], \; H_{12} = \begin{bmatrix} 0 & -\frac{pT_L}{I} \end{bmatrix},$$
$$H_{21} = \begin{bmatrix} 0 \\ -\frac{\alpha(1-\epsilon)I}{T_L} \end{bmatrix},$$
$$H_{22} = \begin{bmatrix} -(M_{22}+M_{44}) + \left(\frac{I'}{I} - \frac{T_L'}{T_L}\right) & \beta S \\ \alpha\epsilon & -(M_{33}+M_{44}) + \left(\frac{I'}{I} - \frac{T_L'}{T_L}\right) \end{bmatrix}.$$

The *Lozinskii* measure of matrix H is defined as $v(H) \leq \max\{g_7, g_8\}$,
where $g_7 = v(H_{11}) + \|H_{12}\|$ and $g_8 = \|H_{21}\| + v(H_{22})$.
Thus,

$$v(H_{11}) = -(M_{22}+M_{33}), \|H_{12}\| = \frac{pT_L}{I}, \text{ and } \|H_{21}\| = \frac{\alpha(1-\epsilon)I}{T_L}.$$

Then,

$$\begin{aligned} v(H_{22}) &= \max\{-(M_{22}+M_{44})+\left(\frac{I^{'}}{I}-\frac{T_L^{'}}{T_L}\right)+\alpha\epsilon, -(M_{33}+M_{44})+\left(\frac{I^{'}}{I}-\frac{T_L^{'}}{T_L}\right)+\beta S\} \\ &= \max\{-(\alpha+p+m+2\mu)+\left(\frac{I^{'}}{I}-\frac{T_L^{'}}{T_L}\right)+\alpha\epsilon, -(d_1+\theta+p+m+2\mu) \\ &+\left(\frac{I^{'}}{I}-\frac{T_L^{'}}{T_L}\right)+\beta S\}. \end{aligned} \tag{3.26}$$

From the fourth equation of system (2.1), we have

$$\begin{aligned} \frac{dT_L}{dt} &= \alpha(1-\epsilon)L - pT_L - mT_L - \mu T_L \\ \frac{T_L^{'}}{T_L} &= \frac{\alpha(1-\epsilon)L}{T_L} - (p+m+\mu) \\ \frac{T_L^{'}}{T_L} + p + m + \mu &= \frac{\alpha(1-\epsilon)L}{T_L}. \end{aligned} \tag{3.27}$$

Substitute above expression in $v(H_{22})$, we have

$$v(H_{22}) = \max\{\alpha\epsilon-(\alpha+\mu)+\frac{I^{'}}{I}-\frac{\alpha(1-\epsilon)L}{T_L}, \beta S-(d_1+\theta+\mu)+\frac{I^{'}}{I}-\frac{\alpha(1-\epsilon)L}{T_L}\}. \tag{3.28}$$

Therefore, we have

$$\begin{aligned} g_7 &= v(H_{11})+\|H_{12}\| = -(M_{22}+M_{33})+\frac{pT_L}{I} = -(\alpha+2\mu+d_1+\theta)+\frac{pT_L}{I} \\ g_8 &= \|H_{21}\|+v(H_{22}) \\ &= \frac{\alpha(1-\epsilon)I}{T_L}+\max\{\alpha\epsilon-(\alpha+\mu)+\frac{I^{'}}{I}-\frac{\alpha(1-\epsilon)L}{T_L}, \beta S-(d_1+\theta+\mu)+\frac{I^{'}}{I}-\frac{\alpha(1-\epsilon)L}{T_L}\} \\ &= \frac{\alpha(1-\epsilon)I}{T_L}+\frac{I^{'}}{I}+\max\{\alpha\epsilon-(\alpha+\mu)-\frac{\alpha(1-\epsilon)L}{T_L}, \beta S-(d_1+\theta+\mu)-\frac{\alpha(1-\epsilon)L}{T_L}\}. \end{aligned}$$

From the third equation of system (2.1), we have

$$\begin{aligned} \frac{dI}{dt} &= \alpha\epsilon L + wqT_I - \mu I - d_1 I - \theta I \\ \frac{I^{'}}{I} &= \frac{\alpha\epsilon L}{I}+\frac{wqT_I}{I}-(\mu+d_1+\theta) \\ \frac{I^{'}}{I}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I} &= -(\mu+d_1+\theta). \end{aligned} \tag{3.29}$$

We then consider

$$\begin{aligned} g_7 &= -(\alpha+2\mu+d_1+\theta)+\frac{pT_L}{I} \\ &= \frac{I^{'}}{I}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I}-(\alpha+\mu)+\frac{pT_L}{I}. \end{aligned}$$

Therefore,

$$\begin{aligned} v(H) &\leq \max\{g_7, g_8\} \\ &\leq \frac{I^{'}}{I} + \max\Bigg\{ -\frac{\alpha\epsilon L}{I} - \frac{wqT_I}{I} - (\alpha+\mu) + \frac{pT_L}{I}, \frac{\alpha(1-\epsilon)I}{T_L} \\ &+ \sup\Bigg\{\alpha\epsilon - (\alpha+\mu) - \frac{\alpha(1-\epsilon)L}{T_L}, \beta S - (d_1+\theta+\mu) - \frac{\alpha(1-\epsilon)L}{T_L}\Bigg\}\Bigg\}. \end{aligned} \tag{3.30}$$

Next, since $v(G) \leq \max\{g_5, g_6\}$,
where

$$g_5 = -(M_{11}+M_{44}) + \frac{\beta S T_L}{I} \text{ and } g_6 = \frac{\beta I^2}{T_L} + v(G_{22}).$$

Then,

$$\begin{aligned} g_5 &= -(M_{11}+M_{44}) + \frac{\beta S T_L}{I} \\ &= -(M_{11}+M_{44}) + \frac{I^{'}}{I} - \frac{I^{'}}{I} + \frac{\beta S T_L}{I} \\ &= \frac{I^{'}}{I} - (\beta S + p + m + \mu) + \frac{\beta S T_L}{I} - \frac{\alpha\epsilon L}{I} - \frac{wqT_I}{I} + d_1 + \theta. \end{aligned}$$

Therefore,

$$\begin{aligned} v(G) \leq \frac{I^{'}}{I} + \max\Bigg\{ & d_1 + \theta - (\beta I + p + m + \mu) + \frac{\beta S T_L}{I} - \frac{\alpha\epsilon L}{I} - \frac{wqT_I}{I}, \\ & \frac{\beta I^2}{T_L} - \frac{\alpha\epsilon L}{I} - \frac{wqT_I}{I} - (\alpha+\mu) + \frac{pT_L}{I}, \frac{\beta I^2}{T_L} + \frac{\alpha(1-\epsilon)I}{T_L} \\ & + \sup\Bigg\{\alpha\epsilon - (\alpha+\mu) - \frac{\alpha(1-\epsilon)L}{T_L}, \beta S - (d_1+\theta+\mu) - \frac{\alpha(1-\epsilon)L}{T_L}\Bigg\}\Bigg\}. \end{aligned} \tag{3.31}$$

Next, since $v(F) \leq \max\{g_3, g_4\}$,
where $g_3 = -(M_{11}+M_{33})$ and $g_4 = \beta I + v(F_{22})$.
Then,

$$\begin{aligned} g_3 &= -(M_{11}+M_{33}) \\ &= -(M_{11}+M_{33}) + \frac{I^{'}}{I} - \frac{I^{'}}{I} \\ &= \frac{I^{'}}{I} - (\beta I + \mu) - \frac{\alpha\epsilon L}{I} - \frac{wqT_I}{I}. \end{aligned}$$

Therefore,

$$v(F) \leq \frac{I^{'}}{I} + \max\Bigg\{ -(\beta I + \mu) - \frac{\alpha\epsilon L}{I} - \frac{wqT_I}{I}, \beta I + d_1 + \theta - (\beta I + p + m + \mu)$$

$$+\frac{\beta ST_L}{I}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I},\beta I+\frac{\beta I^2}{T_L}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I}-(\alpha+\mu)+\frac{pT_L}{I},$$
$$\beta I+\frac{\beta I^2}{T_L}+\frac{\alpha(1-\epsilon)I}{T_L}+\sup\left\{\alpha\epsilon-(\alpha+\mu)-\frac{\alpha(1-\epsilon)L}{T_L},\right.$$
$$\left.\left.\beta S-(d_1+\theta+\mu)-\frac{\alpha(1-\epsilon)L}{T_L}\right\}\right\}. \tag{3.32}$$

Next, since $v(B)\leq \max\{g_1,g_2\}$,
where $g_1=-(M_{11}+M_{22})+\max\{\beta S,p\}$ and $g_2=\alpha+v(B_{22})$.

We then have

$$\begin{aligned}
g_1&=-(M_{11}+M_{22})+\max\{\beta S,p\}\\
&=-(M_{11}+M_{22})+\max\{\beta S,p\}+\frac{I^{'}}{I}-\frac{I^{'}}{I}\\
&=\frac{I^{'}}{I}-(\beta I+\alpha+\mu)+\max\left\{\beta S,p\right\}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I}+d_1+\theta.
\end{aligned}$$

Therefore,

$$\begin{aligned}
v(B)\leq\ &\frac{I^{'}}{I}+\max\left\{-(\beta I+\alpha+\mu)+\sup\left\{\beta S,p\right\}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I}+d_1+\theta,\right.\\
&\alpha-(\beta I+\mu)-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I},\alpha+d_1+\theta-(p+m+\mu)+\frac{\beta ST_L}{I}\\
&-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I},\beta I+\frac{\beta I^2}{T_L}-\frac{\alpha\epsilon L}{I}-\frac{wqT_I}{I}-\mu+\frac{pT_L}{I},\alpha+\beta I+\frac{\beta I^2}{T_L}\\
&\left.+\frac{\alpha(1-\epsilon)I}{T_L}+\sup\left\{\alpha\epsilon-(\alpha+\mu)-\frac{\alpha(1-\epsilon)L}{T_L},\beta S-(d_1+\theta+\mu)-\frac{\alpha(1-\epsilon)L}{T_L}\right\}\right\}.\\
&=\frac{I^{'}}{I}-\bar{b}
\end{aligned}$$

where

$$\begin{aligned}
\bar{b}=\min\Bigg\{&\beta I+\alpha+\mu-\inf\left\{-\beta S,-p\right\}+\frac{\alpha\epsilon L}{I}+\frac{wqT_I}{I}-d_1-\theta,\\
&-\alpha+\beta I+\mu+\frac{\alpha\epsilon L}{I}+\frac{wqT_I}{I},p+m+\mu+\frac{\alpha\epsilon L}{I}+\frac{wqT_I}{I}-\frac{\beta ST_L}{I}\\
&-\alpha-d_1-\theta,\frac{\alpha\epsilon L}{I}+\frac{wqT_I}{I}+\mu-\frac{pT_L}{I}-\beta I-\frac{\beta I^2}{T_L},-\alpha-\beta I-\frac{\beta I^2}{T_L}\\
&-\frac{\alpha(1-\epsilon)I}{T_L}-\inf\left\{-\alpha\epsilon+\alpha+\mu+\frac{\alpha(1-\epsilon)L}{T_L},-\beta S+d_1+\theta+\mu+\frac{\alpha(1-\epsilon)L}{T_L}\right\}\Bigg\}.
\end{aligned} \tag{3.33}$$

Hence, we obtain $v(B)\leq\frac{I^{'}}{I}-\bar{b}$.

Next, consider any solution $S(t),L(t),I(t),T_L(t)$ emanating from the compact absorbing set $\Gamma\subset\Omega$. Let t^* be large enough such that the system is persistent and $(S(t),L(t),I(t),T_L(t))\subset\Gamma$ for

all $t \geq t^*$. Then, along each solution $S(t), L(t), I(t), T_L(t)$ such that $(S(0), L(0), I(0), T_L(0)) \in \Gamma$, for $t > t^*$, $\frac{1}{t}[\ln I(t) - \ln I(0)] < \frac{\bar{b}}{2}$.
Hence,

$$\begin{aligned} \bar{q_2} = \frac{1}{t}\int_0^t v(B)ds &\leq \frac{1}{t}\int_0^t \left(\frac{I'}{I} - \bar{b}\right)ds \\ &= \frac{1}{t}((\ln I(t) - \ln I(0)) - \bar{b}t) \\ &= \left(\frac{\ln I(t) - \ln I(0)}{t}\right) - \bar{b} \\ &< -\frac{\bar{b}}{2}, \end{aligned} \tag{3.34}$$

Thus, $q_2 < 0$ when $\bar{b} > 0$.
Hence, by Theorem 3.5, (S, L, I, T_L) is globally asymptotically stable in *int* Ω when $R_0 > 1$ and $\bar{b} > 0$. We next have to proof the remaining two variables i.e., T_I and R.
Next, consider the fifth equation of the system (2.1),

$$\begin{aligned} \frac{dT_I}{dt} &= \theta I - (1-w)qT_I - wqT_I - d_2T_I - \mu T_I - nT_I \\ &= \theta I - (q + d_2 + \mu + n)T_I, \end{aligned} \tag{3.35}$$

and its limit system is

$$\frac{dT_I}{dt} = \theta I^* - (q + d_2 + \mu + n)T_I, \text{ where } I^* \text{ is a constant.}$$

Since $\theta I^* = (q + d_2 + \mu + n)T_I^*$, we get

$$\frac{dT_I}{dt} = (q + d_2 + \mu + n)T_I^* - (q + d_2 + \mu + n)T_I. \tag{3.36}$$

By using integrating factor method to solve equation (3.36), we obtain

$$T_I = T_I^* + Ce^{-(q+d_2+\mu+n)t}. \tag{3.37}$$

By taking $t \longrightarrow \infty$, we have $\lim_{t\to\infty} T_I(t) = T_I^*$.
Consider the sixth equation of the system (2.1),

$$\frac{dR}{dt} = mT_L + nT_I - \mu R, \tag{3.38}$$

and its limit system is

$$\frac{dR}{dt} = mT_L^* + nT_I^* - \mu R, \text{ where } T_L^* \textit{ and } T_I^* \text{ are constants.}$$

Since $mT_L^* + nT_I^* = \mu R^*$, we get

$$\frac{dR}{dt} = \mu R^* - \mu R. \tag{3.39}$$

TABLE 1. **Numerical values of sensitivity indices of R_0**

Parameters	Index at Parameter Value	Sign
Λ	$+1.0000$	positive
β	$+1.0000$	positive
ϵ	$+1.0000$	positive
α	$+0.3000$	positive
μ	-0.2000	negative
d_1	-0.2000	negative
θ	-0.6000	negative

Again, by using integrating factor method to solve equation (3.39), we obtain

$$R = R^* + Ce^{-\mu t}. \tag{3.40}$$

By taking $t \longrightarrow \infty$, we have $\lim_{t\to\infty} R(t) = R^*$.
Therefore, with all results above E_1 is globally asymptotically stable when $R_0 > 1$, and $\bar{b} > 0$. This completes the proof. □

3.6. Sensitivity analysis**.**

To check what parameter have positive or negative effect to R_0, the sensitivity indices are calculated. We use the technique of the normalized forward sensitivity index [10]. The sensitivity indice to each of the parameter is

$$r_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0}. \tag{3.41}$$

The values of sensitivity indices for our model are presented in Table 1. They are calculated by using all parameters values in Table 2.

Table 1 shows that increasing the values of Λ, β, ϵ and α by 10%, the value of R_0 will increase by 10.00%, 10.00%, 10.00%, and 3.00%, respectively. On the contrary, increasing the value of μ, d_1 and θ by 10%, the value of R_0 will decrease by 2.00%, 2.00%, and 6.00%, respectively. Therefore, fo reduce the value of R_0, one could possibly reduce the value of TB transmission rate (β), and increase treatment rate of active TB individuals (θ). These two parameters are two factors that we could control and affects the value of R_0 the most. The normalized sensitivity are shown in Tabel 1.

TABLE 2. **Parameters values used in numerical study**

Parameter	Description	Value	Reference
Λ	The recruitment rate	4	Assume
β	Tuberculosis transmissionrate from active tuberculosis individuals	0.1	Ullah et al., 2020
μ	The natural death rate of population	0.1	Ullah et al., 2020
α	The transferred rate at which latent individuals leave the latent stage	0.25	Ullah et al., 2020
ϵ	The fraction of latent individuals who become active TB individuals	0.7	Assume
$1-\epsilon$	The fraction latent individuals who get treatment	0.3	Assume
θ	The treatment rate of active TB individuals	0.35	Yavuz et al., 2023
m	The recovery rate of treated latent individuals	0.5	Yavuz et al., 2023
n	The recovery rate of treated active TB individuals	0.06	Yavuz et al., 2023
d_1	The disease induced dealh rate of active TB individuals	0.1	Ullah et al., 2020
d_2	The disease induced dealh rate of treated active TB individuals	0.05	Ullah et al., 2020
q	The rate at which treated active TB individuals leaves compartment	0.1	Assume
w	The fraction of treated active TB individuals reenter active TB class due to incomplete treatment	0.7	Assume
$1-w$	The fraction of treated active TB individuals reenter latent class due to incomplete treatment	0.8	Assume
p	The rate at which treated latent individuals reenter latent class due to incomplete treatment	0.5	Assume

4. Optimal Control

In this section, with the method of Pontryagin et al., 1962 [21] we extend system (2.1) by applying optimal control variables in the model in order to determine the best intervention strategies that helps eradicating the tuberculosis transmission in the specified time.
The optimal control model includes four control variables defined as:
(i) $u_1(t)$ is the preventive control.
(ii) $u_2(t)$ is the screening and put under treatment.
(iii) $u_3(t)$ is the treatment effort for active TB.
(iv) $u_4(t)$ is the campaign to make sure patients obtain complete treatment.
The schematic diagram for the optimal control model is shown in Figure 2.

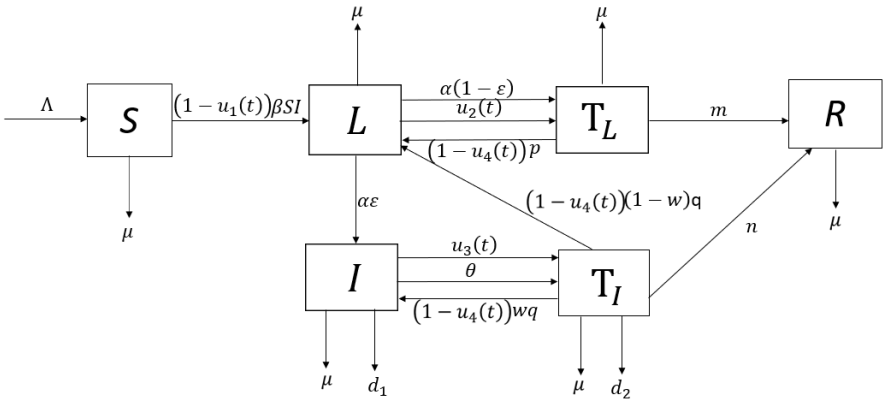


Figure 2. A schematic diagram for the optimal control model of tuberculosis with incomplete treatment.

The model above can be written as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (1-u_1(t))\beta SI - \mu S,\\
\frac{dL}{dt} &= (1-u_1(t))\beta SI + (1-u_4(t))pT_L + (1-u_4(t))(1-w)qT_I\\
&\quad - \alpha\epsilon L - \mu L - u_2(t)L - \alpha(1-\epsilon)L,\\
\frac{dI}{dt} &= \alpha\epsilon L + (1-u_4(t))wqT_I - \mu I - d_1 I - u_3(t)I - \theta I,\\
\frac{dT_L}{dt} &= \alpha(1-\epsilon)L - (1-u_4(t))pT_L - mT_L - \mu T_L + u_2(t)L,\\
\frac{dT_I}{dt} &= \theta I - (1-u_4(t))(1-w)qT_I - (1-u_4(t))wqT_I - d_2 T_I - \mu T_I - nT_I + u_3(t)I,\\
\frac{dR}{dt} &= mT_L + nT_I - \mu R.
\end{aligned} \tag{4.1}$$

Our goal is to reduce the number of latent individuals (L), the number of active TB individuals (I), the number of treated latent individuals (T_L), the number of treated active TB individuals (T_I) by all four controls mentioned above. We first construct an objective function of the model as follows:

$$J(u_1,u_2,u_3,u_4) = \min \int_0^T \Big[W_1L(t) + W_2I(t) + W_3T_L(t) + W_4T_I(t) + \frac{1}{2}(W_5u_1^2(t) + W_6u_2^2(t) + W_7u_3^2(t) + W_8u_4^2(t))\Big]dt, \tag{4.2}$$

with initial conditions

$$S(0) \geq 0,\ L(0) \geq 0,\ I(0) \geq 0,\ T_L(0) \geq 0,\ T_I(0) \geq 0 \text{ and } R(0) \geq 0.$$

Here the constants $W_1, W_2, W_3, W_3, W_4, W_5, W_6, W_7$ and W_8 are weight constants and the terms $W_5u_1^2(t), W_6u_2^2(t), W_7u_3^2(t)$ and $W_8u_4^2(t)$ represent the costs associated with preventive control, screening put under treatment, treatment effort for active TB and campaign to make sure patients obtain complete treatment, respectively. We can determine an optimal solution of this optimal control problem by considering the Lagrangian and the Hamiltonian for the problem.

The Lagrangian of the optimal control problem is given by

$$f(L,I,T_L,T_I,u_1,u_2,u_3,u_4) = W_1L(t) + W_2I(t) + W_3T_L(t) + W_4T_I(t) + \frac{1}{2}\Big(W_5u_1^2(t) + W_6u_2^2(t) + W_7u_3^2(t) + W_8u_4^2(t)\Big). \tag{4.3}$$

Applying Pontryagin's Minimum Principle (PMP), we form the Hamiltonian and derive the optimality system:

$$\begin{aligned} H = {} & W_1L(t) + W_2I(t) + W_3T_L(t) + W_4T_I(t) + \frac{1}{2}\Big(W_5u_1^2(t) + W_6u_2^2(t) \\ & + W_7u_3^2(t) + W_8u_4^2(t)\Big) \\ & + \lambda_S[\Lambda - (1-u_1(t))\beta SI - \mu S] \\ & + \lambda_L[(1-u_1(t))\beta SI + (1-u_4(t))pT_L + (1-u_4(t))(1-w)qT_I \\ & - \alpha\epsilon L - \mu L - u_2(t)L - \alpha(1-\epsilon)L] \\ & + \lambda_I[\alpha\epsilon L + (1-u_4(t))wqT_I - \mu I - d_1I - u_3(t)I - \theta I] \\ & + \lambda_{T_L}[\alpha(1-\epsilon)L - (1-u_4(t))pT_L - mT_L + u_2(t)L - \mu T_L] \\ & + \lambda_{T_I}[\theta I - (1-u_4(t))(1-w)qT_I - (1-u_4(t))wqT_I - d_2T_I - \mu T_I \\ & - nT_I + u_3(t)I] \\ & + \lambda_R[mT_L + nT_I - \mu R], \end{aligned} \tag{4.4}$$

where $\lambda_S, \lambda_L, \lambda_I, \lambda_{T_L}, \lambda_{T_I}$ and λ_R are the adjoint functions associated with the state equations for S, L, I, T_L, T_I and R, respectively.

Theorem 3.8 Let $\widetilde{S},\widetilde{L},\widetilde{I},\widetilde{T_L},\widetilde{T_I}$ and $\widetilde{R}$ be optimal state solution with associated optimal control variable $u_1^*(t)$, $u_2^*(t)$, $u_3^*(t)$ and $u_4^*(t)$ for the optimal control problem (4.1). Then there exist adjoint variables $\lambda_S,\lambda_L,\lambda_I,\lambda_{T_L},\lambda_{T_I}$ and λ_R satisfying:

$$\begin{aligned}
\lambda_S^{'} &= -\Big[-((1-u_1(t))\beta\widetilde{I}+\mu)\lambda_S+(1-u_1(t))\beta\widetilde{I}\lambda_L\Big]\\
\lambda_L^{'} &= -\Big[W_1-(\alpha+\mu)\lambda_L+\alpha\epsilon\lambda_I-u_2(t)\lambda_L+\alpha(1-\epsilon)\lambda_{T_L}+u_2(t)\lambda_{T_L}\Big]\\
\lambda_I^{'} &= -\Big[W_2-(1-u_1(t))\beta\widetilde{S}\lambda_S+(1-u_1(t))\beta\widetilde{S}\lambda_L-(\mu+d_1+u_3(t)+\theta)\lambda_I+(\theta+u_3(t))\lambda_{T_I}\Big]\\
\lambda_{T_L}^{'} &= -\Big[W_3-(1-u_4(t))p\lambda_{T_L}-(m+\mu)\lambda_{T_L}+(1-u_4(t))p\lambda_L+m\lambda_R\Big]\\
\lambda_{T_I}^{'} &= -\Big[W_4-(1-u_4(t))(1-w)q\lambda_{T_I}-(1-u_4(t))wq\lambda_{T_I}-(d_2+\mu+n)\lambda_{T_I}+n\lambda_R\\
&\quad+(1-u_4(t))(1-w)q\lambda_L+(1-u_4(t))wq\lambda_I\Big]\\
\lambda_R^{'} &= -\Big[-\mu\lambda_R\Big],
\end{aligned}$$

with transversality conditions:

$$\lambda_S(T)=0,\ \lambda_L(T)=0,\ \lambda_I(T)=0,\ \lambda_{T_L}(T)=0,\ \lambda_{T_I}(T)=0 \text{ and } \lambda_R(T)=0.$$

Furthermore, the optimal control variable $u_1^*(t)$, $u_2^*(t)$, $u_3^*(t)$ and $u_4^*(t)$ are given by

$$\begin{aligned}
u_1^*(t) &= \max\left\{0,\min\left\{\frac{\big(\lambda_L-\lambda_S\big)\beta\widetilde{SI}}{W_5},u_{1\max}\right\}\right\},\\
u_2^*(t) &= \max\left\{0,\min\left\{\frac{\big(\lambda_L-\lambda_{T_L}\big)\widetilde{L}}{W_6},u_{2\max}\right\}\right\},\\
u_3^*(t) &= \max\left\{0,\min\left\{\frac{\big(\lambda_I-\lambda_{T_I}\big)\widetilde{I}}{W_7},u_{3\max}\right\}\right\},\\
u_4^*(t) &= \max\left\{0,\min\left\{\frac{\big(\lambda_L-\lambda_{T_L}\big)p\widetilde{T_L}+\big(\lambda_L-\lambda_{T_I}\big)(1-w)q\widetilde{T_I}+\big(\lambda_I-\lambda_{T_I}\big)wq\widetilde{T_I}}{W_8},u_{4\max}\right\}\right\}.
\end{aligned}$$

Proof. The adjoint equations are computed and the transversality conditions. From Hamiltonian, and by setting $S(t)=\widetilde{S},L(t)=\widetilde{L},I(t)=\widetilde{I},T_L(t)=\widetilde{T_L},T_I(t)=\widetilde{T_I}$ and $R(t)=\widetilde{R}$, we obtain

$$\begin{aligned}
\lambda_S^{'} &= -\frac{\partial H}{\partial S} = -\Big[-((1-u_1(t))\beta\widetilde{I}+\mu)\lambda_S+(1-u_1(t))\beta\widetilde{I}\lambda_L\Big]\\
\lambda_L^{'} &= -\frac{\partial H}{\partial L} = -\Big[W_1-(\alpha+\mu)\lambda_L+\alpha\epsilon\lambda_I-u_2(t)\lambda_L+\alpha(1-\epsilon)\lambda_{T_L}+u_2(t)\lambda_{T_L}\Big]\\
\lambda_I^{'} &= -\frac{\partial H}{\partial I} = -\Big[W_2-(1-u_1(t))\beta\widetilde{S}\lambda_S+(1-u_1(t))\beta\widetilde{S}\lambda_L-(\mu+d_1+u_3(t)+\theta)\lambda_I\\
&\qquad+(\theta+u_3(t))\lambda_{T_I}\Big]\\
\lambda_{T_L}^{'} &= -\frac{\partial H}{\partial T_L} = -\Big[W_3-(1-u_4(t))p\lambda_{T_L}-(m+\mu)\lambda_{T_L}+(1-u_4(t))p\lambda_L+m\lambda_R\Big]\\
\lambda_{T_I}^{'} &= -\frac{\partial H}{\partial T_I} = -\Big[W_4-(1-u_4(t))(1-w)q\lambda_{T_I}-(1-u_4(t))wq\lambda_{T_I}-(d_2+\mu+n)\lambda_{T_I}
\end{aligned}$$

$$+ n\lambda_R + (1-u_4(t))(1-w)q\lambda_L + (1-u_4(t))wq\lambda_I\Big]$$

$$\lambda_R^{'} = -\frac{\partial H}{\partial R} = -\Big[-\mu\lambda_R\Big]. \tag{4.5}$$

Similarly, by the approach of Pontryagin et al., 1962 [21], we solve the equation, $\frac{\partial H}{\partial u_i} = 0$ at u_i^*; for $i = 1,2,3,4$ and obtain:

$$\frac{\partial H}{\partial u_1} = W_5 u_1(t) + \beta\widetilde{SI}\lambda_S - \beta\widetilde{SI}\lambda_L = 0$$

$$u_1(t) = \frac{\big(\lambda_L - \lambda_S\big)\beta\widetilde{SI}}{W_5}. \tag{4.6}$$

$$\frac{\partial H}{\partial u_2} = W_6 u_2(t) + \lambda_{T_L}\widetilde{L} - \lambda_L\widetilde{L} = 0$$

$$u_2(t) = \frac{\big(\lambda_L - \lambda_{T_L}\big)\widetilde{L}}{W_6}. \tag{4.7}$$

$$\frac{\partial H}{\partial u_3} = W_7 u_3(t) + \lambda_{T_I}\widetilde{I} - \lambda_I\widetilde{I} = 0$$

$$u_3(t) = \frac{\big(\lambda_I - \lambda_{T_I}\big)\widetilde{I}}{W_7}. \tag{4.8}$$

$$\frac{\partial H}{\partial u_4} = W_8 u_4(t) + \lambda_{T_L} p\widetilde{T_L} - \lambda_L p\widetilde{T_L} + \lambda_{T_I}(1-w)q\widetilde{T_I} + \lambda_{T_I} wq\widetilde{T_I}$$

$$-\lambda_L(1-w)q\widetilde{T_I} - \lambda_I wq\widetilde{T_I} = 0$$

$$u_4(t) = \frac{\big(\lambda_L - \lambda_{T_L}\big)p\widetilde{T_L} + \big(\lambda_L - \lambda_{T_I}\big)(1-w)q\widetilde{T_I} + \big(\lambda_I - \lambda_{T_I}\big)wq\widetilde{T_I}}{W_8}. \tag{4.9}$$

It is subject to the constraint $0 \le u_1(t) \le u_{1\max}$, $0 \le u_2(t) \le u_{2\max}$, $0 \le u_3(t) \le u_{3\max}$ and $0 \le u_4(t) \le u_{4\max}$.

Specifically, we have

$$u_1^*(t) = \max\left\{0, \min\left\{\frac{\big(\lambda_L - \lambda_S\big)\beta\widetilde{SI}}{W_5}, u_{1\max}\right\}\right\},$$

$$u_2^*(t) = \max\left\{0, \min\left\{\frac{\big(\lambda_L - \lambda_{T_L}\big)\widetilde{L}}{W_6}, u_{2\max}\right\}\right\},$$

$$u_3^*(t) = \max\left\{0, \min\left\{\frac{\big(\lambda_I - \lambda_{T_I}\big)\widetilde{I}}{W_7}, u_{3\max}\right\}\right\},$$

$$u_4^*(t) = \max\left\{0, \min\left\{\frac{\big(\lambda_L - \lambda_{T_L}\big)p\widetilde{T_L} + \big(\lambda_L - \lambda_{T_I}\big)(1-w)q\widetilde{T_I} + \big(\lambda_I - \lambda_{T_I}\big)wq\widetilde{T_I}}{W_8}, u_{4\max}\right\}\right\}. \tag{4.10}$$

This completes the proof.

□

5. Numerical simulation of optimal control

Numerical simulations are carried out to illustrate the dynamics of the system (4.1). The control strategies are considered in 5 cases as follows:

Strategy A: $u_1 \neq 0$, $u_2 = u_3 = u_4 = 0$, where $u_{1\max} = 0.8$,

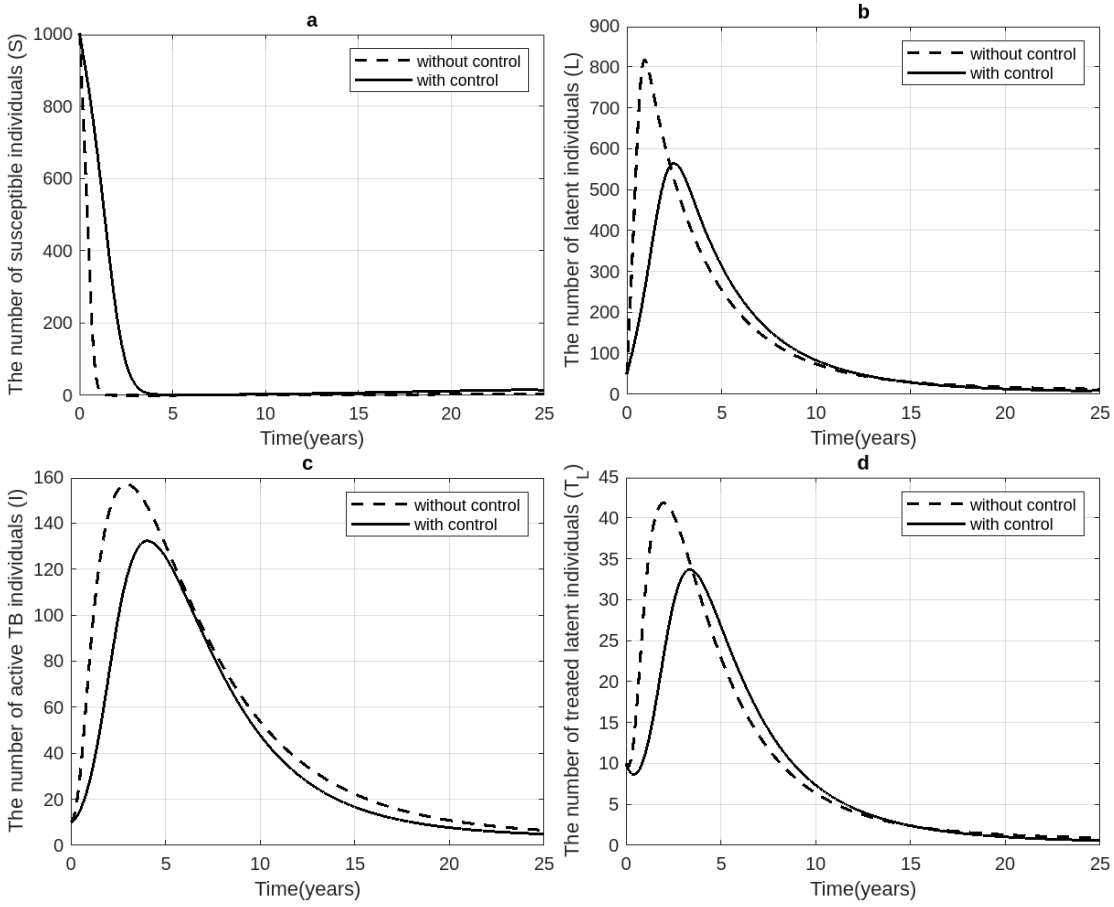
Strategy B: $u_1 = 0$, $u_2 \neq 0$, $u_3 = u_4 = 0$, where $u_{2\max} = 0.8$,

Strategy C: $u_1 = u_2 = 0$, $u_3 \neq 0$, $u_4 = 0$, where $u_{3\max} = 0.8$,

Strategy D: $u_1 = u_2 = u_3 = 0$, $u_4 \neq 0$, where $u_{4\max} = 0.8$,

Strategy E: $u_1 \neq 0$, $u_2 \neq 0$, $u_3 \neq 0$, $u_4 \neq 0$, where $u_{1\max} = u_{2\max} = u_{3\max} = u_{4\max} = 0.8$.

5.1. **Strategy A: The control by the preventive control only.**



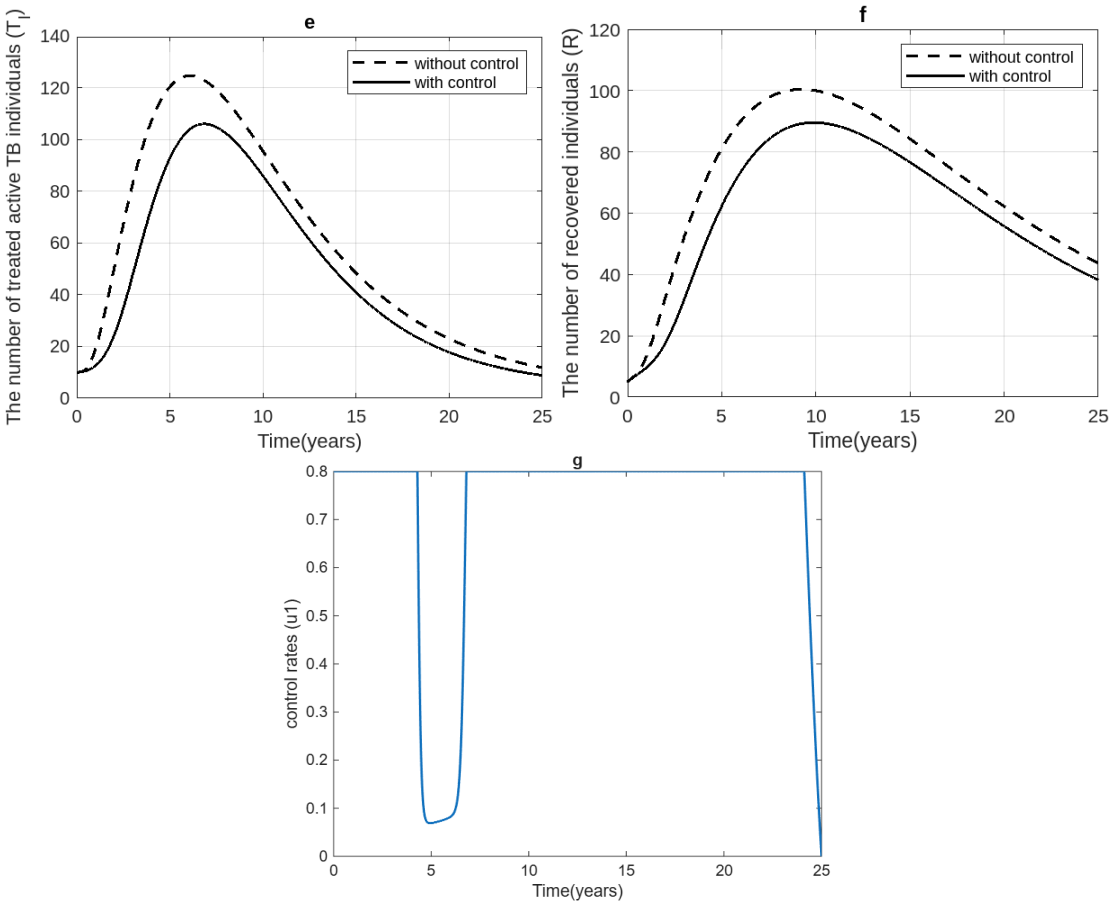
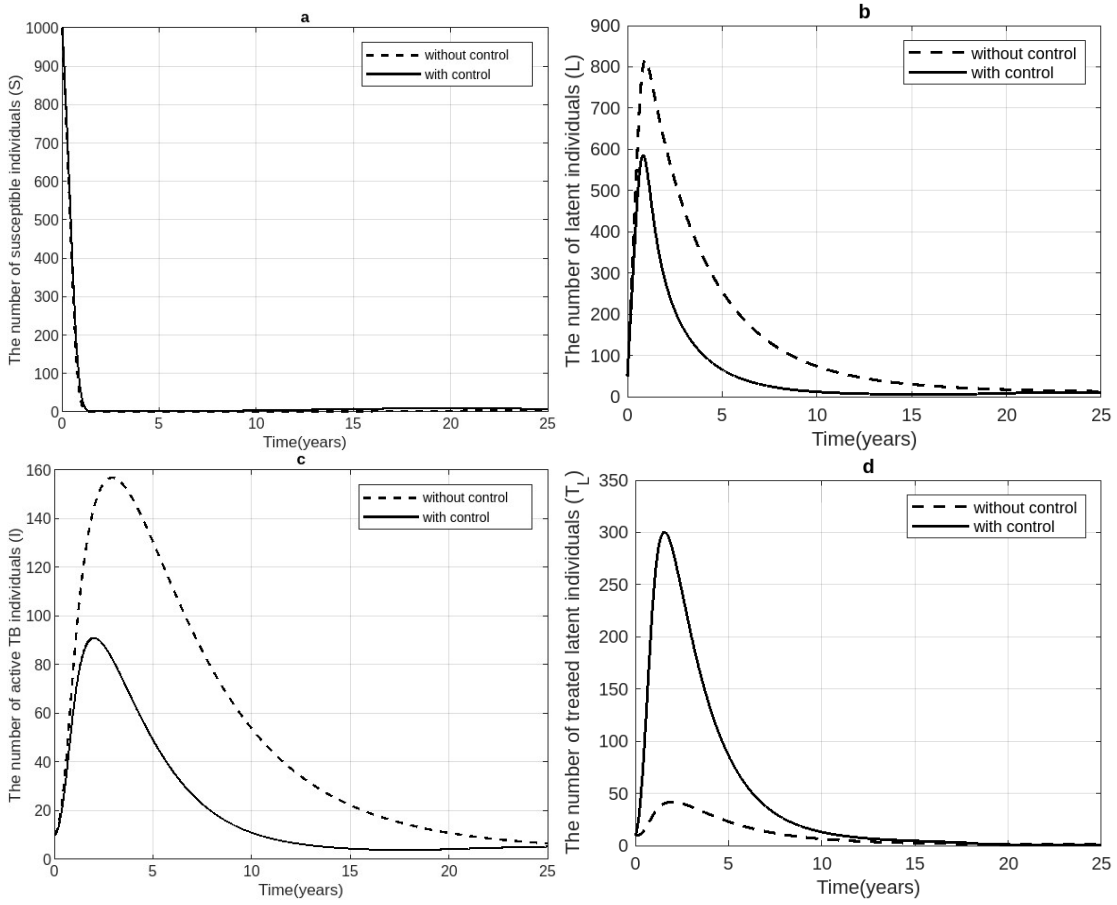


FIGURE 3. Numerical simulations of the optimal control model (4.1) with the preventive control (u_1) only. (a) the number of susceptible individuals (S), (b) the number of latent individuals (L), (c) the number of active TB individuals (I), (d) the number of treated latent individuals (T_L), (e) the number of treated active TB individuals (T_I), (f) the number of recovered individuals (R) and (g) the strategy of control (u_1) when $u_{1\max} = 0.8$, $u_{2\max} = 0, u_{3\max} = 0, u_{4\max} = 0$.

Under this strategy, we use control u_1 to optimize the objective function while u_2, u_3 and u_4 are set to be zero. Figure 3 (a) shows that number of susceptible individuals (S) in both cases drops since the beginning where in control case it drops slower than non-control case. The number of susceptible individuals in both cases almost reaches zero during the 4-14 years and tends to increase slightly after that to reach equilibrium value, where an equilibrium value in control case is slightly larger. Figure 3 (b) and (d) show the same pattern of dynamics i.e., a great reduction in the number of latent individuals (L), and the number of treated active TB individuals (T_L) in control condition. The peak in control case occurs a few years later than in non-control one. Finally, in control case both L and T_L seems to reach lower equilibrium value than in non-control condition. Figure 3 (c) shows that the number of active TB individuals (I) is lower in control

case with the peak of about 130, whereas it reaches the peak of more than 150 in non-control one. It can be seen that the number of active TB individuals (I) in control case is less than in the non-control condition throughout 25 years and reaches a lower equilibrium value at the end. Figure 3 (e) shows that the number of treated active TB individuals (T_I) is largely lower in control case comparing to non-control one throughout 25 years. Figure 3 (f) shows that in control case the number of recovered individuals (R) is lower and reaches lower value of equilibrium state than non-control case throughout 25 years. Finally, Figure 3 (g) shows the strategy of u_1 that it has to keep at a maximum rate of 80% since the beginning for about 4.8 years then drops to about 6% for a few years. After that it goes up to 80% again until 24^{th} year and sharply goes down to towards zero in 25^{th} year. Our results demonstrate that a control u_1 could greatly reduce a TB infection.

5.2. **Strategy B: The control by the screening and put under treatment only.**



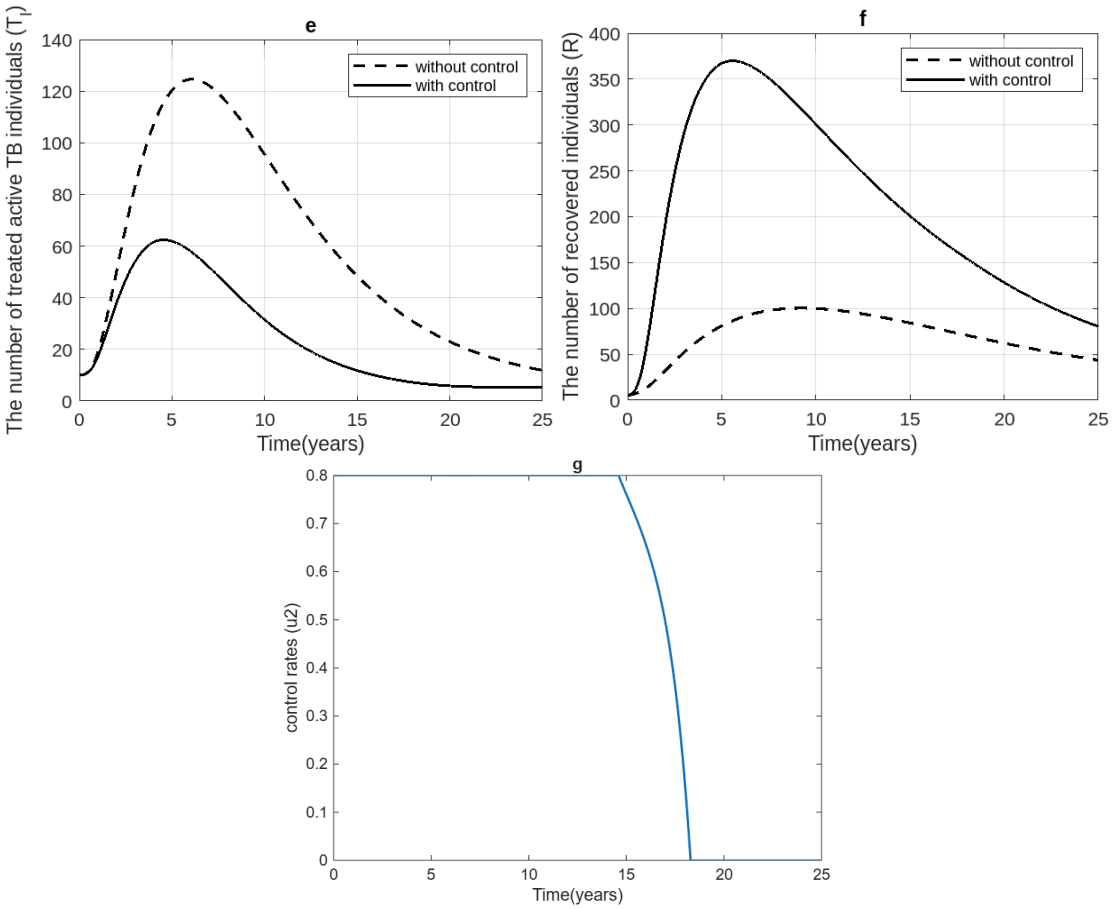
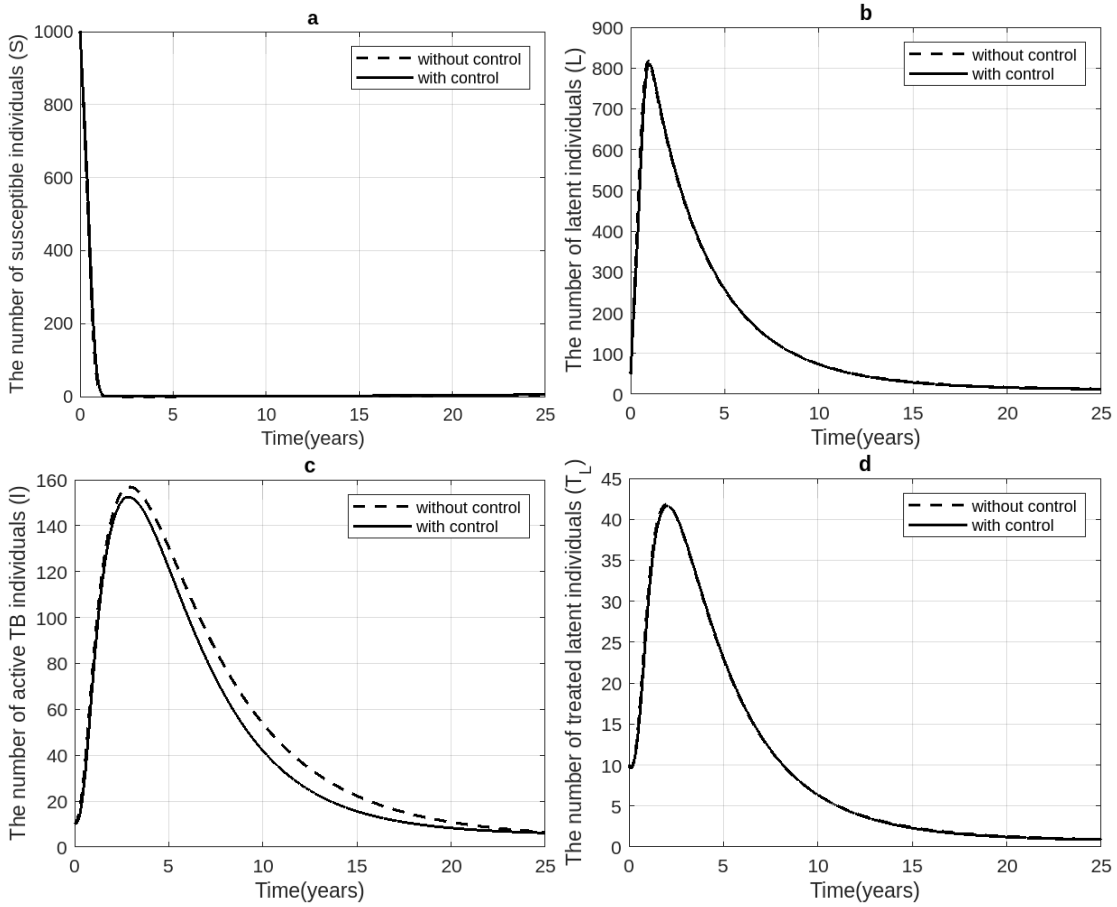


FIGURE 4. Numerical simulations of the optimal control model (4.1) with the screening and put under treatment (u_2) only. (a) the number of susceptible individuals (S), (b) the number of latent individuals (L), (c) the number of active TB individuals (I), (d) the number of treated latent individuals (T_L), (e) the number of treated active TB individuals (T_I), (f) the number of recovered individuals(R) and (g) the strategy of control (u_2) when $u_{1\max} = 0, u_{2\max} = 0.8, , u_{3\max} = 0, u_{4\max} = 0$.

Under this strategy, we use control u_2 to optimize the objective function while u_1, u_3 and u_4 Figure 4 (a) shows that number of susceptible individuals (S) in both case decreases from the start until the first year, after that it stabilizes and increases slightly around 12^{th} year to reach equilibrium value. An equilibrium value in control case is slightly higher. Figure 4 (b) shows that the number of latent individuals (L) in control condition is significantly lower than non-control condition. The peak in control case occurs slightly before in non-control one. Figure 4 (c) shows that the number of active TB individuals (I) is also significantly lower in control case with the peak of about 90, whereas it reaches the peak of more than 159 in non-control one. It can be seen that the number of active TB individuals (I) in control case is less than in the non-control condition throughout 25 years and reaches lower equilibrium value. Figure 4 (d) shows that the number of treated latent individuals (T_L) in control condition is significantly greater than in non-control one although they

reaches the same equilibrium value for both cases. Figure 4 (e) shows a dramatic reduction of the number of treated active TB individuals (T_I) in control case comparing to non-control one and it seems to reach lower equilibrium value. Figure 4 (f) shows that in control case the number of recovered individuals (R) is significantly greater and reaches larger value of equilibrium state than in non-control case. Finally, Figure 4 (g) shows the strategy of $u_2$that it has to keep at a maximum rate of 80% since the beginning until the 14.9^{th} year then it can be dropped to about zero in 18^{th} year and remains zero until 25^{th} year. Our results show that u_2 plays a role in both reducing a TB infection and boosting a number of treated latent and recovered individuals.

5.3. **Strategy C: The control by the treatment effort for active TB only.**



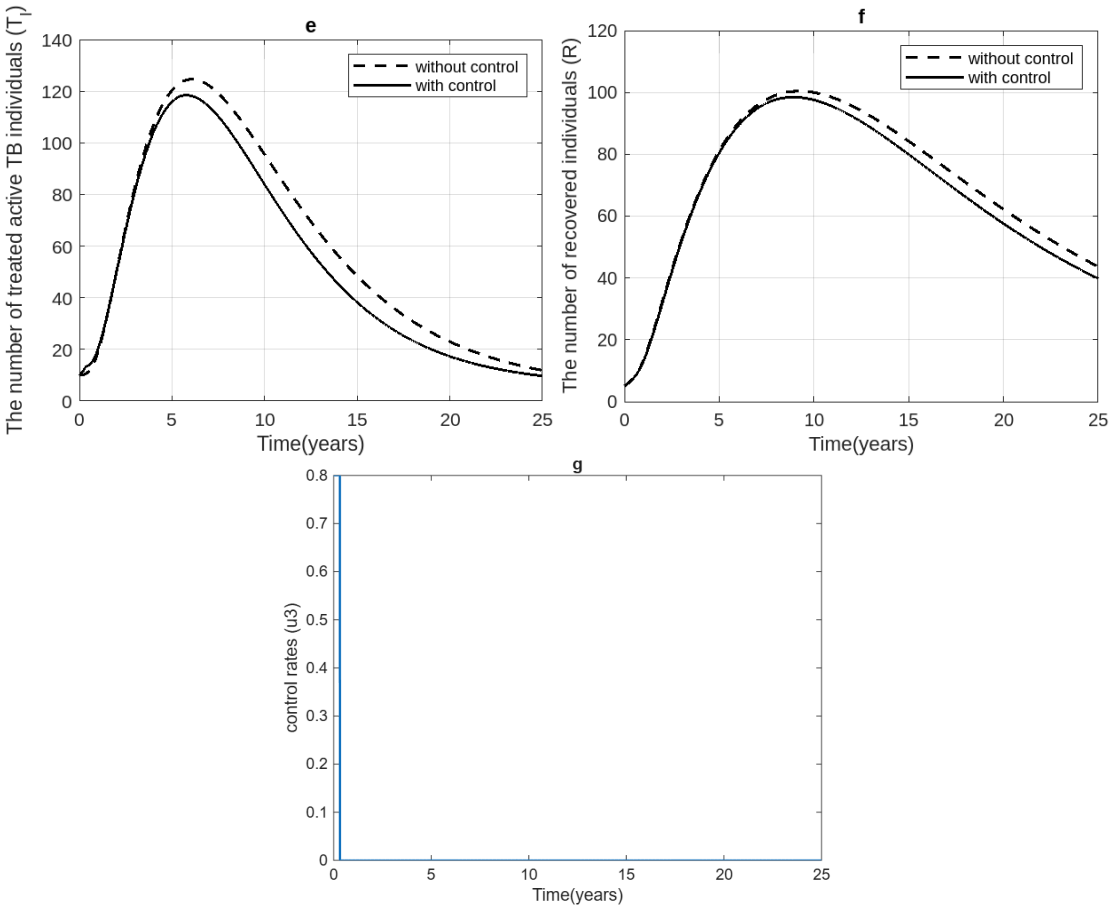
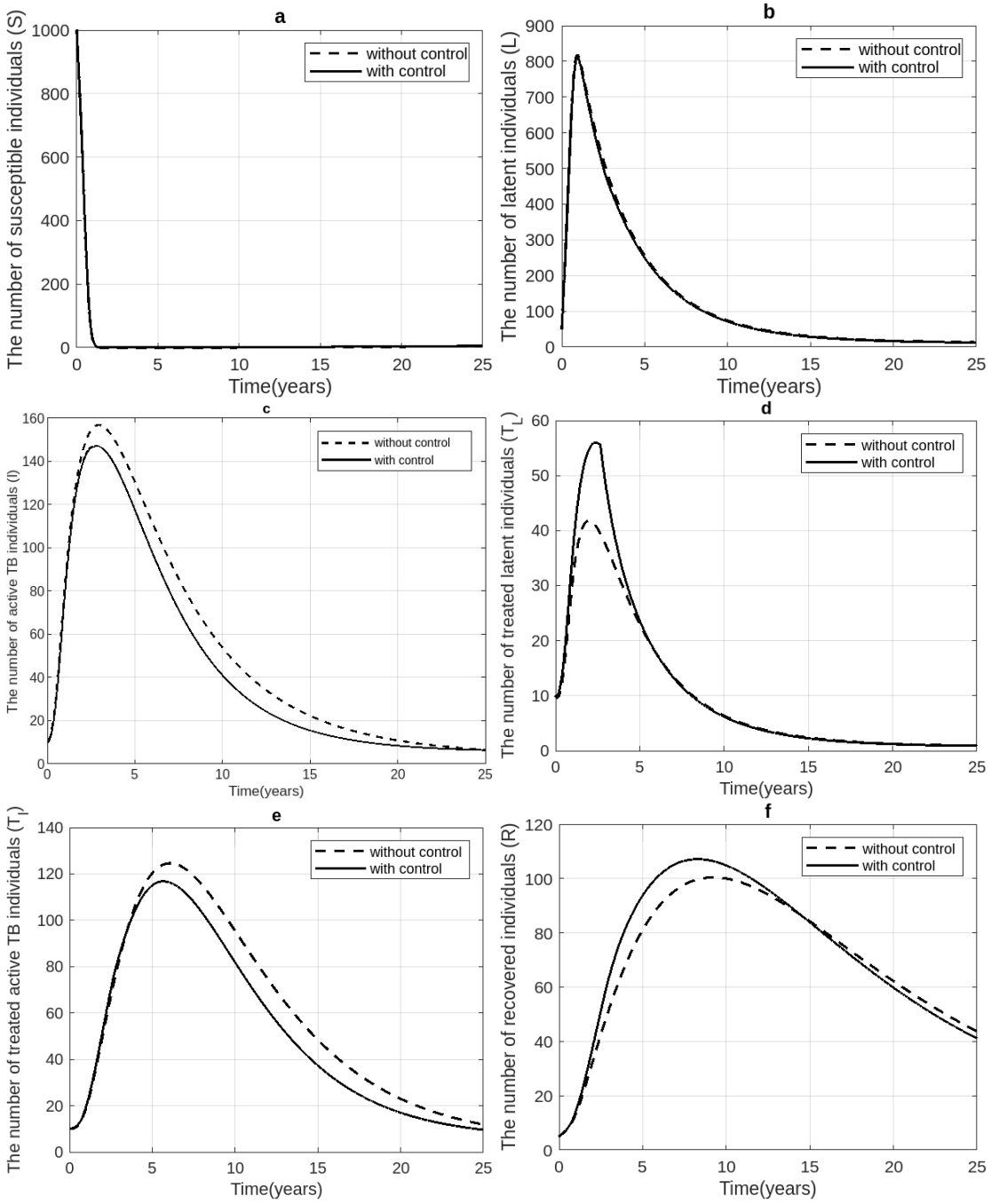


FIGURE 5. Numerical simulations of the optimal control model (4.1) with the treatment effort for active TB (u_3) only. (a) the number of susceptible individuals (S), (b) the number of latent individuals (L), (c) the number of active TB individuals (I), (d) the number of treated latent individuals (T_L), (e) the number of treated active TB individuals (T_I), (f) the number of recovered individuals(R) and (g) the strategy of control (u_3) when $u_{1\max} = 0$, $u_{2\max} = 0, u_{3\max} = 0.8, u_{4\max} = 0$.

Under this strategy, we use control u_3 to optimize the objective function while u_1, u_2 and u_3 are set to be zero. Figure 5 (a), (b) and (d) show that the number of susceptible individuals (S), the number of latent individuals (L), and the number of treated latent individuals (T_L) remain the same for both control and non-control case. Figure 5 (c) shows a slightly reduction of the number of active TB individuals (I) in control case with the peak of about 150, whereas it reaches the peak of more than 159 in non-control one. Figure 5 (e) shows that the number of treated active TB individuals (T_I) is the same for both cases until the 4^{th} year, after that the number of treated active TB individuals (T_I) in control condition is slightly lower than in non-control condition and tends to reach lower equilibrium value. Figure 5 (f) shows similar results as in Figure 5 (e), that is the number of recovered individuals (R) is the same for both cases until the 7^{th} year, and after that the

number of recovered individuals (R) in control case is slightly lower than in non-control one and tends to reach lower equilibrium value. Finally, Figure 5 (g) shows the strategy of u_3 that it has to keep at a maximum rate of 80% for less than a year then it drops to zero immediately and remains zero until the 25^{th} year. Our results show that u_3 plays a role in reducing the number of active TB individuals (I).

5.4. **Strategy D: The control by the campaign to make sure patients obtain complete treatment only.**



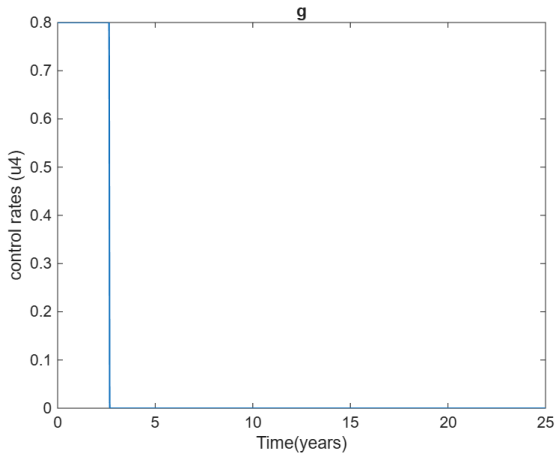


FIGURE 6. Numerical simulations of the optimal control model (4.1) with the campaign to make sure patients obtain complete treatment (u_4) only. (a) the number of susceptible individuals (S), (b) the number of latent individuals (L), (c) the number of active TB individuals (I), (d) the number of treated latent individuals (T_L), (e) the number of treated active TB individuals (T_I), (f) the number of recovered individuals(R) and (g) the strategy of control (u_4) when $u_{1\max} = 0, u_{2\max} = 0, u_{3\max} = 0, u_{4\max} = 0.8$.

Under this strategy, we use control u_4 to optimize the objective function while u_1, u_2, u_3 and u_4 are set to be zero. Similar to Strategy A and B, Figure 6 (a) shows that the number of susceptible individuals (S) is the same in both cases throughout 25 years. Figure 6 (b) shows that the number of latent individuals (L) is almost the same in both cases. Figure 6 (c) and (e) shows the same patterns of dynamics, i.e., the number of active TB individuals (I) and the number of treated active TB individuals (T_I) remains unchanged between control and non-control condition for the first 2 and 4 years, respectively and after that it is slightly lower in control condition compared to non-control one. Then it reaches the same equilibrium value Figure 6 (d) shows that the number of treated latent individuals (T_L) in control condition is greater than a non-control condition, whereas it reaches the same equilibrium value in both cases. Figure 6 (f) shows that in control case the number of recovered individuals (R) is greater than in non-control condition for the first 14 years, after that it tends to be less than the number of recovered individuals (R) in non-control one. Finally, Figure 6 (g) shows the strategy of u_4 that it has to keep at a maximum rate of 80% since the beginning for about 2.5 years then after that it drops to zero immediately and remains zero until the 25^{th} year. Our results demonstrate a role of u_4 that it could increase the number of treated and recovered individuals.

5.5. **Strategy E: combination of all controls.**

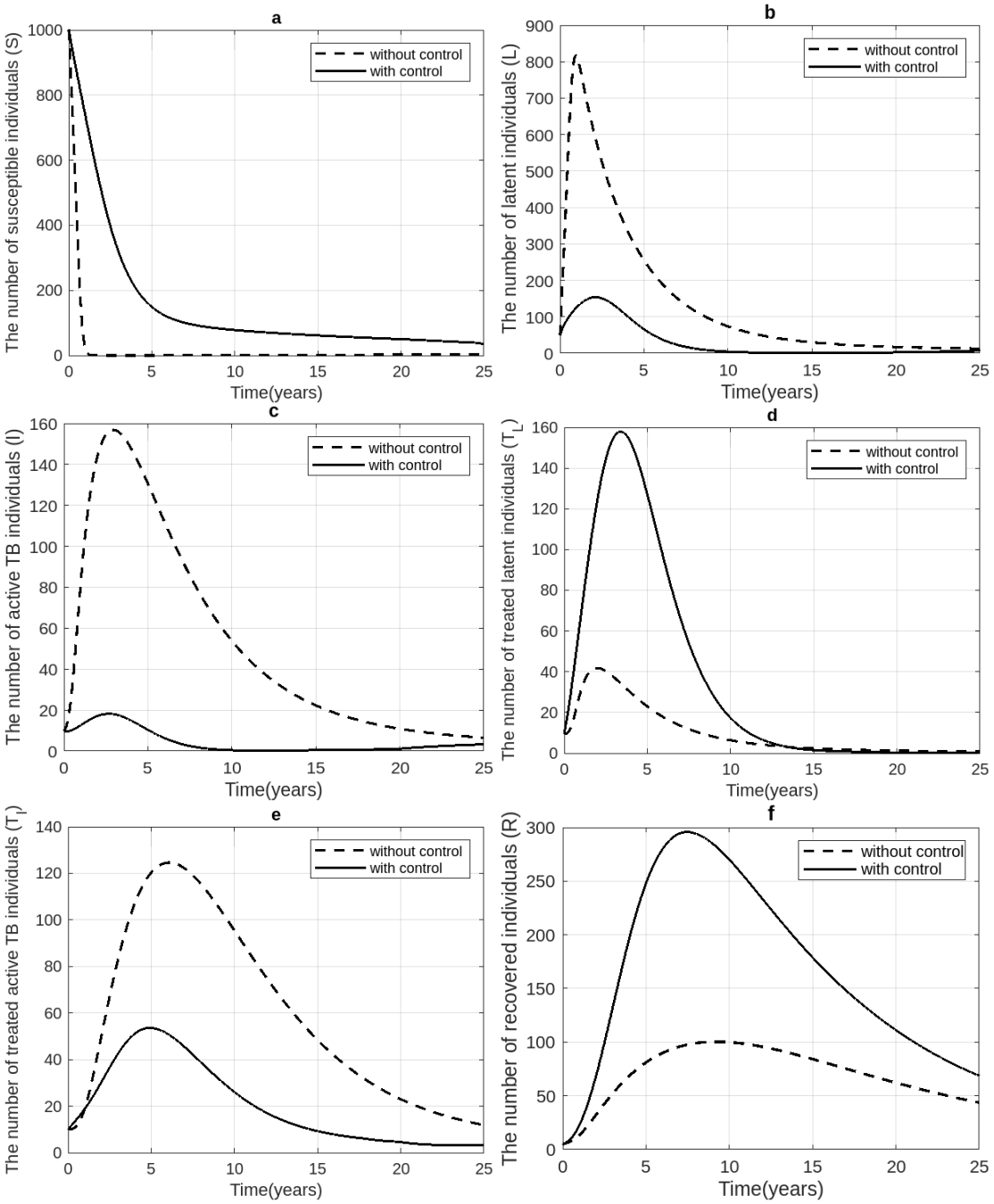


FIGURE 7. Numerical simulations of the optimal control model (4.1) with a combination of all controls (a) the number of susceptible individuals (S), (b) the number of latent individuals (L), (c) the number of active TB individuals (I), (d) the number of treated latent individuals (T_L), (e) the number of treated active TB individuals (T_I), (f) the number of recovered individuals(R) and (g) the strategy of control when $u_{1\max} = 0.8, u_{2\max} = 0.8, u_{3\max} = 0.8, u_{4\max} = 0.8$.

Under this strategy, we use control u_1, u_2, u_3 and u_4 to optimize the objective function, where the maximum value of u_1, u_2, u_3 and u_4 is 0.8. Similar to Strategy A, B, C and D, Figure 7 (a) shows that number of susceptible individuals (S) in control case decreases slower than in non-control condition and it reaches higher equilibrium value. Figure 7 (b) and (c) shows a significant decrease of the number of latent individuals (L) and the number of active TB individuals (I) in control condition comparing to non-control case. Figure 7 (d) shows that the number of treated latent individuals (T_L) in control condition is significantly larger than non-control condition, although it tends towards the same equilibrium value. Figure 7 (e) shows that the number of treated active TB individuals (T_I) is largely lower in control case comparing to non-control one throughout 25 years. It reaches lower equilibrium value in control condition. Figure 7 (f) shows that the number of recovered individuals (R) in control case is significantly larger than in non-control one throughout 25 years.

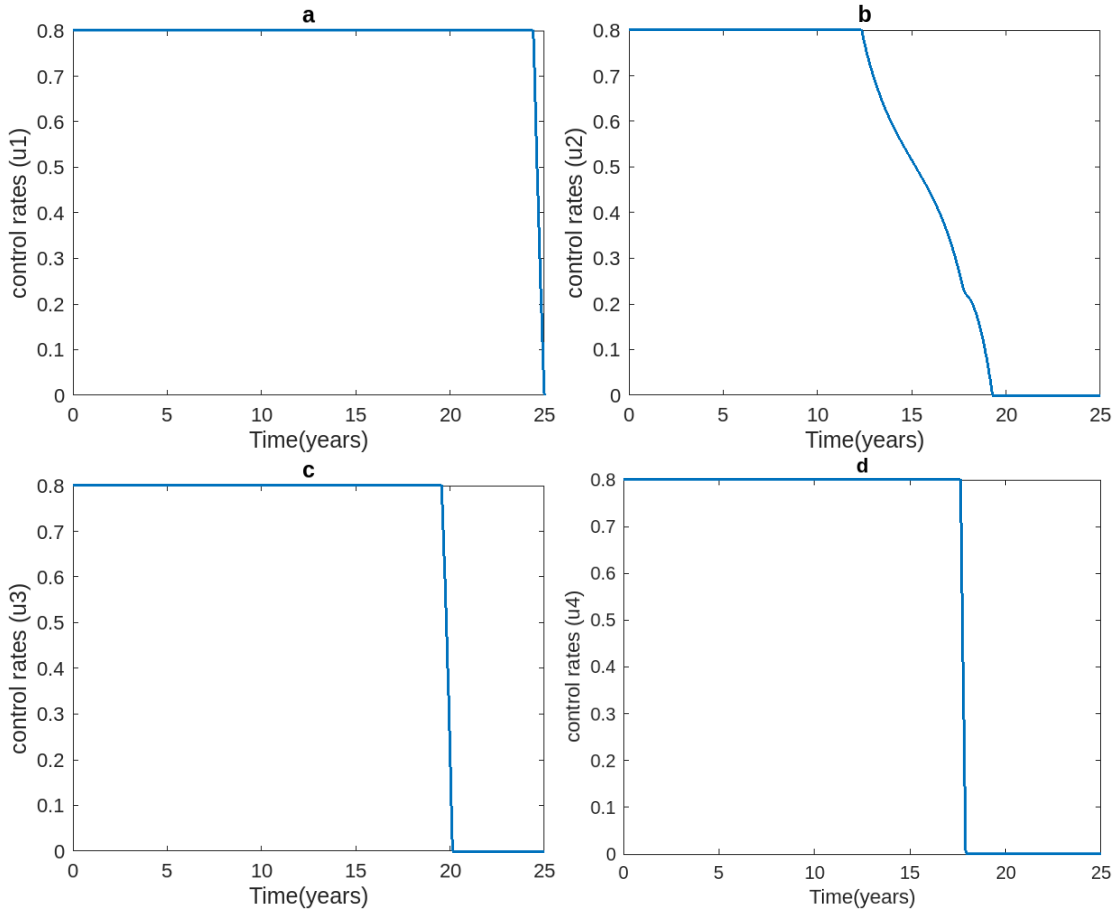


FIGURE 8. Dynamic of control (a) preventive control (u_1), (b) screening and put under treatment (u_2), (c) treatment effort for active TB (u_3) and (d) campaign to make sure patients obtain complete treatment (u_4).

Figure 8 (a) shows the strategy of u_1 that it has to keep at a maximum rate of 80% since the beginning until about 24.8^{th} year and it then sharply goes down to zero towards the 25^{th} year. Figure 8 (b) shows the strategy of u_2 that it has to keep at a maximum rate of 80% since the beginning for about 12 years then drops to zero in 19^{th} year and remains zero towards 25^{th} year. Figure 8 (c) shows the strategy of u_3 that it has to keep at a maximum rate of 80% since the beginning for about 19 years then immediately drops to zero in 20^{th} year and remains zero towards 25^{th} year. Finally, Figure 8 (d) shows the strategy of u_4 that it has to keep at a maximum rate of 80% since the beginning for about 18 years then immediately drops to zero and remains zero towards 25^{th} year. However, our results demonstrate that all three controls give a better strategy than one control alone.

Overall, our results demonstrate that a control u_1 alone could reduce a TB infection for some certain amount, a control u_2 alone could reduce a TB infection and boost a number of treated latent and recovered individuals, a control u_3 alone could slightly reduce the number of active TB individuals (I), and a control u_4 alone could increase the number of treated and recovered individuals. Finally, a combination of all four controls could significantly reduce the number of latent and active TB individuals and significantly increase the number of treated latent individuals and recovered individuals, giving the best result among all five strategies.

6. Conclusions

A mathematical model of tuberculosis transmission with incomplete treatment is proposed in this study. We consider two different classes of treated individuals which are treated latent and treat active TB individuals. Both classes may reenter due to incomplete treatment. Our model therefore is modified from the work of Ullah and the team [24] by adding these two classes into the model and incorporate an incomplete treatment for both classes of population, i.e., incomplete treatment of latent individuals and incomplete treatment of active TB individuals. The model consists of six classes of population which are susceptible (S), latent (L), active TB (I), treated latent (T_L), treated active TB (T_I), and recovered (R) individuals. Model solutions are verified to be nonnegative and bounded. We obtain two equilibrium points i.e., disease-free and endemic. Their stability is analyzed locally and globally. The basic reproduction number is computed with its sensitivity, and it is found to be a threshold for each equilibrium point stability. When the basic reproduction number is less than a unity, the disease-free is obtained to be globally stable. On the other hand, when it is more than a unity, a disease persists and an endemic equilibrium point is stable under certain conditions as proved in Theorem 3.3 and 3.7. The sensitivity analysis result in Table 1 shows that the transmission rate (β) gives a positive effect to the basic reproduction number, whereas the treatment rate of active TB individuals (θ) gives a negative effect. With these results, we therefore extended the study by applying optimal control problem into the model. We add four control variables which are preventive control for transmission, screening of latent and put under treatment, treatment effort for active TB, and campaign to make sure patients obtain complete

treatment. Numerical simulations of optimal control model are performed for five strategies, i.e., each single control and a combination of four controls. The results show that single control alone could reduce active TB patients for some amounts. In particular, Strategy D shows that campaigns to make sure patients obtain complete treatment could lower the number of active TB patients and increase recovered individuals. However, a combination of four controls gives the best result in reducing the number of TB patients and increasing the number of recovered individuals. Our results therefore could be useful as insight information for public health authorities for further action.

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Conflicts of Interest: The authors declare that there are no conflicts of interest regarding the publication of this paper.

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