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An SEITR Model for Tuberculosis Incorporating Migration, General Non-Linear Incidence, and Treatment Effectiveness

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Abstract

Tuberculosis (TB) remains a major global public health challenge, particularly in populations affected by migration and limited access to healthcare. In this study, we formulate and analyze a compartmental SEITR model (Susceptible–Exposed–Infectious–Treated–Recovered) for tuberculosis transmission, incorporating a general nonlinear incidence function of the form $f_1(S)(f_2(I) + f_3(T))$. The model accounts for migration-related population influx and distinguishes between scenarios with and without influx into disease-related compartments. For the model with migration, we establish the existence and global asymptotic stability of a unique endemic equilibrium under suitable conditions. For the reduced model without direct influx into disease-related compartments, we derive the basic reproduction number $\mathcal{R}_0$ using the next-generation matrix method and establish the global asymptotic stability of the disease-free equilibrium for $\mathcal{R}_0 < 1$ and the endemic equilibrium for $\mathcal{R}_0 > 1$, using suitable Lyapunov functions and LaSalle's invariance principle. We further employ the center manifold approach of Castillo-Chavez and Song to demonstrate a forward transcritical bifurcation at the threshold $\mathcal{R}_0 = 1$. The model explicitly accounts for incomplete and fully effective treatment, allowing us to investigate how treatment outcomes influence disease persistence and control. A global sensitivity analysis based on Sobol indices assesses the relative influence of model parameters on $\mathcal{R}_0$. Notably, we identify a critical treatment-efficacy threshold p_c : when $p_c > 1$, even perfect treatment ($p = 1$) cannot drive $\mathcal{R}_0$ below unity, implying that treatment alone cannot eliminate TB in high-burden regimes. Finally, numerical simulations investigate the effects of migration-related influx, bilinear versus saturated incidence, and treatment effectiveness on the epidemic dynamics. These findings contribute to the mathematical understanding of tuberculosis transmission and highlight the importance of treatment outcomes and migration in assessing disease control strategies.



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1. Introduction

Tuberculosis (TB) is an infectious disease caused by *Mycobacterium tuberculosis* (MTB) and predominantly affects the respiratory system, particularly the lungs [1–3]. Transmission mainly occurs through airborne droplets released by individuals with pulmonary TB during coughing, sneezing, or speaking [4]. Despite substantial progress in diagnosis, treatment, and prevention, TB remains a major global health challenge, particularly in low- and middle-income countries. Its epidemiological dynamics are further complicated by demographic heterogeneity, treatment outcomes, population mobility, and interactions with other health conditions such as HIV infection [5–7]. Mathematical modeling therefore provides a valuable framework for understanding TB transmission dynamics and evaluating the potential impact of epidemiological interventions [8–10].

A central component of an epidemiological model is the incidence function, which determines how susceptible individuals acquire infection from infectious individuals. The standard bilinear incidence, typically written as βSI , has been extensively used in TB models because of its mathematical simplicity and epidemiological interpretability [11–16]. However, this formulation assumes that the transmission pressure increases proportionally with the number of infectious individuals. Such an assumption may be restrictive when contact patterns, behavioral responses, heterogeneous mixing, or saturation effects modify the effective transmission process. Nonlinear incidence functions have therefore been introduced to provide more flexible representations of infectious disease transmission [17–25]. For TB in particular, nonlinear transmission mechanisms can account for saturation effects and deviations from simple mass-action transmission. A general formulation of the incidence term, $f_1(S)(f_2(I) + f_3(T))$, allows the contribution of the susceptible population and the transmission pressures exerted by infectious and treated individuals to be represented separately. This structure provides a flexible framework encompassing several commonly used incidence functions as special cases. It is also relevant when individuals undergoing treatment may retain a non-negligible contribution to transmission and when treatment does not necessarily result in immediate or complete epidemiological clearance.

Population mobility provides another important source of complexity in TB transmission dynamics. Migration and immigration can alter the distribution of susceptible and infected individuals between populations and may consequently affect disease persistence and the effectiveness of control measures. Recent TB models have incorporated population movement together with treatment dynamics, drug resistance, media effects, and other epidemiological mechanisms, highlighting the importance of mobility in TB transmission [26,27]. Other studies have investigated general incidence functions in compartmental epidemic models without migration, while subsequent work has incorporated migration into related frameworks [17,23,28]. These studies suggest that both the functional form of the transmission mechanism and population movement may substantially modify the qualitative behavior of epidemic systems.

An important reference for the present study is the model proposed by Ullah et al. [29], which incorporates infectious and treated individuals and distinguishes effective treatment from incomplete treatment. In that model, disease transmission is described by the bilinear incidence term $\lambda S(t)(I(t) + \beta T(t))$. Although this formulation provides a useful representation of TB transmission and treatment dynamics, it relies on a specific bilinear transmission structure and does not incorporate migration into the epidemiological compartments. The model considered in the present work extends this framework by replacing the bilinear incidence term with the general nonlinear structure $f_1(S)(f_2(I) + f_3(T))$, while allowing population fluxes to enter the different epidemiological classes. In fact, the model of Ullah et al. [29] is recovered as a special case by setting $f_1(S) = \lambda S$, $f_2(I) = I$, $f_3(T) = \beta T$,

and setting $\Lambda_i = 0 \forall i \in \{S, E, I, T, R\}$. Thus, the present formulation provides a direct generalization of the model of Ullah et al. while retaining its essential treatment structure.

Beyond the choice of incidence function, the qualitative behavior of epidemiological models near the threshold $R_0 = 1$ also deserves careful attention. Bifurcation analysis has become an important tool in TB modeling because the threshold value $R_0 = 1$ does not always completely determine disease elimination. In particular, the approach developed by Castillo-Chavez and Song has been widely used to show that epidemiological models may exhibit either forward or backward bifurcation; in the latter case, a stable disease-free equilibrium coexists with a stable endemic equilibrium even when $R_0 < 1$ [30–32]. In the context of TB, several studies have shown that mechanisms such as exogenous reinfection, recurrent infection, treatment failure, and limited treatment capacity can lead to a backward bifurcation. These findings indicate that reducing R_0 below unity may not be sufficient to eradicate the disease, and they therefore justify the use of bifurcation analysis in the present work [33,34]. Motivated by this framework, and in line with the general SEITR formulation developed below, we investigate whether the combined effects of nonlinear incidence and incomplete treatment can generate a forward or backward bifurcation and thereby modify the classical threshold dynamics.

Recent developments have also emphasized the importance of explicitly incorporating spatial structure into epidemic models. For example, Wu et al. [35] developed a reaction-diffusion model for cholera transmission incorporating vaccination and wild and drug-resistant strains, and used demographic and epidemiological data from Zimbabwe to investigate spatiotemporal disease dynamics. Their results illustrate how spatial processes and population diffusion can influence disease persistence and localized transmission. Although reaction-diffusion models provide a natural framework for describing continuous spatial spread, the present study considers migration within an ordinary differential equation framework. This distinction allows the analytical effects of population movement to be investigated without introducing an explicit spatial diffusion operator, while also providing a natural basis for future extension toward spatially distributed TB models.

The above considerations motivate the development of a unified SEITR framework in which non-linear transmission, population migration, and incomplete treatment are considered simultaneously. Building on the treatment framework of [29], the present model generalizes the underlying bilinear transmission mechanism and incorporates population fluxes into the epidemiological compartments. This formulation makes it possible to investigate how these mechanisms interact and, in particular, how migration modifies the threshold and long-term dynamics of TB transmission. It also provides a natural framework for comparing the general model with its corresponding formulation in the absence of migration and for examining whether non-linear incidence and incomplete treatment can alter the classical threshold behavior.

The remainder of this paper is organized as follows. Section 2 presents the formulation of the general SEITR model, together with the underlying assumptions, parameter interpretation, and limitations. Section 3 analyzes the general model and establishes the disease-free and endemic equilibria, the basic reproduction number, and their stability properties. Section 4 presents the model without migration and establishes the global asymptotic stability (GAS) of its equilibrium. Section 5 considers a specific non-linear incidence function and develops the corresponding local bifurcation analysis, including the effects of migration. Section 6 is devoted to a global sensitivity analysis based on the Sobol method. Section 7 provides illustrative numerical examples designed to demonstrate the analytical results and compare the dynamics with and without migration and non-linear incidence. Finally, Section 8 summarizes the main findings and discusses possible directions for future research.

2. Model Formulation and Assumptions

We consider a compartmental SEITR model describing the transmission and treatment dynamics of tuberculosis in a population subject to demographic and population fluxes. The population is divided into five epidemiological compartments: susceptible individuals $S(t)$, exposed individuals $E(t)$, infectious individuals $I(t)$, treated individuals $T(t)$, and recovered individuals $R(t)$. The model incorporates a general non-linear incidence function, incomplete treatment, disease-induced mortality, and population fluxes into the different epidemiological classes. The resulting framework is sufficiently general to contain several existing TB models as special cases, including the model of Ullah et al. [29].

The model is described by the following system of ordinary differential equations

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \zeta S, \\ \frac{dE(t)}{dt} = \Lambda_E + f_1(S)(f_2(I) + f_3(T)) - b_1 E + (1-p)\nu T, \\ \frac{dI(t)}{dt} = \Lambda_I + \alpha E - b_2 I, \\ \frac{dT(t)}{dt} = \Lambda_T + \gamma I - b_3 T, \\ \frac{dR(t)}{dt} = \Lambda_R + p\nu T - \zeta R, \end{cases} \quad (1)$$

subject to

$$S(0) > 0, E(0), I(0), T(0), R(0) \geq 0. \quad (2)$$

where

$$b_1 = \zeta + \alpha, \quad b_2 = \zeta + \delta_I + \gamma, \quad b_3 = \zeta + \delta_T + \nu.$$

Here, $S(t)$, $E(t)$, $I(t)$, $T(t)$, and $R(t)$ denote susceptible, exposed, infectious, treated, and recovered individuals, respectively. The parameter ζ represents the natural mortality rate, while δ_I and δ_T denote disease-induced mortality rates in the infectious and treated classes, respectively. Exposed individuals progress to the infectious class at rate α , whereas infectious individuals initiate treatment at rate γ . Treated individuals leave the treatment class at rate ν , with a fraction p successfully completing treatment and entering the recovered class, while the remaining fraction $1 - p$ returns to the exposed class, representing treatment failure or relapse. Here $0 < p \leq 1$.

The compartmental structure of the model is illustrated in Figure 1. The parameters of the system and their meanings are listed in the Table 1.

Table 1. Parameters and their epidemiological interpretation.

Parameter	Meaning
$\Lambda_S, \Lambda_E, \Lambda_I, \Lambda_T$ and Λ_R	Migration-induced population influx into the corresponding epidemiological compartment
α	Progression rate from E to the infectious class I
p	The proportion of effective treatment
δ_I	The mortality rate for I
δ_T	The mortality rate for treated class T
γ	The treatment rate for I
ν	Exit rate from the treatment compartment
ζ	Natural death rate in all classes

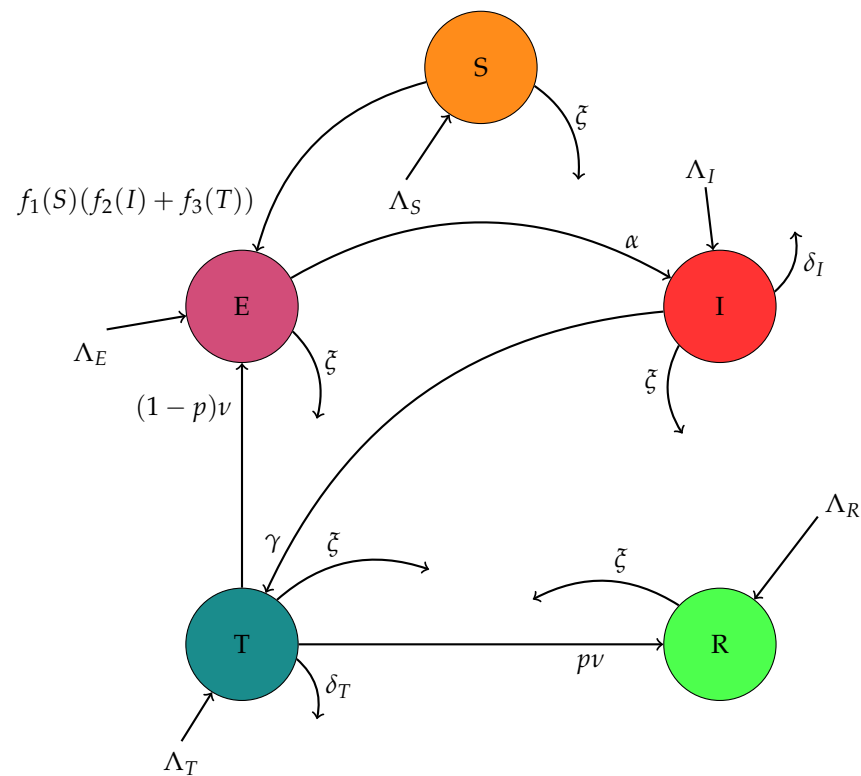


Figure 1. Schematic representation of the SEITR model.

The formulation of system (1)–(2) is based on the following epidemiological assumptions:

- (A1) The population is divided into susceptible, exposed, infectious, treated, and recovered individuals, and individuals within each compartment are assumed to be epidemiologically homogeneous.
- (A2) New infections occur through contact with infectious individuals and, when applicable, individuals undergoing treatment. The transmission process is represented by the general incidence function

$$f_1(S)(f_2(I) + f_3(T)).$$

- (A3) Individuals in the exposed class are not explicitly assumed to contribute to transmission and progress to the infectious class at rate α .
- (A4) Infectious individuals enter treatment at rate γ . Treatment is not necessarily completely effective: a fraction p successfully completes treatment and moves to the recovered class, whereas a fraction $1 - p$ experiences treatment failure or relapse and returns to the exposed class.
- (A5) Natural mortality occurs in all epidemiological classes at rate ξ , while disease-induced mortality occurs in the infectious and treated classes at rates δ_I and δ_T , respectively.
- (A6) The terms $\Lambda_S, \Lambda_E, \Lambda_I, \Lambda_T, \Lambda_R$ represent population influxes into the corresponding epidemiological compartments. These terms provide an aggregated representation of population movement or recruitment from outside the modeled population.
- (A7) The functions f_1, f_2 , and f_3 are sufficiently regular to guarantee the mathematical well-posedness of the model and satisfy the structural assumptions specified below.

To analyze system (1), we assume that $f_1, f_2, f_3 \in C^2([0, \infty))$ and satisfy the following conditions

(H1). For all $S > 0$, $I > 0$, and $T > 0$, we have $f_1(S) > 0$, $f_2(I) > 0$, and $f_3(T) > 0$, respectively.

(H2). $f_1(0) = 0$, $f_2(0) = 0$, and $f_3(0) = 0$.

(H3). The functions f_1 , f_2 , and f_3 are non-decreasing on the interval $[0, +\infty]$.

(H4). The functions $\forall I \geq 0$, $f''(I) \leq 0$, $\forall T \geq 0$, $f''(T) \leq 0$, in particular $(\frac{f_3(T)}{T})$ and $(\frac{f_2(I)}{I})$ are non-increasing on the interval $(0, +\infty)$.

Assumptions (H1)–(H3) are standard structural conditions for general incidence functions, while (H4) imposes a saturation-type property on the transmission contributions of infectious and treated individuals. Such assumptions are useful in establishing global stability results for epidemic systems with non-linear incidence functions [11,19,21].

Despite its generality, the proposed framework involves some simplifying assumptions. First, as a deterministic ODE system, it assumes homogeneous mixing and omits explicit spatial heterogeneity; population influxes capture the epidemiological effects of migration but do not model explicit spatial movement or redistribution. Second, demographic structure (e.g., age, sex, or risk groups) is not incorporated. Third, treatment dynamics are simplified into a single compartment where a fixed fraction p recovers and $(1 - p)$ fails or relapses, without distinguishing between specific drug regimens, resistance profiles, or the precise biological mechanisms of residual infectiousness. Finally, the numerical experiments serve to illustrate the model's analytical properties rather than to provide a data-calibrated prediction for a specific population. These limitations outline natural avenues for future extensions, such as spatially explicit formulations, structured populations, stochasticity, and refined resistance dynamics.

3. Model with Migration

We first investigate the basic qualitative properties of the general SEITR model introduced in Section 2 when population influxes are taken into account. In particular, we establish the positivity and boundedness of solutions and clarify the equilibrium structure induced by the influx terms. Since positive influxes into infected-related compartments may continuously introduce individuals into the epidemic subsystem, the existence of a classical disease-free equilibrium requires particular attention.

3.1. Positive Invariance and Boundedness

Let

$$\Omega = \mathbb{R}_+^5 = \{(S, E, I, T, R) \in \mathbb{R}^5 : S, E, I, T, R \geq 0\}.$$

Under the C^2 -regularity assumptions on f_1, f_2, f_3 , the vector field associated with system (1) is locally Lipschitz on Ω . We first show that Ω is positively invariant.

Indeed, on the boundary of Ω , the corresponding components of the vector field satisfy

$$\dot{S}|_{S=0} = \Lambda_S \geq 0,$$

because $f_1(0) = 0$, while

$$\dot{E}|_{E=0} = \Lambda_E + f_1(S)(f_2(I) + f_3(T)) + (1 - p)\nu T \geq 0,$$

$$\dot{I}|_{I=0} = \Lambda_I + \alpha E \geq 0,$$

$$\dot{T}|_{T=0} = \Lambda_T + \gamma I \geq 0,$$

and

$$\dot{R}|_{R=0} = \Lambda_R + p\nu T \geq 0.$$

Hence, no component of a solution initially belonging to Ω can cross the boundary into the negative region. Therefore,

$$(S(t), E(t), I(t), T(t), R(t)) \in \Omega, \quad t \geq 0,$$

for every non-negative initial condition.

To establish boundedness, define the total population

$$N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$$

and let

$$\Lambda = \Lambda_S + \Lambda_E + \Lambda_I + \Lambda_T + \Lambda_R.$$

Adding the five equations of system (1) yields

$$\dot{N} = \Lambda - \xi N - \delta_I I - \delta_T T.$$

Consequently,

$$\dot{N} \leq \Lambda - \xi N.$$

By the comparison principle,

$$N(t) \leq N(0)e^{-\xi t} + \frac{\Lambda}{\xi} (1 - e^{-\xi t}), \quad t \geq 0.$$

It follows that

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\xi}.$$

In particular, all epidemiological compartments remain bounded and the system is dissipative. Moreover, if

$$N(0) \leq \frac{\Lambda}{\xi},$$

then the region

$$\Omega_\Lambda = \left\{ (S, E, I, T, R) \in \mathbb{R}_+^5 : S + E + I + T + R \leq \frac{\Lambda}{\xi} \right\}$$

is positively invariant.

3.2. Equilibrium Structure Under Population Influx

Let

$$X^* = (S^*, E^*, I^*, T^*, R^*) \in \Omega$$

be an equilibrium of system (1). The equilibrium equations for the infected and treated compartments give

$$I^* = \frac{\Lambda_I + \alpha E^*}{b_2}, \quad T^* = \frac{\Lambda_T + \gamma I^*}{b_3},$$

while

$$R^* = \frac{\Lambda_R + p\nu T^*}{\xi}.$$

These relations show directly how population influxes modify the equilibrium levels of the epidemiological compartments. In particular, a positive influx into an infected-related compartment prevents the corresponding compartment from vanishing at equilibrium.

The existence of a disease-free equilibrium therefore requires special consideration. We define a classical disease-free equilibrium as an equilibrium at which

$$E^* = I^* = T^* = 0.$$

Substituting these conditions into the equilibrium equations gives

$$0 = \Lambda_E, \quad 0 = \Lambda_I, \quad 0 = \Lambda_T.$$

Hence, a classical disease-free equilibrium can exist only if

$$\Lambda_E = \Lambda_I = \Lambda_T = 0.$$

When these conditions hold, the disease-free equilibrium is

$$\mathcal{E}_0 = \left(\frac{\Lambda_S}{\xi}, 0, 0, 0, \frac{\Lambda_R}{\xi} \right),$$

where the last component vanishes when $\Lambda_R = 0$.

Consequently, if population influx is allowed directly into any of the exposed, infectious, or treated compartments, that is, if

$$\Lambda_E > 0, \quad \Lambda_I > 0, \quad \text{or} \quad \Lambda_T > 0,$$

then a classical disease-free equilibrium with $E^* = I^* = T^* = 0$ does not exist. In such a setting, infection is continuously introduced into the population through external influx, and the classical threshold interpretation based on invasion from a disease-free state is therefore not directly applicable. The preceding analysis shows that population influxes into the exposed, infectious, and treated compartments fundamentally alter the disease-free structure of the system. In particular, when any of Λ_E , Λ_I , or Λ_T is positive, a classical disease-free equilibrium is not available, and the standard DFE-based interpretation of the basic reproduction number cannot be directly applied. To characterize the intrinsic transmission threshold of the epidemic, we therefore consider in the next section the corresponding model without population influx into the disease-related compartments, namely $\Lambda_E = \Lambda_I = \Lambda_T = \Lambda_R = 0$. This reduced formulation allows the disease-free equilibrium and the associated basic reproduction number to be defined and analyzed consistently.

It is worth noting that the equation corresponding to the removed compartment (R) can be omitted from the model (1), as it does not contribute to the transmission chain; accordingly, we restrict our analysis to the reduced model

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \zeta S, \\ \frac{dE(t)}{dt} = \Lambda_E + f_1(S)(f_2(I) + f_3(T)) - b_1E + (1-p)vT, \\ \frac{dI(t)}{dt} = \Lambda_I + \alpha E - b_2I, \\ \frac{dT(t)}{dt} = \Lambda_T + \gamma I - b_3T, \end{cases} \quad (3)$$

with the initial conditions

$$S(0) > 0, E(0) \geq 0, I(0) \geq 0, T(0) \geq 0. \quad (4)$$

3.3. Equilibrium Points

In this section, we establish that the model (3) admits a unique endemic equilibrium under the above stated assumptions.

Proposition 1. Assume $D = b_1b_2b_3 - (1-p)v\alpha\gamma > 0$ and for all $S > 0$, $f''_1(S) < 0$. The model (3) has a unique endemic equilibrium state $x_M^* = (S^*, E^*, I^*, T^*)$.

Proof. We solve the system

$$\begin{aligned} \Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \zeta S &= 0, \\ \Lambda_E + f_1(S)(f_2(I) + f_3(T)) - b_1E + (1-p)vT &= 0, \\ \Lambda_I + \alpha E - b_2I &= 0, \\ \Lambda_T + \gamma I - b_3T &= 0. \end{aligned}$$

From Equations (1) and (2)

$$f_1(S)(f_2(I) + f_3(T)) = \Lambda_S - \zeta S = -\Lambda_E + b_1E - (1-p)vT,$$

and from Equations (3) and (4)

$$T = \frac{\alpha\gamma E + \gamma\Lambda_I + b_2\Lambda_T}{b_2b_3}$$

Thus

$$\begin{aligned} E &= \frac{b_2b_3(\Lambda_S + \Lambda_E - \zeta S) + (1-p)v(\gamma\Lambda_I + b_2\Lambda_T)}{b_1b_2b_3 - (1-p)v\alpha\gamma} = E(S), \\ I &= \frac{\alpha}{b_2}E + \frac{\Lambda_I}{b_2} = I(S), \\ T &= \frac{\alpha\gamma E + \gamma\Lambda_I + b_2\Lambda_T}{b_2b_3} = T(S). \end{aligned}$$

Let now the function $H(S) = \Lambda_S - \zeta S - f_1(S)(f_2(I) + f_3(T))$. We have $H(0) = \Lambda_S > 0$, and $H(\frac{\Lambda_S}{\zeta}) = -f_1(\frac{\Lambda_S}{\zeta})(f_2(I(\frac{\Lambda_S}{\zeta}) + f_3(T(\frac{\Lambda_S}{\zeta}))) \leq 0$. Therefore, by the Intermediate Value

Theorem, since H is continuous on $\left[0, \frac{\Lambda_S}{\xi}\right]$, and provided that $H(0)H\left(\frac{\Lambda_S}{\xi}\right) < 0$, there exists at least one $S^* \in \left(0, \frac{\Lambda_S}{\xi}\right)$ such that

$$H(S^*) = 0.$$

The corresponding values $E(S^*)$, $I(S^*)$ and $T(S^*)$ therefore define an endemic equilibrium.

It remains to prove uniqueness. Since

$$\begin{aligned} H'(S) &= -\xi - f_1'(S)(f_2(I) + f_3(T)) \\ &+ f_1(S) \left[\frac{\xi \alpha b_3}{b_1 b_2 b_3 - (1-p) \nu \alpha \gamma} f_2'(I) + \frac{\xi \alpha \gamma}{b_1 b_2 b_3 - (1-p) \nu \alpha \gamma} f_3'(T) \right] \end{aligned}$$

and

$$\begin{aligned} H''(S) &= -f_1''(S)(f_2(I) + f_3(T)) + 2f_1'(S) \left[\frac{\xi \alpha b_3}{b_1 b_2 b_3 - (1-p) \nu \alpha \gamma} f_2'(I) \right. \\ &+ \left. \frac{\xi \alpha \gamma}{b_1 b_2 b_3 - (1-p) \nu \alpha \gamma} f_3'(T) \right] \\ &- f_1(S) \left[\left(\frac{\xi \alpha b_3}{b_1 b_2 b_3 - (1-p) \nu \alpha \gamma} \right)^2 f_2''(I) + \left(\frac{\xi \alpha \gamma}{b_1 b_2 b_3 - (1-p) \nu \alpha \gamma} \right)^2 f_3''(T) \right], \end{aligned}$$

Since $f_1''(S) \leq 0$, and assumption (iv) ensures that $f_2''(I) \leq 0$ and $f_3''(T) \leq 0$, it follows that, for all $S \in (0, S_0)$, $H''(S) \geq 0$. Under the strict concavity of f_1 , we have

$$H''(S) > 0,$$

so that H is strictly convex. Suppose, by contradiction, that there exist two distinct solutions $S_1, S_2 \in (0, S_0)$ satisfying

$$0 < S_1 < S_2 < S_0, \quad H(S_1) = H(S_2) = 0.$$

Since $S_2 \in (S_1, S_0)$, there exists $\lambda \in (0, 1)$ such that

$$S_2 = \lambda S_1 + (1 - \lambda) S_0.$$

By the strict convexity of H , we obtain

$$H(S_2) < \lambda H(S_1) + (1 - \lambda) H(S_0).$$

Using $H(S_1) = 0$ and $H(S_0) < 0$, we consequently have

$$H(S_2) < (1 - \lambda) H(S_0) < 0,$$

which contradicts the assumption that $H(S_2) = 0$.

Therefore, the equation $H(S) = 0$ admits a unique solution $S^* \in (0, S_0)$. $\square$

Theorem 1. Assume that hypotheses **(H1)–(H4)** hold, that $D = b_1 b_2 b_3 - (1 - p) \nu \alpha \gamma > 0$, and that for all $S > 0$, $f_1''(S) < 0$. Then, the unique endemic equilibrium x_M^* is globally asymptotically stable.

Proof. Following the approach of Korobeinikov in [17,20].

Let the the function defined by

$$V = S - S^* - \int_{S^*}^S \frac{f_1(S^*)}{f_1(\tau)} d\tau + E - E^* - E^* \ln \frac{E}{E^*} + C_1 \left(I - I^* - I^* \ln \frac{I}{I^*} \right) + C_2 \left(T - T^* - T^* \ln \frac{T}{T^*} \right)$$

where

$$C_1 = \frac{b_1}{\alpha}, \quad C_2 = \frac{(1-p)\nu T^* + f_1(S^*)f_3(T^*)}{\gamma I^*} = \frac{(1-p)\nu T^* + f_1(S^*)f_3(T^*)}{b_3 T^* - \Lambda_T}.$$

Note that

$$\Lambda_S = f_1(S^*)(f_2(I^*) + f_3(T^*)) + \xi S^*, \quad (5)$$

$$b_1 E^* = \Lambda_E + f_1(S^*)(f_2(I^*) + f_3(T^*)) + (1-p)\nu T^*, \quad (6)$$

$$b_2 I^* = \Lambda_I + \alpha E^*, \quad (7)$$

$$b_3 T^* = \Lambda_T + \gamma I^*. \quad (8)$$

$\dot{V}$ is given by

$$\dot{V} = \dot{S} \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) + \dot{E} \left(1 - \frac{E^*}{E} \right) + C_1 \dot{I} \left(1 - \frac{I^*}{I} \right) + C_2 \dot{T} \left(1 - \frac{T^*}{T} \right)$$

From (5), we have

$$\begin{aligned} \dot{V} &= (\xi(S^* - S) + f_1(S^*)(f_2(I^*) + f_3(T^*)) - f_1(S)(f_2(I) + f_3(T))) \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) \\ &+ (\Lambda_E + f_1(S)(f_2(I) + f_3(T)) - b_1 E + (1-p)\nu T) \left(1 - \frac{E^*}{E} \right) \\ &+ C_1 (\Lambda_I + \alpha E - b_2 I) \left(1 - \frac{I^*}{I} \right) + C_2 (\Lambda_T + \gamma I - b_3 T) \left(1 - \frac{T^*}{T} \right), \end{aligned}$$

and,

$$\begin{aligned} \dot{V} &= \xi S^* \left(1 - \frac{S}{S^*} \right) \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) + f_1(S^*)(f_2(I^*) + f_3(T^*)) \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) \\ &- f_1(S)(f_2(I) + f_3(T)) \\ &+ \Lambda_E \left(1 - \frac{E^*}{E} \right) + f_1(S^*)(f_2(I) + f_3(T)) + f_1(S)(f_2(I) + f_3(T)) \\ &- b_1 E + (1-p)\nu T - \frac{f_1(S)f_2(I)E^*}{E} \\ &- \frac{f_1(S)f_3(T)E^*}{E} + b_1 E^* - \frac{(1-p)\nu TE^*}{E} + C_1 \Lambda_I \left(1 - \frac{I^*}{I} \right) + C_1 \alpha E \\ &- C_1 b_2 I - C_1 \alpha \frac{EI^*}{I} + C_1 b_2 I^* \\ &+ C_2 \Lambda_T \left(1 - \frac{T^*}{T} \right) + C_2 \gamma I - C_2 b_3 T - C_2 \gamma \frac{IT^*}{T} + C_2 b_3 T^*, \end{aligned}$$

Since

$$\begin{aligned}
 C_1 \alpha \frac{EI^*}{I} &= (\Lambda_E + f_1(S^*)f_2(I^*) + f_1(S^*)f_3(T^*) + (1-p)\nu T^*) \frac{EI^*}{E^*I}, \\
 C_1 b_2 I &= (\Lambda_E + f_1(S^*)f_2(I^*) + f_1(S^*)f_3(T^*) + (1-p)\nu T^*) \frac{I}{I^*} + C_1 \Lambda_I \frac{I}{I^*}, \\
 C_1 b_2 I^* &= \Lambda_E + f_1(S^*)f_2(I^*) + f_1(S^*)f_3(T^*) + (1-p)\nu T^* + C_1 \Lambda_I, \\
 C_2 \gamma I &= C_2 \gamma I^* \frac{I}{I^*} = f_1(S^*)f_3(T^*) \frac{I}{I^*} + (1-p)\nu T^* \frac{I}{I^*}, \\
 C_2 \gamma \frac{IT^*}{T} &= C_2 \gamma I^* \frac{IT^*}{I^*T} = (1-p)\nu T^* \frac{IT^*}{I^*T} + f_1(S^*)f_3(T^*) \frac{IT^*}{I^*T}, \\
 C_2 b_3 T^* &= f_1(S^*)f_3(T^*) + (1-p)\nu T^* + C_2 \Lambda_T, \\
 C_2 b_3 T &= C_2 b_3 T^* \frac{T}{T^*} = C_2 \Lambda_T \frac{T}{T^*} + f_1(S^*)f_3(T^*) \frac{T}{T^*} + (1-p)\nu T,
 \end{aligned}$$

By grouping the terms, we obtain

$$\begin{aligned}
 \dot{V} &= \xi S^* \left[1 - \frac{S}{S^*} \right] \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) + f_1(S^*)f_2(I^*) \left[4 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S)E^*f_2(I)}{f_1(S^*)f_2(I^*)E} \right. \\
 &\quad \left. - \frac{EI^*}{E^*I} - \frac{If_2(I^*)}{I^*f_2(I)} \right] \\
 &+ f_1(S^*)f_2(I^*) \left(\frac{f_2(I)}{f_2(I^*)} - \frac{I}{I^*} \right) \left(1 - \frac{f_2(I^*)}{f_2(I)} \right) \\
 &+ f_1(S^*)f_3(T^*) \left[5 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S)E^*f_3(T)}{f_1(S^*)f_3(T^*)E} - \frac{EI^*}{E^*I} - \frac{IT^*}{I^*T} - \frac{Tf_3(T^*)}{T^*f_3(T)} \right] \\
 &+ f_1(S^*)f_3(T^*) \left(\frac{f_3(T)}{f_3(T^*)} - \frac{T}{T^*} \right) \left(1 - \frac{f_3(T^*)}{f_3(T)} \right) \\
 &+ (1-p)\nu T^* \left[3 - \frac{TE^*}{T^*E} - \frac{EI^*}{E^*I} - \frac{IT^*}{I^*T} \right] \\
 &+ \Lambda_E \left(3 - \frac{E^*}{E} - \frac{EI^*}{E^*I} - \frac{I}{I^*} \right) + C_1 \Lambda_I \left(2 - \frac{I^*}{I} - \frac{I}{I^*} \right) + C_2 \Lambda_T \left(2 - \frac{T^*}{T} - \frac{T}{T^*} \right)
 \end{aligned}$$

Due to the fact that the arithmetic mean surpasses the geometric mean,

Then,

$$\begin{aligned}
 4 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S)E^*f_2(I)}{f_1(S^*)f_2(I^*)E} - \frac{EI^*}{E^*I} - \frac{If_2(I^*)}{I^*f_2(I)} &\leq 0, \\
 5 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S)E^*f_3(T)}{f_1(S^*)f_3(T^*)E} - \frac{EI^*}{E^*I} - \frac{IT^*}{I^*T} - \frac{Tf_3(T^*)}{T^*f_3(T)} &\leq 0, \\
 \left[3 - \frac{TE^*}{T^*E} - \frac{EI^*}{E^*I} - \frac{IT^*}{I^*T} \right] &\leq 0, \\
 \left(3 - \frac{E^*}{E} - \frac{EI^*}{E^*I} - \frac{I}{I^*} \right) &\leq 0, \\
 \left(2 - \frac{I^*}{I} - \frac{I}{I^*} \right) &\leq 0, \\
 \left(2 - \frac{T^*}{T} - \frac{T}{T^*} \right) &\leq 0,
 \end{aligned}$$

Taking into consideration (H3)–(H4), we have

$$\begin{aligned}\zeta S^* \left[1 - \frac{S}{S^*} \right] \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) &\leq 0, \\ f_1(S^*) f_2(I^*) \left(\frac{f_2(I)}{f_2(I^*)} - \frac{I}{I^*} \right) \left(1 - \frac{f_2(I^*)}{f_2(I)} \right) &\leq 0, \\ f_1(S^*) f_3(T^*) \left(\frac{f_3(T)}{f_3(T^*)} - \frac{T}{T^*} \right) \left(1 - \frac{f_3(T^*)}{f_3(T)} \right) &\leq 0.\end{aligned}$$

Thus, we have $\frac{dV}{dt} \leq 0$ for all $t \geq 0$. Moreover, the equality $\frac{dV}{dt} = 0$ holds if and only if $(S, E, I, T) = (S^*, E^*, I^*, T^*)$. By invoking LaSalle's Invariance Principle [36,37], it follows that the endemic equilibrium x_M^* is globally asymptotically stable. $\square$

4. Model Without Migration

We assume that $\Lambda_E = \Lambda_I = \Lambda_T = 0$, and we consider Λ_S from now on as the recruitment rate of susceptibles.

4.1. Positivity of Solutions and Invariant Region

Theorem 2. For all $t \geq 0$, the solutions of (3) with initial conditions (4) remain non-negative.

Proof. Let $t_1 = \max\{t, S(t) > 0, E(t) > 0, I(t) > 0, T(t) > 0\}$

By considering the first equation of system (3) along with the initial condition $S(0) > 0$, we obtain

$$\frac{dS(t)}{dt} = \Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \zeta S.$$

Let f be a C^1 function on $[0, S]$. Then, by the mean value Theorem, $\exists c \in (0, S)$ such that

$$f(S) = f'(c)S.$$

Thus,

$$\frac{dS(t)}{dt} = \Lambda_S - S f'_1(c)(f_2(I) + f_3(T)) - \zeta S,$$

and

$$\frac{dS(t)}{dt} + S[f'_1(c)(f_2(I) + f_3(T)) + \zeta] = \Lambda_S,$$

We put

$$\psi(t) = \zeta + f'_1(c)(f_2(I) + f_3(T)).$$

Note that,

$$S' \exp(\zeta t + \int_0^t \psi(\tau) d\tau) + S\psi(t) \exp(\zeta t + \int_0^t \psi(\tau) d\tau) = \Lambda_S \exp(\zeta t + \int_0^t \psi(\tau) d\tau)$$

and

$$(S \exp(\zeta t + \int_0^t \psi(\tau) d\tau))' = \Lambda_S \exp(\zeta t + \int_0^t \psi(\tau) d\tau),$$

Hence

$$\begin{aligned}S(t_1) &= S(0) \exp(-(\zeta t + \int_0^{t_1} \psi(\tau) d\tau)) \\ &+ \left[\exp(-(\zeta t + \int_0^{t_1} \psi(\tau) d\tau)) \right] \times \int_0^{t_1} \Lambda_S \exp(\zeta x + \int_0^x \psi(\tau) d\tau) dx > 0.\end{aligned}$$

Applying the same reasoning, it can be established that $E(t) > 0$, $I(t) > 0$ and $T(t) > 0$, $\forall t \geq 0$. $\square$

We assume that $\Lambda_E = \Lambda_I = \Lambda_T = 0$.

Theorem 3. *The set $\mathcal{A}$, introduced below, remains positively invariant under the dynamics of system (3)*

$$\mathcal{A} = \left\{ (S(t), E(t), I(t), T(t)) \in \mathbb{R}_+^4 \mid S(t) + E(t) + I(t) + T(t) \leq \frac{\Lambda_S}{\xi}, \forall t \geq 0 \right\}.$$

Proof. Assume that the initial condition $(S(0), E(0), I(0), T(0))$ lies within the set $\mathcal{A}$. Let $(S(t), E(t), I(t), T(t))$ denote a solution of the system (3), and define the total population as

$$P(t) = S(t) + E(t) + I(t) + T(t).$$

Then, the time derivative of $P(t)$ satisfies

$$\begin{aligned} \frac{dP(t)}{dt} &= \Lambda_S - \xi P(t) - \delta_I I(t) - \delta_T T(t), \\ &\leq \Lambda_S - \xi P(t). \end{aligned}$$

and

$$\frac{dP(t)}{dt} + \xi P(t) \leq \Lambda_S,$$

By applying the Fundamental Comparison Theorem [38,39], it follows that

$$P(t) \leq P(0)e^{-\xi t} + \frac{\Lambda_S}{\xi}(1 - e^{-\xi t}).$$

Then, $0 < P(t) \leq \frac{\Lambda_S}{\xi}$ as $t \rightarrow \infty$.

If $P(0) \leq \frac{\Lambda_S}{\xi}$ then $P(t) \leq \frac{\Lambda_S}{\xi} \forall t \geq 0$. if $P(0) > \frac{\Lambda_S}{\xi}$ then $\frac{dP(t)}{dt} \leq 0$ and P will decrease to $\frac{\Lambda_S}{\xi}$.

Hence $\mathcal{A}$ is positively invariant. $\square$

4.2. Basic Reproduction Number R_0 and Existence of Equilibrium Points

The basic reproduction number, R_0 , is a substantial concept in epidemiology that measures the average number of secondary infections produced by one infected person in a population where everyone is susceptible. To compute R_0 , we apply the next-generation matrix approach [40,41], in which the matrix N captures the new infection terms, while the matrix G represents the transitions between compartments as defined in model (3).

$$N = \begin{bmatrix} 0 & f_1(S_0)f_2'(0) & f_1(S_0)f_3'(0) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad G = \begin{bmatrix} b_1 & 0 & -(1-p)v \\ -\alpha & b_2 & 0 \\ 0 & -\gamma & b_3 \end{bmatrix}.$$

and

$$NG^{-1} = \frac{1}{b_1 b_2 b_3 - \alpha \gamma (1-p)v} \begin{bmatrix} \alpha b_3 f_1(S_0)f_2'(0) + \alpha \gamma f_1(S_0)f_3'(0) & b_1 b_2 b_3 f_1(S_0)f_2'(0) & X \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

where $X = b_1 b_2 b_3 f_1(S_0) f_3'(0) - f_1(S_0) f_2'(0)(1-p)v$. We assume that

$$b_1 b_2 b_3 - \alpha \gamma (1-p)v > 0.$$

Accordingly, R_0 representing the spectral radius of the matrix NG^{-1} , can be written as

$$R_0 = \frac{\alpha b_3 f_1(S_0) f_2'(0) + \alpha \gamma f_1(S_0) f_3'(0)}{b_1 b_2 b_3 - \alpha \gamma (1-p)v}.$$

For $p = 1$, we have

$$R_{01} = \frac{\alpha b_3 f_1(S_0) f_2'(0) + \alpha \gamma f_1(S_0) f_3'(0)}{b_1 b_2 b_3} < R_0.$$

This result indicates that an increase in the proportion of incomplete treatment is associated with an increase in the basic reproduction number, and consequently with a higher number of new infections. Conversely, when treatment is sufficiently effective and the proportion of incomplete treatment is reduced, the reproduction number decreases, leading to a reduction in the number of new cases. Therefore, ensuring treatment completion and improving treatment effectiveness are essential for reducing disease transmission and controlling the epidemic.

Proposition 2. Suppose that $\Lambda_E = \Lambda_I = \Lambda_T = 0$, that $D = b_1 b_2 b_3 - (1-p)v\alpha\gamma > 0$ and that hypotheses (H1)–(H4) are satisfied.

1. When $R_0 < 1$, the system (3) admits a disease-free state given by $x_{DFE} = (S_0, 0, 0, 0)$, where $S_0 = \frac{\Lambda_S}{\xi}$.
2. When $R_0 > 1$, the system (3) has at least one endemic state denoted by $x_E^* = (S^*, E^*, I^*, T^*)$.

Proof. By setting the equations equal to 0

$$\Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \xi S = 0. \quad (9)$$

$$f_1(S)(f_2(I) + f_3(T)) - b_1 E + (1-p)vT = 0. \quad (10)$$

$$\alpha E - b_2 I = 0. \quad (11)$$

$$\gamma I - b_3 T = 0. \quad (12)$$

Multiplying equation (4) by γ , the equation (5) by b_2 and summing we get

$$E = \frac{b_2 b_3 T}{\alpha \gamma}$$

Then E , I , and T is given as a function of S by $E = \frac{b_2 b_3 (\Lambda_S - \xi S)}{b_1 b_2 b_3 - \alpha \gamma (1-p)v}$, $I = \frac{\alpha b_3 (\Lambda_S - \xi S)}{b_1 b_2 b_3 - \alpha \gamma (1-p)v}$, and $T = \frac{\alpha \gamma (\Lambda_S - \xi S)}{b_1 b_2 b_3 - \alpha \gamma (1-p)v}$.

Therefore, finding the solution to the system (9)–(12) reduces to solving the following equation

$$\Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \xi S = 0. \quad (13)$$

This leads to an equation involving only the variable S .

Define the function $\phi(S) = \Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \xi S$.

Observe that $\phi(S_0) = 0$, hence one solution of (13) is the disease-free equilibrium x_{DFE} .

Furthermore, Equation (13) admits a solution within $(0, S_0)$. Indeed, we note that $\phi(0) = \Lambda_S$, and it is sufficient to verify that $\phi'(S_0) > 0$. ϕ' is given by

$$\phi'(S) = -\xi - f_1'(S)(f_2(I) + f_3(T)) - f_1(S) \left(f_2'(I) \frac{dI}{dS} + f_3'(T) \frac{dT}{dS} \right)$$

we assess this derivative at $S = S_0$, we get

$$\begin{aligned} \phi'(S_0) &= -\xi - f_1(S_0) \left(-f_2'(0) \frac{\xi \alpha b_3}{b_1 b_2 b_3 - \alpha \gamma (1-p) \nu} - f_3'(0) \frac{\xi \alpha \gamma}{b_1 b_2 b_3 - \alpha \gamma (1-p) \nu} \right) \\ &= \xi \left[\frac{\alpha b_3 f_1(S_0) f_2'(0) + \alpha \gamma f_1(S_0) f_3'(0)}{b_1 b_2 b_3 - \alpha \gamma (1-p) \nu} - 1 \right] \\ &= \xi (R_0 - 1) \end{aligned}$$

It follows that $\phi'(S_0) > 0$ whenever $R_0 > 1$.

Therefore, there exists at least one solution $S^* \in (0, S_0)$ to Equation (13).

Consequently, system (3) admits at least one endemic equilibrium (EE), denoted by $x_E^* = (S^*, E^*, I^*, T^*)$. $\square$

4.3. Global Stability

Theorem 4. Suppose that $\Lambda_E = \Lambda_I = \Lambda_T = 0$, that hypotheses (H1)–(H4) are satisfied, and that the basic reproduction number R_0 is less than unity. Then the disease-free equilibrium x_{DFE} is globally asymptotically stable.

Proof. Applying the approach of Korobeinikov in [17,20].

Let the function defined by

$$V_k = x_1 \left(S - \int_{\rho}^S \frac{f_1(S_0)}{f_1(\tau)} d\tau \right) + x_1 E + x_2 I + x_3 T,$$

where $x_1 = \frac{\alpha b_3 f_2'(0) + \alpha \gamma f_3'(0)}{b_1 b_2 b_3 - \alpha \gamma (1-p) \nu}$, $x_2 = \frac{x_1 b_1}{\alpha}$, $x_3 = \frac{f_3'(0) + x_1 (1-p) \nu}{b_3}$ and ρ is an arbitrary positive constant.

V_k' is given by

$$\begin{aligned} \frac{dV_k}{dt} &= x_1 \dot{S} \left(1 - \frac{f_1(S_0)}{f_1(S)} \right) + x_1 \dot{E} + x_2 \dot{I} + x_3 \dot{T} \\ &= x_1 (\Lambda_S - \xi - f_1(S)[f_2(I) + f_3(T)]) \left(1 - \frac{f_1(S_0)}{f_1(S)} \right) + x_1 [f_1(S)(f_2(I) + f_3(T)) - b_1 E] \\ &\quad + x_2 (\alpha E - b_2 I) + x_3 (\gamma I - b_3 T) \\ &= x_1 \xi (S_0 - S) \left(1 - \frac{f_1(S_0)}{f_1(S)} \right) - x_1 f_1(S)[f_2(I) + f_3(T)] + x_1 f_1(S_0)[f_2(I) + f_3(T)] \\ &\quad + x_1 f_1(S)[f_2(I) + f_3(T)] - x_1 b_1 E + x_1 (1-p) \nu T + x_2 \alpha E - x_2 b_2 I + x_3 \gamma I - x_3 b_3 T \\ &= x_1 \xi S_0 \left(1 - \frac{S}{S_0} \right) \left(1 - \frac{f_1(S_0)}{f_1(S)} \right) + x_1 f_1(S_0)[f_2(I) + f_3(T)] - f_2'(0) I - f_3'(0) T \\ &= x_1 \xi S_0 \left(1 - \frac{S}{S_0} \right) \left(1 - \frac{f_1(S_0)}{f_1(S)} \right) + f_2'(0) I \left(\frac{x_1 f_1(S_0)}{f_2'(0)} \frac{f_2(I)}{I} - 1 \right) \\ &\quad + f_3'(0) T \left(\frac{x_1 f_1(S_0)}{f_3'(0)} \frac{f_3(T)}{T} - 1 \right) \end{aligned}$$

Since the functions $T \rightarrow \frac{f_3(T)}{T}$ and $I \rightarrow \frac{f_2(I)}{I}$ are non-increasing then $\frac{f_3(T)}{T} \leq \lim_{T \rightarrow 0} \frac{f_3(T)}{T} = f'_3(0)$ and $\frac{f_2(I)}{I} \leq \lim_{I \rightarrow 0} \frac{f_2(I)}{I} = f'_2(0)$. By the fact that $R_0 = x_1 f_1(S_0)$, we obtain

$$\frac{dV_k}{dt} \leq x_1 \zeta S_0 \left(1 - \frac{S}{S_0}\right) \left(1 - \frac{f_1(S_0)}{f_1(S)}\right) + f'_2(0)I(R_0 - 1) + f'_3(0)T(R_0 - 1)$$

Suppose that $\frac{dV_k}{dt} = 0$. Under this condition, we deduce that either $S = S_0$ and $T = I = 0$, or $R_0 = 1$. Hence, the largest invariant subset of the domain $\mathcal{A}$ contained in $\{(S, E, I, T) \in \mathbb{R}_+^4 : \dot{V}_k = 0\}$ reduces to the disease-free equilibrium x_{DFE} . By invoking LaSalle's invariance principle (see [36,37]), it follows that the disease-free equilibrium x_{DFE} is globally asymptotically stable. $\square$

Theorem 5. Assume that $\Lambda_E = \Lambda_I = \Lambda_T = 0$, that hypotheses **(H1)–(H4)** are satisfied, and that $D = b_1 b_2 b_3 - (1-p)\nu\alpha\gamma > 0$. Furthermore, suppose that $f''_1(S) < 0$ for all $S > 0$ and that the basic reproduction number satisfies $R_0 > 1$. Then, under these assumptions, the unique endemic equilibrium x_E^* is globally asymptotically stable in the feasible region.

Proof. We establish the global asymptotic stability of the endemic equilibrium

$E^* = (S^*, E^*, I^*, T^*)$, by means of an appropriate Lyapunov functional similar to that of [17,20].

Consider the Lyapunov function

$$\begin{aligned} V(S, E, I, T) &= V_S + V_E + V_I + V_T \\ &= S - S^* - \int_{S^*}^S \frac{f_1(\tau)}{f_1(S^*)} d\tau + E - E^* - E^* \ln\left(\frac{E}{E^*}\right) \\ &\quad + c_1 \left(I - I^* - I^* \ln\left(\frac{I}{I^*}\right)\right) \\ &\quad + c_2 \left(T - T^* - T^* \ln\left(\frac{T}{T^*}\right)\right), \end{aligned} \quad (14)$$

where

$$c_1 = \frac{b_1}{\alpha}, \quad c_2 = \frac{(1-p)\nu T^* + f_1(S^*)f_3(T^*)}{\gamma I^*}. \quad (15)$$

The time derivative of V along the solutions of the model is given by

$$\begin{aligned} \frac{dV}{dt} &= V'_S + V'_E + V'_I + V'_T \\ &= S' \left(1 - \frac{f_1(S^*)}{f_1(S)}\right) + E' \left(1 - \frac{E^*}{E}\right) \\ &\quad + c_1 I' \left(1 - \frac{I^*}{I}\right) + c_2 T' \left(1 - \frac{T^*}{T}\right). \end{aligned} \quad (16)$$

For the susceptible component, we obtain

$$V'_S = [\Lambda_S - f_1(S)(f_2(I) + f_3(T)) - \zeta S] \left(1 - \frac{f_1(S^*)}{f_1(S)}\right). \quad (17)$$

For the exposed component,

$$V'_E = [f_1(S)(f_2(I) + f_3(T)) - b_1 E + (1-p)\nu T] \left(1 - \frac{E^*}{E}\right). \quad (18)$$

For the infectious component,

$$V'_I = c_1(\alpha E - b_2 I) \left(1 - \frac{I^*}{I}\right). \quad (19)$$

Finally, for the treated component,

$$V'_T = c_2(\gamma I - b_3 T) \left(1 - \frac{T^*}{T}\right). \quad (20)$$

Using the equilibrium relations

$$\Lambda_S = f_1(S^*)(f_2(I^*) + f_3(T^*)) + \xi S^*, \quad (21)$$

$$b_1 E^* = (1 - p)\nu T^* + f_1(S^*)(f_2(I^*) + f_3(T^*)), \quad (22)$$

$$\alpha E^* = b_2 I^*, \quad (23)$$

and

$$\gamma I^* = b_3 T^*, \quad (24)$$

we can rewrite each term of $\dot{V}$ in a form suitable for establishing its sign.

In particular, substituting (21) into (17) gives

$$\begin{aligned} V'_S = & \xi S^* \left(1 - \frac{S}{S^*}\right) \left(1 - \frac{f_1(S^*)}{f_1(S)}\right) \\ & + f_1(S^*) f_2(I^*) \left(1 - \frac{f_1(S^*)}{f_1(S)}\right) \\ & + f_1(S^*) f_3(T^*) \left(1 - \frac{f_1(S^*)}{f_1(S)}\right) \\ & - f_1(S) (f_2(I) + f_3(T)) + f_1(S^*) (f_2(I^*) + f_3(T^*)). \end{aligned} \quad (25)$$

Similarly, using (22), we obtain

$$\begin{aligned} V'_E = & f_1(S) (f_2(I) + f_3(T)) \\ & + f_1(S^*) f_2(I^*) \left(1 - \frac{f_1(S) E^*}{f_1(S^*) E} \frac{f_2(I)}{f_2(I^*)}\right) \\ & + f_1(S^*) f_3(T^*) \left(1 - \frac{f_1(S) E^*}{f_1(S^*) E} \frac{f_3(T)}{f_3(T^*)}\right) \\ & + (1 - p)\nu T^* - b_1 E + (1 - p)\nu T - (1 - p)\nu T^* \frac{T E^*}{E T^*}. \end{aligned} \quad (26)$$

For the infectious compartment, we have

$$\begin{aligned} V'_I = & c_1(\alpha E - b_2 I) \left(1 - \frac{I^*}{I}\right) \\ = & \frac{b_1}{\alpha} (\alpha E - b_2 I) \left(1 - \frac{I^*}{I}\right). \end{aligned} \quad (27)$$

Using the equilibrium relation

$$\alpha E^* = b_2 I^*,$$

this can be written as

$$\begin{aligned}\dot{V}_I = & b_1 E - (1-p)vT^* \frac{I}{I^*} - f_1(S^*)f_2(I^*) \frac{I}{I^*} \\ & - f_1(S^*)f_3(T^*) \frac{I}{I^*} - (1-p)vT^* \frac{EI^*}{E^*I} \\ & - f_1(S^*)f_2(I^*) \frac{EI^*}{E^*I} - f_1(S^*)f_3(T^*) \frac{EI^*}{E^*I} \\ & + (1-p)vT^* + f_1(S^*)f_2(I^*) + f_1(S^*)f_3(T^*).\end{aligned}$$

For the treated compartment, we obtain

$$\begin{aligned}V'_T = & c_2(\gamma I - b_3 T) \left(1 - \frac{T^*}{T}\right) \\ = & c_2 \gamma I - c_2 \gamma I \frac{T^*}{T} - c_2 b_3 T + c_2 b_3 T^*.\end{aligned}$$

Using

$$\gamma I^* = b_3 T^*,$$

the previous expression may equivalently be represented in terms of the ratios I/I^* and T/T^* .

$$\begin{aligned}\dot{V}_T = & f_1(S^*)f_3(T^*) \frac{I}{I^*} + (1-p)vT^* \frac{I}{I^*} - (1-p)vT^* \frac{IT^*}{I^*T} \\ & - f_1(S^*)f_3(T^*) \frac{IT^*}{I^*T} - (1-p)vT - f_1(S^*)f_2(I^*) \frac{I}{I^*} \\ & + (1-p)vT^* + f_1(S^*)f_2(I^*).\end{aligned}$$

Consequently,

$$\dot{V} = V'_S + V'_E + V'_I + V'_T. \quad (28)$$

Collecting the terms obtained above and using the equilibrium identities (21)–(24), we obtain

$$\begin{aligned}\dot{V} = & \zeta S^* \left(1 - \frac{S}{S^*}\right) \left(1 - \frac{f_1(S^*)}{f_1(S)}\right) \\ & + f_1(S^*)f_2(I^*) \left[4 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S)E^*f_2(I)}{f_1(S^*)Ef_2(I^*)} - \frac{EI^*}{E^*I} - \frac{If_2(I^*)}{I^*f_2(I)}\right] \\ & + f_1(S^*)f_2(I^*) \left[\frac{f_2(I^*)}{f_2(I)} - 1 + \frac{If_2(I^*)}{I^*f_2(I)} - \frac{I}{I^*}\right] \\ & + f_1(S^*)f_3(T^*) \left[5 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S)E^*f_3(T)}{f_1(S^*)Ef_3(T^*)} - \frac{EI^*}{E^*I} - \frac{IT^*}{I^*T} - \frac{Tf_3(T^*)}{T^*f_3(T)}\right] \\ & + f_1(S^*)f_3(T^*) \left[\frac{f_3(T^*)}{f_3(T)} - 1 + \frac{Tf_3(T^*)}{T^*f_3(T)} - \frac{T}{T^*}\right] \\ & + (1-p)vT^* \left[3 - \frac{IT^*}{I^*T} - \frac{E^*T}{ET^*} - \frac{EI^*}{E^*I}\right]\end{aligned} \quad (29)$$

The derivative of V can be written as

$$\begin{aligned}\dot{V} = & \zeta S^* \left[1 - \frac{S}{S^*} \right] \left(1 - \frac{f_1(S^*)}{f_1(S)} \right) + f_1(S^*) f_2(I^*) \left[4 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S) E^* f_2(I)}{f_1(S^*) f_2(I^*) E} \right. \\ & \left. - \frac{EI^*}{E^* I} - \frac{I f_2(I^*)}{I^* f_2(I)} \right] \\ & + f_1(S^*) f_2(I^*) \left(\frac{f_2(I)}{f_2(I^*)} - \frac{I}{I^*} \right) \left(1 - \frac{f_2(I^*)}{f_2(I)} \right) \\ & + f_1(S^*) f_3(T^*) \left[5 - \frac{f_1(S^*)}{f_1(S)} - \frac{f_1(S) E^* f_3(T)}{f_1(S^*) f_3(T^*) E} - \frac{EI^*}{E^* I} - \frac{IT^*}{I^* T} - \frac{T f_3(T^*)}{T^* f_3(T)} \right] \\ & + f_1(S^*) f_3(T^*) \left(\frac{f_3(T)}{f_3(T^*)} - \frac{T}{T^*} \right) \left(1 - \frac{f_3(T^*)}{f_3(T)} \right) \\ & + (1-p) \nu T^* \left[3 - \frac{TE^*}{T^* E} - \frac{EI^*}{E^* I} - \frac{IT^*}{I^* T} \right]\end{aligned}$$

Under hypotheses **(H1)–(H4)**, the arithmetic mean–geometric mean inequality implies that

$$\dot{V} \leq 0. \quad (30)$$

Moreover,

$$\dot{V} = 0$$

can hold only when

$$S = S^*, \quad E = E^*, \quad I = I^*, \quad T = T^*.$$

Hence, the largest invariant subset of

$$\{(S, E, I, T) : \dot{V} = 0\}$$

is the singleton

$$\{(S^*, E^*, I^*, T^*)\}.$$

By LaSalle's invariance principle, the endemic equilibrium $E^* = (S^*, E^*, I^*, T^*)$ is globally asymptotically stable in the feasible region. $\square$

5. Local Bifurcation Analysis

To investigate the local dynamics of system (3) (omitting the migration effect) in a neighborhood of the disease-free equilibrium, we restrict our attention to the following particular choice of the incidence functions

$$f_1(S) = \beta S, \quad f_2(I) = \frac{I}{1 + \alpha_I I}, \quad f_3(T) = \frac{\psi T}{1 + \alpha_T T},$$

where $\beta, \psi > 0$, while α_I and α_T denote the saturation coefficients associated with infectious and treated individuals, respectively.

For notational convenience, we also define

$$b_1 = \zeta + \alpha, \quad b_2 = \zeta + \gamma + \delta_I, \quad b_3 = \zeta + \delta_T + \nu.$$

Under these assumptions, the general system reduces to

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda_S - \beta S \left(\frac{I}{1 + \alpha_I I} + \frac{\psi T}{1 + \alpha_T T} \right) - \xi S, \\ \frac{dE(t)}{dt} = \beta S \left(\frac{I}{1 + \alpha_I I} + \frac{\psi T}{1 + \alpha_T T} \right) - b_1 E + (1 - p)\nu T, \\ \frac{dI(t)}{dt} = \alpha E - b_2 I, \\ \frac{dT(t)}{dt} = \gamma I - b_3 T, \end{cases} \quad (31)$$

Consequently, the basic reproduction number is expressed as

$$R_0 = \frac{\beta \Lambda_S \alpha (b_3 + \psi \gamma)}{\xi (b_1 b_2 b_3 - \alpha \gamma (1 - p) \nu)}.$$

We employ the center manifold approach developed by Castillo-Chavez and Song [30]. This method enables us to determine the nature of the bifurcation occurring when the basic reproduction number crosses the critical threshold $R_0 = 1$. Throughout this section, the transmission coefficient β is chosen as the bifurcation parameter.

The vector field associated with system (31) is of class C^2 in a neighborhood of the disease-free equilibrium since $1 + \alpha_I I > 0$ and $1 + \alpha_T T > 0$ for all biologically feasible solutions.

Theorem 6. *The model (31) undergoes a forward (transcritical) bifurcation at the disease-free equilibrium E_0 when $R_0 = 1$.*

Proof. The disease-free equilibrium of system (31) is

$$E_0 = \left(\frac{\Lambda_S}{\xi}, 0, 0, 0 \right),$$

and the basic reproduction number is

$$R_0 = \frac{\beta \Lambda_S \alpha (b_3 + \psi \gamma)}{\xi (b_1 b_2 b_3 - \alpha \gamma (1 - p) \nu)}.$$

Choosing the transmission rate β as the bifurcation parameter, the critical value corresponding to $R_0 = 1$ is

$$\beta^* = \frac{\xi (b_1 b_2 b_3 - \alpha \gamma (1 - p) \nu)}{\Lambda_S \alpha (b_3 + \psi \gamma)}.$$

The Jacobian matrix of (31) evaluated at E_0 is

$$J(E_0) = \begin{pmatrix} -\xi & 0 & -\beta S^0 & -\beta \psi S^0 \\ 0 & -b_1 & \beta S^0 & \beta \psi S^0 + (1 - p)\nu \\ 0 & \alpha & -b_2 & 0 \\ 0 & 0 & \gamma & -b_3 \end{pmatrix}, \quad S^0 = \frac{\Lambda_S}{\xi}.$$

Its characteristic polynomial is

$$(\lambda + \xi) \left[(\lambda + b_1)(\lambda + b_2)(\lambda + b_3) - \alpha \beta S^0 (\lambda + b_3) - \alpha \gamma (\beta \psi S^0 + (1 - p)\nu) \right] = 0.$$

Using the expression of R_0 , one readily obtains

$$\det(J(E_0)) = \xi (b_1 b_2 b_3 - \alpha \gamma (1 - p) \nu) (1 - R_0),$$

which shows that $\lambda = 0$ is an eigenvalue if and only if $R_0 = 1$, or equivalently $\beta = \beta^*$.

At $\beta = \beta^*$, the characteristic polynomial factorizes as

$$(\lambda + \xi)\lambda(\lambda^2 + A_1\lambda + A_2) = 0,$$

where

$$A_1 = b_1 + b_2 + b_3,$$

and

$$A_2 = b_1b_2 + b_1b_3 + b_2b_3 - \alpha\beta^*S^0.$$

Since

$$\alpha\beta^*S^0 = \frac{b_1b_2b_3 - \alpha\gamma(1-p)\nu}{b_3 + \psi\gamma},$$

$$(b_3 + \psi\gamma)A_2 = b_1b_3^2 + b_2b_3^2 + \psi\gamma b_1b_2 + \psi\gamma b_1b_3 + \psi\gamma b_2b_3 + \alpha\gamma(1-p)\nu > 0.$$

Hence

$$A_1 > 0, \quad A_2 > 0.$$

By the Routh–Hurwitz criterion, the two roots of

$$\lambda^2 + A_1\lambda + A_2 = 0$$

have negative real parts. Consequently, at $R_0 = 1$, the Jacobian matrix possesses a simple zero eigenvalue, whereas all remaining eigenvalues lie strictly in the left half-plane. Therefore, the spectral assumptions of the Castillo–Chavez and Song Theorem are satisfied.

Let $w = (w_1, w_2, w_3, w_4)^T$ and $v = (v_1, v_2, v_3, v_4)^T$ denote the right and left eigenvectors associated with the simple zero eigenvalue, normalized by

$$v \cdot w = 1.$$

Solving

$$J(E_0, \beta^*)w = 0, \quad J(E_0, \beta^*)^T v = 0,$$

gives

$$w = \left(-\frac{\beta^*S^0}{\xi} \left(1 + \frac{\psi\gamma}{b_3} \right), \frac{b_2}{\alpha}, 1, \frac{\gamma}{b_3} \right)^T,$$

and

$$v = \frac{1}{\Delta} \left(0, 1, \frac{b_1}{\alpha}, \frac{\beta^*\psi S^0 + (1-p)\nu}{b_3} \right)^T,$$

where

$$\Delta = \frac{b_1 + b_2}{\alpha} + \frac{\gamma}{b_3^2} (\beta^*\psi S^0 + (1-p)\nu).$$

The vector field is of class C^2 in a neighborhood of E_0 . Moreover, only the first two components are non-linear; hence, the second-order derivatives of F_3 and F_4 vanish identically.

The only non-vanishing second-order partial derivatives of the vector field at (E_0, β^*) are those associated with the non-linear incidence terms of F_1 and F_2 , namely

$$\begin{aligned}\frac{\partial^2 F_1}{\partial S \partial I} &= -\beta^*, & \frac{\partial^2 F_1}{\partial I^2} &= 2\beta^* \alpha_I S^0, \\ \frac{\partial^2 F_1}{\partial S \partial T} &= -\beta^* \psi, & \frac{\partial^2 F_1}{\partial T^2} &= 2\beta^* \psi \alpha_T S^0,\end{aligned}$$

and

$$\begin{aligned}\frac{\partial^2 F_2}{\partial S \partial I} &= \beta^*, & \frac{\partial^2 F_2}{\partial I^2} &= -2\beta^* \alpha_I S^0, \\ \frac{\partial^2 F_2}{\partial S \partial T} &= \beta^* \psi, & \frac{\partial^2 F_2}{\partial T^2} &= -2\beta^* \psi \alpha_T S^0.\end{aligned}$$

All remaining second-order derivatives vanish.

According to the Theorem of Castillo–Chavez and Song, the bifurcation coefficient b is given by

$$b = \sum_{k,i=1}^4 v_k w_i \frac{\partial^2 F_k}{\partial x_i \partial \beta}(E_0, \beta^*).$$

Since the parameter β appears only in the incidence terms and $v_1 = 0$, only the second component contributes. Furthermore,

$$\frac{\partial^2 F_2}{\partial I \partial \beta} = S^0, \quad \frac{\partial^2 F_2}{\partial T \partial \beta} = \psi S^0,$$

whereas all other mixed derivatives with respect to β vanish. Hence,

$$\begin{aligned}b &= v_2 (w_3 S^0 + w_4 \psi S^0) \\ &= v_2 S^0 \left(1 + \frac{\psi \gamma}{b_3}\right),\end{aligned}$$

where we have used

$$w_3 = 1, \quad w_4 = \frac{\gamma}{b_3}.$$

Substituting the normalization constant

$$v_2 = \frac{1}{\Delta},$$

yields

$$b = \frac{S^0 \left(1 + \frac{\psi \gamma}{b_3}\right)}{\frac{b_1 + b_2}{\alpha} + \frac{\gamma}{b_3^2} (\beta^* \psi S^0 + (1-p)\nu)}.$$

Since all model parameters are positive,

$$b > 0.$$

It remains to compute the first bifurcation coefficient a .

The first bifurcation coefficient is defined by

$$a = \sum_{k,i,j=1}^4 v_k w_i w_j \frac{\partial^2 F_k}{\partial x_i \partial x_j}(E_0, \beta^*).$$

Since $v_1 = 0$ and the components F_3 and F_4 are linear, only F_2 contributes. Therefore,

$$a = v_2 \sum_{i,j=1}^4 w_i w_j \frac{\partial^2 F_2}{\partial x_i \partial x_j}(E_0, \beta^*).$$

Using the symmetry of the Hessian matrix together with the previously computed non-vanishing second-order derivatives, we obtain

$$a = 2\beta^* v_2 (w_1 w_3 + \psi w_1 w_4 - \alpha_I S^0 w_3^2 - \psi \alpha_T S^0 w_4^2).$$

Substituting

$$w_3 = 1, \quad w_4 = \frac{\gamma}{b_3},$$

and

$$w_1 = -\frac{\beta^* S^0}{\xi} \left(1 + \frac{\psi \gamma}{b_3}\right),$$

yields

$$a = 2\beta^* v_2 \left[-\frac{\beta^* S^0}{\xi} \left(1 + \frac{\psi \gamma}{b_3}\right)^2 - \alpha_I S^0 - \frac{\psi \alpha_T \gamma^2 S^0}{b_3^2} \right].$$

Since

$$\beta^* > 0, \quad v_2 > 0, \quad S^0 > 0, \quad \alpha_I > 0, \quad \alpha_T > 0, \quad b_3 > 0,$$

every term inside the brackets is strictly negative. Hence,

$$a < 0.$$

Together with the previously established inequality

$$b > 0,$$

all hypotheses of the Castillo–Chavez and Song Theorem are satisfied. Therefore, system (31) undergoes a forward (transcritical) bifurcation at the disease-free equilibrium when $R_0 = 1$. Consequently, for $R_0 > 1$ sufficiently close to unity, a unique endemic equilibrium bifurcates from E_0 and is locally asymptotically stable. $\square$

The bifurcation analysis above is based on the existence of a classical disease-free equilibrium and is therefore carried out for the baseline model without population influx into the disease-related compartments. For the full model with population influx, if $\Lambda_E > 0$, $\Lambda_I > 0$, or $\Lambda_T > 0$, a classical disease-free equilibrium satisfying $E^* = I^* = T^* = 0$ does not exist. Consequently, the standard basic reproduction number based on a disease-free equilibrium is not directly applicable in this setting. Therefore, the usual transcritical bifurcation associated with the crossing of the threshold $\mathcal{R}_0 = 1$ cannot be formulated in the classical DFE-based framework. In this case, population influx into disease-related compartments continuously maintains the epidemic subsystem away from the disease-free state. The effect of such influxes is therefore investigated separately through the equilibrium structure and numerical comparisons presented in the subsequent section.

6. Global Sensitivity Analysis of R_0

In this study, a global sensitivity analysis is performed to rigorously assess the influence of model parameters on the basic reproduction number R_0 , and to identify the most influential parameters contributing to its variation. Unlike local sensitivity analysis,

which is limited to examining small perturbations around fixed values, the global approach allows for a quantitative evaluation across the entire parameter space. It accounts for not only the individual effects of each parameter, but also for their potential interactions. Among the available techniques, the Sobol indices method was chosen for its ability to decompose the variance of R_0 into three main components: the first-order effect (main sensitivity index, MSI), which measures the contribution of a parameter considered in isolation, the total effect (total sensitivity index, TSI), which includes both the direct effect and all interactions with other parameters and the interaction effects, representing the share of variance attributable to parameter combinations. The MSI, TSI, and interaction effects are then computed using the standard formulas defined by Sobol's method.

$$MSI_i = \frac{\text{Var}_i}{\text{Var}_{\text{tot}}} \quad (32)$$

$$TSI_i = 1 - \frac{\text{Var}_{\sim i}}{\text{Var}_{\text{tot}}} \quad (33)$$

$$\sum_{\substack{i=1 \\ i \neq j}}^n S_{i,j} = TSI_i - MSI_i \quad (34)$$

1. Var_i : the variance explained solely by parameter i ,
2. $\text{Var}_{\sim i}$: the variance of the model output when parameter i is held constant,
3. Var_{tot} : the total variance of the basic reproduction number output.

The parameter ranges presented in Table 2 are chosen as broad analytical and exploratory domains to investigate the qualitative behavior of the model and the sensitivity of the basic reproduction number R_0 . These ranges are not intended to represent direct real-world epidemiological observations, population-specific estimates, or calibrated confidence intervals. Rather, they provide a systematic parameter space within which the mathematical properties of the model and the relative influence of the parameters can be examined. In the absence of a specific population-level calibration, uniform distributions are adopted for the global sensitivity analysis to avoid imposing unsupported distributional assumptions on the parameters.

Table 2. Model parameters and their ranges.

Parameter	Range
β	[0, 0.5]
α	[0, 0.1]
Λ_S	[0, 0.5]
ψ	[0, 0.5]
γ	[0, 0.1]
ξ	[0, 0.1]
δ_I	[0, 0.2]
δ_T	[0, 0.2]
ν	[0, 0.2]
p	[0, 0.9]

All parameters follow a uniform distribution.

Sampling was carried out using the Saltelli sampling scheme [42–44], as implemented through the SALib library. A base sample size of $N = 1000$ was used, with the second-order Sobol indices disabled. For each generated parameter vector, the basic reproduction number R_0 was evaluated from its analytical expression. The resulting model outputs were then analyzed using the Sobol variance-based method to compute the first-order indices (S_i) and total-order indices (S_{T_i}). The first-order index quantifies the contribution of

parameter i to the variance of R_0 considered individually, whereas the total-order index accounts for its direct contribution together with all interactions involving that parameter. The difference $S_{T_i} - S_i$ is reported as an aggregate measure of the contribution associated with parameter interactions.

Table 3 presents the first-order and total-order Sobol indices obtained for R_0 . The parameters are ranked according to their total contribution to the variability of the reproduction number. The natural mortality rate ζ exhibits the largest first-order sensitivity index ($MSI_{\zeta} = 0.400$) and the largest total-order index ($TSI_{\zeta} = 0.789$), indicating that it is the most influential parameter over the prescribed parameter domain. The substantial difference between the total-order and first-order indices ($TSI_{\zeta} - MSI_{\zeta} = 0.389$) further indicates important interactions between ζ and other model parameters. The transmission parameter β ranks second, with $MSI_{\beta} = 0.078$ and $TSI_{\beta} = 0.216$. Although its isolated contribution is smaller than that of ζ , its total effect remains substantial, indicating that interactions with other parameters contribute appreciably to the variability of R_0 . The susceptible-population influx Λ_S ranks third, with $MSI_{\Lambda_S} = 0.065$ and $TSI_{\Lambda_S} = 0.217$, showing that population recruitment into the susceptible class can substantially affect the epidemic threshold within the considered parameter domain. The treatment-success parameter p ranks fourth, with $MSI_p = 0.029$ and $TSI_p = 0.180$. Its relatively small first-order contribution but considerably larger total-order effect suggests that its influence on R_0 is partly mediated through interactions with other epidemiological parameters. The disease-induced mortality rate δ_I ranks fifth, with $MSI_{\delta_I} = 0.019$ and $TSI_{\delta_I} = 0.128$. The remaining parameters exhibit comparatively smaller total-order indices, with γ , δ_T , ψ , ν , and α ranking below the five most influential parameters. In particular, the difference between the first-order and total-order indices for several parameters indicates that parameter interactions should not be neglected when interpreting the variability of R_0 .

Overall, the sensitivity analysis identifies ζ , β , Λ_S , p , and δ_I as the five most influential parameters within the prescribed analytical parameter domain. From a control perspective, these results suggest that interventions affecting transmission and treatment success, together with appropriate management of susceptible-population recruitment, may substantially modify the epidemic threshold. These conclusions should be interpreted as model-based sensitivity findings rather than population-specific epidemiological estimates, since the parameter ranges are exploratory and the analysis is not calibrated to a particular dataset. Figure 2 illustrates the first-order and total-order Sobol indices obtained for R_0 .

Table 3. Sobol sensitivity indices for R_0 with interaction effects and ranking.

Parameter	First-Order (MSI)	Total-Order (TSI)	Interaction (TSI - MSI)	Rank
ζ	0.400	0.789	0.389	1
β	0.078	0.216	0.138	2
Λ_S	0.065	0.217	0.152	3
p	0.029	0.180	0.151	4
δ_I	0.019	0.128	0.109	5
γ	0.015	0.070	0.056	6
δ_T	0.002	0.025	0.023	7
ψ	0	0.069	0.069	8
ν	0	0.032	0.032	9
α	0	0.007	0.007	10

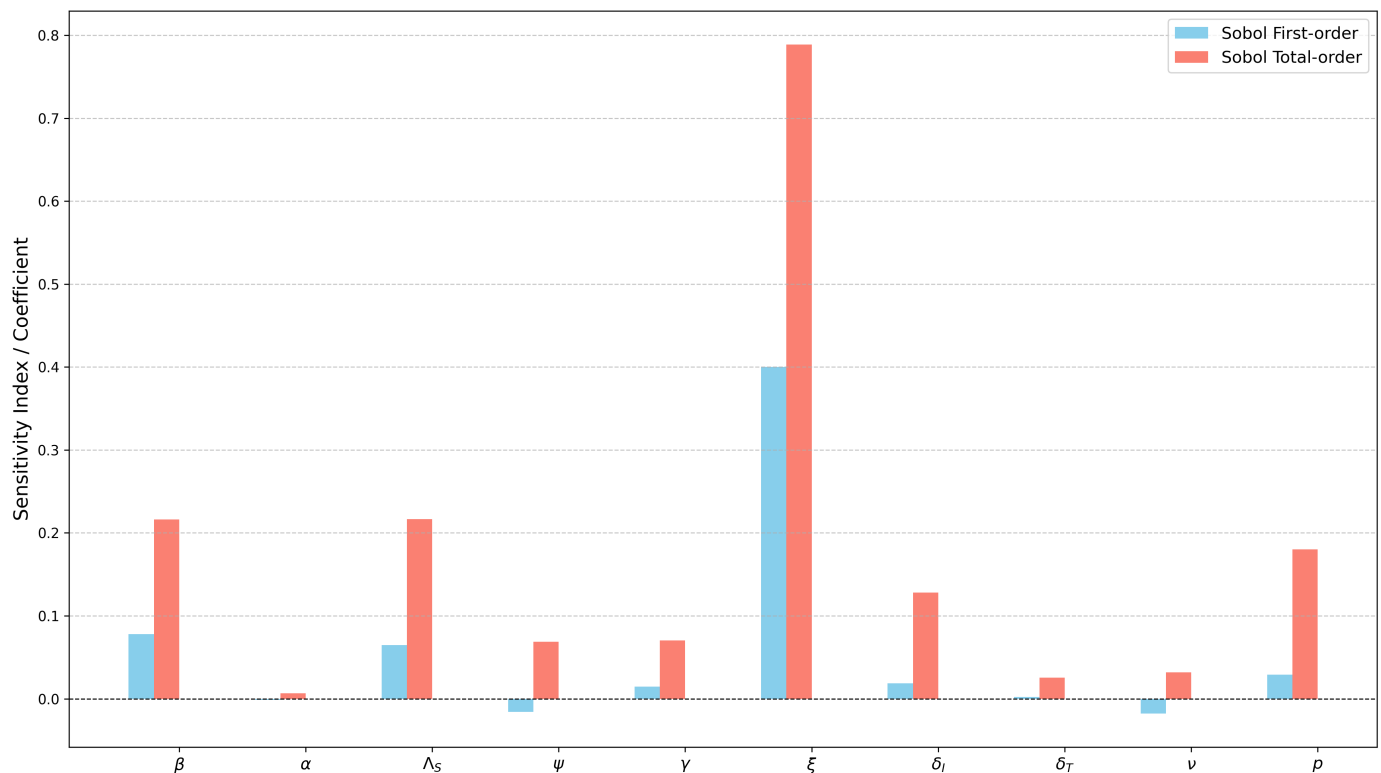


Figure 2. Sensitivity analysis of R_0 with respect to each parameter (Sobol method).

7. Illustrative Examples

This section presents numerical simulations of the SEITR model described by system (31). The simulations complement the analytical results established in the preceding sections and illustrate the effects of key parameters on tuberculosis transmission dynamics. All numerical simulations were performed in Python using the NumPy, SciPy, and Matplotlib libraries. The system of ordinary differential equations was solved using the `solve_ivp` function with the LSODA algorithm. The simulations were conducted over the time interval $[0, 4000]$ using the specified initial conditions and the parameter values reported in Table 4. We investigate the effects of population influxes on disease persistence, compare the temporal dynamics under bilinear and non-linear saturated incidence, and examine the influence of treatment efficacy on the basic reproduction number $\mathcal{R}_0$. A forward bifurcation diagram is also included to illustrate the relationship between the reproduction threshold and the emergence of an endemic equilibrium. These numerical results provide further insight into the epidemiological implications of the mechanisms incorporated into the model and illustrate the analytical findings.

Table 4. Parameters and their values: $R_0 = 0.9 < 1$.

Parameter	Value	Parameter	Value
β	0.001	δ_T	0.001
Λ_S	0.01	p	0.2
ξ	0.001	α_I	0.3
α	0.02	α_T	0.3
γ	0.03	ψ	0.001
ν	0.03	δ_I	0.001

Figure 3 presents the forward transcritical bifurcation diagram obtained by plotting the endemic infectious population I^* against the basic reproduction number $\mathcal{R}_0$. For the

parameter set considered, the critical transmission rate is $\beta_c = 1.10896 \times 10^{-3}$, for which $\mathcal{R}_0(\beta_c) = 1$. The disease-free equilibrium corresponds to the branch $I^* = 0$, which persists for $\mathcal{R}_0 < 1$. At the critical threshold $\mathcal{R}_0 = 1$, corresponding to $\beta = \beta_c$, the endemic branch emerges continuously from the disease-free equilibrium and becomes positive for $\mathcal{R}_0 > 1$. This numerical behavior is consistent with the analytical bifurcation analysis and confirms the occurrence of a forward transcritical bifurcation at $\mathcal{R}_0 = 1$. In particular, the continuous emergence of the endemic branch from the disease-free state illustrates the transition from disease elimination to disease persistence as the transmission rate crosses the critical value β_c .

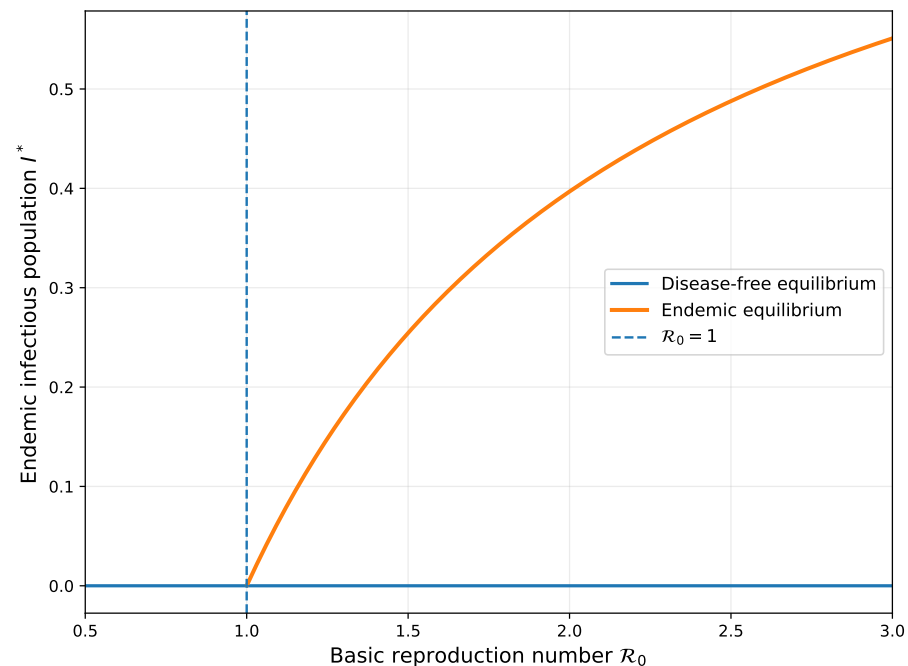


Figure 3. Forward transcritical bifurcation diagram showing the endemic infectious population I^* as a function of the basic reproduction number $\mathcal{R}_0$.

Figure 4 illustrates the SEIT dynamics under two scenarios. Without population influx ($\Lambda_S = \Lambda_E = \Lambda_I = \Lambda_T = 0$), the exposed, infectious, and treated populations peak early and subsequently decay toward zero. With migration ($\Lambda_S = \Lambda_E = \Lambda_T = 0.02$, $\Lambda_I = 0.01$), these compartments converge to positive endemic levels, while the susceptible population approaches a positive level above its zero-influx limit. The comparison highlights the qualitative effect of population influx, shifting the long-term outcome from disease extinction to persistence at an endemic equilibrium, while leaving the transient peak timing broadly similar.

Figure 5 contrasts the system trajectories under bilinear transmission ($\alpha_I = \alpha_T = 0$) against saturated incidence ($\alpha_I = \alpha_T = 0.3$), holding all other parameters and initial conditions fixed. The choice of transmission function profoundly impacts the transient dynamics: bilinear incidence drives pronounced epidemic surges in the exposed, infectious, and treated classes, whereas saturated incidence dampens peak disease prevalence and attenuates the depletion rate of the susceptible pool. Mathematically, because the saturation coefficients (α_I, α_T) vanish upon linearization around the disease-free state, they do not alter the threshold formula for the basic reproduction number $\mathcal{R}_0$. Instead, their inhibitory effect operates exclusively in non-equilibrium regimes by modeling behavioral adjustments or contact saturation during high-prevalence phases. These trajectories demonstrate that while $\mathcal{R}_0$ dictates ultimate threshold outcomes, transient epidemic magnitude and healthcare

burden are heavily governed by the functional form of non-linear transmission. Finally, as these simulations depict a finite time interval, the observed convergence toward similar low-prevalence regimes represents numerical support for asymptotic state equivalence rather than analytical proof of it.

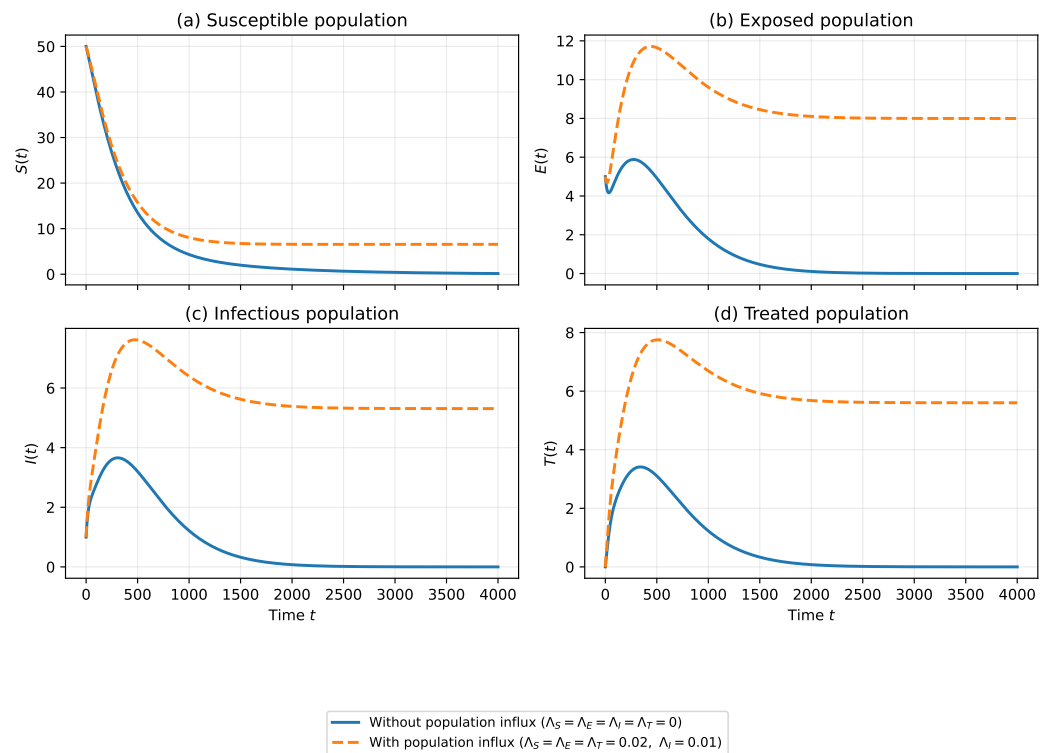


Figure 4. Comparison of model dynamics across all compartments with and without migration.

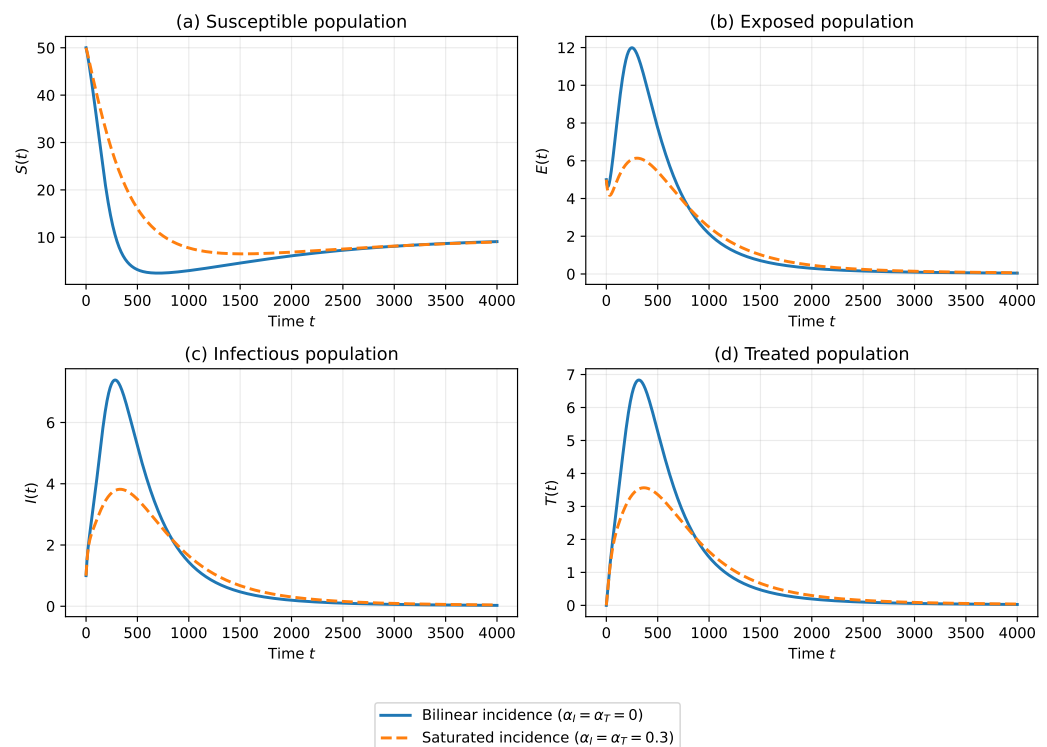


Figure 5. Comparison of transient SEIT dynamics under bilinear ($\alpha_I = \alpha_T = 0$) and saturated ($\alpha_I = \alpha_T = 0.3$) incidence formulations for $\mathcal{R}_0 < 1$. Numerical integration resolved via the LSODA algorithm ($t \in [0, 4000]$ days; tolerances: Abs = 10^{-11} , Rel = 10^{-9}).

Figure 6 illustrates the effect of treatment effectiveness p on the basic reproduction number R_0 for the parameter set corresponding to the endemic regime. The results reveal a pronounced and monotone decrease in R_0 as the proportion of successfully treated individuals increases. Specifically, when treatment is completely ineffective ($p = 0$), the reproduction number reaches $R_0 = 9.141$, indicating substantial transmission potential. Increasing the treatment effectiveness to $p = 0.2, 0.4, 0.6$, and 0.8 reduces R_0 to 4.509, 2.992, 2.239, and 1.789, respectively. Under complete treatment effectiveness ($p = 1$), R_0 attains its minimum value of 1.489. These results demonstrate that increasing the proportion of successful treatment substantially suppresses transmission by reducing the contribution of treatment failure to the replenishment of the exposed class.

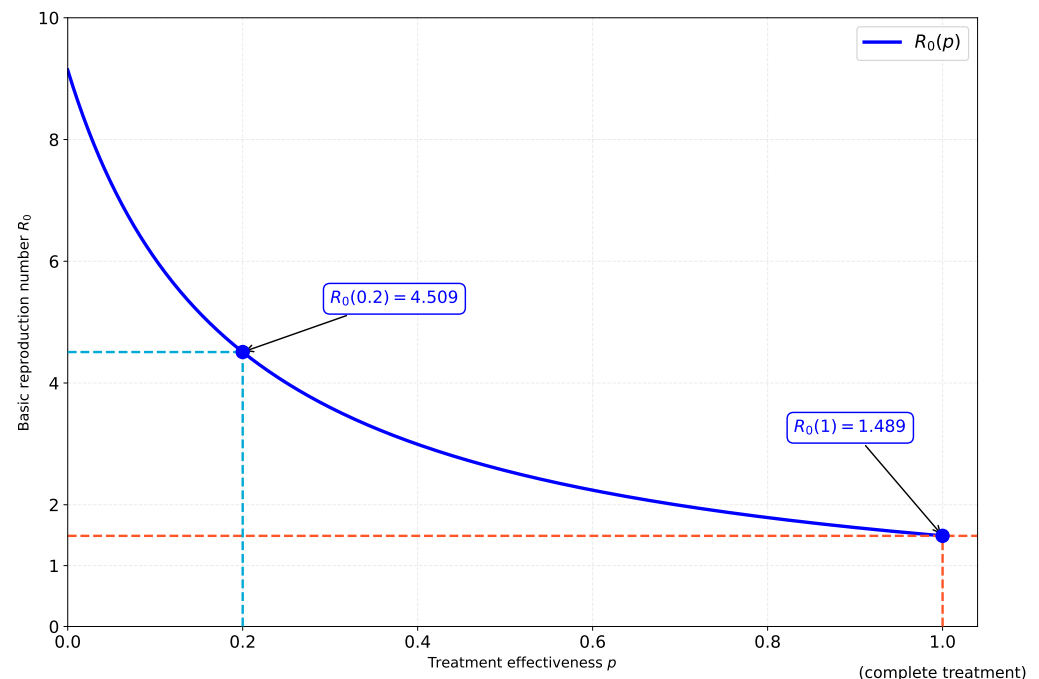


Figure 6. Impact of treatment effectiveness (p) on the basic reproduction number R_0 .

Nevertheless, R_0 remains above unity throughout the biologically admissible interval $0 \leq p \leq 1$. The critical treatment effectiveness obtained from the threshold condition $R_0(p_c) = 1$ is $p_c = 1.585$, which exceeds the maximum feasible value $p = 1$. Hence, even complete treatment effectiveness is insufficient to reduce the reproduction number below the epidemic threshold for the parameter set considered. This finding indicates that treatment alone cannot guarantee disease elimination in this regime and that additional control measures targeting other transmission-related parameters may be required. In particular, the result highlights the importance of combining effective treatment with interventions aimed at reducing the transmission rate and/or the recruitment of susceptible individuals. From a modeling perspective, the figure also confirms the analytical property $\partial R_0 / \partial p < 0$, showing that treatment effectiveness acts as a protective parameter, although its impact is insufficient, by itself, to achieve $R_0 < 1$ under the present parameter configuration.

8. Conclusions and Perspectives

This study established a comprehensive mathematical framework for tuberculosis (TB) transmission by formulating and analyzing an SEITR compartmental model that integrates migration-related population influx alongside a general non-linear incidence mechanism of the form $f_1(S)(f_2(I) + f_3(T))$. By incorporating population fluxes directly into disease-related states and explicitly accounting for both partial and full treatment

efficacy through the parameter p , the model captures the interplay between demographic mobility, transmission saturation, and therapeutic outcomes. Rigorous qualitative analysis revealed fundamental threshold behavior governing disease persistence. For the system incorporating direct migration influx into disease compartments, we established the existence and global asymptotic stability of a unique endemic equilibrium under realistic biological conditions. In the reduced system without direct disease-related influx, the dynamics are completely governed by the basic reproduction number $\mathcal{R}_0$. Using custom Lyapunov functions and LaSalle's invariance principle, we proved that the disease-free equilibrium is globally asymptotically stable for $\mathcal{R}_0 < 1$, whereas a unique endemic equilibrium attracts all trajectories for $\mathcal{R}_0 > 1$. Center manifold theory further confirmed a classic forward transcritical bifurcation at $\mathcal{R}_0 = 1$, confirming that lowering $\mathcal{R}_0$ strictly below unity remains a sufficient condition for disease eradication. Beyond qualitative dynamics, our numerical investigations and global sensitivity analysis using Sobol indices identified key parameters driving $\mathcal{R}_0$, emphasizing the pivotal role of treatment success rates alongside transmission saturation effects. Specifically, while enhancing treatment efficacy p monotonically suppresses $\mathcal{R}_0$, numerical evaluations demonstrated that even perfect treatment performance ($p = 1$) may be insufficient to drive $\mathcal{R}_0 < 1$ when the critical threshold $p_c > 1$ lies outside the feasible biological domain. This highlights that clinical treatment alone cannot guarantee TB elimination in high-burden regimes without multi-pronged interventions. Furthermore, simulations demonstrated how saturated incidence mitigates peak epidemic size compared to standard bilinear dynamics and highlighted how migration influx can sustain endemicity even under high treatment performance. Future research could incorporate explicit spatial movement between geographical regions, heterogeneous migration patterns according to epidemiological status, direct recovery pathways from infectious compartments to the recovered class, and heterogeneous mortality rates. Fractional-order formulations could also be investigated to represent memory effects in disease progression and treatment. Moreover, calibration using appropriate epidemiological data and uncertainty quantification would help evaluate the robustness of model predictions and their applicability to specific populations. Overall, this work unifies global stability analysis, center manifold bifurcation theory, and global sensitivity mechanisms into a robust analytical platform. The findings underscore that effective long-term TB control strategies must simultaneously address clinical treatment success and population movement dynamics.

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