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Dynamical Behaviour of a Modified Tuberculosis Model with Impact of Public Health Education and Hospital Treatment

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Abstract: Tuberculosis (TB), caused by *Mycobacterium tuberculosis* is one of the treacherous infectious diseases of global concern. In this paper, we consider a deterministic model of TB infection with the public health education and hospital treatment impact. The effective reproductive number, R_{ph}, that measures the potential spread of TB is presented by employing the next generation matrix approach. We investigate local and global stability of the TB-free equilibrium point, endemic equilibrium point, and sensitivity analysis. The analyses of the proposed model show that the model undergoes the phenomenon of backward bifurcation when the effective reproduction number (R_{ph}) is less than one, where two stable equilibria, namely, the DFE and an EEP coexist. Further, we compute the sensitivity of the impact of each parameter on the effective reproductive number of the model by employing a normalized sensitivity index formula. Numerical simulation of the proposed model was conducted using Maple 2016 and MatLab R2020b software and compared with the theoretical results for illustration purposes. The investigation results can be useful in providing information to policy makers and public health authorities in mitigating the spread of TB infection by public health education and hospital treatment.

Keywords: tuberculosis infection; public health education; hospital treatment; effective reproduction number; stability analysis; bifurcation analysis; sensitivity analysis

MSC: 97A30; 92B05

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1. Introduction

Tuberculosis (TB) is one of the most hazardous infectious diseases that has become a significant widespread phenomenon, claiming more lives than any other contagious disease every day, according to [1]. Approximately 1/3 of the total population has a TB infection, resulting in millions of deaths and new cases annually (World Health Organization report). The report corroborates that TB is one of the top ten causes of mortality globally of both human and animal populations [2–6]. In 2020, for instance, 10 million individuals developed tuberculosis (TB), and more than 1.5 million died from it, including 214,000 HIV-positive persons (Human Immunodeficiency Virus) [7]. Typically, the signs may not be instantaneous when an individual contracts the disease. Thus, the individual remains asymptomatic for a long time or is latently infected for life [8]. Young adults may become infected by TB when they are most active [5]. Generally, TB-related deaths often happen in middle-income countries, for example, India, which leads the count, followed by Indonesia, China, the Philippines, Pakistan, Nigeria, Bangladesh, and South Africa. Such countries account for over 87% of the entire TB trouble in the world. As a result, it is vital to implement techniques and methods that make it simple to understand how this disease spreads and predict its progression.

TB is a communicable disease caused by *Mycobacterium tuberculosis* affecting mostly the lungs [9,10]. However, it can also attack different organs including the brain, kidney, spine,

central nervous system, or the lymphatic system [2,5,11–13]. It is important to note that the active lung TB disease typically begins with a cough, with sputum or blood on occasion, chest pains, fatigue, unexpected weight reduction, fever, and night sweats, which may last at least three or more weeks at a time. It has been reported by [14] that a pregnant woman who is infected may infect the foetus in some situations. Only individuals who have active TB can spread the disease. The latently infected individuals do not spread the bacteria [5,7]. Transmission starts with one individual, then onto the next, and relies upon the number of infected and expelled drops, the period of contaminated risk exposure, the virulence of the *Mycobacterium tuberculosis*, and the activity of environmental ventilation [7,10,15].

It was reported that behavioural change played a significant role in the transmission of infections [16–18]. Public health educational campaigns on TB disease plays a vital role in TB management and prevention [16,19], and can increase health literacy and awareness of TB among the population. The identification case rate and cure rate of TB patients can be improved by health education on TB disease, as it is an effective choice to minimize the spread of TB. As a result of the emergence of multi-resistance, treatment of TB is very challenging. Drug-resistant TB is a major public health concern across many developing countries, while treatment takes longer and needs more expensive drugs [5]. However, in many developing countries, the treatment of TB is not entirely free. Some TB patients cannot afford the full cost of treatment. As a result, some TB patients are treated at home to save money, as they can not afford to stay in the hospital. Some patients who were not cured choose to be discharged and continue their treatment at home [20].

Several mathematical models have been constructed to study the dynamical behaviour of TB infection. Abimbade et al. [21] formulated and analyzed an optimal control analysis of a TB transmission with incomplete treatment and exogenous re-infection, where they further divided the infected compartment into two groups, namely, uninformed and enlightened. Their findings revealed that both single controls and combinations of three controls have a positive influence on TB burden reduction. Recently, Ojo et al. [22] proposed a mathematical model for TB transmission with control. Their findings demonstrated that minimizing effective contact with infected people and increasing the rate of vaccinating susceptible persons with high vaccine efficacy will bring down the TB burden in a population. Xueyong et al. [16] formulated a TB model with healthy education and treatment. The qualitative analysis of their model exhibited the phenomenon of backward bifurcation, where the stable disease-free equilibrium co-exists with a stable endemic equilibrium, when the reproduction number is less than one. Huo and Zou [20] investigated a TB dynamics model with two types of treatment, namely, treatment at home and treatment in hospital. Their findings revealed that home treatment has a significant detrimental impact on tuberculosis spread. Okuonghae contributed significantly to TB epidemiology. They investigated the dynamics of TB and developed different models with comprehensive observations and results. Okuonghae and Egonmwan [23] presented a deterministic model that explores the impact of diagnosing and treating both latent and active TB infections on the disease's infection dynamics in a population. Castillo and Song [6] studied a comprehensive review of the literature on TB dynamics. They compiled many dynamical models of TB and established a theoretical framework. Recently, Mustapha et al. [24] introduced a TB model with hospitalization and reinfection. A mathematical analysis of the equilibrium points, the basic reproduction number, and sensitivity analysis was discussed. However, the authors of [24] did not include public health education in their model. This factor continues to be one of the most important aspects in people's lives. Public health services contributed to reducing the spread of TB infection.

Inspired by the above discussions, this paper seeks to fill a gap in the references cited, focusing on both public health education and hospital treatment. Infectious individuals in this model are divided into two classes: Infected individuals receiving treatment at home and infected individuals receiving treatment at the hospital. In this paper, we consider the impact of public health education and hospital treatment, comparing with the previous paper of [20]. The key differences are, in this paper, both the susceptible and

infected compartment are divided into two classes, which are susceptible and educated susceptible, and infected individuals at home and in the hospital. The other parts of the paper are assembled as follows: Section 2 is devoted to the model formulation along with its fundamental properties. Section 3 deals with analysis of the existence of equilibria and how it relates to the effective reproduction number. Section 4, bifurcation analysis was conducted theoretically and supported with some numerical experiments to give a visualization of the results. Sensitivity analysis is presented in Section 5. Numerical results and discussions are given in Section 6. Finally, conclusions and future work are given in Section 7.

2. Materials and Methods

In this section, a TB model with the impact of public health education and hospital treatment was considered by stratifying total population into epidemiological classes represented as $N_\iota(t)$, into six compartments, namely, susceptible individuals (S_ι), Educated Susceptible (P_ι), Exposed individuals (E_ι), Infectious individuals at home $(I_{\iota 1})$, Infectious individuals receiving treatment at hospital $(I_{\iota 2})$ and Recovered individuals (R_ι), so that:

$$N_\iota = S_\iota + P_\iota + E_\iota + I_{\iota 1} + I_{\iota 2} + R_\iota. \tag{1}$$

2.1. Clinical Assumptions of the TB Model with Public Health Education and Hospital Treatment

The assumptions used in this model are as follows:

- There is a constant recruitment rate to the susceptible population, and natural cause of death affects individuals in all compartments, with an extra TB-induced death rate in the infected class.
- We assume that, at any moment, the educated susceptible group may act as ignorantly and enter the class of susceptible at a constant rate ϕ, i.e., loss of temporary protection [16].
- We further assumed that some uncured TB patients wanted to be discharged and continue treatment at home at a rate η_2, [20].
- Both infected individuals at home and in the hospital experience the infections effect at the rate λ.
- The infected compartment is divided into two groups, namely, infected individuals who received treatment at home $I_{\iota 1}$, and infected individuals receiving treatment in hospital $I_{\iota 2}$.
- The recovered individual may be again infected by an infectious individual [25].

In this model, it is assumed that the number of susceptible population is generated via recruitment of individuals into the population at a rate Λ. Susceptible individuals are educated at the rate ψ, and then transferred into the educated class. We further assume that education programs provide "temporary protection" at the per-capita rate ϕ [19,26]. We assume that both infected individuals at home and in the hospital experience the infections effect at the rate λ, where

$$\lambda = \frac{\beta(I_{\iota 1} + zI_{\iota 2})}{N_\iota}. \tag{2}$$

In (2), the parameter β, is the transmission rate, while $0 \leq z \leq 1$ is the modification parameter associated with reduced infectiousness of individuals in the hospital (in the $I_{\iota 1}$ compartment) compared to individuals at home. Individuals who have received public health education have a high level of awareness. As a result, educated people are a low-risk population. Education reduces the risk of infection by a factor of $\nu(0 \leq \nu \leq 1)$. The scenario $\nu = 0$ indicates that the education is totally effective to prevent the infection, whereas $\nu = 1$ models the case where the education program is totally ineffective [19,26]. The populations of susceptible and educated susceptible individuals are further decreased

as a result of natural death rate μ. Therefore, the rates of change of the populations of susceptible and educated susceptible individuals are given, respectively, by

$$\frac{dS_\iota}{dt} = \Lambda + \phi P_\iota - \lambda S_\iota - \psi S_\iota - \mu S_\iota,$$

$$\frac{dP_\iota}{dt} = \psi S_\iota - \nu\lambda P_\iota - \phi P_\iota - \mu P_\iota.$$

The population of exposed individuals is generated via the infection of susceptible (S_ι), educated susceptible (P_ι) and recovered (R_ι), at the rate λ, $\nu\lambda$ and $\sigma\lambda$, and decreased as a result of disease symptoms at a rate κ_1, κ_2, and natural death at the rate μ, and $\sigma(0 \leq \sigma \leq 1)$ is taken as the reinfection rate so that

$$\frac{dE_\iota}{dt} = \lambda S_\iota + \nu\lambda P_\iota + \sigma\lambda R_\iota - (\kappa_1 + \kappa_2 + \mu)E_\iota.$$

Similarly, the population of infected individuals at home is generated as result of disease symptoms at a rate κ_1, the rate of TB patients who are not cured from the hospital at a rate η_2 and diminished due to progression to hospital at a rate η_1, recovery by TB patients at home at a rate γ_1, natural death cause μ, and TB-induced death at the rate δ_1, respectively. Thus,

$$\frac{dI_{\iota 1}}{dt} = \kappa_1 E_\iota + \eta_2 I_{\iota 2} - (\eta_1 + \gamma_1 + \mu + \delta_1)I_{\iota 1}.$$

In addition, the population of infected individuals receiving treatment at the hospital is increased as a result of the disease symptoms at a rate κ_2, and hospital admittance of infected individuals receiving treatment at home at a rate η_1, and diminished due to their returning back home to $I_{\iota 1}$ at a rate η_2, natural death cause μ, recovery by TB patients at hospital at a rate γ_2, and TB induced death at the rate δ_2. Therefore,

$$\frac{dI_{\iota_2}}{dt} = \kappa_1 E_\iota + \eta_1 I_{\iota 1} - (\eta_1 + \mu + \gamma_2 + \delta_2)I_{\iota 2}.$$

Finally, the population of recovered individuals is generated via recovery by both infected individuals treated at home γ_1 and at hospital γ_2 at a rate γ_1, γ_2, respectively, and diminished as a result of natural death at a rate μ and progression to the exposed class at a rate $\sigma\lambda$.

$$\frac{dR_\iota}{dt} = \gamma_1 I_{\iota 1} + \gamma_2 I_{\iota 2} - \sigma\lambda R_\iota - \mu R_\iota.$$

Therefore, based on the above description and assumptions, the model of TB with public health education and hospital treatment lead to following a system of non-linear differential equations; the schematic diagram Figure 1 below, and the parameters indicated in the diagram are explained in Table 1.

$$\begin{aligned}
\tfrac{dS_\iota}{dt} &= \Lambda + \phi P_\iota - (\lambda + \psi + \mu)S_\iota,\\
\tfrac{dP_\iota}{dt} &= \psi S_\iota - (\nu\lambda + \phi + \mu)P_\iota,\\
\tfrac{dE_\iota}{dt} &= \lambda S_\iota + \nu\lambda P_\iota + \sigma\lambda R_\iota - (\kappa_1 + \kappa_2 + \mu)E_\iota,\\
\tfrac{dI_{\iota 1}}{dt} &= \kappa_1 E_\iota + \eta_2 I_{\iota 2} - (\eta_1 + \gamma_1 + \mu + \delta_1)I_{\iota 1},\\
\tfrac{dI_{\iota 2}}{dt} &= \kappa_2 E_\iota + \eta_1 I_{\iota 1} - (\eta_2 + \gamma_2 + \mu + \delta_2)I_{\iota 2},\\
\tfrac{dR_\iota}{dt} &= \gamma_1 I_{\iota 1} + \gamma_2 I_{\iota 2} - (\sigma\lambda + \mu)R_\iota,
\end{aligned} \tag{3}$$

where

$$\lambda = \frac{\beta(I_{t1} + zI_{t2})}{N_t}, \tag{4}$$

with the initial conditions given by Equation (5)

$$S_t(0) = S_{t0} > 0,\ P_t(0) = P_{t0} > 0,\ E_t(0) = E_{t0} > 0,\ I_{t1}(0) = I_{t10} > 0,\ I_{t2}(0) = I_{t20} > 0 \tag{5}$$

satisfies the equation

$$\frac{dN_t}{dt} = \Lambda - \mu N_t - \delta_1 I_{t1} - \delta_2 I_{t2}. \tag{6}$$

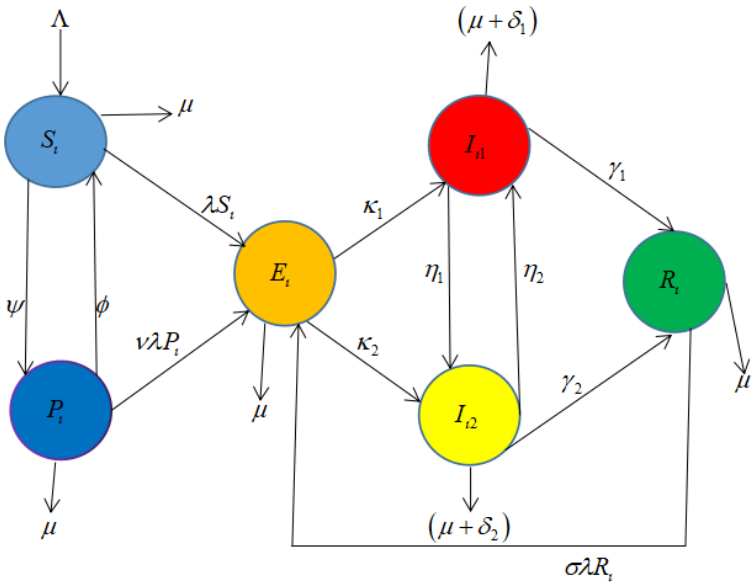

Figure 1. The flow diagram of TB model with public health education and hospital treatment.

Table 1. Interpretation of the Variables and Parameters of the TB Model with Public Education and hospital Treatment (23).

State Variables	Explanation
S_t	The number of individuals who are susceptible
P_t	The number of individuals who are educated susceptible
E_t	The number of individuals who are exposed to TB
I_{t1}	The number of infected individuals at home
I_{t2}	The number of infected individuals at hospital
R_t	The number of individuals who have recovered
N_t	Human population size
Parameters	
Λ	Inflow of recruitment into susceptible class
μ	Per capita natural death rate of humans
ψ	Information dissemination (awareness rate)
ϕ	Rate at which the educated susceptible become susceptible
ν	Reduction of infection rate as a result of awareness
β	Transmission rate
z	Reduction of infection rate as a result of individuals in the hospital
κ_1	Progression rate from E_t to I_{t1}
κ_2	Progression rate from E_t to I_{t2}
γ_1	recovery rate for I_{t1}
γ_2	recovery rate for I_{t2}
σ	Mod. parameter for re-infection among the recovered individuals
η_1	Progression rate from I_{t1} to I_{t2}
η_2	Progression rate from I_{t2} to I_{t1}
δ_1	TB induced mortality rate for I_{t1}
δ_2	TB induced mortality rate for I_{t2}

2.2. Basic Properties of the TB Model with Public Health Education and Hospital Treatment

The basic properties of the TB model (23) are explained in this subsection. In an invariant region, the model is proved to be positive and bounded. When studying the dynamical behaviour of an epidemiologically model, this analysis is crucial because it demonstrates whether the model is epidemiologically relevant and mathematically well-posed, that is, whether the model and its predictions are certain [27].

Positivity of Solutions

To established that the TB infection model system (23) is epidemiologically realistic, all the stated variables are positive all the time.

Theorem 1. *Let initial data be* $\{(S_\iota, P_\iota, E_\iota, I_{\iota 1}, I_{\iota 2}, R_\iota) \geq 0\} \in \Phi$. *Thus, the solution of* $\{S_\iota(t), P_\iota(t), E_\iota(t), I_{\iota 1}(t), I_{\iota 2}(t), R_\iota(t)\}$ *of the model system* (23) *is non-negative for all* $t > 0$.

Proof of Theorem 1. The method described in [28,29], is applied. We use the first equation to consider the non-linear system of (23), which clearly shows that

$$\frac{dS_\iota}{dt} + (\lambda(t) + \psi + \mu)S_\iota > 0,$$

utilizing an integrating factor gives

$$\frac{d}{dt}\left[S_\iota exp\left(\int_0^t (\lambda(\epsilon) + \psi + \mu\right)d\epsilon\right] > 0. \tag{7}$$

Using the initial conditions (5) and integrating (7) results in

$$S_\iota(t) > S_{\iota 0} exp\left[-\left(\int_0^t (\lambda(\epsilon) + \psi + \mu\right)d\epsilon\right] > 0, \forall t > 0.$$

Similarly, it can be shown that $P_\iota(t) > 0$, $E_\iota(t) > 0$, $I_{\iota 1}(t) > 0$, $I_{\iota 2}(t) > 0$, $R_\iota(t) > 0$, $\forall t > 0$. □

2.3. Invariant Region

Theorem 2. *The solution of the TB model system* (23) *is enclosed in the region* Φ *subset* $\in R_+^6$, *given by*

$$\Phi = \left\{(S_\iota, P_\iota, E_\iota, I_{\iota 1}, I_{\iota 2}, R_\iota) \in R_+^6, N_\iota \leq \frac{\Lambda}{\mu}\right\},$$

for the initial conditions (5) *in* Φ.

Proof of Theorem 2. The change in the total population is given by

$$\frac{dN_\iota}{dt} = \Lambda - \mu N_\iota - I_{\iota 1}\delta_1 - I_{\iota 2}\delta_2, \tag{8}$$

In the absence of TB infection, there is no death from TB transmission, (that is, $\delta_1 = \delta_2 = 0$) [30], hence the rate of change of the total population size in Equation (8) is given as

$$\frac{dN_\iota}{dt} \leq \Lambda - \mu N_\iota, \tag{9}$$

The solution of Equation (9) is solved by using the same approach presented in [31,32], and is given by

$$N_\iota(t) \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N_{\iota 0}\right)e^{-\mu t}, \tag{10}$$

where $N_{\iota 0} = N_\iota(0)$.

Using [33] we note that if $N_{\iota 0} < \frac{\Lambda}{\mu}$, $N_\iota \to \frac{\Lambda}{\mu}$ asymptotically as $t \to \infty$ in Equation (10) the total population size $N_\iota \to \frac{\Lambda}{\mu}$, which means that $0 \leq N_\iota \leq \frac{\Lambda}{\mu}$. Therefore, all the feasible solutions in the model converge in the region Φ [34]. □

3. Existence of Equilibrium

3.1. Tuberculosis Free Equilibrium (TFE)

The TFE state denoted as T^0, is generally described as a state in which no disease exists in a given population. The infected population can be defined as a disease type. Using the first fourth equation of system model (23) with $E_\iota = I_{\iota 1} = I_{\iota 2} = R_\iota = 0$ into consideration, we arrive at:

$$T^0 = (S_\iota^0, P_\iota^0, E_\iota^0, I_{\iota 1}^0, I_{\iota 2}^0, R_\iota^0) = \left(\frac{\Lambda}{\mu}\frac{\mu+\phi}{\mu+\psi+\phi}, \frac{\Lambda}{\mu}\frac{\psi}{\mu+\psi+\phi}, 0, 0, 0, 0\right). \tag{11}$$

3.2. Calculation of Effective Reproduction Number (R_{ph}) For the System Model (23)

This section studies the effective reproduction number, which is a threshold parameter that controls the spread of a disease. To obtain the effective reproduction number, we apply the next generation approach described in [35]. The associated incidence matrix (F) and the transition matrix (V) of system model (23), are respectively obtained as:

$$F = \begin{pmatrix} 0 & \frac{\beta(S_\iota^0+\nu P_\iota^0)}{N_\iota^0} & \frac{\beta z(S_\iota^0+\nu P_\iota^0)}{N_\iota^0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} A_1 & 0 & 0 \\ -\kappa_1 & A_2 & -\eta_2 \\ -\kappa_2 & -\eta_1 & A_3 \end{pmatrix}, \tag{12}$$

where

$$A_1 = \kappa_1 + \kappa_2 + \mu,\ A_2 = \eta_1 + \mu + \gamma_1 + \delta_1,\ A_3 = \eta_2 + \mu + \gamma_2 + \delta_2. \tag{13}$$

Thus, the effective reproductive number of system model (23) is calculated from the spectral radius $\rho(FV^{-1})$ as :

$$R_{ph} = \frac{\beta(\phi+\mu+\nu\psi)(\kappa_1 A_3 + \kappa_2\eta_2) + z(\kappa_2 A_2 + \eta_1\kappa_1)}{A_1(A_2A_3 - \eta_1\eta_2)(\psi+\phi+\mu)}. \tag{14}$$

After substituting the values of A_1, A_2 and A_3, Equation (14) becomes

$$R_{ph} = \frac{\beta\,(\nu\,\psi+\mu+\phi)((\eta_2+\mu+\gamma_2+\delta_2)\kappa_1 + \kappa_2\eta_2) + z((\eta_1+\mu+\gamma_1+\delta_1)\kappa_2 + \kappa_1\eta_1)}{C_0 + C_1 + \mu((\eta_1+\mu+\gamma_1+\delta_1)(\eta_2+\mu+\gamma_2+\delta_2) - \eta_1\eta_2)(\psi+\mu+\phi)}. \tag{15}$$

where

$$C_0 = \kappa_1((\eta_1+\mu+\gamma_1+\delta_1)(\eta_2+\mu+\gamma_2+\delta_2) - \eta_1\eta_2)(\psi+\mu+\phi),$$
$$C_1 = \kappa_2((\eta_1+\mu+\gamma_1+\delta_1)(\eta_2+\mu+\gamma_2+\delta_2) - \eta_1\eta_2)(\psi+\mu+\phi).$$

The term effective reproduction number, represented by R_{ph}, is defined as the expected number of secondary cases generated by a single infected individual during the period of infectiousness in a population of susceptible individuals where public health education and hospital treatment are incorporated [36].

3.3. Local Stability of TB Free Equilibrium

To prove the local stability of the TFE, the Jacobian of the proposed model system (23) is used. After that, the Jacobian is used to derive the characteristic equation, from which the eigenvalue result is obtained.

Theorem 3. *The TFE of the proposed model system (23) is locally asymptotically stable when* $R_{ph} < 1$.

Proof of Theorem 3. To prove the system's local stability, the Jacobian of the proposed model system (23) is investigated at TFE, which is then given by:

$$J(T^0) = \begin{pmatrix} -(\mu+\psi) & \phi & 0 & -\frac{\beta S_\iota^0}{N_\iota^0} & -\frac{\beta z S_\iota^0}{N_\iota^0} & 0 \\ \psi & -(\phi+\mu) & 0 & -\frac{\nu\beta P_\iota^0}{N_\iota^0} & -\frac{\nu\beta z P_\iota^0}{N_\iota^0} & 0 \\ 0 & 0 & -A_1 & \frac{\beta(S_0+\nu\beta P_0)}{N_0} & \frac{\beta z(S_0+\nu P_\iota^0)}{N_\iota^0} & 0 \\ 0 & 0 & \kappa_1 & -A_2 & \eta_2 & 0 \\ 0 & 0 & \kappa_2 & \eta_1 & -A_3 & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & -\mu \end{pmatrix}. \quad (16)$$

The characteristic equation of (16) is given by:

$$(\lambda+\mu)(\lambda+\psi+\mu)(\lambda+\phi+\mu)(\lambda+A_1)(\lambda+A_2)(\lambda+A_3) = 0. \quad (17)$$

It can be seen from (17) that:

$$\begin{aligned} \lambda_1 &= -\mu < 0, \\ \lambda_2 &= -(\psi+\mu) < 0, \\ \lambda_3 &= -(\phi+\mu) < 0, \\ \lambda_4 &= -A_1 < 0, \\ \lambda_5 &= -A_2 < 0, \\ \lambda_6 &= -A_3 < 0. \end{aligned} \quad (18)$$

The result reveals that all the eigenvalues $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ are negative given that all the parameters values are greater than zero. As a result, according to the principle of linearized stability [37], the TFE is asymptotically stable. □

3.4. Global Stability of the TB Free Equilibrium

This section investigates the global stability of the TFE point T^0 of the model system (23). To investigate the global stability of the system model (23), we employ the techniques implemented by [38].

$$\begin{aligned} \frac{dX}{dt} &= F(X_\iota, Z_\iota), \\ \frac{dZ}{dt} &= G(X_\iota, Z_\iota); G(X_\iota, 0) = 0. \end{aligned} \quad (19)$$

Here, the components $X_\iota = (S_\iota, P_\iota, R_\iota)$ and $Z_\iota = (E_\iota, I_{\iota 1}, I_{\iota 2})$, where $X_\iota \in \mathbb{R}^3$ denotes the uninfected population and $Z_\iota \in \mathbb{R}^3$ denotes the infected population.

The TB free equilibrium is defined by $T^0 = (X_\iota^*, 0)$.

The fixed point $T^0 = (X_\iota^*, 0)$ is a globally asymptotically stable equilibrium for the system model (23) provided that R_{ph}, which is locally asymptotically stable, and the following two conditions must be satisfied:

$$(H_1)\text{: for } \frac{dX_\iota}{dt} = F(X_\iota, 0), X^*$$

is globally asymptotically stable (GAS),

$$(H_2)\text{: } G(X_\iota, Z_\iota) = AZ_\iota - \hat{G}(X_\iota, Z_\iota), \hat{G}(X_\iota, Z_\iota) \geq 0 \text{ for } (X_\iota, Z_\iota) \in \Phi,$$

If the system model (23) meets the given two criteria, then the following theorem holds.

Theorem 4. *The TB free equilibrium point* $T^0 = (X^*, 0)$ *of the system model* (23) *is globally asymptotically stable provided* $R_{ph} < 1$ *and the conditions* (H_1) *and* (H_2) *are satisfied.*

Proof of Theorem 4. From system model (23) we can get $F(X_\iota, Z_\iota)$ and $G(X_\iota, Z_\iota)$:

$$F(X_\iota, Z_\iota) = \begin{pmatrix} \Lambda + \phi P_\iota - (\lambda + \psi + \mu)S_\iota \\ \psi S_\iota - (\nu\lambda + \phi + \mu)P_\iota \\ \gamma_1 I_{\iota 1} + \gamma_2 I_{\iota 2} - (\sigma\lambda + \mu)R_\iota \end{pmatrix}, \tag{20}$$

$$G(X_\iota, Z_\iota) = \begin{pmatrix} \lambda S_\iota + \nu\lambda P_\iota + \sigma\lambda R_\iota - (\kappa_1 + \kappa_2 + \mu)E_\iota \\ \kappa_1 E_\iota + \eta_2 I_{\iota 2} - (\eta_1 + \gamma_1 + \mu + \delta_1)I_{\iota 1} \\ \kappa_2 E_\iota + \eta_1 I_{\iota 1} - (\eta_2 + \gamma_2 + \mu + \delta_2)I_{\iota 2} \end{pmatrix}. \tag{21}$$

At T^0, Equation (20)

$$\frac{dX}{dt} = F(X_\iota, 0) = \begin{pmatrix} \Lambda + \phi P_\iota - (\psi + \mu)S_\iota \\ \psi S_\iota - (\phi + \mu)P_\iota \\ -\mu R_\iota \end{pmatrix}. \tag{22}$$

From the system (22) above, we see that $X_\iota^* = \left(\frac{\Lambda(\mu+\phi)}{\mu(\mu+\psi+\phi)}, \frac{\Lambda\psi}{\mu(\mu+\psi+\phi)}, 0\right)$ is globally asymptotical stable. This can be verified from the solutions, namely,

$$\begin{aligned} S_\iota(t) &= \tfrac{\Lambda(\mu+\phi)}{\mu(\mu+\psi+\phi)} + \left(S_\iota(0) - \tfrac{\Lambda(\mu+\phi)}{\mu(\mu+\psi+\phi)}\right)exp^{-(\psi+\mu+\phi)t}. \\ P_\iota(t) &= \tfrac{\Lambda\psi}{\mu(\mu+\psi+\phi)} + \left(P_\iota(0) - \tfrac{\Lambda\psi}{\mu(\mu+\psi+\phi)}\right)exp^{-(\psi+\mu+\phi)t}. \\ R_\iota(t) &= R_\iota(0)exp^{-\mu t}. \end{aligned} \tag{23}$$

As $t \to \infty$, the solution $S_\iota(\infty), P_\iota(\infty), R_\iota(\infty) \to \frac{\Lambda(\mu+\phi)}{\mu(\mu+\psi+\phi)}, \frac{\Lambda\psi}{\mu(\mu+\psi+\phi)}, 0$, which implies the global convergence of (22) in Φ, and this satisfies condition H_1.

Next, applying the second condition of the theorem H_2.

From H_2, we have $G(X_\iota, Z_\iota) = AZ_\iota - \hat{G}(X_\iota, Z_\iota), \hat{G}(X_\iota, Z_\iota) \geq 0$ for $(X_\iota, Z_\iota) \in \Phi$.

Therefore, $\hat{G}(X_\iota, Z_\iota) = AZ_\iota - G(X_\iota, Z_\iota)$. where A is an $n \times n$ matrix, Z_ι is a column vector and $G(X_\iota, Z_\iota)$ is a column vector formed from the infectious compartments. We already know that

$$G(X_\iota, Z_\iota) = \begin{pmatrix} G_1(X_\iota, Z_\iota) \\ G_2(X_\iota, Z_\iota) \\ G_3(X_\iota, Z_\iota) \end{pmatrix} = \begin{pmatrix} \lambda S_\iota + \nu\lambda P_\iota + \sigma\lambda R_\iota - (\kappa_1 + \kappa_2 + \mu)E_\iota \\ \kappa_1 E_\iota + \eta_2 I_{\iota 2} - (\eta_1 + \gamma_1 + \mu + \delta_1)I_{\iota 1} \\ \kappa_2 E_\iota + \eta_1 I_{\iota 1} - (\eta_2 + \gamma_2 + \mu + \delta_2)I_{\iota 2} \end{pmatrix}.$$

Now let compute A

$$A = \begin{pmatrix} -(\kappa_1 + \kappa_2 + \mu) & \frac{\beta(\phi+\mu+\nu\psi)}{\phi+\mu+\psi} & \frac{\beta z(\phi+\mu+\nu\psi)}{\phi+\mu+\psi} \\ \kappa_1 & -(\eta_1 + \gamma_1 + \mu + \delta_1) & \eta_2 \\ \kappa_2 & \eta_1 & -(\eta_2 + \gamma_2 + \mu + \delta_2) \end{pmatrix}.$$

The matrix A is a metzler matrix because all its off-diagonal elements are non-negative.

$$A = \begin{pmatrix} -(\kappa_1 + \kappa_2 + \mu) & \frac{\beta(\phi+\mu+\nu\psi)}{\phi+\mu+\psi} & \frac{\beta z(\phi+\mu+\nu\psi)}{\phi+\mu+\psi} \\ \kappa_1 & -(\eta_1 + \gamma_1 + \mu + \delta_1) & \eta_2 \\ \kappa_2 & \eta_1 & -(\eta_2 + \gamma_2 + \mu + \delta_2) \end{pmatrix} \begin{pmatrix} E_\iota \\ I_{\iota 1} \\ I_{\iota 2} \end{pmatrix}.$$

$$AZ_\iota = \begin{pmatrix} \frac{\beta(\phi+\mu+\nu\psi)}{(\phi+\mu+\psi)}(I_{\iota 1} + zI_{\iota 2}) - (\kappa_1 + \kappa_2 + \mu)E_\iota \\ \kappa_1 E_\iota - (\eta_1 + \gamma_1 + \mu + \delta_1)I_{\iota 1} + \eta_2 I_{\iota 2} \\ \kappa_2 E_\iota \eta_1 I_{\iota 1} - (\eta_2 + \gamma_2 + \mu + \delta_2)I_{\iota 2} \end{pmatrix}.$$

Thus, using $\widehat{G}(X_\iota, Z_\iota) = AZ_\iota - G(X_\iota, Z_\iota)$, one obtains the following

$$\widehat{G}(X_\iota, Z_\iota) = \begin{pmatrix} \beta(I_{\iota 1} + zI_{\iota 2})\frac{(\phi+\mu+\nu\psi)}{(\phi+\mu+\psi)} - \frac{S_\iota + \nu P_\iota + \sigma R_\iota}{N_\iota} \\ 0 \\ 0 \end{pmatrix}.$$

Since $\widehat{G}(X_\iota, Z_\iota) \ngeq 0$, H_2 is not satisfied. This suggests that backward bifurcation may occur at T^0 when $R_p h < 1$. □

3.5. Endemic Equilibrium State (T^*)

The stability of (T^0) drives the disease's short-term outbreaks. The stability at the endemic equilibrium points (T^*) characterises its dynamics over a longer duration. We discovered that long-term behaviour has crucial epidemiological implications, such as whether an outbreak of a disease will lead to an endemic scenario or whether the infection will die out. We shall conduct an endemic analysis in this section. T^* can now be determined by equating all of the model system in (23) to zero:

Theorem 5. *The endemic equilibrium state of the model system Equation* (23) *exists if the effective reproduction number* $R_{ph} > 1$.

The proof of Theorem 5 is presented in Appendix A.

Theorem 6. *The TB model with public health education and hospital treatment* (23) *has:*

1. *One or more endemic equilibria when* $R_{ph} < 1$.
2. *A unique endemic equilibrium when* $R_{ph} > 1$.
3. *No endemic equilibrium otherwise.*

4. Local Stability of Endemic Equilibrium

The local stability of endemic equilibrium (T^*) of the model system can be investigated by employing the concept of center manifold presented in [6].

Theorem 7. *The TB model with public health education and hospital treatment* (23) *undergoes backward bifurcation with bistability of* T^* *at* $R_{ph} < 1$ *and* $R_{ph} > 1$.

The proof of Theorem 7 is presented in Appendix B.

Figure 2 demonstrates a backward bifurcation diagram of force of infection λ against the effective reproduction number R_{ph} of the system model (23). We can see from the figure that as R_{ph} increases to one, the disease also increases and this occurs when $R_{ph} < 1$. At $R_{ph} = 1$, DFE and EEP coexists; this means that the disease cannot be eradicated from the population as a result of a high-level endemic. We can also discover that when $R_{ph} > 1$, the disease persists.

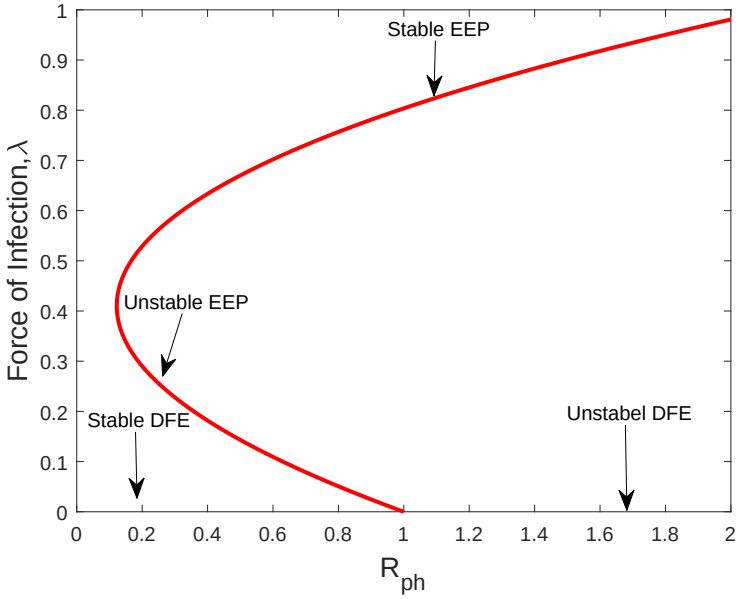

Figure 2. The bifurcation diagram of force of infection against R_{ph} which illustrates a backward bifurcation for the system model (23).

5. Sensitivity Analysis

It is crucial to investigate how sensitive the TB model (23) is to changes in each of its parameters in order to identify control strategies that will assist in the lowering of the infection trajectory. In other words, conducting sensitivity analysis will assist in identifying what should be done or ignored in order to stop the spread of the TB transmission [29,39,40]. When a parameter changes, we can use sensitivity indices to calculate the relative change in a state variable. We employ the normalised forward sensitivity index of a variable to a parameter method provided in [41] in order to perform sensitivity analysis. The ratio of relative change in the variable to relative change in the parameter is known as the sensitivity index. The sensitivity index may also be defined using partial derivatives when the variable is a differentiable function of the parameter. Therefore, as stated in [41], the normalised forward sensitivity index (SI) of a threshold, R_{ph}, that is differentiable with respect to a parameter, η, is defined as:

$$\Upsilon_{\eta}^{R_{ph}} = \frac{\partial R_{ph}}{\partial \eta} \times \frac{\eta}{R_{ph}}. \tag{24}$$

The sensitivity of R_{ph} to each of the 14 parameters presented in Table 2 is calculated using the effective reproduction number given below

$$R_{ph} = \frac{\beta(\phi + \mu + \nu\psi)(\kappa_1 A_3 + \kappa_2\eta_2) + z(\kappa_2 A_2 + \eta_1\kappa_1)}{A_1(A_2A_3 - \eta_1\eta_2)(\psi + \phi + \mu)}, \tag{25}$$

where A_1, A_2, and A_3 are given in Equation (14).

Sensitivity index implied the parameters values obtained in Table 2.

From Table 2, we can see that the parameters have both positive and negative effects on R_{ph}. Positive SI values, such as $\beta, z, \mu, \nu, \phi, \gamma_2, \eta_2$ and κ_1, as shown in Table 2 reveal that an increase in these parameters values increases R_{ph}, which brings about the infection attacking the population. While the parameters $\kappa_2, \mu, \psi, \gamma_1, \eta_1, \delta_1$, and δ_2, have negative SI, that is, an increase in these parameters values decrease R_{ph}, and as a result, the infection gradually fades from the population.

Table 2. Sensitivity Indices of the Model Parameters in Relation to R_{ph}.

Parameters	Description	Sensitivity Index
μ	Per capita natural death rate of humans	−0.6970
ψ	Information dissemination (awareness rate)	−0.2554
ϕ	The rate at which susceptible individuals lose awareness	+0.2454
ν	Reduction in risk of infection due to awareness	+0.1103
β	Transmission rate for contact with I_1	+1.0000
z	Reduction of infection rate as a result of I_2	+0.4476
κ_1	Progression rate from E_i to I_1	+0.4228
κ_2	Progression rate from E_i to I_2	−0.2147
γ_1	Treatment rate for I_{i1}	−0.2573
γ_2	Treatment rate for I_{i2}	+0.5570
η_1	Progression rate from I_{i1} to I_{i2}	−0.6832
η_2	Progression rate from I_{i2} to I_{i1}	+0.1845
δ_1	TB induced mortality rate for I_{i1}	−0.2058
δ_2	TB induced mortality rate for I_{i2}	−0.3714

Figure 3 demonstrates the relationship between the effective reproduction number R_{ph}, ψ, η_1, κ_1, and κ_2. Figure 3a, illustrates a linear relationship between R_{ph} and the awareness rate ψ. We observed from Figure 3a, that R_{ph} decreases with increasing the awareness rate ψ of susceptible individuals. This means that susceptible individuals who are aware of the TB disease and understand how it is transmitted have a great positive impact on the spread of TB infection.

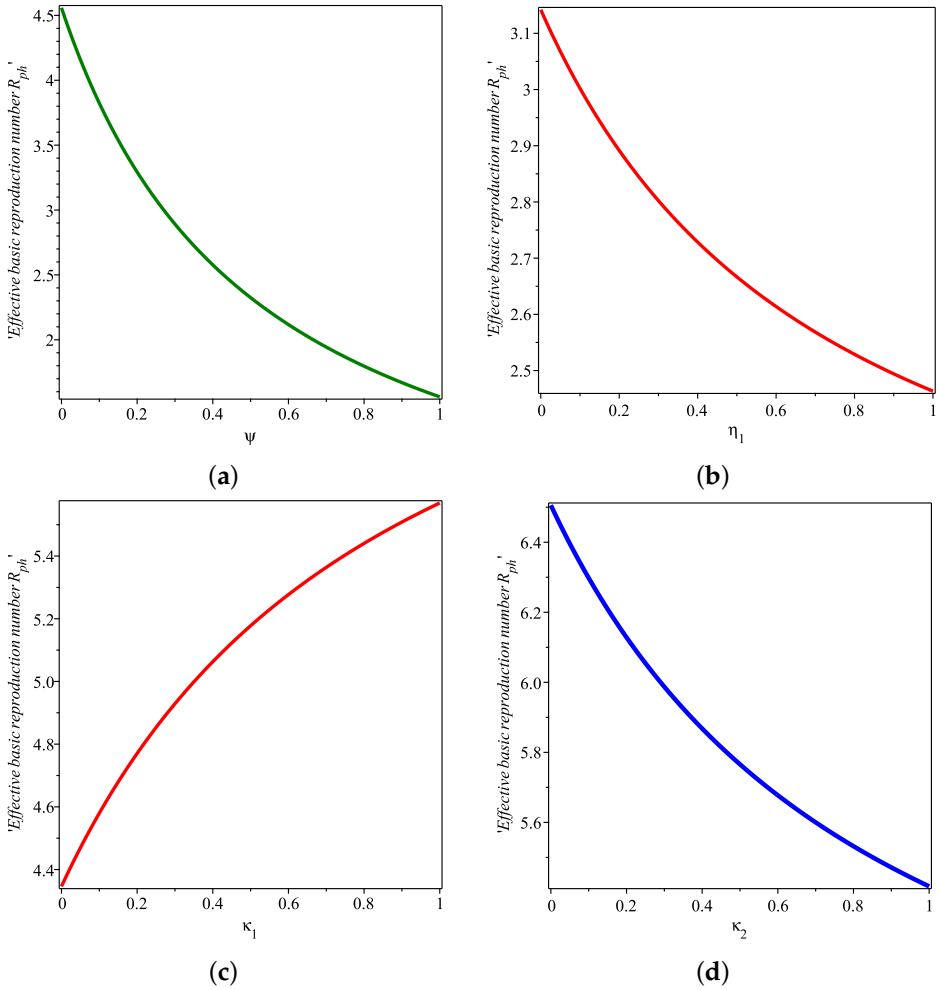

Figure 3. The relationship between ψ (**a**), η_1 (**b**), κ_1 (**c**), and κ_2 (**d**) and the effective reproduction number R_{ph}.

Figure 3b demonstrates a linear relationship between R_{ph} and the rate at which infected individuals progress from $I_{\iota 1}$ class to $I_{\iota 2}$ class. This indicates that R_{ph} decreases with the increasing the rate at which infected individuals $I_{\iota 1}$ at home undergo treatment at the hospital. This means that infected individuals treated at the hospital have a great positive impact on the spread of TB infection too.

Figure 3c,d depicts a relationship between R_{ph}, κ_1 and κ_2. It is clearly seen from the Figure 3c,d that R_{ph} increases with increasing κ_1, while R_{ph} decreases with increasing κ_2. Biologically, this shows that infected individuals receiving treatment at home have a great negative impact on the dynamics of TB disease.

6. Numerical Results and Discussions

The numerical simulation of the model system (23), is performed with the values of the parameters described in Table 3. We used ode45 solver in Matlab, which depends on the Runge-Kutta technique to stimulate model system (23) with the parameter values presented in Table 3, as well as the following initial conditions:

$$S_\iota(0) = 0.6,\ P_\iota(0) = 0.2,\ E_\iota(0) = 0.1,\ I_{\iota 1}(0) = 0.1,\ I_{\iota 2}(0) = 0.1,\ R_\iota(0) = 0.$$

Figure 4 is a graphical representation of system model (23) showing the differences between susceptible and educated susceptible individuals. We can see from Figure 4 that, as time increases, the number of educated susceptible individuals' health continues to increase until it reaches the carrying capacity, whereas, the number of susceptible individuals continues to decrease with time as more and more of them become infected.

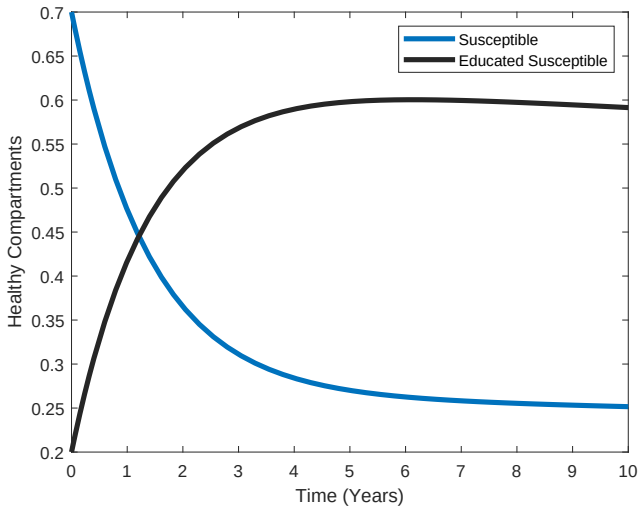

Figure 4. Simulations of system (23) showing the behaviour of susceptible and educated susceptible. Parameters used are $\Lambda = 450{,}862$, $\beta = 0.86, \nu = 0.25, \phi = 0.2, \sigma = 0.65, z = 0.11, \mu = 0.02041$, $\psi = 0.5$, $\eta_1 = 0.02$, $\eta_2 = 0.02$, $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\kappa_1 = 0.03$, $\kappa_2 = 0.3$, $\delta_1 = 0.2$, $\delta_2 = 0.12$.

Figure 5 depicts the impact of the various values of awareness rate (ψ), on the dynamics of S_ι, E_ι, $I_{\iota 1}$ and $I_{\iota 2}$. In general, this figure reflects that when ψ, increases, the number of E_ι, $I_{\iota 1}$ *and* $I_{\iota 2}$ decreases rapidly. This demonstrates that public health education of susceptible individuals has the effect of limiting TB disease spread. As a result, public policymakers must focus on increasing the value of awareness rate ψ in order to prevent and control TB infection in a population.

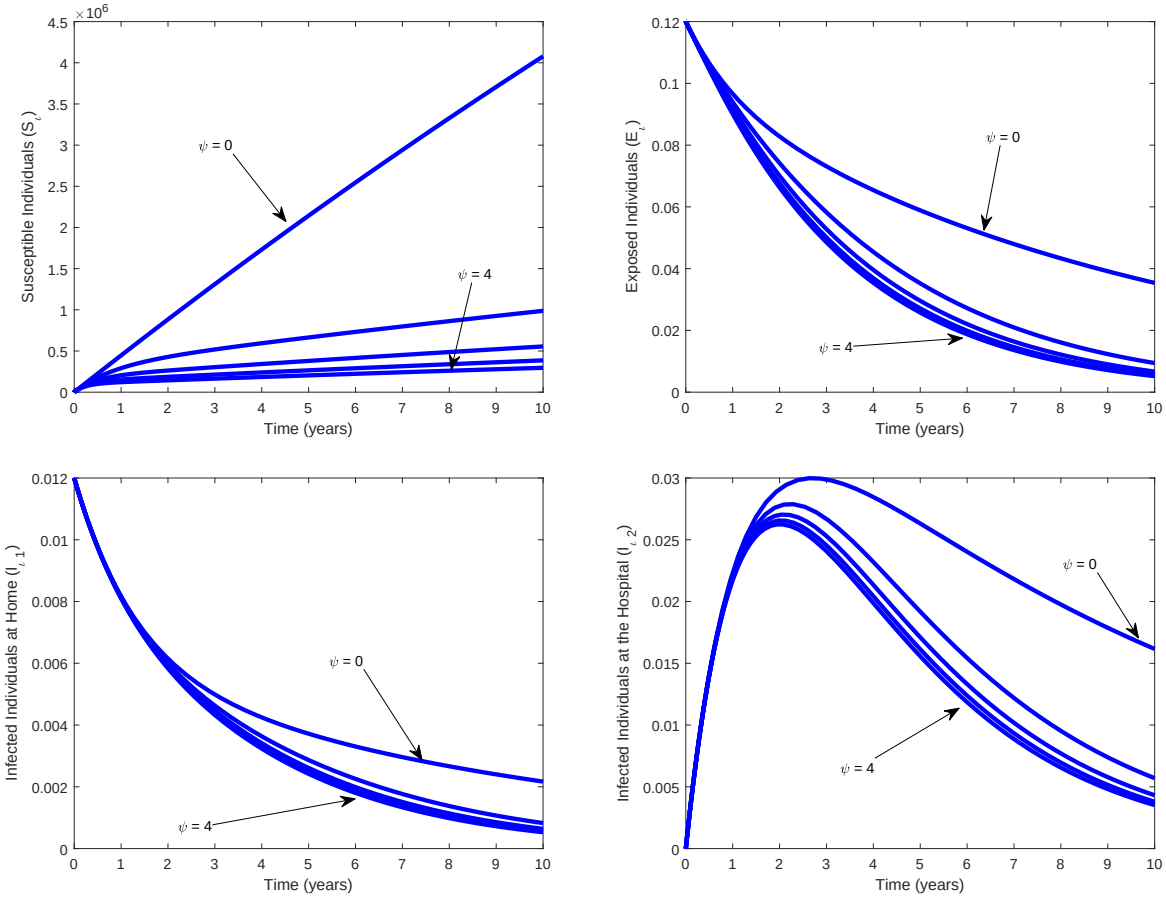

Figure 5. Impact of awareness rate (ψ) on $S_\iota, E_\iota, I_{\iota 1}$, and $I_{\iota 2}$, varying ψ. Other parameters are given as $\Lambda = 450{,}862$, $\beta = 0.86, \nu = 0.25, \phi = 0.2, \sigma = 0.65, z = 0.11, \mu = 0.02041$, $\psi = 0.5$, $\eta_1 = 0.02$, $\eta_2 = 0.02$, $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\kappa_1 = 0.03$, $\kappa_2 = 0.3$, $\delta_1 = 0.2$, $\delta_2 = 0.12$.

Table 3. The Parameters and Baseline Values of the Model with Public Health Education and Hospital Treatment (23).

Parameters	Baseline Values	Ranges	References
Λ	3,768,410 year^{-1}	[3,000,000, 4,000,000]	[42]
μ	0.02041 year^{-1}	[0.0143, 0.03]	[23]
ψ	Variable	[0–1]	Assumed
ϕ	Variable	[0–1]	Assumed
ν	Variable	[0–1]	Assumed
β	Variable	Varied	Assumed
σ	0.25	[0–1]	[25,43]
z	0.11	[0–0.9]	[16]
κ_1	Variable	[0–1]	Assumed
κ_2	Variable	[0–1]	Assumed
γ_1	0.09 year^{-1}	[0–1]	[20]
γ_2	0.72 year^{-1}	[0–1]	[20]
η_1	Variable	[0–1]	assumed
η_2	Variable	[0–1]	assumed
δ_1	0.2 year^{-1}	-	[44]
δ_2	0.02 year^{-1}	-	[45]

Figure 6 investigates the impact of varying the rate at which the educated susceptible lose the awareness ϕ. This figure demonstrates a worst-case scenario in which an increase in loss of awareness in the susceptible individual leads to an increase in the proportion of infectious individuals.

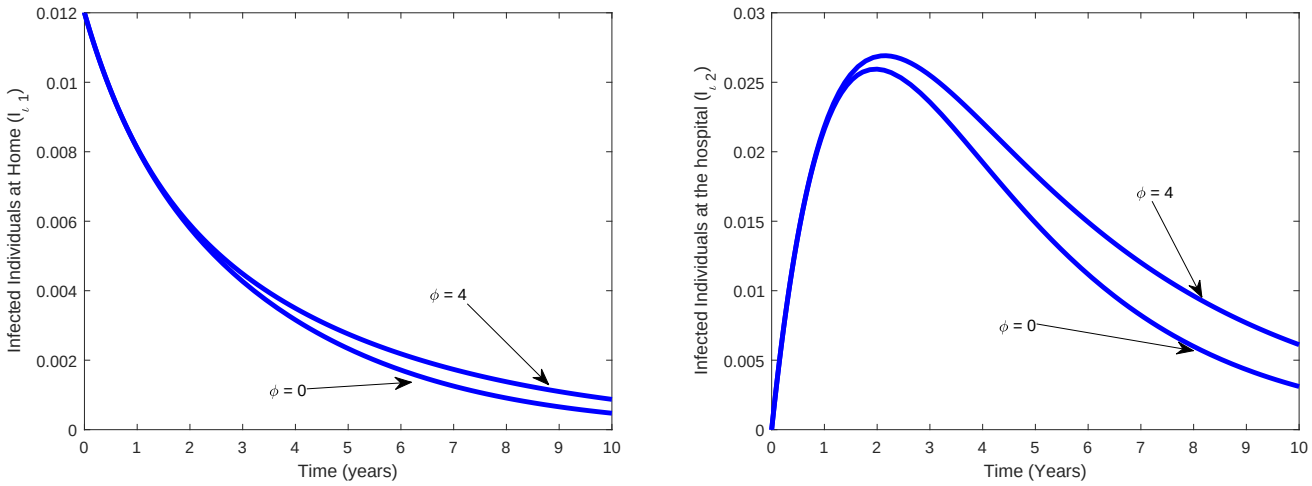

Figure 6. Impact at which educated susceptible lose awareness (ϕ) on I_{i1}, and I_{i2}, varying ϕ. Other parameters are given as $\Lambda = 450{,}862$, $\beta = 0.86, \nu = 0.25, \phi = 0.2, \sigma = 0.65, z = 0.11, \mu = 0.02041$, $\psi = 0.5$, $\eta_1 = 0.02$, $\eta_2 = 0.02$, $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\kappa_1 = 0.03$, $\kappa_2 = 0.3$, $\delta_1 = 0.2$, $\delta_2 = 0.12$.

Figure 7 investigates the effect of varying the reduction of infection by infected individuals receiving treatment at the hospital z. In this figure, z, is varied between 0 to 1. Of course, lowering the value of z, from 1 to 0, as expected, reduces the proportion of infected individuals, although at different rates.

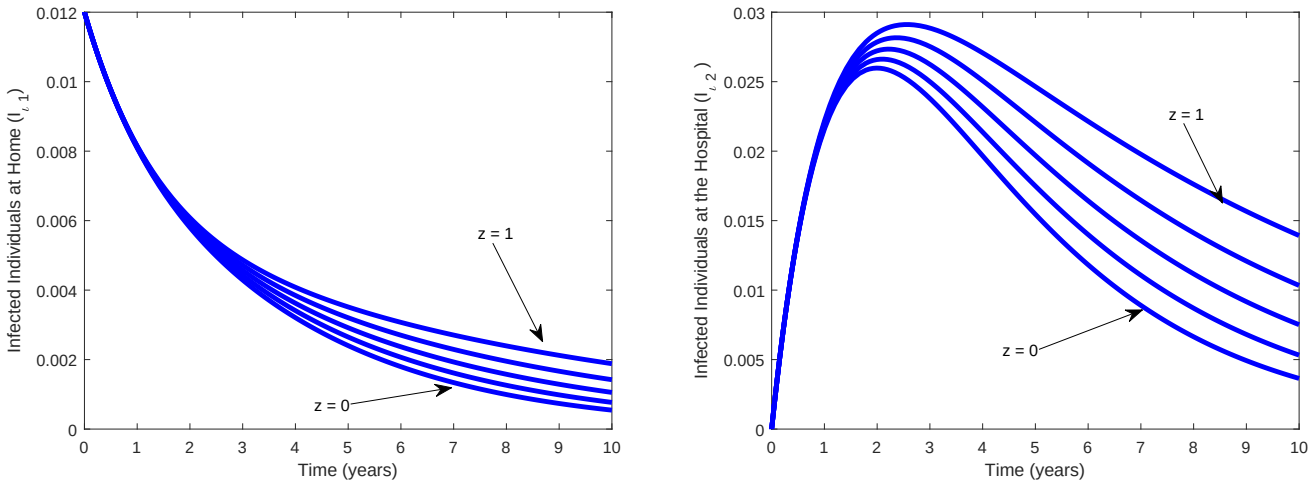

Figure 7. Impact of reduction of infection rate as a result of I_2 (z) on I_{i1}, and I_{i2}, varying z. Other parameters are given as $\Lambda = 450{,}862$, $\beta = 0.86, \nu = 0.25, \phi = 0.2, \sigma = 0.65, z = 0.11, \mu = 0.02041$, $\psi = 0.5$, $\eta_1 = 0.02$, $\eta_2 = 0.02$, $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\kappa_1 = 0.03$, $\kappa_2 = 0.3$, $\delta_1 = 0.2$, $\delta_2 = 0.12$.

Figure 8 illustrates the impact of varying the rate at which infected individuals at home go out for treatment in the hospital, η_1 between 0 and 1 on the individuals treated at home I_{i1} and infected individuals receiving treatment at the hospital, respectively. Obviously, as expected, an increase in the value of η_1, from 0 to 1 greatly reduced the number of infected individuals both at home and at hospital. Epidemiologically, this shows that rate at which infected individuals at home go out for treatment in the hospital have a great impact on reducing the transmission of TB disease in the population.

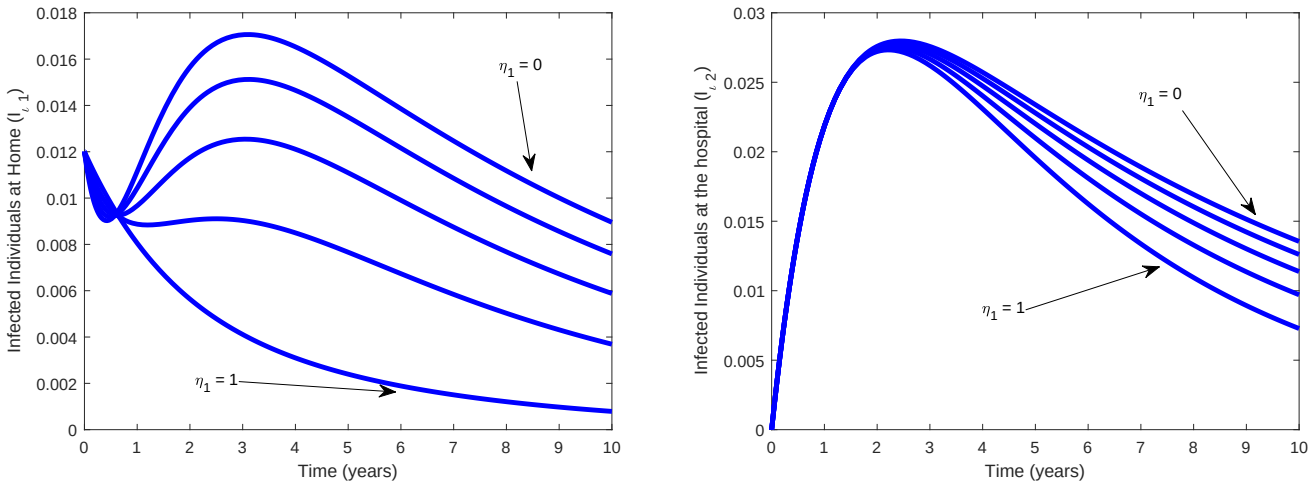

Figure 8. Impact of progression rate (η_1) from I_{i1} to I_{i2} on I_{i1}, and I_{i2}, varying η_1. Other parameters are given as $\Lambda = 450{,}862$, $\beta = 0.86, \nu = 0.25, \phi = 0.2, \sigma = 0.65, z = 0.11, \mu = 0.02041$, $\psi = 0.5$, $\eta_1 = 0.02$, $\eta_2 = 0.02$, $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\kappa_1 = 0.03$, $\kappa_2 = 0.3$, $\delta_1 = 0.2$, $\delta_2 = 0.12$.

7. Conclusions

This paper presented a new deterministic model of TB infection subject to the use of public health education and hospital treatment. To gain insight into its dynamic features, the model was rigorously analyzed. The analyses of the model, which contains six mutually-exclusive epidemiological partitions, show that the model undergoes the phenomenon of backward bifurcation when the effective reproduction number (R_{ph}) is less than one, where two stable equilibria, namely, the DFE and an EEP coexist when the corresponding effective reproductive number is less than one. This backward bifurcation phenomenon of this article is very vital, and this occurs only under education of susceptible individuals and treatment of TB-infected individuals in the hospital. This is mostly telling us that, even if the effective reproduction number is less than unity, while necessary, it is not sufficient for efficiently controlling the spread of a TB epidemic, which is against classical epidemiological theory. In this scenario, TB elimination will depend upon the initial sizes of the sub-populations of the model. The parameters are sensitive to the transmission dynamics of TB diseases, either negatively $(\mu, \nu, \psi, \eta_1, \gamma_1, \gamma_2, \delta_1, \delta_2,$ and $\kappa_2)$ or positively $(\beta_1, \beta_2$ and $\kappa_1)$. According to the numerical investigation, increasing the public health education ψ on the S_ι class, and the progression rate η_1 from I_{i1} to $I_{i2,}$ have a significant great effect on reducing the prevalence of TB burden see Figures 5 and 8. Given that public health education and hospital treatment can reduce the spread of TB infection, this programme should be continued and improved.

In light of the model study in this paper, some gaps in this paper need to be filled; these proposed gaps will allow for possible extension and improvement of the paper. The proposed model can be extended and improved in the future by:

- Considering a stochastic model approach. This will result in more realistic TB model dynamics.
- Since the spread of tuberculosis affects all age groups, it is crucial to consider the dynamics of the TB model by incorporating an age-structured model.
- Real data will also be considered because collecting data for TB patients is difficult in epidemiological models; as a result, we use data collected or estimated from literature sources. Once we have real-world data for TB patients, we can compare it to theoretical outcomes.
- Analyzing the dynamics of the TB model using a fractional order differential equation (FODE). It will be extremely interesting to use a FODE to examine the dynamics of TB model.

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Conflicts of Interest: The authors declare no conflict of interest.

Appendix A

Proof of Theorem 5. At the endemic state, the model system Equation (23) has an equilibrium point called TB endemic equilibrium point.

$$\frac{dS_\iota^*}{dt} = \frac{dP_\iota^*}{dt} = \frac{dE_\iota^*}{dt} = \frac{dI_{\iota 1}^*}{dt} = \frac{dI_{\iota 2}^*}{dt} = \frac{dR_\iota^*}{dt} = 0. \tag{A1}$$

By setting the derivatives of the left hand side of Equation (23) to zero and solving simultaneously we have

$$\begin{aligned}
S_\iota^* &= \tfrac{\Lambda(\nu\lambda^*+\phi+\mu)}{\nu\lambda^{*2}+A_4\lambda^*+A_5},\\
P_\iota^* &= \tfrac{\psi\lambda^*}{\nu\lambda^{*2}+A_4\lambda^*+A_5},\\
E_\iota^* &= \tfrac{\Lambda(\nu\lambda^*+\phi+\mu+\nu\psi)(A_{11}\lambda^* A_{10}\lambda^*)}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})},\\
I_{\iota 1}^* &= \tfrac{\kappa_1\Lambda A_1 A_3(\sigma\lambda^*+\mu)(\nu\lambda^*+\phi+\mu+\nu\psi)\lambda^*}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})},\\
I_{\iota 2}^* &= \tfrac{\kappa_1\Lambda A_1(\sigma\lambda^*+\mu)(\nu\lambda^*+\phi+\mu+\nu\psi)\lambda^*}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})},\\
R_\iota^* &= \tfrac{\kappa_1\Lambda A_0(\gamma_1 A_3+\gamma_2)(\nu\lambda^*+\phi+\mu+\nu\psi)\lambda^*}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})}.
\end{aligned} \tag{A2}$$

Further

$$\lambda^* = \frac{\beta(I_{\iota 1}^* + zI_{\iota 2}^*)}{N_\iota^*}, \tag{A3}$$

First, the nominator

$$\beta(I_{\iota 1}^* + zI_{\iota 2}^*) = \beta\left[\frac{\kappa_1\Lambda A_1 A_3(\sigma\lambda^*+\mu)(\nu\lambda^*+\phi+\mu+\nu\psi)\lambda^*}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})} + \frac{z\kappa_1\Lambda A_1(\sigma\lambda^*+\mu)(\nu\lambda^*+\phi+\mu+\nu\psi)\lambda^*}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})}\right] \tag{A4}$$

$$= \frac{\beta(A_3+z)\kappa_1\Lambda A_1(\sigma\lambda^*+\mu)(\nu\lambda^*+\phi+\mu+\nu\psi)\lambda^*}{(\nu\lambda^{*2}+A_4\lambda^*+A_5)(A_9\lambda^*+A_{10})}, \tag{A5}$$

and the denominator is $N_\iota = S_\iota^* + P_\iota^* + E_\iota^* + I_{\iota 1}^* + I_{\iota 2}^* + R_\iota^*$. After substituting the values of $S_\iota^* + P_\iota^* + E_\iota^* + I_{\iota 1}^* + I_{\iota 2}^* + R_\iota^*$ in Equation (A2), we obtain

$$N_\iota = \frac{\Lambda}{(\nu\lambda^{*2} + A_4\lambda + A_5)(A_9\lambda^* + A_{10})}[(\nu\lambda^* + \phi + \mu + \psi)(A_9\lambda^* + A_{10}) + A_{12}(\lambda^*)], \tag{A6}$$

where

$$\begin{aligned} A_{12}(\lambda^*) = (\nu\lambda^* + \phi + \mu + \nu\psi)(A_{11}\lambda^* + A_{10})\lambda^* + \kappa_1 A_1 A_3(\sigma\lambda^* + \mu)(\nu\lambda^* + \phi + \mu + \nu\psi)\lambda^* \\ + \kappa_1 A_1(\sigma\lambda^* + \mu)(\nu\lambda^* + \phi + \mu + \nu\psi)\lambda^* \kappa_1 A_1(\gamma_1 A_3 + \gamma_2)(\nu\lambda^* + \phi + \mu + \nu\psi) \\ = (\nu\lambda^* + \phi + \mu + \nu\psi)\lambda^*(A_{13}\lambda^* + A_{14}), \end{aligned} \tag{A7}$$

where

$$A_{13} = A_{11} + \kappa_1 A_1 A_3 + \kappa_1 A_1 \sigma,\ A_{14} = A_{10} + \mu\kappa_1 A_1 A_3 + \mu\kappa_1 A_1 + \kappa_1 A_1(\gamma_1 A_3 + \sigma_2).$$

Thus

$$N_\iota = \frac{\Lambda[(\nu\lambda^* + \phi + \mu + \psi)(A_9\lambda^* + A_{10}) + (\nu\lambda^* + \phi + \mu + \psi)(A_{13}\lambda^* + A_{14})]}{(\nu\lambda^{*2} + A_4\lambda^* + A_5)(A_9\lambda^* + A_{10})}. \tag{A8}$$

Hence, from (A3) using (A5) and (A8), to get

$$\lambda^* = \frac{\kappa_1 A_1\beta(A_3 + z)(\sigma\lambda^* + \mu)(\nu\lambda^* + \phi + \mu + \nu\psi)\lambda^*}{[(\nu\lambda^* + \phi + \mu + \psi)(A_9\lambda^* + A_{10}) + (\nu\lambda^* + \phi + \mu + \nu\psi)(A_{13}\lambda^* + A_{14})\lambda^*]}, \tag{A9}$$

$$\Rightarrow$$

$$\lambda^* - \frac{\kappa_1 A_1\beta(A_3 + z)(\sigma\lambda^* + \mu)(\nu\lambda^* + \phi + \mu + \nu\psi)\lambda^*}{[(\nu\lambda^* + \phi + \mu + \psi)(A_9\lambda^* + A_{10}) + (\nu\lambda^* + \phi + \mu + \nu\psi)(A_{13}\lambda^* + A_{14})\lambda^*]} = 0,$$

$$\Rightarrow$$

$$\lambda^* = 0 \text{ or } 1 - \frac{\kappa_1 A_1\beta(A_3 + z)(\sigma\lambda^* + \mu)(\nu\lambda^* + \phi + \mu + \nu\psi)}{[(\nu\lambda^* + \phi + \mu + \psi)(A_9\lambda^* + A_{10}) + (\nu\lambda^* + \phi + \mu + \nu\psi)(A_{13}\lambda^* + A_{14})\lambda^*]} = 0,$$

$$\begin{aligned} \Rightarrow &(\nu\lambda^* + \phi + \mu + \psi)(A_9\lambda^* + A_{10}) + (\nu\lambda^* + \phi + \mu + \nu\psi)(A_{13}\lambda^* + A_{14})\lambda^*] \\ &-\kappa_1 A_1\beta(A_3 + z)(\sigma\lambda^* + \mu)(\nu\lambda^* + \phi + \mu + \nu\psi). \end{aligned} \tag{A10}$$

$$\begin{aligned} &\nu\lambda^*(A_9\lambda^* + A_{10}) + (\phi + \mu + \psi)(A_9\lambda^* + A_{10}) + \nu\lambda^{*2}(A_{13}\lambda^* + A_{14}) + (\phi + \mu + \nu\psi) \\ &(A_{13}\lambda^* + A_{14})\lambda^* - \kappa_1 A_1\beta(A_3 + z)(\sigma\lambda^* + \mu)\nu\lambda^* - \kappa_1 A_1\beta(A_3 + z)(\sigma\lambda^* + \mu)(\phi + \mu + \nu\psi) = 0. \end{aligned} \tag{A11}$$

$$\begin{aligned} &\nu A_9\lambda^{*2} + \nu A_{10}\lambda^* + A_9(\phi + \mu + \psi)\lambda^* + A_{10}(\phi + \mu + \psi) + \nu A_{13}\lambda^{*3} + \nu A_{14}\lambda^{*2} \\ &+ A_{13}(\phi + \mu + \nu\psi)\lambda^{*2} + A_{14}(\phi + \mu + \nu\psi)\lambda^* - \kappa_1 A_1\beta(A_3 + z)\sigma\lambda^{*2} - \mu\kappa_1 A_1\beta(A_3 + z)\nu\lambda^* \\ &- (\phi + \mu + \nu\psi)\kappa_1 A_1\beta(A_3 + z)\sigma\lambda^* - \mu(\phi + \mu + \nu\psi)\kappa_1 A_1\beta(A_3 + z) = 0, \end{aligned} \tag{A12}$$

$$\nu A_{13}\lambda^{*3} + A_{15}\lambda^{*2} + A_{16}\lambda^* + A_{17} = 0. \tag{A13}$$

where

$$\begin{aligned} A_{15} &= \nu A_9 + \nu A_{14} + A_{13}(\phi + \mu + \nu\psi) - \sigma\nu\kappa_1 A_1\beta(A_3 + z) \\ A_{16} &= \nu A_{10} + A_9(\phi + \mu + \psi) + A_{14}(\phi + \mu + \nu\psi) - \kappa_1 A_1\beta(A_3 + z)(\mu\nu + \sigma(\phi + \mu + \nu\psi)) \\ A_{17} &= A_{10}(\phi + \mu + \psi)(1 - R_{ph}). \end{aligned} \tag{A14}$$

Here, R_{ph} is the effective reproduction number given in (15).

Clearly, it is evident that (I_ι) is given by the positive real roots of the polynomial (A13). The number of possible positive real roots of the cubic polynomial (A13) depends on the signs of A_{15}, A_{16}, and A_{17}. The following theorem can be established. □

Appendix B

Proof of Theorem 7. To apply the method in presented in Theorem 4.1 of [6], the following simplification and change of variables are made on the model system (23). Let

$$S_\iota = x_1, P_\iota = x_2, E_\iota = x_3, I_{\iota 1} = x_4, I_{\iota 2} = x_5, R_\iota = x_6, \tag{A15}$$

so that

$$N_\iota = x_1 + x_2 + x_3 + x_4 + x_5 + x_6, \tag{A16}$$

Also, by utilizing the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$, then, the model system (23) becomes $\frac{dX}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ as follows

$$\begin{aligned}
\tfrac{dx_1}{dt} &= f_1 = \Lambda + \phi x_2 - (\lambda + \psi + \mu)x_1,\\
\tfrac{dx_2}{dt} &= f_2 = \psi x_1 - (\nu\lambda + \phi + \mu)x_2,\\
\tfrac{dx_3}{dt} &= f_3 = \lambda x_1 + \nu\lambda x_2 + \sigma\lambda x_6 - (\kappa_1 + \kappa_2 + \mu)x_3,\\
\tfrac{dx_4}{dt} &= f_4 = \kappa_1 x_3 + \eta_2 x_5 - (\eta_1 + \gamma_1 + \mu + \delta_1)x_4,\\
\tfrac{dx_5}{dt} &= f_5 = \kappa_2 x_3 + \eta_1 x_4 - (\eta_2 + \gamma_2 + \mu + \delta_2)x_5,\\
\tfrac{dx_6}{dt} &= f_6 = \gamma_1 x_4 + \gamma_2 x_5 - (\sigma\lambda + \mu)x_6,
\end{aligned} \tag{A17}$$

where

$$\lambda = \frac{\beta(x_4 + z x_5)}{N_\iota}, \tag{A18}$$

The Jacobian of the system (A17), evaluated at the TFE, T^0 (denoted by $J(T^0)$), is given by

$$J(T^0) = \begin{pmatrix}
-(\mu+\psi) & \phi & 0 & -\frac{\beta x_1^0}{N_\iota^0} & -\frac{\beta z x_1^0}{N_\iota^0} & 0\\
\psi & -(\phi+\mu) & 0 & -\frac{\nu\beta x_2^0}{N_\iota^0} & -\frac{\nu\beta z x_2^0}{N_\iota^0} & 0\\
0 & 0 & -A_1 & \frac{\beta(x_1^0+\nu x_2^0)}{N_\iota^0} & \frac{\beta z(x_1^0+\nu x_2^0)}{N_\iota^0} & 0\\
0 & 0 & \kappa_1 & -A_2 & \eta_2 & 0\\
0 & 0 & \kappa_2 & \eta_1 & -A_3 & 0\\
0 & 0 & 0 & \gamma_1 & \gamma_2 & -\mu
\end{pmatrix}, \tag{A19}$$

where A_1, A_2, and A_3 are as in (13), from which it has been shown in (14) that the effective reproduction number, R_{ph} is given by

$$R_{ph} = \frac{\beta(\phi + \mu + \nu\psi)(\kappa_1 A_3 + \kappa_2\eta_2) + z(\kappa_2 A_2 + \eta_1\kappa_1)}{A_1(A_2A_3 - \eta_1\eta_2)(\psi + \phi + \mu)} \tag{A20}$$

Consider the case when $R_{ph} = 1$. Suppose, further, that $\beta = \beta^0$ is chosen as a bifurcation parameter, since R_{ph} is often inconvenient to use directly as bifurcation parameter. Solving for β gives $R_{ph} = 1$ when

$$\beta = \beta^0 = \frac{A_1(A_2A_3 - \eta_1\eta_2)(\psi + \phi + \mu)}{\beta(\phi + \mu + \nu\psi)(\kappa_1A_3 + \kappa_2\eta_2) + z(\kappa_2A_2 + \eta_1\kappa_1)} \tag{A21}$$

The linearized system of the transformed model system (23) with $\beta = \beta^0$ chosen as a bifurcation parameter has a simple zero eigenvalue. We then calculate the right eigenvector W and the left eigenvector V which are associated with the zero eigenvalue of the Jacobian of (A19) at (denoted by J_{β^0}) chosen such that $J(T_0)W = 0$ and $VJ(T_0) = 0$ with $VW = 1$, where

$$W = [w_1, w_2, w_3, w_4, w_5, w_6],$$
$$V = [v_1, v_2, v_3, v_4, v_5, v_6].$$

Then

$$J(T^0)W_i = \begin{pmatrix} -(\mu+\psi) & \phi & 0 & -\frac{\beta x^0}{N_i^0} & -\frac{z\beta x^0}{N_i^0} & 0 \\ \psi & -(\phi+\mu) & 0 & -\frac{\nu\beta x^0}{N_i^0} & -\frac{z\nu\beta x^0}{N_i^0} & 0 \\ 0 & 0 & -A_1 & \frac{\beta x_1^0 + \nu\beta x_2^0}{N_i^0} & \frac{z\beta x_1^0 + \nu z\beta x_2^0}{N_i^0} & 0 \\ 0 & 0 & \kappa_1 & -A_2 & \eta_2 & 0 \\ 0 & 0 & \kappa_2 & \eta_1 & -A_3 & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & -\mu \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}. \tag{A22}$$

i.e.,

$$\begin{aligned} &-(\mu+\psi)w_1 + \phi w_2 - \frac{\beta x_1^0}{N_i^0}w_4 - \frac{z\beta x_1^0}{N_i^0}w_5 = 0, \\ &\psi w_1 - (\mu+\phi)w_2 - \frac{\nu\beta x_2^0}{N_i^0}w_4 - \frac{z\nu\beta x_2^0}{N_i^0}w_5 = 0, \\ &-A_1w_3 + \frac{\beta x_1^0 + \nu\beta x_2^0}{N_i^0}w_4 + \frac{z\beta x_1^0 + \nu z\beta x_2^0}{N_i^0}w_5 = 0, \\ &-\kappa_1w_3 + A_2w_4 + \eta_2w_5 = 0, \\ &\kappa_2w_3 + \eta_1w_4 - A_3w_5 = 0, \\ &\gamma_1w_4 + \gamma_2w_5 - \mu w_6 = 0. \end{aligned} \tag{A23}$$

Solving (A23), gives

$$\begin{aligned} w_1 &= \frac{-A_1(\eta_2+zA_2)\{\phi(\beta x_1^0+\nu\beta x_2^0)+\beta x_1^0((\psi+\mu)(\phi+\mu)-\psi\phi)\}w_5}{((\psi+\mu)(\phi+\mu)-\psi\phi)(A_1A_2N_i^0-\beta(x_1^0+\nu x_2^0)\kappa_1)(\psi+\mu)} < 0, \\ w_2 &= \frac{-A_1(\eta_2+zA_2)x_1^0+\nu x_2^0)w_5}{((\psi+\mu)(\phi+\mu)-\psi\phi)(A_1A_2N_i^0-\beta(x_1^0+\nu x_2^0)\kappa_1)} < 0, \\ w_3 &= \frac{\beta(x_1^0+\nu x_2^0)(\eta_2+zA_2)w_5}{A_1A_2N_i^0-\beta(x_1^0+\nu x_2^0)\kappa_1} > 0, \\ w_4 &= \frac{\{\eta_2A_1N_i^0+\kappa_1\beta(x_1^0+\nu x_2^0)z\}w_5}{A_1A_2N_i^0-\beta(x_1^0+\nu x_2^0)\kappa_1} > 0, \\ w_5 &> 0 \,(\text{can take any value}), \\ w_6 &= \frac{\{A_1N_i^0\gamma_1\eta_2+(A_1A_2N_i^0-\beta(x_1^0+\nu x_2^0)\kappa_1)\gamma_2+\beta(x_1^0+\nu x_2^0)\kappa_1\gamma_1z\}w_5}{\mu(A_1A_2N_i^0-\beta(x_1^0+\nu x_2^0)\kappa_1)} > 0. \end{aligned} \tag{A24}$$

Similarly, calculating the left eigenvector $V = (v_1, v_2, v_3, v_4, v_5, v_6)^T$ with $VJ(T^0) = 0$, gives

$$J(T^0)V_i = (v_1, v_2, v_3, v_4, v_5, v_6)\begin{pmatrix} -(\mu+\psi) & \phi & 0 & -\frac{\beta x^0}{N_t^0} & -\frac{z\beta x^0}{N_t^0} & 0 \\ \psi & -(\phi+\mu) & 0 & -\frac{\nu\beta x^0}{N_t^0} & -\frac{z\nu\beta x^0}{N_t^0} & 0 \\ 0 & 0 & -A_1 & \frac{\beta x^0+\nu\beta x^0}{N_t^0} & \frac{z\beta x^0+\nu z\beta x^0}{N_t^0} & 0 \\ 0 & 0 & \kappa_1 & -A_2 & \eta_2 & 0 \\ 0 & 0 & \kappa_2 & \eta_1 & -A_3 & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & -\mu \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}. \tag{A25}$$

i.e.,

$$\begin{aligned} &-(\mu+\psi)v_1 + \psi v_2 = 0, \\ &\phi v_1 - (\mu+\phi)v_2 = 0, \\ &-A_1 v_3 + \kappa_1 v_4 + \kappa_2 v_5 = 0, \\ &-\tfrac{\beta x_1^0}{N_t^0}v_1 - \tfrac{\nu\beta x_2^0}{N_t^0}v_2 + \tfrac{\beta x_1^0+\nu\beta x_2^0}{N_t^0}v_3 - A_2 v_4 + \eta_1 v_5 + \gamma_1 v_6 = 0, \\ &-\tfrac{z\beta x_1^0}{N_t^0}v_1 - \tfrac{\nu z\beta x_2^0}{N_t^0}v_2 + \tfrac{z\beta x_1^0+\nu z\beta x_2^0}{N_t^0}v_3 - \eta_2 v_4 - A_3 v_5 + \gamma_2 v_6 = 0, \\ &-\mu_1 v_6 = 0. \end{aligned} \tag{A26}$$

Solving (A26), gives

$$\begin{aligned} &v_1 = v_2 = v_6 = 0, \\ &v_3 = \tfrac{\kappa_2\{\kappa_1\beta(x_1^0+\nu x_2^0)+(A_1A_2N_t^0-\beta(x_1^0+\nu x_2^0))\}v_5}{A_1A_2N_t^0-\beta(x_1^0+\nu x_2^0)} > 0, \\ &v_4 = \tfrac{\beta(x_1^0+\nu x_2^0))\kappa_2 v_5}{A_1A_2N_t^0-\beta(x_1^0+\nu x_2^0)\kappa_1} > 0, \\ &v_5 > 0 \,(\text{can take any value}). \end{aligned} \tag{A27}$$

The local dynamics of model system (23) around TB free equilibrium T^0 are totally calculated by a and b represented as

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j}, \tag{A28}$$

$$b = \sum_{k,i=1}^{4} v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \varphi}(0,0). \tag{A29}$$

Now, to determine the coefficients a and b described in Theorem 4.1 of Castillo Chavez and Song [6] as follow:

$$a = \frac{2v_3\beta}{N_t^0}(w_1 + w_2\nu + w_6\sigma)(w_4 + w_5 z). \tag{A30}$$

and

$$b = \frac{\partial^2 f_3}{\partial x_4^0 \partial \beta^0}(0,0) + \frac{\partial^2 f_3}{\partial x_5^0 \partial \beta^0}(0,0) = \frac{x_1^0 + \nu x_2^0 + \sigma x_6^0}{N_t^0} + \frac{z x_1^0 + \nu z x_2^0 + z\sigma x_6^0}{N_t^0} > 0. \quad \text{(A31)}$$

Hence, since $a > 0$ and $b > 0$, in this situation, it follows from Theorem 4.1 of Castillo Chavez and Song that the system model (23) or the transformed model (A17), will undergo a phenomenon of backward bifurcation when $R_{ph} = 1$. Therefore, establishing that TB endemic equilibrium is locally asymptotically stable if $R_{ph} < 1$. □

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