Hindawi
Discrete Dynamics in Nature and Society
Volume 2024, Article ID 5883142, 17 pages
https://doi.org/10.1155/2024/5883142

Research Article

A Model for the Propagation and Control of Pulmonary Tuberculosis Disease in Kenya

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Received 8 November 2023; Revised 10 March 2024; Accepted 21 March 2024; Published 31 March 2024

Academic Editor: Anibal Coronel

Pulmonary tuberculosis is among the leading infectious diseases causing mortality worldwide. Therefore, scaling up intervention strategies to reduce the spread of infections in the population is imperative. In this paper, a population-based compartmental approach has been employed to formulate a mathematical model of pulmonary tuberculosis that incorporates an asymptomatic infectious population. The model includes asymptomatic infectious individuals since they spread infections incessantly to susceptible populations without being noticed, thus contributing to the high rate of infection transmission. Qualitative and numerical analyses were performed to determine the impact of various intervention strategies on controlling infection transmission in the population. Sensitivity and numerical results indicate that increasing screening of latently infected and asymptomatic infectious individuals reduces infection transmission to the susceptible population. Numerical results demonstrate that the combination of vaccination, screening, and treatment of all forms of pulmonary tuberculosis is the most effective intervention in decreasing infection transmission. Furthermore, a combination of screening and treatment of all forms of pulmonary tuberculosis proves more effective than a combination of vaccination and treatment of symptomatic infectious individuals alone. Treating the symptomatic infectious population alone is identified as the least effective intervention for curtailing infection transmission in the susceptible population. These study findings will guide healthcare officials in making decisions regarding the screening of latently infected and asymptomatic infectious pulmonary tuberculosis patients, thereby aiding in the fight against epidemics of this disease.

1. Introduction

Mathematical modeling is one of the valuable tools that explore the transmission dynamics of infectious diseases and assess the impact of various control interventions. Therefore, mathematical modeling of infectious diseases is used to guide public health policies and inform decision-making during epidemics [1, 2]. This paper employs a classical SEIR transmission mechanism to formulate a novel model for pulmonary tuberculosis, aiming to accurately depict its natural progression. The SEIR model, a widely used compartmental model in epidemiology, is utilized to forecast the spread of infectious diseases with latent or exposed phases [3]. During the latent phase of a disease, individuals are neither infectious nor symptomatic [4]. The evolution of infectious diseases, taking into consideration the SEIR model, has been discussed by several authors [5–9]. Research suggests that around one-quarter of the global population harbors latent pulmonary tuberculosis infections [10]. Consequently, an epidemiological model for pulmonary tuberculosis must account for this latent population and hence adopts the classical SEIR framework. Within the SEIR model, the population is categorized into four compartments: Susceptible (S), Exposed (E), Infectious (I), and Recovered (R) [2].

Pulmonary tuberculosis is a contagious disease that primarily affects the lungs. It is caused by microorganisms known as Mycobacterium tuberculosis, which spread when an infectious individual coughs, sneezes, speaks, or sings [4, 10]. The disease exerts a heavy toll on the human population, causing a significant number of deaths worldwide. Every year, approximately 10,000,000 people fall ill with pulmonary tuberculosis across the globe, with about 15% of them succumbing to the disease. In 2022, approximately 10,600,000 people fell ill with tuberculosis worldwide, resulting in 1.6 million deaths [11]. In Kenya, data from Civil Registration Services show that pulmonary tuberculosis accounted for about 5.4% of all reported deaths in 2019 [12]. Therefore, it is imperative to scale up preventive measures aimed at reducing the transmission of infections to the susceptible population. The United Nations' "Agenda 2030 Sustainable Development Goals (SDGs)" has identified tuberculosis as one of the communicable diseases that need to be eradicated worldwide by 2030 for sustainable development [13].

Various mathematical models for pulmonary tuberculosis disease have been formulated, focusing on diverse strategies to control the transmission of infections, such as vaccination, early diagnosis, social protection, and treatment of drug-resistant strains [14–16]. Athithan et al. [17] analyzed a nonlinear model of pulmonary tuberculosis considering case detention and treatment, and their results showed that sustaining treatment and case detention hold great promise in controlling the spread of infections. Salpeter et al. [18] conducted a study on the mathematical modeling of pulmonary tuberculosis with estimates of reproduction number and infection delay function. Their outcome revealed that the risk of infection reactivation decreases rapidly and then gradually, for the first ten years after infection. Houben et al. [19] formulated a mathematical model to estimate the global burden of latent tuberculosis infection, and their results estimated that approximately a quarter of the world's population was infected with latent tuberculosis in 2014. Vaccination and effective contact rates on the spread of pulmonary tuberculosis were assessed using a mathematical model in [20, 21]. Their results showed that vaccination coverage is not sufficient to control pulmonary tuberculosis, and the effective contact rate has a higher impact on the spread of infections. Aparicio et al. [22] explored the strengths and limitations of homogeneous and heterogeneous mixing in tuberculosis epidemics through mathematical modeling, and their results indicated that a decrease in pulmonary tuberculosis incidence was due to a reduction in progression rates. Kasereka et al. [23] simulated a mathematical model of pulmonary tuberculosis transmission in the Democratic Republic of Congo, revealing that monitoring contacts, detection of latent infection, and treatment are the optimal strategies to reduce the transmission of infections in the population.

The main purpose of this study is to investigate the impact of various intervention strategies on controlling the transmission of infections in the population. We formulated and analyzed a deterministic pulmonary tuberculosis model incorporating an asymptomatic infectious population. The justification for incorporating an asymptomatic infectious population stems from a report by the National TB Prevalence Survey of 2016 in Kenya, which showed that 26% of prevalent cases diagnosed during their survey were asymptomatic infectious [24]. We investigated the effects of screening asymptomatic and latently infected populations on controlling the transmission of infections to the susceptible population. This paper is organized as follows: Section 2 presents the model formulation, while in Section 3 mathematical analyses have been performed. Section 4 provides the numerical simulations of the model to illustrate the impact of various intervention strategies on controlling the transmission of infections. Finally, Section 5 presents the conclusion.

2. Pulmonary Tuberculosis Model Formulation Incorporating Asymptomatic Infectious Population

This study adopts a classical SEIR transmission mechanism to formulate a new epidemiological model describing the transmission of pulmonary tuberculosis disease. The model incorporates an asymptomatic infectious population, as they spread infections incessantly to the susceptible population without being noticed, thus contributing to the high rate of transmission. The total human population size at a time t, denoted as $N(t)$, is subdivided into susceptible $S(t)$, vaccinated $V(t)$, latent infected $E(t)$, asymptomatic infectious $I_a(t)$, symptomatic infectious $I_s(t)$, latent infected undergoing treatment $T_E(t)$, asymptomatic infectious undergoing treatment $T_a(t)$, symptomatic infectious undergoing treatment $T_s(t)$, and recovered $R(t)$. Hence, for total human population, we have $N(t) = S(t) + V(t) + E(t) + I_a(t) + I_s(t) + T_E(t) + T_a(t) + T_s(t) + R(t)$.

Individuals are recruited into the population at a constant rate of π. A fraction of the recruits is vaccinated at a constant rate of P and enters the vaccinated class, whereas the rest become susceptible. The susceptible individuals become latently infected after effective contact with any of the following populations: symptomatic infectious, asymptomatic infectious, symptomatic infectious undergoing treatment, and asymptomatic infectious undergoing treatment. The force of infection is represented by $\lambda = \beta(I_s + \eta_1 I_a + \eta_2 T_s + \eta_3 T_a)$, where β represents the transmission rate of pulmonary tuberculosis infections, while η_1, η_2, and η_3 are the dimensionless transmission coefficients accounting for the relative infectiousness of asymptomatic infectious individuals, symptomatic infectious individuals undergoing treatment, and asymptomatic infectious individuals undergoing treatment, respectively, with $\eta_3 < \eta_2 < \eta_1$. This hierarchy assumes that asymptomatic infectious individuals (I_a) are more infectious than symptomatic individuals undergoing treatment (T_s), who are, in turn, more infectious than asymptomatic infectious individuals undergoing treatment (T_a). The dimensionless transmission coefficients, η_1, η_2, and η_3 are considered to be less than 1. The model assumes that vaccination is not 100% effective, and thus the

vaccinated class has a chance of being latently infected at a force of infection given by $\lambda_v = (1-\rho)\lambda = (1-\rho)\beta(I_s + \eta_1 I_a + \eta_2 T_s + \eta_3 T_a)$, where ρ is the vaccine efficacy, such that $0 \le \rho \le 1$. The latent infected individuals, denoted by $E(t)$, are either screened at a constant rate of θ_1 and moved to the latent infected undergoing treatment class $T_E(t)$ or progress to the asymptomatic infectious class $I_a(t)$, at a rate of χ_1. Alternatively, they may develop severe disease and transition to the symptomatic infectious class $I_s(t)$, at a constant rate of χ_2. Asymptomatic infectious individuals are either screened at a rate of θ_2 and moved to the asymptomatic infectious undergoing treatment class $T_a(t)$, or they progress to severe disease and join the symptomatic infectious class $I_s(t)$ at a rate of ω. Symptomatic infectious individuals are identified for treatment at a rate of θ_3 and moved to the symptomatic infectious undergoing treatment class $T_s(t)$. The rate of disease-induced deaths due to pulmonary tuberculosis for individuals in the symptomatic infectious class is given by δ_1, whereas the rate of death due to the disease for symptomatic infectious individuals undergoing treatment is given by δ_2. Treatment for different forms of pulmonary tuberculosis is assumed to be successful, and thus latent infected individuals undergoing treatment, asymptomatic infectious individuals undergoing treatment, and symptomatic infectious individuals undergoing treatment recover at rates of ξ_1, ξ_2, and ξ_3, respectively, and move to the recovered class R. The model assumes that recovered individuals become susceptible again after their immunity wanes at a rate of σ. The rate at which individuals die from causes other than pulmonary tuberculosis is denoted by μ. It is worth noting that all parameters are positive constants.

The compartmental model illustrating the interaction of the human population in various classes is depicted in Figure 1.

The transmission model culminates in a nine-dimensional system of ordinary differential equations as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-P)\pi + \sigma R - (\lambda+\mu)S,\\
\frac{dV}{dt} &= P\pi - ((1-\rho)\lambda + \mu)V,\\
\frac{dE}{dt} &= \lambda S + (1-\rho)\lambda V - (\chi_1 + \chi_2 + \theta_1 + \mu)E,\\
\frac{dI_a}{dt} &= \chi_1 E - (\theta_2 + \omega + \mu)I_a,\\
\frac{dI_s}{dt} &= \chi_2 E + \omega I_a - (\theta_3 + \delta_1 + \mu)I_s,\\
\frac{dT_E}{dt} &= \theta_1 E - (\xi_1 + \mu)T_E,\\
\frac{dT_a}{dt} &= \theta_2 I_a - (\xi_2 + \mu)T_a,\\
\frac{dT_s}{dt} &= \theta_3 I_s - (\delta_2 + \xi_3 + \mu)T_s,\\
\frac{dR}{dt} &= \xi_1 T_E + \xi_2 T_a + \xi_3 T_s - (\sigma+\mu)R.
\end{aligned}\right\} \quad (1)$$

We assume that the model parameters are positive, and the initial conditions of system (1) are given as follows: $S(0) \ge 0$, $V(0) \ge 0$, $E(0) \ge 0$, $I_a(0) \ge 0$, $I_s(0) \ge 0$, $T_E(0) \ge 0$, $T_a(0) \ge 0$, $T_s(0) \ge 0$, and $R(0) \ge 0$.

3. Model Analysis

3.1. Invariant Region. The model system (1) deals with the human population, and thus we need to demonstrate that its solutions are bounded for all time $t > 0$.

Theorem 1. *Given the positive initial conditions, the feasible region is defined as follows:*

$$\Omega = \left\{ \left(S(t), V(t), E(t), I_a(t), I_s(t), T_E(t), T_a(t), T_s(t), R(t)\right) \in R_+^9 : N \le \frac{\pi}{\mu} \right\} \quad (2)$$

Proof. The sum of all equations in system (1) represents the total human population in the model and satisfies the following equation:

$$\begin{aligned}
\frac{dN}{dt} &= \pi - \mu N - \delta_1 I_s - \delta_2 T_s \le \pi - \mu N\\
\Longrightarrow \frac{dN}{dt} &\le \pi - \mu N.
\end{aligned} \quad (3)$$

By integrating inequality (3) and applying the initial conditions, we obtain

$$N(t) \le \frac{\pi}{\mu} + \left(N(0) - \frac{\pi}{\mu}\right)e^{-\mu t}. \quad (4)$$

As $t \longrightarrow \infty$ in inequality (3), the population $N(t) \longrightarrow \pi/\mu$, implying that $0 \le N(t) \le \pi/\mu$. Thus, the feasible solution of the system enters and remains in the region:

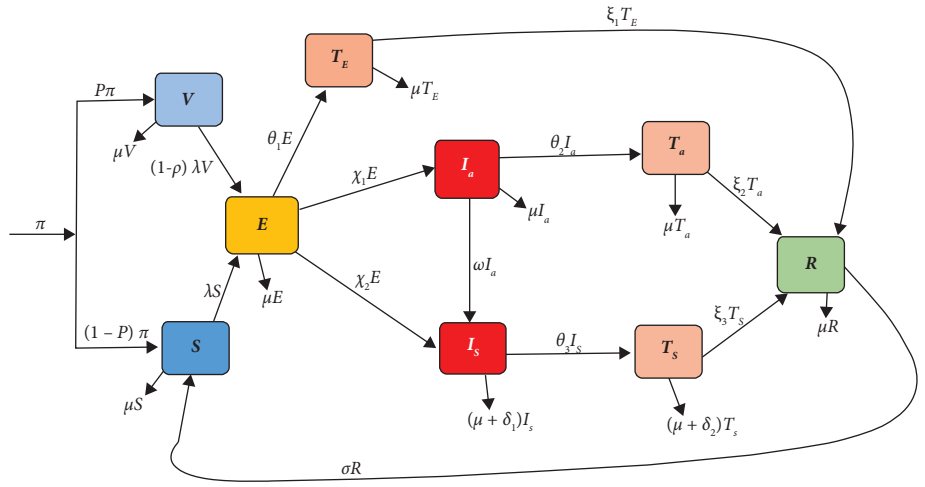

Figure 1: Schematic model diagram for the propagation and control of pulmonary tuberculosis disease in Kenya.

$$\Omega = \left\{ \left(S(t), V(t), E(t), I_a(t), I_s(t), T_E(t), T_a(t), T_s(t), R(t)\right) \in R_+^9 : N \le \frac{\pi}{\mu} \right\}. \tag{5}$$

Therefore, the basic model is well-posed epidemiologically and mathematically, and hence it is sufficient to study its dynamics in Ω. □

3.2. The Basic Reproduction Number and the Control Reproduction Number. The basic reproduction number is a threshold parameter that governs the spread of disease. It is defined as the average number of secondary infections caused by a single infectious individual during his entire infectious period in a population that is entirely susceptible [25].

The control reproduction number is defined as the expected number of secondary infections produced by an index-infected individual in a population that is not entirely susceptible due to the presence of control measures [26].

In the absence of pulmonary tuberculosis, $E = I_a = I_s = T_E = T_a = T_s = R = 0$. Therefore, system (1) has a disease-free equilibrium given by

$$\begin{aligned} B^0 &= \left(S^0, V^0, E^0, I_a^0, I_s^0, T_E^0, T_a^0, T_s^0, R^0\right) \\ &= \left(\frac{(1-P)\pi}{\mu}, \frac{P\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0\right). \end{aligned} \tag{6}$$

We use the next-generation matrix to obtain the control reproduction number as given by [27].

Let $X = (E, I_a, I_S, T_a, T_s)^T$, then it follows from system (1) that

$$\frac{dX}{dt} = f - v, \tag{7}$$

where f and v are matrices representing the new infections and transition terms, respectively, given as follows:

$$\begin{aligned} f &= \begin{bmatrix} \lambda S + (1-\rho)\lambda V \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \\ v &= \begin{bmatrix} (\chi_1 + \chi_2 + \theta_1 + \mu)E \\ (\theta_2 + \omega + \mu)I_a - \chi_1 E \\ (\theta_3 + \delta_1 + \mu)I_S - \chi_2 E - \omega I_a \\ (\xi_2 + \mu)T_a - \theta_2 I_a \\ (\xi_3 + \delta_2 + \mu)T_S - \theta_3 I_S \end{bmatrix}. \end{aligned} \tag{8}$$

The Jacobian matrices of the new infections and transition terms at the disease-free equilibrium are given, respectively, as follows:

$$F = \begin{bmatrix} 0 & \beta\eta_1\left[S^0 + (1-\rho)V^0\right] & \beta\left[S^0 + (1-\rho)V^0\right] & \beta\eta_3\left[S^0 + (1-\rho)V^0\right] & \beta\eta_2\left[S^0 + (1-\rho)V^0\right] \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$
$$V = \begin{bmatrix} \chi_1 + \chi_2 + \theta_1 + \mu & 0 & 0 & 0 & 0 \\ -\chi_1 & \theta_2 + \omega + \mu & 0 & 0 & 0 \\ -\chi_2 & -\omega & \theta_3 + \delta_1 + \mu & 0 & 0 \\ 0 & -\theta_2 & 0 & \xi_2 + \mu & 0 \\ 0 & 0 & -\theta_3 & 0 & \delta_2 + \xi_3 + \mu \end{bmatrix}. \tag{9}$$

The dominant eigenvalue corresponding to the spectral radius $\rho(\mathrm{FV}^{-1})$ of the matrix FV^{-1} is the control reproduction number (R_{cVST}) with vaccination, screening, and treatment of all forms of pulmonary tuberculosis as the intervention strategies. R_{cVST} is given as follows:

$$R_{\mathrm{cVST}} = \frac{\beta\left[\left(\eta_1\chi_1x_3 + \chi_1\omega + \chi_2x_2\right)x_5x_6 + \eta_3\theta_2\chi_1x_3x_6 + \eta_2\theta_3x_5\left(\chi_1\omega + \chi_2x_2\right)\right]\left[(1-P)\pi/\mu + (1-\rho)P\pi/\mu\right]}{x_1x_2x_3x_5x_6}, \tag{10}$$

where

$$\begin{aligned} x_1 &= (\chi_1 + \chi_2 + \theta_1 + \mu), \\ x_2 &= (\theta_2 + \omega + \mu), \\ x_3 &= (\theta_3 + \delta_1 + \mu), \\ x_5 &= (\xi_2 + \mu), \\ x_6 &= (\delta_2 + \xi_3 + \mu). \end{aligned} \tag{11}$$

Without vaccination intervention, the fraction of recruits vaccinated, P, equals zero, and consequently, the parameter, ρ, for vaccine efficacy becomes zero since there will be no vaccinated population. Substituting $P = \rho = 0$ in (10) gives the control reproduction number (R_{cST}) with screening and treatment as the only intervention strategies. R_{cST} is thus given as follows:

$$R_{\mathrm{cST}} = \frac{\beta\left[\left(\eta_1\chi_1x_3 + \chi_1\omega + \chi_2x_2\right)x_5x_6 + \eta_3\theta_2\chi_1x_3x_6 + \eta_2\theta_3x_5\left(\chi_1\omega + \chi_2x_2\right)\right]\pi/\mu}{x_1x_2x_3x_5x_6}. \tag{12}$$

Considering the presence of latent infected and asymptomatic infectious individuals in the population without their screening, the parameters θ_1, θ_2, and ξ_2 become zero. Substituting $\theta_1 = \theta_2 = \xi_2 = 0$ in (10) gives the control reproduction number (R_{cVT_s}) with vaccination and treatment of the symptomatic infectious population as the only intervention strategies. R_{cVT_s} is thus given as follows:

$$R_{\mathrm{cVT}_s} = \frac{\beta\left[\left[\eta_1\chi_1\left(\theta_3 + \delta_1 + \mu\right) + \chi_1\omega + \chi_2\left(\mu + \omega\right)\right]\left(\xi_3 + \delta_2 + \mu\right) + \eta_2\theta_3\left[\chi_1\omega + \chi_2\left(\mu + \omega\right)\right]\right]\left[(1-P)\pi/\mu + (1-\rho)P\pi/\mu\right]}{\left(\chi_1 + \chi_2 + \mu\right)\left(\omega + \mu\right)\left(\theta_3 + \delta_1 + \mu\right)\left(\delta_2 + \xi_3 + \mu\right)}. \tag{13}$$

When there is no vaccination of recruits and screening of both latent infected and asymptomatic infectious populations, the parameters θ_1, θ_2, ξ_2, P, and ρ become zero. Substituting $\theta_1 = \theta_2 = \xi_2 = \mathrm{P} = \rho = 0$ in (10) gives the control reproduction number (R_{cT_s}) with the treatment of the symptomatic infectious population as the only intervention strategy. R_{cT_s} is thus given as follows:

$$R_{\mathrm{cT}_s} = \frac{\beta\left[\left[\eta_1\chi_1\left(\theta_3+\delta_1+\mu\right)+\chi_1\omega+\chi_2\left(\mu+\omega\right)\right]\left(\xi_3+\delta_2+\mu\right)+\eta_2\theta_3\left[\chi_1\omega+\chi_2\left(\mu+\omega\right)\right]\right]\pi/\mu}{\left(\chi_1+\chi_2+\mu\right)\left(\omega+\mu\right)\left(\theta_3+\delta_1+\mu\right)\left(\delta_2+\xi_3+\mu\right)}. \tag{14}$$

Considering no intervention measures in place, that is, when there is no vaccination of the recruits, screening of both latent and asymptomatic infectious populations, and treatment of all forms of pulmonary tuberculosis, the parameters P, ρ, θ_1, θ_2, θ_3, ξ_2, ξ_3, and δ_2 become zero. Substituting $P = \rho = \theta_1 = \theta_2 = \theta_3 = \xi_2 = \xi_3 = \delta_2 = 0$ in (10) gives the basic reproduction number (R_o) given as follows:

$$R_o = \frac{\beta\left[\eta_1\chi_1\left(\delta_1+\mu\right)+\chi_1\omega+\chi_2\left(\mu+\omega\right)\right]\pi/\mu}{\left(\chi_1+\chi_3+\mu\right)\left(\omega+\mu\right)\left(\delta_1+\mu\right)}. \tag{15}$$

3.3. Local Stability of Disease-Free Equilibrium

Theorem 2. *The disease-free equilibrium point is locally asymptotically stable if* $R_{\mathrm{cVST}} < 1$ *and unstable if* $R_{\mathrm{cVST}} > 1$.

Proof. To prove the local stability of the disease-free equilibrium, we obtain the Jacobian matrix of the system at the disease-free equilibrium as follows:

$$J\left(B^0\right) = \begin{bmatrix} -\mu & 0 & 0 & -\beta\eta_1 S^0 & -\beta S^0 & 0 & -\beta\eta_3 S^0 & -\beta\eta_2 S^0 & \sigma \\ 0 & -\mu & 0 & -H_1 & -H_2 & 0 & -H_3 & -H_4 & 0 \\ 0 & 0 & -x_1 & H_5 & H_6 & 0 & H_7 & H_8 & 0 \\ 0 & 0 & \chi_1 & -x_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \chi_2 & \omega & -x_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta_1 & 0 & 0 & -x_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta_2 & 0 & 0 & -x_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta_3 & 0 & 0 & -x_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi_1 & \xi_2 & \xi_3 & -x_7 \end{bmatrix}, \tag{16}$$

where

$$
\begin{aligned}
x_1 &= (\chi_1 + \chi_2 + \theta_1 + \mu),\\
x_2 &= (\theta_2 + \omega + \mu),\\
x_3 &= (\theta_3 + \delta_1 + \mu),\\
x_4 &= (\xi_1 + \mu),\\
x_5 &= (\xi_2 + \mu),\\
x_6 &= (\delta_2 + \xi_3 + \mu),\\
x_7 &= (\sigma + \mu),\\
H_1 &= (1-\rho)\beta\eta_1 V^0,\\
H_2 &= (1-\rho)\beta V^0,\\
H_3 &= (1-\rho)\beta\eta_3 V^0,\\
H_4 &= (1-\rho)\beta\eta_2 V^0,\\
H_5 &= \beta\eta_1\left[S^0 + (1-\rho)V^0\right],\\
H_6 &= \beta\left[S^0 + (1-\rho)V^0\right],\\
H_7 &= \beta\eta_3\left[S^0 + (1-\rho)V^0\right],\\
H_8 &= \beta\eta_2\left[S^0 + (1-\rho)V^0\right].
\end{aligned}
\tag{17}
$$

Evaluating the eigenvalues of (16), we obtain

$$
\{-(\mu+\lambda)\}\{-(\mu+\lambda)\}\{-(x_7+\lambda)\}
\begin{vmatrix}
-x_1-\lambda & H_5 & H_6 & 0 & H_7 & H_8\\
\chi_1 & -x_2-\lambda & 0 & 0 & 0 & 0\\
\chi_2 & \omega & -x_3-\lambda & 0 & 0 & 0\\
\theta_1 & 0 & 0 & -x_4-\lambda & 0 & 0\\
0 & \theta_2 & 0 & 0 & -x_5-\lambda & 0\\
0 & 0 & \theta_3 & 0 & 0 & -x_6-\lambda
\end{vmatrix} = 0. \tag{18}
$$

Either

$$
\{-(\mu+\lambda)\}\{-(\mu+\lambda)\}\{-(x_7+\lambda)\} = 0, \tag{19}
$$

or

$$
\begin{vmatrix}
-x_1-\lambda & H_5 & H_6 & 0 & H_7 & H_8\\
\chi_1 & -x_2-\lambda & 0 & 0 & 0 & 0\\
\chi_2 & \omega & -x_3-\lambda & 0 & 0 & 0\\
\theta_1 & 0 & 0 & -x_4-\lambda & 0 & 0\\
0 & \theta_2 & 0 & 0 & -x_5-\lambda & 0\\
0 & 0 & \theta_3 & 0 & 0 & -x_6-\lambda
\end{vmatrix} = 0. \tag{20}
$$

Simplifying (19), we have

$$(\mu+\lambda)(\mu+\lambda)(x_7+\lambda)=0. \tag{21}$$

By the Routh–Hurwitz criteria, (21) has strictly negative roots given as follows:

$$\begin{aligned}\lambda_1&=\lambda_2\\&=-\mu<0,\\\lambda_3&=-x_7<0.\end{aligned} \tag{22}$$

The characteristic polynomial of (20) is obtained as follows:

$$M_1\lambda^6+M_2\lambda^5+M_3\lambda^4+M_4\lambda^3+M_5\lambda^2+M_6\lambda+M_7=0, \tag{23}$$

where $M_1, M_2, M_3, M_4, M_5, M_6$, and M_7 are determined using Mathematica software as follows:

$$\begin{aligned}
M_1&=1>0,\\
M_2&=x_1+x_2+x_3+x_4+x_5+x_6,\\
M_3&=x_6(x_1+x_2+x_3+x_4+x_5)+x_5(x_1+x_2+x_4)+x_4(x_1+x_2+x_3)+x_3(x_1+x_2)+x_1x_2+H_5\chi_1-H_6\chi_2,\\
M_4&=x_6[x_5(x_1+x_2+x_3+x_4)+x_4(x_1+x_2+x_3)+x_3(x_1+x_2)+x_1x_2-(H_5\chi_1+H_6\chi_2)]\\
&\quad+x_5[x_4(x_1+x_2+x_3)+x_3(x_1+x_2)+x_1x_2-(H_5\chi_1+H_6\chi_2)]+x_4[x_3(x_1+x_2)+x_1x_2-(H_5\chi_1+H_5\chi_2)]\\
&\quad+x_3(x_1x_2-H_5\chi_1)-x_2H_6\chi_2-\omega H_6\chi_1-\theta_2H_7\chi_1-\theta_3H_8\chi_2,\\
M_5&=x_6[x_4x_5(x_1+x_2+x_3)+x_3x_5(x_1+x_2)+x_2x_5x_1+x_3x_4(x_1+x_2)+x_1x_2(x_3+x_4)-H_5\chi_1(x_3+x_4+x_5)\\
&\quad-H_6\omega\chi_1-H_6\chi_2(x_2+x_4+x_5)-H_7\theta_2\chi_1]+x_5[x_1x_2(x_3+x_4)+x_3x_4(x_1+x_2)-H_5\chi_1(x_3+x_4)-H_6\omega\chi_1\\
&\quad-H_6\chi_2(x_2+x_4)-H_8\theta_3\chi_2]+x_4[x_3x_2x_1-H_5\chi_1x_3-H_6\omega\chi_1-H_6\chi_2x_2-H_7\theta_2\chi_1-H_8\theta_3\chi_2]\\
&\quad-H_7\theta_2\chi_1x_3-H_8\theta_3\chi_2x_2-H_8\theta_3\omega\chi_1,\\
M_6&=x_6[x_1x_2(x_3x_4+x_3x_5+x_4x_5)+x_3x_4(x_1x_5+x_2x_5)-H_5\chi_1(x_3x_4+x_3x_5+x_4x_5)-H_6\omega\chi_1(x_4+x_5)\\
&\quad-H_6\chi_2(x_2x_4+x_2x_5+x_4x_5)-H_7\theta_2\chi_1(x_3+x_4)]+x_5[x_1x_2x_3x_4-H_5\chi_1x_3x_4-H_6\chi_2x_4x_5\\
&\quad-H_6\omega\chi_1x_4-H_8\theta_3\chi_2(x_2+x_4)-H_8\omega\theta_3\chi_1]+x_4[-H_7\theta_2\chi_1x_3-H_8\theta_3(x_2\chi_2+\chi_1\omega)]\\
M_7&=x_1x_2x_3x_4x_5x_6-x_4[x_5x_6[x_3H_5\chi_1+H_6(\chi_2x_2+\chi_1\omega)]+H_7\chi_1\theta_2x_3x_6+H_8\theta_3x_5(\chi_2x_2+\chi_1\omega)].
\end{aligned} \tag{24}$$

By the Routh–Hurwitz criteria,

$$M_1>0, M_3>0,\ M_5>0 \text{ and } M_7>0. \tag{25}$$

From $M_7>0$, we have

$$\begin{aligned}
&x_1x_2x_3x_4x_5x_6-x_4[x_5x_6[x_3H_5\chi_1+H_6(\chi_2x_2+\chi_1\omega)]+H_7\chi_1\theta_2x_3x_6+H_8\theta_3x_5(\chi_2x_2+\chi_1\omega)]>0\\
&\Longrightarrow[x_5x_6[x_3H_5\chi_1+H_6(\chi_2x_2+\chi_1\omega)]+H_7\chi_1\theta_2x_3x_6+H_8\theta_3x_5(\chi_2x_2+\chi_1\omega)]<x_1x_2x_3x_5x_6\\
&\Longrightarrow\frac{[x_5x_6[x_3H_5\chi_1+H_6(\chi_2x_2+\chi_1\omega)]+H_7\chi_1\theta_2x_3x_6+H_8\theta_3x_5(\chi_2x_2+\chi_1\omega)]}{x_1x_2x_3x_5x_6}<1.
\end{aligned} \tag{26}$$

Substituting $H_5 = \beta\eta_1[S^0 + (1-\rho)V^0]$, $H_6 = \beta[S^0 + (1-\rho)V^0]$, $H_7 = \beta\eta_3[S^0 + (1-\rho)V^0]$, $H_8 = \beta\eta_2[S^0 + (1-\rho)V^0]$, $S^0 = (1-P)\pi/\mu$, and $V^0 = P\pi/\mu$ in inequality (26) and rearranging yield

$$\frac{\beta\left[\left(\eta_1\chi_1x_3 + \chi_1\omega + \chi_2x_2\right)x_5x_6 + \eta_3\theta_2\chi_1x_3x_6 + \eta_2\theta_3x_5\left(\chi_1\omega + \chi_2x_2\right)\right]\left[(1-P)\pi/\mu + (1-\rho)P\pi/\mu\right]}{x_1x_2x_3x_5x_6} < 1. \tag{27}$$

However,

$$\frac{\beta\left[\left(\eta_1\chi_1x_3 + \chi_1\omega + \chi_2x_2\right)x_5x_6 + \eta_3\theta_2\chi_1x_3x_6 + \eta_2\theta_3x_5\left(\chi_1\omega + \chi_2x_2\right)\right]\left[(1-P)\pi/\mu + (1-\rho)P\pi/\mu\right]}{x_1x_2x_3x_5x_6} = R_{\mathrm{cVST}}. \tag{28}$$

Therefore, (27) can be expressed as follows:

$$R_{\mathrm{cVST}} < 1. \tag{29}$$

From (29), the disease-free equilibrium is locally asymptotically stable if $R_{\mathrm{cVST}} < 1$. This implies that each infectious individual infects, on average, less than one susceptible person during the infectious period, resulting in the disease dying out [27]. □

3.4. The Endemic Equilibrium. The endemic equilibrium $B^* = (S^*, V^*, E^*, I_a^*, I_s^*, T_E^*, T_a^*, T_s^*R^*)$ is evaluated by equating the model system of (1) to zero. The steady-state solution for the model equations is as follows:

$$\begin{aligned}
S^* &= \frac{(1-P)\pi + \sigma R^*}{\lambda^{**} + \mu},\\
V^* &= \frac{P\pi}{(1-\rho)\lambda^{**} + \mu},\\
E^* &= \frac{\lambda^{**}(1-P)\pi + \lambda^{**}\sigma R^* + (1-\rho)\lambda^{**}P\pi}{\left(\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)},\\
I_a^* &= \frac{\chi_1\left[\lambda^{**}(1-P)\pi + \lambda^{**}\sigma R^* + (1-\rho)\lambda^{**}P\pi\right]}{\left(\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)\left(\theta_2 + \omega + \mu\right)},\\
I_s^* &= \frac{\left[\chi_2\left(\theta_2 + \omega + \mu\right) + \omega\chi_1\right]\left[\lambda^{**}(1-P)\pi + \lambda^{**}\sigma R^* + (1-\rho)\lambda^{**}P\pi\right]}{\left(\theta_3 + \delta_1 + \mu\right)\left(\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)\left(\theta_2 + \omega + \mu\right)},\\
T_E^* &= \frac{\theta_1\left[\lambda^{**}(1-P)\pi + \lambda^{**}\sigma R^* + (1-\rho)\lambda^{**}P\pi\right]}{\left(\xi_1 + \mu\right)\left(\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)},\\
T_a^* &= \frac{\theta_2\chi_1\left[\lambda^{**}(1-P)\pi + \lambda^{**}\sigma R^* + (1-\rho)\lambda^{**}P\pi\right]}{\left(\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)\left(\theta_2 + \omega + \mu\right)\left(\xi_2 + \mu\right)},\\
T_s^* &= \frac{\left[\chi_2\left(\theta_2 + \omega + \mu\right) + \omega\chi_1\right]\theta_3\left[\lambda^{**}(1-P)\pi + \lambda^{**}\sigma R^* + (1-\rho)\lambda^{**}P\pi\right]}{\left(\theta_3 + \delta_1 + \mu\right)\left(\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)\left(\theta_2 + \omega + \mu\right)\left(\delta_2 + \xi_3 + \mu\right)},\\
R^* &= \frac{\left[\lambda^{**}S^* + (1-\rho)\lambda^{**}P\pi\right]\left[D_1 + D_2 + D_3\right]}{\left(\lambda^{**} + \mu\right)\left((1-\rho)\lambda^{**} + \mu\right)\left(\chi_1 + \chi_2 + \theta_1 + \mu\right)\left(\theta_2 + \omega + \mu\right)\left(\theta_3 + \delta_1 + \mu\right)A_1A_2A_3A_4},
\end{aligned} \tag{30}$$

where

$$
\begin{aligned}
D_1 &= \xi_1\theta_1(\theta_3+\delta_1+\mu)(\theta_2+\omega+\mu)(\delta_2+\xi_3+\mu)(\xi_2+\mu),\\
D_2 &= \xi_2\theta_2\chi_1(\theta_3+\delta_1+\mu)(\delta_2+\xi_3+\mu)(\xi_1+\mu),\\
D_3 &= \xi_3\theta_3[\chi_2(\theta_2+\omega+\mu)+\omega\chi_1](\xi_1+\mu)(\xi_2+\mu),\\
A_1 &= (\xi_1+\mu),\\
A_2 &= (\xi_2+\mu),\\
A_3 &= (\delta_2+\xi_3+\mu),\\
A_4 &= (\sigma+\mu).
\end{aligned}
\tag{31}
$$

3.4.1. Global Stability of Endemic Equilibrium Point. We consider the method of the Lyapunov function to prove the global stability of the endemic equilibrium point. We propose a logarithmic Lyapunov function L defined by

$$
\begin{aligned}
L(S^*,V^*,E^*,I_a^*,I_s^*,T_E^*,T_a^*,T_s^*,R^*) &= \left(S-S^*+S^*\ln\frac{S^*}{S}\right)+\left(V-V^*+V^*\ln\frac{V^*}{V}\right)+\left(E-E^*+E^*\ln\frac{E^*}{E}\right)\\
&+\left(I_a-I_a^*+I_a^*\ln\frac{I_a^*}{I_a}\right)+\left(I_s-I_s^*+I_s^*\ln\frac{I_s^*}{I_s}\right)+\left(T_E-T_E^*+T_E^*\ln\frac{T_E^*}{T_E}\right)\\
&+\left(T_a-T_a^*+T_a^*\ln\frac{T_a^*}{T_a}\right)+\left(T_s-T_s^*+T_s^*\ln\frac{T_s^*}{T_s}\right)+\left(R-R^*+R^*\ln\frac{R^*}{R}\right).
\end{aligned}
\tag{32}
$$

The derivatives of L along the solution of the model system (1) give

$$
\begin{aligned}
\frac{\mathrm{d}L}{\mathrm{d}t} &= \left(\frac{S-S^*}{S}\right)\frac{dS}{dt}+\left(\frac{V-V^*}{V}\right)\frac{dV}{dt}+\left(\frac{E-E^*}{E}\right)\frac{dE}{dt}+\left(\frac{I_a-I_a^*}{I_a}\right)\frac{dI_a}{dt}+\left(\frac{I_s-I_s^*}{I_s}\right)\frac{dI_s}{dt}\\
&+\left(\frac{T_E-T_E^*}{T_E}\right)\frac{dT_E}{\mathrm{dt}}+\left(\frac{T_a-T_a^*}{T_a}\right)\frac{dT_a}{\mathrm{dt}}+\left(\frac{T_s-T_s^*}{T_s}\right)\frac{dT_s}{\mathrm{dt}}+\left(\frac{R-R^*}{R}\right)\frac{\mathrm{dR}}{\mathrm{dt}}.
\end{aligned}
\tag{33}
$$

Substituting the expressions dS/dt, dV/dt, dE/dt, dI_a/dt, dI_S/dt, dT_E/dt, dT_a/dt, dT_S/dt, and dR/dt from the model system (1) into (33) and simplifying give

$$
\frac{\mathrm{dL}}{\mathrm{dt}} = Q-W,
\tag{34}
$$

where

$$
\begin{aligned}
Q &= \pi+\sigma R+\frac{\sigma R^*S^*}{S}+\frac{\lambda S^*E^*}{E}(1-\rho)\lambda V+\frac{(1-\rho)\lambda V^*E^*}{E}+\chi_1E+\frac{\chi_1I_a^*E^*}{I_a}+\chi_2E\\
&+\frac{\chi_2I_s^*E^*}{I_s}+\omega I_a+\frac{\omega I_s^*I_a^*}{I_s}+\theta_1E+\frac{\theta_1T_E^*E^*}{T_E}+\theta_2I_a+\frac{\theta_2I_a^*T_a^*}{T_a}+\theta_3T_s+\frac{\theta_3I_s^*T_s^*}{T_s}\\
&+\xi_1T_E+\frac{\xi_1T_E^*R^*}{R}+\xi_2T_a+\frac{\xi_2T_a^*R^*}{R}+\xi_3T_s+\frac{\xi_3T_s^*R^*}{R}+\frac{\pi PV^*}{V}+\lambda S,\\
W &= \frac{\pi S^*}{S}+\frac{\pi PS^*}{S}+\frac{\sigma RS^*}{S}+\frac{\lambda SE^*}{E}+\frac{\pi PV^*}{V}+(1-\rho)\lambda V^*+\frac{(1-\rho)\lambda E^*}{E^*}+\chi_1E^*+\frac{\chi_1EI_a^*}{I_a}+\chi_2E^*
\end{aligned}
$$

$$
\begin{aligned}
&+\chi_2 E I_s^* + \omega I_a^* + \frac{\omega I_a I_s^*}{I_s} + \theta_1 E^* + \frac{\theta_1 E T_E^*}{T_E} + \theta_2 I_a^* + \frac{\theta_2 I_a T_a^*}{T_a} + \theta_3 I_s^* + \frac{\theta_3 I_s T_s^*}{T_s} + \xi_1 T_E^* \\
&+\frac{\xi_1 T_E R^*}{R} + \xi_2 T_a^* + \frac{\xi_2 T_a R^*}{R} + \xi_3 T_s^* + \frac{\xi_2 T_s R^*}{R} + \lambda S^* \\
&+\frac{(S-S^*)^2}{S}[\lambda+\mu] + \frac{(V-V^*)^2}{V}[(1-\rho)\lambda+\mu] + \frac{(E-E^*)^2}{E}[\chi_1+\chi_2+\theta_1+\mu] \\
&+\frac{(I_a-I_a^*)^2}{I_a}[\theta_2+\omega+\mu] + \frac{(I_s-I_s^*)^2}{I_s}[\theta_3+\delta_1+\mu] \\
&+\frac{(T_E-T_E^*)^2}{T_E}[\xi_1+\mu] + \frac{(T_a-T_a^*)}{T_a}[\xi_2+\mu] + \frac{(T_s-T_s^*)^2}{T_s}[\delta_2+\xi_3+\mu] + \frac{(R-R^*)^2}{R}[\sigma+\mu].
\end{aligned}
\tag{35}
$$

If $Q \leq W$, then $dL/dt \leq 0$ and $dL/dt = 0$ if and only if $S = S^*, V = V^*, E = E^*, I_a = I_a^*, I_s = I_s^*, T_E = T_E^*, T_a = T_a^*, T_s = T_s^*, R = R^*$.

Therefore, the largest compact invariant set in $\{(S^*, V^*, E^*, I_a^*, I_s^*, T_E^*, T_a^*, T_s^* R^*) \in \Omega : dL/dt = 0\}$ is the singleton endemic equilibrium point B^*. Thus, from LaSalle's invariance principle [28], we conclude that as $t \longrightarrow \infty$, the solution of the model system (1) approaches the endemic equilibrium B^* when the control reproduction number $R^*_{cVST} > 1$. Therefore, the endemic equilibrium point B^* is globally asymptotically stable in the invariant set Ω if $Q < W$.

3.5. Sensitivity Analysis on Control Reproduction Numbers. In this section, we present a sensitivity analysis of the reproduction numbers to determine the relative importance of the various parameters responsible for pulmonary tuberculosis transmission and prevalence in the population. We employed the normalized forward sensitivity index for this model as used by [29]. The normalized sensitivity index which measures the relative change in a parameter K with respect to the reproduction number R_c is given by

$$
\Lambda_K^{R_c} = \frac{\partial R_c}{\partial K} \times \frac{K}{R_c}. \tag{36}
$$

The parameter values in Table 1 are used to calculate the sensitivity indices of the reproduction numbers for the parameters β, P, χ_1, χ_2, θ_1, θ_2, and θ_3.

The calculated sensitivity indices of reproduction numbers are given in Table 2. A positive sensitivity index shows that the reproduction number is an increasing function of the corresponding parameter whereas a negative sensitivity index shows that the reproduction number is a decreasing function of the corresponding parameter. Thus, increasing a parameter with a positive sensitivity index holding other parameters constant increases the reproduction number whereas increasing a parameter with a negative sensitivity index while other factors are held constant decreases the reproduction number [37].

From Table 2, it is observed that β, χ_1, and χ_2 have positive sensitivity index values, and thus an increase in these parameters results in a corresponding increase in the number of the infected population. On the other hand, θ_1, θ_2, θ_3, and P have negative sensitivity index values, and thus an increase in these parameters results in a decrease in the number of infected populations. For instance, if the transmission rate, β, is increased by 10%, the reproduction numbers increase by 10%. On the other hand, increasing screening rate of latent infected, θ_1, by 10% decreases R_{cVST} by 6.673668% while increasing screening rate of asymptomatic infectious, θ_2, by 10% decreases R_{cVST} by 0.751639%.

4. Numerical Simulations of the Model

Numerical simulations of the model system (1) have been done to explore pulmonary tuberculosis epidemic behavior. The simulations were carried out in MATLAB ordinary differential equations inbuilt solver, ode45 function. Numerical simulations have been performed using data from the Kenyan population. The baseline parameters used for numerical simulations are given in Table 1. The initial values states are gives as $S(0) = 1182969$, $V(0) = 30920683$, $E(0) = 17923767$, $I_a(0) = 127595$, $I_s(0) = 200000$, $T_E(0) = 500000$, $T_a(0) = 50000$, and $T_s(0) = 113155$, $R(0) = 2753131$ as obtained from published Kenyan data [30–34]. The simulation results are presented graphically as shown in Figures 2–7.

4.1. Effects of Various Intervention Strategies on the Control Reproduction Number. Equations (10), (12)–(14) represent the control reproduction numbers with various intervention strategies: R_{cVST} for vaccination, screening, and treatment of all infected cases; R_{cST} for screening and treatment of all infected cases; R_{cVT_S} for vaccination and treatment of symptomatic infectious population; and R_{cT_S} for treatment of symptomatic infectious population alone. Figure 2 illustrates the effects of varying reproduction numbers with respect to transmission rate, β. It is observed that $R_{cVST} < R_{cST} < R_{cVT_S} < R_{cT_S}$. This indicates that a combination of vaccination, screening, and treatment of all infected cases is the most effective control measure in reducing infection transmission in the population. Following this, a combination of screening and treatment of all infected cases proves effective, while the combination of vaccination

Table 1: Baseline parameters used in simulation.

Parameter	Description	Value	Reference
η_3	Transmission coefficient for asymptomatic individuals undergoing treatment	0.00000126	Estimated
η_2	Transmission coefficient for symptomatic individuals undergoing treatment	0.00002	Estimated
η_1	Transmission coefficient for the asymptomatic infectious individuals	0.0003	Estimated
σ	The rate at which the immunity of recovered wanes	0.003 year^{-1}	[30]
μ	Natural death rate	0.0147 year^{-1}	[31]
π	Recruitment rate	0.021 year^{-1}	[32]
χ_1	Progression rate of latent infected to asymptomatic individuals	0.05 year^{-1}	[33]
δ_2	Rate of tuberculosis disease-induced deaths during treatment	0.065 year^{-1}	[30]
χ_2	Progression rate of latent infected to infectious individuals with disease symptoms	0.1 year^{-1}	[33]
β	Transmission rate of pulmonary tuberculosis	0.15 year^{-1}	[30]
θ_1	Rate of screening latently infected individuals	0.34 year^{-1}	[34]
ρ	Vaccine efficacy	0.5	[11]
δ_1	Rate of tuberculosis disease-induced deaths	0.5 year^{-1}	[34]
θ_2	Rate of screening asymptomatic infectious individuals	0.5 year^{-1}	[35]
ω	The rate at which asymptomatic infectious individuals exhibit symptoms of the disease	0.6 year^{-1}	[35]
θ_3	The rate at which symptomatic individuals are treated	0.68 year^{-1}	[30]
ξ_3	The recovery rate of treated symptomatic individuals	0.75 year^{-1}	[32]
P	Vaccination rate	0.8 year^{-1}	[31]
ξ_2	The recovery rate of treated asymptomatic infectious individuals	0.8 year^{-1}	[30]
ξ_1	The recovery rate of latent treated	0.85 year^{-1}	[36]

Table 2: Sensitivity indices of reproduction numbers with respect to some model parameters.

Parameter	Sensitivity index
Sensitivity indices of R_{cVST}	
θ_2	−0.751639
θ_1	−0.673668
P	−0.666667
θ_3	−0.5690921
χ_1	+0.11309
χ_2	+0.589703
β	+1
Sensitivity indices of R_{cST}	
θ_2	−0.751362
θ_1	−0.673668
θ_3	−0.0519066
χ_1	+0.113028
χ_2	+0.589756
β	+1
Sensitivity indices of R_{cVT_s}	
P	−0.6666667
θ_3	−0.0519052
χ_1	+0.024444
χ_2	+0.0648092
β	+1
Sensitivity indices of R_{cT_s}	
θ_3	−0.569053
χ_1	+0.0245257
χ_2	+0.0647274
β	+1

and treatment of symptomatic infectious ranks as the third appropriate strategy. Furthermore, it is noted that treating symptomatic cases alone is the least effective strategy in reducing infection transmission in the population.

4.2. Effects of Varying Screening and Treatment Rates on the Control Reproduction Number. In the model flowchart depicted in Figure 1, θ_1 represents the rate of screening latent infected individuals, with Figure 3 illustrating the effects of screening the latent infected population on the control reproduction number. Similarly, according to the model flowchart shown in Figure 1, θ_2 represents the rate of screening the asymptomatic infectious population, with Figure 4 displaying the effects of screening this population on the control reproduction number. It is observed that increasing screening rates for both latent infected and asymptomatic infectious populations reduce the control reproduction number, consequently decreasing the rate of infection transmission. Screening and treating latent infections reduce reactivation, subsequently decreasing infection transmission in the population. The asymptomatic infectious population, experiencing no symptoms, continues their daily routines without seeking medical intervention. This behavior leads to more interactions with susceptible individuals, contributing to a high rate of infection transmission in the population. Therefore, it is prudent to screen and treat asymptomatic infectious individuals since they spread infections incessantly without being noticed.

In the model flowchart depicted in Figure 1, θ_3 represents the rate of treating the symptomatic infectious population, while Figure 5 shows the effects of varying treatment on the control reproduction number for this population. It is observed that increasing the treatment rate of the symptomatic infectious population reduces the control reproduction number. This reduction is attributed to the decrease in the population that is infectious, consequently reducing the rate of infection transmission in the population.

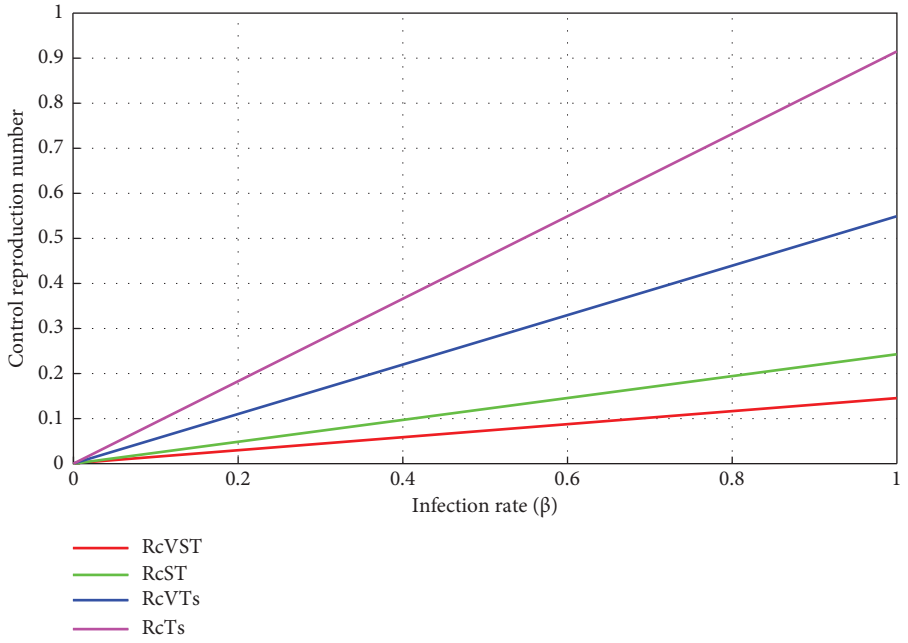

Figure 2: Effects of various intervention strategies on the control reproduction number.

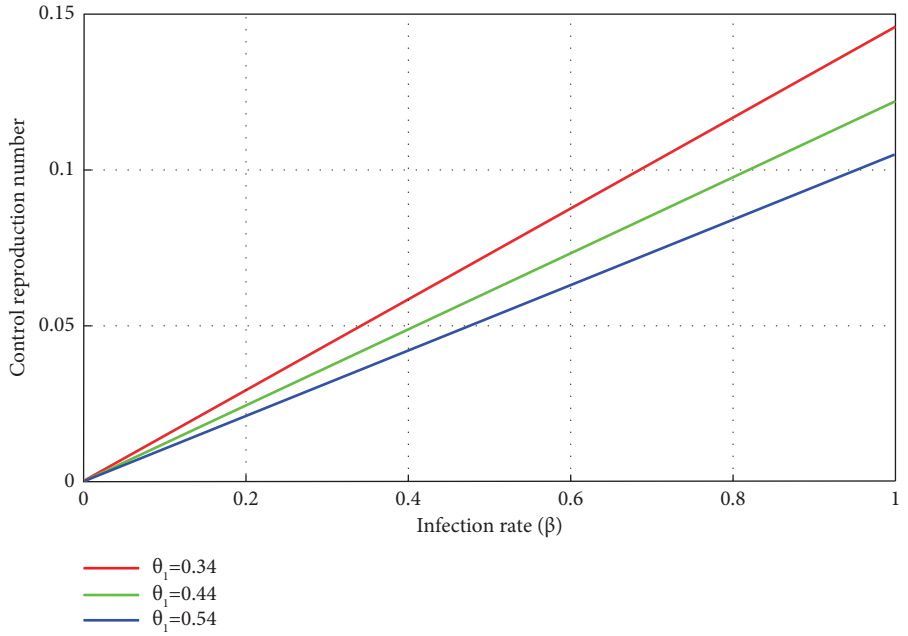

Figure 3: Effects of varying the screening rate for latent infected individuals on the control reproduction number.

4.3. Effects of Asymptomatic Infectious Screening on the Symptomatic Population. Figure 6 illustrates the effects of varying the screening rate for asymptomatic infectious individuals on the symptomatic population. It is observed that an increase in screening for asymptomatic infectious individuals significantly reduces the symptomatic population. This reduction is attributed to the decreased progression of the asymptomatic infectious population to severe pulmonary tuberculosis disease. Asymptomatic infectious individuals often delay seeking healthcare and are not promptly identified for pulmonary tuberculosis testing. However, their detection is crucial for diagnosing the less advanced form of pulmonary tuberculosis and facilitating early treatment. Ultimately, this contributes to the reduction of disease transmission, decreased case fatality, and prevention of adverse consequences of the disease.

4.4. Effects of Screening Latent Infected on the Asymptomatic Infectious Population. Figure 7 illustrates the effects of screening latent infected individuals on the asymptomatic

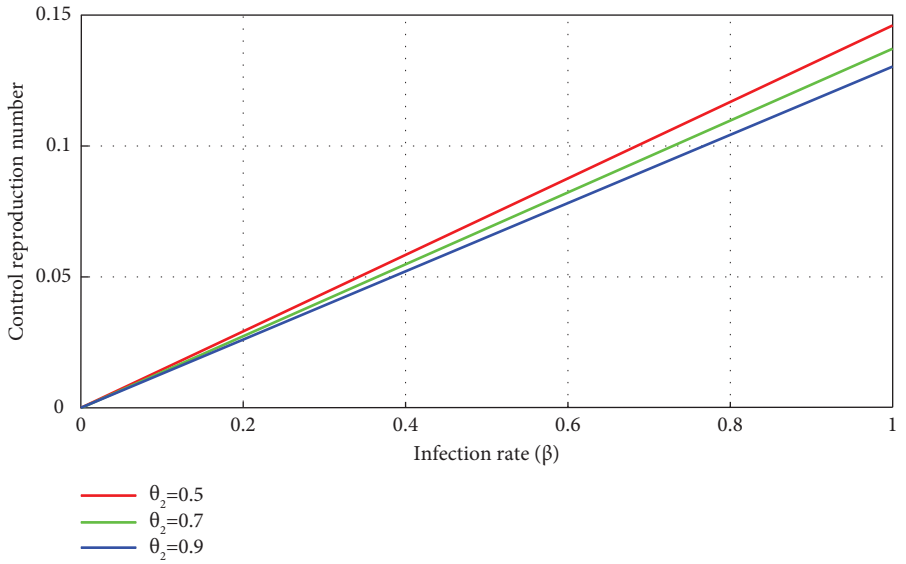

Figure 4: Effects of varying the screening rate for asymptomatic infectious individuals on the control reproduction number.

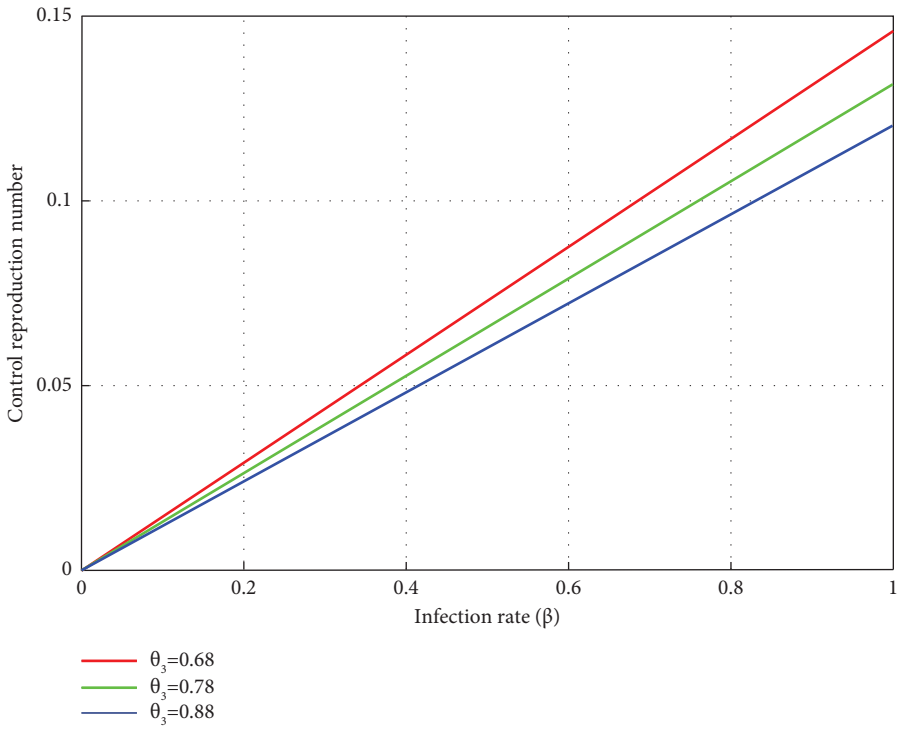

Figure 5: Effects of varying the treatment rate for symptomatic infectious individuals on the control reproduction number.

infectious population. It is observed that an increase in screening of latent infected individuals results in a decrease in the asymptomatic infectious population. This reduction is due to decreased reactivation of latent infections as a result of treatment. The decrease in the asymptomatic infectious population reduces the incessant spread of infections to susceptible populations. Therefore, screening the latent infected population proves to be an effective strategy for reducing the transmission of infections in the population.

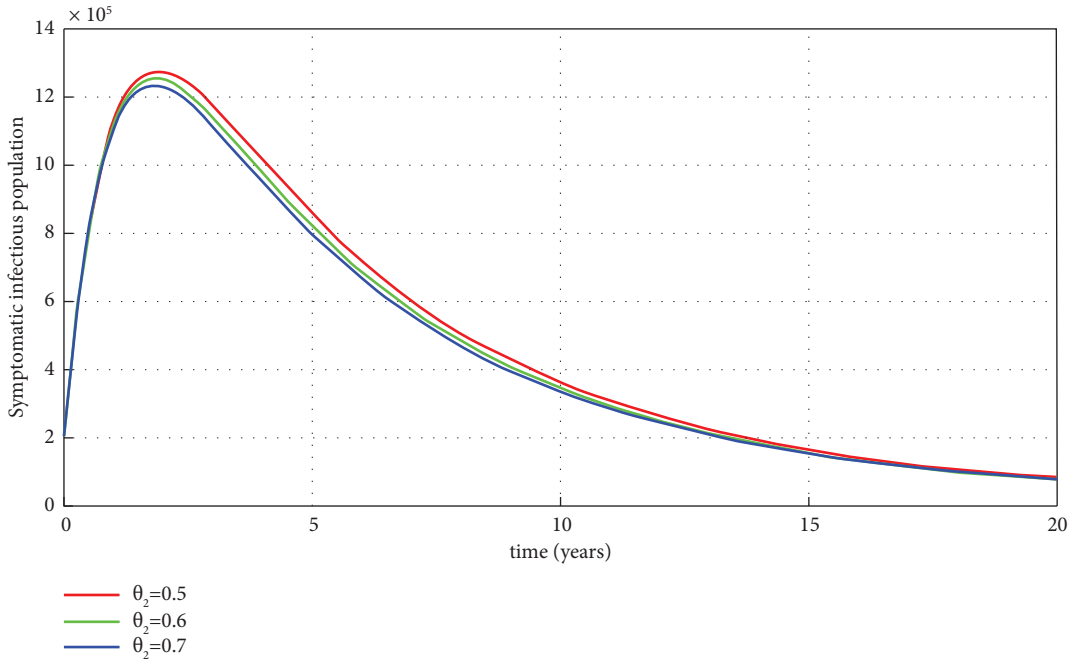

Figure 6: Effects of varying the screening rate for asymptomatic individuals on the symptomatic infectious population.

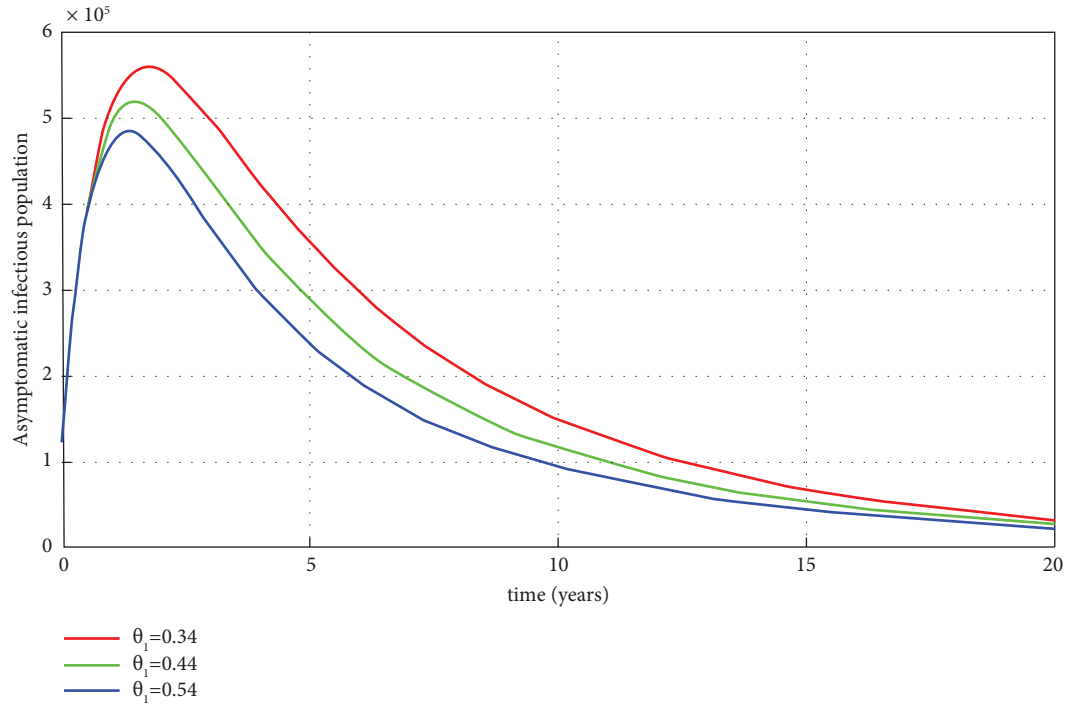

Figure 7: Effects of varying the screening rate for latent infected individuals on the asymptomatic infectious population.

5. Conclusion

In this paper, a mathematical model of pulmonary tuberculosis incorporating an asymptomatic infectious population has been formulated. The asymptomatic infectious population was targeted since they spread infections incessantly to susceptible populations without being noticed, thus contributing to a high rate of infection transmission. This was based on research findings from a survey conducted in Kenya in 2016, which showed that 26% of active

pulmonary tuberculosis cases were asymptomatic infectious and did not seek medical care. The model considered intervention strategies that include vaccination, screening of both latent infected and asymptomatic infectious populations, and treatment of all forms of pulmonary tuberculosis disease. Qualitative as well as numerical analyses have been performed to determine effective intervention strategies that reduce the transmission of pulmonary tuberculosis infections in the population. However, the limitations of this model, which will be considered in our next research paper, include the impact of natural immunity on the progression of individuals from latent infection to pulmonary tuberculosis disease and the waning of vaccine efficacy.

Qualitative and numerical results demonstrate that increasing the screening of asymptomatic and latently infected individuals reduces the transmission of infections to the susceptible population. The numerical analysis indicates that the combination of vaccination, screening, and treatment of all forms of pulmonary tuberculosis disease is the most effective intervention in decreasing disease transmission. Furthermore, the results suggest that a combination of screening and treatment of all forms of pulmonary tuberculosis disease is more effective than a combination of vaccination and treatment of symptomatic infectious individuals alone. Treating the symptomatic population alone is identified as the least effective intervention for curtailing infection transmission in the susceptible population.

Therefore, this study recommends that more attention should be directed toward screening and treatment of latent infected and the asymptomatic infectious populations. Screening and treating latent infections reduce the development of pulmonary tuberculosis disease and consequently decreases the rate of infection transmission in the population. Additionally, screening and treating the asymptomatic infectious population reduce the incessant spread of infections to susceptible individuals and, consequently, decreases the rate of infection transmission.

The limitations of this study, which could be considered in future studies, include the optimal control theory in the presence of vaccination, screening, and treatment, as well as the stochastic- and fractional-order approaches of the model.

Data Availability

The data used to support the findings of this study are included in the article.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding publication of this paper.

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