

Mathematical Modelling of the Dynamics of Tuberculosis and Tobacco Smoking

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Abstract: Tuberculosis (TB) remains a major global public health challenge, particularly in low- and middle-income countries, with tobacco smoking worsening disease outcomes. This study developed a deterministic compartmental model to investigate the combined dynamics of TB transmission and tobacco smoking, incorporating vaccination, smoking behaviour, smoking cessation, waning immunity, recovery, and TB-induced mortality. The model stability and the existence of equilibrium points were determined. The basic reproduction number (R_0) was obtained using the next-generation matrix, while sensitivity analysis identified parameters with the greatest influence on TB transmission. Numerical simulations were conducted using MATLAB to investigate the effects of smoking, vaccination, vaccine efficacy, and smoking cessation on the dynamics of tuberculosis. Results indicate that the effective TB transmission rate and smoking modifying factor are key drivers of transmission, whereas vaccination and smoking cessation contribute to reducing the spread of TB. The model provided quantitative insights into the interaction between smoking and TB and offers a mathematical framework for evaluating combined behavioural and vaccination interventions for TB control.

Keywords: Tuberculosis; Tobacco smoking; Mathematical modelling; Basic reproduction number; Vaccination; Smoking cessation.

I. INTRODUCTION

Tuberculosis (TB), caused by the bacterium *Mycobacterium tuberculosis*, remains a major global public health concern, particularly in low- and middle-income countries [1, 2]. Despite being preventable and curable, TB continues to cause mortality worldwide. According to the World Health Organisation report in 2022, an estimated 10.6 million people developed TB and approximately 1.6 million died from the disease globally in 2021 [3]. TB is transmitted mainly through airborne droplets released by individuals with active pulmonary TB, which makes effective prevention and control essential [4].

Tobacco smoking is a risk factor for TB. Smoking damages the respiratory system and affects immune function, increasing susceptibility to *M. tuberculosis* infection and the risk of disease progression and poor treatment outcomes [5, 6]. Compared with non-smokers, smokers have been reported to have approximately twice the risk of developing active TB and may experience higher rates of recurrence, treatment failure, and mortality [7]. Consequently, smoking cessation represents a potentially important component of comprehensive TB control strategies [8, 9]. Vaccination, particularly with the Bacillus Calmette–Guérin (BCG) vaccine, also remains an important preventive measure, although its protection is partial and may wane over time [10, 11].

Mathematical modelling provides a useful framework for understanding TB transmission and evaluating intervention strategies. However, existing TB models often consider smoking, vaccination, smoking cessation, or waning immunity separately. This study develops and analyses a deterministic compartmental model that incorporates tobacco smoking, smoking cessation, vaccination, waning immunity, TB infection, recovery, and disease-induced mortality. The model is analysed through equilibrium analysis, computation of the basic reproduction number, sensitivity analysis, and numerical

simulations. The study provides quantitative insights into the combined effects of smoking, smoking cessation, vaccination, and waning immunity on TB transmission and contributes to the development of effective TB control strategies.

II. MODEL FORMULATION

A mathematical model was developed to investigate the dynamics of tuberculosis transmission and tobacco smoking. The population is assumed to mix homogeneously; BCG vaccination provides partial protection against TB; vaccination occurs at a constant rate; susceptible smokers may quit smoking, while quitters may relapse to smoking; non-smokers are assumed not to initiate smoking during the study period; Quitters are assumed to have a lower risk of TB infection than active smokers and Recovered individuals may lose immunity and return to the corresponding susceptible classes. The model is divided into eight compartments, namely: susceptible non-smokers (S_n), susceptible smokers (S_m), vaccinated individuals (V), quitters (Q), infected non-smokers (I_n), infected smokers (I_m), recovered non-smokers (R_n), and recovered smokers (R_m).

Figure 1 below shows an illustration of the developed model:

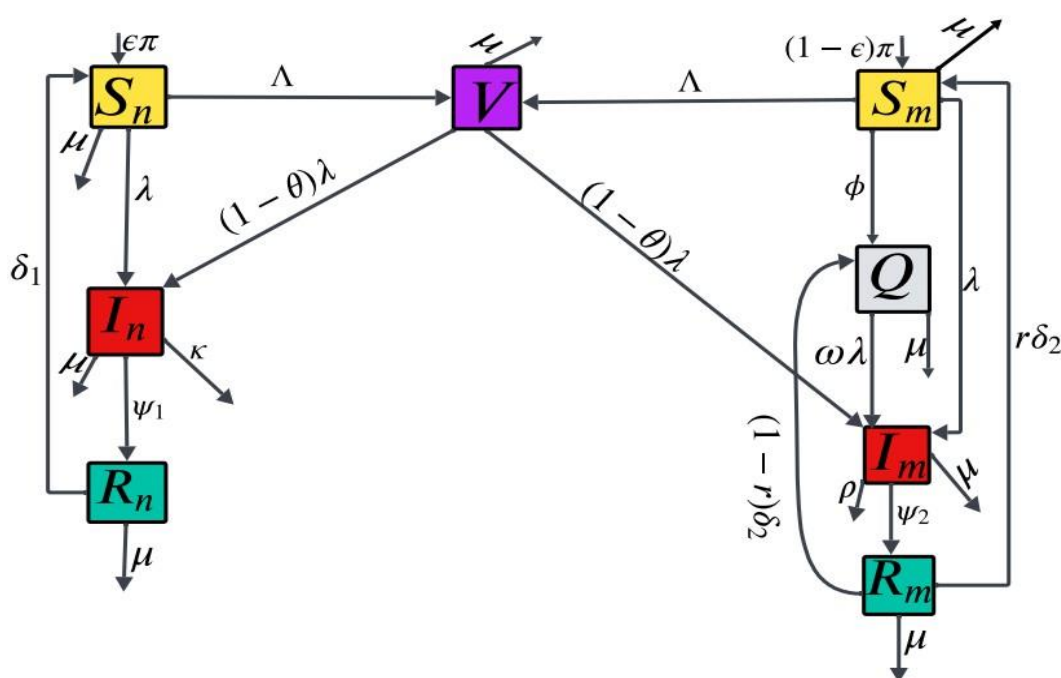


Figure 1: flow chart

The defining parameters can be seen in Table 1, which are assumed constant and non-negative.

Parameters	Description
π	Recruitment rate
κ	Rate at which infected non-smokers progress to death due to TB
ρ	Rate at which infected smokers progress to death due to TB
Λ	Rate at which susceptible are vaccinated
μ	per capita natural mortality rate
ψ_1	Rate at which infected non-smokers recover
ψ_2	Rate at which infected smokers recover
ϕ	Rate at which susceptible smokers quit smoking
β	Transmission rate
δ_1	Rate at which recovered non-smokers lose immunity
δ_2	Rate at which recovered smokers lose immunity
θ	Vaccine efficacy
ω	quitter infection modifying factor

Table 1: Description of parameters.

The following system of differential equations was developed:

$$\begin{cases} \frac{dS_n}{dt} = \epsilon\pi + \delta_1 R_n - \lambda S_n - \Lambda S_n - \mu S_n, \\ \frac{dS_m}{dt} = (1 - \epsilon)\pi + r\delta_2 R_m - \lambda S_m - \Lambda S_m - \phi S_m - \mu S_m, \\ \frac{dV}{dt} = \Lambda S_n + \Lambda S_m - 2(1 - \theta)\lambda V - \mu V, \\ \frac{dQ}{dt} = \phi S_m + (1 - r)\delta_2 R_m - \omega\lambda Q - \mu Q, \\ \frac{dI_n}{dt} = \lambda S_n + (1 - \theta)\lambda V - \kappa I_n - \psi_1 I_n - \mu I_n, \\ \frac{dI_m}{dt} = \lambda S_m + (1 - \theta)\lambda V + \omega\lambda Q - \psi_2 I_m - \rho I_m - \mu I_m, \\ \frac{dR_n}{dt} = \psi_1 I_n - \delta_1 R_n - \mu R_n, \\ \frac{dR_m}{dt} = \psi_2 I_m - \delta_2 R_m - \mu R_m. \end{cases}$$

The total population is given by:

$$N = S_n + S_m + V + Q + I_n + I_m + R_n + R_m.$$

The force of TB infection is defined as

$$\lambda = \beta \left(\frac{I_n + pI_m}{N} \right),$$

where β is the effective TB transmission rate and $p > 1$ is the smoking-modifying factor accounting for the increased contribution of infectious smokers to transmission.

i) Disease-Free Equilibrium and Stability

The disease-free equilibrium (DFE) represents a steady state at which tuberculosis is absent from the population [12]. Setting the infected compartments to zero, that is,

$$I_n^* = I_m^* = 0,$$

implies

$$R_n^* = R_m^* = 0, \quad \lambda^* = 0.$$

Substituting these conditions into the model equations and setting the derivatives to zero gives the disease-free populations

$$\begin{aligned} S_n^* &= \frac{\epsilon\pi}{\Lambda + \mu}, & S_m^* &= \frac{(1 - \epsilon)\pi}{\phi + \Lambda + \mu}, \\ V^* &= \frac{\Lambda\pi(\epsilon\phi + \Lambda + \mu)}{\mu(\Lambda + \mu)(\phi + \Lambda + \mu)}, \end{aligned}$$

and

$$Q^* = \frac{\phi(1 - \epsilon)\pi}{\mu(\phi + \Lambda + \mu)}.$$

Hence, the disease-free equilibrium is given by:

$$E_0 = \left(\frac{\epsilon\pi}{\Lambda + \mu}, \frac{(1 - \epsilon)\pi}{\phi + \Lambda + \mu}, \frac{\Lambda\pi(\epsilon\phi + \Lambda + \mu)}{\mu(\Lambda + \mu)(\phi + \Lambda + \mu)}, \frac{\phi(1 - \epsilon)\pi}{\mu(\phi + \Lambda + \mu)}, 0, 0, 0, 0 \right).$$

ii) Basic Reproduction Number (R_0)

The basic reproduction number, R_0 , was obtained using the next-generation matrix. It represents the expected number of secondary infections generated by a typical infectious individual in a wholly susceptible population [13]. Following the next-generation matrix approach, the infected compartments are I_n and I_m . The new-infection and transition matrices evaluated at the disease-free equilibrium E_0 are

$$F = \begin{pmatrix} \frac{\beta X}{N^*} & \frac{\beta p X}{N^*} \\ \frac{\beta Y}{N^*} & \frac{\beta p Y}{N^*} \end{pmatrix}, \quad V = \begin{pmatrix} \kappa + \psi_1 + \mu & 0 \\ 0 & \rho + \psi_2 + \mu \end{pmatrix},$$

where

$$X = S_n^* + (1 - \theta)V^*, \quad Y = S_m^* + (1 - \theta)V^* + \omega Q^*,$$

and

$$N^* = S_n^* + S_m^* + V^* + Q^* = \frac{\pi}{\mu}.$$

The basic reproduction number is obtained as the spectral radius of the next-generation matrix FV^{-1} . Thus,

$$\mathcal{R}_0 = \frac{\mu \beta X}{\pi(\kappa + \psi_1 + \mu)} + \frac{\mu \beta p Y}{\pi(\rho + \psi_2 + \mu)}.$$

Substituting the disease-free equilibrium values gives

$$\mathcal{R}_0 = \beta \left[\frac{\epsilon \mu (\phi + \Lambda + \mu) + (1 - \theta) \Lambda (\epsilon \phi + \Lambda + \mu)}{(\Lambda + \mu)(\phi + \Lambda + \mu)(\kappa + \psi_1 + \mu)} + \frac{p[(1 - \epsilon)(\mu + \omega \phi) + (1 - \theta) \Lambda (\epsilon \phi + \Lambda + \mu)]}{(\phi + \Lambda + \mu)(\rho + \psi_2 + \mu)} \right].$$

The DFE is locally asymptotically stable when $R_0 < 1$, indicating that TB transmission declines following the introduction of infection, whereas $R_0 > 1$ indicates the potential for TB invasion and persistence in the population [14, 15].

iii) Local Stability of the Disease-Free Equilibrium

Local stability is a situation in which, when a system is put somewhere near the point, it will move itself to the equilibrium point in some time [16]. The local stability of the disease-free equilibrium (DFE) is determined by evaluating the Jacobian matrix of the infected subsystem at the DFE [17]. Using the next-generation formulation, the Jacobian is given by

$$J = F - V,$$

where

$$F = \begin{pmatrix} \frac{\beta x}{N^*} & \frac{\beta p x}{N^*} \\ \frac{\beta y}{N^*} & \frac{\beta p y}{N^*} \end{pmatrix}, \quad V = \begin{pmatrix} \kappa + \psi_1 + \mu & 0 \\ 0 & \rho + \psi_2 + \mu \end{pmatrix},$$

with

$$x = S_n^* + (1 - \theta)V^*, \quad y = S_m^* + (1 - \theta)V^* + \omega Q^*.$$

Hence,

$$J = \begin{pmatrix} \frac{\beta x}{N^*} - (\kappa + \psi_1 + \mu) & \frac{\beta p x}{N^*} \\ \frac{\beta y}{N^*} & \frac{\beta p y}{N^*} - (\rho + \psi_2 + \mu) \end{pmatrix}.$$

For a 2×2 system, the Routh–Hurwitz criterion requires

$$\text{Tr}(J) < 0, \quad \det(J) > 0$$

for local asymptotic stability. Direct calculation gives

$$\text{Tr}(J) = \frac{\beta(x + py)}{N^*} - (\kappa + \psi_1 + \rho + \psi_2 + 2\mu),$$

and

$$\det(J) = (\kappa + \psi_1 + \mu)(\rho + \psi_2 + \mu) - \frac{\beta}{N^*}[x(\rho + \psi_2 + \mu) + py(\kappa + \psi_1 + \mu)].$$

Since $N^* = \pi/\mu$, the condition $\det(J) > 0$ is equivalent to

$$\frac{\mu\beta}{\pi} \left[\frac{x}{\kappa + \psi_1 + \mu} + \frac{py}{\rho + \psi_2 + \mu} \right] < 1.$$

Substituting the DFE values of S_n^*, S_m^*, V^* , and Q^* , the left-hand side becomes exactly the basic reproduction number $\mathcal{R}_0$. Thus,

$$\mathcal{R}_0 < 1.$$

Therefore, the disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$, whereas it is unstable when $\mathcal{R}_0 > 1$.

iv) Endemic Equilibrium

The endemic equilibrium (EE) represents a steady state at which TB persists in the population,

hence $I_n^* > 0$ and $I_m^* > 0$ [16]. Setting the right-hand sides of the model equations to zero

and solving for the non-infected compartments in terms of I_n^* and I_m^* leads to:

$$\begin{cases} S_n^* = \frac{(\delta_1 + \mu)\epsilon\pi + \delta_1\psi_1 I_n^*}{(\delta_1 + \mu)(\lambda^* + \Lambda + \mu)} \\ S_m^* = \frac{(\delta_2 + \mu)(1 - \epsilon)\pi + r\psi_2\delta_2 I_m^*}{(\delta_2 + \mu)(\lambda^* + \Lambda + \phi + \mu)} \\ V^* = \frac{\Lambda}{2(1 - \theta)\lambda^* + \mu} \left(\frac{(\delta_1 + \mu)\epsilon\pi + \delta_1\psi_1 I_n^*}{(\delta_1 + \mu)(\lambda^* + \Lambda + \mu)} + \frac{(\delta_2 + \mu)(1 - \epsilon)\pi + r\psi_2\delta_2 I_m^*}{(\delta_2 + \mu)(\lambda^* + \Lambda + \phi + \mu)} \right) \\ Q^* = \frac{1}{(\omega\lambda^* + \mu)} \left(\frac{\phi((1 - \epsilon)\pi(\delta_2 + \mu) + r\delta_2\psi_2 I_m^*)}{(\delta_2 + \mu)(\lambda^* + \Lambda + \phi + \mu)} + \frac{(1 - r)\delta_2\psi_2 I_m^*}{(\delta_2 + \mu)} \right) \\ R_n^* = \frac{\psi_1 I_n^*}{\delta_1 + \mu} \\ R_m^* = \frac{\psi_2 I_m^*}{\delta_2 + \mu} \end{cases}$$

and

$$N^* = S_n^* + S_m^* + V^* + Q^* + I_n^* + I_m^* + R_n^* + R_m^*.$$

Thus, the EE is given by:

$$E^* = (S_n^*, S_m^*, V^*, Q^*, I_n^*, I_m^*, R_n^*, R_m^*),$$

where all components are non-negative and satisfy the above equilibrium relations.

v) Sensitivity analysis

Sensitivity analysis is very important in determining which parameters dominate the results or have a high impact on R_0 and should be targeted by intervention strategies [19].

Parameters	Description	Value (per year)	Reference
π	Recruitment rate	0.029×100000	[5]
κ	Rate at which infected non-smokers progress to death due to TB	0.04	[20]
ρ	Rate at which infected smokers progress to death due to TB	0.08	Assumed
Λ	Rate at which susceptible are vaccinated	0.1	[21]
μ	per capita natural mortality rate	0.0162	[22]
ψ_1	Rate at which infected non-smokers recover	0.233	[23]
ψ_2	Rate at which infected smokers recover	0.171	Assumed
ϕ	Rate at which susceptible smokers quit smoking	0.043	[24]
β	Transmission rate	0.2	Assumed
δ_1	Rate at which recovered non-smokers loss immunity	0.01	[25]
δ_2	Rate at which recovered smokers loss immunity	0.2	Assumed
θ	Vaccine efficacy	0.5	[26]
ω	quitters infection modifying factor	0.5	Assumed
p	Smoking modifying factor	2.5	Assumed

Table 2: Description, dimension and values of parameters with references.

The normalised forward sensitivity index of a variable, u , that depends differentially on a parameter, p , is defined as [27]:

$$\gamma_p^u = \frac{\partial u}{\partial p} \times \frac{p}{u}$$

The sensitivity indices of parameters β , p , θ , κ , ψ_1 , ψ_2 , ρ , Λ , ϕ , ω and μ , are given in the table below:

Parameters	Sensitivity Index	Effect on R_0
β	+1.010	Strong positive
θ	-0.843	strong negative
p	+0.723	strong positive
ψ_2	-0.462	Negative
ρ	-0.217	Negative
ψ_1	-0.227	Negative
κ	-0.039	Weak negative
Λ	+0.046	Weak positive
ϕ	-0.021	Weak negative
ω	+0.063	Weak positive

Table 3: Normalized Forward Sensitivity Indices of the Basic Reproduction Number (R_0)

Normalised forward sensitivity analysis was performed to identify key parameters in the model that influence the basic reproduction number R_0 . A positive sensitivity index indicates that an increase in the parameter increases R_0 and hence increases disease transmission, while a negative sensitivity index indicates that an increase in the parameter decreases R_0 and hence reduces disease transmission.

Considering the simulation results in Table 3, the TB transmission rate β has the highest sensitivity index of +1.010, making it the most influential parameter affecting the transmission dynamics of tuberculosis. An increase in β by 1% results in approximately a 1.01% increase in R_0 , showing that tuberculosis transmission is highly dependent on the frequency of effective contacts between susceptible and infectious individuals. Therefore, reducing close contact with infectious individuals would play a major role in controlling the spread of the disease.

The vaccine efficacy (θ) has a sensitivity index of -0.843 , making it the parameter with the greatest negative influence on R_0 . An increase in vaccine efficacy by 1% leads to approximately a 0.843% decrease in R_0 , indicating that improving the protective effectiveness of vaccination can help reduce the spread of tuberculosis.

The smoking modification factor p has a sensitivity index of +0.723, indicating that smoking enhances the transmission of tuberculosis. An increase of 1% in p leads to approximately a 0.723% increase in R_0 . This shows that smokers contribute more to tuberculosis transmission than non-smokers, highlighting the need to control smoking as an intervention in tuberculosis control.

The recovery rates of infected smokers (ψ_2) and infected non-smokers (ψ_1) have sensitivity indices of -0.462 and -0.227 , respectively. These results indicate that increasing recovery rates reduces R_0 , showing the importance of minimising the infectious period and reducing secondary infections.

The disease-induced death rates among smokers ρ and non-smokers κ have sensitivity indices of -0.217 and -0.039 , respectively. Although increases in these parameters reduce R_0 , this is not a desirable control strategy because it represents an increase in loss of life and should therefore be interpreted as a mathematical consequence of the model.

The smoking cessation rate ϕ has a sensitivity index of -0.021 , indicating that increasing smoking cessation slightly reduces the transmission potential of tuberculosis. Although those who quit smoking remain susceptible to TB infection, smoking cessation contributes positively to disease control by lowering the overall infectious burden. The quitter infection modifying factor ω has a positive sensitivity index of +0.063, indicating that infection among quitters increases R_0 and highlighting the need for continued health monitoring and preventive interventions among quitters.

The vaccination rate Λ has a small positive sensitivity index of 0.046, indicating that small changes in vaccination around the baseline values have a limited influence on R_0 . This does not mean that vaccination is ineffective, but rather that minimal changes in vaccination coverage have a very small effect compared with changes in the effective contact rate.

III. NUMERICAL SIMULATION

The model system in (1) was solved numerically using MATLAB to illustrate the TB dynamics and assess the effects of vaccination, smoking cessation, and tobacco smoking. Parameter values were obtained from published literature where available, while unavailable values were assumed within biologically reasonable ranges.

For a population of 100,000, the initial conditions were selected using reported TB and tobacco-smoking estimates from the region. A tobacco-smoking prevalence of 10.1% implies 10,100 smokers, while the reported TB incidence of 214 per 100,000 and the proportion of TB patients who smoke were used to estimate the initial infected populations [28, 29, 30]. Thus,

$$(S_n, S_m, V, Q, I_n, I_m, R_n, R_m) = (89686, 10100, 0, 0, 169, 45, 0, 0).$$

The vaccinated, quitter, and recovered compartments were initially set to zero to examine their subsequent development under vaccination, smoking cessation, and treatment.

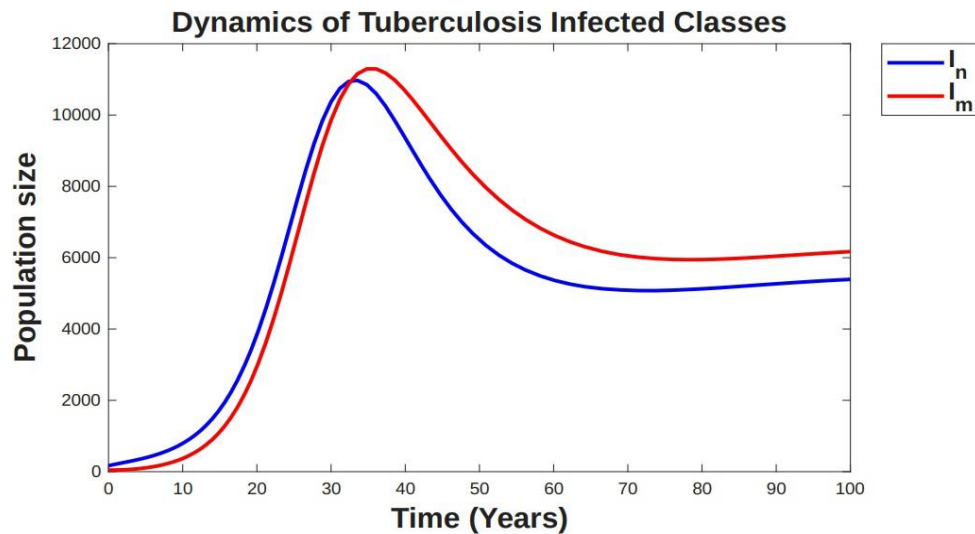


Figure 2: Dynamics of the model compartments.

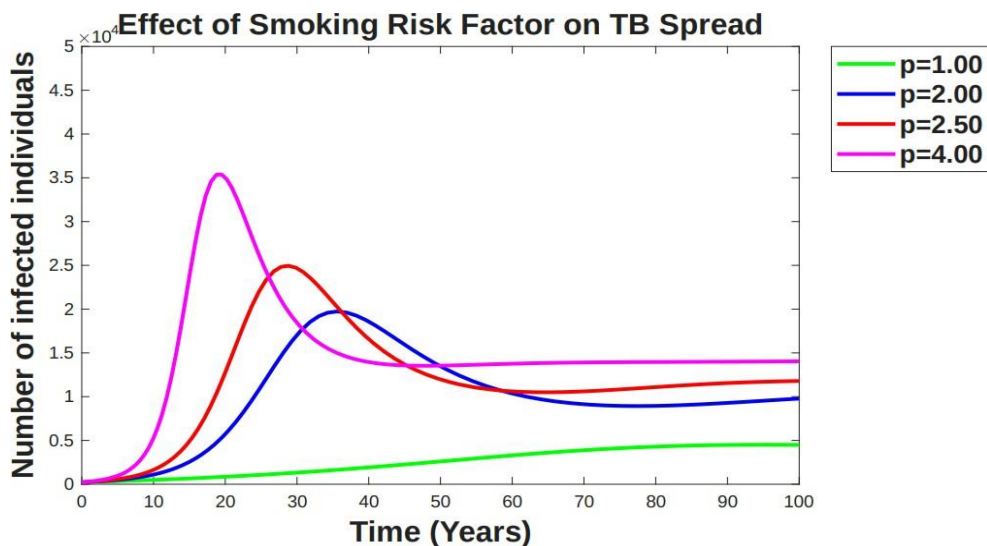


Figure 3: Effect of the Smoking modifying factor (p).

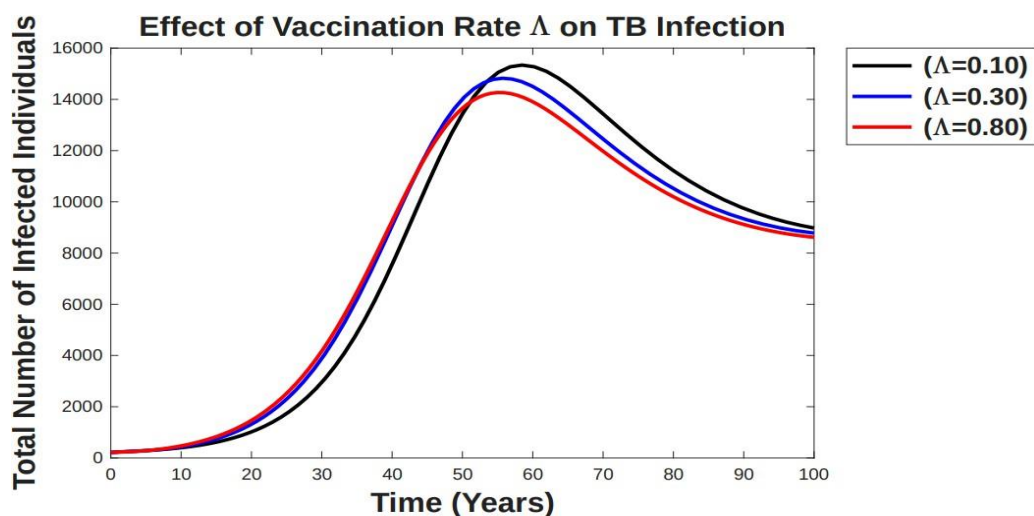


Figure 4: Effect of vaccination rate Λ on TB dynamics.

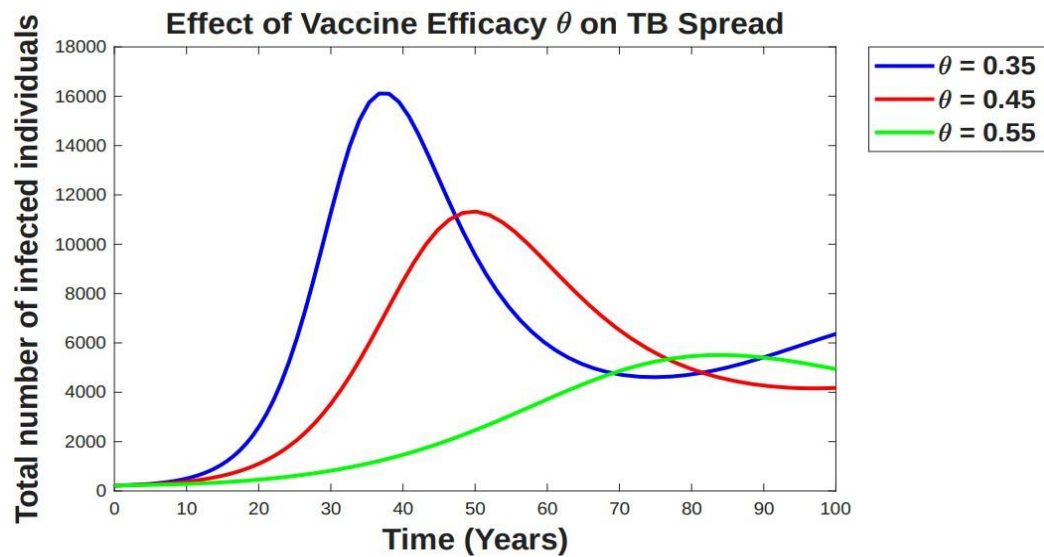


Figure 5: Effect of vaccine efficacy θ on TB dynamics.

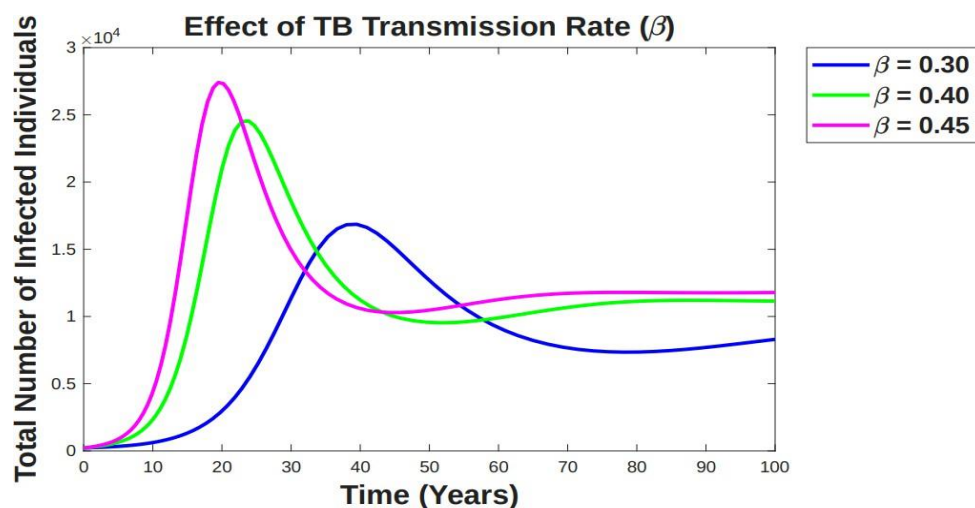


Figure 6: Effect of transmission rate β on TB dynamics.

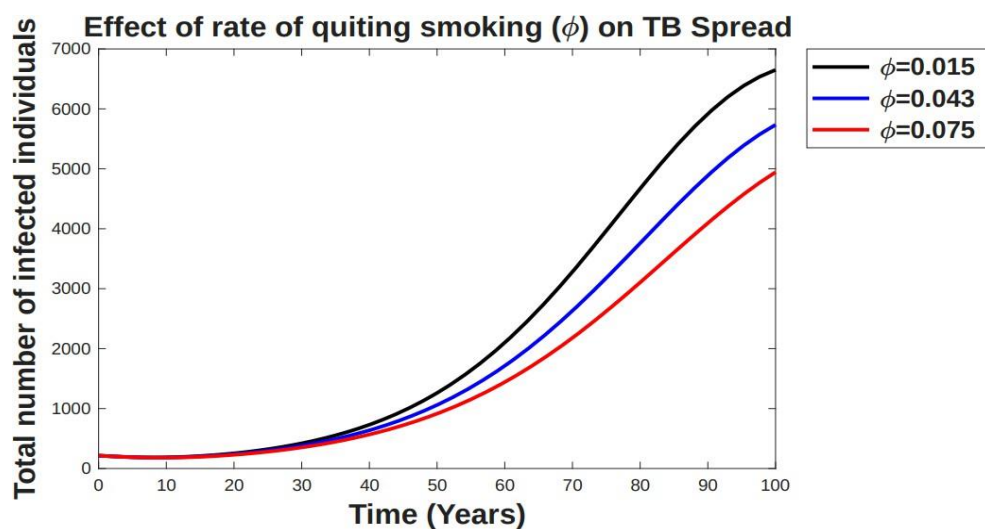


Figure 7: Effect of smoking cessation rate ϕ on TB dynamics.

Figure 2 shows the dynamics of I_n and I_m . The infected classes initially increase, reach a peak, and then decline toward endemic levels. Throughout the simulation, I_m remains higher than I_n , indicating the greater contribution of smoking to TB transmission through the smoking-modifying factor p .

Figure 3 presents the effect of varying p on the total infected population. Increasing p from 1 to 2, 2.5, and 4 results in faster disease growth and higher infection levels. Thus, a larger smoking-modifying factor increases the contribution of infected smokers to TB transmission and consequently increases the overall TB burden. The results highlight smoking reduction and cessation as important interventions for TB control.

Figure 4 presents the effect of the vaccination rate Λ on the total infected population. Increasing Λ reduces the epidemic peak and the long-term number of infected individuals. At $\Lambda = 0.1$, the infected population reaches its highest peak, whereas higher vaccination rates progressively suppress and delay the epidemic. All trajectories eventually approach endemic levels, but the equilibrium infection level decreases with increasing vaccination. These results demonstrate that increasing vaccination coverage can substantially reduce TB transmission and persistence.

Figure 5 illustrates the effect of vaccine efficacy θ on TB dynamics. Increasing vaccine efficacy reduces TB transmission in the population. Although TB persists at endemic levels, higher vaccine efficacy results in a lower long-term infection burden. Thus, improving vaccine effectiveness can enhance TB control.

Figure 6 illustrates the effect of the transmission rate β on the total infected population for $\beta = 0.30, 0.40$, and 0.45 . Increasing β results in a faster increase in infections, earlier epidemic peaks, and higher peak values. The peak occurs at approximately 35, 20, and 15 years for $\beta = 0.30, 0.40$, and 0.45 , respectively. Higher transmission rates also produce greater endemic infection levels. These results confirm that β is a key driver of TB transmission and that reducing effective transmission is essential for controlling the disease.

Figure 7 presents the effect of the smoking cessation rate ϕ on TB dynamics for $\phi = 0.015, 0.043$, and 0.075 . Increasing the cessation rate progressively reduces the number of infected individuals, with the effect becoming more pronounced over time. The lowest cessation rate, $\phi = 0.015$, produces the greatest infection burden, whereas $\phi = 0.075$ results in the lowest number of infections. The trajectories are initially similar because changes in smoking status accumulate gradually, but increasingly diverge as cessation takes effect. These findings highlight smoking cessation as an important complementary intervention for reducing the long-term burden of TB.

IV. CONCLUSION

In this study, a mathematical model of the dynamics of tuberculosis and tobacco smoking was developed. The model incorporated tobacco smoking, smoking cessation, and vaccination. The disease-free and endemic equilibria were established, and the disease-free equilibrium was shown to be locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. Under the values of the baseline parameters, $R_0 > 1$, indicating the potential for the persistence of TB

in the population. Sensitivity analysis identified the TB transmission rate β and smoking modifying factor p as major positive contributors to TB transmission, highlighting the importance of reducing effective transmission and smoking-related infectiousness. Numerical simulations further showed that increasing vaccination rate and vaccine efficacy reduces TB infection, while increasing the smoking cessation rate lowers the long-term infection burden.

In general, the findings demonstrate that smoking is an important factor in the dynamics of tuberculosis and should be considered when developing mathematical models and interven-

tion strategies. Integrating vaccination, smoking cessation, and measures that reduce TB transmission may therefore provide a more effective approach to controlling TB and reducing its long-term burden.

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