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Analysing of Tuberculosis in Turkey through SIR and SEIR Models

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Abstract
Tuberculosis (TB), caused by bacterium Mycobacterium tuberculosis, primarily affects the lungs but can also affects other parts of the body. This research investigates TB transmission dynamics using Susceptible-Infected-Recovered (SIR) and Susceptible-Exposed-Infected-Recovered (SEIR) models, both employing Ordinary Differential Equations (ODEs). The models contain two non-negative equilibria: the disease-free equilibrium point (DFEP) and the endemic equilibrium point (EEP). The stability of the models is determined using the Routh-Hurwitz Criterion. The basic reproduction number, R_0 is calculated using the Next Generation Matrix method. When $R_0 < 1$, the disease does not spread and when $R_0 > 1$, it become endemic. Model parameters are derived from TB reported data, with simulations conducted in MATLAB using the function ode15s. This research compares SIR and SEIR models using TB reported data to assess their accuracy and investigate the effects of varying transmission (β) and recovery (γ) rates on infection rates. Higher β increases infections, while higher γ values lead to faster recovery and reduce infections population. The findings benefit healthcare by showing that with $R_0 < 1$, current measures are effective but require monitoring. The analysing β and γ identifies effective interventions such as vaccination and treatment. Furthermore, comparing SIR and SEIR models helps policymakers choose better models for predicting TB outbreaks and evaluating public health strategies.

Keywords: SIR; SEIR; Infectious disease; Tuberculosis; Basic reproduction number

1. Introduction

Originating over thousands of years ago, TB has persisted through the annals of human history, leaving an indelible mark on societies, cultures, and civilizations. Its resilience and ability to adapt have often outpaced our attempts to mitigate and eradicate it, leading to TB's recognition as a global health emergency by the World Health Organization (WHO) in the early 1990s [1].

Globally, TB remains one of the top ten causes of death and the leading cause from a single infectious agent [2]. The WHO reported an alarming 10 million new TB cases worldwide in 2020 alone. While the disease spans continents, its burden is notably severe in certain regions. For instance, in 2020, countries such as India, Indonesia, China, the Philippines, and Pakistan accounted for almost two-thirds of the global total [2].

To address this pressing global health issue, leveraging mathematical models such as the SIR (Susceptible-Infectious-Recovered) and SEIR (Susceptible-Exposed-Infectious-Recovered) models becomes crucial. These models play a pivotal role in understanding the dynamics of tuberculosis transmission, predicting its future trends, and assessing the impact of interventions. The SIR model simplifies the population into compartments of susceptible, infectious, and recovered individuals, while the SEIR model extends this by adding an exposed compartment, accounting for individuals who are infected but not yet infectious.

These mathematical models provide valuable insights into designing effective strategies for TB control, optimizing resource allocation, and ultimately reducing the burden of this disease. By quantifying the intricate interplay of various factors, such as population demographics, transmission dynamics, and intervention efficacy, SIR and SEIR models offer a systematic approach to inform evidence-based decisions and policy recommendations.

1.1. Tuberculosis Disease

Tuberculosis (TB) has been present for millions of years, with the genus Mycobacterium originating more than 150 million years ago. The disease has been associated with a high mortality rate throughout history, and in the Middle Ages, it was known as "king's evil" and believed to be curable by a royal touch. It wasn't until 1720 that the infectious origin of TB was first conjectured by the English physician Benjamin Marten, and in 1882, Robert Koch was able to isolate the tubercle bacillus, which was a significant breakthrough in the fight against [3].

Genetic studies have been instrumental in tracing the evolution and spread of the TB bacterium, revealing a complex history intertwined with human migration [4]. The researcher discusses how the genetic diversity of TB strains mirrors the patterns of human settlement and movement, suggesting a millennia-long association between the pathogen and its host. This evolutionary perspective is crucial in comprehending the resilience and adaptability of TB.

Moreover, the transmission of tuberculosis is influenced by several key factors that interact with one another. High population density can lead to increased transmission rates [5]. The strength and reach of healthcare infrastructure significantly affects disease management and containment. Furthermore, socioeconomic status influences susceptibility to TB, with those in less favorable economic conditions often facing greater exposure and risk [5]. Comprehensive healthcare access is vital, as insufficient medical services may lead to delayed treatment and contribute to the spread of TB. Understanding the interplay between these factors is essential for the development and implementation of effective TB control measures [5].

However, many researchers discussed the strategies to prevent and control the TB transmission. prevent and control the TB transmission, [6] discuss the global strategies for TB control, emphasizing the importance of robust health systems and international collaboration in combating this disease. Besides, [7] discussed several prevention strategies for TB in Korea. One of the key strategies is the management of latent TB infection (LTBI) through screening and treatment of high-risk groups. This involves identifying individuals who have been infected with TB bacteria but do not have active TB disease and providing them with appropriate treatment to prevent the development of active TB in the future.

2. SIR Model for Tuberculosis

SIR model is one of a mathematical model to analyze the simulation of the spreading of TB. This research will focus on the modification of the basic SIR model initially proposed by Kermack and McKendrick [8]. The model has two equilibrium points called disease-free equilibrium points (DFEP) and endemic equilibrium point (EEP). Figure 1 shows the flow of SIR model for Tuberculosis. Total population size is as $N(t) = S(t) + I(t) + R(t)$.

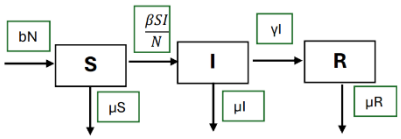

Figure 1 Schematic diagram of SIR model transmission for Tuberculosis

Based on Figure 1, the compartment can be understood as follows:

$$\frac{dS(t)}{dt} = bN - \frac{\beta S(t)I(t)}{N} - \mu S(t), \quad (1)$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t)}{N} - (\gamma + \mu)I(t), \quad (2)$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \mu R(t). \quad (3)$$

With subject to $S(0) \geq 0$, $I(0) \geq 0$, and $R(0) \geq 0$. Based on SIR model, β is the transmission rate. Meanwhile, γ represents the recovery rate The population dynamics are also influenced by μ, the birth rate, which adds new susceptible individual, and b, the death rate, which is the natural death rate in the population. Let $b = \mu$,

Hence, SIR model presented by:

$$\frac{dS(t)}{dt} = -\beta S(t)I(t) - \mu(1 - S(t)). \tag{4}$$

$$\frac{dE(t)}{dt} = \beta S(t)I(t) - (\gamma + \mu)I(t). \tag{5}$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \mu R(t). \tag{6}$$

2.1. Equilibrium Point

To find equilibrium points, let Equation (4) to Equation (6) equal to zero [9]. Thus, the equation can be written as

$$\frac{dS(t)}{dt} = \frac{dI(t)}{dt} = \frac{dR(t)}{dt} = 0. \tag{7}$$

2.1.1. The Disease-Free Equilibrium Point (DFEP)

The DFEP occurs when the infected population, $I = 0$, In other words, there is no active infection in the population. Hence, get $S = 1$ and DFEP can be written as:

$$D_1 = (S_1^*, I_1^*, R_1^*) = (1,0,0). \tag{8}$$

2.1.2. The Endemic Equilibrium Point (EEP)

The EEP is the situation where the infected population,$I \neq 0$, indicating that the disease persists in the population and obtained:

$$D_2 = (S_2^*, I_2^*) = \left(\frac{\gamma+\mu}{\beta}, \frac{\mu(\beta-\gamma-\mu)}{\beta(\mu+\gamma)}, \frac{\gamma(\beta-\gamma-\mu)}{\beta(\mu+\gamma)}\right). \tag{9}$$

2.2. Jacobian Matrices

The Jacobian matrix for SIR model is as follow:

$$J(D_n) = (S_n^*, I_n^*) = \begin{bmatrix} -\beta I - \mu & \beta S & 0 \\ \beta I & \beta S - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}, \tag{10}$$

Substituting the values for the disease-free equilibrium point (DFEP) and endemic equilibrium point (EEP) yields the final Jacobian matrix, yields the following matrix:

$$J(D_1) = (1,0) = \begin{bmatrix} -\mu & \beta & 0 \\ 0 & \beta - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}, \qquad J(D_2) = \left(\frac{\gamma+\mu}{\beta}, \frac{\mu(\beta-\gamma-\mu)}{\beta(\mu+\gamma)}, \frac{\gamma(\beta-\gamma-\mu)}{\beta(\mu+\gamma)}\right) = \begin{bmatrix} \frac{-\mu\beta}{\mu+\gamma} & -\gamma-\mu & 0 \\ \frac{\mu(\beta-\gamma-\mu)}{\mu+\gamma} & 0 & 0 \\ 0 & \gamma & -\mu \end{bmatrix}.$$

2.3 Basic Reproduction Number (R_0) for SIR Model

From the above SIR model, an epidemic occurs if the number of infected classes increases. Therefore, $\frac{dI}{dt} > 1$ and at the outset of an epidemic, nearly everyone is susceptible. Hence, can say that $S \approx 1$, substitute the $S = 1$, where the susceptible class is equal to the population, and it becomes:

$$R_0 = \frac{\beta}{\gamma+\mu} > 1. \tag{13}$$

2.4 Stability Analysis at DFEP and EEP for SIR Model

The DFEP system is stable if and only if $R_0 > 1$. To determine the stability analysis at DFEP, need to find the $det(J - \lambda I)$ of Equation (11). Thus, the characteristics polynomial is as follow:

$$P(\lambda) = \lambda^3 - (-3\mu + \beta - \gamma)\lambda^2 - (2\mu\beta - 2\gamma\mu - 3\mu^2) + \mu^2(\lambda + \mu - \beta). \tag{14}$$

From the Equation (14), 3 is the highest power. For determining the stability of DFEP, the Routh-Hurwitz criteria test is used. Based on the Routh-Hurwitz criterion [10] the sufficient and necessary condition for stability of DFEP are the eigenvalues must be negative real part. From Equation (14), the eigenvalues are as follows:

$$\lambda_1 = \beta - \gamma - \mu \qquad \lambda_2 = -\mu \qquad \lambda_3 = -\mu$$

From observation, all eigenvalue λ_1, λ_2 and $\lambda_3 < 0$ when $\beta < \gamma + \mu$. It is shown that the DFEP is stable.

Next, is stability at EEP, using the Equation (11), same approach as DFEP and obtained that the characteristics polynomial is as follows:

$$P(\lambda) = \lambda^3 + (\mu+\mu R_0)\lambda^2 + (\mu^2 R_0 + \mu\beta - \frac{\mu\beta}{R_0})\lambda + \mu^2\beta - \frac{\mu^2\beta}{R_0} \quad (15)$$

Let a_1, a_2 and a_3 be coefficient of λ_1, λ_2 and λ_3 and a_4 be the constant of the Equation (15). From Routh–Hurwitz stability criterion analysis, if $a_1 > 0, a_2 > 0$ and $a_1 a_2 - a_3 > 0$, then, all the roots of the characteristic equation have a negative real part; hence, the equilibrium point (EEP) is stable. Thus, the EEP remains stable within the population.

3. SEIR Model for Tuberculosis

The SEIR epidemic model represents an extension of the SIR model. Within this model describing TB transmission, it includes four compartments: susceptible $(S(t))$, exposed $(E(t))$, infected $(I(t))$, and recovered $(R(t))$.

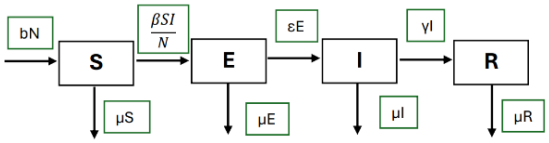

Figure 2 Schematic diagram of SIR model transmission for Tuberculosis

Based on Figure 2, the compartment can be understood as follows:

$$\frac{dS(t)}{dt} = bN - \frac{\beta S(t)I(t)}{N} - \mu S(t), \quad (16)$$

$$\frac{dE(t)}{dt} = \frac{\beta S(t)I(t)}{N} - (\gamma+\in)E(t), \quad (17)$$

$$\frac{dI(t)}{dt} = E \in (t) - (\gamma + \mu)I(t), \quad (18)$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \mu R(t). \quad (19)$$

With subject to $S(0) \geq 0$, $I(0) \geq 0$, and $R(0) \geq 0$. Where a new compartment $E(t)$ denotes individuals who are infected but do not yet show symptoms. From SEIR model, the parameter $\in$ represents the rate at which exposed individuals become infected, where $\frac{1}{\in}$ is the mean latent period, while other parameter are the same as SIR model above. After solving the Equation (16) to Equation (19), the system of SEIR model is presented by:

$$\frac{dS(t)}{dt} = -\beta S(t)I(t) + \mu(1 - S(t)) \quad (20)$$

$$\frac{dE(t)}{dt} = \beta S(t)I(t) - (\in +\mu)E(t) \quad (21)$$

$$\frac{dI(t)}{dt} = E \in (t) - (\gamma + \mu)I(t) \quad (22)$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \mu R(t) \quad (23)$$

3.1 Equilibrium Point

To find equilibrium points, let Equation (20) to Equation (23) equal to zero [9]. Thus, the equation can be written as:

$$\frac{dS(t)}{dt} = \frac{dI(t)}{dt} = \frac{dE(t)}{dt} = \frac{dR(t)}{dt} = 0. \quad (24)$$

3.1.1. The Disease-Free Equilibrium Point (DFEP)

The DFEP occurs when the infected population, $I = 0$, In other words, there is no active infection in the population. Hence, get $S = 1$ and DFEP can be written as

$$D_1 = (S_1^*, I_1^*, E_1^*, R_1^*) = (1,0,0,0). \quad (25)$$

3.1.2. Basic Reproduction Number (R_0), for SEIR Model

The basic reproduction number, R_0 represents the average number of secondary infections produced by a single infected individual in a completely susceptible population. For the SEIR model of tuberculosis, the reproduction number is given by the following expression:

$$R_0 = \frac{-\beta\epsilon}{(\mu+\epsilon)(\mu+\gamma)}. \tag{26}$$

3.1.3. The Endemic Equilibrium Point (EEP)

The EEP is the situation where the infected population, $I \neq 0$, indicating that the disease persists in the population and obtained in terms of R_0,

$$D_2 = (S_2^*, E_2^*, I_2^*, R_2^*) = (\frac{1}{R_0}, \frac{\gamma(R_0-1)}{R_0(\epsilon+\mu)}, \frac{\mu(R_0-1)}{\beta}, \frac{\gamma(R_0-1)}{\beta}). \tag{27}$$

3.2. Jacobian Matrices

The Jacobian matrix for SEIR model is as follow:

$$J(D_n) = (S_n^*, E_n^*, I_n^*, R_n^*) = \begin{bmatrix} -\beta I - \mu & 0 & -\beta S & 0 \\ \beta I & -(\epsilon+\mu) & \beta S & 0 \\ 0 & \epsilon & -(\gamma+\mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}. \tag{28}$$

Substituting the values in Equation (25) and Equation (27) into Equation (28) to get Jacobian for DFEP and EEP,respectively as follows:

$$J(D_1) = (1,0,0,0) = \begin{bmatrix} \mu & 0 & -\beta & 0 \\ 0 & -(\epsilon+\mu) & \beta & 0 \\ 0 & \epsilon & -(\gamma+\mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}$$

$$J(D_2) = \left(\frac{1}{R_0}, \frac{\gamma(R_0-1)}{R_0(\epsilon+\mu)}, \frac{\mu(R_0-1)}{\beta}, \frac{\gamma(R_0-1)}{\beta}\right) = \begin{bmatrix} -\mu R_0 & 0 & \frac{-\beta}{R_0} & 0 \\ \mu(R_0-1) & -(\epsilon+\mu) & \frac{\beta}{R_0} & 0 \\ 0 & \epsilon & -(\gamma+\mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}.$$

3.3. Stability Analysis at DFEP and EEP for SEIR Model

The stability for DFEP and EEP in SEIR model is same approach as in SIR model. Thus, the characteristics polynomial for DFEP is as follows:

$$P(\lambda) = a_0\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4.$$

Where

$$a_0 = 1,$$
$$a_1 = 4\mu + \gamma + \epsilon,$$
$$a_2 = \gamma\epsilon + 3\mu\epsilon + 3\gamma\mu + 6\mu^2 - R_0(\gamma+\mu)(\epsilon+\mu),$$
$$a_3 = 2\gamma\epsilon\mu + 3\gamma\mu^2 + 4\mu^3 - 2\mu R_0(\gamma+\mu)(\epsilon+\mu),$$
$$a_4 = \mu^2(\gamma\epsilon + \mu\epsilon + \gamma\mu + \mu^2 - R_0(\gamma+\mu)(\epsilon+\mu).$$

Clearly $a_0 > 0$, $a_1 > 0, a_2 > 0, a_3 > 0$ and $a_4 > 0$, hence the eigenvalues will be negative. Thus, DFEP will be stable if $R_0 < 1$.

Next is stability analysis for EEP, the characteristics polynomial is as follows:

$$P(\lambda) = a_0\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4$$

where,

$$a_0 = 1,$$
$$a_1 = 3\mu + \gamma + \mu R_0 \ ,$$
$$a_2 = \epsilon\gamma + 2\epsilon\mu + \mu R_0\epsilon + 2\gamma\mu + \gamma\mu R_0 + 3\mu^2 + 3\mu^2 R_0 - \epsilon\beta$$
$$a_3 = \epsilon\gamma\mu + \epsilon\gamma\mu R_0 + \gamma\mu^2 + 2\gamma\mu^2 R_0 + \epsilon\mu^2 + 2\epsilon\mu^2 R_0 + \mu^3 + 3\mu^3 R_0 - \frac{2\epsilon\beta\mu}{R_0}$$
$$a_4 = \mu(\gamma\epsilon\mu R_0 + \gamma\mu^2 R_0 + \epsilon\mu^2 R_0 + \mu^3 R_0 - \frac{\epsilon\beta\mu}{R_0}$$

According to the Hurwitz criterion [10], $a_1 > 0$, $a_3 > 0$ and $a_1 a_2 - a_3 > 0$, hence the EEP is stable. It is clear that, $a_1 > 0$, $a_3 > 0$ and $a_1 a_2 - a_3 > 0$. Thus, the equilibrium point at EEP is locally asymptotically stable if and only if $R_0 > 1$; otherwise, it is unstable.

4. Numerical Simulation of SIR and SEIR Model

4.1. Parameter value of SIR and SEIR

The numerical simulation for SIR and SEIR models will be based on the epidemiological data from [11]. The initial conditions for both models are established using specific values to illustrate the numerical results, which are shown in Table 4.

Table 4. Model parameters and initial data

Parameter	Description	SIR Value	SEIR value
$\boldsymbol{\beta}$	Transmission rate of infected population	0.872	0.872
$\in$	Rate of progression to infectious stage from exposed	-	1.428
$\boldsymbol{\gamma}$	Recovery rate	0.897	0.938
$\boldsymbol{\mu}$	Natural death rate	0.0049	0.0049
$\boldsymbol{S(0)}$	Initial number of susceptible	67610005	67595153
$\boldsymbol{E(0)}$	Initial number of exposed	-	14852
$\boldsymbol{I(0)}$	Initial number of infected	20535	20535
$\boldsymbol{R(0)}$	Initial number of recovered	1230000	1230000
$\boldsymbol{R_0}$	Reproduction number	0.9664	0.9211

The data from 2005 to 2015 regarding the epidemic are taken into consideration. The total initial population $N(0) = 68860540$, mirroring Turkey's reported population in 2005 [12]. The initial number of infected individuals $I(0)$ is obtained from a report [13] indicating 20535 cases. Estimation of the initial number of exposed individuals $E(0)$ involves a comparison of infection-to-exposure ratios in literature and adaptation to Turkey's data.

4.2 Prediction cases of SIR and SEIR models

This numerical simulation will focus on infected populations to compare which models, SIR or SEIR, provide more accurate predictions of infected populations based on reported data.

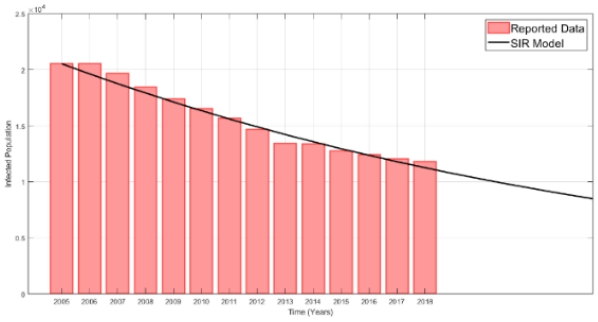

Figure 3 Bar diagram of SIR prediction model and reported data

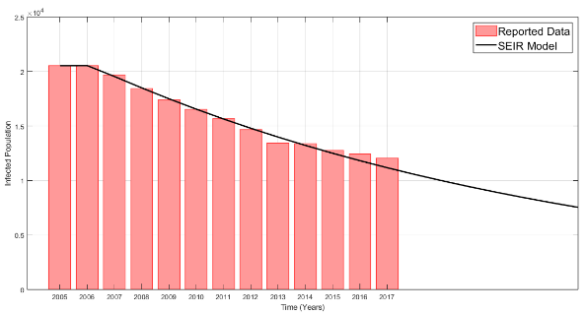

Figure 4 Bar diagram of SEIR prediction model and reported data

Figure 3 shows a bar chart illustrating the reported infected data from 2005 to 2015 and a line graph representing the predictions made by the SIR model. The value of SIR parameter listed in Table 4. It showcases the annual total infection counts of actual cases show at Table 5 alongside the model's predictions. The results indicate that the model does a good job approximating the real infection counts.

Figure 4 follows the same approach as Figure 3 but this time it compares the actual yearly infection cases to the prediction made by the SEIR model. From observation, it is clear that the model's predictions closely match the real data, indicating that the SEIR model is also effective in approximating the actual infection counts.

To show accuracy of the numerical solutions of SIR and SEIR, the relative error is calculated by using

$$h(t) = \frac{|I_c(t) - I_d(t)|}{I_d(t)},$$

Where $I_c(t)$ and $I_d(t)$ are the model prediction and the corresponding data at time t, respectively.

Table 5. Model parameters and initial data [2,13-22].

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Year	Reported data	SIR	ERROR(SIR)	SEIR	ERROR(SEIR)
2005	20535	20535	-	20535	-
2006	20526	19628	0.04375	20543	0.00085
2007	19694	18757	0.04758	19543	0.00767
2008	18452	17917	0.02900	18497	0.00244
2009	17402	17111	0.01672	17494	0.00529
2010	16551	16338	0.01287	16543	0.00048
2011	15679	15597	0.00523	15643	0.00230
2012	14691	14889	0.01348	14790	0.00674
2013	13409	14211	0.05981	13984	0.04288
2014	13378	13563	0.01383	13222	0.1166
2015	12772	12944	0.01347	12499	0.2137
2016	12417	12352	0.00523	11817	0.04832
2017	12046	11787	0.02150	11171	0.07260
2018	11786	11248	0.04565	10561	0.10394
2019	-	10733	-	9984	-
2020	-	10241	-	9438	-
2021	-	9772	-	8922	-
2022	-	9324	-	8435	-
2023	-	8896	-	7973	-
2024	-	8487	-	7537	-

Table 5 presents the reported infected data, model predictions, and their relative errors for the years 2005 to 2018. The reported data sources include various WHO (World Health Organization) reports from 2007 to 2017. Table 5 shows that for SIR model, the lowest errors occurred in 2011 and 2016. For SEIR model, the lowest error was in 2010. That indicates that both models have particular years where their predictions were exceptionally accurate compared to the actual reported data.

4.3. Variation in Transmission Rate (β), and Recovery Rate (γ) for SIR model

The aim is to investigate the impact of varying β and γ values on the infected population. Figure 5 and Figure 6 are simulated based on the parameters listed in Table 4.

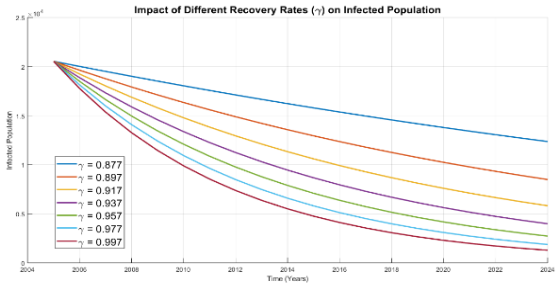

Figure 5 Total number of infected individuals for different values of transmission rate (β) (SIR)

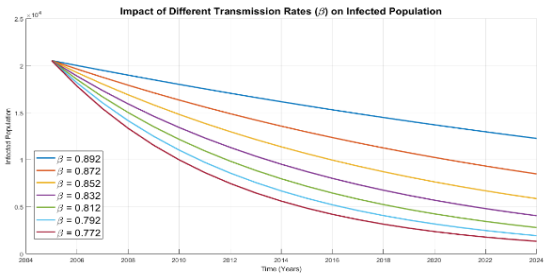

Figure 6 Total number of infected individuals for different values of recovery rate (γ) (SIR)

Figure 5 and Figure 6 explore how changing β and γ values affect the number of infected individuals. In Figure 5, it can be observed that as β increases to its highest value of 0.892 (represented by the blue line), the infected population also increases correspondingly. Conversely, when β decreases to its lowest value of 0.772 (red line), the infected population decreases accordingly. This implies that an increase in the transmission rate leads to a decrease in the infected population.

While in Figure 6, the total number of infected individuals for different values of γ (SIR model) is depicted. From observation, when $\gamma = 0.877$ (represented by the blue line), the infected population increases. However, an increase in the γ value to 0.997 results in a decrease in the number of infected individuals. This observation indicates that an increase in the recovery rate leads to a decrease in the number of infected individuals.

4.4 Variation in Transmission Rate, and Recovery Rate for SEIR model

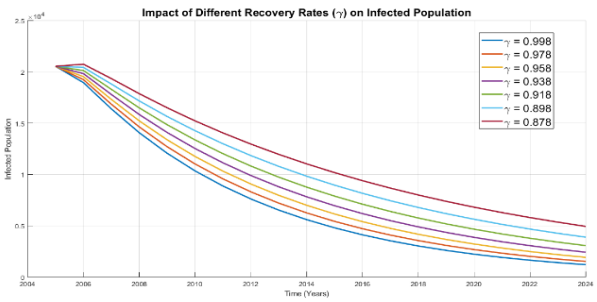

Figure 7 Total number of infected individuals for different values of transmission rate (β) (SEIR)

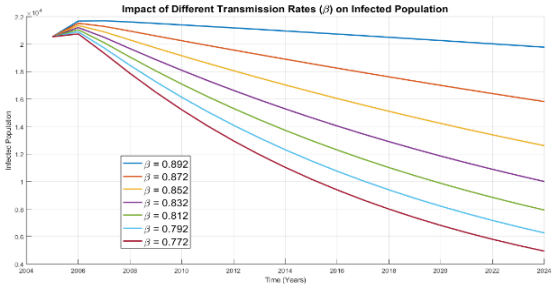

Figure 8 Total number of infected individuals for different values of recovery rate (γ) (SEIR)

In Figure 7, the impact of seven β values (ranging from 0.772 to 0.892) on the infected population is observed. For instance, when β is at its highest (0.892), the infected population initially rises from 2005 to 2006 and then gradually decreases until 2024. Conversely, lowering β to 0.772 follows a similar pattern of an initial increase followed by a continuous decrease. This indicates that reducing the transmission rate leads to fewer infected individuals.

The initial rise in infections occurs because at the outbreak's start, many people are susceptible. With a high transmission rate, the infection spreads rapidly among these susceptible individuals, causing a sharp increase in infections initially. As more people get infected, the susceptible population shrinks, slowing new infections and resulting in a peak before declining as recoveries outpace new infections.

In Figure 8, the impact of seven γ values (ranging from 0.878 to 0.998) in the SEIR Model on the infected population is analyzed. Lower γ values (like 0.878) show a higher peak of infected individuals initially, followed by a decrease until 2024. Conversely, higher γ values (like 0.998) result in a lower peak and a quicker decline in infections. This indicates that a faster recovery rate leads to fewer infected individuals.

For lower γ values, slower recovery means infected individuals remain infectious longer, leading to more transmissions initially. As the epidemic progresses, even with slower recoveries, the susceptible population decreases, causing fewer new infections and decreasing the infected population. Higher γ values lead to quicker recoveries, reducing the infectious period and resulting in a lower peak and faster decline in infections.

Conclusion

This research used data on Tuberculosis (TB) and conducted simulations in MATLAB to compare two mathematical models: the SIR (Susceptible-Infectious-Recovered) and the SEIR (Susceptible-Exposed-Infectious-Recovered) models. The SEIR model is likely to provide a more accurate representation of TB dynamics because it accounts for the latent period before an infected individual becomes infectious. This additional detail can result in more precise predictions, especially in diseases like TB, where there is a significant latency period. By analyzing how different rates of transmission (β) and recovery (γ) impact infections, while higher recovery rate led to faster healing and a reduced infected population. Moreover, the results of this research have significant when the basic reproduction number, $R_0 < 1$, current measures are effective but need continuous monitoring. Analysing β and γ identifies effective interventions like vaccination and treatment. Furthermore, comparing SIR and SEIR models helps policymakers choose better models for spreading TB outbreaks and evaluating public health strategies.

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