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Adv Syst Sci Appl 2022; **04**; 144-161
Published online at http://ijassa.ipu.ru.

Analysis and Dynamics of Tuberculosis Outbreak: A Mathematical Modelling Approach

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Abstract: Tuberculosis (TB) is an infectious disease caused by mycobacterium disease which causes major ill health in humans. Control strategies like vaccines, early detention, treatment and isolation are required to minimize or eradicate this deadly pandemic disease. This article presents a novel mathematical modelling approach to tuberculosis disease using Vaccinated-Susceptible-Latent-Mild-Chronic-Isolated-Treated model. We examined if the epidemiology model is well posed and then obtained two equilibria points (disease free and endemic equilibrium). We also showed that TB disease free equilibrium is locally and globally asymptotically stable if $R_0 < 1$. We solved the model analytically using Homotopy Perturbation Method (HPM) and the graphical representations and interpretations of various effects of the model parameters in order to measure the impact for effective disease control are presented. The findings show that infected populations will be reduced when the isolation and treatment rates and their effectiveness are high.

Keywords: tuberculosis; Homotopy Perturbation Method; infectious disease; basic reproduction number; vaccination

1. INTRODUCTION

Tuberculosis (TB) is the third greatest killer worldwide caused by an infectious agent [12]. According to World Health Organization (WHO), one-third of the world's population is currently infected by the TB bacillus bacteria. Being a disease of poverty, the vast majority of TB deaths are in developing countries with more than half occurring in Asia. Furthermore, over 95% of these deaths occurred in low- and middle-income countries where the cost of diagnosis and treatment is high, and not readily accessible.

Tuberculosis is a chronic bacteria infectious disease caused by Mycobacterium tuberculosis which poses a major health, social and economic burden globally, especially in low- and middle-income countries [5]. The surge in HIV-TB co-infection and the growing emergence of multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB) strains has further fueled TB epidemic. TB usually affects the lungs (pulmonary TB) but it can also affect other sites as well (extra-pulmonary TB). Tuberculosis is transmitted by tiny

airborne droplets which are expelled into the air when a person with active pulmonary TB coughs or talks [21,22].

Diagnosis of latent TB infections (LTBI) and prompt treatment of active cases remains an important component of effective TB control as shown in previous studies. On the other hand, undetected TB infection and delay in the treatment of active TB cases leads to more severe disease conditions in the infected person which could result in wider disease spread in the community [3, 7, 10, 9].

Some researchers have proposed some mathematical models to solve problems arising from TB models and we shall discuss some of them as follows: [8] presents a Susceptible-Exposed-Infected-Recovered (SEIR) tuberculosis model which incorporated treatment of infectious individuals and chemoprophylaxis (treatment for the latently infected). The model assumed that the latently infected individuals develop the active disease as a result of endogenous re-activation, exogenous re-infection and disease relapse. [4] presents the mathematical model of a tuberculosis transmission dynamics incorporating first and second line treatment. [7] proposed a seven-compartment model which included diagnosed and undiagnosed infectious population and their result shows that high vaccination rate is required to eradicate TB. In [16], the authors develop a mathematical model for control of tuberculosis epidemiology by incorporating some control strategies, the findings of the study show that using multiple controls is the best way to control the spread of TB. Several studies have been conducted utilizing a mathematical model method in order to identify ways to control diseases in the population [1,13, 18-20]. [17] presented a deterministic SEIR model to described the transmission dynamics of TB in Ashanti region of Ghana and the results showed that the region has herd immunity against TB infection. In this study we consider some control strategies such as, treatment, isolation and vaccination into the mathematical formulation of TB outbreak with assumptions that people in each compartment have equal natural death rate and infection does not confer immunity to the treated and recovered individuals.

The rest of the article is divided as follows: Method which includes model formulation and mathematical analysis of the model are described in “section 2. Next, section consists of the semi-analytic solution of the model formulated. Numerical simulation and graphical representation of results is given in 4, while the discussion of the results is presented in section 5. Finally, in section 6, we have provided conclusions of this article.

2. METHOD

2.1. Model formulation

In this section, the TB transmission model is formulated. Using a compartmental approach, the total host population can be partitioned into seven compartments according to their epidemiological status. The groups are the Vaccinated $\big(V(t)\big)$, Susceptible $\big(S(t)\big)$,Latent $\big(E(t)\big)$, Mild TB $\big(I_m(t)\big)$, Chronic TB $\big(I_c(t)\big)$, isolated infectious $\big(J(t)\big)$ and Treated $\big(T(t)\big)$ individual, where t is the time variable. It is assumed that once the treatment of active TB cases is interrupted, there is no more treatment. Vaccination reduces the risk of infection by a factor $\theta \in (0,1)$ and the efficacy of the vaccine is ω. Let a constant π stands for the number of newborn babies into the population, then $\theta\pi$ are the individuals in the vaccinated class while $(1-\theta)\pi$ are the susceptible individual. The susceptible class also increases with a waning rate of vaccine at ω, due to fact that vaccine does not confer a total immunity. We assume that$\mu$is per capital natural death rate and $d_i(i = 1,2,3)$ is the disease induced death rate in classes$I_m(t)$, $I_c(t)$ and$J(t)$ respectively. It is natural to assume that $d_2 \geq d_3 \geq d_1$ due to the treatment of active TB cases reducing the disease induced death rate are the transmission coefficients from class $S(t)$, $E(t)$ and $T(t)$ respectively. We assume that $\lambda_1 > \lambda_2 > \lambda_3$ because the treatment of active TB cases reduces the infectivity of active TB cases. Take $\rho(0 < \rho < 1)$ as the fraction of the latent persons who have fast TB progression. The

proportion ϕ of individual in the exposed class will progress to the chronic class via endogenous us reactivation. $\sigma$is the reactivation rate from the latent persons to infected class. $\gamma$is the reactivation rate of the individual in the mild TB $\left(I_m(t)\right)$ to the chronic TB $\left(I_c(t)\right)$. The parameters are the recovery rates of the individual in the classes $\left(I_m(t)\right)$, $\left(I_c(t)\right)$ and $J(t)$ respectively. And the parameter is the rate of isolation of the individuals in chronic class $\left(I_c(t)\right)$.

In this article, the TB dynamic model describing the compartment is based on the following assumptions:

That a proportion of the population of newborn is immunized against TB infection through vaccination.

That the immunity conferred on individuals by treatment expires after some time at given rate.

That people in each compartment have equal natural death rate of μ

That there are no immigrants and emigrants. The only way of entry into the population is through new-born babies and the only way of exit is through death from natural causes or death from TB related causes.

That the infection does not confer immunity to the treated and recovered individuals and so they go back to the susceptible class at a given rates.

That all newborns are previously uninfected by TB and therefore join either the immunized compartment or the susceptible compartment depending on whether they are vaccinated or not.

We combine the basic assumptions, model parameters, variables and the TB infection processes to formulate a schematic diagram for TB infection as shown in Figure 1. The model equations are given as follow

$$\frac{dV}{dt} = \pi\theta - (\mu + \omega)V\,, \tag{1}$$

$$\frac{dS}{dt} = \pi(1-\theta) - \lambda_1 S - \mu S + \omega V, \tag{2}$$

$$\frac{dE}{dt} = \lambda_1 S + \lambda_3 T - \lambda_2 E - (\mu + \sigma)E, \tag{3}$$

$$\frac{dI_m}{dt} = (1-\rho)\sigma E - (\gamma + \mu + r_1 + d_1)I_m + (1-\phi)\lambda_2 E, \tag{4}$$

$$\frac{dI_c}{dt} = \rho\sigma E + \gamma I_m - (r_2 + r_3 + \mu + d_2)I_c + \phi\lambda_2 E, \tag{5}$$

$$\frac{dJ}{dt} = r_3 I_c - (\mu + d_3 + r_4)J \quad , \tag{6}$$

$$\frac{dT}{dI} = r_4 J + r_2 I_c + r_1 I_m - (\mu + \lambda_3)T\,, \tag{7}$$

where $\lambda_1 = \beta(I_c + \varepsilon_1 I_m + \varepsilon_2 J)$,$\lambda_2 = \alpha(I_c + \varepsilon_3 I_m + \varepsilon_4 J)$ and $\lambda_3 = \gamma(I_c + \varepsilon_5 I_m + \varepsilon_6 J)$.

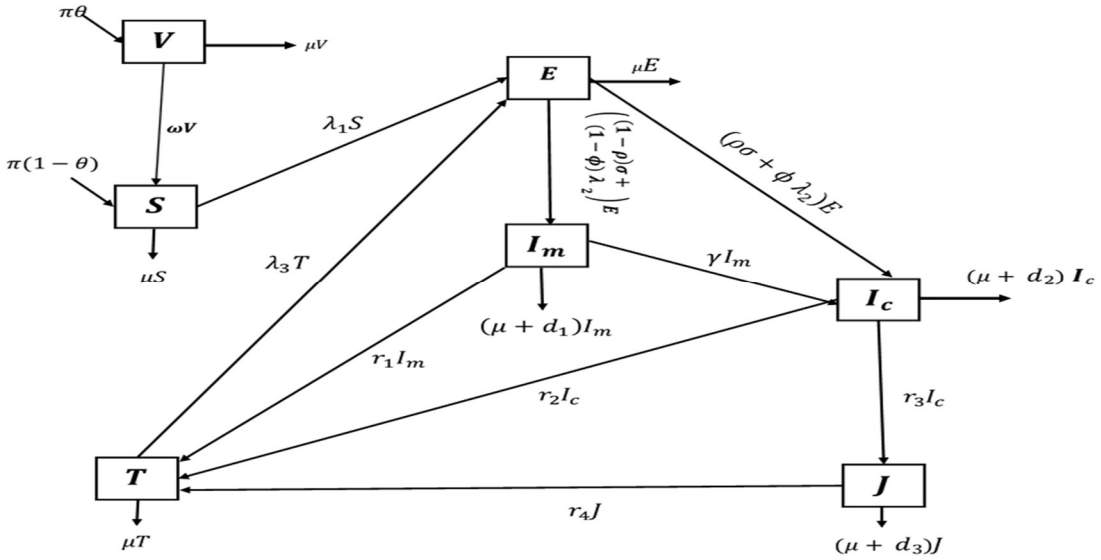

Figure 1: Schematic Representation of the Model

Table 1. Notation and definition of other parameters

Symbol	Description
r_1	Treatment rate of individuals in mild class
r_2	The treatment rate for those is $I_c$class
r_3	The progression rate from classesI_c to J
r_4	The treatment rate for those in isolated class
β	Transmission rate among the susceptible
d_1	Death rate for mild TB individual
d_2	Death rate for chronic TB individuals
d_3	Death rate for isolated class
α	Effective transmission rate from latent class to infected class
ε_1	Relative infectiousness of humans with mild TB compared to humans in the chronic class
ε_2	Relative infectiousness of humans with TB in the isolated class compared to humans in the chronic class
ε_3	Relative infectiousness of humans with mild TB due to endogenous reactivation compared to humans in the chronic class.
ε_4	Relative infectiousness of humans on isolation after endogenous reactivation compared to humans in the chronic class
ε_5	Relative infectiousness of humans with mild TB due to exogenous re-infection compared to humans in the chronic class
ε_6	Relative infectiousness of humans on isolation after exogenous re-infection compared to humans in the chronic class
ρ	Fraction of the latent persons who have fast TB progression
σ	Reactivation rate from the latent individuals
ϕ	Proportion of individual in the exposed class
γ	Reactivation rate of the individual in the mild TB class
μ	Natural death rate

2.2. The positive invariant region

The entire population size N can be determined from by adding Equations (1)–(7). Hence, $N(t) = S(t) + V(t) + E(t) + I_m(t) + I_c(t) + J(t) + T(t)$ then,

$$\frac{dN}{dt} = \pi - \mu N(t) - d_1 N - d_2 N - d_3 N \tag{8}$$

In the absence of the disease $(d_1 = d_2 = d_3 = 0)$ then (8) gives

$$\frac{dN}{dt} = \pi - \mu N \tag{9}$$

Theorem 1:

The system (1) to (7) has solution which are contained in the feasible region Ω *for all* $t > 0$.

Proof.

Let $\Omega = (S, V, E, I_m I_c, J, T) \in R^7$ be any solution of the system (1)–(7) with non-negative initial condition. Using theorem of differential inequality, Equation (9) gives

$\frac{dN}{dt} \leq \pi - \mu N$, then $0 \leq N \leq \frac{\pi}{\mu}$, Hence $\pi - \mu N \geq ke^{-\mu t}$, where k is constant. Thus, the feasible set of the model is given by

$$\Omega = \left\{(S, V, I_m, I_c, J, T) \in R^7 : S, V, I_m, I_c, J, T \geq 0, N \leq \frac{\pi}{\mu}\right\},$$

which is positive invariant (i.e., solution remain positive for all time t) and the model is epidemiologically meaningful and mathematically well pose. □

2.3. Positivity of solutions

Since Equations (1)–(7) represent the population in each compartment and all model parameters are all positive, then it lies in a region Ω defined by

$$\Omega = \left\{(S, V, I_m, I_c, J, T) \in R^7 : S, V, I_m, I_c, J, T \geq 0, N \leq \frac{\pi}{\mu}\right\}.$$

Theorem 2:

Let the initial value for the model equation be given as

$$\left\{\left(S(0), V(0), E(0), I_m(0), I_c(0), J(0), T(0)\right)\right\} \in n$$

Then the solution set $\{S(t), V(t), E(0), I_m(t), I_c(t), J(t), T(t)\}$ *of the system (1)–(7) is positive for all* $t > 0$.

Proof.

From Equation (2)

$$\frac{dS}{dt} = \pi(1-\theta) + \omega V - (\lambda_1 + \mu)S \text{ then, } \frac{dS}{dt} \geq -(\lambda_1 + \mu)S$$

On Integrating it gives $\int \frac{dS}{S} \geq -\int (\lambda_1 + \mu)\, dt$.

Hence, $S(t) \geq S(0)e^{-(\lambda_1+\mu)t}$.

Applying the same approach to other equations in the model equations, we have:

$$V(t) \geq V(0)e^{-(\mu+\omega)t}, E(t) \geq E(0)e^{-(\sigma+\mu+\lambda_2)t}, I_m(t) \geq I_m(0)e^{-(\gamma+\mu+r_1+d_1)t},$$

$I_c(t) \geq I_c(0)e^{-(r_2+r_3+\mu+d_3)t}, J(t) \geq J(0)e^{-(\mu+d_3+r_3)t}, T(t) \geq T(0)e^{-(\mu+\lambda_3)t}$. Therefore, the solution to the model equations is positive for all $t > 0$.□

2.4. Equilibrium points of the model

The equilibrium state is the point in which there is zero disturbance on the system under consideration. That is, the rate of change of the model variables with time is zero. Thus, at equilibrium,

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI_m}{dt} = \frac{dI_c}{dt} = \frac{dJ}{dt} = \frac{dT}{dt} = 0 \tag{10}$$

Let $E^* = (V, S, E, I_m, I_c, J, T) = (V^*, S^*, E^*, I_m{}^*, I_c{}^*, J^*, T^*)$ be arbitrarily equilibrium point

Substituting Equations (10) into Equations (1)–(7) gives:

$$\pi\theta - (\mu + \omega)V = 0, \tag{11}$$

$$\pi(1-\theta) - \lambda_1 S - \mu S + \omega V = 0, \tag{12}$$

$$\lambda_1 S + \lambda_3 T - \lambda_2 E - (\mu + \sigma)E = 0, \tag{13}$$

$$(1-\rho)\sigma E - (\gamma + \mu + r_1 + d_1)I_m + (1-\phi)\lambda_2 E = 0, \tag{14}$$

$$\rho\sigma E + \gamma I_m - (r_2 + r_3 + \mu + d_2)I_c + \phi\lambda_2 E = 0, \tag{15}$$

$$r_3 I_c - (\mu + d_3 + r_4)J = 0, \tag{16}$$

$$r_4 J + r_2 I_c + r_1 I_m - (\mu + \lambda_3)T = 0. \tag{17}$$

From Equation (11) we have

$$V = \frac{\pi\theta}{(\mu+\omega)}. \tag{18}$$

Substitute Equation (18) into equation (12) we have

$$\frac{1}{\lambda_1+\mu}\left(\pi(1-\theta) + \frac{\omega\pi\theta}{\mu+\omega}\right) = S. \tag{19}$$

From Equation (16) we have $J = K_1 I_c$ where $K_1 = \frac{r_3}{r_4+\mu+d_3}$.

From (15) we have $[(1-\rho)\sigma + (1-\phi)\lambda_2]E = [r_2 + r_3 + \mu + d_2]I_m \Rightarrow E = K_2 I_m$ where $K_2 = \frac{r_2+r_3+\mu+d_3}{(1-\rho)\sigma+(1-\phi)\lambda_2}$, from Equation (16) we have $(r_2 + r_3 + \mu + d_2)I_c = \rho\sigma E + \phi\lambda_2 E + \gamma I_m$, then $I_c = K_3 I_m$ where $K_3 = \frac{(\rho\sigma+\phi\lambda_2)K_2+\gamma}{r_2+r_3+\mu+d_2}$ further substitution gives $J = K_1 K_3 I_m$ and from Equation (17) we have: $T = \frac{r_4 J + r_2 I_c + r_1 I_m}{\mu+\lambda_3}$.

then $T = K_4 I_m$ where $K_4 = \frac{(r_4 K_1 K_3 + r_2 K_3 + r_1)}{\mu+\lambda_3}$,

$$\left.\begin{aligned}\lambda_1 &= \beta(I_c + \varepsilon_1 I_m + \varepsilon_2 J)\\ \lambda_2 &= \alpha(I_c + \varepsilon_3 I_m + \varepsilon_4 J)\\ \lambda_3 &= \gamma_1(I_c + \varepsilon_5 I_m + \varepsilon_6 J)\end{aligned}\right\} \text{ and } \left.\begin{aligned}J &= K_1 K_3 I_m\\ I_c &= K_3 I_m\end{aligned}\right\} \text{ by combining both equations:}$$

$$\left.\begin{aligned}\lambda_1 &= \beta I_m(K_3 + \varepsilon_2 K_1 K_3 + \varepsilon_1)\\ \lambda_2 &= \alpha I_m(K_3 + \varepsilon_4 K_1 K_3 + \varepsilon_3)\\ \lambda_3 &= \gamma_1 I_m(K_3 + \varepsilon_6 K_1 K_3 + \varepsilon_5)\end{aligned}\right\}$$

Further substitution gives $[\beta K_5 S + \gamma_1 K_7 - \alpha K_4 K_6 I_m - (\mu + \sigma)K_2]I_m = 0$.

Therefore, $I_m = 0$ or $\beta K_5 S + \gamma_1 K_7 - \alpha K_6 K_4 I_m - (\mu + \sigma)K_2 = 0$

$$\text{where } \left.\begin{aligned}K_5 &= K_3 + \varepsilon_1 + \varepsilon_2 K_1 K_3\\ K_6 &= K_3 + \varepsilon_3 + \varepsilon_4 K_1 K_3\\ K_7 &= K_3 + \varepsilon_5 + \varepsilon_6 K_1 K_3\end{aligned}\right\} \text{ and}$$

$$\left.\begin{aligned}\lambda_1 &= \beta K_5 I_m\\ \lambda_2 &= \alpha K_6 I_m\\ \lambda_3 &= \gamma_1 K_7 I_m\end{aligned}\right\}. \tag{20}$$

2.5. Disease free equilibrium (D.F.E)

The disease-free equilibrium state is the point at which there exist no infection in the given population.

At Disease Free Equilibrium, we let

$$(V, S, E, I_m, I_c, J, T) = E^* = (V^*, S^*, E^*, I_m^*, I_c^*, J^*, T^*).$$

Lemma 1:

The D.F.E of the model exists and is given by

$$E^0 = (V^*, S^*, E^*, I_m^*, I_c^*, J^*, T^*) = \left(\frac{\pi\theta}{(\mu+\omega)}, \frac{\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)}, 0,0,0,0,0\right).$$

Proof.

Suppose $I_m = 0$. Then Equation (20) becomes $\left.\begin{matrix}\lambda_1 = 0\\ \lambda_2 = 0\\ \lambda_3 = 0\end{matrix}\right\}$ and also $\left.\begin{matrix}J^* = 0\\ E^* = 0\\ I_c^* = 0\\ T^* = 0\end{matrix}\right\}$.

From (19)

$$S^* = \frac{\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)}.$$

Thus, the lemma is proved and

$$\begin{pmatrix} V^* \\ S^* \\ E^* \\ I_m{}^* \\ I_c{}^* \\ J^* \\ T^* \end{pmatrix} = \begin{pmatrix} \frac{\pi\theta}{(\mu+\omega)} \\ \frac{\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}. \tag{21}$$

The above equation is the Disease-Free Equilibrium.

2.7. Effective reproduction number

In biomathematics, the basic reproduction number *(R_0)* is the average number of infected contacts per infected individual. It is one of the fundamental concepts to determine the future of an epidemics in a population. When $R_0 < 1$ The infection will die out in the long run, but if $R_0 > 1$. The infection will be able to spread in a population. In this model, the spectral radius of the equation is given as largest eigenvalue given as $R_0 = \rho f v^{-1}$

Following the procedure in [14, 15], the next generation matrix operator is used to estimate the effective reproduction number such that the Jacobian matrices for the new infection terms and the remaining transfer terms are obtained below

$$F_i = \begin{pmatrix} \lambda_1 S + \lambda_3 T \\ \big((1-\phi)\lambda_2\big)E \\ (\phi\lambda_2)E \\ 0 \end{pmatrix}$$

$$V_i = \begin{pmatrix} (\mu+\sigma)E \\ (\gamma_1 + r_1 + d_1 + \mu)I_m - (1-\rho)\sigma E \\ (r_2 + r_3 + d_2 + \mu)I_c - \gamma_1 I_m - \rho\sigma E \\ (\mu + d_3 + r_4)J - r_3 I_C \end{pmatrix}$$

$$F(DFE) = \begin{pmatrix} 0 & \frac{\beta\varepsilon_1\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} & \frac{\beta\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} & \frac{\beta\varepsilon_2\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (\mu+\sigma) & 0 & 0 & 0 \\ -(1-\rho)\sigma & (\gamma_1+\mu+r_1+d_1) & 0 & 0 \\ -\rho\sigma & -\gamma_1 & (r_2+r_3+d_2+\mu) & 0 \\ 0 & 0 & -r_3 & (\mu+r_4+d_3) \end{pmatrix}$$

$$\text{Let } V = \left.\begin{pmatrix} Q_1 & 0 & 0 & 0 \\ -C_1 & Q_2 & 0 & 0 \\ -C_2 & -\gamma_1 & Q_3 & 0 \\ 0 & 0 & -r_3 & Q_4 \end{pmatrix}\right\}$$

where

$$\begin{gathered} Q_{1=}(\mu+\sigma), \\ Q_2 = \gamma+\mu+r_1+d_1, \\ Q_3 = r_2+r_3+d_2+\mu, \\ Q_4 = d_3+r_4+\mu, \end{gathered}$$

therefore

$$V^{-1} = \begin{pmatrix} \frac{1}{(\mu+\sigma)} & 0 & 0 & 0 \\ \frac{(1-\rho)\sigma}{Q_1Q_2} & \frac{1}{Q_2} & 0 & 0 \\ \frac{(1-\rho)\gamma\sigma+\rho\sigma Q_2}{Q_1Q_2Q_3} & \frac{\gamma}{Q_2Q_3} & \frac{1}{Q_3} & 0 \\ \frac{r_3[(1-\rho)\gamma\sigma+\rho\sigma Q_2]}{Q_1Q_2Q_3Q_4} & \frac{r_3\gamma}{Q_2Q_3Q_4} & \frac{r_3}{Q_3Q_4} & \frac{1}{Q_4} \end{pmatrix}$$

$$FV^{-1} =$$

$$\begin{pmatrix} \frac{\beta\varepsilon_1\sigma x(1-\rho)}{Q_1Q_2}+\frac{\beta x\sigma[(1-\rho)\gamma_1+\rho Q_2]}{Q_1Q_2Q_3}+\frac{\beta\varepsilon_2 xr_3\sigma[(1-\rho)\gamma_1+\rho Q_2]}{Q_1Q_2Q_3Q_4} & \frac{\beta\varepsilon_1 x}{Q_2}+\frac{\beta x\gamma_1}{Q_2Q_3}+\frac{\beta\varepsilon_2 xr_3\gamma_1}{Q_2Q_3Q_4} & \frac{\beta x}{Q_3}+\frac{\beta\varepsilon_2 xr_3}{Q_3Q_4} & \frac{\beta\varepsilon_2 x}{Q_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{22}$$

where $x = \frac{\pi(\mu+\omega-\mu\ )}{\mu(\mu+\omega)}$.

From (22), we calculate the eigen-values to determine the basic reproduction number, R_o by taking the spectral radius (dominant eigenvalue) of the matrix FV^{-1}, This is computed by $|J-\lambda I|=0$, hence the matrix becomes

$$|J-\lambda I| = \begin{vmatrix} T_1-\lambda_1 & T_2 & T_3 & T_4 \\ 0 & -\lambda_2 & 0 & 0 \\ 0 & 0 & -\lambda_3 & 0 \\ 0 & 0 & 0 & -\lambda_4 \end{vmatrix} = 0$$

where

$$T_1 = \frac{\beta\varepsilon_1\sigma x(1-\rho)}{Q_1Q_2}+\frac{\beta x\sigma[(1-\rho)\gamma_1+\rho Q_2]}{Q_1Q_2Q_3}+\frac{\beta x\varepsilon_2 r_3\sigma[(1-\rho)\gamma_1+\rho Q_2]}{Q_1Q_2Q_3Q_4}$$

$$T_2 = \frac{\beta\varepsilon_1 x}{Q_2}+\frac{\beta x\gamma_1}{Q_2Q_3}+\frac{\beta\varepsilon_2 xr_3\gamma_1}{Q_2Q_3Q_4}, T_3 = \frac{\beta x}{Q_3}+\frac{\beta\varepsilon_2 xr_3}{Q_3Q_4}, T_4 = \frac{\beta\varepsilon_2 x}{Q_4}$$

and

$$x = \frac{\pi(\mu + \omega - \mu\theta)}{\mu(\mu + \omega)}.$$

This implies that

$$\lambda_1 = T_1 = \frac{\beta\varepsilon_1\sigma x(1-\rho)}{Q_1Q_2} + \frac{\beta x\sigma[(1-\rho)\gamma_1 + \rho Q_2]}{Q_1Q_2Q_3} + \frac{\beta\varepsilon_2 x r_3\sigma[(1-\rho)\gamma_1 + \rho Q_2]}{Q_1Q_2Q_3Q_4}$$
$$\lambda_2 = 0, \lambda_3 = 0, \lambda_4 = 0.$$

Therefore

$$R_0 = \frac{\beta\sigma x[(1-\rho)(\varepsilon_1 Q_3 + \gamma_1) + \rho Q_2]}{Q_1Q_2Q_3} + \frac{\beta\varepsilon_2 x r_3\sigma[(1-\rho)\gamma_1 + \rho Q_2]}{Q_1Q_2Q_3Q_4}$$
$$= \frac{\beta\sigma\pi(\mu + \omega - \mu\theta)([(1-\rho)(\varepsilon_1 Q_3 + \gamma_1) + \rho Q_2]Q_4 + \varepsilon_2 r_3[(1-\rho)\gamma_1 + \rho Q_2])}{\mu(\mu + \omega)(\mu + \sigma)Q_2Q_3Q_4}$$

where

$$Q_2 = \gamma + \mu + r_1 + d_1,$$
$$Q_3 = r_2 + r_3 + d_2 + \mu,$$
$$Q_4 = d_3 + r_4 + \mu.$$

2.8. Local stability of disease-free equilibrium

Theorem 3:

The Disease Equilibrium of the model equations (1)–(7) is locally asymptotically Stable (LAS) if $R_0 < 1$.

Proof.

Using Jacobian stability techniques, the Jacobian matrix at D.F.E is given by:

$$J(E^0) = \begin{pmatrix} -(\mu+\omega) & 0 & 0 & 0 & 0 & 0 & 0 \\ \omega & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu+\sigma) & \frac{\beta\varepsilon_1\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} & \frac{\beta\varepsilon_1\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} & \frac{\beta\varepsilon_1\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)} & 0 \\ 0 & 0 & (1-\rho)\sigma & -(\gamma_1+\mu+d_1+r_1) & 0 & 0 & 0 \\ 0 & 0 & \rho\sigma & \gamma & -(r_2+r_3+\mu+d_3) & 0 & 0 \\ 0 & 0 & 0 & 0 & r_3 & -(\mu+d_3+r_4) & 0 \\ 0 & 0 & 0 & r_1 & r_2 & r_4 & -\mu \end{pmatrix}.$$

Let

$$\left.\begin{aligned} Q_1 &= (\mu+\sigma), \\ Q_2 &= (\gamma+\mu+d_1+r_1), \\ Q_3 &= -(r_2+r_3+\mu+d_3), \\ Q_4 &= (\mu+d_3+r_4), \\ Q_5 &= (\mu+\omega), \\ B_1 &= \tfrac{\beta\varepsilon_1\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)}, \\ B_2 &= \tfrac{\beta\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)}, \\ B_3 &= \tfrac{\beta\varepsilon_1\pi(\mu+\omega-\mu\theta)}{\mu(\mu+\omega)}, \\ C_1 &= (1-\rho)\sigma \end{aligned}\right\}, \tag{23}$$

$$J(E^0) = \begin{pmatrix} -Q_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ \omega & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -Q_1 & B_1{}^* & B_2 & B_3 & 0 \\ 0 & 0 & C_1 & Q_2 & 0 & 0 & 0 \\ 0 & 0 & \rho\sigma & \gamma & -Q_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & r_3 & -Q_4 & 0 \\ 0 & 0 & 0 & r_1 & r_2 & r_4 & -\mu \end{pmatrix}.$$

Then,

$$|J - \lambda I| = \begin{pmatrix} -Q_5 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\mu Q_5 - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -Q_1 - \lambda & B_1 & B_2 & B_3 & 0 \\ 0 & 0 & 0 & Q_6 - \lambda & C_1B_2 & C_1B_3 & 0 \\ 0 & 0 & 0 & 0 & Q_7 - \lambda & Q_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & -Q_9 - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -Q_9Q_{10} - \lambda \end{pmatrix}$$

and

$$\begin{aligned} &Q_6 = C_1B_1 - Q_1Q_2, \\ &Q_7 = (C_1B_1 - Q_1Q_2)(\rho\sigma B_2 - Q_1Q_3) - (\gamma Q_1 - \rho\sigma B_1)C_1B_2, \\ &Q_8 = (C_1B_1 - Q_1Q_2)\rho\sigma B_3 - (\gamma Q_1 - \rho\sigma B_1)C_1B_3, \\ &Q_9 = -([(C_1B_1 - Q_1Q_2)(\rho\sigma B_2 - Q_1Q_3) - (\gamma Q_1 - \rho\sigma B_1)C_1B_2]Q_4), \\ &+r_3[(C_1B_1 - Q_1Q_2)\rho\sigma B_3 - (\gamma Q_1 - \rho\sigma B_1)C_1B_3], \\ &Q_{10} = \mu(C_1B_1 - Q_1Q_2)[(C_1B_1 - Q_1Q_2)(\rho\sigma B_2 - Q_1Q_3) - (\gamma Q_1 - \rho\sigma B_1)C_1B_2] \end{aligned}$$

where

$$-\left.\begin{aligned} &Q_5 - \lambda = 0, \\ &-\mu Q_5 - \lambda = 0, \\ &-Q_1 - \lambda = 0, \\ &Q_6 - \lambda = 0, \\ &Q_9 - \lambda = 0, \\ &-Q_{13}Q_{15} - \lambda = 0 \end{aligned}\right\}. \tag{24}$$

From Equation (24) $\lambda_6 = -Q_{13}$,

$$\lambda_6 = -\begin{Bmatrix} [(C_1B_1 - Q_1Q_2)(\rho\sigma B_2 - Q_1Q_3) - (\gamma Q_1 - \rho\sigma B_1)C_1B_2]Q_4 \\ +r_3[(C_1B_1 - Q_1Q_2)\rho\sigma B_3 - (\gamma Q_1 - \rho\sigma B_1)C_1B_3] \end{Bmatrix}. \tag{25}$$

Substitute equation (23) into equation (25), we have

$$= -\beta\delta\pi(\mu + \omega - \mu\theta)\left[\frac{(1-\rho)(\varepsilon_1Q_3 + \gamma)Q_4 + \rho Q_2Q_4 + r_3\varepsilon_2(1-\rho)\gamma + \rho Q_2}{\mu(\mu+\omega)Q_1Q_2Q_3Q_4}\right].$$

Therefore,

$$R_0 = \beta\delta\pi(\mu + \omega - \mu\theta)\left[\frac{(1-\rho)(\varepsilon_1Q_3+\gamma)Q_4+\rho Q_2Q_4+r_3\varepsilon_2(1-\rho)\gamma+\rho Q_2}{\mu(\mu+\omega)Q_1Q_2Q_3Q_4}\right]. \tag{26}$$

The DFE is locally asymptotically stable since all the eigenvalues of (26) are negatives for $R_0 < 1$, hence the proof is established. □

3. NUMERICAL METHOD

3.1. Homotopy Perturbation Method (HPM)

The fundamental of Homotopy Perturbation Method (HPM) was first proposed by [11]. The Homotopy Perturbation Method, which provides analytical approximate solution, is applied to various linear and non-linear equations. [2, 18] used Homotopy Perturbation Method to solve a Susceptible-Infected-Recovered (SIR) model of infectious diseases. The Homotopy Perturbation Method is a series expansion method used in the solution of nonlinear partial differential equations [18].

To show the simple concepts of this method, we consider the following non-linear differential equation given as equations according to [2].

$$A_3(U) - f(r) = 0, r \in \Omega \tag{27}$$

Subject to the boundary condition

$$B_3\left(U, \frac{\partial U}{\partial n}\right) = 0, r \in \Gamma \tag{28}$$

Where A_3 is a general differential operator, B_3 a boundary operator, $f(r)$ is a known analytical function and Γ is the boundary of the domain Ω. The operator A_3 can be divided into two parts L and N, where L is the linear part, and N is the nonlinear part. Equation (27) can be written as:

$$L(U) + N(U) - f(r) = 0, r \in \Omega.$$

The Homotopy Perturbation structure is shown as follows

$$H(V, h) = (1 - h)[L(V) - L(U_0)] + h[A(V) - f(r)] = 0 \tag{29}$$

where

$$V(r, P): \Omega \in [0,1] \to R. \tag{30}$$

In equation (29) $P \in [0,1]$ is an embedding parameter and $U_0$is the approximation that satisfies the boundary condition. It can be assumed that the solution of the equation (30) can be written as power series in h given as equations (31) to (32):

$$V = V_0 + hV_1 + h^2V_2 + \ldots \tag{31}$$

And the best approximation for the solution is: $U = lim\, v = v_0 + hv_1 + h^2v_2 + \cdots$

$$h \to 1. \tag{32}$$

The series (31) is convergent for most cases. However, the convergent rate depends on the nonlinear operator A (V)

3.2. Solution of the model equations using HPM

From differential equations 1 to 7

$$\frac{dS}{dt} + (\mu + \lambda_1)S - \omega V - \pi(1 - \theta) = 0, \tag{33}$$

$$\frac{dV}{dt} + (\mu + \omega)V - \theta\pi = 0, \tag{34}$$

$$\frac{dE}{dt} + (\sigma + \lambda_2 + \mu)E - \lambda_3 T - \lambda_1 S = 0, \tag{35}$$

$$\frac{dI_m}{dt} + (\gamma + \mu + r_1 + d_1)I_m - (1-\rho)\sigma E - (1-\phi)\lambda_2 E = 0, \quad (36)$$

$$\frac{dI_c}{dt} + (\mu + d_2 + r_3 + r_2)I_c - \rho\sigma E - \gamma I_m - \phi\lambda_2 E = 0, \quad (37)$$

$$\frac{dJ}{dt} + (\mu + d_3 + r_4)J - r_3 I_c = 0, \quad (38)$$

$$\frac{dT}{dI} + (\mu + \lambda_3)T - r_4 J - r_2 I_c - r_1 I_m = 0. \quad (39)$$

With the initial condition given as

$$S(0) = S_0, V(0) = V_0, E(0) = E_0, I_m(0) = I_{m0}, I_c(0) = I_{c0}, J(0) = J_0, T(0) = T_0. \quad (40)$$

Let

$$S = s_0 + hs_1 + h^2 s_2 + \ldots, \quad (41)$$

$$V = u_0 + hu_1 + h^2 u_2 + \ldots, \quad (42)$$

$$E = v_0 + hv_1 + h^2 v_2 + \ldots, \quad (43)$$

$$I_m = w_0 + hw_1 + h^2 w_2 + \cdots, \quad (44)$$

$$I_c = x_0 + hx_1 + h^2 x_2 + \ldots, \quad (45)$$

$$J = y_0 + hy_1 + h^2 y_2 + \ldots, \quad (46)$$

$$T = z_0 + hz_1 + h^2 z_2 + \ldots. \quad (47)$$

Applying HPM into equation (33)

$$(1-h)\frac{dS}{dt} + h\left[\frac{dS}{dt} + (\mu + \lambda_1)S - \omega V - \pi(1-\theta)\right] = 0. \quad (48)$$

Substitute equation (41) and (42) into (48)

$$(s_0^1 + hs_1^1 + h^2 s_2^1 + \ldots) + h\begin{bmatrix}(\mu + \lambda_1)(s_0 + hs_1 + h^2 s_2 + \ldots) - \omega \\ (u_0 + hu_1 + h^2 u_2 + \ldots) - \pi(1-\theta)\end{bmatrix} = 0.$$

Collecting the coefficient of power of h, we have

$$h^0 : s_0^1 = 0, \quad (49)$$

$$h^1 : s_1^1 + (\mu + \lambda_1)s_0 - \omega u_0 - \pi(1-\theta) = 0, \quad (50)$$

$$h^2 : s_2^1 + (\mu + \lambda_1)s_1 - \omega u_1 = 0. \quad (51)$$

From Equation (49) $s_0^1 = 0$, integrating both side $s_0 = D_1$ and applying initial condition $s_0(0) = S_0 = s_0$, $D_1 = S_0$ and $s_0 = S_0$.
From Equation (51)

$$s_1^1 = \pi(1-\theta) + \omega v_0 - (\mu + \lambda_1)s_0.$$

Integrate both sides and applying the initial condition we get:

$$s_1(t) = (\pi(1-\theta) + \omega v_0 - (\mu + \lambda_1)s_0)t. \quad (52)$$

Substitute Equations (41) and (42) into (52)

$$s_1(t) = (\pi(1-\theta) + \omega E_0 - (\mu + \lambda_1)S_0)t. \quad (53)$$

Applying HPM to Equation (34)

$$(1-h)\frac{dV}{dt} + h\left[\frac{dV}{dt} + (\mu + \omega)V - \pi\theta\right] = 0. \quad (54)$$

Substitute Equation (42) into (54)

$$(u_0^1 + hu_1^1 + h^2u_2^1 + \ldots) + h[(\mu + \omega)(u_0 + hu_1 + h^2u_2 + \ldots) - \pi\theta] = 0.$$

Collecting the coefficient of power of h, we have

$$h^0: u_0^1 = 0, \tag{55}$$

$$h^1: u_1^1 + (\mu + \omega)u_0 - \pi\theta = 0, \tag{56}$$

$$h^2: u_2^1 + (\mu + \omega)u_1 = 0. \tag{57}$$

From Equation (56)

$$u_1^1 = \pi\theta - (\mu + \omega)u_0.$$

Integrate both sides and applying the initial condition we have:

$$u_1(t) = (\pi\theta - (\mu + \omega)u_0)t. \tag{58}$$

Substitute Equation (42) into (60)

$$u_1(t) = (\pi\theta - (\mu + \omega)V_0)t. \tag{59}$$

From Equation (51)

$$s_2^1 = \omega u_1 - (\mu + \lambda_1)s_1. \tag{60}$$

Substitute Equation (59) and (53) into (60)

$$s_2^1 = \omega(\pi\theta - (\mu + \omega)V_0)t - (\mu + \lambda_1)(\pi(1 - \theta) + \omega E_0 - (\mu + \lambda_1)S_0)t,$$

$$s_2^1 = \big(\omega(\pi\theta - (\mu + \omega)V_0) - (\mu + \lambda_1)(\pi(1 - \theta) + \omega E_0 - (\mu + \lambda_1)S_0)\big)t.$$

Integrating both sides and applying the initial condition

$$s_2(t) = \big(\omega(\pi\theta - (\mu + \omega)V_0) - (\mu + \lambda_1)(\pi(1 - \theta) + \omega E_0 - (\mu + \lambda_1)S_0)\big)\frac{t^2}{2}. \tag{61}$$

Substitute the initial condition and equation (53) and (61) into (41)

$$S(t) = s_0 + hs_1 + h^2s_2 + \ldots,$$

$$S(t) = \lim_{\{h \to 1\}}(s_0 + hs_1 + h^2s_2 + \cdots),$$

$$S(t) = s_0 + s_1 + s_2 + \ldots,$$

hence,

$$S(t) = S_0 + (\pi(1 - \theta) + \omega E_0 - (\mu + \lambda_1)S_0)t + \big(\omega(\pi\theta - (\mu + \omega)V_0) - (\mu + \lambda_1)(\pi(1 - \theta) + \omega E_0 - (\mu + \lambda_1)S_0)\big)\frac{t^2}{2}.$$

Following the same process for other equations, we have:

$$V(t) = V_0 + (\pi\theta - (\mu + \omega)V_0)t + \big((\mu + \omega)(\pi\theta - (\mu + \omega)V_0)\big)\frac{t^2}{2},$$

$$E(t) = E_0 + \begin{pmatrix}\lambda_2 T_0 + \lambda_1 S_0 - \\ (\sigma + \lambda_2 + \mu)E_0\end{pmatrix}t + \begin{pmatrix}\lambda_2(r_4 J_0 + r_2 I_{c0} + r_1 I_{m0} - (\mu + \lambda_3)T_0) + \lambda_1 \\ (\pi(1 - \theta) + \omega E_0 - (\mu + \lambda_1)S_0) \\ -(\lambda_2 + \mu + \sigma)(\lambda_2 T_0 + \lambda_1 S_0 - (\sigma + \lambda_2 + \mu)E_0)\end{pmatrix}\frac{t^2}{2},$$

$$I_m(t) = I_{m0} + \big((1 - \rho)\sigma E_0 + (1 - \phi)\lambda_2 E_0 - (\gamma + \mu + r_1 + d_1)I_{m0}\big)t + \begin{pmatrix}\big((1 - \rho)\sigma + (1 - \theta)\big)(\lambda_2 T_0 + \lambda_1 S_0 - (\sigma + \lambda_2 + \mu)E_0) - (\gamma + r_1 + d_1 + \mu) \\ \Big(\big((1 - \rho)\sigma + (1 - \phi)\lambda_2\big)E_0 - (\gamma + \mu + r_1 + d_1)I_{m0}\Big)\end{pmatrix}\frac{t^2}{2},$$

$$I_c(t) = I_{c0} + (\phi\lambda_2 E_0 + \rho\sigma E_0 + \gamma I_{m0} - (\mu + d_2 + r_2 + r_3)xI_{c0})t + \begin{pmatrix} (\rho\sigma + \phi\lambda_2)(\lambda_2 T_0 + \lambda_1 S_0 - (\sigma + \lambda_2 + \mu)E_0) + \\ \gamma\big((1-\rho)\sigma E_0 + (1-\phi)\lambda_2 E_0 - (\gamma + \mu + r_1 + d_1)I_{m0}\big) - \\ (\mu + d_2 + r_2 + r_3)(\phi\lambda_2 E_0 + \rho\sigma E_0 + \gamma I_{m0} - (\mu + d_2 + r_2 + r_3)xI_{c0}) \end{pmatrix}\frac{t^2}{2},$$

$$J(t) = J_0 + \begin{pmatrix} r_3 I_{c0} - (\mu + d_3 + r_4) \\ J_0 \end{pmatrix} t + \begin{pmatrix} r_3(\phi\lambda_2 E_0 + \rho\sigma E_0 + \gamma I_{m0} - (\mu + d_2 + r_2 + r_3)xI_{c0}) \\ -(\mu + d_3 + r_4)(r_3 I_{c0} - (\mu + d_3 + r_4)J_0) \end{pmatrix}\frac{t^2}{2},$$

$$T(t) = T_0 + (r_4 J_0 + r_2 I_{c0} + r_1 I_{m0} - (\mu + \lambda_3)T_0)t + \begin{pmatrix} r_4(r_3 I_{c0} - (\mu + d_3 + r_4)J_0) + \\ r_2\begin{pmatrix}\phi\lambda_2 E_0 + \rho\sigma E_0 + \gamma I_{m0} - \\ (\mu + d_2 + r_2 + r_3)xI_{c0}\end{pmatrix} + \\ r_1\begin{pmatrix}(1-\rho)\sigma E_0 + (1-\phi)\lambda_2 E_0 - \\ (\gamma + \mu + r_1 + d_1)I_{m0}\end{pmatrix} \\ -(\mu + \lambda_3)\begin{pmatrix} r_4 J_0 + r_2 I_{c0} + r_1 I_{m0} \\ -(\mu + \lambda_3)T_0 \end{pmatrix} \end{pmatrix}\frac{t^2}{2}.$$

4. RESULTS

4.1. Numerical parameters

In this section, we give the values and source of the parameters used for simulating the model. We used the following initial values for $S(t)$ = 160,840,589, $E(t)$ = 1,700,000, $I_m(t)$ = 90000, $I_c(t)$ = 10,400,000, $J(t)$ = 1,000,000, $T(t)$ = 1,109,000 and $V(t)$ = 8,000,000. $N = S(t) + E(t) + I_m(t) + I_c(t) + J(t) + T(t) + V(t) = 206{,}139{,}589$.

Table 2. The parameters value used for the model

Parameters	Value	Source
π	2,895,131	Estimated
μ	0.018	Estimated
d_1	0.0365	Assumed
d_2	0.68	[21]
d_3	0.1	[22]
r_1	0.02	Assumed
r_2	0.02	Assumed
r_3	0.00375	[21]
θ	0.020	[22]
ρ	0.075	[22]
ϕ	0.3	Assumed
λ_2	0.2	Assumed
λ_3	0.2	Assumed
σ	0.01	Assumed

4.2. Graphical representation of solutions of the model equation

The graphical representations are from the analytical solutions of the model equations. They are plotted using MAPLE software.

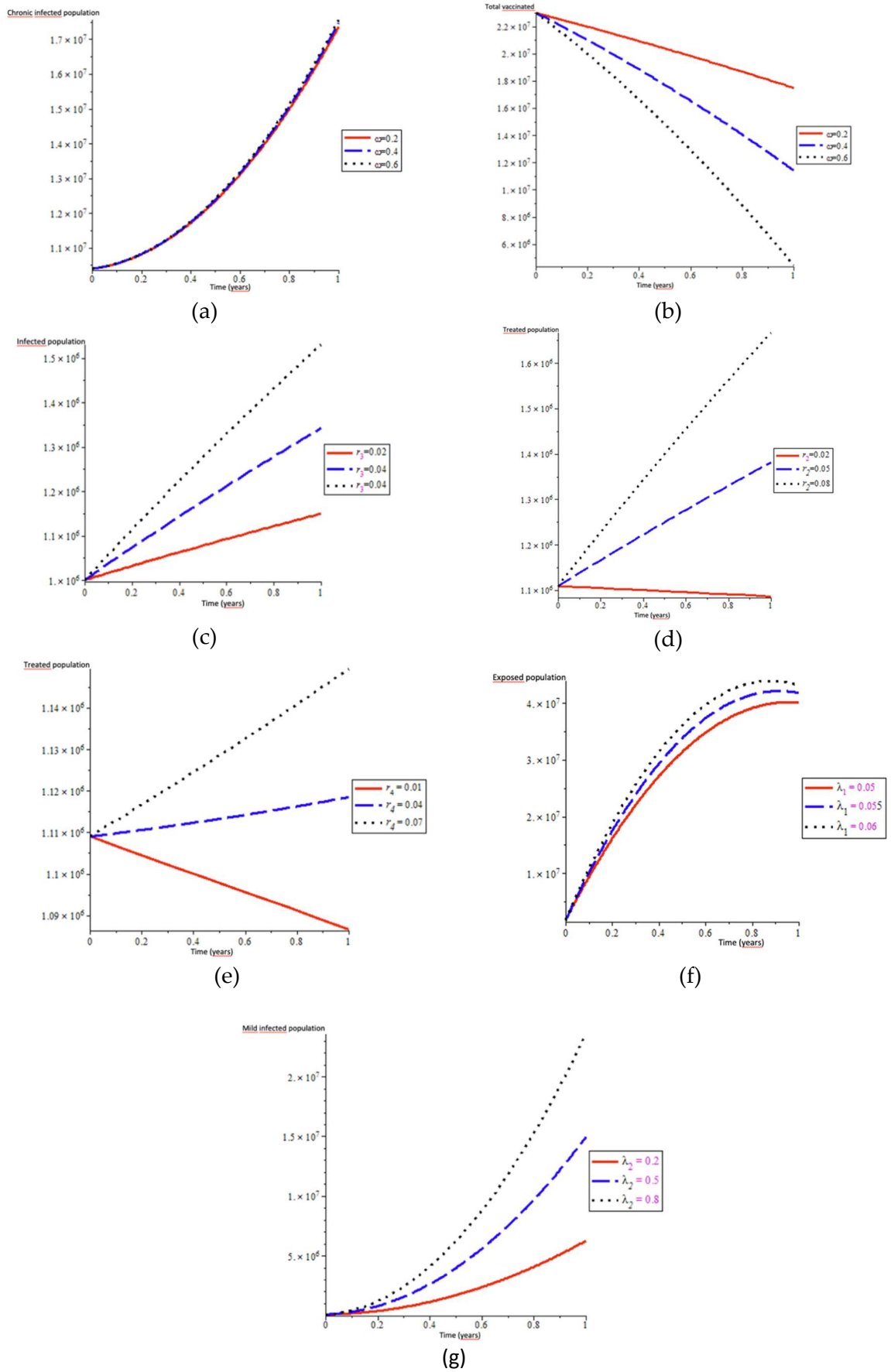

Fig. 2. (a) Effect of waning rate of vaccine on the chronic class. (b) Effect of waning rate of vaccine on vaccinated class. (c) Effect of isolation rate on the isolated compartment. (d) Treated individual against time for different values of recovery rate for chronic class. (e) Effect of recovery rate for those in isolated class. (f) Impact of effective interaction between susceptible and infected classes. (g) Mild TB individual against time for different values of transmission rate.

5. DISCUSSION

Figure 2a is the graph of chronic infected individuals against time for different values of waning rate of vaccine. We carried out simulations by varying waning rate of vaccine as 0.2, 0.4 and 0.6. It could be observed that different level of waning rate does not have effect on the infected population. For different level of waning rate, the TB infection continues to persist in the given population.

Figure 2b is the graph of vaccinated individuals against time for different values of waning rate of vaccine. It was observed that the vaccinated population decreases with increase in the vaccination rate. Therefore, high waning rate of vaccine reduces the vaccinated population and thus puts them at the risk of contracting the disease.

Figure 2c is the graph of isolated infectious individuals against time at different values of Progression rate from chronic TB class to isolated infected class. It was observed that the number of isolated Individuals increases as Progression rate from chronic TB class to isolated infected class Increases.

Figure 2d is the graph of treated TB individuals against time. It was observed that the number of treated TB individual increases as the recovery rate among the chronic TB individual increases. This implies that increase in the progression rate will lead to increase in number of individuals with chronic TB disease.

Figure 2e is the graph of recovered individual against time. The lower the treated rate the lower the number of recovered individuals. The lowest percentage almost decrease to zero. This shows that as the recovered are treated, they move to Chronic TB population.

Figure 2f is the graph of exposed individual against time for different values of contact rate. We can observe that infected individuals increase as contact rate increases. The figure illustrates the great influence of effective contact rate on the exposed population.

Figure 2g is the graph of mild TB individual against time. It was observed that the number of mild TB individual increases as the transmission rate from the exposed to the chronic individuals increases.

6. CONCLUSION

In this study, a mathematical model of tuberculosis transmission dynamics incorporating treatment, isolation and vaccination using the system of first order ordinary differential equations was developed and analyzed. It was discovered that the model has two equilibria. The equilibrium states were obtained and analyzed for their stability relatively to the effective reproduction number. The result shows that, the disease-free equilibrium was stable. We are able to show that the tuberculosis infectious free equilibrium is locally and globally asymptotically stable if $R_0 < 1$. The analytical solution was obtained using Homotopy Perturbation Method and effective reproduction number was computed in order to measure the relative impact for individual or combined intervention for effective disease control.

The graphs illustrate the impact of a combined effect of contact rate, waning rate of vaccine and rate of isolation. One can observe that this combines effects reduce the size of infected compartments. Thus, the simultaneous increase of effectiveness of vaccination rate, isolation rate and treatment rate are effective control measures against TB infection.

The model shows that the spread of tuberculosis infection depends largely on the contact rate, hence the ministry of health and other health workers should emphasize on the improvement in early detection of tuberculosis infection cases, so that transmission can be minimized. Infected individuals should be isolated and treated immediately and individuals infected with tuberculosis should be given antiretroviral drugs immediately. In future work, we intent to incorporate optimal control strategy into the model for greater insight into the dynamics.

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