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A Two-Group Model of TB in a Crowded Environment

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Abstract: In this paper, we introduce a two-group epidemic model of the dynamics of tuberculosis in a prison system. The total population of inmates consists of two groups: The sentenced inmates and those inmates who are awaiting trial. The threshold parameter for local stability is computed and analysed. We also prove the global stability of the disease free equilibrium using a Lyapunov function. We apply the model to South African data on tuberculosis and we observe consistency between the model prediction and the data. Numerical results are presented to illustrate the analytical results.

Keywords: Global stability, Inflow of infecteds, Remand inmates, Removal rate, Sentenced inmates, Two-group TB model.

1 Introduction

Tuberculosis (TB) is a major cause of illness and it ranks among the top ten causes of death worldwide [1]. Although TB disease is prevalent in a variety of communities, its transmission rate is higher in crowded communities, especially under poverty conditions and among people living in small buildings that lack ventilation. Prisons have TB prevalence which is relatively higher than that in the ambient community [2]. In many countries, prisoners exceed the prison capacity. For the case of South Africa, see [3] in this regard. Modeling of infectious diseases in prisons has been addressed in [5], [6] [7] and [8]. Paper [5] includes an analysis of SI-model for a prison population, while [8] presents a disease model with four compartments on a prison. A model of TB in a prison is shown in [6]. The modeling in [7] informs specific measures for reducing the transmission of TB in a specific prison system. Paper [9] also presents a study of TB in a prison using a model presented originally in [10]. In a prison complex, there are different categories of inmates. We may find remand inmates as well as prisoners that have already been sentenced. With compartmental modeling, it is possible that these two categories of inmates can be distinguished by following the two-group modeling approach.

In this paper, we propose a two-group deterministic compartmental TB model that considers the dynamics of TB in a prison system. Our model divides the prison population into two groups: The sentenced individuals and the remand individuals. This division of the prison population will enable us to accurately monitor the dynamics of TB disease in each group. We compute the parameters of the model using South African data, as in [9]. Furthermore, we use mathematical analysis combined with simulations to investigate the behaviour of the model. With two sub-populations, our current model is completely different from the other related papers. The two-group structure aims for better accuracy. Another highlight of the present paper is the proof of global stability of the disease free equilibrium in the restricted model (without inflow of infected). The latter theorem informs the possibility or not of eliminating TB from the prison on condition that the new incoming inmates are free from TB infection.

2 The model

To investigate the dynamics of TB in a prison population, we divide the total prison population (N) into sentenced sub-population (N_s) and the awaiting trial (remand) sub-population (N_a). We further subdivide the sentenced sub-population into compartments, such as susceptible class (S), exposed class (E), infectious class (I), treatment class (T) the awaiting trial sub-population as susceptible class (U), exposed class (L), infectious class

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(J) and treatment class (H). Susceptible individuals are recruited into the sentenced and awaiting trial sub-population at a constant rate ρA and μB, respectively. We assume that there is inflow of susceptible, exposed and infected individuals into the susceptible class, exposed class and the infected class at a rate $f_S\rho A, f_E\rho A, f_I\rho A, f_U\mu B, f_L\mu B$ and $f_J\mu B$, respectively into the sentenced and the remand sub-population. We assume that $f_S+f_E+f_I=1$ and $f_U+f_L+f_J=1$. People under TB treatment are considered to be unfit to commit crime and we assume that there is no inflow of infectives into the treatment class for any of the two sub-populations. The model is described by eight ordinary differential equation, as follows:

$$\begin{aligned}
\dot{S} &= f_S\rho A-\alpha S(I+\varphi J)-\rho S,\\
\dot{E} &= f_E\rho A+\alpha S(I+\varphi J)-\zeta E(I+\varphi J)\\
&\quad -(\rho+k)E+gT,\\
\dot{I} &= f_I\rho A+\zeta E(I+\varphi J)+kE-(\rho+\delta+p)I,\\
\dot{T} &= pI-(\rho+g)T,\\
\dot{U} &= f_U\mu B-\beta U(J+\psi I)-\mu U,\\
\dot{L} &= f_L\mu B+\beta U(J+\psi I)-\xi L(J+\psi I)\\
&\quad -(\mu+\iota)L+hH,\\
\dot{J} &= f_J\mu B+\xi L(J+\psi I)+\iota L-(\mu+\gamma+q)J,\\
\dot{H} &= qJ-(\mu+h)H.
\end{aligned} \tag{1}$$

It is important to note that in general populations, removal of individuals out of the system only occurs due to death. In this model, as in model [9], removal occurs by death or by discharge from prison. These rates of removal from different sub-populations are denoted by ρ and μ, respectively. The rate at which individuals die due to TB disease is denoted by δ and γ in each sub-population, respectively. Susceptible individuals who are in the sentenced and awaiting trial sub-population acquire TB infection at a rate $\alpha S(I+\varphi J)$ and $\beta U(J+\psi I)$ and move into the exposed classes E and L, respectively. Parameters α and β are the transmission coefficients from susceptible classes to the exposed class, respectively. The cross-effect between the sentenced and awaiting trial individuals is represented by φ and ψ, respectively. Exposed individuals leave the exposed class (E) and (L) for infectious class (I) and (J) at a rate kE and ιJ, respectively. Exposed individuals who become infectious in the sentenced and awaiting trial sub-population move to the infectious class at a rate $\zeta E(I+\varphi J)$ and $\xi L(J+\psi I)$, where ζ and ξ represent the transmission coefficient from exposed class to the infectious class, respectively. Infectious individuals receive treatment and move to the treatment class at a rate pI and qJ, where p and q are treatment rates, respectively. Successfully treated individuals move to exposed class at a rate gT and hH, respectively.

The total population for sentenced and awaiting trial sub-population is given by

$$N_s(t)=S(t)+E(t)+I(t)+T(t)$$

and

$$N_a(t)=U(t)+L(t)+J(t)+H(t),$$

respectively.

We first analyse the model without the inflow of infecteds, i.e. when

$$f_E=f_I=f_L=f_J=0, \text{ and } f_S=f_U=1,$$

and a disease free equilibrium exist. In this special case, system (1) has a disease free equilibrium

$$\begin{aligned}
P_0^* &= (S_0,E_0,I_0,T_0,U_0,L_0,J_0,H_0)\\
&= (P_{s0}^*,P_{a0}^*),
\end{aligned}$$

where $P_{s0}^*=(A,0,0,0)$ and $P_{a0}^*=(B,0,0,0)$. Convergence to P_0^* means that the disease will vanish from the prison system. This is similar to the convergence to P_{s0}^* which is the sentenced subgroup and P_{a0}^* the awaiting trial individuals.

3 Invariant region and positivity of solutions

It is important to prove that all the state variables of system (1) are non-negative for all time. In this section, a solution of the system (1) will be denoted by

$$X(t)=(S(t),E(t),I(t),T(t),U(t),L(t),J(t),H(t)). \tag{2}$$

Proposition 1. *Given any $t_0>0$, suppose that we have a solution $X(t)$ of system (1) over an interval $0<t<t_0$ with*

$N_s(0)<A$ *and* $N_a(0)<B$

and with $X(t)\in\mathbb{R}_+^8$ for all $t_0\geq 0$. Then, $N_s(t)\leq A$ and $N_a(t)\leq B$.

Proof. From model system (1), we get

$$\frac{dN_s}{dt}=\rho(A-N_s)-\delta I, \tag{3}$$

$$\frac{dN_a}{dt}=\mu(B-N_a)-\gamma J, \tag{4}$$

respectively. Thus we have $\frac{dN_s}{dt}<0$ for $N_s>A$ and $\frac{dN_a}{dt}<0$ for $N_a>B$. This completes the proof.

We consider all solutions of system (1) in the following positively invariant subset $\Lambda\in\mathbb{R}^8$:

$$\Lambda=\Lambda_s\times\Lambda_a,$$

where

$$\begin{aligned}
\Lambda_s &= \{(S,E,I,T)\in[0,\infty)\mid S+E+I+T\leq A\},\\
\Lambda_a &= \{(U,L,J,H)\in[0,\infty)\mid U+L+J+H\leq B\}.
\end{aligned}$$

Theorem 2. *There is a unique solution $X(t)$ of system (1) on $t \geq 0$ for any given initial value $X(0) \in \mathbb{R}^8_+$.*

Proof. Suppose to the contrary that there exists $t_0 > 0$ such that

$$X(t_0)) \notin \mathbb{R}^8_+.$$

Let

$$\tau = \inf \{t > 0 : X(t)) \notin \mathbb{R}^8_+\}.$$

We define a function V_0 as follows:

$$\begin{aligned} V_0(t) = &\Big(S(t) - \ln S(t)\Big) + \Big(E(t) - \ln E(t)\Big) \\ &+\Big(I(t) - \ln I(t)\Big) + \Big(T(t) - \ln T(t)\Big) \\ &+\Big(U(t) - \ln U(t)\Big) + \Big(L(t) - \ln L(t)\Big) \\ &+\Big(J(t) - \ln J(t)\Big) + \Big(H(t) - \ln H(t)\Big). \end{aligned} \tag{5}$$

Each of the terms bracketed in (5) is positive for every $t < \tau$.

Taking the derivative of V_0, we now have

$$\begin{aligned} \dot{V}_0 = &\Big[\Big(1 - \frac{1}{S}\Big)\Big(\rho A - \alpha S(I + \varphi J) - \rho S\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{E}\Big)\Big(\alpha S(I + \varphi J) - \zeta E(I + \varphi J) \\ &-(\rho + k)E + gT\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{I}\Big)\Big(\zeta E(I + \varphi J) + kE - (\rho + \delta + p)I\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{T}\Big)\Big(pI - (\rho + g)T\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{U}\Big)\Big(\mu B - \beta U(J + \psi I) - \mu U\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{L}\Big)\Big(\beta U(J + \psi I) - \xi L(J + \psi I) \\ &-(\mu + \iota)L + hH\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{J}\Big)\Big(\xi L(J + \psi I) + \iota L - (\mu + \gamma + q)J\Big)\Big] \\ &+\Big[\Big(1 - \frac{1}{H}\Big)\Big(qJ - (\mu + h)H\Big)\Big]. \end{aligned} \tag{6}$$

Expanding (6), we get

$$\begin{aligned} \dot{V}_0 = &\rho A + \mu B - \rho S - \frac{1}{S} f_S \rho A + (I + \varphi J) + \rho - \rho E \\ &-\frac{1}{E} f_E \rho A - \frac{1}{E} \alpha S(I + \varphi J) + \zeta(I + \varphi J) + (\rho + k) \\ &-\frac{1}{E} gT - (\rho + \delta)I \\ &-\frac{1}{I} f_I \rho A - \frac{1}{I} \zeta E(I + \varphi J) - \frac{1}{I} kE + (\rho + \delta + p) \\ &-\rho T - \frac{1}{T} pI + (\rho + g) \\ &-\mu U - \frac{1}{U} f_U \mu B + \beta(J + \psi I) + \mu - \mu L - \frac{1}{L} f_L \mu B \\ &-\frac{1}{L} \beta U(J + \psi I) + \xi(J + \psi I) + (\mu + \iota) \\ &-\frac{1}{L} hH - (\mu + \gamma)J - \frac{1}{J} f_J \mu B - \frac{1}{J} \xi L(J + \psi I) \\ &-\frac{1}{J} \iota L + (\mu + \gamma + q) - \mu H - \frac{1}{H} qJ + (\mu + h). \end{aligned} \tag{7}$$

Eliminating the negative terms in (7), we obtain the following inequality:

$$\begin{aligned} \dot{V}_0 \leq &\rho A + \mu B + (\alpha + \zeta)(I + \varphi J) + (\beta + \xi)(J + \psi I) \\ &+4\rho + 4\mu + k + \delta + p + g + \iota + \gamma + q + h. \end{aligned} \tag{8}$$

Consequently, we have

$$\dot{V}_0 \leq M_1, \tag{9}$$

where

$$\begin{aligned} M_1 = &\rho A + \mu B + (\alpha + \zeta)(A + \varphi B) + (\beta + \xi)(B + \psi A) \\ &+4\rho + 4\mu + k + \delta + p + g + \iota + \gamma + q + h. \end{aligned} \tag{10}$$

Taking the integral in (9) from 0 to t, we obtain

$$\begin{aligned} V_0(t) &= V_0(0) + \int_0^t \dot{V}_0(s) ds \\ &\leq V_0(0) + M_1 t \ \leq \ V_0(0) + M_1 \tau. \end{aligned} \tag{11}$$

Now, we note that

$$\lim_{x \to 0^+} (x - \ln x) = \infty,$$

so we must have

$$\lim_{t \to \tau^-} V(t) = \infty.$$

On the other hand, we have from (11) that V_0 is bounded over the interval $[0, \tau)$. This is a contradiction.

The basic reproduction number, R_0 of model system (1), is computed using the next generation matrix approach which has been developed by Van den Driessche and Watmough [11]. Using the notation in [11], the matrices F and V for new infection term and the remaining transfer terms, respectively, are given by:

$$F=\begin{pmatrix} 0 & \alpha A & 0 & \alpha\varphi A & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta\psi B & 0 & \beta B & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and V is the matrix:

$$\begin{pmatrix} \rho+k & 0 & -g & 0 & 0 & 0 \\ -k & \rho_0 & 0 & 0 & 0 & 0 \\ 0 & -p & \rho+g & 0 & 0 & 0 \\ 0 & 0 & 0 & \mu+\iota & 0 & -h \\ 0 & 0 & 0 & -\iota & \mu_0 & 0 \\ 0 & 0 & 0 & 0 & -q & \mu+h \end{pmatrix},$$

with $\rho_0=(\rho+\delta+p)$ and $\mu_0=(\mu+\gamma+q)$. The matrix FV^{-1} can now be calculated as

$$\begin{pmatrix} \frac{k\alpha A(\rho+g)}{\theta_1} & \frac{\alpha A\lambda_1}{\theta_1} & \frac{k\alpha gA}{\theta_1} & \frac{\alpha\iota\psi A(\mu+h)}{\theta_2} & \frac{\alpha\psi A\lambda_2}{\theta_2} & \frac{\alpha\iota\psi hA}{\theta_2} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta\psi kB(\rho+g)}{\theta_1} & \frac{\beta\psi B\lambda_1}{\theta_1} & \frac{\beta\psi gkB}{\theta_1} & \frac{\beta\iota B(\mu+h)}{\theta_2} & \frac{\beta B\lambda_2}{\theta_2} & \frac{\beta\iota hB}{\theta_2} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where $\rho_1=(\rho+k+g)$,
$\mu_1=(\mu+\iota+h)$,
$\lambda_1=(\rho+g)(\rho+k)$,
$\lambda_2=(\mu+h)(\mu+\iota)$,
$\theta_1=gk(\rho+\delta)+\rho\rho_0\rho_1$
and
$\theta_2=h\iota(\mu+\gamma)+\mu\mu_0\mu_1$.

The basic reproduction number, R_0, is defined as the spectral radius of the next generation matrix [11] and takes the form:

$$R_0=\frac{1}{2}(R_{s1}+R_{a2})+\frac{1}{2}\sqrt{(R_{s1}-R_{a2})^2+4R_{a1}R_{s2}}, \qquad (12)$$

with

$$R_{s1}=\frac{k\alpha A(\rho+g)}{\theta_1}, \qquad R_{s2}=\frac{\beta\psi kB(\rho+g)}{\theta_1},$$
$$R_{a1}=\frac{\alpha\iota\psi A(\mu+h)}{\theta_2} \text{ and } R_{a2}=\frac{\beta\iota B(\mu+h)}{\theta_2}.$$

The parameter R_0 gives a threshold condition that the disease will be eradicated from the prison system if $R_0\leq 1$, and if $R_0>1$, then the disease will persist into the prison system.

4 Global stability of disease free equilibrium

We now investigate the global stability of a disease free equilibrium of system (1) using the Lyapunov function approach. To conduct the analytical analysis of global stability of disease free equilibrium, we assume that there is no inflow of infecteds, i.e. $f_E=f_I=f_L=f_J=0$. We introduce the following invariants of model (1), which will serve to describe global asymptotic stability of P_0^*.
Let

$$\alpha_0=\max\left\{\alpha,\frac{\rho\zeta}{k}\right\} \text{ and } \beta_0=\max\left\{\beta,\frac{\mu\xi}{\iota}\right\}. \qquad (13)$$

We introduce the following two numbers:

$$R_{g1}=\frac{k(\rho+g)(\alpha_0A+\beta_0\psi B)}{\rho(\rho+\delta+p)(\rho+k+g)+gk(\rho+\delta)},$$

and

$$R_{g2}=\frac{\iota(\mu+h)(\varphi\alpha_0A+\beta_0B)}{\mu(\mu+\gamma+q)(\mu+\iota+h)+h\iota(\mu+\gamma)}.$$

Note that

$$\rho_0(\rho+k)(\rho+g)-pkg=\rho\rho_0\rho_1+gk(\rho+\delta),$$
$$\mu_0(\mu+\iota)(\mu+h)-q\iota h=\mu\mu_0\mu_1+h\iota(\mu+\gamma).$$

Global stability of the disease free equilibrium is extremely important since it means that whatever the initial state, the disease definitely vanishes from the population.

Theorem 3. *Consider the case when there is no inflow of infected cases in system (1), i.e. $f_E=f_I=0=f_L=f_J$. Suppose that $R_{g1}<1$, and $R_{g2}<1$. Then, the disease free equilibrium is globally asymptotically stable.*

Proof. The condition $R_{g1}<1$ implies that:

$$k(\rho+g)(\alpha_0A+\beta_0\psi B)-gk(\rho+\delta)-\rho\rho_0\rho_1<0,$$
$$k(\rho+g)(\alpha_0A+\beta_0\psi B)+pkg-\rho_0(\rho+k)(\rho+g)<0.$$

It is possible to find, consecutively, $a_0>0$, $\varepsilon_1>0$ and $\varepsilon_2>0$ such that:

$$[a_0+k(\rho+g)][\alpha_0A+\beta_0\psi B]+p(kg+\varepsilon_1)$$
$$-\rho_0[(\rho+k)(\rho+g)-\varepsilon_2]<0. \qquad (14)$$

Let a_0 be as above and

$$a_1 = k(\rho+g),$$
$$a_2 = (\rho+k)(\rho+g)-\varepsilon_2 > 0,$$
$$a_3 = kg+\varepsilon_1.$$

Likewise, the condition $R_{g2} < 1$ implies that:

$$\iota(\mu+h)(\varphi\alpha_0 A+\beta_0 B)-h\iota(\mu+\gamma)-\mu\mu_0\mu_1 < 0,$$
$$\iota(\mu+h)(\varphi\alpha_0 A+\beta_0 B)+q\iota h-\mu_0(\mu+\iota)(\mu+h) < 0.$$

It is also possible to find, consecutively, $b_0 > 0$, $\varepsilon_3 >$ and $\varepsilon_4 > 0$ such that:

$$[b_0+\iota(\mu+h)][\varphi\alpha_0 A+\beta_0 B]+q(\iota h+\varepsilon_3 -\mu_0[(\mu+\iota)(\mu+h)-\varepsilon_4] < 0. \quad (15)$$

Let b_0 be as above. Now, we introduce numbers b_i, as follows:

$$b_1 = \iota(\mu+h),$$
$$b_2 = (\mu+\iota)(\mu+h)-\varepsilon_4 > 0,$$
$$b_3 = \iota h+\varepsilon_3.$$

We define the following function V, which we shall prove to be a Lyapunov function to guarantee the global asymptotically stable:

$$V = a_0(A-S)+a_1E+a_2I+a_3T +b_0(B-U)+b_1L+b_2J+b_3H.$$

We calculate the time derivative:

$$\begin{aligned}\dot{V} &= -a_0[\rho(A-S)+\alpha S(I+\varphi J)]\\ &\quad +a_1[\alpha S(I+\varphi J)-\zeta E(I+\varphi J)-(\rho+k)E+gT]\\ &\quad +a_2[\zeta E(I+\varphi J)+kE-\rho_0 I]+a_3[\rho A+pI\\ &\quad -(\rho+g)T]-b_0[\mu(B-U)+\beta U(J+\psi I)]\\ &\quad +b_1[\beta U(J+\psi I)-\xi L(J+\psi I)-(\mu+\iota)L+hH]\\ &\quad +b_2[\xi L(J+\psi I)+\iota L-(\mu+\gamma+q)J]\\ &\quad +b_3[\mu B+qJ-(\mu+h)H],\\ &= -a_0\rho(A-S)+E[a_2k-a_1(\rho+k)]\\ &\quad +I[\alpha S(a_0+a_1)+\zeta E(a_2-a_1)+\beta\psi U(b_0+b_1)\\ &\quad +\xi\psi L(b_2-b_1)\\ &\quad +a_3p-a_2\rho_0]+T[a_1g-a_3(\rho+g)]\\ &\quad -b_0\mu(B-U)+L[b_2\iota-b_1(\mu+\iota)]+J[\beta U(b_0+b_1)\\ &\quad +\xi L(b_2-b_1)+\alpha\varphi S(a_0+a_1)+\zeta\varphi E(a_2-a_1)\\ &\quad +b_3q-b_2\mu_0]+H[b_1h-b_3(\mu+h)]. \quad (16)\end{aligned}$$

Therefore from (16) we now have the following inequality:

$$\begin{aligned}\dot{V} &\le -a_0\rho(A-S)+E[a_2k-a_1(\rho+k)]\\ &\quad +I[\alpha_0A(a_0+a_1)+\beta_0\psi B(b_0+b_1)+a_3p-a_2\rho_0]\\ &\quad +T[a_1g-a_3(\rho+g)]-b_0\mu(B-U)\\ &\quad +L[b_2\iota-b_1(\mu+\iota)]\\ &\quad +J[\beta_0B(b_0+b_1)+\alpha_0\varphi A(a_0+a_1)+b_3q-b_2\mu_0]\\ &\quad +H[b_1h-b_3(\mu+h)].\end{aligned}$$

We obtain $\dot{V}$ as

$$\dot{V} \le -a_0\rho(A-S)+p_EE+p_II+p_TT -b_0\mu(B-U)+p_LL+p_JJ+p_HH, \quad (17)$$

where the coefficients are

$$\begin{aligned}p_E &= a_2k-a_1(\rho+k),\\ p_I &= \alpha_0A(a_0-a_1)+\beta_0\psi B(b_0+b_1)+a_3p-a_2\rho_0,\\ p_T &= a_1g-a_3(\rho+g),\\ p_L &= b_2\iota-b_1(\mu+\iota),\\ p_J &= \beta_0B(b_0+b_1)+\alpha_0\varphi A(a_0+a_1)+b_3q-b_2\mu_0,\\ p_H &= b_1h-b_3(\mu+h).\end{aligned}$$

Now, we check that these coefficients are negative,

$$\begin{aligned}p_E &= a_2k-a_1(\rho+k)\\ &= k(\rho+k)(\rho+g)-\varepsilon_2k-k(\rho+k)(\rho+g)\\ &= -\varepsilon_2k.\end{aligned}$$

Also

$$p_L = -\varepsilon_4\iota < 0.$$

Now, we have

$$\begin{aligned}p_T &= a_1g-a_3(\rho+g) = kg(\rho+g)-(kg+\varepsilon_1)(\rho+g)\\ &= -\varepsilon_1(\rho+g) < 0.\end{aligned}$$

Similarly,

$$p_H = -\varepsilon_3(\mu+h) < 0. \quad (18)$$

Now we check

$$\begin{aligned}p_I &= \alpha_0A(a_0-a_1)+\beta_0\psi B(b_0+b_1)+a_3p-a_2\rho_0,\\ &= \alpha_0A[a_0-k(\rho+g)]+\beta_0\psi B[b_0+\iota(\mu+h)]\\ &\quad +p(kg+\varepsilon_1)-\rho_0(\rho+k)(\rho+g)-\varepsilon_2\rho_0.\end{aligned}$$

Hence, by condition (14), $p_I < 0$.
Likewise,

$$\begin{aligned}p_J &= \beta_0B[b_0+\iota(\mu+h)]+\alpha_0\varphi A[a_0+k(\rho+g)]\\ &\quad +q(\iota h+\varepsilon_3)-\mu_0(\mu+\iota)(\mu+h)-\varepsilon_4\mu_0\\ &\le 0.\end{aligned}$$

This proves that

$$\dot{V}(A-S,E,I,T,B-U,L,J,H). \le 0$$

Hence, $\dot{V}$ is negative definite. Therefore, P_0^* is globally asymptotically stable.

Theorem 3 asserts that TB can be eradicated in a prison system of the type of this model if there is no inflow of infected individuals, and the invariants R_{g1} and R_{g2} are below the threshold value of unity.

5 Numerical values of parameters

Statistics on the duration of the incarceration of remand detainees were unavailable. A 2011 report [3, 2nd paragraph on p5] implies that remand incarceration in South Africa had a median value of the above 3 months, and that there are cases of remand custody in prison that continues for years. On this basis, we suggest a rough estimate of 12 weeks for the average duration of remand custody. This value is significantly higher than the nominal value that was used in [9]. It gives us a value of μ_p as

$$\mu_p = \frac{1}{12} \text{ week}^{-1} = \frac{52}{12} \text{ year}^{-1} = 4.333 \text{ year}^{-1}.$$

Similarly, as in [9], we calculate an estimate for the removal rates μ and ρ using Table 1. We further assume, as in [9], that on average, a sentenced inmate completes 75% of sentenced time. Then, we obtain the release rate

$$\rho_p = 0.1249 \text{ year}^{-1}.$$

In [9], we calculated a value for the general mortality rate excluding deaths due to TB. This numerical value (0.003628) will be taken as the common value of μ_m and ρ_m. Thus, we can calculate the values of μ and ρ as:

$$\mu = \mu_p + \mu_m - \mu_m\mu_p = 4.335 \text{ year}^{-1},$$
$$\rho = \rho_p + \rho_m - \rho_m\rho_p = 0.1281 \text{ year}^{-1}.$$

Since the remand inmates do not stay in prison for a long time, we can estimate the disease mortality for this group as being the same as in the outside population. We assume the value $\gamma = 0.3 \text{ year}^{-1}$ as in the reference [4].

For sentenced inmates, the disease induced mortality rate is assumed to yield the same expected number of deaths due to TB as in the model of [9]. Thus, we take

$$\delta = 0.01876 \times \frac{N}{N_s} = 0.02616 \text{ year}^{-1}.$$

The rest of the parameters are evaluated along the same lines as in [9].

4.1. Effective Contact rates

The contact rates for sentenced and awaiting trial individuals are computed using a lower bound for the effective rates c_0 as in [9], given by:

$$\alpha = 7.1351\times10^{-5} \text{ year}^{-1}$$

and

$$\beta = 1.8303\times10^{-4} \text{ year}^{-1},$$

respectively. The transmission coefficients between the exposed class and the infectious class are given by

$$\zeta = \frac{k}{2N_s} \text{ and } \xi = \frac{\iota}{2N_a},$$

respectively, as in [9].

4.2. Other parameters

For sentenced individuals, the progression rate from the exposed class to the infectious class is the same as in [9], which is $k = 0.05$. As for remand inmates, we assume ι to be the same as the general population $\iota = 0.1$ [10].

Sentenced inmates receive treatment at the rate $p = 0.5$ and the remand inmates at $q = 0.3$, from [9]. Sentenced inmates recover and progress to the exposed class after a successful treatment at a rate of $g = 2(10/N_s)$ and remand inmates at $h = 2(10/N_a)$, as in [9].

The inflow of infectives is obtained from [9]

$$f_S = 0.2,\ f_U = 0.2,\ f_E = 0.74,\ f_L = 0.74,\ f_I = 0.06,$$
$$\text{and } f_J = 0.06.$$

The initial values are obtained by splitting the values of the initial values of the one-group model in [9]. The remand population constitutes a fraction $\frac{2}{7}$ of the prison population, so we deduce the following initial values:

$$S = 23500,\ E = 78800,\ I = 2300,\ T = 13000,$$
$$U = 9300,\ L = 31000,\ J = 900,\ \text{and } H = 5200.$$

Table 1: Model parameters

Parameter	Estimated value	Source
μ	4.335 year^{-1}	[12], [9]
ρ	0.1281 year^{-1}	[12], [9]
γ	0.3 year^{-1}	[9]
δ	$0.02616 \text{ year}^{-1}$	[9]
p	0.50 year^{-1}	[10]
q	0.30 year^{-1}	[10]
k	0.05 year^{-1}	[10]
ι	0.1 year^{-1}	[10], [9]
h	$20/N_s$	[10], [9]
g	$20/N_a$	[10], [9]
ζ	$k/2N_s$	[9]
ξ	$\iota/2N_a$	[9]

Table 2: Contact rates parameters and inflow of infectives

Parameter	Estimated value	Source
α	0.000071351 year^{-1}	[9]
β	0.00018303 year^{-1}	[9]
f_S, f_E, f_I	0.2, 0.74, 0.06	[9]
f_U, f_L, f_J	0.2, 0.74, 0.06	[9]

Table 3: Initial conditions

Parameter	Estimated value	Source
N	164000	[13]
N_s	118000	[13]
N_a	46000	[13]
S	23500	[13], [9]
E	78800	[13], [9]
I	2300	[13], [9]
T	13000	[13], [9]
U	9300	[13], [9]
L	31000	[13], [9]
J	900	[13], [9]
H	5200	[13], [9]

6 Numerical Simulations

A two-group model system (1) is simulated using South African real data and these parameter values are presented in Table 1, Table 2 and Table 3.

5.1. Simulations without the inflow of infective

We proceed using parameters of Table 1 and Table 3 to analyse the simulation results in the absence of the inflow of infectives. The trajectory plot of the two-group model system (1) are presented in Figure 1 when $R_{g1} < 1$ and $R_{g2} < 1$. To obtain different values of R_{g1} and R_{g2}, we vary the values of $\alpha = 0.0000142$ and $\beta = 0.00000904$. We observed that the trajectories of the two-group model (1) converges to disease free equilibrium. Therefore, the disease will disappear in both sub-populations as Theorem 3 asserts. A slight increase in the contact rate α and β leads to convergence of disease free equilibrium in the sentenced sub-population, while the remand population is experiencing endemicity. This occurs because inmates are not screened for TB immediately on admission in the remand population, while inmates in the sentenced population are screened and put under treatment. The simulation results can be seen in Figure 2 with $R_{g1} = 0.957$ and $R_{g2} = 1.25$.

In the absence of cross-effect between the two sub-populations and the consequent decrease in the contact rates, the disease in the prison system will be eradicated due to the treatment that is administered. This can be seen in Figure 3 with $R_{g1} = 0.920$ and $R_{g2} = 0.968$. Quite obviously, in the absence of treatment

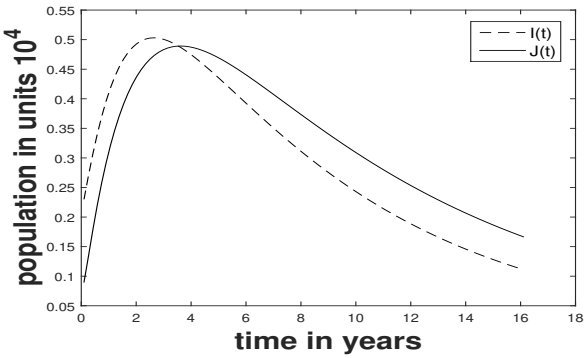

Fig. 1: $\alpha = 0.0000142$, $\beta = 0.00000904$ and $R_{g1} = 0.922, R_{g2} = 0.997$.

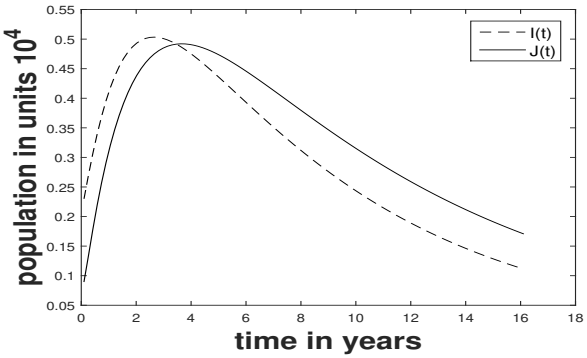

Fig. 2: $\alpha = 0.0000146$, $\beta = 0.0000121$, and $R_{g1} = 0.957$, $R_{g2} = 1.25$.

and with the cross-effect, the disease becomes persistent and the inmates will be at high risk of getting infected. TB transmission is driven exclusively by the systematic and prolonged exposure of susceptible to infectious individuals and this can be seen in Figure 4 with $R_{g1} = 3.12$ and $R_{g2} = 1.66$. Hence, sentenced inmates are considered to be in an active sub-population and are at risk of TB infection due to close and frequent contacts with infectious inmates.

5.2. Simulations with the inflow of infective

Now, we introduce the inflow of infectives in Figure 5 and use the contact rates in Table 2. The results show that the two-group model system (1) always has an endemic equilibrium and is globally asymptotically stable, which indicates that the disease will persist in the presence of the inflow of infectives. A further simulation that illustrates the dynamics of the infections classes I and J, when there is an inflow of infectives and a reduction in

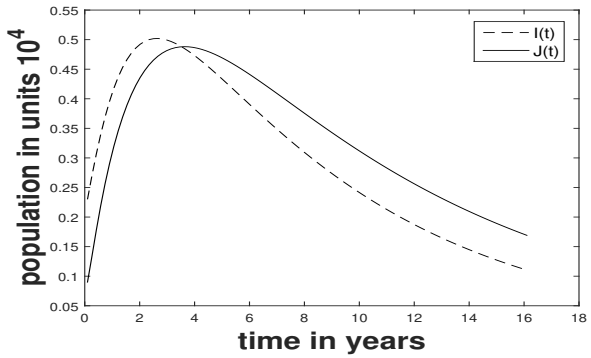

Fig. 3: $\alpha = 0.0000146, \beta = 0.0000121, \varphi = 0, \psi = 0$ and $R_{g1} = 0.920, R_{g2} = 0.949$.

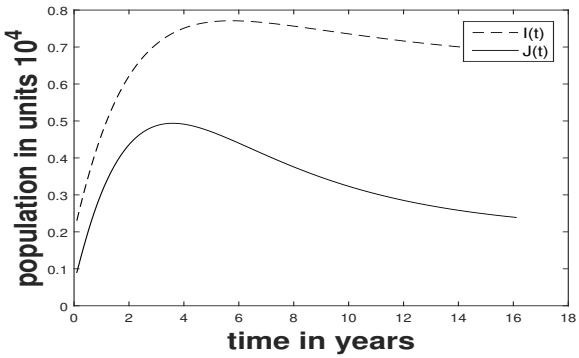

Fig. 5: $\alpha = 0.000071351$, $\beta = 0.00018303$ and $R_{g1} = 5.05, R_{g2} = 9.76$.

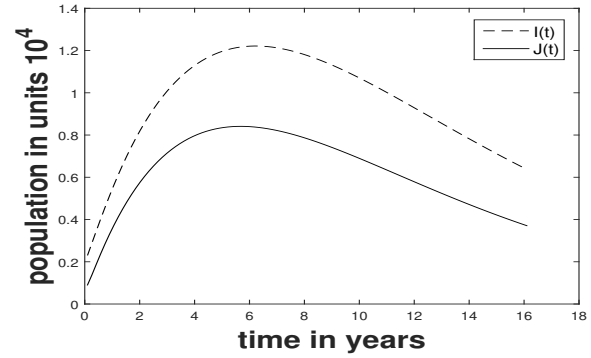

Fig. 4: $\alpha = 0.0000146$, $\beta = 0.0000121, \varphi = 0, \psi = 0$ $p = 0$, $q = 0$ and $R_{g1} = 3.12, R_{g2} = 1.67$.

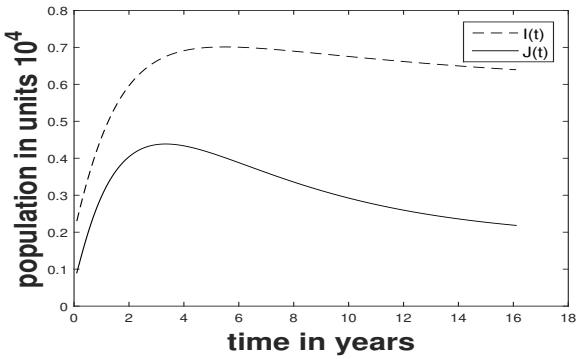

Fig. 6: $\alpha = 0.0000142$, $\beta = 0.00000904$ and $R_{g1} = 0.922, R_{g2} = 0.997$.

contact rates, has been presented in Figure 6. Figure 6, reveals that the disease will still persist in both population groups even though $R_{g1} < 1$ and $R_{g2} < 1$.
In Figure 7, it is noticeable that even though the contact rate has been reduced, the infectious class of sentenced inmates increases drastically in the absence of treatment with $R_{g1} = 3.28$ and $R_{g2} = 1.24$ in the remand population. The situation will worsen if we use the contact rates in Table 2 with the absence of treatment. This can be seen in Figure 8. The inflow of infectives prohibits the prison system to converge towards disease free equilibrium.

7 Conclusion

In this paper, we introduced a two-group model to monitor TB disease in a prison system. We divided the total prison population into sentenced sub-population and remand sub-population. Our simulation results have shown that when we have inflow of infecteds, the disease cannot be eliminated even though the basic reproduction number R_0 is less than unity. The cross effect between the two groups, as shown in Figure 3, also poses a major problem to reduction or removal of TB prevalence in one of the two groups. The theoretical analysis Theorem 3 together with simulation results confirm that subject to certain conditions, the disease free equilibrium is globally asymptotically stable. Therefore, more consideration should be given to monitor the inflow of infecteds to reduce the number of infectious individuals in order to eliminate the disease in the prison population. Then, it becomes unimportant to admit infected inmates to a prison facility which has an elimination plan in place. Since TB flourishes in crowded environments, social distancing and wearing of face masks should have a positive impact on curbing the spread of the disease. Most importantly, screening the inmates on admission together with comprehensive curative and preventive services for latent cases and active cases are important for reduction of TB prevalence in a prison.

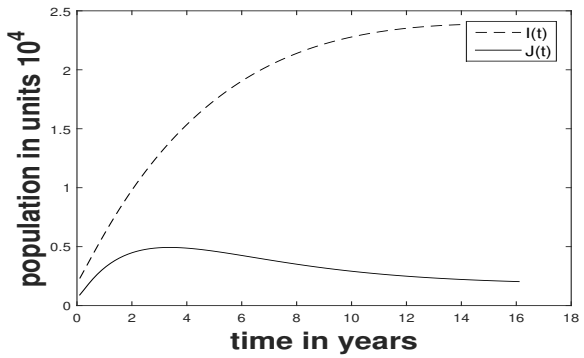

Fig. 7: $\alpha = 0.0000146,\ \beta = 0.0000150,\ q = 0,\ p = 0$ and $R_{g1} = 3.28, R_{g2} = 1.24$.

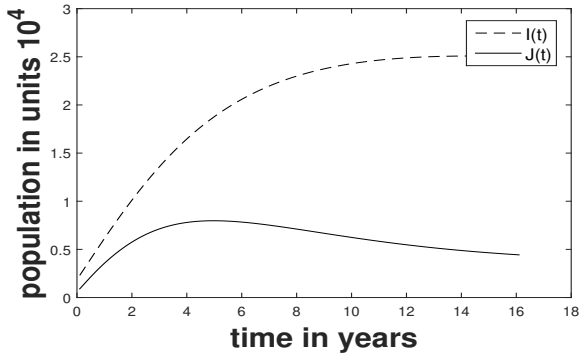

Fig. 8: $\alpha = 0.000071351,\ \beta = 0.00018303,\ q = 0,\ p = 0$ and $R_{g1} = 17.10, R_{g2} = 13.30$.

The paper [2] gives valuable insights into the challenges to curbing TB in prisons in Africa. An important topic for future research on the theme of TB population dynamics in a prison is the matter of reducing the average time that remand inmates spend in prison. The importance of such a reduction, for several reasons, has been highlighted in paper [3] for instance. This and other intervention strategies can be investigated through optimal control theory.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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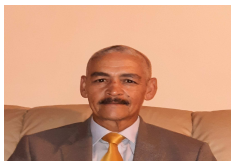

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