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Research Article

A Stochastic TB Model for a Crowded Environment

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We propose a stochastic compartmental model for the population dynamics of tuberculosis. The model is applicable to crowded environments such as for people in high density camps or in prisons. We start off with a known ordinary differential equation model, and we impose stochastic perturbation. We prove the existence and uniqueness of positive solutions of a stochastic model. We introduce an invariant generalizing the basic reproduction number and prove the stability of the disease-free equilibrium when it is below unity or slightly higher than unity and the perturbation is small. Our main theorem implies that the stochastic perturbation enhances stability of the disease-free equilibrium of the underlying deterministic model. Finally, we perform some simulations to illustrate the analytical findings and the utility of the model.

1. Introduction

Tuberculosis (TB) continues to be a major global health problem that is responsible for 1.5 million deaths worldwide each year [1]. TB is most prevalent in communities with socioeconomical problems but is not confined to such. The authors in [2, 3] associate TB infection with poverty and underdevelopment of some countries. It has been observed globally that one of the major factors driving TB infection is overcrowding. TB mostly occurs in poorest countries that are not developed and particularly where a population is overcrowded and in countries that are influenced by war. Conflict is the most common cause of large population displacement, which often results in relocation to temporary settlements such as camps. Factors including malnutrition and overcrowding in camp settings further increase the exposure to TB infection in these populations. Following up on a paper of Ssematimba et al. [3] regarding internally displaced people's camps in Uganda, Buonomo and Lacitignola [2] proposed a model that considers the dynamics of TB in concentration camps with a case study in Uganda. Another type of crowded environment which provides favourable conditions for TB to flourish is prisons and more so if the prison is full beyond its capacity. There are more than 10 million inmates in prisons all over the world. The United States of America is in the top rank with about 2.2 million inmates while South Africa is in rank 11 [4]. South African prison has approximately 160000 inmates in custody, of which 120000 are sentenced individuals while the rest are awaiting trial. This means that a large number of inmates are kept in remand population and some of them might not be found guilty at the end of the process, after having been exposed to high risk of TB infection.

Mathematical models have been used to model TB by considering the size of the area and how size and density affect the extent to which TB can invade a certain population [2, 3, 5–7]. Quite obviously, considering the manner in which TB is aerially transmitted from one person to another, the prison situation provides favourable conditions for TB to flourish. TB is an infectious disease caused by bacillus Mycobacterium tuberculosis that most often affects the lungs (pulmonary TB) and can affect other parts as well such as brain, kidneys, and spine (extrapulmonary TB) [8, 9]. The TB infection can take place when an infected individual releases some droplet nuclei which can remain airborne in any indoor area for up to four hours. The tubercle bacillus can persist in a dark area for several hours but it is exceptionally sensitive to sunshine. The risk of infection increases as the length of prison stay increases and the sentenced offenders are more likely to get TB infection as compared to the awaiting trial inmates.

Against this background the paper [10] offers a model for the population dynamics of TB in a prison or prison system.

In particular, it computes the parameters relevant to South Africa for the given model, using publicly available data. The current paper considers a stochastic form of the model in [10]. It is well understood that stochastic differential equation (sde) attempts to reflect the effect of random disturbances in or on a system. A second reason for studying sde models is that it is good to know that a given model carries some resilience against small disturbances. In this case we consider the transmission parameters to be stochastically perturbed, similarly to [11]. Stochastic pertubation has been studied by Yang and Mao [12]; they considered a multigroup SEIR epidemic model. In most cases, it has been observed in [12, 13] that introducing a stochastic perturbation into an unstable disease-free equilibrium model system of ordinary differential equation may lead to a system being stable in sde. Stochastic differential equation models for various diseases have been studied and similar work has been done in [11, 12, 14–16].

Our paper focuses on the analysis of TB in prisons as prisons have been recognized as institutions with very high TB burden as compared to a general population [17]. For a deterministic model of similar type, in [10] we computed parameter values pertaining to South Africa. For the stochastic model in this paper the focus is on mathematical analysis. In Section 2, the model is introduced, based on the paper of Buonomo and Lacitignola [2]. The existence and uniqueness of the solution to the stochastic models is investigated by using the Lyapunov method in Section 3. Stability of the disease-free equilibrium for stochastic models is shown in Section 4. We show our results by means of numerical simulations and conclude in Section 5.

2. The Model

We introduce a stochastic compartmental model which is based on the deterministic model in the paper of Buonomo and Lacitignola [2]. We divide the population, which is of size $N(t)$ at time t, into four compartments, namely, the class $S(t)$ of susceptible individuals $S(t)$, the class $E(t)$ of individuals infected with TB who are not infectious, the class $I(t)$ of individuals infected with active TB who are infectious, and the class $T(t)$ of individuals under treatment. It is important to note that in general populations removal of individuals out of the system is only by death. In this model, as in [10], the removal is by death or by discharge from prison, and the discharge is the dominant factor. This rate of removal is denoted by μ. The disease induced mortality rate is denoted by δ. Individuals are recruited into the susceptible class $S(t)$ at a constant rate μA. Susceptible individuals get infected with active TB at a rate c_1SI, where c_1 is the effective contact rate between the infectious and susceptible individuals. Individuals leave the exposed class $E(t)$ for infectious class $I(t)$ at rate kE. Exposed individuals who are infectious move to the infectious class $I(t)$ at a rate c_3EI, where c_3 is the effective contact rate between the exposed and infectious individuals. Successfully treated individuals who were infectious move to exposed class at a rate c_2TI, where c_2 is the effective contact rate between the treated and infectious individuals. Exposed and infectious individuals move into class $T(t)$ at the rates r_1 and r_2, respectively.

Let us assume $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq t_0}, \mathbb{P})$ to be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq t_0}$ which is right continuous. Let $W_i(t)$ $(i = 1, 2)$ be two mutually independent Brownian motions. Let us fix a nonnegative number σ, which shall serve as the intensity of the perturbation. We also fix two other positive numbers p and q with $p+q = 1$ that will balance the perturbation. The stochastic perturbations are similar to those in the model of [11].

Model System (1)

$$\begin{aligned} dS &= \left[f_S\mu A - c_1SI - \mu S\right]dt \\ &\quad - \sigma\left(pESdW_1(t) + qISdW_2(t)\right), \\ dE &= \left[f_E\mu A + c_1SI + c_2TI - c_3EI - \left(\mu + r_1 + k\right)E\right]dt \\ &\quad + \sigma pESdW_1(t), \\ dI &= \left[f_I\mu A + kE - \left(\mu + r_2 + \delta\right)I + c_3EI\right]dt \\ &\quad + \sigma qISdW_2(t), \\ dT &= \left[r_1E + r_2I - c_2TI - \mu T\right]dt. \end{aligned} \tag{1}$$

It is noticed that if $f_E + f_I > 0$ then system (1) does not have a disease-free equilibrium. We will first investigate the model without the inflow of infected cases, i.e., when $f_E = f_I = 0$. In this case the disease-free state

$$E_0 = \left(S_0, E_0, I_0, T_0\right) = (A, 0, 0, 0) \tag{2}$$

is an equilibrium point. The underlying deterministic model of (1) is the model given by the same system of equations in the special case $\sigma = 0$, i.e., without stochastic perturbation as in [10]. The underlying deterministic model coincides with the model of Buonomo and Lacitignola [2]. The basic reproduction number of the underlying deterministic model has already been computed in paper [2] and is given by the following formula:

$$R_0 = \frac{kc_1A}{\mu_1\mu_2}, \tag{3}$$

where $\mu_1 = \mu + r_1 + k$ and $\mu_2 = \mu + r_2 + \delta$.

We now present the following set:

$$\begin{aligned} \Delta_A = \Big\{x \in \mathbb{R}^4 : x_1, x_2, x_3, x_4 > 0 \ x_1 + x_2 + x_3 + x_4 \\ \leq A\Big\}. \end{aligned} \tag{4}$$

Remark 1. For the rest of the paper we will assume that the sample paths are restricted to Ω_0, which is defined as follows:

$$\begin{aligned} \Omega_0 = \big\{w \in \Omega \mid (S(t,w), E(t,w), I(t,w), T(t,w)) \\ \in \Delta_A \quad \text{for all } t \geq 0\big\}. \end{aligned} \tag{5}$$

Lemma 2 (see [13]). *For $k \in \mathbb{N}$, let $X(t) = (X_1(t), X_2(t), \ldots, X_k(t))$ be a bounded $\mathbb{R}^k$-valued function and let $(t_{0,n})$ be*

any increasing unbounded sequence of positive real numbers. Then there is family of sequences $(t_{l,n})$ such that for each $l \in 1, 2, \ldots, k$, $(t_{l,n})$ is a subsequence of $(t_{l-1,n})$ and the sequence $X_l(t_{l,n})$ converges to a chosen limit point of the sequence $X_l(t_{l-1,n})$.

3. Existence and Uniqueness of Positive Global Solutions

Proposition 3. *Suppose that we have a solution*

$$X(t) = (S(t), E(t), I(t), T(t)) \tag{6}$$

of system (1) over an interval $t \in [0, \tau)$ with $S(0) + E(0) + I(0) + T(0) < A$ and with $X(t) \in \mathbb{R}^4_{++}$ for all $0 \le t \le \tau$, a.s., then $S(t) + E(t) + I(t) + T(t) \le A$.

Proof. Given any solution in $X(t)$ satisfying the conditions of Proposition 3, then we have the total population in system (1) obeying the following ordinary differential equation:

$$\frac{d(N-A)}{dt} = -\mu(N-A) - \delta I \le -\mu(N-A) \quad \text{a.s.} \tag{7}$$

Therefore, similarly to [11], for instance, $N(0) < A$ implies that $N(t) < A$ for all $t \in [0, \tau)$. □

In this section, we investigate the existence and uniqueness of global positive solutions of stochastic models by using the Lyapunov method. This method is popularly applied for such problems; see [23, 24], for instance.

Theorem 4. *There is a unique solution $(S(t), E(t), I(t), T(t)) \in \mathbb{R}^4_+$ to system (1) on $t \ge 0$ for any given initial value $(S(0), E(0), I(0), T(0)) \in \mathbb{R}^4_+$, and the solution will remain in $\mathbb{R}^4_+$ with probability one; namely, $(S(t), E(t), I(t), T(t)) \in \mathbb{R}^4_+$ for all $t \ge 0$ almost surely.*

Sketch of the proof. Since the coefficients in (1) satisfy the Lipschitz condition locally, for any given initial value $(S(0), E(0), I(0), T(0))$, there is a unique local solution $(S(t), E(t), I(t), T(t))$ on $t \in [0, \tau_{en})$, where τ_{en} is the explosion time. Our a im is to show that this solution is global and positive almost surely; i.e., $\tau_{en} = \infty$ a.s.

Let $r_0 > 0$ such that $S(0), E(0), I(0), T(0) > r_0$. For each integer $r \le r_0$, we define the stopping times

$$\begin{aligned}\tau_r = \inf\{&t \in [0, \tau_{en}] : S(t) \le r \text{ or } E(t) \le r \text{ or } I(t) \\ &\le r \text{ or } T(t) \le r\}.\end{aligned} \tag{8}$$

Let

$$\begin{aligned}\tau = \lim_{r\to 0} \tau_r = \inf\{&t \in [0, \tau_{en}) : S(t) \le 0 \text{ or } E(t) \\ &\le 0 \text{ or } I(t) \le 0 \text{ or } T(t) \le 0\}.\end{aligned} \tag{9}$$

For this purpose we introduce a function V as follows:

$$V = \ln\frac{A}{S} + \ln\frac{A}{E} + \ln\frac{A}{I} + \ln\frac{A}{T}. \tag{10}$$

We note that, by Proposition 3, each of the terms

$$\begin{gathered}\ln\frac{A}{S}, \\ \ln\frac{A}{E}, \\ \ln\frac{A}{I}, \\ \ln\frac{A}{T}\end{gathered} \tag{11}$$

is positive, and

$$\lim_{u\to 0^+} \frac{A}{u} = +\infty. \tag{12}$$

By Itô's formula, for all $t \ge 0$, $s \in [0, t \wedge \tau_r]$, we have

$$\begin{aligned}dV(X(s)) = &-\frac{1}{S(s)}\left(f_S\mu A - c_1 S(s) I(s) - \mu S(s)\right. \\ &\left. + \frac{(\sigma p E(s))^2}{2} + \frac{(\sigma q I(s))^2}{2}\right) ds - \frac{1}{E(s)}\left(f_E\mu A\right. \\ &+ c_1 S(s) I(s) + c_2 T(s) I(s) - c_3 E(s) I(s) \\ &\left. - (\mu + r_1 + k) E(s) + \frac{(\sigma p S(s))^2}{2}\right) ds \\ &- \frac{1}{I(s)}\left(f_I\mu A + kE(s) - (\mu + r_2 + \delta) I(s)\right. \\ &\left. + c_3 E(s) I(s) + \frac{(\sigma q S(s))^2}{2}\right) ds - \frac{1}{T(s)}(r_1 E(s) \\ &+ r_2 I(s) - c_2 T(s) I(s) - \mu T(s)) ds + \sigma p (E(s) \\ &- S(s)) dW_1(s) + \sigma q (I(s) \\ &- S(s)) dW_2(s).\end{aligned} \tag{13}$$

After eliminating some negative terms we have the following inequality:

$$dV(X(s)) \le M_1 ds + dM_2(s), \tag{14}$$

where

$$\begin{aligned}M_1 = &4\mu + r_1 + r_2 + k + d + I(c_1 + c_2) + c_3(E + I) \\ &+ \frac{\sigma^2}{2}\left(p^2E^2 + q^2I^2\right) + \frac{1}{2}(\sigma(p+q)S)^2,\end{aligned} \tag{15}$$

and

$$dM_2(s) = \sigma p (E - S) dW_1(s) + \sigma q (I - S) dW_2(s). \tag{16}$$

Taking the integral in (14) from 0 to $t \wedge \tau_{r0}$, we have

$$\int_0^{t\wedge\tau_r} dV(X(s)) \le \int_0^{t\wedge\tau_r} M_1 ds + \int_0^{t\wedge\tau_r} dM_2(s). \tag{17}$$

By taking expectations, the latter inequality yields

$$\begin{aligned}&\mathbb{E}\left[V\left(S\left(t\wedge\tau_r\right),E\left(t\wedge\tau_r\right),I\left(t\wedge\tau_r\right),T\left(t\wedge\tau_r\right)\right)\right]\\&\quad\le V\left(X\left(0\right)\right)+M_1t.\end{aligned}\tag{18}$$

Now we note that

$$\begin{aligned}&\mathbb{E}V\left[S\left(t\wedge\tau_r\right),E\left(t\wedge\tau_r\right),I\left(t\wedge\tau_r\right),T\left(t\wedge\tau_r\right)\right]\\&\quad=\mathbb{E}\left[\Psi_{(\tau_r\le t)}V\left(S\left(t\wedge\tau_r\right),E\left(t\wedge\tau_r\right),I\left(t\wedge\tau_r\right),\right.\right.\\&\quad\left.\left.T\left(t\wedge\tau_r\right)\right)\right]+\mathbb{E}\left[\Psi_{(\tau_r>t)}V\left(S\left(t\wedge\tau_r\right),E\left(t\wedge\tau_r\right),\right.\\&\quad\left.I\left(t\wedge\tau_r\right),T\left(t\wedge\tau_r\right)\right)\right]\ge\mathbb{E}\left[\Psi_{(\tau_r\le t)}V\left(S\left(\tau_r\right),E\left(\tau_r\right),\right.\\&\quad\left.I\left(\tau_r\right),T\left(\tau_r\right)\right)\right],\end{aligned}\tag{19}$$

where $\Psi_{(\cdot)}$ is the indicator function. If $\tau_r<\infty$, then there are some components of $S(\tau_r)$, $E(\tau_r)$, $I(\tau_r)$, $T(\tau_r)$ equal to r, and therefore $(S(\tau_r),E(\tau_r),I(\tau_r),T(\tau_r))\ge\ln(A/r)$.

Thus we have

$$\begin{aligned}&\mathbb{E}\left[V\left(S\left(t\wedge\tau_r\right),E\left(t\wedge\tau_r\right),I\left(t\wedge\tau_r\right),T\left(t\wedge\tau_r\right)\right)\right]\\&\quad\ge\ln\left(\frac{A}{r}\right)\mathbb{P}\left(\tau_r\le t\right).\end{aligned}\tag{20}$$

Combining (14) and (18) gives, for all $t\ge0$,

$$\mathbb{P}\left(\tau\le t\right)\le\frac{V\left(X\left(0\right)\right)+M_1t}{\ln\left(A/r\right)}\tag{21}$$

Letting $r\to0$, we obtain, for all $t\ge0$, $\mathbb{P}(\tau\le t)=0$. Hence $\mathbb{P}(\tau=\infty)=1$. As $\tau_{en}=\tau=\infty$ a.s. Therefore, the solution of model (1) will not explode at a finite time with probability one. This completes the proof. □

4. Stability of Disease-Free Equilibrium

Let us choose a positive number a_3 and two nonnegative numbers a_1 and a_2. Specific values will be assigned to these numbers in different analyses.

Let us assume that

$$a_3\ge\frac{k}{\mu_1}.\tag{22}$$

Now we define a stochastic process $Z(X(t))$

$$Z\left(X\left(t\right)\right)=a_1\left(A-S\left(t\right)\right)+a_2T\left(t\right)+a_3E\left(t\right)+I\left(t\right)\tag{23}$$

and a process

$$V\left(X\left(t\right)\right)=\ln Z\left(X\left(t\right)\right).\tag{24}$$

For $w\in\Omega_0$, we note that $Z(X(t))>0$ and therefore $V(X(t))$ are defined for all $w\in\Omega_0$. For convenience, we introduce the variables:

$$\begin{aligned}Q_Z&=\frac{A-S}{Z},\\T_Z&=\frac{T}{Z},\\E_Z&=\frac{E}{Z},\\I_Z&=\frac{I}{Z}\end{aligned}\tag{25}$$

and for a stochastic process $x(t)$ we shall write

$$\left\langle x\right\rangle_s=\frac{1}{s}\int_0^s x\left(u\right)du.\tag{26}$$

4.1. On the Lyapunov Exponent of Z. The Lyapunov exponent of a quantity $q(t), t\ge0$ is defined as

$$\limsup_{t\to\infty}\frac{1}{t}\ln q\left(t\right).\tag{27}$$

The infinitesimal generator $\mathscr{L}$ of system (1) (see Øksendal [25]) will play an important role in the sequel. Now we can calculate $\mathscr{L}V$ and express it as a function of $X(t)$. From Lemma 2 it follows that for each $w\in\Omega_0$ there is an increasing sequence (t_n^w) with the following properties (but we shall suppress w and write (t_n)):

For every $w\in\Omega$,

$$\lim_{n\to\infty}\left\langle\mathscr{L}V\left(X\right)\right\rangle_{t_n}=\limsup_{t\to\infty}\left\langle\mathscr{L}V\left(X\right)\right\rangle_t\tag{28}$$

and the limits below, which shall be denoted by q,τ,j,i, do exist:

$$\begin{aligned}q&=\lim_{n\to\infty}\left\langle Q_Z\right\rangle_{t_n},\\\tau&=\lim_{n\to\infty}\left\langle T_Z\right\rangle_{t_n},\\j&=\lim_{n\to\infty}\left\langle E_Z\right\rangle_{t_n},\\i&=\lim_{n\to\infty}\left\langle I_Z\right\rangle_{t_n}.\end{aligned}\tag{29}$$

We write

$$\Lambda=\limsup_{t\to\infty}\left\langle\mathscr{L}V\left(X\right)\right\rangle_t.\tag{30}$$

Let

$$c_*=\max\left\{c_1,c_2,c_3\left(\frac{\mu}{k}-1\right)\right\}.\tag{31}$$

We can write

$$\int_0^t dV=\int_0^t\mathscr{L}Vdt+M\left(t\right),\tag{32}$$

where

$$M(t) = \int_0^t \frac{1}{Z}\sigma p(E-S)\,dW_1 + \int_0^t \frac{1}{Z}\sigma q(I-S)\,dW_2, \quad (33)$$

and we note that by the strong law of large numbers [16],

$$\lim_{n\to\infty} \frac{1}{t}M(t) = 0 \quad \text{a.s.} \quad (34)$$

Therefore

$$\begin{aligned}
&\limsup_{t\to\infty} \frac{1}{t}V(X(t)) \\
&\quad = \limsup_{t\to\infty} \frac{1}{t}\int_0^t \mathscr{L}V(X(s))\,ds \,(\text{a.s.}) \\
&\quad = \lim_{n\to\infty} \frac{1}{t_n}\int_0^{t_n} \mathscr{L}V(X(s))\,ds \,(\text{a.s.}).
\end{aligned} \quad (35)$$

Now we expand $\mathscr{L}V$:

$$\begin{aligned}
\mathscr{L}V = {} & \frac{-a_1}{Z}\left[\mu A - c_1 SI - \mu S\right] \\
& - \frac{a_1^2\sigma^2}{2Z^2}\left(p^2E^2S^2 + q^2I^2S^2\right) \\
& + \frac{a_2}{Z}\left[r_1E + r_2I - c_2TI - \mu T\right] \\
& + \frac{a_3}{Z}\left[c_1SI + c_2TI - c_3EI - (\mu + r_1 + k)E\right] \\
& - \frac{a_3^2}{2Z^2}\sigma^2\left(p^2E^2S^2\right) \\
& + \frac{1}{Z}\left[kE - (\mu + r_2 + \delta)I + c_3EI\right] \\
& - \frac{1}{2Z^2}\left(\sigma^2q^2I^2S^2\right) - a_1a_3\left(\sigma pES\right)^2 \\
& - a_1\left(\sigma qIS\right)^2.
\end{aligned} \quad (36)$$

With regard to the calculation of $\mathscr{L}V$ we note the following:

$$\begin{aligned}
& a_3I_Z\{c_1S + c_2T - c_3E\} + c_3I_ZE \\
& \quad = a_3I_Z\left\{c_1S + c_2T + c_3\left(\frac{1}{a_3} - 1\right)E\right\} \\
& \quad \le a_3I_Zc_*(S + T + E) \le a_3I_Zc_*A.
\end{aligned} \quad (37)$$

Therefore,

$$\begin{aligned}
\mathscr{L}V \le {} & a_3I_Zc_*A - I_Z(\mu_2 - a_2r_2) \\
& + E_Z(a_2r_1 - a_3\mu_1 + k) - a_2\mu T_Z \\
& + I_Z(a_1c_1S - a_2c_2T) - a_1\mu Q + B,
\end{aligned} \quad (38)$$

where

$$\begin{aligned}
B = {} & -\frac{(a_1\sigma)^2}{2}\left[(pE_ZS)^2 + (qI_ZS)^2\right] - \frac{a_3^2}{2}\left[(\sigma pE_ZS)^2\right] \\
& - \frac{1}{2}\left[(\sigma qI_ZS)^2\right] - a_1a_3\left(\sigma pE_ZS\right)^2 - a_1\left(\sigma qI_ZS\right)^2.
\end{aligned} \quad (39)$$

This yields the inequality:

$$\begin{aligned}
\mathscr{L}V \le {} & I_Z\left((a_1c_1 + a_3c_*)A - \mu_2 + a_2r_2\right) \\
& + E_Z(k - a_3\mu_1 + a_2r_1) - a_2\mu_2T_Z - a_1\mu Q_Z \\
& + B.
\end{aligned} \quad (40)$$

In the expression for B, if we ignore the multiples of a_1 (they are negative), then we obtain an inequality:

$$B \le -\frac{(\sigma S)^2}{2}\left\{\left(pa_3E_Z\right)^2 + \left(qI_Z\right)^2\right\}. \quad (41)$$

4.2. Stability Theorems. We now introduce another invariant R_σ, which enables us to formulate stability theorems for the stochastic model (1). As a corollary of the main theorem we can deduce a global stability theorem for disease-free equilibrium. Let

$$R_\sigma = \frac{kc_*A}{\mu_1\mu_2}. \quad (42)$$

In the model of Buonomo and Lacitignola [2], we have backward bifurcation at $R_0 = 1$. Therefore, the condition $R_0 < 1$ does not imply global stability of the underlying deterministic model. As a corollary to the main theorem, Theorem 6, will follow the fact that for the model in [2] the disease-free equilibrium is globally asymptotically stable when $R_\sigma < 1$. In preparation for our main theorem we introduce a function $h(x)$ as follows:

$$h(x) = \frac{p^2(1-x)^2 + q^2x^2}{x}; \quad x > 0. \quad (43)$$

Then

$$\lim_{x\to\infty} h(x) = \infty \text{ and if } q \ne 0, \text{then } \lim_{x\to 0^+} h(x) = \infty. \quad (44)$$

Also we note that

$$h'(x) = \frac{1}{x^2}\left[-p^2 + x^2\right]. \quad (45)$$

Therefore $h'(x) = 0 \Leftrightarrow x = p$ and we know that $p \le 1$. Since h has only one critical value on the interval $(0, \infty)$, in view of (44), it follows that the critical point is an absolute minimum of h on the interval $(0, \infty)$.

Therefore the minimum value $h_{\min}$ of h over $[0, 1]$ is

$$\begin{aligned}
h_{\min} &= \frac{p^2(1-p) + q^2p}{p} = p(1-p) + \left(1 - p^2\right) \\
&= (1-p)(p + 1 + p) = (1-p)(1 + 2p).
\end{aligned} \quad (46)$$

Proposition 5. *If*

$$R_\sigma - \frac{(\sigma A)^2 h_{\min}}{2\mu_2} < 1, \quad (47)$$

then (I, E) *converges exponentially to zero almost surely.*

Proof. We introduce the function V of (24), with $a_1 = a_2 = 0$. Now note that (47) is equivalent to

$$\frac{kc_* A}{\mu_1} - \frac{(\sigma A)^2 h_{\min}}{2} - \mu_2 < 0. \tag{48}$$

We choose a number $\epsilon > 0$ sufficiently small such that

$$\frac{k+\epsilon}{\mu_1} c_* A - \mu_2 - \frac{(\sigma A)^2}{2} h_{\min} < 0. \tag{49}$$

Now we choose

$$a_3 = \frac{k+\epsilon}{\mu_1}. \tag{50}$$

From inequality (40) it follows that

$$\mathscr{L}V \le \left[a_3 c_* A - \mu_2\right] I_Z + \left[k - a_3\mu_1\right] E_Z - B_1, \tag{51}$$

where

$$B_1 = \frac{(\sigma A)^2}{2}\left\{p^2 \left(a_3 E_Z\right)^2 + \left(qI_Z\right)^2\right\}. \tag{52}$$

Now note that we can express B_1 as follows:

$$\begin{aligned} B_1 &= \frac{(\sigma A)^2}{2}\left\{p^2 \left(1 - I_Z\right)^2 + \left(qI_Z\right)^2\right\} \\ &= \frac{(\sigma A)^2}{2} I_Z h\left(I_Z\right). \end{aligned} \tag{53}$$

Therefore, we have

$$B_1 \ge \frac{(\sigma A)^2}{2} I_Z h_{\min}, \tag{54}$$

and, consequently,

$$\begin{aligned} \mathscr{L}V \le &\left[a_3 c_* A - \mu_2 - \frac{(\sigma A)^2}{2} h_{\min}\right] I_Z \\ &+ \left[k - a_3\mu_1\right] E_Z. \end{aligned} \tag{55}$$

Therefore

$$\Lambda \le \left[a_3 c_* A - \mu_2 - \frac{(\sigma A)^2}{2} h_{\min}\right] i + \epsilon j \tag{56}$$

and since i and j cannot both be zero, it follows that $\Lambda < 0$. This completes the proof. □

Theorem 6. *(a) If $(E(t), I(t))$ almost surely converges exponentially to 0, then*

$$\lim_{t\to\infty} S(t) = A \quad \text{(a.s.)} \quad \text{and} \quad \lim_{t\to\infty} T(t) = 0 \quad \text{(a.s.)}. \tag{57}$$

(b) If

$$R_\sigma - \frac{(\sigma A)^2 h_{\min}}{2\mu_2} < 1, \tag{58}$$

then disease-free equilibrium is almost surely exponentially stable.

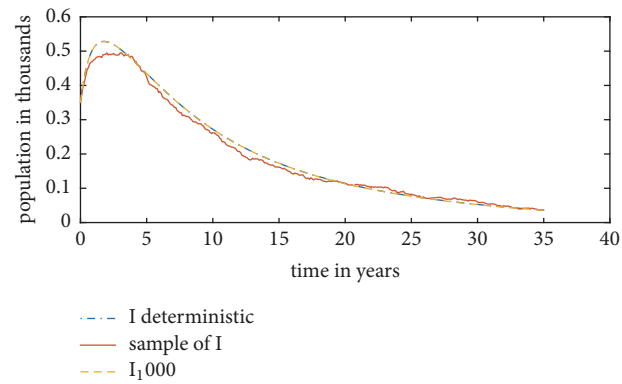

FIGURE 1: $R_0 = 1.3917$, $R_\sigma = 1.1653$, $c_1 = 0.000065$, and $\sigma = 0.04$.

Proof. (a) Suppose to the contrary that we have

$$\lim_{t\to\infty} (A - S(t)) + T(t) > 0 \quad \text{(a.s.)}. \tag{59}$$

Let Z be the same as that in (23), with $a_1 = a_2 = a_3 = 1$. Since $(E(t), I(t))$ almost surely converges exponentially to 0 while

$$\lim_{t\to\infty} (A - S(t)) + T(t) > 0 \quad \text{(a.s.)}, \tag{60}$$

it follows that $j = 0$ and $i = 0$ (a.s.). Thus from inequality (40) it follows that

$$\Lambda \le -\mu_2 T_Z - \mu Q_Z \quad \text{(a.s.)}. \tag{61}$$

Therefore $\Lambda < 0$. This implies that Z converges to 0, and thus

$$\lim_{t\to\infty} (A - S(t)) + T(t) = 0 \quad \text{(a.s.)}, \tag{62}$$

which is a contradiction. This completes the proof of (a).

(b) This follows from Proposition 5 and Theorem 6(a). □

5. Numerical Simulation

The simulations presented here illustrate the analytical results of our model in (1). The parameter values have already been calculated in the paper [10], by using real data, mostly from [18, 20, 21]. We will now use those parameter values, listed in Table 1, and vary the value of c_1 and σ in order for us to be able to find different values of R_0 and R_σ. We first consider a model without the inflow of infective cases and then with the inflow of infective cases.

We give some numerical simulations to show different dynamic outcomes of the deterministic model and its stochastic version. We illustrate by means of simulations the possible disease eradication in the absence of the inflow of infective cases. This will be shown in Figures 1, 2, and 3. Over these three cases we vary the value of c_1 and σ so as to obtain different values of R_0 and R_σ.

In Figure 1, we present a case in which we take $c_1 = 0.000065$, $\sigma = 0.04$ and then we obtain $R_0 = 1.3917$ and $R_\sigma = 1.1653$. This situation does not satisfy the conditions of Theorem 6, and indeed the I-class does not appear to

Table 1: Model parameters and initial conditions.

Parameter	Estimated value	Source
μ	0.18192	[10], data from [18, 19]
d	0.01876	[10], data from [18, 20]
c_1	0.00007893	[10], see also [21]
c_2	$20/A$	[10, 22]
c_3	$k(2A)$	Estimated from [10]
r_1	0.30	[2]
r_2	0.50	[2]
k	0.05	[2, 21]
A	160000	[18]
f_S, f_E, f_I	0.2, 0.74, 0.06	[18]
$S_{t_{15}}$	32000	[10], data from [18]
$E_{t_{15}}$	107000	[10], data from [18]
$I_{t_{15}}$	3500	[10], data from [18]
$T_{t_{15}}$	17100	[10], data from [18]

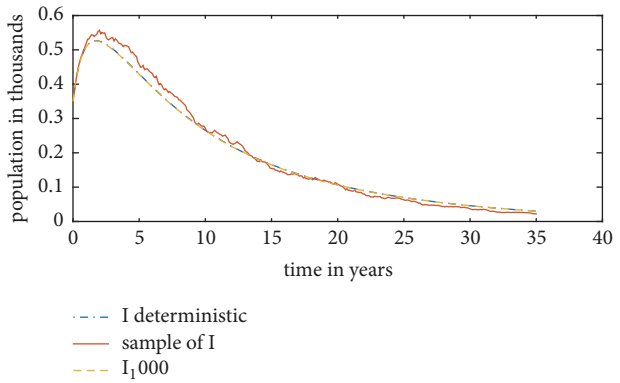

Figure 2: $R_0 = 1.3275$, $R_\sigma = 0.9737$, and $c_1 = 0.000062$, $\sigma = 0.05$.

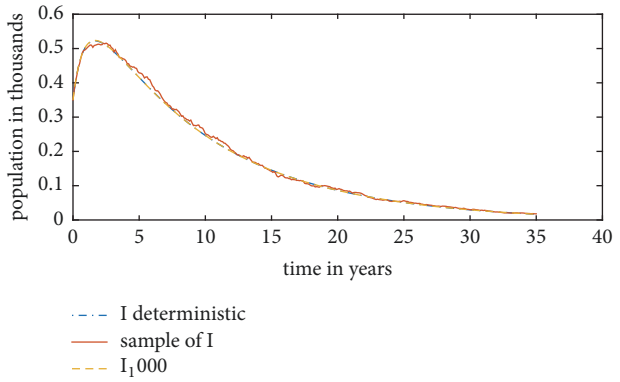

Figure 3: $R_0 = 1.1562$, $R_\sigma = 0.0.9298$, and $c_1 = 0.000054$, $\sigma = 0.04$.

converge to zero. This means that the disease will persist in our prison population.

In Figure 2, we notice that when the perturbation is sufficiently big, then the disease will possibly be eliminated for a stochastic model even if for the deterministic model it does not seem be the case. We have chosen $c_1 = 0.000062, \sigma = 0.05$ and then we calculate $R_0 = 1.3275$ and $R_\sigma = 0.9737$.

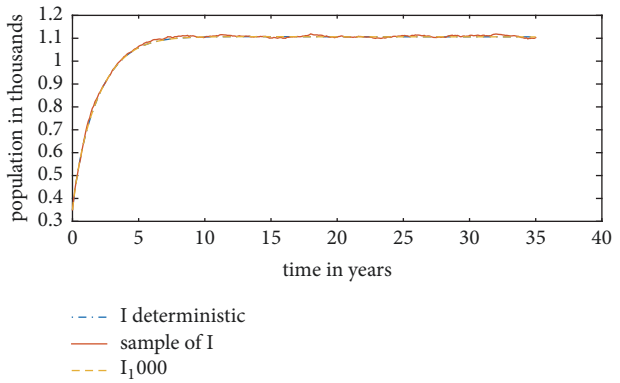

Figure 4: $R_0 = 1.6900$, $R_\sigma = 1.4635$, and $c_1 = 0.00007893$, $\sigma = 0.04$ with $f_S = 0.2$, $f_E = 0.74$, and $f_I = 0.06$.

In Figure 3, a choice of $c_1 = 0.000054$ and $\sigma = 0.04$ yields $R_0 = 1.1562$ and $R_\sigma = 0.9298$. This choice of parameters satisfies the conditions in Theorem 6, and surely the infectious class seems to converge to zero.

We now study model (1) with the inflow of infectives and present a sample computation. We choose $c_1 = 0.00007893$ as in Table 1 and $\sigma = 0.04$. Then the values of R_0 and R_σ can be calculated as $R_0 = 1.6900, R_\sigma = 1.4635$. In Figure 4, it is observed that when the basic reproduction number for the underlying deterministic model is above unity, then the disease will persist into our prison system. It is also seen that the inflow of infective cases play a part in influencing the number of TB infected cases in the prison system.

6. Conclusion

A stochastic SEIT model was presented and analysed to assess the impact of active TB on a crowded environment, specifically in prisons. We started off by verifying that there is a unique global positive solution for the system of stochastic differential equation in (1). It was noted that whenever the basic reproduction number is significantly greater than

unity then the disease will persist in the prison population through our simulations in Figures 1 and 2. It has also been observed for a stochastic model that when the perturbation is sufficiently big then the disease tends to vanish and this can be seen in Figure 2. It is more important to study smaller perturbation. It has been observed that whenever $R_\sigma < 1$, then I and E almost surely converge exponentially to zero in step with Theorem 6, in the absence of the inflow of infective. These results can also be seen in Figure 3. By introducing the inflow of infective cases into the prison system, TB remains endemic, as can be seen in Figure 4. By screening the inflow on admission and providing for them a separate accommodation, TB infection in a prison system can be greatly reduced.

Data Availability

All the data used in this research are publicly available and are cited in the article. No new data were generated for this article.

Disclosure

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Conflicts of Interest

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

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