A Stochastic Approach of Tuberculosis Model With Effects of Case Detection and Treatment

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ABSTRACT

In this paper we are deriving and extending a non-linear deterministic mathematical model of tuberculosis with emphasis on case detection and treatment into its equivalent stochastic model. By revising the analysis in the deterministic method, different Equilibria in the model were found using convenient technique and analyzed their condition for stability and reproduction number were computed. Some dynamical behavior of the model and their approximations are illustrated through analytic and numerical simulation for both deterministic and stochastic model which shows strong oscillation for stochastic trajectory this lead to a narrow gap between the curves of deterministic and stochastic. This happened because of random white translated by a supplementary term added whose trajectories come to superimpose the deterministic trajectories. Some preliminary result shows that case detection play an important role towards eliminating the spread of TB.

1 Introduction

Tuberculosis (TB) epidemic is widely acknowledged to be the most severe health crisis of modern times globally, the best estimate is that 10.0 million people (range, 9.0˜11.1 million) developed TB disease in 2017: 5.8 million men, 3.2 million women and 1.0 million children. There were cases in all countries and age groups, but overall 90% were adults (aged ≥ 15 years) [2]. Most TB disease occurs in resource-limited countries. *Mycobacterium tuberculosisis* the bacterial agent which makes the growth of Tuberculosis (TB) in humans. The usual target of this agent is lungs, but can strikes any part of the body such as the kidney, spine, and brain. TB is among the most deadly infectious diseases in the world[2]. There have been few victories in the efforts to contain it. This is true despite remarkable advances in our understanding of the molecular biology of the virus and its effects on the body-advances that have led to major therapeutic discoveries in the epidemic. We can also note that some research shows a total elimination of the disease is not possible at this moment, this is due to the difficulty of developing an effective vaccine, the expensive and time taken for diagnostic process and the compulsion of many months of treatment.

To control the transmission level of the disease case detection was identified as a major factor for controlling and containing the spread of TB. We can see for instance In countries with a high incidence of TB, WHO guidance issued in 2018 includes a new recommendation to consider testing and treatment for people aged 5 years or more who are household contacts of bacteriologically confirmed pulmonary TB cases by prioritizing a vaccine to lower the risk of infection, a vaccine or new drug treatment to cut the risk of TB disease in the 1.7 billion people already latently infected, rapid diagnostics for use at the point of care and simpler, shorter drug regimens for treating TB disease [2].

Mathematical modeling as a means of describing a system using mathematical concepts and language are best tool in studying and understanding the transmission dynamics of infectious diseases spread like TB with emphases in providing deeper insight into the control and elimination strategies. There is an Wide-ranging of work for a number century of deterministic models or rather compartmental ordinary differential equation (ODE) models describing transmission dynamics of TB, many of which developed as a variations of the standard SEIR model (S, susceptible, E exposed, I,

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infected and R recovered, see [7] for a review of SEIR models). The impact of treatment and its relationship with case detection on the dynamics of TB was observed in [1], which shows the prospects of controlling the spread of TB is bright if only the treatment strategy can be sustained and case detection significantly improved upon. To further reduce the burden of TB, increasing the case detection rate will not only lower the backward bifurcation range, in the presence of exogenous re-infection, but could also lower the reproduction number, reducing the severity of the TB epidemic [3]. chemotheraphy of human TB infection on the impact of the first line drug regimen on active disease control under the stipulated time of TB treatment with The efficacy of each drug was explored in [4] and observations show that low drug efficacy values result in extension of treatment period. However, direct progression from the susceptible to the infectious class is often allowed to mimic primary disease; recovery is proved to be possible due to antibiotic treatment. Recovered individuals can move back to the latent class again by having contact with effected class, since treatment given is not completely give protection against future exposure, as in [5].

We observed that most of these models proposed do not consider the unpredictable biological and epidemiological conditions i.e. the random nature perturbations that the dynamic system experienced. It is therefore not only worthy to predict the state of biological process, but also to know the random aspect of this process (product by a Wiener process and a "White noise") [8]. Only stochastic models capture the inherent randomness in disease transmission observed in real-life outbreaks, which can strongly influence the outcome of an emerging epidemic because case numbers will initially be small compared with the population size [9]. This necessitate to construct a stochastic model that contains in addition to the deterministic term, an additional random term. We speak of the deterministic model if the random term is omitted and the stochastic model other- wise. The deterministic model is therefore a particular case of the stochastic model, and can have several associated stochastic models. The dynamical behavior of a stochastic tuberculosis model with antibiotic resistance were discuss in [10] By constructing a suitable stochastic Lyapunov function, also a sufficient conditions for the existence of a unique ergodic stationary distribution of the positive solutions to model were establish. Reducing the time between onset of active disease and initiation of treatment was found one of the effective means of reducing disease burden as in [11]. A Pulmonary tuberculosis therapy with emphasis on insights from a prototype model with rifampin, were a description of the time-course of TB and its treatment from the first day of infection to the last day of therapy were presented In [6],. The model reproduces some important characteristics of the antibacterial effect of rifampin observed in patients. Numerous stochastic models have been developed from deterministic model to study various aspects of control parameters through analysis and numerical simulations of models see [8, 15, 16, 17] for some insight.

The purpose of this paper is to develop Stochastic Model using the deterministic model proposed in [1]. We considered both deterministic and stochastic models in finding and establishing the Equilibria of the models while analyzing the model via deterministic and diffusion counterparts with same approach as in [1, 8, 10, 12, 15, 17]. Perform Numerical simulation. To further investigated the difference and dynamical behavior of the two models a numerical simulations were perform using suitable parameters as estimated in [1, 10].

The rest of the article is organized as follows. In Section 2, we describe the proposed ODE model of [1] with same assumptions . In Section 3, we revised the classical deterministic analysis models. In Section 4, We extended the model described in section 2 to its equivalent stochastic model. This approach has been used recently in the study of many epidemic models; see for example [8, 10, 15]. Finally Numerical simmulation & experiments, summary and conclusions are presented in section 5 and 6 respectively.

2 Model Formulation

In order to review transmission dynamics of this disease, an SEIR-model is presented by dividing the overall population to four (4) epidemiological subgroups or classes as: Susceptible $x(t)$, Exposed $y(t)$, TB Infected $v(t)$ and Recovered class $z(t)$ at time t. Hence we denote the overall population by $N = x(t) + y(t) + v(t) + z(t)$. Also by

assumptions whole population is varying mixed homogeneously (i.e every individuals has equal chances of being exposed or infected with TB while in contact with infectious group of the population) [1]. Let ϕ denotes the fraction of TB-infected persons that are diagnose with TB-infection that are undergoing for treatment. Therefore spreading or transmission due to these will be minimal unlike those infected persons not been diagnose.

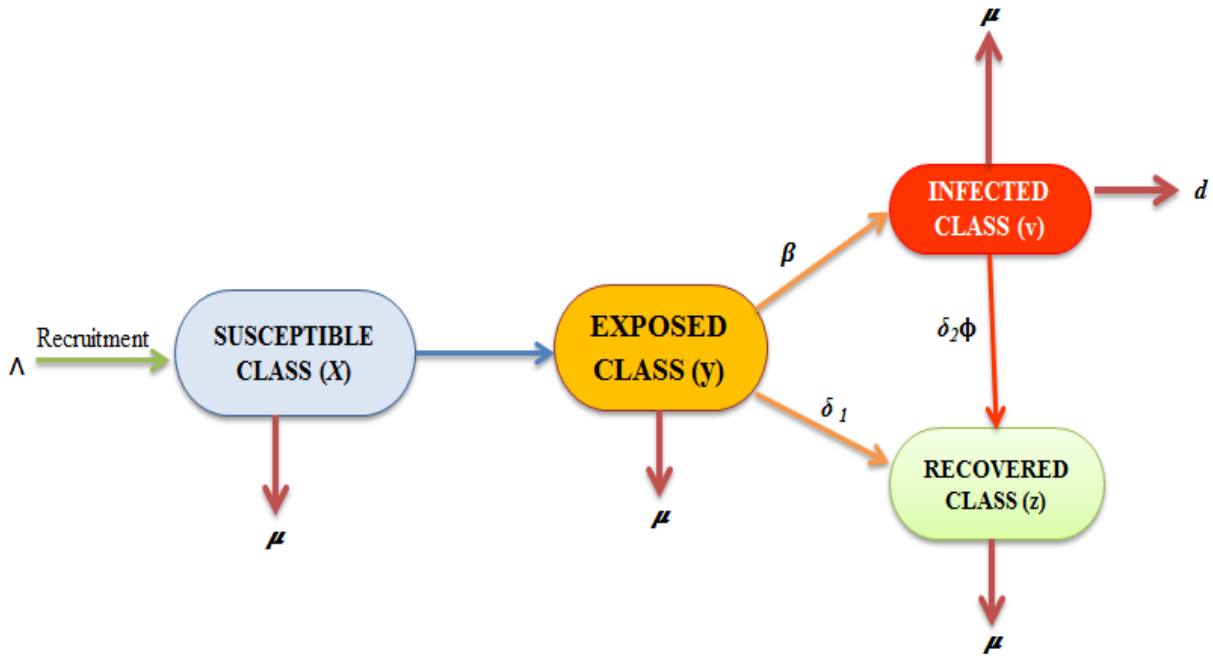

Fig. 1 Flowchart showing TB transmission in the model

We can also note that, individuals immunity level vary one person to another, with this we let a fraction of ψ (where $0 < \psi < 1$) of persons with new case of infection evolves Tuberculosis rapid and moves to infected class Then we lets $(1-\psi)$ denotes a fraction of persons with new infection transit to TB exposed subgroup/class before gradually moving to fully TB-infected class. Again, if exposed persons are in association with TB-infected persons will eventually be infected after some time if not treated. By Considering above assumptions, we consider a model that was recommended by [1]:

$$\begin{aligned}
\frac{dx}{dt} &= \Lambda - \mu x - [\alpha_1\phi + \alpha_2(1-\phi)]xv \\
\frac{dy}{dt} &= (1-\psi)[[\alpha_1\phi + \alpha_2(1-\phi)]xv - \beta vy - (\mu + \delta_1 + \theta)y \\
\frac{dv}{dt} &= \psi[\alpha_1\phi + \alpha_2(1-\phi)]xv + \beta vy - (\mu + d + \delta_2\phi)v + \theta y \\
\frac{dz}{dt} &= \delta_1 y + \delta_2\phi v - \mu z
\end{aligned} \tag{1}$$

See **table (1)** below for various parameters definitions as used in the model **??**. By re-assigning some parameter model 1 can be further simplified as follows:

$$\begin{aligned}
\frac{dx}{dt} &= \Lambda - \mu x - k_1 xv, \\
\frac{dy}{dt} &= (1-\psi)k_1 xv - \beta vy - k_2 y, \\
\frac{dv}{dt} &= \psi k_1 xv + \beta vy - k_3 v + \theta y, \\
\frac{dz}{dt} &= \delta_1 y + \delta_2 \phi v - \mu z,
\end{aligned} \tag{2}$$

The description of parameters and variables used in model 1 and 2 are presented in **Table 1** below

Table 1 :

Parameters Used in Model & their definition

Parameters	**Definitions**
Λ	Rate of Recruitment into Popn.
μ	Natural removal/death
ϕ	Rate of case detection
α_1	Transmission rate (determined)
α_2	Transmission rate (undetermined)
ψ	Fast progression rate to infection class
β	Rate of contact between $expose\&Infected$
θ	Progression rate to Infected class from Expose class
δ_1, δ_2	Rates of recovery (treatment cases)
d	Death removal rate (infection cases)

where $k_1 = [\alpha_1\phi + \alpha_2(1-\phi)]$, $k_2 = (\mu + \delta_1 + \theta)$, $k_3 = (\mu + d + \delta_2\phi)$. one can verify that, the given system of equation in 1 characterise a dynamical systems on biological feasible region with convenient domain which is given as:

$$\Theta = \{(x, y, v, z) \in \mathbb{R}^4_+ : x + y + v + z \leq \frac{\Lambda}{\mu}\}$$

and $\dot{x} \geq 0$, $\dot{y} \geq 0$, $\dot{v} \geq 0$, $\dot{z} \geq 0$, , $\dot{N} \leq 0$ when $x = 0, y = 0, v = 0, z = 0$ and $N = \frac{\Lambda}{\mu}$ respectively.
It is easy to show that the given domain Θ is positively invariant set.
Also we can observed that the rate at which the total population N change can be written as

$$N' = \Lambda - \mu N - \mu(y + v + z) - \mu y - (\mu + d)v - \mu z$$

One can verify using a standard comparison theorem that

$$N(t) \leq N(0)e^{-dt} + \frac{\Lambda}{\mu}(1 - e^{-dt}).$$

If $N(0) \leq \frac{\Lambda}{\mu}$, then clearly $N(t) \leq \frac{\Lambda}{\mu}$ which implies the positively invariant of Θ under the system in (1). Thus we can deduce that there exist a non-negative solution of the system for time $t \geq 0$.

2.1 Basic Reproduction Number

The basic reproduction number $\mathcal{R}_o$ is obtain by using Next-Generation matrix methods and follows from [1] as:

$$\mathcal{R}_o = \frac{[(1-\psi)\theta + \psi k_2]k_1\Lambda}{\mu k_2 k_3}$$

3 Model Analysis

Let $X(x, y, v, z) = (x_1, x_2, x_3, x_4)$ so that the overall population is $N = x_1 + x_2 + x_3 + x_4$. Therefore the corresponding F is obtained by using Theorem 3.1 of [15].

$$F(X) = \begin{pmatrix} A - \mu x_1 - k_1 x_1 x_3 \\ (1-\psi)k_1 x_1 x_3 - \beta x_2 x_3 - k_2 x_2 \\ \psi k_1 x_1 x_3 + \beta x_2 x_3 - k_3 x_3 + \theta x_2 \\ \delta_1 x_2 + \delta_2 \phi x_3 - \mu x_4 \end{pmatrix} \tag{3}$$

The function F is *Lipschitz continuous*. Then, the dynamic behavior of above process $(X(t), t \geq 0)$, (see for example in [15]), can be approximated by a system of 1st order ordinary differential equations as:

$$X'(t) = F(X), \quad \text{as} \quad N \to \infty \tag{4}$$

3.1 The Existence of Equilibria

The equilibria points of the system 2 is obtain by equating the compartment of the system (2) equal to 0 [7] i.e $\frac{dx}{dt} = 0$, $\frac{dy}{dt} = 0$, $\frac{dv}{dt} = 0$ and $\frac{dz}{dt} = 0$. hence the following two (2) equilibria of the model exist:

3.1.1 The Disease-free Equilibrium

The following is given as disease-free equilibrium (DFE) is given by

$$E^{(0)} = (x^0, y^0, v^0, z^0) = (\frac{\Lambda}{\mu}, 0, 0, 0). \tag{5}$$

3.1.2 Endemic Equilibrium

The endemic equilibrium (EE) is given by

$$E^* = (x^*, y^*, v^*, z^*) \tag{6}$$

with,

$$x^* = \frac{\Lambda}{\mu + k_1 v^*},$$
$$y^* = \frac{(1-\psi)k_1 x^* v^*}{\beta v^* + k_2},$$
$$z^* = \frac{\delta_1 y* + \delta_2 \phi v^*}{\mu},$$

and v^* this is obtained as the root of the following quadratic equation,

$$g(v^*) = A_1(v^*)^2 + B_1 v^* + C_1 = 0 \tag{7}$$

Where

$A_1 = \beta k_1 k_3$,

$B_1 = \beta\mu k_3 + k_1 k_2 k_3 - \beta k_1 \Lambda$ and

$C_1 = \mu k_2 k_3 - \theta(1-\psi)k_1\Lambda - \psi k_1 k_2 \Lambda = \mu k_2 k_3(1 - R_0)$.

Hence the number of positive root of 7 tells about the count of positive-endemic equilibrium of model 2 above. The following cases shed more light:

1. If $C_1 < 0$ that is only when the relevant reproduction number R_0 is above unity, then a unique equilibrium point.
2. When $B_1 < 0$ and $C_1 < 0$ or If $B_1^2 - 4A_1C_1 = 0$, then $f(x_3^*) = 0$, then we have exactly 1 point of endemic-equilibrium.
3. If $C_1 > 0$, $B_1 < 0$ and $B_1^2 - 4A_1C_1 > 0$, i.e associated threshold number is below unity, then the system will have exactly (2) endemic equilibria (say E_1^* and E_2^*).
4. Otherwise, no equilibria (endemic) exist thus, if $A_1C_1 > 0$, $B_1 > 0$.

When $R_0 < 1$, thus

$$R_0^c = 1 - \frac{B_1^2}{4A_1\mu k_2 k_3} \tag{8}$$

such that $4A_1C_1 > (=or<)0$ if $R_0 > (=or<) R_0^c$,

where R_0^c is defined to be the backward-bifurcation points and it appear for $R_0^c < R_0 < 1$ (see in case 3. above). For details of bifurcation analysis See for e.g in [1]

3.2 Stability Analysis

3.2.1 The Local Stability of Disease-free Equilibrium

Theorem 1 *The disease-free equilibrium (DFE) point* $E^{(0)} = (x^0, y^0, v^0, z^0) = (\frac{\Lambda}{\mu}, 0, 0, 0)$ *in (5), is locally asymptotically stable when* $R_0 < 1$ *and unstable when* R_0 *is less than unity.*

Proof The Jacobian matrix of (2) is given by

$$J(X) = \begin{pmatrix} -\mu - k_1 v & 0 & -k_1 x & 0 \\ (1-\psi)k_1 v & -\beta v - k_2 & (1-\psi)k_1 x - \beta y & 0 \\ \psi k_1 v & \beta v + \theta & \psi k_1 x + \beta y - k_3 & 0 \\ 0 & \delta_1 & \delta_2\phi & -\mu \end{pmatrix} \tag{9}$$

Now evaluating (5) in (9) yields:

$$J(E^0) = \begin{pmatrix} -\mu & 0 & -k_1\frac{\Lambda}{\mu} & 0 \\ 0 & -k_2 & (1-\psi)k_1\frac{\Lambda}{\mu} & 0 \\ 0 & \theta & \psi k_1\frac{\Lambda}{\mu} - k_3 & 0 \\ 0 & \delta_1 & \delta_2\phi & -\mu \end{pmatrix} \tag{10}$$

Here, matrix (10) posses four (4) eigenvalues as follows:

$$\begin{aligned} r_1 &= -\mu, \\ r_2 &= -\mu, \\ r_3 &= -\frac{\sqrt{D}}{2\mu} + \frac{k_2\mu + k_3\mu - k_1\Lambda\mu}{2\mu}, \\ r_4 &= \frac{\sqrt{D}}{2\mu} - \frac{k_2\mu + k_3\mu - k_1\Lambda\mu}{2\mu}. \end{aligned} \tag{11}$$

Where,
$D = (k_3\mu - k_1\Lambda\psi - k_2\mu)^2 - 4\theta k_1\Lambda\mu(\psi - 1)$. Therefore, the stability of (5) is obtained by the eigenvalues in r_3 and r_4, since the remaining eigenvalues will be negative for nonnegative parameters. Hence, the disease-free equilibrium point (5) is *stable* iff $r_3, r_4 < 0$. □

3.3 The Local stability of Endemic equilibrium (EE) point

Theorem 2 *The endemic equilibrium (EE) of model (2) is locally asymptotically stable when $a_3a_2 - a_1 > 0$, the values of a_3, a_2 and a_1 are the coefficient in the polynomial of the variational matrix corresponding to EE.*

Proof On evaluating EE point of (6) on the Jacobian matrix (9) we obtained the following variational matrix as:

$$J(E^*) = \begin{pmatrix} e_{11} & 0 & e_{13} \\ e_{21} & e_{22} & e_{23} \\ e_{31} & e_{32} & e_{33} \end{pmatrix} \tag{12}$$

Where,

$$e_{11} = -\mu - k_1v^*, \quad e_{13} = -\frac{k_1\Lambda}{\mu + k_1v^*}, \quad e_{21} = (1-\psi)k_1v^*, \quad e_{22} = -\beta v^* - k_2,$$
$$e_{23} = \frac{(1-\psi)k_1\Lambda}{\mu + k_1v^*} - \beta y^*, \quad e_{31} = pk_1v^*, \quad e_{32} = \beta v^* + \theta, \quad e_{33} = \frac{\psi k_1\Lambda}{\mu + k_1v^*} + \beta y^* - k_3,$$

Hence matrix (12) has three (3) eigenvalues, which will the solutions of the following polynomial:

$$l^3 + a_3l^2 + a_2l + a_1 = 0$$

where,

$$a_3 = (e_{11} + e_{22} + e_{33}),$$
$$a_2 = [(e_{22}e_{33} - e_{23}e_{32}) + (e_{11}e_{33} - e_{31}e_{13}) + (e_{11}e_{22} - 0)],$$
$$a_1 = [e_{11}(e_{22}e_{33} - e_{23}e_{32}) - 0 + e_{13}(e_{21}e_{32} - e_{31}e_{22})].$$

Thus, by employing Routh-Hurwitz criteria (as in [1]), the endemic equilibrium point (6) will be locally-asymptotically *stable* subject to the following circumstances are met.

$$a_3 > 0 \quad \textit{and} \quad \begin{vmatrix} a_3 & a_1 \\ 1 & a_2 \end{vmatrix} > 0.$$

Here obviously $a_3 > 0$, so EE is locally-asymptotically stable if the determinant matrix above satisfied the remaining inequality. □

4 Stochastic model

To derive an equivalence SDE model of (2), Assume $X(t) = (X_1(t),\ X_2(t),\ X_3(t),\ X_4(t))^{tr}$ to be a random variables (continuous) for $(x(t),\ y(t),\ v(t),\ z(t))^T$ which denotes TB-Susceptible, TB-Exposed, TB-Infected and TB-Recovered/Treated individuals at time t in the population respectively as mentioned earlier. The model (2) (deterministic), comprises of nine (9) possible states changes below. looking at possible states changes and their relevant probabilities, we can compute the following expectations $E(\Delta X)$ and $E((\Delta X)(\Delta X)^T)$ By neglecting terms higher than $o(\Delta t)$ (see for example in [10]).
The expectation of ΔX is then defined to be a drift vector $\mathbf{f}$ multiply by Δt:

Table 2 Possible changes in the four classes with their probabilities

Possibility state change Δx_j	**Probability** P_j
$\Delta x_1 = [1,0,0,0]^T$	$P_1 = \Lambda \Delta t$
$\Delta x_2 = [-1,0,0,0]^T$	$P_2 = \mu X_1 \Delta t$
$\Delta x_3 = [-1,1,0,0]^T$	$P_3 = k_1 X_1 X_3 \Delta t$
$\Delta x_4 = [0,-1,1,0]^T$	$P_4 = (\psi k_1 X_1 + \beta X_2) X_3 \Delta t$
$\Delta x_5 = [0,-1,0,0]^T$	$P_5 = k_2 X_2 \Delta t$
$\Delta x_6 = [0,0,-1,0]^T$	$P_6 = k_3 X_3 \Delta t$
$\Delta x_7 = [0,0,1,0]^T$	$P_7 = \theta X_2 \Delta t$
$\Delta x_8 = [0,0,0,1]^T$	$P_8 = (\delta_1 X_2 + \delta_2 \phi X_3) \Delta t$
$\Delta x_9 = [0,0,0,-1]^T$	$P_9 = \mu X_4 \Delta t$
$\Delta x_{10} = [0,0,0,0]^T$	$P_{10} = 1 - \sum_{j=1}^{9} P_j \Delta t$

$$E(\Delta X) = \sum_{j=1}^{9} p_j \Delta x_j = \mathbf{f}(x, y, v, z)\Delta t, \tag{13}$$

,

Where

$$\mathbf{f} = \begin{pmatrix} \Lambda - \mu X_1 - k_1 X_1 X_3 \\ (1-\psi) k_1 X_1 X_3 - \beta X_2 X_3 - k_2 X_2 \\ \psi k_1 X_3 + \beta X_2 X_3 - k_3 X_3 + \theta X_2 \\ \delta_1 X_2 + \delta_2 \phi X_3 - \mu X_4 \end{pmatrix} \tag{14}$$

One can clearly observed that, the drift vector $\mathbf{f}$ is the same as RHS of the ODE model in (2). By neglecting the term of order $o(\Delta)^2$. Thus, we found $V(\Delta X)$ as:

$$V(\Delta X) \approx E((\Delta X)(\Delta X)^T) = \sum_{j=1}^{8} p_j \Delta x_j (\Delta x_j)^T \tag{15}$$

$$E((\Delta X)(\Delta X)^T) = \begin{pmatrix} v_{11} & v_{12} & 0 & 0 \\ v_{21} & v_{22} & v_{23} & 0 \\ 0 & v_{32} & v_{33} & 0 \\ 0 & 0 & 0 & v_{44} \end{pmatrix} .\Delta t = V.\Delta t, \tag{16}$$

Thus, above define a *diffusion-matrix* (see in [10, 12, 13]) which is symmmetric, positive-definite matrix with its components obtained as:

$$
\begin{aligned}
v_{11} &= P_1 + P_2 + P_3 = \Lambda + dX_1 + k_1X_1X_3,\\
v_{12} &= v_{21} = -P_3 = -k_1X_1X_3,\\
v_{22} &= P_3 + P_4 + P_5 = k_1X_1X_3 + (\psi k_1X_1 + \beta X_2)X_3 + k_2X_2,\\
v_{23} &= v_{32} = -P_4 = -(\psi k_1X_1X_3 + \beta X_2X_3)\\
v_{33} &= P_4 + P_6 + P_7 = \psi k_1X_1X_3 + \beta X_2X_3 + k_3X_3 + \theta X_2\\
v_{44} &= P_8 + P_9 = \delta_1X_2 + \delta_2\phi X_3 + \mu X_4.
\end{aligned}
$$

Even though many work shows that there is no explicit way for obtaining the square-root of above matrix with such dimension of V (see for e.g [10, 12]). Hence, We go with methods in [12, 13, 14] and found an equivalent matrix, G such that $V = GG^T$, where the matrix G is of dimension 4×9 as:

$$
G = \begin{pmatrix}
\sqrt{\Lambda} & -\sqrt{\mu X_1} & -\sqrt{k_1X_1X_3} & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \sqrt{k_1X_1X_3} & -\sqrt{a} & -\sqrt{k_2X_2} & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \sqrt{a} & 0 & -\sqrt{k_3X_3} & \sqrt{\theta X_2} & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \sqrt{b} & \sqrt{dX_4}
\end{pmatrix} \tag{17}
$$

wherein, $a = (\psi k_1X_1 + \beta X_2)X_3$, $b = \delta_1X_2 + \delta_2\phi X_3$
Then, we can write the Ito stochastic differential model as:

$$
\begin{cases}
d\mathbf{X}(t) &= \mathbf{f}(t, \mathbf{X})dt + G(t, \mathbf{X})d\mathbf{W}(t),\\
\mathbf{X}(0) &= (X_1(0), X_2(0), X_3(0), X_4(0))^t r,
\end{cases} \tag{18}
$$

wherein, $\mathbf{W}(t) = (W_1(t), W_2(t), ..., W_9(t))^T$, is a vector of nine independent Wiener-processes. The drift vector $\mathbf{f}$ is given in (14), and the diffusion matrix G is a 4×9 matrix in (17). Hence the following SDE model is then constructed as follows:

$$
\begin{aligned}
dx &= \big(\Lambda - \mu x - k_1xv\big)dt + \sqrt{\Lambda}\, dW_1 - \sqrt{\mu x}\, dW_2 - \sqrt{k_1xv}\, dW_3;\\
dy &= \big[(1-\psi)k_1xv - \beta vy - k_2y\big]dt + \sqrt{k_1xv}\, dW_3\\
&\quad - \sqrt{\psi k_1xv + \beta vy}\, dW_4 - \sqrt{k_2y}\, dW_5;\\
dv &= \big(\psi k_1xv + \beta vy - k_3v + \theta y\big)dt + \sqrt{\psi k_1xv + \beta vy}\, dW_4\\
&\quad - \sqrt{k_3v}\, dW_6 + \sqrt{\theta y}\, dW_7;\\
dz &= \big(\delta_1y + \delta_2\phi v - \mu z\big)dt + \sqrt{\delta_1y + \delta_2\phi v}\, dW_8 - \sqrt{\mu z}\, dW_9.
\end{aligned} \tag{19}
$$

5 Numerical Experiments and Discussion

In this part of the work wen illustrate fews of the numerical simulation of models (1) and (19). the values of basic parameter used for TB model (1) are adopted from [1] and some parameters are assumed. The following were considered as values for the set of parameters for Tuberculosis deterministic Model. (1).

$$
\begin{gathered}
\Lambda = 18,\ \mu = 0.075,\ \phi = 0.9,\ \alpha_1 = 0.001,\ \alpha_2 = 0.003\\
\psi = 0.4,\ \beta = 0.005,\ \theta = 0.0001,\ \delta_1 = \delta_2 = 0.08,\ d = 0.032
\end{gathered}
$$

We can see the stability of of disease free equilibrium (DFE) E^0 in plotted in figure 2 for initial values of $x(0) = 150$, $y(0) = 50$, $v(0) = 60$ and $z(0) = 80$. The results of DFE is obtained $E^0 = (240, 0, 0, 0)$.

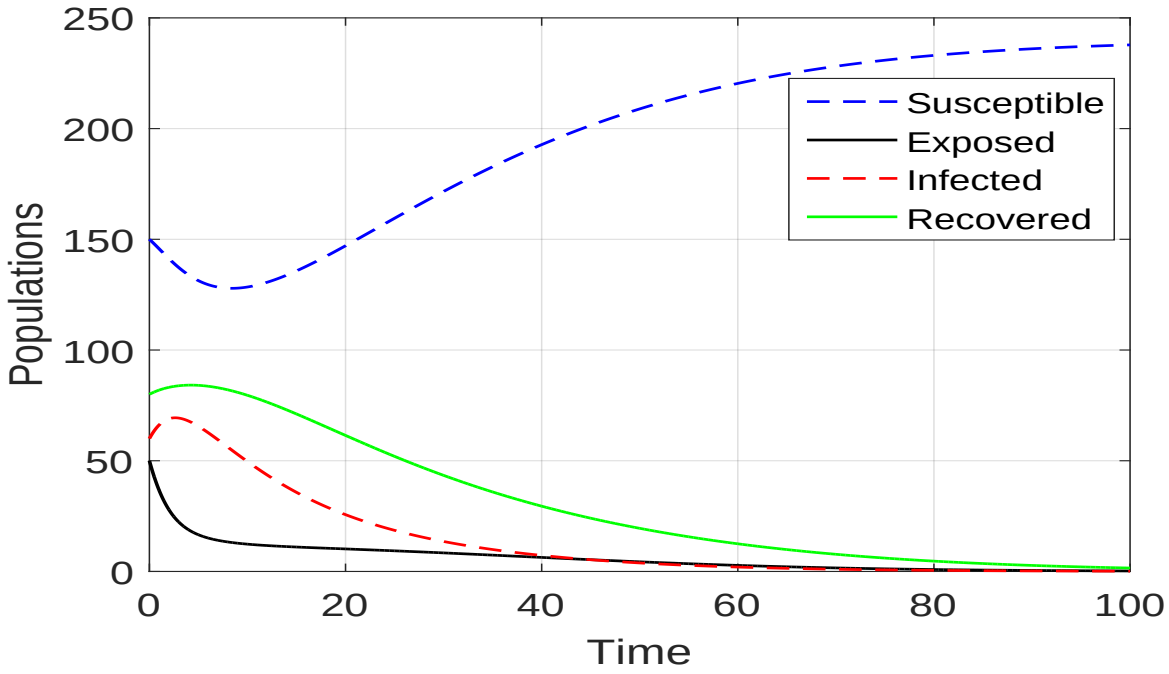

Fig. 2 Variation of Population with time for $R_0 < 1$

The impact of case-detection showcase an important role in the dynamics of TB disease, this is illustrated in figure (3) where the infected class are simulated for different values of ϕ, the plot shows for values of $\phi = 0.9$ the infections decreases to zero over time and if $\phi = 0.6$ the infections increases which tell about the significant effect of case detection is very vital towards eliminating the diseases.

In Figure (4), at time $t = 0$ and from an initial state, the path of the ODE system (1) and that of SDE model (19) act relatively same for some point over time. Thus one can observed that the Stochastic model trajectories are oscillating this happen as a results of the random white noise presence in the environment. We can not dismiss the effect of random white-noise which is forcing the solutions of the SDE model to strongly oscillate over time. Also trajectories of Exposes class/subgroup of deterministic model (1) were compared to that of stochastic model (19). A significance difference is observed in the two plots this is also has to do with random white noise presence in the stochastic model and this is clearly true because of nature TB disease transmission since any individual/persons within host-population get a chance of been exposed randomly by the infected individual/persons through interactions.

In figure (6) a plot of Infected class of deterministic and stochastic verses time is shown. The presence of random white noise display a strong oscillation for stochastic trajectory this lead to a narrow gap between the two curves. This happened because of random white-noise as earlier discuss, translated by a supplementary term added whose trajectories comes to superimpose the deterministic trajectories.

6 Summary and Conclusion

In this chapter we have derive and extended nonlinear deterministic model of (TB) by considering the effect of case-detection and its treatment to a Stochastic Differential Equation Model (SDE). We have dynamically study the behavior of the two models by revising the analysis in the deterministic method. Different Equilibria were found for the models using convenient techniques and analyzed their condition for stability for values of basic reproduction number below unity $\mathcal{R}_0 < 1$ and above unity $\mathcal{R}_0 > 1$. A bright picture by influence of treatment (see also [1]) together with

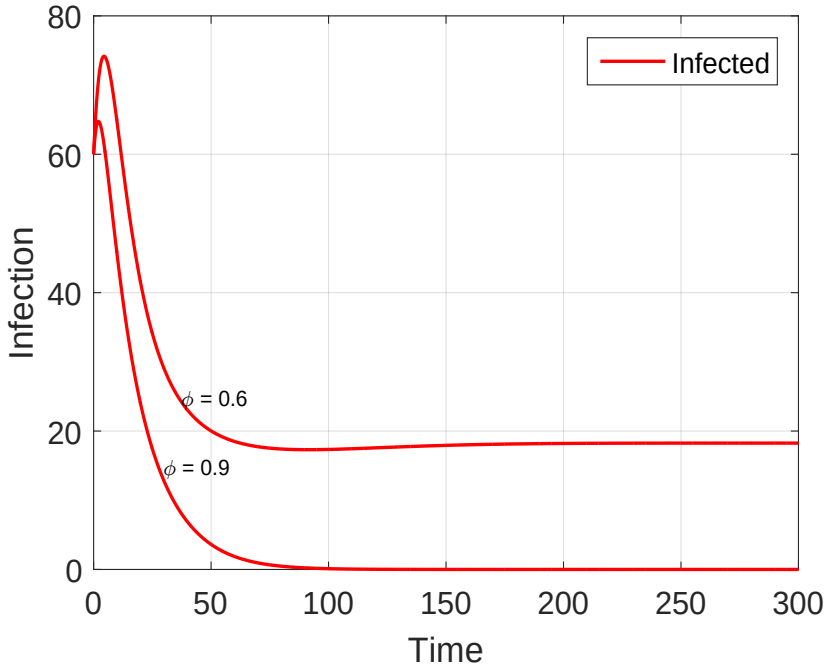

Fig. 3 Variation of Infective Class with time for different values of ϕ

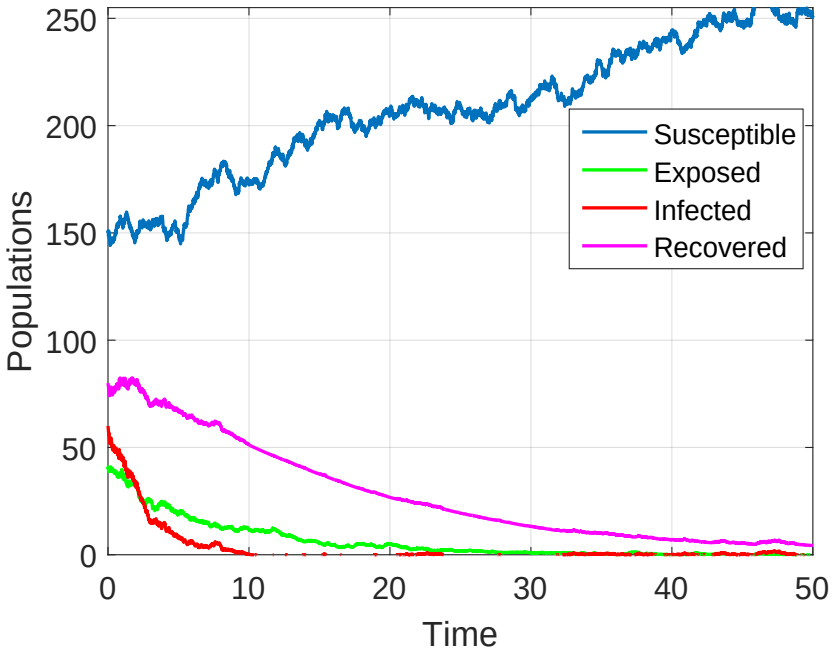

Fig. 4 Variation of Population with time for SDE

case-detection on the eradication strategies of TB disease were quantitatively seen in the model. Generally, the work lamented the likelihood of TB disease containment of its spread is eventually possible, if the treatment guidelines can be strengthen and case-detection be strongly improve this is proves through some analytic and numerical simulation for ODE and SDE models.

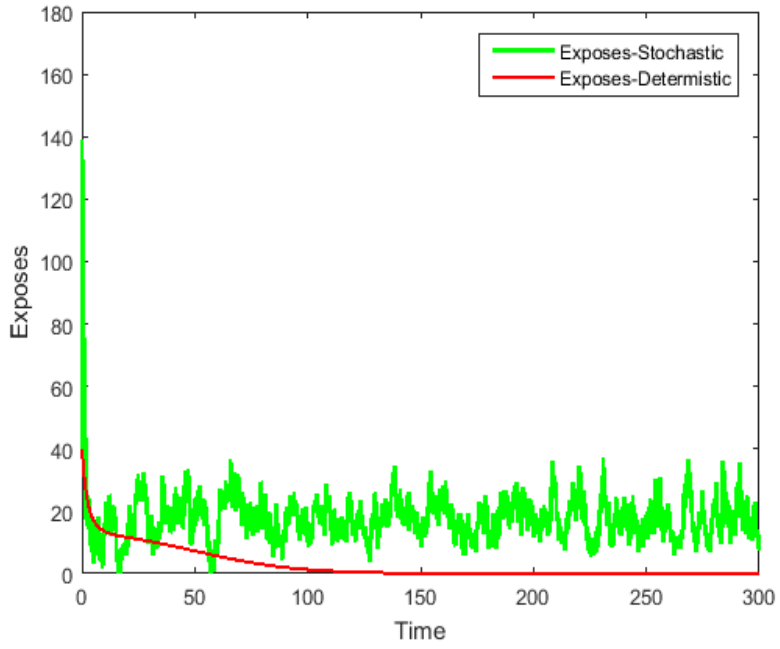

Fig. 5 Plots of Expose Popn verses time for Deterministic and SDE models

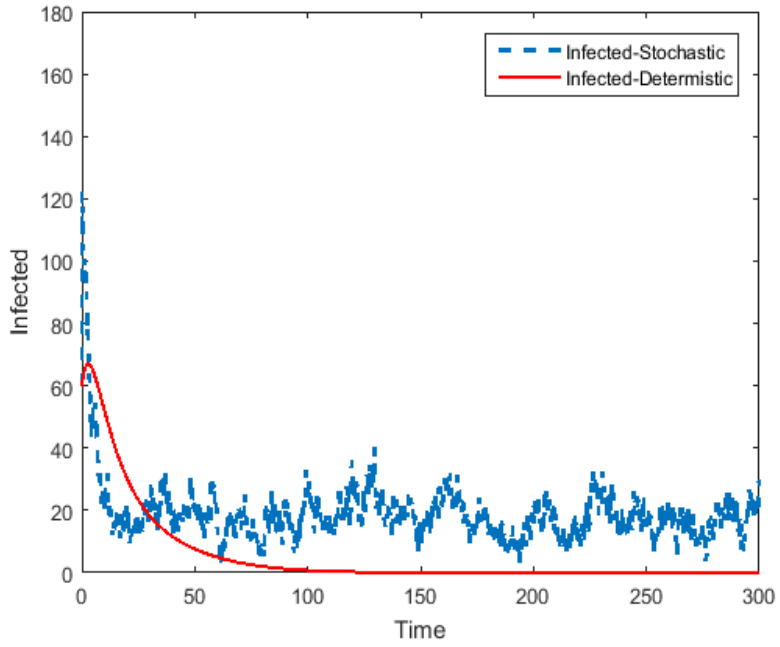

Fig. 6 Plots of Infected Popn verses time for Deterministic and SDE Models

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