

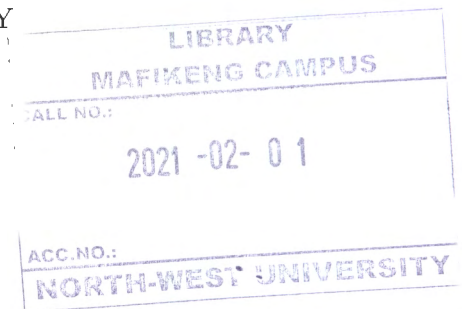
A theoretical analysis of stochastic epidemiological models under delay and fractional brownian motion

By

Mainza Mbokoma (26966395)

SUBMITTED IN FULFILLMENT OF THE ACADEMIC
REQUIREMENTS FOR THE DEGREE OF
MASTERS OF SCIENCE
IN THE
SCHOOL OF MATHEMATICAL SCIENCES
NORTH-WEST UNIVERSITY
MAFIKENG

FEBRUARY 2016



Supervisor: Prof. S.C. OUKOUOMI NOUTCHIE

Abstract

This study examined selected nonlinear Stochastic Differential Equations arising in disease modeling. The main objective of this study was to include randomness into the dynamics of tuberculosis and HIV. Stochasticity to the models was introduced through perturbation of parameters which is a standard method in stochastic population modeling. The well-posedness analysis of SDEs included the existence of non-negative solutions as required in the dynamics of population modeling. A detailed stability analysis of results, analytical properties and asymptotical behavior of solutions was also done. The mean reverting process was approximated for one of the variables in the TB and HIV models and the mean and variance of the process was found.

Preface

This study was conducted under the supervision of Professor S.C. Oukouomi Noutchie, Department of Mathematical Sciences, North-West University, Mafikeng Campus.

The study is the original work of the author and has not been submitted in any form for a degree or diploma at any other University.

Signed:

.....

M. Mbokoma (Student)

.....

Professor S.C. Oukouomi Noutchie (Supervisor)

Dedication

I dedicate this study to my mother, Mary Ng'andwe Chileka, my aunt, Eunice Mbokoma Banda and all my family members and friends. This is the fruit of your patience.



Acknowledgements

I am deeply grateful to the Almighty God for guiding me throughout my studies. I thank my supervisor, Professor S.C. Oukouomi Noutchie for accepting to supervise me. I appreciate his patience, guidance and support during my studies. I am grateful for the received financial support from the NWU (Mafikeng campus) and UNZA. I thank the staff of the Department of Mathematical Sciences, North-West University (Mafikeng campus) for the conducive environment offered during my studies as well as the opportunity to serve as a student assistant in the department. I extend gratitude to the Department of Mathematics and Statistics of the University of Zambia for giving me the opportunity to further my studies. I thank my friends and housemates, Hope, Chrisper and Dennis for making it possible for me to study at the NWU and for their support. Finally, I am sincerely grateful to Yolanda Mutemwa for her love, friendship and support during my stay at the graduate school.

Notation and conventions

$A := B$	A is defined by B
$[a, b], (a, b)$	closed and open intervals from a to b respectively
$\mathbb{N}, \mathbb{N}_0, \mathbb{Z}$	$\{1, 2, \dots\}, \{0, 1, 2, \dots\}, \{\dots, -2, -1, 0, 1, 2, \dots\}$
$a \vee b, a \wedge b$	maximum and minimum of a and b respectively
$A \subseteq B$	A is contained in B or $A = B$
$f(\cdot), g(\cdot_1, \cdot_2)$	The functions $x \rightarrow f(x), (x_1, x_2) \rightarrow g(x_1, x_2)$
1_A	The indicator function of the set A
$(\Omega, \mathcal{F}, P)$	Probability space
$\sigma(Z_i, i \in I)$	The σ -algebra generated by the family $(Z_i)_{i \in I}$ of sets
$\mathcal{B}^n, \mathcal{B}(M)$	The σ -algebra of Borel set in $\mathbb{R}^n$ and in $M \subset \mathbb{R}^n$ respectively
$P, E[X], \text{Var}[X]$	Probability, expected value and variance of X respectively
$X_n \stackrel{P}{=} X$	X_n converges P -stochastically to X
$X_n \stackrel{L}{=} X$	X_n converges in Law to X
$\mathcal{N}(\mu, \sigma^2)$	Normal distribution with mean μ and variance σ^2
$\mathcal{L}^p(I, \mathbb{R}^n)$	p -integrable function $f : I \rightarrow \mathbb{R} \left(\int_I f ^p < \infty \right)$
$\mathcal{C}(I, \mathbb{R}^n)$	$\{f : I \rightarrow \mathbb{R}^n f \text{ is continous}\}$
$\ f\ _\infty$	$\sup_x f(x) $
$\xi(t)$	white noise
$W(t)$ or $B(t)$	Wiener process or Brownian motion at time t
$X(t)$	Solution process to SDE at time t
$(\mathcal{F}_t)$	Filtration for $(W(t), t \geq 0)$
A^t	Transpose of the matrix A
$ A $	Norm of the $n \times m$ matrix $A : A ^2 = \sum_{i=1}^n \sum_{j=1}^m a_{i,j}^2 = \text{tr}(AA^t)$
$\text{tr}(A) = \sum_{i=1}^n a_{ii}$	trace of the matrix A , where a_{ii} denotes the entry on the i -th row and i -th column of A
$ x $	The norm of $x \in \mathbb{R}^n : x ^2 = \sum_{i=1}^n x_i^2 = xx^t$

Contents

Abstract	i
Preface	ii
Dedication	iii
Acknowledgements	iv
Notations and conventions	v
1 Introduction	1
2 Preliminary and auxiliary results	3
2.1 Fundamentals of probability theory	3
2.2 Stochastic processes	7
2.3 Stochastic differential equations	10
2.4 Fractional brownian motion	14
2.4.1 Stochastic differential equations driven by fractional brownian motion	15
2.4.2 Itô's formula with respect to fractional brownian motion	16
3 Mathematical analysis of a stochastic tuberculosis model	21
3.1 Introduction	21
3.2 Deterministic model	23
3.2.1 Basic reproduction ratio	25
3.2.2 Global stability of the disease-free equilibrium	25

3.2.3	Existence and uniqueness of endemic equilibrium	26
3.2.4	Global stability of endemic equilibrium	27
3.3	Stochastic model	30
3.3.1	Non-negative solutions	31
3.3.2	Asymptotic behaviour	34
3.3.3	Mean reverting process	40
3.4	Conclusion	42
4	Stochastic model and analysis of the dynamics of AIDS and use of condoms	43
4.1	Introduction	43
4.2	Deterministic model	44
4.3	Stochastic model	54
4.3.1	Non-negative solutions	55
4.3.2	Asymptotic behaviour and stability	59
4.3.3	Stability at the disease-free equilibrium	62
4.4	Results and discussion	68
5	Stochastic model for internal HIV dynamics driven by fractional brownian motion	69
5.1	Introduction	69
5.2	Deterministic model	70
5.3	Stochastic model	73
5.3.1	Non-negative solutions	75
5.3.2	Asymptotic behaviour	77
5.3.3	Mean reverting process	83
5.4	Conclusion	85
6	Conclusion	86
	References	88

1

Introduction

A stochastic model is a tool for estimating probability distributions of potential outcomes by allowing for random variation in one or more inputs over time. The random variation is usually based on fluctuations observed in historical data for a selected period, using standard time-series techniques. Distributions of potential outcomes are derived from a large number of simulations (stochastic projections) which reflect the random variation in the input(s). Stochastic differential equations (SDEs) were first initiated and developed by Itô (1942). The theory provides a useful tool to introduce the notion of inherent randomness into deterministic models and characterise their long-term behaviour. Understanding and predicting rare events caused by large fluctuations is often crucial in describing dynamics of complex disordered many-body systems. Rare events may lead to diverse phenomena such as crystal nucleation and growth, self-assembly of macromolecules, protein folding, population extinction and loss of bio-diversity. Such complex systems are, in general, intrinsically far from equilibrium and therefore, defy many standard techniques of statistical mechanics. An important category of such complex systems includes systems containing a discrete, large yet finite population of interacting agents such as molecules, bacteria, cells, animals or even humans. Such systems are intrinsically disordered as they experience constant fluctuations due to the discreteness of particles and stochastic nature of the interactions between them. The stochastic dynamics of such systems can often be accurately described by assuming the Markov property and using the corresponding master equation. The latter can be viewed as a comprehensive description, effectively accounting for the internal degrees of freedom of the particles through the interaction rates. When the population size is sufficiently large, the system will typically dwell in a close vicinity of the peak (or one of the peaks) of its probability distribution of population sizes. Most of the time, the system weakly fluctuates about this peak, whereas it only rarely undergoes a large fluctuation of the order of the typical population size. Nevertheless, it is precisely these extreme rare events which may be of great importance in many applications, especially if they have irreversible or devastating consequences, such as population extinction, population explosion or population switching between metastable states. The notion of stochastic differential should always be clearly defined from a mathematical point of view as it is not unique and depends on the understanding

of a random process and its intrinsic derivatives. The purpose of this study is to include stochastic and fractional stochastic calculus into some existing deterministic models in epidemiology in order to capture the effects of unpredictable events.

In chapter 2 some important terminologies used in the study are defined. The first section presents the fundamental aspects of measure theory and provides a brief introduction to probability theory. The second section discusses stochastic processes, the notion of Brownian motion and Markov processes. The third section examines Stochastic Differential Equations and their solvability in relation to Itô's theorem. The last section discusses Stochastic differential equation driven by fractional Brownian motion and provides an Itô's theorem with respect to fractional Brownian motion.

Chapter 3 focuses on the stochastic model of tuberculosis. The results of S. Bowong, J. Tewa and J. Kamgang [9] are extended by making their deterministic model into a stochastic one and showing that the new model has non-negative solutions. A detailed analysis on the stability of the model is also presented. The mean reverting process that approximates one of the variables is also found and for this process, its mean and variance are also found.

Chapter 4 examines a stochastic model representing the spread of HIV-AIDS in a homosexual population. The effects of environmental noise on the spread of the disease are examined. A detailed analysis on asymptotic stability is done for both in probability and in pth moment.

Chapter 5 focuses on a stochastic model driven by fractional Brownian motion to describe the dynamics of HIV-1 infection. The stochastic model studied is changed from Dalal et al [20] to a stochastic model driven by fractional Brownian motion. The new model is shown to have non-negative results. The stability of the model is also discussed.

Chapter 6 provides a summary of results of the investigations.



2

Preliminary and auxiliary results

2.1. Fundamentals of probability theory

A brief introduction of measure theory is given as far as it is needed in modern probability. The measure theoretic foundations of probability theory is explained. There is a lot of literature that can be read for further information on the introduction to measure theory, for example [11], [12], [14],[24] or [39].

Definition 2.1.1. Let Ω be a non-empty set. A collection $\mathcal{F}$ of subsets of Ω is called a sigma-algebra (σ -algebra) if it satisfies the following properties:

- (i) $\phi, \Omega \in \mathcal{F}$,
- (ii) if $X \in \mathcal{F}$, then $X^c \in \mathcal{F}$, where $X^c := \Omega - X$, is the compliment of X in Ω ,
- (iii) if $X_1, X_2, \dots \in \mathcal{F}$, then $\bigcup_{k=1}^{\infty} X_k, \bigcap_{k=1}^{\infty} X_k \in \mathcal{F}$.

Definition 2.1.2. The pair $(\Omega, \mathcal{F})$ of a set Ω and a σ -algebra $\mathcal{F}$ is the measurable space and the elements of $\mathcal{F}$ are measurable sets. The function $P : \mathcal{F} \rightarrow [0, 1]$ is a probability measure on $(\Omega, \mathcal{F})$ provided that:

- (a) $P(\phi) = 0, P(\Omega) = 1$,
- (b) if $X_1, X_2, X_3, \dots \in \mathcal{F}$, then $P(\bigcup_{k=1}^{\infty} X_k) \leq \sum_{k=1}^{\infty} P(X_k)$,
- (c) for any disjoint sets $X_1, X_2, \dots \in \mathcal{F}$, $P(\bigcup_{k=1}^{\infty} X_k) = \sum_{k=1}^{\infty} P(X_k)$.

Hence, for any $X, Y \in \mathcal{F}$ and $X \subseteq Y$, then $P(X) \leq P(Y)$.

The subsets A of Ω that belong to $\mathcal{F}$ are known as events and $P(A)$ is interpreted as the probability that an event A will occur. A property which is true except for an event of probability zero is said to hold almost surely (abbreviated "a.s.").

Definition 2.1.3. A probability space is the triple $(\Omega, \mathcal{F}, P)$ such that Ω is any set, $\mathcal{F}$ is a σ -algebra and P is a probability measure.

Definition 2.1.4. A probability space $(\Omega, \mathcal{F}, P)$ is said to be a complete probability space if for all $B \in \mathcal{F}$ with $P(B) = 0$ and all $A \subset B$ one has $A \in \mathcal{F}$.

Often, the study of probability spaces is restricted to complete probability spaces.

Definition 2.1.5. Let $(\Omega, \mathcal{F}, P)$ be a probability space. A set A is a null set if there exist $B \in \mathcal{F}$, such that $B \supset A$ with $P(B) = 0$.

In other words a set is null if it is contained in a measurable set which has probability 0.

Definition 2.1.6. Let the class of subsets of Ω be denoted by $\mathcal{H}$. Then the σ -algebra $\sigma(\mathcal{H})$ generated by $\mathcal{H}$, is the smallest σ -algebra $\mathcal{F}$ on Ω such that $\mathcal{H} \subseteq \mathcal{F}$. It is obtained by finding the intersection of all the σ -algebras on Ω that have $\mathcal{H}$ as a subclass.

$$\sigma(\mathcal{H}) = \bigcap \{ \mathcal{F}_i \mid \mathcal{F}_i \text{ is } \sigma\text{-algebra of } \Omega, \mathcal{H} \subseteq \mathcal{F}_i, i = 1, 2, \dots \}.$$

Definition 2.1.7. The Borel σ -algebra $\mathcal{B}$ is the smallest σ -algebra that contains all open (or closed) subsets of $\mathbb{R}^n$.

Definition 2.1.8. Consider a probability space $(\Omega, \mathcal{F}, P)$. Then, an n -dimensional random variable is a function $Y : \Omega \rightarrow \mathbb{R}^n$ such that for every $A \in \mathcal{B}$, $Y^{-1}(A) \in \mathcal{F}$.

Equivalently, it is concluded that Y is $\mathcal{F}$ -measurable.

Remark 2.1.9. In probability, it is important to understand that $\mathcal{F}(X)$ (σ -algebra generated by X) is interpreted as “containing all relevant information” about the random variable X .

Lemma 2.1.10. [36]

Let $Y : \Omega \rightarrow \mathbb{R}^n$ be a random variable. Then, the σ -algebra

$$\mathcal{F}(Y) := \{Y^{-1}(A) \mid A \in \mathcal{B}\}$$

is the σ -algebra generated by Y . It is the smallest sub- σ -algebra of $\mathcal{F}$ with respect to which Y is measurable.

Definition 2.1.11. Let $(\Omega, \mathcal{F}, P)$ be a probability space and X a random variable, then the expectation (or mean value) of X is given by

$$E(X) := \int_{\Omega} X dP$$

and the Variance of X is given by

$$\text{Var}(X) := \int_{\Omega} |X - E(X)|^2 dP$$

where $|\cdot|$ is just the Euclidean norm.

It is observed that

$$\text{Var}(X) = E(|X - E(X)|^2) = E(|X|^2) - |E(X)|^2.$$

One can explicitly compute the mean and variance using

$$\text{Var}(X) = \int_{\mathbb{R}^n} |x - E(X)|^2 f(x) dx \text{ and } E(X) := \int_{\mathbb{R}^n} x f(x) dx$$

Therefore, $E(X)$ and $\text{Var}(X)$ can be computed using ordinary Riemann integrals over $\mathbb{R}^n$. This is important because one cannot observe the probability space $(\Omega, \mathcal{F}, P)$, but one can only “see” what values X takes in $\mathbb{R}^n$. As a matter of fact, in probability theory, all the quantities of interest are computed in terms of the density function f in $\mathbb{R}^n$.

Definition 2.1.12. Let $(\Omega, \mathcal{F}, P)$ be a probability space and $X : \Omega \rightarrow \mathbb{R}^n$ a random variable, then

(i) the function $F_X : \mathbb{R}^n \rightarrow [0, 1]$ is the distribution function of X such that

$$F_X(x) := P(X \leq x) \text{ for every } x \in \mathbb{R}^n,$$

(ii) the joint distribution function for the random variables $X_1, X_2, \dots, X_m : \Omega \rightarrow \mathbb{R}^n$ is the function $F_{X_1, X_2, \dots, X_m} : (\mathbb{R}^n)^m \rightarrow [0, 1]$ defined by

$$F_{X_1, X_2, \dots, X_m}(x_1, x_2, \dots, x_m) := P(X_1 \leq x_1, X_2 \leq x_2, \dots, X_m \leq x_m)$$

for every $x_i \in \mathbb{R}^n, i = 1, 2, \dots, m$.

It should be noted that $x = (x_1, x_2, \dots, x_n), y = (y_1, y_2, \dots, y_n) \in \mathbb{R}^n$, $x < y$ means $x_i < y_i$ for $i = 1, 2, \dots, n$.

Definition 2.1.13. Let $F = F(y)$ be the distribution function for the random variable $Y : \Omega \rightarrow \mathbb{R}^n$. If there exist an integrable non-negative function $f : \mathbb{R}^n \rightarrow \mathbb{R}$ such that

$$F(y) = F(y_1, y_2, \dots, y_n) = \int_{-\infty}^{y_1} \cdots \int_{-\infty}^{y_n} f(x_1, \dots, x_n) dx_n \cdots dx_1,$$

then f is the probability density function of Y .

Therefore, for every $A \in \mathcal{B}$, it is concluded that

$$P(Y \in A) = \int_A f(y) dy$$

which is an important expression since the integral on the right hand side can be calculated explicitly.

Definition 2.1.14. (i) Events $X, Y \in \mathcal{F}$ are said to be independent if

$$P(X \cap Y) = P(X)P(Y).$$

(ii) Let $\mathcal{F}_i \subseteq \mathcal{F}$ for $i = 1, 2, \dots$ be σ -algebras, then $\{\mathcal{F}_i\}_{i=1}^\infty$ are independent if for any choice of $i = 1, 2, \dots, k$ and events $X_i \in \mathcal{F}_i$, one obtains

$$P(X_1 \cap X_2 \cap \cdots \cap X_k) = P(X_1)P(X_2) \cdots P(X_k).$$

(iii) If one considers the random variables $X_i : \Omega \rightarrow \mathbb{R}^n$ ($i = 1, 2, \dots$), then $X_1, X_2, \dots$ are independent random variables if for every choice of Borel sets $\mathcal{A}_1, \mathcal{A}_2, \dots, \mathcal{A}_k \subseteq \mathbb{R}^n$, $k \geq 2$ one obtains

$$P(X_1 \in \mathcal{A}_1, X_2 \in \mathcal{A}_2, \dots, X_k \in \mathcal{A}_k) = P(X_1 \in \mathcal{A}_1)P(X_2 \in \mathcal{A}_2) \cdots P(X_k \in \mathcal{A}_k).$$

This is the same as saying that the σ -algebras $\{\mathcal{F}(X_i)\}_{i=1}^\infty$ generated by the random variables X_i are independent.

The concept of independence and its ramifications are the hallmarks of probability theory.

Theorem 2.1.15. [39]

The random variables $Y_1, Y_2, \dots, Y_n : \Omega \rightarrow \mathbb{R}^n$ are independent if and only if

$$F_{Y_1, Y_2, \dots, Y_n}(y_1, y_2, \dots, y_n) = F_{Y_1}(y_1)F_{Y_2}(y_2) \cdots F_{Y_n}(y_n) \quad \text{for every } y_i \in \mathbb{R}^n, i = 1, 2, \dots, n.$$

Equivalently, using the probability density functions of the random variables one obtains

$$f_{Y_1, Y_2, \dots, Y_n}(y_1, y_2, \dots, y_n) = f_{Y_1}(y_1)f_{Y_2}(y_2) \cdots f_{Y_n}(y_n) \quad \text{for all } y_i \in \mathbb{R}^n, i = 1, 2, \dots, n,$$

where the functions f are the appropriate density functions.

Theorem 2.1.16. [39]

Let $X_1, X_2, \dots, X_n$ be independent random variables such that

$$E(|X_i|) < \infty \quad (i = 1, 2, \dots, n),$$

then $E(|X_1 X_2 \cdots X_n|) < \infty$ and

$$E(X_1 X_2 \cdots X_n) = E(X_1)E(X_2) \cdots E(X_n).$$

Theorem 2.1.17. [39]

Let $X_1, X_2, \dots, X_n$ be independent random variables such that

$$\text{Var}(X_i) < \infty, \quad i = 1, 2, \dots, n,$$

then

$$\text{Var}(X_1 + X_2 + \cdots + X_n) = \text{Var}(X_1) + \text{Var}(X_2) + \cdots + \text{Var}(X_n).$$

Definition 2.1.18. Let $(\Omega, \mathcal{F}, P)$ be the probability space with events $B_1, B_2, \dots, B_n, \dots$. Then the event

$$\bigcap_{m=1}^{\infty} \bigcup_{n=m}^{\infty} B_n = \{\omega \in \Omega \mid \omega \text{ belong to infinitely many of the } B_m\}$$

is called “ B_m infinitely often”, abbreviated “ B_m i.o.”

Lemma 2.1.19. (Borel-Cantelli Lemma) [11]

$P(B_n \text{ i.o.}) = 0$ whenever $\sum_{n=1}^{\infty} P(B_n) < \infty$.

Definition 2.1.20. (i) Let $X, Y \in \mathcal{F}$ be any two events in the probability space $(\Omega, \mathcal{F}, P)$ such that $P(Y) > 0$, then the probability of X , given Y denoted as $P(X|Y)$ is defined as

$$P(X|Y) := \frac{P(X \cap Y)}{P(Y)}.$$

(ii) Let the joint probability density function of two random variables X and Y be $f(x, y)$, then the conditional probability density function of X , given $Y = y$, defined for all values of y , such that $f_Y(y) > 0$, is given by

$$f_{X|Y}(x|y) = \frac{f(x, y)}{f_Y(y)}, \quad f_Y(y) = \int_X f(x, y) dx.$$

Definition 2.1.21. Consider two random variables X and Y . The conditional expectation of X , given that $Y = y$, defined for all values of y , such that $f_Y(y) > 0$, is given by

$$E(X|Y = y) = \int_{-\infty}^{\infty} x f_{X|Y}(x|y) dx.$$

2.2. Stochastic processes

Definition 2.2.1. (i) A stochastic process is a parametrised collection of random variables $\{X(t)|t \geq 0\}$ that assume values in $\mathbb{R}^n$ and are defined on the probability space $(\Omega, \mathcal{F}, P)$.

(ii) Every point $\omega \in \Omega$ has a corresponding sample path $t \rightarrow X(t, \omega)$.

Remark 2.2.2. (a) One thinks of t as “time” and each ω as an individual “particle” or “experiment”. $X(t, \omega)$ represents the position (or result) at time t of the particle (experiment) ω .

(b) The idea here is that when one observes the random values $X(\cdot)$ as one runs an experiment at different times, one is actually observing $\{X(t, \omega)|t \geq 0\}$, which is the sample path for a fixed $\omega \in \Omega$. In general, a different sample path is observed if one reruns the experiment.

Definition 2.2.3. Let $X : \Omega \rightarrow \mathbb{R}$ be a random variable. Then X is said to be normally distributed with mean μ and variance σ^2 (or X is an $\mathcal{N}(\mu, \sigma^2)$ random variable) if it has the following probability density function:

$$f(x) = \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{(x-\mu)^2}{2\sigma^2}}, \quad (x \in \mathbb{R}).$$

Definition 2.2.4. A Wiener process (or Brownian motion) is a real-valued stochastic process $W(\cdot)$ that satisfies the following properties:

(i) $W(0) = 0$ a.s.,

(ii) $W(t)$ is an independent increment process, i.e. for $t_1 < t_2 < t_3 < t_4$, then $W(t_2) - W(t_1)$, $W(t_3) - W(t_2)$ and $W(t_4) - W(t_3)$ are independent stochastic variables,

- (iii) $W(t) - W(s)$ has a normal distribution with mean 0 and variance $t - s$, i.e. $W(t) - W(s) \sim \mathcal{N}(0, t - s)$ for $t > s$.

Lemma 2.2.5. [46]

Let $W(\cdot)$ be a one-dimensional Wiener process. Then

$$E[W(s)W(t)] = s \wedge t = \min\{t, s\} \text{ for every } s \geq 0, t \geq 0.$$

Definition 2.2.6. Let $W(\cdot)$ be an n -dimensional Wiener process defined on the probability space $(\Omega, \mathcal{F}, P)$, then

- (i) the history of the Wiener process up to (and including) time t is the σ -algebra $\mathcal{W}(t) := \mathcal{F}(W(s) | 0 \leq s \leq t)$.
- (ii) The future of the Wiener process after time s is the σ -algebra $\mathcal{F}^+(s) := \mathcal{F}(W(t) - W(s) | t \geq s)$.

Definition 2.2.7. Consider a real valued stochastic process $X(\cdot)$, such that $E(|X(t)|) < \infty$ for every $t \geq 0$.

- (i) $X(\cdot)$ is a martingale if for every $t \geq s \geq 0$, $X(s) = E(X(t) | \mathcal{F}(s))$ a.s.
- (ii) $X(\cdot)$ is a submartingale if for every $t \geq s \geq 0$, $X(s) \leq E(X(t) | \mathcal{F}(s))$ a.s.
- (iii) $X(\cdot)$ is a supermartingale if for every $t \geq s \geq 0$, $X(s) \geq E(X(t) | \mathcal{F}(s))$ a.s.

Definition 2.2.8. Let $\mathcal{F}(\cdot) \subseteq \mathcal{F}$ be a family of σ -algebras, it is said that $\mathcal{F}(\cdot)$ is non-anticipating (with respect to the Wiener process $W(\cdot)$) if the following holds:

- (a) $\mathcal{F}(s) \subseteq \mathcal{F}(t)$ for every $0 \leq s \leq t$,
- (b) $\mathcal{F}(t) \subseteq \mathcal{W}(t)$ for every $t \geq 0$,
- (c) $\mathcal{F}(t)$ is independent of $\mathcal{F}^+$ for every $t \geq 0$. Mostly, $\mathcal{F}(\cdot)$ is called a filtration.

Remark 2.2.9. All the available information at time t is contained in $\mathcal{F}(t)$ i.e.

$$\mathcal{F}(t) = \mathcal{F}(W(s) (0 \leq s \leq t), X_0),$$

where the random variable X_0 is independent of $\mathcal{F}^+(t)$.

Definition 2.2.10. A real-valued stochastic process $X(\cdot)$ is said to be non-anticipating (with respect to $\mathcal{F}(\cdot)$) if for each time $t \geq 0$, $X(t)$ is $\mathcal{F}(t)$ -measurable. i.e. the stochastic process $X(t)$ depends only on the information available in the σ -algebra $\mathcal{F}(t)$ for every $t \geq 0$.

Definition 2.2.11. Suppose that $(X, \mathcal{A})$ and $(Y, \mathcal{B})$ are measurable spaces. The product σ -algebra $\mathcal{A} \otimes \mathcal{B}$ is the σ -algebra on $X \times Y$ generated by the collection of all measurable rectangles,

$$\mathcal{A} \otimes \mathcal{B} = \sigma(\{A \times B : A \in \mathcal{A}, B \in \mathcal{B}\}).$$

Definition 2.2.12. The stochastic process $X(t)$ is said to be measurable if for every $B \in \mathcal{B}(\mathbb{R}^n)$, the set $\{(t, \omega); X(t, \omega) \in B\}$ belongs to the product σ -algebra $\mathcal{B}([0, \infty)) \otimes \mathcal{F}$. In other words, if the mapping

$$(t, \omega) \rightarrow X(t, \omega) : ([0, \infty) \times \Omega, \mathcal{B}([0, \infty)) \otimes \mathcal{F}) \rightarrow (\mathbb{R}^n, \mathcal{B}(\mathbb{R}^n))$$

is measurable.

Definition 2.2.13. The stochastic process $X(t)$ is said to be adapted to the filtration $\mathcal{F}(t)$ if for every $t \geq 0$, the random variable $X(t)$ is $\mathcal{F}(t)$ -measurable.

Definition 2.2.14. The stochastic process $X(t)$ is progressively measurable with respect to the filtration $\mathcal{F}(t)$ if, for every $t \geq 0$ and $B \in \mathcal{B}(\mathbb{R}^n)$, the set $\{(s, \omega); 0 \leq s \leq t, \omega \in \Omega, X(s, \omega) \in B\}$ belongs to the product σ -algebra $\mathcal{B}([0, t]) \otimes \mathcal{F}$. In other words, if the mapping

$$(s, \omega) \rightarrow X(s, \omega) : ([0, t] \times \Omega, \mathcal{B}([0, t]) \otimes \mathcal{F}) \rightarrow (\mathbb{R}^n, \mathcal{B}(\mathbb{R}^n))$$

is measurable for every $t \geq 0$.

Definition 2.2.15. Let $(\Omega, \mathcal{F})$ be a measurable space with filtration $\mathcal{F}(t)$. Then a random time T of the filtration is a stopping time if the event $\{T \leq t\}$ belongs to the σ -algebra $\mathcal{F}(t)$, for each $t \geq 0$. A random time T is an optional time of the filtration if $\{T < t\} \in \mathcal{F}(t)$, for each $t \geq 0$.

Definition 2.2.16. A stochastic process $X(t)$ defined on the probability space $(\Omega, \mathcal{F}, P)$ is a Markov process if

$$P(X(t+s) \in \mathcal{A} | \mathcal{F}(t)) = P(X(t+s) \in \mathcal{A} | X(t))$$

for all Borel sets $\mathcal{A} \in \mathcal{B}$ and $s > 0$.

Remark 2.2.17. The definition reveals that the probabilities of the future values of $X(t)$ given the current value $X(s)$, can be predicted just as if one knew the entire history of the process before time s . That is, the process only “knows” its value at the present time s and does not “remember” how it got there.

Theorem 2.2.18. [14]

An n -dimensional real valued Wiener process $W(\cdot)$ defined on the probability space $(\Omega, \mathcal{F}, P)$ is a Markov process. That is,

$$P(W(t) \in \mathcal{A} | W(s)) = \frac{1}{(2\pi(t-s))^{\frac{n}{2}}} \int_{\mathcal{A}} \exp -\frac{|x - W(s)|^2}{2(t-s)} dx \quad \text{a.s.}$$

for all $0 \leq s < t$, and the Borel sets $\mathcal{A}$.

2.3. Stochastic differential equations

There are interesting processes in robotics, optimal control and economics which could be described as a differential equation with non-deterministic dynamics. Suppose one describes the original processes by the following ordinary differential equation:

$$(ODE) \quad \begin{cases} dX(t) = \mu(X(t))dt & (t > 0) \\ X(0) = x_0, & x_0 \in \mathbb{R}^n \end{cases}$$

where $\mu : \mathbb{R}^n \rightarrow \mathbb{R}^n$ is a smooth vector field and the solution is the trajectory $X(\cdot) : [0, \infty) \rightarrow \mathbb{R}^n$. $X(t)$ is the state of the system at $t \geq 0$.

However, in various applications trajectories of systems that are experimentally measured are modeled by (ODE), and in fact, they do not behave as predicted.

Therefore, it would be reasonable if the (ODE) is modified, somehow, so that the possibility of random effects disturbing the system is included. This is done by writing the differential equation as follows:

$$(1) \quad \begin{cases} \dot{X}(t) = \mu(X(t)) + \sigma(X(t))\xi(t) & (t > 0) \\ X(0) = x_0, & x_0 \in \mathbb{R}^n \end{cases}$$

where $\mu(X(t))$ is a drift coefficient and $\sigma(X(t))$ is a diffusion coefficient of the process. $\sigma : \mathbb{R}^n \rightarrow \mathbb{M}^{n \times m}$ (= space of $n \times m$ matrices) and $\xi(\cdot) := m$ -dimensional white noise.

White noise is the derivative of the Wiener process with respect to time. That is, $\dot{W}(\cdot) = \xi(\cdot)$. Rewriting (1) one obtains:

$$\frac{dX(t)}{dt} = \mu(X(t)) + \sigma(X(t))\frac{dW(t)}{dt}.$$

Multiplying through by dt we have:

$$(SDE) \quad \begin{cases} dX(t) = \mu(X(t))dt + \sigma(X(t))dW(t) \\ X(0) = x_0 \end{cases}$$

The expression obtained is a stochastic differential equation. The integrated version of (SDE) is

$$(2) \quad X(t) = x_0 + \int_0^t \mu(X(s)) ds + \int_0^t \sigma(X(s)) dW, \text{ for every } t \geq 0.$$

A definition of the following stochastic integral is provided before solving the stochastic integral equation above

$$\int_0^t G dW$$

for some wide class of stochastic processes G , so that the RHS of (2) at least makes sense.

Definition 2.3.1. (i) A partition P of an interval $[0, t]$ is a finite collection of points $P := \{0 = t_0 < t_1 < \dots < t_m = t\}$ in $[0, t]$

(ii) Let P be a partition of $[0, t]$ and fix a constant $0 \leq \lambda \leq 1$ so that

$$\tau_k := \lambda t_{k+1} + (1 - \lambda)t_k \quad (k = 0, \dots, m-1).$$

For a constant $0 \leq \lambda \leq 1$ and partition P ,

$$R = R(P, \lambda) := \sum_{k=0}^{m-1} W(\tau_k)(W(t_{k+1}) - W(t_k)) \text{ is defined.}$$

This is an approximation of the Riemann sum for $\int_0^T W dW$.

Lemma 2.3.2. [59]

For a partition P^n of $[0, t]$ and a fixed $0 \leq \lambda \leq 1$, one defines

$$R_n := \sum_{k=0}^{m_n-1} W(\tau_k^n)(W(t_{k+1}^n) - W(t_k^n)).$$

Taking the limit in $\mathcal{L}^2(\Omega)$, $\lim_{n \rightarrow \infty} R_n = \frac{W(t)^2}{2} + (\lambda - \frac{1}{2})t$ is obtained, hence

$$E((R_n - \frac{W(t)^2}{2} - (\lambda - \frac{1}{2})t)^2) \rightarrow 0.$$

In particular, the limit of the Riemann sum approximations depends on the choice of intermediate points $t_k^n \leq \tau_k^n \leq t_{k+1}^n$, where $\tau_k^n = (1 - \lambda)t_k^n + \lambda t_{k+1}^n$.

Choosing $\lambda = 0$ corresponds to the Itô's definition of $\int_0^T W dW$. i.e.

$$\int_0^T W dW = \frac{W^2(T)}{2} - \frac{T}{2} \quad (\text{Itô's integral}).$$

Another definition is due to Stratonovich, when one considers $\lambda = \frac{1}{2}$; so that

$$\int_0^T W dW = \frac{W^2(T)}{2} \quad (\text{Stratonovich integral}).$$

Theorem 2.3.3. (Properties of Itô's integral) [59]

Consider stochastic processes $F, G \in \mathcal{L}^2(0, t)$ and some constants $a, b \in \mathbb{R}$, then one obtains

$$(i) \quad \int_0^t aF + bG dW = a \int_0^t F dW + b \int_0^t G dW,$$

$$(ii) \quad E\left(\int_0^t F dW\right) = 0,$$

$$(iii) \quad E\left(\left(\int_0^t F dW\right)^2\right) = E\left(\int_0^t F^2 ds\right),$$

$$(iv) \quad E\left(\int_0^t F dW\right)\left(\int_0^t G dW\right) = E\left(\int_0^t FG ds\right),$$

$$(v) \quad E \left(\int_0^t G dW \right) \left(\int_0^t G dW \right)' = E \int_0^t G G' ds.$$

Theorem 2.3.4. (Itô's formula) [53]

Let $dX = Fdt + GdW$ be a stochastic differential for $X(\cdot)$, such that $G \in \mathcal{L}^2(0, T)$, $F \in \mathcal{L}^1(0, T)$. For a continuous function $u : \mathbb{R} \times [0, T] \rightarrow \mathbb{R}$, it is assumed that there exist continuous partial derivatives $\frac{\partial u}{\partial t}$, $\frac{\partial u}{\partial x}$ and $\frac{\partial^2 u}{\partial x^2}$. Let

$$Y(t) := u(X(t), t),$$

then the differential of Y is given by

$$\begin{aligned} dY &= \frac{\partial u}{\partial t} dt + \frac{\partial u}{\partial x} dX + \frac{1}{2} \frac{\partial^2 u}{\partial x^2} G^2 dt \\ &= \left(\frac{\partial u}{\partial t} + \frac{\partial u}{\partial x} F + \frac{1}{2} \frac{\partial^2 u}{\partial x^2} G^2 \right) dt + \frac{\partial u}{\partial x} G dW. \end{aligned}$$

Lemma 2.3.5. [2]

$$(i) \quad d(W^2(t)) = dt + 2W(t)dW(t)$$

$$(ii) \quad d(tW(t)) = t dW(t) + W(t) dt$$

Theorem 2.3.6. (Itô's Product rule) [47]

Consider the two differentials for X and Y

$$\begin{cases} dX = G_1 dt + H_1 dW \\ dY = G_2 dt + H_2 dW \end{cases} \quad 0 \leq t \leq T$$

For $G_i \in \mathcal{L}^1(0, T)$, $H_i \in \mathcal{L}^2(0, T)$, ($i = 1, 2$). Then

$$d(XY) = Y dX + X dY + H_1 H_2 dt.$$

Note: $H_1 H_2 dt$ is Itô correction term. Therefore, Itô's integration-by-parts is given by

$$\int_s^r Y dX = X(r)Y(r) - X(s)Y(s) - \int_s^r X dY - \int_s^r H_1 H_2 dt.$$

Notation

- (i) Let X_0 be an n -dimensional random variable that is independent of an m -dimensional Wiener process $W(\cdot)$. Hereafter, for $t \geq 0$, we shall take

$$\mathcal{F}(t) := \mathcal{F}(X_0, W(s) \ (0 \leq s \leq t))$$

the σ -algebra generated by X_0 and the history of the Wiener process up to (and including) time t .

(ii) The components of the following functions (not random variables)

$$\mu : \mathbb{R}^n \times [0, t] \rightarrow \mathbb{R}^n$$

$$\sigma : \mathbb{R}^n \times [0, t] \rightarrow \mathbb{M}^{n \times m}$$

can be displayed as follows:

$$\mu = (\mu_1, \mu_2, \dots, \mu_n), \quad \sigma = \begin{pmatrix} \sigma_{11} & \cdots & \sigma_{1m} \\ \vdots & \ddots & \vdots \\ \sigma_{n1} & \cdots & \sigma_{nm} \end{pmatrix}.$$

So that $|\mu| = \sqrt{\mu_1^2 + \mu_2^2 + \cdots + \mu_n^2}$ and $|\sigma| = \sqrt{\text{tr}(\sigma^t \sigma)}$.

Definition 2.3.7. Consider the following Itô stochastic differential equation

$$(SDE) \quad \begin{cases} dX(t) = \mu(X(t))dt + \sigma(X(t))dW(t) \\ X(0) = x_0 \end{cases}$$

then for $0 < t < T$, $X(\cdot)$ is the solution of the SDE, provided that:

- (i) $X(\cdot)$ is progressively measurable with respect to $\mathcal{F}(\cdot)$,
- (ii) $\mu(X(t)) \in \mathcal{L}_n^1(0, T)$,
- (iii) $\sigma(X(t)) \in \mathcal{L}_{n \times m}^2(0, T)$,
- (iv) $X(t) = x_0 + \int_0^t \mu(X(s), s) ds + \int_0^t \sigma(X(s), s) dW$, almost surely for every $0 \leq t \leq T$.

Definition 2.3.8. A function $f : \mathbb{S} \rightarrow \mathbb{T}$ is called Lipschitz continuous on $A \subset \mathbb{S}$ if there exist some constant $L > 0$ such that

$$\forall x, y \in A : |f(x) - f(y)| \leq L|x - y|$$

and it is called locally Lipschitz continuous if for each $z \in \mathbb{S}$ there exists a neighbourhood U of z such that f is Lipschitz continuous on U .

Lemma 2.3.9. (Gronwall inequalities) [27]

Consider two non-negative, continuous functions $\phi(t)$ and $f(t)$ defined for every $t \in [0, T]$ and let C be a constant. If

$$\phi(t) \leq C + \int_0^t f(s)\phi(s) ds, \quad \text{for every } 0 \leq t \leq T,$$

then

$$\phi(t) \leq Ce^{\int_0^t f(s) ds}, \quad \text{for every } 0 \leq t \leq T.$$

Gronwall inequalities are useful in proving the existence and uniqueness theorem for stochastic differential equations.

Theorem 2.3.10. (*Existence and Uniqueness theorem*) [59]

Let $\mu : \mathbb{R}^n \times [0, T] \rightarrow \mathbb{R}^n$ and $\sigma : \mathbb{R}^n \times [0, T] \rightarrow \mathbb{M}^{n \times m}$ be continuous functions satisfying the conditions below:

- (a) $|\mu(x, t) - \mu(y, t)| \leq K|x - y|$, $|\sigma(x, t) - \sigma(y, t)| \leq K|x - y|$ for all $0 \leq t \leq T$, $x, y \in \mathbb{R}^n$,
- (b) $|\mu(x, t)| \leq K(1 + |x|)$, $|\sigma(x, t)| \leq K(1 + |x|)$ for every $0 \leq t \leq T$, $x \in \mathbb{R}^n$ and a constant K ,
- (c) Let X_0 be an n -dimensional real-valued random variable that is independent of $\mathcal{F}^+(t)$ and $E(|X_0|^2) < \infty$,

then for $0 \leq t \leq T$, there is a unique solution $X \in \mathcal{L}_n^2(0, T)$ of the stochastic differential equation:

$$\begin{cases} dX = \mu(X, t)dt + \sigma(X, t)dW \\ X(0) = X_0. \end{cases}$$

2.4. Fractional brownian motion

A stochastic process $X(t)$ defined on a probability space $(\Omega, \mathcal{F}, P)$ is said to be a Gaussian process if for all $t_1, t_2, \dots, t_n \in \mathbb{R}_{\geq 0}$, the random vector $(X(t_1), X(t_2), \dots, X(t_n))$ follows a Gaussian distribution. The distribution of a Gaussian process $(X(t))_{t \geq 0}$ is uniquely determined by its mean function given by

$$m(t) = E(X(t)),$$

and its covariance function given by

$$R(s, t) = E((X(t) - m(t))(X(s) - m(s))).$$

Definition 2.4.1. A fractional Brownian motion (FBM) with Hurst parameter $H \in (0, 1)$ is a continuous Gaussian process $B_H(t), t \geq 0$ that has a mean function 0 and a covariance function given by

$$R(s, t) = E(B_H(s)B_H(t)) = \frac{1}{2}(|s|^{2H} - |s - t|^{2H} + |t|^{2H}).$$

It is observed that for $H = \frac{1}{2}$, the covariance function becomes $R(s, t) = \min(s, t)$.

Consequently, a fractional Brownian motion with Hurst parameter $H = \frac{1}{2}$ is a Wiener process.

Let $B_H(t), t \geq 0$ be a fractional Brownian motion with Hurst parameter H , then one obtains

$$E(B_H(s) - B_H(t)) = 0,$$

and

$$E((B_H(s) - B_H(t))^2) = E(B_H^2(s)) - 2E(B_H(t)B_H(s)) + E(B_H^2(t)) = |t - s|^{2H}.$$

A characteristic that mostly distinguish fractional brownian motion from brownian motion is the fact that fractional brownian motion is not a semimartingale for $1/2 < H < 1$ (Lin [45]). There

is therefore a need to carefully define the stochastic integral from first principles with respect to fractional Brownian motion. See for example, Heyde and Dai [17], Lin [45] and Norris and Gripenberg [30] for their contributions in relation to this study. It is essential that a corresponding Itô's formula be derived for stochastic differential equations driven by fractional brownian motion.

2.4.1 Stochastic differential equations driven by fractional brownian motion

In order to understand the theory of stochastic differential equations with respect to fractional brownian motion, it is necessary to explain Riemann-Stieltjes Integral since it is an important notion to understanding stochastic integration. Let us first recall the basic Riemann Integral.

Definition 2.4.2. Let $f : \mathbb{R} \rightarrow \mathbb{R}$ be a continuous function, then the Riemann integral over $[a, b] \subset \mathbb{R}$ is defined by

$$\int_0^t f(t)dt = \lim_{\|\Delta_m\| \rightarrow 0} \sum_{j=1}^m f(\tau_j)(t_j - t_{j-1}),$$

where $\Delta_m = \{t_0, t_1, t_2, \dots, t_m\}$ is a partition of $[a, b]$ such that $a = t_0 < t_1 < \dots < t_{n-1} < t_m = b$, $\|\Delta_m\| = \max_{1 \leq j \leq m} (t_j - t_{j-1})$ and $\tau_j \in [t_{j-1}, t_j]$ is an evaluation point.

Definition 2.4.3. Let $f : [a, b] \rightarrow \mathbb{R}$ be continuous, then the p -variation of a function f is given as

$$\sum_{k=1}^n (f(t_k^n) - f(t_{k-1}^n))^p,$$

where $a = t_0^n < t_1^n < \dots < t_n^n = b$ is a partition of the interval which tends to 0 as $n \rightarrow \infty$.

Definition 2.4.4. A continuous function $f : [a, b] \rightarrow \mathbb{R}$ is said to be a function of bounded variation if

$$\sup_{\pi \in \mathcal{P}} \sum_{i=1}^n |f(t_i) - f(t_{i-1})| < \infty, \quad \forall t > 0,$$

where $\mathcal{P} = \{\pi = \{t_0, t_1, \dots, t_n\} | \pi \text{ is a partition of } [a, b]\}$.

Definition 2.4.5. Let $f : [a, b] \rightarrow \mathbb{R}$ be a function of bounded variation and $g : [a, b] \rightarrow \mathbb{R}$ be a continuous function. Then Riemann-Stieltjes integral is defined as follows:

$$\int_a^b g(t)df(t) = \lim_{\|\Delta_m\| \rightarrow 0} \sum_{j=1}^m g(\tau_j)(f(t_j) - f(t_{j-1})),$$

where $\Delta_m = \{t_0, t_1, \dots, t_m\}$ is a partition of $[a, b]$ such that $a = t_0 < t_1 < \dots < t_m = b$, $\|\Delta_m\| = \max_{1 \leq j \leq m} (t_j - t_{j-1})$ and τ_j is an evaluation point in the interval $[t_{j-1}, t_j]$.

It should be noted that if $f(t) = t$, then the Riemann-Stieltjes integral is just the Riemann integral.

Proposition 2.4.6. *Let g be a continuous function and $f \in \mathcal{C}^1$, then*

$$\int_a^b g(t)df(t) = \int_a^b g(t)f'(t)dt$$

and if f and g are of bounded variation, then

$$\int_a^b g(t)df(t) = g(b)f(b) - g(a)f(a) - \int_a^b f(t)dg(t).$$

Many researchers have suggested various methods of defining a stochastic integral with respect to fractional brownian motion (Heyde and Dai [17], Lin [45] and Norris and Gripenberg [30]). In this study the definition given by Heyde and Dai is considered. $(\Omega, \mathcal{F}, P)$ is taken to be a complete probability space associated with the usual normalised fractional brownian motion $B_H(t)$ on an interval $[0, T]$, for $1/2 < H < 1$.

Definition 2.4.7. *Consider two stochastic processes $\mu(t, \omega)$ and $\sigma(t, \omega) : [0, T] \times \Omega \rightarrow \mathbb{R}$. Then a stochastic process $X(t)$ for $t \in [0, T]$ is said to have a stochastic differential equation with respect to fractional Brownian motion $B_H(t)$*

$$dX(t) = \mu(t)dt + \sigma(t)dB_H(t),$$

if for every $(t, \omega) \in [0, T] \times \Omega$, then the equation below holds

$$X(t, \omega) = X_0(\omega) + \int_0^t \mu(s, \omega)ds + \int_0^t \sigma(s, \omega)dB_H(s)$$

for a random variable X_0 . The stochastic integral $\int_0^t \mu(s, \omega)ds$ for every $\omega \in \Omega$ is a usual Riemann-Stieltjes integral while $\int_0^t \sigma(s, \omega)dB_H(s)$ is defined as that given by Heyde and Dai [17].

2.4.2 Itô's formula with respect to fractional brownian motion

Consider stochastic differential equations driven by Brownian motion

$$dX(t) = \mu(t, X(t))dt + \sigma(t, X(t))dB(t),$$

Itô's formula is a powerful tool used when dealing with this kind of calculus. When it comes to stochastic differential equations driven by fractional Brownian motion

$$dX(t) = b(t, X(t))dt + B(t, X(t))dB_H(t),$$

a version of Itô's formula is needed that can play a similar role when dealing with this kind of equation. The following theorem gives the Itô's formula with respect to the fractional brownian motion.

Theorem 2.4.8. (*Itô's formula with respect to fractional Brownian motion*) [18]

Let a complete probability space $(\Omega, \mathcal{F}, P)$ be given and $B_H(t)$ be a fractional brownian motion with $1/2 < H < 1, t \in [0, T]$ and $B_H(0) = 0$ a.e. (therefore, $E[B_H(t)] \equiv 0$ for every $t \in [0, T]$). Let $\mu(t, \omega), \sigma(t, \omega)$ and $X(\tau, \omega)$ be stochastic processes such that for any $[t_0, t_1] \subseteq [0, T]$,

1. $\mu(t, \omega)$ is Riemann-Stieltjes integrable on $[t_0, t_1]$ for every $\omega \in \Omega$;
2. $\int_0^{t_1} \sigma(t) dB_H(t)$ exists as described in Heyde and Dai [17];
3. One of the following holds
for every $0 \leq s \leq r \leq t_2 \leq t_3 \leq t_4 \leq T, \{\mu(t) : 0 \leq t \leq T\}$ and $\{B_H(t) : 0 \leq t \leq T\}$ are such that

$$\begin{aligned} & E\{((\sigma(t_3) - \sigma(s))(\sigma(r) - \sigma(s))(B_H(t_4) - B_H(t_3)))(B_H(t_3) - B_H(t_2)))\} \\ &= E\{(\sigma(t_3) - \sigma(s))(\sigma(r) - \sigma(s))\} E\{(B_H(t_4) - B_H(t_3))(B_H(t_3) - B_H(t_2))\}, \end{aligned}$$

or,

the second derivative $d^2\sigma(t)/dt^2$ exists, and for every $0 \leq s \leq t_1 < t_2, t_3 \leq t_4 \leq T, \{\sigma'(t) = d\sigma(t)/dt : s < t \leq \max\{r, t_3\}\}$ and $(B_H(r), B_H(t_2), B_H(t_3), B_H(t_4))$ are such that for any random variables ξ and η such that ξ and η are measurable with respect to the sigma-algebra $\sigma\{\sigma'(t) : s \leq t \leq \max\{r, t_3\}\}$ and $E|\xi|^4 < \infty, E|\eta|^4 < \infty$, then one obtains the following

$$\begin{aligned} & E\{((\sigma'(s)(r-s) + \xi)(\sigma'(s)(t_3-s) + \eta)(B_H(t_2) - B_H(r))(B_H(t_4) - B_H(t_3)))\} \\ &= E\{(\sigma'(s)(r-s) + \xi)(\sigma'(s)(t_3-s) + \eta)\} E\{(B_H(t_2) - B_H(r))(B_H(t_4) - B_H(t_3))\}, \end{aligned}$$

and

$$\sup_{0 \leq t \leq T} E\left|\frac{dB}{dt}(t, \omega)\right|^4 < \infty, \quad \sup_{0 \leq t \leq T} E\left|\frac{d^2B}{dt^2}(t, \omega)\right|^4 < \infty;$$

4.

$$X(t) - X(t_0) = \int_{t_0}^t \mu(\tau, \omega) d\tau + \int_{t_0}^t \sigma(\tau, \omega) dB_H(\tau),$$

where for every $\omega \in \Omega$, the first integral is just the usual Riemann-Stieltjes integral, while the second one is an Itô integral defined in Dai and Heyde [17]. Consider a two variable function $U(t, x) : [0, T] \times \mathbb{R} \rightarrow \mathbb{R}$ that has uniformly continuous partial derivatives $\partial U/\partial t, \partial U/\partial x$ and $\partial^2 U/\partial x^2$. Assuming further that

$$\sup_{0 \leq t \leq T} E|U(t, X(t))|^2 < \infty, \quad (2.1)$$

$$\sup_{0 \leq t \leq T} E\left|\frac{\partial U}{\partial t}(t, X(t))\right|^2 < \infty, \quad (2.2)$$

$$\sup_{0 \leq t \leq T} E\left|\frac{\partial U}{\partial x}(t, X(t))\right|^2 < \infty, \quad (2.3)$$

$$\sup_{0 \leq t \leq T} E\left|\frac{\partial^2 U}{\partial x^2}(t, X(t) + O_{L_2}(1))\right|^2 < \infty, \quad (2.4)$$

$$\sup_{0 \leq t \leq T} E|\mu(t)|^2 < \infty, \quad (2.5)$$

$$\sup_{0 \leq t \leq T} E|\sigma(t)|^2 < \infty, \quad (2.6)$$

$$E|B(t) - B(s)| < C|t - s|^\beta, \beta \geq 0,$$

where $O_{L_2}(1)$ means a term such that $E|O_{L_2}(1)|^2 < \infty$. Let $Y(t) = U(t, X(t))$. If for any $0 \leq t \leq T$,

$$\int_0^t \sigma(\tau, \omega) \frac{\partial U}{\partial x}(\tau, X(\tau)) dB_H(\tau)$$

exists in the sense described in Dai and Heyde [17], then the result that follows holds

$$\begin{aligned} Y_t = Y_0 &+ \int_0^t \left\{ \frac{\partial U}{\partial t}(\tau, X(\tau)) + b(\tau, \omega) \frac{\partial U}{\partial x}(\tau, X(\tau)) \right\} d\tau \\ &+ \int_0^t B(\tau, \omega) \frac{\partial U}{\partial x}(\tau, X(\tau)) dB_H(\tau) \end{aligned}$$

or equivalently,

$$dY_t = \left\{ \frac{\partial U}{\partial t}(t, X(t)) + \mu(t, \omega) \frac{\partial U}{\partial x}(t, X(t)) \right\} dt + \sigma(t, \omega) \frac{\partial U}{\partial x}(t, X(t)) dB_H(t). \quad (2.7)$$

Remarks on Theorem 2.4.8.

- (i) Contrary to the usual Itô formula (i.e. with respect to Brownian motion), there is no term

$$\frac{1}{2} \sigma^2(\tau, \omega) \frac{\partial^2 U}{\partial x^2}(t, X(t))$$

in (2.7), since $E(B_H(t + \delta) - B_H(t))^2 = |\delta|^{2H}$, where $2H > 1$.

- (ii) The requirements on $\mu(t), \sigma(t), X(t)$ and $U(t, X(t))$ such as Conditions 1, 2 and 4 in the theorem above, and the moment conditions (2.1) – (2.6) are standard.
- (iii) The two conditions in 3 are critical for Itô's formula to be true in the case of fractional Brownian motion. Many stochastic processes can be chosen as $\sigma(t)$. For example,

$$\sigma(t) = A_1(t) + A_2,$$

where A_1 and A_2 are two random variables with $E[A_1^2] < \infty$ and A_1 is independent of $\{B_H(\tau)\}$.

Lemma 2.4.9. Assuming that stochastic processes $\mu(\tau)$ and $\sigma(\tau)$ satisfy the conditions of Theorem 2.4.3, then for every $t, s \in [0, T]$ such that $|t - s| \rightarrow 0$, one obtains

$$\int_s^t \mu(\tau) d\tau + \int_s^t \sigma(\tau) dB_H(\tau) = \mu(s)(t - s) + \sigma(s)(B_H(t) - B_H(s)) + O_{L_2}(|t - s|),$$

where $O_{L_2}(|t - s|)$ means a term such that

$$(E|O_{L_2}(|t - s|)|^2)^{1/2} = O(|t - s|).$$

Proofs to both Theorem 2.4.3 and Lemma 2.4.4 are given in [18].

Definition 2.4.10. Let $g(t) : \mathbb{R} \rightarrow \mathbb{R}$ be a bounded Borel function. Defining

$$\int_0^t g(B_H(s)) dB_H(s) = \int_0^{B_H(t)} g(s) ds$$

$$\int_0^{B_H(t)} g(s) ds = \lim_{|\beta^n| \rightarrow 0} \sum_i g(t_{i-1}^n) (B_H(t_i^n) - B_H(t_{i-1}^n))$$

where $\beta^n : t_0 = 0 < t_1^n < t_2^n < \dots < t_q^n = t$ is a sequence of partitions on $[0, t]$ is given as in Dai and Heyde [17].

Theorem 2.4.11. [45]

Let $f(s, x)$ and $g(s)$ be Borel functions such that

- (i) $g : [0, \infty) \rightarrow \mathbb{R}$ is bounded,
- (ii) $|f(s, x)| \leq K(1 + |x|)$,
- (iii) $|f(s, x) - f(s, y)| \leq K|x - y|$

for a positive constant K . Then, the stochastic differential equation

$$\begin{cases} dX(t) = f(t, X(t))dt + g(t)dB_H(t) \\ X_0 = B(\omega) \end{cases}$$

has a unique solution with continuous paths, where $B(\omega) \in \mathcal{L}^2(\Omega)$.

Theorem 2.4.12. [18]

The stochastic differential equation

$$\begin{cases} dS(t) = aS(t)dt + bS(t)dB_H(t) \\ S(t_0) = B(\omega) \end{cases}$$

has a unique solution given by

$$S_t = B \exp\{a(t - t_0) + b(B_H(t) - B_H(t_0))\},$$

where a and b are constants and $B(\omega)$ is a positive random variable such that $E|B(\omega)|^2 < \infty$.

The following proposition gives some properties of the fractional Itô integral.

Proposition 2.4.13. [4]

- (i) For all $a < b \in \mathbb{R}$, one obtains $B_H(b) - B_H(a) = \int_a^b dB_H(t)$.



(ii) Let $X : M \rightarrow (\mathcal{L}^2)$ be a fractional Itô integrable. Then,

$$\int_M X(t)dB_H(t) = \int_{\mathbb{R}} 1_M(t)X(t)dB_H(t),$$

where 1_M denotes the indicator function of M and $(\mathcal{L}^2) := \mathcal{L}^2(\Omega, \mathcal{G}, P)$ such that $\mathcal{G}$ is the σ -algebra generated by $\{I(f); f \in \mathcal{L}^2(\mathbb{R})\}$.

(iii) Let $X : M \rightarrow (\mathcal{L}^2)$ be a fractional Itô integrable. Then,

$$E\left[\int_M X(t)dB_H(t)\right] = 0.$$

3

Mathematical analysis of a stochastic tuberculosis model

3.1. Introduction

Tuberculosis (TB) in humans, is an airborne infective bacterial sickness caused by *Mycobacterium tuberculosis* (also known as *M. tuberculosis* or MTB for short). It usually affects the lungs. *M. tuberculosis*, together with other several similar mycobacterial types (*M. canetti*, *M. pinnipedii*, *M. caprae*, *M. africanum*, *M. microti*, *M. mungi* and *M. bovis*), make up what is referred to as *Mycobacterium TB complex* (WHO report [28]). It has been discovered that not all of these species cause TB in humans, but most of them do. In main regions, most cases of the tuberculosis are known to be caused by the *M. tuberculosis*. Tubercle bacilli is one of the mycobacterium organisms that causes TB in humans. The MTB (*Mycobacterium tuberculosis*) are carried by air particles, known as droplet nuclei, which have a diameter of about 1 to 5 microns. The infective droplet nuclei are produced when a person with laryngeal or pulmonary TB coughs, sneezes, shouts, laughs, spits, sings or talks. Depending on conditions of the environment, the tubercle bacilli drops are carried by such droplets and can be suspended for many hours in the air. MTB is remitted through air, not through contact. The spread of bacteria occurs when an individual inhales the droplet nuclei which harboured MTB, and the droplet nuclei passes through the nasal passage or the mouth, then goes through the respiratory tract, progresses to the bronchi and finally reaches the alveoli in the lungs. Transmission is heightened by regular and long exposure to an infective individual, usually at home, the workplace, school or other public places. Tubercle bacilli are consumed by the alveolar macrophage (or dust cell); most of the tubercle bacilli are either inhibited or destroyed. A small amount of tubercle bacilli, which are inhibited, multiplies within the cells of the alveoli and are then released after the death of the macrophage. If alive, small amounts of the tubercle bacilli enter the bloodstream and spread everywhere, including areas where the disease is most likely to mature (bones, apex of the lungs, kidneys, lymph node joints, the brain, urogenital and digestive tracts, nervous systems and the skin). Within a period of 2 - 8 weeks, some white blood cells known

as macrophages, surround and ingest the tubercle bacilli and form a granuloma which is a barrier shell that keeps the bacilli under control and contained. If the tubercle bacilli is not kept under control by the immune system, it will multiply rapidly thus causing TB. The process can happen in the lungs, brain, kidneys or bones and other parts of the body.

The universal burden of TB has multiplied over the years regardless of the prevailing control measures currently being implemented. These measures include vaccines such as Bacille Calmette-Guerin (BCG) and the World Health Organisation's direct observation therapy strategy (DOTS) that concentrates on finding cases and giving short course therapy (Colijn *et al.* [16]). According to WHO, there were about 1.8 million deaths resulting from 14 million infections in 2007, mostly in developing countries (WHO [74]). In 2003, there were about 8.8 million new TB infections in Africa resulting in 1.7 million deaths (WHO [73]). Out of 9 million cases of tuberculosis that developed in 2013, about 56% came from the Western Pacific Regions and South-East Asia. An additional 25% was from Africa and the continent had the highest deaths and infection rates relative to population size. China and India alone accounted for about 11% and 24% of total cases respectively (WHO [28]).

Waalder *et al.* [70] are the pioneers of mathematical modeling for the transmission dynamics of TB. Their model comprises of a system of linear difference equations. They divided the population in three different epidemiological classes: non-infected (susceptible); latent (infected but not infectious); and infectious (infected cases). They expressed the rate of infection as a function of the number of individuals that are infectious. Their study provided many researchers with the basic starting point in Mathematical modeling of the transmission dynamics of TB in communities. Brogger and Ferebee [13] came up with models that improved on Waalder's model. Brogger did not only introduce heterogeneity (age) but also changed the method for calculating rates of infection. The infection rate in Brogger's model was a mixture of linear and nonlinear infection terms. Using Waalder and Brogger's models as a template, ReVelle *et al.* [62] came up with the first nonlinear system of ordinary differential equations for modeling transmission dynamics of Tuberculosis. They used differential equations to formulate the connection between the prevalence of TB and rate of infection in his model. When modeling the rate of infection, they did not use the regular mass action law given by the bilinear function of Kermack and McKendrick [37]. ReVelle *et al.* were the first (in the context of dynamics of transmission of TB) to critically explain why the rate of infection depends linearly on prevalence using the probabilistic approach that is common today.

Adetunde [1] studied a Susceptible-Latent-Infected-Recovered (SLIR) model for the dynamical behaviour of tuberculosis in the upper east region of the northern part of Ghana. His model exhibited two equilibria (the endemic equilibrium and disease free equilibrium). Egbetade *et al.* [23] suggested a mathematical model for treatment of TB epidemics in a population. They came up with a proof for the existence and uniqueness of a solution theorem for the model established. Additionally, they demonstrated that if the basic reproductive ratio is less than one, then the infection is cleared from the population. Other studies in this field have been done by Kalu and Inyama [35], Ibrahim *et al.* [31] and Oname and Inyama [60].

These models are usually deterministic, and assume that all input variables are deterministic functions of time, ignoring completely the randomness of these variables. Since biological processes involved in the dynamics of TB are stochastic rather than deterministic, neglecting their built-in randomness may lead to misleading and erroneous results.

In this study, these limitations were overcome by extending the study of S. Bowong, J.J. Tewa and J.C. Kamgang [9] (on Stability analysis on the transmission dynamics of TB) by converting their deterministic model to a system of stochastic differential equations. An analysis of a stochastic model representing transmission dynamics of tuberculosis was done. Stochasticity was introduced to the model through perturbation of parameters which is an accepted method in modeling stochastic dynamics of a population. It was proven that the system or model under review has solutions that are non-negative, which is fundamental in the dynamics of population modeling. In addition, a detailed analysis was done to observe the asymptotic behaviour of the model. The mean reverting process was also used to approximate one of the variables and found its variance and mean.

3.2. Deterministic model

A basic model of tuberculosis infection that incorporates both fast and slow progressions, efficient chemoprophylaxis (given only to individuals who are in a latent stage of infection) and therapeutic treatments (given to the infectious individuals) was considered.

S. Bowong, J.J. Tewa and J.C. Kamgang [9] used nonlinear models to study the transmission dynamics of tuberculosis models. Their model was based on the TB transmission work frame proposed in [56, 57]. The population was divided into three groups depending on an individual's epidemiological state; they are categorised as susceptible (non-infected), latently infected (infected non-cases) or infectious (infected cases). The sizes of these groups are represented by x, y and z respectively. There is a constant rate of recruitment into the susceptible class only and it is denoted as λ . The natural death rate (caused by disease unrelated reasons) is proportionate to the size of the population, and it occurs at a constant rate $\mu > 0$. The mortality rate induced by tuberculosis only affects those in class z and has a constant rate $\delta \geq \mu$. Transmission of M. tuberculosis happens when there is adequate contact or interaction between an infectious and susceptible individual. Since individuals who are latently infected are not infectious, they are not able to transmit bacteria. A susceptible individual, has on average, βz contacts in every unit time that would remit the disease. Therefore, the rate of infection for susceptibles is βxz . A fraction α of individuals recently infected is assumed to directly progress to the infective class, at the same time the rest have a suppressed infection and join the latent class y . When an individual is infected with M. tuberculosis and the infection is dormant, he/she remains in latency stage throughout his/her life except when the infection is reactivated. To take treatment into account, let ηy be the number of individuals latently-infected and receive efficient chemoprevention or chemoprophylaxis, and γ be the rate of effective per capita therapy. Here, it is assumed that chemoprevention reduces the reactivation rate of latently infected individuals y and the introduction of therapeutics will move

an individual at once from a state of being infectious z to a state of latency y . The time it takes for individuals in a latency state who are not on chemoprophylaxis to be infective is assumed to be exponentially distributed, with $\frac{1}{\kappa}$ as the average waiting time. Thus an individual leaves the group y at the rate $\kappa(1 - \eta)y$, while infectious individuals enter the latent class y at a constant rate γz respectively. The following three-dimensional model describes the transmission dynamics of TB, including therapeutic treatment:

$$\begin{aligned}\frac{dx(t)}{dt} &= \lambda - \mu x(t) - \beta x(t)z(t), \\ \frac{dy(t)}{dt} &= \beta(1 - \alpha)x(t)z(t) + \gamma z(t) - [\mu + \kappa(1 - \eta)]y(t), \\ \frac{dz(t)}{dt} &= \kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) - (\gamma + \mu + \delta)z(t)\end{aligned}\tag{3.1.1}$$

with appropriate initial conditions. The above mathematical model explains the transmission dynamics of tuberculosis under treatment effects. We give the description of the parameters and variables used in the above model below:

- $x(t)$ number of susceptible individuals at time t ;
- $y(t)$ number of individuals at time t in the latent class;
- $z(t)$ number of infectious individuals at time t ;
- λ rate of recruitment into the susceptible class;
- μ natural mortality rate (not caused by the disease);
- δ mortality rate caused by the disease;
- α proportion of newly-infected individuals;
- β transmission coefficient of bacteria;
- η proportion of individuals in the latent class who are on effective chemoprophylaxis;
- γ rate of effective per capita therapy; and
- κ time before a latent infected person who is not receiving chemoprophylaxis become infective.

It is assumed that all the parameters $\lambda, \mu, \delta, \kappa, \eta$ and γ are non-negative and any other parameters are positive with $\alpha \in [0, 1]$. Since the model in (3.1.1) deals with human beings, non-negativity of the variables is further assumed at time $t = 0$. Moreover, when one adds all the equations in model (3.1.1) one obtains the total dynamics $T(t) = x(t) + y(t) + z(t)$ of the system given by

$$\dot{T}(t) = \lambda - \mu T(t) - \delta z(t).$$

Definition 3.2.1. *Given a dynamical system $\dot{x} = f(x)$ and a trajectory $x(t, x_0)$ where x_0 is an initial point. Let $\mathcal{D} := \{x \in \mathbb{R}^n | \phi(x) = 0\}$ where ϕ is a real valued function. The set $\mathcal{D}$ is said to be positively invariant if $x_0 \in \mathcal{D}$ implies that $x(t, x_0) \in \mathcal{D} \forall t \geq 0$.*

Intuitively, this means that once a trajectory of the system enters $\mathcal{D}$, it will never leave it again.

Definition 3.2.2. Given a vector space X over the field $\mathbb{F}$ of real or complex numbers, a set S is called *absorbing* if for all $x \in X$ there exists a real number r such that

$$\forall \alpha \in \mathbb{F} : |\alpha| \geq r \Rightarrow x \in \alpha S \text{ with } \alpha S := \{\alpha s \mid s \in S\}.$$

It should be noted that when $T(t) = \frac{\lambda}{\mu}$ as $t \rightarrow \infty$, there is no disease in the population and $\frac{\lambda}{\mu}$ is the upper bound of $T(t)$ provided that $T(0) \leq \frac{\lambda}{\mu}$. In addition, if $T(0) > \frac{\lambda}{\mu}$, then $T(t)$ will decrease to this level. Therefore, the following possible region is obtained:

$$\mathcal{D} = \{(x(t), y(t), z(t)) \in \mathbb{R}_+^3, 0 \leq T(t) \leq \frac{\lambda}{\mu} + \epsilon\}$$

which is a compact positively invariant set for $\epsilon > 0$ and forward absorbing set for every $\epsilon > 0$. Also, every solution in $\mathbb{R}_+^3$ approaches $\mathcal{D}$ so that the analysis can be restricted to this region. For the system, the conventional continuation as well as the existence and uniqueness of solutions will still be satisfied in the region $\mathcal{D}$.

3.2.1 Basic reproduction ratio

Most models in epidemiology have a threshold value used to decide if the disease will become endemic or be eradicated from the population. The basic reproductive ratio, is known as the average number of cases generated by an infectious person during his/her infective period in an uninfected population, and is denoted by R_0 . If $R_0 < 1$, then each infectious person infects less than one new individual on average and the epidemic is expected to be eliminated. On the contrary, if $R_0 > 1$, then the number of new infections caused by every infectious person is greater than one and the epidemic infests the entire population. Hence, the basic reproductive ratio R_0 is usually considered as the threshold number established when a disease will invade and persist in a new host population. S. Bowong, J.J. Tewa and J.C. Kamgang determined R_0 for this model to be

$$R_0 = \frac{\beta x_0 [\mu \alpha + \kappa(1 - \eta)]}{(\mu + \delta) [\mu + \kappa(1 - \eta)] + \mu \gamma}$$

The disease free equilibrium is obtained for the model when one sets the right hand side of (3.1.1) to zero and solving explicitly for x, y and z .

When $R_0 < 1$ one obtains a locally asymptotically stable disease free equilibrium, and when $R_0 > 1$, it is unstable. It is expected that the number of infectious individuals will die out and the number of non-infectious individuals will approach $\frac{\lambda}{\mu}$ whenever $R_0 < 1$, and when $R_0 > 1$, one expects the number of individuals not infected to approach a unique endemic equilibrium.

3.2.2 Global stability of the disease-free equilibrium

The global properties of a disease-free equilibrium will now be studied. For the disease-free equilibrium, the property for global stability is given in the following theorem:

Theorem 3.2.3. *The disease-free equilibrium $(\frac{\lambda}{\mu}, 0, 0)$ for model (3.1.1) is globally asymptotically stable when $R_0 \leq 1$ in the non-negative octant $\mathbb{R}_+^3$.*

PROOF.

Let the Lyapunov-LaSalle function be defined as follows:

$$Y(y, z) = \kappa(1 - \eta)y(t) + [\mu + \kappa(1 - \eta)]z(t).$$

The time derivative along with the trajectories of model (3.1.1) will satisfy

$$\begin{aligned} \dot{Y}(y, z) &= \kappa(1 - \eta)\dot{y}(t) + [\mu + \kappa(1 - \eta)]\dot{z}(t), \\ &= \kappa(1 - \eta)[(1 - \alpha)\beta x(t)z(t) + \gamma z(t) - [\kappa(1 - \eta) + \mu]y(t)] \\ &\quad + [\kappa(1 - \eta) + \mu][\kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) - (\gamma + \mu + \delta)z(t)], \\ &= [\beta x(t)[\alpha\mu + \kappa(1 - \eta)] - \gamma\mu - [\kappa(1 - \eta) + \mu](\mu + \delta)]z(t). \end{aligned}$$

Since $x \leq x_0$,

$$\begin{aligned} \dot{Y}(y, z) &\leq [\beta x_0(t)[\alpha\mu + \kappa(1 - \eta)] - \gamma\mu - [\mu + \kappa(1 - \eta)](\mu + \delta)]z(t) \\ &\leq \frac{1}{[\mu + \kappa(1 - \eta)](\delta + \mu) + \gamma\mu} \left\{ \frac{\beta x_0[\alpha\mu + \kappa(1 - \eta)]}{[\mu + \kappa(1 - \eta)](\delta + \mu) + \gamma\mu} - 1 \right\} z(t) \\ &\leq \frac{(R_0 - 1)z(t)}{[\mu + \kappa(1 - \eta)](\mu + \delta) + \gamma\mu} \text{ is obtained.} \end{aligned}$$

Thus, $R_0 \leq 1$ implies that $\dot{Y}(y, z) \leq 0$. Furthermore, $\dot{Y}(y, z) = 0$ provided that $R_0 = 0$ or $z(t) = 0$. Hence in $\{(x(t), y(t), z(t)) \in \mathbb{R}^3 \geq 0, \dot{Y}(y, z) = 0\}$, the biggest compact invariant set is the singleton $\{(x_0, 0, 0)\}$. Consequently, all the trajectories starting in $\mathcal{D}$ approach $(x_0, 0, 0)$ when $t \rightarrow \infty$ (LaSalle Lyapunov theorem [41]). Therefore, global asymptotic stability is obtained on the non-negative octant $\mathbb{R}_+^3$ for $R_0 \leq 1$ since $\mathcal{D}$ is an absorbing set. The reason for considering a compact positively invariant set is to set up the stability of $(x_0, 0, 0)$ because $Y(y, z)$ is not positive definite. In most cases, the LaSalle's invariance principle will only prove the attractiveness of equilibrium. Since $\mathcal{D}$ allows to conclude for stability [41, 42, 43], this verifiable truth is frequently neglected in the literature that uses LaSalle's invariance principle. Hence, the proof is complete. ■

3.2.3 Existence and uniqueness of endemic equilibrium

In this section, the uniqueness and existence of the endemic equilibrium for the deterministic model (3.1.1) is examined. The basic reproduction ratio R_0 is used to achieve this objective. S. Bowong, J.J. Tewa and J.C. Kamgang [9] analysed the deterministic model in their study (Stability analysis of the transmission dynamics of tuberculosis model). They demonstrated that a unique disease-free equilibrium exists if $R_0 \leq 1$ and whenever $R_0 > 1$, there exist a unique endemic equilibrium

$X^* = (x^*, y^*, z^*)$ obtained by equating all equations to zero in the model (3.1.1), and it yields

$$\begin{cases} \lambda - \beta x^* z^* - \mu x^* = 0 \\ \beta(1 - \alpha)x^* z^* - [\kappa(1 - \eta) + \mu]y^* + \gamma z^* = 0 \\ \beta \alpha x^* z^* + \kappa(1 - \eta)y^* - (\mu + \delta + \gamma)z^* = 0 \end{cases} \quad (3.1)$$

Using the first and second equations in (3.1), one obtains

$$x^* = \frac{\lambda}{\mu + \beta z^*}, \quad y^* = \frac{z^*}{\mu + \kappa(1 - \eta)} \left[\frac{\beta(1 - \alpha)\lambda}{\beta z^* + \mu} + \gamma \right], \quad (3.2)$$

then substituting (3.2) into the third equation of (3.1) yields

$$z^* \left(-\frac{\beta}{\mu} z^* + R_0 - 1 \right) = 0. \quad (3.3)$$

There are two solutions for equation (3.3): $z^* = 0$ which exists at the disease-free equilibrium or

$$z^* = \frac{\mu(R_0 - 1)}{\beta}.$$

Thus, if $R_0 > 1$, then $z^* > 0$ and one may easily prove that the endemic equilibrium is defined by

$$\begin{aligned} x^* &= \frac{\lambda}{\mu R_0}, \\ y^* &= \frac{\mu(R_0 - 1)}{\beta[\mu + \kappa(1 - \eta)]} \left[\frac{\beta(1 - \alpha)\lambda}{\mu R_0} + \gamma \right], \\ z^* &= \frac{\mu(1 - R_0)}{\beta} \end{aligned} \quad (3.1.2)$$

From this, one obtains the following results:

Lemma 3.2.4. *If $R_0 > 1$, then (3.1.1) has a unique endemic equilibrium $X^* = (x^*, y^*, z^*)$. Where x^*, y^* , and z^* are as defined in (3.1.2).*

3.2.4 Global stability of endemic equilibrium

Here, the global behaviour of an endemic equilibrium X^* is examined for the model (3.1.1). The following result is obtained:

Theorem 3.2.5. *The unique endemic equilibrium X^* is globally asymptotically stable on $\mathcal{D} \setminus \{y(t) = z(t) = 0\}$ whenever $R_0 > 1$.*

PROOF.

Let's define the Lyapunov function as follows:

$$V(x(t), y(t), z(t)) = (x(t) - x^* \ln(x(t))) + A(y(t) - y^* \ln(y(t))) + B(z(t) - z^* \ln(z(t)))$$

where A and B are positive constants to be determined later. Differentiating the above function with respect to t gives

$$\begin{aligned}
 \dot{V}(x(t), y(t), z(t)) &= \left(1 - \frac{x^*}{x(t)}\right) \dot{x}(t) + A \left(1 - \frac{y^*}{y(t)}\right) \dot{y}(t) + B \left(1 - \frac{z^*}{z(t)}\right) \dot{z}(t) \\
 &= \left(1 - \frac{x^*}{x(t)}\right) (\lambda - \mu x(t) - \beta x(t) z(t)) \\
 &\quad + A \left(1 - \frac{y^*}{y(t)}\right) (\beta(1 - \alpha) x(t) z(t) + \gamma z(t) - [\mu + \kappa(1 - \eta)] y(t)) \\
 &\quad + B \left(1 - \frac{z^*}{z(t)}\right) (\kappa(1 - \eta) y(t) + \beta \alpha x(t) z(t) - (\mu + \delta + \gamma) z(t)). \quad (3.4)
 \end{aligned}$$

Considering equation (3.1) at the endemic equilibrium X^* , one obtains

$$\lambda = \beta x^* z^* + \mu x^*, \quad \mu + \kappa(1 - \eta) = \beta(1 - \alpha) \frac{x^* z^*}{y^*} + \gamma \frac{z^*}{y^*}, \quad \mu + \delta + \gamma = \beta \alpha x^* + \kappa(1 - \eta) \frac{y^*}{z^*} \quad (3.5)$$

Substituting (3.5) into (3.4) gives:

$$\begin{aligned}
 \dot{V}(x(t), y(t), z(t)) &= \left(1 - \frac{x^*}{x(t)}\right) [\beta x^* z^* + \mu x^* - \beta x(t) z(t) - \mu x(t)] \\
 &\quad + A \left(1 - \frac{y^*}{y(t)}\right) \left[\beta(1 - \alpha) x(t) z(t) + \gamma z(t) - \beta(1 - \alpha) x^* z^* \frac{y(t)}{y^*} - \gamma z^* \frac{y(t)}{y^*}\right] \\
 &\quad + B \left(1 - \frac{z^*}{z(t)}\right) \left[\kappa(1 - \eta) y(t) + \beta \alpha x(t) z(t) - \beta \alpha x^* z(t) - \kappa(1 - \eta) y^* \frac{z(t)}{z^*}\right] \\
 &= -\frac{\mu(x(t) - x^*)^2}{x(t)} + \beta x^* z^* \left(1 - \frac{x(t) z(t)}{x^* z^*}\right) \\
 &\quad + A \left(1 - \frac{y^*}{y(t)}\right) \left[(1 - \alpha) \beta x^* z^* \left(\frac{x(t) z(t)}{x^* z^*} - \frac{y(t)}{y^*}\right) + \gamma z^* \left(\frac{z(t)}{z^*} - \frac{y(t)}{y^*}\right)\right] \\
 &\quad + B \left(1 - \frac{z(t)}{z^*}\right) \left[\beta \alpha x^* z^* \left(\frac{x(t) z(t)}{x^* z^*} - \frac{z(t)}{z^*}\right) + \kappa(1 - \eta) y^* \left(\frac{y(t)}{y^*} - \frac{z(t)}{z^*}\right)\right]. \quad (3.6)
 \end{aligned}$$

Using (3.1) at the endemic equilibrium X^* , it can be deduced that

$$y^* = \frac{1}{\mu + \kappa(1 - \eta)} [\beta(1 - \alpha) x^* z^* + \gamma z^*]$$

Equation (3.6) may thus be rewritten as:

$$\begin{aligned}
 \dot{V}(x(t), y(t), z(t)) &= -\frac{\mu(x(t) - x^*)^2}{x(t)} + \beta x^* z^* \left[\left(1 - \frac{x^*}{x(t)}\right) \left(-\frac{x(t) z(t)}{x^* z^*} + 1\right)\right. \\
 &\quad + A(1 - \alpha) \left(1 - \frac{y^*}{y(t)}\right) \left(\frac{x(t) z(t)}{x^* z^*} - \frac{y(t)}{y^*}\right) \\
 &\quad + B \alpha \left(1 - \frac{z(t)}{z^*}\right) \left(\frac{x(t) z(t)}{x^* z^*} - \frac{z(t)}{z^*}\right) \\
 &\quad + \frac{B \kappa(1 - \eta)(1 - \alpha)}{\mu + \kappa(1 - \eta)} \left(1 - \frac{z(t)}{z^*}\right) \left(\frac{y(t)}{y^*} - \frac{z(t)}{z^*}\right) \Big] \\
 &\quad + \gamma z^* \left[A \left(1 - \frac{y^*}{y(t)}\right) \left(\frac{z(t)}{z^*} - \frac{y(t)}{y^*}\right) + \frac{B \kappa(1 - \eta)}{\mu + \kappa(1 - \eta)} \left(1 - \frac{z(t)}{z^*}\right) \left(\frac{y(t)}{y^*} - \frac{z(t)}{z^*}\right)\right]. \quad (3.7)
 \end{aligned}$$

Now, let $(r, s, u) = \left(\frac{x(t)}{x^*}, \frac{y(t)}{y^*}, \frac{z(t)}{z^*}\right)$, then equation (3.7) becomes

$$\begin{aligned}\dot{V}(x(t), y(t), z(t)) &= -\frac{\mu(x(t) - x^*)^2}{x(t)} + \beta x^* z^* \left[\left(1 - \frac{1}{r}\right)(-ru + 1) + A(1 - \alpha) \left(1 - \frac{1}{s}\right)(ru - s) \right. \\ &\quad \left. + B\alpha \left(1 - \frac{1}{u}\right)(ru - u) + \frac{B\kappa(1 - \eta)(1 - \alpha)}{\mu + \kappa(1 - \eta)} \left(1 - \frac{1}{u}\right)(s - u) \right] \\ &\quad + \gamma z^* \left[A \left(1 - \frac{1}{s}\right)(u - s) + \frac{B\kappa(1 - \eta)}{\mu + \kappa(1 - \eta)} \left(1 - \frac{1}{u}\right)(s - u) \right] \\ &= -\frac{\mu(x(t) - x^*)^2}{x(t)} + f(r, s, u),\end{aligned}\tag{3.8}$$

where

$$\begin{cases} f(r, s, u) &= \beta x^* z^* f_1(r, s, u) + \gamma z^* f_2(s, u), \\ f_1(r, s, u) &= -\left(1 - \frac{1}{r}\right)(1 - ru) + A(1 - \alpha) \left(\frac{1}{u} - 1\right)(s - ru) \\ &\quad + B\alpha \left(1 - \frac{1}{u}\right)(ru - u) + \frac{B\kappa(1 - \eta)(1 - \alpha)}{\mu + \kappa(1 - \eta)} \left(1 - \frac{1}{u}\right)(s - u), \\ f_2(s, u) &= A \left(1 - \frac{1}{s}\right)(u - s) + \frac{B\kappa(1 - \eta)}{\mu + \kappa(1 - \eta)} \left(1 - \frac{1}{u}\right)(s - u). \end{cases}\tag{3.9}$$

The constants A and B can be chosen to be in the form $A = A(\alpha)$ and $B = B(\alpha)$ such that the function f is not positive for all $r, s, u \in \mathbb{R}^+$ so that the derivative of $V(x(t), y(t), z(t))$ with respect to time is less than zero. For one to be able to cancel the coefficients of s and u in the expressions of f_1 and f_2 respectively,

$$A = \frac{\kappa(1 - \eta)}{\mu\alpha + \kappa(1 - \eta)} \quad \text{and} \quad B = \frac{\mu + \kappa(1 - \eta)}{\mu\alpha + \kappa(1 - \eta)} \quad \text{are chosen.}\tag{3.10}$$

Substituting equation (3.10) into equation (3.9) gives:

$$\begin{cases} f_1(r, s, u) = 1 + u + \frac{1}{r} + \frac{\kappa(1 - \eta)(1 - \alpha)}{\alpha\mu + \kappa(1 - \eta)} \left(1 - \frac{ru}{s} - u - \frac{s}{u}\right) + \frac{\alpha[\mu + \kappa(1 - \eta)]}{\alpha\mu + \kappa(1 - \eta)} (1 - u - r), \\ f_2(s, u) = \frac{\kappa(1 - \alpha)}{\alpha\mu + \kappa(1 - \alpha)} \frac{(s - u)^2}{su}. \end{cases}\tag{3.11}$$

From equation (3.11), it is clear that the function $f_2 \leq 0$, it is equal to zero when $s = u$. Also, when one differentiates f_1 with respect to α

$$\frac{\partial f_1}{\partial \alpha}(r, s, u) = -\frac{\kappa(1 - \eta)[\mu + \kappa(1 - \eta)]}{[\mu\alpha + \kappa(1 - \eta)]^2} \left(1 + r - \frac{ru}{s} - \frac{s}{u}\right) \text{ is obtained.}$$

If one fixes r, s and u , then $\frac{\partial f_1}{\partial \alpha}$ has a constant sign for $\alpha \in [0, 1]$. Thus, f_1 is maximum at $\alpha = 0$ or $\alpha = 1$.

Suppose $\alpha = 1$ and substituting for $f_1(r, s, u)$ in equation (3.11) gives

$$f_1(r, s, u) = 2 - r - \frac{1}{r},$$

hence, by using of arithmetic-geometric means inequality, $\dot{V}(t) \leq 0$, it's equal to zero only if $x^* = x(t)$ and $s = u$. LaSalle's extension implies that solutions of model system eq. (3.1.1) that intersects with the interior of $\mathcal{D}$ limit to an invariant set contained in $\Gamma = \{(x(t), y(t), z(t)) \in$

$\mathcal{D}, x(t) = x^*, y(t) = y^*, z(t) = z^*, \}$. Then, it follows that the only invariant set in Γ is the one that contains the endemic equilibrium point X^* . Therefore, all solutions of model system eq. (3.1.1) intersect the interior of $\mathcal{D}$ limit to X^* .

Similarly, if $\alpha = 0$, then the function $f_1(r, s, u)$ becomes

$$f_1(r, s, u) = 2 - \frac{ru}{s} - \frac{1}{r} - \frac{s}{u}.$$

As in the previous case, using of arithmetic-geometric means inequality, the only invariant set contained in $\mathcal{D}$ is the singleton X^* . This proves that every solution intersecting $\mathcal{D}$ will limit to the endemic equilibrium point X^* .

Since the set is absorbing, the endemic equilibrium X^* is globally asymptotically stable on $\mathbb{R}_+^3$ for all non-negative initial conditions. This concludes the proof. ■

All the models above are deterministic in nature and do not introduce stochastic effects to the transmission dynamics of the disease. The other models found in the literature (S. Bowong, J. Tewa and J. Kamgang [9] have the same dynamic equations but used the Lyapunov function to show global stability for the endemic equilibrium. The Lyapunov function was also used in articles [51, 52] to determine the global dynamics of SIR and SEIR models.

3.3. Stochastic model

Since transmission of the M. tuberculosis and infection of TB are influenced by various complex biological processes the existence of randomness in the transmission dynamics of the disease could be believed. Taking into consideration all these factors, randomness can be presented to the system by having the parameters μ, δ and β replaced by $\mu \rightarrow \mu + \sigma_1 \dot{W}_1(t), \delta \rightarrow \delta + \sigma_1 \dot{W}_1(t)$ and $\beta \rightarrow \beta + \sigma_2 \dot{W}_2(t)$. This is the starting point for making the system (or model) stochastic, which is done by perturbation of parameters (which is a standard approach in stochastic population modeling). Ideally one is supposed to introduce environmental stochasticity even to other parameters, such as the rate at which infectious individuals move to the latent class γ and λ the rate of recruitment per unit time of susceptible individuals, but doing this would make our analysis more complicated. The new system of stochastic differential equations is now given as follows:

$$\begin{aligned} dx(t) &= (\lambda - \mu x(t) - \beta x(t)z(t))dt - \sigma_1 x(t)dW_1(t), \\ dy(t) &= (\beta(1 - \alpha)x(t)z(t) + \gamma z(t) - [\mu + \kappa(1 - \eta)]y(t))dt - \sigma_1 y(t)dW_1(t), \\ dz(t) &= (\kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) - (\gamma + \mu + \delta)z(t))dt - \sigma_2 z(t)dW_2(t) \end{aligned} \quad (3.1.3)$$

with suitable initial conditions,

where $W_1(t)$ and $W_2(t)$ are independent Wiener processes (or Brownian motions). When parameters such as the death rate of the disease is unpredictable (or has randomness), it is required that environmental noise be introduced to the parameter as explained in [3, 10, 48, 49]. It should be noted that the same noise intensity σ and the Wiener process $W(t)$ are obtained for both the

susceptible and latent infected individuals, but different for infectious individuals. This is due to the fact that biological factors that would affect the rates of mortality for susceptible individuals and individuals in latent stage are highly likely to be identical, but the biological factors that influence infectious individuals are different.

Therefore, since even without comprehensive biological information, it is likely that the Wiener process $W(t)$ and σ (noise intensity) are different for infectious and noninfectious individuals, it is probable that an initial simplifying approximation would be to assume that they are identical. Since infectious and noninfectious individuals would have distinct biological systems, it is reasonable for both $W(t)$ and σ to be different among infectious and noninfectious individuals.

When there are no infected individuals or individuals in a latent state that are present, note that the status would be $(x(t), y(t), z(t)) = (\frac{\lambda}{\mu}, 0, 0)$, which is a point of equilibrium for the deterministic model but it is different for the non-deterministic model although the co-ordinates $(y, z) = (0, 0)$ are still a stochastic equilibrium point for the stochastic model. However, the state of the first co-ordinate is different for the stochastic model and it will later be proved that it stochastically changes around $\frac{\lambda}{\mu}$.

3.3.1 Non-negative solutions

When modeling of population dynamics is involved it is crucial that one should not worry about negative values. Therefore, one has to first prove the non-negativity of solutions.

The usual complete probability space is denoted by $(\Omega, \mathcal{F}, P)$, with filtration $\{\mathcal{F}_t\}_{t \geq 0}$ that satisfies the usual requirements (i.e. it is right continuous and increasing, and all the P -null sets are in $\mathcal{F}_0$). Let $W(t)$ be a one-dimensional Wiener process defined on the complete probability space. Also, let $\mathbb{R}_{++}^3 = \{\tilde{x} \in \mathbb{R}^3 : x_i > 0 \text{ and } x_i \in \tilde{x} \text{ for every } 1 \leq i \leq 3\}$ and let $\tilde{x}(t) = (x(t), y(t), z(t))$.

The following result helps to prove the subsequent theorem.

Lemma 3.3.1. *For every $v > 0$, the following inequality is obtained:*

$$v \leq 2(v + 1 - \log(v)) - (4 - 2\log 2).$$

PROOF.

For every $v > 0$, let $f(v) = v + 2 - 2\log(v)$.

Differentiating $f(v)$ and equating to 0, $v = 2$ is obtained which is the minimum value $f(v)$. The result is thus obtained. $\blacksquare$

Using this result, one can now prove the following theorem.

Theorem 3.3.2. *Let $\lambda, \mu, \gamma, \beta, \delta$ and κ be real positive numbers and $0 < \eta, \alpha < 1$. Then, there exists a unique solution $\tilde{x}(t)$ to (3.1.3) for every initial value $\tilde{x}_0 \in \mathbb{R}_{++}^3$ and $\forall t \geq 0$. With probability 1, this solution will remain in $\mathbb{R}_{++}^3$, that is, $\tilde{x}(t) \in \mathbb{R}_{++}^3$ for every $t \geq 0$ a.s.*

PROOF.

Since the coefficients of the equations in (3.1.3) are locally Lipschitz continuous, then for every

given initial condition $\tilde{x}_0 \in \mathbb{R}_{++}^3$, there is a unique local solution $\tilde{x}(t)$ for every $t \in [0, \tau_e]$, where τ_e is the explosion time [2, 25]. In order to prove that the solution is global, all one has to do is show that $\tau_e < \infty$ almost surely. Consider a constant $r_0 \geq 0$ that is large enough so that all components of $\tilde{x}_0$ are contained in the interval $[\frac{1}{r_0}, r_0]$. The stopping time for each integer $r \geq r_0$ is defined as:

$$\tau_r = \inf\{t \in [0, \tau_e) : \text{at least one of } x, y \text{ or } z \in (\frac{1}{r}, r)\},$$

where throughout this section, one sets $\inf \emptyset = \infty$ (where $\emptyset$ is the empty set). Clearly, τ_r increases as $r \rightarrow \infty$. Let $\tau_\infty = \lim_{r \rightarrow \infty} \tau_r$, then $\tau_e \geq \tau_\infty$ almost surely. If it is proven that $\tau_\infty = \infty$ almost surely, then $\tau_e = \infty$ and $\tilde{x}(t) \in \mathbb{R}_{++}^3$ almost surely for every $t \geq 0$. In other words, for the proof to be complete, it must be proven that $\tau_\infty = \infty$ almost surely. If this statement was not true, then there would be a pair of real numbers $\varepsilon \in (0, 1)$ and $\mathcal{T} > 0$ such that

$$P\{\tau_\infty \leq \mathcal{T}\} > \varepsilon.$$

Then there is an integer $r_1 \leq r_0$ such that

$$P\{\tau_\infty \leq \mathcal{T}\} \geq \varepsilon \quad \text{for every } r \geq r_1. \quad (3.12)$$

Let a $\mathcal{C}^2$ -function $U : \mathbb{R}_{++}^3 \rightarrow \mathbb{R}_{++}^3$ be defined by

$U(\tilde{x}) = 1 + x - \log(x) + 1 + y - \log(y) + 1 + z - \log(z)$. It can be seen that this function is non-negative using $v + 1 - \log(v) \geq 0, \forall v > 0$. Using Itô's formula (Theorem 2.3.4),

$$\begin{aligned} dU(\tilde{x}(t)) &= \left[\left(1 - \frac{1}{x(t)}\right) [\lambda - \mu x(t) - \beta x(t)z(t)] + \left(1 - \frac{1}{y(t)}\right) [\beta(1 - \alpha)x(t)z(t) + \gamma z(t) \right. \\ &\quad \left. - (\mu + \kappa(1 - \eta))y(t)] + \left(1 - \frac{1}{z(t)}\right) [\kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) - (\mu + \gamma + \delta)z(t)] \right. \\ &\quad \left. + \sigma_1^2 + \frac{\sigma_2^2}{2} \right] dt + \sigma_1(2 - x(t) - y(t))dW_1(t) + \sigma_2(1 - z(t))dW_2(t) \\ &= \left[\lambda - \mu x(t) - \beta x(t)z(t) + \beta(1 - \alpha)x(t)z(t) + \gamma z(t) - (\mu + \kappa(1 - \eta))y(t) \right. \\ &\quad \left. + \kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) - (\gamma + \mu + \delta)z(t) - \frac{\lambda}{x(t)} + \mu + \beta z(t) - \frac{\beta(1 - \alpha)x(t)z(t)}{y(t)} \right. \\ &\quad \left. - \frac{\gamma z(t)}{y(t)} + (\mu + \kappa(1 - \eta)) - \frac{\kappa(1 - \eta)y(t)}{z(t)} - \beta\alpha x(t) + (\gamma + \mu + \delta) + \sigma_1^2 + \frac{\sigma_2^2}{2} \right] dt \\ &\quad + \sigma_1(2 - x(t) - y(t))dW_1(t) + \sigma_2(1 - z(t))dW_2(t) \text{ is obtained.} \end{aligned}$$

Hence,

$$\begin{aligned} dU(\tilde{x}(t)) &= [\lambda + 3\mu + \kappa(1 - \eta) + \gamma + \delta + \sigma_1^2 + \frac{\sigma_2^2}{2} + \beta(1 - \alpha)x(t)z(t) \\ &\quad + \gamma z(t) + \kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) + \beta z(t)] dt \\ &\quad + \sigma_1(2 - x(t) - y(t))dW_1(t) + \sigma_2(1 - z(t))dW_2(t) \end{aligned}$$

let

$$c_1 = \lambda + 3\mu + \kappa(1 - \eta) + \gamma + \delta + \sigma_1^2 + \frac{\sigma_2^2}{2} \text{ and } c_2 = \beta(1 - \alpha) + \gamma + \kappa(1 - \eta) + \beta\alpha + \beta$$

According to Lemma 3.3.1, $v \leq 2(v + 1 - \log(v))$ so

$$\beta(1 - \alpha)x(t)z(t) + \gamma z(t) + \kappa(1 - \eta)y(t) + \beta\alpha x(t)z(t) + \beta z(t) \leq c_2 U(\tilde{x}).$$

Therefore,

$$dU(\tilde{x}(t)) \leq (c_1 + c_2 U(\tilde{x}))dt + \sigma_1(2 - x(t) - y(t))dW_1(t) + \sigma_2(1 - z(t))dW_2(t).$$

Hence,

$$dU(\tilde{x}(t)) \leq c_3(1 + U(\tilde{x}))dt + \sigma_1(2 - x(t) - y(t))dW_1(t) + \sigma_2(1 - z(t))dW_2(t),$$

where $c_3 = \max(c_1, c_2)$. Therefore, if $t_1 \leq T$,

$$\begin{aligned} \int_0^{\tau_r \wedge t_1} dU(\tilde{x}(t)) &\leq \int_0^{\tau_r \wedge t_1} c_3(1 + U(\tilde{x}(t)))dt + \int_0^{\tau_r \wedge t_1} \sigma_1(2 - x(t) - y(t))dW_1(t) \\ &\quad + \int_0^{\tau_r \wedge t_1} \sigma_2(1 - z(t))dW_2(t). \end{aligned}$$

Taking expectations, one obtains

$$\begin{aligned} E\left[\int_0^{\tau_r \wedge t_1} dU(\tilde{x}(t))\right] &\leq E\left[\int_0^{\tau_r \wedge t_1} c_3(1 + U(\tilde{x}(t)))dt\right] + E\left[\int_0^{\tau_r \wedge t_1} \sigma_1(2 - x(t) - y(t))dW_1(t)\right] \\ &\quad + E\left[\int_0^{\tau_r \wedge t_1} \sigma_2(1 - z(t))dW_2(t)\right], \end{aligned}$$

which implies

$$\begin{aligned} E[U(\tilde{x}(\tau_r \wedge t_1))] &\leq U(\tilde{x}_0) + E\left[\int_0^{\tau_r \wedge t_1} c_3(1 + U(\tilde{x}(t)))dt\right] + E\left[\int_0^{\tau_r \wedge t_1} \sigma_1(2 - x(t) - y(t))dW_1(t)\right] \\ &\quad + E\left[\int_0^{\tau_r \wedge t_1} \sigma_2(1 - z(t))dW_2(t)\right]. \end{aligned}$$

Using property (ii) of Itô's integrals (theorem 2.3.3),

$$\begin{aligned} E[U(\tilde{x}(\tau_r \wedge t_1))] &\leq U(\tilde{x}_0) + E\left[\int_0^{\tau_r \wedge t_1} c_3(1 + U(\tilde{x}(t)))dt\right] \\ &\leq U(\tilde{x}_0) + c_3 t_1 + c_3 E\left[\int_0^{\tau_r \wedge t_1} U(\tilde{x}(t))dt\right] \\ &\leq U(\tilde{x}_0) + c_3 T + c_3 E\left[\int_0^{\tau_r \wedge t_1} U(\tilde{x}(t))dt\right] \\ &\leq U(\tilde{x}_0) + c_3 T + c_3 \int_0^{t_1} E[U(\tilde{x}(\tau_r \wedge t))]dt \text{ is obtained} \end{aligned}$$

According to the Gronwall inequality (Lemma 2.3.8),

$$f = c_3, \quad \phi = E[U(\tilde{x}(\tau_r \wedge t_1))], \quad c_0 = U(\tilde{x}_0) + c_3 T,$$

then,

$$\begin{aligned} E[U(\tilde{x}(\tau_r \wedge t_1))] &\leq (U(\tilde{x}_0) + c_3 T)e^{\int_0^{t_1} c_3 ds} \\ &= (U(\tilde{x}_0) + c_3 T)e^{c_3 t_1} \\ &\leq (U(\tilde{x}_0) + c_3 T)e^{c_3 T} \end{aligned} \tag{3.13}$$

set $\Delta_r = \{\tau_r \leq T \text{ for } r \leq r_1\}$ and by (3.12), $P(\Delta_r) \geq \epsilon$. It should be noted that for all $\omega \in \Delta_r$, at least one of $x(\tau_r, \omega)$, $y(\tau_r, \omega)$ or $z(\tau_r, \omega)$ will be equal to either r or $\frac{1}{r}$, thus $U(\tilde{x}(\tau_r, \omega))$ is not less

than the minimum between

$$1 + r - \log(r) \text{ and } 1 + \frac{1}{r} - \log(\frac{1}{r}) = 1 + \frac{1}{r} + \log(r).$$

Therefore,

$$U(\tilde{x}(\tau_r, \omega)) \geq 1 + r - \log(r) \wedge 1 + \frac{1}{r} + \log(r).$$

It then follows from (3.12) and (3.13) that

$$\begin{aligned} (U(\tilde{x}_0) + c_3 \mathcal{T})e^{c_3 \mathcal{T}} &\geq E[1_{\Delta_r}(\omega)U(\tilde{x}(\tau_r, \omega))] \\ &\geq \epsilon[(1 + r - \log(r)) \wedge (1 + \frac{1}{r} + \log(r))], \end{aligned}$$

where 1_{Δ_r} is an indicator function of Δ_r . If one allows $r \rightarrow \infty$, then it will lead to a contradiction $\infty > (U(\tilde{x}_0) + c_3 \mathcal{T})e^{c_3 \mathcal{T}} = \infty$. Therefore, $\tau_\infty = \infty$ almost surely. ■

The next step is to investigate how the system behaves asymptotically and attempt to obtain more analytical results.

3.3.2 Asymptotic behaviour

The disease-free equilibrium for the deterministic model was found to be $(\frac{\lambda}{\mu}, 0, 0)$, but not for the stochastic model. However, $y = z = 0$ at the disease-free equilibrium for the stochastic model. Instead of the first co-ordinate to be a fixed constant $\frac{\lambda}{\mu}$, it follows a stochastic process that fluctuates around $\frac{\lambda}{\mu}$. Conditions for exponential stability of $y(t), z(t)$ are first found and a stable distribution for $x(t)$ is obtained.

Definition 3.3.3. (See[[47], p.119]). Denote a complete probability space by $(\Omega, \mathcal{F}, P)$ with a right continuous filtration $\{\mathcal{F}_t\}_{t \geq 0}$ and all the P -null sets to be contained in $\mathcal{F}_0$.

Suppose that $0 \leq t_0 < T < \infty$, and let $\tilde{x}_0$ be an $\mathbb{R}^n$ -valued random variable that is $\mathcal{F}_{t_0}$ -measurable and $E|\tilde{x}_0|^2 < \infty$. Let $G : \mathbb{R}^n \times [t_0, T] \rightarrow \mathbb{R}^{n \times m}$ and $F : \mathbb{R}^n \times [t_0, T] \rightarrow \mathbb{R}^n$ be Borel measurable functions such that $G(0, t) = 0$ and $F(0, t) = 0$ for every $t \geq t_0$. Let an Itô-type n -dimensional stochastic differential equation be defined as

$$d\tilde{x}(t) = F(\tilde{x}(t), t)dt + G(\tilde{x}(t), t)dW(t) \quad (3.14)$$

for $t_0 \leq t \leq T$, with an initial condition $\tilde{x}(t_0) = \tilde{x}_0$, at time t , the stochastic differential equation has a solution $\tilde{x}(t, t_0, \tilde{x}_0)$.

Then, the trivial solution of equation (3.14) is exponentially stable almost surely if

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| < 0 \text{ a.s.}$$

for every $x_0 \in \mathbb{R}^n$.

Theorem 3.3.4. $y(t)$ and $z(t)$ are exponentially stable almost surely if the following conditions hold, that is, they will exponentially tend to their equilibrium position 0 with probability 1.

$$(i) \ 2(\beta x(t) - (\mu + \delta)) - \sigma_2^2 < 0$$

$$(ii) [\beta x(t) - (\mu + \delta) - \mu]^2 < (\sigma_1^2 + 2\mu)(\sigma_2^2 - 2[\beta x(t) - (\mu + \delta)])$$

PROOF.

Let us consider $d(y(t) + z(t))$,

$$\begin{aligned} d(y(t) + z(t)) &= [\beta(1 - \alpha)x(t)z(t) + \gamma z(t) - (\mu + \kappa(1 - \eta))y(t) + \kappa(1 - \eta)y(t) \\ &\quad + \beta\alpha x(t)z(t) - (\gamma + \mu + \delta)z(t)]dt - \sigma_1 y(t)dW_1(t) - \sigma_2 z(t)dW_2(t) \\ &= [\beta x(t)z(t) - \mu y(t) - (\mu + \delta)z(t)]dt - \sigma_1 y(t)dW_1(t) - \sigma_2 z(t)dW_2(t), \end{aligned}$$

Let $\tilde{y} = (y, z)$ and $U(\tilde{y}) = \log(y + z)$ for $y, z \in (0, \infty)$. Using Itô's formula one obtains

$$\begin{aligned} dU(\tilde{y}(t)) &= \left(\frac{\beta x(t)z(t)}{y(t) + z(t)} - \frac{\mu y(t)}{y(t) + z(t)} - \frac{(\mu + \delta)z(t)}{y(t) + z(t)} - \frac{1}{2} \frac{\sigma_1^2 y^2(t)}{(y(t) + z(t))^2} - \frac{1}{2} \frac{\sigma_2^2 z^2(t)}{(y(t) + z(t))^2} \right) dt \\ &\quad - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t) \\ &= \frac{1}{2(y(t) + z(t))^2} [2(y(t) + z(t))(\beta x(t)z(t) - \mu y(t) - (\mu + \delta)z(t)) - \sigma_1^2 y^2(t) - \sigma_2^2 z^2(t)] dt \\ &\quad - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t) \\ &= \frac{1}{2(y(t) + z(t))^2} [2(\beta x(t)y(t)z(t) - \mu y^2(t) - (\mu + \delta)y(t)z(t) + \beta x(t)z^2(t) - \mu y(t)z(t) \\ &\quad - (\mu + \delta)z^2(t)) - \sigma_1^2 y^2(t) - \sigma_2^2 z^2(t)] dt - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t), \end{aligned}$$

one can write the expression

$$2[\beta x(t)y(t)z(t) - \mu y^2(t) - (\mu + \delta)y(t)z(t) + \beta x(t)z^2(t) - \mu y(t)z(t) - (\mu + \delta)z^2(t)] - \sigma_1^2 y^2(t) - \sigma_2^2 z^2(t)$$

as follows:

$$\begin{pmatrix} y(t) & z(t) \end{pmatrix} \begin{pmatrix} -2\mu - \sigma_1^2 & \beta x(t) - (\mu + \delta) - \mu \\ \beta x(t) - (\mu + \delta) - \mu & 2(\beta x(t) - (\mu + \delta)) - \sigma_2^2 \end{pmatrix} \begin{pmatrix} y(t) \\ z(t) \end{pmatrix}.$$

Hence, $dU(\tilde{y}(t))$ can be expressed as follows

$$\begin{aligned} dU(\tilde{y}) &= \frac{1}{2(y(t) + z(t))^2} \left\{ \begin{pmatrix} y(t) & z(t) \end{pmatrix} \begin{pmatrix} -2\mu - \sigma_1^2 & \beta x(t) - (\mu + \delta) - \mu \\ \beta x(t) - (\mu + \delta) - \mu & 2(\beta x(t) - (\mu + \delta)) - \sigma_2^2 \end{pmatrix} \begin{pmatrix} y(t) \\ z(t) \end{pmatrix} \right\} dt \\ &\quad - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t). \end{aligned}$$

Consider the matrix

$$\begin{pmatrix} -2\mu - \sigma_1^2 & \beta x(t) - (\mu + \delta) - \mu \\ \beta x(t) - (\mu + \delta) - \mu & 2(\beta x(t) - (\mu + \delta)) - \sigma_2^2 \end{pmatrix}.$$

This matrix is negative-definite since $-2\mu - \sigma_1^2 < 0$ and its determinant is positive, let the largest (negative) eigenvalue be denoted as $\lambda_{\max}$, then

$$\begin{pmatrix} y(t) & z(t) \end{pmatrix} \begin{pmatrix} -2\mu - \sigma_1^2 & \beta x(t) - (\mu + \delta) - \mu \\ \beta x(t) - (\mu + \delta) - \mu & 2(\beta x(t) - (\mu + \delta)) - \sigma_2^2 \end{pmatrix} \begin{pmatrix} y(t) \\ z(t) \end{pmatrix} \leq \lambda_{\max}(y^2(t) + z^2(t)) \\ = -|\lambda_{\max}|(y^2(t) + z^2(t)).$$

Therefore,

$$dU(\tilde{y}(t)) \leq \left(-|\lambda_{\max}| \frac{1}{2(y(t) + z(t))^2} (y^2(t) + z^2(t)) \right) dt - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t). \quad (3.15)$$

As $(y^2 + z^2) \geq 2yz$ and $2(y^2 + z^2) = y^2 + z^2 + y^2 + z^2 \geq y^2 + z^2 + 2yz = (y + z)^2$ i.e $2(y^2 + z^2) \geq (y + z)^2 \Rightarrow \frac{1}{2}(y + z)^2 \leq (y^2 + z^2)$,
using $\frac{1}{2}(y + z)^2 \leq (y^2 + z^2)$, equation (3.15) becomes

$$\begin{aligned} dU(\tilde{y}(t)) &\leq -\frac{1}{4}|\lambda_{\max}|dt - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t) \\ \Rightarrow d(\log(y(t) + z(t))) &\leq -\frac{1}{4}|\lambda_{\max}|dt - \frac{\sigma_1 y(t)}{(y(t) + z(t))^2} dW_1(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))^2} dW_2(t). \end{aligned}$$

When integrated the inequality above and using the following result due to Mao [47]

$$\limsup_{t \rightarrow \infty} \frac{1}{t} |W_i(t)| = 0 \text{ for } i = 1, 2$$

one obtains

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log(y(t) + z(t)) \leq -\frac{1}{4}|\lambda_{\max}| < 0 \text{ a.s.}$$

Therefore, $y(t) \rightarrow 0$ and $z(t) \rightarrow 0$ a.s $t \rightarrow \infty$. This completes the proof. ■

It should be noted that Theorem 3.3.4 above does not give any condition concerning the value of R_0 , it does not particularly presume that $R_0 < 1$. Also, it should be noted that when $\sigma_1 = \sigma_2 = 0$, then conditions in the Theorem above cannot be satisfied for the deterministic model.

In Theorem 3.3.4 (i) and (ii) the restrictions on the variances do not have a clear meaning in themselves biologically. Nevertheless, notice that the term $\beta x(t)z(t)$ is the rate at which susceptibles become infected. Under the condition $\beta z(t) < 1$, an infectious individual infects less than one susceptible on average during the whole period of being infective, which implies that $R_0 < 1$, condition (i) in Theorem 3.3.4 will always hold. The conditions in Theorem 3.3.4 will always be satisfied provided that the variances σ_1^2 and σ_2^2 are sufficiently large. This result is interesting because whenever the variances for the noise are sufficiently large, then the number of infectious individuals in the population will eventually reduce to zero, no matter what values other parameters have, even when $R_0 > 1$.

Attention is now shifted to the susceptible population $x(t)$. Stability in distribution for $x(t)$ is shown, that is, it becomes stable around the mean value $\frac{\lambda}{\mu}$. To achieve this, a new stochastic

process $x_2(t)$ is set up defined by the following stochastic differential equation with initial condition $x_2(0) = x(0)$,

$$dx_2(t) = (\lambda - \mu x_2(t))dt - \sigma_1 x_2(t)dW_1(t).$$

One wants to demonstrate that $x(t)$ can be approximated by $x_2(t)$ in limit when t becomes large, that is,

$$\lim_{t \rightarrow \infty} (x_2(t) - x(t)) = 0 \quad \text{in probability.}$$

Another function $x_1(t)$ is introduced to help prove the theorem. It is defined by the stochastic differential equation with initial condition $x_1(0) = x(0)$

$$dx_1(t) = (\lambda - (\mu + \epsilon)x_1(t))dt - \sigma_1 x_1(t)dW_1(t). \quad (3.16)$$

Theorem 3.3.5. *If conditions of Theorem 3.3.4 are satisfied, then*

$$\lim_{t \rightarrow \infty} (x_2(t) - x(t)) = 0 \quad \text{in probability.}$$

PROOF.

The equation of $x(t)$ is

$$dx(t) = (\lambda - \mu x(t) - \beta x(t)z(t))dt - \sigma_1 x(t)dW_1(t).$$

We first prove that

$$\liminf_{t \rightarrow \infty} (x(t) - x_1(t)) \geq 0 \quad \text{a.s.}$$

Therefore, consider

$$\begin{aligned} d(x(t) - x_1(t)) &= (-\mu(x(t) - x_1(t)) + \epsilon x_1(t) - \beta x(t)z(t))dt - \sigma_1(x(t) - x_1(t))dW_1(t) \\ &= (-(\mu + \epsilon)(x(t) - x_1(t)) + (\epsilon - \beta z(t))x(t))dt - \sigma_1(x(t) - x_1(t))dW_1(t), \end{aligned}$$

and the solution is given by

$$x(t) - x_1(t) = \varphi(t) \int_0^t \varphi^{-1}(s)(\epsilon - \beta z(s))x(s)ds$$

where

$$\varphi(t) = \exp \left\{ -(\mu + \epsilon + \frac{\sigma_1^2}{2})t - \sigma_1 W_1(t) \right\}.$$

Using the results of Theorem 3.3.4 where it was shown that $z(t) \rightarrow 0$ a.s as $t \rightarrow \infty$, it is concluded that for nearly every $\omega \in \Omega$, there is a $T = T(\omega)$ such that

$$z(t) < \frac{\epsilon}{\beta}, \forall t \geq T.$$

Therefore, for every $\omega \in \Omega$, if $t > T$, then

$$x(t) - x_1(t) = \varphi(t) \left(\int_0^T \varphi^{-1}(s)(\epsilon - \beta z(s))x(s)ds + \int_T^t \varphi^{-1}(s)(\epsilon - \beta z(s))x(s)ds \right).$$

Hence, $x(t) - x_1(t) \geq \varphi(t)k(T)$, where

$$k(T) = \int_0^T \varphi^{-1}(s)(\epsilon - \beta z(s))x(s)ds$$

clearly, as $t \rightarrow \infty$ then $\varphi(t) \rightarrow 0$ and $|k(T)| < \infty$ a.s.

Therefore,

$$\liminf_{t \rightarrow \infty} |x(t) - x_1(t)| \geq 0 \quad \text{a.s.} \quad (3.17)$$

Next, it is proven that $\liminf_{t \rightarrow \infty} (x_2(t) - x(t)) \geq 0$ a.s. For this, one considers

$$d(x_2(t) - x(t)) = (\mu(x(t) - x_2(t)) + \beta x(t)z(t))dt + \sigma_1(x(t) - x_2(t))dW_1(t).$$

This implies that

$$d(x_2(t) - x(t)) \geq (\mu(x(t) - x_2(t)))dt + \sigma_1(x(t) - x_2(t))dW_1(t).$$

Let $\phi = x_2 - x$. Hence,

$$d\phi(t) \geq -\mu\phi(t)dt - \sigma_1\phi(t)dW_1(t).$$

For every $\omega \in \Omega$, either

$$\omega \in \Omega_1 = \{\omega : \phi(t_0) > 0 \text{ for some } t_0 \geq 0\}$$

or

$$\omega \in \Omega_2 = \{\omega : \phi(t_0) \leq 0 \text{ for every } t_0 \geq 0\}.$$

For $\omega \in \Omega_1$, let $t_1 = \sup\{t : t \geq t_0 \text{ and } \phi(t)\} > 0$. If $t_1 < \infty$, then in $[t_0, t_1]$

$$d\phi(t) = (-\mu + f(t))\phi(t)dt - \sigma_1\phi(t)dW_1(t) \quad (3.18)$$

with a random variable $f(t)$. To find the solution of (3.18), let $Y = \log \phi(t)$ and Itô's formula is used to obtain

$$\phi(t) = \phi(t_0) \exp \left\{ \int_{t_0}^t (-\mu + f(s) - \frac{\sigma_1^2}{2})ds - \sigma_1 W_1(t) \right\} > 0.$$

Hence,

$$\phi(t_1) = \phi(t_0) \exp \left\{ \int_{t_0}^{t_1} (-\mu + f(s) - \frac{\sigma_1^2}{2})ds - \sigma_1 W_1(t) \right\} > 0.$$

Thus, $\phi(t) > 0$ in $[t_1, t_1 + \delta_1]$ for some δ_1 . This is a contradiction. Hence, $t_1 = \infty$ and $\phi(t) \geq 0$ for every $t \geq t_0$. So one obtains $\liminf_{t \rightarrow \infty} \phi(t) \geq 0$ a.s.

A case for $\omega \in \Omega_2$ is now considered where $\phi(t) \leq 0$ for every $t \geq 0$. Then,

$$d\phi(t) = (-\mu - f(t))\phi(t)dt - \sigma_1\phi(t)dW_1(t), \quad (3.19)$$

where the random variable $f(t) \geq 0$. Then one finds the solution of (3.19) by letting $Y = \log \phi(t)$ and using Itô's formula to obtain

$$\phi(t) = \phi(0) \exp \left\{ \int_0^t (-\mu - f(s) - \frac{\sigma_1^2}{2})ds - \sigma_1 W_1(t) \right\} \geq 0, \quad \text{since } x_2(0) = x(0).$$

Hence, $\liminf_{t \rightarrow \infty} \phi(t) \geq 0$ a.s. Therefore,

$$\liminf_{t \rightarrow \infty} (x_2(t) - x(t)) \geq 0 \quad \text{a.s.} \quad (3.20)$$

Next one considers $d(x_1(t) - x_2(t))$,

$$d(x_1(t) - x_2(t)) = (-\mu(x_1(t) - x_2(t)) - \epsilon x_1(t))dt - \sigma_1(x_1(t) - x_2(t))dW_1(t),$$

whose solution is

$$x_1(t) - x_2(t) = -\epsilon \exp \left\{ -(\mu + \frac{\sigma_1^2}{2})t - \sigma_1 W_1(t) \right\} \int_0^t \exp \left\{ (\mu + \frac{\sigma_1^2}{2})s - \sigma_1 W_1(s) \right\} x_1(s) ds.$$

It should be noted that $x_1(s) \geq 0$, since it is a solution of the stochastic differential equation (3.16) which is linear and can be solved explicitly to give

$$x_1(t) = \lambda \int_0^t \exp \left\{ -(\mu + \epsilon + \frac{\sigma_1^2}{2})(t-s) - \sigma_1(W_1(t) - W_1(s)) \right\} ds \quad (3.21)$$

Therefore,

$$|x_1(t) - x_2(t)| = \epsilon \int_0^t x_1(s) \exp \left\{ -(\mu + \frac{\sigma_1^2}{2})(t-s) - \sigma_1(W_1(t) - W_1(s)) \right\} ds$$

since $W_1(t) - W_1(s) \sim \mathcal{N}(0, t - s)$, one obtains

$$\begin{aligned}
 E[\exp[-\sigma_1(W_1(t) - W_1(s))]] &= \int_{-\infty}^{\infty} e^{-\sigma_1 u} \frac{1}{\sqrt{2\pi(t-s)}} e^{-\frac{u^2}{2(t-s)}} du \\
 &= \frac{1}{\sqrt{2\pi(t-s)}} \int_{-\infty}^{\infty} \exp\left\{-\frac{1}{2(t-s)}(u^2 + 2(t-s)\sigma_1 u)\right\} du \\
 &= \frac{1}{\sqrt{2\pi(t-s)}} \int_{-\infty}^{\infty} e^{\left\{-\frac{1}{2(t-s)}(u^2 + 2(t-s)\sigma_1 u) + (t-s)^2\sigma_1^2 - (t-s)^2\sigma_1^2\right\}} du \\
 &= \frac{e^{\frac{(t-s)\sigma_1^2}{2}}}{\sqrt{2\pi(t-s)}} \int_{-\infty}^{\infty} \exp\left\{-\frac{1}{2(t-s)}(u + (t-s)\sigma_1)^2\right\} du \\
 &= e^{\frac{(t-s)\sigma_1^2}{2}}.
 \end{aligned}$$

Hence,

$$E[\exp[-\sigma_1(W_1(t) - W_1(s))]] = \exp\left\{\frac{\sigma_1^2}{2}(t-s)\right\}. \quad (3.22)$$

Therefore,

$$\begin{aligned}
 E|x_1(t) - x_2(t)| &= \epsilon E\left[\int_0^t x_1(s) \exp\left\{-\left(\mu + \frac{\sigma_1^2}{2}\right)(t-s) - \sigma_1(W_1(t) - W_1(s))\right\} ds\right] \\
 &= \epsilon \int_0^t E[x_1(s) \exp\left\{-\left(\mu + \frac{\sigma_1^2}{2}\right)(t-s) - \sigma_1(W_1(t) - W_1(s))\right\}] ds \\
 &= \epsilon \int_0^t E[x_1(s) \exp\left\{-\left(\mu + \frac{\sigma_1^2}{2}\right)(t-s)\right\}] E[\exp[-\sigma_1(W_1(t) - W_1(s))]] ds
 \end{aligned}$$

as $x_1(s)$ is independent of $W_1(t) - W_1(s)$,

$$\begin{aligned}
 &= \epsilon \int_0^t E[x_1(s) \exp\left\{-\left(\mu + \frac{\sigma_1^2}{2}\right)(t-s)\right\}] \exp\left\{\left(\frac{t-s}{2}\right)\sigma_1^2\right\} ds \\
 &= \epsilon \int_0^t E[x_1(s) \exp\{-\mu(t-s)\}] ds \\
 &= \epsilon \int_0^t \exp\{-\mu(t-s)\} E(x_1(s)) ds. \quad (3.23)
 \end{aligned}$$

Taking the expectation of (3.21) and using (3.22), one obtains

$$E[x_1(s)] = \lambda \int_0^t \exp\{-(\mu + \epsilon)(t-s)\} ds \leq \frac{\lambda}{\mu + \epsilon}.$$

Substituting this result in (3.23), one obtains

$$\begin{aligned}
 E|x_1(t) - x_2(t)| &\leq \frac{\lambda}{\mu + \epsilon} \int_0^t \exp\{-\mu(t-s)\} ds \\
 &= \frac{\lambda}{\mu + \epsilon} \left(\frac{1}{\mu} - \frac{e^{-\mu t}}{\mu}\right) \\
 &= \frac{\lambda \epsilon e^{-\mu t}}{\mu(\mu + \epsilon)} (e^{\mu t} - 1).
 \end{aligned}$$

Hence, one obtains

$$\lim_{\epsilon \rightarrow 0} \lim_{t \rightarrow \infty} E|x_1(t) - x_2(t)| = 0.$$

This implies that

$$\lim_{\epsilon \rightarrow 0} \lim_{t \rightarrow \infty} |x_1(t) - x_2(t)| = 0 \quad \text{in probability} \quad (3.24)$$

Combining (3.17), (3.20) and (3.24), one obtains the required result. Therefore, the proof is complete. $\blacksquare$

3.3.3 Mean reverting process

Since the process $x(t)$ was estimated by $x_2(t)$, one wants to know more about the process $x_2(t)$. This is done by finding parameters like the variance and mean for this process.

The differential for the mean reverting process is

$$dx_2(t) = (\lambda - \mu x_2(t))dt - \sigma_1 x_2(t)dW_1(t).$$

Its explicit solution is given by

$$x_2(t) = x_2(0) \exp \left\{ -\left(\mu + \frac{\sigma_1^2}{2}\right)t - \sigma_1 W_1(t) \right\} + \lambda \int_0^t \exp \left\{ -\left(\mu + \frac{\sigma_1^2}{2}\right)(t-s) - \sigma_1 (W_1(t) - W_1(s)) \right\} ds$$

taking the expectation

$$E[x_2(t)] = x_2(0) E[\exp \left\{ -\left(\mu + \frac{\sigma_1^2}{2}\right)t \right\}] E[e^{-\sigma_1 W_1(t)}] + \lambda \int_0^t E[\exp \left\{ -\left(\mu + \frac{\sigma_1^2}{2}\right)(t-s) \right\}] E[e^{-\sigma_1 (W_1(t) - W_1(s))}] ds$$

Since $W_1(t) \sim \mathcal{N}(0, t)$, then

$$\begin{aligned} E[e^{-\sigma_1 W_1(t)}] &= \int_{-\infty}^{\infty} e^{-\sigma_1 u} \frac{1}{\sqrt{2\pi t}} e^{-\frac{u^2}{2t}} du \\ &= \frac{1}{\sqrt{2\pi t}} \int_{-\infty}^{\infty} e^{-\frac{1}{2t}(u^2 + 2t\sigma_1 u)} du \\ &= \frac{1}{\sqrt{2\pi t}} \int_{-\infty}^{\infty} e^{-\frac{1}{2t}[u^2 + 2t\sigma_1 u - t^2\sigma_1^2 + t^2\sigma_1^2]} du \\ &= \frac{1}{\sqrt{2\pi t}} \int_{-\infty}^{\infty} e^{-\frac{1}{2t}[(u+t\sigma_1)^2 - t^2\sigma_1^2]} du \\ &= e^{-\frac{t\sigma_1^2}{2}} \frac{1}{\sqrt{2\pi t}} \int_{-\infty}^{\infty} e^{-\frac{(u+t\sigma_1)^2}{2t}} du \\ &= e^{-\frac{t\sigma_1^2}{2}}. \end{aligned} \tag{3.25}$$

Substituting (3.22) and (3.25) in the expression for $E[x_2(t)]$, one obtains

$$\begin{aligned} E[x_2(t)] &= x_2(0) E[e^{-\mu t}] + \lambda \int_0^t E[e^{-\mu(t-s)}] ds \\ &= x_2(0) e^{-\mu t} + \frac{\lambda}{\mu} (1 - e^{-\mu t}) \end{aligned}$$

therefore taking the limit of $E[x_2(t)]$ as $t \rightarrow \infty$ we have

$$\lim_{t \rightarrow \infty} E[x_2(t)] = \frac{\lambda}{\mu}$$

To obtain the second moment, $V(x_2(t)) = x_2^2$ is considered and the Itô's formula is used to get

$$d(x_2^2(t)) = (\sigma_1^2 - 2\mu)x_2^2(t)dt + 2\lambda x_2 dt - 2\sigma_1 x_2^2(t)dW_1(t).$$

Hence,

$$x_2^2(t) = x_2^2(0) + \int_0^t (\sigma_1^2 - 2\mu)x_2^2(s)ds + 2\lambda \int_0^t x_2(s)ds - 2\sigma_1 \int_0^t x_2^2(s)dW_1(s).$$

Taking the expectation, one obtains

$$E[x_2^2(t)] = E[x_2^2(0)] + \int_0^t (\sigma_1^2 - 2\mu)E[x_2^2(s)]ds + 2\lambda \int_0^t E[x_2(s)]ds.$$

The expectation of the stochastic integral is zero (property (ii) of theorem 2.3.3).

When one differentiates with respect to t ,

$$\frac{d}{dt}E[x_2^2(t)] = (\sigma_1^2 - 2\mu)E[x_2^2(t)] + 2\lambda E[x_2(t)] \text{ is obtained.}$$

Also,

$$\begin{aligned} \frac{d}{dt} \{E[x_2^2(t)]e^{-(\sigma_1^2 - 2\mu)t}\} &= e^{-(\sigma_1^2 - 2\mu)t} \frac{d}{dt}E[x_2^2(t)] + E[x_2^2(t)] \frac{d}{dt}e^{-(\sigma_1^2 - 2\mu)t} \\ &= e^{-(\sigma_1^2 - 2\mu)t} \left[(\sigma_1^2 - 2\mu)E[x_2^2(t)] + 2\lambda E[x_2(t)] \right] - (\sigma_1^2 - 2\mu)E[x_2^2(t)]e^{-(\sigma_1^2 - 2\mu)t} \\ &= 2\lambda E[x_2(t)]e^{-(\sigma_1^2 - 2\mu)t}. \end{aligned}$$

Integrating and multiplying by $e^{(\sigma_1^2 - 2\mu)t}$ one obtains

$$E[x_2^2(t)] = E[x_2^2(0)]e^{(\sigma_1^2 - 2\mu)t} + 2\lambda e^{(\sigma_1^2 - 2\mu)t} \int_0^t e^{-(\sigma_1^2 - 2\mu)s} E[x_2(s)]ds$$

When we substitute for the value of $E[x_2(s)]$ in the above expression we get

$$E[x_2^2(t)] = E[x_2^2(0)]e^{(\sigma_1^2 - 2\mu)t} + 2\lambda e^{(\sigma_1^2 - 2\mu)t} \left\{ \int_0^t e^{(\sigma_1^2 - 2\mu)s} (z(0)e^{-\mu s} + \frac{\lambda}{\mu}(1 - e^{-\mu s})) ds \right\}$$

Simplifying we have

$$\begin{aligned} E[x_2^2(t)] &= E[x_2^2(0)]e^{(\sigma_1^2 - 2\mu)t} + 2\lambda e^{(\sigma_1^2 - 2\mu)t} \left\{ z(0) \int_0^t e^{(\sigma_1^2 - 2\mu)s} e^{-\mu s} ds + \frac{\lambda}{\mu} \int_0^t e^{(\sigma_1^2 - 2\mu)s} (1 - e^{-\mu s}) ds \right\} \\ &= E[x_2^2(0)]e^{(\sigma_1^2 - 2\mu)t} + 2\lambda e^{(\sigma_1^2 - 2\mu)t} \left\{ -\left(\frac{z(0) - \frac{\lambda}{\mu}}{\sigma_1^2 - \mu}\right)(e^{-(\sigma_1^2 - \mu)t} - 1) - \frac{\lambda}{\mu(\sigma_1^2 - 2\mu)}(e^{-(\sigma_1^2 - 2\mu)t} - 1) \right\} \end{aligned}$$

given that $\sigma_1^2 \neq \mu, 2\mu$

$$\begin{aligned} &= E[x_2^2(0)]e^{(\sigma_1^2 - 2\mu)t} - 2\lambda \left(\frac{z(0) - \frac{\lambda}{\mu}}{\sigma_1^2 - \mu}\right)e^{-\mu t} + 2\lambda \left(\frac{z(0) - \frac{\lambda}{\mu}}{\sigma_1^2 - \mu}\right)e^{(\sigma_1^2 - 2\mu)t} - \frac{2\lambda^2}{\mu(\sigma_1^2 - 2\mu)} \\ &\quad + \frac{2\lambda^2}{\mu(\sigma_1^2 - 2\mu)}e^{(\sigma_1^2 - 2\mu)t}. \end{aligned}$$

Taking the limit, one needs $\sigma_1^2 < 2\mu$ for $\lim_{t \rightarrow \infty} E[x_2^2(t)] < \infty$, then one obtains

$$\lim_{t \rightarrow \infty} E[x_2^2(t)] = \frac{2\lambda^2}{\mu(2\mu - \sigma_1^2)}.$$

The term above is positive since $\sigma_1^2 - 2\mu < 0$. Therefore for the mean reverting process the asymptotic variance is

$$\lim_{t \rightarrow \infty} \text{Var}(x_2(t)) = \lim_{t \rightarrow \infty} [E[x_2^2(t)] - [E(x_2(t))]^2] = \frac{\lambda^2 \sigma_1^2}{\mu^2(2\mu - \sigma_1^2)}.$$

If $\sigma_1^2 = \mu$, then $\lim_{t \rightarrow \infty} E[x_2^2(t)] = 2\frac{\lambda^2}{\mu^2}$ and $\lim_{t \rightarrow \infty} \text{Var}(x_2(t)) = 2\frac{\lambda^2}{\mu^2} - \frac{\lambda^2}{\mu^2} = \frac{\lambda^2}{\mu^2}$.

Finally, if $\sigma_1^2 \geq 2\mu$ then $\lim_{t \rightarrow \infty} \text{Var}(x_2(t)) = \infty$.

Hence, if $\sigma_1^2 < 2\mu$, then there is a finite variance for the limit process given by

$$\frac{2\lambda^2}{\mu(2\mu - \sigma_1^2)},$$

However, if $\sigma_1^2 \geq 2\mu$, then an infinite variance is obtained for the limit process.

Also, for $\sigma_1^2 = \mu$ a finite variance $\frac{\lambda^2}{\mu^2}$ is obtained.

3.4. Conclusion

This chapter has presented the stochastic model that describes the dynamics of transmission for tuberculosis. It was shown that the solutions are non-negative. The aspect of stability for the stochastic model was studied. The number of infectious individuals and the number of infected individuals were shown in latent stage to asymptotically tend to zero exponentially with probability one. In addition, it was revealed that $x(t)$ can be approximated by a mean reverting process $x_2(t)$. Previously, the models that have been studied for the transmission dynamics of tuberculosis in the literature use ordinary differential equations that are deterministic, neglecting the stochastic effects. In this study it was shown that stochastic differential equations give additional options in modeling the transmission dynamics of TB. By reproducing the results obtained from the deterministic model [9] and making some improvements to it, it was shown that the stochastic model considered adds a new dimension of results to the dynamics of transmission of the disease. It adds a different perspective to this particular problem and gives researchers a different approach to be considered in future. Since most of the problems in the real world are not deterministic in nature, but rather stochastic, then including effects of stochasticity into the model gives a more realistic way of modeling the dynamics of transmission of TB.

For instance, the stochastic model enabled one to study the limiting asymptotic distribution of the number of susceptible and infected individuals in a latent state and infectious individuals. The limiting asymptotic variance expression for the distribution of the number of susceptible individuals was also derived. Models that are deterministic are less versatile compared to models that are stochastic because stochastic models incorporate random effects such as environmental stochasticity and one was able to find quantities such as the variance, probabilities of extinction and probability distributions of variables, which are characteristics that are impossible to include in deterministic models.

4

Stochastic model and analysis of the dynamics of AIDS and use of condoms

4.1. Introduction

The first time AIDS was diagnosed as a new distinct disease was in 1981. It appears to have started in America, Australia and Western parts of Europe, mainly among men who were homosexual or bisexual and drug abusers who used injections in certain urban regions. In some parts of Central Africa and the Caribbean, it was among individuals with multiple sexual partners (WHO [75]). HIV was discovered in 1983 as the virus that causes AIDS. It takes about 10 years on average for HIV infection to progress to AIDS. No efficient medication that can cure AIDS has been discovered so far and infected people never recover from it. This means they remain infective throughout their entire life. Until today, HIV is being spread relentlessly throughout the world since its discovery in 1981, and it is now a worldwide pandemic. The HIV virus spreads in the human body by attacking and weakening the immune system, particularly by decreasing the CD4 cells (or T-helper cells). The pathogeny of HIV-AIDS is the function of its life expectancy, cell environment in the body of the host, and the amount of virus in the infected person's body. Factors such as genetic distinctions between individuals, age, virulence levels of a particular virus strain, and coinfection with other pathogenic bacteria influence the rate of progression and severity of the disease. HIV-AIDS is a composite mixture of several pandemics in the world, and its really a defining public health crisis for this generation. The three well-known forms in which HIV spreads to other uninfected people are through direct association with body fluids or blood of an infected individual, sexual contact with a person infected with the virus, and mother-to-child infection.

The greatest number of infected people are currently in Africa (mostly sub-Sahara). However, in various regions in the world, such as Western Europe, Latin America, USA and the Caribbean, the major mode of transmission is between homosexual men and intravenous drug users (UNAIDS [68]; MAP [50]).

Despite the increased access to treatment and funding, as well as political obligation towards it,

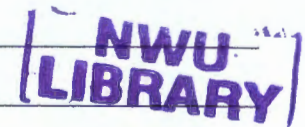
the HIV-AIDS pandemic has continuously outpaced the universal feedback. The HIV pandemic remains exceedingly dynamic, the virus keeps developing and changing its characteristics as it exploits new chances to spread.

There has been considerable amount of work done in Mathematics to model the transmission dynamics of AIDS or HIV, and such studies are generally fixed to the ordinary differential equations. Different kinds of models have been studied by many researchers. Anderson and Blythe [5, 6, 7] included heterogenousness to the target population to model the spread of HIV-AIDS. They derived distributions that describe the variation in the incubation cycle of the virus using a group of hazard functions. Likewise, J.A. Jacquez, C.P. Simon and J.S. Koopman [33, 34] modeled the spread of HIV among the homosexual community by dividing the population into n subpopulations, considering how sexually active the individuals were. This examination was done through mixed patterns. The model discussed by Greenhalgh et al. [29] in their paper (A mathematical treatment of AIDS and condom use) is considered in this study. In their model, they did not account for the built-in randomness associated with the dynamics of HIV-AIDS transmission. An investigation of how environmental noise affects the transmission dynamics of the infection represented by the system of differential equations considered will be done.

In this chapter, the modeling for the spread of the HIV virus between the homogenised populations of homosexuals divided in different risk subgroups is discussed depending on tendencies of people towards the use of condoms.

A discussion of stochasticity to the mathematical model of HIV-AIDS and condom use is done through perturbation of parameters acceptable for use in the stochastic population modeling. Firstly, it is shown that the stochastic system has solutions that are non-negative as required in the dynamics of population modeling. A thorough asymptotic stability analysis is done by studying its p^{th} moment and stability in probability. The aim is to find out the influence of environmental noise towards the stability of the system.

4.2. Deterministic model



A model with two population groups is considered for infected and susceptible homosexual males. It is assumed that every individual belongs to any one of two different groups that are sexually active, with α_1 and α_2 as their respective risk levels, where α_1 and α_2 is the probability that individuals do not insist on using condoms in their partnership. For instance, a person from the second group with $\alpha_2 = 0$ will always want to use a condom, while a person from the second group with $\alpha_2 = 1$ will never insist on using a condom. In addition, $0 \leq \alpha_1 \leq \alpha_2 \leq 1$. This means the second group is more at risk than the first one. If a person from the first group has sexual contact with a person from the second group, then $1 - \alpha_1\alpha_2$ is the probability that the person will use a condom. A definition of the variables to be used in the model is given.

- $x(t)$ total number of all susceptible individuals in the first group at time t ;
 $y(t)$ total number of all infected individuals in the first group at time t ;
 $z(t)$ total number of all susceptible individuals in the second group at time t ; and
 $z_1(t)$ total number of all infected individuals in the second group at time t .

The following four-dimensional deterministic model is part of the model discussed by Greenhalgh *et al.* [29]:

$$\begin{aligned}
 \frac{dx(t)}{dt} &= \lambda_1 - \mu x(t) - B_1 x(t) + C_{21} z(t) - C_{12} x(t), \\
 \frac{dy(t)}{dt} &= B_1 x(t) - (\mu + \sigma + D_{12}) y(t) + D_{21} z_1(t), \\
 \frac{dz(t)}{dt} &= \lambda_2 - \mu z(t) - B_2 z(t) + C_{12} x(t) - C_{21} z(t), \text{ and} \\
 \frac{dz_1(t)}{dt} &= B_2 z(t) - (\mu + \sigma + D_{21}) z_1(t) + D_{12} z_1(t),
 \end{aligned} \tag{4.1.1}$$

with relevant initial conditions, where $B_k = \beta c \alpha_k G(t)$, for $k = 1, 2$ and $G(t) = \frac{\alpha_1 y(t) + \alpha_2 z_1(t)}{x(t) + y(t) + z(t) + z_1(t)}$.

A full description of all parameters used in the model is given below:

- λ_1 rate at which new susceptible people are recruited in the first group;
- λ_2 rate at which new susceptible people are recruited in the second group;
- μ per capita rate at which people are leaving populations that are sexually mixing for reasons that are not disease-related;
- σ per capita rate at which individuals who are infected develop AIDS;
- β the mean sexual risk by every infected partner when they share bodily fluids;
- c number of partners on average per unit time for each individual;
- α_1 probability for an individual in the first group not wanting to use a condom;
- α_2 probability for an individual in the second group not wanting to use a condom;
- C_{12} per capita migration rate of a susceptible person to the unsafe group from the safe group;
- C_{21} per capita migration rate of a susceptible person to the safe group from the unsafe group;
- D_{12} per capita migration rate for an infected person to the unsafe group from the safe group; and
- D_{21} per capita migration rate for an infected person to the safe group from the unsafe group.

Proportional mixing within groups is assumed. The number of partners for a person in the first group is $c\delta t + o(\delta t)$ for time $[t, t + \delta t)$. $G(t)$ is the probability of not using a condom whenever there is an encounter at a time t with an infected partner. The number of infections in the first group in $[t, t + \delta t)$ is $\beta c \alpha_1 G(t) \delta t + o(\delta t)$ and a person does not insist on using a condom and becomes infected. Also $\beta c \alpha_2 G(t) \delta t + o(\delta t)$ is the number of infections in the second group in $[t, t + \delta t)$.

For this model, Greenhalgh *et al.* [29] came up with an expression for the basic reproductive ratio, known as the number of cases produced on average by an infected person during his/her infective period in an uninfected population (denoted by R_0). They demonstrated that the model

has a unique solution at the disease-free equilibrium, and it is also locally asymptotically stable whenever $R_0 < 1$ and it is unstable when $R_0 > 1$. Furthermore, there is a unique endemic equilibrium, whenever $R_0 > 1$, then two subcritical endemic equilibria occurs when $R_0 < 1$.

A special case of model (4.1.1) was considered by [19] for more complete results. The special case examined is such that individuals from the first group are completely safe in their sexual practices (i.e $\alpha_1 = 0$). In addition, the infected migrate to the safe group from the unsafe group, and this migration must not be in the opposite direction, and no migration for susceptible individuals (that is $D_{12} = C_{21} = C_{12} = 0$). This special case of migration is exclusively critical since individuals infected with HIV find out through receiving a positive HIV test that they are infected and adopt safer sexual practices accordingly. The new model equations are:

$$\begin{aligned}\frac{dx(t)}{dt} &= \lambda_1 - \mu x(t), \\ \frac{dy(t)}{dt} &= D_{21}z_1(t) - (\mu + \sigma)y(t), \\ \frac{dz(t)}{dt} &= \lambda_2 - \beta c \alpha_2^2 \frac{z_1(t)z(t)}{T(t)} - \mu z(t), \text{ and} \\ \frac{dz_1(t)}{dt} &= \beta c \alpha_2^2 \frac{z_1(t)z(t)}{T(t)} - (\mu + \sigma + D_{21})z_1(t),\end{aligned}\tag{4.1.2}$$

where $T(t) = x(t) + y(t) + z(t) + z_1(t)$ with suitable initial conditions. For this model, people in the safe group (that is $\alpha_1 = 0$) have no contribution to the spread of HIV-AIDS. The resulting model is therefore, completely a one-group system with regard to the spread of the virus.

We will first review some results obtained by Greenhalgh *et al.* [29] from their deterministic model before stochasticity is introduced to the model.

A study on the long-term behaviour of the spread of disease in the two-group models (4.1.1) and (4.1.2) is done. In particular, conditions for the endurance or eradication of HIV in these models are examined. By studying the behaviour of these simpler models, one can have an idea as to how the generalised heterogeneous model and multi-group model may behave. For example, if one discovers that the basic reproduction number governs whether or not the disease spreads or will die out in the two-group models, then one might reasonably expect this also to be the case for more general models (models with multiple subpopulations).

The disease-free equilibrium for the model was found to be

$$(x^*, y^*, z^*, z_1^*) = \left(\frac{\lambda_1 + \lambda_2 \nu_{21}}{\mu + C_{12}(1 - \nu_{21})}, 0, \frac{\lambda_2 + \lambda_1 \nu_{12}}{\mu + C_{21}(1 - \nu_{12})}, 0 \right),$$

where $\nu_{ij} = \frac{C_{ij}}{\mu + C_{ij}}$, for $j, i = 1, 2$ and $j \neq i$ is defined as the ratio of the number of individuals leaving the x subpopulation that move into the z , given no infection of susceptibles. The other individuals of this group are accounted for by background mortalities (μ). The parameter ν_{ij} is

therefore important in determining the sizes of the two susceptible subpopulations at any given time. A similar ratio $\Delta_{ij} = \frac{D_{ij}}{\mu + \sigma + D_{ij}}$ for $j, i = 1, 2$, and $j \neq i$ is defined as the proportion of the number of people leaving one infected subpopulation and move into another infected subpopulation. The non-zero equilibrium quantities x^* and z^* are given in terms of recruitment rates, the migration rates between the susceptible subpopulations and background mortality. The ratios of the $x(t)$ and $z(t)$ subpopulations to the total susceptible population at this disease-free state are respectively given by

$$\Gamma = \frac{x^*}{x^* + z^*} = \frac{\Lambda + (1 - \Lambda)\nu_{21}}{\nu_{21} + \frac{1 - \nu_{21}}{1 - \nu_{12}}},$$

and

$$1 - \Gamma = \frac{z^*}{x^* + z^*} = \frac{\frac{1 - \nu_{21}}{1 - \nu_{12}} - \Lambda(1 - \nu_{21})}{\nu_{21} + \frac{1 - \nu_{21}}{1 - \nu_{12}}},$$

where $\Lambda = \frac{\lambda_1}{\lambda_1 + \lambda_2}$ is the proportion of the susceptibles who are recruited into the $x(t)$ subpopulation. An examination of the stability of the above solution is done in the theorem that follows.

$$R_0 = \frac{\beta c \alpha_1^2 \Gamma}{\mu + \sigma + D_{12}} \frac{1}{1 - \Delta_{12} \Delta_{21}} + \frac{\beta c \alpha_2^2 (1 - \Gamma)}{\mu + \sigma + D_{21}} \frac{1}{1 - \Delta_{12} \Delta_{21}} + \frac{\beta c \alpha_1 \alpha_2 (D_{12} \Gamma + D_{21} (1 - \Gamma))}{(\mu + \sigma + D_{12})(\mu + \sigma + D_{21})} \frac{1}{1 - \Delta_{12} \Delta_{21}} \text{ are first defined.}$$

and

$$R_0^* = \frac{\beta c \alpha_2^2 (1 - \Lambda)}{\mu + \sigma + D_{21}}$$

Theorem 4.2.1. [29]

The model has a unique solution for the disease-free equilibrium, that is, locally stable asymptotically if $R_0 < 1$ and whenever $R_0 > 1$, it is unstable. Furthermore, the corresponding solution of the disease-free equilibrium for the special case (model 4.1.2) is globally stable asymptotically if $R_0^ \leq 1$ and whenever $R_0^* > 1$, it is unstable.*

PROOF.

Firstly, it is shown that the solution for the disease-free equilibrium of (4.1.1) is locally stable asymptotically if $R_0 < 1$ and unstable whenever $R_0 > 1$. The RHS of (4.1.1) is denoted as (r_1, r_2, r_3, r_4) respectively, the Jacobian matrix $\mathbf{A} = \partial(r_1, r_2, r_3, r_4)/\partial(x, y, z, z_1)$ evaluated at disease-free equilibrium is

$$\mathbf{A}_0^* = \begin{bmatrix} -(\mu + C_{12}) & -\beta c \alpha_1^2 \Gamma & C_{12} & -\beta c \alpha_1 \alpha_2 \Gamma \\ 0 & \beta c \alpha_1^2 \Gamma - (\mu + \sigma + D_{12}) & 0 & D_{21} + \beta c \alpha_1 \alpha_2 \Gamma \\ C_{12} & -\beta c \alpha_1 \alpha_2 (1 - \Gamma) & -(\mu + C_{21}) & -\beta c \alpha_2^2 (1 - \Gamma) \\ 0 & \beta c \alpha_1 \alpha_2 (1 - \Gamma) + D_{12} & 0 & \beta c \alpha_2^2 (1 - \Gamma) - (\mu + \sigma + D_{21}) \end{bmatrix}.$$

Working on the characteristic equation $|\mathbf{A}_0^* - \lambda \mathbf{I}| = 0$, the characteristic polynomial may be expressed as

$$P(\lambda) = P_1(\lambda)P_2(\lambda) = (\lambda^2 + a_{11}\lambda + a_{12})(\lambda^2 + a_{21}\lambda + a_{22}),$$

where

$$\begin{aligned} a_{11} &= (\mu + C_{12}) + (\mu + C_{21}), \\ a_{12} &= (\mu + C_{12})(\mu + C_{21}) - C_{12}C_{21}, \\ a_{21} &= (\mu + \sigma + D_{12}) + (\mu + \sigma + D_{21}) - \beta c \alpha_1^2 \Gamma - \beta c \alpha_2^2 (1 - \Gamma), \quad \text{and} \\ a_{22} &= (\mu + \sigma + D_{12})(\mu + \sigma + D_{21}) - D_{12}D_{21} - \beta c \alpha_1^2 \Gamma (\mu + \sigma + D_{21}) \\ &\quad - \beta c \alpha_2^2 (1 - \Gamma)(\mu + \sigma + D_{12}) - \beta c \alpha_1 \alpha_2 \Gamma D_{12} - \beta c \alpha_1 \alpha_2 (1 - \Gamma) D_{21}. \end{aligned}$$

Conditions on $P(\lambda)$ are adjusted so that its roots have strictly negative real parts. Since $P(\lambda)$ is a product of two quadratics $P_1(\lambda)$ and $P_2(\lambda)$, the Routh-Hurwitz conditions (Willems [72]) require a_{11}, a_{12}, a_{21} and a_{22} to be strictly positive. One can clearly see that $a_{11} > 0$ and $a_{12} > 0$. Now $a_{21} > 0$ implies that $(\mu + \sigma + D_{12}) > \beta c \alpha_1^2 \Gamma$ and $(\mu + \sigma + D_{21}) > \beta c \alpha_2^2 (1 - \Gamma)$, hence, $a_{21} > 0$. It then follows that $a_{22} > 0$, is the only condition for stability of the disease-free solution, and is equivalent to $R_0 < 1$. The special case $R_0 = 1$ implies that $a_{22} = 0$, which in turn, implies that $a_{12} > 0$. So the dominant eigenvalue is zero and the stability for the equilibrium solution cannot easily be determined. Finally, for $R_0 > 1$, one obtains $a_{22} < 0$, and a single real negative root (and a single positive root) for $P_2(\lambda)$. Consequently, the disease free solution is unstable for $R_0 > 1$.

It is now shown that the result of the disease-free solution for the model (4.1.2) is globally stable asymptotically. Let $Q = [0, \infty]^4$ be the non-negative region in $\mathbb{R}^4$. If the initial condition $(x(0), y(0), z(0), z_1(0))$ are in Q then $(x(t), y(t), z(t), z_1(t))$ is in Q for every $t \geq 0$. As an inductive step, there is a need for upper bounds on $T(t)$ and $\frac{z(t)}{x(t)+z(t)}$. Since

$$\begin{aligned} \frac{dT(t)}{dt} &= \frac{d}{dt}(x(t) + y(t) + z(t) + z_1(t)) \\ &= \lambda_1 + \lambda_2 - \mu T(t) - \sigma(y(t) + z_1(t)) \end{aligned}$$

it can be deduced that $\limsup_{t \rightarrow \infty} T(t) \leq \frac{\lambda_1 + \lambda_2}{\mu}$. Using the first and third equations of (4.1.2), with $S(t) = z(t) + x(t)$ one obtains

$$\begin{aligned} \frac{d}{dt} \left(\frac{z(t)}{S(t)} \right) &= \frac{\lambda_2}{S(t)} - \beta c \alpha_2^2 \frac{z_1(t)z(t)}{T(t)S(t)} - \mu \frac{z(t)}{S(t)} - (\lambda_1 + \lambda_2) \frac{z(t)}{S^2(t)} \\ &\quad + \beta c \alpha_2^2 \frac{z_1(t)}{T(t)} \left(\frac{z_1(t)}{S(t)} \right)^2 + \mu \frac{z_1(t)}{S(t)} \\ &= \frac{1}{S(t)} \left(\lambda_2 - \beta c \alpha_2^2 \frac{z_1(t)z(t)}{T(t)} \left(1 - \frac{z(t)}{S(t)} \right) \right) - (\lambda_1 + \lambda_2) \frac{z(t)}{S(t)} \\ &\leq \frac{1}{S(t)} \left(\lambda_2 - \frac{z(t)}{S(t)} (\lambda_1 + \lambda_2) \right) \\ &\leq 0 \quad \text{for } \frac{z(t)}{S(t)} \geq 1 - \Lambda. \end{aligned}$$

This implies that $\limsup_{t \rightarrow \infty} \frac{z(t)}{S(t)} \leq 1 - \Lambda$.

Thus, for every given $\epsilon > 0$, there exists t_0 such that $\frac{z(t)}{S(t)} \leq (1 - \Lambda)(1 + \epsilon_0)$ for all $t \geq t_0$. Focus is now placed on the model equation for the progression of the virus in the $z_1(t)$ subpopulation. Let $f(t) = z_1(t)/(\mu + \sigma + D_{21})$, then for $t \geq t_0$,

$$\begin{aligned} \frac{df(t)}{dt} &= z_1(t) \left(\frac{\beta c \alpha_2^2}{\mu + \sigma + D_{21}} \frac{z(t)}{T(t)} - 1 \right) \\ &\leq z_1(t) \left(\frac{\beta c \alpha_2^2}{\mu + \sigma + D_{21}} \frac{z(t)(1 - \Lambda)}{T(t)} (1 + \epsilon_0) - 1 \right) \\ &= z_1(t) \left((1 + \epsilon_0) \frac{R_0^*}{T(t)} - 1 \right) \\ &= z_1(t) (R_0^*(1 + \epsilon_0) - 1) - R_0^*(1 + \epsilon_0) \frac{z_1(t)(z_1(t) + y(t))}{T(t)} \end{aligned}$$

The cases $R_0^* < 1$ and $R_0^* = 1$ are considered separately. For $R_0^* < 1$, choose ϵ_0 small enough so that $R_0^*(1 + \epsilon_0) - 1 = \bar{\epsilon} < 0$,

$$\begin{aligned} \frac{df(t)}{dt} &\leq \bar{\epsilon} z_1(t) \\ &= (\mu + \sigma + D_{21}) \bar{\epsilon} f(t) = \epsilon f(t), \end{aligned}$$

where $\epsilon = (\mu + \sigma + D_{21}) \bar{\epsilon} < 0$. Rearranging and integrating the last expression on $[t_0, t]$ gives $0 \leq f(t) \leq f(t_0) \exp[\epsilon(t - t_0)]$ which implies that $\lim_{t \rightarrow \infty} f(t) = 0$. Therefore, $z_1(t) \rightarrow 0$ as $t \rightarrow \infty$. Then, for $R_0^* = 1$, there is t_1 such that $T \leq 2(\lambda_1 + \lambda_2)/\mu$ for all $t \geq t_1$. Given an $\epsilon > 0$ choose ϵ_0 such that $4(\lambda_1 + \lambda_2)\epsilon_0/\mu = \epsilon$, then

$$\begin{aligned} \frac{df(t)}{dt} &= z_1(t) \left(\epsilon_0 - (1 + \epsilon_0) \frac{(y(t) + z_1(t))}{T} \right) \\ &\leq z_1(t) \left(\epsilon_0 - (1 + \epsilon_0) \frac{\mu(y(t) + z_1(t))}{2(\lambda_1 + \lambda_2)} \right), \quad \text{for } t \geq \max(t_1, t_0). \end{aligned}$$

Therefore, for $t \geq \max(t_1, t_0)$, either $y(t) + z_1(t) \leq \epsilon(1 + \epsilon_0) < \epsilon$ or

$$\frac{df(t)}{dt} \leq -\epsilon_0(\mu + \sigma + D_{21})f(t),$$

there must be $t_2 > \max(t_0, t_1)$ with $z_1(t) < \epsilon$ for $t > t_2$. Hence, $z_1(t) \rightarrow 0$ as $t \rightarrow \infty$. This implies that as $t \rightarrow \infty$, then $y(t) \rightarrow 0$, $x(t) \rightarrow \frac{\lambda_1}{\mu}$ and $z(t) \rightarrow \frac{\lambda_2}{\mu}$. This concludes the proof. $\blacksquare$

Having examined the behaviour of the disease-free solutions in the models and the conditions necessary for the disease to be eradicated, an examination of the behaviour of the models is done

when the disease spreads. Firstly

$$\begin{aligned} \Phi = & -\frac{\mu}{\mu + \sigma} \frac{1 - \Gamma}{1 - \Lambda} \left(\Lambda + \frac{\alpha_2}{\alpha_1} (1 - \Lambda) + \frac{\eta_{21}}{1 - \eta_{21}} \frac{\alpha_2}{\alpha_1} \frac{\eta_{12}}{1 - \eta_{12}} \right) \\ & - \left\{ \left(\frac{\alpha_1}{\alpha_2} (1 - \Gamma) \frac{\Lambda}{1 - \Lambda} - \Gamma \right) \left(\frac{\beta c \alpha_1^2}{\mu + \sigma + D_{12}} + \frac{\beta c \alpha_1 \alpha_2 \Delta_{12}}{\mu + \sigma + D_{21}} \right) \right. \\ & \left. \times \frac{1}{1 - \Delta_{12} \Delta_{21}} \right\} \end{aligned} \quad (4.1)$$

and

$$\varrho = 2 \frac{\alpha_2}{\alpha_1} \left(\frac{\mu + C_{12} + C_{21}}{\mu + \sigma} \right) \frac{(1 - \Gamma)^2}{(1 - \Lambda)^2} \text{ are defined.} \quad (4.2)$$

Theorem 4.2.2. [29]

In the two group model in (4.1.1),

- (i) if $R_0 > 1$, then a unique endemic equilibrium exists,
- (ii) if $R_0 \in (\sqrt{\varrho^2 + 2\varrho} - \varrho, 1)$ and $\Phi > 1 - R_0 + \sqrt{(1 - R_0)2\varrho}$, then the two endemic equilibria can occur,
- (iii) if $R_0 = 1$ and $\Phi > 0$, then a single endemic equilibrium exists.

If neither (i), (ii) nor (iii), occurs then there are no endemic equilibria. The special case model (4.1.2) has a unique endemic equilibrium and it is locally asymptotically stable whenever $R_0^* > 1$.

PROOF.

The conditions necessary for non-trivial equilibrium solutions of the model (4.1.1) are shown. It is assumed that there is disease present in the population. By setting the time derivatives in the model equations equal to zero, one solves for the subpopulation quantities at equilibrium, (x^*, y^*, z^*, z_1^*) . Then, it becomes easy to derive the relationship

$$a_1 F^{*2} + a_2 F^* + a_3 = 0, \quad (4.3)$$

where $F^* = \frac{\alpha_1 y^* + \alpha_2 z_1^*}{T^*}$ is positive,

$$\begin{aligned} a_1 = & \frac{\beta^2 c^2 \alpha_1 \alpha_2}{\mu + \sigma}, \\ a_2 = & \beta c \left\{ \frac{\mu}{\mu + \sigma} \left(\alpha_1 \Lambda + \alpha_2 (1 - \Lambda) + \alpha_1 \frac{\nu_{21}}{1 - \nu_{21}} + \alpha_2 \frac{\nu_{12}}{1 - \nu_{12}} \right) + \alpha_1 (1 - \Lambda) + \Lambda \alpha_2 \right. \\ & - \frac{\beta c \alpha_1^2 \alpha_2 \Lambda}{\mu + \sigma + D_{12}} \frac{1}{1 - \Delta_{12} \Delta_{21}} - \frac{\beta c \alpha_1 \alpha_2^2 (1 - \Lambda)}{\mu + \sigma + D_{21}} \frac{1}{1 - \Delta_{12} \Delta_{21}} \\ & \left. - \frac{\beta c \alpha_1 \alpha_2^2 \Lambda}{\mu + \sigma + D_{21}} \frac{\Delta_{12}}{1 - \Delta_{12} \Delta_{21}} - \frac{\beta c \alpha_1^2 \alpha_2 (1 - \Lambda)}{\mu + \sigma + D_{12}} \frac{\Delta_{21}}{1 - \Delta_{12} \Delta_{21}} \right\}, \quad \text{and} \\ a_3 = & \frac{\mu + C_{12} + C_{21}}{1 - \Delta_{12} \Delta_{21}} \left\{ 1 - \Delta_{12} \Delta_{21} - \frac{\beta c \alpha_1^2 \Gamma}{\mu + \sigma + D_{12}} - \frac{\beta c \alpha_2^2 (1 - \Gamma)}{\mu + \sigma + D_{21}} \right. \\ & \left. - \frac{\beta c \alpha_1 \alpha_2 (D_{12} \Gamma + D_{21} (1 - \Gamma))}{(\mu + \sigma + D_{12})(\mu + \sigma + D_{21})} \right\}. \end{aligned}$$

It can easily be seen that $a_1 > 0$, but signs of a_3 and a_2 are not fixed (the problem of determining the existence of endemic equilibrium solutions using (4.3)). A definition of a quadratic function $y = q(F^*)$ is given, whose coefficients are in terms of the model parameters, and its roots are the equilibrium solutions ($y = q(F^*) = 0$ and $F^* = \hat{F}$). With $y = q(F^*) = a_1 F^{*2} + a_2 F^* + a_3$ as given above in (4.3) one obtains

$$\hat{F} = \frac{-a_2 \pm \sqrt{a_2^2 - 4a_1a_3}}{2a_1}.$$

Therefore, the possibilities are zero, one or two endemic equilibrium solutions. The two existing conditions for positive real roots in terms of a_1, a_2 and a_3 are as follows:

- (i) one strictly positive real root (F^*): when $a_3 < 0$ or $a_3 = 0$ and $a_2 < 0$;
- (ii) two distinct strictly positive real roots (F^*): when $a_3 > 0$ or $a_2 < 0$ and $a_2^2 > 4a_1a_3$.

For different types and direction of migration of individuals and different risk levels $0 \leq \alpha_1 < \alpha_2 \leq 1$, the coefficients a_1, a_2 and a_3 change accordingly. One may therefore investigate the influence of these characteristics on the conditions for persistence. The following parametrisation is useful:

$$a_1 = \frac{\beta^2 c^2 \alpha_1 \alpha_2}{\mu + \sigma}, \quad (4.4)$$

$$a_2 = \beta c \alpha_1 \frac{1 - \Lambda}{1 - \Gamma} (1 - R_0 - \Phi), \quad (4.5)$$

$$a_3 = (\mu + C_{12} + C_{21})(1 - R_0) \quad (4.6)$$

The coefficient a_3 is given by some model parameters multiplied by $1 - R_0$. The parameter Φ denotes terms that remain when a_3 is substituted into a_2 (there are some infectivity terms in common). On inspection, it seems that Φ may either be positive or negative. It is possible to set the following implication:

$$R_0 < 1 \Rightarrow \left(\frac{\beta c \alpha_1^2}{\mu + \sigma + D_{12}} + \frac{\beta c \alpha_1 \alpha_2 \Delta_{12}}{\mu + \sigma + D_{21}} \right) \frac{1}{1 - \Delta_{12} \Delta_{21}} < 1.$$

It is straightforward that

$$\frac{a_2}{a_1} (1 - \Gamma) \frac{\Lambda}{1 - \Lambda} - \Gamma > -1,$$

and it follows that $R_0 < 1$ ensures that $\Phi < 1$. We now define

$$\Upsilon^* = \frac{\beta c \alpha_2}{\mu + \sigma} \left(\frac{1 - \Gamma}{1 - \Lambda} \right) F^* \text{ is now defined, and}$$

the relationship in (4.3) may be written as

$$\Upsilon^{*2} + (1 - R_0 - \Phi) \Upsilon^* + \frac{1}{2} \varrho (1 - R_0) = 0, \quad (4.7)$$

where

$$\begin{aligned} \varrho &= 2 \frac{a_2}{a_1} \left(\frac{\mu + C_{12} + C_{21}}{\mu + \sigma} \right) \frac{(1 - \Gamma)^2}{(1 - \Lambda)^2} \\ &= 2 \frac{a_2}{a_1} \frac{\mu}{\mu + \sigma} \left(1 + \frac{\nu_{12}}{1 - \nu_{12}} + \frac{\nu_{21}}{1 - \nu_{21}} \right) \frac{(1 - \Gamma)^2}{(1 - \Lambda)^2}. \end{aligned}$$

Using condition (i) for existence of positive roots and (4.6), one obtains $R_0 > 1$, or $R_0 = 1$ and $\Phi > 0$, as a condition of existence for a single endemic equilibrium solution. From condition (ii), for existence of positive roots and (4.5), (4.6) and the knowledge that $R_0 < 1$ implies $\Phi < 1$, the values of Φ are required in the range

$$1 - R_0 < \Phi < 1 \quad \text{or} \quad 0 < R_0 < 1 \quad (4.8)$$

for two endemic equilibria to exist. Further examination of Φ in (4.1) reveals that there are stronger upper bounds on Φ than those of unity. Therefore, the parameters R_0 and Φ together in (4.8), determine a necessary but not sufficient region for the parameter space of two endemic equilibria. A development of the condition $a_2^2 > 4a_1a_3$ in condition (ii) (for positive real roots) is done in terms of the parameters R_0 and Φ . From (4.7) this condition is equivalent to the quadratic expression

$$\Phi^2 - 2(1 - R_0)\Phi + (1 - R_0)(1 - R_0 - 2\varrho) > 0, \quad (4.9)$$

which is satisfied for $\Phi > \Phi_c = 1 - R_0 + \sqrt{2\varrho(1 - R_0)}$. This critical value Φ_c is one of the roots of the quadratic expression in (4.9); the other (smaller) root is less than $1 - R_0$, and so does not satisfy the condition in (4.8). One must now also satisfy $\Phi_c < 1$, in order to agree with (4.8). That is,

$$1 > R_0 > \sqrt{\varrho^2 + 2\varrho} - \varrho = R_0^{\min} > 0 \text{ is required}$$

for $\varrho > 0$. Thus, $R_0^{\min}$ is the lower bound for the minimum value of R_0 for which two endemic solutions are permissible. This completes the proof for the first part of the theorem.

Next, it is proven that model (4.1.2) has a unique solution for the endemic equilibrium and it is locally stable asymptotically for $R_0^* > 1$. It is first assumed that $R_0^* > 1$. At equilibrium,

$$x^* = \frac{\lambda_1}{\mu}; \quad (4.10)$$

$$y^* = \frac{D_{21}}{\mu + \sigma} z_1^*; \quad (4.11)$$

$$0 = \lambda_2 - \mu z^* - \beta c \alpha_2^2 \frac{z_1^*}{T^*} z^*; \quad \text{and} \quad (4.12)$$

$$0 = \beta c \alpha_2^2 \frac{z_1^*}{T^*} z^* - (\mu + \sigma + D_{21}) z_1^*. \quad (4.13)$$

where $T^* = x^* + y^* + z^* + z_1^*$. In addition, combining (4.12) and (4.13) gives

$$z^* = \frac{\lambda_2}{\mu} - \frac{\mu + \sigma + D_{21}}{\mu} z_1^* \quad (4.14)$$

It should be noted that $z_1^* = 0$ implies $y^* = 0$. Now assuming $z_1^* \neq 0$, from (4.13), $\beta c \alpha_2^2 \frac{z^*}{T^*} = \mu + \sigma + D_{21}$. Alternatively, one can write this as

$$\frac{z^*}{x^* + y^* + z^* + z_1^*} = \frac{\mu + \sigma + D_{21}}{\beta c \alpha_2^2}, \quad (4.15)$$

$$< \frac{1}{1 - \Lambda} \frac{\mu + \sigma + D_{21}}{\beta c \alpha_2^2} = \frac{1}{R_0^*} < 1. \quad (4.16)$$

Using the relationships in (4.10), (4.11) and (4.14), the left hand side of (4.15) may be expressed exclusively in terms of z_1^* . One obtains

$$\frac{\lambda_2 - (\mu + \sigma + D_{21})z_1^*}{\lambda_1 + \lambda_2 - \frac{\sigma}{\mu + \sigma}(\mu + \sigma + D_{21})z_1^*} = \frac{\mu + \sigma + D_{21}}{\beta c \alpha_2^2}. \quad (4.17)$$

In order to prove existence, the left hand side of (4.17) is treated as a function in z_1^* , say $k(z_1^*)$. Differentiating $k(z_1^*)$ with respect to z_1^* one obtains

$$\frac{dk(z_1^*)}{dz_1^*} = \frac{-(\lambda_1 + \lambda_2 - \frac{\sigma}{\mu + \sigma})(\mu + \sigma + D_{21})}{(\lambda_1 + \lambda_2 - \frac{\sigma}{\mu + \sigma}(\mu + \sigma + D_{21})z_1^*)^2},$$

and the function has been shown to be monotone decreasing with respect to z_1^* . Limiting values of $k(z_1^*)$ in the positive quadrant are

$$k(0) = 1 - \Lambda \text{ and } k\left(\frac{\lambda_2}{\mu + \sigma + D_{21}}\right) = 0.$$

Now, from (4.15)

$$\begin{aligned} \frac{z^*}{x^* + y^* + z^* + z_1^*} &= \frac{\mu + \sigma + D_{21}}{\beta c \alpha_2^2} < \frac{1}{1 - \Lambda} \frac{\mu + \sigma + D_{21}}{\beta c \alpha_2^2} < 1, \\ &\Rightarrow \frac{\mu + \sigma + D_{21}}{\beta c \alpha_2^2} < 1 - \Lambda = k(0). \end{aligned}$$

Since $k(z_1^*)$ is monotone decreasing, the requirement that $R_0^* > 1$ guarantees the existence of a unique quantity $z_1^* = \hat{z}_1 > 0 \in (0, \lambda_2/(\mu + \sigma + D_{21}))$ such that $k(\hat{z}_1) = (\mu + \sigma + D_{21})/\beta c \alpha_2^2 \in (0, 1 - \Lambda)$. It can easily be shown that x^* , z^* and y^* have unique positive values. Hence, it has been established that a unique endemic equilibrium exists for model (4.1.2). It is now demonstrated that it is locally stable asymptotically.

Linearising the system of (4.1.2) about the endemic equilibrium (x^*, y^*, z^*, z_1^*) , the corresponding Jacobian matrix evaluated at this solution is

$$\mathbf{A}_e = \begin{bmatrix} -\mu & 0 & 0 & 0 \\ 0 & -(\mu + \sigma) & 0 & 0 \\ \beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}} & \beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}} & -\mu - \beta c \alpha_2^2 \frac{z_1^* (T^* - z^*)}{T^{*2}} & -\beta c \alpha_2^2 \frac{z^* (T^* - z_1^*)}{T^{*2}} \\ -\beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}} & -\beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}} & \beta c \alpha_2^2 \frac{z_1^* (T^* - z^*)}{T^{*2}} & \beta c \alpha_2^2 \frac{z^* (T^* - z_1^*)}{T^{*2}} - (\mu + \sigma + D_{21}) \end{bmatrix},$$

determining the characteristic equation of $\mathbf{A}_e$, $\det(\mathbf{A}_e - \lambda \mathbf{I}) = 0$, where λ are eigenvalues of $\mathbf{A}_e$, and $\mathbf{I}$ is a 4×4 identity matrix. One immediately obtains $\lambda = -\mu$ as one of the eigenvalues. The other eigenvalues are specified by the cubic polynomial $\lambda^3 + c_1 \lambda^2 + c_2 \lambda + c_3 = 0$, where

$$c_1 = \frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu + (\mu + \sigma), \quad (4.18)$$

$$c_2 = (\mu + \sigma) \frac{\beta c \alpha_2^2 z_1^*}{T^*} + (\mu + \sigma + D_{21}) \frac{\beta c \alpha_2^2 z_1^*}{T^*} \left(1 - \frac{z^*}{T^*}\right) + \mu \beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}} \quad (4.19)$$

$$+ \mu(\mu + \sigma) + D_{21} \beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}}, \quad (4.20)$$

$$c_3 = (\mu + \sigma)(\mu + \sigma + D_{21}) \frac{\beta c \alpha_2^2 z_1^*}{T^*} \left(1 - \frac{z^*}{T^*}\right) + \mu(\mu + \sigma + D_{21}) \beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}}. \quad (4.21)$$

According to the Routh-Hurwitz criterion, one obtains $c_1 > 0$, $c_3 > 0$ and $c_1 c_2 > c_3$. Clearly, $c_1 > 0$ and $c_3 > 0$. It remains to show that $c_1 c_2 > c_3$. Tidying up (4.20) one obtains

$$c_2 = (\mu + \sigma) \left(\frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu \right) + (\mu + D_{21}) \frac{\beta c \alpha_2^2 z_1^*}{T^*} + \sigma \frac{\beta c \alpha_2^2 z_1^*}{T^*} \left(1 - \frac{z^*}{T^*} \right).$$

Writing c_1 in the form

$$c_1 = (\mu + \sigma) + \left(\frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu \right),$$

after some straight forward cancelation of terms one obtains

$$\begin{aligned} c_1 c_2 - c_3 &= (\mu + \sigma) \left(\frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu \right)^2 + (\mu + D_{21}) \left(\frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu \right) \frac{\beta c \alpha_2^2 z_1^*}{T^*} \\ &\quad + \sigma \left(\frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu \right) \frac{\beta c \alpha_2^2 z_1^*}{T^*} \left(1 - \frac{z^*}{T^*} \right) + (\mu + \sigma)^2 \left(\frac{\beta c \alpha_2^2 z_1^*}{T^*} + \mu \right) \\ &\quad + \sigma D_{21} \beta c \alpha_2^2 \frac{z^* z_1^*}{T^{*2}}. \end{aligned}$$

Thus, $c_1 c_2 > c_3$ and the unique endemic equilibrium solution is locally stable asymptotically. ■

In this study, the case $\alpha_1 \alpha_2 = 0$ (i.e when there is interrelations between members of the different groups, one of them will always insist on using a condom) is considered. This means that safe sex is practiced whenever a person from the first group has sexual relations with a person from the second group. Also, ($C_{12} = C_{21} = D_{12} = 0$) is assumed. The resulting simplified model enables us to study in detail, the effect of the model parameters on a long term behaviour of the disease, especially the influence of the risk levels and migration rates. The model (4.2.1) becomes

$$\begin{aligned} \frac{dx(t)}{dt} &= \lambda_1 - \mu x(t) - \beta c \alpha_1^2 \frac{x(t)y(t)}{T(t)}, \\ \frac{dy(t)}{dt} &= \beta c \alpha_1^2 \frac{x(t)y(t)}{T(t)} - (\mu + \sigma)y(t) + D_{21}z_1(t), \\ \frac{dz(t)}{dt} &= \lambda_2 - \beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} - \mu z(t), \text{ and} \\ \frac{dz_1(t)}{dt} &= \beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} - (\mu + \sigma + D_{21})z_1(t). \end{aligned} \tag{4.1.3}$$

The equilibrium and stability analysis of this deterministic model will be the same as (4.1.1). In the results of (4.1.1), $\alpha_1 \alpha_2 = 0$ and $C_{12} = C_{21} = D_{12} = 0$ are substituted in order to obtain the results for this model. The interest is to study how environmental noise affects this system of differential equations.

4.3. Stochastic model

The parameter σ is defined as per capita rate of individuals infected develop AIDS. Essentially, σ can be estimated by adding its average value and the error term assumed to be normally distributed,

so that the probability for an event in $[t, t + \Delta t)$ of a single infected person is approximately $\mathcal{N}(\sigma\Delta t, \sigma_1^2\Delta t) + o(\Delta t)$. Therefore, σ is replaced by $\sigma + \sigma_1\dot{W}(t)$, where $\dot{W}(t)$ is the white noise and $W(t)$ is a Wiener process. $\sigma_1 > 0$ (and is smaller than σ) is the noise intensity. This is a usual approach for stochastic modeling in population dynamics. In addition, this introduces stochasticity to the system and allows one to obtain analytical results for the model (see [3, 10, 26, 48]).

The new system of stochastic differential equations is now given as follows:

$$\frac{dx(t)}{dt} = \lambda_1 - \mu x(t) - \beta c \alpha_1^2 \frac{x(t)y(t)}{T(t)}, \quad (4.22)$$

$$dy(t) = \left[\beta c \alpha_1^2 \frac{x(t)y(t)}{T(t)} - (\mu + \sigma)y(t) + D_{21}z_1(t) \right] dt - \sigma_1 y(t) dW(t), \quad (4.23)$$

$$\frac{dz(t)}{dt} = \lambda_2 - \beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} - \mu z(t), \text{ and} \quad (4.24)$$

$$dz_1(t) = \left[\beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} - (\mu + \sigma + D_{21})z_1(t) \right] dt - \sigma_1 z_1(t) dW(t), \quad (4.25)$$

with suitable initial conditions. Since the stochasticity is introduced through parameter σ , the noise term is the same in both Equations (4.23) and (4.25). In the following sections, the analytical results for the new model are established.

4.3.1 Non-negative solutions

The usual complete probability space is denoted by $(\Omega, \mathcal{F}, P)$, with the filtration $\{\mathcal{F}_t\}_{t \geq 0}$ which satisfies the usual conditions (i.e. increasing and continuous on the right, and $\mathcal{F}_0$ has all P -null sets). $W(t)$ is considered to be a one-dimensional Wiener process (or Brownian motion) defined on a complete probability space $(\Omega, \mathcal{F}, P)$. In addition, let $\mathbb{R}_{++}^4 = \{x \in \mathbb{R}^4 : x_i > 0 \text{ for every } 1 \leq i \leq 4 \text{ whenever } x = (x_1, x_2, x_3, x_4)\}$ and $\mathbb{R}_+^4 = \{y \in \mathbb{R}^4 : y_i \geq 0 \text{ for all } 1 \leq i \leq 4 \text{ whenever } y = (y_1, y_2, y_3, y_4)\}$ and let the solution to the model be given by $\tilde{x}(t) = (x(t), y(t), z(t), z_1(t))^T$.

Lemma 3.3.1 is used to prove the following theorem.

Theorem 4.3.1. *Let $\sigma_1, \mu, \sigma, D_{21}, \beta$ be real positive constants and $0 < \alpha_1, \alpha_2 < 1$. Then, for every initial condition $\tilde{x}_0 \in \mathbb{R}_{++}^4$, there is a unique solution $\tilde{x}(t)$ to Equations (4.22) – (4.25) for every $t \geq 0$ and this solution will always be in $\mathbb{R}_{++}^4$ with probability 1, i.e $\tilde{x}(t) \in \mathbb{R}_{++}^4$ almost surely, for every $t \geq 0$.*

PROOF.

Considering that the coefficients for the equations are locally Lipschitz continuous, then for every given initial value $\tilde{x}_0 \in \mathbb{R}_{++}^4$ there is a unique local solution $\tilde{x}(t)$ for every $t \in [0, \tau_e)$, and τ_e is an explosion time (see [2, 25]). One needs to show that $\tau_e = \infty$ a.s for the solution to be global. A constant $k_0 \geq 0$ is chosen which is large enough so that all the components of $\tilde{x}_0$ are in the interval $[\frac{1}{k_0}, k_0]$. For every integer $k \geq k_0$, the stopping time

$$\tau_k = \inf\{t \in [0, \tau_e) : \text{at least one of } x(t), y(t), z(t) \text{ or } z_1(t) \notin (1/k, k)\} \text{ is defined,}$$

throughout the study $\inf \emptyset = \infty$ ($\emptyset$ is the empty set). Hence, as $k \rightarrow \infty$, τ_k is also increasing. Let $\tau_\infty = \lim_{k \rightarrow \infty} \tau_k$, from which one obtains $\tau_\infty \leq \tau_e$. If it is proven that $\tau_\infty = \infty$ almost surely, then $\tau_e = \infty$ and $\tilde{x}(t) \in \mathbb{R}_{++}^4$ almost surely for every $t \geq 0$. This means that to complete the proof, all one needs to show is that $\tau_\infty = \infty$ a.s. If one supposes that the statement is untrue, then there are some constants $T > 0$ and $\varepsilon \in (0, 1)$ such that

$$P\{\tau_\infty \leq T\} > \varepsilon.$$

Therefore, there is an integer $k_1 \geq k_0$ such that,

$$P\{\tau_k \leq T\} \geq \varepsilon \quad \text{for all } k \geq k_1. \quad (4.26)$$

Let $U : \mathbb{R}_{++}^4 \rightarrow \mathbb{R}_{++}^4$ be a C^2 -function defined by

$$U(t) = [1 + x - \log(x)] + [1 + y - \log(y)] + [1 + z - \log(z)] + [1 + z_1 - \log(z_1)].$$

Non-negativity of $U(t)$ is seen from $1 + x - \log x \geq 0 \quad \forall x > 0$. Using Itô's formula, one obtains

$$\begin{aligned} dU(\tilde{x}) &= \left(1 - \frac{1}{x}\right) \left(\lambda_1 - \mu x - \beta c \alpha_1^2 \frac{xy}{T(t)}\right) dt + \left[\left(1 - \frac{1}{y}\right) D_{21} z_1 - (\mu + \sigma)y + \beta c \alpha_1^2 \frac{xy}{T(t)} + \frac{1}{2} \sigma_1^2\right] dt \\ &\quad + \left[\left(1 - \frac{1}{z_1}\right) \left(\beta c \alpha_2^2 \frac{zz_1}{T(t)} - (\mu + \sigma + D_{21})z_1\right) + \frac{1}{2} \sigma_1^2\right] dt + \left(1 - \frac{1}{z}\right) \left(\lambda_2 - \mu z - \beta c \alpha_2^2 \frac{zz_1}{T(t)}\right) dt \\ &\quad + (1 - y)\sigma_1 dW(t) + (1 - z_1)\sigma_1 dW(t) \\ &\leq \left(\lambda_1 \left(1 - \frac{1}{x}\right) + \mu + \beta c \alpha_1^2 \frac{y}{T(t)}\right) dt + \left[\left(1 - \frac{1}{y}\right) (D_{21} z_1 - (\mu + \sigma)y + \beta c \alpha_1^2 \frac{xy}{T(t)}) + \frac{1}{2} \sigma_1^2\right] dt \\ &\quad + (1 - y)\sigma_1 dW(t) + \left(\lambda_2 \left(1 - \frac{1}{z}\right) + \mu + \beta c \alpha_2^2 \frac{z_1}{T(t)}\right) dt + \left(\beta c \alpha_2^2 \frac{zz_1}{T(t)} + \mu + \sigma + D_{21} + \frac{1}{2} \sigma_1^2\right) dt \\ &\quad + (1 - z_1)\sigma_1 dW(t) \\ &\leq \left[4\mu + 2\sigma + D_{21} + \beta c \alpha_1^2 + \beta c \alpha_2^2 + \sigma_1^2 + \left(1 - \frac{1}{x}\right) \lambda_1 + \left(1 - \frac{1}{y}\right) D_{21} z_1 + \left(1 - \frac{1}{z}\right) \lambda_2\right] dt \\ &\quad + (2 - y - z_1)\sigma_1 dW(t). \end{aligned}$$

Hence,

$$dU(\tilde{x}(t)) \leq (r_1 + \lambda_1 + \lambda_2 + D_{21} z_1) dt + (2 - y - z_1)\sigma_1 dW(t),$$

$$\text{where } r_1 = 4\mu + 2\sigma + D_{21} + \beta c \alpha_1^2 + \beta c \alpha_2^2 + \sigma_1^2.$$

Using lemma 3.3.1 and how U has been defined, $z_1 \leq 2U(\tilde{x}) - (4 - 2\log 2) \Rightarrow z_1 \leq 2U(\tilde{x})$.

Therefore, one obtains

$$\begin{aligned} dU(\tilde{x}(t)) &\leq (r_1 + \lambda_1 + \lambda_2 + 2D_{21}U(\tilde{x})) dt + (2 - y - z_1)\sigma_1 dW(t) \\ &\leq r_2(1 + U(\tilde{x})) dt + (2 - y - z_1)\sigma_1 dW(t), \end{aligned}$$

where $r_2 = \max\{r_1 + \lambda_1 + \lambda_2, 2D_{21}\}$. Hence, if $t_1 \leq T$,

$$\int_0^{\tau_k \wedge t_1} dU(\tilde{x}(t)) \leq \int_0^{\tau_k \wedge t_1} r_2(1 + U(\tilde{x})) dt + \int_0^{\tau_k \wedge t_1} (2 - y - z_1)\sigma_1 dW(t)$$

which implies that

$$EU(\tilde{x}(\tau_k \wedge t_1)) \leq U(\tilde{x}_0) + E \int_0^{\tau_k \wedge t_1} r_2(1 + U(\tilde{x}(t))) dt,$$

The expectation of third integral is zero according to theorem 2.2.3

$$\begin{aligned}
&\leq U(\tilde{x}_0) + r_2 t_1 + r_2 E \int_0^{\tau_k \wedge t_1} U(\tilde{x}(t)) dt \\
&\leq U(\tilde{x}_0) + r_2 T + r_2 E \int_0^{t_1} U(\tilde{x}(\tau_k \wedge t)) dt \\
&\leq U(\tilde{x}_0) + r_2 T + r_2 \int_0^{t_1} EU(\tilde{x}(\tau_k \wedge t)) dt
\end{aligned}$$

Following Gronwall inequality (lemma 2.3.8),

$$f = r_2, \phi = EU(\tilde{x}(\tau_k \wedge t)) \text{ and } r_0 = U(\tilde{x}_0) + r_2 T$$

Therefore, one obtains

$$EU(\tilde{x}(\tau_k \wedge T)) \leq (U(\tilde{x}_0) + r_2 T)e^{r_2 T} = r_3. \quad (4.27)$$

Let $\Delta_k = \{\tau_k \leq T\}$ for all $k \geq k_1$ and from equation (4.26), then $P(\Delta_k) \geq \varepsilon$. For every $\omega \in \Delta_k$, at least one of $z_1(\tau_k, \omega)$, $z(\tau_k, \omega)$, $y(\tau_k, \omega)$ or $x(\tau_k, \omega)$ will be equal to either $1/k$ or k . Therefore, $U(\tilde{x}(\tau_k, \omega))$ is not less than either $1 + 1/k - \log(1/k) = 1 + 1/k + \log(k)$ or $1 + k - \log(k)$.

Therefore,

$$U(\tilde{x}(\tau_k, \omega)) \geq 1 + 1/k + \log(k) \wedge 1 + k - \log(k).$$

From (4.26) and (4.27), it follows that

$$\begin{aligned}
(U(\tilde{x}_0) + r_2 T)e^{r_2 T} &\geq E[1_{\Delta_k}(\omega)U(\tilde{x}(\tau_k, \omega))] \\
&\geq \varepsilon([1 + 1/k + \log(k)] \wedge [1 + k - \log(k)]),
\end{aligned}$$

where 1_{Δ_k} is an indicator function on Δ_k . If one lets $k \rightarrow \infty$ it will lead to a contradiction, that is, $\infty > r_3 = \infty$. It can therefore be concluded that, $\tau_\infty = \infty$ almost surely. This concludes the proof. $\blacksquare$

Since some components of the initial conditions can be zero, it will be interesting if one considered what will happen if this is to occur, i.e. $\tilde{x}_0 \in \mathbb{R}_+^4$.

Theorem 4.3.2. *Let $\sigma_1, \mu, \sigma, D_{21}, \beta$ be real positive numbers and $0 \leq \alpha_1, \alpha_2 \leq 1$. Therefore, for every initial value $\tilde{x}_0 \in \mathbb{R}_+^4$, the solution of equations (4.22) – (4.25) will be in $\mathbb{R}_+^4$ almost surely, i.e., $\tilde{x}(t) \in \mathbb{R}_+^4$ with probability 1 for every $t \geq 0$.*

PROOF.

There are four cases to consider:

- (i) $y(0) = 0, z_1(0) = 0, x(0) \geq 0, z(0) \geq 0$;

Whenever $y(0) = z_1(0) = 0$, one gets $y(t) = z_1(t) = 0$ for every $t \geq 0$ a.s. Using this result and equations (4.22) and (4.24), one obtains $x(t) \geq 0$ and $z(t) \geq 0$.

(ii) $x(0) > 0, z_1(0) = 0, y(0) \geq 0, z(0) \geq 0$;

If $z_1(0) = 0$ then $z_1(t) = 0$ for every $t \geq 0$ a.s. From (4.23) and if one lets $Y = \log y(t)$, and using Itô's formula, one obtains,

$$\begin{aligned} d \log y(t) &= \left(D_{21} \frac{z_1(t)}{y(t)} - (\mu + \sigma) + \beta c \alpha_1^2 \frac{x(t)}{T(t)} - \frac{\sigma_1^2}{2} \right) dt - \sigma_1 dW(t) \\ &= \left[\beta c \alpha_1^2 \frac{x(t)}{T(t)} - (\mu + \sigma + \frac{\sigma_1^2}{2}) \right] dt - \sigma_1 dW(t) \\ &\leq \left[\beta c \alpha_1^2 - (\mu + \sigma + \frac{\sigma_1^2}{2}) \right] dt - \sigma_1 dW(t) \\ &\Rightarrow y(t) \leq y(0) \exp \left[\left(\beta c \alpha_1^2 - (\sigma + \mu + \frac{\sigma_1^2}{2}) \right) t - \sigma_1 W(t) \right]. \end{aligned}$$

Hence, for every $t \geq 0$, $y(t) > 0$ a.s. Just like in (i) $x(t) \geq 0$, and $z(t) \geq 0$.

(iii) $x(t) \geq 0, z(t) \geq 0, y(0) = 0, z_1(0) > 0$;

From (4.22), one can write

$$\frac{dx(t)}{dt} \geq \lambda_1 - (\beta c \alpha_1^2 + \mu)x(t).$$

From this, one gets

$$x(t) \geq x(0)e^{-(\beta c \alpha_1^2 + \mu)t} + \frac{\lambda_1}{\beta c \alpha_1^2 + \mu} \left(1 - e^{-(\beta c \alpha_1^2 + \mu)t} \right).$$

Hence, $x(\Delta t) > 0$ for positive and small Δt . Also, if $D_{21} > 0$, then $y(\Delta t) = D_{21}z_1(0)\Delta t + o(\Delta t)$ a.s.

Similarly, from (4.24), one gets

$$\begin{aligned} \frac{dz(t)}{dt} &\geq \lambda_2 - \beta c \alpha_2^2 z(t) - \mu z(t) \\ \Rightarrow z(t) &\geq z(0)e^{-(\beta c \alpha_2^2 + \mu)t} + \frac{\lambda_2}{\beta c \alpha_2^2 + \mu} \left(1 - e^{-(\beta c \alpha_2^2 + \mu)t} \right). \end{aligned}$$

Hence, $z(t) > 0$ for Δt small and positive.

$$\frac{dz_1(t)}{dt} = \beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} - (\mu + \sigma + D_{21})z_1(t) - \sigma_1 z_1(t) \dot{W}(t) \text{ is now considered.}$$

Let $X = \log z_1(t)$, then using Itô's formula, one gets

$$\begin{aligned} d \log z_1(t) &= \left(\beta c \alpha_2^2 \frac{z(t)}{T(t)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) dt - \sigma_1 dW(t) \\ \log z_1(t) &= \int_0^t \left(\beta c \alpha_2^2 \frac{z(s)}{T(s)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) ds - \int_0^t \sigma_1 dW(s) + C \\ \Rightarrow z_1(t) &= C_1 \exp \left[\int_0^t \left(\beta c \alpha_2^2 \frac{z(s)}{T(s)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) ds - \int_0^t \sigma_1 dW(s) \right], \end{aligned}$$

so, $z_1(\Delta t) > 0$ a.s for a positive small Δt . When one changes the origin of time to a small positive Δt and following theorem 4.3.1, one obtains $x(t), y(t), z(t), z_1(t) > 0$ a.s.

(iv) $z(0) \geq 0, x(0) \geq 0, z_1(0) > 0, y(0) > 0$;

If one has to change the starting time to a positive small Δt , then without loss of generality, it can be concluded that $z(0) > 0, x(0) > 0$ and following theorem 4.3.1, one gets $x(t), y(t), z(t), z_1(t) > 0$ almost surely.



4.3.2 Asymptotic behaviour and stability

The model of differential equations currently being studied is partly deterministic and partly stochastic. The equations of the system are analysed individually in the next theorem. A study of the asymptotic behaviour of variables in the model is done. A lemma (see [46]) is first stated to help prove the theorem.

Lemma 4.3.3. [46]

Let $F(t)$ and $V(t)$ be continuous and increasing adapted processes for all $t \geq 0$ and $F(0) = V(0) = 0$ almost surely. A real valued continuous local martingale $A(t)$ is considered such that $A(0) = 0$ a.s. and let ξ be a non-negative $\mathcal{F}_0$ -measurable random variable and $E(\xi) < \infty$.

$Y(t) = \xi + F(t) - V(t) + A(t)$ for $t \geq 0$ is defined such that

if $Y(t) \geq 0$, then

$$\left\{ \lim_{t \rightarrow \infty} A(t) < \infty \right\} \subset \left\{ \lim_{t \rightarrow \infty} F(t) < \infty \right\} \cap \left\{ \lim_{t \rightarrow \infty} V(t) < \infty \right\} \text{ a.s.,}$$

where $D \subset E$ almost surely means $P(D \cap E^c) = 0$. If $\lim_{t \rightarrow \infty} F(t) < \infty$ a.s., then for almost every $\omega \in \Omega$

$$\lim_{t \rightarrow \infty} Y(t, \omega) < \infty, \quad \lim_{t \rightarrow \infty} V(t, \omega) < \infty$$

and

$$\lim_{t \rightarrow \infty} A(t, \omega) < \infty \quad \text{exist.}$$

To prove the next theorem, let $R_1 = \frac{\beta c \alpha_2^2 \frac{\lambda_2}{\lambda_1 + \lambda_2}}{\mu + \sigma + D_{21} + \sigma_1^2/2}$.

Theorem 4.3.4. The stochastic differential equations (4.22)–(4.25) is globally stable asymptotically whenever $R_1 < 1$ for every initial value $\tilde{x}_0 \in \mathbb{R}_+^4$, it tends to the equilibrium position of $(\frac{\lambda_1}{\mu}, 0, \frac{\lambda_2}{\mu}, 0)$, asymptotically with probability 1.

PROOF.

First, the infected individuals in the two groups are considered, that is, $y(t)$ and $z_1(t)$. There is a need to find upper bounds for $\frac{x(t)}{T(t)}$ and $\frac{z(t)}{T(t)}$ before we can proceed. From (4.22), one obtains

$$\frac{dx(t)}{dt} \leq \lambda_1 - \mu x(t).$$

Solving, one gets

$$x(t) \leq x(0)e^{-\mu t} + \frac{\lambda_1}{\mu} (1 - e^{-\mu t})$$

$$\lim_{t \rightarrow \infty} x(t) \leq \frac{\lambda_1}{\mu} \tag{4.28}$$

$$\Rightarrow \limsup_{t \rightarrow \infty} x(t) \leq \frac{\lambda_1}{\mu}.$$

Similarly, from (4.24), one obtains $\frac{dz(t)}{dt} \leq \lambda_1 - \mu z(t)$

and when evaluated we have

$$z(t) \leq z(0)e^{-\mu t} + \frac{\lambda_2}{\mu} (1 - e^{-\mu t})$$

Hence,

$$\Rightarrow \limsup_{t \rightarrow \infty} z(t) \leq \frac{\lambda_2}{\mu} \text{ a.s.}$$

From (4.28) and (4.24) we get

$$\begin{aligned} \limsup_{t \rightarrow \infty} \frac{z(t)}{T(t)} &= \limsup_{t \rightarrow \infty} \frac{z(t)}{x(t) + y(t) + z(t) + z_1(t)} \\ &\leq \limsup_{t \rightarrow \infty} \frac{z(t)}{x(t) + z(t)} \\ &\leq \frac{\frac{\lambda_2}{\mu}}{\frac{\lambda_1}{\mu} + \frac{\lambda_2}{\mu}} = \frac{\lambda_2}{\lambda_1 + \lambda_2} \end{aligned}$$

$$\Rightarrow \limsup_{t \rightarrow \infty} \frac{z(t)}{T(t)} \leq \frac{\lambda_2}{\lambda_1 + \lambda_2}$$

$\forall \epsilon > 0 \exists t_1$ such that,

$$\frac{z(t)}{T(t)} \leq \frac{\lambda_2}{\lambda_1 + \lambda_2} + \epsilon \quad \forall t \geq t_1. \quad (4.29)$$

For $\tau \geq t_1$,

$$\frac{1}{\tau} \int_0^\tau \frac{z(s)}{T(s)} ds = \frac{1}{\tau} \int_0^{t_1} \frac{z(s)}{T(s)} ds + \frac{1}{\tau} \int_{t_1}^\tau \frac{z(s)}{T(s)} ds.$$

Now using (4.29), one obtains,

$$\begin{aligned} \frac{1}{\tau} \int_0^\tau \frac{z(s)}{T(s)} ds &\leq \frac{1}{\tau} \int_0^{t_1} ds + \frac{1}{\tau} \int_{t_1}^\tau \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} + \epsilon \right) ds \\ &= \frac{t_1}{\tau} + \frac{1}{\tau} (\tau - t_1) \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} + \epsilon \right) \\ &= \frac{t_1}{\tau} \left[1 - \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} + \epsilon \right) \right] + \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} + \epsilon \right) \quad \text{a.s.} \end{aligned}$$

Therefore, one obtains

$$\limsup_{\tau \rightarrow \infty} \frac{1}{\tau} \int_0^\tau \frac{z(s)}{T(s)} ds \leq \frac{\lambda_2}{\lambda_1 + \lambda_2} + \epsilon \text{ a.s.}$$

Since ϵ is arbitrary,

$$\limsup_{\tau \rightarrow \infty} \frac{1}{\tau} \int_0^\tau \frac{z(s)}{T(s)} ds \leq \frac{\lambda_2}{\lambda_1 + \lambda_2} \quad \text{a.s. is obtained.} \quad (4.30)$$

In the same way,

$$\limsup_{t \rightarrow \infty} \frac{x(t)}{T(t)} \leq \frac{\lambda_1}{\lambda_1 + \lambda_2}.$$

The next step is to examine the infected population, that is, $y(t)$ and $z_1(t)$. For $z_1(t)$, if $z_1(0) > 0$, then, one defines a function $Y(z_1) = \log(z_1)$, $z_1 \in (0, \infty)$. Using Itô's formula, one obtains,

$$dY(z_1(t)) = \left[\beta c \alpha_2^2 \frac{z(t)}{T(t)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right] dt - \sigma_1 dW(t).$$

Then,

$$\int_0^\tau dY(z_1(t)) = \int_0^\tau \left(\beta c \alpha_2^2 \frac{z(s)}{T(s)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) ds - \int_0^\tau \sigma_1 dW(s).$$

Simplifying and dividing by $\frac{1}{\tau}$ yields

$$\frac{1}{\tau} Y(z_1(\tau)) = \frac{1}{\tau} Y(z_1(0)) + \frac{1}{\tau} \int_0^\tau \left(\beta c \alpha_2^2 \frac{z(s)}{T(s)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) ds - \frac{1}{\tau} \int_0^\tau \sigma_1 dW(s).$$

Using the result from [47], one notices that

$$\lim_{\tau \rightarrow \infty} \frac{W(\tau)}{\tau} = 0. \quad (4.31)$$

Using (4.30) and (4.31) and substituting the value of $Y(z_1(\tau))$, one obtains

$$\begin{aligned} \limsup_{\tau \rightarrow \infty} \frac{1}{\tau} \log z_1(\tau) &= \beta c \alpha_2^2 \lim_{\tau \rightarrow \infty} \frac{1}{\tau} \int_0^\tau \frac{z(s)}{T(s)} ds - (\mu + \sigma + D_{21} + \frac{\sigma_1^2}{2}) - \lim_{\tau \rightarrow \infty} \frac{1}{\tau} \int_0^\tau \sigma_1 dW(s) \\ &\leq \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21} + \frac{\sigma_1^2}{2}) \\ &< 0 \quad \text{since } R_1 < 1 \end{aligned}$$

$$\lim_{\tau \rightarrow \infty} \frac{1}{\tau} \log z_1(\tau) \leq \kappa < 0, \quad (4.32)$$

where $\kappa = \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21} + \frac{\sigma_1^2}{2})$,
from (4.32),

$$\Rightarrow z_1(t) \leq e^{\kappa t} \quad \forall t \geq 0 \text{ a.s.} \quad (4.33)$$

For $y(t)$ one can express (4.23) as

$$y(t) = y(0) + \beta c \alpha_1^2 \int_0^t \frac{x(s)y(s)}{T(s)} ds + D_{21} \int_0^t z_1(s) ds - (\mu + \sigma) \int_0^t y(s) ds - \sigma_1 \int_0^t y(s) dW(s).$$

From (4.33), one obtains,

$$\lim_{t \rightarrow \infty} \int_0^t z_1(s) ds \leq \int_0^\infty e^{\kappa t} < \infty \quad \text{almost surely.}$$

Using Lemma 4.3.3, one gets

$$\lim_{t \rightarrow \infty} y(t) < \infty$$

$$\Rightarrow \lim_{t \rightarrow \infty} \int_0^t y(s) ds = \int_0^\infty y(s) ds < \infty \quad \text{almost surely.}$$

It has to be proven that $\lim_{t \rightarrow \infty} y(t) = 0$ almost surely

If not, there exists an $\Omega_1 \subset \Omega$ such that $P(\Omega_1) > 0$ and for every $\omega \in \Omega_1$,

$$\liminf_{t \rightarrow \infty} y(t, \omega) > 0.$$

Therefore, for every fixed $\omega_1 \in \Omega_1$, one gets,

$$\liminf_{t \rightarrow \infty} y(t, \omega_1) = \rho(\omega_1) > 0$$

and there is a $T_1 > 0$ such that, $y(t, \omega) > \frac{1}{2} \rho(\omega_1)$ for every $t \geq T_1$. Hence,

$$\int_0^\infty y(\tau, \omega_1) d\tau = \int_0^{T_1} y(\tau, \omega_1) d\tau + \int_{T_1}^\infty y(\tau, \omega_1) d\tau \geq \int_{T_1}^\infty y(\tau, \omega_1) d\tau = \infty.$$

This implies that $\Omega_1 \subset \Omega_2$, where $\Omega_2 = \{\omega : \int_0^\infty y(s, \omega) ds = \infty\}$. Therefore, $P(\Omega_2) > 0$.

Hence, one gets

$\lim_{t \rightarrow \infty} y(t) = 0$ almost surely.

If susceptible individuals of group two are considered, that is, $z(t)$, by (4.33), one obtains

$$\lim_{t \rightarrow \infty} \frac{z_1(t)}{T(t)} = 0 \quad \text{a.s.} \quad (4.34)$$

Given any $\epsilon > 0$ and by (4.34), there is a T' such that

$$\beta c \alpha_2^2 \frac{z_1(t)}{T(t)} < \epsilon \quad \forall t \geq T'.$$

Therefore, for every $t \geq T'$,

$$\begin{aligned} \frac{dz(t)}{dt} &\geq \lambda_2 - (\mu + \epsilon)z(t) \text{ almost surely} \\ \Rightarrow z(t) &\geq z(0)e^{-(\mu+\epsilon)t} + \frac{\lambda_2}{\mu+\epsilon} \left(1 - e^{-(\mu+\epsilon)t}\right). \end{aligned}$$

It is now easy to see that $\liminf_{t \rightarrow \infty} z(t) \geq \frac{\lambda_2}{\mu}$ almost surely.

One then obtains,

$$\lim_{t \rightarrow \infty} z(t) = \frac{\lambda_2}{\mu}$$

It has been proven that as $t \rightarrow \infty$, $x(t) \rightarrow \frac{\lambda_1}{\mu}$, $z(t) \rightarrow \frac{\lambda_2}{\mu}$, $y(t) \rightarrow 0$, $z_1(t) \rightarrow 0$. This completes the proof. ■

It was observed that infectious populations eventually die out with time while susceptible population stabilises around $\frac{\lambda_1}{\mu}$, $\frac{\lambda_2}{\mu}$ for the first and the second groups respectively.

4.3.3 Stability at the disease-free equilibrium

Two kinds of stability are examined for the disease-free equilibrium. Moment exponential stability and almost sure exponential stability are discussed. Firstly, a definition of stability (check [47]) is given.

Let

$$ds(t) = F(s(t), t)dt + G(s(t), t)dW(t) \quad (4.35)$$

be a general n -dimensional stochastic differential equation. The solution to the equation is denoted by $s(t; s_0)$ for every $t \geq 0$ with the initial value $s(0) = s_0$. If

$$G(0, t) = 0 \quad \text{and} \quad F(0, t) = 0 \quad \text{for every } t \geq 0.$$

Then, $s(t) \equiv 0$ is a solution to equation (4.35) and corresponds to an initial value $s(t_0) = 0$. This solution is known as the equilibrium position or the trivial solution.

Definition 4.3.5. *The trivial solution of equation (4.35) is almost surely stable exponentially if*

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log |s(t; s_0)| < 0 \quad \text{almost surely}$$

for every $s_0 \in \mathbb{R}^n$.

Definition 4.3.6. The trivial solution of equation (4.35) is the p^{th} moment stable exponentially if there are some constants $\lambda, C > 0$ such that

$$E|s(t; s_0)|^p \leq C|s_0|^p e^{-\lambda t} \quad \text{for all } t \geq 0$$

for every $s_0 \in \mathbb{R}^n$. It is stable in mean square exponentially whenever $p = 2$.

A near disease-free equilibrium population, that is, $x(t) = \frac{\lambda_1}{\mu} + \varsigma_1$, $z(t) = \frac{\lambda_2}{\mu} + \varsigma_2$, and $y(t), z_1(t)$ to be very small is considered.

Almost sure exponential stability

Considering infected populations, one obtains

$$\begin{aligned} d\tilde{V}(t) &= \begin{pmatrix} \beta c \alpha_1^2 \frac{x(t)}{T(t)} - (\mu + \sigma) & D_{21} \\ 0 & \beta c \alpha_2^2 \frac{z(t)}{T(t)} - (\mu + \sigma + D_{21}) \end{pmatrix} \tilde{V}(t) dt - \sigma_1 \tilde{V}(t) dW(t) \\ &\approx \begin{pmatrix} \beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) & D_{21} \\ 0 & \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) \end{pmatrix} \tilde{V}(t) dt - \sigma_1 \tilde{V}(t) dW(t), \end{aligned}$$

where $\tilde{V}(t) = (y(t), z_1(t))^T$ and $\frac{x(t)}{T(t)} \rightarrow \frac{\lambda_1}{\lambda_1 + \lambda_2}$, $\frac{z(t)}{T(t)} \rightarrow \frac{\lambda_2}{\lambda_1 + \lambda_2}$.

One now gets the solution.

Let $X = \log(y(t))$ and $Y = \log(z_1(t))$, and using Itô's formula yields

$$\begin{aligned} d \log(y(t)) &= \left(\beta c \alpha_1^2 \frac{x(t)}{T(t)} - (\mu + \sigma) + D_{21} \frac{z_1(t)}{y(t)} - \frac{\sigma_1^2}{2} \right) dt - \sigma_1 dW(t) \\ &= \left(\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) + D_{21} - (\mu + \sigma) - \frac{\sigma_1^2}{2} \right) dt - \sigma_1 dW(t) \\ \Rightarrow \log(y(t)) &= \left(\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) + D_{21} - (\mu + \sigma) - \frac{\sigma_1^2}{2} \right) t - \sigma_1 W(t) + C \\ \Rightarrow y(t) &= y(0) \exp \left[\left(\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) + D_{21} - \frac{\sigma_1^2}{2} \right) t - \sigma_1 W(t) \right]. \end{aligned}$$

Similarly,

$$\begin{aligned} d \log(z_1(t)) &= \left(\beta c \alpha_2^2 \frac{z(t)}{T(t)} - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) dt - \sigma_1 dW(t) \\ &= \left(\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) dt - \sigma_1 dW(t) \\ z_1(t) &= z_1(0) \exp \left[\left(\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) t - \sigma_1 W(t) \right]. \end{aligned}$$

$$\text{Therefore,} \quad \tilde{V}(t) = \exp \left[\left(F - \frac{1}{2} \sigma_1^2 I \right) t - \sigma_1 I W(t) \right] \tilde{V}(0),$$

where I is an identity matrix. Hence,

$$F = \begin{pmatrix} \beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) & D_{21} \\ 0 & \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) \end{pmatrix}.$$

Suppose $R_1 = \frac{\beta c \alpha_2^2 \frac{\lambda_2}{\lambda_1 + \lambda_2}}{\mu + \sigma + D_{21} + \sigma_1^2/2} < 1$ and $\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) < \mu + \sigma + \frac{\sigma_1^2}{2}$, then

$$F - \frac{1}{2} \sigma_1^2 I = \begin{pmatrix} \beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) - \frac{1}{2} \sigma_1^2 & D_{21} \\ 0 & \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) - \frac{1}{2} \sigma_1^2 \end{pmatrix}.$$

Computing the eigenvalues λ' and λ''

$$\det \left[\left(F - \frac{1}{2} \sigma_1^2 I \right) - \lambda I \right] = \begin{vmatrix} \beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) - \frac{1}{2} \sigma_1^2 - \lambda' & D_{21} \\ 0 & \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) - \frac{1}{2} \sigma_1^2 - \lambda'' \end{vmatrix}$$

$\det \left[\left(F - \frac{1}{2} \sigma_1^2 I \right) - \lambda I \right] = 0$, implies that

$$\left(\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) - \frac{1}{2} \sigma_1^2 - \lambda' \right) \left(\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) - \frac{1}{2} \sigma_1^2 - \lambda'' \right) = 0$$

$$\Rightarrow \lambda' = \beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma) - \frac{\sigma_1^2}{2} \text{ or } \lambda'' = \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - (\mu + \sigma + D_{21}) - \frac{1}{2} \sigma_1^2.$$

Since λ' and λ'' are real negative numbers, then

$$\| \exp \left[\left(F - \frac{1}{2} \sigma_1^2 I \right) t \right] \| \leq C e^{-2\lambda t}$$

**NWU
LIBRARY**

for some constants $C, \lambda > 0$ (check [[47], page127]). Following Mao's method of analysis [[47], page127], one obtains

$$\lim_{t \rightarrow \infty} \frac{1}{t} \log |\tilde{V}(t)| \leq -2\lambda \quad \text{a.s.}$$

Therefore, $\exists t_0$ such that for every $t \geq t_0$

$$|\tilde{V}(t)| \leq |\tilde{V}(t_0)| e^{-\lambda(t-t_0)} \quad \text{almost surely.}$$

Without loss of generality, let it be assumed that $\lambda_i < \mu, i = 1, 2$.

Since $\frac{dx(t)}{dt} \leq \lambda_1 - \mu x(t)$ one gets

$$x(t) \leq \frac{\lambda_1}{\mu} \left(1 - e^{-\mu t} \right) + x(0) e^{-\mu t}, \text{ hence,}$$

$$|x(t) - \frac{\lambda_1}{\mu}| \leq |x(0) - \frac{\lambda_1}{\mu}| e^{-\mu t}.$$

Next, let $\xi(t) = -\frac{\lambda_2}{\mu} + z(t)$ (then $z(t) = \frac{\lambda_2}{\mu} + \xi(t)$), from (4.24) one gets

$$\begin{aligned} \frac{d\xi(t)}{dt} &= \lambda_2 - \beta c \alpha_2^2 \frac{z_1(t)}{T(t)} \left(\xi(t) + \frac{\lambda_2}{\mu} \right) - \mu \left(\xi(t) + \frac{\lambda_2}{\mu} \right) \\ &= -\mu \xi(t) - \beta c \alpha_2^2 \frac{z_1(t)}{T(t)} \left(\xi(t) + \frac{\lambda_2}{\mu} \right) \\ &= -\mu \xi(t) - \beta c \alpha_2^2 \frac{z(t) z_1(t)}{T(t)}. \end{aligned}$$

Integrating for $t \geq t_0$ (and integrating factor I.F = $e^{\mu t}$), one gets

$$\begin{aligned}
 \left[\frac{d\xi(t)}{dt} + \mu\xi(t) \right] e^{\mu t} &= \left[-\beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} \right] e^{\mu t} \\
 \Rightarrow \int_{t_0}^t \frac{d}{ds} \left(\xi(s) e^{\mu s} \right) ds &= \int_{t_0}^t \left(-\beta c \alpha_2^2 \frac{z(s)z_1(s)}{T(s)} \right) e^{\mu s} ds \\
 \Rightarrow \xi(t) &= \xi(t_0) e^{-\mu(t-t_0)} - e^{-\mu t} \int_{t_0}^t \beta c \alpha_2^2 \frac{z(s)z_1(s)}{T(s)} e^{\mu s} ds \\
 \Rightarrow |\xi(t)| &\leq |\xi(t_0)| e^{-\mu(t-t_0)} + \beta c \alpha_2^2 e^{-\mu t} \int_{t_0}^t |\tilde{V}(0)| e^{-\lambda s + \mu s} ds \\
 &\leq \left[|\xi(t_0)| + \frac{\beta c \alpha_2^2 |\tilde{V}(0)|}{\mu - \lambda} \right] e^{-\lambda(t-t_0)}.
 \end{aligned}$$

Therefore, for $t \geq t_0$, one gets,

$$|\tilde{x}(t) - \left(\frac{\lambda_1}{\mu}, 0, \frac{\lambda_2}{\mu}, 0 \right)| \leq R e^{-\lambda(t-t_0)},$$

where R is a constant. Hence, there is an almost surely locally exponentially stable disease-free equilibrium.

Moment exponential stability

Let

$$\begin{aligned}
 a_{11} &= \left[\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) - \left(\mu + \sigma + \frac{\sigma_1^2}{2} \right) \right] t - \sigma_1 W(t), \\
 a_{12} &= D_{12} t \quad \text{and} \\
 a_{22} &= \left[\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) - \left(\mu + \sigma + D_{21} \right) - \frac{\sigma_1^2}{2} \right] t - \sigma_1 dW(t).
 \end{aligned}$$

Close to the disease-free equilibrium

$$z_1(t) = z_1(0) e^{a_{22}(t)}.$$

Let $\xi(t) = z(t) - \frac{\lambda_2}{\mu}$, then one gets

$$\begin{aligned}
 \frac{d\xi(t)}{dt} &= -\mu\xi(t) - \beta c \alpha_2^2 \frac{z(t)z_1(t)}{T(t)} \\
 \Rightarrow \xi(t) &= \xi(0) e^{-\mu t} - e^{-\mu t} \int_0^t \beta c \alpha_2^2 \frac{z(s)z_1(s)}{T(s)} e^{\mu s} ds.
 \end{aligned}$$

It should be noted that,

$$\int_0^t \beta c \alpha_2^2 \frac{z(s)z_1(s)}{T(s)} e^{\mu s} ds \leq \beta c \alpha_2^2 \int_0^t z_1(0) e^{a_{22}(s) + \mu s} ds, \quad \text{since } \frac{z_1(t)z(t)}{T(t)} \leq z_1(t).$$

Hence,

$$\begin{aligned} |\xi(t)| &\leq |\xi(0)|e^{-\mu t} + \beta c \alpha_2^2 z_1(0) e^{-\mu t} \int_0^t e^{a_{22}(s)+\mu s} ds \\ &\leq 2 \max \left(|\xi(0)|e^{-\mu t}, \beta c \alpha_2^2 z_1(0) e^{-\mu t} \int_0^t e^{a_{22}(s)+\mu s} ds \right). \end{aligned}$$

If $A = \begin{pmatrix} a_{11} & a_{12} \\ 0 & a_{22} \end{pmatrix}$ then following the properties of matrix exponential, one gets

$$e^A = \begin{cases} \begin{pmatrix} e^{a_{11}} & \frac{a_{12}}{a_{11}-a_{22}}(e^{a_{11}} - e^{a_{22}}) \\ 0 & e^{a_{22}} \end{pmatrix} & \text{if } a_{22} \neq a_{11}, \\ \begin{pmatrix} e^{a_{11}} & a_{12}e^{a_{11}} \\ 0 & e^{a_{22}} \end{pmatrix} & \text{if } a_{22} = a_{11}. \end{cases}$$

From the above, the disease-free equilibrium solution can be written as

$$\tilde{V}(t) = \exp \left[\left(F - \frac{1}{2} \sigma_1^2 I \right) t - \sigma_1 I W(t) \right] \tilde{V}(0).$$

Then,

$$\left(F - \frac{1}{2} \sigma_1^2 I \right) t - \sigma_1 I W(t) = \begin{pmatrix} a_{11}(t) & a_{12}(t) \\ 0 & a_{22}(t) \end{pmatrix}. \quad (4.36)$$

Suppose $\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2} \right) + D_{21} \neq \beta c \alpha_1^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right)$, then

$$\tilde{V}(t) = \begin{pmatrix} e^{a_{11}(t)} & \frac{a_{12}}{a_{11}-a_{22}}(e^{a_{11}(t)} - e^{a_{22}(t)}) \\ 0 & e^{a_{22}(t)} \end{pmatrix} \tilde{V}(0) = \begin{pmatrix} p e^{a_{11}(t)} + q e^{a_{22}(t)} \\ z_1(0) e^{a_{22}(t)} \end{pmatrix},$$

where p and q are constants. Hence,

$$|\tilde{V}(t)| \leq r e^{2 \max(a_{11}(t), a_{22}(t))}, \quad \text{where } r = 5 \max(|p|^2, |q|^2, z_1(0)^2).$$

Moreover, from (4.22), one gets,

$$\begin{aligned} \frac{dx(t)}{dt} &\leq \lambda_1 - \mu x(t) \\ \Rightarrow x(t) &\leq \frac{\lambda_1}{\mu} (1 - e^{-\mu t}) + x(0) e^{-\mu t} \\ \Rightarrow |x(t) - \frac{\lambda_1}{\mu}| &\leq |x(0) - \frac{\lambda_1}{\mu}| e^{-\mu t}. \end{aligned}$$

Let $\tilde{h}(t) = (x(t) - \frac{\lambda_1}{\mu}, y(t), z(t) - \frac{\lambda_2}{\mu}, z_1(t))$

$$\begin{aligned} |\tilde{h}(t)|^2 &= \left(x(t) - \frac{\lambda_1}{\mu} \right)^2 + y^2(t) + \left(z(t) - \frac{\lambda_2}{\mu} \right)^2 + z_1^2(t) \\ &= |\tilde{V}(t)|^2 + \left(x(t) - \frac{\lambda_1}{\mu} \right)^2 + \left(z(t) - \frac{\lambda_2}{\mu} \right)^2 \\ &\leq 3 \max(|\tilde{V}(t)|^2, |x(t) - \frac{\lambda_1}{\mu}|^2, |z(t) - \frac{\lambda_2}{\mu}|^2) \\ &\leq 3 \max \left(r_1 e^{\max(2a_{11}(t), 2a_{22}(t), -2\mu t)}, r_2 e^{-2\mu t} \left| \int_0^t e^{a_{22}(s)+\mu s} ds \right|^2 \right), \end{aligned}$$

where $r_1 = \max(r^2, 4|\xi(0)|^2, |x(0) - \frac{\lambda_1}{\mu}|^2)$ and $r_2 = 4|\beta c \alpha^2 z_1(0)|^2$. Therefore, for every $p > 0$

$$\begin{aligned} |\tilde{h}(t)|^p &\leq 3^{\frac{p}{2}} \max\left(r_1^{\frac{p}{2}} e^{\max(pa_{11}(t), pa_{22}(t), -p\mu t)}, r_2^{\frac{p}{2}} e^{-p\mu t} \left| \int_0^t e^{a_{22}(s) + \mu s} ds \right|^p\right), \\ E|\tilde{h}(t)|^p &\leq 3^{\frac{p}{2}} r_1^{\frac{p}{2}} \left(Ee^{pa_{11}(t)} + Ee^{pa_{22}(t)} + e^{-p\mu t}\right) + r_2^{\frac{p}{2}} t^{p-1} e^{-p\mu t} E\left(\int_0^t e^{p(a_{22}(s) + \mu s)} ds\right), \\ &\text{for } p \geq 1, \text{ using Hölder's inequality,} \\ &\leq 3^{\frac{p}{2}} r_1^{\frac{p}{2}} \left(e^{\left(\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2}\right) - (\mu + \sigma + \frac{\sigma_1^2}{2})\right)pt + \frac{p^2 \sigma_1^2}{2}t} + e^{\left(\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2}\right) - (\mu + \sigma + D_{21}) - \frac{\sigma_2^2}{2}\right)pt + \frac{p^2 \sigma_1^2}{2}t} + e^{-p\mu t}\right) \\ &\quad + r_2^{\frac{p}{2}} t^{p-1} e^{-p\mu t} \int_0^t Ee^{p(a_{22}(s) + \mu s)} ds. \end{aligned}$$

The last integral is

$$\begin{cases} t, & \text{if } k = \frac{p^2 \sigma_1^2}{2} + \mu \\ \frac{e^{p(\mu - k)t + \frac{p^2 \sigma_1^2}{2}t} - 1}{p(\mu - k) + \frac{p^2 \sigma_1^2}{2}}, & \text{if } k \neq \frac{p^2 \sigma_1^2}{2} + \mu \end{cases}.$$

Therefore,

$$\begin{aligned} E|\tilde{V}(t)|^p &\leq 3^{\frac{p}{2}} r_1^{\frac{p}{2}} \left(e^{\left(\beta c \alpha_1^2 \left(\frac{\lambda_1}{\lambda_1 + \lambda_2}\right) - (\mu + \sigma + \frac{\sigma_1^2}{2})\right)pt + \frac{p^2 \sigma_1^2}{2}t} + e^{\left(\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2}\right) - (\mu + \sigma + D_{21}) - \frac{\sigma_2^2}{2}\right)pt + \frac{p^2 \sigma_1^2}{2}t} + e^{-p\mu t}\right) \\ &\quad + r_3 t^{p-1} e^{-p\mu t} + r_4 t^{p-1} e^{\left(\beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2}\right) - (\mu + \sigma + D_{21}) - \frac{\sigma_2^2}{2}\right)pt + \frac{p^2 \sigma_1^2}{2}t} + r_5 t^p e^{-p\mu t} \end{aligned}$$

for some constants r_3, r_4 and r_5 .

For $(\sigma + \mu) - \max(0, \beta c \alpha_2^2 (\frac{\lambda_2}{\lambda_1 + \lambda_2}) - D_{21}) > \frac{(p-1)\sigma_1^2}{2}$, the p^{th} moment exponentially stable disease-free equilibrium. On the other hand, if $\frac{(p-1)\sigma_1^2}{2} + \frac{\lambda_1}{\lambda_1 + \lambda_2} > \mu + \sigma$, choose $\tilde{V}(0) = (1, 0)^T$, then one gets

$$\begin{aligned} \tilde{V}(t) &= (e^{a_{11}(t)}, 0)^T \\ \Rightarrow |\tilde{V}(t)|^p &= e^{pa_{11}(t)} \\ \Rightarrow E|\tilde{h}(t)|^p &\geq Ee^{pa_{11}(t)} = e^{p\left[\beta c \alpha_1^2 \frac{\lambda_1}{\lambda_1 + \lambda_2} \frac{(p-1)\sigma_1^2}{2} - (\mu + \sigma)\right]t}. \end{aligned}$$

In this case, the p^{th} moment exponentially unstable disease-free equilibrium is obtained for every positive p .

In the same way, if

$$\frac{(p-1)\sigma_1^2}{2} > (\mu + \sigma + D_{21}) - \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2}\right),$$

then for every $p > 0$, the disease-free equilibrium is again p^{th} moment exponentially unstable.

In the case where $\beta c \alpha_1^2 (\frac{\lambda_1}{\lambda_1 + \lambda_2}) + D_{21} = \beta c \alpha_2^2 (\frac{\lambda_2}{\lambda_1 + \lambda_2})$, then

$$\tilde{V}(t) = \begin{pmatrix} e^{a_{11}(t)} & a_{12}(t)e^{a_{11}(t)} \\ 0 & e^{a_{22}(t)} \end{pmatrix} \tilde{V}(0).$$

Finally, using the same reasoning shows that if

$$(\mu + \sigma) - \max(\beta c \alpha_2^2 (\frac{\lambda_2}{\lambda_1 + \lambda_2}) - D_{21}, 0) > \frac{(p-1)\sigma_1^2}{2},$$

then the disease-free equilibrium is p^{th} moment exponentially stable for $p \geq 1$ and p^{th} is not moment exponentially stable for every $p > 0$ whenever

$$(\mu + \sigma) - \max(0, \beta c \alpha_2^2 (\frac{\lambda_2}{\lambda_1 + \lambda_2}) - D_{21}) < \frac{(p-1)\sigma_1^2}{2}.$$

4.4. Results and discussion

This study examined a stochastic model representing the spread of HIV-AIDS in a homosexual population. The main objective of the study was to analyse the effect of environmental noise to the dynamics of the spread of the disease. Using the system of stochastic differential equations, it was discovered that environmental noise brings stability to the system. Both stability in probability and the almost sure exponential stability are attained, but does not achieve exponential stability for the p^{th} moment whenever $p \geq 1$.

Suppose a recently infected person enters the disease-free equilibrium and τ is the time that the newly infected person will be infectious, the approximate probability $\mathcal{F}_{t+\Delta t}$ that the infected person goes out of the infected class during the time interval $[t, t + \Delta t]$ is given by $(\mu + \sigma + D_{21})\Delta t + \sigma_1(W(t + \Delta t) - W(t))$. Therefore, given $\mathcal{F}_t$, then the probability that an infected person will still be infectious at time t is

$$P(\tau \geq t | \mathcal{F}_t) \approx \min\{e^{-(\mu + \sigma + D_{21})t - \sigma_1 W(t)}, 1\}.$$

Hence,

$$\begin{aligned} P(\tau \geq t) &\approx E(P(\tau \geq t | \mathcal{F}_t)) \\ &\approx E(\exp [- (\mu + \sigma + D_{21})t - \sigma_1 W(t)]) \\ &\approx \exp \left[- \left((\mu + \sigma + D_{21}) - \frac{\sigma_1^2}{2} \right) t \right], \end{aligned}$$

provided that $\mu + \sigma + D_{21} > \frac{\sigma_1^2}{2}$,

$$E[\tau] \approx \frac{1}{\mu + \sigma + D_{21} - \frac{\sigma_1^2}{2}}$$

and $R_2 \approx \beta c \alpha_2^2 \left(\frac{\lambda_2}{\lambda_1 + \lambda_2} \right) E[T]$ is the number of cases caused by an infected person who enters the disease-free equilibrium.

Hence for $p = 2$, if $R_2 < 1$ and $(\mu + \sigma) > \frac{\sigma_1^2}{2}$ then we have the exponentially mean square stable disease free equilibrium, however when $R_2 > 1$ or $\frac{\sigma_1^2}{2} > (\mu + \sigma)$ then we have the exponentially mean square unstable disease free equilibrium.

In addition, it was observed that significance of the biological parameters is unaffected, and the noise does not change the system in any way. One particular case is the importance of using condoms to reduce the spread of HIV-AIDS. It is a parameter that is very important in the stochastic model as it helps in determining the possibility of eliminating the infection from the given population. Nevertheless, an improvement to the deterministic model is made by including noise to the model. Since the real world is stochastic (not entirely deterministic) in nature, it is always very significant to include stochasticity (random effects) to the deterministic model. In this study, it was shown as one of the ways this could be done. From this study it can be concluded that the introduction of environmental noise to the deterministic system (or model) brings stability to the system.

5

Stochastic model for internal HIV dynamics driven by fractional brownian motion

5.1. Introduction

From the time HIV was discovered in 1981, it has relentlessly been spreading to every part of the world and has become a dominant global pandemic. It attacks the immune system, specifically by decreasing the CD4 cells. The production and development of HIV infection is the function of the cellular environment of the host, life cycle of the virus, and the amount of virus in the body of an infected individual. Factors such as genetic differences between individuals, age, virulence levels of a particular virus strain, and coinfection with other pathogenic bacteria influence the progression rate and the severity of the disease. Viral replication in the host starts at the cell surface receptor CD4 on the entry site of HIV. The infective cells discharge virions (a complete viral particle made up of the infective form of a virus), that infects other healthy cells. The primary target of HIV are the CD4 cells, which become infected when they encounter the virus. Active HIV replication occurs at every stage of infection. Throughout a period of time, even when small amounts of virus can be detected in the blood, a considerable number of virus accumulate inside every infected cell. This kind of interaction between the immune system and the virus is known as HIV internal viral dynamics. In this chapter, a stochastic model driven by fractional Brownian motion for the host virus interaction is formulated.

Modeling the interaction between the CD4 cells and HIV-1 virus has been an extensive research area for many years [8, 15, 21]. Mathematical modeling have come to play a significant role in these biological systems. Predictions about the behaviour of the system has been made possible through the use of Mathematics. This chapter presents some analytical results for the stochastic model driven by fractional Brownian motion (particularly, an expression for the variance and mean of the limit process.

When stochastic models are used, there are more real benefits to be gained than when deterministic models are used. Naturally, real life is not deterministic but stochastic. When a biological

phenomenon such as the internal HIV viral dynamics are modeled, there is a need to consider the stochastic effects. The effects of environmental stochasticity are modeled on some of the parameters in the model. More useful results are generated by stochastic models rather than models that are deterministic, since a distribution of the predicted outcomes can be constructed by running a stochastic model many times, for instance, the number of infected cells at time t , while a deterministic model will only give one predicted value. Having a distribution for the predicted outcomes is more versatile as it helps to practically study stochastic quantities that are important. For instance, the probability that the infectious virus particles have died out at a given time and the variance of the number of infectious virus particles at a given time, which cannot be studied using deterministic models. Quantities such as the mean value of the number of cells can be modeled more accurately by stochastic models since they take into account the effects due to random variation on these quantities which deterministic models cannot.

Other studies done by N. Dalal *et al.* [19] and [20] introduce stochasticity to the model for the model of AIDS and use of condoms and the model for internal HIV dynamics respectively using the method of perturbation of parameters which is a standard method in modeling stochastic dynamics of a population. They demonstrated non-negativity of solutions to the model established in their studies as required in the dynamics of population modeling. In addition, they performed a thorough asymptotic stability analysis (both in p^{th} moment and in probability). The purpose of this study is to show how a certain type of stochastic perturbation (noise driven by fractional Brownian motion) may help stabilise the system.

The first step is to prove that the solutions are non-negative which is a very critical condition for modeling stochastic differential equations in population dynamics. Attention is then shifted to the stability aspect for the three variables under consideration. Next, it is shown that the number of virus particles and infected cells will both almost surely tend to zero (their disease-free equilibrium value) under certain conditions. Then, a mean reverting process is introduced to show that the number of healthy cells tend to this mean reverting process in probability. Finally, details of the parameters of the mean reverting process are given.

5.2. Deterministic model

Highly Active Antiretroviral Treatment (HAART) can reverse or slow down the production and development of HIV-1 to a certain extent. In general, HAART constrains the formation process of virus particles. This results in an increased quantity of CD4 cells and a reduced quantity of viral load. In this chapter, a stochastic model of viral dynamics driven by fractional Brownian motion is done including the effects of HAART.

Schaedeli and Verotta [69] used models that are nonlinear to represent the HIV-1 dynamics that incorporates various factors related to recovery. They started by first giving a nonlinear model for the dynamics of HIV-1 virus, then included HIV medication adherence, exposure to drugs and insurgency of resistant HIV-1 strains. They used clinical trial data for AIDS patients using a

combination of antiretroviral drugs for treatment to demonstrate the application of their model. Perelson and Nelson [58] maintain that models with intracellular delays have a more accurate biological representation and change the estimated values of kinetic parameters compared to models without delays. They constructed and analysed a set of models that include the dynamics of both uninfected and infected T cells, a combination of antiretroviral therapy and intracellular delays. They demonstrated that for any unperfect drug effect, the estimated death rate of productively infected cells increases when they fitted data with delay models compared to the values obtained when non-delay models are used. They also obtained general results on the stability of the system. About a decade ago, Ciupe *et al.* [15] examined a model for the dynamics of HIV-1 infection made up of three different stages beginning with initial infection, then latent stage and eventually AIDS or drug therapy. In this chapter, the dynamics of primary infection and the early stages of latency were modeled. They revealed that when time delays are allowed in the model there is a better prediction of viral load data compared to when a model has no time delay. They also established that the primary infection model predicts the turnover rates for productively infected T cells and viral totals to be much longer than those observed from patients receiving antiviral drug therapy. They compared the results using Monte Carlo techniques and analysis for three different models and showed how each predicts rather dramatic differences between the fitted parameters. The following three-dimensional model (proposed by Dalal, Greenhalgh and Mao [20]) describe the viral dynamics in the presence of HIV-1 infection and HAART:

$$\begin{aligned}\frac{dx(t)}{dt} &= \lambda - \delta x(t) - \beta(1 - \gamma)x(t)z(t), \\ \frac{dy(t)}{dt} &= \beta(1 - \gamma)x(t)z(t) - \alpha y(t), \\ \frac{dz(t)}{dt} &= (1 - \eta)N\alpha y(t) - \mu z(t) - \beta(1 - \gamma)x(t)z(t),\end{aligned}$$

with suitable initial conditions. The model above mathematically captures the dynamics of HIV-1 interacting with CD4 cells.

Generally, HAART is a combination of protease inhibitor (PI) drugs and reverse transcriptase inhibitor (RTI) drugs. In the early stages of HIV replication, RTI drugs prevents the HIV RNA from being converted to DNA. Therefore, RTI drugs block the uninfected cells from being infected. The protease inhibitor drugs are fashioned to prevent HIV from being properly assembled during the final stage in the replication cycle of the virus. They also cause recently generated virus to be noninfective [22]. The parameters and variables used in the above model are described as follows:

-
- $x(t)$ is the concentration of uninfected cells;
 $y(t)$ is the concentration of infected cells;
 $z(t)$ is the concentration of virus particles;
 $(1 - \eta)$ is the effect of the protease inhibitor drug;
 $(1 - \gamma)$ is the effect of reverse transcriptase inhibitor drug;
 λ is the total production rate per unit time of healthy cells;
 δ is per capita death rate for healthy cells;
 β is the transmission coefficient between infective virus particles and uninfected cells;
 α is per capita death rate for infected cells;
 N is the average number of infective virus particles produced by an infected cell in the absence of HAART throughout its infectious lifetime; and
 μ is per capita death rate for infective virus particles.

It should be noted that when an uninfected cell is infected by an infectious virus particle, it absorbs the virus particle which effectively dies. Consequently, the term $\beta(1 - \gamma)x(t)z(t)$ appears in all the equations of the model. The model above has a unique disease-free equilibrium given by $(\lambda/\delta, 0, 0)$. Every cell that is newly infected and enters the disease-free equilibrium, remains infected for time $(1/\alpha)$ and during this time, it generates $N(1 - \eta)$ infectious virus particles. Assuming that the system is still near the disease-free equilibrium, every infectious virus particle has an approximate time of survival of $(\mu + \beta(1 - \gamma)\lambda/\delta)$ and during this time, the number of cells it will infect is

$$\frac{(1 - \gamma)\beta\frac{\lambda}{\delta}}{(\mu + (1 - \gamma)\beta\frac{\lambda}{\delta})} = \frac{(1 - \gamma)\beta\lambda}{\delta\mu + (1 - \gamma)\beta\lambda}.$$

In this particular case, one can define the basic reproductive ratio R_0 as the average number of healthy cells expected to be infected (secondary infected cells) by a single infected cell entering the disease-free population at equilibrium. Secondary infected cells are cells that are infected by an infectious virus particle that originated from an initial infected cell. R_0 is useful because it helps determine whether or not an infectious disease can spread through a population. Therefore,

$$R_0 = \frac{(1 - \gamma)\beta\lambda N(1 - \eta)}{(\delta\mu + \beta\lambda(1 - \gamma))}.$$

In this case, R_0 can also be thought of as the number of secondary infected virus particles that one infected virus particle generates on average as it enters the disease-free population at equilibrium. In this case, a secondary infectious virus particle is an infectious virus particle generated by an infected cell which was infected by the primary infectious virus particle. The same expression for R_0 is obtained.

Tuckwell and Wan [67] analysed the deterministic model. They showed that if $R_0 \leq 1$, then there is a unique disease-free equilibrium and if $R_0 > 1$, then there is a unique endemic equilibrium in

addition to the disease-free equilibrium given by

$$\begin{aligned} x^* &= \frac{\mu}{[N(1-\eta)-1]\beta(1-\gamma)}, \\ y^* &= \frac{N(1-\eta)\beta\lambda(1-\gamma) - (1-\gamma)\beta\lambda - \delta\mu}{(N(1-\eta)\alpha\beta(1-\gamma) - 1)}, \\ z^* &= \frac{N(1-\eta)\beta\lambda(1-\gamma) - (1-\gamma)\beta\lambda - \delta\mu}{(1-\gamma)\beta\mu}. \end{aligned}$$

Furthermore, they demonstrated that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, while if $R_0 > 1$, then the unique endemic equilibrium is locally asymptotically stable while the disease-free equilibrium is unstable. Therefore, if $R_0 < 1$, then the number of infected virus particles and infected cells is expected to die out in the long-run and one expects the number of healthy cells to approach λ/δ , while if $R_0 > 1$, the number of infectious virus particles, infected cells and healthy cells are expected to approach their unique endemic equilibrium values.

Perelson *et al.* [61] considered a simpler form of the deterministic model. They took the number of uninfected cells to be constant. Also, Bonhoeffer *et al.* [8] analysed a simpler form of the deterministic model where they approximated the term $\beta(1-\gamma)x(t)z(t)$ to be zero. In both studies they did not include any effect of stochasticity in their analysis. They showed that there is a basic reproductive ratio R_0 which shows how the system behaves. For $R_0 > 1$, there is a unique endemic equilibrium but for $R_0 \leq 1$, the system has a locally asymptotically stable unique disease-free equilibrium. Later, they modified the basic model to add the effects of resistance. Di Mascio *et al.* [21] considered Bonhoeffer *et al.*'s simplified version of the deterministic model and modified it to add the effects of HAART in the same way as they did in [20].

All the models above are deterministic and do not introduce the effects of stochasticity to their models. There are many other models in literature ([15],[22],[38],[69]) that are similar to the model but have introduced different or extra variables such as two types of infectious virus particles, two types of infected cells or CD8 cells.

5.3. Stochastic model

The death of CD4 cells involves a range of mechanisms, which includes apoptosis and syncytium formation among other things [54]. The rate at which virions are cleared, can be affected by various factors such as immune elimination and entry and binding into cells [61]. Since there are various complex biological phenomena that affect the rates of death of both the virus and infected cells, it is believed that in these death rates, there is randomness involved.

For a long time, many scientists have believed that HIV depletes the CD4+ T cells (primary target), by cutting off the production of new T-cells. However, on the other hand, it has been shown in some studies that HIV does not cut off such production but rather speeds up the division of existing T cells. There is an immediate fall in the rate of production of T-cells that is accompanied by an even greater decrease in the death rate of CD4 T-cells after an initiation of highly active antiretroviral

therapy (HAART). Therefore, the increased count of CD4+ T-cells seen following HAART is not due to an increased production of new T cells but instead it is caused by a slowdown in the loss of existing T cells. Due to these contradicting views, there is reason to believe that there is randomness in the death rates of CD4 cells.

Accounting for these factors one can add the effects of randomness to the system by replacing the parameters δ, α and μ in the model by $\delta \rightarrow \delta + \sigma_1 \dot{B}_{H_1}(t)$, $\alpha \rightarrow \alpha + \sigma_1 \dot{B}_{H_1}(t)$ and $\mu \rightarrow \mu + \sigma_2 \dot{B}_{H_2}(t)$. This is the first step to introducing stochasticity into the model. It would be more appealing to also introduce environmental stochastic variations into the other parameters such as the total rate of production of healthy cells λ and the transmission coefficient β , but this will make the analysis more difficult.

N. Dalal *et al.* [20] introduced stochasticity to the system by adding a term with white noise (ordinary time derivative of Brownian motion) to the model. In this chapter, consideration is given to the noise driven by fraction Brownian motion B_H , where $H \in (0, 1)$ is the Hurst parameter, (for $H = 1/2$ one gets a standard Brownian motion).

Therefore, one obtains the following system of stochastic differential equations driven by fractional Brownian motion:

$$dx(t) = (\lambda - \delta x(t) - \beta(1 - \gamma)x(t)z(t))dt - \sigma_1 x(t)dB_{H_1}(t), \quad (5.1)$$

$$dy(t) = (\beta(1 - \gamma)x(t)z(t) - \alpha y(t))dt - \sigma_1 y(t)dB_{H_1}(t), \quad (5.2)$$

$$dz(t) = ((1 - \eta)N\alpha y(t) - \mu z(t) - \beta(1 - \gamma)x(t)z(t))dt - \sigma_2 z(t)dB_{H_2}(t), \quad (5.3)$$

with suitable initial conditions,

where $B_{H_1}(t)$ and $B_{H_2}(t)$ are independent fractional Brownian motions. When parameters such as death rate have randomness in them, the standard method is to introduce environmental noise to the parameter as in [3, 10, 48, 49]. The same fractional Brownian motion $B_H(t)$ and the noise intensity σ is obtained for infected and healthy CD4 cells, but different for virus particles and CD4 cells. This is because the biological factors that affect the death rates of healthy and infected CD4 cells are believed to be identical, but the biological factors affecting the virus particles and CD4 cells are different.

Therefore, even without detailed biological facts, it is plausible that the fractional Brownian motion $B_H(t)$ and the noise intensity σ are not the same for infected and healthy CD4 cells, it is possible as a first simplifying approximation to assume that they are similar. Since the virus particles and CD4 cells are considerably distinct biological entities, it is highly likely that both $B_H(t)$ and σ are different between infective virus particles and CD4 cells.

Note how the situation will be like without the infectious virus particles and infected cells present

$$(x, y, z) = (\lambda/\delta, 0, 0)$$

which is a point of equilibrium for the deterministic model but not for the stochastic model, though the last two co-ordinates $(y, z) = (0, 0)$ are still a stochastic equilibrium position. The situation

is different for the first co-ordinate of the system, and it is shown that it stochastically fluctuates about λ/δ .

5.3.1 Non-negative solutions

When dealing with models in population dynamics, it is fundamental that one does not have uncertainties about negative values. Therefore, one must first show that the solutions are non-negative.

Let $(\Omega, \mathcal{F}, P)$ denote the usual complete probability space with filtration $\{\mathcal{F}_t\}_{t \geq 0}$ that satisfies the conventional properties (i.e. it is right continuous and increasing and all the P -null sets are contained in $\mathcal{F}_0$). Let $B_H(t)$ be a one-dimensional fractional Brownian motion defined on the complete probability space. Furthermore, let $\mathbb{R}_{++}^3 = \{\tilde{x} \in \mathbb{R}^3 : x_i > 0 \text{ and } x_i \in \tilde{x} \text{ for all } 1 \leq i \leq 3\}$ such that $\tilde{x}(t) = (x(t), y(t), z(t))$.

Using the result in Lemma 3.3.1 one can now prove the following theorem.

Theorem 5.3.1. *Assuming that $\sigma_1, \sigma_2, \mu, \delta, \lambda, \alpha, N$ are positive real numbers and $0 < \gamma, \eta < 1$, then there is a unique solution $\tilde{x}(t)$ for equations (5.1 – 5.3) for every initial value $\tilde{x}_0 \in \mathbb{R}_{++}^3$ and $\forall t \geq 0$. With probability 1, this solution remains in $\mathbb{R}_{++}^3$, that is, $\tilde{x}(t) \in \mathbb{R}_{++}^3$ for every $t \geq 0$ a.s.*

PROOF.

Since the coefficients of the equations in (5.1 – 5.3) are locally Lipschitz continuous, then for every given initial condition $\tilde{x}_0 \in \mathbb{R}_{++}^3$, there is a unique local solution $\tilde{x}(t)$ for every $t \in [0, \tau_e]$, where τ_e is the explosion time [2, 25]. In order to prove that the solution is global, all one has to do is show that $\tau_e < \infty$ almost surely. A constant $c_0 \geq 0$ that is large enough so that all components of $\tilde{x}_0$ are contained in the interval $[\frac{1}{c_0}, c_0]$ is considered. The stopping time for each integer $c \geq c_0$ is defined as.

$$\tau_c = \inf \left\{ t \in [0, \tau_e) : \text{at least one of } x, y \text{ or } z \in \left(\frac{1}{c}, c \right) \right\},$$

where throughout this section, one sets $\inf \emptyset = \infty$ (where $\emptyset$ is the empty set). Clearly, τ_c increases as $c \rightarrow \infty$. Let $\tau_\infty = \lim_{c \rightarrow \infty} \tau_c$, then $\tau_e \geq \tau_\infty$ almost surely. If it is proven that $\tau_\infty = \infty$ almost surely, then $\tau_e = \infty$ and $\tilde{x}(t) \in \mathbb{R}_{++}^3$ almost surely for every $t \geq 0$. In other words, for the proof to be complete, it must be proven that $\tau_\infty = \infty$ almost surely. If this statement was not true, then there will be a pair of real numbers $\varepsilon \in (0, 1)$ and $\mathcal{T} > 0$ such that

$$P\{\tau_\infty \leq \mathcal{T}\} > \varepsilon.$$

Then, there is an integer $c_1 \leq c_0$ such that

$$P\{\tau_\infty \leq \mathcal{T}\} \geq \varepsilon \quad \text{for every } c \geq c_1. \quad (5.4)$$

Let a C^2 -function $U : \mathbb{R}_{++}^3 \rightarrow \mathbb{R}_{++}^3$ be defined by

$U(\tilde{x}) = 1 + x - \log(x) + 1 + y - \log(y) + 1 + z - \log(z)$. It can be seen that this function is non-negative

using $v + 1 - \log(v) \geq 0, \forall v > 0$. Following Itô's formula with respect to FBM (Theorem 2.4.8), one obtains

$$\begin{aligned}
dU(\tilde{x}(t)) &= \left(1 - \frac{1}{x(t)}\right)(\lambda - \delta x(t) - (1 - \gamma)\beta x(t)z(t))dt - \sigma_1 x(t) \left(1 - \frac{1}{x(t)}\right)dB_{H_1}(t) \\
&\quad + \left(1 - \frac{1}{y(t)}\right)(\beta(1 - \gamma)x(t)z(t) - \alpha y(t))dt - \sigma_1 x(t) \left(1 - \frac{1}{y(t)}\right)dB_{H_1}(t) \\
&\quad + \left(1 - \frac{1}{z(t)}\right)((1 - \eta)N\alpha y(t) - \mu z(t) - \beta(1 - \gamma)x(t)z(t))dt - \sigma_2 z(t) \left(1 - \frac{1}{z(t)}\right)dB_{H_2}(t) \\
&= \left[\left(1 - \frac{1}{x(t)}\right)(\lambda - \delta x(t) - \beta(1 - \gamma)x(t)z(t)) + \left(1 - \frac{1}{y(t)}\right)(\beta(1 - \gamma)x(t)z(t) - \alpha y(t))\right. \\
&\quad \left.+ \left(1 - \frac{1}{z(t)}\right)((1 - \eta)N\alpha y(t) - \mu z(t) - \beta(1 - \gamma)x(t)z(t))\right]dt \\
&\quad + \sigma_1(2 - x(t) - y(t))dB_{H_1}(t) + \sigma_2(1 - z(t))dB_{H_2}(t) \\
&= \left[\lambda - \delta x(t) - \beta(1 - \gamma)x(t)z(t) + \beta(1 - \gamma)x(t)z(t) - \alpha y(t) + (1 - \eta)N\alpha y(t) - \mu z(t)\right. \\
&\quad \left.- \beta(1 - \gamma)x(t)z(t) - \frac{\lambda}{x(t)} + \delta + \beta(1 - \gamma)z(t) - \frac{(1 - \gamma)}{y(t)}\beta x(t)z(t) + \alpha - \frac{(1 - \eta)}{z(t)}N\alpha y(t)\right. \\
&\quad \left.+ \mu + \beta(1 - \gamma)x(t)\right]dt + \sigma_1(2 - x(t) - y(t))dB_{H_1}(t) + \sigma_2(1 - z(t))dB_{H_2}(t).
\end{aligned}$$

Hence,

$$\begin{aligned}
dU(\tilde{x}(t)) &\leq [\lambda\delta + \alpha + \mu + \beta(1 - \gamma)x(t)z(t) + (1 - \eta)N\alpha y(t) + (1 - \eta)N\alpha z(t)]dt \\
&\quad + \sigma_1(2 - x(t) - y(t))dB_{H_1}(t) + \sigma_2(1 - z(t))dB_{H_2}(t).
\end{aligned}$$

Let $r_1 = \lambda + \delta + \alpha + \mu$ and $r_2 = 2(1 - \eta)N\alpha + 2\beta(1 - \gamma)$.

By lemma 3.3.1, $u \leq 2(u + 1 - \log(u))$ so

$(1 - \eta)N\alpha y(t) + \beta(1 - \gamma)z(t) + \beta(1 - \gamma)y(t) \leq r_2 U(\tilde{x}(t))$ Therefore

$$dU(\tilde{x}(t)) \leq (r_1 + r_2 U(t))dt + \sigma_1(2 - x(t) - y(t))dB_{H_1}(t) + \sigma_2(1 - z(t))dB_{H_2}(t).$$

Hence,

$$dU(\tilde{x}(t)) \leq r_3(1 + U(\tilde{x}(t)))dt + \sigma_1(2 - x(t) - y(t))dB_{H_1}(t) + \sigma_2(1 - z(t))dB_{H_2}(t),$$

where $\max(r_1, r_2)$. Therefore, if $t_1 \leq \mathcal{T}$,

$$\begin{aligned}
\int_0^{\tau_c \wedge t_1} dU(\tilde{x}(t)) &\leq \int_0^{\tau_c \wedge t_1} r_3(1 + U(\tilde{x}(t)))dt + \int_0^{\tau_c \wedge t_1} \sigma_1(2 - x(t) - y(t))dB_{H_1}(t) \\
&\quad + \int_0^{\tau_c \wedge t_1} \sigma_2(1 - z(t))dB_{H_2}(t).
\end{aligned} \tag{5.5}$$

Taking the expectation, one gets

$$E[U(\tilde{x}(\tau_c \wedge t_1))] \leq U(\tilde{x}_0) + E \int_0^{\tau_c \wedge t_1} r_3(1 + U(\tilde{x}(t)))dt.$$

The expectation of the last two integrals in (5.5) is zero, by prop 2.4.13(iii)

$$\begin{aligned} \Rightarrow E[U(\tilde{x}(\tau_c \wedge t_1))] &\leq U(\tilde{x}_0) + r_3 t_1 + r_3 E \int_0^{\tau_c \wedge t_1} U(\tilde{x}(t)) dt \\ &\leq U(\tilde{x}_0) + r_3 \mathcal{T} + r_3 E \int_0^{t_1} U(\tilde{x}(\tau_c \wedge t)) dt \\ &= U(\tilde{x}_0) + r_3 \mathcal{T} + r_3 \int_0^{t_1} E[U(\tilde{x}(\tau_c \wedge t))] dt. \end{aligned}$$

Following the Gronwall inequality,

$f = 1, \phi = E[U(\tilde{x}(\tau_c \wedge t_1))], C_0 = U(\tilde{x}_0) + r_3 \mathcal{T}$, one obtains

$$\begin{aligned} E[U(\tilde{x}(\tau_c \wedge t_1))] &\leq (U(\tilde{x}_0) + r_3 \mathcal{T}) e^{\int_0^{t_1} r_3 ds} \\ &= (U(\tilde{x}_0) + r_3 \mathcal{T}) e^{r_3 t_1} \\ &\leq (U(\tilde{x}_0) + r_3 \mathcal{T}) e^{r_3 \mathcal{T}}. \end{aligned} \tag{5.6}$$

Set $\Delta_c = \{\tau_c \leq \mathcal{T} \text{ for } c \leq c_1\}$ and by (5.4), $P(\Delta_c) \geq \epsilon$. Note that for all $\omega \in \Delta_c$, at least one of $x(\tau_c, \omega), y(\tau_c, \omega)$ or $z(\tau_c, \omega)$ will be equal to either c or $\frac{1}{c}$. Thus, $U(\tilde{x}(\tau_c, \omega))$ is not less than the minimum between $1 + c - \log(c)$ and $1 + \frac{1}{c} - \log(\frac{1}{c}) = 1 + \frac{1}{c} + \log(c)$.

Therefore, $U(\tilde{x}(\tau_c, \omega)) \geq 1 + c - \log(c) \wedge 1 + \frac{1}{c} + \log(c)$.

It then follows from (5.4) and (5.5) that

$$\begin{aligned} (U(\tilde{x}_0) + r_3 \mathcal{T}) e^{r_3 \mathcal{T}} &\geq E[1_{\Delta_c}(\omega) U(\tilde{x}(\tau_c, \omega))] \\ &\geq \epsilon[(1 + c - \log(c)) \wedge (1 + \frac{1}{c} + \log(c))], \end{aligned}$$

where 1_{Δ_c} is an indicator function of Δ_c . If one lets $c \rightarrow \infty$, then it will lead to a contradiction $\infty > (U(\tilde{x}_0) + r_3 \mathcal{T}) e^{r_3 \mathcal{T}} = \infty$. Therefore, $\tau_\infty = \infty$ almost surely. $\blacksquare$

Next, an investigation of how the system behaves asymptotically is done in an attempt to get more analytical results.

5.3.2 Asymptotic behaviour

Theorem 5.3.2. *$y(t)$ and $z(t)$ are exponentially stable almost surely if the following conditions hold, that is, they will exponentially tend to their equilibrium position 0 almost surely (with probability 1).*

$$(i) \quad N\alpha(1 - \eta) - \alpha < 0$$

$$(ii) \quad [\frac{1}{2}(N\alpha(1 - \eta) - \alpha - \mu)]^2 < \mu(\alpha - (1 - \eta)N\alpha)$$

PROOF.

Firstly,

$$\begin{aligned} d(y(t) + z(t)) &= [\beta(1 - \gamma)x(t)y(t) - \alpha y(t) + (1 - \eta)N\alpha y(t) - \mu z(t) - \beta(1 - \gamma)x(t)y(t)]dt \\ &\quad - \sigma_1 y(t)dB_{H_1}(t) - \sigma_2 z(t)dB_{H_2}(t) \text{ is considered.} \end{aligned}$$

Let $x' = (y, z)$ and $V(x') = \log(y + z)$ for $y, z \in (0, \infty)$.

Using Itô's formula with respect to fractional Brownian motion, one obtains

$$\begin{aligned} dV(x'(t)) &= \left(\frac{(1-\eta)N\alpha y(t)}{y(t)+z(t)} - \frac{\alpha y(t)}{y(t)+z(t)} - \frac{\mu z(t)}{y(t)+z(t)} \right) dt - \frac{\sigma_1 y(t)}{y(t)+z(t)} dB_{H_1}(t) - \frac{\sigma_2 z(t)}{y(t)+z(t)} dB_{H_2}(t) \\ &= \frac{1}{y(t)+z(t)} \left((1-\eta)N\alpha y(t) - \alpha y(t) - \mu z(t) \right) dt - \frac{\sigma_1 y(t)}{y(t)+z(t)} dB_{H_1}(t) - \frac{\sigma_2 z(t)}{y(t)+z(t)} dB_{H_2}(t) \\ &= \frac{1}{(y(t)+z(t))^2} (y(t)+z(t)) ((1-\eta)N\alpha y(t) - \alpha y(t) - \mu z(t)) dt \\ &\quad - \frac{\sigma_1 y(t)}{y(t)+z(t)} dB_{H_1}(t) - \frac{\sigma_2 z(t)}{y(t)+z(t)} dB_{H_2}(t). \end{aligned}$$

One can express

$$(y(t)+z(t))((1-\eta)N\alpha y(t) - \alpha y(t) - \mu z(t))$$

as follows:

$$\begin{pmatrix} y(t) & z(t) \end{pmatrix} \begin{pmatrix} (1-\eta)N\alpha - \alpha & \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} \\ \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} & -\mu \end{pmatrix} \begin{pmatrix} y(t) \\ z(t) \end{pmatrix}.$$

Hence, $dV(x'(t))$ can be expressed as follows:

$$\begin{aligned} dV(x'(t)) &= \frac{1}{(y(t)+z(t))^2} \left\{ \begin{pmatrix} y(t) & z(t) \end{pmatrix} \begin{pmatrix} (1-\eta)N\alpha - \alpha & \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} \\ \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} & -\mu \end{pmatrix} \begin{pmatrix} y(t) \\ z(t) \end{pmatrix} \right\} dt \\ &\quad - \frac{\sigma_1 y(t)}{y(t)+z(t)} dB_{H_1}(t) - \frac{\sigma_2 z(t)}{y(t)+z(t)} dB_{H_2}(t). \end{aligned}$$

The matrix

$$\begin{pmatrix} (1-\eta)N\alpha - \alpha & \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} \\ \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} & -\mu \end{pmatrix} \text{ is considered.}$$

The matrix is negative definite since $(1-\eta)N\alpha - \alpha < 0$ and its determinant is positive. Let the greatest (negative) eigen value be denoted as $\lambda_{\max}$, then

$$\begin{aligned} \begin{pmatrix} y(t) & z(t) \end{pmatrix} \begin{pmatrix} (1-\eta)N\alpha - \alpha & \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} \\ \frac{1}{2} - \frac{\alpha}{2} - \frac{\mu}{2} & -\mu \end{pmatrix} \begin{pmatrix} y(t) \\ z(t) \end{pmatrix} &\leq \lambda_{\max} (y^2(t) + z^2(t)) \\ &= -|\lambda_{\max}| (y^2(t) + z^2(t)). \end{aligned}$$

Therefore,

$$dV(x'(t)) \leq \left(-|\lambda_{\max}| \frac{1}{(y(t)+z(t))^2} (y^2(t) + z^2(t)) \right) dt - \frac{\sigma_1 y(t)}{y(t)+z(t)} dB_{H_1}(t) - \frac{\sigma_2 z(t)}{y(t)+z(t)} dB_{H_2}(t). \quad (5.7)$$

Since $(y^2 + z^2) \geq 2yz$ and $2(y^2 + z^2) = y^2 + z^2 + y^2 + z^2 \geq y^2 + z^2 + 2yz = (y+z)^2 \Rightarrow 2(y^2 + z^2) \geq (y+z)^2$,

using $2(y^2 + z^2) \geq (y + z)^2$ in (5.7), one gets

$$\begin{aligned} dV(x'(t)) &\leq -\frac{1}{2}|\lambda_{\max}|dt - \frac{\sigma_1 y(t)}{(y(t) + z(t))}dB_{H_1}(t) - \frac{\sigma_2 z(t)}{(y(t) + z(t))}dB_{H_2}(t) \\ &\leq -\frac{1}{2}|\lambda_{\max}|dt \\ \Rightarrow d(\log(y(t) + z(t))) &\leq -\frac{1}{2}|\lambda_{\max}|dt \\ \Rightarrow \log(y(t) + z(t)) &\leq -\frac{1}{2}|\lambda_{\max}|t + C \end{aligned}$$

$$\Rightarrow \limsup_{t \rightarrow \infty} \frac{1}{t} \log(y(t) + z(t)) \leq -\frac{1}{2}|\lambda_{\max}| < 0 \text{ a.s.}$$

Therefore, $y(t) \rightarrow 0$ and $z(t) \rightarrow 0$ almost surely as $t \rightarrow \infty$. This completes the proof. $\blacksquare$

Stability in distribution is now shown for $x(t)$ in the sense that it stabilises around the mean value λ/δ . To achieve this, another stochastic process $S(t)$ is introduced, its defined by the initial condition $S(0) = x(0)$ and the stochastic differential equation is given by

$$dS(t) = (\lambda - \delta S(t))dt - \sigma_1 S(t)dB_{H_1}(t).$$

It is shown that $x(t)$ can be approximated by $S(t)$ as t becomes large, so that $\lim_{t \rightarrow \infty} (S(t) - x(t)) = 0$ in probability.

Another function $x_1(t)$ is introduced to help prove the theorem, it is defined by the following stochastic differential equation with initial condition $x_1(0) = x(0)$,

$$dx_1(t) = (\lambda - (\delta + \varepsilon)x_1(t))dt - \sigma_1 x_1(t)dB_H(t). \quad (5.8)$$

Theorem 5.3.3. *If conditions of Theorem 5.3.2 are satisfied, then*

$\lim_{t \rightarrow \infty} (S(t) - x(t)) = 0$ in probability.

PROOF.

The differential for $x(t)$ is

$$dx(t) = [\lambda - \delta x(t) - \beta(1 - \gamma)x(t)z(t)]dt - \sigma_1 x(t)dB_{H_1}(t).$$

One first proves that

$$\liminf_{t \rightarrow \infty} (x(t) - x_1(t)) \geq 0 \text{ a.s.}$$

Therefore,

$$\begin{aligned} d(x(t) - x_1(t)) &= (-\sigma(x(t) - x_1(t)) + \varepsilon x_1(t) - \beta(1 - \gamma)x(t)z(t))dt - \sigma_1(x(t) - x_1(t))dB_{H_1}(t) \\ &= (-\delta + \varepsilon)(x(t) - x_1(t)) + (\varepsilon - \beta(1 - \gamma)z(t))x(t)dt - \sigma(x(t) - x_1(t))dW_1(t) \end{aligned}$$

is considered and the solution given by

$$x(t) - x_1(t) = \varphi(t) \int_0^t \varphi^{-1}(s)(\varepsilon - \beta(1 - \gamma)z(s))x(s)ds,$$

where

$$\varphi(t) = \exp \{ -(\sigma + \varepsilon)t - \sigma_1 B_{H_1}(t) \}.$$

Using the results of Theorem 5.3.2, where it was shown that $z(t) \rightarrow 0$ a.s as $t \rightarrow \infty$, it is concluded that, for nearly every $\omega \in \Omega$, there is a $T = T(\omega)$ such that



$$z(t) < \frac{\varepsilon}{\beta(1-\gamma)}, \forall t \geq T.$$

Therefore, for every $\omega \in \Omega$, if $t > T$, then

$$x(t) - x_1(t) = \varphi(t) \left(\int_0^T \varphi^{-1}(s)(\varepsilon - \beta(1-\gamma)z(s))x(s)ds + \int_T^t \varphi^{-1}(s)(\varepsilon - \beta(1-\gamma)z(s))x(s)ds \right)$$

Hence, $x(t) - x_1(t) \geq \varphi(t)k(T)$, where

$$k(T) = \int_0^T \varphi^{-1}(s)(\varepsilon - \beta(1-\gamma)z(s))x(s)ds$$

clearly, as $t \rightarrow \infty$ then $\varphi(t) \rightarrow 0$ and $|k(T)| < \infty$ a.s.

Therefore,

$$\liminf_{t \rightarrow \infty} |x(t) - x_1(t)| \geq 0 \quad \text{a.s.} \quad (5.9)$$

It is now shown that $\liminf_{t \rightarrow \infty} (S(t) - x(t)) \geq 0$ almost surely. To do this,

$d(S(t) - x(t)) = (-\delta(S(t) - x(t)) + \beta(1-\gamma)x(t)z(t))dt - \sigma_1(S(t) - x(t))dB_{H_1}(t)$ is considered.

This implies that

$$d(S(t) - x(t)) \geq (-\mu(S(t) - x(t)))dt - \sigma_1(S(t) - x(t))dB_{H_1}(t).$$

Let $\phi = S - x$, hence

$$d\phi(t) \geq -\delta\phi(t)dt - \sigma_1\phi(t)dB_{H_1}(t).$$

For every $\omega \in \Omega$, either

$$\omega \in \Omega_1 = \{\omega : \phi(t_0) > 0 \text{ for some } t_0 \geq 0\}$$

or

$$\omega \in \Omega_2 = \{\omega : \phi(t_0) \leq 0 \text{ for every } t \geq 0\}.$$

For $\omega \in \Omega_1$, let $t_1 = \sup\{t : t \geq t_0 \text{ and } \phi(t) > 0\}$. If $t_1 < \infty$, then in $[t_0, t_1]$,

$$d\phi(t) = (-\sigma + f(t))\phi(t)dt - \sigma_1\phi(t)dB_{H_1}(t), \quad (5.10)$$

with a random variable $f(t)$. To find the solution of (5.10), let $Y = \log \phi(t)$ and use Itô's formula with respect to fractional Brownian motion to obtain

$$\phi(t) = \phi(t_0) \exp \left\{ \int_{t_0}^t (-\sigma + f(s))ds - \sigma_1 B_{H_1}(t) \right\} > 0.$$

Hence,

$$\phi(t_1) = \phi(t_0) \exp \left\{ \int_{t_0}^{t_1} (-\sigma + f(s))ds - \sigma_1 B_{H_1}(t) \right\} > 0.$$

Thus, $\phi(t) > 0$ in $[t_1, t_1 + \delta_1]$ for some δ_1 . This is a contradiction. Hence, $t_1 = \infty$ and $\phi(t) \geq 0$ for every $t \geq t_0$. So one gets $\liminf_{t \rightarrow \infty} \phi(t) \geq 0$ a.s.

A case for $\omega \in \Omega_2$ is considered where $\phi(t) \leq 0$ for every $t \geq 0$. Then,

$$d\phi(t) = (-\sigma - f(t))\phi(t)dt - \sigma_1\phi(t)dB_{H_1}(t), \quad (5.11)$$

where the random variable $f(t) \geq 0$. The solution for (5.11) is found by letting $Y = \log \phi(t)$ and Itô's formula with respect to fractional Brownian motion is used to obtain

$$\phi(t) = \phi(0) \exp \left\{ \int_0^t (-\sigma - f(s))ds - \sigma_1 B_{H_1}(t) \right\} \geq 0, \quad \text{since } x_2(0) = x(0).$$

Hence, $\liminf_{t \rightarrow \infty} \phi(t) \geq 0$ a.s. Therefore,

$$\liminf_{t \rightarrow \infty} (S(t) - x(t)) \geq 0 \quad \text{a.s.} \quad (5.12)$$

Next,

$d(x_1(t) - S(t)) = (-\sigma(x_1(t) - S(t)) - \varepsilon x_1(t))dt - \sigma(x_1(t) - S(t))dB_{H_1}(t)$ is considered,

whose solution is

$$x_1(t) - S(t) = -\varepsilon \exp\{-\sigma t - \sigma_1 B_{H_1}(t)\} \int_0^t \exp\{(\sigma + \sigma_1 B_{H_1}(s))\} x_1(s) ds.$$

Note that $x_1(s) \geq 0$, since it is a solution of the stochastic differential equation (5.8) which is linear and can be solved explicitly to give

$$x_1(t) = \lambda \int_0^t \exp\{-(\sigma + \varepsilon)(t-s) - \sigma_1(B_{H_1}(t) - B_{H_1}(s))\} ds. \quad (5.13)$$

Therefore,

$$|x_1(t) - S(t)| = \varepsilon \int_0^t x_1(s) \exp\{-\sigma(t-s) - \sigma_1(B_{H_1}(t) - B_{H_1}(s))\} ds$$

since $B_{H_1}(t) - B_{H_1}(s) \sim \mathcal{N}(0, (t-s)^{2H_1})$, one gets

$$\begin{aligned} E[\exp\{-\sigma_1(B_{H_1}(t) - B_{H_1}(s))\}] &= \int_{-\infty}^{\infty} e^{-\sigma_1 u} \frac{1}{\sqrt{2\pi(t-s)^{2H_1}}} e^{-\frac{u^2}{2(t-s)^{2H_1}}} du \\ &= \frac{1}{\sqrt{2\pi(t-s)^{2H_1}}} \int_{-\infty}^{\infty} \exp\left\{-\frac{1}{2(t-s)^{2H_1}}(u^2 + 2(t-s)^{2H_1}\sigma_1 u)\right\} du \\ &= \frac{1}{\sqrt{2\pi(t-s)^{2H_1}}} \int_{-\infty}^{\infty} e^{\left\{-\frac{1}{2(t-s)^{2H_1}}(u^2 + 2(t-s)^{2H_1}\sigma_1 u) + (t-s)^{4H_1}\sigma_1^2 - (t-s)^{4H_1}\sigma_1^2\right\}} du \\ &= \frac{1}{\sqrt{2\pi(t-s)^{2H_1}}} \int_{-\infty}^{\infty} e^{-\frac{1}{2(t-s)^{2H_1}}\left\{(u+(t-s)^{2H_1}\sigma_1)^2 - (t-s)^{4H_1}\sigma_1^2\right\}} du \\ &= \frac{e^{\frac{(t-s)^{2H_1}\sigma_1^2}{2}}}{\sqrt{2\pi(t-s)^{2H_1}}} \int_{-\infty}^{\infty} \exp\left\{-\frac{1}{2(t-s)^{2H_1}}(u + (t-s)^{2H_1}\sigma_1)^2\right\} du \\ &= e^{\frac{(t-s)^{2H_1}}{2}\sigma_1^2}. \end{aligned}$$

Hence,

$$E[\exp\{-\sigma_1(B_{H_1}(t) - B_{H_1}(s))\}] = \exp\left\{\frac{(t-s)^{2H_1}}{2}\sigma_1^2\right\} \quad (5.14)$$

Therefore,

$$\begin{aligned} E|x_1(t) - S(t)| &= \varepsilon E\left[\int_0^t x_1(s) \exp\{-\delta(t-s) - \sigma_1(B_{H_1}(t) - B_{H_1}(s))\} ds\right] \\ &= \varepsilon \int_0^t E[x_1(s) \exp\{-\delta(t-s) - \sigma_1(B_{H_1}(t) - B_{H_1}(s))\}] ds \\ &= \varepsilon \int_0^t E[x_1(s) \exp\{-\delta(t-s)\}] E[\exp\{-\sigma_1(B_{H_1}(t) - B_{H_1}(s))\}] ds \end{aligned}$$

as $x_1(s)$ is independent of $B_{H_1}(t) - B_{H_1}(s)$,

$$\begin{aligned} &= \varepsilon \int_0^t E[x_1(s) \exp\{-\delta(t-s)\}] \exp\left\{\frac{(t-s)^{2H_1}}{2}\sigma_1^2\right\} ds \\ &= \varepsilon \int_0^t E[x_1(s) \exp\left\{\frac{(t-s)^{2H_1}}{2}\sigma_1^2 - \delta(t-s)\right\}] ds \\ &= \varepsilon \int_0^t \exp\left\{\frac{(t-s)^{2H_1}}{2}\sigma_1^2 - \delta(t-s)\right\} E(x_1(s)) ds. \end{aligned} \quad (5.15)$$

Taking the expectation of (5.13) and using (5.14),

$$\begin{aligned}
 E[x_1(s)] &= \lambda \int_0^t E \left[\exp\{-(\delta + \varepsilon)(t-s) - \sigma_1(B_{H_1}(t) - B_{H_1}(s))\} \right] ds \\
 &= \lambda \int_0^t e^{-(\delta + \varepsilon)(t-s)} E[e^{-\sigma_1(B_{H_1}(t) - B_{H_1}(s))}] ds \\
 &= \lambda \int_0^t e^{-(\delta + \varepsilon)(t-s)} e^{\frac{(t-s)^{2H_1}}{2} \sigma_1^2} ds \\
 &= \lambda \int_0^t e^{\frac{\sigma_1^2}{2}(t-s)^{2H_1}} e^{-(\delta + \varepsilon)(t-s)} ds \text{ is obtained.}
 \end{aligned}$$

Using integration by parts, one gets

$$\begin{aligned}
 E[x_1(t)] &= \frac{\lambda}{\delta + \varepsilon} \left(1 - e^{\frac{\sigma_1^2}{2} t^{2H_1} - (\delta + \varepsilon)t} \right) + \frac{\lambda \sigma_1^2 H_1}{\delta + \varepsilon} \int_0^t (t-s)^{2H_1-1} e^{\frac{\sigma_1^2}{2}(t-s)^{2H_1} - (\delta + \varepsilon)(t-s)} ds \\
 &\geq \frac{\lambda}{\delta + \varepsilon} \left(1 - e^{\frac{\sigma_1^2}{2} t^{2H_1} - (\delta + \varepsilon)t} \right) + \frac{\lambda \sigma_1^2 H_1}{\delta + \varepsilon} \int_0^t (t-s)^{2H_1-1} e^{\frac{\sigma_1^2}{2}(t-s)^{2H_1}} ds \\
 &= \frac{\lambda}{\delta + \varepsilon} \left[\left(1 - e^{\frac{\sigma_1^2}{2} t^{2H_1} - (\delta + \varepsilon)t} \right) + \left(1 - e^{\frac{\sigma_1^2}{2} t^{2H_1}} \right) \right] \\
 &= \frac{\lambda}{\delta + \varepsilon} \left[2 - e^{\frac{\sigma_1^2}{2} t^{2H_1} - (\delta + \varepsilon)t} - e^{\frac{\sigma_1^2}{2} t^{2H_1}} \right] \\
 &\leq \frac{2\lambda}{\delta + \varepsilon} \\
 \Rightarrow E[x_1(t)] &\geq \frac{2\lambda}{\delta + \varepsilon}.
 \end{aligned}$$

Substituting this result in (5.15), one obtains

$$\begin{aligned}
 E|x_1(t) - S(t)| &\geq \frac{2\lambda\varepsilon}{\delta + \varepsilon} \int_0^t e^{\frac{\sigma_1^2(t-s)^{2H_1}}{2} - \delta(t-s)} ds \\
 &\leq \frac{2\lambda\varepsilon}{\delta + \varepsilon} \left[\frac{1}{\delta} \left(2 - e^{\frac{\sigma_1^2}{2} t^{2H_1} - \delta t} - e^{\frac{\sigma_1^2}{2} t^{2H_1}} \right) \right] \\
 &= \frac{2\lambda\varepsilon}{\delta + \varepsilon} \left[\frac{1}{\delta} \left(2 - e^{\frac{\sigma_1^2}{2} t^{2H_1}} (e^{-\delta t} + 1) \right) \right].
 \end{aligned}$$

Hence, it is concluded that

$$\lim_{\varepsilon \rightarrow 0} \lim_{t \rightarrow \infty} E|x_1(t) - S(t)| = 0.$$

This implies that

$$\lim_{\varepsilon \rightarrow 0} \lim_{t \rightarrow \infty} |x_1(t) - S(t)| = 0 \quad \text{in probability} \quad (5.16)$$

combining (5.9), (5.12) and (5.16) gives the needed result. Therefore, the proof is complete. $\blacksquare$

5.3.3 Mean reverting process

One wants to know more about the stochastic process $S(t)$, that is, being used to approximate $x(t)$. This is done by finding its mean and variance.

The mean reverting process has the following differential equation:

$$dS(t) = (\lambda - \delta S(t))dt - \sigma_1 S(t)dB_{H_1}(t),$$

whose solution is given by

$$S(t) = S(0) \exp(-\delta t - \sigma_1 B_{H_1}(t)) + \lambda \int_0^t \exp\{-\sigma(t-s) - \sigma_1(B_{H_1}(t) - B_{H_1}(s))\} ds.$$

Taking the expectation, one gets

$$E[S(t)] = S(0)E[\exp(-\delta t)]E[e^{-\sigma_1 B_{H_1}(t)}] + \lambda \int_0^t E[\exp\{-\delta(t-s)\}]E[e^{-\sigma_1(B_{H_1}(t) - B_{H_1}(s))}] ds.$$

Since $B_{H_1}(t) \sim N(0, t^{2H_1})$, then

$$\begin{aligned} E[e^{-\sigma_1 B_{H_1}(t)}] &= \int_{-\infty}^{\infty} e^{-\sigma_1 u} \frac{1}{\sqrt{2\pi t^{2H_1}}} e^{-\frac{u^2}{2t^{2H_1}}} du \\ &= \frac{1}{\sqrt{2\pi t^{2H_1}}} \int_{-\infty}^{\infty} e^{-\frac{1}{2t^{2H_1}}(u^2 + 2t^{2H_1}\sigma_1 u)} du \\ &= \frac{1}{\sqrt{2\pi t^{2H_1}}} \int_{-\infty}^{\infty} e^{-\frac{1}{2t^{2H_1}}[u^2 + 2t^{2H_1}\sigma_1 u - (t^{2H_1}\sigma_1)^2 + (t^{2H_1}\sigma_1)^2]} du \\ &= \frac{1}{\sqrt{2\pi t^{2H_1}}} \int_{-\infty}^{\infty} e^{-\frac{1}{2t^{2H_1}}[(u + t^{2H_1}\sigma_1)^2 - (t^{2H_1}\sigma_1)^2]} du \\ &= e^{-\frac{t^{2H_1}\sigma_1^2}{2}} \frac{1}{\sqrt{2\pi t^{2H_1}}} \int_{-\infty}^{\infty} e^{-\frac{(u + t^{2H_1}\sigma_1)^2}{2t^{2H_1}}} du \\ &= e^{-\frac{t^{2H_1}\sigma_1^2}{2}}. \end{aligned} \tag{5.17}$$

Substituting (5.14) and (5.17) in the expression for $E[S(t)]$, one obtains

$$\begin{aligned} E[S(t)] &= S(0)E[e^{-\delta t}]e^{-\frac{1}{2}t^{2H_1}\sigma_1^2} + \lambda \int_0^t E[e^{-\delta(t-s)}]e^{\frac{1}{2}(t-s)^{2H_1}\sigma_1^2} ds \\ &= S(0)e^{\frac{1}{2}\sigma_1^2 t^{2H_1} - \delta t} + \lambda \int_0^t e^{\frac{1}{2}(t-s)^{2H_1}\sigma_1^2 - \delta(t-s)} ds \\ &\leq S(0)e^{\frac{1}{2}\sigma_1^2 t^{2H_1} - \delta t} + \frac{\lambda}{\delta} \left[2 - e^{\frac{1}{2}\sigma_1^2 t^{2H_1}} - e^{\frac{1}{2}\sigma_1^2 t^{2H_1} - \delta t} \right] \\ &\leq S(0)e^{\frac{1}{2}\sigma_1^2 t^{2H_1} - \delta t} + \frac{\lambda}{\delta}. \end{aligned}$$

Therefore, taking the limit, one obtains

$$\begin{aligned} \lim_{t \rightarrow \infty} E[S(t)] &\leq \frac{\lambda}{\delta} \quad \text{if } \delta t \geq \frac{1}{2}\sigma_1^2 t^{2H_1}, \\ \text{and } \lim_{t \rightarrow \infty} E[S(t)] &= \infty \text{ for } \delta t < \frac{1}{2}\sigma_1^2 t^{2H_1}. \end{aligned}$$

To find the second moment, $V[S(t)] = S^2$ is considered and Itô's formula with respect to fractional Brownian motion is used to obtain

$$\begin{aligned} dV[S(t)] &= \left[\frac{\partial V}{\partial t} + b(t, \omega) \frac{\partial V}{\partial S} \right] dt + B(t, \omega) \frac{\partial V}{\partial S} dB_{H_1}(t) \\ &= 2S(t)[\lambda - \delta S(t)]dt - 2\sigma_1 S^2(t)dB_{H_1}(t). \end{aligned}$$

$$\begin{aligned} \text{Therefore, } dS^2(t) &= 2\lambda S(t)dt - 2\delta S^2(t)dt - 2\sigma_1^2 S^2(t)dB_{H_1}(t) \\ \Rightarrow S^2(t) &= S^2(0) + 2\lambda \int_0^t S(s)ds - 2\delta \int_0^t S^2(s)ds - 2\sigma_1 \int_0^t S^2(s)dB_{H_1}(s). \end{aligned}$$

Taking the expectation, one gets

$$E[S^2(t)] = E[S^2(0)] + 2\lambda \int_0^t E[S(s)]ds - 2\delta \int_0^t E[S^2(s)]ds - 2\sigma_1 \int_0^t E[S^2(s)]dB_{H_1}(s).$$

The last integral is zero, by proposition 2.4.13(iii).

Hence,

$$E[S^2(t)] = E[S^2(0)] + 2\lambda \int_0^t E[S(s)]ds - 2\delta \int_0^t E[S^2(s)]ds$$

Differentiating with respect to t , one gets

$$\frac{d}{dt}E[S^2(t)] = 2\lambda E[S(t)] - 2\delta E[S^2(t)]$$

and

$$\begin{aligned} \frac{d}{dt}[E[S^2(t)]e^{2\delta t}] &= e^{2\delta t} \frac{d}{dt}E[S^2(t)] + E[S^2(t)] \frac{d}{dt}e^{2\delta t} \\ &= e^{2\delta t}[2\lambda E[S(t)] - 2\delta E[S^2(t)]] + 2\delta e^{2\delta t}E[S^2(t)] \\ &= 2\lambda E[S(t)]e^{2\delta t}. \end{aligned}$$

Integrating and multiplying by $e^{-2\delta t}$, one obtains

$$E[S^2(t)] = E[S^2(0)]e^{-2\delta t} + 2\lambda e^{-2\delta t} \int_0^t e^{2\delta s} E[S(s)]ds.$$

Now, substituting for $E[S(s)]$, one gets

$$\begin{aligned} E[S^2(s)] &\leq E[S^2(0)]e^{-2\delta s} + 2\lambda e^{-2\delta s} \int_0^s e^{2\delta u} \left[\frac{\lambda}{\delta} e^{\frac{\sigma_1^2}{2} s^{2H_1} - \delta s} \right] ds \\ &= E[S^2(0)]e^{-2\delta s} + 2\lambda e^{-2\delta s} \left[\frac{\lambda}{\delta} \int_0^s e^{2\delta u} du + S(0) \int_0^s e^{\frac{\sigma_1^2}{2} s^{2H_1} + \delta s} ds \right] \\ &= E[S^2(0)]e^{-2\delta s} + 2\lambda e^{-2\delta s} \left[\frac{\lambda}{2\delta^2} (e^{2\delta s} - 1) + S(0) \int_0^s e^{\frac{\sigma_1^2}{2} s^{2H_1} + \delta s} ds \right] \\ \Rightarrow E[S^2(t)] &\leq E[S^2(0)]e^{-2\delta t} + \frac{\lambda^2}{\delta^2} (1 - e^{-2\delta t}) + 2\lambda S(0)e^{-2\delta t} \int_0^t e^{\frac{\sigma_1^2}{2} s^{2H_1} + \delta s} ds. \end{aligned}$$

Taking the limit as $t \rightarrow \infty$,

$$\lim_{t \rightarrow \infty} E[S^2(t)] \leq \frac{\lambda^2}{\delta^2} \text{ is obtained.}$$

Therefore, the maximum value of $E[S^2(t)]$ is $\frac{\lambda^2}{\delta^2}$.

One can now compute the asymptotic variance of the mean reverting process

$$\lim_{t \rightarrow \infty} V(S(t)) = \lim_{t \rightarrow \infty} E[S^2(t)] - \lim_{t \rightarrow \infty} E[S(t)]^2 = 0.$$

5.4. Conclusion

This chapter has examined a stochastic model driven by fractional Brownian motion in order to describe the dynamics of HIV-1 infection. It was proven that the solutions are non-negative. The stability of the model was studied. It was shown that the number of virus particles and infected cells almost surely tend asymptotically to zero exponentially. It was also shown that $x(t)$ approaches the mean reverting process $S(t)$ in probability.

Most models of internal HIV dynamics studied in the past by most researchers are deterministic in nature. Perelson *et al.* [61] considered the number of healthy cells to be constant and modeled the dynamics of infectious virus particles and infected cells. Nelson and Perelson [58], Di Mascio *et al.* [21] and Bonhoeffer *et al.* [8] studied models similar to the current deterministic model, but approximated the term $\beta(1-\gamma)x(t)z(t)$ to be zero. Tuckwell and Wan [67] studied the deterministic model and included the term $\beta(1-\gamma)x(t)z(t)$ to obtain stability and equilibrium results.

N. Dalal *et al.* [20] studied a stochastic differential equation model for internal HIV dynamics. Their model is similar to the current one but is driven by standard Brownian motion instead of the fractional Brownian motion. Different results were obtained because in the analysis, the Itô's formula with respect to fractional Brownian motion was used which is different from the one used in [20].

It was shown in this study that the stochastic model gives another option in the modeling of viral dynamics and adds a different perspective to this problem, and this gives researchers a different method to consider in future. Since most real world problems are not deterministic, including stochastic effects into the model gives a more realistic way to model viral dynamics.

6

Conclusion

This study has successfully managed to include randomness to the existing deterministic epidemiological models turning them into stochastic ones. Since most real world problems are not deterministic, including stochastic effects into the model gives us a more realistic way to model viral dynamics. In this chapter we give a summary of our findings.

In chapter three of this study, a stochastic model that describes the dynamics of transmission for tuberculosis was examined. In the past, the models studied for the transmission dynamics of tuberculosis in the literature used ordinary differential equations that are deterministic, neglecting the stochastic effects. By reproducing the results obtained from the deterministic model in [9] and making some improvements on it, it was shown that the stochastic model considered adds a new dimension of results to the dynamics of transmission of the disease. Including effects of stochasticity into the model gives a more realistic way of modeling the dynamics of transmission of TB since there is a lot of randomness in the real world. It was proven that the model has positive solutions. The stability of the stochastic model was studied. It was shown that the number of infectious and infected individuals in latent stage asymptotically tend to zero exponentially with probability one. In addition, it was demonstrated that the susceptible population $x(t)$ can be approximated by a mean reverting process $x_2(t)$.

In chapter four, a stochastic model representing the spread of HIV-AIDS in a homosexual population was considered. The effects of environmental noise to the dynamics of the spread of the disease was also discussed. Using the system of stochastic differential equations, it was discovered that environmental noise brings stability to the system. Both the stability in probability and the almost sure exponential stability were attained, but did not achieve exponential stability for the p^{th} moment whenever $p \geq 1$. From this study, it can be concluded that the introduction of environmental noise to the deterministic system (or model) brings stability to the system.

Chapter five discussed a stochastic model driven by fractional Brownian motion to describe the dynamics of HIV-1 infection. Using Itô's formula with respect to fractional Brownian motion, one was able to obtain new results. It was proven that the solutions are non-negative. The stability of the model was later studied. It was shown that the number of virus particles and infected cells

almost surely tend asymptotically to zero exponentially. It was later shown that $x(t)$ approach a mean reverting process $S(t)$ in probability.

From these results, it can be concluded that a certain type of stochastic perturbation can help bring stability to a deterministic system.

Bibliography

- [1] Adetunde I.A (2008): The mathematical models of the dynamical behaviour of tuberculosis disease in the upper region of the northern part of Ghana: a case study of Bawku. *Current Research in Tuberculosis*, 1(1): 1-6.
- [2] Arnold L, *Stochastic Differential equations: Theory and Applications*, Wiley, New York, 1974.
- [3] Bahar A, Mao X, Stochastic delay LotkaVolterra model, *J. Math. Anal. Appl.* 292 (2004) 364-380.
- [4] Bender C, An S-transform approach to integration with respect to a fractional Brownian motion, *Bernoulli* 9(6), 2003, 955-983
- [5] Blythe S.P, Anderson R.M, Distributed incubation and infections periods in models of transmission dynamics of human immunodeficiency virus (HIV), *IMA J. Math. Appl. Med. Biol.* 5 (1988) 1-19.
- [6] Blythe S.P, Anderson R.M., Variable infectiousness in HIV transmission models, *IMA J. Math. Appl. Med. Biol.* 5 (1988) 181-200.
- [7] Blythe S.P, Anderson R.M., Heterogenous sexual activity models of HIV transmission in male homosexual populations, *IMA J. Math. Appl. Med. Biol.* 5 (1988) 237-260.
- [8] Bonhoeffer S, Shaw G, May R, Nowak M, Virus dynamics and drug therapy, *Proc. Natl. Acad. Sci. USA* 94 (1997) 6971-6976.
- [9] Bowong S, Tewa J.J, Kamgang J, Stability analysis of the transmission dynamics of tuberculosis models. *WMJS*. Vol. 7 (2011) No. 2, pp. 83-100.
- [10] Braumann C.A, Variable effort harvesting models in random environments: Generalisation to density-dependent noise intensities, *Math. Biosci.* 177-178 (2002) 229-245.
- [11] Breiman L, *Probability*, Addison-Wesley, New York, 1968.
- [12] Bremaud P, *An introduction to probabilistic modeling*, Springer, New York, 1988.
- [13] Brogger S. 1967. Systems analysis in tuberculosis control: A model, *Amer. Rev. Resp. Dis.* 95, 419-434.
- [14] Chung K.L, *Elementary probability theory with stochastic processes*, Springer, New York, 1975.
- [15] Ciupe M, Bivort B, Bortz D, Nelson P, Estimating kinetic parameters from HIV primary infection data through the eyes of three different mathematical models, *Math. Biosci.* 200 (2006) 1-27.

-
- [16] Colijn C, Cohen T and Murray M (2006): Mathematical models of tuberculosis: accomplishments and future challenges. *Proc. Natl. Aca. Sci.* 103(11)1-28.
 - [17] Dai W and Heyde C.C, Stochastic integrals with respect to fractional Brownian motion, (1996), preprint.
 - [18] Dai W and Heyde C.C, Itô's formula with respect to fractional Brownian motion and its applications, *Journal of Applied Mathematics and Stochastic Analysis* 9, Number 4, 1996, 439-448.
 - [19] Dalal N, Greenhalgh D, Mao X, A stochastic model of AIDS and condom use. *J. Math. Anal. Appl.* 325 (2007), no. 1, 36-53.
 - [20] Dalal N, Greenhalgh D, Mao X, A stochastic model for internal HIV dynamics. *J. Math. Anal. Appl.* 341 (2008), no. 2, 1084-1101.
 - [21] Di Mascio M, Ribeiro R, Markowitz M, Ho D, Perelson A, Modeling the long-term control of viraemia in HIV-1 infected patients treated with antiretroviral therapy, *Math. Biosci.* 188 (2004) 47-62.
 - [22] Ding A, Wu H, Relationships between antiviral treatment effects and biphasic viral decay rates in modelling HIV dynamics, *Math. Biosci.* 160(1999) 63-82.
 - [23] Egbetade S.A, Ibrahim M.O and Ejieji C.N (2013): On existence of a vaccination model of tuberculosis disease epidemic. *International of Engineering and Science*, 2(7): 41-44.
 - [24] Feller W, An introduction to probability theory and its applications, Vol. 2, John Wiley and Sons, New York - London - Sydney, 1966.
 - [25] Freidman A, Stochastic differential equations and applications, Vol. 1 and 2, Academic press, New York, (1975, 1977).
 - [26] Gard T.C, Introduction to stochastic differential equations, Marcel Dekker, New York, 1988.
 - [27] Gihman I.I and Skorohod A.V, Stochastic differential equations, Springer-Verlag, Berlin, 1978.
 - [28] Global tuberculosis report (2014): www.who.int/tb/publications/global_report.
 - [29] Greenhalgh D, Doyle M, Lewis F, A mathematical treatment of AIDS and condom use, *IMA J. Math. Appl. Med. Biol.* 18 (2001) 225-262.
 - [30] Gripenberg G and Norros I, On the prediction of fractional Brownian motion, *J. Appl. Prob.* 33 (1996), 400-410.
 - [31] Ibrahim M.O, Ejieji C.N and Egbetade S.A (2013): A mathematical model for the epidemiology of tuberculosis with estimate of the basic reproduction number. *IOSR Journal of Mathematics*, 5(5): 46-52.
 - [32] James M., Hyman E., Ann Stanley, Using mathematical models to understand the AIDS epidemic, *Math. Biosci.* 90 (1988) 415-473.
 - [33] Jacquez J.A, Simon C.P, Koopman J.S, Structured mixing: Heterogenous mixing by the definition of activity groups, in: C. Castillo-Chavez (Ed.), *Mathematical and Statistical Approaches to AIDS Epidemiology*, in: *Lecture Notes in Biomath.*, vol. 83, Springer, Berlin, 1989, pp. 301-315.
-

-
- [34] Jacquez J.A, Simon C.P, Koopman J.S, The reproduction number in deterministic models of contagious disease, *Comments Theor. Biol.* 2 (1988) 159-209.
 - [35] Kalu A.U, and Inyama S.C (2012): Mathematical model of the role of vaccination and treatment on the transmission dynamics of tuberculosis. *Gen. Math. Notes*, 11(1): 10-23.
 - [36] Karatzas I and Shreve S.E, *Brownian motion and stochastic calculus*, 2nd ed, Springer, New York, 1991.
 - [37] Kermack W.O and McKendrick A.G (1927): A contribution to the mathematical theory of epidemics. *Proc. R. Soc.* 115: 700-721.
 - [38] Korthals Altes H, Wodarz D, Jansen V, The dual role of CD4 T helper cells in the infection dynamics of HIV and their importance for vaccination, *J. Theoret. Biol.* 214 (2002) 633-646.
 - [39] Lamperti J, *Probability*, Benjamin Inc., New York, 1966.
 - [40] Lamperti J, A simple construction of certain diffusion processes, *J. Math. Kyôto* (1964), 161-170.
 - [41] LaSalle J.P, *Stability theory for ordinary differential equations, stability theory for ordinary differential equations*. *J Differ Eq* 1968; 41:57-65.
 - [42] LaSalle J.P, *The stability of dynamical systems*, Philadelphia, PA: Society for Industrial and Applied Mathematics; 1976. With an appendix: "Limiting equations and stability of nonautonomous ordinary differential equations" by Z. Artstein, *Regional Conference Series in Applied Mathematics*.
 - [43] LaSalle J.P, *Stability theory and invariance principles, Dynamical systems (Proc. Internat. Sympos., Brown Univ., Providence, R.I., 1974), Vol. I, pp. 211-222. Academic Press, New York, 1976.* 34-35
 - [44] Lin X, So J, Global stability of the endemic equilibrium and uniform persistence in epidemic models with subpopulations. *Journal of the Australian Mathematical Society*, 1993, 34(3): 282-295.
 - [45] Lin S.J, *Stochastic analysis of fractional Brownian motions, Stochastics and stochastics reports* 55.1-2 (1995): 121-140.
 - [46] Lipster R.S and Shiriyayev A.N, *Theory of Martingales*, Kluwer Academic, Dordrecht, 1986.
 - [47] Mao X, *Stochastic differential equations and applications*, Horwood Publishing Limited, Chichester, 1997.
 - [48] Mao X, Marion G, Renshaw E, Environmental Brownian noise suppresses explosions in population dynamics, *Stochastic Process. Appl.* 97 (2002) 95-110.
 - [49] Mao X, Yuan C, Zou J, *Stochastic differential delay equations of population dynamics*, *J. Math. Anal. Appl.* 304 (2005) 296-320.
 - [50] MAP 1998 The status and trend of HIV epidemics around the world. Provisional Report (available on www.unaids.org or www.unaids.com).
 - [51] McCluskey C, Global stability for a class of mass action systems allowing for latency in tuberculosis. *Journal of Mathematical Analysis and Applications*, 2008, 338: 518-555.
-

-
- [52] McCluskey C, Lyapunov functions for tuberculosis models with fast and slow progression. *Mathematical Biosciences and Engineering*, 2006, 3: 603-614.
- [53] McKean H, *Stochastic Integrals*, Academic Press, New York, 1969.
- [54] Mechanisms of CD4 cell destruction, www.aidsmap.com, 2006.
- [55] Moghadas S, Gumel A, Analysis of a model for transmission dynamics of tb. *Canadian Applied Mathematics Quarterly*, 2002, 10: 411-427.
- [56] Murphy B, Singer B, Kirschner D, Comparing epidemic tuberculosis in demographically distinct populations. *Mathematics in Bioscience*, 2002, 180: 161-185.
- [57] Murphy B, Singer B, Kirschner D, On the treatment of tuberculosis in heterogeneous populations. *Journal of Theoretical Biology*, 2003, 223: 391-404.
- [58] Nelson P, Perelson A, Mathematical analysis of delay differential equation models of HIV-1 infection, *Math. Biosci.* 179 (2002) 73-94.
- [59] Oksendal B.K, *Stochastic differential equations: An introduction with applications*, 4th ed, Springer-Verlag, Berlin 1995.
- [60] Oname A and Inyama S.C (2014): Stochastic model and simulation of the prevalence of measles. *Int. J. of Math. Sci. & Engg. Appls*, 8(1): 311-323.
- [61] Perelson A, Neumann A, Markowitz M, Leonard J, Ho D, HIV-1 dynamics in vivo: Virion clearance rate, infected cell life-span, and viral generation time, *Science* 271 (1996) 1582-1586.
- [62] ReVelle C.S, Lynn W.R and Feldman F (1967): Mathematical models for the allocation of tuberculosis control activities in developing nations. *Am. Rev.Respir. Dis.* 96: 893-909.
- [63] Song B, Castillo-Chavez C, Aparicio J, Tuberculosis models with fast and slow dynamics: the role of close and casual contacts. *Mathematical Biosciences*, 2002, 180: 187-205.
- [64] Sussmann H, On the gap between deterministic and stochastic ordinary differential equations, *Ann. Probability*. 6 (1978), 19-41.
- [65] Sussman H, An interpretation of stochastic differential equations as ordinary differential equations which depend on the sample point. *Bull. Amer. Math. Soc.* 83 (1977) 296 - 298.
- [66] Tewa J.J, Dimi J.L, Bowong S, Lyapunov functions for a dengue disease transmission model. *Chaos Solitons & Fractals*, 2009, 36(2): 936-941.
- [67] Tuckwell H.C, Wan F.Y.M, Nature of equilibria and effects of drug treatments in some simple viral population dynamical models, *IMA J. Math. Appl. Med. Biol.* 17 (2000) 311-327.
- [68] UNAIDS 1998 AIDS epidemic update: December 1998 AIDS joint United Nations programme on HIV/AIDS (available on www.unaids.org or www.unaids.com).
- [69] Verotta D, Schaedeli F, Non-linear dynamics models characterising long-term virological data from AIDS clinical trials, *Math. Biosci.* 176(2002) 163-183.
-

- [70] Waller H.T, Geser A and Anderson S (1962): The use of mathematical models in the study of the epidemiology of tuberculosis. *Am. J. Publ. Health*, 52: 1002-1013.
- [71] Wiener N, Paley R and Zygmund A, Notes on random functions, *Math. Z.* 37 (1959) 647 - 668.
- [72] Willems J.L 1970 *Stability Theory of Dynamical Systems*. New York: Wiley.
- [73] World Health Organization (2010): Tuberculosis Fact sheet. www.who.org.
- [74] World Health Organization (2007): Vaccine preventable diseases: Monitoring system, Global summary. www.who.org.
- [75] World Health Organisation (1993): Global programme on AIDS. The HIV/AIDS Pandemic: 1993 Overview. Geneva: WHO.
- [76] Zhien Ma, Jia Li, *Dynamical Modeling and Analysis of Epidemics*, World Scientific Publishing Co. Pte. Ltd, 2009.