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DETERMINISTIC AND STOCHASTIC MODEL FOR THE TRANSMISSION OF LASSA FEVER

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ABSTRACT

Many models on the transmission dynamics of Lasser fever were based on purely deterministic approach. This approach does not put into cognizance randomness which is inherent in disease transmission resulting from differences in immunity levels, contact patterns, hygienic practices and mutation rates among so many other possibilities. In this work, we attempt to demonstrate the impact of uncertainties in the mode of transmission of Lassa fever by subjecting the dynamics to some white noise modeled by the Brownian motion as a Wiener process. An existing deterministic model involving the Susceptible, Exposed, Infected and Recovered (SEIR) individuals was transformed into a stochastic differential equation model by applying the procedure proposed by Allen et al (2008). The resulting system of Stochastic Differential Equations (SDE) was solved numerically using the Milstein scheme for SDEs. An algorithm for the method was developed and implemented in Python programming language. Numerical simulations of the model was done using four sets of parameters; λ , $\mu, \gamma, \kappa, \beta$ representing the natural birth rate, the natural death rate , the recovering rate from infected to recovered, transmission rate from exposed to infected ,transmission rate from susceptible to exposed are carried out to investigate the transmission dynamic of Lassa fever. The results of the simulations indicate that randomness does affect transmission of Lassa fever. We therefore recommend that factors such as social behavior, hygienic practices, contact patters, mutation rate should be considered while formulating mathematics models of disease transmission.

Keywords: Deterministic model, Stochastic model, Disease transmission, Lassa fever

INTRODUCTION

Lassa Fever is an active Viral Hemorrhagic Fever (VHF) that is known to be endemic in Various West African countries including Nigeria. A viral disease majorly caused by Lassa Virus. The earliest record of the disease was in the 1950s but the virus was isolated by the Centre For Disease Control (CDC), Atlanta, USA in 1969, from a sample taken from a missionary workers in a town called Lassa in the Yedseram River valley in the present Borno State of Northern Nigeria (Tara, 2004). It is transmitted through Human – Human; Human - Environment – Human; Reservoir – Humans and Reservoir - Vector – Human. These various methods of transmission make Lassa fever to be endemic whenever the outbreak occurs, (Fisher-Hoch, Tomori, Nasidi, Perez-Oronoz, Fakile, Hutwagner, 1995).

So much efforts have been done over the years to study mathematically the dynamics of the spread and control of Lassa fever since its initial discovery in Nigeria such as the work of Ogabi, Olusa and Madufor (2012), Bawa, Abdulraham and Jimoh (2013) and others, just to mention a few. However, their works were based purely on deterministic approach.

Deterministic models constitute a vast majority of the models in existing literature and are formulated in terms of Ordinary Differential Equation (ODE). Consequently, they predict the same dynamic for an infective process given the same initial conditions. Real life scenarios are often not straight forward as the approach posits as there are several effects that can introduce some form of uncertainties to real life dynamics. It is our view therefore that uncertainty should be included when modelling the dynamics of the spread of a disease. This agrees with the view of Gamboa and Lopez-Herrero (2013) that stochastic models analogous to ODE models which takes into account the random nature of the events capable of addressing issues such as probabilities of major outbreaks, disease extinction and general statistical analysis of some relevant epidemic descriptors are suitable for disease epidemic models.

The main advantage of deterministic modeling over stochastic modeling lay on the simplicity to analyze. Stochastic modeling should be preferred over deterministic modeling because, most natural way of studying the spread of disease is stochastic because it defines the probability of transmission of disease between individuals (Andersson & Briton, 2000). Moreso, some phenomena are genuinely stochastic and do not satisfy the law of large number. In such cases, deterministic models are not the most appropriate approach for modeling the start of an epidemic because the number of infectious individuals is small. Furthermore, it terms of extinction of endemic diseases, it can only be analyzed by stochastic model because the extinction occurs when the epidemic process deviates from expected level, (Allen, 2007). Kloeden & Platen (1999) put it concisely by stating that stochastic models are, in general, more realistic since the spread of diseases is stochastic in nature.

According to Britton (2009), deterministic models are primarily aimed at large community but stochastic models seem to be more appropriate to describe evolution of infective process evolving in a small community. For example, consider an epidemic outbreak in an uniformly homogenous community, it seems sensible to assume some uncertainty/randomness in the final number infected. Also, if the basic reproduction number, $R_o > 1$ and the community is large but the outbreak is initiated by only one (or a few) initial infective, it should be possible that, by chance, the epidemic never takes off.

Taylor and Karlin (1998), stated that a deterministic model predicts a single outcome from a given set of circumstances. A stochastic model predicts a set of possible outcomes weighted by their likelihoods or probabilities. A deterministic model is specified by a set of equations that describe exactly how the system will evolve over time. In a stochastic model, the evolution is at least random and if the process is run

several times, it will not give identical results. Different runs of a stochastic process are often called realization of the process. Deterministic models are generally easier to analyze than stochastic model.

Related Works

Several works have been done on stochastic epidemic models. Tuckwell and William (2006) formulated some properties of a simple stochastic epidemic model of SIR type. The model is Markovian of the SIR type in which the total population is constant and individual meet a random number of other individuals at each time step. In the model with a finite recovery time R, simulations revealed large variability in both the total number of infected individuals and in the total duration of the epidemic, even when the variability in number of contacts per day is small. In the case of no recovery, $R = \infty$, a formal diffusion approximation was obtained for the number of cases infected.

Ogwuche, Iortyer, Emonyi and Ali M. (2023) formulated an SDE model for the transmission of Tuberculosis (TB). In their work, a deterministic model for the transmission of TB was presented and then transformed into a system of stochastic differential equation model. The Euler- Maruyama method was used for the simulation.

Gamboa and Lopez – Herrero (2018) formulated the number of periodic inspections during outbreaks of discrete – time stochastic SIS epidemic model. The underlying Mathematical model involves a discrete time Markov chain with a single absorbing state, the number of inspections in an outbreak is a first passage time into this absorbing state. Cumulative probabilities were numerically determined from a recursive algorithm and expected value comes from explicit expressions.

Allen and Linda (2017) formulated a primer on stochastic epidemic models. The paper focused on the formulation, numerical simulation and analysis of stochastic epidemic model. Specially, models were formulated for continuous – time Markov chain and stochastic differential equations. Some well – known examples were used for illustration and analytical methods for approximating the probability of a disease outbreak were also discussed.

Rao (2014) developed a dynamic analysis of a stochastic SIR epidemic. He assumed that stochastic perturbations are of a white noise type which is directly proportional to the distance of three variables from the steady state values, respectively. By constructing suitable Lyapunov functions and applying Itô's formula, some qualitative properties were obtained. Finally, he deducted that when the intensities of noise satisfy some conditions and are not sufficiently large, the population of the stochastic model may be stochastically permanent.

Lopez – Herrero (2013) formulated an epidemic transmission on SEIR stochastic models with non – linear incidence rate. Within a stochastic frame work, two random variables were defined to describe the variation of the number of secondary cases produced by an index case of a closed population. Computational algorithms were presented in order to characterize both random variables. He stated that a possible extension of his paper could be to study the epidemic expansion when control measures such as vaccination or isolation are implemented.

Allen (2008) developed stochastic differential equation model using a procedure similar to that used in the advancement of many ordinary differential equation models. He considered two-state dynamical process where $S_1(t)$ and $S_2(t)$ represent the values of two states in the system at time t. He assumed that in a small time interval Δt,S_1 can change by $-\lambda_1$,0 or λ_1 and S_2 can change by $-\lambda_2$,0 or λ_2 where $\lambda_1,\lambda_2 \geq 0$. He further let $\Delta S \equiv [S_1, S_2]^T$which is the change in a small-time interval Δt.There are eight possible changes for the two states in the time interval Δt not including the case where there is no change in the time interval. The changes $\lambda_i$are assumed to be non-negative. All probabilities may depend on $S_1(t), S_2(t)$ and time t. He also assumed that the probabilities for the change are proportional to the time interval Δt.He finalized by calculating the covariance matrix for the change.

In summary, many researchers in recent times have worked on the deterministic Mathematical modeling of Lassa fever outbreak but no work has been extensively done stochastic Mathematical modeling of Lassa fever.

MATERIALS AND METHODS

Consider four (4) typical compartmental deterministic mathematical model using the S(t),E(t), I(t) and R(t) to give a better understanding on the transmission dynamic of Lassa Fever. N(t) is the total population size given by:

$$N(t) = S(t) + E(t) + I(t) + R(t) \quad (1)$$

Parameters of the Model

The parameter of the basic Lassa Fever model is defined as follows:

Table 1: Parameters of a basic Lassa Fever Model

Symbol	Description
μ	Natural death rate
λ	Natural birth rate
γ	Recovery rate from infected to recovered state
κ	Transmission rate from exposed to infected state
β	Transmission rate from susceptible to exposed state

Assumptions of the Model

i. All the recruits are neither immune nor infected.
ii. Recruitment into the Susceptible class is done by birth.
iii. The virus does not kill the vector (their death can be naturally or accidental) and individuals in each class can die a natural death.
iv. Humans cannot transmit the infection to rodents as it is assumed that they have no Lassa fever in their bodies.
v. Infected immigrants are not included because it is assumed that most people who are sick will not travel.
vi. There is no immigration of the recovered humans. Humans leave the population through natural death.
vii. The infective period of the vector ends with its death and therefore the vector does not recover from being infective.

The Model Equations

The dynamics of transmission of Lassa fever can be represented schematically as shown Figure 1.

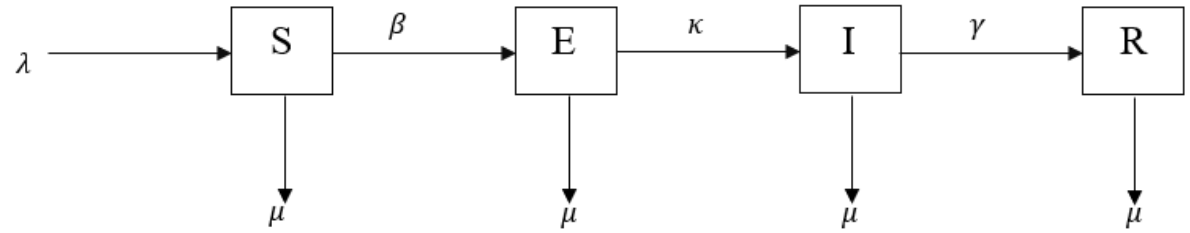

Figure 1: The Basic Lassa fever Transmission Diagram

The assumptions in section 3.3 and the model flowchart together lead to the following system of ordinary differential equations which describe the transmission dynamics of the disease as:

$$\frac{dS}{dt} = \lambda - (\beta + \mu)S$$
$$\frac{dE}{dt} = \beta - (\mathrm{k} + \mu)E$$
$$\frac{dI}{dt} = \mathrm{k}E - (\gamma + \mu)I \quad (2)$$
$$\frac{dR}{dt} = \gamma I - \mu R$$

Basic Properties of the Deterministic Model

3.5.1 Invariant Property

Theorem 3.1: The Closed Set

D = **{**(S, E, I, R,)ϵR_+^4: S+E+I+R ≥ 0**}**

Is positively invariant for the model equation (3.2) with non – negative initial condition in R_+^4

Proof:

Considering the total population size N which can be determined by N= S+E+I+R as stated in (3.1).

Differentiating (3.1) gives

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \quad (3)$$

Substituting (3.2) into (3.3), we have:

$$\frac{dN}{dt} = \lambda - \beta S - \mu S + \beta S - \mathrm{K}E - \mu E + \mathrm{K}E - (\gamma + \mu)I + \gamma I - \mu R$$
$$\frac{dN}{dt} = \lambda - (S + E + I + R)\mu$$
$$\frac{dN}{dt} = \lambda - N\mu$$
$$\frac{dN}{dt} \leq \lambda - N\mu$$
$$\frac{dN}{dt} + N\mu \leq \lambda$$

and using Birkhoff and Rota(1989) Theorem on differential inequality, we have

$0 \leq N \leq \frac{\lambda}{\mu}$, hence

$\lambda - N\mu \geq \mathrm{K}e^{-ut}$ Where K is constant

Taking limit as t ⟶ ∞

$$N \leq \frac{\lambda}{\mu} \quad (4)$$

Based on (3.4), all feasible solution of the human population of the model system (2) are in the region.

$$\mathrm{D} = \{(\mathrm{S}, \mathrm{E}, \mathrm{I}, \mathrm{R}) \epsilon R_+^4 : \mathrm{N} \leq \frac{\lambda}{\mu}\} \quad (5)$$

which is a positively invariant set under the flow induced by the model (2). Hence the system (2) is epidemiologically meaningful and mathematically well posed in the domain D. Therefore, in this domain it is sufficient to consider the dynamic of the flow generated by the model (3.2). In addition, the existence, uniqueness and continuation of results hold for the system.

Existence and stability of steady-state solutions

The E = (S^*,E^*,I^*,R^*) is the steady state of the system (3.2) which can be calculated by setting the right hand side of the model (3.2)to zero, giving us the following;

$$\lambda - \beta S - \mu S = 0$$
$$\beta S - \mathrm{K}E - \mu E = 0 \quad (6)$$
$$\mathrm{K}E - (\gamma + \mu)I = 0$$
$$\gamma I - \mu R = 0$$

Existence of disease-free equilibrium (DFE)

Disease-free equilibrium points are steady-state solutions where there is no disease. We defined the 'diseased' classes as the human population that is infected, that is; I and E, in the system (2)

At equilibrium states the rate of change of the state varies with respect to time is Zero, i.e.

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

Let E_0 denotes the disease-free equilibrium. We set $S^* = E^* = I^* = R^* = 0$

Hence, the DFE of the basic Lassa fever model (3.2) is given by

$$E_0 = (S^* E^*, I^*, R^*,) = [\frac{\lambda}{\mu}, O, 0, 0] \quad (7)$$

The existence of the trivial equilibrium point

For as long as the birth rate λ is not zero, the population will not be extinct. This implies that there is no trivial equilibrium point, thus

$(S^*, E^* I^*, R^*) \neq (0, 0, 0, 0)$

The Basic Reproduction Number, R_o

The basic reproduction number denoted by R_o is the number of secondary cases of infection emanating from a single infection source i.e. produced by a typical infectious rodent or an individual. R_o= 1 implies that the disease is at threshold below which the generation of secondary case is insufficient to maintain the infection within human community. If R_o< 1, it implies that, an infected individual produces less than one new infected individual during the infectious period and the infection can be eradicated. Conversely if Ro>I, it implies that each infected individual produces on average more than one new infected individual and the disease will invade the population i.e. the DFE is unstable and invasion is always possible.

To obtain R_o of our model, we explore using the next generation matrix operator approach developed by van den Driessche and Watmough (2002). Let the next generation matrix be G.It is comprised of two parts; F and V^{-1} where

$$\mathrm{F} = \left[\frac{\partial F_i(X_0)}{\partial x_j}\right] \quad (8)$$
$$\mathrm{V} = \left[\frac{\partial V_{i(X_0)}}{\partial x}\right] \quad (9)$$

Where
F_i =The new infections
V_i =Transfers of infections from one compartment to another
X_0 = The disease free equilibrium
R_0 = The dorminant eigenvalue of the matrix
G= FV^{-1} (10)
The infection classes are E and I, Hence
$F_1 = \beta S, \qquad F_2 = 0$
$\frac{\partial F_1}{\partial I}/E_0 = \beta S^* = \frac{\beta\lambda}{\mu}, \quad \frac{\partial F_1}{\partial E}/E_0 = 0$
$\frac{\partial F_2}{\partial I}/E_0 = 0, \quad \frac{\partial F_2}{\partial E}/E_0 = 0$
Using

$$\text{F}=\begin{pmatrix}\frac{\partial F_1}{\partial I}/E_0 & \frac{\partial F_1}{\partial E}/E_0\\ \frac{\partial F_2}{\partial I}/E_0 & \frac{\partial F_2}{\partial E}/E_0\end{pmatrix}=\begin{pmatrix}\frac{\beta\lambda}{\mu} & 0\\ 0 & 0\end{pmatrix} \quad (11)$$

Next
Let
$V_1 = -[-(\kappa+\mu)E]$
$= \quad [(\kappa+\mu)E]$
$V_2 = -[\kappa E - (\gamma+\mu)I]$
$= -\kappa E + (\gamma+\mu)I$
$\frac{\partial V_1}{\partial I}/E_0 = 0, \quad \frac{\partial V_1}{\partial E}/E_0 = (\kappa+\mu)$
$\frac{\partial V_2}{\partial I}/E_0 = (\gamma+\mu), \quad \frac{\partial V_2}{\partial E}/E_0 = -\kappa$

$$\text{V}=\begin{pmatrix}\frac{\partial V_1}{\partial I}/E_0 & \frac{\partial V_1}{\partial E}/E_0\\ \frac{\partial V_2}{\partial I}/E_0 & \frac{\partial V_2}{\partial E}/E_0\end{pmatrix}=\begin{pmatrix}0 & \kappa+\mu\\ \gamma+\mu & -\kappa\end{pmatrix} \quad (12)$$

Recall that if

$$\text{A}=\begin{pmatrix}a & b\\ c & d\end{pmatrix} \text{then } \text{A}^{-1}=\frac{1}{\text{ad}-\text{bc}}\begin{pmatrix}\text{d} & -\text{b}\\ -\text{c} & \text{a}\end{pmatrix}$$

Hence

$$V^{-1}=\frac{1}{0-(\gamma+\mu)(\kappa+\mu)}\begin{pmatrix}-\kappa & -(\kappa+\mu)\\ -(\gamma+\mu) & 0\end{pmatrix}$$

$$=\begin{pmatrix}\frac{\kappa}{(\gamma+\mu)(\kappa+\mu)} & \frac{-(\kappa+\mu)}{-(\gamma+\mu)(\kappa+\mu)}\\ \frac{-(\gamma+\mu)}{-(\gamma+\mu)(\kappa+\mu)} & 0\end{pmatrix}$$

$$=\begin{pmatrix}\frac{\kappa}{(\gamma+\mu)(\kappa+\mu)} & \frac{1}{(\gamma+\mu)}\\ \frac{1}{(\kappa+\mu)} & 0\end{pmatrix} \quad (13)$$

Therefore

$$\text{G}=FV^{-1}=\begin{pmatrix}\frac{\beta\lambda}{\mu} & 0\\ 0 & 0\end{pmatrix}\begin{pmatrix}\frac{\kappa}{(\gamma+\mu)(\kappa+\mu)} & \frac{1}{(\gamma+\mu)}\\ \frac{1}{\kappa+\mu} & 0\end{pmatrix}$$

$$=\begin{pmatrix}\frac{\beta\lambda\kappa}{(\gamma+\mu)(\kappa+\mu)\mu} & \frac{\beta\lambda}{\mu(\kappa+\mu)}\\ 0 & 0\end{pmatrix}$$

We have to find the most dominant eigenvalue of G
$|G-\xi I| = 0$

$$\begin{vmatrix}\frac{\beta\lambda\text{K}}{\mu(\gamma+\mu)(\text{K}+\mu)} & \frac{\beta\lambda}{\mu(\text{K}+\mu)}\\ 0 & -\xi\end{vmatrix}=0$$

$$-\xi\left[\frac{\beta\lambda\text{K}}{\mu(\gamma+\mu)(\text{K}+\mu)}\right]-0=0$$

$$\xi = 0 \; or \; \xi=\frac{\beta\lambda\text{K}}{\mu(\gamma+\mu)(\text{K}+\mu)}$$

Therefore, by calculation, R_0 is defined. Mathematically,

$$R_0=\frac{\beta\lambda\text{K}}{\mu(\gamma+\mu)(\kappa+\mu)} \quad (14)$$

Formulation of the Stochastic Model for the Transmission of Lassa Feaver

Using the first modeling procedure developed by Allen et al (2008), we derive the stochastic model for the deterministic model (3.1) above

Table 2: Table of transition probabilities

Change	Probability	Event
$[\mathbf{1000}]^T$	$P_1 = \lambda\Delta t$	Birth of a susceptible human.
$[-\mathbf{1000}]^T$	$P_2 = \mu S\Delta t$	Susceptible dies natural death.
$[-\mathbf{1100}]^T$	$P_3 = \beta S\Delta t$	Susceptible becomes exposed.
$[\mathbf{0-100}]^T$	$P_4 = \mu E\Delta t$	Exposed dies natural death.
$[\mathbf{0-110}]^T$	$P_5 = KE\Delta t$	Exposed becomes infected.
$[\mathbf{00-10}]^T$	$P_6 = \mu I\Delta t$	Infected dies natural death.
$[\mathbf{00-11}]^T$	$P_7 = \gamma I\Delta t$	Infected becomes recovered.
$[\mathbf{000-1}]^T$	$P_8 = \mu R\Delta t$	Recovered dies natural death.

The drift vector is defined as:

$$\vec{F} = \sum_{j=1}^{8} p_j \overrightarrow{\lambda_j} \quad (15)$$

Where $\overrightarrow{\lambda_j}$ and p_j are the random changes and the transition probabilities as defined in the table above.

$$\vec{F} = P_1\begin{bmatrix}1\\0\\0\\0\end{bmatrix}+P_2\begin{bmatrix}-1\\0\\0\\0\end{bmatrix}+\text{P}_3\begin{bmatrix}-1\\1\\0\\0\end{bmatrix}+P_4\begin{bmatrix}0\\-1\\0\\0\end{bmatrix}+P_5\begin{bmatrix}0\\-1\\1\\0\end{bmatrix}+P_6\begin{bmatrix}0\\0\\-1\\0\end{bmatrix}+P_7\begin{bmatrix}0\\0\\-1\\1\end{bmatrix}+P_8\begin{bmatrix}0\\0\\0\\-1\end{bmatrix}$$

$$=\begin{bmatrix}\lambda\\0\\0\\0\end{bmatrix}+\begin{bmatrix}-\mu S\\0\\0\\0\end{bmatrix}+\begin{bmatrix}-\beta S\\\beta S\\0\\0\end{bmatrix}+\begin{bmatrix}0\\-\mu E\\0\\0\end{bmatrix}+\begin{bmatrix}0\\-\text{K}E\\\text{K}E\\0\end{bmatrix}+\begin{bmatrix}0\\0\\-\mu I\\0\end{bmatrix}+\begin{bmatrix}0\\0\\-\gamma I\\\gamma I\end{bmatrix}+\begin{bmatrix}0\\0\\0\\-\mu R\end{bmatrix}$$

Hence, the drift vector $\vec{F}$ of order 4x 1, is given by

$$\vec{F} = \begin{bmatrix} \lambda - (\beta + \mu)S \\ \beta S - (\mathrm{K} + \mu)E \\ \mathrm{K}E - (\gamma + \mu)I \\ \gamma I - \mu R \end{bmatrix} \quad (16)$$

Similarly, the Covariance matrix which is the volatility coefficient is defined as

$$\mathrm{V} = \sum_{j=1}^{8} P_{\mathrm{j}} \vec{\lambda}_{\mathrm{j}} (\vec{\lambda}_{\mathrm{j})\,\mathrm{T}} \quad (17)$$

$$\mathrm{V} = P_1 \begin{bmatrix} 1 \\ 0 \\ 0 \\ 0 \end{bmatrix} [1\ 0\ 0\ 0] + P_2 \begin{bmatrix} -1 \\ 0 \\ 0 \\ 0 \end{bmatrix} [-1\ 0\ 0\ 0] + P_3 \begin{bmatrix} -1 \\ 1 \\ 0 \\ 0 \end{bmatrix} [-1\ 1\ 0\ 0] + P_4 \begin{bmatrix} 0 \\ -1 \\ 0 \\ 0 \end{bmatrix} [0\ -1\ 0\ 0] +$$

$$P_5 \begin{bmatrix} 0 \\ -1 \\ 1 \\ 0 \end{bmatrix} [0\ -1\ 1\ 0] + P_6 \begin{bmatrix} 0 \\ 0 \\ -1 \\ 0 \end{bmatrix} [0\ 0\ -1\ 0] + P_7 \begin{bmatrix} 0 \\ 0 \\ -1 \\ 1 \end{bmatrix} [0\ 0\ -1\ 1] + P_8 \begin{bmatrix} 0 \\ 0 \\ 0 \\ -1 \end{bmatrix} [0\ 0\ 0\ -1]$$

Multiplying the covariance matrix, we have:

$$\mathrm{V} = \begin{bmatrix} \lambda + \mu S + \beta S & -\beta S & 0 & 0 \\ -\beta S & \beta S + \mu E + \mathrm{K}E & -\mathrm{K}E & 0 \\ 0 & -\mathrm{K}E & \mathrm{K}E + \mu I + \gamma I & -\gamma I \\ 0 & 0 & -\gamma I & \gamma I + \mu R \end{bmatrix} \quad (18)$$

Consequently, the resulting stochastic differential equation model of the equivalent ordinary differential equation model in equation(3.2) is given by:

$$d\vec{X}(t) = \vec{F}\left(t, \vec{X}(t)\right) dt + \vec{V}^{\frac{1}{2}}\left(t, \vec{X}(t)\right) d\vec{W}(t)\ ;\ \vec{X}(0) = [X_1(0), X_2(0), X_3(0), X_4(0)]^{\mathrm{T}} \quad (19)$$

where $\vec{F}$ and $\vec{V}$ are as defined in equation (3.16) and (3.17) respectively.

Method of Solution

The Euler-Maruyama method is used for the simulation. For the Ito SDE

$$\mathrm{d}X_t = \mathrm{a}(t, X_t)dt + b(t, X_t)\, dW_t \quad (4.1)$$

The Euler – Maruyama method is given by:

$$Y_{n+1} = Y_n + a(\tau_n, Y_n)(\tau_{n+1} - \tau_n) + b(\tau_n, Y_n)(W_{\tau_{n+1}} \uparrow - W_{\tau_n}) \quad (20)$$

f the Ito process.

The Euler Scheme

According Kloeden, P.E. and Platen Eckhard (2007), one of the simplest time discrete approximations of an Ito process is the Euler approximation, or the Euler-Maruyama approximation as it is sometimes called. Considering an Ito process $\mathrm{X} = \{X_t, t_0 \le t \le T\}$satisfying the scalar stochastic differential equation:

$$\mathrm{d}X_t = \mathrm{a}(t, X_t)dt + b(t, X_t)\, dW_t \quad (21)$$

on $t_0 \le \mathrm{t} \le \mathrm{T}$ with the initial value

$$X_{t_0} = X_0 \quad (22)$$

For a given discretization $t_0 = \tau_0 < \tau_1 < \cdots < \tau_n < \cdots \tau_N = T$ of the time interval $[t_0, T]$,an Euler approximation is a continuous time stochastic process $\mathrm{Y} = \{Y(t), t_0 \le t \le T\}$ satisfying the iterative scheme

$$Y_{n+1} = Y_n + a(\tau_n, Y_n)(\tau_{n+1} - \tau_n) + b(\tau_n, Y_n)(W_{\tau_{n+1}} - W_{\tau_n}) \quad (23)$$

In the 1- dimensional case, d= m= 1, the Euler scheme has the form

$$Y_{n+1} = Y_n + \mathrm{a}\Delta + b\Delta W \quad (24)$$

Where $\Delta = \tau_{n+1} - \tau_n = I_{(0)} = J_{(0)}$ (25)

Is the length of the time discretization subinterval $[\tau_n, \tau_{n+1}]$ and

$$\Delta W = W_{\tau_{n+1}} - W_{\tau_n} \quad (26)$$

$$Y_{n+1}^k = Y_n^k + a^k\Delta + b^k\Delta W, \quad (27)$$

For k = 1 , …, d, where the drift and diffusion coefficients are d- dimensional vectors $\mathrm{a} = (a^1, \ldots, a^d)$ and $\mathrm{b} = (b^1, \ldots, b^d)$.

For the general multi- dimensional case with d, m = 1, 2…the kth component of the Euler scheme has the form

$$Y_{n+1}^k = Y_n^k + a^k\Delta + \sum_{j=1}^{m} b^{k,j}\Delta W^j \quad (28)$$

where

$$\Delta W^j = W_{\tau_{n+1}}^j - W_{\tau_n}^j = I_{(j)} = J_{(j)} \quad (29)$$

Is the $\mathrm{N}(0; \Delta)$ distributed increment of the jth component of the m- dimensional standard Wiener process W on $[\tau_n, \tau_{n+1}]$ and ΔW^{j_1} *and* ΔW^{j_2} are independent for $j_1 \neq j_2$. The diffusion coefficient $\mathrm{b} = [b^{k,j}]$is a d× m matrix.

RESULTS AND DISCUSSION

The resulting model in equation (3.18) was simulated using the Euler-Maruyama scheme in (20) using the parameter and initial condition:

Table 3: Table of value for the parameters

Parameter	Value
λ	0.02
μ	0.02
β	0.05
K	0.1
γ	0.05
S_0	1000
E_0	10
I_0	1
R_0	0

The result for the simulation using these values for five realizations are as shown in Figure 4.1 – 4.5

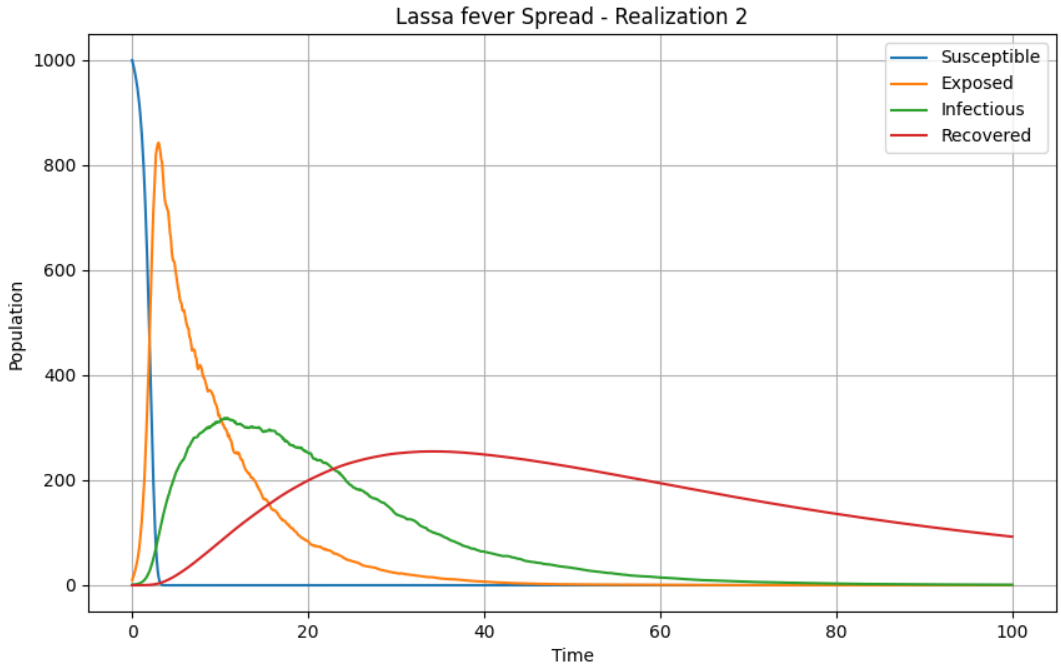

Figure 2: Realization 1 of Lassa fever transmission dynamics

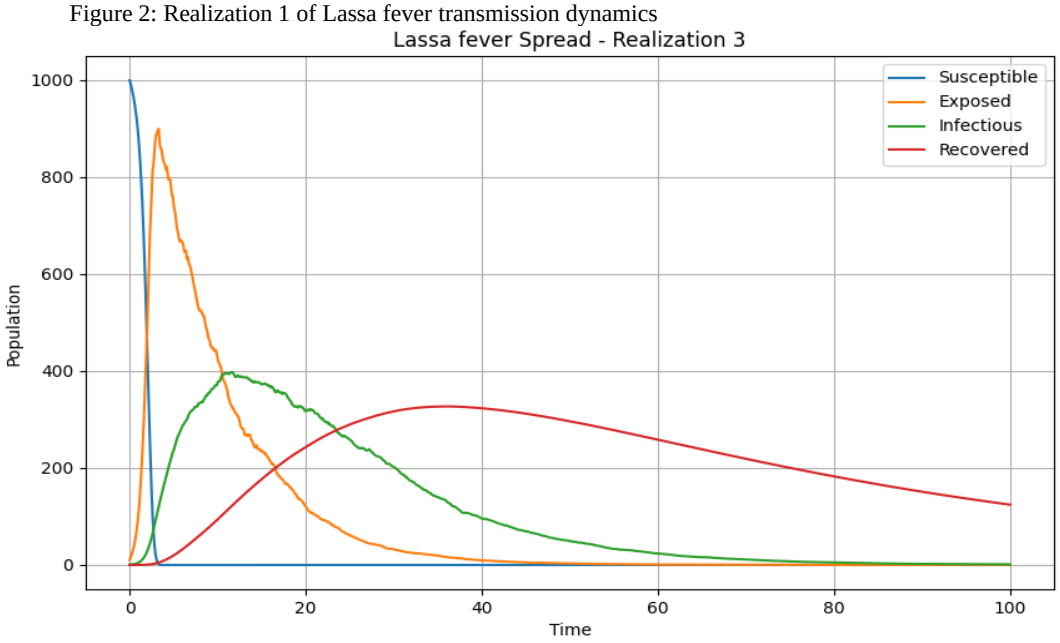

Figure 3: Realization 2 of Lassa fever transmission dynamics

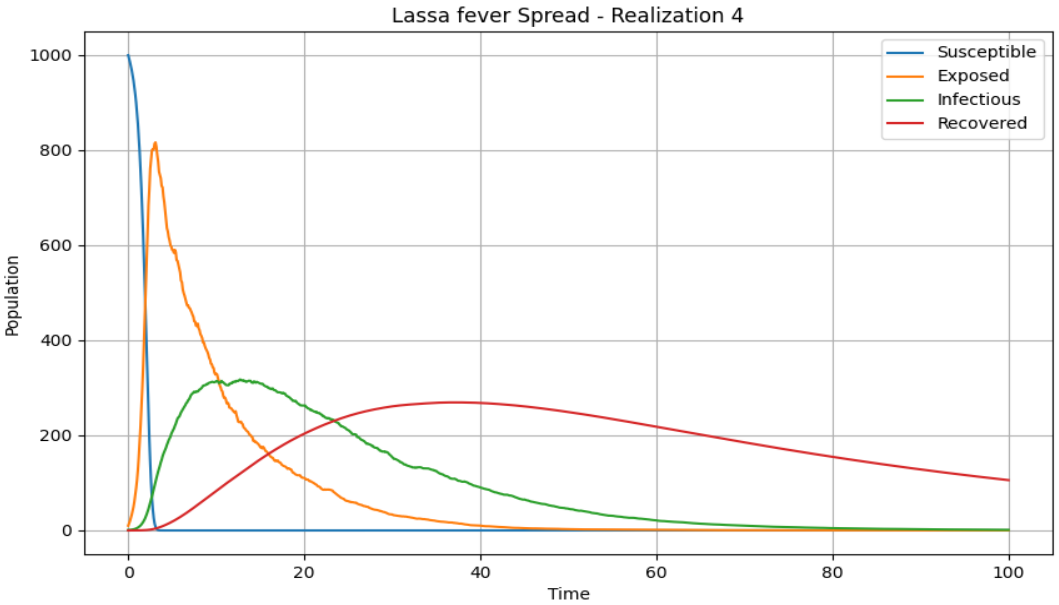

Figure 4: Realization 3 of Lassa fever transmission dynamics

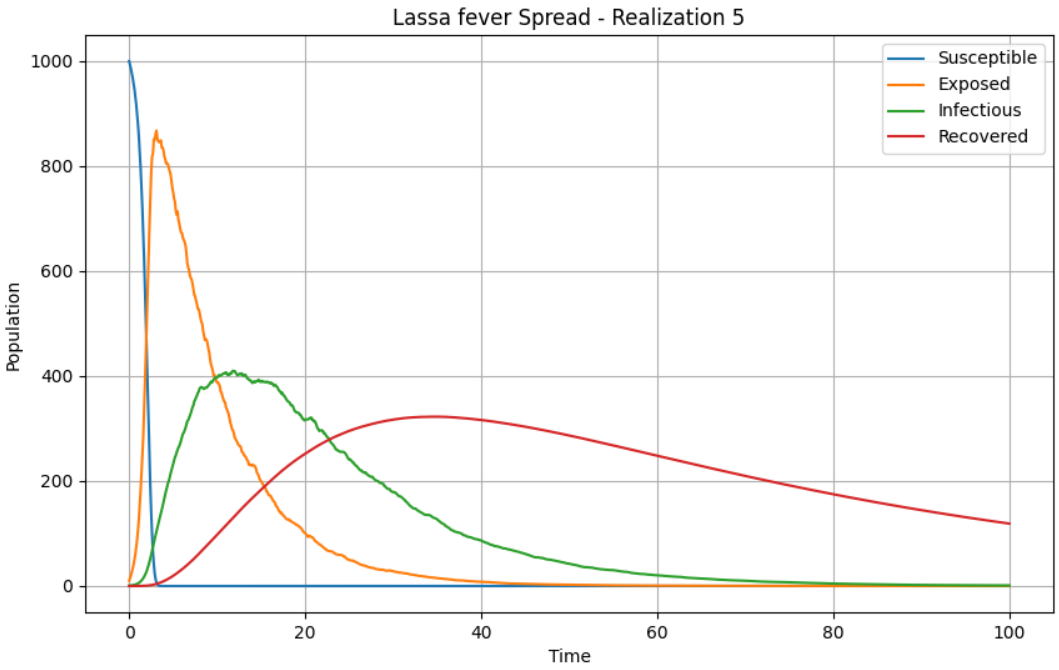

Figure 5: Realization 4 of Lassa fever transmission dynamics

Discussion of Results

The dynamics of the spread of Lassa fever can be effectively modeled in form of a stochastic differential equations.

From the results of the simulations done as shown in figure (2) to figure (5) represent different realization of the simulations using the parameter values indicated on Table 3. The Exposed and the Infectious population can be observed to be under some randomness mimicking the uncertainties in the dynamic of transmission. The infected population and exposed population reached its peak within time interval 0-20 and then exponentially decreases with time to zero. Specifically, the results in the figures indicate the decreasing population of susceptible population, random fluctuations in exposed and infected population to the final trend where the infected population decreases with time due to the decrease in the exposed population. This shows that spreading of the disease is being reduced due to the preventive measures which are being practiced.

CONCLUSION

In this work, a deterministic and a stochastic differential equation model is developed and investigated for the transmission dynamics of Lassa fever epidemic. The model, which is a multidimensional Ito diffusion process, includes susceptible individuals, exposed individuals, infected individual and recovered individuals. A deterministic model was formed and resulting model was transformed into a stochastic differential equation model by applying the procedure proposed by Allen et. al. (2008). As most nonlinear Stochastic Differential Equations (SDEs) are not easy to solve analytically, Euler Maruyama Method for SDEs is used to solve and analyze the model with the aid of Python software.

REFERENCES

Anderson, H. and Britton, T. (2000). Stochastic Epidemic Models and their Statistical Analysis. Lecture notes in Statistics, 151.Springer-verlag, New York.

Allen, E.J. (2007) Modeling with Stochastic Differential Equations. Springer, Dordrecht, The Netherlands.

Allen. E. J. Allen. L.J.S., Arciniega, A. and Greenwood, P.E. (2008). Construction of Equivalent Stochastic Differential Equation Models. *Stochastic Analysis Application.* 26(2):274-297.

Allen Linda, J.S. (2017). A Primer on Stochastic Epidermic models: Formulation, Numerical Simulation and Analysis. *Journal of Infectious Disease Modeling*, 2: 128-142.

Bawa, M.,Abdulraham, S. and Jimoh, O.R. (2013). Stability Analysis of Disease Free Equilibrium state for Lassa fever Disease. *International Journal of Science, Technology, Mathematics and Education*, 9(2): 115-123.

Bernoulli, H. and Blower, J. (2000). Mathematical modelling for the Control of Smallpox. *Bio. Medical Journal*, 19: 670-676.

Britton Tom (2009). Stochastic Epidemic Models: A survey. *Stockholm University*, SE-106, 91, Stockholm, Sweden.

Fisher-Hoch, S.P., Tomori, O., Nasidi, A., Perez-Oronoz, G.I., Fakile, Y., Hutwagner, I. and Cormick, M.C. (1995). Review of cases of nosocomial. Lassa fever in Nigeria: The High price of Poor Medical Practice. *Bio Medical Journal*, 311: 857-859.

Gamboa Maria and Lopez-Herrero, M.J. (2018). The Number of Periodic Inspections during Outbreaks of Discrete-time Stochastic SIS Epidemic Models.*Faculty of Statistical Studies, Complutense University of Madrid* 28040 Madrid, Spain.

Kloeden, P.E. and Platen, E. (1999) Numerical Solution to Stochastic Differential Equations. Springer, Berlin.

Kloeden, P.E. and Platen, E. (2007). Numerical solution of Stochastic Differential Equation. Springer-verlag. Berlin Heidelberg, New York, 3-540-54062-8.

Lopez-Herrero, M.J. (2013). Epidemic Transmission on SEIR Stochastic Models with Nonlinear Incidence rate. *Mathematical Methods Applied Science Journal*, 40:2532-2541.

Ogabi, C.O., Olusa, T.V. and Madufor, M.A. (2012). Controlling Lassa fever Transmission in Northern part of Edo state, Nigeria using SIR model. *New York Science Journal*, 5 (12), http://www.science pub.net/new york .

Ogwuche O. I. Iortyer E. K, Emonyi A. and Ali M. (2023), A stochastic differential equation (SDE) based model for the spread of Tuberculosis, FUDMA Journal of Sciences, vol. 7(5), pp 9-17, DOI*: https://doi.org/10.33003/fjs-2023-0705-1990*

Tara, K.H. (2004). Virology notes in Lassa fever. Retrieved on March 10, 2012 from www.taraharper.com/v lassa.html.

Taylor Howard and Karlin Samuel (1998). An Introduction to Stochastic Modeling. Third Edition. *Academic press*. Accessed July 23, 2020.

Tuckwell, H.C. and Williams, R.J. (2006). Some Properties of a Simple Stochastic Epidemic Model of SIR type. *Mathematical Biosciences Journal*, 3:76-9.