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daniel.okuonghae@uniben.edu
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Application of quadratic Lyapunov function for SIR model with demography

A.L. Ozioko*, I.S.S. Abang[†], G.C.E. Mbah[‡], and M.O. Omeike[§]

Abstract

Lyapunov functions enable the stability analysis of dynamic systems described by ordinary differential equations without finding the solution of such equations. In this paper, we construct a quadratic Lyapunov function for SIR model with demography in analogy with a linear system, and show that the SIR model with demography is asymptotically stable using this quadratic Lyapunov function. This quadratic Lyapunov function satisfies the disease free and endemic equilibrium.

Keywords: Lyapunov function; asymptotic; stability; differential equation

1 Introduction

Lyapunov functions are scalar functions used to prove the stability of equilibrium of ordinary differential equations. It is very important to stability theory in dynamical systems and control theory. Lyapunov function is a necessary and sufficient condition for stability. There are many ways to construct Lyapunov functions and much remains to be done in this regard since there isn't a universal constructive method for finding simple and explicit Lyapunov functions which helps to determine the stability or instability of a system as well as acting as control.

Lyapunov himself [7] indicated the method of constructing the Lyapunov functions for an independent linear system. In [10], they constructed a Lyapunov function for a second and third order respectively with non-linear functions. Their method consists of finding a quadratic form function for a linear system, and analogy in the subsequent selection, a Lyapunov function for the non-linear system. The method also gives us the possibility of obtaining the Lyapunov function in a rather small proximity to the equilibrium position of the non-linear system [1]. Further results in the construction of Lyapunov functions were obtained in the connection with the so-called Aizerman problem [12]. The formulation of this problem prompts the idea of constructing Lyapunov function for non-linear systems by analogy with linear systems. [4] have studied the singular points of third order non-linear ordinary differential equations. According to them, only one singular point of each of the different equations is stable and the rest of the singular points are unstable. Also, they have not investigated the stability character of the system over some regions around a singular point.

For an autonomous polynomial system of differential equations, how to compute the Lyapunov function at equilibria is a basic problem. In [8], the author transformed the problem of computing the Lyapunov function into a quantifier elimination problem. The disadvantage of the method is that the computation complexity of quantifier elimination is doubly exponential in the

*Corresponding Author: Department of Mathematics, Federal University Lokoja, Nigeria; Email: arinze.luke@fulokoja.edu.ng

[†]NCTM, an Agency of Federal Ministry of Science and Technology, PRODA Training Institute, Enugu, Nigeria; Email: sunnyscott2010@gmail.com

[‡]Department of Mathematics, University of Nigeria, Nsukka, Nigeria; Email: godwin.mbah@unn.edu.ng

[§]Department of Mathematics, Federal University of Agriculture, Abeokuta, Nigeria; Email: moomeike@yahoo.com

number of total variables. In order to avoid this problem,[14] propose a symbolic method; they first construct a special semi algebraic system using the local properties of a Lyapunov function as well as its derivative and solving these inequations using cylindrical algebraic decomposition (CAD). Lyapunov functions are indispensable in verifying some quantitative properties of solutions in ordinary differential equations. The problems encountered in the applications are in the construction of appropriate Lyapunov functions for some non-linear ordinary differential equations [2]. Lyapunov function [9] is a good tool in the stability analysis of dynamical systems.

The SIR model is a classical model in mathematical epidemiology [13]. Particularly Kermack and McKendrick[5] use a SIR model to prove the existence of a threshold. The model of Kermack and McKendrick is without demography, i.e. without vital dynamics. As a conceptual model, the classical SIR models are very important [13] . However, the asymptotic behavior of the model changes when introducing vital dynamics. The global stability using Lyapunov functions of SIR model with a total constant population is proved in [3] and [13].

In this work, we established the application of quadratic lyapunov function [10] for the SIR model with demography in analogy with the linear system.

2 Methodology and Discussions

Consider the ordinary differential equation

$$\ddot{x} + a\ddot{x} + b\dot{x} + cx = 0, \quad (1)$$

resulting to the third scalar first order shown as follows:

$$\begin{aligned} \dot{x} &= y, \\ \dot{y} &= z, \\ \dot{z} &= -az - by - cx, \end{aligned} \quad (2)$$

and the system of third- dimensional linear equation

$$\dot{X} = AX, \quad (3)$$

where A is positive definite metrics. Define

$$2V(x, y, z) = X^T AX = A_1x^2 + A_2y^2 + A_3z^2 + 2A_4xy + 2A_5xz + 2A_6yz \quad (4)$$

such that

$$\dot{V} = -cA_5x^2 - (bA_6 - A_4)y^2 - (aA_3 - A_6)z^2 - (cA_6 + bA_5 - A_1)xy - (cA_3 + aA_5 - A_4)xz - (aA_6 + bA_3 - A_2 - A_5)yz. \quad (5)$$

We have the following possibilities:

$$\begin{aligned} \dot{V} &\propto -x^2, \\ \dot{V} &\propto -y^2, \\ \dot{V} &\propto -z^2, \\ \dot{V} &\propto -(x^2 + y^2 + z^2). \end{aligned} \quad (6)$$

CASE I:

If $\dot{V} = -cA_5x^2$, then

$$\begin{aligned} cA_5 &> 0, \\ bA_6 - A_4 &= 0, \\ aA_3 - A_6 &= 0, \\ cA_6 + bA_5 - A_1 &= 0, \\ cA_3 + aA_5 - A_4 &= 0, \\ aA_6 + bA_3 - A_2 - A_5 &= 0, \end{aligned} \tag{7}$$

such that

$$\begin{aligned} A_1 &= \frac{(a^2c + ab^2 - cb)A_5}{k}, \\ A_2 &= \frac{(a^3 + c)A_5}{k}, \\ A_3 &= \frac{aA_5}{k}, \\ A_4 &= \frac{a^2bA_5}{k}, \\ A_6 &= \frac{a^2A_5}{k}, \end{aligned} \tag{8}$$

where $ab - c = k > 0$. Substituting (8) in (4) and set $A_5 = 1$ since $A_5 > 0$,

$$\begin{aligned} 2V(x, y, z) &= \frac{(a^2c + ab^2 - cb)}{k}x^2 + \frac{(a^3 + c)}{k}y^2 + \frac{a}{k}z^2 + 2\frac{a^2b}{k}xy + 2xz + 2\frac{a^2}{k}yz \\ &= \frac{a^2c}{k}x^2 + bx^2 + \frac{a^3 + c}{k}y^2 + \frac{a}{k}z^2 + \frac{2a^2b}{k}xy + 2xz + \frac{2a^2}{k}zy \\ &= bx^2 + \frac{2a^2b}{k}xy + \frac{a^3 + c}{k}y^2 + \frac{a^2c}{k}x^2 + 2xz + \frac{a}{k}z^2 + \frac{2a^2}{k}zy \\ &= b\left(x + \frac{a^2y}{k}\right)^2 + \left(\frac{k(a^3 + c) - ba^4}{k}\right)y^2 + \frac{a^2c}{k}\left(x + \frac{kz}{a^2c}\right)^2 + \left(\frac{a^3c - k^2}{ka^2c}\right)z^2 + \\ &\quad \frac{2a^2}{k}zy \\ &= b\left(x + \frac{a^2y}{k}\right)^2 + \left(\frac{k(a^3 + c) - ba^4}{k}\right)y^2 + \frac{a^2c}{k}\left(x + \frac{kz}{a^2c}\right)^2 + \\ &\quad \frac{a^3c - k^2}{ka^2c}\left(z + \frac{a^4cy}{a^3c - k^2}\right)^2 - \frac{a^6c}{k(a^3c - k^2)}y^2. \end{aligned} \tag{9}$$

This is positive definite and the corresponding time derivative

$$\dot{V}(x, y, z) = -cx^2. \tag{10}$$

Therefore

$$2V_1(x, y, z) = \frac{a^2c}{k}x^2 + bx^2 + \frac{a^3 + c}{k}y^2 + \frac{a}{k}z^2 + \frac{2a^2b}{k}xy + 2xz + \frac{2a^2}{k}zy \tag{11}$$

is a Lyapunov function for the third order differential equation (1) since $c > 0$ and $ab - c = k > 0$.

CASE II

If $\dot{V} = -(bA_6 - A_4)y^2$, then

$$\begin{aligned} cA_5 &= 0, \\ bA_6 - A_4 &> 0, \\ aA_3 - A_6 &= 0, \\ cA_6 + bA_5 - A_1 &= 0, \\ cA_3 + aA_5 - A_4 &= 0, \\ aA_6 + bA_3 - A_2 - A_5 &= 0, \end{aligned} \tag{12}$$

such that

$$\begin{aligned} A_5 &= 0, \\ A_1 &= caA_3, \\ a_2 &= (a^2 + b)A_3, \\ A_4 &= cA_3, \\ A_6 &= aA_3, \\ (ab - c)A_3 &> 0. \end{aligned} \tag{13}$$

Substituting (13) in (4) and set $A_3 = 1$,

$$\begin{aligned} 2V(x, y, z) &= cax^2 + (a^2 + b)y^2 + z^2 + 2cxy + 2ayz \\ &= cax^2 + 2cxy + a^2y^2 + by^2 + 2ayz + z^2 \\ &= ac\left(x + \frac{y}{a}\right)^2 + \left(a^2 - \frac{c}{a}\right)y^2 + b\left(y + \frac{az}{b}\right)^2 + \left(\frac{b - a^2}{b}\right)z^2, \end{aligned} \tag{14}$$

which is positive definite and the time derivative of V is

$$\dot{V}(x, y, z) = -(ab - c)y^2. \tag{15}$$

Since $ab - c > 0$,

$$2V_2(x, y, z) = cax^2 + (a^2 + b)y^2 + z^2 + 2cxy + 2ayz \tag{16}$$

is a Lyapunov function for (1).

CASE III

If $\dot{V} = -(aA_3 - A_6)z^2$, then

$$\begin{aligned} cA_5 &= 0, \\ bA_6 - A_4 &= 0, \\ aA_3 - A_6 &> 0, \\ cA_6 + bA_5 - A_1 &= 0, \\ cA_3 + aA_5 - A_4 &= 0, \\ aA_6 + bA_3 - A_2 - A_5 &= 0, \end{aligned} \tag{17}$$

which implies

$$\begin{aligned}
 A_5 &= 0, \\
 A_1 &= cA_6, \\
 a_2 &= \left(a + \frac{b^2}{c}\right)A_6, \\
 A_4 &= bA_6, \\
 A_3 &= \frac{b}{c}A_6, \\
 \left(\frac{ab}{c} - 1\right)A_6 &> 0.
 \end{aligned}
 \tag{18}$$

Setting $A_6 = 1$ and substitute (18) in (4),

$$\begin{aligned}
 2V(x, y, z) &= cx^2 + \left(a + \frac{b^2}{c}\right)y^2 + \frac{b}{c}z^2 + 2bxy + 2yz \\
 &= cx^2 + 2bxy + ay^2 + \frac{b^2}{c}y^2 + 2yz + \frac{b}{c}z^2 \\
 &= c\left(x + \frac{by}{c}\right)^2 + \left(\frac{ac - b^2}{c}\right)y^2 + \frac{b^2}{c}\left(y + \frac{cz}{b^2}\right)^2 + \left(\frac{b^3 - c^2}{cb^2}\right)z^2,
 \end{aligned}
 \tag{19}$$

which is positive definite and the corresponding time derivative

$$\dot{V}(x, y, z) = -\left(\frac{ab}{c} - 1\right)z^2
 \tag{20}$$

and $\left(\frac{ab}{c} - 1\right) > 0$. Therefore

$$2V_3(x, y, z) = cx^2 + \left(a + \frac{b^2}{c}\right)y^2 + \frac{b}{c}z^2 + 2bxy + 2yz
 \tag{21}$$

is suitable Lyapunov function for (7).

Remark 2.1 *The sum of two or more Lyapunov functions result to another Lyapunov function that satisfies the Lyapunov criteria.*

3 SIR Model with Demographic Term

We consider a population N divided into classes of susceptible, infectious and removed individuals, with numbers at time t denoted by $S(t)$, $I(t)$ and $R(t)$ respectively, that is $N = S(t) + I(t) + R(t)$. We assume that there is no vertical transmission, then all offspring are susceptible. We assume that the natality Λ compensates for the deaths and the state variables are proportions of the population. The parameter is the rate of recovery. Note that, in our model the disease confers a permanent immunity. The parameter β is the effective per capita contact rate of infectious individuals [13]. The dynamic of this model is given by the following system:

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - \beta SI - \mu S, \\
 \frac{dI}{dt} &= \beta SI - \gamma I - \mu I, \\
 \frac{dR}{dt} &= \gamma I - \mu R.
 \end{aligned}
 \tag{22}$$

Since the variable R in SIR model (22) does not appear in the equations of S and I , for model (22) we only need to consider the subsystem [3]:

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \beta SI - \mu S, \\ \frac{dI}{dt} &= \beta SI - \gamma I - \mu I.\end{aligned}\tag{23}$$

From model (22) we have,

$$\begin{aligned}\frac{dN}{dt} &= \Lambda - \mu N - \gamma I, \\ &\leq \Lambda - \mu N.\end{aligned}\tag{24}$$

Lemma 3.1 *The system (22) with initial conditions $S(0) > 0$, $I(0) \geq 0$, and $R(0) \geq 0$ have a positive invariant solution in the region $\Omega = \left\{ (S, I, R) \in \mathbb{R}_+^3 : N = S(t) + I(t) + R(t) \leq \frac{\Lambda}{\mu} \right\}$.*

Proof. Without loss of generality,

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t).$$

Multiplying through by the integrating factor $I.F = e^{\mu t}$ and integrate both side, we have

$$N(t)e^{\mu t} \leq \frac{\Lambda e^{\mu t}}{\mu} + K.\tag{25}$$

Furthermore, applying the initial conditions $t = 0$ in (25), we have

$$K \geq N(0) - \frac{\Lambda}{\mu}.\tag{26}$$

Substituting (26) into (25), we have

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t},\tag{27}$$

where the $N(0) = N_0$ is the initial value of the total populations. As $t \rightarrow \infty$ in (27), the population size $N(t)$ approaches $\frac{\Lambda}{\mu}$, ie

$$0 \leq N(t) \leq \frac{\Lambda}{\mu}.$$

However $N = S(t) + I(t) + R(t) \leq \frac{\Lambda}{\mu}$. This implies that $S(t) \leq \frac{\Lambda}{\mu}$, $I(t) \leq \frac{\Lambda}{\mu}$ and $R(t) \leq \frac{\Lambda}{\mu}$. Hence the solutions are bounded.

In particular, If $N(0) < \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$. Therefore the region $\Omega \in \mathbb{R}_+^3$ is positively invariant. Therefore the model the model (22) makes biological sense

The basic reproduction ratio is given by

$$R_0 = \frac{\beta}{\gamma + \mu}.$$

3.1 Stability of Disease Free Equilibrium (DFE)

The system (22) has disease free equilibrium which is given as $(S, I) = (1, 0)$.

Theorem 3.1 *If $R_0 \leq 1$ then the DFE is globally asymptotically stable on Ω .*

Proof. The quadratic Lyapunov function (4) for the given equation (23) is

$$2V(S, I) = A_1 S^2 + A_2 I^2 + 2A_4 SI,$$

such that

$$\dot{V}(S, I) = -(\beta A_1 I + A_1 \mu - \beta A_4 I) S^2 - (\beta A_4 S - A_2 \beta S + \gamma A_2 + \mu A_2) I^2 - (\mu A_4 + \gamma A_4 + \mu A_4) SI + A_1 \Lambda S + \Lambda A_4 I. \quad (28)$$

The only possibility in (25) is that

$$\dot{V}(S, I) = -(\beta A_4 S - A_2 \beta S + \gamma A_2 + \mu A_2) I^2,$$

such that

$$\begin{aligned} \beta A_4 S - A_2 \beta S + \gamma A_2 + \mu A_2 &> 0, \\ \beta A_1 I + A_1 \mu - \beta A_4 I &= 0, \\ \mu A_4 + \gamma A_4 + \mu A_4 &= 0, \\ A_1 &= 0, \\ A_4 &= 0. \end{aligned} \quad (29)$$

Since $A_2 > 0$, set $A_2 = 1$ and we obtain

$$\dot{V}(S, I) = -(\gamma + \mu - \beta S) I^2,$$

such that

$$2V(S, I) = I^2.$$

Finally, the quadratic Lyapunov function for the disease free equilibrium is

$$2V(S, I) = I^2,$$

which is positive definite and the corresponding time derivative

$$\begin{aligned} \dot{V}(S, I) &= -(\gamma + \mu - \beta S) I^2 \\ &= -(\gamma + \mu) \left[1 - \frac{\beta}{\gamma + \mu} S \right] I^2 \\ &= -(\gamma + \mu) [1 - R_0 S] I^2 \\ &< 0, \end{aligned} \quad (30)$$

is negative definite since $\gamma + \mu - \beta S > 0$, $R_0 < 1$ and $S \approx 1$, hence asymptotically stable on Ω .

3.2 Global Stability of Endemic Equilibrium (EE)

The endemic equilibrium is give by $(S^*, I^*) = \left(\frac{1}{R_0}, \frac{\mu}{\beta} (R_0 - 1) \right)$.

Theorem 3.2 *If $R_0 > 1$, the DFE is unstable and there exists a unique endemic equilibrium $(S^*, I^*) = \left(\frac{1}{R_0}, \frac{\mu}{\beta} (R_0 - 1) \right)$ and this endemic equilibrium is stable on Ω and globally asymptotically stable if $S < \frac{1}{R_0}$.*

Proof. Consider the quadratic Lyapunov function defined by

$$2V(S, I) = I^2.$$

Clearly V is positive definite, that is $V(S, I) \geq 0$. The derivative along trajectories of (22) is given by:

$$\begin{aligned}\dot{V}(S, I) &= -(\gamma + \mu - \beta S)I^2 \\ &= -(\gamma + \mu)[1 - R_0 S]I^2 \\ &= -(\gamma + \mu)\left[1 - R_0 \frac{1}{R_0}\right]I^2 \\ &\leq 0.\end{aligned}\tag{31}$$

Then we conclude $\dot{V}(S, I)$ is semi-definite positive. Then the endemic equilibrium is stable by Lyapunov theorems.

If $S < \frac{1}{R_0}$, then

$$\dot{V}(S, I) < 0.$$

Hence the Endemic Equilibrium is global asymptotically stable.

4 Conclusion

The construction of Lyapunov function is the fact that corresponding to any positive definite quadratic form $V(x)$, there exist another positive definite $U(x)$ such that $\dot{V} = -U$ is satisfied. We obtain the quadratic Lyapunov Function of SIR model near the equilibrium in analogy with the linear system without necessarily solving the differential equation. Although many methods for finding Lyapunov functions are available, quadratic Lyapunov stability analysis [10] provides suitable function that satisfied the disease free equilibrium and the endemic equilibrium of SIR Model instead of finding separately Lyapunov function for both disease free equilibrium and endemic equilibrium.

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Dynamics of an epidemiological model of drinking

I. Abubakar^{*†}, S. Usaini^{*‡}, and K.I. Falade^{*§}

Abstract

In this paper, we review and carry out the bifurcation analysis of the drinking model proposed by Manthey *et al.* in 2008. We show that the model exhibits a drinking-free equilibrium point and a problem drinking-free equilibrium which exists when the reproductive number $\mathcal{R}_0^Y > 1$. Each of these equilibria is a stable node if the threshold parameters $\mathcal{R}_0^Y < 1, \mathcal{R}_0^Z < 1$ and $\mathcal{R}_1 < 1, \mathcal{R}_0^Y > 1$ respectively. Moreover, the existence of multiple endemic equilibria reveals the complex dynamics of the model by the occurrence of two saddle-node bifurcations leading to the phenomenon of backward bifurcation. Taking the transmission rate of non-drinkers to problem drinkers κ as a bifurcation parameter, we show the two saddle-nodes SN and SN_2 for the extinction and persistence of the drinking problem. Before the disappearance of E_1 and E_2 after SN in the interval $(0, \mathcal{R}_0^{Z_{c1}})$ the system is bistable with E_0 and E_2 being the stable equilibrium points. The system is rendered mono stable after the SN before the SN_2 emerged in $(\mathcal{R}_0^{Z_{c1}}, \mathcal{R}_0^{Z_{c2}})$ with E_0 as the only stable equilibrium point. Then after the emergence of SN_2 in the interval $(\mathcal{R}_0^{Z_{c2}}, \kappa^*)$, the system becomes bistable again with E_0 and E_4 . But for $\kappa > \kappa^*$, there is endemic persistence of the drinking problem with E_4 as the only stable attractor. When such a scenario does not exist a forward bifurcation occurs for which the drinking-free equilibrium exchanges stability at $\kappa = \kappa^c$ with drinking-present equilibrium point. Numerical simulations of the model support these extinction and persistence regions of the drinking problem.

Keywords: campus drinking; bifurcation; stable node; saddle-node

1 Introduction

With recent improvement of our living standard, the life styles of people are becoming more and more diversified and drinking plays a significant role in daily life of some people. However the current situation of alcoholism around the world is really terrifying. In Europe for instance, alcohol is the third leading risk factor for disease and mortality after tobacco and high blood pressure [19]. The world health organization reported that more than 3 million people died because of alcohol and other substance abuse, which account to 5.3% of all death and 5.1% of the global disease burden attributable to alcohol consumption [20]. It was also presented in [14] that alcohol use rates are very high among college students in the US. In fact, approximately two of five such students were heavy drinkers, meaning that having had five or more drinks in a row in the past 2 weeks. Alcohol use is higher among male than female students as well. White students are highest in heavy drinking, black students are lowest and Hispanic students are intermediate. Use of alcohol alone is higher among college students than among non college age-mates. Therefore, alcoholism is not only a global public health issues, but also a societal problem that need to be addressed.

To address the problem of alcohol drinking among youth, several mathematical models have been proposed to analyze the spread and control of drinking behaviour, See for instance

^{*}Department of Mathematics, Kano University of Science and Technology, Wudil, Nigeria

[†]E-mail: ibrahimjaba258@gmail.com

[‡]Corresponding Author: salisu.usaini@kustwudil.edu.ng

[§]Email: faladekazeem2013@gmail.com

[1, 2, 3, 4, 11] and the references therein. Sánchez *et al.* [15], proposed a simple mathematical model to describe the dynamics of drinking behaviors of individuals in shared drinking environments. Despite its limitations, the model has generated some useful insights. More precisely, the basic reproductive number (as a function of treatment), is not always the key to controlling drinking within the population due to high rates of recovery and relapse. The high relapse rates will occur when treatment programs have only short-term positive effects. The drinking behaviours of college students motivated the study using mathematical model on how the ‘disease’ of drinking spread on campus [13]. The model suggests that the pattern of recruiting new members plays a significant role in the reduction of campus alcohol problems and that the reproductive numbers are not sufficient to predict whether drinking behaviour will persist on campus. More precisely, campus alcohol abuse may be reduced by minimizing the ability of problem drinkers to directly recruit non-drinkers. Bhunu developed a deterministic mathematical model for the spread of alcoholism to gain insights into its growing health and social problem. The associated reproduction number is calculated and Local asymptotic stabilities of equilibrium points were carried out. The analysis of the reproduction number reveals that encouraging and supporting moderate drinkers to quit alcohol consumption is more effective in the control of alcoholism than supporting and encouraging alcoholics to quit and vice-versa. Numerical simulations of the model support this results and also shown that encouraging and supporting all alcohol consumers to quit drinking will be the best strategy [4]. A mathematical model of alcohol abuse was presented in [17]. The basic reproduction number $\mathcal{R}_0$ is determined and they proved that the problem-free equilibrium is both locally and globally asymptotically stable when $\mathcal{R}_0 < 1$. Sensitivity analysis of the model parameters indicates that the transmission coefficient β_1 from moderate and occasional drinker to heavy drinker, is the most useful parameter to target for the reduction of $\mathcal{R}_0$.

In a recent development, the threshold dynamics of a multi-group SEAR alcoholism epidemic model with public health education was analysed [12]. It was proved that the basic reproduction number $\mathcal{R}_0$ plays the role of a threshold for the long-time behaviour of the model. They showed that the model exhibits global asymptotic stable alcohol-free and alcohol-present equilibrium points. Sensitivity analysis of the model parameters and numerical simulations were presented to illustrate the results. Zhang *et al.* proposed a delayed drinking model to carry out the bifurcation and optimal control analysis. A threshold value of the time delay for the local stability of drinking-present equilibrium and the existence of Hopf bifurcation were obtained. In addition, optimal strategies to lower down the number of drinkers were presented. Numerical simulations to support the obtained results and the effects of some parameters on dynamics of the model were provided [21]. An alcohol consumption model with health education and three time delays was formulated and analyzed [11]. The alcoholism generation number was determined and two equilibrium points of the model were obtained. The corresponding global dynamics of the model are analyzed respectively in four cases with different time delays. Furthermore, the effects of health education and three time delays in controlling the alcohol problem were discussed. Some numerical simulation results were also given to support the theoretical predictions.

We know that when a disease transmission model posses multiple endemic equilibria, the model exhibits complex dynamics such as backward bifurcation, saddle-node bifurcation and forward hysteresis [6, 7, 10, 18]. The existence of two endemic equilibrium points of the drinking model proposed by Manthey *et al.* [13], motivate us to review and investigate its complex dynamics. In fact, we show the occurrence of the phenomena of backward bifurcation and saddle-node bifurcation when the transmission rate of non-drinkers to problem drinkers κ is varied (increasing the threshold parameter $\mathcal{R}_0^Z$). The existence of these phenomena indicates the extinction and persistence regions of drinking problem.

The remaining paper is organized as follows: In Section 2, we describe the model. Section 3 presents the existence and stability results of the model equilibria. In order to complete the investigation, the special cases of the model are analyzed in Section 4. In Section 5, conditions

for host population persistence and extinction for each of these cases are derived. Regions of host population persistence and extinction for certain parameter ranges are illustrated *via* numerical simulations also in Section 5.

2 Model description

For the drinking model proposed in [13], the student population is divided into three distinct classes of non-drinkers (X), social drinkers (Y) and problem drinkers (Z) and the population is assumed to be constant. It is well known that the definitions of non-drinkers, social drinkers, and problem drinkers are not consistently used in the literature [8]. Therefore, the following definitions that would be of interest to college administrators and parents who are concerned with the negative consequences of campus-drinking problem are presented in [13].

- (a) Non-drinkers are susceptible which we define as students who have not taking or consume alcohol for an extended period of time, typically one year. Also student who stop drinking is regarded as non-drinkers
- (b) Social drinkers are defined as students that consume alcohol occasionally and in moderation
- (c) Problem drinkers are defined as student whose drinking habit and associated behavior have negative consequence for themselves and others

Since students are only in campus for a relatively short period of time typically five year or less then it's not easy to access whether or not a student who has stopped drinking has permanently recovered. Hence, the model does not include a recovered class and developed on the assumptions that

- (i) Student joint campus in any of the three drinking state and mix randomly with the rest of the campus population. Once in campus, student may transit from any drinking state to another.
- (ii) Transition to higher drinking state is as a result of social interaction and transition to lower drinking state results from recovery process [8].
- (iii) There is a net positive flow towards a higher drinking state. This is an effect that has been termed the 'college effect' in the literature [1]. Such an effect is more pronounced in the first 2 years of college and declines in the last 2 years during which time students transition towards adult drinking patterns [4].
- (iv) The student population is homogeneous with respect to variables such as age, academic class rank and socio-economic status. More precisely, the homogeneity here means that students within each compartment are similar with regard to their drinking behaviors.
- (v) The model does not consider the effect of breaks and vacations.
- (vi) The interaction terms αXY , κXZ , and γYZ , respectively modelled the conversion from non-drinkers to social drinkers, non-drinkers to problem drinkers, and social drinkers to problem drinkers.
- (vii) The conversions in return of (vi) are to be the result of recovery process and are implemented via the terms βY , ϵZ and δZ , respectively.

Thus the dynamics of campus drinking is modelled using the following system of nonlinear differential equations (equation 1), where X, Y and Z have been resealed as proportion. The respective rates of transfer between the three classes are shown in the flow diagram in Figure 1.

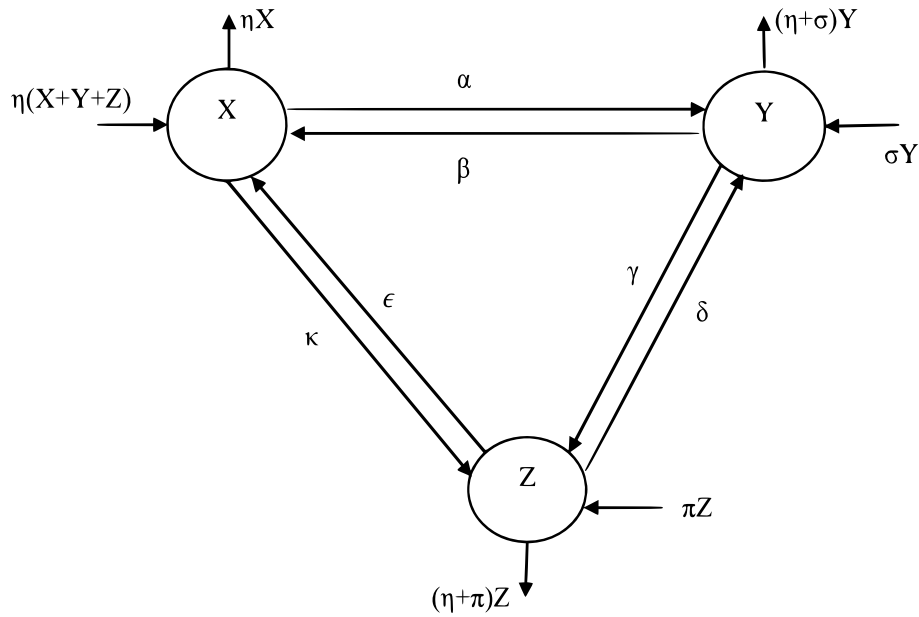


Figure 1: Flow diagram of model (1)

Parameter	Description	Typical value
α	Transmission rate of non- drinkers to social drinkers	1.0
β	Recovery rate of social drinkers	0.2
γ	Transmission rate of social drinkers to problem drinkers	1.0
δ	Recovery rate of problem drinkers to social drinkers	0.2
ϵ	Recovery rate of problem drinkers to non-drinkers	0.2
η	Departure rate from campus environment	0.25
κ	Transmission of non-drinkers to problem drinkers	1.5
σ	Entrance rate of social drinkers	0.25
π	Entrance rate of problem drinkers	0.1

Table 1: Parameter description

$$\begin{aligned}
\frac{dX}{dt} &= \eta - \eta X - \alpha XY - \kappa XZ + \beta Y + \epsilon Z, \\
\frac{dY}{dt} &= \sigma Y - (\eta + \sigma)Y + \alpha XY - \beta Y - \gamma YZ + \delta Z, \\
\frac{dZ}{dt} &= \pi Z - (\eta + \pi)Z + \gamma YZ + \kappa XZ - \delta Z - \epsilon Z,
\end{aligned} \tag{1}$$

with $X + Y + Z = 1$. In system (1), the parameters α, γ , and κ are the mean transmission rates which measure the effectiveness of the interactions between non-drinkers and social drinkers, social drinkers and problem drinkers, and non-drinkers and problem drinkers, respectively. β is the recovery rate that represents the mean rate at which social drinkers revert to the non-drinking class. The recovery rates δ and ϵ are the mean rates at which problem drinkers move back to the social drinking and the non-drinking classes, respectively. Parameter η denotes the net departure rate from the campus environment. The other parameters of the model are described in Table 1.

For model (1) with initial condition $X > 0, Y > 0, Z > 0$ to be biologically meaningful, we need to show that the solutions of the model with positive initial conditions are non-negative

and remain positive for all time $t > 0$. Moreover, the region these solutions exist is positively invariant and attractive.

We can use elementary techniques for solving ordinary differential equations to show that the state variables (X, Y and Z) are positive. In fact, using elementary integrating factor technique we have from the first equation of (1) the integrating factor

$$\rho(t) = e^{(\eta + \alpha Y + \kappa Z)},$$

so that

$$\frac{d(\rho X)}{dt} = \rho(\eta + \beta Y + \epsilon Z).$$

Integrating this, gives

$$\rho(t)X(t) = \rho(0)X(0) + (\eta + \beta Y + \epsilon Z) \int_0^t \rho ds \geq 0.$$

Similarly, from the second equation of (1)

$$\rho(t)Y(t) = \rho(0)Y(0) + (\eta + \beta Y + \delta Z) \int_0^t \rho ds \geq 0.$$

Then one can see by inspection from the third equation of (1) that

$$Z(t) = Z(0) \exp \left[\int_0^t \{ \gamma Y(s) + \kappa X(s) - (\eta + \delta + \epsilon) \} ds \right] \geq 0.$$

Hence, $X(t), Y(t), Z(t) \geq 0$ for all $t \geq 0$.

Summing the three equations in (1), we have

$$\frac{d(X + Y + Z)}{dt} = \eta[1 - (X + Y + Z)].$$

It follows that $(X + Y + Z) \leq 1$, so that the feasible region

$$\mathcal{D} = \{(X, Y, Z) \in \mathbb{R}_+^3 : 0 < X + Y + Z \leq 1\}$$

is positively invariant for system (1).

3 Existence and stability of equilibria

3.1 Drinking problem free equilibria

This is an equilibrium which represents a free drinking environment (i.e. when the compartments of social and problem drinkers are empty). Thus, this equilibrium point is given by

$$E_0 = (X_0, Y_0, Z_0) = (1, 0, 0).$$

To identify condition under which a culture of drinking can be established a threshold parameter as in disease transmission models called the basic reproduction number is required. Considering the two level of infective, two reproductive numbers are obtained in the absence of drinking. The first reproductive number, denoted by $\mathcal{R}_0^Y$ is obtained from the second equation of system (1) as follows

$$\mathcal{R}_0^Y = \frac{\alpha}{\eta + \beta}.$$

This reproductive number is defined as the average number of secondary cases a typical social drinker generated in a nondrinking campus environment. Note that the secondary cases are cases of new social drinkers recruited from the nondrinking class. Similarly, from the third equation of system (1), we have the second reproductive number

$$\mathcal{R}_0^Z = \frac{\kappa}{\eta + \delta + \epsilon}.$$

The threshold parameter $\mathcal{R}_0^Z$ is defined similar to the $\mathcal{R}_0^Y$. The secondary cases here are the cases of new problem drinkers recruited from the nondrinking class.

Moreover, the reproduction numbers $\mathcal{R}_0^Y$ and $\mathcal{R}_0^Z$ are simply obtained from the product of the transmission rates α and κ and the times spent in the Y and Z classes $\left(\frac{1}{\eta + \delta + \epsilon} \& \frac{\alpha}{\eta + \beta}\right)$, respectively.

The Jacobian matrix of the system (1) evaluated at any equilibrium point is the following.

$$J(X, Y, Z) = \begin{pmatrix} -\eta - \alpha Y - \kappa Z & -\alpha X + \beta & -\kappa X + \epsilon \\ \alpha Y & -Q_1 + \alpha X - \delta Z & -\alpha Y + \delta \\ \kappa Z & \gamma Z & \alpha Y + \kappa X - Q_1 \end{pmatrix} \quad (2)$$

where $Q_1 = \eta + \beta, Q_2 = \eta + \delta + \epsilon$.

Theorem 3.1 The drinking-free equilibrium (DFE), E_0 , of model (1) is a stable node on $\mathcal{D}$ if $\mathcal{R}_0^Y < 1, \mathcal{R}_0^Z < 1$ and is a saddle point otherwise.

Proof. Using (2), the Jacobian matrix evaluated at the drinking free equilibrium is

$$J(1, 0, 0) = \begin{pmatrix} -\eta & -\alpha + \beta & -\kappa + \epsilon \\ 0 & -Q_1 + \alpha & \delta \\ 0 & 0 & \kappa - Q_2 \end{pmatrix}. \quad (3)$$

It is easy to see that the eigenvalues of $J(1, 0, 0)$ are

$$-\eta, \alpha - Q_1 = Q_1(\mathcal{R}_0^Y - 1), \kappa - Q_2 = Q_2(\mathcal{R}_0^Z - 1).$$

These eigenvalues are real and negative whenever $\mathcal{R}_0^Y$ and $\mathcal{R}_0^Z$ are less than unity. Thus, E_0 is a stable node and a saddle point if $\mathcal{R}_0^Y$ and $\mathcal{R}_0^Z$ are greater than unity or one of the two is greater than one. Hence the proof. ■

3.2 Problem drinking-free equilibrium

At the problem drinking-free equilibrium, $X \neq 0, Y \neq 0$ and $Z = 0$. Therefore, from the second equation of (1), we obtain

$$X = \frac{\eta + \beta}{\alpha} = \frac{1}{\mathcal{R}_0^Y}. \quad (4)$$

Then using (4) in the first equation of (1) with $Z=0$, we have

$$Y = 1 - \frac{\eta + \beta}{\alpha} = 1 - \frac{1}{\mathcal{R}_0^Y}.$$

Thus, in the absence of problem drinkers on campus the second equilibrium point is given by

$$E_1 = (X_1, Y_1, Z_1) = \left(\frac{1}{\mathcal{R}_0^Y}, 1 - \frac{1}{\mathcal{R}_0^Y}, 0\right). \quad (5)$$

Thus, this equilibrium point exists when $\mathcal{R}_0^Y > 1$ and different from E_0 . It is considered as second ‘drinking-free’ equilibrium if social drinking is not regarded as a problem. In this

case, Z is the only infective compartment of system (1) and so, the third reproductive number, $\mathcal{R}_1$, which is the average number of secondary cases a typical problem drinker can produce in a campus environment with only non-drinkers and social drinkers. Here, the secondary cases are cases of new problem drinkers recruited from the non-drinking and social drinking classes. The threshold parameter $\mathcal{R}_1$ is then computed from the third equation of (1) as follows

$$\mathcal{R}_1 = \frac{\gamma(\alpha - \eta - \beta) + \kappa(\alpha + \beta)}{\alpha(\eta + \delta + \epsilon)} = \frac{\gamma\left(1 - \frac{1}{\mathcal{R}_0^Y}\right) + \frac{\kappa}{\mathcal{R}_0^Y}}{\eta + \delta + \epsilon}.$$

Theorem 3.2 The problem drinking-free equilibrium E_1 is a stable node on $\mathcal{D}$ if $\mathcal{R}_1 < 1$, $\mathcal{R}_0^Y > 1$ and a saddle point otherwise.

Proof. The Jacobian matrix evaluated at E_1 is obtained from (2) as follows

$$J(X_1, Y_1, 0) = \begin{pmatrix} -\eta - \alpha Y_1 & -\alpha X_1 + \beta & -\kappa X_1 + \epsilon \\ \alpha Y_1 & -Q_1 + \alpha X_1 & -\alpha Y_1 + \delta \\ 0 & 0 & \alpha Y_1 + \kappa X_1 - Q_2 \end{pmatrix}. \quad (6)$$

It can be observed that one of the eigenvalues of $J(X_1, Y_1, 0)$ is

$$\alpha Y_1 + \kappa X_1 - Q_2 = Q_2(\mathcal{R}_1 - 1)$$

and the other two are of the 2×2 sub-matrix $J^*(X_1, Y_1, 0)$ of $J(X_1, Y_1, 0)$ obtained by deleting the third row and the third column of $J(X_1, Y_1, 0)$.

$$J^*(X_1, Y_1, 0) = \begin{pmatrix} -\alpha X_1 + \beta & -\kappa X_1 + \epsilon \\ -Q_1 + \alpha X_1 & -\alpha Y_1 + \delta \end{pmatrix}.$$

Now, using (5) the characteristics equation of $J^*(X_1, Y_1, 0)$ is

$$\lambda^2 + \left[\eta + \alpha \left(1 - \frac{1}{\mathcal{R}_0^Y} \right) \right] \lambda + \eta \alpha \left(1 - \frac{1}{\mathcal{R}_0^Y} \right) = 0$$

so that

$$\lambda = \frac{-\left[\eta + \alpha \left(1 - \frac{1}{\mathcal{R}_0^Y} \right) \right] \pm \left[\eta - \alpha \left(1 - \frac{1}{\mathcal{R}_0^Y} \right) \right]}{2}$$

implying that $\lambda_1 = -\eta$ and $\lambda_2 = -\alpha \left(1 - \frac{1}{\mathcal{R}_0^Y} \right)$. Then the three eigenvalues of $J(X_1, Y_1, 0)$ are $Q_2(\mathcal{R}_1 - 1)$, $-\eta$, $-\alpha \left(1 - \frac{1}{\mathcal{R}_0^Y} \right)$, which are real and negative when $\mathcal{R}_1 < 1$ and $\mathcal{R}_0^Y > 1$. Moreover, the first eigenvalue is positive if $\mathcal{R}_1 > 1$ and the third one is positive when $\mathcal{R}_0^Y < 1$. It follows that the problem drinking-free equilibrium E_1 is a stable node if $\mathcal{R}_1 < 1$ and $\mathcal{R}_0^Y > 1$ and a saddle point otherwise. ■

The result of this theorem shows that, it is possible for non-drinkers and social drinkers to coexists at equilibrium provided $\mathcal{R}_1 < 1$ and $\mathcal{R}_0^Y > 1$.

3.3 Endemic equilibria

With the persistence of the problem of drinking abuse in college campuses, all the three drinking states can be present at a time in a campus. From the third equation of (1), we obtain at endemic state

$$X = \frac{\eta - \gamma Y + \delta + \epsilon}{\kappa}. \quad (7)$$

Then using (7) in the second equation of system (1), we have

$$\sigma Y - (\eta + \sigma)Y + \alpha \frac{\eta - \gamma Y + \delta + \epsilon}{\kappa} Y - \beta Y - \gamma Y Z + \delta Z = 0$$

and, so

$$Z = \frac{\alpha\gamma Y^2 + (\eta\kappa + \beta\kappa - \alpha\eta - \alpha\delta - \alpha\epsilon)Y}{\kappa(\delta - \gamma Y)}. \quad (8)$$

Substituting the expressions of X and Z in (7) and (8), respectively, into equation $X + Y + Z = 1$, we obtain after algebraic manipulation the following quadratic equation in Y .

$$aY^2 + bY + c = 0, \quad (9)$$

where

$$\begin{aligned} a &= \gamma(\kappa - \alpha - \gamma), \\ b &= -\kappa\gamma + \alpha\delta + \epsilon\gamma + \eta\alpha - \delta\kappa + \alpha\epsilon - \beta\kappa + \eta\gamma + 2\gamma\delta - \eta\kappa \\ &= -\gamma(\eta + \delta + \epsilon)(\mathcal{R}_0^Z - 1) - \kappa(\delta + \beta + \eta) + \delta(\alpha + \gamma) + \alpha(\eta + \epsilon), \\ c &= \delta(\eta + \delta + \epsilon)(\mathcal{R}_0^Z - 1). \end{aligned} \quad (10)$$

Then the following theorem provides condition under which an equilibrium proportion of social drinkers can exist.

Theorem 3.3 The system of equations (1) has

- (i) a unique endemic equilibrium if $a > 0, c < 0$.
- (ii) a unique endemic equilibrium if $a > 0, b < 0$ and $c = 0$ or $b^2 - 4ac = 0$.
- (iii) two endemic equilibria if $a > 0, b < 0, c > 0$ and $b^2 - 4ac > 0$.
- (iv) two endemic equilibria if $a < 0, b > 0, c < 0$ and $b^2 - 4ac > 0$.
- (v) no endemic equilibrium otherwise.

Proof. The proof follows from the properties of the roots of quadratic equation if $\mathcal{R}_0^Z$ is less than or greater than unity. For $\mathcal{R}_0^Z = 1$, the statements can be verified by inspection.

The possible existence of multiple endemic equilibrium points is given in (iii) and (iv) of the Theorem above which suggest the possibility of backward bifurcation, where the stable DFE coexist with a stable endemic equilibrium, when the associated reproductive number is less than unity.

4 Bifurcation analysis and numerical results

To investigate the dynamics of model (1) using the Center manifold theory as presented in [5], we make the following transformation on the model. Let $X = p_1, Y = p_2$ and $Z = p_3$ so that the total population is $1 = p_1 + p_2 + p_3$. In vector notation, we have $P = (p_1, p_2, p_3)^T$ with the system (1) written in the form $\frac{dP}{dt} = (f_1, f_2, f_3)^T$. It follows that

$$\begin{aligned} \frac{dP}{dt} &= f_1 = \eta - \eta p_1 - \alpha p_1 p_2 - \kappa p_1 p_3 + \beta p_2 + \epsilon p_3, \\ \frac{dY}{dt} &= f_2 = \sigma p_2 - (\eta + \sigma)p_2 + \alpha p_1 p_2 - \beta p_2 - \gamma p_2 p_3 + \delta p_3, \\ \frac{dZ}{dt} &= f_3 = \pi p_3 - (\eta + \pi)p_3 + \gamma p_2 p_3 + \kappa p_1 p_3 - \delta p_3 - \eta p_3. \end{aligned} \quad (11)$$

The trace and determinant of $J(E_0)$ as presented in (3) are respectively

$$\text{tr}(J(E_0)) = -\eta - Q_1 + \alpha + \kappa - Q_2 \neq 0,$$

and

$$\det(J(E_0)) = -\eta(\alpha - Q_1)(\kappa - Q_2) = -\eta Q_1 Q_2 (\mathcal{R}_0^Y - 1)(\mathcal{R}_0^Z - 1).$$

This determinant is zero if either $\mathcal{R}_0^Y = 1$ or $\mathcal{R}_0^Z = 1$ or $\mathcal{R}_0^Y = \mathcal{R}_0^Z = 1$. The first two relations are equivalent to $\alpha = \eta + \beta$ or $\kappa = \eta + \delta + \epsilon$. Thus, $J(E_0)$ have a simple zero eigenvalue and so, the Center Manifold Theory can be applied. Let the left eigenvectors for $J(E_0)$ be $V = (v_1, v_2, v_3)^T$ then using standard computation of eigenvectors, we have

$$v = \left(1, \frac{\alpha - \beta}{\eta + \beta + \alpha}, \frac{\delta(\alpha - \beta) + (\kappa - \epsilon)(\eta + \beta + \alpha)}{Q_2(\eta + \beta + \alpha)(\mathcal{R}_0^Z - 1)} \right). \quad (12)$$

Similarly, the right eigenvectors corresponding to the simple zero eigenvalue $w = (w_1, w_2, w_3)^T$ are the following.

$$w = \left(\frac{-\delta(\beta - \alpha) + Q_1(\epsilon - \kappa)(\mathcal{R}_0^Y - 1)}{\eta Q_1(\mathcal{R}_0^Y - 1)}, \frac{-\delta}{Q_1(\mathcal{R}_0^Y - 1)}, 1 \right). \quad (13)$$

The nonzero partial derivatives of the vector field f of (11) evaluated at E_0 are as follows

$$\begin{aligned} \frac{\partial^2 f_1}{\partial x_1 \partial x_2} &= \frac{\partial^2 f_1}{\partial x_2 \partial x_1} = -\alpha, & \frac{\partial^2 f_1}{\partial x_1 \partial x_3} &= \frac{\partial^2 f_1}{\partial x_3 \partial x_1} = -\kappa, \\ \frac{\partial^2 f_2}{\partial x_1 \partial x_2} &= \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = \alpha, & \frac{\partial^2 f_2}{\partial x_2 \partial x_3} &= \frac{\partial^2 f_2}{\partial x_3 \partial x_2} = -\gamma, \\ \frac{\partial^2 f_3}{\partial x_1 \partial x_3} &= \frac{\partial^2 f_3}{\partial x_3 \partial x_1} = \kappa, & \frac{\partial^2 f_3}{\partial x_2 \partial x_3} &= \frac{\partial^2 f_3}{\partial x_3 \partial x_2} = \gamma. \end{aligned} \quad (14)$$

Computation of a

Using equations (12), (13) and (14), we have

$$\begin{aligned} a &= \sum_{k,i,j=1}^3 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} \\ &= 2 \frac{-\delta(\beta - \alpha) + Q_1(\epsilon - \kappa)(\mathcal{R}_0^Y - 1)}{\eta Q_1(\mathcal{R}_0^Y - 1)} \left(\frac{\alpha \delta}{Q_1(\epsilon - \kappa)(\mathcal{R}_0^Y - 1)} - \kappa \right) \\ &\quad + 2 \frac{-\delta(\beta - \alpha) + Q_1(\epsilon - \kappa)(\mathcal{R}_0^Y - 1)}{\eta Q_1(\mathcal{R}_0^Y - 1)} \frac{\delta \phi(\beta - \alpha) + Q_1 \varphi(\epsilon - \kappa)(\mathcal{R}_0^Y - 1)}{Q_1 Q_2 \varphi(\mathcal{R}_0^Y - 1)(\mathcal{R}_0^Z - 1)} \\ &\quad - 2 \frac{\delta \gamma [\varphi(\kappa - \epsilon) - (\beta - \alpha)(Q_2 \varphi + \delta)]}{Q_1 Q_2 \varphi(\mathcal{R}_0^Y - 1)(\mathcal{R}_0^Z - 1)}. \end{aligned} \quad (15)$$

with $\phi = Q_1(\mathcal{R}_0^Y - 1) + Q_2(\mathcal{R}_0^Z - 1)$, $\varphi = \eta + \beta + \alpha$.

Computation of b

Now, using the vectors v and w as presented in (12) and (13), respectively with the following associated partial derivatives

$$\frac{\partial^2 f_1}{\partial x_3 \partial \kappa^*}(E_0) = -1, \quad \frac{\partial^2 f_3}{\partial x_3 \partial \kappa^*}(E_0) = 1$$

we have

$$b = \sum_{k,i} v_k x_i \frac{\partial^2 f_k}{\partial x_i \partial \kappa^*} = \frac{(\delta + \eta)(\eta + \beta) + \eta \alpha + \delta \beta}{Q_2(\eta + \beta + \alpha)(\mathcal{R}_0^Z - 1)}$$

where $\kappa = \kappa^*$ is a bifurcation parameter. Since b is positive if $\mathcal{R}_0^Z > 1$, it follows that model (1) will undergo backward bifurcation if $a > 0$. On the other hand, the model will exhibit a forward bifurcation when $a < 0$.

We find the bifurcation curve by solving for the bifurcation parameter κ^* in equation (9) as follows.

$$\kappa(Y) = \frac{\gamma(\alpha + \gamma)Y^2 - [\gamma(\eta + \delta + \epsilon) + \delta(\alpha + \delta) + \alpha(\eta + \epsilon)]Y + \delta(\eta + \delta + \epsilon)}{\gamma Y^2 - (\gamma + \delta + \beta + \eta)Y + \delta}.$$

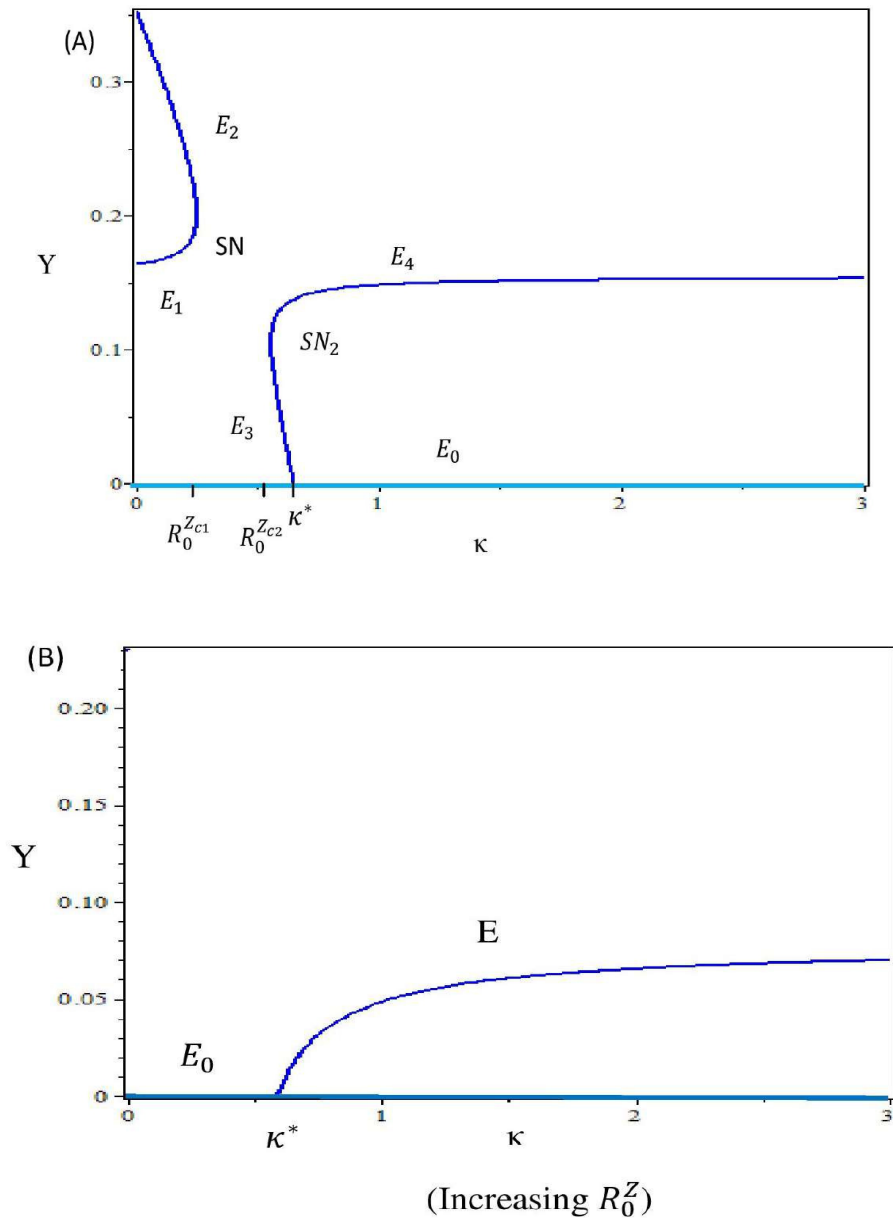


Figure 2: Bifurcation diagrams for varying the transmission rate of non-drinkers to problem drinkers κ (A) showing backward bifurcation, SN indicate the saddle-node for which the proportion of social drinkers collapses, SN_2 indicates another saddle-node for which the problem will persist in the campus and (B) forward bifurcation.

We use the parameter values $\alpha = 1.25, \beta = 0.0000002, \gamma = 1, \delta = 0.2, \epsilon = 0.2$, and $\eta = 0.25$ to plot the bifurcation diagram in figure 2(A) which shows how social drinkers proportion changes

with varying the bifurcation parameter $\kappa = \kappa^*$ or equivalently the basic reproductive number $\mathcal{R}_0^Z$. The threshold parameter $\mathcal{R}_0^Z$ is varied by altering the transmission rate of non-drinkers to problem drinkers, κ . If $\mathcal{R}_0^Z < 1 < \mathcal{R}_0^{Z_{c1}}$, the drinking problem invades the population as the stable drinking-free equilibrium E_0 coexists with a stable endemic equilibrium point E_2 . These equilibrium points coalesce and disappear by a saddle-node (SN) bifurcation at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c1}}$. Thus, the proportion of social drinkers collapses. However, if $\mathcal{R}_0^Z$ is between $\mathcal{R}_0^{Z_{c1}}$ and $\mathcal{R}_0^{Z_{c2}}$ the only stable equilibrium point is the drinking-free equilibrium E_0 . After the extinction regime another critical threshold parameter $\mathcal{R}_0^{Z_{c2}}$ exists for which a second saddle-node bifurcation occurs for the persistence of the drinking problem. This scenario resulting to the emergence of two endemic equilibrium points (E_3 and E_4) at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c2}}$ after the extinction regime ($\mathcal{R}_0^{Z_{c1}}, \mathcal{R}_0^{Z_{c2}}$). In this case, the drinking-free equilibrium (E_0) and endemic equilibrium E_4 are locally stable in the interval $(\kappa^*, \mathcal{R}_0^{Z_{c2}})$ due to the occurrence of backward bifurcation at the bifurcation point $\kappa = \kappa^*$. But for $\kappa > \kappa^*$ the equilibrium E_0 loses stability and a locally stable endemic equilibrium is only E_4 .

It follows that, the critical threshold parameter $\mathcal{R}_0^{Z_{c1}}$ is a tipping point for an unexpected population collapse. This leads to a mono stable system whenever $\mathcal{R}_0^Z > \mathcal{R}_0^{Z_{c1}}$ such that the only global attractor is the drinking-free equilibrium point E_0 . More precisely, there is a saddle-node bifurcation for $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c1}}$, after which the proportion of social drinkers goes extinct. Moreover, there is another critical threshold parameter $\mathcal{R}_0^{Z_{c2}}$ which is greater than $\mathcal{R}_0^{Z_{c1}}$, for the occurrence of a second saddle-node bifurcation. This scenario gives rise to two endemic equilibrium points E_3 and E_4 , as rapidly as the endemic attractor disappears at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c1}}$. They re-emerge after the extinction regime ($\mathcal{R}_0^{Z_{c1}}, \mathcal{R}_0^{Z_{c2}}$).

One can observe from Figure 2(A) that these two saddle-node bifurcations, if they exist are separate from each other and not related by sharing the same saddle-node as in the scenarios with three endemic equilibrium points [6, 7, 9]. This discontinuity has a profound implication for control measure aimed at impacting the threshold parameter $\mathcal{R}_0^Z$. In fact, increasing $\mathcal{R}_0^Z$ before and after the extinction regime (i.e., below $\mathcal{R}_0^{Z_{c1}}$ and above $\mathcal{R}_0^{Z_{c2}}$) can be beneficial for the social drinkers sub-population since it facilitates endemic persistence not extinction. On the other hand, reducing $\mathcal{R}_0^Z$ below $\mathcal{R}_0^{Z_{c2}}$ but above $\mathcal{R}_0^{Z_{c1}}$ can be detrimental to social drinkers, as it causes eradication of this sub-population. Moreover, decreasing $\mathcal{R}_0^Z$ below $\mathcal{R}_0^{Z_{c2}}$ and above $\mathcal{R}_0^{Z_{c1}}$ can be beneficial to the host population since it leads to the stability of the system at drinking-free equilibrium point E_0 .

In Figure 2(B), we show using the parameter values $\alpha = 1.207, \beta = 1.2, \gamma = 1, \delta = 0.2, \epsilon = 0.141$, and $\eta = 0.25$ a forward (transcritical) bifurcation for which the stable drinking-free equilibrium point E_0 loses stability at $\kappa > \kappa^*$ or equivalently $\mathcal{R}_0^Z > 1$ due to the emergence of a unique stable endemic equilibrium point (E). In this case, the classical requirement for drinking problem control (reducing κ below κ^* or equivalently $\mathcal{R}_0^Z$ below 1) could be sufficient to eradicate the drinking problem in the campus.

4.1 Numerical simulations

We plot the numerical solutions of model (1) using Runge-Kutta RK5 with parameter values as presented in Table 1 to see the impact of the transmission rate of non-drinkers to problem drinkers on the dynamics of the model. Figure 3(A) depicts the persistence of drinking problem when $\mathcal{R}_0^Z = 0.3846153846 < 1$. That is, the social and problem drinkers recruit the non drinkers. This scenario correspond to the persistence before $\mathcal{R}_0^{Z_{c1}}$ in Figure 2(A). In Figure 3(B) an extinction scenario corresponding to Figure 2(A) which occur between $\mathcal{R}_0^{Z_{c1}}$ and $\mathcal{R}_0^{Z_{c2}}$ with only a stable drinking-free equilibrium is shown. Such a scenario occurs when $\mathcal{R}_0^Z = 0.5 < 1$. Another persistence scenario is depicted in Figure 3(C) showing that the drinking problem persist in the campus when $\mathcal{R}_0^Z = 2.307692308 > 1$.

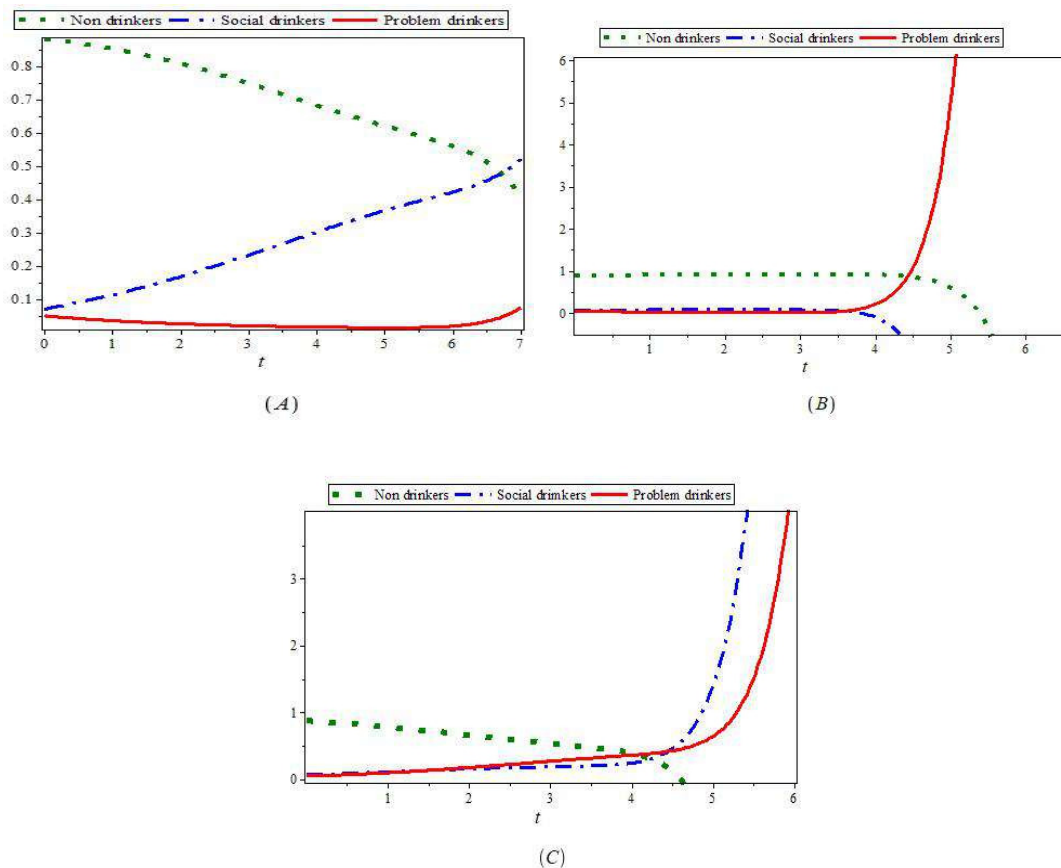


Figure 3: Time diagrams of model (1) with: (A) using the parameter values in Table 1 showing that when $R_0^Z = 0.3846153846 < 1$ drinking problem persist on campus; (B) using $\kappa = 0.75, \epsilon = 0.25, \eta = 0.5, \delta = 0.5$ and other parameter values are as in Table 1 showing that when $R_0^Z = 0.5 < 1$ there is a drinking problem free campus; (C) parameter values as in Table 1 showing that when $R_0^Z = 2.307692308 > 1$ drinking Problem persist on the campus. The graphs of non-drinkers (dot), social drinkers (dash dot) and problem drinkers(solid).

5 Discussion and conclusion

We review the drinking model presented in [13] and investigate its complex dynamics noting that the model exhibits multiple endemic equilibrium points. In particular, the model have a drinking-free equilibrium and problem drinking-free equilibrium point which exists if $\mathcal{R}_0^Y > 1$. These equilibrium points are stable nodes if $\mathcal{R}_0^Y < 1, \mathcal{R}_0^Z < 1$ and $\mathcal{R}_1 < 1, \mathcal{R}_0^Y > 1$, respectively and are saddle points otherwise. Then when the drinking problem persist on campus two equilibrium points emerged called endemic or drinking present equilibrium points. The existence of multiple drinking-present equilibrium points enable us to use Center manifold theory as presented in [5] to investigate the existence of the phenomenon of a backward bifurcation in section 4. In fact, we show in Figure 2 the bifurcation diagram of such phenomenon by varying the bifurcation parameter $\kappa = \kappa^*$ or equivalently the basic reproductive number $\mathcal{R}_0^Z$. That is, the threshold parameter $\mathcal{R}_0^Z$ is varied by altering the transmission rate of non-drinkers to problem drinkers, κ . More precisely, figure 2(A) shows that if $\mathcal{R}_0^Z < 1 < \mathcal{R}_0^{Z_{c1}}$, the drinking problem invades the population as the stable drinking-free equilibrium E_0 coexists with a stable endemic equilibrium point E_2 . These equilibrium points coalesce and disappear by a saddle-node (SN) bifurcation at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c1}}$. Thus, the proportion of social drinkers collapses. However, if $\mathcal{R}_0^Z$ is between $\mathcal{R}_0^{Z_{c1}}$ and $\mathcal{R}_0^{Z_{c2}}$ the only stable equilibrium point is the drinking-free equilibrium E_0 . After the extinction regime another critical threshold parameter $\mathcal{R}_0^{Z_{c2}}$ exists for which a second saddle-node

bifurcation occurs resulting in the persistence of the drinking problem. This scenario results to the emergence of two endemic equilibrium points (E_3 and E_4) at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c2}}$ after the extinction regime ($\mathcal{R}_0^{Z_{c1}}, \mathcal{R}_0^{Z_{c2}}$) due to the occurrence of the phenomenon of backward bifurcation. In this case, the stable drinking-free equilibrium (E_0) coexist with a stable endemic equilibrium E_4 in the interval $(\mathcal{R}_0^{Z_{c2}}, \kappa^*)$. Then for $\kappa > \kappa^*$ the equilibrium E_0 loses stability and a locally stable endemic equilibrium is the only E_4 . This result is similar to the findings in [10, 18].

Thus, the critical threshold parameter $\mathcal{R}_0^{Z_{c1}}$ is a tipping point for an unexpected population collapse. This leads to a mono stable system whenever $\mathcal{R}_0^Z > \mathcal{R}_0^{Z_{c1}}$ such that the only global attractor is the drinking-free equilibrium point E_0 . In particular, there is a saddle-node (SN) bifurcation at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c1}}$, after which the proportion of social drinkers goes extinct. Moreover, there is another critical threshold parameter $\mathcal{R}_0^{Z_{c2}}$ which is greater than $\mathcal{R}_0^{Z_{c1}}$, for the occurrence of a second saddle-node bifurcation (SN_2). This scenario gives rise to two endemic equilibrium points E_3 and E_4 , as rapidly as the endemic attractor disappears at $\mathcal{R}_0^Z = \mathcal{R}_0^{Z_{c1}}$. These equilibrium points re-emerge after the extinction regime ($\mathcal{R}_0^{Z_{c1}}, \mathcal{R}_0^{Z_{c2}}$).

We observe from Figure 2(A) that these two saddle-node bifurcations, if they exist are separate from each other and not related by sharing the same saddle-node as in the scenarios with three endemic equilibrium points [9, 6, 7]. This discontinuity has a profound implication for control measure aimed at impacting the threshold parameter $\mathcal{R}_0^Z$. In fact, increasing $\mathcal{R}_0^Z$ before and after the extinction regime (i.e., below $\mathcal{R}_0^{Z_{c1}}$ and above $\mathcal{R}_0^{Z_{c2}}$) can be beneficial for the social drinkers sub-population since it facilitates endemic persistence not extinction. On the other hand, reducing $\mathcal{R}_0^Z$ below $\mathcal{R}_0^{Z_{c2}}$ but above $\mathcal{R}_0^{Z_{c1}}$ can be detrimental to social drinkers, as it causes eradication of this sub-population. Moreover, decreasing $\mathcal{R}_0^Z$ below $\mathcal{R}_0^{Z_{c2}}$ and above $\mathcal{R}_0^{Z_{c1}}$ can be beneficial to the host population since it leads to the stability of the system at drinking-free equilibrium point E_0 .

We also show in Figure 2(B), a forward (transcritical) bifurcation for which the stable drinking-free equilibrium point E_0 loses stability at $\kappa > \kappa^*$ or equivalently $\mathcal{R}_0^Z > 1$ due to the emergence of a unique stable endemic equilibrium point (E). In this case, the classical requirement for drinking problem control (reducing κ below κ^* or equivalently $\mathcal{R}_0^Z$ below 1) could be sufficient to eradicate the problem in the campus.

In order to complete our investigations, we simulate the numerical solution of model (1) using Runge-Kutta RK5 with Maple 18 software and the parameter values presented in Table 1. Indeed, we show in Figure 3 the persistence and extinction scenarios of the drinking problem. To be more precise, when $\mathcal{R}_0^Z = 0 : 3846153846 < 1$ ($\kappa \ll \kappa^*$) and $\mathcal{R}_0^Z = 2 : 3076692308 > 1$ ($\kappa > \kappa^*$) the drinking problem persist in the campus environment (see Figures, 3(A), and 3(C), respectively for the persistence). This scenario is similar to that of Figure 2(A) below $\mathcal{R}_0^{Z_{c1}}$ and above $\mathcal{R}_0^{Z_{c2}}$. On the other hand, Figure 3(B) shows that the drinking problem goes to extinct if $\mathcal{R}_0^Z = 0.5 < 1$ since the population collapse as in the extinction regime ($\mathcal{R}_0^{Z_{c1}}, \mathcal{R}_0^{Z_{c2}}$) shown in Figure 3(A).

In conclusion, although the campus alcohol abuse may be reduced by minimizing the ability of problem drinkers to directly recruit non-drinkers as reported in [13] but our analysis reveals that such strategy is to be applied with caution. This is because the occurrence of two separate saddle-node bifurcations make the control more difficult. Indeed, reducing the direct recruitment of non-drinkers to problem drinkers below the first saddle-node SN and above the second one SN_2 can leads to the persistence of drinking problem in the campus.

Conflict of interest

All the authors state that there is no conflict of interest.

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On the constrained optimal control problems with mixed constraints

A.S. Afolabi*

Abstract

The analytical solutions of optimal control problems with mixed constraints are examined. The analytical solutions are obtained by applying the first order optimality conditions on the Lagrangian function. The method of fundamental matrix is then used to solve the resulting non-homogeneous system of first order ordinary differential equations. This leads to the analytical solutions of the state and control variables and the objective function value. Two examples of optimal control problems constrained by ordinary differential equations and inequalities are considered.

Keywords: analytical solution; Halmitonian; Lagrangian; fundamental matrix; fundamental solution

1 Introduction

Optimization involves finding the minimum or maximum value of a function called extremum. In many real life processes such as engineering and epidemiology, decision Makers have to take many technological and managerial decisions at several stages in order to minimize the effort required or maximize the benefit derived. Such decisions are crucial because it ensures optimum use of available resources.

Constrained optimization is the process of minimizing or maximizing an objective functional with respect to some decision variables in the presence of some constrained on the decision variables. These constraints create more problems while finding the minimum or maximum value of the objective function as compared to unconstrained ones. In most situation, the constraints have some effects on the objective functional. However, there are few situations under which the constraints do not have any influence on the minimum point. Here, the constrained minimum of the problem is the same as the unconstrained minimum ([20], [21]). Majority of the available methods are designed for unconstrained optimization, where no imitations are placed on the design variables.

Optimal Control theory deals with finding a control law for a dynamical system such that an objective function is optimized over a given period of time. It deals with systems that can be controlled. The theory of optimal control is well developed for many decades ([22]). It requires a performance index or cost functional $J(t, x(t), u(t))$, a set of state variables $(t, x(t) \in X)$, a set of control variables $(u(t) \in U(t))$ in a time t , with $t_0 \leq t \leq t_f$. To maximized a given objective functional, one must obtain the associated state variable $x(t)$ and a piecewise continuous control $u(t)$. Mathematical control theory deals with the basic principles underlying the analysis and design of control systems arising from some areas of applied Mathematics. To control any system means to influence its behaviour so as to achieve a desired goal. Mathematical techniques are

*Department of Mathematical Sciences, Federal University of Technology, Akure, Ondo State, Nigeria; email: asafolabi@futa.edu.ng

built to implement this influence [4].

One of such techniques is Pontryagin's principle. Pontryagin's maximum (or minimum) principle was formulated in 1956 by the Russian mathematician Lev Pontryagin and his students as Pontryagin's maximum principle and proved historically based on maximizing the Hamiltonian [5]. For the validity of the statement to be established, the sufficient conditions must be satisfied in order to determine the nature of the turning point [7].

A brief historical survey of the development of the theory of optimal control and calculus of variations was given by ([18]). The calculus of variations uses variations, which are small changes in functionals in order to minimize or maximize the functionals. EulerLagrange equation of the calculus of variations is used to find functions that minimize or maximize functionals. Several other well-defined new areas in optimization theory were reviewed in [15]. A number of numerical techniques have also been developed over the years for optimal control problems that have analytical solutions and the ones that did not have analytical solutions by [2], [3], [6], [12], [13], [14], [16],[17]and [19].

In the early years of optimal control, the favoured approach for solving optimal control problems was that of indirect method. In an indirect method, the calculus of variations is employed to obtain the first-order optimality conditions [10]. [8] considered an optimal control problem involving equality and inequality state-control constraints with fixed initial and final endpoint state constraints. The control variables belong to the class of piecewise continuous functions while and state variables belong to the class of piecewise smooth functions respectively. The paper provided a direct derivation of second order necessary conditions, based on a variational approach where a certain quadratic form is non-negative. The inequality constraints are treated as equalities of the active constraints for this set, thus producing a restrictive set of optimality conditions.

The analytic and numeric solutions of discretized constrained optimal control problems were obtained ([11]). The analytic and numeric solutions of general continuous linear quadratic optimal control problem were presented. The associated general Riccati differential equation was solved by numerical-analytical approach using variational iteration method. Numerical solutions of the constrained optimal control problem were obtained via quadratic programming of the discretized continuous optimal control problems by shooting method and the conjugate gradient method (CGM). The results showed that both the analytical and numerical solutions agreed favourably. In the paper, the analytical solution and numerical solution by penalty function methods were presented and convergence analysis was conducted.

The analytical solutions of optimal control problems constrained by ordinary differential equations were obtained. The necessary condition of optimality was applied to the Hamiltonian function in order to obtain the analytical conditions of this class of problems. This leads to the optimal state, control and adjoint variables and hence the optimal objective function value of optimal control problems constrained by ordinary differential equations [1]. The theoretical analysis of [1] is extended to optimal control problems with mixed constrained in this paper. The analytical solutions of the state, control variables together with the objective function values were obtained. The effects of active and inactive inequality constraints on the state and control variables were also examined.

2 Methodology

Consider an optimal control problem with mixed constrained given as

$$\begin{aligned} &\text{Minimize } f(x, u, t), \\ &\text{subject to } h(x, u, t) = 0, \\ &\quad g(x, u, t) \leq 0, \end{aligned} \quad (1)$$

where $f : \mathbb{R}^n \times \mathbb{R}^m \times \mathbb{R} \rightarrow \mathbb{R}$, $h : \mathbb{R}^n \times \mathbb{R}^m \times \mathbb{R} \rightarrow \mathbb{R}^p$, $g : \mathbb{R}^n \times \mathbb{R}^m \times \mathbb{R} \rightarrow \mathbb{R}^r$ are given functions, $m \leq n$, $p \leq n$, $r \leq n$, $x \in \mathbb{R}^n$ and $u \in \mathbb{R}^m$. The components of h and g are denoted by $h_1, h_2, \dots, h_p$ and $g_1, g_2, \dots, g_r$, respectively. Equation (1) can be converted to an equality constrained problem by introducing a vector of additional variables $z = (z_1, z_2, \dots, z_r)$. The problem then becomes

$$\begin{aligned} &\text{Minimize } f(x, u, t), \\ &\text{subject to } h_i(x, u, t) = 0, \quad i = 1, 2, \dots, p, \\ &\quad g_j(x, u, t) + z_j^2(x, u, t) = 0, \quad j = 1, 2, \dots, r. \end{aligned} \quad (2)$$

2.1 Analytical Solution of Optimal Control Problems with Mixed Constraints and Real Coefficients

$$\text{Minimize } I(x, u) = \int_0^T (px^2(t) + qu^2(t))dt, \quad (3)$$

$$\text{subject to } \dot{x}(t) = ax(t) + bu(t), \quad (4)$$

$$cx(t) + du(t) \leq 0, \quad x(0) = x_0, \quad (5)$$

where a, b, c, d are real constants and $p, q > 0$.

Theorem 1.1 *Given the optimal control variable $u^*(t)$ and the solution $x^*(t)$ of the state system (4) that minimizes $I(x, u, t)$ over U (where U is the permissible set of controls), then there exists an adjoint variable $\mu(t)$ satisfying*

$$\dot{\mu}(t) = -2px(t) - a\mu(t) - c\lambda, \quad t \in [0, T], \quad (6)$$

and with the transversality condition

$$\mu(T) = 0, \quad (7)$$

$$u^*(t) = \frac{-b\mu(t) - d\lambda}{2q}. \quad (8)$$

Proof. With appropriate conditions on the end points, adjoint variable $\mu(t)$ can be introduced by forming the required augmented functional from (3)-(5). The Hamiltonian function is given as

$$H(x, u, \mu) = px^2(t) + qu^2(t) + \mu(ax(t) + bu(t)), \quad (9)$$

and the Lagrangian function is defined as

$$L(x, u, \mu, \lambda) = px^2(t) + qu^2(t) + \mu(ax(t) + bu(t)) + \lambda(cx(t) + du(t)). \quad (10)$$

From the knowledge of calculus of variation, it seems plausible that the optimal solutions for our initial optimal control problem ought to be the Euler-Lagrange equations for L regarded as function of four variables (x, u, μ, λ) . Thus, the E-L system can be written as

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{x}} \right] = \frac{\partial L}{\partial x}, \quad (11)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{u}} \right] = \frac{\partial L}{\partial u}, \quad (12)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{\mu}} \right] = \frac{\partial L}{\partial \mu}, \quad (13)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{\lambda}} \right] = \frac{\partial L}{\partial \lambda}. \quad (14)$$

Equations (11)-(14) now give the following equations

$$\dot{\mu}(t) = -2px(t) - a\mu(t) - c\lambda, t \in [0, T], \quad (15)$$

$$u^*(t) = \frac{-b\mu(t) - d\lambda}{2q}, \quad (16)$$

$$\dot{x}(t) = ax(t) - b \left(\frac{b\mu(t) + d\lambda(t)}{2q} \right), \quad (17)$$

$$cx + du = 0. \quad (18)$$

The optimality system of equations (15) and (17) represent a linear 2-point boundary value non-homogeneous ordinary differential equations, which are the necessary conditions for an optimal control $u^*(t)$. The two-point boundary value problem can be written in matrix form as

$$\begin{pmatrix} \dot{x}(t) \\ \dot{\mu}(t) \end{pmatrix} = \begin{pmatrix} -2p & -a \\ a & \frac{-b^2}{2q} \end{pmatrix} \begin{pmatrix} x(t) \\ \mu(t) \end{pmatrix} + \begin{pmatrix} -c \\ \frac{-bd}{2q} \end{pmatrix} \lambda(t). \quad (19)$$

Let

$$M = \begin{pmatrix} -2p & -a \\ a & \frac{-b^2}{2q} \end{pmatrix}. \quad (20)$$

The eigenvalues of M are

$$\lambda_1 = -\frac{4pq - A + b^2}{4q}, \quad (21)$$

$$\lambda_2 = -\frac{4pq + A + b^2}{4q}, \quad (22)$$

where $A = \sqrt{-(b^2 + 4aq - 4pq)(-b^2 + 4aq + 4pq)}$ and its eigenvectors are

$$U_1 = \begin{pmatrix} \frac{4b^2 - (4pq - A + b^2)}{4aq} \\ 1 \end{pmatrix}, \quad (23)$$

and

$$U_2 = \begin{pmatrix} \frac{4b^2 - (4pq + A + b^2)}{4aq} \\ 1 \end{pmatrix}. \quad (24)$$

Thus, the solution of the complementary part of (19) is given by

$$V(t) = c_1 \vec{U}_1 e^{\lambda_1 t} + c_2 \vec{U}_2 e^{\lambda_2 t}. \quad (25)$$

The general solution can be written in matrix form as

$$\begin{pmatrix} x(t) \\ \mu(t) \end{pmatrix} = \begin{pmatrix} \frac{4b^2 - (4pq - A + b^2)}{4aq} e^{\lambda_1 t} & \frac{4b^2 - (4pq + A + b^2)}{4aq} e^{\lambda_2 t} \\ e^{\lambda_1 t} & e^{\lambda_2 t} \end{pmatrix} \begin{pmatrix} c_1 \\ c_2 \end{pmatrix}. \quad (26)$$

Hence, applying the method of fundamental matrix [9], this implies that

$$X(t) = \psi(t)C \quad (27)$$

where

$$\psi(t) = \begin{pmatrix} \frac{4b^2-(4pq-A+b^2)}{4aq} e^{\lambda_1 t} & \frac{4b^2-(4pq+A+b^2)}{4aq} e^{\lambda_2 t} \\ e^{\lambda_1 t} & e^{\lambda_2 t} \end{pmatrix} \quad (28)$$

is a 2×2 matrix with the fundamental solutions $x_1(t)$ and $x_2(t)$ along the columns.

Hence,

$$\psi^{-1}(t) = \frac{1}{-2A} \begin{pmatrix} e^{\lambda_2 t} & -\frac{4b^2-(4pq+A+b^2)}{4aq} e^{\lambda_2 t} \\ -e^{\lambda_1 t} & \frac{4b^2-(4pq-A+b^2)}{4aq} e^{\lambda_1 t} \end{pmatrix}. \quad (29)$$

This implies that

$$\psi^{-1}(t)g(s) = \begin{pmatrix} -\frac{e^{\lambda_2 t}}{2A} & \frac{4b^2-(4pq+A+b^2)}{8aqA} e^{\lambda_2 t} \\ \frac{e^{\lambda_1 t}}{2A} & -\frac{4b^2-(4pq-A+b^2)}{8aqA} e^{\lambda_1 t} \end{pmatrix} \begin{pmatrix} -c \\ \frac{-bd}{2q} \end{pmatrix} \lambda(s) \quad (30)$$

$$\psi^{-1}(s)g(s) = \begin{pmatrix} \frac{c\lambda(s)e^{\lambda_2 s}}{2A} - bd\left(\frac{4b^2-(4pq+A+b^2)}{16aq^2A}\right)\lambda(s)e^{\lambda_2 s} \\ -\frac{c\lambda(s)e^{\lambda_1 s}}{2A} + bd\left(\frac{4b^2-(4pq-A+b^2)}{16aq^2A}\right)\lambda(s)e^{\lambda_2 s} \end{pmatrix}, \quad (31)$$

where $g(t) = \begin{pmatrix} -c \\ \frac{-bd}{2q} \end{pmatrix} \lambda(t)$ is the non-homogeneous part of equation (19).

$$\int \psi^{-1}(s)g(s)d(s) = \int \begin{pmatrix} \frac{c\lambda(s)e^{\lambda_2 s}}{2A} - bd\left(\frac{4b^2-(4pq+A+b^2)}{16aq^2A}\right)\lambda(s)e^{\lambda_2 s} \\ -\frac{c\lambda(s)e^{\lambda_1 s}}{2A} + bd\left(\frac{4b^2-(4pq-A+b^2)}{16aq^2A}\right)\lambda(s)e^{\lambda_2 s} \end{pmatrix} d(s) \quad (32)$$

$$\psi(t) \int \psi^{-1}(s)g(s)d(s) = \begin{pmatrix} \frac{4b^2-(4pq-A+b^2)}{4aq} e^{\lambda_1 t} & \frac{4b^2-(4pq+A+b^2)}{4aq} e^{\lambda_2 t} \end{pmatrix} \int \begin{pmatrix} \frac{c\lambda(s)e^{\lambda_2 s}}{2A} - bd\left(\frac{4b^2-(4pq+A+b^2)}{16aq^2A}\right)\lambda(s)e^{\lambda_2 s} \\ -\frac{c\lambda(s)e^{\lambda_1 s}}{2A} + bd\left(\frac{4b^2-(4pq-A+b^2)}{16aq^2A}\right)\lambda(s)e^{\lambda_2 s} \end{pmatrix} d(s) \quad (33)$$

This implies that the general solution is given by

$$X(t) = \psi(t)C + \psi(t) \int \psi^{-1}(s)g(s)d(s). \quad (34)$$

3 Solved examples

Example 3.1

$$\text{Minimize } I(t, x(t), u(t)) = \int_0^4 u^2(t)dt \quad (35)$$

$$\dot{x} = x + u, \quad x(0) = 0 \quad (36)$$

$$u(t) \leq 5 \quad (37)$$

Solution:

The Hamiltonian function, H , is given by

$$H(x, u, \mu) = u^2(t) + \mu(x(t) + u(t)), \quad (38)$$

and the Lagrangian function L is given by

$$L(x, u, \mu, \lambda) = u^2(t) + \mu(x(t) + u(t)) + \lambda(u(t) - 5). \quad (39)$$

From the knowledge of calculus of variations, it seems plausible that the optimal solutions for our initial optimal control problem ought to be the Euler-Lagrangian equations for L regarded as function of four variables $(x(t), u(t), \mu(t), \lambda(t))$. Thus, the E-L system can be written as

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{x}} \right] = \frac{\partial L}{\partial x}, \quad (40)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{u}} \right] = \frac{\partial L}{\partial u}, \quad (41)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{\mu}} \right] = \frac{\partial L}{\partial \mu}, \quad (42)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{\lambda}} \right] = \frac{\partial L}{\partial \lambda}. \quad (43)$$

Equation (40) implies that

$$\dot{\mu}(t) = -\mu, \quad (44)$$

whether the constraint is active or not. The transversality boundary condition implies that $\mu(4) = \frac{\partial \psi}{\partial x} = -1$ where $\psi = -x(4)$ is the terminal cost. Hence,

$$\mu(t) = -e^{4-t}. \quad (45)$$

Assuming that the control constraint is initially active for some period of time, then $\lambda \geq 0, u = 5$, then

$$L_u = 2u(t) + \mu + \lambda = 0, \quad (46)$$

$$\lambda(t) = -10 - \mu = e^{4-t} - 10. \quad (47)$$

For $\lambda \geq 0$, then

$$\lambda(t) = e^{4-t} - 10 \geq 0. \quad (48)$$

Hence,

$$t \leq 4 - \ln(10) \text{ and } \lambda \geq 0. \quad (49)$$

On the other hand, if the constraint is inactive, then

$$L_u = 2u(t) + \mu = 0 \quad (50)$$

$$L_u = 2u(t) + \mu = 0 \quad (51)$$

$$u(t) = -\frac{1}{2}\mu(t). \quad (52)$$

The optimal control for this problem is given as

$$u^*(t) = \begin{cases} 5, & \text{if } t \leq 4 - \ln(10) \\ \frac{1}{2}e^{4-t}, & \text{if } t \geq 4 - \ln(10) \end{cases}$$

To obtain the state variable in the two arcs, we examine the two control inputs obtained above. When $u(t) = 5$, equation (3.2) becomes

$$\dot{x}(t) = x(t) + 5, \quad (53)$$

since $x(0) = 0$, then

$$x(t) = 5e^t - 5. \quad (54)$$

Similarly, in view of (52), equation (3.2) becomes

$$\dot{x}(t) = x(t) - \frac{1}{2}e^{4-t}, \quad (55)$$

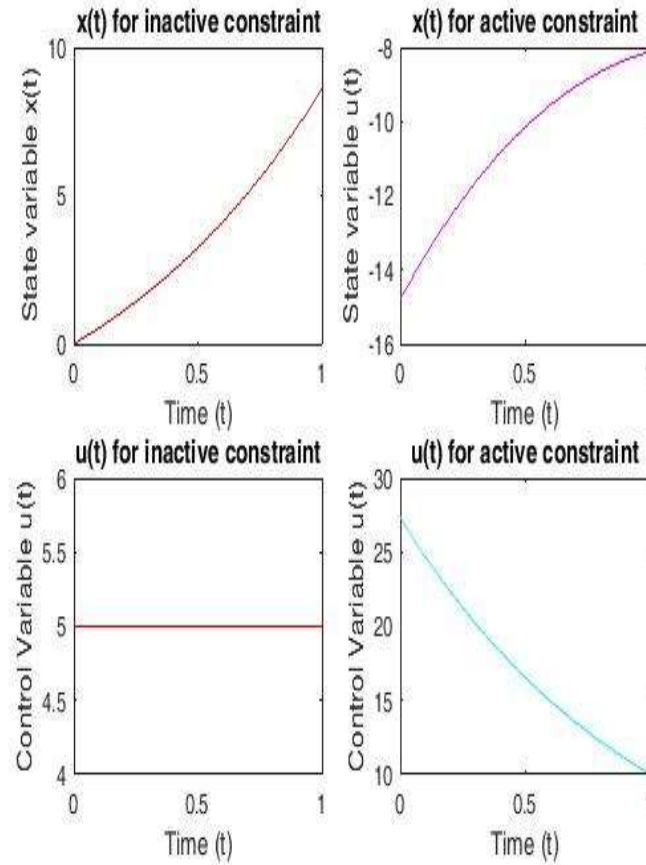


Figure 1: State Variable $x(t)$ and Control Variable $u(t)$ for Active and Inactive forms of the Inequality Constraint Against Time (t)

since $x(0) = 0$, then

$$x(t) = -\frac{1}{4}e^{4-t} + (5 - 25e^{-4})e^t. \quad (56)$$

Hence, the state variable is given by

$$x^*(t) = \begin{cases} 5e^t - 5, & \text{if } t \leq 4 - \ln(10) \\ -\frac{1}{4}e^{4-t} + (5 - 25e^{-4})e^t, & \text{if } t \geq 4 - \ln(10) \end{cases}$$

Hence, when $u^*(t) = 5$ and $x^*(t) = 5e^t - 5$ for $t \leq 4 - \ln(10)$, then

$$\text{Minimize } I(t, x(t), u(t)) = (5e^4 - 5) + \int_0^4 5^2 dt = 372.9908. \quad (57)$$

Similarly, when $u^*(t) = \frac{1}{2}e^{4-t}$ and $x^*(t) = -\frac{1}{4}e^{4-t} + (5 - 25e^{-4})e^t$ for $t \geq 4 - \ln(10)$, then

$$\text{Minimize } I(t, x(t), u(t)) = -\frac{1}{4}e^{4-4} + (5 - 25e^{-4})e^4 + \int_0^4 \left(\frac{1}{2}e^{4-t}\right)^2 dt = 992.7302. \quad (58)$$

Example 3.2

$$\text{Minimize } I(t, x(t), u(t)) = \int_0^1 (x^2(t) + u^2(t)) dt, \quad (59)$$

$$\dot{x} = 4x + 5u, \quad x(0) = 1 \quad (60)$$

$$2x(t) + 4u(t) \leq 0. \quad (61)$$

Solution:

The Hamiltonian function, H , is given by

$$H(x, u, \mu) = x^2(t) + u^2(t) + \mu(-4x - 5u), \quad (62)$$

and the Lagrangian function, L , is given by

$$L(x, u, \mu, \lambda) = x^2(t) + u^2(t) + \mu(-4x - 5u) + \lambda(2x(t) + 4u(t)). \quad (63)$$

From the knowledge of calculus of variations, it seems plausible that the optimal solutions for our initial optimal control problem ought to be the Euler-Lagrangian equations for L regarded as function of four variables $(x(t), u(t), \mu(t), \lambda(t))$. Thus, the E-L system can be written as

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{x}} \right] = \frac{\partial L}{\partial x}, \quad (64)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{u}} \right] = \frac{\partial L}{\partial u}, \quad (65)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{\mu}} \right] = \frac{\partial L}{\partial \mu}, \quad (66)$$

$$\frac{d}{dt} \left[\frac{\partial L}{\partial \dot{\lambda}} \right] = \frac{\partial L}{\partial \lambda}. \quad (67)$$

Equation (64) implies that

$$\dot{\mu}(t) = -2x(t) + 4\mu(t) - 2\lambda(t). \quad (68)$$

Equation (65) implies that

$$2u(t) - 5\mu(t) + 4\lambda(t) = 0. \quad (69)$$

This implies that

$$\lambda^*(t) = \frac{-2u(t) + 5\mu(t)}{4}. \quad (70)$$

Equation (66) leads to the state equation whereas equation (67) implies that

$$2x(t) + 4u(t) = 0. \quad (71)$$

Assuming that the control constraint is initially active for some period of time, then $\lambda \geq 0$, then

$$u^*(t) = \frac{x(t)}{2} \quad (72)$$

In view of equation (72), equation (60) becomes

$$\dot{x}(t) = \frac{13}{2}x(t). \quad (73)$$

Since $x(0) = 1$, then

$$x^*(t) = e^{\frac{13}{2}t}. \quad (74)$$

The optimal control given by equation (72) then becomes

$$u^*(t) = \frac{e^{\frac{13}{2}t}}{2}. \quad (75)$$

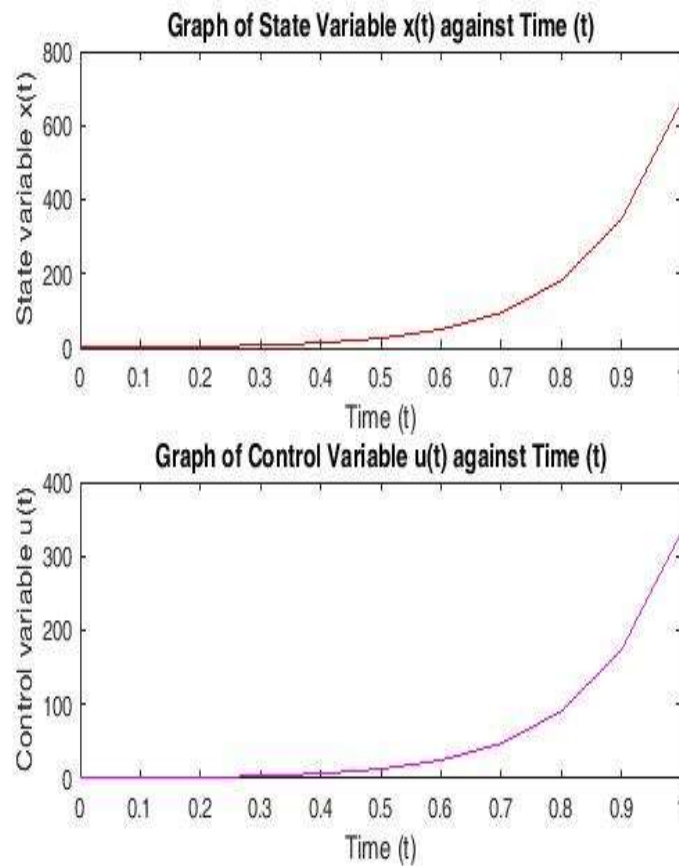


Figure 2: Graphical Representation of the Solution of Optimal Control Problem Constrained by Algebraic-differential Equations

In view of equations (70), (74) and (75), equation (68) then becomes

$$\dot{\mu}(t) = -\frac{3}{2}\mu + \frac{3}{2}e^{\frac{13}{2}t}, \quad (76)$$

whether the constraint is active or not. The transversality boundary condition implies that $\mu(1) = \frac{\partial \psi}{\partial x} = 0$ where $\psi = 0$ is the terminal cost. Hence,

$$\mu^*(t) = \frac{3}{16}e^{-\frac{3t}{2}}(e^{8t} - e^8). \quad (77)$$

It follows immediately that Equation (70) becomes

$$\lambda^*(t) = -\frac{1}{2}\left(\frac{e^{\frac{13}{2}t}}{2}\right) + \frac{5}{4}\left(\frac{3}{16}e^{-\frac{3t}{2}}(e^{8t} - e^8)\right). \quad (78)$$

Hence,

$$\lambda^*(t) = \frac{15}{64}e^{-\frac{3t}{2}}(e^{8t} - e^8) - \frac{1}{4}e^{\frac{13}{2}t}. \quad (79)$$

The objective function value is given as 42540.

4 Concluding remarks

The analytical solutions of continuous quadratic optimal control problems constrained by ordinary differential equations and inequalities have been presented. The analytical solution was obtained by applying the first order optimality conditions on the Lagrangian function. The resulting system of non-homogeneous first order ordinary differential equations was then solved using the method of fundamental matrix to obtain the optimal values of the state, control and adjoint variables. This leads to the optimal value of the cost functional. Two examples are considered and the results show the effects of active and inactive constraints on the state and control variables and the objective function values.

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On the reproduction number of a model for the transmission dynamics of Malaria and Pneumonia co-infection with mass action incidence

M.O. Adeniyi*, T.O. Oluyo[†], and O.Y. Oludoun[‡]

Abstract

Pneumonia and malaria are two of the world's most lethal diseases. The geographic overlap of these diseases in Sub-Saharan Africa facilitates co-infection. The effect of the basic reproduction number on the transmission dynamics of malaria and pneumonia co-infection is investigated in this study. The co-infection model for malaria and pneumonia revealed that there is a relationship between the reproduction number for malaria and pneumonia. The study also discovered that pneumonia disease can aggravate malaria in the population. Considering the global stability of the model, the disease free equilibrium (DFE) account for co-infected persons must be less than unity to ensure global stability of the model.

Keywords: malaria; malaria-pneumonia; disease-free equilibrium; global stability; basic reproduction number; pneumonia

1 Introduction

Mathematical models of infectious diseases allow us to deduce the state and progression of an outbreak based on the information available. Although the burden of infectious diseases has surpassed the burden of chronic diseases and psychiatric illnesses in many countries, infectious diseases and health conditions continue to cause morbidity and mortality in many countries and communities. Malaria, measles, hepatitis B, cholera, and pneumonia epidemics are characterized in Sub-Saharan Africa owing to poor hygiene, sanitation, malnutrition, and perinatal conditions, with poverty being the most visible feature of disease spread in the region [20, 18, 17].

Malaria is a febrile illness caused by Plasmodium parasites that are transmitted to humans through mosquito bites from infected female Anopheles mosquitoes [3]. Human malaria is caused by five parasitic species, of which Plasmodium falciparum is the deadliest and most common on the African continent. According to the most recent WHO report, approximately 241 million cases of malaria were recorded in 2020, a 24 percent increase over the previous year, resulting in a 35 percent increase in deaths compared to the previous year [24].

Pneumonia is the leading cause of respiratory morbidity in over two million children under the age of five. It's a lung infection brought on by bacteria, viruses, fungi, or other pathogens [10]. Streptococcus pneumoniae, also known as pneumococcus, is the most common cause. Pneumococcus spreads through micro aspiration of oropharyngeal organisms and inhalation of aerosols. Children become severely ill with high fevers and rapid breathing. Infants usually suffer from convulsions, unconsciousness, hypothermia, lethargy, and feeding problems [10, 12,

*Department of Mathematical Science, Lagos State University of Science and Technology, Lagos State, Nigeria; Email: adeniyi.m@mylaspotech.edu.ng

[†]Department of Applied Mathematics, Ladoke Akintola University of Technology, Ogbomosho, Oyo State, Nigeria; Email: tooluyo@lautech.edu.ng

[‡]Corresponding Author: Department of Mathematics, Bowen University, Iwo, Osun State, Nigeria; Email: oludoun.olajumoke@lmu.edu.ng

21]. Fungal pneumonia is uncommon, but it can occur in people who have weakened immune systems due to AIDS, immuno-suppressive drugs, or other medical issues [7]. Infections caused by parasites typically enter the body through the skin or mouth and travel through the body to the lungs, usually through the blood [23]. Some people are more susceptible to pneumonia than others. Infection is especially dangerous for people over the age of 65, infants, and children under the age of five [6]. People with weakened immune systems are more likely to become infected with cytomegalovirus (CMV) [8].

Pneumonia and malaria are two of the world's most lethal diseases. In Sub-Saharan Africa, the geographic overlap of these diseases facilitates co-infection. Oluyo and Adeniyi [15] showed the consequences of co-infection of pneumonia and malaria parasites and that their combination leads to worse outcomes, posing a major public health concern.

1.1 Related works

Lawi et al. [11] presented and analyzed a deterministic model of malaria and pneumonia dynamics. The findings revealed that increasing the treatment rate leads to a reduction in new disease incidences. Due to increased mobility, a decrease in pneumonia cases would result in an increase in malaria cases. A study by Niger and Gumel [13] examined the role of repeated exposure on the transmission dynamics of malaria in a human population using a deterministic model. The study revealed that when the associated reproduction threshold is less than unity, a stable disease-free equilibrium coexists with a stable endemic equilibrium. According to a thorough qualitative analysis of the model, which includes three immunity stages and the absence of reinfection, it was shown that when the reproduction threshold exceeds unity, mass action incidence has a globally asymptotically stable endemic equilibrium. Olaniyi and Obabiyi [16] developed a model considering the transmission of *Plasmodium falciparum* malaria between humans and mosquitoes using a seven-dimensional ordinary differential equation with non-linear forces of infection in the form of saturated incidence rates. It was shown that antibodies were produced in both human and mosquito populations in response to the presence of parasite-causing malaria. Consequentially, humans must increase their antibody production in order to combat parasite invasion in the bloodstream. Also, an individual's immunity state, that is, the infected person's general health and nutritional status, is a factor in preventing or aiding the occurrence of malaria. Under different levels of sanitation strategy, a deterministic mathematical model for malaria was considered in [19]. According to the study, the results revealed that the malaria model has a backward bifurcation, implying that malaria eradication requires more than just lowering $R_m < 1$, and that other factors such as sanitation must be considered and maintained, better sanitation reduces the number of mosquito bites and the rate of malaria transmission. Oluyo and Adeniyi [15] conducted a study on a thorough mathematical analysis of the malaria-pneumonia co-infection model. The local and global stabilities of the malaria only, pneumonia only, and full malaria-pneumonia models were investigated. Their result showed that the malaria only model exhibits a backward bifurcation, implying that malaria will be cleared from the population if a critical threshold is met, while the pneumonia only model reveals that pneumonia disease is cleared from the population if a critical threshold is met. Using a nonlinear mathematical model, [14] proposed and analyzed the transmission dynamics of malaria disease in a population, the model was qualitatively evaluated to incorporate time-dependent variable controls, with the goal of reducing the spread of malaria disease.

This paper is organized as follows; the section 2 entails the mathematical formulation of the malaria and pneumonia co-infection model. In Section 3, the mathematical analysis of the model which is sub sectioned into section 3.1 as the feasible region and well-posedness of the model, section 3.2, the equilibrium analysis of the model, the basic reproduction number of the model was shown in section 3.3, the stability analysis of the malaria and pneumonia co-infection was shown in section 3.4. Section 4, present numerical simulation, discussion of result and concluding remarks are presented in Section 5.

2 Mathematical formulation

The development of the malaria and pneumonia co-infection model in the host(human) and vector(mosquito) populations starts with dividing the total human-mosquito population into two distinct compartments written as $N_h(t)$ and $N_m(t)$, representing the total population sizes for humans and mosquitoes at a given time t , respectively. Consequently, within the scope of this discussion from this point on, the use of the time t dependency is suppressed. Following the modification of the work of Lawi et al [11], Oluyo and Adeniyi [15] studied a mathematical model on the co-infection of malaria and pneumonia with mass action incidence. In this study, the human population will be decomposed further into the following seven epidemiological compartments namely: susceptible humans S_h , exposed humans to malaria E_h , exposed humans to pneumonia E_p , exposed humans to both malaria and pneumonia E_{hp} , infected humans with malaria I_h , infected humans with pneumonia I_p and infected humans with both malaria and pneumonia I_{hp} such that $N_h = S_h + E_h + E_p + E_{hp} + I_h + I_p + I_{hp}$. For clarity of exposition, the states variables are identified with their corresponding class. For example, taking into consideration human from the class E_h , it is considered that E_h is a function and not a class. In general, the vector population is sub-divided the population into three classes: susceptible mosquito S_m , exposed mosquito E_m and infected mosquito I_m . Thus, the total mosquito population is $N_m = S_m + E_m + I_m$.

According to Filipe et al [9], the rate of infection between human and mosquito populations depend on many factors such as biting rate of the mosquito α (i.e. the number of bites per mosquito) and the transmission rate β_{mh} . The force of malaria infection for humans (i.e. from mosquitoes to humans) is defined as the product of the average number of bites per human α and the transmission rate β_{mh} from a mosquito in the I_m class to a susceptible humans in S_h class i.e.,

$$\lambda_h = \alpha\beta_{mh}I_m. \quad (1)$$

Similarly, the force of infection for mosquitoes (i.e. from human to mosquito) is given as

$$\lambda_m = \alpha\beta_{hm}(I_h + \delta I_{hp}), \quad (2)$$

where β_{hm} is the transmission rate from an infected human in the I_h or I_{hp} class to a susceptible mosquito in S_m class with δ defined as a modification parameter that accounts for the infectiousness of co-infected individuals in comparison to their counterparts, such that $0 < \delta < 1$. Force of infection for pneumonia denoted by λ_p is defined as the contact rate per human C , the transmission rate of pneumonia from a human in I_p or I_{hp} class to a susceptible human in S_h class i.e.

$$\lambda_p = \beta_{hm}C(I_p + gI_{hp}), \quad (3)$$

where g denote the modification parameter for the co-infected individual's relative infectiousness in comparison to their counterparts such that $0 < g < 1$.

From these assumptions a flow diagram is drawn which is summarized using a system of nonlinear ordinary differential equations as follows:

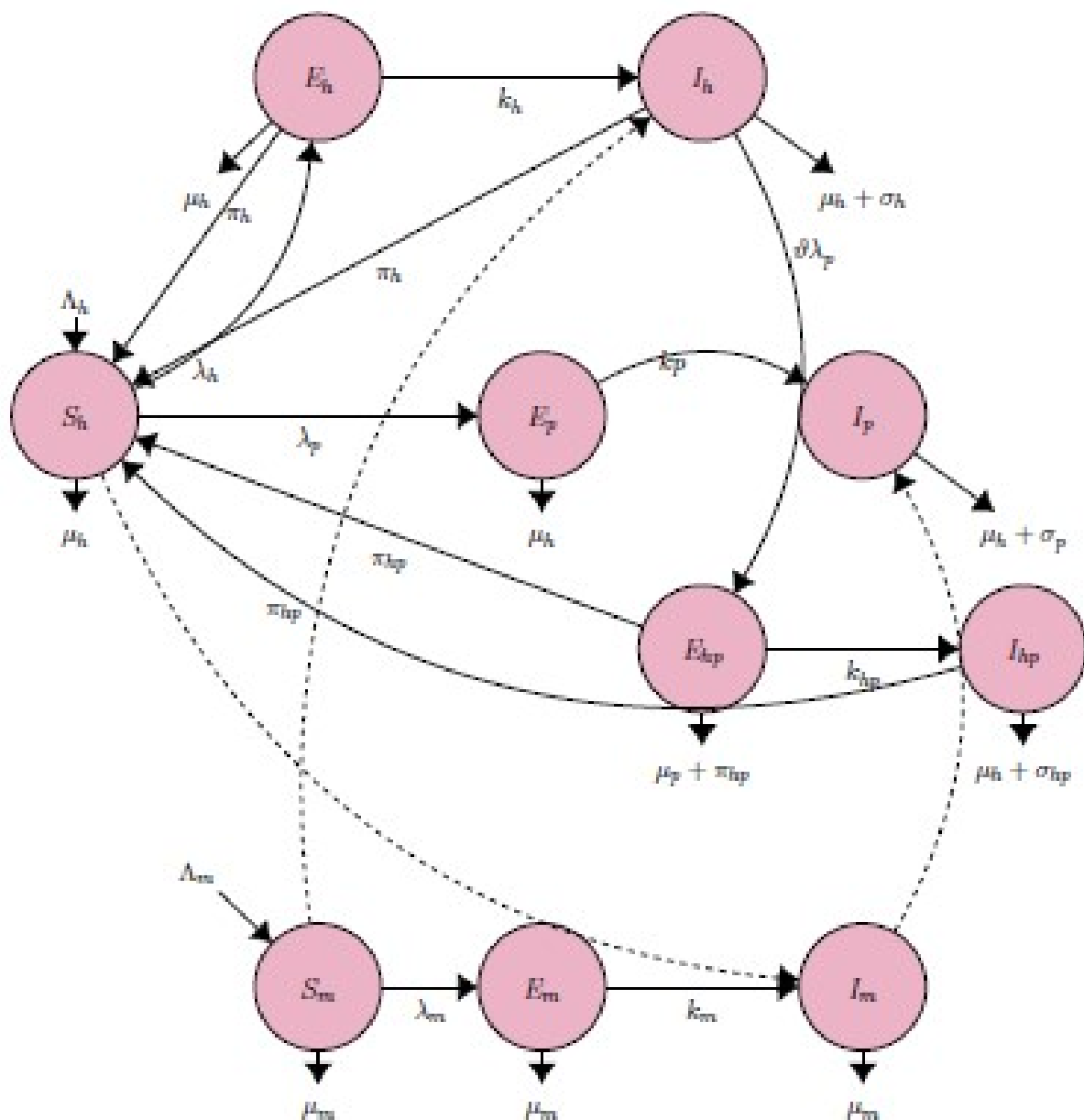


Figure 1: Model flow diagram

$$\begin{aligned}
 S'_h &= \Lambda_h - \lambda_h S_h - \lambda_p S_h - \mu_h S_h + \pi_h E_h + \pi_p E_p + \pi_{hp} E_{hp} + \pi_h I_h + \pi_p I_p + \pi_{hp} I_{hp}, \\
 E'_h &= \lambda_h S_h - A_0 E_h, \\
 I'_h &= k_h E_h - \vartheta \lambda_p I_h - A_1 I_h, \\
 E'_p &= \lambda_p S_h - A_2 E_p, \\
 I'_p &= k_p E_p - \varepsilon \lambda_h I_p - A_3 I_p, \\
 E'_{hp} &= \vartheta \lambda_p I_h + \varepsilon \lambda_h I_p - A_4 E_{hp}, \\
 I'_{hp} &= k_{hp} E_{hp} - A_5 I_{hp}, \\
 S'_m &= \Lambda_m - \lambda_m S_m - \mu_m S_m, \\
 E'_m &= \lambda_m S_m - A_6 E_m, \\
 I'_m(t) &= k_m E_m - \mu_m I_m, \\
 &\left(S_{h0}, E_{h0}, I_{h0}, E_{p0}, I_{p0}, E_{hp0}, I_{hp0}, S_{m0}, E_{m0}, I_{m0} \right)^T,
 \end{aligned} \tag{4}$$

where $A_0 = (k_h + \pi_h + \mu_h)$, $A_1 = (\sigma_h + \pi_h + \mu_h)$, $A_2 = (k_p + \pi_p + \mu_h)$, $A_3 = (\sigma_p + \pi_p + \mu_h)$, $A_4 = (k_{hp} + \pi_{hp} + \mu_h)$, $A_5 = (\sigma_{hp} + \pi_{hp} + \mu_h)$, $A_6 = (k_m + \mu_m)$.

For convenience, the model parameter descriptions is shown in Table 1 . Further, the following assumptions are made:

- (i) The model's parameters are all strictly positive.
- (ii) This model assumes that all individuals in the population are equally likely to become infected if they come into effective contact with an infectious individual or infectious mosquito, and that infection transmission occurs at a mass action incidence rate.
- (iii) It is assumed that an infected individual will recover with no permanent immunity and revert to the susceptible class.
- (iv) Since birth, immigration, emigration, and death occur in the population, it is assumed that human populations are not constant.
- (v) Individuals exposed to malaria as well as those exposed to pneumonia have a less than one in a million chance of survival until the infectious state.
- (vi) The chance of a mosquito exposed to malaria surviving until the infectious state is less than or equal to one.
- (vii) The exposed class of human and mosquito in the population is therefore included as a result of assumptions (v) and (vi) above.

Variable and Parameter Descriptions	
S_h	Susceptible class of human population to malaria
E_h	Human population exposed to malaria
I_h	Human population infectious with malaria
E_p	Human population exposed to pneumonia
I_p	Human population infectious with pneumonia
E_{hp}	Human population exposed to both malaria and pneumonia
I_{hp}	Human population that is infectious with malaria and Pneumonia
S_m	Susceptible class of vector population to malaria
E_m	Vector population exposed to malaria
I_m	Infectious class of the vector population
Λ_h	Constant per capita recruitment rate into susceptible human population
Λ_m	Constant per capita recruitment rate into susceptible vector population
μ_h	Natural death rate of human population
μ_m	Natural death rate of vector population
ϑ	Modification parameter according to the increased susceptibility to infection with Pneumonia
σ_h	Malaria induced mortality rate
σ_p	Pneumonia induced mortality rate
σ_{hp}	Malaria and pneumonia induced mortality rate
ε	The expected decrease in contact due to ill health as a result of pneumonia disease; $0 < \varepsilon < 1$
π_h	Rate of recovery from malaria to the susceptible class
π_p	Rate of recovery from pneumonia to the susceptible class
π_{hp}	Rate of recovery from malaria and pneumonia to the susceptible class
λ_h	Force of malaria infection for humans
λ_m	Force of infection for mosquitoes
λ_p	Transmission rate for pneumonia
k_h	The per capita rate of progression of humans from the exposed class E_h to the infectious class I_h .
k_p	The per capita rate of progression of humans from the exposed class E_p to the infectious class I_p .
k_{hp}	The per capita rate of progression of humans from the exposed class E_{hp} to the infectious class I_{hp} .
k_m	The per capita rate of progression of mosquitoes from the exposed class E_m to the infectious class I_m .

Table 1: Parameter description

3 Mathematical analysis of the model

3.1 Feasible Region and Well-posedness of the Model

In this section, the feasibility and well-posedness of the model are studied using the approach in [2].

Suppose system (4) can be rewritten compactly as

$$\begin{aligned} X'(t) &= \phi(X(t)), \\ X(0) &= X_0. \end{aligned} \quad (5)$$

It is assumed that $\phi \in \mathbf{C}'$ provides sufficient conditions to ensure that system (5) is well-posed; however, because the concern is with the model's stability near equilibrium points, additional assumptions on ϕ are required in order to invoke a variation of the centre manifold theorem, which was proven by Castillo-Chavez, 2004 [4] . Consequently, it is assumed that ϕ is twice continuously differentiable i.e. $\phi \in \mathbf{C}^2 \subset \mathbf{C}'$. In addition, the functions under investigation should have a bounded first derivative $\mathbf{C}'_{\mathbf{B}}(\mathfrak{R}_+^{10})$ for system (5) to be feasible in an epidemiological sense.

Theorem 3.1 (Feasible region of the model)

Suppose $(S_h, E_h, I_h, E_p, I_p, E_{hp}, I_{hp}, S_m, E_m, I_m)^T$ with initial data $X(0) \geq 0$, then, the solutions x of the malaria and pneumonia model (5) will remain non-negative for all time $t > 0$. Further, define the following region of attraction Γ_{hm} given by

$$\Gamma_{hm} = \left\{ x \in \mathbf{C}'_{\mathbf{B}} \cap \mathbf{C}'_{\mathbf{B}}(\mathfrak{R}_+^{10}) : \Gamma_h \times \Gamma_m \subset \mathfrak{R}_+^7 \times \mathfrak{R}_+^3 \right\},$$

where $\Gamma_h = \left\{ (S_h, E_h, I_h, E_p, I_p, E_{hp}, I_{hp}, S_m, E_m, I_m) : S_h + E_h + I_h + E_p + I_p + E_{hp} + I_{hp} \leq \frac{\Lambda_h}{\mu_h} \right\}$,

and $\Gamma_m = \left\{ (S_m, E_m, I_m) : S_m + E_m + I_m \leq \frac{\Lambda_m}{\mu_m} \right\}$

attracts all solution initiating in the interior positive invariant.

The proof of Theorem 1 follows from [15].

3.2 Equilibrium analysis of the model

The feasibility of all equilibrium points of the model (4) are considered in this section. The equilibrium points of system (4) are solutions of the dynamical system (4) obtained by equating the first derivative of all system variables to zero. In particular, for the malaria and pneumonia model in (4), the following feasible steady states are obtained:

- (i) **Malaria and pneumonia free equilibrium (MPFE)** denoted by ξ_0 , this represent the solutions of the dynamical variable of system (4) corresponding to the case where there is no presence of both malaria and pneumonia diseases in the population. The (MPFE), ξ_0 is feasible if the associated basic reproduction number for malaria and pneumonia diseases denoted by R_{hm} and R_p respectively are both less than unity or otherwise. Thus,

$$\xi_0 = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0 \right), \quad (6)$$

where $R_{hm} = \sqrt{R_h R_m}$, $R_h = \frac{\alpha \beta_{mh} k_h \Lambda_h}{\mu_h A_0 A_1}$, $R_m = \frac{\alpha \beta_{hm} k_m \Lambda_m}{\mu_m^2 A_6}$ and $R_p = \frac{\Lambda_h k_p \beta_p C}{\mu_h A_2 A_3}$ respectively. The co-infection reproduction number denoted by R_c is defined to be

$$R_c = \max\{R_{hm}, R_p\}. \quad (7)$$

A detailed insight and explanation on the basic reproduction number is considered in section 3.3.

- (ii) **Malaria free endemic equilibrium** (*MFEE*) written as ξ_1 . (*MFEE*), ξ_1 depicts the solution of (4) corresponding to the case where there is absence of malaria disease but there is presence of pneumonia disease in the population. This scenario is feasible if the basic reproduction for pneumonia infection R_p is greater than unity. Then,

$$\xi_1 = \left(S_{h1}, 0, 0, E_{p1}, I_{p1}, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0 \right). \quad (8)$$

- (iii) **Pneumonia free endemic equilibrium** (*PFEE*) denoted by ξ_2 , is a situation in the population representing the absence of pneumonia but persistence of malaria disease. This equilibrium is feasible if $R_{hm} > 1$. Thus,

$$\xi_2 = \left(S_{h2}, E_{h2}, I_{h2}, 0, 0, 0, 0, S_{m2}, E_{m2}, I_{m2} \right). \quad (9)$$

- (iv) **Malaria and Pneumonia endemic equilibrium** (*MPPE*) written as ξ^* is a scenario where both malaria and pneumonia diseases persist in the population. ξ^* is feasible if both $R_{hm} > 1$ and $R_p > 1$. We define

$$\xi^* = \left(S_h^*, E_h^*, I_h^*, E_p^*, I_p^*, E_{hp}^*, I_{hp}^*, S_m^*, E_m^*, I_m^* \right). \quad (10)$$

Remark 3.1 The detailed calculations on how the equilibrium points in (6), (8), (9), and (10) were derived are provided in Appendix A.

3.3 The basic reproduction number

The basic reproduction number R_{hm} and R_p are important dimensionless threshold in epidemiology. They are used to predict whether a disease goes to extinction or persist in the affected population. According to [5], the term basic reproduction number, R_{hm} is defined as the average number of secondary infections produced by a typical single infection in a completely susceptible population. Particularly, if $R_{hm} > 1$ (or $R_{hm} < 1$), it means that on average, an infected human(mosquito) will produce more than (or less than) one secondary infected mosquito (human) during the whole infectious period in an entirely susceptible population and thus the disease will persist (or die out) in the population, respectively. R_h and R_m represent the dynamics of the model when only malaria disease is transmitted in the population through cross-infection of humans and mosquitoes in the absence of no control, while R_p captures the dynamics of the model when only pneumonia disease is transmitted in the population through contact between a susceptible and infected individual when no control is available. R_c describe the co-infection dynamics of the model when malaria and pneumonia are transmitted among people in the population. The basic reproduction number R_0 can be obtained by applying the next generation matrix technique [5, 25] and implemented in [1, 14]. The derivation of the basic reproduction numbers R_{hm} and R_p are shown in the Appendix.

A deep insight is sought to understand the qualitative information embedded in the basic reproduction number. The basic reproduction numbers R_{hm} and R_p is decomposed further to obtain information that are epidemiologically meaningful. From epidemiological point of view, it is of interest to have R_{hm} and R_p below unity. R_{hm} is decomposed to give

$$R_{hm} = \alpha \sqrt{\frac{\Lambda_h}{\mu_h}} \sqrt{\frac{\beta_{mh}}{(\sigma_h + \pi_h + \mu_h)}} \sqrt{\frac{k_h}{(k_h + \pi_h + \mu_h)}} \sqrt{\frac{k_m}{(k_m + \mu_m)}} \sqrt{\frac{\beta_{hm}}{\mu_m}} \sqrt{\frac{\Lambda_m}{\mu_m}},$$

$$R_{hm} = \alpha \prod_{i=1}^6 \sqrt{Q_i} \quad , \quad i = 1, 2, \dots, 6. \quad (11)$$

Similarly,

$$R_p = C \frac{\Lambda_h}{\mu_h} \frac{k_p}{(k_p + \pi_h + \mu_h)} \frac{\beta_p}{(\sigma_p + \pi_h + \mu_h)},$$

$$R_p = C \prod_{j=1}^3 q_j, \quad i = 1, 2, 3. \quad (12)$$

The first factor of Eqn. 11, α , the biting rates of the *anopheles* mosquitoes. This is usually less than unity because the *anopheles* mosquito normally spread less than 100 *sporozoites* per bite [22]. The size of each of the other factors in Eqn. 11 is analyzed as follows:

- (i) The term $Q_1 = \frac{\Lambda_h}{\mu_h}$ refers to the ratio of the human recruitment rate to the natural death rate of humans. In normal settings, the human recruitment rate rank high than the natural death rate of humans on the sensitivity order of many epidemic models documented in the literature.
- (ii) The term $Q_2 = \frac{\beta_{mh}}{(\sigma_h + \pi_h + \mu_h)}$ is the ratio of the transmission probability of the mosquito-to-human to the reciprocal of the sum of the malaria-induced death rate, recovery rate and natural death rate of humans. This quantity is always greater than zero monotonically decreasing with respect to the malaria induced death in humans plus the recovery rate and the natural death in humans. This is true if the probability of infection is less than unity while the term in the denominator of Q_2 increases. However, because increasing σ_h and μ_h is not practical in a real scenario, this particular term Q_2 provide no control over R_{hm} .
- (iii) The term $Q_3 = \frac{k_h}{(k_h + \pi_h + \mu_h)}$ is the reciprocal of the host (human) progression rate to malaria infected class divided by itself and the sum of human recovery π_h and the death rates, μ_h . The upper bound of this term is one and is monotonically decreasing with respect to the sum of the human recovery π_h and mortality rates, μ_h . This fact is consistent if there are few humans exposed to the parasites to invade, then fewer malaria infections will arise. Since increasing μ_h is not realistic, Q_3 does not offer any control over R_{hm} .
- (iv) The term $Q_4 = \frac{k_m}{(k_m + \mu_m)}$ depicts the reciprocal of the vector latent period k_m divided by itself plus the vector death rate, μ_m . As in Q_3 , the quantity Q_4 is bounded above, always less than unity and monotonically decreases with respect to the mosquito death rate μ_m . Hence, the higher the mosquito mortality rate, the smaller Q_4 . Thus, Q_4 will offer realistic control over R_{hm} if the fatality rate of the mosquitoes can be increased.
- (v) The term $Q_5 = \frac{\beta_{hm}}{\mu_m}$ is the ratio of the transmission probabilities of human-to-mosquito to the mosquito natural death rate. Since the mosquito natural death rate μ_m is always smaller compared to the transmission probability of malaria from infected human to a susceptible mosquito, then Q_5 is always greater than unity, hence this term will increase R_{hm} .
- (vi) Q_6 is the ratio of the mosquito recruitment rate to mosquito mortality rate μ_m . From other studies, $\Lambda_m > \mu_m$, thus Q_6 will have a positive effect on R_{hm} .

R_p is decomposed and analyzed as follows:

The parameter C , the contact rate between susceptible humans and infected human with pneumonia. This is normally less than unity from literature. The other factors are considered as follows:

- (i) q_1 is the same as Q_1 , hence the result follows.
- (ii) q_2 is the term $q_2 = \frac{k_p}{(k_p + \pi_p + \mu_h)}$ representing the reciprocal of the human progression rate to pneumonia infected class divided by itself and the sum of human recovery from pneumonia π_p and the human natural death rate μ_h . Clearly, 1 is the upper bound of q_2 and is monotonically decreasing with respect to the sum of the human recovery rate from pneumonia and the fatality rate μ_h . Indeed, this is the case when there are few humans exposed to the bacteria invasion which may lead to few pneumonia infections cases. Further, since increasing μ_h is not practical in a real life situation, q_2 control over R_p is insignificant.
- (iii) $q_3 \frac{\beta_p}{(\sigma_p + \pi_p + \mu_h)}$ refer to the ratio of the pneumonia transmission probability to the reciprocal of the sum of the pneumonia induced deaths in humans, pneumonia recovery rate and natural death rate of humans. As in the case of Q_2 , q_3 is always greater than zero and monotonically decreasing with respect to the pneumonia-induced and natural death rates in humans. This is the case whenever the probability of pneumonia infection is less than unity while the reciprocal of q_3 has no significant control on R_p .

By combining Eqns. (3.7) and (3.8), it can be shown that the basic reproduction number for malaria infection, R_{hm} and pneumonia infection, R_p is related by

$$R_{hm} = Q^* \sqrt{R_p}, \quad (13)$$

where $Q^* = \sqrt{\frac{\alpha^2 Q_2 Q_3 Q_4 Q_5 Q_6}{C q_2 q_3}}$.

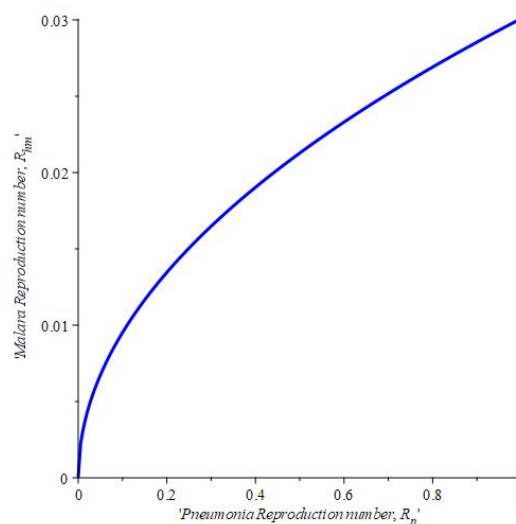


Figure 2: The graph of the Malaria Reproduction number R_{hm} against Pneumonia Reproduction number R_p

It can be seen from Figure 2 that increase in the basic reproduction number for pneumonia, R_p will increase the basic reproduction number for malaria. This implies that increase in pneumonia can induce malaria. In Figure 3, the contour plot showing the relationship between R_{hm} and R_p is shown.

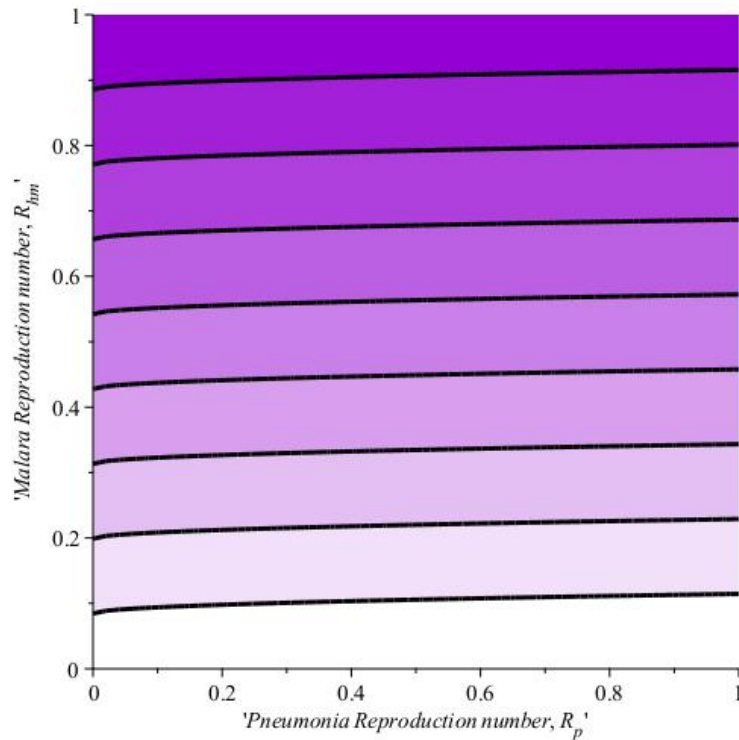


Figure 3: Contour plot of the malaria and pneumonia co-infection

Eqn. (3.7) is re-emphasized on as follows: First, if the biting rate of the mosquitoes $\alpha = 0$, it follows that $R_{hm} = 0$ as the quantity totally nullify R_{hm} which give rise to the following epidemiological scenario:

1. Humans are not bitten by mosquitoes and
2. Mosquitoes are not transmitting the disease

Further, suppose the transmission probability from human-to-mosquito β_{hm} is equal to zero, then R_{hm} is zero meaning that infected humans are not transmitting the malaria disease to susceptible mosquitoes. However, if certain quantities such as the death rate of the vector, μ_m and human death rate μ_h are zero then R_{hm} is undefined. Realistically, μ_h and μ_m cannot be zero because all mosquitoes and humans experience death at a stage in their life cycle. The study on control strategies will form the foundation of a separate research.

3.4 Stability analysis of the model

In the following section, the local asymptotic stability (LAS) of ξ_0 , ξ_1 and ξ_2 is computed through the following theorem:

Theorem 3.2 Consider the model system (2.4), then the following holds:

- (i) The malaria and pneumonia free equilibrium, ξ_0 is feasible and locally asymptotically stable if $R_{hm} < 1$ and $R_p < 1$ otherwise unstable.
- (ii) The malaria-free endemic equilibrium, ξ_1 is feasible and locally asymptotically stable if $R_p > 1$.
- (iii) The pneumonia-free endemic equilibrium, ξ_2 is feasible and locally asymptotically stable if $R_{hm} > 1$.

The proof to Theorem 3.2 is given in Appendix A.

Parameter	Values	Source
Λ_h	0.071	[15]
Λ_m	0.00011	[15]
μ_h	0.04	[15]
μ_m	0.0000457	[15]
ϑ	0.5	[15]
σ_p, σ_h	0.01	[15]
σ_{hp}	0.1	[15]
ε	0.1	Estimated
π_p, π_h	0.1	[15]
k_{hp}, k_p	0.5	[15]
α, π, τ	0.5	Estimated

Table 2: Parameter values

3.5 Global stability analysis of the Malaria and Pneumonia co-infection free equilibrium, ξ_0

Theorem 2 (i) suggest that the malaria and pneumonia co-infection free equilibrium ξ_0 is locally asymptotically stable if R_{hm} and R_p holds, however, this does not capture the co-infection scenario in details. It is therefore important to consider the global asymptotic stability (GAS). To do this, the global asymptotic stability analysis is motivated through the following theorem:

Theorem 3.3 *The malaria and pneumonia co-infection free equilibrium ξ_0 is globally asymptotically stable if ξ_0 is locally asymptotically stable and $R_{mp} \leq 1$, where R_{mp} is the co-infection threshold defined as*

$$R_{mp} = \frac{k_{hp}}{A_4 A_5} (A_3 g R_p + A_1 \delta).$$

The proof of Theorem 3.3 is provided in the Appendix B.

Remark 3.2 *It can be deduced that the global stability of ξ_0 depends on R_{mp} . Indeed, R_{mp} take into account the average number of secondary infections arising from co-infectious individual with pneumonia plus malaria infected humans.*

3.6 Endemic equilibrium point and bifurcation analysis

When a disease persist in the population in the course of time, then an endemic equilibrium occurs. The persistence of a disease depend on a threshold called the reproduction number R_0 whether it is less or greater than unity. Recent studies, [[2]] has revealed that for disease eradication, bringing $R_0 < 1$ may not be sufficient but may also depend on other parameters. The endemic equilibrium usually arise from other studies in the literature when $R_0 > 1$, however, there are situations where the endemic equilibrium co-exist with the disease free equilibrium for $R_0 < 1$, this phenomenon is called bifurcation. Bifurcation can be either forward (super-critical) or backward (sub-critical) which have serious implications in epidemiology. A forward bifurcation is shown when $R_0 > 1$ from below, and looses its stability so that a small positive super-threshold equilibrium that is asymptotically stable, then the disease free equilibrium become unstable. However, when $R_0 < 1$ and a small positive sub-threshold equilibrium appears, and the disease free equilibrium together with a larger positive equilibrium are locally asymptotically stable.

Considering the components of the endemic equilibrium ξ^* , it is desirable to study in details the stability of the endemic equilibrium around the malaria and pneumonia co-infection

free equilibrium using bifurcation analysis through the well known Centre Manifold Theorem discussed in [[4]]. In the following, the bifurcation analysis of the malaria and pneumonia co-infection model is studied. Particularly, the following theorem is proven to study the bifurcation behaviour of the model:

Theorem 3.4 (*Bifurcation analysis for the malaria and pneumonia co-infection model*) Let $R_{hp} = 1$ and the positive quantities a and b defined as follows

$$a = -\frac{2\nu_4(\mu_h^2 + (k_p + \pi_p + \sigma_p)\mu_h + k_p\sigma_p)r_5^2 A_2 A_3 (g A_2 A_3 k_{hp} \vartheta + A_4 A_5 k_p)}{\Lambda_h k_p^3 A_4 A_5}, \quad b = \frac{\nu_4 r_5 \beta_p \Lambda_h (g A_2 A_3 k_{hp} \vartheta + A_4 A_5 k_p)}{A_4 A_5 k_p \mu_h},$$

then the malaria and pneumonia model exhibits a forward bifurcation if $a < 0$ and $b > 0$.

The proof of Theorem 3.4 can be found in Appendix C.

4 Discussion and conclusion

In this work, the effect of the basic reproduction number ' R_0 ' on the transmission dynamics of malaria and pneumonia co-infection is studied. The malaria and pneumonia co-infection model (2.4) revealed that there exist a relationship between the reproduction number for malaria (R_{hm}) and reproduction number for pneumonia (R_p) given by (3.9). The study further revealed that pneumonia disease may induce malaria disease in the population. Considering the global stability of the Disease Free Equilibrium (DFE) of the model, the threshold (R_{mp}) that account for the co-infected persons is required to be less than unity to guarantee global stability of the model. A short summary highlighting the conclusions of this work is given below:

1. In section 3.1, the mathematical and epidemiological wellposedness of the model was shown given that the initial conditions are satisfied. Further, in Theorem 1, a feasible region Γ_{hm} that is mathematically and epidemiologically suitable was provided.
2. In Section 3.2, the model equilibrium points were obtained.
3. In Section 3.3, the basic reproduction number for malaria (R_{hm}) and (R_p) and the co-infection reproduction number (R_c) were discussed in details by decomposing them into components in order to carefully analyze the size contribution of each component involved. The analysis revealed that increasing the mosquito mortality rate (μ_m) will offer realistic control over R_{hm} . Also, the analyses revealed that the transmission probabilities of human-to-mosquito, β_{hm} and the recruitment rate of mosquitoes (Λ_m) will increase the size of the reproduction number.
4. In Section 3.4, stability analysis of the model were discussed. Precisely, in Theorem 2 conditions for the local stability of ξ_0 , ξ_1 and ξ_2 using the associated basic reproduction number was established.
5. In Section 3.5, the global stability of the malaria and pneumonia free equilibrium, ξ_0 using the method of Lyapunov function was established. Theorem 3 also provide that for the global stability to be established, the co-infection threshold R_{mp} must be less or equal to unity.
6. In Section 3.6, the Centre Manifold Theorem was used to study the stability of the endemic equilibrium around the malaria and pneumonia co-infection free equilibrium. The analysis revealed that the co-infection model exhibit a transcritical (forward) bifurcation.

As a guide for future research, it will be of interest to study the quantities that are related to control measures that can effectively increase mosquito fatality, reduce mosquito recruitment while also reducing the transmission rates of malaria and pneumonia.

Conclusively, the malaria and pneumonia co-infection model has provided us with a mathematical understanding the relationship between the basic reproduction numbers for malaria and pneumonia transmission dynamics.

Appendix A. Feasibility of equilibrium points

The feasibility of $MPFE$, ξ_0 is trivial. The next focus is on the feasibility of ξ_1 , ξ_2 and ξ^*

Feasibility of ξ_1

Consider the case when $\lambda_h = \lambda_m = 0$ in (4), so that the following equation are obtained at equilibrium:

$$\Lambda_h - \beta_p C I_p S_h - \mu_h S_h + \pi_p E_p + \pi_p I_p = 0, \quad (A.1)$$

$$\beta_p C I_p S_h - A_2 E_p = 0, \quad (A.2)$$

$$k_p E_p - A_3 I_p = 0, \quad (A.3)$$

$$\Lambda_m - \mu_m S_m = 0. \quad (A.4)$$

It is clear from (A.4) that $S_{m1} = \frac{\Lambda_m}{\mu_m}$. This is the case as there are no malaria infection in the population. Thus, the susceptible vectors are not infected and pose no malaria threat. From eqn. (A.3),

$$E_p = \frac{A_3}{k_p} I_p. \quad (A.5)$$

Using (A.5) in (A.1) yield

$$S_h = \frac{k_p \Lambda_h + \pi_p (k_p + A_3) I_p}{k_p (\beta_p C I_p + \mu_h)}. \quad (A.6)$$

Taking into account of (A.5) and (A.6) in (A.2),

$$f(I_p) = \Lambda_h k_p \beta_p C - \mu_h A_2 A_3 - \beta_p C (\sigma_p (k_p + \mu_h) + \mu_h A_2) I_p = 0. \quad (A.7)$$

Now,

$$\begin{aligned} f(0) &= \Lambda_h k_p \beta_p C - \mu_h A_2 A_3 \\ &= \mu_h A_2 A_3 (R_p - 1) > 0 \quad \text{if } R_p > 1. \end{aligned} \quad (A.8)$$

Further,

$$f(H_0) = -\mu_h A_2 A_3 < 0, \quad (A.9)$$

where $H_0 = \frac{\Lambda_h k_p}{(\sigma_p (k_p + \mu_h) + \mu_h A_2)}$. Also,

$$f'(I_p) = -\beta_p C (\sigma_p (k_p + \mu_h) + \mu_h A_2) < 0. \quad (A.10)$$

Thus, $f(I_p)$ has a unique positive root $I_p = I_{p1}$ in the interval $(0, H_0)$ if $R_p > 1$. In particular, eqn (A.7) yield

$$\begin{aligned} I_p = I_{p1} &= \frac{\Lambda_h k_p \beta_p C - \mu_h A_2 A_3}{\beta_p C (\sigma_p (k_p + \mu_h) + \mu_h A_2)} \\ &= \frac{\mu_h A_2 A_3 (R_p - 1)}{\beta_p C (\sigma_p (k_p + \mu_h) + \mu_h A_2)}. \end{aligned} \quad (A.11)$$

Using (A.11) in (A.5) yield

$$E_p = E_{p1} = \frac{\mu_h A_2 A_3^2 (R_p - 1)}{k_p \beta_p C (\sigma_p (k_p + \mu_h) + \mu_h A_2)}. \quad (A.12)$$

Further, using (3.17) in (3.12) yield

$$S_h = S_{h1} = \frac{k_p \Lambda_h + \pi_p (k_p + A_3) I_{p1}}{k_p (\beta_p C I_{p1} + \mu_h)}. \quad (A.13)$$

Thus, the malaria-free endemic equilibrium, ξ_1 is

$$\xi_1 = (S_{h1}, 0, 0, E_{p1}, I_{p1}, 0, 0, S_{m1}, 0, 0). \quad (A.14)$$

Feasibility of ξ_2

Suppose that $\lambda_p = 0$ which implies that $E_p = I_p = E_{hp} = I_{hp} = 0$ in (2.4), the following algebraic equations are obtained at equilibrium:

$$\Lambda_h - \alpha \beta_{mh} I_m S_h - \mu_h S_h + \pi_h E_h + \pi_h I_h = 0, \quad (A.15)$$

$$\alpha \beta_{mh} I_m S_h - A_0 E_h = 0, \quad (A.16)$$

$$k_h E_h - A_1 I_h = 0, \quad (A.17)$$

$$\Lambda_m - \alpha \beta_{hm} I_h S_m - \mu_m S_m = 0, \quad (A.18)$$

$$\alpha \beta_{hm} I_h S_m - A_6 E_m = 0, \quad (A.19)$$

$$k_m E_m - \mu_m I_m = 0. \quad (A.20)$$

From (A.20)

$$E_m = \frac{\mu_m I_m}{k_m}. \quad (A.21)$$

By adding (A.18) and (A.19) taking into account (A.21) yield

$$S_m = \frac{k_m \Lambda_m - \mu_m A_6 I_m}{\mu_m k_m}. \quad (A.22)$$

Using (A.21) and (A.22) in (A.19) while making I_h subject gives

$$\alpha \beta_{hm} I_h \left(\frac{k_m \Lambda_m - \mu_m A_6 I_m}{\mu_m k_m} \right) - \frac{A_6 \mu_m I_m}{k_m} = 0.$$

Thus

$$I_h = I_{h2} = \frac{\mu_m^2 A_6 I_m}{\alpha \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m)}. \quad (\text{A.23})$$

In view of (A.23), Equation (A.17) can be written as

$$E_h = E_{h2} = \frac{\mu_m^2 A_1 A_6 I_m}{\alpha k_h \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m)}. \quad (\text{A.24})$$

From (A.15), the expression for S_h is

$$S_h = \frac{\Lambda_h + \pi_h E_h + \pi_h I_h}{\alpha \beta_{mh} I_m + \mu_h}. \quad (\text{A.25})$$

The substitution of (A.23) and (A.24) in (A.25), implies that

$$S_h = S_{h2} = \frac{\Lambda_h \alpha k_h \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m) - (k_h + \mu_h) A_1 A_6 \mu_m^2 I_m + k_h \pi_h \mu_m^2 A_6 I_m}{\mu_h \alpha k_h \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m)}. \quad (\text{A.26})$$

By substituting (A.24) and (A.26) in (A.16) gives

$$\beta_{mh} \left[\frac{\Lambda_h \alpha k_h \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m) - (k_h + \mu_h) A_1 A_6 \mu_m^2 I_m + k_h \pi_h \mu_m^2 A_6 I_m}{\mu_h k_h \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m)} \right] - \frac{A_0 A_1 A_6 \mu_m^2}{\alpha k_h \beta_{hm} (k_m \Lambda_m - \mu_m A_6 I_m)} =$$

Further simplification of the above equation yield

$$\begin{aligned} f(I_m) &= \alpha^2 \beta_{mh} \beta_{hm} k_m k_h \Lambda_m \Lambda_h - \mu_h \mu_m^2 A_0 A_1 A_6 - \mu_m \alpha A_6 \beta_{mh} \left[\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_3 + k_h \sigma_h + k_h \mu_h) \right] I_m = 0 \\ &= \mu_h \mu_m^2 A_0 A_1 A_6 \left(\frac{\alpha^2 \beta_{mh} \beta_{hm} k_m k_h \Lambda_m \Lambda_h}{\mu_h \mu_m^2 A_0 A_1 A_6} - 1 \right) - \mu_m \alpha A_6 \beta_{mh} \left[\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_3 + k_h \sigma_h + k_h \mu_h) \right] I_m \\ &= \mu_h \mu_m^2 A_0 A_1 A_6 (R_{hm}^2 - 1) - \mu_m \alpha A_6 \beta_{mh} \left[\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_3 + k_h \sigma_h + k_h \mu_h) \right] I_m = 0. \end{aligned} \quad (\text{A.27})$$

Then

$$f(0) = \mu_h \mu_m^2 A_0 A_1 A_6 (R_{hm}^2 - 1) > 0 \quad \text{if} \quad R_{hm} > 1, \quad (\text{A.28})$$

$$f(H_1) = \mu_h \mu_m^2 A_0 A_1 A_6 < 0, \quad (\text{A.29})$$

$$\text{where } H_1 = \frac{\mu_h \mu_m^2 A_0 A_1 R_{hm}^2}{\mu_m \left[\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_1 + k_h (\sigma_h + \mu_h)) \right]}.$$

Also,

$$f'(I_m) = -\mu_m \alpha \beta_{mh} A_6 \left[\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_1 + k_h (\sigma_h + \mu_h)) \right] < 0. \quad (\text{A.30})$$

Thus, $f(I_m)$ has a unique positive root $I_m = I_{m2}$ in $(0, H_1)$ if $R_{hm} > 1$. In particular, from (A.27) we have that

$$I_m = \frac{\mu_h \mu_m A_0 A_1 (R_{hm}^2 - 1)}{\alpha \beta_{mh} [\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)]}. \quad (A.31)$$

Substituting (A.31) in (A.21), yields

$$E_m = E_{m2} = \frac{\mu_h \mu_m^2 A_0 A_1 (R_{hm}^2 - 1)}{k_m \alpha \beta_{mh} [\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)]}. \quad (A.32)$$

Further, from (A.22) using (A.31), S_m is non-negative if $R_{hm} > 1$. i.e.

$$\begin{aligned} S_m = S_{m2} &= \frac{1}{\mu_m k_m} \left[k_m \Lambda_m - \frac{\mu_h \mu_m^2 A_0 A_1 A_6 (R_{hm}^2 - 1)}{\alpha \beta_{mh} [\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)]} \right] \\ &= \frac{\mu_h \mu_m^2 A_0 A_1 A_6 + \mu_h k_m \Lambda_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)}{\mu_m k_m \alpha \beta_{mh} [\alpha \Lambda_h k_h \beta_{hm} + \mu_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)]}. \end{aligned} \quad (A.33)$$

By using (A.31) in (A.23), (A.24) and (A.26) respectively gives

$$I_h = I_{h2} = \frac{\mu_h \mu_m^3 A_0 A_1 A_6 (R_{hm}^2 - 1)}{\alpha \beta_{hm} [\mu_h \mu_m^2 A_0 A_1 A_6 + \alpha^2 \beta_{mh} \Lambda_m k_m \mu_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)]}; \quad (A.34)$$

$$E_h = E_{h2} = \frac{\mu_h \mu_m^3 A_0 A_1^2 A_6 (R_{hm}^2 - 1)}{k_h \alpha \beta_{hm} [\mu_h \mu_m^2 A_0 A_1 A_6 + \alpha^2 \beta_{mh} \Lambda_m k_m \mu_m (\mu_h A_1 + k_h \sigma_h + k_h \mu_h)]}; \quad (A.35)$$

and

$$S_h = S_{h2} = \frac{\Lambda_h + \pi_h E_{h2} + \pi_h I_{h2}}{\alpha \beta_{mh} I_{m2} + \mu_h}. \quad (A.36)$$

Thus, the pneumonia-free endemic equilibrium is feasible if $R_{hm} > 1$ i.e.

$$\xi_2 = (S_{h2}, E_{h2}, I_{h2}, 0, 0, 0, 0, S_{m2}, E_{m2}, I_{m2}). \quad (A.37)$$

Feasibility of ξ^*

It is of note for this case that $\lambda_h > 0$, $\lambda_p > 0$ and $\lambda_m > 0$, implies that each of the state variables is non-negative. Thereafter, solving the full system of (2.4) as follows by equating the left-hand side of (2.4) to zero:

$$\Lambda_h - \lambda_h S_h - \lambda_p S_h - \mu_h S_h + \pi_h E_h + \pi_p E_p + \pi_{hp} E_{hp} + \pi_h I_h + \pi_p I_p + \pi_{hp} I_{hp} = 0, \quad (A.38)$$

$$\lambda_h S_h - A_0 E_h = 0, \quad (A.39)$$

$$k_h E_h - \vartheta \lambda_p I_h - A_1 I_h = 0, \quad (A.40)$$

$$\lambda_p S_h - A_2 E_p = 0, \quad (A.41)$$

$$k_p E_p - \varepsilon \lambda_h I_p - A_3 I_p = 0, \quad (\text{A.42})$$

$$\vartheta \lambda_p I_h + \varepsilon \lambda_h I_p - A_4 E_{hp} = 0, \quad (\text{A.43})$$

$$k_{hp} E_{hp} - A_5 I_{hp} = 0, \quad (\text{A.44})$$

$$\Lambda_m - \lambda_m S_m - \mu_m S_m = 0, \quad (\text{A.45})$$

$$\lambda_m S_m - A_6 E_m = 0, \quad (\text{A.46})$$

$$k_m E_m - \mu_m I_m = 0. \quad (\text{A.47})$$

From (A.45),

$$S_m = S_m^* = \frac{\Lambda_m}{\lambda_m + \mu_m}. \quad (\text{A.48})$$

Using (A.48) in (A.46) to get

$$E_m = E_m^* = \frac{\Lambda_m \lambda_m}{A_6 (\lambda_m + \mu_m)}, \quad (\text{A.49})$$

In view of (A.47), I_m is obtained as

$$I_m = I_m^* = \frac{k_m \Lambda_m \lambda_m}{\mu_m A_6 (\lambda_m + \mu_m)}. \quad (\text{A.50})$$

It can be seen from (A.39) and (A.41) that

$$E_h = \frac{\lambda_h}{A_0} S_h, \quad (\text{A.51})$$

and

$$E_p = \frac{\lambda_p}{A_2} S_h. \quad (\text{A.52})$$

The substitution of (A.51) and (A.52) in (A.40) and (A.42) respectively yields

$$I_h = \frac{k_h \lambda_h}{A_0 (\vartheta \lambda_p + A_1)} S_h, \quad (\text{A.53})$$

and

$$I_p = \frac{k_p \lambda_p}{A_2 (\varepsilon \lambda_h + A_3)} S_h. \quad (\text{A.54})$$

From (A.43) using (A.53) and (A.54), gives

$$E_{hp} = \frac{\left[k_h \vartheta A_2 (\varepsilon \lambda_h + A_3) + k_p \varepsilon A_0 (\vartheta \lambda_p + A_1) \right]}{A_0 A_2 A_4 (\varepsilon \lambda_h + A_3) (\vartheta \lambda_p + A_1)} \lambda_p \lambda_h S_h. \quad (\text{A.55})$$

Using (A.55) in (A.44) to get

$$I_{hp} = \frac{k_{hp} \left[k_h \vartheta A_2 (\varepsilon \lambda_h + A_3) + k_p \varepsilon A_0 (\vartheta \lambda_p + A_1) \right]}{A_0 A_2 A_4 A_5 (\varepsilon \lambda_h + A_3) (\vartheta \lambda_p + A_1)} \lambda_p \lambda_h S_h. \quad (A.56)$$

By substituting (A.51), (A.52), (A.53), (A.54), (A.55) and (A.56) in (A.38) while making S_h subject yield

$$S_h = \frac{A_0 A_2 A_4 (\varepsilon \lambda_h + A_3) (\vartheta \lambda_p + A_1) \Lambda_h}{D_0 \lambda_p \lambda_h^2 + D_1 \lambda_p \lambda_h + D_2 \lambda_h^2 + D_3 \lambda_h + D_4 \lambda_h \lambda_p^2 + D_5 \lambda_p^2 + D_6 \lambda_p + \mu_h A_0 A_2 A_4}, \quad (A.57)$$

where

$$\begin{aligned} D_0 &= A_2 \vartheta \varepsilon (k_{hp} (1 - \pi_{hp}) + \mu_h) + \mu_h A_2 A_4 \vartheta \varepsilon, \\ D_1 &= (k_h A_2 A_3 \vartheta \varepsilon + \mu_h) + k_p A_0 A_1 \varepsilon \varepsilon (k_{hp} (1 - \pi_{hp}) + \mu_h) + \mu_h A_2 A_4 A_3 \vartheta + \mu_h A_0 A_1 A_4 \varepsilon + \\ &\mu_h A_0 A_2 A_4 \vartheta \varepsilon, D_2 = A_2 A_4 k_h \varepsilon (\sigma_h + \mu_h) + \mu_h A_1 A_2 A_4 \varepsilon, D_3 = k_h A_2 A_3 A_4 \varepsilon (\sigma_h + \mu_h) + \mu_h A_2 A_4 (A_1 A_3 + \\ &\varepsilon A_0), \\ D_4 &= k_p \vartheta \varepsilon A_0 ((1 - \pi_{hp}) k_{hp} + \mu_h) + \mu_h A_0 A_4 \vartheta \varepsilon, D_5 = k_p A_0 A_4 \vartheta (\sigma_p + \mu_h) + \mu_h A_0 A_3 A_4 \vartheta A_0, \\ D_6 &= k_p A_0 A_1 A_4 \vartheta (\sigma_p + \mu_h) + \mu_h A_0 A_4 (A_1 A_3 + \vartheta A_2 A_3). \end{aligned}$$

Substituting (A.57) in (A.51), (A.52), (A.53), (A.54), (A.55) and (A.56) yield the corresponding values for E_h , E_p , I_h , I_p , E_{hp} and I_{hp} as a functions of λ_h and λ_p . Thus, the components of malaria and pneumonia endemic equilibrium, ξ^* has a unique positive endemic equilibrium if $\lambda_h > 0$, $\lambda_m > 0$ and $\lambda_p > 0$. Hence

$$\xi^* = \left(S_h^*, E_h^*, I_h^*, E_p^*, I_p^*, E_{hp}^*, I_{hp}^*, S_m^*, E_m^*, I_m^* \right). \quad (A.58)$$

Appendix B. Prove of the stability analysis of the model

Proof.

(i) The Jacobian matrix of system (2.4) evaluated at ξ_0 is

$$J(\xi_0) = \begin{bmatrix} -\mu_h & \pi & \pi & \tau & -\frac{\beta_p C \Lambda_h}{\mu_h} + \tau & \phi & -\frac{\beta_p C g \Lambda_h}{\mu_h} + \phi & 0 & 0 & -\frac{\alpha \beta_{mh} \Lambda_h}{\mu_h} \\ 0 & -a_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha \beta_{mh} \Lambda_h}{\mu_h} \\ 0 & k_h & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -a_2 & \frac{\beta_p C \Lambda_h}{\mu_h} & 0 & \frac{\beta_p C g \Lambda_h}{\mu_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & k_p & -a_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -a_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & k_{hp} & -a_5 & 0 & 0 & 0 \\ 0 & 0 & -\frac{\alpha \beta_{hm} \Lambda_m}{\mu_m} & 0 & 0 & 0 & -\frac{\alpha \beta_{hm} \delta \Lambda_m}{\mu_m} & -\mu_m & 0 & 0 \\ 0 & 0 & \frac{\alpha \beta_{hm} \Lambda_m}{\mu_m} & 0 & 0 & 0 & \frac{\alpha \beta_{hm} \delta \Lambda_m}{\mu_m} & 0 & -a_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & k_m & -\mu_m \end{bmatrix}. \quad (A.59)$$

By inspection, it is easy to see that two eigenvalues of (A.59) are $\lambda_1 = -\mu_h$ and $\lambda_2 = -\mu_m$ while the remaining eigenvalues are obtained from the following 8×8

$$A = \begin{bmatrix} -A_0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha \beta_{mh} \Lambda_h}{\mu_h} \\ k_h & -A_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -A_2 & \frac{\beta_p C \Lambda_h}{\mu_h} & 0 & \frac{\beta_p C g \Lambda_h}{\mu_h} & 0 & 0 \\ 0 & 0 & k_p & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & k_{hp} & -A_5 & 0 & 0 \\ 0 & \frac{\alpha \beta_{hm} \Lambda_m}{\mu_m} & 0 & 0 & 0 & \frac{\alpha \beta_{hm} \delta \Lambda_m}{\mu_m} & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_m & -\mu_m \end{bmatrix}. \quad (A.60)$$

By Routh-Hurwitz condition, all the eigenvalues of (A.60) are negative if

$$(i) \text{ Trace}(A) < 0 \quad (ii) \text{ Det}(A) > 0.$$

Clearly,

$$\bullet \text{ Trace}(A) = -A_0 - A_1 - A_2 - A_3 - A_4 - A_5 - A_6 - \mu_m < 0.$$

•

$$\begin{aligned} \text{Det}(A) &= \frac{A_4 A_5 (k_h \alpha^2 \beta_{mh} \Lambda_h k_m \beta_{hm} \Lambda_m - A_0 \mu_m^2 A_6 A_1 \mu_h) (\beta_p C \Lambda_h k_p - A_2 A_3 \mu_h)}{\mu_h^2 \mu_m} \\ &= \frac{A_0 A_1 A_2 A_3 A_4 A_5 A_6 \mu_m^2 \mu_h^2 \left(\frac{k_m k_h \alpha^2 \beta_{mh} \beta_{hm} \Lambda_h \Lambda_m}{A_0 A_1 A_6 \mu_m^2 \mu_h} - 1 \right) \left(\frac{\beta_p C \Lambda_h k_p}{A_2 A_3 \mu_h} - 1 \right)}{\mu_h^2 \mu_m} \\ &= A_0 A_1 A_2 A_3 A_4 A_5 A_6 \mu_m (R_{hm}^2 - 1) (R_p - 1) > 0 \quad \text{if } R_{hm} < 1 \quad R_p < 1. \end{aligned}$$

So that the eigenvalues of *matrix* A are all negative, and hence, the eigenvalues of (A.59) are all negative if $R_{hm} < 1$ and $R_p < 1$. Thus, malaria and pneumonia free equilibrium, ξ_0 is locally asymptotically stable otherwise unstable.

- (ii) It is easy to obtain the upper triangular matrix of the Jacobian matrix of the model system (2.4) evaluated at ξ_1 as

$$J(\xi_1) = \begin{bmatrix} -G_1 - \mu_h & \pi & \pi & \tau & -G_2 + \tau & \phi & -kG_2 + \phi & 0 & 0 & -G_3 \\ 0 & -A_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_3 \\ 0 & 0 & -G_1 - A_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_2 & G_2 & 0 & kG_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_3 & 0 & 0 & 0 & 0 & -G_5 \\ 0 & 0 & 0 & 0 & 0 & -A_4 & 0 & 0 & 0 & G_5 \\ 0 & 0 & 0 & 0 & 0 & 0 & -A_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_4 - \mu_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m \end{bmatrix} \quad (A.61)$$

where $A_7 = \frac{\mu_h A_2 A_3}{\beta_p C(\sigma_p(k_p + \mu_h) + \mu_h A_2)}$, $G_1 = \beta_p C A_7 (R_p - 1)$, $G_2 = \frac{\beta_p C(k_p \Lambda_h + \pi_{hp}(k_p + A_3) A_7 (R_p - 1))}{k_p(\beta_p C A_7 (R_p - 1) + \mu_h)}$,
 $G_3 = \frac{\alpha \beta_{mh}(k_p \Lambda_h + \pi_{hp}(k_p + A_3) A_7 (R_p - 1))}{k_p(\beta_p C A_7 (R_p - 1) + \mu_h)}$,

$G_4 = \alpha \beta_{hm} A_7 (R_p - 1)$, $G_5 = \alpha \varepsilon \beta_{mh} A_7 (R_p - 1)$. It is easy to see that

$$Trace(\xi_1) = -2G_1 - \mu_h - A_0 - A_1 - A_2 - A_3 - A_4 - A_5 - G_4 - 2\mu_m - A_6 < 0 \quad R_p > 1$$

$Determinant(\xi_1) = (-G_1 - \mu_h) A_0 (-G_1 - A_1) A_2 A_3 A_4 A_5 (G_4 + \mu_m) A_6 \mu_m > 0 \quad R_p > 1$. Hence all eigenvalues of (A.61) are negative which implies that the malaria-free endemic equilibrium, ξ_1 of system (2.4) is feasible and locally asymptotically stable if $R_p > 1$ otherwise unstable.

- (iii) The upper triangular matrix of the Jacobian matrix of the model system (2.4) evaluated at ξ_1 gives

$$J(\xi_2) = \begin{bmatrix} -G_6 - \mu_h & \pi & \pi & \tau & -G_7 + \tau & \phi & -kG_7 + \phi & 0 & 0 & -G_8 \\ 0 & -A_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_8 \\ 0 & 0 & -A_1 & 0 & -G_9 & 0 & -kG_9 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_2 & G_7 & 0 & kG_7 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -G_{10} - A_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -A_4 & kG_9 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -A_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m \end{bmatrix} \quad (A.62)$$

where $A_8 = \frac{\mu_h \mu_m A_0 A_1}{\alpha \beta_{mh}(\alpha \Lambda_h k_h \beta_{hm} + \mu_m(\mu_h A_1 + k_h \mu_h + k_h \sigma_h))}$, $A_9 = \frac{\mu_h \mu_m^2 A_0 A_1 A_6 + \mu_h k_m \Lambda_m(\mu_h A_1 + k_h \mu_h + k_h \sigma_h)}{\mu_m k_m \alpha \beta_{mh}(\alpha \Lambda_h k_h \beta_{hm} + \mu_m(\mu_h A_1 + k_h \mu_h + k_h \sigma_h))}$,

$$A_{10} = \frac{\mu_h \mu_m^3 A_0 A_1 A_6}{\alpha \beta_{hm} (\mu_h \mu_m^2 A_0 A_1 A_6 + \alpha^2 \beta_{mh} \Lambda_m k_m \mu_m (\mu_h A_1 + k_h \mu_h + k_h \sigma_h))}, \quad G_6 = \alpha \beta_{mh} A_8 (R_{hm}^2 - 1),$$

$$G_7 = \frac{\beta_p C}{\alpha \beta_{mh} A_8 (R_{hm}^2 - 1) + \mu_h} \left(\Lambda_h + \frac{\pi_h A_1 A_{10} (R_{hm}^2 - 1)}{k_h} + \pi_h A_{10} (R_{hm}^2 - 1) \right),$$

$$G_8 = \frac{\alpha \beta_{mh}}{\alpha \beta_{mh} A_8 (R_{hm}^2 - 1) + \mu_h} \left(\Lambda_h + \frac{\pi_h A_1 A_{10} (R_{hm}^2 - 1)}{k_h} + \pi_h A_{10} (R_{hm}^2 - 1) \right),$$

$G_9 = \vartheta \beta_p C A_{10} (R_{hm}^2 - 1)$, $G_{10} = \alpha \varepsilon \beta_{mh} A_8 (R_{hm}^2 - 1)$. Straight forward calculation yield

$\lambda_1 = -(G_6 + \mu_h)$, $\lambda_2 = -A_0$, $\lambda_3 = -A_1$, $\lambda_4 = -A_2$, $\lambda_5 = -(G_{10} + A_3)$, $\lambda_6 = -A_4$, $\lambda_7 = -A_5$, $\lambda_8 = -\mu_m$, $\lambda_9 = -A_6$ and $\lambda_{10} = -\mu_m$. Observe that λ_1 and λ_5 are both negative if $R_{hm} > 1$, thus, all eigenvalues of (A.62) are negative meaning that the pneumonia-free endemic equilibrium, ξ_2 of system (2.4) is feasible and locally asymptotically stable if $R_{hm} > 1$ and unstable if $R_{hm} < 1$.

Appendix B. Prove of the global stability of Malaria and Pneumonia co-infection free equilibrium point

Proof. Consider the following Lyapunov function

$$\begin{aligned} L(E_h, I_h, E_p, I_p, E_{hp}, I_{hp}, E_m, I_m) = & \frac{k_h}{\mu_m^2 A_0} E_h + \frac{1}{\mu_m^2} I_h + \frac{k_p}{\mu_m^2 A_2} E_p + \frac{1}{\mu_m^2} I_p + \frac{1}{\mu_m^2} E_{hp} \\ & + \frac{A_4}{\mu_m^2 k_{hp}} I_{hp} + \frac{A_1}{\mu_m \alpha \beta_{hm} \Lambda_m} E_m + \frac{A_1 A_6}{\mu_m \alpha \beta_{hm} \Lambda_m k_m} I_m. \end{aligned} \quad (B.1)$$

The differentiation of (B.1) with respect to time t along the solutions of system (2.4) yield

$$L' = \left(\frac{k_h \alpha \beta_{mh} S_h}{\mu_m^2 A_0} - \frac{A_1 A_6}{\alpha \beta_{hm} \Lambda_m k_m} \right) I_m + \left(\frac{A_1 S_m}{\mu_m \Lambda_m} - \frac{A_1}{\mu_m^2} \right) I_h + \left(\frac{k_p \beta_p C S_h}{\mu_m^2 A_2} - \frac{A_3}{\mu_m^2} \right) I_p + \left(\frac{k_p \beta_p C g S_h}{\mu_m^2} + \frac{A_1 \delta S_m}{\mu_m \Lambda_m} - \frac{A_4 A_5}{\mu_m^2 k_{hp}} \right) E_{hp}$$

At the DFE, $S_h = \frac{\Lambda_h}{\mu_h}$ and $S_m = \frac{\Lambda_m}{\mu_m}$

$$L' = \frac{A_1 A_6}{\alpha \beta_{hm} \Lambda_m k_m} \left(\frac{\alpha^2 \beta_{hm} \beta_{mh} k_h k_m \Lambda_h \Lambda_m}{\mu_h \mu_m^2 A_0 A_1 A_6} - 1 \right) I_m + \frac{A_3}{\mu_m^2} \left(\frac{k_p \beta_p C \Lambda_h}{\mu_h A_2 A_3} - 1 \right) I_p + \frac{A_4 A_5}{k_{hp} \mu_m^2} \left(\frac{k_{hp} k_p \beta_p C g \Lambda_h}{\mu_m^2 A_2 A_4 A_5} + \frac{k_{hp} \delta A_1}{A_4 A_5} - 1 \right) E_{hp}$$

$$L' = \frac{A_1 A_6}{\alpha \beta_{hm} \Lambda_m k_m} (R_{hm}^2 - 1) I_m + \frac{A_3}{\mu_m^2} (R_p - 1) I_p + \frac{A_4 A_5}{\mu_m^2 k_{hp}} (R_{mp} - 1) I_{hp}, \quad (B.2)$$

where

$$R_{mp} = \frac{k_{hp}}{A_4 A_5} (A_3 g R_p + \delta A_1). \quad (B.3)$$

Thus, the malaria and pneumonia free equilibrium, ξ_0 is globally asymptotically stable if $R_{hm} < 1$, $R_p < 1$ and $R_{mp} < 1$

Remark 4.1 The condition for the global stability of the malaria and pneumonia free equilibrium, ξ_0 require that $R_{mp} < 1$ in addition to $R_{hm} < 1$ and $R_p < 1$. $R_{mp} < 1$ account for those individuals who are infectious with both diseases.

Appendix C. Prove of Theorem 3.4

Proof. Consider the dynamical system (2.4) with contact rate C as a bifurcation parameter, then the stability of the endemic equilibrium can be studied by considering the existence and stability of non-trivial equilibrium states in a neighbourhood of the bifurcation point. The idea here is that the Disease Free Equilibrium (DFE), ξ_0 is considered to be a manifold (ξ_0, C) such that the local stability of ξ_0 changes at the point (ξ_0, C^*) . Suppose from (3.3) at the critical point $R_c = R_p = 1$, then

$$\frac{\Lambda_h k_p \beta_p C}{\mu_h A_2 A_3} = 1.$$

Therefore, the bifurcation parameter at the critical value $R_p = 1$ is

$$C = \frac{\mu_h A_2 A_3}{\Lambda_h k_p \beta_p} = C^*. \quad (C.1)$$

This means that x_{i0} is locally asymptotically stable when $C < C^*$ and unstable if $C > C^*$. Further, for easy representation, we rewrite system (2.4) as

$$\dot{x} = F(x, C),$$

where $x = (S_h, E_h, I_h, E_P, I_P, E_{hP}, I_{hP}, S_m, E_m, I_m)^T$ such that X_i is the i th component of X and $F = (F_1, F_2, F_3, F_4, F_5, F_6, F_7, F_8, F_9, F_{10})^T$, i.e.,

$$\begin{aligned} F_1 &= \Lambda_h - \alpha\beta_{mh}x_{10}x_1 - \beta_p C(gx_7 + x_5)x_1 - \mu_h x_1 + \pi_h x_3 + \pi_h x_2 + \pi_p x_5 + \pi_p x_4 + \pi_{hp} x_7 + \pi_{hp} x_6, \\ F_2 &= \alpha\beta_{mh}x_{10}x_1 - A_0 x_2, \\ F_3 &= k_h x_2 - \vartheta\beta_p C(gx_7 + x_5)x_3 - A_1 x_3, \\ F_4 &= \beta_p C(gx_7 + x_5)x_1 - A_2 x_4, \\ F_5 &= -\varepsilon\alpha\beta_{mh}x_{10}x_5 + k_p x_4 - A_3 x_5, \\ F_6 &= \vartheta\beta_p C(gx_7 + x_5)x_1 + \varepsilon\alpha\beta_{mh}x_{10}x_1 - A_4 x_6, \\ F_7 &= k_{hp} x_6 - A_5 x_7, \\ F_8 &= \Lambda_m - \alpha\beta_{hm}(\delta x_7 + x_3)x_8 - \mu_m x_8, \\ F_9 &= \alpha\beta_{hm}(\delta x_7 + x_3)x_8 - a_6 x_9, \\ F_{10} &= k_m x_9 - \mu_m x_{10}. \end{aligned} \quad (C.2)$$

The Jacobian of (C.2) evaluated at (ξ_0, C^*) denoted by $J(\xi_0, C^*)$ is obtained as

$$J(\xi_0, C^*) = \begin{bmatrix} -\mu_h & \pi_h & \pi_h & \pi_p & -\frac{a_2 a_3}{k_p} + \pi_p & \pi_{hp} & -\frac{a_2 a_3 g}{k_p} + \pi_{hp} & 0 & 0 & -\frac{\alpha\beta_{mh}\Lambda_h}{\mu_h} \\ 0 & -a_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha\beta_{mh}\Lambda_h}{\mu_h} \\ 0 & k_h & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -a_2 & \frac{a_2 a_3}{k_p} & 0 & \frac{a_2 a_3 g}{k_p} & 0 & 0 & 0 \\ 0 & 0 & 0 & k_p & -a_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -a_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & k_{hp} & -a_5 & 0 & 0 & 0 \\ 0 & 0 & -\frac{\alpha\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & -\frac{\alpha\beta_{hm}\delta\Lambda_m}{\mu_m} & -\mu_m & 0 & 0 \\ 0 & 0 & \frac{\alpha\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & \frac{\alpha\beta_{hm}\delta\Lambda_m}{\mu_m} & 0 & -a_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & k_m & -\mu_m \end{bmatrix}. \quad (C.3)$$

According to the Centre Manifold Theorems as proved by Castillo-Chavez and Song [4], $J(\xi_0, C^*)$ have a zero simple eigenvalue while others have negative real parts. In order to determine the coefficients a and b , we first obtain the values of the left and right eigen vectors of

$J(\xi_0, C^*)$ denoted respectively as

$$\nu = (\nu_1, \nu_2, \nu_3, \nu_4, \nu_5, \nu_6, \nu_7, \nu_8, \nu_9, \nu_{10}),$$

and

$$r = (r_1, r_2, r_3, r_4, r_5, r_6, r_7, r_8, r_9, r_{10})^T.$$

Observe that the $J(\xi_0, C^*)$ is not symmetric, thus the left and right eigen vectors are not equivalent. The solutions of the following equations

$$\nu J(\xi_0, C^*) = 0, \quad (C.4)$$

and

$$J(\xi_0, C^*)r = 0, \quad (C.5)$$

yields the following solutions

$$\nu = \left(0, 0, 0, \nu_4, \frac{A_2\nu_4}{k_p}, \frac{gA_2A_3k_{hp}\nu_4}{A_4A_5k_p}, \frac{gA_2A_3\nu_4}{A_5k_p}, 0, 0, 0\right), \quad (C.6)$$

and

$$r = \left(-\frac{(\mu_h^2 + (k_p + \pi_p + \sigma_p)\mu_h + k_p\sigma_p)r_5}{\mu_h k_p}, 0, 0, \frac{a_3r_5}{k_p}, r_5, 0, 0, 0, 0, 0\right), \quad (C.7)$$

where ν_4 and r_5 are free positive left and right eigenvectors respectively. The following nonzero partial derivatives of F at x_{i0} for system (C.2) are obtained as

$$\frac{\partial^2 F_1}{\partial x_1 \partial x_5} = -\beta_p C, \frac{\partial^2 F_1}{\partial x_1 \partial x_7} = -\beta_p C g, \frac{\partial^2 F_1}{\partial x_1 \partial x_{10}} = -\alpha \beta_{mh}, \frac{\partial^2 F_2}{\partial x_1 \partial x_{10}} = \alpha \beta_{mh} C, \frac{\partial^2 F_3}{\partial x_3 \partial x_5} = -\vartheta \beta_p C, \frac{\partial^2 F_3}{\partial x_3 \partial x_7} = -\vartheta \beta_p C g,$$

$$\frac{\partial^2 F_4}{\partial x_1 \partial x_5} = \beta_p C, \frac{\partial^2 F_4}{\partial x_1 \partial x_7} = \beta_p C g, \frac{\partial^2 F_5}{\partial x_5 \partial x_{10}} = -\varepsilon \alpha \beta_{mh}, \frac{\partial^2 F_6}{\partial x_1 \partial x_{10}} = \varepsilon \alpha \beta_{mh}, \frac{\partial^2 F_6}{\partial x_1 \partial x_5} = \vartheta \beta_p C, \frac{\partial^2 F_6}{\partial x_1 \partial x_7} = \vartheta \beta_p C g$$

$$\frac{\partial^2 F_8}{\partial x_3 \partial x_8} = -\alpha \beta_{hm}, \frac{\partial^2 F_8}{\partial x_7 \partial x_8} = -\alpha \beta_{hm} \delta, \frac{\partial^2 F_9}{\partial x_3 \partial x_8} = \alpha \beta_{hm}, \frac{\partial^2 F_9}{\partial x_7 \partial x_8} = \alpha \beta_{hm} \delta, \frac{\partial^2 F_1}{\partial x_5 \partial C} = -\frac{\beta_p \Lambda_h}{\mu_h}, \frac{\partial^2 F_1}{\partial x_7 \partial C} = -\frac{\beta_p g \Lambda_h}{\mu_h}$$

$$\frac{\partial^2 F_4}{\partial x_5 \partial C} = \frac{\beta_p \Lambda_h}{\mu_h}, \frac{\partial^2 F_4}{\partial x_7 \partial C} = \frac{\beta_p g \Lambda_h}{\mu_h}, \frac{\partial^2 F_6}{\partial x_5 \partial C} = \frac{\vartheta \beta_p \Lambda_h}{\mu_h}, \frac{\partial^2 F_6}{\partial x_7 \partial C} = -\frac{\vartheta \beta_p g \Lambda_h}{\mu_h}.$$

Thus, the computation of the coefficient of b yield

$$\begin{aligned} b &= \sum_{i,k=1}^{10} \nu_k r_i \frac{\partial^2 F_k}{\partial x_i \partial C}(\xi_0, C^*) \\ &= \nu_1 r_5 \frac{\partial^2 F_1}{\partial x_5 \partial C}(\xi_0, C^*) + \nu_1 r_7 \frac{\partial^2 F_1}{\partial x_7 \partial C}(\xi_0, C^*) + \nu_4 r_5 \frac{\partial^2 F_4}{\partial x_5 \partial C}(\xi_0, C^*) + \nu_4 r_7 \frac{\partial^2 F_4}{\partial x_7 \partial C}(\xi_0, C^*) \\ &\quad + \nu_6 r_5 \frac{\partial^2 F_6}{\partial x_5 \partial C}(\xi_0, C^*) + \nu_6 r_7 \frac{\partial^2 F_6}{\partial x_7 \partial C}(\xi_0, C^*) \\ &= \frac{\Lambda_h \beta_p (\vartheta \nu_6 - \nu_1 + \nu_4) (g r_7 + r_5)}{\mu_h} \\ &= \frac{\nu_4 r_5 \beta_p \Lambda_h (g \vartheta A_2 A_3 k_{hp} + A_4 A_5 k_p)}{A_4 A_5 k_p \mu_h} > 0. \end{aligned} \quad (C.8)$$

Note that b is always positive since ν_4 and r_5 are positive. It then follows that the bifurcation behaviour of the dynamical system (C.2) is totally determined by the sign of the coefficient a .

$$\begin{aligned}
 a &= \sum_{i,k=1}^{10} \nu_k r_i r_j \frac{\partial^2 F_k}{\partial x_i \partial x_j} (\xi_0, C^*) \\
 &= 2 \left[\nu_1 r_1 r_5 \frac{\partial^2 F_1}{\partial x_1 \partial x_5} (\xi_0, C^*) + \nu_1 r_1 r_7 \frac{\partial^2 F_1}{\partial x_1 \partial x_7} (\xi_0, C^*) + \nu_1 r_1 r_{10} \frac{\partial^2 F_1}{\partial x_1 \partial x_{10}} (\xi_0, C^*) + \nu_2 r_1 r_{10} \frac{\partial^2 F_2}{\partial x_1 \partial x_{10}} (\xi_0, C^*) \right. \\
 &\quad + \nu_3 r_3 r_5 \frac{\partial^2 F_3}{\partial x_3 \partial x_5} (\xi_0, C^*) + \nu_3 r_3 r_7 \frac{\partial^2 F_3}{\partial x_3 \partial x_7} (\xi_0, C^*) + \nu_4 r_1 r_5 \frac{\partial^2 F_4}{\partial x_1 \partial x_5} (\xi_0, C^*) + \nu_4 r_1 r_7 \frac{\partial^2 F_4}{\partial x_1 \partial x_7} (\xi_0, C^*) \\
 &\quad + \nu_5 r_5 r_{10} \frac{\partial^2 F_5}{\partial x_5 \partial x_{10}} (\xi_0, C^*) + \nu_6 r_1 r_5 \frac{\partial^2 F_6}{\partial x_1 \partial x_5} (\xi_0, C^*) + \nu_6 r_1 r_7 \frac{\partial^2 F_6}{\partial x_1 \partial x_7} (\xi_0, C^*) + \nu_6 r_1 r_{10} \frac{\partial^2 F_6}{\partial x_1 \partial x_{10}} (\xi_0, C^*) \\
 &\quad \left. + \nu_8 r_3 r_8 \frac{\partial^2 F_8}{\partial x_3 \partial x_8} (\xi_0, C^*) + \nu_8 r_7 r_8 \frac{\partial^2 F_8}{\partial x_7 \partial x_8} (\xi_0, C^*) + \nu_9 r_3 r_8 \frac{\partial^2 F_9}{\partial x_3 \partial x_8} (\xi_0, C^*) + \nu_9 r_7 r_8 \frac{\partial^2 F_9}{\partial x_7 \partial x_8} (\xi_0, C^*) \right] \\
 &= -\frac{2\nu_3 r_3 r_7 \vartheta \mu_h A_2 A_3 g}{\Lambda_h k_p} + \frac{2\nu_6 r_1 r_7 \vartheta \mu_h A_2 A_3 g}{\Lambda_h k_p} - \frac{2\nu_1 r_1 r_7 \mu_h A_2 A_3 g}{\Lambda_h k_p} + \frac{2\nu_4 r_1 r_7 \mu_h A_2 A_3 g}{\Lambda_h k_p} - \frac{2\nu_3 r_3 r_5 \vartheta \mu_h A_2 A_3}{\Lambda_h k_p} \\
 &\quad + \frac{2\nu_6 r_1 r_5 \vartheta \mu_h A_2 A_3}{\Lambda_h k_p} - 2\nu_8 r_7 r_8 \alpha \beta_{hm} \delta + 2\nu_9 r_7 r_8 \alpha \beta_{hm} \delta - 2\nu_5 r_5 r_{10} \alpha \varepsilon \beta_{mh} + 2\nu_6 r_1 r_{10} \alpha \varepsilon \beta_{mh} \\
 &\quad - \frac{2\nu_1 r_1 r_5 \mu_h A_2 A_3}{\Lambda_h k_p} + \frac{2\nu_4 r_1 r_5 \mu_h A_2 A_3}{\Lambda_h k_p} - 2\nu_8 r_3 r_8 \alpha \beta_{hm} + 2\nu_9 r_3 r_8 \alpha \beta_{hm} - 2\nu_1 r_1 r_{10} \alpha \beta_{mh} + 2\nu_2 r_1 r_{10} \alpha \beta_{mh} \\
 &= -\frac{2\nu_4 (\mu_h^2 + (k_p + \pi_p + \sigma_p) \mu_h + k_p \sigma_p) r_5^2 A_2 A_3 (g A_2 A_3 k_{hp} \vartheta + A_4 A_5 k_p)}{\Lambda_h k_p^3 A_4 A_5} < 0.
 \end{aligned} \tag{C.9}$$

It follows that the sign of a is negative, thus the bifurcation is transcritical (forward).

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Existence and uniqueness analysis of Atangana-Baleanu fractional order network model of Noro virus

V.B. Otugene*, P.C. Agina[†], H. Edogbanya[‡], and D.C. Digwo[§]

Abstract

This manuscript is concerned to the study of noro virus model and its complications with the Atangana-Baleanu fractional order derivative which is a new approach for such type of biological models. Further for the corresponding results, existence and uniqueness of the solution is examined by engaging the fixed point theory and Picard-Lindelof approach

Keywords: $SEII_tR$ Noro-virus model; Atangana-Baleanu fractional derivative; Picard-Lindelof approach; fixed point theory

1 Introduction

Gastroenteritis is still ranked second among the childhood killer diseases globally [19]. the greater burden of this disease can be observed in developing countries especially sub-Saharan Africa [26]. Decades ago, rotavirus was the predominant pathogen implicated in most cases of diarrhoea among children below five years of age but recent studies have shown that human norovirus (HuNoVs) has become the major aetiological agent responsible for viral gastroenteritis among children below five years of age, replacing rotavirus subsequent to the introduction of rotavirus vaccine in most countries [7].

Globally, an estimated 677 million episodes of HuNoV associated gastroenteritis and 200,000 mortalities have been recorded annually (Pires et al., 2015). Noroviruses belong to the caliciviridae family and are non-enveloped, single-stranded RNA viruses that are extremely infectious with just a few virions capable of causing gastroenteritis among infected individuals [2]. They are classified into ten genogroup (GI-GX) based on genetic similarity but most cases of acute gastroenteritis in humans were caused by two genogroups (GI and GII) while other genogroups can cause infections in the feline, canine, bovine etc. population [8,23,24]. Further genotypic classification based on the partial sequence of RNA-dependent RNA polymerase (RdRp) in ORF1 and complete sequence of the capsid in ORF2 led to the identification of 52 (49 accepted genotypes and 3 tentative genotypes) genotypes within the genogroups [8]. Noroviruses are formidable pathogens due to their ability to undergo genetic recombination and mutation leading to the emergence of a new strain every 2-3 years [16].

HuNoVs affects all ages causing self limiting infections in healthy individuals with an incubation period of 12-48h [20]. But causes severe disease among children, the elderly and immunocompromised persons often accompanied with symptoms including: diarrhoea, stomach

*Corresponding Author: College of Information and Communication Technology, Salem University, Nigeria; E-Mail: otugenevictorbamigwojo@gmail.com

[†]Department of Mathematics, Chukwuemeka Odumegwu Ojukwu University, Anambra State, Nigeria; E-Mail: pc.agina@coou.edu.ng

[‡]Mathematics Department. Federal University Lokoja, Nigeria; E-Mail: helenedogbanya@fulokoja.edu.ng

[§]Department of Microbiology, University of Nigeria, Nsukka; E-Mail: daniel.digwo.pg00245@unn.edu.ng

cramps, profuse vomiting, leading to loss of fluids that might result to severe dehydration [20]. Specifically, HuNoVs can result in chronic gastroenteritis among immunocompromised patients e.g. transplant patients with infection lasting from several weeks to a year (Brown et al., 2017). Despite causing gastroenteritis in some individuals, certain population of individuals (5% – 30%) infected with HuNoVs become asymptomatic and several reasons have been provided for this observation including: lack of histo-blood group antigens (HBGA) absent in this population, patients recovering from a previous HuNoV infection and are still shedding the virions in their stool or passage of non-replicating virus through the human gastrointestinal tract [15]. Norovirus is transmitted mainly through the fecal-oral route and close contact with infected persons. The virus has high stability in the environment and survives on fomites for long periods [19].

Norovirus is responsible for sporadic outbreaks in enclosed spaces such cruise ships, military camps, and long term care facilities [21]. It is the primary pathogen implicated in most cases of food borne gastroenteritis due to several outbreaks linked to consumption of partially cooked, seafoods or ready to eat foods contaminated by infected food handlers [17,21]. Despite, the outbreaks and huge burden of disease linked to HuNoV, efforts to develop a vaccine for the prevention of the infection are underway although there is still limiting issues such as the lack of robust cell lines to culture and study these viruses. there is the challenge of lack of data especially from developing countries on the burden of the infections due to poor medical diagnostic facilities for viral pathogens [18].

The insight that the transmission dynamics of epidemic disease can be formulated using mathematical language dates back to 1766 when Daniel Bernouli published a paper where he described the effect of smallpox variolation on life expectancy [10]. Other early mathematical models in epidemiology were introduced in 1927 when Kermack and Mckendrick published a series of papers that described the dynamic of disease transmission in term of system of differential equation [12]. Epidemiological mathematical models provide several aspect for understanding the dynamic of the spread of an epidemiv and suggestion of effective control strategies. A large number of studies and publications have been made on modeling and analysis of epidemiology disease. However, the major of the studies were restricted to integer order differential equations.

Resent studies has revealed that fractional differential equations being a branch of mathematical analysis that studies calculus of derivatives and integral of arbitrary order can be used to model several phenomenon in different fields including epidemiology [10]. The non local property in the sense that the n th derivatives of a function $g(x)$ at x is a local property of a function only when n is an integer, whereas a non integer fraction derivative of $g(x)$ at $x=b$ depends on all values of $g(x)$ including those far away from b is the advantage of describing mathematical models using fractional derivatives. In other words, in the case of fractional order derivatives since it involves derivatives and integral of arbitrary real and complex orders, the future state depends on the present and previous.

Using fractional derivatives in modeling a dynamical system helps to describe the hereditary properties and efficacy of the memory of essential features in many biological mechanism [14]. Many authors contributed to the development of fractional calculus starting from 1695 when L'Hospital asked Leibiz what if the order of a derivative is $n=1/2$? Other contributor include Euler 1730 and Langrange in 1812. other prominent name in fractional derivatives and integral include Reimann Liouvill, Hadamard, Caputo, Caputo-Fabrizio and recently Atangana- Baleanu. Atangana identified previous mistakes and advanced corrections to the use of fractional calculus related to fundamental theorem of calculus. Baleanu et al a fractional mathematical model for tumor-immune surveillance mechanism and investigated the effect of chemotherapy on the model. Sene studied a fractional diffusion equation in the context of the fractional operator. Kolade and Owolabi performed analysis and numerical simulation of a system using a two-step family of Adams- Bashforth method to approximate the A-B fractional derivatives [28].

A-B fractional derivatives operator involving the Mittagge -Leffler kernel is used to analyse SEIRA mathematical model [25]. The above surveyed works shows that fractional derivatives

have many applications in mathematical modeling and analysis of real life phenomena .The recent A-B fractional developpe has earned popularity and respect due to its immense application in biological, physical,medical enginnering and several other nonlinear analysis.

Motivated by the above argument , we study in the this paper $SEIIR$ (susceptible-exposed-infected-infected on treatment-recovered cases).The authors have structured the rest of this paper as follows. In section 2 , we described and formulated the rota virus model with fractional order. In section 3 we established the existence and uniqueness of solution by exerting the fixed point theory and the Pichard Lindelof approach. In section 4 positivity and boundedness of solution in the sense of Atangana - Baleanu operator was shown.

2 Model description and formulation

In this section , we present the Atangana- Baleanu fractional derivative representation of HBV mathematical model. The basic definitions of Atangana- Baleanu is given by :

Definition 1 : Let $g \in C^1(a, b)$, $a < b$, be a function, and let $\alpha \in [0, 1]$. The Atangana- Baleanu (AB) fractional derivative in Caputo type of order α given by [23,24]

$$D_t^\alpha g(t) = \frac{F(\alpha)}{1-\alpha} \int_0^t \frac{dg}{dk} E_\alpha \left[-\frac{\alpha}{1-\alpha} (t-k)^\alpha \right] dk, \quad (1)$$

where $F(\alpha)$ is the normalization function given by $F(\alpha) = 1 - \alpha + \frac{\alpha}{\Gamma(\alpha)}$, characterized by $F(0) = F(1) = 1$, and the Mittag-Leffler function $E_\alpha(z)$ with C the set of complex number given by

$$E_\alpha = \sum_{\beta=0}^{\infty} \frac{z^\beta}{\Gamma(1+\alpha\beta)}, \alpha, z \in C, R(\alpha) > 0.$$

Definition 2 : The AB fractional integral of the function $g \in C^1(a, b)$, $a < b$, is given by [25,25,33]

$$I_t^\alpha g(t) = \frac{1-\alpha}{F(\alpha)} g(t) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_a^t g(k)(t-k)^{\alpha-1} dk. \quad (2)$$

Lemma 1 ([25]) The AB fractional derivative and AB fractional integral of the function $g \in C^1(a, b)$ satisfies the Newton-Leibniz equality

$$I_t^\alpha (D_t^\alpha) = g(t) - g(a).$$

Lemma 2 ([26,27]) For two functions $f, g \in \beta^1(a, b)$, $b > a$, the AB fractional derivative satisfies the following inequality:

$$||D_t^\alpha f(t) - D_t^\alpha g(t)|| \leq \beta ||f(t) - g(t)||.$$

The mathematical model with integer order used in this study is expressed by the equation:

$$\begin{aligned}
\frac{dS(t)}{dt} &= \beta - \mu S(t) - \alpha_1 C(N)(I(t) + Q(t)S(t), \\
\frac{dE(t)}{dt} &= \alpha_1 C(N)(I(t) + Q(t)S(t) - \lambda_2 E(t), \\
\frac{dI(t)}{dt} &= \lambda_2 I(t) - (\mu + \alpha_3 + \lambda_1)Q(t), \\
\frac{dI_T(t)}{dt} &= (1 - \rho)\alpha_3 I(t) - (\mu + \lambda_2)I_T(t), \\
\frac{dQ(t)}{dt} &= \rho\alpha_3 I(t) - \Psi Q(t), \\
\frac{dR(t)}{dt} &= \alpha_4 I_T(t) - (\mu + \alpha_5)R(t),
\end{aligned} \tag{3}$$

where $S(t)$ represent number of individuals susceptible to noro virus, $E(t)$ represent exposed individuals, $I(t)$ represent individuals infected with noro virus, I_t represent infected individual on treatment, $Q(t)$ is contaminated surface, $R(t)$ represent recovered individuals. The recruitment rate of the population is denoted by β , μ is death rate due to disease, $\lambda_1, \lambda_2, \dots, \lambda_5$ is movement rate from one compartment to another, $\delta_1, \dots, \delta_4$ is rate at which individuals die due to natural cause. $C(N) = \frac{\alpha N}{1 + bN}$ derived in [28] is the saturating contact rate, α, b are positive contacts and the total population is given by: $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$. The natural death rate term is μN .

In the absence of disease the differential equation of total population is $\frac{dN}{dt} = \beta - \alpha_3 N$ and thus $\lim_{t \rightarrow \infty} N(t) = \frac{\beta}{\alpha_3}$ and it is the carrying capacity of the demographic structure under consideration. Observed that $C(N) \approx \alpha N$ for small N and $C(N) \approx \frac{\alpha}{b}$ for large N . We assume here also that a patient that recovered from noro virus is susceptible to the virus as he/she has no permanent immunity.

Thus the AB fractional order mathematical model taking into account the assumptions, the saturating contact rate is giving by the system of differential equations:

$$\begin{aligned}
{}_0^{ABC}D_t^\alpha S(t) &= G_1(t, S), \\
{}_0^{ABC}D_t^\alpha E(t) &= G_2(t, E), \\
{}_0^{ABC}D_t^\alpha I(t) &= G_3(t, I), \\
{}_0^{ABC}D_t^\alpha I_T(t) &= G_4(t, I_T), \\
{}_0^{ABC}D_t^\alpha Q(t) &= G_5(t, Q), \\
{}_0^{ABC}D_t^\alpha R(t) &= G_6(t, R),
\end{aligned} \tag{4}$$

where the kernels are given by

$$\begin{aligned}
G_1(t, S) &= \beta - \mu S(t) - b_0 \frac{(I(t) + Q(t))}{k(N)} S(t), \\
G_2(t, E) &= b_0 \frac{(I(t) + Q(t))}{k(N)} S(t) - \lambda_2 E(t), \\
G_3(t, I) &= \lambda_2 I(t) - (\mu + \alpha_3 + \lambda_1)Q(t), \\
G_4(t, I_T) &= (1 - \rho)\alpha_3 I(t) - (\mu + \lambda_2)I_T(t), \\
G_5(t, Q) &= \rho\alpha_3 I(t) - \Psi Q(t), \\
G_6(t, R) &= \alpha_4 I_T(t) - (\mu + \alpha_5)R(t),
\end{aligned}$$

$S(0) = S_0, E(0) = E_0, I(0) = I_0, I_T(0) = I_T(0), Q(0) = Q_0, R(0) = R_0$ are the initial conditions and $b_0 = \alpha\alpha_1$, and α_1 is the probability per unit time of transmitting the infection between

individuals taking part in contact or a contaminated surface, $K(N) = 1 + BN$. In the presence of endemic, $\frac{dN}{dt} = \beta - \mu N - \alpha_3$ and this tells us that the population size is not constant.

3 Existence and uniqueness of solutions

We shall deliberate the existence and uniqueness of the solution of the fractional order model (4). To demonstrate the existence and uniqueness of the solution to (4) we engage the famous theorem referred to as the Banach fixed point theorem. For an extensive study of the fixed point and contraction, we refer the reader to [21] and the reference therein.

To show the existence and uniqueness of solution we proceed as follows. Applying the AB fractional integral defined in (2) to model (4), we have:

$$\begin{aligned} S(t) - S(0) &= \frac{1-\alpha}{F(\alpha)}G_1(t, S) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_1(k, S)(t-k)^{\alpha-1}dk, \\ E(t) - E(0) &= \frac{1-\alpha}{F(\alpha)}G_2(t, E) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_2(k, E)(t-k)^{\alpha-1}dk, \\ I(t) - I(0) &= \frac{1-\alpha}{F(\alpha)}G_3(t, I) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_3(k, I)(t-k)^{\alpha-1}dk, \\ I_T(t) - I_T(0) &= \frac{1-\alpha}{F(\alpha)}G_4(t, I_T) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_4(k, I_T)(t-k)^{\alpha-1}dk, \\ Q(t) - Q(0) &= \frac{1-\alpha}{F(\alpha)}G_5(t, Q) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_5(k, Q)(t-k)^{\alpha-1}dk, \\ R(t) - R(0) &= \frac{1-\alpha}{F(\alpha)}G_6(t, R) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_6(k, R)(t-k)^{\alpha-1}dk. \end{aligned} \quad (5)$$

The set $B = H(J)xH(J)x...andH(J) = C[0, T]$ is a Banach space of real value continuous functions defined on the interval $J = [0, T]$ with the corresponding norm defined by:

$$\|(S, E, I, I_T, Q, R)\| = \|S\| + \|E\| + \|I\| + \|I_T\| + \|Q\| + \|R\|, \text{ where, } \|S\| = \sup_{t \in J} |S(t)|, \|E\| = \sup_{t \in J} |E(t)|, \|I\| = \sup_{t \in J} |I(t)|, \|I_T\| = \sup_{t \in J} |I_T(t)|, \|Q\| = \sup_{t \in J} |Q(t)|, \|R\| = \sup_{t \in J} |R(t)|.$$

Theorem 1 (Lipschits Condition and Contraction)

For each of the kernels, $G_1(t, S), G_2(t, P), \dots, G_6(t, Y)$ in (2) there exist $L_i > 0, i = 1, 2, 3, 4, 5, 6$ such that

$$\begin{aligned} \|G_1(t, S) - G_1(t, S_1)\| &\leq L_1\|S(t) - S_1(t)\|, \\ \|G_2(t, E) - G_1(t, P_2)\| &\leq L_2\|E(t) - E_1(t)\|, \\ \|G_3(t, Q) - G_1(t, I_1)\| &\leq L_3\|I(t) - I_1(t)\|, \\ \|G_4(t, I_T) - G_1(t, W_1)\| &\leq L_4\|I_T(t) - I_{T1}(t)\|, \\ \|G_5(t, Q) - G_5(t, Q_1)\| &\leq L_5\|Q(t) - Q_1(t)\|, \\ \|G_6(t, R) - G_6(t, R_1)\| &\leq L_6\|R(t) - R_1(t)\|, \end{aligned}$$

are contraction for $0 \leq L_i \leq 1 \forall i = 1, 2, 3, 4, 5, 6$.

Proof.

$$\begin{aligned} \|G_1(t, S) - G_1(t, S_1)\| &= \|\beta - \mu S(t) - b_0 \frac{(I(t) + Q(t))}{k(N)} S(t) - (\beta - \mu S_1(t) - b_0 \frac{(I(t) + Q(t))}{k(N)} S_1(t))\| \\ &\leq \left(b_0 \frac{m_2 + m_1}{K(N)} \right) \|(S_1(t) - S(t))\| \\ &\leq L_1 \|(S_1(t) - S(t))\|, \end{aligned}$$

where $L_1 = \left(b_0 \frac{m_3 + m_5}{K(N)}\right) + \mu \|S\|$ is $\sup_{t \in J} = M_1, \|E\|$ is $\sup_{t \in J} = M_2, \|I\|$ is $\sup_{t \in J} = M_3, \|I_T\|$ is $\sup_{t \in J} = M_4, \|Q\|$ is $\sup_{t \in J} = M_5, \|R\|$ is $\sup_{t \in J} = M_6$.

In a similar way, we can show the existence of $L_1 = 2, 3, 4, 5, 6$, and a contraction principle for $G_2(t, E), G_3(t, I), G_4(t, I_T), G_5(t, Q), G_6(t, R), 0 \leq L_i < 1$.

We now define the recursive form of (5) for $t = t_n, n = 1, 2, 3, \dots$

$$\begin{aligned} S_n(t) &= \frac{1-\alpha}{F(\alpha)} G_1(t, S_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_1(k, S_{n-1})(t-k)^{\alpha-1} dk, \\ E_n(t) &= \frac{1-\alpha}{F(\alpha)} G_2(t, E_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_2(k, E_{n-1})(t-k)^{\alpha-1} dk, \\ I_n(t) &= \frac{1-\alpha}{F(\alpha)} G_3(t, I_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_3(k, I_{n-1})(t-k)^{\alpha-1} dk, \\ I_{Tn}(t) &= \frac{1-\alpha}{F(\alpha)} G_4(t, I_{Tn-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_4(k, I_{Tn-1})(t-k)^{\alpha-1} dk, \\ Q_n(t) &= \frac{1-\alpha}{F(\alpha)} G_5(t, Q_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_5(k, Q_{n-1})(t-k)^{\alpha-1} dk, \\ R_n(t) &= \frac{1-\alpha}{F(\alpha)} G_6(t, R_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_6(k, R_{n-1})(t-k)^{\alpha-1} dk. \end{aligned} \quad (6)$$

The difference between successive terms in (6) are expressed as follows:

$$\begin{aligned} A_{1n}(t) &= S_n - S_{n-1} = \frac{1-\alpha}{F(\alpha)} (G_1(t, S_{n-1}) - G_1(t, S_{n-2})) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_1(t, S_{n-1}) - G_1(t, S_{n-1}))(t-k)^{\alpha-1} dk, \\ A_{2n}(t) &= E_n - E_{n-1} = \frac{1-\alpha}{F(\alpha)} (G_2(t, E_{n-1}) - G_2(t, E_{n-2})) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_2(t, E_{n-1}) - G_2(t, E_{n-1}))(t-k)^{\alpha-1} dk, \\ A_{3n}(t) &= I_n - I_{n-1} = \frac{1-\alpha}{F(\alpha)} (G_3(t, I_{n-1}) - G_3(t, I_{n-2})) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_3(t, I_{n-1}) - G_3(t, I_{n-1}))(t-k)^{\alpha-1} dk, \\ A_{4n}(t) &= I_{Tn} - I_{Tn-1} = \frac{1-\alpha}{F(\alpha)} (G_4(t, I_{Tn-1}) - G_4(t, I_{Tn-2})) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_4(t, I_{Tn-1}) - G_4(t, I_{Tn-1}))(t-k)^{\alpha-1} dk, \\ A_{5n}(t) &= Q_n - Q_{n-1} = \frac{1-\alpha}{F(\alpha)} (G_5(t, Q_{n-1}) - G_5(t, Q_{n-2})) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_5(t, Q_{n-1}) - G_5(t, Q_{n-1}))(t-k)^{\alpha-1} dk, \\ A_{6n}(t) &= R_n - R_{n-1} = \frac{1-\alpha}{F(\alpha)} (G_6(t, R_{n-1}) - G_6(t, R_{n-2})) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_6(t, R_{n-1}) - G_6(t, R_{n-1}))(t-k)^{\alpha-1} dk. \end{aligned} \quad (7)$$

Taking the norm of both sides of (5), we have

$$\begin{aligned}
 \|A_{1n}(t)\| &= \|S_n(t) - S_{n-1}(t)\| \frac{1-\alpha}{F(\alpha)} \|(G_1(t, S_{n-1}) - G_1(t, S_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_1(t, S_{n-1}) - G_1(t, S_{n-1}))\| (t-k)^{\alpha-1} dk, \\
 \|A_{2n}(t)\| &= \|PE_n - E_{n-1}\| = \frac{1-\alpha}{F(\alpha)} \|(G_2(t, E_{n-1}) - G_2(t, E_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_2(t, E_{n-1}) - G_2(t, E_{n-1}))\| (t-k)^{\alpha-1} dk, \\
 \|A_{3n}(t)\| &= \|I_n - I_{n-1}\| = \frac{1-\alpha}{F(\alpha)} \|(G_3(t, I_{n-1}) - G_3(t, I_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_3(t, I_{n-1}) - G_3(t, I_{n-1}))\| (t-k)^{\alpha-1} dk, \\
 \|A_{4n}(t)\| &= \|I_{Tn} - I_{Tn-1}\| = \frac{1-\alpha}{F(\alpha)} \|(G_4(t, I_{Tn-1}) - G_4(t, I_{Tn-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_4(t, W_{n-1}) - G_4(t, W_{n-1}))\| (t-k)^{\alpha-1} dk, \\
 \|A_{5n}(t)\| &= \|Q_n - Q_{n-1}\| = \frac{1-\alpha}{F(\alpha)} \|(G_5(t, Q_{n-1}) - G_5(t, Q_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_5(t, Q_{n-1}) - G_5(t, Q_{n-1}))\| (t-k)^{\alpha-1} dk, \\
 \|A_{6n}(t)\| &= \|R_n - R_{n-1}\| = \frac{1-\alpha}{F(\alpha)} \|(G_6(t, R_{n-1}) - G_6(t, R_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_6(t, R_{n-1}) - G_6(t, R_{n-1}))\| (t-k)^{\alpha-1} dk.
 \end{aligned} \tag{8}$$

The first equation in (8) is reduced to the following expression

$$\begin{aligned}
 \|A_{1n}(t)\| &= \|S_n(t) - S_{n-1}(t)\| \leq \frac{1-\alpha}{F(\alpha)} \|(G_1(t, S_{n-1}) - G_1(t, S_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_1(t, S_{n-1}) - G_1(t, S_{n-1}))\| (t-k)^{\alpha-1} dk, \\
 \|A_{1n}(t)\| &\leq \frac{1-\alpha}{F(\alpha)} L_1 \|S_{n-1}(t) - S_{n-2}(t)\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} L_1 \int_0^t \|S_{n-1}(t) - S_{n-2}(t)\| (t-k)^{\alpha-1} dk, \\
 \|A_{1n}(t)\| &\leq \frac{1-\alpha}{F(\alpha)} L_1 \|S_{n-1}(t) - S_{n-2}(t)\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} L_1 \|S_{n-1}(t) - S_{n-2}(t)\| \int_0^t (t-k)^{\alpha-1} dk, \\
 \|A_{1n}(t)\| &\leq L_1 \|S_{n-1}(t) - S_{n-2}(t)\| \left| \frac{1-\alpha}{F(\alpha)} + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (t-k)^{\alpha-1} dk \right| \\
 \|A_{1n}(t)\| &\leq L_1 \|A_{1(n-1)}\| \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right|.
 \end{aligned}$$

Therefore,

$$\|A_{1n}(t)\| \leq L_1 \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right| \|A_{1(n-1)}\| \tag{9}$$

In a similarly way , we reduced the remaining expression of (8) to the form:

$$\begin{aligned}
 \|A_{2n}(t)\| &\leq L_2 \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right| \|A_{2(n-1)}(t)\| \\
 \|A_{3n}(t)\| &\leq L_3 \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right| \|A_{3(n-1)}(t)\| \\
 \|A_{4n}(t)\| &\leq L_4 \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right| \|A_{4(n-1)}(t)\| \\
 \|A_{5n}(t)\| &\leq L_5 \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right| \|A_{5(n-1)}(t)\| \\
 \|A_{6n}(t)\| &\leq L_6 \left| \frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right| \|A_{6(n-1)}(t)\|.
 \end{aligned} \tag{10}$$

Theorem 2: The fractional model given in (2) has a solution if we can find M_0 satisfying

$$\left(\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_i \leq 1, i = 1, 2, \dots, 6. \tag{11}$$

From (8) and (9) we have:

$$\begin{aligned}
 \|A_{1n}(t)\| &\leq \|S(0)\| \left| \left(\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_1 \right|^n, \\
 \|A_{2n}(t)\| &\leq \|E(0)\| \left| \left(\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_2 \right|^n, \\
 \|A_{3n}(t)\| &\leq \|I(0)\| \left| \left(\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_3 \right|^n, \\
 \|A_{4n}(t)\| &\leq \|I_T(0)\| \left| \left(\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_4 \right|^n, \\
 \|A_{5n}(t)\| &\leq \|Q(0)\| \left| \left(\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_5 \right|^n, \\
 \|A_{6n}(t)\| &\leq \|R(0)\| \left| \left(\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right) L_6 \right|^n.
 \end{aligned} \tag{12}$$

The existence of the solution is confirmed by theorem 1, and we need now to show that the functions $S(t)$, $P(t)$, $Q(t)$, $W(t)$, $X(t)$ and $Y(t)$ are solutions of model (2). To show this we assumed that the following is satisfied:

$$\begin{aligned}
 S(t) - S(0) &= S_n(t) - a_{1n}(t), \\
 E(t) - E(0) &= E_n(t) - a_{2n}(t), \\
 I(t) - I(0) &= Q_n(t) - a_{3n}(t), \\
 I_T(t) - I_T(0) &= I_{Tn}(t) - a_{4n}(t), \\
 Q(t) - Q(0) &= Q_n(t) - a_{5n}(t), \\
 R(t) - R(0) &= R_n(t) - a_{6n}(t).
 \end{aligned} \tag{13}$$

From (13),

$$\begin{aligned}
 \|a_{1n}(t)\| &= \frac{1-\alpha}{F(\alpha)} \|(G_1(t, S_{n-1}) - G_1(t, S_{n-2}))\| + \\
 &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t \|(G_1(k, S_{n-1}) - G_1(k, S_{n-1}))\| (t-k)^{\alpha-1} dk,
 \end{aligned}$$

$$\begin{aligned} \|a_{1n}(t)\| &\leq \frac{1-\alpha}{F(\alpha)} L_1 \|S_n(t) - S_{n-1}(t)\| \\ &\quad \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} L_1 \|S_n(t) - S_{n-1}(t)\|. \end{aligned}$$

If we were to repeat the process recursively, we have

$$\|a_{1n}(t)\| \leq \left[\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right]^{n+1} L_1^n \|S_n(t) - S_{n-1}(t)\|^n,$$

which at $t = M_0^\alpha$ yields,

$$\|a_{1n}(t)\| \leq \left[\frac{1-\alpha}{F(\alpha)} + \frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)} \right]^{n+1} L_1^n \|S_n(t) - S_{n-1}(t)\|^n. \quad (14)$$

Applying the limit to both sides of (13) as $n \rightarrow \infty$, we have see that $\|a_{1n}(t)\| \rightarrow 0$ for

$$\left[\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right] L_1 \leq 1.$$

In a similar fashion, we show that

$$\|a_{2n}(t)\| \rightarrow 0, \|a_{3n}(t)\| \rightarrow 0, \|a_{4n}(t)\| \rightarrow 0, \|a_{5n}(t)\| \rightarrow 0, \|a_{6n}(t)\| \rightarrow 0,$$

for

$$\left[\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right] L_i \leq 1 \quad i = 2, 3, 4, 5, 6.$$

Theorem 1 and 2 guaranty the existence of solution of model (2) by the Banach fixed point theorem. we shall show the uniqueness of the solution in Theorem 3.

Theorem 3: (Uniqueness of Solution)

The fractional model (2) has a unique solution provided that

$$\left[\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right] L_i \leq 1 \quad i = 2, 3, 4, 5, 6. \quad (15)$$

Proof.

We assume that $S_1(t), E_1(t), I_1(t), I_{T1}(t), Q_1(t), R_1(t)$, are solution to model (4). Then,

$$\begin{aligned} S(t) - S_1(t) &= \frac{1-\alpha}{F(\alpha)} (G_1 S(t) - G_1 S_1(t)) + \\ &\quad \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_1 S(t) - G_1 S_1(t)) (t-k)^{\alpha-1} dk. \end{aligned}$$

Taking the norm of both sides , we have:

$$\begin{aligned} \|S(t) - S_1(t)\| &= \frac{1-\alpha}{F(\alpha)} \|L_1\| \|S(t) - S_1(t)\| + \\ &\quad \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \|L_1\| \|S(t) - S_1(t)\|. \end{aligned}$$

Since

$$\left(1 - \|L_1\| \left(\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)} \right) \right) > 0,$$

we obtain $\|S(t) - S_1(t)\| = 0$. Thus we have $S(t) = S_1(t)$. We can also show that $P(t) = P_1(t), Q(t) = Q_1(t), W(t) = W_1(t), X(t) = X_1(t), Y(t) = Y_1(t)$. We have completed the proof of Theorem 3.

4 Positivity and boundedness

The model (4) will be meaningful epidemiologically, if the solution of system (4) with no negative initial data will remain non-negative for all time $t \geq 0$.

Lemma (1). For all time $t \geq 0$ and initial data $N(0) \geq 0$, where $N(t) = (S(t), E(t), I(t), I_T(t), Q(t), R(t))$. The solution of model (4) are non-negative for all $t \geq 0$ if there exist; furthermore $\limsup N(t) \leq \frac{\beta}{\mu}$. The epidemiologically feasible region of model (2) is given by

$$\Omega = \{(S(t), E(t), I(t), I_T(t), Q(t), R(t)) \in \mathbb{R}_+^6 \mid S(t) + P(t) + Q(t) + W(t) + X(t) + Y(t) \leq N \leq \frac{\beta}{\mu}\}. \quad (16)$$

Proof. Applying the AB fractional integral(2) of the first equation of (4) we have:

$$I_t^\alpha D_t^\alpha \left(S(t) e^{\int_0^t (\mu + b_0 \frac{(I(t) + Q(t))}{k(N)}) dS} \right) = \beta I_t^\alpha \left(e^{\int_0^t (\mu + b_0 \frac{(I(t) + Q(t))}{k(N)}) dS} \right).$$

It is obvious that

$$S(t) = \beta e^{-\int_0^t (\mu + b_0 \frac{(I(t) + Q(t))}{k(N)}) dS} \left[\frac{1 - \alpha}{F(\alpha)} e^{\int_0^t (\mu + b_0 \frac{(I(t) + Q(t))}{k(N)}) dS} + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + b_0 \alpha_1 \zeta) dS} \int_0^t (t - w) dw \right] > 0. \quad (17)$$

Similar argument goes for the remaining equations of (2)

$$\begin{aligned}
 \mathbf{E}(\mathbf{t}) &= b_0 \frac{(I(t) + Q(t))}{k(N)} S(t) e^{-\int_0^t \lambda_2 dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t \lambda_2 dt} + \right. \\
 &\quad \left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t \lambda_2 dt} (t-w) dw \right] > 0, \\
 \mathbf{I}(\mathbf{t}) &= \lambda_2 E(t) e^{-\int_0^t (\mu + \alpha_3 + \lambda_1) dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \alpha_3 + \lambda_1) dt} + \right. \\
 &\quad \left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \alpha_3 + \lambda_1) dt} (t-w) dw \right] > 0, \\
 \mathbf{IT}(\mathbf{t}) &= (1-\rho)\alpha_3 I(t) e^{-\int_0^t (\mu + \lambda_2) dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \lambda_2) dt} + \right. \\
 &\quad \left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \lambda_2) dt} (t-w) dw \right] > 0, \\
 \mathbf{Q}(\mathbf{t}) &= \rho\alpha_3 I(t) e^{-\int_0^t \Psi dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t \Psi dt} + \right. \\
 &\quad \left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t \Psi dt} (t-w) dw \right] > 0, \\
 \mathbf{R}(\mathbf{t}) &= \alpha_4 I_T(t) e^{-\int_0^t (\mu + \alpha_5) dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \alpha_5) dt} + \right. \\
 &\quad \left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \alpha_5) dt} (t-w) dw \right] > 0.
 \end{aligned} \tag{18}$$

It follows from (17) and (18) that each of the solution of (2) is non negative and remain in $\mathbb{R}_+^6$.

We now establish the boundedness of the solution of the fractional model (4) taking into account that all the parameters used in the model as said earlier are non negative. we shall continue by summing up all the equation of model (2) which gives

$$D_t^\alpha N = \beta - \mu N - \lambda_2 I_T - \Psi Q(t) - \alpha_5 R(t),$$

$$D_t^\alpha N(t) - \mu N(t) \leq \beta,$$

$$\left(D_t^\alpha N(t) - \mu N(t) \right) e^{\int_0^t \mu dt} \leq \beta e^{\int_0^t \mu dt},$$

$$N(t) \leq \frac{\beta}{\mu}.$$

It is not difficult to observe that $N(t) \rightarrow \frac{\beta}{\mu}$ as $t \rightarrow \infty$. Hence (16) is the biologically feasible region of (4).

5 Conclusion

In this work, we studied a $SEIIR$ epidemic mathematical involving the Atangana-Baleanu fractional order which does not contain a singular kernel. Further, the existence and uniqueness of solution for equilibrium solutions have been proved by engaging the Banach fixed point theorem.

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Some new results on a free boundary value problem related to autoignition of combustible fluid in insulation materials

R.O. Olayiwola^{*†}, J.T. Fadepo^{*}, A.S. Gimba^{*}, A.W. Abubakar[‡], A.B. Zhiri^{*},
and F.E. Okoosi^{*}

Abstract

Autoignition of combustible fluids in insulation materials is one of the major problems facing the processing industries and many developing nations because it leads to serious environmental problem. This paper presents an analytical solution to a free boundary value problem related to autoignition of combustible fluids in insulation materials. The aim is to ascertain whether such a system is safe or if it will undergo ignition for a particular set of conditions. The conditions for the existence of unique solution of the model is established by actual solution method. The properties of solution is examined. The analytical solution is obtained via polynomial approximation method, which show the influence of the parameters involved on the system. The effect of changes in parameters such as the Frank-Kamenetskii number, Lewis number, Nusselt number, condensed reactant diffusion coefficient and the endothermicity are presented graphically and discussed.

Keywords: autoignition; combustible fluids; free boundary; heat transfer; insulation materials; polynomial approximation method

1 Introduction

The fuel's autoignition is usually caused by a leaking of a combustible liquid into insulation material surrounding a hot pipe. Leakage of flammable liquids into isolation layer during a transportation or technological process can lead to spontaneous fire of the fuel. Since the insulation is a porous medium containing an oxidizer, the exothermic oxidation reaction can lead to a substantial self-heating of the fuel and its ignition and, as a result, to destruction of the pipeline [10].

Models of combustion are characterized by two phenomena; ignition and explosion [2, 5].

The investigation of the autoignition process has been carried out by many authors. McIntosh *et al.* [7] presented a mathematical model for the autoignition of combustible liquid in an inert porous material. The simple model takes a spatially uniform approach to both the energy equation and the liquid equation for the fluid and predicts a watershed temperature and thermal runaway. McIntosh and Griffiths [6] presented the mathematical foundations for a simple theory for investigating the

^{*} Corresponding Author; E-mail: olayiwola.rasaq@futminna.edu.ng

[†] Department of Mathematics, Federal University of Technology, Minna, Nigeria.

[‡] Department of Mathematics, Air Force Institute of Technology, Kaduna, Nigeria.

phenomenon of ignition of flammable fluids in lagging material that are used for insulation of hot pipework, for transport of heat transfer fluids, or other similar situations. A theory to explain these findings was presented which shows that there is a watershed temperature beyond which substantial self-heating will take place. Although the theory does not take account of diffusion, it simulates the main physics of the phenomenon. Truscott *et al.* [12] examined an initial value problem related to autoignition of combustible fluids in insulation materials and investigated the effect of diffusion on the system. Brindley *et al.* [3] developed the theoretical background and the limiting criteria for safe operation on autoignition of combustible fluids in insulation materials and discussed the magnitudes of the parameters that may be used to compare theory with experiment.

Popoola and Ayeni [8] extended the model in [12] to a free boundary value problem by assuming that the reactant is not confined to a fixed space. Shchepakina [11] investigated the critical conditions for autoignition of combustible fluids in porous insulation materials. This work concentrated on the critical case which is concerned with the phenomenon of delayed loss of stability in the dynamical model. The realizability conditions for the critical regime are obtained. Shchepakina [10] investigated an autoignition of liquid fuel due to leaking into insulation porous material surrounding a pipeline. By application of the geometric theory of singular perturbations, he described all possible scenarios of the process, reveal the critical condition for the autoignition, and calculate the maximal safe temperature of fuel's heating.

In this paper, an approximate analytical solution capable of predicting the temperature distribution in a process of autoignition of combustible fluids in insulation materials is presented. For this we shall follow the method proposed in [9].

2 Model formulation

The work in [8] is extended by considering the model geometry which may be represented by a single characteristic dimension to be sphere. The model variables expressed as a function of spatial co-ordinate r , $0 \leq r \leq R$, and time t , $t > 0$, are the matrix temperature $T(r, t)$, the concentration of condensed reactant $C_f(r, t)$ and the concentration of oxygen $C_{ox}(r, t)$. The formulation of our model is guided by the following assumptions:

- 1 Initially, a known amount of fluid is soaked uniformly within the block of insulation and there is air present throughout the porous structure.
- 2 The initial temperature of the system is known.
- 3 The reactant is adsorbed in the liquid state on the pore surface within the insulation matrix. It may react in this condensed state or it may evaporate.
- 4 The exothermic oxidation occurs by reaction between gaseous oxygen in the pores and the condensed reactant.
- 5 The order of reaction with respect to the gas-phase oxygen concentration and the condensed fluid concentration is greater than one.
- 6 The vaporization is related to the condensed fluid concentration and its temperature dependence is expressed in an Arrhenius-like exponential form.
- 7 The convective heat transport of gaseous components is ignored, as are pressure gradients within the matrix.

Based on the above assumptions, the respective conservation equations in spherical coordinate system are thus:

$$\frac{\partial T}{\partial t} = \frac{k}{\rho c r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial T}{\partial r} \right) + \frac{QA}{\rho c} C_f^\alpha C_{ox}^\beta e^{-\frac{E}{RT}} - \frac{Q_v F}{\rho c} C_f e^{-\frac{E_v}{RT}} \quad (1)$$

$$\frac{\partial C_f}{\partial t} = \frac{D_f}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial C_f}{\partial r} \right) - AC_f^\alpha C_{ox}^\beta e^{-\frac{E}{RT}} - FC_f e^{-\frac{E_v}{RT}} \quad (2)$$

$$\frac{\partial C_{ox}}{\partial t} = \frac{D_0}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial C_{ox}}{\partial r} \right) - \nu AC_f^\alpha C_{ox}^\beta e^{-\frac{E}{RT}}, \quad (3)$$

with the initial and boundary conditions:

$$\left. \begin{aligned} T(r,0) &= T_0, & \frac{\partial T}{\partial r} \Big|_{r=0} &= 0, & k^* \frac{\partial T}{\partial r} \Big|_{r=s(t)} &= h(T_0 - T|_{r=s(t)}) \dot{s}(t) \\ C_f(r,0) &= C_{f0}, & \frac{\partial C_f}{\partial r} \Big|_{r=0} &= 0, & D_f^* \frac{\partial C_f}{\partial r} \Big|_{r=s(t)} &= -k_{mf} (C_{f0} + C_f|_{r=s(t)}) \dot{s}(t) \\ C_{ox}(r,0) &= C_{ox0}, & \frac{\partial C_{ox}}{\partial r} \Big|_{r=0} &= 0, & D_0^* \frac{\partial C_{ox}}{\partial r} \Big|_{r=s(t)} &= -k_{mox} (C_{ox0} + C_{ox}|_{r=s(t)}) \dot{s}(t) \\ \dot{s}(t) &= \frac{1}{t^\gamma}, & 0 < r < s(t) & \end{aligned} \right\} \quad (4)$$

where C_{ox} is the concentration of Oxygen, C_f is the concentration of condensed reactant, D_0 is the Oxygen diffusion coefficient, D_f is the condensed reactant diffusion coefficient, ρ is the density of the reactant, k is the thermal conductivity of the medium, c is the heat capacity of the medium, Q is the enthalpy of oxidation (exothermicity), Q_v is the enthalpy of vaporization (endothermicity), R is the universal gas constant, T is the temperature of the medium, E is the activation energy (reaction), E_v is the activation energy (vaporization), T_0 is the initial temperature of the medium, ν is the stoichiometry coefficient, C_{ox0} is the initial oxygen concentration within the insulation block, C_{f0} is the initial concentration of the uniformly distributed fluid, t is the time, A is the pre-exponent factor (reaction), F is the pre-exponent factor (vaporization), α and β are the order of reaction, r is the spatial coordinate, $s(t)$ is the moving boundary, D_0^* is the effective Oxygen diffusion coefficient, D_f^* is the effective condensed reactant diffusion coefficient, k^* is the effective thermal conductivity of the medium, k_{mf} is the condensed reactant convective mass transfer coefficient, k_{mox} is the Oxygen convective mass transfer coefficient, h is the convective heat transfer coefficient, $hT_0 \dot{s}(t)$ is the heat energy released per unit time by the reaction, $hT|_{r=s(t)} \dot{s}(t)$ is the energy flux induced by the motion of the boundary preserve energy conservation, $k_{mf} C_{f0} \dot{s}(t)$ is the number of moles per unit time of condensed fluid, $k_{mf} C_f|_{r=s(t)} \dot{s}(t)$ is the mass flux of condensed fluid induced by the motion of the boundary preserve mass conservation, $k_{mox} C_{ox0} \dot{s}(t)$ is the number of moles per unit time of oxidizer that diffused into the system for the reaction, $k_{mox} C_{ox}|_{r=s(t)} \dot{s}(t)$ is the mass flux of oxidizer.

3 Method of solution

3.1 Dimension analysis

Dimensionless variables for space and time is been introduced as:

$$r' = \frac{2r}{\sqrt{D_f} s(t)}, \quad t' = \frac{4t}{s(t)^2}. \quad (5)$$

Dimensionless variables for medium temperature, condensed fluid concentration and oxygen concentration is been introduced as follows:

$$\theta = \frac{E}{RT_0^2}(T - T_0), \quad \psi = \frac{C_f}{C_{f0}}, \quad \phi = \frac{C_{ox}}{C_{ox0}}, \quad E_v = nE, \quad \epsilon = \frac{RT_0}{E}, \quad (6)$$

where $s(t) = (1 - \gamma)^{-1} t^{1-\gamma}$ is moving boundary. Here, we choose $\gamma = \frac{1}{2}$ as in [8].

Using (5) and (6), and after dropping the prime, equations (1) - (4) become

$$\frac{\partial \theta}{\partial t} = \frac{Le}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \theta}{\partial r} \right) + \delta \psi^\alpha \phi^\beta e^{\frac{\theta}{1+\epsilon\theta}} - \delta_1 \psi e^{\frac{n\theta}{1+\epsilon\theta}} \quad (7)$$

$$\frac{\partial \psi}{\partial t} = \frac{1}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \psi}{\partial r} \right) - \sigma_1 \psi^\alpha \phi^\beta e^{\frac{\theta}{1+\epsilon\theta}} - \sigma_2 \psi e^{\frac{n\theta}{1+\epsilon\theta}} \quad (8)$$

$$\frac{\partial \phi}{\partial t} = \frac{\alpha_1}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \psi}{\partial r} \right) - \beta_1 \psi^\alpha \phi^\beta e^{\frac{\theta}{1+\epsilon\theta}} \quad (9)$$

$$\left. \begin{aligned} \theta(r,0) &= 0, & \frac{\partial \theta}{\partial r} \Big|_{r=0} &= 0, & \frac{\partial \theta}{\partial r} \Big|_{r=\frac{2}{\sqrt{D_f}}} &= -Nu\theta \Big|_{r=\frac{2}{\sqrt{D_f}}} \\ \psi(r,0) &= 1, & \frac{\partial \psi}{\partial r} \Big|_{r=0} &= 0, & \frac{\partial \psi}{\partial r} \Big|_{r=\frac{2}{\sqrt{D_f}}} &= -Sh_f \left(1 + \psi \Big|_{r=\frac{2}{\sqrt{D_f}}} \right) \\ \phi(r,0) &= 1, & \frac{\partial \phi}{\partial r} \Big|_{r=0} &= 0, & \frac{\partial \phi}{\partial r} \Big|_{r=\frac{2}{\sqrt{D_f}}} &= -Sh_{ox} \left(1 + \phi \Big|_{r=\frac{2}{\sqrt{D_f}}} \right) \\ 0 < r &< \frac{2}{\sqrt{D_f}} \end{aligned} \right\} \quad (10)$$

where

$$\begin{aligned}
 Le = \frac{k}{\rho c D_f} \text{ is the Lewis number, } \delta = \frac{QA(s(t))^2 C_{f0}^\alpha C_{ox0}^\beta e^{-\frac{E}{RT_0}}}{4 \in T_0 \rho c} \text{ is the Frank-Kamenetskii number} \\
 \text{(reaction), } \delta_1 = \frac{Q_v F(s(t))^2 C_{f0} e^{-\frac{E}{RT_0}}}{4 \in T_0 \rho c} \text{ is the Frank-Kamenetskii number (vaporization),} \\
 \alpha_1 = \frac{D_0}{D_f}, \quad \sigma_1 = \frac{A(s(t))^2 C_{f0}^{\alpha-1} C_{ox0}^\beta e^{-\frac{E}{RT_0}}}{4}, \quad \sigma_2 = \frac{F(s(t))^2 e^{-\frac{E}{RT_0}}}{4}, \\
 \beta_1 = \frac{vA(s(t))^2 C_{f0}^\alpha C_{ox0}^{\beta-1} e^{-\frac{E}{RT_0}}}{4}, \quad Sh_f = \frac{k_{mf} \sqrt{D_f}}{D_f^*} \text{ is the Sherwood number (condensed fluid),} \\
 Sh_{ox} = \frac{k_{mox} \sqrt{D_f}}{D_0^*} \text{ is the Sherwood number (oxidizer), } \quad Nu = \frac{h \sqrt{D_f}}{k^*} \text{ is the Nusselt number.}
 \end{aligned}$$

3.2 Existence and uniqueness of solution

Theorem 3.1: Let $Le = \alpha_1 = \sigma_1 = \sigma_2 = \beta_1 = Nu = Sh_f = Sh_{ox} = 1$ and $\delta_1 = -\frac{\delta}{2}$. Then there exists a solution of (7) – (9) which satisfies (10).

Proof: Multiply (8) and (9) by $\frac{\delta}{2}$ and adding the resulting equations, we obtain

$$\begin{aligned}
 \frac{\partial \varphi}{\partial t} &= \frac{1}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \varphi}{\partial r} \right) \\
 (11) \quad \varphi(r, 0) &= \delta, \quad \left. \frac{\partial \varphi}{\partial r} \right|_{r=0}, \quad \left. \frac{\partial \varphi}{\partial r} \right|_{r=\frac{2}{\sqrt{D_f}}} = - \left(\delta + \varphi \Big|_{r=\frac{2}{\sqrt{D_f}}} \right), \quad (12)
 \end{aligned}$$

where

$$\varphi(r, t) = \theta(r, t) + \frac{\delta}{2} (\psi(r, t) + \phi(r, t)).$$

Assuming a polynomial solution of the form (see, [9]):

$$\varphi(r, t) = c_0(t) + c_1(t)r + c_2(t)r^2, \quad (13)$$

we obtain the approximated solution of equations (11) and (12) as

$$\phi(r, t) = \frac{\delta \sqrt{D_f}}{4} + \left(1 + \frac{\sqrt{D_f}}{4}\right) \left(b + (\delta - b)e^{-at}\right) - \frac{\sqrt{D_f}}{4} \left(\delta + (b + (\delta - b)e^{-at})\right) r^2 \quad (14)$$

where

$$a = \frac{240D_f}{40D_f + 8\sqrt{D_f} - 32}, \quad b = -\frac{12}{D_f} \delta.$$

Then, we obtain

$$\psi(r, t) = \frac{2}{\delta} \left(\left(\frac{\delta \sqrt{D_f}}{4} + \left(1 + \frac{\sqrt{D_f}}{4}\right) \left(b + (\delta - b)e^{-at}\right) - \frac{\sqrt{D_f}}{4} \left(\delta + (b + (\delta - b)e^{-at})\right) \right) - \theta(r, t) \right) - \phi(r, t) \quad (15)$$

$$\phi(r, t) = \frac{2}{\delta} \left(\left(\frac{\delta \sqrt{D_f}}{4} + \left(1 + \frac{\sqrt{D_f}}{4}\right) \left(b + (\delta - b)e^{-at}\right) - \frac{\sqrt{D_f}}{4} \left(\delta + (b + (\delta - b)e^{-at})\right) \right) - \theta(r, t) \right) - \psi(r, t) \quad (16)$$

$$\theta(r, t) = \left(\frac{\delta \sqrt{D_f}}{4} + \left(1 + \frac{\sqrt{D_f}}{4}\right) \left(b + (\delta - b)e^{-at}\right) - \frac{\sqrt{D_f}}{4} \left(\delta + (b + (\delta - b)e^{-at})\right) \right) - \frac{2}{\delta} (\psi(r, t) + \phi(r, t)) \quad (17)$$

Hence, there exists a solution of problem (7) – (10). This completes the proof.

3.3 Properties of solution

Following thermal ignition theory, it is necessary to assume that there is always sufficient oxygen and fuel present in subcritical conditions so that the behaviour of the system is then governed solely by the energy conservation equation (7). That is ψ and ϕ are assumed constant.

Lemma 3.1: Let $\epsilon \rightarrow 0$. Then the parabolic differential equation (7) with initial and boundary conditions (10) has a unique solution.

Proof: It suffices to show that the Lipschitz condition is satisfied. That is if we are able to show that $|f(r, t, \theta_1) - f(r, t, \theta_2)| \leq k|\theta_1 - \theta_2|$.

From (7),

$$f(r, t, \theta) = \delta \psi^\alpha \phi^\beta e^\theta - \delta_1 \psi e^{n\theta}.$$

By mean value theorem

$$\frac{\partial f}{\partial \theta} = \delta \psi^\alpha \phi^\beta e^\theta - \delta_1 n \psi e^{n\theta}.$$

But

$$\max \frac{\partial f}{\partial \theta} \text{ is achieved when } n=0 \text{ and } e^\theta = 1 \text{ i.e. } n=0 \text{ and } \theta=0.$$

Hence

$$k = \max \frac{\partial f}{\partial \theta} = \delta \psi^\alpha \phi^\beta.$$

Therefore,

$$|f(r, t, \theta_1) - f(r, t, \theta_2)| \leq \delta \psi^\alpha \phi^\beta |\theta_1 - \theta_2|.$$

Since the Lipschitz condition is satisfied, equation (7) has a unique solution. This completes the proof.

For a well stirred reaction when the activation energy for reaction is the same as activation energy for vaporization, i.e. $n=1$ and in a high activation energy situation $\epsilon \rightarrow 0$, equation (7) reduces to

$$\frac{d\theta}{dt} = \delta \psi^\alpha \phi^\beta e^\theta - \delta_1 \psi e^\theta, \quad \theta(0) = 0 \quad (18)$$

Solving (18), we obtain

$$\text{As } t = \frac{1}{(\delta \psi^\alpha \phi^\beta - \delta_1 \psi)}, \quad \theta(t) \rightarrow \infty. \quad (19)$$

Hence, $\theta(t) \rightarrow \infty$ is thermal runaway and $t = \frac{1}{(\delta \psi^\alpha \phi^\beta - \delta_1 \psi)}$ is actually the time of thermal runaway.

3.4 Analytical solution via polynomial approximation method

In the presence of diffusion, assuming constant ψ and ϕ , equation (7) can be solve analytically in the limit $\epsilon \rightarrow 0$. Follow the idea in [1], we assume the expression for $\exp(\theta)$, that is,

$$\exp(\theta) \approx 1 + (e-2)\theta. \quad (20)$$

Then (7) and (10) can be written as:

$$\frac{\partial \theta}{\partial t} = \frac{Le}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \theta}{\partial r} \right) + \delta \psi^\alpha \phi^\beta (1 + (e-2)\theta) - \delta_1 \psi (1 + (e-2)\theta)^n \quad (21)$$

$$\theta(r, 0) = 0, \quad \left. \frac{\partial \theta}{\partial r} \right|_{r=0} = 0, \quad \left. \frac{\partial \theta}{\partial r} \right|_{r=\frac{2}{\sqrt{D_f}}} = -Nu\theta \Big|_{r=\frac{2}{\sqrt{D_f}}} \quad (22)$$

Here, we assume polynomial solution of the form (see, [9]):

$$\theta(r, t) = a_0(t) + a_1(t)r + a_2(t)r^2. \quad (23)$$

Applying the boundary conditions as given in (22), we obtain

$$a_1(t) = 0, \quad a_2(t) = -\frac{Nu\sqrt{D_f}}{4} \theta \Big|_{r=\frac{2}{\sqrt{D_f}}}, \quad a_0(t) = \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) \theta \Big|_{r=\frac{2}{\sqrt{D_f}}} \quad (24)$$

Then, equation (23) becomes

$$\theta(r, t) = \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) \theta \Big|_{r=\frac{2}{\sqrt{D_f}}} - \frac{Nu\sqrt{D_f}}{4} \theta \Big|_{r=\frac{2}{\sqrt{D_f}}} r^2 \quad (25)$$

For long spherical shape (see, [4]), we have

$$\bar{\theta} = 3 \int_0^1 r^2 \theta dr \quad (26)$$

where $\bar{\theta}$ is the average temperature.

Equations (26) gives the relations

$$\bar{\theta} = \left(\frac{8}{D_f \sqrt{D_f}} \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) - \frac{96Nu}{20D_f^2} \right) \theta \Big|_{r=\frac{2}{\sqrt{D_f}}} \quad (27)$$

And

$$\frac{\partial}{\partial t} \bar{\theta} = \left(\frac{8}{D_f \sqrt{D_f}} \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) - \frac{96Nu}{20D_f^2} \right) \frac{\partial}{\partial t} \theta \Big|_{r=\frac{2}{\sqrt{D_f}}}. \quad (28)$$

Integrating (21) with respect to x , yield the following equation

$$\frac{\partial}{\partial t} \theta \Big|_{r=\frac{2}{\sqrt{D_f}}} + p \theta \Big|_{r=\frac{2}{\sqrt{D_f}}} = q. \quad (29)$$

Solving (29) gives

$$\theta|_{r=\frac{2}{\sqrt{D_f}}} = \frac{q}{p}(1-e^{-pt}). \quad (30)$$

Substituting equation (30) into equation (25) gives

$$\theta(r,t) = \frac{q}{p} \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) (1-e^{-pt}) - \frac{qNu\sqrt{D_f}}{4p} (1-e^{-pt}) r^2, \quad (31)$$

where

$$p = \frac{\left(\frac{12LeNu}{D_f} + \frac{8\delta\psi^\alpha\phi^\beta(e-2)}{D_f\sqrt{D_f}} \left(\frac{3Nu}{5\sqrt{D_f}} - \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) \right) + \delta_1\psi n(e-2) \left(\left(1 + \frac{Nu\sqrt{D_f}}{4} \right) - \frac{12Nu}{5D_f^2} \right) \right)}{\left(\frac{8}{D_f\sqrt{D_f}} \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) - \frac{96Nu}{20D_f^2} \right)},$$

$$q = \frac{\frac{8}{D_f\sqrt{D_f}} (\delta\psi^\alpha\phi^\beta - \delta_1\psi)}{\left(\frac{8}{D_f\sqrt{D_f}} \left(1 + \frac{Nu\sqrt{D_f}}{4} \right) - \frac{96Nu}{20D_f^2} \right)}$$

The computation was done on equations (31) using computer symbolic algebraic package MAPLE 2021 version.

4 Results and discussion

Simulation was carried out to show the impact of the model parameters by employing Polynomial Approximation Method (PAM) on the equation (7). The equation was solved over spatial co-ordinate and a specific period of time using Polynomial Approximation Method. The computation equation (31) was done using MAPLE 2021 version. The parameter values used in the simulations are

$$N_u = 4, 8, 12, D_f = 0.3, 0.5, 0.7, Le = 0.1, 0.5, 1.0, \phi = 2.75, \psi = 0.1, n = 2, \alpha = 2, \beta = 2, \delta_1 = 2, 4, \cdot$$

$$6, \delta = 0.4, 0.6, 0.8$$

Numerical simulation of Frank-Kamenetskii number, Lewis number, Nusselt number, condensed reactant diffusion coefficient and endothermicity number was conducted to see whether or not these parameters contribute significantly to the medium temperature.

Figure 1 depicts the graph of medium temperature $\theta(r,t)$ against spatial co-ordinate r and time t for different values of Frank-Kamenetskii number δ . It is observed that the temperature of the medium increases with time and decreases along spatial co-ordinate but this medium temperature decreases as Frank-Kamenetskii number increases.

Figure 2 displays the graph of medium temperature $\theta(r,t)$ against spatial co-ordinate r and time t for different values of Lewis number Le . It is observed that the temperature of medium increases

with time and decreases along spatial co-ordinate but this medium temperature increases as Lewis number increases.

Figure 3 shows the graph of medium temperature $\theta(r, t)$ against spatial co-ordinate r and time t for different values of Nusselt number Nu . It is observed that the temperature of medium increases with time and decreases along spatial co-ordinate but this medium temperature decreases as Nusselt number increases.

Figure 4 displays the graph of medium temperature $\theta(r, t)$ against spatial co-ordinate r and time t for different values of condensed reactant diffusion coefficient D_f . It is observed that the temperature of medium increases with time and decreases along spatial co-ordinate but this medium temperature increases as condensed reactant diffusion coefficient increases.

Figure 5 depicts the graph of medium temperature $\theta(r, t)$ against spatial co-ordinate r and time t for different values of endothermicity number δ_1 . It is observed that the temperature of medium increases with time and decreases along spatial co-ordinate but this medium temperature increases as endothermicity number increases.

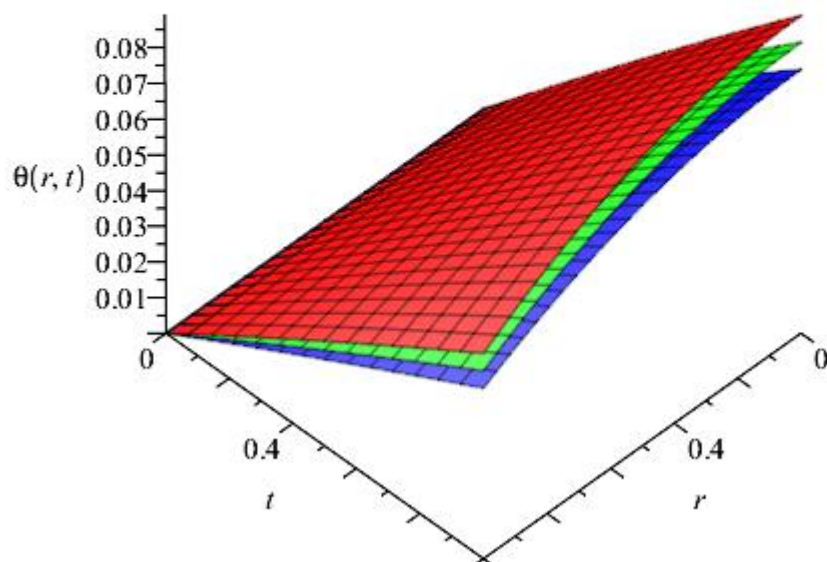


Figure 1: Effects of Frank-Kamenetskii number on medium temperature

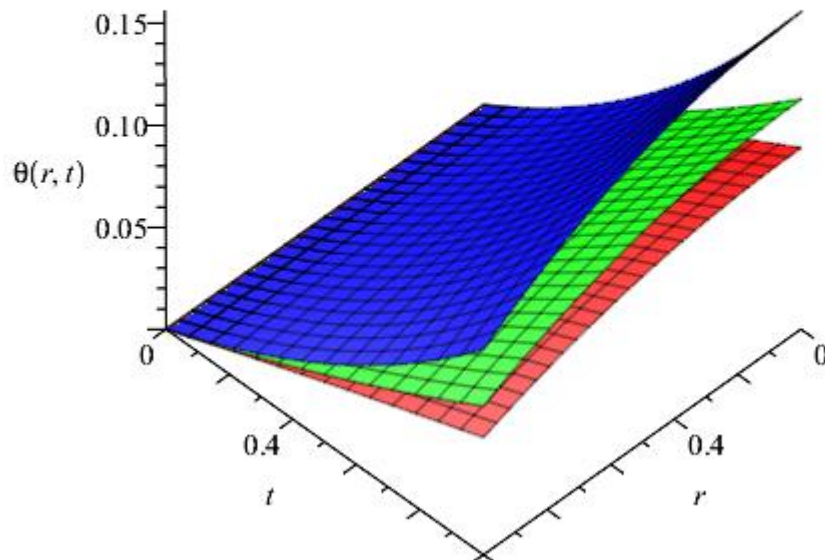


Figure 2: Effects of Lewis number on medium temperature

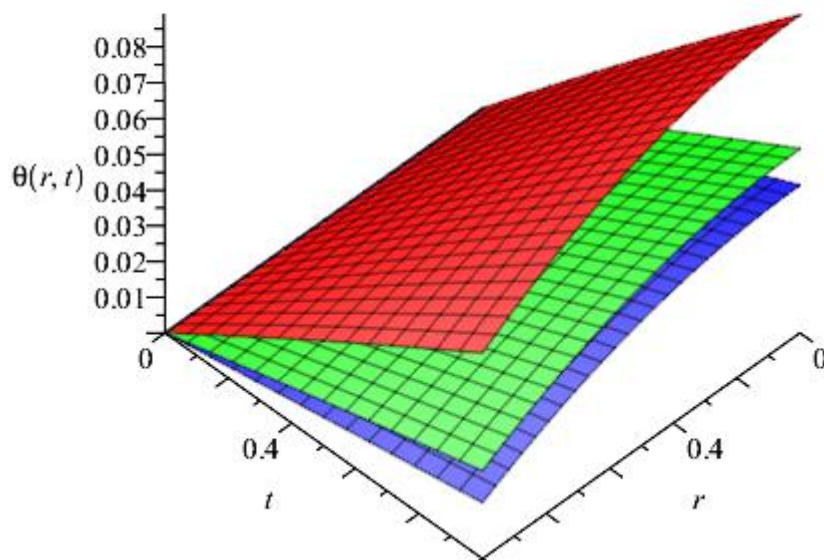


Figure 3: Effects of Nusselt number on medium temperature

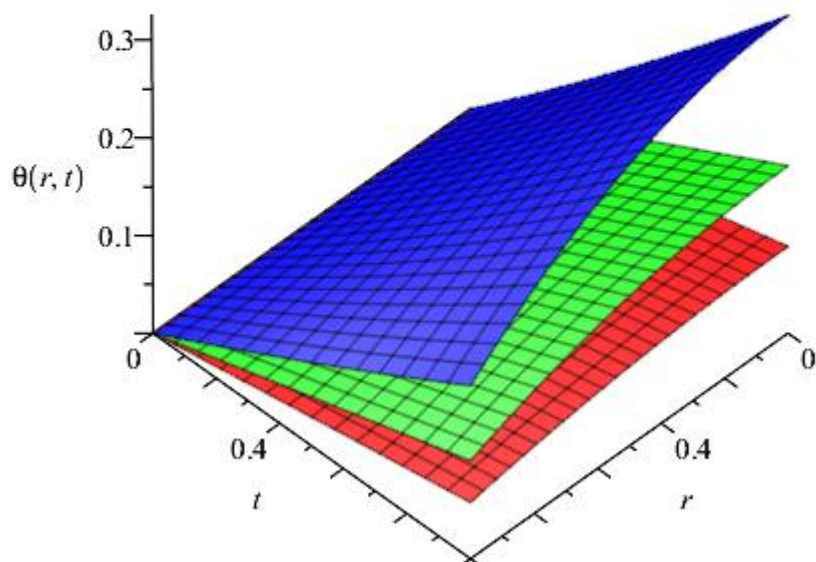


Figure 4: Effects of condensed reactant diffusion coefficient on medium temperature

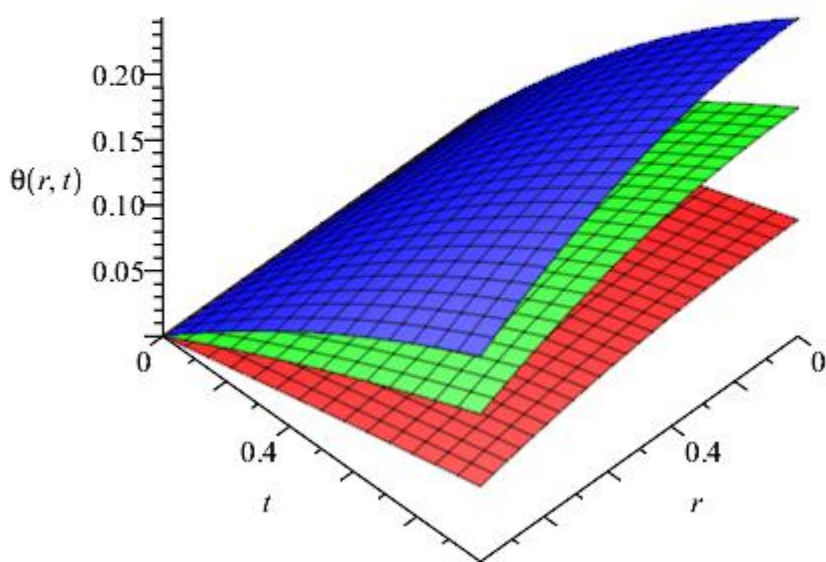


Figure 5: Effects of endothermicity number on medium temperature

It is worth pointing out that the effects observed in Figures 1 - 5, are important to guide insulation materials manufacturers so as to provide safety precautions during storage and usage.

5 Conclusion

For a well stirred reaction and in a high activation energy situation $\epsilon \rightarrow 0$, we have found that thermal runaway could still occur when the activation energy for reaction is the same as activation energy for vaporization, i.e. $n=1$. That is $\theta(t) \rightarrow \infty$ as $t \rightarrow \frac{1}{(\delta\psi^\alpha\phi^\beta - \delta_1\psi)}$. The existence of the unique solution of the problem implies that the problem represents a physical situation under specific conditions. In the presence of diffusion, the analytical simulations showed that increase in both Frank-Kamenetskii and Nusselt numbers reduced the medium temperature while increase in Lewis number, condensed reactant diffusion coefficient and endothermicity number enhanced the medium temperature. Therefore, the established conditions and the results obtained are not expected to guide manufacturers of insulation materials but provide safety precautions during storage and usage.

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Statistical analysis of academic performance among pupils in primary schools. a yardstick for an unbiased decision - making

D.M.O. Omebo*, A.Y. Emmanuel†, T.D. Ailobhio‡§, and G.I. Fanen‡**

Abstract

This paper is an extract of the research findings that was carried out in Ofu local government area of Kogi state in 2021 which was used to resolve the conflict that broke out as a result of the proposal to establish Government model Science College by the Kogi state Government. The bone of contention then was either to establish model boys' college or model girls' college in the local government. This necessitated the research to find out if performance depends on sex or not. An analysis of variance for categorical data (CATANOVA) was used in carrying out the test. It was discovered that performance depends on sex, and that male pupils performed better than the female pupils. Hence, Government boys model college was recommended. All the findings were based on the performance of male and female pupils in primary six using common entrance examination results for 2019 since there was no common entrance examination for 2020 due to covid-19 pandemic.

Keywords: sex; Chi- square; catanova; performance.

1 Introduction

The growth of statistics over time and its role in problem solving cannot be over emphasized. This has made itself felt in almost every phase of human endeavor. Statistics provides models and theories that are needed to study situations involving uncertainties. One of such problems that these models and theories can be employed is that which confronted Ofu Local government area of Kogi state in 2021.

* Corresponding Author. Department of Statistics, Federal University of Lafia, PMB 146, Lafia, Nigeria;
E-mail: damebo2002@yahoo.co.uk

† Department of Mathematics, Federal University of Lafia, PMB 146, Lafia, Nigeria;
E-mail: atanyiemmanuel@gmail.com

‡ Department of Statistics, Federal University of Lafia, PMB 146, Lafia, Nigeria;

§ E-mail: ailobhiodorcass@gmail.com

** E-mail: fanen.ibi@science.fulafia.edu.ng

The Kogi state government of Nigeria announced the proposal to establish a new single sex model college to be located in Ofu local government Area of the state. As usual both sexes began to give reasons why it should be boys model college or girls model college. All efforts to resolve this problem to no avail. This necessitated research to find out if performance is dependent on sex or not. This is because the sex that performed better will be able to feed the college with the desired quality and quantity of students needed by the new college.

There are three districts in Ofu Local Government. These include Ugwolawo, Igalaogba and Itobe districts. Data on performance was categorize into male and female.

This study is therefore aimed at finding out if performance between the males and females in the local government is the same or not. Hence, this study investigates the comparative academic performance between male and female pupils in primary six across the districts of Ofu local government area of Kogi state.

1.1 Aim and objectives of the study

- To ascertain if performance is dependent on sex
- To determine which of the sex should have the school
- To resolve the conflict already created by this agitation.

1.2 Nature and scope of study

There are 53 primary schools in Ofu Local Government Area of Kogi State out of which samples of 20 primary schools were taken. The primary schools in kogi state like their counterparts in other states are controlled by the state primary Education Board which is now called state universal basic Education Board (SUBEB) which in turn is controlled by the state ministry of education. It is this state ministry of education that provides an approved syllabus to be followed by the primary schools and at the end of the six-year programme, the pupils sit for common entrance examination which qualify them to go to secondary schools. The examination is made up of three papers. These are: Paper 1(English language), paper 2 (Mathematics) and Paper 3 (General paper). At the end of the examination, successful candidates are classified into five groups – Distinction(A), Good (B), Credit (C), Fair (D) and Pass (E). Similarly, unsuccessful candidates are awarded (F). All findings are based on the analysis of this examination results from the sampled schools from the various districts.

1.3 Sampling techniques

Out of the 53 primary schools in Ofu Local government Area, I sampled 20 primary schools according to the classification below.

Sampling distribution of the primary schools in Ofu local government area

Districts	Number of schools
Ugwolawo	9
Igalaogba	6
Itobe	5

The researcher used stratified random sampling techniques to represent the entire Ofu Local Government Area. The local Government has three districts so that communities were used as the

stratifying factor. Ofu Local Government Area comprises of eighteen communities, which is grouped into three districts forming the strata, from which simple random sampling was used. Ugwolawo district consists of Ugwolawo, Ogegume, Ojofa, Obagwu, Umomi, Akpagidigbo, Alome, Ikagbo, and Ugbogbo, while Igalaogba district is made up of Aloma, Igah, Ejule, Ogbonicha, Ogbulu and Ikeje. Finally, Itobe district comprises of Ojodu, Adumu, Aloji, Itobe and Ochadamu. Also, I used proportional allocation of sample size according to the weight (number of the communities that each group consists in terms of number of primary schools in each group).

2 Literature review

Even though many studies have been carried out to ascertain the dependence of performance on one factor or the other, majority of them if not all were carried out using the results from examinations such as first school leaving certificate (FSLCE), Senior school certificate examination (SSCE), University matriculation Examinations (UME) among others as their only source of data for the analysis which this study frown at due to gross examination malpractice usually associated with the conduct and marking of the scripts by the authorities concern.

In order to ascertain if boys are more brilliant than girls, [5] investigated into the performance of boys and girls in a sample of 32 primary schools Nsukka Educational Zone. From the report of his findings, he asserted that boys are more brilliant than girls all within primary school age.

Another research carried out by [1] on how the shift system affects the academic performance of primary school pupils in Enugu using qualitative data based on 1978 only, he came up with the following results: first, that the morning session pupils performed better than the afternoons. Secondly, that this disparity in performance was as a result of the fact that shift one had relatively more qualified and trained teachers than shift two. [12] carried out research on school location and academic performance of pupils in English language in primary schools. He reported that there is a significant difference in the academic performance of pupils in urban and rural primary schools in English language.

3 Methodological frameworks

The methodological framework for this study is a two-way analysis of variance for categorical data (CATANOVA).

A two-way Catanova with chi-square tests for interaction.

Consider a two-way experimental situation with a k -dimensional vector of responses $\{ (n_{ijk}) \mid K, i=1,2,\dots,I, j=1,2,\dots,J \}$ recorded in frequencies in the $(ij)^{\text{th}}$ plot. We assume a multinomial probability model, namely

$$\begin{aligned} \Pr(\{n_{ijk}\} \mid \{\pi_{ijk}\}) &= \frac{n!}{\prod_k \{n_{ijk}!\}} (\pi_{ijk})^{\prod_k n_{ijk}} \\ n_{ijk} &= 0, 1, \dots, n_{ij} \\ 0 &\leq \pi_{ijk} \leq 1 \\ N_{ij} &= \sum_k n_{ijk} \quad \text{is held fixed} \\ N_{ijk}, n_{i'j'k} &\text{ are independent } \forall i \neq i' \end{aligned}$$

The hypothesis to be tested are:

HR: $\prod_{ijk} = \prod_{jk}$, there is no row effect

HC: $\prod_{ijk} = \prod_{ik}$, there is no column effect

HRC: $\prod_{ijk} = \prod_{k}$, there is no row x column interaction.

The expectation of the sample estimate of $\prod_{ijk}$ is given by the linear model

$$E(\prod_{ijk}) = \mu_k + \tau_{ik} + \beta_{jk} + \lambda_{ijk},$$

where μ_k , β_{jk} and λ_{ijk} assume their usual meaning of constant, row effect, column effect and interaction respectively.

The hypothesis, HR, HC, and HRC will be seen to be respectively equivalent to

H_i: $\tau_{ik} = 0 \quad \forall i$, H_B: $\beta_{jk} = 0 \quad \forall j$ and $\lambda_{ijk} = 0 \quad \forall i, j$.

The parameters: $\prod_{ijk}$ may be considered fixed or random with probability density $h(\prod_{ijk})$, over $\{0,1\}$ depending on whether I and J are random or not, [13,14] Specially,

$g(\prod_{ijk})$ if I and J are random, $0 \leq \prod_{ijk} \leq 1$

$h(\prod_{ijk}) = g(\prod_{jk})$ if I is fixed and J random $0 \leq \prod_{jk} \leq 1$

$g(\prod_{ik})$ if I is random and J fixed, $0 \leq \prod_{ik} \leq 1$.

We shall now consider the sum of squares (SS) for $\{n_{ijk}\}_k$. This may be obtained, [6,7,8,9,10, 13] also [2,3], as the trace of the sample variance- covariance matrix. That is

$$SS(n_{ijk}) = \sum_k \text{var}(n_{ijk})$$

Upon further simplification, we have that

$$TSS = n - \sum_k n_{2..k}/n, \text{ as the total sum of squares}$$

$$BRSS = n - \sum_{ik} n_{2i.k}/n_i, \text{ as the between row sum of squares}$$

$$BCSS = n - \sum_{jk} n_{2.jk}/n_j, \text{ as the between column sum of squares}$$

$$WUSS = n - \sum_{ijk} n_{2ijk}/n_{ij}, \text{ as the within unit sum of squares,}$$

where

$$N = \sum_i n_i = \sum_j n_j = \sum_{ij} n_{ij}$$

$$RSS = TSS - BRSS = \sum_k n_{2..k}/n - \sum_k n_{2..k}/n$$

$$CSS = TSS - BCSS = n - \sum_{jk} n_{2.jk}/n_j - \sum_k n_{2..k}/n$$

$$NSS = BRSS + BCSS - TSS - WUSS$$

$$= n - \sum_{ijk} n_{2ijk}/n_{ij} - \sum_{ik} n_{2i.k}/n_i - \sum_{jk} n_{2.jk}/n_j + \sum_k n_{2..k}/n.$$

We may reject HR, HC and HRC respectively at specified α -level of error if

$$\chi^2_R \geq \chi^2_{(I-1)(J-1)(K-1)}(\alpha)$$

where

$$\chi^2_R = (K-1)(n-1)RSS / TSS$$

$$\chi^2_C = (K-1)(n-1)c_{SS} / TSS$$

$$\chi^2_{NT} = (K-1)(n-1)NSS / TSS$$

3.1 Materials and methods

Below is the data layout for a two – way analysis of variance for categorical data (CATANOVA).
 This was given by Table 1 [4]

	Districts	Ugwolawo								Igalaogba								Itobe							
i	Response s	A	B	C	D	E	F	T		A	B	C	D	E	F	T	A	B	C	D	E	F	T		
	Males	n ₁₁₁	n ₁₁₂	n ₁₁₃	n ₁₁₄	n ₁₁₅	n ₁₁₆	n ₁₁₇	n ₁₁₈	n ₁₁₉	n ₁₂₀	n ₁₂₁	n ₁₂₂	n ₁₂₃	n ₁₂₄	n ₁₂₅	n ₁₃₁	n ₁₃₂	n ₁₃₃	n ₁₃₄	n ₁₃₅	n ₁₃₆	n ₁₃₇		
	Females	n ₂₁₁	n ₂₁₂	n ₂₁₃	n ₂₁₄	n ₂₁₅	n ₂₁₆	n ₂₁₇	n ₂₁₈	n ₂₁₉	n ₂₂₀	n ₂₂₁	n ₂₂₂	n ₂₂₃	n ₂₂₄	n ₂₂₅	n ₂₃₁	n ₂₃₂	n ₂₃₃	n ₂₃₄	n ₂₃₅	n ₂₃₆	n ₂₃₇		
	n.jk	n _{.11}	n _{.12}	n _{.13}	n _{.14}	n _{.15}	n _{.16}	n _{.17}	n _{.18}	n _{.19}	n _{.20}	n _{.21}	n _{.22}	n _{.23}	n _{.24}	n _{.25}	n _{.31}	n _{.32}	n _{.33}	n _{.34}	n _{.35}	n _{.36}	n _{.37}		

Table 1. Data layout for two-way Catanavo

DISTRICTS

	Ugwolawo							Igalaogba							Itoke						T							
sex	A	B	C	D	E	F	ni 1.	A	B	C	D	E	F	ni 2.	A	B	C	D	E	F	n i3 .	ni. k						ni. .
ma le	1 3 9	62	58	1 0	5 5	8 3	40 7	71	75	4 5	2 4	3 6	52	30 3	10 7	5 2	5 3	5 3	4 5	7 5	3 3 7	3 1 7	18 9	1 5 6	3 9 6	1 3 6	2 1 0	10 47
fe ma le	8 0	51	48	1 1	5 0	8 5	32 5	39	48	2 9	3 7	3 9	51	24 3	43	2 0	2 6	6 3	3 0	6 4	1 8 9	1 6 2	11 9	1 0 3	5 4 9	1 1 9	2 0 0	75 7
To tal	2 1 9	11 3	10 6	2 1	1 0 5	1 6 8	73 2	11 0	12 3	7 4	6 1	7 5	10 3	54 6	15 0	7 2	7 9	1 1	7 5	1 3 9	5 2 6	4 7 9	30 8	2 5 9	9 3	2 5 5	4 1 0	18 04

Table 2: Two – way Catanova test on performance of male and female pupils of primary in Ofu local Government Area of Kogi State in common entrance examination for the year 2021

$$\begin{aligned}
 \text{Total sum of squares (TSS)} &= n - \sum_k n2..k/n \\
 &= 1804 - (479^2 + 308^2 + 259^2 + 93^2 + 255^2 + 410^2) / 1804 \\
 &= 1804 - 633160 / 1804 \\
 &= 1804 - 350.9756 \\
 \text{TSS} &= 1453.0244.
 \end{aligned}$$

$$\begin{aligned}
 \text{Between sex sum of squares (BSSS)} &= n - \sum_{ik} n2i.k/n i \\
 &= 1804 - (317^2 + 189^2 + 156^2 + 39^2 + 136^2 + 210^2) / 1047 + (162^2 + 119^2 + 103^2 \\
 &+ 54^2 + 119^2 + 200^2) / 757 \\
 &= 1804 - (214.5778 + 142.7886) \\
 &= 1804 - 357.3664 \\
 \text{BRSS} &= 1446.6336.
 \end{aligned}$$

$$\begin{aligned}
 \text{Between districts sum of squares (BDSS)} &= n - \sum_{jk} n2.jk/nj \\
 &= 1804 - (219^2 + 113^2 + 106^2 + 21^2 + 105^2 + 168^2) / 732 + (110^2 + 123^2 + 74^2 \\
 &+ 11^2 + 75^2 + 139^2) / 526 \\
 &= 1804 - (152.5355 + 96.4469 + 112.1521) \\
 &= 1804 - 361.1345 \\
 \text{BDSS} &= 1442.8655.
 \end{aligned}$$

$$\begin{aligned}
 \text{Row sum of squares (RSS)} &= \text{TSS} - \text{BDSS} \\
 &= 1453.0244 - 1446.6336 \\
 &= 6.3908 \\
 \text{RSS} &= 6.3908.
 \end{aligned}$$

$$\begin{aligned}\text{Column sum of squares (CSS)} &= \text{TSS} - \text{BDSS} \\ &= 1453.0244 - 1442.8655 \\ &= 10.1588.\end{aligned}$$

Source of variance	Degree of freedom	Sum of squares
Row or factor levels or sexes	1	6.3908
Districts	2	10.1588
Sex x districts	10	1.3334
Residual	1790	1435.1414
Total	1803	1453.0244

Table 3: Catanova table

Ho: Performance is independent of sex.

H1: Performance is dependent on sex.

$$\begin{aligned}\text{The test statistic} &= \chi^2_{\text{cal}} = k - 1(n-1)\text{RSS} / \text{TSS} \\ &= (6-1)1804 - 1) 6.3908 / 1453.0244 \\ &= 39.6504\end{aligned}$$

$$\chi^2_{\text{tab}} = \chi^2_{(1-1)(K-1)}^{(0.05)} = \chi^2_{(2-1)(6-1)}^{(0.05)} = 11.070$$

Decision Rule: Reject Ho if χ^2_{cal} is greater than χ^2_{tab}

Conclusion:

Since $\chi^2_{\text{cal}} = 39.6504 > \chi^2_{\text{tab}} = 11.070$, we reject Ho and conclude that performance depends on the sex.

To determine which sex perform better than the other, we obtain the proportions of students that passed and failed for each of the sex as follows:

Proportion of males that made grade A is:

$$P_{MA} = n_{MA} / N_m = 317 / 1047 = 0.30.$$

Proportion of females that made grade A is:

$$P_{FA} = n_{FA} / N_F = 162 / 757 = 0.21.$$

Proportion of males that made grade B is:

$$P_{MB} = n_{mb} / N_m = 189 / 1047 = 0.18.$$

Proportion of females that made grade B is

$$P_{FB} = n_{FB} / N_F = 119 / 757 = 0.16.$$

Proportion of males that made grade C is

$$P_{MC} = n_{MC}/N_M = 156/1047 = 0.15.$$

Proportion of females that made grade C is
 $P_{FC} = n_{FC}/N_F = 103/575 = 0.14.$

Proportion of males that made grade D is
 $P_{MD} = n_{MD}/N_M = 39/1047 = 0.04.$

Proportion of females that made grade D is
 $P_{FD} = n_{FD}/N_F = 54/757 = 0.07.$

Proportion of males that made grade E is
 $P_{ME} = n_{ME}/N_M = 136/1047 = 0.16.$

Proportion of males that made grade F is
 $P_{MF} = n_{MF}/N_M = 210/1047 = 0.20.$

Proportion of females that made grade F is
 $P_{FF} = n_{FF}/N_F = 200/757 = 0.26.$

The proportion of males that made grades A, B, and C are more than the females that made grades A, B, and C. Likewise, the proportion of males that made grade D, E, and F are less than that of the females that made grades D, E and F. We conclude that male pupils in the Local Government perform better than their female counterpart.

4 Conclusion

From the foregoing, it shows that performance depends on sex. This means that performance of any chosen pupil depends on sex of the pupil. From the result obtained, pupils from male pupils performed better than the females. The researcher therefore recommended that boys model science college should be established since more qualified males will be available to go the school

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Solution of substance abuse and domestic violence mathematical model using Homotopy perturbation method

E. Azuaba*, T. J. Oluwafemi^{†‡}, N. I. Akinwande[†], D. Bako[†], and J. Dayap[§]

Abstract

In this study, homotopy perturbation method was applied for the approximate solution of a mathematical model of substance abuse and domestic violence. The system of model equations developed was transformed into series of differential equations which enables the series to converge which gives the approximate solution required.

Keywords: substance abuse; domestic violence; homotopy perturbation method (hpm)

1 Introduction

Domestic Violence the violence occurring in the family or domestic unit, including, inter alia, physical and mental aggression, emotional and psychological abuse, rape and sexual abuse, incest, marital rape, or rape between regular or occasional partners and cohabitants, crimes committed in the name of honor, female genital and sexual mutilation, and other traditional practices harmful to women, such as forced marriages [3]. Intimate partner violence is a more specific term defining one of the most common forms of violence against women which refers to a pattern of assaultive and coercive behavior by an individual against her/his partner that may include physical injury, psychological abuse, sexual assault, progressive isolation, stalking, deprivation, intimidation, and reproductive coercion [10]. Domestic violence is now considered as a global health issue and a threat for women's health [5,10]. The worldwide prevalence of IPV among all ever partnered women was 30.0% [4]. 5.3 million cases of domestic violence among women 18 years of age and older is happening in the U.S., leading to about 2 million injuries and 1,400 deaths every year [10].

* Corresponding Author: Department of Mathematical Sciences, Bingham University Karu, Nassarawa State, Nigeria; E-mail: emmanuel.azuaba@binghamuni.edu.ng

[†]Department of Mathematics, Federal University of Technology, Minna, Nigeria

[‡]General Studies Department, Newgate College of Health Technology, Minna, Nigeria

[§]Department of Mathematics and Sciences, School of Arts and Sciences, University of San Jose-Recoletos, 6000 Cebu City, Philippines

Domestic violence is not only against women, but men have also been found to be abused by their partners [5]. One of the most common risks found in several cases of domestic violence is that the perpetrators were tested positive for drugs and/or were under the influence of alcohol, some studies found that there are strong correlations between alcohol addiction, drugs and marijuana during the act of violence [7,3,5].

Homotopy Perturbation Method (HPM) is a power series expansion method used in the solution of nonlinear partial differential equations which transforms the original non-linear differential equations into a series of linear differential equation [8,6]. [1] obtained the approximate solution of a deterministic mathematical model of zika virus using Homotopy Perturbation Method. [8] applied HPM in solving non-linear differential model equations. [6] solved a mathematical model of Ebola Virus disease transmission dynamics using HPM, the model is a coupled non-linear differential equation.[9] applied HPM to solve a system of non-linear ordinary differential equations used to model Rabies disease. The results obtained was compared with Runge-Kutta fourth order and nonstandard finite difference method and it shows to be a reliable method of solution.

Mathematical Model has been formulated to study dynamics of domestic violence [2]. In this work, a new mathematical model was formulated in cooperating violent individuals who are substance abuser.

2 Model formulation

$$\frac{dS}{dt} = \Lambda - \beta AS - \beta_1 VS - \mu S, \quad (1)$$

$$\frac{dA}{dt} = \beta AS + \beta_2 AV - (\phi_1 + \delta_1 + \varepsilon + \mu)A, \quad (2)$$

$$\frac{dV}{dt} = \beta_1 VS - (\kappa_1 + \phi_2 + \theta + \mu)V, \quad (3)$$

$$\frac{dV_a}{dt} = \beta_2 AV + \kappa_1 V - (\phi_3 + \theta + \mu)V_a, \quad (4)$$

$$\frac{dV_v}{dt} = \tau V_v + \alpha V V_v + \alpha b V_a V_v - (\phi_4 + \delta_2 + \mu)V_v, \quad (5)$$

$$\frac{dR}{dt} = \phi_1 A + \phi_2 V + \phi_3 V_a + \phi_4 V_v - \mu R, \quad (7)$$

Variables:

S = Susceptible Individuals

A = Substance Abusers

V = Violent Individuals

V_a = Violent Individuals who are substance Abusers

V_v = Victims of Domestic Violence

R = Recovered

Parameters:

β = Contact Rate

ϕ = Recovery/ Treatment rate

δ_1 = Substance abused induced death rate

δ_2 = Violence induced death rate

κ_1 = movement from V to V_a
 θ = Rate at which V and V_a are caught
 ε = rate at which substance abusers are apprehended
 τ = Victim recruitment
 α = contact that results into domestic violence
 b = parameter defined by increase in violence due to substance abuse

3 Method and materials

To illustrate the HPM, we consider the following non-linear differential equations given by

$$K(U) - f(r) = 0, r \in \Omega, \quad (8)$$

subject to the boundary conditions;

$$B\left(U, \frac{\partial U}{\partial n}\right) = 0, r \in \Gamma, \quad (9)$$

where K is the general differential operator, B is the boundary operator, $f(r)$ is the analytical function and Γ is the boundary of the domain Ω . The operator can be divided into two major parts L and N being the linear and non-linear component respectively, where equations (8) can be re-written as

$$L(U) + N(U) - f(r) = 0, r \in \Omega. \quad (10)$$

The HPM is structured as follows:

$$H(v, p) = (1 - p)[L(v) - L(u_0)] + p[A(v) - f(r)] = 0, \quad (11)$$

where

$$v(r, p): \Omega \in [0, 1] \rightarrow R,$$

and $p \in [0, 1]$ is an embedding parameter and u_0 is the initial approximation that satisfies the boundary conditions. The solution to equation (11) can be assumed as power series in p as follows:

$$V = V_0 + pV_1 + p^2V_2 + \dots \quad (12)$$

Setting $p = 1$, the approximate solution of (1), therefore can be obtained as

$$U = \lim_{p \rightarrow 1} V = V_0 + pV_1 + p^2V_2 + \dots \quad (13)$$

4 Solution of the model equation

$$\frac{dS}{dt} - \Lambda + \beta AS + \beta_1 VS + \mu S = 0, \quad (14)$$

$$\frac{dA}{dt} - \beta AS - \beta_2 AV + (\phi_1 + \delta_1 + \varepsilon + \mu)A = 0, \quad (15)$$

$$\frac{dV}{dt} - \beta_1 VS + (\kappa_1 + \phi_2 + \theta + \mu)V = 0, \quad (16)$$

$$\frac{dV_a}{dt} - \beta_2 AV - \kappa_1 V + (\phi_3 + \theta + \mu)V_a = 0, \quad (17)$$

$$\frac{dV_v}{dt} - \tau V_v - \alpha VV_v - \alpha b V_a V_v + (\phi_4 + \delta_2 + \mu)V_v = 0, \quad (18)$$

$$\frac{dR}{dt} - \phi_1 A - \phi_2 V - \phi_3 V_a - \phi_4 V_v + \mu R = 0, \quad (19)$$

with initial conditions

$$S(0) = S_0; A(0) = A_0; V(0) = V_0; V_a(0) = V_{a_0}; V_v(0) = V_{v_0}; R(0) = R_0.$$

Let

$$S = a_0 + pa_1 + p^2 a_2 + \dots \quad (20)$$

$$A = b_0 + pb_1 + p^2 b_2 + \dots \quad (21)$$

$$V = c_0 + pc_1 + p^2 c_2 + \dots \quad (22)$$

$$V_a = d_0 + pd_1 + p^2 d_2 + \dots \quad (23)$$

$$V_v = e_0 + pe_1 + p^2 e_2 + \dots \quad (24)$$

$$R = x_0 + px_1 + p^2 x_2 + \dots \quad (25)$$

Applying HPM to equation (14) – (19) using (20) – (25) we have the followings:

From equation (14)

$$(1 - p) \frac{dS}{dt} + p \left[\frac{dS}{dt} - \Lambda + \beta AS + \beta_1 VS + \mu S \right] = 0. \quad (26)$$

Substituting (20) – (25) into (26) and simplifying we have

$$(a'_0 + pa'_1 + p^2 a'_2 + \dots) + p(a_0 + pa_1 + p^2 a_2 + \dots)(\beta(b_0 + pb_1 + p^2 b_2 + \dots) + \beta_1(c_0 + pc_1 + p^2 c_2 + \dots) + \mu) - \Lambda = 0. \quad (27)$$

Comparing the coefficient we get

$$p^0: a'_0 = 0 \quad (28)$$

$$p^1: a'_1 + \beta a_0 b_0 + \beta_1 a_0 c_0 + \mu a_0 = 0 \quad (29)$$

$$p^2: a'_2 + \beta(a_0 b_1 + a_1 b_0) + \beta_1(a_1 c_0 + a_0 c_1) = 0. \quad (30)$$

From (15),

$$(1 - p) \frac{dA}{dt} + p \left[\frac{dA}{dt} - \beta AS - \beta_2 AV + (\phi_1 + \delta_1 + \varepsilon + \mu)A \right] = 0. \quad (31)$$

Substituting (20) – (25) into (31) and simplifying we have

$$(b'_0 + pb'_1 + p^2b'_2 + \dots) + \omega_1 p(b_0 + pb_1 + p^2b_2 + \dots) - p(b_0 + pb_1 + p^2b_2 + \dots)(\beta(a_0 + pa_1 + p^2a_2 + \dots) + \beta_2(c_0 + pc_1 + p^2c_2 + \dots)) = 0. \quad (32)$$

Comparing

$$p^0: b'_0 = 0 \quad (33)$$

$$p^1: b'_1 + \omega_1 b_0 - \beta a_0 b_0 - \beta_2 b_0 c_0 = 0 \quad (34)$$

$$p^2: b'_2 + \omega_1 b_1 - \beta(a_0 b_1 + a_1 b_0) - \beta_2(b_1 c_0 + b_0 c_1) = 0. \quad (35)$$

From (16),

$$(1 - p) \frac{dV}{dt} + p \left[\frac{dV}{dt} - \beta_1 VS + (\kappa_1 + \phi_2 + \theta + \mu)V \right] = 0. \quad (36)$$

Substituting (20) – (25) into (36) and simplifying we have

$$(c'_0 + pc'_1 + p^2c'_2 + \dots) + p(c_0 + pc_1 + p^2c_2 + \dots)(\omega_2 - \beta_1(a_0 + pa_1 + p^2a_2 + \dots)) = 0 \quad (37)$$

Comparing

$$p^0: c'_0 = 0, \quad (38)$$

$$p^1: c'_1 + \omega_2 c_0 - \beta_1 a_0 c_0 = 0, \quad (39)$$

$$p^2: c'_2 + \omega_2 c_1 - \beta_1(a_1 c_0 + a_0 c_1) = 0. \quad (40)$$

From (17),

$$(1 - p) \frac{dV_a}{dt} + p \left[\frac{dV_a}{dt} - \beta_2 AV - \kappa_1 V + (\phi_3 + \theta + \mu)V_a \right] = 0. \quad (41)$$

Substituting (20) – (25) into (41) and simplifying we have

$$(d'_0 + pd'_1 + p^2d'_2 + \dots) + p\omega_3(d_0 + pd_1 + p^2d_2 + \dots) - p(c_0 + pc_1 + p^2c_2 + \dots)(\kappa_1 + \beta_2(b_0 + pb_1 + p^2b_2 + \dots)) = 0. \quad (42)$$

Comparing

$$p^0: d'_0 = 0, \quad (43)$$

$$p^1: d'_1 + \omega_3 d_0 - \beta_2 b_0 c_0 - \kappa_1 c_0 = 0, \quad (44)$$

$$p^2: d'_2 + \omega_3 d_1 - \beta_2 (b_1 c_0 + b_0 c_1) - \kappa_1 c_1 = 0. \quad (45)$$

From (18),

$$(1-p) \frac{dV_v}{dt} + p \left[\frac{dV_v}{dt} - \tau V_v - \alpha V V_v - \alpha b V_a V_v + (\phi_4 + \delta_2 + \mu) V_v \right] = 0. \quad (46)$$

Substituting (20) – (25) into (46) and simplifying we have

$$(e'_0 + p e'_1 + p^2 e'_2 + \dots) + p(e_0 + p e_1 + p^2 e_2 + \dots)(\omega_4 - \tau - \alpha - \alpha b(d_0 + p d_1 + p^2 d_2 + \dots)) = 0, \quad (47)$$

Comparing

$$p^0: e'_0 = 0, \quad (48)$$

$$p^1: e'_1 + \omega_4 e_0 - \tau e_0 - \alpha e_0 - \alpha b e_0 d_0 = 0, \quad (49)$$

$$p^2: e'_2 + \omega_4 e_1 - \tau e_1 - \alpha e_1 - \alpha b(e_0 d_1 + e_1 d_0) = 0, \quad (50)$$

From (19),

$$(1-p) \frac{dR}{dt} + p \left[\frac{dR}{dt} - \phi_1 A - \phi_2 V - \phi_3 V_a - \phi_4 V_v + \mu R \right] = 0. \quad (51)$$

Substituting (20) – (25) into (51) and simplifying we have

$$(x'_0 + p x'_1 + p^2 x'_2 + \dots) + p\mu(x_0 + p x_1 + p^2 x_2 + \dots) - p\phi_1(b_0 + p b_1 + p^2 b_2 + \dots) - p\phi_2(c_0 + p c_1 + p^2 c_2 + \dots) - p\phi_3(d_0 + p d_1 + p^2 d_2 + \dots) - p\phi_4(e_0 + p e_1 + p^2 e_2 + \dots) = 0 \quad (52)$$

Comparing

$$p^0: x'_0 = 0, \quad (53)$$

$$p^1: x'_1 + \mu x_0 - \phi_1 b_0 - \phi_2 c_0 - \phi_3 d_0 - \phi_4 e_0 = 0, \quad (54)$$

$$p^2: x'_2 + \mu x_1 - \phi_1 b_1 - \phi_2 c_1 - \phi_3 d_1 - \phi_4 e_1 = 0. \quad (55)$$

Given that

$$\omega_1 = (\phi_1 + \delta_1 + \varepsilon + \mu); \omega_2 = (\kappa_1 + \phi_2 + \theta + \mu); \omega_3 = (\phi_3 + \theta + \mu); \omega_4 = (\phi_4 + \delta_2 + \mu). \quad (56)$$

From (28),

$$a'_0 = 0. \quad (57)$$

Implying,

$$a_0 = S_0, \quad (58)$$

so that

$$b_0 = A_0, \quad (59)$$

$$c_0 = V_0, \quad (60)$$

$$d_0 = V_{a_0}, \quad (61)$$

$$e_0 = V_{v_0}, \quad (62)$$

$$x_0 = R_0. \quad (63)$$

From (29),(34),(39),(44),(49) and (54) integrating we have

$$a_1 = (-S_0(\beta A_0 + \beta_1 V_0 + \mu))t, \quad (64)$$

$$b_1 = (A_0(\beta S_0 + \beta_2 V_0 - \omega_1))t, \quad (65)$$

$$c_1 = (V_0(\beta_1 S_0 - \omega_2))t, \quad (66)$$

$$d_1 = (\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0})t, \quad (67)$$

$$e_1 = (V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4))t, \quad (68)$$

$$x_1 = (\phi_1 A_0 + \phi_2 V_0 + \phi_3 V_{a_0} + \phi_4 V_{v_0} - \mu R_0)t. \quad (69)$$

Solving (30),(35),(40),(45),(50) and (55) integrating and substituting (64) – (69) we have

$$a_2 = [-(\beta(S_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) - A_0 S_0(\beta A_0 + \beta_1 V_0 + \mu)) + \beta_1(S_0 V_0(\beta_1 S_0 - \omega_2) - V_0 S_0(\beta A_0 + \beta_1 V_0 + \mu)))] \frac{t^2}{2} \quad (70)$$

$$b_2 = [\beta(S_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) - A_0 S_0(\beta A_0 + \beta_1 V_0 + \mu)) - \omega_1 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + \beta_2(V_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + A_0 V_0(\beta_1 S_0 - \omega_2))] \frac{t^2}{2} \quad (71)$$

$$c_2 = [\beta_1(S_0 V_0(\beta_1 S_0 - \omega_2) - S_0 V_0(\beta A_0 + \beta_1 V_0 + \mu)) - \omega_2 V_0(\beta_1 S_0 - \omega_2)] \frac{t^2}{2} \quad (72)$$

$$d_2 = [\beta_2(V_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + A_0 V_0(\beta_1 S_0 - \omega_2)) + \kappa_1 V_0(\beta_1 S_0 - \omega_2) - \omega_3(\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0})] \frac{t^2}{2} \quad (73)$$

$$e_2 = [V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4)(\tau + \alpha + \alpha b V_{a_0}) + \alpha b V_{v_0}(\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0}) - \omega_4 V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4)] \frac{t^2}{2} \quad (74)$$

$$x_2 = [\phi_1 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + \phi_2 V_0(\beta_1 S_0 - \omega_2) + \phi_3(\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0}) + \phi_4 V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4) - \mu(\phi_1 A_0 + \phi_2 V_0 + \phi_3 V_{a_0} + \phi_4 V_{v_0} - \mu R_0)] \frac{t^2}{2}. \quad (75)$$

Substituting (58) – (75) into (20) – (25) and setting $p = 1$ we have

$$S(t) = S_0 + (-S_0(\beta A_0 + \beta_1 V_0 + \mu))t + [-(\beta(S_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) - A_0 S_0(\beta A_0 + \beta_1 V_0 + \mu)) + \beta_1(S_0 V_0(\beta_1 S_0 - \omega_2) - V_0 S_0(\beta A_0 + \beta_1 V_0 + \mu))] \frac{t^2}{2} \quad (76)$$

$$A(t) = A_0 + (A_0(\beta S_0 + \beta_2 V_0 - \omega_1))t + [\beta(S_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) - A_0 S_0(\beta A_0 + \beta_1 V_0 + \mu)) - \omega_1 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + \beta_2(V_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + A_0 V_0(\beta_1 S_0 - \omega_2))] \frac{t^2}{2} \quad (77)$$

$$V(t) = V_0 + (V_0(\beta_1 S_0 - \omega_2))t + [\beta_1(S_0 V_0(\beta_1 S_0 - \omega_2) - S_0 V_0(\beta A_0 + \beta_1 V_0 + \mu)) - \omega_2 V_0(\beta_1 S_0 - \omega_2)] \frac{t^2}{2} \quad (78)$$

$$V_a(t) = V_{a_0} + (\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0})t + [\beta_2(V_0 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + A_0 V_0(\beta_1 S_0 - \omega_2)) + \kappa_1 V_0(\beta_1 S_0 - \omega_2) - \omega_3(\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0})] \frac{t^2}{2} \quad (79)$$

$$V_v = V_{v_0} + (V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4))t + [V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4)(\tau + \alpha + \alpha b V_{a_0}) + \alpha b V_{v_0}(\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0}) - \omega_4 V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4)] \frac{t^2}{2} \quad (80)$$

$$R = R_0 + (V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4))t + [\phi_1 A_0(\beta S_0 + \beta_2 V_0 - \omega_1) + \phi_2 V_0(\beta_1 S_0 - \omega_2) + \phi_3(\beta_2 A_0 V_0 - \kappa_1 V_0 - \omega_3 V_{a_0}) + \phi_4 V_{v_0}(\tau + \alpha + \alpha b V_{a_0} - \omega_4) - \mu(\phi_1 A_0 + \phi_2 V_0 + \phi_3 V_{a_0} + \phi_4 V_{v_0} - \mu R_0)] \frac{t^2}{2} \quad (81)$$

Equation (76) – (81) are the general solutions to the model (1) – (7).

5 Conclusion

In this article a semi-analytical approach that is Homotopy Perturbation Method (HPM) was applied to obtain the general solution of a substance abuse and domestic violence mathematical model. The model equations were transformed into series form so as to convey faster. We concluded that the Homotopy perturbation method is a powerful technique to solve nonlinear differential equations.

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Application of stochastic model in estimation of stock return rates in capital market investments

I.U. Amadi* and O.T. Okpoye†

Abstract

The rigidity of financial assets lies on capital investments which geared towards bringing returns to the investors. Therefore, this paper studied the problem of system of Stochastic Differential Equations (SDE) of time-varying investments of Dangote cement PLC where multiplicative inverse effects, additive effects, additive inverse effects and multiplicative effects were used as key parameter in the model to obtain stock rate of returns. The problem was solved analytical by adopting Ito's theorem which gave four different investment solutions. In addition, the validity of analytical solutions was clearly confirmed graphically and discussed consequently.

Keywords: stock price; stochastic analysis; asset price; return rates and SDE

1 Introduction

In capital investments monies are been invested in a business such that the return rates will be appropriately utilized to cover day-to-day trading activities expenses. An investor may need additional capital assets; in order to improve in its trading events. So capital investments are established on the basis of: to acquire additional capital assets for expansion which enables the the business to increase in unit production, create new ideas on products or even add value to the business and to explore on technological advancements in order to increase efficiency and reduce costs and to replace worn-out assets. In absence of capital investments, trading business will definitely have a hard time getting off the ground.

Nevertheless, the effect of stock market operations and performance has enlarged significantly in the development of financial of markets as investors gain their normal returns to earn a living. However, stock trading is normally known in making high returns. Return on investment for capital market is a measure to properly evaluate gain of an investment. It is a major parameter often used by

*Corresponding Author. Department of Mathematics/Statistics Captain Elechi Amadi Polytechnic, Rumuola, Port Harcourt, Nigeria; E-mail:amadiuchenna@gmail.com

†Department of Mathematics/Statistics, Ignatius Ajuru University of Education, Rumuolumeni, Port Harcourt, Nigeria; E-mail:Tariokpoye@gmail.com

a trader or investors to regulate profitability of expenditure [1]. In stock market the dynamics of stock prices are reflected by uncertain movement of their value over time. That is why it better to properly understand the type of physical quantities to model in order to obtain realistic results of stock variables.

For instance [2] worked on stochastic model of the fluctuation of stock market price. Here conditions for determining the equilibrium price, sufficient conditions for dynamic stability and convergence to equilibrium of the growth rate of the value function of shares. In another dimension, [3] considered a stochastic model of price changes at the floor of stock market. In their research the equilibrium price and the market growth rate of shares were determined. See [4] for considerable extensions and constrains subsequently in this particular area of study. [5] Investigated the stochastic analyses of Markov chain in finite states. The transition matrix replicated the use of 3-states transition probability matrix which enables them to proffer precise condition of obtaining expected mean rate of return of each stock.

In similar manner, [6] examined stochastic analysis of stock market expected returns and Growth-rates. The precise conditions for obtaining the drifts, volatilities and Growth-rates of four different stocks were also considered. In the work of [7] the problem of stock price fluctuations using stochastic differential equations, principal component analysis and KS goodness of fit test were studied. The analytical solutions were detailed; the computational and graphical results were presented and discussed respectively; as if it that was not enough, [2] studied the stochastic analysis of stock market expected returns for investors. The detailed conditions for obtaining the drifts, volatilities and variances of four different stocks were considered. [8] Considered a combination of deterministic and stochastic systems with its random parameters in the model. The analytical solution to the proposed model is presented in detail which determined the insurance quantities or variables

However, [9] considered the unstable nature of stock market forces using proposed differential equation model. In the work of [10] studied stability analysis of stochastic model of price change at the floor of a stock market. In their research précised conditions are obtained which determines the equilibrium price and growth rate of stock shares.

The work in [11] considered stochastic analysis of the behavior of stock prices. Results reveal that the proposed model is efficient for the prediction of stock prices. In the same vein, [12] studied the stochastic model of some selected stocks in the Nigerian Stock Exchange (NSE), in their research the drift and volatility coefficients for the stochastic differential equations were determined.[13], built the geometric Brownian Motion and studied the accuracy of the model with detailed analysis of simulated data.[14] Studied the solution of differential equations and stochastic differential equations of time-varying investment returns; where precise conditions are obtained which govern stock return rate through multiplicative and multiplicative inverse trends series.

This paper, considered four systems of stochastic differential equations in time varying investments of Dangote cement PLC whose rate of returns is assumed to follow: multiplicative inverse effects, additive effects, additive inverse effects and multiplicative effects; all having quadratic functions over time. These investment equations were solved analytically independently by adopting Ito's theorem and four solutions were obtained. To verify and to test for the validity of the analytical solutions, we used closing stock prices of Dangote cement PLC and estimate other

relevant parameter values in the proposed solutions; this way the effects of rate of returns were displayed.

This paper is aimed at solving an accurate analytical solution of time independent equations by stating precise conditions of predicting stock returns in Dangote cement PLC which was not considered by previous efforts. This paper extends the works of [1] by solving the pricing effects of stock variables as quadratic functions in SDE domain.

This paper is arranged in following manners: Section 2.1 presents the mathematical formulation of the problem, method of solution is seen in Subsection 2.1.1, Results are seen in Section 3.1, while the discussion of results is presented in Section 3.1.1 and paper is concluded in Section 4.1.

2 Mathematical formulation of the problem

Here we Considered time varying investment of Dangote cement PLC through closing stock prices of 2018-2021 which said to have a complete probability space $(\Omega, \mathcal{F}, \mathbb{P})$ endowed with a Wiener process such that a finite time investment horizon $T > 0$. Investors are always geared towards maximizing investment returns. Therefore, we assumed the company's rate of returns to follow multiplicative inverse effects, additive effects, additive inverse effects and multiplicative effects; all having quadratic functions over time. Hence this rate of return is defined in the system of stochastic differential equations of (2- 5) below:

Thus the stochastic process describing the process is of the form:

$$dS(t) = \theta dt + \sigma dZ^{(1)}(t), \quad (1)$$

where θ is an expected rate of returns on stock, σ is the volatility of the stock, dt is the relative change in the price during the period of time and $Z^{(1)}$ is a Wiener process.

$$dS_{K_1}(t) = \left\{ (\theta_1 \theta_2)^{-1} \right\}^2 S_{K_1}(t) dt + \sigma S_{K_1}(t) dZ^{(1)}(t), \quad (2)$$

$$dS_{K_2}(t) = (\theta_1 + \theta_2)^2 S_{K_2}(t) dt + \sigma S_{K_2}(t) dZ^{(2)}(t), \quad (3)$$

$$dS_{K_3}(t) = \left\{ (\theta_1 + \theta_2)^{-1} \right\}^2 S_{K_3}(t) dt + \sigma S_{K_3}(t) dZ^{(3)}(t), \quad (4)$$

$$dS_{K_4}(t) = (\theta_1 \theta_2)^2 S_{K_4}(t) dt + \beta S_{K_4}(t) dZ^{(4)}(t), \quad (5)$$

where $S_{K_1}(t), S_{K_2}(t), S_{K_3}(t)$ and $S_{K_4}(t)$ are underlying stocks with the following initial conditions:

$$S_{K_1}(0) = S_{0(K_1)}, t > 0 \quad (6)$$

$$S_{K_2}(0) = S_{0(K_2)}, t > 0 \quad (7)$$

$$S_{K_3}(0) = S_{0(K_3)}, t > 0 \quad (8)$$

$$S_{K_4}(0) = S_{0(K_4)}, t > 0. \quad (9)$$

Though, the price evolution of a risky assets are usually modeled as the trajectory of a risky assets that are usually of a diffusion process defined on some underlying probability space, with the geometric Brownian motion the paramount tool used as the established reference model [13].

Theorem 1.1: (Ito's formula) Let $(\Omega, \beta, \mu, F(\beta))$ be a filtered probability space $X = \{X, t \geq 0\}$ be an adaptive stochastic process on $(\Omega, \beta, \alpha, F(\beta))$ processing a quadratic variation (X) with SDE defined as:

$$dX(t) = g(t, X(t))dt + f(t, X(t))dW(t)$$

$$t \in \mathfrak{R} \text{ and for } u = u(t, X(t)) \in C^{1 \times 2}(\Pi \times \square)$$

$$du(t, X(t)) = \left\{ \frac{\partial u}{\partial t} + g \frac{\partial u}{\partial x} + \frac{1}{2} f^2 \frac{\partial^2 u}{\partial x^2} \right\} d\tau + f \frac{\partial u}{\partial x} dW(t).$$

2.1 Method of solution

The investment equation (2)-(5) consist of a system of stochastic differential equations and whose solutions are not trivial. We applying Theorem 1.1 solving for $S_{K_1}(t)$, $S_{K_2}(t)$, $S_{K_3}(t)$, and $S_{K_4}(t)$ To grab this problem we note that

$$S_{K_1}(t), S_{K_2}(t), S_{K_3}(t) \text{ and } S_{K_4}(t) < \infty \text{ for all } S \in [0,1).$$

From (2), Thus, generalizing and considering a function $f(S_{K_1}(t), t)$, hence it has to do with partial derivatives. Expansion of $f(S_{K_1}(t), dS_{K_1}(t), t + dt)$ in a Taylor series about $(S_{K_1}(t), t)$ gives

$$df = \frac{\partial f}{\partial S_{K_1}(t)} dS_{K_1}(t) + \frac{\partial f}{\partial t} dt + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_1}^2(t)} dS_{K_1}^2(t) + \dots \quad (10)$$

Substituting (2) in (10) gives

$$df = \frac{\partial f}{\partial S_{K_1}(t)} \left\{ \sigma S_{K_1}(t) dZ^{(1)}(t) + \left\{ (\theta_1 \theta_2)^{-1} \right\}^2 S_{K_1}(t) dt \right\} + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_1}^2(t)} dS_{K_1}^2(t) dS_{K_1}^2(t)$$

$$df = \sigma S_{K_1}(t) \frac{\partial f}{\partial S_{K_1}(t)} dZ^{(1)}(t) + \left(\left\{ (\theta_1 \theta_2)^{-1} \right\}^2 S_{K_1}(t) \frac{\partial f}{\partial S_{K_1}(t)} + \frac{1}{2} \sigma^2 S_{K_1}^2(t) \frac{\partial^2 f}{\partial S_{K_1}^2(t)} dS_{K_1}^2(t) \right) dt.$$

Now considering the SDE in (2).

Let $f(S_{K_1}(t)) = \ln S_{K_1}(t)$, the partial derivatives becomes

$$\frac{\partial f}{\partial S_{K_1}(t)} = \frac{1}{S_{K_1}(t)}, \frac{\partial^2 f}{\partial S_{K_1}^2(t)} = -\frac{1}{S_{K_1}^2(t)}, \frac{\partial f}{\partial t} = 0. \quad (11)$$

Following Theorem 1.1(Ito's), substituting () and simplifying gives

$$df = \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} dt + \sigma dZ^{(1)}. \quad (12)$$

Since the RHS of (12) is independent of $f(S_{K_1}(t))$, the stochastic is computed as follows:

$$\begin{aligned} f(S_{K_1}(t)) &= f_0 + \int_0^t \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} dt + \int_0^t \sigma dZ^{(1)}(t) \\ &= f_0 + \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(1)}(t), \text{ Since } f(S_{K_1}(t)) = \ln S_{K_1}(t) \text{ a found solution for } S_{K_1}(t) \text{ becomes} \\ \ln S_{K_1}(t) &= \ln S_{0(K_1)} + \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(1)}(t), \ln S_{K_1}(t) - \ln S_{0(K_1)} = \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(1)}(t) \\ \ln \left(\frac{S_{K_1}(t)}{S_{0(K_1)}} \right) &= \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(1)}(t) \\ S_{K_1}(t) &= S_{0(K_1)} \exp \left\{ \left((\theta_1 \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(1)}(t) \end{aligned} \quad (13)$$

This is the complete solution of investment equation whose rate of returns and asset price valuation is multiplicative inverse effects with a quadratic function.

From (3), thus, generalizing and considering a function $f(S_{K_2}(t), t)$, hence it has to do with partial derivatives. Expansion of $f(S_{K_2}(t), dS_{K_2}(t), t + dt)$ in a Taylor series about $(S_{K_2}(t), t)$ gives

$$df = \frac{\partial f}{\partial S_{K_2}(t)} dS_{K_2}(t) + \frac{\partial f}{\partial t} dt + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_2}^2(t)} dS_{K_2}^2(t) + \dots \quad (14)$$

Substituting (3) in (14) gives

$$df = \frac{\partial f}{\partial S_{K_2}(t)} \left\{ \sigma S_{K_2}(t) dZ^{(2)}(t) + (\theta_1 + \theta_2)^2 S_{K_2}(t) dt \right\} + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_2}^2(t)} dS_{K_2}^2(t) dS_{K_2}^2(t)$$

$$df = \sigma S_{K_2}(t) \frac{\partial f}{\partial S_{K_2}(t)} dZ^{(2)}(t) + \left\{ (\theta_1 + \theta_2)^2 S_{K_2}(t) \frac{\partial f}{\partial S_{K_2}(t)} + \frac{1}{2} \sigma^2 S_{K_2}^2(t) \frac{\partial^2 f}{\partial S_{K_2}^2(t)} dS_{K_2}^2(t) \right\} dt.$$

Now considering the SDE in (3).

Let $f(S_{K_2}(t)) = \ln S_{K_2}(t)$, the partial derivatives becomes

$$\frac{\partial f}{\partial S_{K_2}(t)} = \frac{1}{S_{K_2}(t)}, \quad \frac{\partial^2 f}{\partial S_{K_2}^2(t)} = -\frac{1}{S_{K_2}^2(t)}, \quad \frac{\partial f}{\partial t} = 0. \quad (15)$$

Following Theorem 1.1 (Ito's), substituting (15) and simplifying gives

$$df = \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} dt + \sigma dZ^{(2)}. \quad (16)$$

Since the RHS of (16) is independent of $f(S_{K_2}(t))$, the stochastic is computed as follows:

$$f(S_{K_2}(t)) = f_0 + \int_0^t \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} dt + \int_0^t \sigma dZ^{(2)}(t)$$

$$= f_0 + \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t), \text{ Since } f(S_{K_2}(t)) = \ln S_{K_2}(t) \text{ a found solution for } S_{K_2}(t) \text{ becomes}$$

$$\ln S_{K_2}(t) = \ln S_{0(K_2)} + \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t), \ln S_{K_2}(t) - \ln S_{0(K_2)} = \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t)$$

$$\ln \left(\frac{S_{K_2}(t)}{S_{0(K_2)}} \right) = \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t)$$

$$S_{K_2}(t) = S_{0(K_2)} \exp \left\{ \left\{ (\theta_1 + \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t) \right\}. \quad (17)$$

This is the complete solution of investment equation whose rate of returns and asset price valuation is additive effects with a quadratic function.

From (4), thus, generalizing and considering a function $f(S_{K_3}(t), t)$, hence it has to do with partial derivatives. Expansion of $f(S_{K_3}(t), dS_{K_3}(t), t + dt)$ in a Taylor series about $(S_{K_3}(t), t)$ gives

$$df = \frac{\partial f}{\partial S_{K_3}(t)} dS_{K_3}(t) + \frac{\partial f}{\partial t} dt + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_3}^2(t)} dS_{K_3}^2(t) + \dots \quad (18)$$

Substituting (4) in (18) gives,

$$df = \frac{\partial f}{\partial S_{K_3}(t)} \left\{ \sigma S_{K_3}(t) dZ^{(3)}(t) + \left\{ (\theta_1 + \theta_2)^{-1} \right\}^2 S_{K_3}(t) dt \right\} + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_3}^2(t)} dS_{K_3}^2(t) dS_{K_3}^2(t)$$

$$df = \sigma S_{K_3}(t) \frac{\partial f}{\partial S_{K_3}(t)} dZ^{(3)}(t) + \left\{ \left\{ (\theta_1 + \theta_2)^{-1} \right\}^2 S_{K_3}(t) \frac{\partial f}{\partial S_{K_3}(t)} + \frac{1}{2} \sigma^2 S_{K_3}^2(t) \frac{\partial^2 f}{\partial S_{K_3}^2(t)} dS_{K_3}^2(t) \right\} dt.$$

Now considering the SDE in (4).

Let $f(S_{K_3}(t)) = \ln S_{K_3}(t)$, the partial derivatives becomes

$$\frac{\partial f}{\partial S_{K_3}(t)} = \frac{1}{S_{K_3}(t)}, \quad \frac{\partial^2 f}{\partial S_{K_3}^2(t)} = -\frac{1}{S_{K_3}^2(t)}, \quad \frac{\partial f}{\partial t} = 0. \quad (19)$$

Following Theorem 1.1 (Ito's), substituting (19) and simplifying gives

$$df = \left\{ \left\{ (\theta_1 + \theta_2)^{-1} \right\}^2 - \frac{1}{2} \sigma^2 \right\} dt + \sigma dZ^{(3)}. \quad (20)$$

Since the RHS of (20) is independent of $f(S_{K_3}(t))$, the stochastic is computed as follows:

$$\begin{aligned}
 f(S_{K_3}(t)) &= f_0 + \int_0^t \left\{ \left((\theta_1 + \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} dt + \int_0^t \sigma dZ^{(3)}(t) \\
 &= f_0 + \left\{ \left((\theta_1 + \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t), \text{ Since } f(S_{K_3}(t)) = \ln S_{K_3}(t) \text{ a found solution for } S_{K_3}(t) \text{ becomes} \\
 \ln S_{K_2}(t) &= \ln S_{0(K_2)} + \left\{ \left((\theta_1 + \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(2)}(t), \ln S_{K_3}(t) - \ln S_{0(K_3)} = \left\{ \left((\theta_1 + \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(3)}(t) \\
 \ln \left(\frac{S_{K_3}(t)}{S_{0(K_3)}} \right) &= \left\{ \left((\theta_1 + \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(3)}(t) \\
 S_{K_3}(t) &= S_{0(K_3)} \exp \left\{ \left\{ \left((\theta_1 + \theta_2)^{-1} \right)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(3)}(t) \right\}. \quad (21)
 \end{aligned}$$

This is the complete solution of investment equation whose rate of returns and asset price valuation is additive inverse effects with a quadratic function.

From (5), Thus, generalizing and considering a function $f(S_{K_4}(t), t)$, hence it has to do with partial derivatives. Expansion of $f(S_{K_4}(t), dS_{K_4}(t), t + dt)$ in a Taylor series about $(S_{K_4}(t), t)$ gives

$$df = \frac{\partial f}{\partial S_{K_4}(t)} dS_{K_4}(t) + \frac{\partial f}{\partial t} dt + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_4}^2(t)} dS_{K_4}^2(t) + \dots \quad (22)$$

Substituting (5) in (22) gives

$$\begin{aligned}
 df &= \frac{\partial f}{\partial S_{K_4}(t)} \left\{ \sigma S_{K_4}(t) dZ^{(1)}(t) + (\theta_1 \theta_2)^2 S_{K_4}(t) dt \right\} + \frac{1}{2} \frac{\partial^2 f}{\partial S_{K_4}^2(t)} dS_{K_4}^2(t) dS_{K_4}^2(t) \\
 df &= \sigma S_{K_4}(t) \frac{\partial f}{\partial S_{K_4}(t)} dZ^{(4)}(t) + \left\{ (\theta_1 \theta_2)^2 S_{K_4}(t) \frac{\partial f}{\partial S_{K_4}(t)} + \frac{1}{2} \sigma^2 S_{K_4}^2(t) \frac{\partial^2 f}{\partial S_{K_4}^2(t)} dS_{K_4}^2(t) \right\} dt.
 \end{aligned}$$

Now considering the SDE in (5).

Let $f(S_{K_4}(t)) = \ln S_{K_4}(t)$, the partial derivatives becomes

$$\frac{\partial f}{\partial S_{K_4}(t)} = \frac{1}{S_{K_4}(t)}, \frac{\partial^2 f}{\partial S_{K_4}^2(t)} = -\frac{1}{S_{K_4}^2(t)}, \frac{\partial f}{\partial t} = 0 \quad (23)$$

Following Theorem 1.1 (Ito's), substituting (23) and simplifying gives

$$df = \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} dt + \sigma dZ^{(4)} . \quad (24)$$

Since the RHS of (24) is independent of $f(S_{K_2}(t))$, the stochastic is computed as follows:

$$\begin{aligned} f(S_{K_4}(t)) &= f_0 + \int_0^t \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} dt + \int_0^t \sigma dZ^{(4)}(t) \\ &= f_0 + \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(4)}(t), \text{ Since } f(S_{K_4}(t)) = \ln S_{K_4}(t) \text{ a found solution for } S_{K_4}(t) \text{ becomes} \\ \ln S_{K_4}(t) &= \ln S_{0(K_4)} + \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(4)}(t), \ln S_{K_4}(t) - \ln S_{0(K_4)} = \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(4)}(t) \\ \ln \left(\frac{S_{K_4}(t)}{S_{0(K_4)}} \right) &= \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(4)}(t) \\ S_{K_4}(t) &= S_{0(K_4)} \exp \left\{ \left\{ (\theta_1 \theta_2)^2 - \frac{1}{2} \sigma^2 \right\} t + \sigma dZ^{(4)}(t) \right\}. \end{aligned} \quad (25)$$

This is the complete solution of investment equation whose rate of returns and asset price valuation is multiplicative effects with a quadratic function.

3 Results

This Section presents the graphical results for the problems in (2)-(5) whose solutions are in (13)-(25). Hence the following parameter values were used in the simulation study:

Dangote₂₀₁₈: $\theta_1 \theta_2 = 21.7, S_{K_1} 0 = 128.91, \sigma = 2.5, dZ = 1.00, t = 1$

Dangote₂₀₁₉: $S_{K_2} 0 = 166.11, \theta_1 + \theta_2 = 22.4, \sigma = 2.5, dZ = 1.00, t = 1$

Dangote₂₀₁₉: $S_{K_3} 0 = 270, \theta_1 + \theta_2 = 22.4, \sigma = 2.5, dZ = 1.00, t = 1$

Dangote₂₀₁₉: $S_{K_2} 0 = 190, \theta_1 \theta_2 = 22.4, \sigma = 2.5, dZ = 1.00, t = 1$

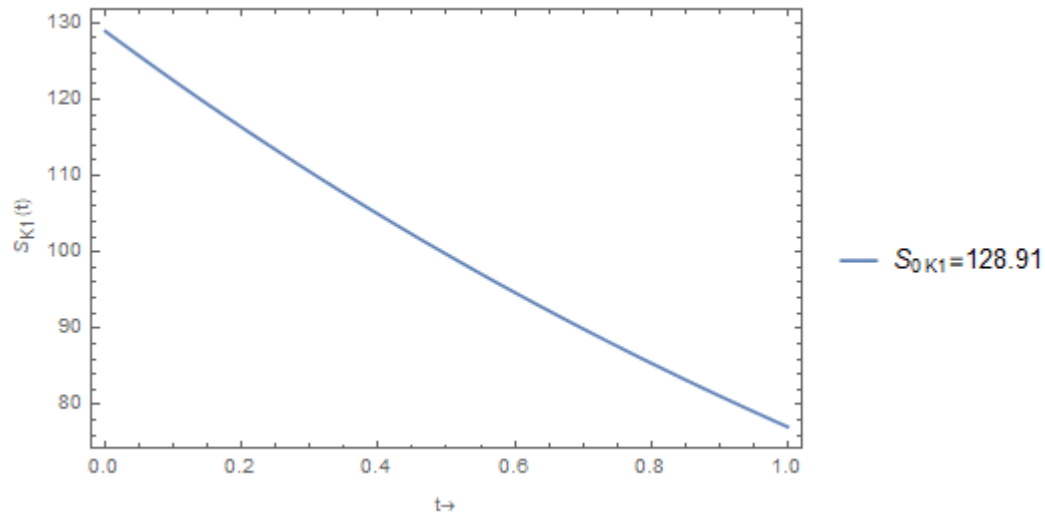


Figure 1: Plot of Dangote stock rate of returns which follows multiplicative inverse effects.

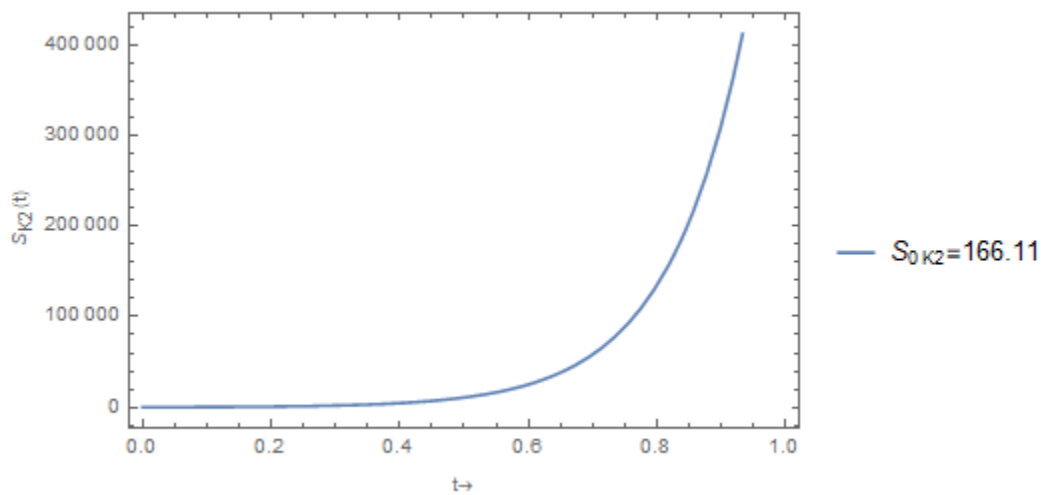


Figure 2: Plot of Dangote stock rate of returns which follows additive effects.

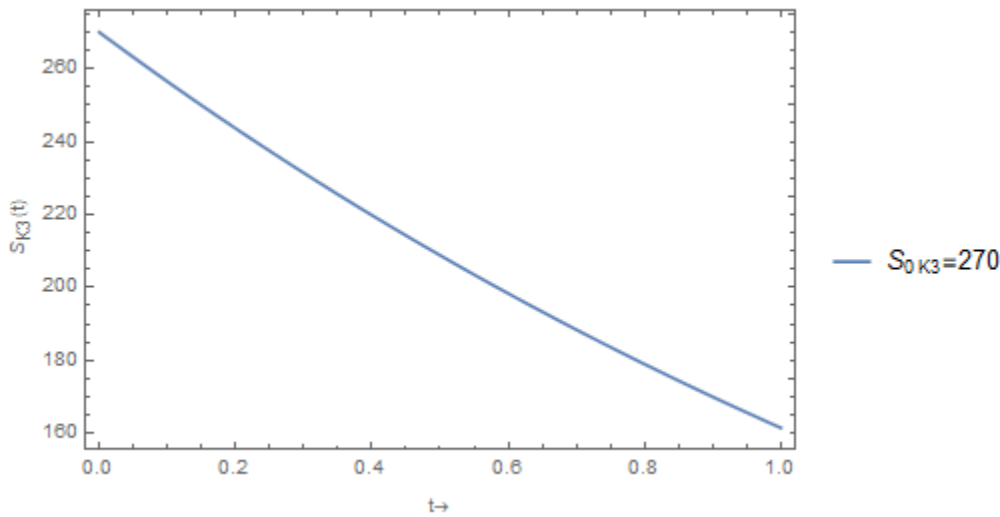


Figure 3: Plot of Dangote stock rate of returns which follows additive inverse effects.

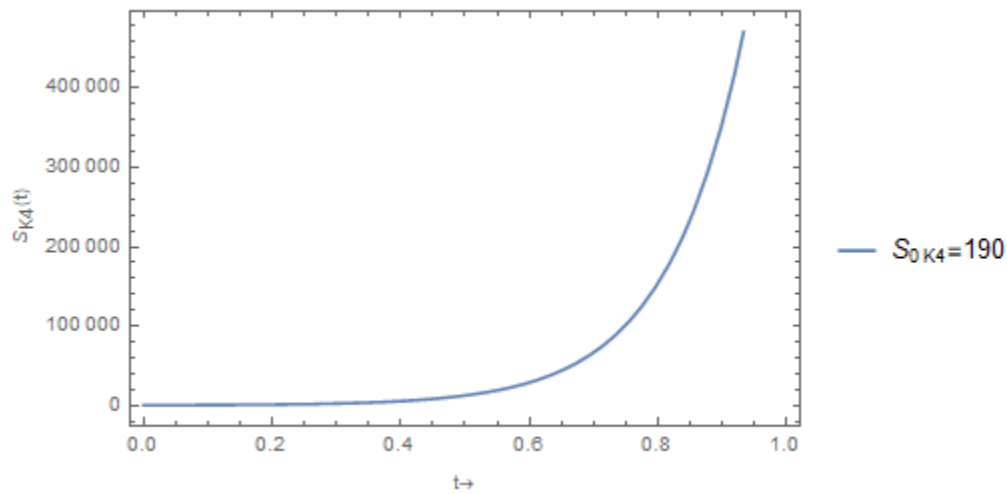


Figure 4: Plot of Dangote stock rate of returns which follows multiplicative effects

3.1 Discussion of results

As seen in Figures 1 and 2 respectively that the plots are linear; this means that there's a linear relationship between return rates and time of trading activities of price changes. Carefully examination of the plots shows that asset return increases linearly as time increases throughout the trading days, with the tendency of being 1. It also describes changes over short and long periods of time, which informs an investor for proper decision making. These results also agree with what the principle of multiplicative inverse and correlation as it affects trend line. Multiplicative inverse of

trend of a number is a number by which the multiplication result is 1. Therefore, in terms of prediction multiplicative inverse is more efficient and reliable than multiplicative effect. The range of regression is within $-1 \leq r \leq 1$; by this formation means that total sure of high rate of returns in Dangote cement PLC which is indexed with millions of naira. This result is line with that of [1].

In Figures 2 and 4, it can be observed that the two plots have exponential growth of stock returns of time-varying investment. The trends stipulate that a little increase of time of investment; will also increase increase rate of return of capital investments. In the same vein, time decreases the value of asset return approaches to zero. This result agrees with the concept of multiplicative trend which indicates a non-linear trend (curved trend line). This informs investors that when return rates follow multiplicative trend it grows unboundedly. With this remark, profit making is inevitable throughout the trading days.

Nevertheless, stochastic systems are effective in the mathematical modeling of stock returns for long- and short-term investment plans worth millions of naira.

4 Conclusions

In absence of capital investments, trading business will definitely have a hard time getting off the ground. Therefore, this paper studied the problem of stochastic systems of time-varying investments of Dangote cement PLC where multiplicative inverse effects, additive effects, additive inverse effects and multiplicative effects were used as key parameter in the model to obtain stock rate of returns. The analytical solution of problem showed: (i) a little increase on time of investment; will also increase increases rate of return of capital investments, (ii) stock rates return increases linearly as time also increases, (iii) Multiplicative inverse and additive inverse effects is more effective and reliable than multiplicative effects and additive effect on the ground that it is a sure event (iv) Dangote cement PLC is making high rate of returns based on stochastic analysis of stock price data.

Finally, comparing two or more companies in terms of stock returns and applying stochastic systems will be an interesting area to explore.

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Mathematical modeling of rape under the influence of human disturbance and noise

C. Nkutura^{*} and M.N. Onwubuya[†]

Abstract

To leverage rape decline, a guide from epidemiological modeling was used to develop a new deterministic model for the dynamics of rape during covid-19 and the world at large. This was used to qualitatively assess the role of convictions and loss of immunity to rape on the transmission process of rape in a society. This study showed that loss of immunity to rape and its rate of previous convicted and jailed rape culprit individuals regress to being susceptible to rape cases induced a backward bifurcation when associated to rape reproduction number ($R_{ef} < 1$) is less than one. This means that imprisonment of the convicted rape individuals will help to eradicate rape out from the entire world even at covid-19 pandemic. The study showcased that if the cause of backward bifurcation is removed, the rape-free equilibrium (R_e) of the model is globally asymptotically stable when the reproduction number is less than one. This study also showed that the rape endemic equilibrium (R_{ee}) is locally-asymptotically stable when the reproduction number is greater than one ($R_{ef} > 1$). The analytical solutions suggest that Policymakers should provide policies that would check one time convicted and jailed individuals from being susceptible to rape after their released from prison. Self-recovered individuals should be assisted and engaged with employment as to avoid the stigmatization of rape.

Keywords: rape; Covid-19; pandemic; SCPV model; noise and human disturbance.

1 Introduction

Rape is the abuse of humanity and personality trait to the victims and society at large. It is a sexual assault that involves sexual intercourse or other forms of sexual penetration being carried out by physical force or coercion against the person's consent. Rape is an element of the crime of genocide when committed with the intention to destroy a targeted ethnic group partly or completely. It can be notable in various form as child rape and abuse, incest rape, sexual slavery, gang rape, prison rape, marital rape, acquaintance rape, war rape and statutory rape, [16,18]. Rape is predominantly committed by male factors against females. Globally, rape occurs in all continents of the world and the female partners are always helpless when involved according to [20, 21, 22] stated that about 35% of women worldwide have been raped in their life time.

^{*} Corresponding Author. Department of Mathematics, School of Foundation Studies, Rivers State College of Health Science and Management Technology, Port Harcourt, Nigeria; E-mail: nkuturachristiana@rschst.edu.ng, christienkutura245@gmail.com

[†] Department of Statistics, Delta State Polytechnic, Oghara, Nigeria

Several reasons have been identified for the causes of rape in a society. Thus, it can be caused by some of these factors drug abuse and alcohol addicts, lack of sex education, rape myths by men against women, the desire to control, parental negligence, Peer and family factors, Societal factors and poverty. Drug facilitated sexual assault is also known as predator-prey rape [12]. it is a sexual assault carried out after the victim has become incapacitated due to having consumed alcoholic beverages or other drugs, [13]. Alcohol has used to play an activating role during sexual assault, when the rapists must have taken some drugs like Cocaine, Marijuana and Tramadol which contributed to psychological havoc or trauma on the victims, [10,11, 14]. The major cost of rape is risk of being caught, punished, convictions and jailed. Nevertheless, the probability of being caught depends on the effectiveness of security and legal system of such countries, [15]. One may wish to read the detail article on rape and its possible mode of control in [1].

The various body of literature existing on the social, political and management aspects of rape and attempt to provide policies to curb rape. Scientifically, several attempts have been made to use mathematical models to study the dynamics of rape in a population. Amongst others were [9,17] used elementary model of population growth first order differential equation to study the growth rate of domestic violence and predictions using differential equations in a population; consideration was given in a community full of individuals prone to rape as case study. The work done by [7] studied rape as a disease epidemic process and used [5] classical SIR epidemiological mathematical models for modelling the spread of rape. He considered the population as made up of individuals susceptible to rape (S), rapists (I) and individuals who recover and are immune to rape (R) where the notations for the state variables were adjusted differently in [6]. [13] examined the impact of chaos theory in social systems afflicted with rape.

Currently, this study presents a new deterministic model that studies the population dynamics of rape in a population at covid-19 pandemic under the influence of human disturbance and noise. The model treats rape as a disease as was done by [19], thus, the model could fit well for an epidemiological setting even at covid-19 pandemic. In this present study, instead of having just three classes used by [7, 21], a modification of the SIR models to SCPVR models was made and I proposed dividing the population into five classes which includes the rape culprits (C), rape victims or prey (P) and individuals who are convicted and jailed for rape they committed (V). Also, the model considered human disturbance and noise factors. Hence, this paper is organized as follows: the model formulation is section 2 and section 3 provide discussion and conclusions.

2 Model Formulation

The total population at time t , denoted by $N(t)$ is subdivided into five mutually exclusive compartments of susceptible individuals to rape ($S(t)$); individual for rape culprit ($C(t)$); individuals who are victims of rape or prey to rape ($P(t)$), rape individuals who are convicted and jailed ($V(t)$) and individuals who recover from rape ($R(t)$) respectively. So that the total population becomes

$$N(t) = S(t) + C(t) + P(t) + V(t) + R(t).$$

Rape interaction in the population is modelled using a standard incidence function. The population of individuals who are susceptible to rape $S(t)$ is increased by the recruitment of a proportion ρ , ($0 \leq \rho \leq 1$) of immigrants (who are susceptible) into the population at a rate γ , 'recovered' individuals who become susceptible to rape again at a rate π and convicted individuals who after their release become susceptible to rape again at a rate ψ . The susceptible population is decreased by rape at a rate λ that is the rape force of infection, where

$$\lambda = \frac{\beta C}{N-V},$$

and β is the rape transmission rate. The susceptible population is further decreased by natural death at a rate μ ; natural removal occurs in all the compartments at this rate. Thus,

$$\frac{dS}{dt} = \rho\gamma - \lambda S - \mu S - \pi R - \psi V.$$

The population of rape culprit individuals $C(t)$ is increased by the rape interaction that effects the subsequently infects susceptible individuals at the rate λ . Because the human disturbance or predation is negative on the culprit and positive on the victim or prey, so that it becomes $a_1 = Eq$ (q is the predating coefficient and E is total effort applied on predating on the population of the culprit). The noise term is assumed to be sinusoidal with amplitude A and frequency is ω of the noise, and phase shift φ . The population is decreased by trial, conviction and subsequently jailing which leads to removal from the society at a rate γ , recovery at a rate α and natural death μ . In this study, the noise term from culprit is called black noise. This is colored noise because it does not last long in the culprit and it shifts per time from when it was conceived and ends at the time action has been executed by the culprit; gives

$$\frac{dC}{dt} = \lambda S - (\gamma + \alpha + \mu)C - qEC + A \sin(\omega t + \varphi).$$

The population of rape victims or prey individuals $P(t)$ is increased by the rape interaction, that effects subsequently infect susceptible individuals at the rate λ . Because the human disturbance or predation is positive on the victim or prey, so that it becomes $b_1 = Eq$ (q is the predating coefficient and E is total effort applied on predating on the population of the culprit as to stop the action). The noise term is assumed to be sinusoidal with amplitude A and frequency is ω of the noise. The population is decreased by trial, conviction and subsequently trauma and sickness which leads to removal from the society at a rate δ , recovery at a rate α and natural death μ . In this study, the noise coming from the victims or prey is called green noise. This green noise is a limiting case of the black noise because it last long in the victims-prey; gives

$$\frac{dP}{dt} = \lambda S - (\delta + \alpha + \mu)P + A \sin \omega t.$$

The population of convicted and jailed individuals $V(t)$ is increased by culprit individuals who are caught and convicted at the rate η (this population is in a form of isolation and without interaction with the outside world) and decreased by such individuals who are later become susceptible to rape after their release. Considering the period spent in jail at the rate Ω , convicted individuals who after serving their become resistant to rape at rate ξ and natural death μ . This gives

$$\frac{dV}{dt} = \eta C - (\xi + \Omega + \mu)V.$$

The population of rape individuals who were not caught and convicted but recover due to counselling and public enlightenment programmes on the need to shun rape considering that the society is an imperfect one where not all persons who commit rape face convictions. $R(t)$ is increased by rape individuals who abandon a rape lifestyle at a rate α . This population is decreased

by developing resistance to rape environment at a rate $\emptyset$, becoming susceptible to rape again after some time at a rate π and natural death μ , yields

$$\frac{dR}{dt} = \alpha C - (\emptyset + \pi + \mu)R.$$

Based on the above formulations and assumptions, the model for the dynamics and spread of rape at covid-19 in a population consist of the following system of nonlinear (deterministic) differential equations. Table 1 describes the associated state variables and parameters in the model.

$$\frac{dS}{dt} = \rho\gamma - \lambda S - \mu S - \pi R - \psi V, \quad (1)$$

$$\frac{dC}{dt} = \lambda S - (\gamma + \alpha + \mu)C - qEC + A \sin(\omega t + \varphi), \quad (2)$$

$$\frac{dP}{dt} = \lambda S - (\delta + \alpha + \mu)P + A \sin \omega t, \quad (3)$$

$$\frac{dV}{dt} = \eta C - (\xi + \Omega + \mu)V, \quad (4)$$

$$\frac{dR}{dt} = \alpha C - (\emptyset + \pi + \mu)R, \quad (5)$$

with initial conditions

$$S(0) = S_0, \quad C(0) = C_0, \quad P(0) = P_0, \quad V(0) = V_0, \quad R(0) = R_0.$$

Some of the assumptions and features of this model are below.

1. Assume that we are dealing an imperfect society whereby not all culprit individuals are caught and convicted.
2. Assume that the culprit individuals who likely recovered due to counseling and enlightenment campaigns abandon their rape lifestyle and recover from rape tendencies.
3. Assume that the culprit individuals are not rape victims or prey
4. Incorporating a class of convicted individuals who are jailed and are in complete isolation from the outside.
5. Assume that the human disturbance is negative on the culprit and positive on the victim or prey; hence it was ignored on victim class.
6. Assume the noise term (white and colored noise) to be sinusoidal with amplitude A , frequency is ω and phase shift φ .

Table 1 shows the variables and parameters description.

Dependent Variables	Interpretation
S	Population of individuals susceptible to rape
C	Population of culprit individuals
P	Population of rape victims or prey individuals
V	Population convicted individuals who are jailed (isolated)
R	Population of individuals who recover from rape
Parameters	Interpretation
ρ	Recruitment rate
γ	Proportion of immigrants who are susceptible to rape
λ	Susceptible population is decreased by rape
μ	Natural mortality rate in all the compartments
π	Rate of the 'recovered' individuals become susceptible to rape
ψ	Convicted individuals become susceptible to rape after release
α	Rape recovery rate
β	Rape transmission rate
q	Predating coefficient by human beings
E	Total effort applied on predating the population of the culprit
A	Amplitude of the noise from culprit and victim individuals
ω	Frequency of the noise from culprit and victim individuals
φ	Phase shift of the noise from culprit
δ	Rate at which the victim individuals are removal from the society
Ω	Conviction rate
ξ	Rate at which convicted individuals become resistant to rape
η	Rate at which culprit individuals caught and convicted
$\emptyset$	Rate of developing resistance to rape environment

Table 1: Variables and parameters description

2.1 Basic properties

For model (1-5) to be meaningful, it is important to prove that all the state variables are non-negative for all time t . thus, the solutions of model (1-5) with positive initial data will remain positive for all $t \geq 0$.

2.1.1 Well-posed model and positivity of the solutions

Model (1) controls and monitors human population therefore all the state variables and parameters of the model are non-negative. Considering the domain

$$D = \left\{ S, C, P, V, R \in R^5 : 0 \leq N \leq \frac{\rho\gamma}{\mu} \right\}. \quad (6)$$

It can be shown that the set of D is a positive invariant set and a global attractor of this system. That is any phase trajectory initiated anywhere in the non-negative region R_+^5 of the phase space eventually enters the region D and remains in D thereafter.

Let $S_0, C_0, P_0, V_0, R_0 \geq 0, S_0 + C_0 + P_0 + V_0 + R_0 = N_0$. Then, there exists solutions $S(t), C(t), P(t), V(t), R(t)$ for the dynamical system (1) with initial data at $t = 0$, that defined for all $t \geq 0$. In fact, $S(t), C(t), P(t), V(t), R(t)$ are non-negative and $S(t) + C(t) + P(t) + V(t) + R(t) = N(t)$ for all t . if $C_0 = 0, R_0 = 0, V_0 = 0$, then $C(t) \equiv 0$ and $V(t) \equiv 0$.

Since $N = S + C + P + V + R$, it follows that the rate of change of the total population is given by

$$\frac{dN}{dt} = \rho\gamma - \mu N, \quad (7)$$

therefore, that $N(t) \rightarrow \frac{\rho\gamma}{\mu}$ as $t \rightarrow \infty$. That is the total population is asymptotically constant. The well-posedness of the model follows a straight forward application of the classical theory in [11].

Lemma 2.1 *The region D is positively invariant for the model equations (1-5) and if $C_0 > 0$, then $S(t) > 0, R(t) > 0, P(t) > 0$ and $C(t) > 0$ for all $t > 0$ and V is bounded by $\dot{V} = \max \left\{ V_0, \frac{\eta}{\mu + \xi + \Omega} \right\}$.*

Proof. The right hand side of system (1-5) is continuously differentiable and hence it is locally Lipschitz and therefore there exists a unique solution $S(t), C(t), P(t), V(t), R(t)$ to system (1-5) with the initial data S_0, C_0, P_0, V_0, R_0 that is defined on a maximal forward interval of existence, [11]. Consider the set $D \subset R_+^5$, since $C(t) \geq 0$ from system of equation (1-5), yields

$$C(t) \geq C_0 \exp \int_0^t \left[\frac{\beta C}{N-V} - Eq + A \sin(\omega t + \varphi) - K_1 \right] dt \quad (8)$$

$$P(t) = \left[P_0 + \int_0^t \eta C(\xi) e^{K_2(\xi-t_0)} d\xi \right] e^{K_2(t_0-t)} (A \sin \omega t) \quad (9)$$

$$V(t) = \left[V_0 + \int_0^t \eta C(\xi) e^{K_3(\xi-t_0)} d\xi \right] e^{K_3(t_0-t)} \quad (10)$$

$$R(t) = \left[R_0 + \int_0^t \alpha C(\xi) e^{K_4(\xi-t_0)} d\xi \right] e^{K_4(t_0-t)}. \quad (11)$$

So that $S(t) \geq 0$, $C(t) \geq 0$, $P(t) \geq 0$, $V(t) \geq 0$, $R(t) \geq 0$, $\forall t > 0$, where $K_1 = \gamma + \alpha + \mu$, $K_2 = \delta + \alpha + \mu$, $K_3 = \xi + \Omega + \mu$, $K_4 = \phi + \pi + \mu$.

To show that V is bounded by $\dot{V} = \max \left\{ V_0, \frac{\eta}{\mu + \xi + \Omega} \right\}$, is to show whether $V(t) \leq \omega$, $\forall t \geq 0$ and that $\omega \geq \frac{\eta}{K_3}$ if $V_0 \leq \omega$. Suppose that the above inequalities do not hold, then there exists a time, t_1 , with $V'(t_1) > 0$ and $V'(t_1) > \omega$. Using the equation for V in (1), gives $\frac{dV(t_1)}{dt} = \eta C(t_1) - K_3 V(t_1) \leq \eta(C(t_1) - 1) < 0$. (12)

Since $(C(t_1) - 1) < 0$ and $\eta > 0$. This is the contradiction which implies that $V(t) < \omega$, $\forall t \geq 0$. Suppose that $V(0) > \omega \geq \frac{\eta}{K_3}$. In order to show that $V(t) \leq V(0) - \forall t \geq 0$, we assume that the last inequality does not hold. Hence, there exists a time $t_2 > 0$ such that $V(t_2) \geq V(0)$ and

$$V'(t_2) > 0.$$

Since $V(t_2) > \frac{\eta}{K_3}$. Then

$$\frac{dV(t_2)}{dt} = \eta C(t_2) - K_3 V(t_2) \leq \eta(C(t_2) - 1) < 0, \text{ but } V'(t_2) > 0. \quad (13)$$

Hence, we reach a contradiction and $V(t)$ is bounded from above by $\dot{V}$, where

$$\dot{V} = \max \left\{ V_0, \frac{\eta}{K_3} \right\}.$$

End of proof.

Lemma 2.1 guarantees the positivity of the different groups of individuals making up the total population, S, C, P, V and R for all time, $t \geq 0$.

3 Asymptotic stability of rape-free equilibrium (RFE)

The rape-free equilibrium of the model (1-5) is given by

$$E_0 = (S^*, C^*, P^*, V^*, R^*) = \left(\frac{\rho\gamma}{\mu}, 0, 0, 0, 0 \right) \quad (14)$$

Setting $A = 0$, depicting no noise and no rape action.

The linear stability of E_0 can be established using the next generation operator method on the model equ (1-5), [5,2,10]. Using the notations in [4], it follows that F and V , which stands for the new rape infection terms and remaining transition terms respectively are given by $F = \rho\beta$ and $V = \alpha + \mu + \gamma$.

It follows that the effective rape reproduction number of the model equation (1-5) denoted by $R_{rn} = FV^{-1} = \frac{\rho\beta}{\alpha+\mu+\gamma}$.

The result above follows from theorem 2 in [4].

Lemma 3.1 *The CFE (E_0) of the model equ (1-5) is locally-asymptotically stable (LAS) if $R_{rn} < 1$ and unstable if $R_{rn} > 1$.*

The threshold quantity, R_{rn} is the rape reproduction number. It represents the average number of secondary cases of rape generated by a typical rape individual in a population that is completely susceptible to rape (using a typical language in epidemiology, [14]. The implication of Lemma 3.1 is that when R_{rn} is less than one ($R_{rn} < 1$), rape can be eliminated from the population if the initial sizes of the sub-population of the model are in basin of attraction of the rape-free equilibrium (E_0). Hence, a small influx of rape-infected individuals into the country will not generate large outbreaks of rape and it dies out in time completely.

3.1 Backward Bifurcation Analysis

To describe the bifurcation type in the model equations (1-5), it will go a long way to help in determining the factors that could hinder efforts in tackling rape in the population. Hence, the following results were assumed.

Theorem 3.1 That the model equation (1-5) exhibits a backward bifurcation at $R_{rn} = 1$ whenever a bifurcation coefficient denoted by a , is positive.

Proof.

Let $E_1 = (S^{**}, C^{**}, P^{**}, V^{**}, R^{**})$ represents any arbitrary endemic equilibrium of the model; that is an equilibrium in which at least one of the infected components is non-zero. The existence of backward bifurcation will be explored using the Manifold theory, [2,3]. To apply this theory, it is appropriate to carry out the following change of variables. Let: $S = x_1, C = x_2, P = x_3, V = x_4, R = x_5$. By using the vector notation, $X = (x_1, x_2, \dots, x_5)^T$, the model can be written in the form

$\frac{dX}{dt} = F(X)$, with $F = (f_1, f_2, \dots, f_5)^T$, yields:

$$\dot{x}_1 \equiv f_1 = \gamma\rho - \lambda x_1 - \mu x_1 + \pi x_5 + \psi x_4 \quad (15)$$

$$\dot{x}_2 \equiv f_2 = \lambda x_1 - (\eta + \alpha + \mu)x_2 \quad (16)$$

$$\dot{x}_3 \equiv f_3 = \lambda x_1 - (\delta + \alpha + \mu)x_3 + \quad (17)$$

$$\dot{x}_4 \equiv f_4 = \eta x_2 - (\xi + \Omega + \mu)x_4 \quad (18)$$

$$\dot{x}_5 \equiv f_5 = \alpha x_2 - (\emptyset + \pi + \mu)x_5 \quad (19)$$

where $K_1 = \eta + \alpha + \mu + qE$, $K_2 = \delta + \alpha + \mu$, $K_3 = \xi + \Omega + \mu$, $K_4 = \emptyset + \pi + \mu$, $K_5 = qE$ and the force of infection is given by

$$\lambda = \frac{\beta x_2}{N - x_4} \text{ and } N = x_1 + x_2 + x_3 + x_4 + x_5.$$

Put $\beta = \beta^*$ as a bifurcation parameter. Solving for $\beta = \beta^*$ from $R_{rn} = 1$, gives

$$\beta = \beta^* = \frac{K_1}{\rho}. \quad (20)$$

The Jacobian of the transformed model equation (15–19) evaluated at the rape-free equilibrium (E_0) with $\beta = \beta^*$ is given by

$$J^* = J(E_0)|_{\beta=\beta^*} = \begin{pmatrix} -\mu & -\rho\beta^* & \pi & 0 & \xi \\ 0 & \rho\beta^* - K_1 & 0 & 0 & 0 \\ 0 & \alpha & -K_2 & 0 & 0 \\ 0 & 0 & \emptyset & -\mu & \psi \\ 0 & \eta & 0 & 0 & -K_3 \end{pmatrix} \quad (21)$$

The matrix J^* has a right eigenvector as $w = (w_1, w_2, \dots, w_5)^T$, where

$$\begin{aligned} w_1 &= \frac{-p\beta w_2 + \pi w_3 + \xi w_2}{\mu} \\ w_2 &= w_2 > 0, \\ w_3 &= \frac{\alpha}{K_2} w_2 > 0, \\ w_4 &= \frac{\emptyset\alpha}{\mu K_2} + \frac{\psi\eta}{\mu K_3} w_2 > 0, \\ w_5 &= \frac{\eta}{K_3} w_2 > 0. \end{aligned} \quad (22)$$

Thus, J^* has a left eigenvector $v = (v_1, v_2, \dots, v_5)$ satisfies that $v \cdot w = 1$, with

$$v_1 = v_3 = v_4 = v_5 = 0, \text{ and } v_2 > 0.$$

This follows from Theorem 4.1 in [2]. To compute the associated non-zero partial derivatives of $F(x)$ evaluated at rape-free equilibrium, E_0 by defining the associated bifurcation coefficients, a and b , giving by:

$$a = \sum_{k,i,j=1}^5 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0), \text{ and } b = \sum_{k,i=1}^5 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*} (0,0),$$

when computed gives

$$a = \frac{2\beta^* \mu v_2 w_2^2}{\gamma} \left\{ \frac{1-\rho}{\mu} \left(\frac{\pi \alpha K_3 + \eta \xi K_2}{K_2 K_3} \right) - \left(\frac{(1-\rho)K_1}{\mu} + \frac{\eta \psi}{\mu K_3} + \frac{\emptyset \alpha}{\mu K_2} + \frac{\alpha}{K_2} + 1 \right) \right\} \quad (23)$$

and

$$b = v_2 w_2 \rho > 0. \quad (24)$$

Hence, the bifurcation coefficient b is positive, it follows from Theorem 4.1 in [2] that the model equation (1-5) and the transformed model (15-19) will undergo a backward bifurcation if the backward bifurcation coefficient a , given by equation (23) is positive.

The occurrence of backward bifurcation has been observed in numerous disease transmission dynamics as in [2,3] showed a typical case of co-existence of a stable rape-free equilibrium and a stable rape endemic equilibrium when the associated rape reproduction number of the model is less than one. This implies that the model equation (1-5) is the typical tool of having a rape reproduction number (R_{rn}) less than one, while the jailed equation is to deteriorated rape in the

population where it is common. Putting $\pi = \xi = 0$ in (23) and setting all parameters of the model (1-5) positive and $\beta^* > 0$, the bifurcation parameter a reduces to;

$$a = -\frac{2\beta^* \mu v_2 w_2^2}{\gamma} \left(\frac{(1-\rho)K_1}{\mu} + \frac{\eta\psi}{\mu K_3} + \frac{\phi\alpha}{\mu K_2} + \frac{\alpha}{K_2} + 1 \right) < 0 \quad (25)$$

with $v_2 > 0$ and $w_2 > 0$.

From Theorem 4.1 in the model equation (1-5) does not undergo backward bifurcation, i.e. if $\pi = \xi = 0$. Then it shows that the rate at which individuals 'recover' from rape practices due to certain interventions like public enlightenment, counseling, jail e.t.c and previously convicted individuals become susceptible to rape for $\pi \neq 0$ and $\xi \neq 0$ are the causes of backward bifurcation in the rape transmission model. Therefore, the mathematical analysis in shows that previously convicted and recovered individuals becoming susceptible to rape will make it difficult to have rape eradicated from the population.

3.2 Global Asymptotic Stability: Special Case $\pi = \xi = 0$.

Considering the model equation (1-5) is with $\pi = \xi = 0$.

Theorem 3.2 The RFE of the model (1-5) with $\pi = \xi = 0$ is globally-asymptotically stable (GAS) in D whenever $R_m < 1$.

Proof. Consider the model (1-5) with $\pi = \psi = 0$ and linear Lyapunow function $F = \rho C(t)$. By Lyapunow derivative with respect to t .

$$\begin{aligned} \dot{F} &= \rho \dot{C} \\ &= \left[\frac{\rho \beta S C}{N - V} - (a - \mu - \eta) \right] C \\ &= a + \frac{C}{a + \mu + \eta} \left[\frac{\rho \beta}{a + \mu + \eta} \frac{S}{N - V} - 1 \right] \end{aligned} \quad (26)$$

From (26), $S(t) \leq N(t) - V(t)$ in D for all $t > 0$, then

$$\dot{F} \leq \frac{1}{a + \mu + \eta} (R_m - 1) C \quad (27)$$

Thus, $F \leq 0$ if $R_m \leq 1$ with $\dot{F} = 0$, if and only if $C=0$. Therefore, F is a Laypnow function in D and it follows from LaSalle's Invariance Principle [8], that every solution to the equations in (1-5) with $\pi = \xi = 0$ with initial conditions in D converges to E_0 as $t \rightarrow \infty$ i.e.

$(C(t), R(t), V(t)) \rightarrow (0,0,0)$ as $t \rightarrow \infty$. Substituting $C = R = V = 0$ into equation (1-5) gives $S(t) \rightarrow \frac{\rho\gamma}{\mu}$ as $t \rightarrow \infty$. Hence $(S, C, P, V, R) \rightarrow \left(\frac{\rho\gamma}{\mu}, 0, \frac{(1-\rho)\gamma}{\mu}, 0, 0 \right)$ as $t \rightarrow \infty$ for $R_m \leq 1$, so that the RFE, E_0 , is GAS in D if $R_m \leq 1$ for the case $\pi = \xi = 0$.

The above result signifies that in the absence of recovered individuals and persons previously convicted and jailed for rape becoming susceptible to rape again, rape will be eliminated from the population of the society if the rape reproduction number or threshold $R_m < 1$.

3.3 Existence and stability of rape endemic equilibrium

To create the existence of the rape endemic equilibrium of the model equation (1–5), let $E_1 = \{S^{**}, C^{**}, P^{**}, V^{**}, R^{**}\}$ represents any arbitrary rape endemic equilibrium of the model (1–5). The equation (1-5) are solved in terms of the rape force of infection at a steady state to give

$$S^{**} = \frac{1}{\lambda^{**} - \mu} \left[\frac{\eta \xi K_2 C^{**} + \rho \gamma K_2 K_3}{K_2 K_3} \right] \quad (29)$$

$$C^{**} = \frac{\lambda^{**} K_2 K_3 \rho \gamma}{K_1 K_2 K_3 (\lambda^{**} + \mu) - \lambda^{**} (K_3 \alpha \pi + K_2 \eta \xi)}, \quad (30)$$

$$R^{**} = \frac{\alpha C^{**}}{K_2}; \quad T^{**} = \frac{1}{\mu} \left[\frac{K_2 \theta \alpha C^{**} + \psi \eta K_2 C^{**} + K_2 K_3 (1 - \rho) \gamma}{K_2 K_3} \right]; \quad V^{**} = \frac{\eta C^{**}}{K_3} \quad (31)$$

Recall, that the rape force of infection at steady state, λ^{**} , is expressed as

$$\lambda^{**} = \frac{\beta C^{**}}{S^{**} + C^{**} + R^{**} + T^{**}}$$

$$H \lambda^{**2} - \lambda^{**} (p \beta \mu K_2 K_3) = 0$$

where $H = \alpha[(p - 1)\pi + p(\mu + \theta)]K_3 + [\gamma((p - 1)\varepsilon + p\psi) + (1 - p)K_1]K_2$, and further reduces to

$$H \lambda^{**} - \mu K_1 K_2 (R_0 - 1) \quad (32)$$

$$\lambda^{**} = \frac{\mu K_1 K_2 K_3}{H} (R_0 - 1) \quad (33)$$

Hence, a unique rape endemic equilibrium exists (i.e. $\lambda^{**} > 0$) when $R_0 > 1$ as far as $H > 0$. Clearly, if $\pi = \varepsilon = 0$, then $H > 0$ and there exists a unique rape endemic equilibrium for this case. Thus, the following result is established.

Theorem 3.3 The model (1-5) has a unique endemic (positive) equilibrium when $\pi = \varepsilon = 0$ whenever $R_0 > 1$.

The possibility of the existence of a rape endemic equilibrium exists even $R_0 < 1$. This occurs when $H < 0$ which is possible when $\pi \neq 0$ and $\varepsilon \neq 0$ since $0 \leq 1$. This further proves the possibility of a backward bifurcation when $R_0 < 1$ whereby the rape free equilibrium will co-exist with a rape endemic equilibrium. Therefore, the general implication of this as stated before that individual who ‘recover’ from rape after an intervention programme and previously convicted individuals should strive not to become susceptible to rape again. The relevant government agencies may determine and formulate policies that could help with preventive strategies cum corrective measures.

3.4 Local Asymptotic Stability of the Rape Endemic Equilibrium

Since the total population is asymptotically constant, the result in [2] guarantee that the following system of equations from (34-38).

$$\frac{dS}{dt} = \rho\gamma - \lambda S - \mu S - \pi R - \psi V \quad (34)$$

$$\frac{dC}{dt} = \lambda S - (\gamma + \alpha + \mu)C - qEC + A \sin(\omega t + \varphi) \quad (35)$$

$$\frac{dP}{dt} = \lambda S - (\delta + \alpha + \mu)P + A \sin \omega t \quad (36)$$

$$\frac{dV}{dt} = \eta C - (\xi + \Omega + \mu)V \quad (37)$$

$$\frac{dR}{dt} = \alpha C - (\phi + \pi + \mu)R \quad (38)$$

where $\gamma = \frac{\beta C}{N-V}$ with $N = \frac{\rho\gamma}{\mu}$, has the same asymptotic qualitative dynamics as those of system. Putting $K_2 = -qEC + A \sin(\omega t + \varphi)$, $K_4 = A \sin \omega t$

The Jacobian matrix for system (34-38) at the endemic equilibrium, ε_1 is

$$J(E_1) = \begin{pmatrix} -A - \mu & B & 0 & -D + \psi & \pi \\ A & 0 & B - K_1 + K_6 & 0 & 0 \\ A & 0 & 0 & B - K_3 + K_7 & 0 \\ 0 & \eta & 0 & -K_4 & 0 \\ 0 & \alpha & 0 & 0 & -K_5 \end{pmatrix}$$

where $A = \frac{\beta C^{**}}{N-V^{**}}$, $B = \frac{\beta S^{**}}{N-V^{**}}$ and $D = \frac{\beta S^{**} C^{**}}{(N-V^{**})^2}$

Hence, the characteristic equation associated with the local stability of $J(\varepsilon_1)$ are:

$$A_4(\varphi)Q^4 - A_3(\varphi)Q^3 - A_2(\varphi)Q^2 + A_1(\varphi)Q + A_0 = 0 \quad (39)$$

where

$$\begin{aligned} A_4 &= 1, \\ A_3 &= B + \mu + K_2 + K_3 \\ A_2 &= -D\gamma - B\mu + (A - B + \mu)K_3 + K_2(A - B + \mu + K_3) \\ &\quad + K_1(A + \mu + K_2 + K_3), \\ A_1 &= -D\gamma\mu + A\gamma\xi + A\alpha\pi + (-B\mu + (A + \mu)K_1)K_3 \\ &\quad + K_2(-D\gamma - B\mu + (A - B + \mu)K_3 + K_1(A + \mu + K_3)), \\ A_0 &= +A\alpha\pi K_3 + K_2(\gamma(D\mu + A\xi) + (B\mu - (A + \mu)K_1)K_3) \end{aligned}$$

and Q_i represents the eigenvalues of the Jacobian with φ is the model's parameter vector. The local stability of the rape endemic equilibrium is geared towards the roots of equation (39). Therefore, the rape endemic equilibrium point is locally asymptotically stable if the roots of equations (39) are negative real roots.

4 Numerical Simulations

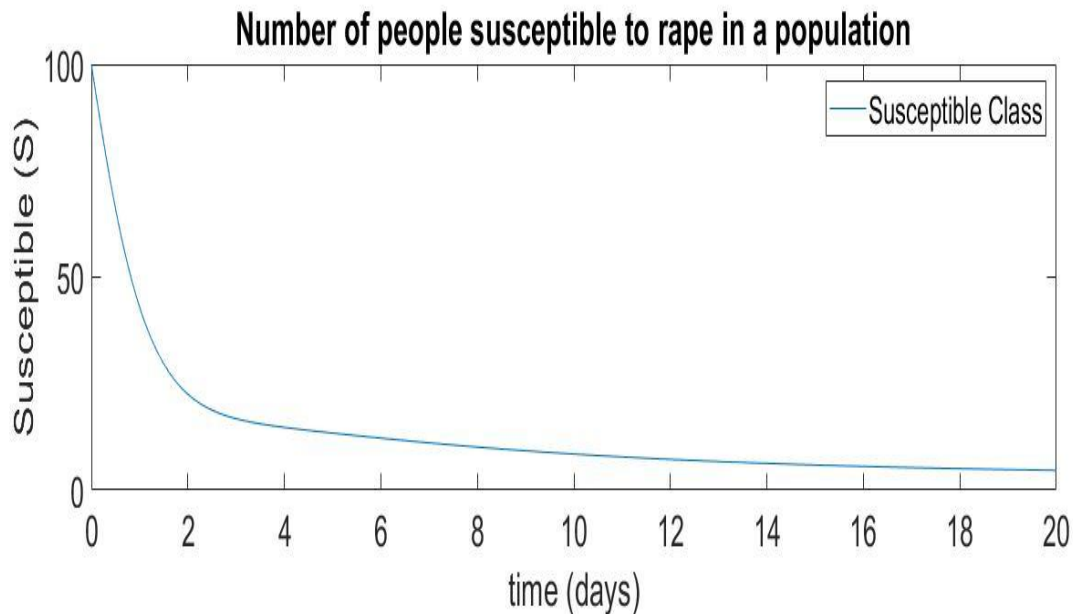


Figure 1: Graph of Susceptible Individuals to Rape cases in a Population. Parameter Values are: $\pi=3.142$, $P=0.5$, $\delta=0.90$, $\varrho=0.45$, $\beta=0.72$, $k_1=0.75$, $k_2=0.75$, $\mu=0.005$, $\varphi=0.67$, $\phi=0.81$, $\gamma=0.80$, $\xi=0.45$, with initial condition $(S(0), P(0), V(0), C(0), R(0)) = (100, 50, 50, 20, 10)$.

Figure 1 above showed that the susceptible population or individuals reduces slowly with respect time and move to either the culprit-victim individuals asymptotically.

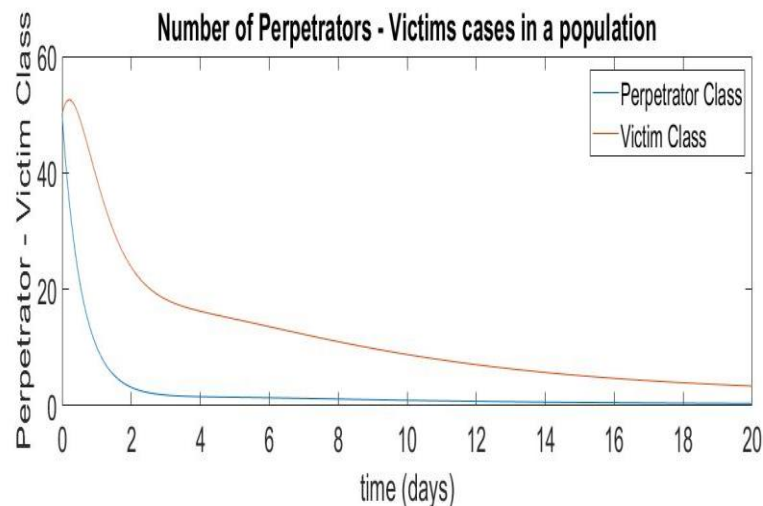


Figure 2: Graph of Rape Perpetrator-Victim cases in a Population. The parameter values are: $\pi=3.142$, $P=0.5$, $\delta=0.90$, $\varrho=0.45$, $\beta=0.72$, $k_1=0.75$, $k_2=0.75$, $\mu=0.005$, $\varphi=0.67$, $\phi=0.81$, $\gamma=0.80$, $\xi=0.45$, with initial condition $(S(0), P(0), V(0), J(0), R(0)) = (100, 50, 50, 20, 10)$.

In figure 2, the graph depicts that rape victims do not go to extinction because in the natural the stigma lives with them mostly females, while the perpetrators or culprits

sometimes vanishes as a result rape control measures and if the culprit are males.

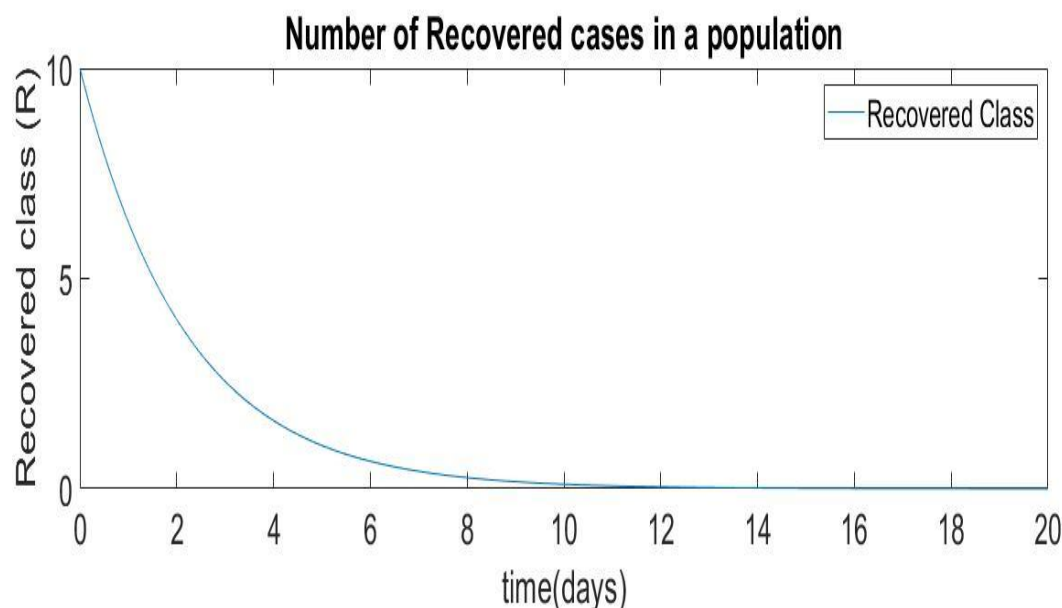


Figure 3: Graph of Recovered Individuals from Rape cases in a Population. The parameter values are: $\pi=3.142$, $P=0.5$, $\delta=0.90$, $\varrho=0$, $\beta=0.72$, $k_1=0.75$, $k_2=0.75$, $\mu=0.005$, $\varphi=0$, $\phi=0.81$, $\gamma=0.80$, $\tau=0.45$, with initial condition $(S(0), P(0), V(0), C(0), R(0)) = (100, 50, 50, 20, 10)$.

Figure 3 above showed that the recovered individuals reduce to annihilation asymptotically with respect time through natural death or migration to another environment.

4.1 Discussions of Findings

The study designed and discussed the asymptotic behavior of a new deterministic model for the dynamics of rape in a population with noise and human disturbance. The model equation (1-5) has a locally-asymptotically stable rape free equilibrium whenever the associated rape reproduction number (R_m) is less than one. This model undergoes the phenomenon of backward bifurcation, where the stable rape-free equilibrium co-exists with a stable rape endemic equilibrium. The analysis showed that this phenomenon is caused by the rates at which individuals who previously recovered from rape, following some intervention programme and individuals who previously were convicted and jailed for rape revert to being susceptible to rape.

It is also shown that the model has a globally-asymptotically stable rape-free equilibrium (DFE) when individuals who recover from rape and those previously convicted and jailed for rape never become susceptible to rape but have developed total resistance to rape. The Jailed model was identified as the general measure to curb and control rape, both at Covid-19 and other crisis respectively. To restrain rape globally, an SCPVR models was developed which implies Susceptible to rape S, Culprit C, Prey P, Convicted and Jailed because of rape committed V and recover from rape R, models. The study identifies two equilibrium points such as Pandemic free equilibrium state P_e and the rape free equilibrium state, R_e . The MATLAB ODE45 software was used for the

simulations. However, the graphs show that the susceptible class increases with time in a ratio of 1:1 and the recovered class goes into extinction with the presence of human disturbance and jail class which is the control measure of rape cases in any given population. The results and simulations mean that the rape culprit goes to life imprisonment because of the action. The simulation of this study with noise showed a sinusoidal and an exponential increase; which implies that the action of the rape culprit was high on the victim.

We can see from the line graph that the recovered class tends to zero with respect to time. We can also affirm that the model is a good for the control of rape cases in a population within a stipulated time. The numerical simulation graph of this is depicted in rape cases with respect to time. The perpetrator-victim classes decline with respect to time also. This shows that the proposed model can be used to control the high rise in number of rape cases during the Covid-19 which is realistic to real life situation.

5 Conclusion

In conclusion, the findings from the model analysis, shows that the convicted and jail for life imprisonment is the only possible measure to mitigate and control the impact of rape cases in any rape society. This study could be extended to multiple rape scenarios that led to pregnancy.

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The transmission dynamics of Lassa fever virus

I.D. Ofem* and H.O. Adagba†

Abstract

Lassa fever Virus (LFV) is a rare, acute arena viral hemorrhagic fever caused by Lassa virus, which is often fatal if untreated. In this research work, we developed a model that describes the dynamics of Lassa fever Virus (LFV) disease in human population compartments involved with vital dynamics (birth and death rates), incorporating exposed quarantine individuals of 6 to 21 days window period and treatment of infected individuals of 21 days (+) of infectious period in the trends of infection progression which are influenced by public health education campaign. A system of Nonlinear differential equations was formulated for the transmission dynamic of Lassa fever virus (LFV). The model has the Basic Reproduction Number (R_0) and effective reproductive number (R_e), the Stability analysis of the model indicated that the local and global disease-free equilibrium (DFE) is stable and indeed when $(R_0) < 1$ and unstable when $(R_0) > 1$. Numerical results show that the infected individuals should be quarantined and given health education campaigns before treatment, because both complement each other in reducing the spread of Lassa fever virus as well as increasing the rate of recovery of the infected individuals.

Keywords: Lassa fever virus; transmission dynamics; human population compartments

1 Introduction

In this chapter we reviewed works done by other researchers on mathematical modeling, especially on Lassa fever Virus (LFV). According to (World Health Organization, 2017) Lassa fever Virus (LFV) is an acute, hemorrhagic viral disease that occurs mainly in West African countries and was first discovered in the town of Lassa in the North-East Nigeria in 1969. Though it was first described in the 1950s, but the virus was not identified until 1969, when two missionary nurses died from it in the town of Lassa in Borno, Nigeria (Richmond and Baglolle 2003). Lassa fever is a zoonotic disease, i.e., it can be transmitted from an infected animal to a human. Multimammate rats of the genus *Mastomys* is the reservoir host of the virus (Walker *et al*, 2019). It is associated with acute and potentially fatal haemorrhagic illness caused by Lassa virus (Hallam *et al*, 2018). The Lassa fever virus is a zoonotic disease that is primarily transmitted to humans from contact with infected animals; usually a rodent of the genus *Mastomys* commonly known as multimammate rat (World Health Organization, 2017 &

* Corresponding Author. Department of Statistics, Cross River Institute of Technology and Management, Ugep, Cross Rivers, Nigeria; E-mail: ofeminyang29@gmail.com

† Department of Industrial Mathematics and Applied Statistics, Ebony State University, Abakiliki, Nigeria; E-mail: henela2012@gmail.com

2018). The *Mastomys* rats infected with Lassa fever do not become ill, but can transmit the virus to humans and other primates through the shed of their urine and faeces (World Health Organization, 2017). Lassa fever is known to be endemic in Benin, Ghana, Liberia, Mali, Sierra Leone, and Nigeria, but probably exists in other African countries as well (World Health Organization, 2017 & 2018). The Lassa fever virus usually last for 2-21 days in humans, but because the clinical course of the disease is so variable and nonspecific, clinical diagnosis of the disease in affected patients at early stages has been difficult (World Health Organization, 2017; Sulaiman & Ibrahim, *et al.*, 2018), and confirmatory laboratory diagnosis is not within the confines of many centers in West Africa (World Health Organization, 2017; Andrew, *et al.* (2013). About 80% of people infected with Lassa fever are asymptomatic (World Health Organization, 2017). The incubation period of the disease ranges from 6-21 days and the onset of the disease, when it is symptomatic, is usually gradual, starting with fever, general weakness, and malaise (World Health Organization, 2017).

After few days of infection, follows headache, sore throat, muscle pain, chest pain, nausea, vomiting, diarrhea, and abdominal pain (World Health Organization, 2017). In severe cases of the disease, facial swelling, fluid in the lung cavity, bleeding from the mouth, nose, vagina or gastrointestinal tract and low blood pressure may also occur (World Health Organization, 2017). Protein may also be noted in the urine of an infected person (World Health Organization, 2017). However, shock, seizures, tremor, disorientation, and coma may also be seen in the later stages of infection (World Health Organization, 2017). Deafness occurs in 25% of patients who survived the disease (World Health Organization, 2017). In half of the infected cases, hearing returns partially after 1-3 months of recovery, and transient hair loss and gait disturbance may occur during recovery (World Health Organization, 2017). Death usually occurs within the first 14 days of onset in fatal cases (World Health Organization, 2017). It is reported that, 1 out of each 5 infections results in severe disease case, where the virus affects several vital organs such as the liver, spleen and the kidneys (World Health Organization, 2017).

The disease is especially severe in late pregnancies with maternal death and or fetal loss occurring in more than 80% of the cases during third trimester (World Health Organization, 2017). The overall case-fatality rate is 1% and observed case-fatality rate among patients hospitalized with severe cases of the Lassa fever is 15% (World Health Organization, 2017). However, when the presence of the disease is confirmed in a community, prompt isolation of affected patients, good infection prevention and control practices, and rigorous contact tracing can stop outbreaks (World Health Organization, 2017). Early supportive care with rehydration and symptomatic treatment of the infected persons improves survival and chances of recovery (World Health Organization, 2017). The antiviral drug ribavirin seems to be an effective treatment for the Lassa fever virus if given early in the course of clinical illness (World Health Organization, 2018). There is no evidence to support the role of ribavirin as post-exposure prophylactic treatment for the Lassa fever (World Health Organization, 2018). Generally, there is no licensed vaccine for Lassa fever virus (World Health Organization, 2018; Thomas, *et al.*, 2005), opined that no experimental vaccine has completely protected non-human primates against Lassa fever.

The Lassa fever virus is usually transmitted to humans via contact with food or household items contaminated with rodents' urine or faeces (World Health Organization, 2017). Humans become infected with the Lassa fever virus from exposure to urine or faeces of an infected *Mastomys* rats (World Health Organization, 2017). The Lassa virus may also be transmitted among humans through direct contact with the blood, urine, faeces or other bodily secretions of an infected person with Lassa fever (World Health Organization, 2017). Currently, there is no epidemiological evidence supporting airborne spread of the Lassa fever virus among humans, but, person-to-person infections and laboratory transmission can also occur, especially in hospitals lacking adequate infection prevention

and control measures (World Health Organization, 2017). Person-to-person transmission occurs in both community and healthcare settings where the virus may be spread by contaminated medical equipment, such as re-used needles (World Health Organization, 2017). Sexual transmission of the Lassa fever virus has also been reported (World Health Organization, 2017). Infections in humans typically occurs by direct or indirect exposure to animal excrement through the respiratory or gastrointestinal tracts (Okuonghae D. et al, 2006). Inhalation of the tiny particles of infectious material (aerosol) is believed to be the most significant means of exposure (Okuonghae D. et al, 2006). It is possible to acquire the infection through broken skin or mucous membranes that are directly exposed to infectious materials and so, transmission from person-to-person has been established (World Health Organization, 2017; Okuonghae D. et al, 2006). Outbreaks of the Lassa fever virus occurs regularly in West Africa with the most recent one in Nigeria (Vanguard News, 2018). From 1 January 2018 through 18 March 2018, about 1495 suspected cases and 119 deaths have been reported in Nigeria from 20 States of the Federation including Anambra, Bauchi, Benue, Delta, Ebonyi, Edo, Ekiti, FCT Abuja, Gombe, Imo, Kaduna, Kogi, Lagos, Cross River, Nassarawa, Ondo, Osun, Plateau, Rivers and Taraba (World Health Organization, 2018).

During this period, 376 cases were confirmed, 9 were classified probable, 1084 were reported negative and 26 are pending; awaiting laboratory results (World Health Organization, 2018). Among the 376 confirmed and the 9 classified probable cases, 95 deaths were reported giving a case-fatality rate of the confirmed and probable cases to be 24.7% (World Health Organization, 2018). Similar reports about the current ongoing Lassa fever outbreaks and fatality cases in Nigeria can be found in (Ayodemola, 2018; Vanguard News, 2018). Since 1 January 2018, the number of Lassa fever cases increased from 10 to 70 weekly reported cases (World Health Organization, 2018). However, since mid-February 2018, there has been a downward trend in the weekly reported number of cases (World Health Organization, 2018). The Nigeria Center for Disease Control NCDC has built a laboratory in Ebonyi State that has the equipment needed to identify the Lassa fever virus, which is the fourth of its kind to be established by the institution in the country (Vanguard News, 2018).

Mathematical modeling in epidemiology provides an understanding of the mechanisms that influence the spread of diseases, and suggests control strategies. For some diseases, it is found that for a period of time, a part of the infectious class does not show the symptoms (Mohammed, AL-Smadi and Ghaleb, 2014). For modeling such diseases SEIR models are used (Mohammed, AL-Smadi and Ghaleb, 2014). A general SEIR model with vertical transmission for the dynamics of an infectious disease was studied by El-Sheikh and El-Marouf (2003), where a fraction p and q of the offspring from the exposed and the infectious classes, respectively, were assumed to be infected at birth. This study shows that some part of infectious class manifest symptoms. The work of El-Sheikh & El-Marouf (2003) showed that once the disease appears, it eventually persists at the unique endemic equilibrium level. They finally used mathematical tools of differential analysis, persistence theory, Hopf-Andronov-Poincaré bifurcation, and linear system theory to deduce the existence of a family of periodic solutions that bifurcate from a positive interior equilibrium.

A mathematical model for the transmission dynamics of Lassa fever virus can also be found in the work of Faniran (2017). In developing the model, Faniran (2017) neglected the latency/exposure period in the successive trends of infection progression in both humans and the animals. They also neglected human-to-human transmission of the virus in the human host and considered only animal-to-human transmission. This work will add knowledge to this model on human -to -human transmission of Lassa fever virus (LFV). Okuonghae and Okuonghae (2006) formulated a SIS model

coupled to a population of rat species, for the transmission of Lassa fever disease. The equilibrium states of the model were obtained and examined the endemic and epidemic situations. They further calculated the basic reproductive number and gave conditions for disease outbreak. Onuorah et al (2016) developed a Lassa fever model using the sex structure approach. Their model represented the transmission dynamics of the Lassa fever disease using a set of ordinary differential equations. They obtained the disease-free equilibrium. Their computation of basic reproduction number is 0.129 which is less than the one.

Akinpelu and Akinwande (2018) formulated a mathematical model for Lassa fever and Sensitivity analysis. The model was divided into five compartments susceptible (S), latent (L), infected (I), isolated (Is) and recovered (R) classes. The equilibrium states, basic reproduction number were obtained using generation matrix and their stabilities were analyzed using Descartes' rule of sign and comparison test. However, their work lacked global stability which this study has covered. In the work of Nwasuka et al (2019) A mathematical model is formulated and analyzed to study the controllability of Lassa fever incorporating separation of infected individuals and treatment measures. The model assumes that humans susceptible acquired the Infection via interaction with the infected rodent populations at a constant rate and also the model assumes that treatment is only given to separated human population without quarantining of exposed individuals. Therefore, this work covered quarantining of exposed individuals of 6 days to 21 days. Another mathematical model for the transmission dynamics of the Lassa fever infection was developed by Omale and Edibo (2015). In the development of the model, they also neglected the latency/exposure period in the successive trends of infection progression in both humans and the animals and considered re-infection after recovery from the treatment intervention in the humans. They considered linear incidence transmission in the animals and the force of infection in the model did not account for animal-to-human transmission. This work will cover latency/exposure period in the successive trends of infection.

A deterministic mathematical model for transmission dynamics of Lassa fever with quarantine policy was constructed in the work of Tolulope, Akinyemi and Bamidele (2015). In their work, they divided the infectious human population into two; the quarantined and un-quarantined humans and considered both as contributing to human-to-human transmission of the virus in the human host. They subdivided the animal population into adults and infant reservoirs and assumed only the adults contributes in animal-to-human transmission of the Lassa virus. In the model, they assumed humans recover with permanent immunity. This work assumed that human recovery is not with permanent immunity. The work of Onuorah et al (2016) subdivided the human host into males and females and the animal reservoirs into active and inactive reservoirs when developing their model. They incorporated sexual transmission of the virus among sexually active humans as one of the means of transmitting the disease in humans. Sensitivity analysis of parameters in the basic reproduction number of their model revealed that, the reproduction number is most sensitive to parameters representing human birth, condom efficacy and compliance rates. However, their work lacked a qualitative and numerical analysis which this study has covered. In the work of Adewale et al (2016), they developed a mathematical model for the transmission dynamics of the Lassa fever virus with proposed isolation of infectious humans as control strategy on the spread of the disease among humans. Most importantly, they showed that the disease free equilibrium is stable while the endemic equilibrium may or may not be stable depending on the model parameters. This model is important to this study, however, the researchers intend to use a different model which is the S, E, E_q, I, I_T, R model to determine the transmission dynamics of Lassa fever virus (LFV).

The model in James et al (2015) studied the transmission dynamics of Lassa fever virus in animal and humans. The SIR model in their work did not consider the exposed quarantining of

individuals between the 6 to 21 days window period in the transmission dynamics of the virus which this study covered. In Ojo et al (2021), the authors developed and critically analyzed a six compartmental model to study the effect of control variables on the control of Lassa fever in Nigeria. The population is stratified into the human population of susceptible, exposed, infectious, and recovered individuals, and also the rodent population of susceptible and infectious rodents. The model in Mark N. C. *et al* (2015) which is the bases of this work, studied a mathematical model on the dynamics of Lassa fever Virus (LFV) in human population. The model in their work consists of only five (5) compartments. They considered quarantining of infectious treated individuals after window period (i.e. 21 days ⁽⁺⁾) and neglected exposed quarantine individuals of 6 to 21days of window period of the model which this study covered. A number of gaps have been revealed in the foregoing literature, but it is not clear enough about the extent to which the Lassa fever virus (LFV) disease was analyzed. The study attempted to investigate the transmission dynamics of Lassa fever virus (LFV) whose numerical results show that the infected individuals should be quarantined and given health education campaigns before treatment, because both complement each other in reducing the spread of Lassa fever virus as well as increasing the rate of recovery of the infected individuals.

1.1 Aims and objectives of the study

The aim of this paper is to examines the transmission dynamics of Lassa fever Virus (LFV), and other specific objectives of the study are;

1. Carry out a detailed study of Lassa fever Virus (LFV)
2. Investigate the existence and stability of the equilibrium state of the model.
3. Interpret numerically, the results of the analysis.
4. Use the study in providing useful contribution that will aid in the awareness of the dynamics and possible control measures of the disease.

2 Model formulation

The population (N) is divided into six (6) compartments; susceptible individuals (S), Exposed individuals (E), Exposed quarantine (E_q), infected individuals (I), Infectious Treated (I_T), and Recovered individuals (R), So that, $N(t) = S(t) + E(t) + E_q(t) + I(t) + I_T(t) + R(t)$.

S/N	PARAMETERS	PARAMETERS DESCRIPTION
1	$S(t)$	Number of susceptible at time t
2	$E(t)$	Number of exposed at time t
3	(E_q)	Number of exposed quarantine at time t
4	(I)	Number of infected at time t
5	(I_T)	Number of infected treated at time
6	(R)	Number of recovered at time t
7	$N(t)$	Total population at time t

Table 1: Description of variables

S/ N	VARIABLE	VARIABLE DESCRIPTION
1	π	Recruitment rate into susceptible class
2	λ	Rate at which susceptible individuals move to exposed class
3	ϵ	Rate of maturity from exposed to the infected class
4	θ	Rate at which the recovered individuals move to susceptible class
5	τ_1	Rate at which exposed-quarantine individuals move to susceptible class.
6	τ_2	Rate at which exposed-quarantine individuals move to infectious class
7	ν	Rate at which infected individuals move to infectious treated class for treatment.
8	ρ	Rate at which Exposed individuals move to quarantine class
9	f	Rate at which treated infectious individuals recover from the disease
10	μ	Natural death rate
11	β	contact rate between the susceptible and infectious individuals
12	δ_1	Rate at which untreated infected individuals died due to infectious
13	δ_2	Rate at which infected individuals died during treatment
14	κ	proportion from S to I (fast progression)

Table 2: Description of Parameters

2.1 Assumptions of the model

The following assumptions were made during the study:

1. The mixing of individual is homogeneous, meaning that all individuals have equal likelihood of getting infected if they come in adequate contact with infectious individuals and the transmission of infection occurs with a standard incident (βSI)
2. Individuals in each group have homogeneous susceptibility but the susceptibilities of individuals from different groups are distinct and different.
3. We assumed that all recruits are neither infected with Lassa fever virus (LFV).
4. The rate at which recovered individuals become susceptible (θ) is negligible.
5. That some infectious individuals under treatment died as a result of the disease, while some recovered from the disease
6. That some individual died natural death

Model diagram of Lassa fever virus

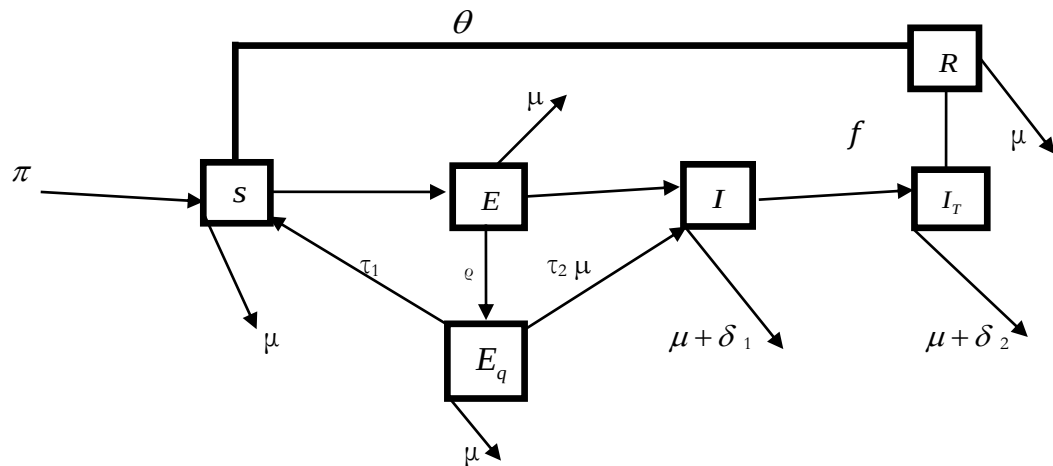


Figure 1. Schematic Diagram

Extended Model Equations

$$\begin{aligned}
 \frac{\partial S}{\partial t} &= \pi - (\lambda + \mu) + \theta R + \tau_1 E_q \\
 \frac{\partial E}{\partial t} &= \lambda S - (\mu + \epsilon + \rho) E \\
 \frac{\partial E_q}{\partial t} &= \rho E - (\mu + \tau_1 + \tau_2) E_q \\
 \frac{\partial I}{\partial t} &= \epsilon E - (\nu + (\mu + \delta_1)) I + \tau_2 E_q \\
 \frac{\partial I_T}{\partial t} &= \nu I - (f + (\mu + \delta_2)) I_T \\
 \frac{\partial R}{\partial t} &= f I_T - (\mu + \theta) R
 \end{aligned}
 \tag{1}$$

3 Model analysis

In this section, the model equations (1) will be analyzed quantitatively in order to understand the transmission dynamics of Lassa fever virus (LFV). We will therefore, consider the following proof; invariant region, positivity of solution, disease free equilibrium (DFE), endemic equilibrium (EE), basic reproduction number, effective reproduction number, local and global stability analysis of the model.

3.1 Invariant region

In this section, we think of the solutions as having six dimensions, so that at any instance of time (t), we have a vector of solutions with six elements (representing the state variables), real and positive solution. First, we established the positivity of invariant region or set to the above biological model (1) using the total population as $N = S + E + E_q + I + I_T + R$.

Theorem 3.1

The solutions of the system (1) are feasible for all $t > 0$, if they enter invariant region Ω .

Proof:

Let $\Omega = (S, E, E_q, I, I_T, R) \in R_+^6$ be any solution of the system (1) with non-negative initial conditions. Using the total population of our model as $N = S + E + E_q + I + I_T + R$.

Hence,

$$\frac{\partial N}{\partial t} = \frac{\partial S}{\partial t} + \frac{\partial E}{\partial t} + \frac{\partial E_q}{\partial t} + \frac{\partial I}{\partial t} + \frac{\partial I_T}{\partial t} + \frac{\partial R}{\partial t}.$$

Considering the force of infection $\lambda = \beta SI$ in our model equation (1), the above equation becomes

$$\frac{\partial N}{\partial t} = \pi - \mu N - dI, \quad (2)$$

so that,

$$\begin{aligned} \frac{\partial N}{\partial t} &\leq \pi - \mu N \\ \frac{\partial N}{\partial t} + \mu N &\leq \pi, \end{aligned}$$

where the integrating factor is given as;

$$\begin{aligned} I.F &= \ell^{\int \mu dt} \\ I.F &= \ell^{\mu t}. \end{aligned}$$

Thus, the above differential equation become;

$$\frac{\partial}{\partial t} (N \ell^{\mu t}) \leq \pi \ell^{\mu t}$$

$$\begin{aligned}\partial(N\ell^{\mu t}) &\leq \pi\ell^{\mu t} dt \\ \int \partial(N\ell^{\mu t}) &\leq \pi \int \ell^{\mu t} dt + C\end{aligned}$$

The above integral becomes

$$\begin{aligned}N\ell^{\mu t} &\leq \frac{\pi}{\mu}\ell^{\mu t} + C \\ N(t) &\leq \frac{\pi}{\mu} + C\ell^{-\mu t},\end{aligned}\tag{3}$$

where C is the constant of integration. Given that at time $t = 0$, $N(t) = N(0)$, then

$$\begin{aligned}N(0) &\leq \frac{\pi}{\mu} + C \\ C &= N(0) - \frac{\pi}{\mu}.\end{aligned}$$

Hence, equation (4) becomes

$$N(t) \leq \left(1 - \ell^{-\mu t}\right) \frac{\pi}{\mu} + N(0)\ell^{-\mu t}.\tag{4}$$

We therefore, apply Birkoff and Rota's theorem on equation (5) as $t \rightarrow \infty$, we obtain

$N(t) \leq \frac{\pi}{\mu}$, hence all the feasible solutions of the model (2) enter the region or set of

$\Omega = \left\{ (S, E, E_q, I, I_T, R) \leq \frac{\pi}{\mu} \right\}$ which is positive invariant, attracting and its sufficient to consider

solution in Ω . Thus, in this region or set of our model is biologically feasible whenever $N > \frac{\pi}{\mu}$,

then $\frac{\partial N}{\partial t} < 0$ which mean that the population reduces asymptotically to the carrying capacity and

whenever $N \leq \frac{\pi}{\mu}$ every solution with initial condition in Ω in that region for $t > 1$, so the model

is well posed to Ω and biologically meaningful. This ends the proof.

3.2 Positivity of solution

For the system S, E, E_q, I, I_T, R to be biologically meaningful, it is important to prove that all solutions with non-negative initial conditions will remain positive. Here, we will take each differential equations of the dynamical system (2) to illustrates that the corresponding solutions are positive.

Theorem 3.2

Let the initial data be

$\{S(0), E(0), E_q(0), I(0), I_T(0), R(0) \geq 0\} \in R_+^6$. Then, the solution set $\{S(t), E(t), E_q(t), I(t), I_T(t), R(t)\}$ of the system (1) is positive for all $t > 0$.

Proof:

From the first equation of the model system (1)

$$\frac{\partial S}{\partial t} = \pi - (\lambda + \mu)S + \theta R + \tau_1 E_q$$

$$\Rightarrow \dot{S} = \pi + \theta R + \tau_1 E_q - (\lambda + \mu)S$$

$$\dot{S} \geq \pi + \theta R + \tau_1 E_q - (\lambda + \mu)S$$

$$\frac{\dot{S}}{S} \geq \pi + \theta R + \tau_1 E_q - (\lambda + \mu)$$

After integrating we have,

$$\dot{S}(t) \geq S(0) \left(e^{\int -(\lambda + \mu) dt} \right) \geq 0$$

Then, $(\lambda + \mu) \geq 0$

Using the second equation of system (2), we have

$$\frac{\partial E}{\partial t} = \lambda S - (\mu + \epsilon + \rho)E$$

$$\Rightarrow \dot{E} = \lambda S - (\mu + \epsilon + \rho)E$$

$$\frac{\dot{E}}{E} = \lambda S - (\mu + \epsilon + \rho)$$

After integration we get,

$$\dot{E}(t) \geq E(0) \left(e^{\int -(\mu + \epsilon + \rho) dt} \right) \geq 0$$

Then, $(\mu + \epsilon + \rho) \geq 0$

Again from the third equation of system (2), we have

$$\frac{\partial E_q}{\partial t} = \rho E - (\mu + \tau_1 + \tau_2)E_q$$

$$\Rightarrow \dot{E}_q = \rho E - (\mu + \tau_1 + \tau_2)E_q$$

$$\frac{\dot{E}_q}{E_q} = \rho E - (\mu + \tau_1 + \tau_2)$$

After integration we have

$$\dot{E}_q(t) \geq E_q(0) \left(e^{\int^{-(\mu+\tau_1+\tau_2)} dt} \right) \geq 0$$

$$\text{Then, } (\mu + \tau_1 + \tau_2) \geq 0.$$

From the fourth equation of model (1), we get

$$\frac{\partial I}{\partial t} = E + \tau_2 E_q - (\nu + (\mu + \delta_1))I$$

$$\Rightarrow \dot{I} = E + \tau_2 E_q - (\nu + (\mu + \delta_1))I$$

$$\frac{\dot{I}}{I} = E + \tau_2 E_q - (\nu + (\mu + \delta_1))$$

After integrating we have

$$\dot{I}(t) \geq I(0) \left(e^{\int^{-(\nu+(\mu+\delta_1))} dt} \right) \geq 0$$

$$\text{Then, } (\nu + (\mu + \delta_1)) \geq 0.$$

From the fifth equation of our model (2)

$$\frac{\partial I_T}{\partial t} = \nu I - (f + (\mu + \delta_2))I_T$$

$$\Rightarrow \dot{I}_T = \nu I - (f + (\mu + \delta_2))I_T$$

$$\frac{\dot{I}_T}{I_T} = \nu I - (f + (\mu + \delta_2))$$

After integrating we get

$$\dot{I}_T \geq I_T(0) \left(e^{\int^{-(f+(\mu+\delta_2))} dt} \right) \geq 0$$

$$\text{Then, } (f + (\mu + \delta_2)) \geq 0.$$

From the sixth equation of model system (1)

$$\frac{\partial R}{\partial t} = fI_T - (\mu + \theta)R$$

$$\dot{R} = fI_T - (\mu + \theta)R$$

$$\frac{\dot{R}}{R} = fI_T - (\mu + \theta)$$

After integrating we have,

$$\dot{R} \geq R(0) \left(e^{\int -(\mu + \theta) dt} \right) \geq 0$$

Then,

$$(\mu + \theta) \geq 0$$

Therefore, all the solutions of the equations (2) are positive for all $t > 0$ which ends the proof.

3.3 Existence of diseases free equilibrium (DFE) points

Equilibrium state in our model represent the absent of Lassa fever virus in a given susceptible population. Thus, all the infected classes will be set to zero and the entire population will comprise of only susceptible individuals over those who died natural death.

Theorem 3.3

The disease-free equilibrium point of our model exist at the point where

$$e_0 = (S, E, E_q, I, I_T, R) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0 \right)$$

Proof:

At equilibrium state the rate of change of each variable is equal to zero, that is;

$$\frac{\partial S}{\partial t} = \frac{\partial E}{\partial t} = \frac{\partial E_q}{\partial t} = \frac{\partial I}{\partial t} = \frac{\partial I_T}{\partial t} = \frac{\partial R}{\partial t} = 0$$

$$\text{Let, } (S, E, E_q, I, I_T, R) = (S^0, E^0, E_q^0, I^0, I_T^0, R^0)$$

Then from the first equation of our model (2)

$$\frac{\partial S}{\partial t} = \pi - (\lambda + \mu)S + \theta R + \tau_1 E_q$$

The rate of change of R, E_q is equal to zero, therefore, taking $R = E_q = 0$

$$\Rightarrow \pi - (\lambda + \mu)S^0 = 0$$

$$S^0 = \frac{\pi}{\mu + \lambda}$$

Taking $\lambda = \beta SI$ as the force of infection we have,

$$S^0 = \frac{\pi}{\mu + \beta SI}$$

Taking the rate of change of (I) to be equal to zero i.e. $I = 0$, we have

$$S^0 = \frac{\pi}{\mu} \quad (5)$$

Also taking the rate of change of E, E_q, I, I_T, R of our model (2) to be equal to zero i.e. $E = E_q = I = I_T = R = 0$, then the 2nd to sixth equations of our model become zero (0).

Hence,

$$\varepsilon_0 = (S, E, E_q, I, I_T, R) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0 \right). \quad (6)$$

Therefore, equation (6) represents the state in which there is no infection in the population and it is known as the disease-free equilibrium point (DFE).

3.4 Existence of endemic equilibrium (EE) points

The endemic equilibrium state of the system (1) is the state where the disease cannot be totally eradicated but remains in the population. For the disease to persist in the population, the susceptible class, exposed class, exposed quarantined class, infected class, infected treatment class and Recovered class must not be zero at equilibrium state. In order to obtain the endemic equilibrium state, we solve equation (1) of our model simultaneously taking into account of the fact that

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

Theorem 3.4

If $(S^*, E^*, E_q^*, I^*, I_T^*, R^*)$ is equilibrium state, then

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

Proof:

From equation two of our model (1)

$$\begin{aligned} \frac{\partial E}{\partial t} &= \lambda S - (\mu + \epsilon + \rho)E \\ \Rightarrow \lambda S^* - (\mu + \epsilon + \rho)E^* &= 0 \\ E^* &= \frac{\lambda S^0}{(\mu + \epsilon + \rho)} \end{aligned}$$

We substitute equation (8) into (9) to have thus;

$$\begin{aligned} E^* &= \frac{\lambda \frac{\pi}{\mu}}{(\mu + \epsilon + \rho)} \\ E^* &= \frac{\lambda \pi}{\mu(\mu + \epsilon + \rho)} \end{aligned} \quad (8)$$

We substitute (8) into (3) of our model (2) thus;

$$\begin{aligned}
 \frac{\partial E_q}{\partial t} &= \rho E - (\mu + \tau_1 + \tau_2)E_q \\
 \Rightarrow \rho E - (\mu + \tau_1 + \tau_2)E_q &= 0 \\
 E_q^* &= \frac{\rho E^*}{(\mu + \tau_1 + \tau_2)} \\
 E_q^* &= \frac{\rho \lambda \pi}{\mu(\mu + \epsilon + \rho)} \\
 E_q^* &= \frac{\rho \lambda \pi}{\mu(\mu + \epsilon + \rho)} \times \frac{1}{(\mu + \tau_1 + \tau_2)} \\
 E_q^* &= \frac{\rho \lambda \pi}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)}. \tag{9}
 \end{aligned}$$

We substitute (8) and (9) into fourth equation of model (1) to yield

$$\begin{aligned}
 \frac{\partial I}{\partial t} &= \epsilon E - (\nu + (\mu + \delta_1))I + \tau_2 E_q \\
 \Rightarrow \epsilon E - (\nu + (\mu + \delta_1))I + \tau_2 E_q &= 0 \\
 I^* &= \frac{\epsilon E^* + \tau_2 E_q^*}{(\nu + (\mu + \delta_1))} \\
 I^* &= \frac{\epsilon \left(\frac{\lambda \pi}{\mu(\mu + \epsilon + \rho)} \right) + \tau_2 \left(\frac{\rho \lambda \pi}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)} \right)}{(\nu + (\mu + \delta_1))} \\
 I^* &= \frac{\epsilon \lambda \pi (\mu + \tau_1 + \tau_2) + \tau_2 \rho \lambda \pi}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)} \times \frac{1}{(\nu + (\mu + \delta_1))} \\
 I^* &= \frac{\lambda \pi [\epsilon (\mu + \tau_1 + \tau_2) + \tau_2 \rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(\nu + (\mu + \delta_1))}. \tag{10}
 \end{aligned}$$

We substitute (10) into fifth equation of our model (2) to yield

$$\begin{aligned}
 \frac{\partial I_T}{\partial t} &= \nu I - (f + (\mu + \delta_2))I_T \\
 \Rightarrow \nu I - (f + (\mu + \delta_2))I_T &= 0 \\
 I_T^* &= \frac{\nu I^*}{(f + (\mu + \delta_2))} \\
 I_T^* &= \frac{\nu \left(\frac{\lambda \pi [\epsilon (\mu + \tau_1 + \tau_2) + \tau_2 \rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(\nu + (\mu + \delta_1))} \right)}{(f + (\mu + \delta_2))}
 \end{aligned}$$

$$I_T^* = \frac{v\lambda\pi[(\mu + \tau_1 + \tau_2) + \tau_2\rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))}. \quad (11)$$

We substitute (11) into sixth equation of our model (2) to have

$$\begin{aligned} \frac{\partial R}{\partial t} &= fI_T - (\mu + \theta)R \\ \Rightarrow fI_T - (\mu + \theta)R &= 0 \\ R^* &= \frac{fI_T^*}{(\mu + \theta)} \\ R^* &= \frac{f\left(\frac{v\lambda\pi[(\mu + \tau_1 + \tau_2) + \tau_2\rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))}\right)}{(\mu + \theta)} \\ R^* &= \frac{fv\lambda\pi[(\mu + \tau_1 + \tau_2) + \tau_2\rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))(\mu + \theta)}. \end{aligned} \quad (12)$$

Finally, we substitute (12) into first of our model (1) to yield

$$\begin{aligned} \frac{\partial S}{\partial t} &= \pi - (\lambda + \mu) + \theta R + \tau_1 E_q \\ \pi - (\lambda + \mu)S^* + \theta R^* + \tau_1 E_q^* &= 0 \\ S^* &= \frac{\pi + \theta R^* + \tau_1 E_q^*}{(\lambda + \mu)} \\ S^* &= \frac{\pi + \theta\left(\frac{fv\lambda\pi[(\mu + \tau_1 + \tau_2) + \tau_2\rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))(\mu + \theta)}\right) + \tau_1\left(\frac{\rho\lambda\pi}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)}\right)}{(\lambda + \mu)} \\ S^* &= \frac{\pi + \theta\frac{fv\lambda\pi[(\mu + \tau_1 + \tau_2) + \tau_2\rho]}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))(\mu + \theta)} + \frac{\tau_1\rho\lambda\pi}{\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)}}{(\lambda + \mu)} \\ S^* &= \frac{\pi}{(\lambda + \mu)} + \frac{\theta fv\lambda\pi[(\mu + \tau_1 + \tau_2) + \tau_2\rho]}{\mu(\mu + \epsilon + \rho)(v + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))(\mu + \theta)} + \frac{\tau_1\rho\lambda\pi}{\mu(\lambda + \mu)(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)} \\ S^* &= \frac{\pi\mu(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(\mu + \theta) + \theta fv\lambda\pi\epsilon(\mu + \tau_1 + \tau_2) + \tau_2\rho + \tau_1\rho\lambda\pi(v + (\mu + \delta_1))(\mu + \theta)}{\mu(\lambda + \mu)(\mu + \epsilon + \rho)(\mu + \tau_1 + \tau_2)(v + (\mu + \delta_1))(f + (\mu + \delta_2))(\mu + \theta)} \end{aligned}$$

$$\begin{aligned}
 S^* &= \frac{\pi\mu(\mu+\epsilon+\rho)(\mu+\tau_1+\tau_2)(v+(\mu+\delta_2))(\mu+\theta)+\theta f v \lambda [(\mu+\tau_1+\tau_2)+\tau_2\rho]}{\mu(\lambda+\mu)(\mu+\epsilon+\rho)(\mu+\tau_1+\tau_2)(v+(\mu+\delta_1))(f+(\mu+\delta_2))(\mu+\theta)} \\
 S^* &= \frac{\pi\mu(\mu+\theta)(\mu+\epsilon+\rho)(\mu+\tau_1+\tau_2)(v+(\mu+\delta_1))(f+(\mu+\delta_2))+\tau_1\rho\lambda\pi [((v+(\mu+\delta_1))(f+(\mu+\delta_2)))]}{\mu(\lambda+\mu)(\mu+\epsilon+\rho)(\mu+\tau_1+\tau_2)(v+(\mu+\delta_1))(f+(\mu+\delta_2))(\mu+\theta)} \\
 S^* &= \frac{+\theta f v \pi \lambda [(\mu+\tau_1+\tau_2)+\tau_2\rho]}{\mu(\lambda+\mu)(\mu+\epsilon+\rho)(\mu+\tau_1+\tau_2)(v+(\mu+\delta_1))(f+(\mu+\delta_2))(\mu+\theta)}
 \end{aligned}
 \tag{13}$$

Hence, equations (8), (9), (10), (11), (12) and (13) represents the state in which there is an infection in the population and it is known as the endemic equilibrium point (EE).

3.5 Basic reproduction number (R_0)

The basic reproduction number denoted by R_0 is an important parameter used to determine how long an infectious disease will prevail or can last in a given population, when $R_0 < 1$. It means an average infected individual produces less than one new secondary case during the infectious period and this implies that with time the disease will die out of the population, thereby giving it a clean health bill. But if $R_0 > 1$, it means the disease will persist in the population. So, for the disease to die out of the population, R_0 must be less than one. Therefore, reproduction number represents the rate of spreading of an infection in a given susceptible population, hence considering model system (1) without control measures, the infectious classes are E, E_q, I, I_T .

To derive the basic reproduction number R_0 of the disease-free equilibrium (DFE), we employ the next generation operator technique described by Diekmann and Heesterbeek (2000), and which was subsequently analyzed by Vanden and Watmough (2002). This method is described as follows; assuming that there are n compartments so that the first m compartments correspond to infected individuals. Let $f_i(x)$ be the rate of appearance of new infectious in compartment i and $V_i^+(x)$ be the rate of transfer of individuals into compartment i by all other means, other than the endemic. Also let $V_i^-(x)$ be the transfer of individuals out of the compartment i . The disease transmission model consists of the system of equation $x_i' = f_i(x) - V_i^+(x)$ where $V_i^+(x) = V_i^-(x) - V_i^+(x)$.

One other important step is to obtain the disease-free equilibrium point x_0 , we therefore, compute matrices F and V which are $m \times m$ matrices, where m represents the infected classes, define by $F = \left[\frac{\partial F_i}{\partial x_j}(x_0) \right]$ and $V = \left[\frac{\partial V_i}{\partial x_j}(x_0) \right]$ With $1 \leq i, j \leq m$, and F is a non-negative and V is a non-singular M matrix, (a matrix with inverse, belong to the class of positive matrices). Since F is non-negative and V is non-singular, then V^{-1} is non-negative and FV^{-1} is also non-negative. We then compute matrix FV^{-1} , defined as the next generation matrix (Diekmann et al., 1990). This is given as $R_0 = \rho(K)$, where $K = FV^{-1}$, Computing reproduction number, we rewrite equations (1) of our model starting with the Exposed compartment of the population: E, E_q, I, I_T, S, R and then followed by the uninfected classes, then the model equations become;

$$\left. \begin{aligned} \frac{\partial E}{\partial t} &= \lambda S - (\mu + \epsilon + \rho)E \\ \frac{\partial E_q}{\partial t} &= \rho E - (\mu + \tau_1 + \tau_2)E_q \\ \frac{\partial I}{\partial t} &= \epsilon E - (\nu + (\mu + \delta_1))I + \tau_2 E_q \\ \frac{\partial I_T}{\partial t} &= \nu I - (f + (\mu + \delta_2))I_T \\ \frac{\partial S}{\partial t} &= \pi - (\lambda + \mu) + \theta R + \tau_1 E_q \\ \frac{\partial R}{\partial t} &= f I_T - (\mu + \theta)R \end{aligned} \right\} \quad (14)$$

Considering our model (14) and only the infectious classes (E, E_q, I, I_T) only, we then define f_i and V_i to compute R_0 as follows;

$$f_i = F = \begin{bmatrix} \beta SI \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

So that

$$\frac{\partial f_i}{\partial x_j} \varepsilon_0 = F = \begin{bmatrix} 0 & 0 & \beta S & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Also

$$V_i = V = \begin{bmatrix} (\mu + \epsilon + \rho)E \\ -\rho E + (\mu + \tau_1 + \tau_2)E_q \\ -\epsilon E + \tau_2 E_q + (\nu + (\mu + \delta_1))I \\ -\nu I + (f + (\mu + \delta_2))I_T \end{bmatrix}$$

where,

$$K_1 = \mu + \epsilon + \rho,$$

$$K_2 = \mu + \tau_1 + \tau_2$$

$$K_3 = \nu + (\mu + \delta_1)$$

$$K_4 = f + (\mu + \delta_2)$$

We take the partial derivative of ν with respect to E, E_q, I, I_T we have

$$\frac{\partial f_i}{\partial x_j} \varepsilon_0 = V = \begin{bmatrix} K_1 & 0 & 0 & 0 \\ -\rho & K_2 & 0 & 0 \\ -\epsilon & -\tau_2 & K_3 & 0 \\ 0 & 0 & -\nu & K_4 \end{bmatrix}.$$

We form the matrix of the co-factor of the determinant

$$V = \begin{bmatrix} K_2 K_3 & -\rho K_3 & -\rho \tau_2 + \epsilon K_2 & K_2 K_3 \\ 0 & K_1 K_3 & K_1 \tau_2 & 0 \\ 0 & 0 & K_1 K_2 & 0 \\ K_2 K_3 & \rho K_3 & -\rho \tau_2 + \epsilon K_2 & K_2 K_3 \end{bmatrix}.$$

We take the adjoin of the matrix V to give

$$V^T = \begin{bmatrix} K_2 K_3 & 0 & 0 & K_2 K_3 \\ \rho K_3 & K_1 K_3 & 0 & \rho K_3 \\ \rho \tau_2 + \epsilon K_2 & K_1 \tau_2 & K_1 K_2 & \rho \tau_2 + \epsilon K_2 \\ K_2 K_3 & 0 & 0 & K_2 K_3 \end{bmatrix}.$$

We form the inverse of the matrix V

$$V^{-1} \frac{1}{K_1 K_2 K_3} = \begin{bmatrix} \frac{1}{K_1} & 0 & 0 & \frac{1}{K_1} \\ \frac{\rho}{K_1 K_2} & \frac{1}{K_2} & 0 & \frac{\rho}{K_1 K_2} \\ \frac{\rho \tau_2 + \epsilon}{K_1 K_3} & \frac{\tau_2}{K_2 K_3} & \frac{1}{K_3} & \frac{\rho \tau_2 + \epsilon}{K_1 K_3} \\ \frac{1}{K_1} & 0 & 0 & \frac{1}{K_1} \end{bmatrix}.$$

Furthermore,

$$|FV^{-1} - \lambda I| = \begin{vmatrix} \frac{\beta S \rho}{K_1 K_2 K_3} - \lambda & 0 & 0 & \frac{\beta S}{K_1 K_2 K_3} \\ 0 & 0 - \lambda & 0 & 0 \\ 0 & 0 & 0 - \lambda & 0 \\ 0 & 0 & 0 & 0 - \lambda \end{vmatrix} = 0 \quad (15)$$

Then, $\lambda^3 \left(\frac{\beta S \rho}{K_1 K_2 K_3} - \lambda \right) = 0$.

Therefore, either $\lambda^3 = 0$ or $\lambda = \frac{\beta S \rho}{K_1 K_2 K_3}$,

From (15), we obtain four eigenvalues which are by; $\lambda_1 = \frac{\beta S \rho}{K_1 K_2 K_3}$,

$$\lambda_2 = 0, \lambda_3 = 0 \text{ and } \lambda_4 = 0$$

Clearly, λ_1 is the dominant eigenvalue and it becomes the basic reproduction number of our model (2). Therefore, the basic associated reproduction number is

$$(R_0) = \rho(FV^{-1}) = \frac{\beta S \rho}{K_1 K_2 K_3} = 1.20 > 1$$

3.6 Effective reproductive number (R_e)

We incorporate quarantine, health education campaign and treatment as control or intervention strategy to eliminate Lassa fever virus (LFV) disease. When these control measures are deployed to a model, the basic reproduction number of the infectious

disease becomes the effective reproduction (R_e), [27]. Considering our model (14) and the infectious classes (E, E_q, I, I_T) only, we compute R_e as follows;

$$f_i = F = \begin{bmatrix} \beta(1-\phi)SI \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

So that,

$$\frac{\partial f_i}{\partial x_j} \varepsilon_0 = F = \begin{bmatrix} 0 & 0 & \beta(1-\phi)S & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Also,

$$V_i = V = \begin{bmatrix} (\mu + \epsilon + \rho)(1 - \phi)E \\ -\rho(1 + \phi)E + (\mu + \tau_1 + \tau_2)(1 + \phi)E_q \\ -\epsilon(1 + \phi)E + \tau_2(1 + \phi)E_q + (\nu + (\mu + \delta_1)(1 + \phi)I \\ -\nu(1 + \phi)I + (f + (\mu + \delta_1)(1 + \phi)I_T \end{bmatrix},$$

where,

$$K_1 = (\mu + \epsilon + \rho)(1 + \phi)$$

$$K_2 = (\mu + \tau_1 + \tau_2)(1 + \phi) \quad K_3 = \nu + (\mu + \delta_1)(1 + \phi)$$

$$K_4 = f + (\mu + \delta_2)(1 + \phi)$$

We take the partial derivative of ν with respect to E, E_q, I, I_T we have;

$$\frac{\partial f_i}{\partial x_j} \varepsilon_0 = V = \begin{bmatrix} K_1(1 + \phi) & 0 & 0 & 0 \\ -\rho(1 + \phi) & K_2(1 + \phi) & 0 & 0 \\ -\epsilon(1 + \phi) & -\tau_2(1 + \phi) & K_3(1 + \phi) & 0 \\ 0 & 0 & -\nu(1 + \phi) & K_4(1 + \phi) \end{bmatrix}$$

We form the matrix of the co-factor of the determinant

$$V = \begin{bmatrix} K_2K_3(1 + \phi) & -\rho K_3(1 + \phi) & -\rho\tau_2(1 + \phi) + \epsilon K_2(1 + \phi) & K_2K_3(1 + \phi) \\ 0 & K_1K_3(1 + \phi) & K_1\tau_2(1 + \phi) & 0 \\ 0 & 0 & K_1K_2(1 + \phi) & 0 \\ K_2K_3(1 + \phi) & \rho K_3(1 + \phi) & -\rho\tau_2(1 + \phi) + \epsilon K_2(1 + \phi) & K_2K_3(1 + \phi) \end{bmatrix}$$

We take the adjoin of the matrix V to give

$$V^T = \begin{bmatrix} K_2K_3(1 + \phi) & 0 & 0 & K_2K_3(1 + \phi) \\ \rho K_3(1 + \phi) & K_1K_3(1 + \phi) & 0 & \rho K_3(1 + \phi) \\ \rho\tau_2(1 + \phi) + \epsilon K_2(1 + \phi) & K_1\tau_2(1 + \phi) & K_1K_2(1 + \phi) & \rho\tau_2(1 + \phi) + \epsilon K_2(1 + \phi) \\ K_2K_3(1 + \phi) & 0 & 0 & K_2K_3(1 + \phi) \end{bmatrix}$$

We form the inverse of the matrix V

$$V^{-1} \frac{1}{K_1 K_2 K_3 (1+\phi)} = \begin{bmatrix} \frac{1}{K_1(1+\phi)} & 0 & 0 & \frac{1}{K_1(1-\phi)} \\ \frac{\rho(1-\phi)}{K_1 K_2 (1+\phi)} & \frac{1}{K_2(1+\phi)} & 0 & \frac{\rho}{K_1 K_2} \\ \frac{\rho \tau_2 (1-\phi) + \epsilon (1-\phi)}{K_1 K_3 (1+\phi)} & \frac{\tau_2 (1-\phi)}{K_2 K_3 (1+\phi)} & \frac{1}{K_3 (1+\phi)} & \frac{\rho \tau_2 (1-\phi) + \epsilon (1-\phi)}{K_1 K_3 (1+\phi)} \\ \frac{1}{K_1(1+\phi)} & 0 & 0 & \frac{1}{K_1(1+\phi)} \end{bmatrix}$$

Furthermore,

$$|FV^{-1} - \lambda I| = \begin{vmatrix} \frac{\beta S \rho (1-\phi)}{K_1 K_2 K_3 (1+\phi)} - \lambda & 0 & 0 & \frac{\beta S (1-\phi)}{K_1 K_2 K_3 (1+\phi)} \\ 0 & 0 - \lambda & 0 & 0 \\ 0 & 0 & 0 - \lambda & 0 \\ 0 & 0 & 0 & 0 - \lambda \end{vmatrix} = 0 \quad (16)$$

Then, $\lambda^3 \left(\frac{\beta S \rho (1-\phi)}{K_1 K_2 K_3 (1+\phi)} - \lambda \right) = 0$.

Therefore, either $\lambda^3 = 0$ or $\lambda = \frac{\beta S \rho (1-\phi)}{K_1 K_2 K_3 (1+\phi)}$,

From (16), we obtain four eigenvalues which are by; $\lambda_1 = \frac{\beta S \rho (1-\phi)}{K_1 K_2 K_3 (1+\phi)}$,

$$\lambda_2 = 0, \lambda_3 = 0 \text{ and } \lambda_4 = 0$$

Clearly, λ_1 is the dominant eigenvalue and it becomes the effective reproduction number (R_e) of our model (2). Therefore, the basic associated reproduction number is

$$(R_e) = \frac{\beta S \rho (1-\phi)}{K_1 K_2 K_3 (1+\phi)} = 0.85 < 1.$$

Hence, this implies that, since $R_e = 0.85 < 1$, therefore, quarantine, health education campaigns can reduce the spread of Lassa fever virus (LFV) disease. However, when quarantine and health education campaigns are given to all individuals to reduce the spread of Lassa fever virus, it increases the effectiveness of the treatment given to the infected individuals, hence increasing the recovery rate (θ), also reduces the rate of getting infected (β), reducing the rate at which untreated infected individuals died due to infectious (δ_1) and the rate at which infected individuals died during treatment (δ_2). Thus, the effective reproduction number with quarantine and health education campaign $R_e = 0.85 < 1$ and is less than the basic reproduction number

$R_0 = 1.20$, i.e. ($R_e < R_0$), it implies that Lassa fever virus (LFV) can be controlled in a complete susceptible population (see figure 4.2).

3.7 Local stability analysis of Lassa fever disease free equilibrium (DFE)

Local stability of an equilibrium point means that, if a system is put somewhere near the point, then the system will move itself to the equilibrium point in some time. Epidemiologically, this implies that Lassa fever virus (LFV) will be eliminated from the population whenever $R_0 < 1$ if the initial size of the sub-populations is in the basin of the disease-free equilibrium (DFE), that is a small influx of Lassa fever virus (LFV) infectious individuals into the community will not generate a large Lassa fever virus outbreak, and the disease will die out in time.

Theorem 3.7

Lassa fever Virus free equilibrium of system (2) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

To analyze the local stability of the disease-free equilibrium (DFE) and established theorem (3.8.7), we obtain the Jacobean matrix of the Lassa fever model (1) becomes.

$$\left. \begin{aligned} f_1 &= \pi - (\lambda + \mu)S + \theta R + \tau_1 E_q \\ f_2 &= \lambda S - (\mu + \epsilon + \rho)E \\ f_3 &= \rho E - (\mu + \tau_1 + \tau_2)E_q \\ f_4 &= \epsilon E - (\nu + (\mu + \delta_1))I + \tau_2 E_q \\ f_5 &= \nu I - (f + (\mu + \delta_2))I_T \\ f_6 &= fI_T - (\mu + \theta)R \end{aligned} \right\} \quad (14)$$

The Jacobean matrix of our model (14) is describe as follows

$$J(DFE) = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial E_q} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial I_T} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial E_q} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial I_T} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial E_q} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial I_T} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial E_q} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial I_T} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial E_q} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial I_T} & \frac{\partial f_5}{\partial R} \\ \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial E_q} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial I_T} & \frac{\partial f_6}{\partial R} \end{bmatrix}$$

$$J(DFE) = \begin{bmatrix} -(\beta S + \mu) & 0 & \tau_1 & 0 & 0 & 0 \\ \beta S & -(\mu + \epsilon + \rho) & 0 & 0 & 0 & 0 \\ 0 & \rho & -(\mu + \tau_1 + \tau_2) & 0 & 0 & 0 \\ 0 & \epsilon & 0 & -(\nu + (\mu + \delta_1)) & 0 & 0 \\ 0 & 0 & 0 & 0 & -(f + (\mu + \delta_2)) & 0 \\ 0 & 0 & 0 & 0 & f & -(\mu + \delta_2) \end{bmatrix}$$

where,

$$A_1 = -(\beta S + \mu)$$

$$A_2 = -(\mu + \epsilon + \rho)$$

$$A_3 = -(\mu + \tau_1 + \tau_2)$$

$$A_4 = -(\nu + (\mu + \delta_1))$$

$$A_5 = -(f + (\mu + \delta_2))$$

$$A_6 = -(\mu + \delta_2)$$

$$J(DFE) = \begin{bmatrix} -A_1 - \lambda & 0 & \tau_1 & 0 & 0 & 0 \\ \beta S & -A_2 - \lambda & 0 & 0 & 0 & 0 \\ 0 & \rho & -A_3 - \lambda & 0 & 0 & 0 \\ 0 & \epsilon & 0 & -A_4 - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_5 - \lambda & 0 \\ 0 & 0 & 0 & 0 & f & -A_6 - \lambda \end{bmatrix} = 0 \quad (15)$$

where

$$\lambda_1 = -A_1$$

$$\lambda_2 = -A_2$$

$$\lambda_3 = -A_3$$

$$\lambda_4 = -A_4$$

$$\lambda_5 = -A_5$$

$$\lambda_6 = -A_6$$

The characteristic equation is therefore given by

$(-A_1 - \lambda)(-A_2 - \lambda)(-A_3 - \lambda)(-A_4 - \lambda)(-A_5 - \lambda)(-A_6 - \lambda) = 0$, where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ corresponding to $\lambda_j, j = 1, 2, \dots, 6$ are the eigenvalues. From [15], since all the roots are negative sign that is $\lambda_j < 0$. We conclude by Routh Hurwitz stability criterion that states that any system can be stable if and only if all the roots of the first column have the same sign. Hence, the model (1) is asymptotically stable.

3.8 Global stability analysis of the disease-free equilibrium (DFE)

By global stability, we mean stability relative to initial states in which the number of each species is positive. It follows that if a perturbation shifts the state of the system from a feasible equilibrium to a feasible state and the system is thereafter left alone, the natural dynamics of the system will drive the state of the system into a neighborhood of the equilibrium. It also means that the system will come to the equilibrium point from any possible starting point (i.e., there is no nearby condition).

Theorem 3.8

The disease free equilibrium is globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof :

Other methods like Lyapunov function can be used to proof global stability, which many authors have used in their various works. We adopt the Comparison theorem to prove the global stability of the disease free equilibrium (GAS), we have to make use of F and M matrices used in calculating the reproduction number (R_0) of our work. The rate of change of the variables of infectious classes of our system (14) (E, E_q, I, I_T, R) can be written as:

$$\frac{\partial x}{\partial t} = (F - V)X - JX, X = (E, E_q, I, I_T, R) \quad \frac{\partial x}{\partial t} = (F - V)X - JX, X = (E, E_q, I, I_T, R)$$

At the disease free equilibrium (DFE), $(S, E, E_q, I, I_T, R) = (\frac{\pi}{\mu}, 0, 0, 0, 0, 0)$,

consequently, equation (14) becomes;

$$(E, E_q, I, I_T, R) = \begin{bmatrix} E \\ E_q \\ I \\ I_T \\ R \end{bmatrix} \leq (F - V) \begin{bmatrix} E \\ E_q \\ I \\ I_T \\ R \end{bmatrix} \quad (16)$$

From [15], all eigenvalues of the matrix $F - V$ have negative real parts. i.e. $|(F - V) - \lambda I| = 0$

$$((F - V) - \lambda I) = \begin{bmatrix} -A_1 - \lambda & 0 & \tau_1 & 0 & 0 & 0 \\ \beta S & -A_2 - \lambda & 0 & 0 & 0 & 0 \\ 0 & \rho & -A_3 - \lambda & 0 & 0 & 0 \\ 0 & \epsilon & 0 & -A_4 - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_5 - \lambda & 0 \\ 0 & 0 & 0 & 0 & f & -A_6 - \lambda \end{bmatrix}$$

The characteristics polynomial of the above matrix was found and can be written as;

$$g(x) = \lambda^6 + F_1\lambda^5 + F_2\lambda^4 + F_3\lambda^3 + F_4\lambda^2 + F_5\lambda + F_6 = 0$$

Since, $F_1 > 0, F_2 > 0, F_3 > 0, F_4 > 0, F_5 > 0, F_6 > 0$, from our local stability result, where $\rho(FV^{-1}) < 1$ if $R_0 < 1$, which is equivalent to $(F - V)$ having eigenvalues with negative real parts when $R_0 < 1$, it follows that the linearized differential inequality system (16) is stable when $R_0 < 1$.

Consequently, $(E, E_q, I, I_T, R) \rightarrow (0, 0, 0, 0, 0)$ as $t \rightarrow \infty$. Thus, by the comparison theorem, $(E, E_q, I, I_T, R) \rightarrow (0, 0, 0, 0, 0)$ as $t \rightarrow \infty$. Substituting $E = E_q = I = I_T = R = 0$ in (2) $S(t) \rightarrow \infty$ as $t \rightarrow \infty$. Thus, $(S(t), E(t), E_q(t), I(t), I_T, R(t)) \rightarrow (S^0, 0, 0, 0, 0, 0)$ as $t \rightarrow \infty$ for $R_0 < 1$, thus, our Lassa fever disease free equilibrium is globally asymptotically stable.

4 Numerical simulations of Lassa fever model

In this paper, we give illustration of the analytical results of the work by carrying out numerical simulation of the model using epidemiological parameters given in Table 3. Parameters were obtained from different literature and those unavailable were assumed based on the available information. Some numerical solutions of the model for different initial population sizes is presented using the various values of the parameter and to validate that these solutions are in agreement with qualitative behaviors of the model population: $N = S_0(t) = 123600, E_0(t) = 263840, E_{q0}(t) = 185000, I_0(t) = 182700, I_{T0}(t) = 215000, R_0(t) = 290574$, the units of values are per day.

Parameters	Parameter description	Approx. values/days	Source
π	Recruitment rate	7342	Assumed
λ	Rate at which susceptible move to exposed class	4.377×10^{-10}	Assumed
ϵ	Rate of maturity from exposed to infected class	10^6	James T. et al, 2015
θ	Recovery rate to susceptible	0.01	James T. et al, 2015
τ_1	Rate of exposed-quarantine to susceptible class	0.42	James T. et al, 2015
τ_2	Rate of exposed-quarantine to infected class	0.05	James T. et al, 2015
ν	Rate of infected class to infectious treated class	0.006	James T. et al, 2015
ρ	Rate of exposed class to exposed-quarantine class	1/24	James T. et al, 2015
f	Rate of infectious-treated to recover class	0.05	James T. et al, 2015
μ	Natural death rate	0.0182	James T. et al, 2015
β	Contact rate between susceptible and infected	0.5	Assumed

δ_1	Lassa fever infected death rate	0.197	James T. et al, 2015
δ_2	Those who died by LFV during treatment	1/30	Assumed
ϕ	Level of re-infection	0.08	Assumed

Table 3: Parameters values of the model

The graph of the basic and effective reproduction numbers are plotted and interpreted as follows:

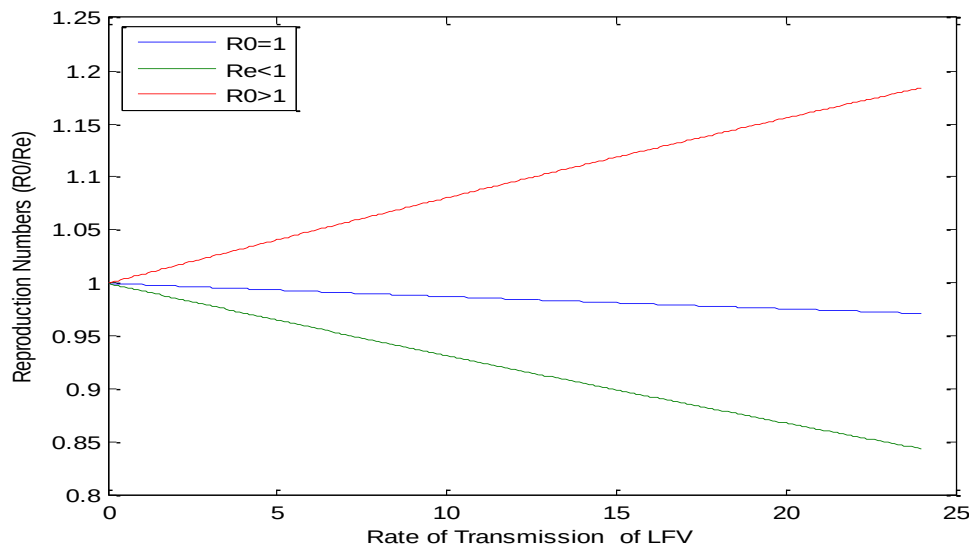


Figure 2: Effect of the rate of contact (β) on the Reproduction Numbers

Figure 2 shows the effect of rate of contact (β) on the basic and effective Reproduction Numbers. Figure 2 gives the following relationship; $R_0 = 1$, means if care is not taken the disease may invade the susceptible population in case of a backward bifurcation. $R_0 > 1$, means even if the infected individuals are treated, effect of the treatment might be very small. Hence, gives little or no recovery to the infected individuals. $R_e < 1$, means quarantine and health education campaign measures reduces the rate of spread of virus and increases the effectiveness of treatment given to infected individuals which also increases the rate of recovery. From the above scenario, it would be observed that when the susceptible are affected with Lassa fever virus, then the basic reproduction number (R_0) increases from $R_0 = 1.00$ to $R_0 = 1.20$ and when they are considered for quarantine and health education campaigns, then the effective reproduction number (R_e) decreases from $R_e = 1.00$ to $R_e = 0.85$. This implies that, when many susceptible individuals are getting infected due to the rate at which the virus is spread, it increases the basic reproduction number (R_0). Also, when few susceptible individuals are getting infected because of the introduction of quarantine and health education campaigns, this decreases the effective reproduction number (R_e).

Simulation of different Epidemiological classes with different parameter values of Lassa fever model

In this section, graphs of different epidemiological classes are plotted to show the transmission dynamics of Lassa fever Virus (LFV) disease.

From figure 3, the diagram shows the change in the dynamics of population with respect to time by varying the rate of spread of LFV model of susceptible population.

Figure 3a, it would be observed that the susceptible population at time $(t) = 0$ day is $p = 20,000$ individuals, the population then increases from $p = 20,000$ individuals to $p = 290,000$ individuals

at time $(t) \geq 60$ days, this is caused by the influx from recruitment rate (π) , recovery rate (θ) ,

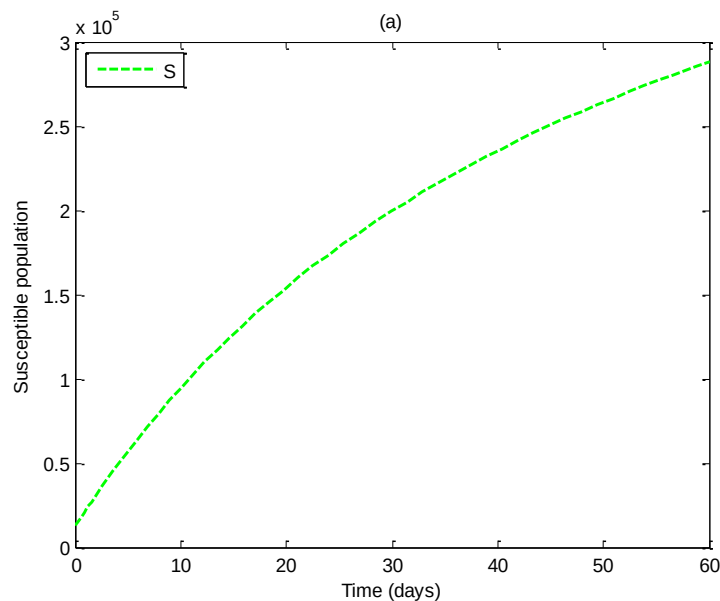


Figure 3a

non-manifested symptom, individual's rate (τ_1) , the rate (λ) of movement of those who are exposed and those who died natural death (μ) . This is because most individuals are now aware of Lassa fever virus (LFV), its mode of transmission and ways of quarantine and treatment for infected individuals. It is very important to control the recruitment rate into the susceptible population as well as encouraging sufficient behavioral change among them so as to effectively control and eradicate the LFV epidemic from the population/community. Although most of the infected persons under-went treatment and recovered, thereby reducing the spread of the virus to possibly $R_e < 1$.

Finally, it will be observed that the lower the rate of spread of Lassa fever virus among the susceptible population, the slower the rate of progression to exposed population and the higher the rate of spread of LFV, the higher the rate of progression of the disease to exposed population, because time keeps reducing as the rate of spread of the Virus increases. Hence, the transmission rate of LFV infection reduced and thereby reducing the susceptible population.

From the figure above, the diagram shows the change in the dynamics of population with respect

to time by varying the rate of spread of LFV model of exposed population.

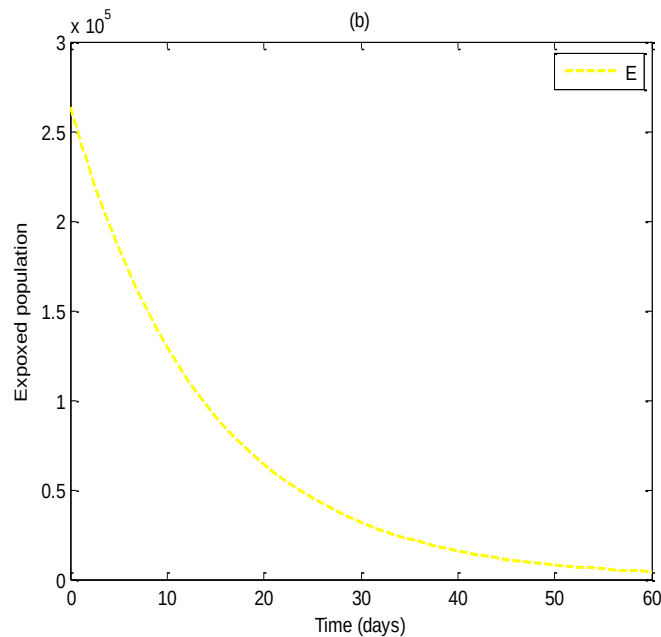


Figure 3b

Figure 3b, it would be also observed that the exposed population at time $(t) = 0$ day is $p = 260,000$ individuals, the population then decreases from $p = 260,000$ individuals to $p = 10,000$ individuals at time $(t) \leq 60$ days, this is caused by the influx of exposed rate (λ) from the susceptible population to exposed population and the going out of infected rate (ϵ) from exposed population to infected population, quarantine rate (ρ) and those who died natural death (μ). This implies that the exposed population decreases as the day increases with the help of quarantine and health education campaigns given to individuals. This is because individual awareness about virus and its methods of prevention has gone a long way to reduce the spread. This is in line with Okuonghae and Okuonghae (2006) who opined that the most effective way to reduce the virus epidemic infection is to carry out quarantine exercise and to educate the people who will go for checkup in order to become aware of their status. We also observed from the graph that if the days are increased further than 60 days, we would be discovered that at time $(t) = 70$ days to $t = 100$ day the population of exposed individuals would be asymptotically tending to zero (0) tolerant of the virus.

From the figure above, the diagram shows the change in the dynamics of population with respect to time by varying the rate of spread of LFV model of exposed-quarantine population. Figure 3c, depicts the exposed-quarantine population at time $(t) = 0$ day and $p = 180,000$ individuals, the population then continuously decreases from $p = 180,000$ to $p = 10,000$ individuals at time $(t) \geq 60$ days, this is caused by the influx of quarantine rate (ρ) and going out of non-manifested symptoms individuals rate (τ_1) , manifested symptoms individuals rate (τ_2) and those who died natural death (μ) . This implies that quarantine, public health education campaigns and treatment as control measures are sufficient for the timely control and eradication of the disease. Hence, these control strategies have significantly reduced the contraction and spread of the virus in the population/community. It can also be observed that if the days are increased further than 60 days, it would be discovered that at time $(t) = 70$ to $t = 100$ days the population of exposed-quarantined individuals would be asymptotically tending to zero (0) tolerant of the virus.

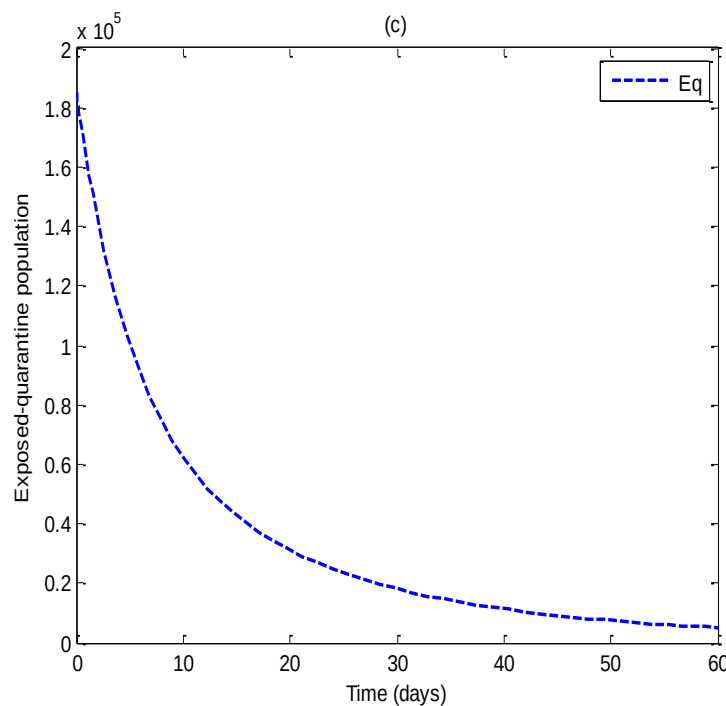


Figure 3c

Figure 3d depicts the population of infected individuals over time, it would be observed that the population of infected is $(p) = 180,000$ individuals at time $(t) = 0$ day, the population then decreases constantly from $p = 180,000$ individuals to $p = 10,000$ individuals at time $(t) \geq 60$ days due to the influx of virus exposed individuals rate (ϵ) , manifested symptoms individuals rate (τ_2) after 21 days of quarantined and going out of infectious rate (ν) individuals to undergo treatment, those who died by the virus (δ_1) and those who died natural death (μ) . This implies that health education campaigns facilitate good movement of infectious individuals to undergo treatment. This is in line with World Health Organization, (2017) who opined that prompt isolation of

affected patients, good infection prevention, control practices and rigorous contact tracing can stop the virus outbreaks. It is seen that figure 3d is similar to figure 3c respectively.

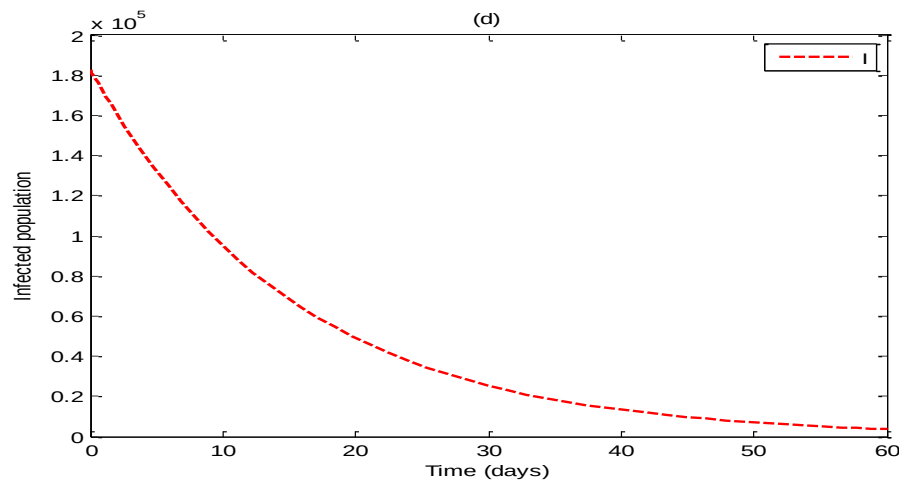


Figure 3d

From the figure above, the diagram shows the change in the dynamics of population with respect to time by varying the rate of spread of LFV model of infected-treated population. Figure 3e, depict the population of infected-treated individuals over time, we observed that the population of infected is $(p) = 220,000$ individuals at time $(t) = 0$ day, it would be also observed that the

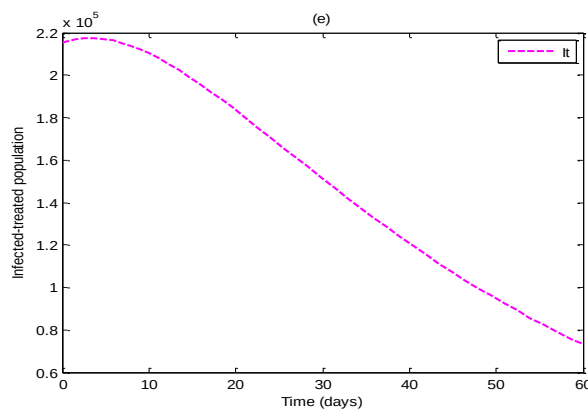


Figure 3e

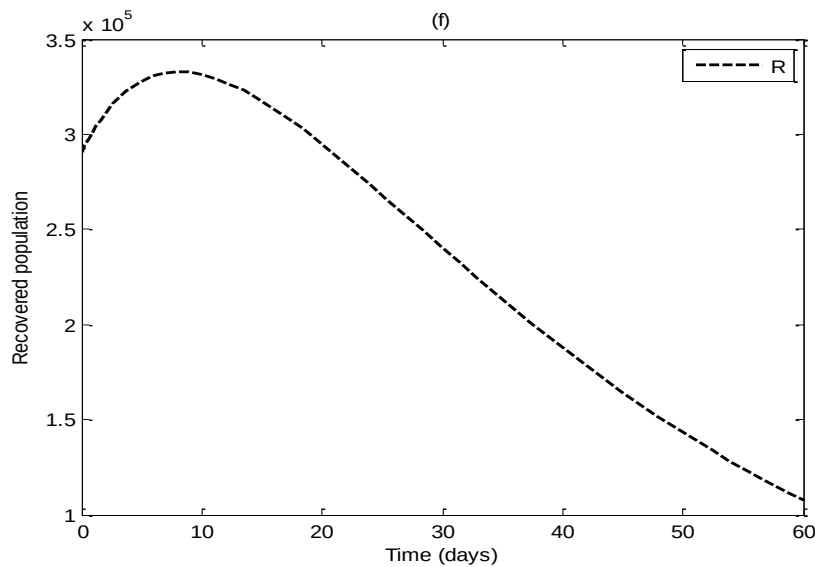


Figure 3f

population then decreases constantly from $p = 220,000$ individuals to $p = 70,000$ individuals at time $(t) \geq 60$ days due to the influx of infected individuals rate (ν) undergoing treatment, recovery rate (f) , the rate (μ) of those who died natural death and the rate (δ_2) of those who died by Lassa fever virus. This implies that quarantine and health education campaigns yielded an effective treatment on infected-treated humans within the possible shorted time of the day. This is in line with World health organization (WHO, 2018) who reported that timely quarantine of exposed and infected individuals should be observed and also a test to know their virus status be carried out. From the figure above, the diagrams show the change in the dynamics of population with respect to time by varying the rate of spread of LFV model of recovered population.

Figure 3f, depicts the population of recovered individuals over time, it will be observed that the population of recovered is $(p) = 300,000$ individuals at time $(t) = 0$ day, it then increased gradually to its peak as $(p) = 340,000$ individuals at time $(t) = 10$ days and subsequently decreases from $p = 340,000$ individuals to $p = 11,000$ individuals at time $(t) \geq 60$ days. This is because of the availability of all the control strategies employed to eradicate the virus. Here, many infected persons were given proper awareness about the virus, some were quarantined, treated and recovered to susceptible population while some infected persons died by the virus. Hence, the three control strategies should always be implemented to reduce the spread of the virus.

From the figure above, the diagram shows the change in the dynamics of population with respect to time by varying the rate of spread of LFV model of the total population. Figure 3g, is a summary of all the population of figures 3a to 3f. It will be observed here that the graphs intercept with each other at some points, this really showed the transmission dynamics of Lassa fever virus (LFV). The graph here still contains the same information of figure 3a to 3f. The points of interception of the graph are labelled A, B, C, D, E, F, G, H and interpreted as follows. The point of intercept A: This shows the interception between Susceptible population (S) and Exposed population (E). It is observed here that the number of exposed individuals and susceptible individuals are $(p) =$

240,000 individuals at time $(t) = 32$ days. This implies that the exposed individuals are to be quarantined beyond 32 days to avoid more spread of the virus.

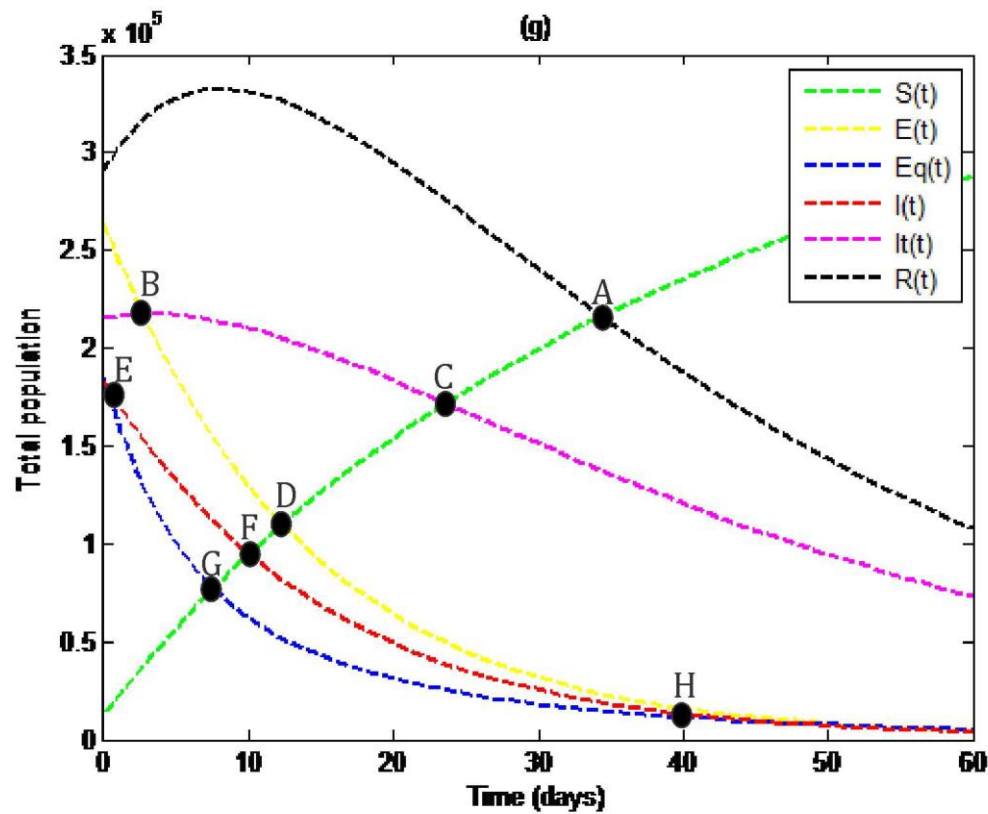


Figure 3g

The point of intercept B: This depicts the interception between exposed population (E) and infected-treated population (I_T). It is observed that the number of exposed individuals who were quarantined before treatment are $(p) = 220,000$ individuals at time $(t) = 3$ days. This implies that the combination of quarantine and health education campaigns enhance positive and sufficient behavioral changed on individuals who under-went treatment. This is because there is a good turn-up of individuals who responded for treatment at time $(t) = 3$ days. The point of intercept C: This shows the interception between susceptible population (S) and infected-treated population (I_T). This depicts that the number of susceptible individuals who contacted the virus and under-went treatment are $(P) = 180,000$ individuals at time $(t) = 24$ days. This is because the level of individual awareness of the virus is very high. The point of intercept D: This shows the interception of exposed population (E) and susceptible population (S). This depicts that the number of exposed individuals with the virus from the susceptible population are $(P) = 120,000$ individuals at time $(t) = 12$ days. It's implied that this number of exposed individuals were injected with Ribavirin vaccine (antibody) within 12 days of exposure to the virus and the vaccine is only for a short-term protection.

The point of intercept E: This shows the interception between infected population (I) and exposed-quarantine population (E_q). It is observed from the graph that the number of infected individuals who were quarantine due to the virus are $(p) = 170,000$ individuals at time $(t) = 1$ day. This is because the infected individuals were controlled at early stage (window period) of the virus

through quarantined. The point of intercept F: This depicts the interception between the susceptible population (S) and infected population (I). It is observed here that the number of susceptible individuals who were infected with the virus are $(p) = 100,000$ individuals at time $(t) = 10$ days. This is because some of these susceptible individuals do not practice safety and healthy life style like maintaining a standard community hygiene to control the rat population by regular hand washing, storing foods in rodent proof containers, keeping pet cat at home, avoiding blood and other body fluids when caring for sick people. The point of intercept G: This shows the interception between susceptible population (S) and exposed-quarantined population (E_q). It is observed that some of exposed-quarantined individuals who did not manifest symptoms of the virus during quarantined period, later returned to susceptible population (P) = 90,000 individuals at time $(t) = 8$ days. This means that the susceptible individuals were not infectious, since they did not manifest symptoms of the virus at time $(t) = 8$ days.

The point of intercept H: This depict the interception between exposed population (E), infected population (I) and exposed-quarantined population (E_q). It is observed here that the number of the sub-populations are asymptotically tending to stability with $(p) = 10,000$ individuals at time $(t) = 40$ days to time $(t) \geq 60$ days. This implies that exposed and infected individuals were quarantined, treated and healed, thereby reducing the spread of the virus because health education campaign was given to them.

5 Conclusion

In this work, the transmission dynamics of Lassa fever virus is formulated and rigorously analyzed. The epidemiological and mathematical findings of this work are summarized and concluded below: Based on the results of this work, we conclude that Lassa fever virus (LFV) is a dangerous virus that does not respect individual or a community if not dealt with. Not keeping to Lassa fever virus (LFV) protocol reduces the rate of recovery in infected individuals under treatment as seen in figure 7.2e. Quarantine and health education campaigns reduced the rate of spread of the disease as described in figure 7.2f, therefore infected individuals must be encouraged to take health education to understand very well why they must be quarantined and take their treatment as advised, because these increased their rate of recovery as described in figure 7.2f.

It may be hypothesized that the most effective way to reduce the rate of spread of Lassa fever virus and the prevalent rate is to give people health education in all classes as described in figure 1. Therefore, education programs must reach all communities at all social levels, to increase the awareness about Lassa fever virus (LFV).

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A Bayesian analysis and estimation of random censored data: application to pharmacovigilance data on Malaria drugs

Y.S. Jackson^{*†}, S.C. Nwaosu[‡], N.P Dibal^{*}, and Z. Wudiri[§]

Abstract

In this study, we propose and derive a new Hierarchical Bayesian Weibull Regression model on random censored data using the Jeffrey's prior. We derive the posterior density, marginal and obtain their kernels. The model parameters were estimated using Integrated Nested Laplace Approximation (INLA) and assess the model performance using Kulback Leibler Divergence and Deviance Information Criterion. The data is obtained from a pharmacovigilance study conducted by NAFDAC in collaboration with World Health Organization in Federal republic of Nigeria across six geopolitical zones. The shape parameter θ , were selected in the closed interval between zero and one with their respective priors distributions $\psi^j(\theta)$. It is evident that choice of prior $\psi^j(\theta) = 0.9$ results in the posterior for θ to be 2.52 for model I. In model II, we use the prior $\psi^j(\theta) = 0.001$ and obtain the posterior estimate of 2.514. We obtain the parameter estimate for model III using classical Integrated Nested Laplace Approximation with shape parameter 2.673. For choice of prior $\psi^j(\theta) = 80$, the posterior for θ is 3.007 in model IV. When we tune the prior to higher density region the posterior distribution for θ increases. For model I, II and III, the density is Rayleigh, but model IV tends to Normal for $\theta = 3.007$. It implies that higher prior change the shape of the posterior distribution. The predictors on recovery are not sensitive to low priors. The DIC and KLD show that the posterior converges to the real density with KLD of zero for all the models but model IV has the minimum DIC, which will perform better.

Keywords: Jeffrey's prior; hierarchical Bayes model; integrated nested Laplace approximation; latent Gaussian Models; random censoring

^{*} Department of Mathematical Sciences, University of Maiduguri, Maiduguri, Nigeria

[†] Corresponding Author. E-mail: samailajyaga@unimaid.edu.ng

[‡] Department of Math/Statistics/Computer Science, University of Agriculture, Makurdi, Nigeria

[§] Department of Community Medicine, College of Medical Sciences, University of Maiduguri, Maiduguri, Nigeria

1 Introduction

Latent Gaussian models are class of hierarchical models with some good number of hyperparameters. The response variable in the model is assume to be non-Gaussian and the latent field have Gaussian density with non-Gaussian hyperparameters. There are quite good approximate methods for performing Bayesian analysis for hierarchical structured models such as the MCMC method, Lindley's approximation, Tierney-Kadane, Lindely-Smith, some can be approximated to Gaussian distribution using Laplace approximation method. In this paper, we proposed a new hierarchical Bayesian Weibull regression model with using Jeffreys prior on random censored data. In Bayesian, statistics there is more concern on the elicitation of prior distribution..

In this study, we use Jeffreys prior for the shape parameter of Weibull density and normal priors for regression parameters and frailty parameter was assign a gamma prior distribution. A Weibull regression model with accelerated failure time was discussed [1]. Accelerated failure time regression model is a linear function of covariates and extreme value distribution. In [2] Weibull regression model was used on time to first and second recurrence of kidney infection with covariates for survival function and inclusion of additive random effect using Bayesian method. They analyze time to first and second recurrence of infection for kidney patients on dialysis. Using the Cox model with multiplicative frailty parameter for each individual. The survival distribution used is a truncated Weibull due to the censored observations. They used the non-informative prior for the regression co-efficient and the precision of random effect.

Bayesian method was used to analyzed data on photo carcinogenicity in four groups of mice each containing 20 mice with [3], they recorded a survival time of death event and the survival distribution assumed a Weibull with failure time and covariate vector with base line hazard function due to censored observations and implemented a truncated Weibull regression model with lower bound corresponding to the censoring time, and regression co-efficient β , where assumed a priori distribution to follow independent normal distribution with zero mean and vague precision of 0.0001. The shape parameter is distributed as **Gamma(1, 0.0001)** which coincides with the work of [2]. Bayesian analysis in the presence of covariates for multivariate survival data was developed by [4] and used different frailties (latent variables) to capture correlations among the survival times for the same individual. They use the Weibull and generalized gamma distribution considering right censored lifetime data and the MCMC method for Bayesian analysis and use gamma prior for the scale parameter and gamma prior for the latent parameter resulting to a hierarchical Bayesian model with two levels due to the hyperparameters in the model.

In determining the best estimator of the two-parameter Weibull distribution, [5] adopted the frequentist maximum likelihood estimator, least squares regression estimator and the Bayesian estimator by using two loss functions, which are, the quadratic loss function, and the linear exponential loss function. The Bayes estimates were obtained using the Lindleys approximation, the comparison was made through simulation to determine the performance of these methods. From the result, they found the Bayesian estimator with linear exponential loss function (LINEX), is found to be the best compared to the conventional MLE and Least squares method. [5] used the similar approach on censored data using the Lindleys approximation for the Bayesian approach with a standard non-informative prior and proposed generalization of the non-informative priors. The Bayes estimator using LINEX loss function with the proposed prior gives better result. They study also coincides with the work of [6] but

the difference is that they use Jeffreys prior and the extension of Jeffreys prior for the Bayesian paradigm on censored data. The parameters, was estimated using the Lindleys approximation.

The Bayesian with extension of Jeffreys prior gives better result compared to the MLE. A two-parameter Weibull density considering the properties of Bayes estimator with different loss functions was estimated in [7], the simulation scheme was done using non-informative priors and informative prior such as gamma prior, exponential-gamma prior, gamma-gamma prior and finally determine the prior and posterior predictive distributions. The parameters were estimated using the quadratic loss, weighted loss, square error loss, and square logarithmic loss functions. Numerical integration was tool for parameter estimation. The posterior risk of joint exponential gamma prior along with quadratic loss are most appropriate for the shape parameter while the joint lognormal-gamma prior with quadratic loss function are suitable for the scale parameter [8] use similar method but used the modified Weibull density. [6] Compared the Bayesian method on interval-censored data for Weibull distribution. The study considered the MLE and Bayes estimates. The Bayesian estimation method cannot solve for the parameters analytically instead, the MCMC method. The full conditional distribution for the scale and shape parameters are obtain using Metropolis Hastings Algorithm (M-H Algorithm), and Lindley's approximation to determine the shape parameter. The result shows that the Bayesian using the M-H Algorithm for estimating the shape and scale parameter based on interval-censored data is the best.

The work in [9] Performed approximate Bayesian inference in a sub-class of structured additive regression called the latent Gaussian models. They use integrated nested Laplace approximation on structured model and assume the response variable belong to the exponential family. The mean is linked to a structured additive linear predictor through a given log-link function and account for effects of various covariates in an additive way. They also provide accurate and fast deterministic approximations to all posterior marginal for the latent field and posterior marginal for the hyper-parameters. Model comparison, was obtained using the marginal likelihood and Deviance Information Criterion (DIC) also [10] investigates the use of INLA to solve Bayesian inferential problem in Bayesian survival analysis, the study uses the exponential and Weibull distributed lifetimes with and without censoring and frailty. Cox model, was fit with piece wise linear baseline hazard and express the model as latent Gaussian model so that INLA proposed by [11] can be a good tool for computation of posterior marginal. The model was tested using time to infection of kidney dialysis patients.

The work in [12] developed a Bayesian Semi-parametric model for occurrence of Asthma attacks in respiratory trials; they decompose the intensity function into product of various terms, first is the baseline intensity and second is the covariate and lastly the frailty term to take care of heterogeneity among different individuals with ability to have quick response to the event. The computations, was done using the INLA and the result turns out that this method has the highest speed and accuracy compared to Monte Carlo based inference.

2 Methods: Bayes theorem for Parametric Inference

Consider a general problem in which we have data y and we require inference about a parameter θ . In Bayesian analysis, θ is unknown and viewed as a random quantity. Thus, it possesses a density function $\psi(\theta)$. Using Bayes theorem, the marginal distribution for θ given the data y is

$$\psi(\theta|y) = \frac{\psi(y|\theta)\psi(\theta)}{\psi(y)} \quad (1)$$

where $\psi(\theta|y)$ is the posterior density, $\psi(\theta)$ is the prior density, $\psi(y|\theta)$ is the likelihood, $\psi(y)$ is the marginal for the data (Congdon, 2003).

3 Non-Informative Prior Distributions

Jeffrey's prior is a non-informative prior that is invariant under reparameterization [16]. The invariance property makes the prior distribution to be more formal than the application of uniform prior distribution. There are so many formal rule for selecting prior but we restrict to the Jeffreys prior. [13], explicitly define Jeffrey's prior in terms of lebesgue measure on the parameter space. Consider a density function $\psi_1(t|\theta)$ with respect to σ -finite dominating measure $\mu(dt)$ on a sample space $(\mathcal{X}, \mathcal{B})$. Θ is a non-empty open subset of k -dimensional parameter space $\mathbb{R}^k$. The function $\ln L(\psi_1(t|\theta))$ is a well-defined differentiable function of θ . We construct a well-defined score function $S_1(t|\theta)$ and its covariance exist. The score function, is given by the expression

$$S_1(\theta) = \left\{ \frac{\partial^2}{\partial \theta_i \partial \theta_j} \ln L(f(t|\theta)), i, j = 1, 2, \dots, n \right\} \quad (2)$$

The Fisher Information is derive from the covariance of the score function.

The Jeffreys prior with respect to the parameter θ is

$$\pi^J(\theta) \propto (\det(I_1(\theta)))^{1/2}.$$

4 Hierarchical Bayesian model

Bayesian statistics assumes that the parameters of a given distribution are random hereby having a probability density. Hierarchical Bayes model is a complex Bayesian model that consist of a sampling distribution (Data model) conditional on the parameter θ and the parameter has a distribution $\psi(\theta|\gamma)$ conditional on hyper-parameter γ . Finally if γ has a distribution making the model in different layers or levels. Such analogue in Bayesian modeling is termed as the hierarchical Bayes model. Example of a Hierarchical Bayesian model equation is

$$\psi(y, \theta, \gamma) = \psi(y|\theta)f(\theta|\gamma)\psi(\gamma). \quad (3)$$

It is full joint distribution of all quantities. The inference about θ and γ is simply obtained through their joint posterior distribution

$$\psi(\theta, \gamma | y) = \frac{\psi(\theta, \gamma, y)}{\psi(\gamma)} = \frac{\psi(y|\theta)\psi(\theta|\gamma)\psi(\gamma)}{\iint \psi(y|\theta)\psi(\theta|\gamma)\psi(\gamma)d\theta d\gamma}. \quad (4)$$

And inference about γ is given by its marginal posterior

$$\psi(\gamma|y) = \int \psi(\theta, \gamma|y)d\theta \propto \psi(\gamma) \int \psi(y|\theta)\psi(\theta|\gamma)d\theta. \quad (5)$$

We notice that γ is un-identifiable. The likelihood involves only θ , so prior belief about γ is influenced by the data y only through θ .

5 Model Formulation

Let T be a recovery time with density function ψ and non-recovery function S_r . Define a random censoring time C with distribution function G having probability density function g and non-recovery function S_g . Each unit has a recovery time T_i and a censoring time C_i . We define n independent and identically distributed random pair (Y_i, φ_i) , where

$$Y_i = \text{Min}(T_i, C_i) \quad (6)$$

$$\varphi = \begin{cases} 1, & \text{for } T_i \leq C_i \\ 0, & \text{for } C_i < T_i \end{cases} \quad (7)$$

The times T_i and C_i are assumed to be independent [14]. Let ϑ and S_v be distribution and non-recovery function. By independence assumption, it easily follows that the non-recovery function is

$$S_v(y) = [S_r(y)][S_g(y)]. \quad (8)$$

The distribution function of Y is

$$v(y) = 1 - S_r(y)S_g(y). \quad (9)$$

The likelihood function for the n independent and identically distributed random pairs (Y_i, φ_i) is

$$L = \left[\prod_{i=1}^n [S_g(y_i)]^{\varphi_i} [g(y_i)]^{1-\varphi_i} \right] \left[\prod_{i=1}^n [\psi(y_i)]^{\varphi_i} [S_r(y_i)]^{1-\varphi_i} \right] \quad (10)$$

If C , is not parameterized, then the first factor plays role in the maximization process, hence the likelihood function can be taken to be

$$L = \prod_{i=1}^n [(\psi(y_i))^{\varphi_i} [S_r(y_i)]^{1-\varphi_i}]. \quad (11)$$

It has the same form as the likelihood derived for right and left censoring [10]. The joint pdf for the pair, (Y, φ) is given by

$$P(Y, \varphi) = [(g(y))(S_r(y))]^{1-\varphi}[(\psi(y))(S_g(y))]^\varphi. \quad (12)$$

6 Weibull Regression Model

The time T_i for recovery, is distributed as Weibull with density function

$$\psi(t|\theta, \lambda) = \theta \lambda t^{\theta-1} \exp(-\lambda t^\theta), \quad (13)$$

where $t > 0$, the shape parameter $\theta > 0$, and $\lambda > 0$, is the scale parameter. For Weibull density, we have a vector of covariate that affect t . Let the covariate be Z , where Z' is a $(p \times 1)$ vector

$$\lambda_i = \exp(\beta_1 Z_1 + \beta_2 Z_2 + \dots + \beta_p Z_p) = \exp(Z_i^T \beta). \quad (14)$$

Density function for modeling event time is the product of the hazard function and the non-recovery function. The parameterization require modeling function for the mean of the distribution through λ . Covariate is link through λ and the parameter θ provides the shape of the distribution. Considering the change of parameter, we define the density for the recovery time with covariate Z as

$$\psi(t_i|\theta, z, \beta) = \theta \exp(Z_i^T \beta) t^{\theta-1} \exp(-\exp(Z_i^T \beta) t^\theta), \quad (15)$$

where the vector β is the co-efficient of regression parameters for covariates Z , t is the recovery time, and θ is the shape parameter. The hazard function, is given by

$$h_r(t) = \frac{\theta \exp(Z_i^T \beta) t^{\theta-1} \exp(-\exp(Z_i^T \beta) t^\theta)}{\exp(\exp(Z_i^T \beta) t^\theta)}. \quad (16)$$

The non-recovery function for each person at time t is express as

$$S_r(t|z_i, \theta) = \exp(-\exp(z_i^T \beta) t^\theta), \quad (17)$$

after substitution, we obtain the non-recovery function as we link equation (15), (17) into equation (12) the joint distribution becomes

$$\psi(t|z, \beta, \theta) = \theta^\varphi \exp \varphi(Z_i^T \beta) t^{\varphi(\theta-1)} \exp(-\exp(Z_i^T \beta) t^\theta). \quad (18)$$

7 Constructing Jeffreys Prior $\pi^J(\theta)$ for Bayesian HW-R Model

We have two methods for constructing prior for the shape parameter of Bayesian HW-R model, in first method, we modified the work of [6]) and in the second method, we establish another method for constructing Jeffrey's prior for the shape parameter of a Weibull distribution.

Method I

Jeffrey's prior as derived in [6] shows the joint distribution of both the scale and shape parameter. We will modify the prior, adopting some assumptions. Jeffreys prior for the shape parameter θ is obtained by determining the Marginal density from the joint Jeffreys prior distribution for scale and shape parameter (θ, λ) , secondly we assume Independence between the shape and scale parameter (θ, λ) . The joint prior for two-parameter Weibull distribution is

$$\psi^j(\theta, \lambda) \propto \sqrt{\det I(\theta, \lambda)} \quad (19)$$

$$I(\theta, \lambda) = -E_{(\theta, \lambda)} \begin{bmatrix} \frac{\partial^2 \ln L(\psi(t|\theta, \lambda))}{\partial \theta^2} & \frac{\partial^2 \ln L(\psi(t|\theta, \lambda))}{\partial \theta \partial \lambda} \\ \frac{\partial^2 \ln L(\psi(t|\theta, \lambda))}{\partial \theta \partial \lambda} & \frac{\partial^2 \ln L(\psi(t|\theta, \lambda))}{\partial \lambda^2} \end{bmatrix}, \quad (20)$$

where $I(\theta, \lambda)$ is the Fisher Information matrix (covariance of the score function), we derive Jeffreys prior as follows. Since we are considering the model with correction for censoring, we will determine the Jeffreys prior from the general representation with likelihood function

$$L(\psi(t|\lambda, \theta)) = \prod_{i=1}^n (\theta \lambda t^{\theta-1} \exp(-\lambda t^\theta))^{\varphi_i} (\exp(-\lambda t^\theta))^{1-\varphi_i}. \quad (21)$$

Before we determine the prior, we need to form Fisher's Information matrix. We derive Fisher's Information matrix from equation (21) by taking the second order partial derivative of log-likelihood function for each parameter forming the matrix

$$\begin{aligned} I(\theta, \lambda) &= \begin{bmatrix} \frac{1}{\theta^2} & \frac{\ln \theta + \Gamma'(2)}{\theta \lambda} \\ \frac{\ln \theta + \Gamma'(2)}{\theta \lambda} & \frac{\sum \varphi_i + (\ln \theta)^2 + \Gamma''(2) + 2 \ln \theta \Gamma'(2)}{\lambda^2} \end{bmatrix} \\ &= \frac{\sum \varphi_i + (\ln \theta)^2 + \Gamma''(2) + 2 \ln \theta \Gamma'(2)}{(\theta \lambda)^2} - \left[\frac{\ln \theta + \Gamma'(2)}{\theta \lambda} \right]^2 \\ &= \frac{\sum \varphi_i + \Gamma''(2) - (\Gamma'(2))^2}{(\theta \lambda)^2} = \frac{\sum \varphi_i + r}{(\theta \lambda)^2}, \end{aligned} \quad (22)$$

where

$$\Gamma'(2) = \int_0^\infty y \ln(y) \exp(-y) dy, \quad \Gamma''(2) = \int_0^\infty y \ln^2(y) \exp(-y) dy$$

$$r = \Gamma''(2) - (\Gamma'(2))^2.$$

Since we are only interested in the value of the prior for θ we ignore the constant term and use the kernel function for the prior distribution

$$\psi^j(\theta, \lambda) = \sqrt{\frac{\sum \varphi_i + r}{(\theta \lambda)^2}} \propto \frac{1}{\theta \lambda}. \quad (23)$$

We will derive the density for the shape parameter θ by looking for its marginal distribution. Using Bayes theorem for parametric inference

$$\psi^j(\theta|\lambda) = \frac{\psi^j(\theta, \lambda)}{\psi(\lambda)}, \quad (24)$$

where $\psi^j(\theta, \lambda)$ is the joint prior distribution in equation (23), and $\psi(\lambda)$ is the marginal distribution for scale parameter, $1/\lambda$, since the distribution of the parameters are conditionally independent. The distribution of $\psi^j(\theta)$ given by

$$\psi^j(\theta) \propto \frac{1}{\theta} \quad (25)$$

$$\psi^j(\theta) = \frac{k}{\theta}. \quad (26)$$

Finally, Jeffreys prior for shape parameter of a Weibull density is Bernoulli distribution with parameter zero and one 'B (0,1)'.

Method II

On expanding equation (42), the log-likelihood function, is given by

$$\begin{aligned} \ln L(\psi(t|\theta, \lambda, \varphi)) = & \left(\sum_{i=1}^m \varphi_i \right) \log \theta + \left(\sum_{i=1}^m \varphi_i \right) \log \lambda - \lambda \sum_{i=1}^m t_i \varphi_i \\ & - \lambda \sum_{i=1}^m t_i (1 - \varphi_i) + (\theta - 1) \varphi_i \sum_{i=1}^m \log t_i. \end{aligned} \quad (27)$$

The Fisher Information for this likelihood, is obtained by taking the expectation of the second order partial derivative of the log-likelihood function with respect to each parameter, which is shown as

$$\begin{aligned} I(\theta) = & -E \left[\frac{\partial^2 \ln L(f(t|\theta, \lambda))}{\partial \theta^2} \right] = -E \left[-\frac{\sum_{i=1}^m \varphi_i}{\theta^2} \right] \\ = & \left(\frac{\sum_{i=1}^m E(\varphi_i)}{\theta^2} \right) \propto \frac{1}{\theta^2}. \end{aligned} \quad (28)$$

Before we continue with the derivation, we need to establish a theorem that will enable us to get the expected value of the indicator variable φ_i

Theorem 1

Let $\{y_r\}_{r=1}^m$ be a finite sequence of sampled patients having attribute d with survival times $\{t_r\}_{r=1}^m$, and random censoring times $\{c_r\}_{r=1}^n$ with censoring indicator $\{\varphi_r\}_{r=1}^m$. We define a probability space $D = \{\Omega, \varphi, P(\varphi)\}$, with $P(\varphi)$, a probability measure satisfying $0 \leq P(\varphi) \leq 1$, and $\sum P(\varphi) = 1$. For m number of patients there will be n censored patients and $(m - n)$ uncensored patients with the following characterization.

$$\begin{aligned} E(\varphi) &= (m - n)P \\ \text{Var}(\varphi) &= (m - n)P(1 - P). \end{aligned}$$

Proof:

Let $P(\varphi_y)$ be the probability that y^{th} individual is censored and $(1 - P(\varphi_y))$ be the probability of being uncensored. φ_y , is distributed as Bernoulli with the parameter P with probability mass function

$$P(\varphi_y = r) = P^{\varphi_y} (1 - P)^{1 - \varphi_y} \text{ for } r, \varphi = 0, 1 \quad (29)$$

The first characterization is the mean which is derive as follows

$$E(\varphi_y) = \sum_{y=1}^m \varphi_y P(\varphi_y = r). \quad (30)$$

Since there are n number of censored observations and $(m - n)$ number of uncensored observation the indicator variable assumes values

$$\varphi = \begin{cases} 1, & \text{if uncensored} \\ 0, & \text{if censored} \end{cases}$$

Equation (30) becomes

$$E(\varphi) = \sum_{y=1}^m \varphi_y P(\varphi_y = r) = (m - n)P, \quad (31)$$

where

$$y, x = 1, 2, \dots, m, \text{ for } P_y = P_x.$$

The second prove is obtain directly using the variance of a Bernoulli distribution

$$V(\varphi) = \sum_{i=1}^m \varphi_y^2 P(\varphi_y) - \left(\sum_{i=1}^m \varphi_y P(\varphi_y) \right)^2 = (m - n)P(1 - P). \quad (32)$$

Applying Theorem 1 to equation (28) we get

$$V(\varphi) = \frac{\sum_{i=1}^m E(\varphi_i)}{\theta^2} = \frac{\sum_{i=1}^m (m - n)P_y}{\theta^2} \propto \frac{k}{\theta^2}, 0 < k < h. \quad (33)$$

The Fisher information $I(\theta)$ is finally, $I(\theta) \propto k/\theta^2$ and Jeffreys prior becomes

$$\psi^J(\theta) \propto \left[\frac{k}{\theta^2} \right]^{1/2} = \frac{k^{1/2}}{\sqrt{\theta^2}} \propto \frac{1}{\theta} \quad (34)$$

This result is the same with the modified version of Alomari et al (2012)

8 Analysis of Bayesian HW-R Model using Jeffrey's prior $\pi^J(\theta)$

In Bayesian analysis, the basic concept is the determination of posterior density using the Bayes method of parametric inference. The posterior density for Bayesian HW-R model using Bayes theorem is given by

$$\psi(\theta, \beta, \phi|t) \propto L(\psi(t|\theta, \beta, \eta)) \psi(\beta) \psi^J(\theta) \psi(\phi) \quad (35)$$

$$= \frac{L(\psi(t|\theta, \beta, \eta)) \psi(\beta|\mu, \Phi) \psi^J(\theta) f(\phi)}{\iint L(\psi(t|\theta, \beta, \eta)) \psi(\beta|\mu, \Phi) \psi^J(\theta) \psi(\phi) d\theta d\beta}. \quad (36)$$

With prior specification for hyperparameters. The first one is the gamma prior for parameter ϕ with density function

$$\psi(\phi) = \frac{b^a}{\Gamma(a)} \exp(-b\phi). \quad (37)$$

Secondly is the normal or Gaussian prior distribution for the parameter of predictor variables β .

$$\psi(\beta) = \frac{\exp - \left(\frac{1}{2} (\beta - \hat{\beta})^T \Phi^{-1} (\beta - \hat{\beta}) \right)}{(\sqrt{2\pi}) |\Phi|^{-1/2}}. \quad (38)$$

Finally, the prior distribution for the shape parameter of Weibull distribution, which is derived using Jeffreys rule. It is given, as

$$\psi^J(\theta) = \frac{k^{1/2}}{\theta}, \quad 0 \leq k \leq h. \quad (39)$$

The posterior density and marginal in equation (35) after substitution and Simplification becomes

$$\begin{aligned} \psi(\theta, \beta, \phi|t) \propto \theta^{r-1} \exp \left(\sum_{i=1}^n \left(\varphi_i + z_i^T \beta + \varphi_i(\theta - 1) \ln(t_i) \right) - t_i^\theta \exp(z_i^T \beta) \right. \\ \left. - \frac{1}{2} (\beta - \hat{\beta})^T \Phi^{-1} (\beta - \hat{\beta}) - a\phi \right). \end{aligned} \quad (40)$$

8.1 Kernel for posterior and marginal densities

Kernel for Posterior density

$$\begin{aligned} \psi(\theta, \beta, \phi|t) \propto \theta^{r-1} \exp \left(\sum_{i=1}^n \left(z_i^T \beta + \varphi_i(\theta - 1) \ln(t_i) \right) - t_i^\theta \exp(z_i^T \beta) \right. \\ \left. - \left(\frac{1}{2} (\beta - \hat{\beta})^T \Phi^{-1} (\beta - \hat{\beta}) \right) - a\phi \right). \end{aligned} \quad (41)$$

Posterior marginal for θ in Kernel form

$$\psi(\theta|\beta, \phi, t) \propto \theta^{r-1} \exp \left[(\theta - 1) \sum_{i=1}^m \varphi_i \ln t_i - t_i^\theta e^{(z_i^T \beta)} \right]. \quad (42)$$

Posterior Marginal for ϕ in Kernel form

$$\psi(\phi|\beta, \theta, t) \propto \exp(-a\phi) \quad (43)$$

Posterior Marginal for β in Kernel form

$$\psi(\beta|\theta, \phi, t, z) \propto \exp \left\{ -\frac{1}{2} (\beta - \hat{\beta})^T \Phi^{-1} (\beta - \hat{\beta}) + \sum z_i^T \beta \right\}. \quad (44)$$

9 Parameter Estimation

Parameter estimation is one of the most difficult aspect Bayesian analyses more especially complex models that the prior distribution is not conjugate and class of hierarchical models which sometimes MCMC method and Numerical Integration cannot give accurate estimates due to the hierarchical structure. Lindley (1980) developed a method for approximating posterior distribution for integral of the form

$$I = \frac{\int U(\lambda, \theta) \exp[\ln L(\psi(\lambda, \theta)) + \rho(\lambda, \theta)] d\theta}{\int \exp[\ln L(\lambda, \theta) + \rho(\lambda, \theta)] d\theta}, \quad (45)$$

where $U(\lambda, \theta)$, is the function of λ and θ only, $\ln L(\psi(\lambda, \theta))$ is the log likelihood and $\rho(\lambda, \theta) = \ln L[\psi(\lambda, \theta)]$. Lindleys method evaluates this form of integral.

9.1 Laplace Approximation

The Laplace method approximate the log-likelihood of the posterior density as a quadratic form with mode $(\hat{\theta})$. The method approximates equation (45) using the Taylor's series expansion on $\psi^*(\theta)$. The expansion yielded

$$\ln[L(\psi^*(\theta))] \approx b - \frac{1}{2}(\theta - \hat{\theta})^T H_1(\theta - \hat{\theta}) \quad (46)$$

$L(\psi^*(\theta))$, is the likelihood function for density $\psi^*(\theta)$ evaluated at θ , b is a constant and H_1 is defined as

$$H_1 = -\frac{\partial^2 \ln[L(\psi^*(\theta))]}{\partial \theta^2} = -\nabla^2 \ln[L(\psi^*(\theta))] \text{ at } \theta = \hat{\theta}.$$

The posterior density $f^*(\theta)$, is approximated by an un-normalized Gaussian density

$$\psi(\theta) \equiv \psi^*(\theta) \exp\left(-\frac{\nabla^2}{2} \ln[L(\psi^*(\theta))] (\theta - \hat{\theta})^T (\theta - \hat{\theta})\right). \quad (47)$$

The normalizing constant for Gaussian density is approximated using normalizing constant of this Gaussian:

$$C \equiv \psi^*(\theta) \sqrt{\frac{2\pi}{\nabla^2 \ln[L(f(\theta))]}}. \quad (48)$$

9.2 Quasi-Newton Optimization

Quasi-Newton method is one of the ideas in solving non-linear optimization problem. It is like the steepest descent that require only the gradient of the objective function that is good enough to produce super-linear convergence. It can solve unconstrained, constrained and large scale optimization problems. The Quasi-Newton method is performed using the following steps. Consider a non-linear objective function

$$\psi(\theta_\sigma, \omega) = a\theta_\sigma^r + b\theta_\sigma^{r-1} + \dots + q + u\omega^t + v\omega^{t-1} + \dots + z \quad (49)$$

where $a, b, \dots \in \mathbb{R}$, and θ_σ, ω are variables

Given a starting point $\theta_{(0)} \in \text{domain } \psi$ for $\theta = (\theta_\sigma, \omega)$

Compute the Quasi-Newton direction $\Delta\theta = -H_{k-1}^{-1}\psi'(\theta_{k-1})$

Determine the step size t_k

Compute $\theta_k = \theta_{k-1} + t_k\psi'(\theta_k)$

Compute H_k

Broyden, Fletcher, Goldfarb, and Shanno (BFGS) for updating Hessian (H_k)

BFGS is the popular Quasi-Newton method; it is use for updating the Hessian H_k . Following the model of the objective function at the current iterate θ_k , we use the finite difference approximation method

$$\psi(\theta_k + \Delta\theta) = \psi(\theta_k) + \Delta\theta_k \psi'(\theta_k) + \frac{\Delta\theta_k^T}{2!} H_k \Delta\theta_k + \dots \quad (50)$$

H_k , is a $n \times n$ symmetric positive definite matrix and is revised or updated at every iteration. The function value and gradient of this model at $\Delta\theta_k = 0$ match ψ_k and $\Delta\psi_k$, the minimizer $\Delta\theta_k$ of this convex quadratic model is written as $\Delta\theta = -H_{k-1}^{-1} \psi'(\theta_{k-1})$, will be use as search direction and new iterate is

$$\theta_{k+1} = \theta_k + t_k \psi'(\theta_k), \quad (51)$$

where t_k is the step length, satisfying the Wolfe condition.

The approximate Hessian H_k is used in place of the true Hessian and is updated using

$$H_k = H_{k-1} + \frac{vv^T}{v^T s} - \frac{H_{k-1} s s^T H_{k-1}}{s^T H_{k-1} s}, \quad (52)$$

where

$$s = \theta_k - \theta_{k-1}, \quad v = \Delta\psi(\theta_k) - \psi\Delta(\theta_{k-1})$$

The inverse update is obtain for $v^T s > 0$ and strictly convex function ψ is given by

$$H_k^{-1} = \left(I - \frac{s v^T}{v^T s} \right) H_{k-1}^{-1} \left(I - \frac{v^T s}{v^T s} \right) + \frac{s s^T}{v^T s}. \quad (53)$$

The BFGS update satisfies the secant condition as $H_k s > v$ i.e.

$H_k(\theta_k - \theta_{k-1}) = \Delta\psi(\theta_k) - \psi\Delta(\theta_{k-1})$, and θ_{k+1} is obtain as

$$\theta_{k+1} = \theta_k - \frac{\psi'(\theta_k)}{H_k} \quad (54)$$

$$H_k = \frac{\psi'(\theta_k) - \psi'(\theta_{k-1})}{\theta_k - \theta_{k-1}}. \quad (55)$$

10 Integrated Nested Laplace Approximation (INLA) Method

Numerical approach in Bayesian analysis for Latent Gaussian models is discussed in [11] and [15]. This approach makes the Laplace approximations very accurate when applied to Latent Gaussian Model (LGM).

Numerical computation of the Latent Gaussian Model

The posterior density $\psi(x, w|t)$ for Gaussian Latent Field x and hyperparameters w conditional on t is given by the expression

$$\psi(x, w|t) \propto \psi(w) \psi(x|w) \prod_{i=1}^n \psi(t_i|x_i), \quad (56)$$

$\psi(x_i|t)$: The posterior marginal for Latent Gaussian Field for all i

$\psi(w_i|t)$: Posterior for the marginal, for hyperparameters, which is not Gaussian.

Numerical computation of posterior marginal

The posterior marginal for the hyper-parameters, conditionally on time t is express as

$$\psi(w|t) \propto \frac{\psi(x, t, w)}{\psi(x|t, w)}. \quad (57)$$

We locate the mode in log scale and the posterior marginal will be interpolated, and we use the Gaussian Approximation to the conditional for the marginal for hyper- parameters

$$\psi(w|t) \propto \frac{\psi(x, w|t)}{\psi_G(x|w, t)} \quad \text{at } x = x_{\text{mode}}(w). \quad (58)$$

For the Latent field, the marginal for $x_i|t$ we do similarly

$$\psi(x_i|t) \approx N_c \frac{\psi(x, t|w)}{\psi_G(x_{-i}|x_i, t, w)} \quad \text{at } x_{-i} = x_{\text{mode}}(w, x_i). \quad (59)$$

Taking $\psi_G(x_{-i}|x_i, t, w)$ as the Gaussian Approximation which gives us Laplace Approximation

$$\begin{aligned} \psi(x|t, w) &\propto \exp\left(-\frac{1}{2}x^T \Phi x + \sum_{i=1}^n \ln L(\psi(t_i|x_i))\right) \\ \psi_G(x|w, t) &\approx \exp\left(-\frac{1}{2}(x - \mu)^T (\Phi + \text{diag}(\Phi_{(\theta, \phi)})) (x - \mu)\right). \end{aligned} \quad (60)$$

Implementing INLA for Bayesian HW-R model

Consider the linear predictor of a structured additive model

$$\eta = \beta_0 + \beta_1 Z_1 + \dots \beta_n Z_n + \phi \sigma + \epsilon, \quad (61)$$

where Z 's are the covariates

β_0 = Intercept for the linear predictor model

β 's = linear effects of covariates Z (fixed)

σ = The variable accounting for frailty term in the linear predictor

ϕ = is the co-efficient for frailty term in the model.

ϵ = is an unstructured term accounting for unexplained variation in the model

Computing Posterior marginal for $\psi(\theta, \phi|\beta, t,)$ using Quasi-Newton method

In computing the posterior marginal for the kernel $\psi(\theta, \phi|\beta, t,)$, we first, locate the mode for $\ln \psi(\theta, \phi|\beta, t,)$, by optimizing with respect to θ using Quasi-Newton method which build up to an approximation to the second derivative of $\ln \psi(\theta, \phi|\beta, t,)$ using difference between successive gradient vectors and the gradient is obtain using finite difference.

The kernel is

$$\begin{aligned} \psi(\theta, \phi|\beta, t) &\propto \theta^{r-1} \exp\left\{(\theta - 1) \sum_{k=1}^m \varphi_k \ln t_k - t_k^\theta e^\eta - a\phi\right\} \\ \ln \psi(\theta, \phi|\beta, t,) &= (r - 1) \ln \theta + \sum_{k=1}^m \varphi_k (\theta - 1) \ln t_k - t_k^\theta e^\eta - a\phi. \end{aligned} \quad (62)$$

We locate the mode for θ_k and ϕ_k by using the Quasi-Newton method. The quadratic model for the objective function, is given by the second order approximation

$$\mathcal{M}_k(\Delta\hat{\theta}_k) = \psi(\hat{\theta}_k) + \Delta\hat{\theta}_k \psi'(\hat{\theta}_k) + \frac{\Delta\hat{\theta}_k^T}{2!} H_k \Delta\hat{\theta}_k + \dots \quad (63)$$

where $\hat{\theta} = (\hat{\theta}, \hat{\phi})$, $\Delta\hat{\theta} = (\hat{\theta} - \hat{\theta}_k)$ and $\mathcal{M}_k(\Delta\hat{\theta}_k)$ is the objective function.

Using finite difference, the Hessian is obtain using the formula

$$H_k(\theta) = \frac{\left(\frac{\partial \ln \psi(\hat{\theta}_k, \phi | \beta, t,)}{\partial \hat{\theta}_k} - \frac{\partial \ln \psi(\hat{\theta}_{k-1}, \phi | \beta, t,)}{\partial \hat{\theta}_{k-1}} \right)}{\theta_k - \theta_{k-1}}. \quad (64)$$

For $\hat{\theta}_k$

$$\frac{\partial \ln \psi(\hat{\theta}_k, \phi | \beta, t,)}{\partial \hat{\theta}_k} = \frac{r-1}{\hat{\theta}_k} + \sum_{k=1}^m \varphi_k \ln t_k - t_k^{\hat{\theta}_k} \ln t_k e^\eta. \quad (65)$$

For $\hat{\theta}_{k-1}$

$$\frac{\partial \ln \psi(\hat{\theta}_{k-1}, \phi | \beta, t,)}{\partial \hat{\theta}_{k-1}} = \frac{r-1}{\hat{\theta}_{k-1}} + \sum_{k=1}^m \varphi_k \ln t_k - t_k^{\hat{\theta}_{k-1}} \ln t_k e^\eta. \quad (66)$$

Using this we compute the Hessian for the objective function to be

$$H_k = \frac{\left\{ r-1 \left(\frac{1}{\hat{\theta}_k} - \frac{1}{\hat{\theta}_{k-1}} \right) - e^\eta \left(t_k^{\hat{\theta}_k} \ln t_k + t_k^{\hat{\theta}_{k-1}} \ln t_k \right) \right\}}{\hat{\theta}_k - \hat{\theta}_{k-1}}. \quad (67)$$

The value of $\hat{\theta}_k$ can be iterated to get new value for $\hat{\theta}_{k+1}$ using

$$\hat{\theta}_{k+1} = \hat{\theta}_k - \left\{ \left(\frac{\partial \ln \psi(\hat{\theta}_k, \phi | \beta, t,)}{\partial \hat{\theta}_k} \right) / H_k \right\}, \quad (68)$$

$$\hat{\theta}_{k+1} = \hat{\theta}_k - \frac{\frac{r-1}{\hat{\theta}_k} + \sum_{k=1}^m \varphi_k \ln t_k - t_k^{\hat{\theta}_k} \ln t_k e^\eta}{\left\{ \frac{r-1 \left(\frac{1}{\hat{\theta}_k} - \frac{1}{\hat{\theta}_{k-1}} \right) - e^\eta \left(t_k^{\hat{\theta}_k} \ln t_k + t_k^{\hat{\theta}_{k-1}} \ln t_k \right)}{\hat{\theta}_k - \hat{\theta}_{k-1}} \right\}}. \quad (69)$$

We routinely get new values of Hessian and compute new values for $\hat{\theta}$. Finally; we construct an interpolant and compute the marginal using numerical integration from this interpolant. We standardize values by converting $\hat{\theta}$ using

$$\hat{\theta}(z) = \hat{\theta} + A \lambda^{\frac{1}{2}} Z, \quad (70)$$

where $\lambda^{\frac{1}{2}}$, A are obtained from Eigen decomposition of inverse Hessian obtained from the finite difference approximation and $Z \sim N(\mathbf{0}, \mathbf{I})$ which corrects for scale and rotation.

Computing posterior marginal for latent variables $\Psi(x_i|t)$

We use numerical integration to obtain the posterior marginal for x_i

$$\psi(x_k|t) = \sum_{i \in t} \psi(x_k|\hat{\theta}_k, t) \psi(\hat{\theta}_k|t) \Delta_k, \quad (71)$$

which is the sum over the values of $\hat{\theta}_k$ with area weight Δ_k , we then use laplace approximation to provide the accurate approximation using the set of weighted points $\{\hat{\theta}_k\}$ to be use in the integration.

11 Assessing Model Performance

11.1 Deviance Information Criterion

The models, will be assessed using the deviance Information Criterion (DIC), the DIC is given by the expression

$$D(\theta) = -2\text{Ln}\mathcal{L}(\theta) + \mathcal{C}, \quad (72)$$

θ is the vector of unknown parameters of the model, $\mathcal{L}(\theta)$ is the likelihood function of the model and $\mathcal{C}$ is a constant that does not need to be known. The DIC define by [17] is well applied in the Bayesian model comparison, it has the form

$$\text{DIC} = \mathcal{D}(\hat{\theta}) + 2n_{\mathcal{D}}, \quad (73)$$

where $\mathcal{D}(\hat{\theta})$, is the deviance evaluated at the posterior mean and $n_{\mathcal{D}}$ is the effective number of parameter for the model.

11.2 Kulback Leibler Divergence (KLD)

Kullback and Leibler introduce another information measure aside the Bayesian information and Akaike Information criterion to assess the distance between the true and approximated Bayesian posterior distribution. Let $f(\theta|y)$ be the posterior density for the true model and $g(\theta|y)$ be the probability density for the approximated model on the measurable space. The Kullback Leibler Divergence, is defined by the relation

$$I(\psi, g) = E \left[\frac{\psi(\theta|y)}{g(\theta|y)} \right] = \int \log \left[\frac{\psi(\theta|y)}{g(\theta|y)} \right] \psi(\theta|y) dy, \quad (74)$$

$I(\psi, g)$ is non-negative and it reach 0 when $\psi(\theta|y)$ is the same as $g(\theta|y)$ almost surely, and is interpreted as information lost when $g(\theta|y)$ is used to approximate $\psi(\theta|y)$. The smaller values of $I(\psi, g)$ we consider the model g to be closer to the true distribution $\psi(\theta|y)$

12 Results

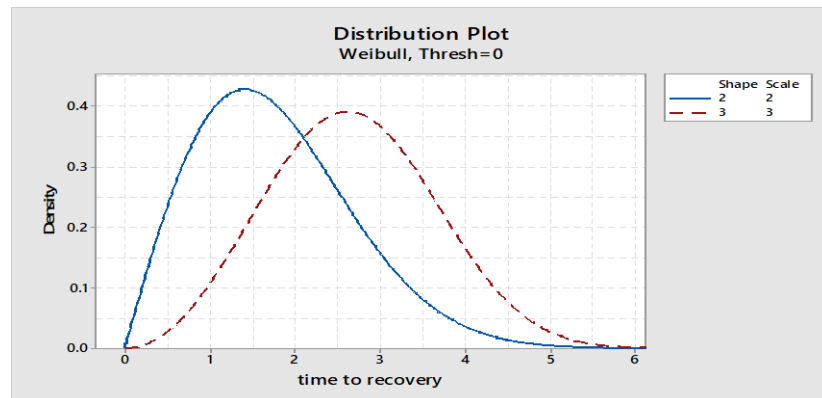


Figure 1a: Density Plot for Weibull distribution with Scale $\lambda = 2$ and Shape Parameter $\theta = 2$, Scale $\lambda = 3$ and shape $\theta = 3$

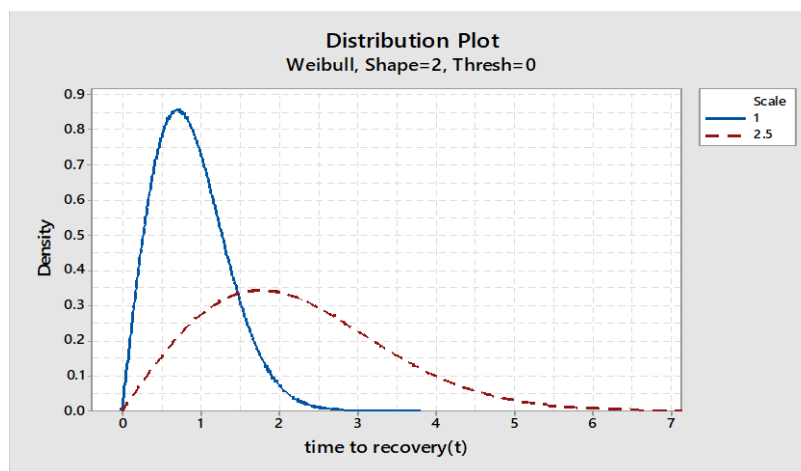


Figure 1b: Density Plot for Weibull distribution with Scale parameter $\lambda = 1$, $\lambda = 2.5$ with shape parameter $\theta = 2$

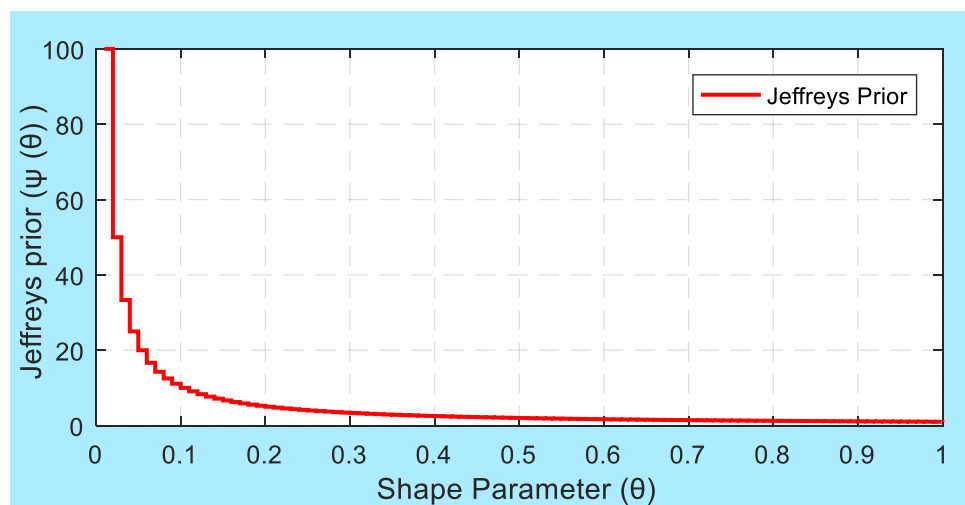


Figure 2: Density plot for Jeffrey's prior for shape parameter θ in the close interval $[0, 1]$

Specification for the drug precision ϕ						
Variables	Mean	S.E	0.025 Quant	0.5Quant	0.975 Quant	Mode
β_0	-4.6920	0.4306	-5.5479	-4.6881	-3.8584	-4.6754
$\beta_{(\text{Sex})}$	0.0093	0.1247	-0.2350	0.0091	0.2546	0.0087
$\beta_{(\text{Age})}$	-0.0039	0.0063	-0.0165	-0.0039	0.0083	-0.0037
$\beta_{(\text{Wght})}$	0.0081	0.0052	-0.0020	0.0081	0.0182	0.0081
$\theta_{(\text{Shape})}$	2.52	0.1272	2.277	2.518	2.777	2.515

Table 1: INLA estimate for Bayesian HW-R using Jeffreys prior, $\psi^J(\theta) = 0.9$ and no prior

Variables	Mean	S.E	0.025 Quant	0.5Quant	0.975 Quant	Mode
β_0	-4.6815	0.4304	-5.5372	-4.6776	-3.8482	-4.6696
$\beta_{(\text{Sex})}$	0.0095	0.1247	-0.2348	0.0092	0.2548	0.0088
$\beta_{(\text{Age})}$	-0.0039	0.0063	-0.0165	-0.0038	0.0084	-0.0037
$\beta_{(\text{Wght})}$	0.0081	0.0052	-0.0020	0.0081	0.0182	0.0081
$\theta_{(\text{Shape})}$	2.512	0.1272	2.271	2.512	2.771	2.509

Table 2: INLA estimate for Bayesian HW-R using Jeffreys prior, $\psi^J(\theta) = 0.001$ with Gamma (1, 0.001) prior specification for the drug precision ϕ

Variables	Mean	S.E	0.025 Quant	0.5Quant	0.975 Quant	Mode
β_0	-4.9686	0.4339	-5.8309	-4.9647	-4.1285	-4.9567
$\beta_{(\text{Sex})}$	0.0061	0.1247	-0.2382	0.0059	0.2514	0.0054
$\beta_{(\text{Age})}$	-0.0042	0.0063	-0.0168	-0.0042	0.0080	-0.0041
$\beta_{(\text{Wght})}$	0.0085	0.0052	-0.0017	0.0085	0.0186	0.0084
$\theta_{(\text{Shape})}$	2.673	0.1273	2.425	2.671	2.934	2.668

Table 3: INLA estimate for Bayesian HW-R with no prior specification for θ .

Variables	Mean	S.E	0.025 Quant	0.5Quant	0.975 Quant	Mode
β_0	-5.5796	0.4429	-6.4593	-5.5758	-4.7217	-5.5681
$\beta_{(\text{Sex})}$	-0.0029	0.1248	-0.2472	-0.0031	0.2426	-0.0035
$\beta_{(\text{Age})}$	-0.0049	0.0063	-0.0175	-0.0048	0.0074	-0.0047
$\beta_{(\text{Wght})}$	0.0092	0.0052	-0.0010	0.0092	0.0194	0.0092
$\theta_{(\text{Shape})}$	3.007	0.1334	2.752	3.005	3.276	3.001

Table 4: INLA estimate for Bayesian HW-R using Jeffrey's prior Jeffrey's prior $\psi^J(\theta) = 80$ with Gamma (1, 0.001) prior specification for the drug precision ϕ .

Variables	Mean	S.E	0.025 Quant	0.5Quant	0.975 Quant	Mode
I	18643.4	1.842E+04	1282.9	13233.4	67312.8	3490.4
II	976.60	938.94	75.43	705.00	3563.90	211.35
III	18691.46	1.846E+04	1286.54	13258.45	67506.76	3500.54
IV	992.14	957.67	77.717	714.82	3529.05	217.49

Table 5: Hyper-Parameter (Random effect component) for drug precision ϕ , using Gamma (1, 0.001) Prior for Model I, II, IV and No prior for Model III.

Model	Marginal Log-likelihood	DIC	Rank	KLD
I	-642.38	1122.23	3	0
II	-652.98	1122.72	4	0
III	-596.55	1116.17	2	0
IV	-576.73	1111.35	1	0

KLD: Kullback Leibler Divergence DIC: Deviance Information Criterion

Table 6: Assessing Model performance using the Deviance Information Criterion, Marginal Log-likelihood and Kulback Leibler Divergence

13 Summary and Conclusion

The study proposed a Bayesian HW-R model using Jeffreys prior on random censored data. The model was tested using data for recovery times of patients tested positive of malaria and where administered drugs. The study, also determine the posterior density for the proposed model and established a new method for obtaining Jeffreys prior for random censored data. Also, modify the work of Alomari [6] and obtain the Jeffreys prior for shape parameter since the scale parameter is an exponential function of the predictors, which was thus, incorporated forming a structured additive model for the linear predictor through log link function.

The predictor's influence the recovery time only through the predictor. The model used is termed the Latent Gaussian Model. Consider the result in Table1, $\psi^J(\theta) = 0.9$, the posterior mean with this prior yield an estimate of $\theta = 2.52$, also the result in table 2 for prior $\psi^J(\theta) = 0.001$, has posterior mean of $\theta = 2.514$. The prior in table 1 is higher than the prior in table 2 as such the posterior estimate for θ was influenced by the choice of θ in the θ -coordinate. When the prior is non-informative, the likelihood takes part in maximizing the posterior density. Table 3 has no prior specification for the shape parameter θ , the estimated value is $\theta = 2.673$. It is clear that the prior in table 2 decreases the posterior estimate from 2.673 to 2.52 for $\psi^J(\theta) = 0.9$ and 2.514 for $\psi^J(\theta) = 0.001$, but the distribution is Rayleigh for value of θ within the range of 2, the estimate without prior specification uses only the data at hand that's why the estimate is 2.673. Table 4 is exceptional because when we specified the prior to be high in θ -coordinate, $\psi^J(\theta) = 80$, the posterior for θ increases suddenly to the peak with posterior

estimate $\theta = 3.007$ which approximately changed the density to normal, while the other three follows the Rayleigh density as shown in fig1 and fig1.b.

When the value for $\psi^j(\theta)$ in θ -coordinate increases, the posterior estimate for θ increases and change the shape of the distribution. For prior density $\psi^j(\theta)$ with censoring, the posterior density attains normality with increase in prior for shape parameter θ in θ -Space. The assessment of covariate effect with normal prior shows small ignorable differences in the parameter for table 1 and 2. Table 4 shows a very large difference because of high prior updating. In addition, the random component of the predictor, η of the structured additive class of Latent Gaussian Model assumes gamma (1, 0.001) prior. The posterior for drug precision ϕ in model I and III is not large and model II and IV shows no large difference in estimating the drug precision estimate. Finally, assessing model performance, the Jeffreys prior with increase in $\psi^j(\theta)$ in θ -space increases the posterior estimate for θ . Performance evaluation was done using the deviance Information Criterion (DIC) and Kullback Leibler Divergence (KLD). Using this two approaches model IV is the best with minimum DIC and KLD shows that the posterior density converges to the true density with KLD=0.

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Modelling heat and mass transfer of a CO₂ binary mixture: a mathematical approach

R.O. Olayiwola^{*†}, A.T. Cole^{*}, M.D. Shehu^{*}, F.A. Oguntolu^{*}, E.E. Iyeme[‡], and A.W. Abubakar[§]

Abstract

This paper presents an analytical solutions for describing heat and mass transfer between a droplet of organic solvent and a compressed antisolvent taking into consideration the viscous energy dissipation and heat and mass transfer between the surface and the droplet by convection. The solvent and antisolvent are assumed to be fully miscible and have the same temperature. Both the initial temperature of the mixture and the initial carbon dioxide concentration are also assumed to depend on the space variable. The governing equations formulated based on the conservation of total mass, chemical species, momentum and energy were solved analytically using polynomial approximation method. The results obtained are presented graphically and discussed. The results revealed the effects of operating parameters on droplet lifetime. These results might be used for interpretation or experiments planning of the more complex real supercritical antisolvent process.

Keywords: carbon dioxide; drug particles; polynomial approximation method; precipitation; solvent; supercritical fluid.

1 Introduction

Particle formation technologies are important in the pharmaceutical industries, and in the production of photographic materials, ceramics, explosives, and dyes. The focus in drug formulation is to develop techniques that give more consistent and controlled particle size, morphology, and uniformity [4]. In current drug delivery technology, very small drug particles are of interest since they can travel faster to the target organ and distribute more evenly in the body [7].

Conventional methods for manipulating pharmaceutical particles include jet milling, spray drying, and emulsion techniques [2]. Jet milling uses particle-to-particle impact forces to break up the products into smaller pieces [10]. However, the jet milling process generally does not produce a uniform particle size distribution and running the process consumes a lot of energy [2]. Freeze drying results in particles of broad size ranges and usually require subsequent milling and sieving [12]. Spray drying usually gives

^{*}Department of Mathematics, Federal University of Technology, Minna, Nigeria

[†]Corresponding Author. E-mail: olayiwola.rasaq@futminna.edu.ng

[‡]Department of Mathematics, Cross River University of Technology, Calabar, Nigeria

[§]Department of Mathematics, Air Force Institute of Technology, Kaduna, Nigeria

particles of controllable size, but the extreme temperatures used during the operation can denature biological materials [17].

Conventional drug particle precipitation uses organic solvents as antisolvents for precipitation or as emulsifiers for emulsion process [18]. Traces of residual organic solvents, such as methylene chloride, that may still be in the drug particles have motivated researchers to find alternative methods for particle formation [16]. An alternative to reduce this problem is to use supercritical carbon dioxide (CO_2) as an antisolvent to precipitate drug particles from solution since CO_2 is nonflammable, non-toxic, inexpensive, renewable, and environmentally benign [11].

A supercritical fluid is any substance at a temperature and pressure above its critical point, where distinct liquid and gas phases do not exist. It can effuse through solids like a gas, and dissolve materials like a liquid. Carbon dioxide and water are the most commonly used supercritical fluids [14].

In order to successfully design a CO_2 antisolvent process, it is important to understand the effects of the operating parameters on particle characteristics. However, Bristow *et al.* [3] explained experimental particle size and morphology results in light of current hydrodynamic, kinetic and thermodynamic theories.

In the face of experimental difficulties, mathematical modeling appears as a useful tool to determine important parameters of the process. In view of this, Kumar *et al.* [9] developed a mathematical model of the droplet moving in a supercritical carbon dioxide environment. The solvent chosen in their study is ethanol. They solved the equations using MATLAB software. Chong *et al.* [5] presented a numerical procedure of mathematical model for mass transfer between a droplet of organic solvent and a compressed antisolvent which is applicable to the supercritical antisolvent (SAS) method of particle formation. Their model equations were put into the form that allowed the application of the MATLAB standard solver pdepe. Wu *et al.* [19] modelled the mixing of a hydrocarbon droplet, containing a mixture of toluene and either n-decane or tetralin, in a reservoir of supercritical or near-critical water. Their study provided microscale information, such as multi-component partitioning, and insight into large scale mixing in applications. Almeida *et al.* [1] used a three-dimensional mathematical model to study the development of a jet of solution (ethanol and minocycline) expanded in pressurized carbon dioxide in order to interpret the process of development of regions of supersaturation of the solution. The commercial code ANSYS FLUENT was used to solve the model relating the impact of the flow of solution in the mixing chamber of the precipitation process.

In this paper, a mathematical model capable of predicting the composition of hydrocarbon and carbon dioxide in SAS process is presented. We assume constant density, diffusivity and thermal conductivity. The droplet is moving through CO_2 continuum and heat and mass transfer take place to and from the droplet which led to the supersaturation and then fine particle of solute. To simulate the flow analytically, we assume that the medium is not stagnant so the convective flux can be considered.

2 Model formulation

A ‘pseudo’ droplet of solvent (hydrocarbon droplet) immersed in a compressed antisolvent (carbon dioxide) in miscible conditions is considered. The space variable is r , $0 \leq r \leq R$, and time is t , $t > 0$. The state variables depending on (r, t) are the mixture temperature T , carbon dioxide mole fraction

X_c , solvent mole fraction X_h , radial velocity of droplet u and pressure p . The formulation of our model is guided by the following assumptions:

1. The solvent and the antisolvent are fully miscible so only one equation is needed to describe the mass transfer.
2. The medium is not stagnant so the convective flux can be considered.
3. The radial velocity (velocity of droplet) is non-zero because of temperature change and mixing of carbon dioxide and hydrocarbons.
4. The heat and mass transfer between the surface and the mixture take place by convection.
5. The spherical symmetry is considered, making the problem one-dimensional.
6. The viscous energy dissipation is considered.
7. There is no heat source.

The diffusive flux is assumed to be proportional to the concentration gradient as described by Fick's law:

$$N_c = -\rho D \nabla X_c + X_c N. \quad (1)$$

The mass balance on carbon dioxide reads:

$$\frac{\partial}{\partial t}(\rho X_c) + \nabla \cdot (-\rho D \nabla X_c + X_c N) = 0. \quad (2)$$

The continuity equation required to find the convective flux N is:

$$\frac{\partial \rho}{\partial t} + \nabla \cdot N = 0. \quad (3)$$

Multiplying (3) by X_c and combining with (2) yields:

$$\rho \frac{\partial X_c}{\partial t} + N \nabla \cdot X_c = \nabla \cdot (\rho D \nabla X_c). \quad (4)$$

In a similar manner, momentum and energy conservation equations can be obtained respectively as:

$$\rho \frac{\partial u}{\partial t} + N \nabla \cdot u = -\frac{\partial p}{\partial x} + \frac{\partial}{\partial x} \left(2\mu \frac{\partial u}{\partial x} - \frac{2}{3} \mu \nabla \cdot u \right) \quad (5)$$

$$c_p \left(\rho \frac{\partial T}{\partial t} + N \nabla \cdot T \right) = \nabla \cdot (\lambda \nabla T) + \rho c_p D \frac{1000}{M} \frac{\partial X_c}{\partial x} \frac{\partial T}{\partial x} + \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 - \frac{2}{3} \left(\frac{\partial u}{\partial x} \right)^2 \right). \quad (6)$$

Introducing an apparent flux to replace convective flux (i.e., $N = \rho u$) yields in one-dimensional Cartesian coordinates:

$$\frac{\partial \rho}{\partial t} + \frac{\partial}{\partial x}(\rho u) = 0 \quad (7)$$

$$\rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right) = -\frac{\partial p}{\partial x} + \frac{\partial}{\partial x} \left(2\mu \frac{\partial u}{\partial x} - \frac{2}{3} \mu \frac{\partial u}{\partial x} \right), \quad (8)$$

$$\rho \left(\frac{\partial X_c}{\partial t} + u \frac{\partial X_c}{\partial r} \right) = \frac{\partial}{\partial x} \left(\rho D \frac{\partial X_c}{\partial x} \right), \quad (9)$$

$$\rho c_p \left(\frac{\partial T}{\partial t} + u \frac{\partial T}{\partial x} \right) = \frac{\partial}{\partial x} \left(\lambda \frac{\partial T}{\partial x} \right) + \rho c_p D \frac{1000}{M} \frac{\partial X_c}{\partial x} \frac{\partial T}{\partial x} + \mu \left(2 \left(\frac{\partial u}{\partial x} \right)^2 - \frac{2}{3} \left(\frac{\partial u}{\partial x} \right)^2 \right). \quad (10)$$

We can eliminate the continuity equation (7) by means of streamline function (see, [13]),

$$\eta(x, t) = (\rho^2)^{-\frac{1}{2}} \int_0^x \rho(s, t) ds \quad (11)$$

The coordinate transformation becomes,

$$\frac{\partial}{\partial x} \rightarrow \frac{\partial}{\partial \eta} \frac{\partial \eta}{\partial x} = \frac{\partial}{\partial \eta} \quad (12)$$

$$\frac{\partial}{\partial t} \rightarrow \frac{\partial}{\partial \eta} \frac{\partial \eta}{\partial t} + \frac{\partial}{\partial t} = -u \frac{\partial}{\partial \eta} + \frac{\partial}{\partial t}. \quad (13)$$

We make the additional assumptions that c_p , λ , μ and ρD are constant. Although these assumptions could be relaxed in the future, they considerably simplify the equations. The equations can be simplified as

$$\frac{\partial u}{\partial t} = -\frac{1}{\rho} \frac{\partial p}{\partial \eta} + \nu \frac{\partial}{\partial \eta} \left(2 \frac{\partial u}{\partial \eta} - \frac{2}{3} \frac{\partial u}{\partial \eta} \right) \quad (14)$$

$$\frac{\partial X_c}{\partial t} = D \frac{\partial}{\partial \eta} \left(\frac{\partial X_c}{\partial \eta} \right) \quad (15)$$

$$\frac{\partial T}{\partial t} = \frac{\lambda}{\rho c_p} \frac{\partial}{\partial \eta} \left(\frac{\partial T}{\partial \eta} \right) + \frac{1000D}{M} \frac{\partial X_c}{\partial \eta} \frac{\partial T}{\partial \eta} + \frac{\nu}{c_p} \left(2 \left(\frac{\partial u}{\partial \eta} \right)^2 - \frac{2}{3} \left(\frac{\partial u}{\partial \eta} \right)^2 \right). \quad (16)$$

In spherical coordinates system (see, [6]), (14) – (16) become

$$\frac{\partial u}{\partial t} = -\frac{1}{\rho} \frac{\partial p}{\partial r} + \nu \frac{\partial}{\partial r} \left(2 \frac{\partial u}{\partial r} - \frac{2}{3} \frac{1}{r^2} \frac{\partial}{\partial r} (r^2 u) \right) \quad (17)$$

$$\frac{\partial X_c}{\partial t} = \frac{D}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial X_c}{\partial r} \right) \quad (18)$$

$$\frac{\partial T}{\partial t} = \frac{\lambda}{\rho c_p r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial T}{\partial r} \right) + \frac{1000D}{M} \frac{\partial X_c}{\partial r} \frac{\partial T}{\partial r} + \frac{\nu}{c_p} \left(2 \left(\frac{\partial u}{\partial r} \right)^2 + 4 \left(\frac{u}{r} \right)^2 - \frac{2}{3} \left(\frac{\partial u}{\partial r} + \frac{2u}{r} \right)^2 \right), \quad (19)$$

where u is the radial velocity (velocity of droplet).

The mole fraction of the solvent (hydrocarbon droplet) is directly deduced by the relation:

$$X_h = 1 - X_c \quad (20)$$

Darcy's law

$$u = -\frac{K}{\mu} \frac{\partial p}{\partial r}. \quad (21)$$

To avoid potential convergence problems, the initial and boundary conditions is formulated as follows:
 Initial condition:

At $t = 0$ and $\forall r$,

$$u = U_s, \quad T = \frac{1}{2} T_{c0} (1 + \tanh(\alpha(r - R))), \quad X_c = \frac{1}{2} X_{c0} (1 + \tanh(\alpha(r - R))) \quad (22)$$

Boundary conditions:

$$\left. \begin{aligned} \frac{\partial u}{\partial r} \Big|_{r=0} &= 0, & \frac{\partial u}{\partial r} \Big|_{r=R} &= q \\ \frac{\partial X_c}{\partial r} \Big|_{r=0} &= 0, & D^* \frac{\partial X_c}{\partial r} \Big|_{r=R} &= k_m X_c \Big|_{r=R} \\ \frac{\partial T}{\partial r} \Big|_{r=0} &= 0, & \lambda^* \frac{\partial T}{\partial r} \Big|_{r=R} &= h T \Big|_{r=R}, \quad T_{c0} > T_{h0} \\ p \Big|_{r=R} &= p_{out} \end{aligned} \right\}, \quad (23)$$

where X_c is the mole fraction of the carbon dioxide, X_h is the mole fraction of the solvent (hydrocarbon droplet), D is the diffusion coefficient of the solvent in the carbon dioxide, ρ is the density of the binary mixture, λ is heat conductivity of the mixture, K is the permeability, R is the droplet radius, T is the temperature of the mixture, M is the molecular weight, T_{c0} is the initial temperature of carbon dioxide, T_{h0} is the initial temperature of hydrocarbon, X_{c0} is the initial concentration of carbon dioxide, X_{h0} is the initial concentration of hydrocarbon, p is the pressure, u is the radial velocity of droplet, U_s is the surface velocity, μ is the dynamic viscosity, c_p is the constant pressure specific heat, r is the radial coordinate, K is the permeability, D^* is the effective diffusion coefficient, λ^* is the effective heat conductivity, k_m is the convective mass transfer coefficient, h is the convective heat transfer coefficient, p_{out} is the outlet pressure, q is constant.

3 Method of solution

3.1 Dimensional analysis

Dimensionless variables for space and time is been introduced as:

$$r' = \frac{r}{R}, \quad t' = \frac{t}{t^*}, \quad t^* = \frac{R}{U_s} \quad (24)$$

Dimensionless variables for radial velocity of droplet, mixture temperature, carbon dioxide mole fraction and hydrocarbon mole fraction is been introduced as follows:

$$\theta = \frac{T}{T_{c0}}, \quad \phi = \frac{X_c}{X_{c0}}, \quad \psi = \frac{X_h}{X_{c0}}, \quad u' = \frac{u}{U_s}, \quad p' = \frac{p}{\rho U_s^2} \quad (25)$$

where t^* is reference values for time.

Using (24) and (25), and after dropping the prime, equations (17) - (23) become

$$\frac{\partial u}{\partial t} = -\frac{\partial p}{\partial r} + \frac{4}{3} \frac{1}{R_e} \left(\frac{\partial^2 u}{\partial r^2} - \frac{1}{r} \left(\frac{\partial u}{\partial r} - \frac{u}{r} \right) \right) \quad (26)$$

$$\frac{\partial \phi}{\partial t} = \frac{1}{P_{em}} \frac{1}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \phi}{\partial r} \right) \quad (27)$$

$$\frac{\partial \theta}{\partial t} = \frac{1}{P_e} \frac{1}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \theta}{\partial r} \right) + \frac{\beta}{P_{em}} \frac{\partial \phi}{\partial r} \frac{\partial \theta}{\partial r} + \frac{4}{3} g \frac{E_c}{R_e} \left(\frac{\partial u}{\partial r} - \frac{u}{r} \right)^2 \quad (28)$$

$$\psi(r, t) = \sigma - \phi(r, t) \quad (29)$$

$$\frac{\partial p}{\partial r} = -\frac{1}{D_a R_e} u \quad (30)$$

$$\left. \begin{aligned} u(r, 0) &= 1, & u_r(0, t) &= 0, & u_r(1, t) &= \gamma \\ \phi(r, 0) &= \frac{1}{2} (1 + \tanh(\alpha'(r-1))), & \phi_r(0, t) &= 0, & \phi_r(1, t) &= -Sh\phi(1, t) \\ \theta(r, 0) &= \frac{1}{2} (1 + \tanh(\alpha'(r-1))), & \theta_r(0, t) &= 0, & \theta_r(1, t) &= -Nu\theta(1, t) \\ p_r(1, t) &= \delta \end{aligned} \right\} \quad (31)$$

where

$R_e = \frac{RU_s}{\nu}$ is the Reynolds number, $P_{em} = \frac{RU_s}{D}$ is the Peclet mass number, $P_e = \frac{\rho c_p RU_s}{\lambda}$ is the Peclet energy number, $E_c = \frac{U_s^2}{g c_p T_0}$ is the Eckert number, $\sigma = \frac{1}{X_0}$, $\beta = \frac{1000 X_0}{M}$, $\alpha' = \alpha R$,

$D_a = \frac{K}{R^2}$ is the Darcy number, $Sh = \frac{Rk_m}{D^*}$ is the Sherwood number, $Nu = \frac{Rh}{\lambda^*}$ is the Nusselt number, $\gamma = \frac{Rq}{U_s}$, $\delta = \frac{p_{out}}{\rho U_s^2}$

3.2 Solution via Polynomial Approximation Method

Here, we assume polynomial solution of the form (see, [15]):

$$\phi(r, t) = a_0(t) + a_1(t)r + a_2(t)r^2 \quad (32)$$

$$u(r, t) = b_0(t) + b_1(t)r + b_2(t)r^2 \quad (33)$$

$$\theta(r, t) = c_0(t) + c_1(t)r + c_2(t)r^2. \quad (34)$$

Applying the boundary conditions as given in (31), we obtain

$$\begin{aligned} a_1(t) = b_1(t) = c_1(t) = 0, \quad a_2(t) = -\frac{Sh}{2}\phi|_{r=1}, \quad b_2(t) = \frac{\gamma}{2}, \quad c_2(t) = -\frac{Nu}{2}\theta|_{r=1}, \\ a_0(t) = \left(1 + \frac{Sh}{2}\right)\phi|_{r=1}, \quad b_0(t) = u|_{r=1} - \frac{\gamma}{2}, \quad c_0(t) = \left(1 + \frac{Nu}{2}\right)\theta|_{r=1} \end{aligned} \quad (35)$$

Then, equation (32) – (33) become

$$\phi(r, t) = \left(1 + \frac{Sh}{2}\right)\phi|_{r=1} - \frac{Sh}{2}\phi|_{r=1}r^2 \quad (36)$$

$$u(r, t) = u|_{r=1} - \frac{\gamma}{2} + \frac{\gamma}{2}r^2 \quad (37)$$

$$\theta(r, t) = \left(1 + \frac{Nu}{2}\right)\theta|_{r=1} - \frac{Nu}{2}\theta|_{r=1}r^2 \quad (38)$$

For long spherical shape (see, [8]), we have

$$\bar{\phi} = 3 \int_0^1 r^2 \phi dr \quad (39)$$

$$\bar{u} = 3 \int_0^1 r^2 u dr \quad (40)$$

$$\bar{\theta} = 3 \int_0^1 r^2 \theta dr \quad (41)$$

where $\bar{\phi}$ is the average mole fraction, $\bar{u}$ is the average velocity, $\bar{\theta}$ is the average temperature.

Equations (39) – (41) give the relations

$$\bar{\phi} = \left(1 + \frac{Sh}{2}\right)\phi|_{r=1}, \quad \bar{u} = u|_{r=1} - \frac{2\gamma}{5}, \quad \bar{\theta} = \left(1 + \frac{Nu}{2}\right)\theta|_{r=1} \quad (42)$$

and

$$\frac{\partial}{\partial t}\bar{\phi} = \left(1 + \frac{Sh}{2}\right)\frac{\partial}{\partial t}\phi|_{r=1}, \quad \frac{\partial}{\partial t}\bar{u} = \frac{\partial}{\partial t}u|_{r=1}, \quad \frac{\partial}{\partial t}\bar{\theta} = \left(1 + \frac{Nu}{2}\right)\frac{\partial}{\partial t}\theta|_{r=1} \quad (43)$$

Integrating (26) – (28) with respect to r , yield the following equations

$$\left. \frac{\partial}{\partial t} \phi \right|_{r=1} + a \phi|_{r=1} = 0 \quad (44)$$

$$\left. \frac{\partial}{\partial t} u \right|_{r=1} - b u|_{r=1} = c \quad (45)$$

$$\left. \frac{\partial}{\partial t} \theta \right|_{r=1} + (A - B e^{-at}) \theta|_{r=1} = D(F - G e^{bt} + E^2 e^{2bt}) \quad (46)$$

Solving (44) – (46) gives

$$\phi|_{r=1} = \frac{1}{2} e^{-at} \quad (47)$$

$$u|_{r=1} = \left(\left(\frac{c}{b} + 1 \right) e^{bt} - \frac{c}{b} \right) \quad (48)$$

$$\theta|_{r=1} = \left(\begin{array}{l} FD \left[\frac{1}{A} \left(1 - e^{-\left(At + \frac{B}{a} e^{-at} \right)} \right) - \right. \\ \left. \frac{B}{A} \left(\left(e^{-\left(At + \frac{B}{a} e^{-at} \right)} - e^{(1-A)t} \right) + \frac{B\sqrt{\pi}}{\sqrt{-aB}} e^{-\left(At + \frac{B}{a} e^{-at} \right)} \operatorname{erf} \left(\frac{\sqrt{-aB}}{a} (e^{-t} - 1) \right) \right) \right] - \\ \\ DG \left[\frac{1}{A+b} \left(e^{bt} - e^{-\left(At + \frac{B}{a} e^{-at} \right)} \right) - \right. \\ \left. \frac{B}{A+b} \left(\left(e^{-\left(At + \frac{B}{a} e^{-at} \right)} - e^{(1-A)t} \right) + \frac{B\sqrt{\pi}}{\sqrt{-aB}} e^{-\left(At + \frac{B}{a} e^{-at} \right)} \operatorname{erf} \left(\frac{\sqrt{-aB}}{a} (e^{-t} - 1) \right) \right) \right] + \\ \\ DE^2 \left[\frac{1}{A+2b} \left(e^{2bt} - e^{-\left(At + \frac{B}{a} e^{-at} \right)} \right) - \right. \\ \left. \frac{B}{A+2b} \left(\left(e^{-\left(At + \frac{B}{a} e^{-at} \right)} - e^{(1-A)t} \right) + \frac{B\sqrt{\pi}}{\sqrt{-aB}} e^{-\left(At + \frac{B}{a} e^{-at} \right)} \operatorname{erf} \left(\frac{\sqrt{-aB}}{a} (e^{-t} - 1) \right) \right) \right] \end{array} \right) + h(1) e^{-\left(At + \frac{B}{a} e^{-at} \right)} \quad (49)$$

Substituting equations (47) – (49) into equations (36) – (38) give

$$\phi(r, t) = \frac{1}{2} \left(1 + \frac{Sh}{2} \right) e^{-at} - \frac{Sh}{4} r^2 e^{-at} \quad (50)$$

$$u(r, t) = \left(\left(\frac{c}{b} + 1 \right) e^{bt} - \frac{c}{b} \right) - \frac{\gamma}{2} + \frac{\gamma}{2} r^2 \quad (51)$$

$$\theta(r, t) = \left(1 + \frac{Nu}{2}\right) \theta|_{r=1} - \frac{Nu}{2} \theta|_{r=1} r^2 \quad (52)$$

$$\psi(r, t) = \sigma - \frac{1}{2} \left(1 + \frac{Sh}{2}\right) e^{-at} + \frac{Sh}{4} r^2 e^{-at} \quad (53)$$

$$p(r, t) = \delta + \frac{1}{DaRe} \left(\left(\left(\frac{c}{b} + 1 \right) e^{bt} - \frac{c}{b} \right) - \frac{\gamma}{3} \right) - \frac{1}{DaRe} \left(\left(\left(\frac{c}{b} + 1 \right) e^{bt} - \frac{c}{b} \right) - \frac{\gamma}{2} + \frac{\gamma}{6} r^2 \right) r, \quad (54)$$

where

$$\begin{aligned} a &= \frac{3Sh}{\left(1 + \frac{Sh}{5}\right) P_{em}}, & b &= \frac{1}{Re} \left(4 + \frac{1}{Da}\right), & c &= -\frac{1}{Re} \left(\frac{4}{3} + \frac{1}{5Da}\right), \\ A &= \frac{Nu}{\left(1 + \frac{Nu}{5}\right) P_e}, & B &= \frac{\beta Nu Sh}{10 \left(1 + \frac{Nu}{5}\right) P_{em}}, & D &= \frac{4gEc}{3 \left(1 + \frac{Nu}{5}\right) Re}, \\ E &= \left(\frac{c}{b} + 1\right), & F &= \left(\frac{4\gamma c}{3b} + \frac{4\gamma^2}{9}\right), & G &= E \left(\frac{4\gamma}{3} + \frac{2c}{b}\right), \\ h(1) &= e^{\frac{B}{a}} \left[\frac{1}{2} - \left(FD \left(\frac{1}{A} \left(1 - e^{-\frac{B}{a}}\right) - \frac{B}{A} \left(e^{\frac{B}{a}} - 1\right)\right) - DG \left(\frac{1}{A+b} \left(1 - e^{-\frac{B}{a}}\right) - \frac{B}{A+b} \left(e^{\frac{B}{a}} - 1\right)\right) + \right. \right. \\ &\quad \left. \left. DE^2 \left(\frac{1}{A+2b} \left(1 - e^{-\frac{B}{a}}\right) - \frac{B}{A+2b} \left(e^{\frac{B}{a}} - 1\right)\right) \right) \right] \end{aligned}$$

The computations were done on equations (50) – (54) using computer symbolic algebraic package MAPLE 2021.

4 Results and Discussion

4.1 Analysis of Results

The transport and mixing processes are simulated analytically for a droplet of solvent (hydrocarbon) and a compressed antisolvent (carbon dioxide) in miscible conditions using polynomial approximation method. Analytical solutions given by equations (50) - (54) are computed using computer symbolic algebraic package MAPLE 2021. The numerical results obtained from the method are shown in Figures 1 to 18.

4.2 State Variables Dynamics

We performed the numerical simulations of the system of differential equations of the state variables to determine the changes in the various state variables with time and space. There seems to be a continuous increase in the mixture temperature, droplet velocity, hydrocarbon concentration and pressure as the carbon dioxide concentration decreases with time. Our findings showed an inverse relationship among the mixture temperature, droplet velocity, hydrocarbon concentration, pressure and carbon dioxide concentration as shown in Figures 1 – 5.

Figure 1 depicts the graph of mixture temperature $\theta(r, t)$ against time t for different values of droplet radius r . It is observed that the temperature of the mixture increases with time and this mixture temperature is higher at the centre of the droplet than at the end of the domain.

Figure 2 shows the graph of carbon dioxide concentration $\phi(r, t)$ against time t for different values of droplet radius r . It is observed that the concentration of carbon dioxide decreases with time and this concentration is higher at the centre of the droplet than at the end of the domain.

Figure 3 displays the graph of hydrocarbon concentration $\psi(r, t)$ against time t for different values of droplet radius r . It is observed that the concentration of hydrocarbon increases with time and this concentration is higher at the end of the domain than at the centre of the droplet.

Figure 4 manifests the graph of droplet velocity $u(r, t)$ against time t for different values of droplet radius r . It is observed that the velocity of droplet increases with time and this velocity is higher at the end of the domain than at the centre of the droplet.

Figure 5 depicts the graph of pressure $p(r, t)$ against time t for different values of droplet radius r . It is observed that the pressure increases with time and this pressure increase throughout the domain except at the end of the domain where it constant.

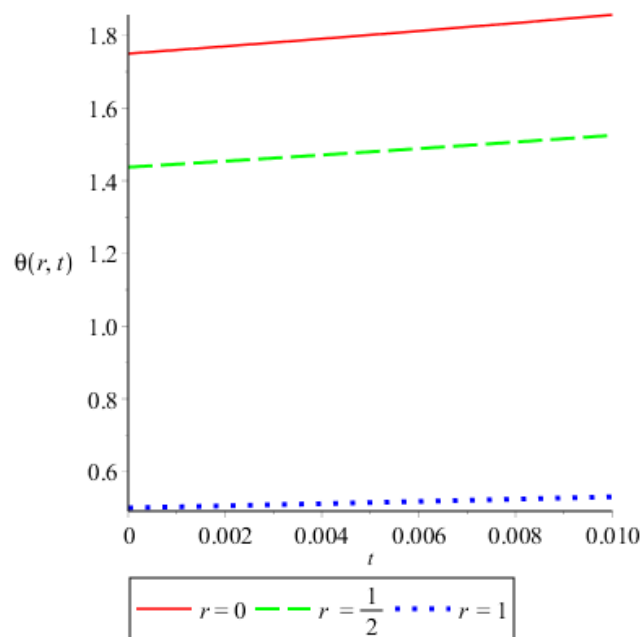


Figure 1: Mixture Temperature

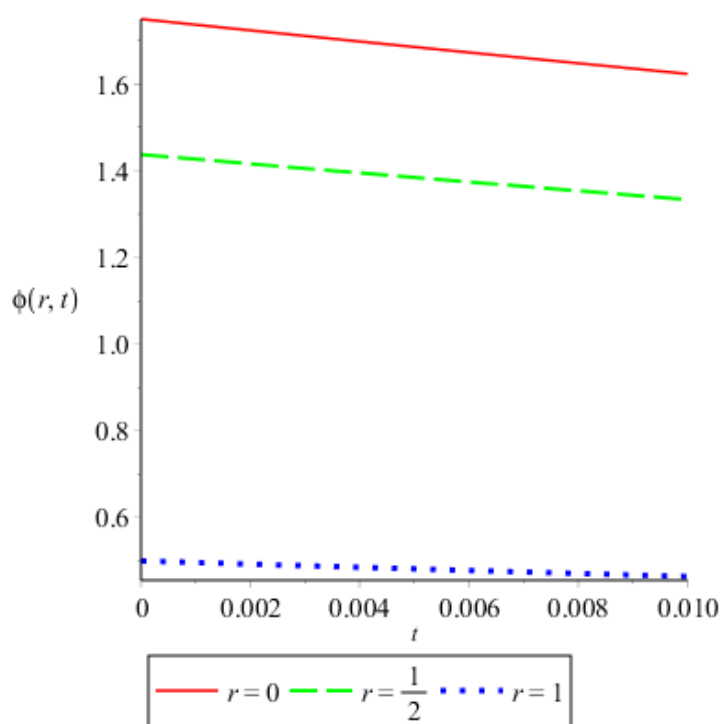


Figure 2: Carbon dioxide Concentration

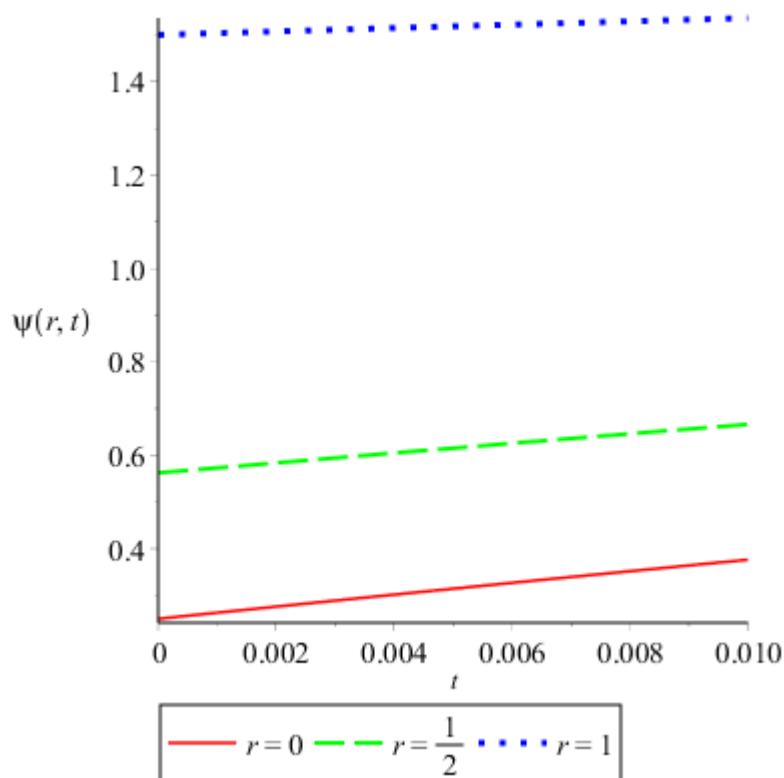


Figure 3: Hydrocarbon Concentration

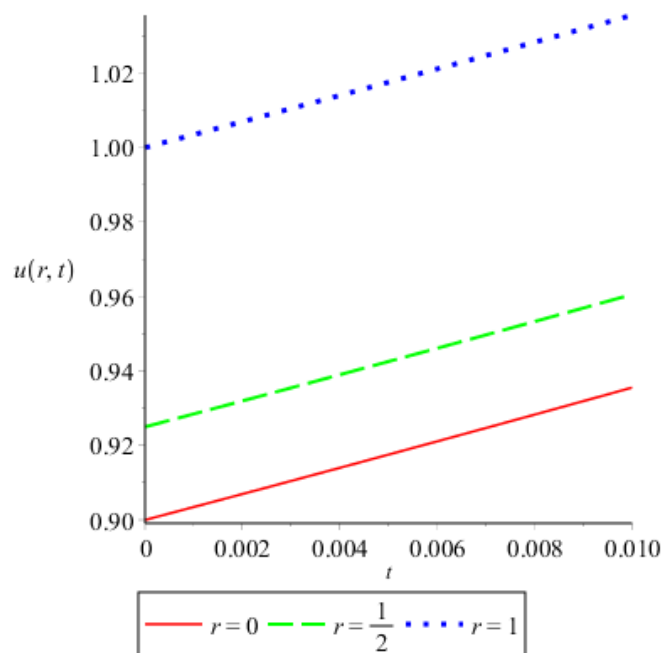


Figure 4: Droplet Velocity

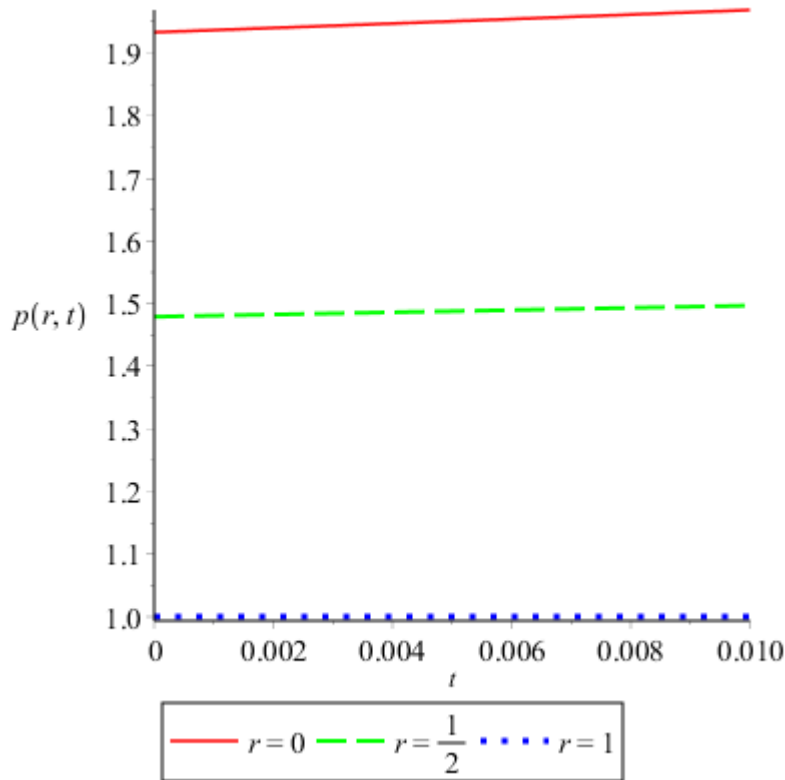


Figure 5: Pressure

4.3 Effects of Nusselt Number, Nu

Numerical analysis of the Nusselt number was conducted to see whether or not the Nusselt number contribute significantly to the mixture temperature.

Figure 6 discloses the graph of mixture temperature $\theta(r, t)$ against time t and droplet radius r for different values of Nusselt number Nu . It is observed that the temperature of mixture increases with time and decreases along droplet radius but this temperature increases as Nusselt number increases.

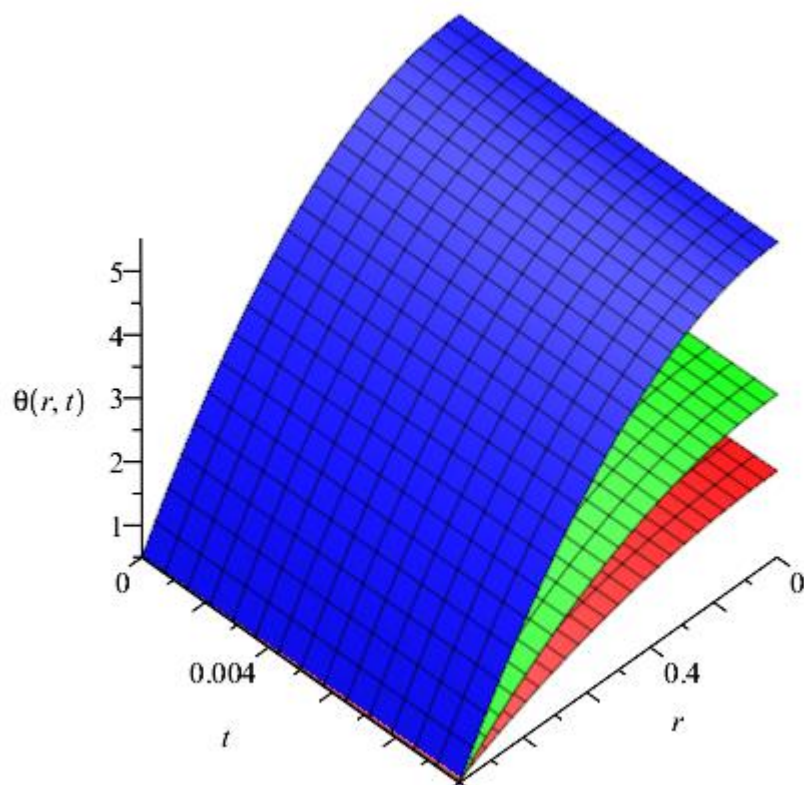


Figure 6: Effects of Nusselt number, Nu on mixture temperature

4.4 Effects of Peclet Energy Number, P_e

Numerical analysis of the Peclet energy number was conducted to see whether or not the Peclet energy number contribute significantly to the mixture temperature.

Figure 7 depicts the graph of mixture temperature $\theta(r, t)$ against time t and droplet radius r for different values of Peclet energy number P_e . It is observed that the temperature of mixture increases with time and decreases along droplet radius but this temperature increases as Peclet energy number increases.

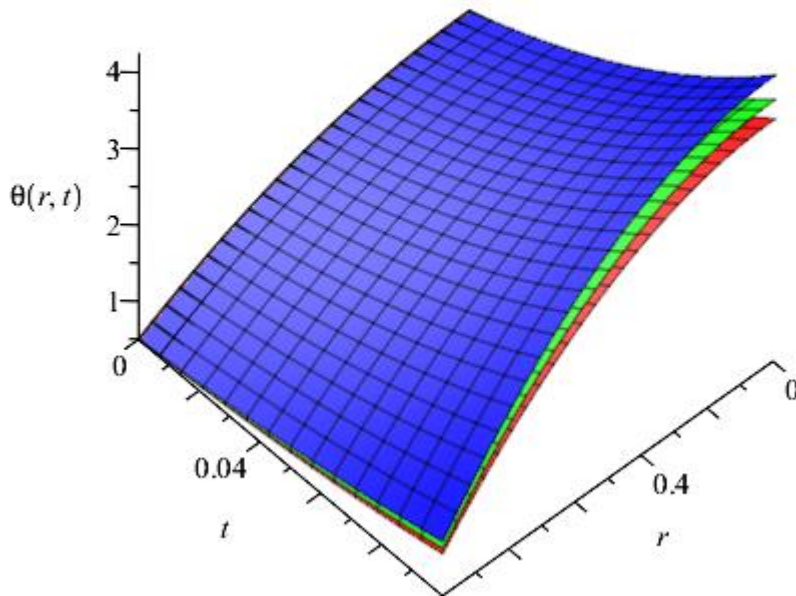


Figure 7: Effects of Peclet energy number, P_e on mixture temperature

4.5 Effects of Eckert Number, E_c

Numerical analysis of the Peclet energy number was conducted to see whether or not the Peclet energy number contribute significantly to the mixture temperature.

Figure 8 shows the graph of mixture temperature $\theta(r, t)$ against time t and droplet radius r for different values of Eckert number E_c . It is observed that the temperature of mixture increases with time and decreases along droplet radius but this temperature increases as Eckert number increases.

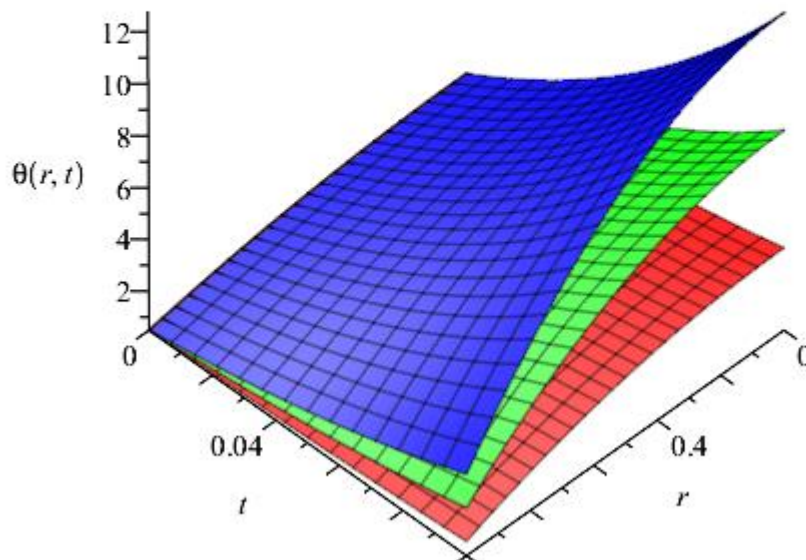


Figure 8: Effects of Eckert number, E_c on mixture temperature

4.6 Effects of Darcy Number, D_a

Numerical analysis of the Darcy number was conducted to see whether or not the Darcy number contribute significantly to the mixture temperature, droplet velocity and pressure.

Figure 9 shows the graph of mixture temperature $\theta(r, t)$ against time t and droplet radius r for different values of Darcy number D_a . It is observed that the temperature of mixture increases with time and decreases along droplet radius but this temperature increases as Darcy number decreases.

Figure 10 displays the graph of droplet velocity $u(r, t)$ against time t and droplet radius r for different values of Darcy number D_a . It is observed that the velocity of droplet increases with time and increases along droplet radius but this velocity increases as Darcy number decreases. Figure 11 displays the graph of pressure $p(r, t)$ against time t and droplet radius r for different values of Darcy number D_a . It is observed that the pressure increases with time and decreases along droplet radius but this pressure increases as Darcy number decreases.

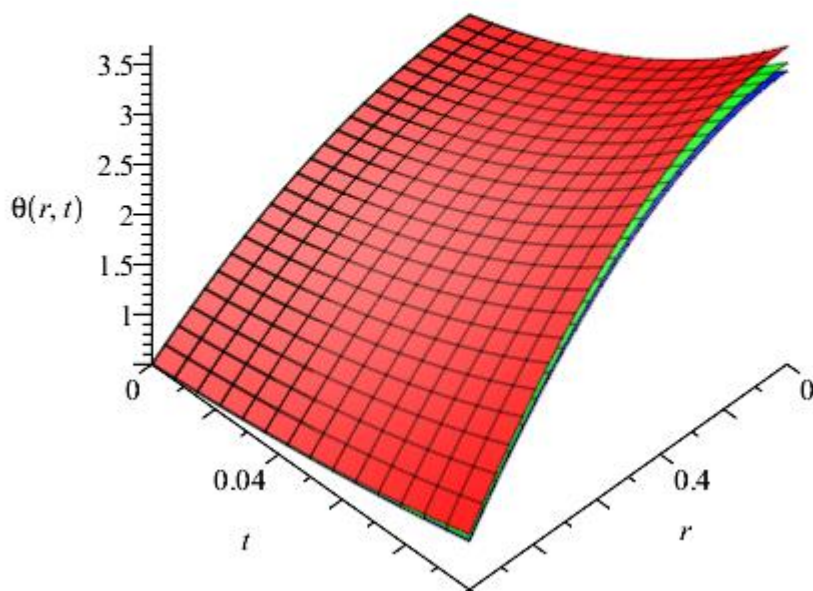


Figure 9: Effects of Darcy number, D_a on mixture temperature

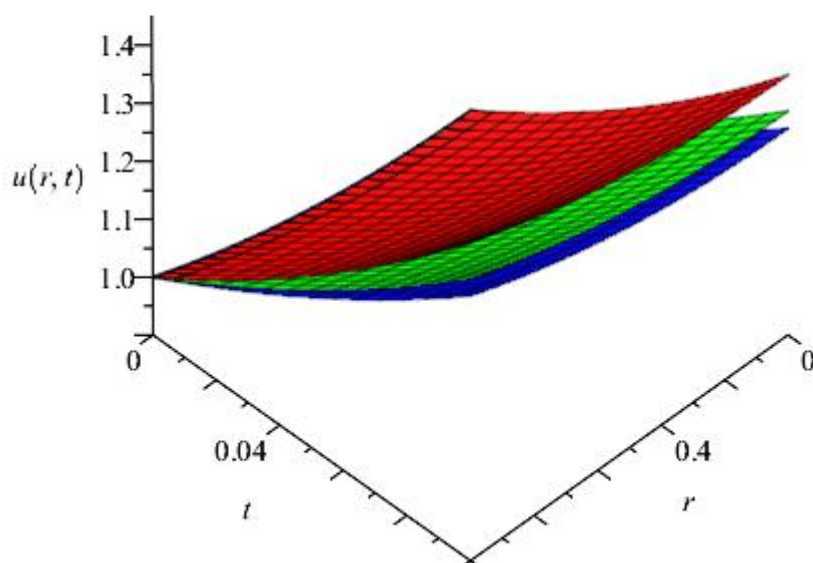


Figure 10: Effects of Darcy number, D_a on droplet velocity

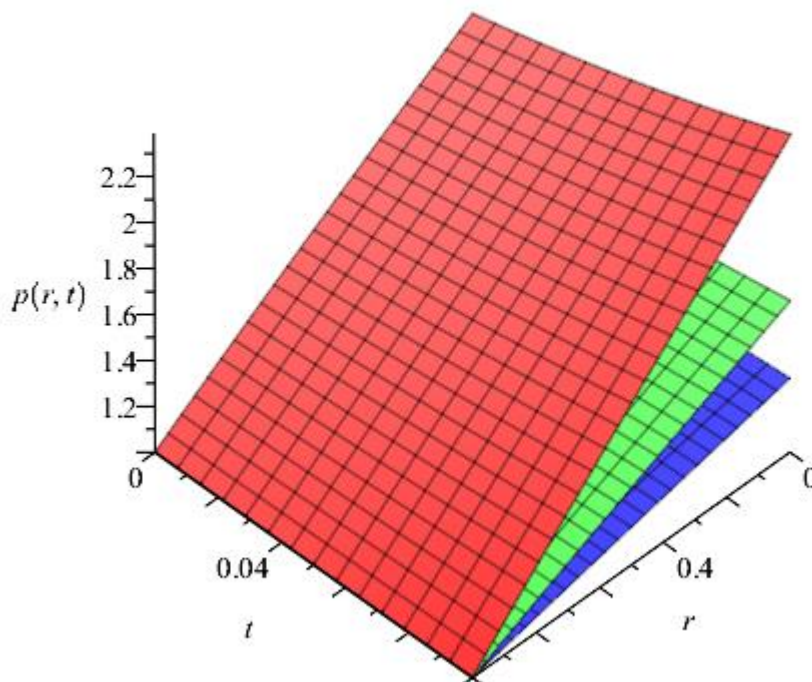


Figure 11: Effects of Darcy number, D_a on pressure

4.7 Effects of Sherwood Number, Sh

Numerical analysis of the Sherwood number was conducted to see whether or not the Sherwood number contribute significantly to the mixture temperature and carbon dioxide concentration.

Figure 12 shows the graph of mixture temperature $\theta(r, t)$ against time t and droplet radius r for different values of Sherwood number Sh . It is observed that the temperature of mixture increases with time and decreases along droplet radius but this temperature increases as Sherwood number decreases.

Figure 13 displays the graph of carbon dioxide concentration $\phi(r, t)$ against time t and droplet radius r for different values of Sherwood number Sh . It is observed that the concentration of carbon dioxide increases with time and decreases along droplet radius but this concentration increases as Sherwood number increases.

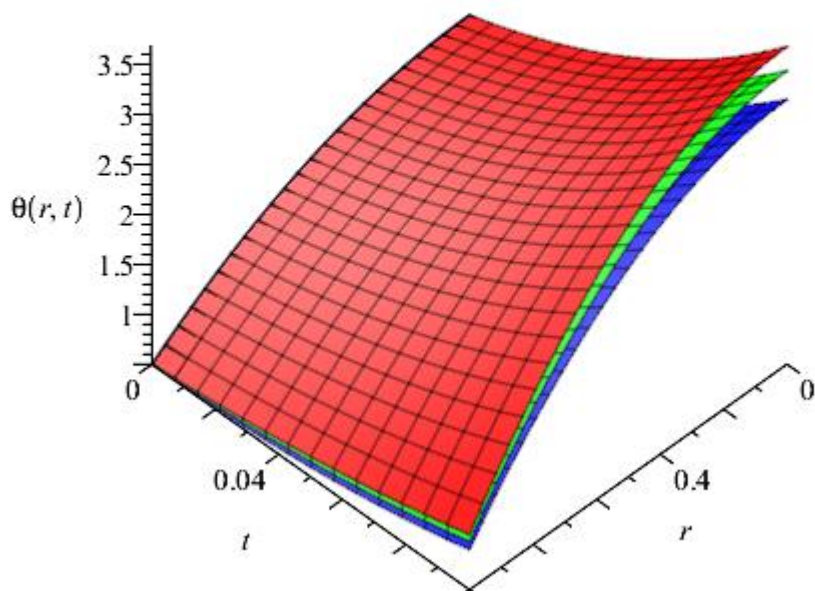


Figure 12: Effects of Sherwood number, Sh on mixture temperature

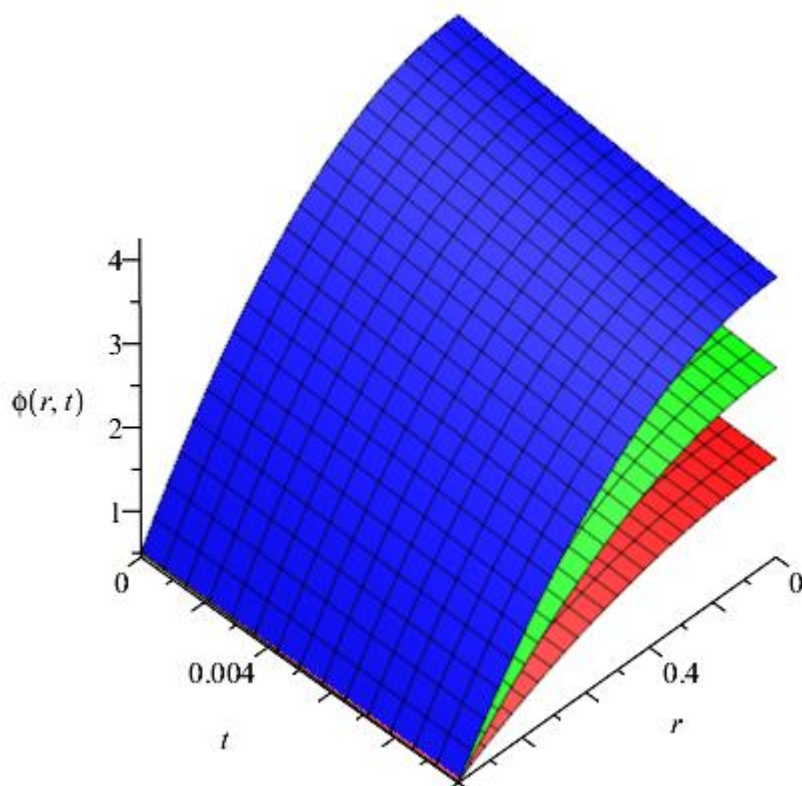


Figure 13: Effects of Sherwood number, Sh on Carbon dioxide concentration

4.8 Effects of Peclet Mass Number, P_{em}

Numerical analysis of the Peclet mass number was conducted to see whether or not the Peclet mass number contribute significantly to the carbon dioxide and hydrocarbon concentrations.

Figure 14 shows the graph of carbon dioxide concentration $\phi(r, t)$ against time t and droplet radius r for different values of Peclet mass number P_{em} . It is observed that the concentration of carbon dioxide decreases with time and decreases along droplet radius but this concentration increases as Darcy number increases.

Figure 15 displays the graph of hydrocarbon concentration $\psi(r, t)$ against time t and droplet radius r for different values of Peclet mass number P_{em} . It is observed that the concentration of hydrocarbon increases with time and increases along droplet radius but this concentration increases as Darcy number decreases.

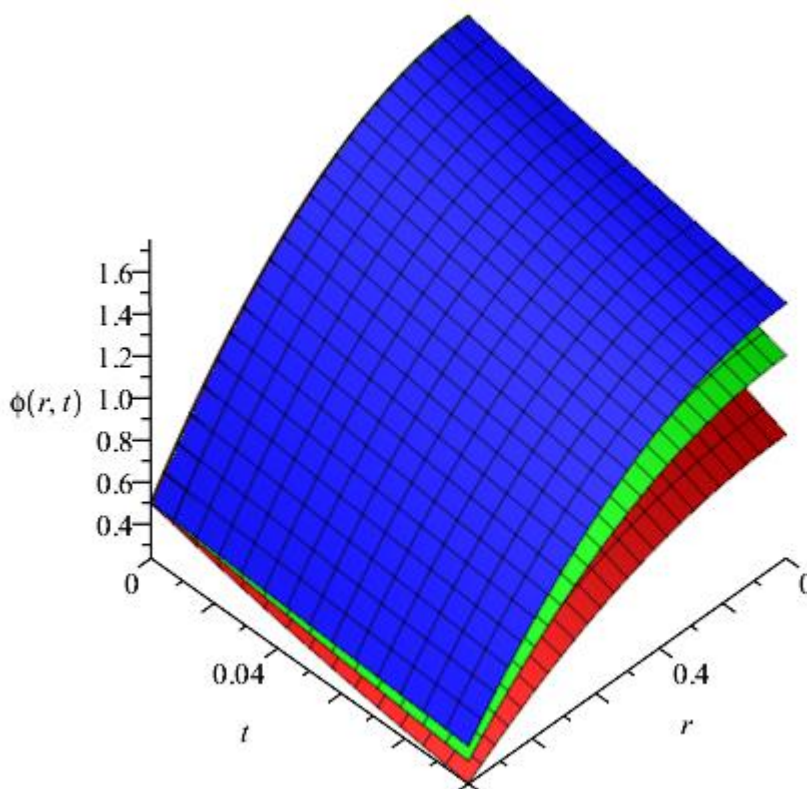


Figure 14: Effects of Peclet mass number, P_{em} on Carbon dioxide concentration

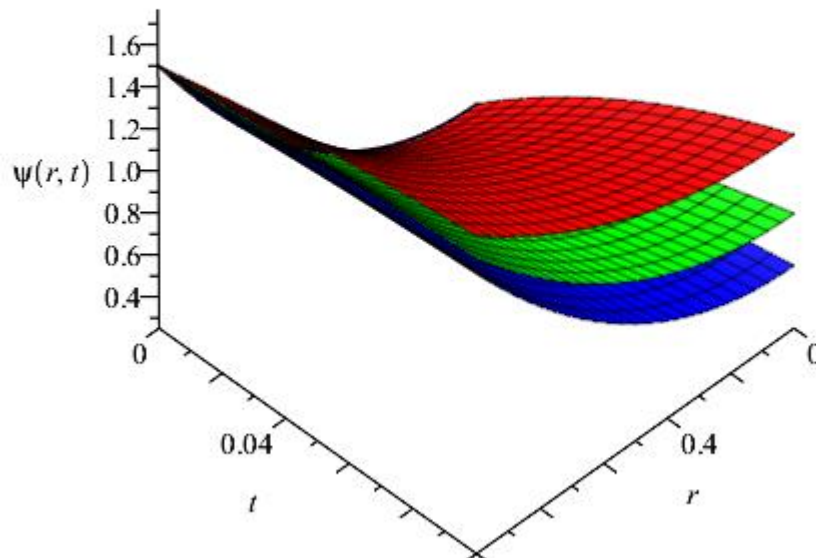


Figure 15: Effects of Peclet mass number, P_{em} on hydrocarbon concentration

4.9 Effects of Reynolds Number, R_e

Numerical analysis of the Reynolds number was conducted to see whether or not the Reynolds number contribute significantly to the mixture temperature, droplet velocity and pressure.

Figure 16 shows the graph of mixture temperature $\theta(r, t)$ against time t and droplet radius r for different values of Reynolds number R_e . It is observed that the temperature of mixture increases with time and decreases along droplet radius but this temperature increases as Reynolds number decreases. Figure 17 displays the graph of droplet velocity $u(r, t)$ against time t and droplet radius r for different values of Reynolds number R_e . It is observed that the velocity of droplet increases with time and increases along droplet radius but this velocity increases as Reynolds number decreases.

Figure 18 displays the graph of pressure $p(r, t)$ against time t and droplet radius r for different values of Reynolds number R_e . It is observed that the pressure increases with time and decreases along droplet radius but this pressure increases as Reynolds number decreases.

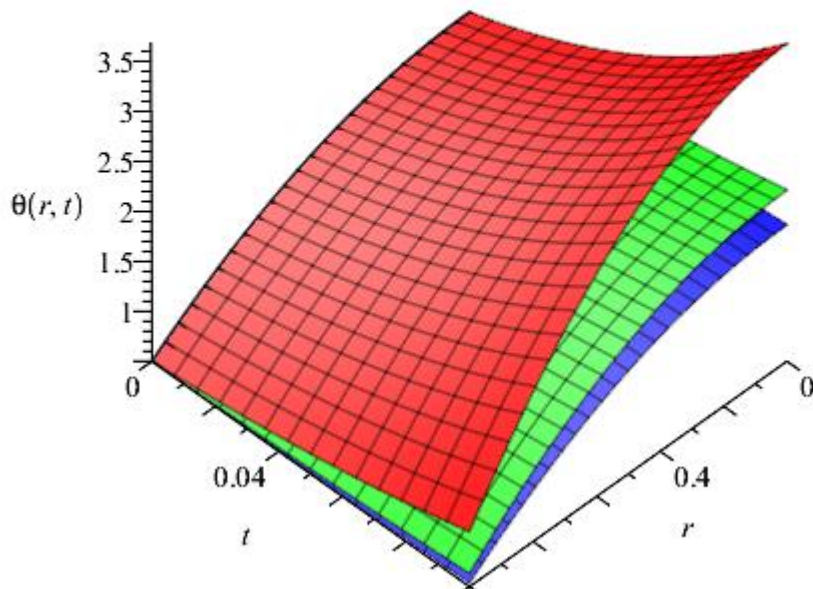


Figure 16: Effects of Reynolds number, R_e on mixture temperature

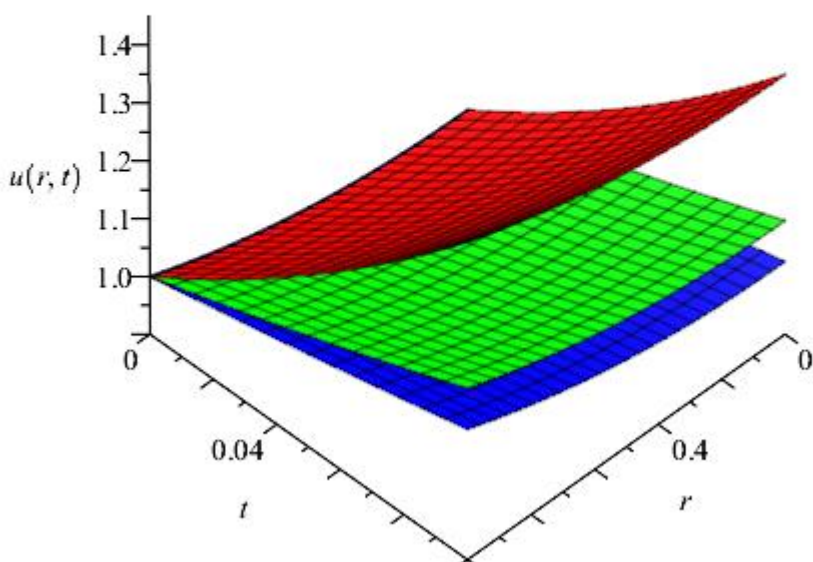


Figure 17: Effects of Reynolds number, R_e on droplet velocity

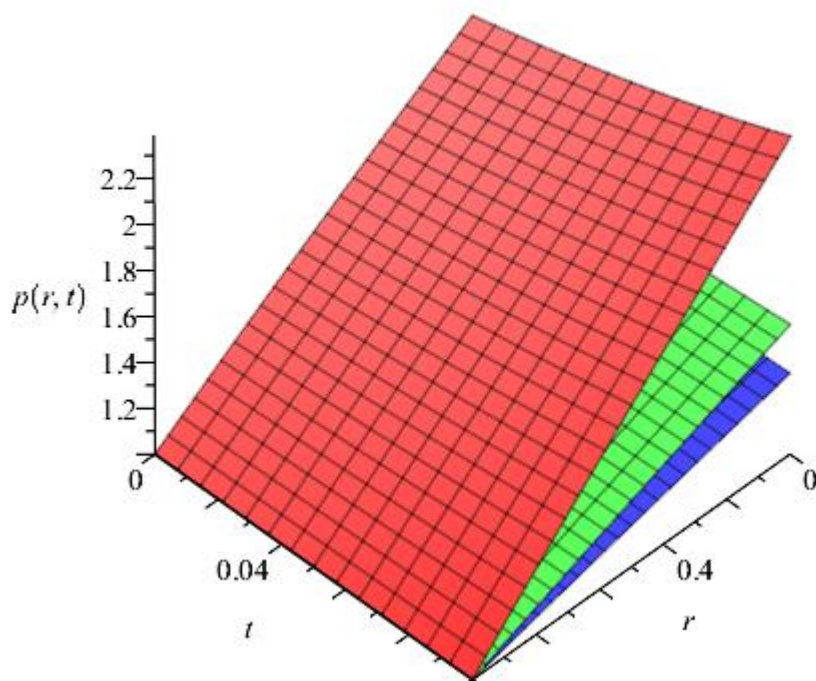


Figure 18: Effects of Reynolds number, R_e on pressure

It is worth pointing out that the effects observed in Figures 1 to 18, are important for designing and optimizing different CO_2 antisolvent processes for the formulation of small crystalline drug products.

5 Conclusion

This research work developed a model describing heat and mass transfer between a droplet of solvent (hydrocarbon) and a compressed antisolvent (carbon dioxide) in miscible conditions taking into consideration the viscous energy dissipation and transfer of heat and mass between the reservoir surface and the droplet by convection, in order to investigate the role of operating parameters on droplet lifetime. This model which relies on several assumptions is based on the conservation of total mass, chemical species, momentum and energy written in transient state mode of operation. The governing parameter of the problem are the Eckert number (E_c), Peclet energy number (P_e), Reynolds number (R_e), Peclet mass number (P_{em}), Darcy number (D_a), Nusselt number (Nu) and Sherwood number (Sh). The study revealed the following:

The mixture temperature, hydrocarbon concentration, pressure and velocity of droplet are increasing function of time.

The CO_2 concentration is a decreasing function of time.

The mixture temperature is higher at the centre of droplet than at the end of domain.

There is higher concentration of CO_2 at the centre of droplet than at the end of domain.

There is higher concentration of hydrocarbon at the end of domain than at the centre of droplet.

The velocity of droplet is higher at the end of domain than at the centre of droplet.
The pressure is higher at the end of domain than at the centre of droplet.

The results highlighted above showed that the particle formation in drug production could be controlled by the governing parameters involved. These results are useful in pharmaceutical industries for achieving very small drug particles of interest. The results of this study may be of importance to engineers and scholars attempting to develop programming standards and to researchers interested in the theoretical aspects of computer programming.

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A stochastic analysis of stock price variation assessments in Oando Nigeria, Plc

I.U. Amadi* and M.J.O. Vivian†

Abstract

This paper applied stochastic analysis of Markov chain in the closing stock price data of Oando Nigeria Plc (2017-2021). The stock prices were transformed into 3-steps transition probability matrix solution for each independent year. The criteria of obtaining future stock price changes of the year were considered. From the analytical solution of stochastic analysis, showed that the closing stock price of 2017 has the highest mean rate of returns and growth rates respectively: 2.4112 and 69%. Also stock prices of 2018 has the best probability of price not changing in the near future: 26% , which is a guide to investor for proper decision making. In all, the impact of future price changes were assessed which shows various levels of pricing effects on the aspect of percentage increase and decrease as it affects the original prices, Oando Nigeria PLC is profit making organization, hence the growth rates are all indexed in millions of naira.

Keywords: stock prices; stochastic analysis; Markov chain; Oando Nigeria Plc

1 Introduction

Stock market prices are highly violated; hence they are highly affected by some unforeseen circumstances. For instance, the rise and fall of dollar exchange rate political crisis and flooding etc . The impact of the activities can result to unstable nature of stock market price which causes panic buying of goods and services. So, it is worth developing a mathematical model which can enhance realistic result for the purpose of practical findings. Therefore, Markov chain is adopted to examine the closing stock price of Oando Nigeria Plc; since stock price is a formation of stochastic process. The stochastic analysis of these data of Oando Plc will aid investors to find prospects on the independent years of stock prices throughout the trading days.

*Corresponding Author. Department of Mathematics/ Statistics, Captain Elecchi Amadi Polytechnic Rumuola, Port Harcourt, Nigeria; E-mail: amadiuchennainno@gmail.com.

†Department of Mathematics/ Statistics, Ignatius Ajuru university of Education, Rumuolumeni, Port Harcourt, Nigeria; E-mail: oleremeakaevivian@gmail.com .

Nevertheless, a lots scholar has written extensively on the modeling stock price using Markov chain and results obtained in various ways. For instance, [8] examined stock market prices due to its fluctuations and influences in financial lives and economic health of a country. Their findings showed that stock price is a random work and no investor can alter the fairness and unfairness of a stock price as defined by expectation. [12] studied Markov process of stock market performance. In their result oil, India exhibits a higher chance of being stable with no significant increase or decrease. [9] considered Markov chain model on share price movement. Result showed GT Bank shares change hands more than the FB Bank. [2] examined the Markov chain model on forecasting of stocks prices in Nigeria. In their result Markov chain model was determined based on probability transition matrix. [4] Explored the stochastic analyses of Markov chain in finite states. Their work replicated the use of 3-states transition probability matrix which enables them to proffer precise condition of obtaining expected mean rate of return of each stock. [13] considered stochastic analysis of share prices. Results showed precise condition of determining expected mean return time for stock price; improving investment decision based on highest transition probabilities. In the same manner, [5] studied the behavior of stock market using Markov chain. The study reveals that regardless of bank's current share price steady state probabilities of share price remain the same all through the iteration. [8] Studied long run prospects of security prices in Nigeria where the data were collected from the randomly selected banks from the banking sector of Nigeria. The analysis suggested that the price level of Nigerian bank were likely to remain relatively stable in the long run irrespective of the current situations.[14] examined the long run behavior of the closing price of shares of eight Nigerian banks using Markov chain model. They computed limiting distribution transition probability matrix of the of share price and found that despite of the current situation in the market there is hope for Nigerian bank stocks. It was concluded that the results derive from the study will be useful to investors.

More so, because of unstable nature of stock price movements; which can be linked as stochastic processes. Therefore, the method of Markov chain was used to study the closing stock prices of Oando Nigeria plc for the period of 2017-2021. The stock price data replicated 3-states transition probability matrix solution for each independent year and the last three months of each year were used as row vector to determine the future stock price changes. From the analytical solution of stochastic analysis the mean rate of returns, growth rates and other price changes were considered. This paper extends the [5] by assessing the investment of Oando Nigeria independently for the period of (2017-2021), stating the future stock price changes and their investments growth rates of different years.

This paper is aimed at presenting transitions probability matrix solution of Oando Nigeria PLC for five independent states, to obtain the expected mean rate of returns, Growth rates, and the transition probability future changes of stock prices.

This paper is arranged: section 2 presents mathematical preliminaries, the problem formulation is seen in subsection 2.1, Results are pressed in section 3 and the paper is concluded in section 4.

2 Mathematical preliminaries

A stochastic process $X(t)$ is a relations of random variables $\{X_t(\gamma), t \in T, \gamma \in \Omega\}$, i.e, for each t in the index set T , $X(t)$ is a random variable. Now we understand t as time and call $X(t)$ the state of the procedure at time t . In view of the fact that a stochastic process is a relation of random variables, its requirement is similar to that for random vectors.

It can also be seen as a statistical event that evolves time in accordance to probabilistic laws. Mathematically, a stochastic process may be defined as a collection of random variables which are ordered in time and defines at a set of time points which may be continuous or discrete.

Definition 1: A stochastic process X is said to be a Markov chain if Markov property is satisfied:

$$P(X_{n+1} = j / X_0, X_1, \dots, X_n) = P(X_{n+1} = j / X_n) \quad (1.1)$$

For all $n \geq 0$ and $i, j \in S$ (state space) .

It is sufficient to know that the Markov property given (1.1) is equivalent to easy of the following for each $j \in S$.

$$P(X_{n+1} = j / X_{n_1}, X_{n_2}, \dots, X_{n_k}) = P(X_{n+1} = j / X_{n_k}) \quad (1.2)$$

(for any $n_1 < n_2 < \dots, n_k \leq n$) .

Assuming $X_n = i$ implies that the chain is in the *ith* state at the *nth* step.it can also be said that the chain' having the value *i*' or 'being in state *i*'. The idea behind the chain is described by its transition probabilities:

$$P(X_{n+1} = j / X_n = i) \quad (1.3)$$

They are dependent on i, j and n .

Definition 2: The chain X is said to be homogeneous if the following are stated below

$$P(X_{n+1} = j / X_n = i) = P(X_1 = j / X_0 = i) \quad (1.4)$$

for all n, i, j .

The transition matrix $P = (P_{ij})$ is $n \times n$ matrix of transition probabilities.

$$P_{ij} = P(X_{n+1} = j / X_n = i) \quad (1.5)$$

Hence, the transition probabilities with homogenous Markov chain are always stationary at a point.

Theorem 1: Suppose P is a stochastic matrix which implies the following:

i) P has non-negative entries or $P_{ij} \geq 0$

$$(ii) \sum_j P_{ij} = \sum_j P(X_{n+1} = j / X_n = i) = \sum_j P(X_1 = j / X_0 = i)$$

which is stationarity or point of convergence.

Proof: (i) each associated entry in P is a transition probability P_{ij} and being probability $P_{ij} \geq 0$.

$$(ii) \sum_j P_{ij} = \sum_j P(X_{n+1} = j / X_n = i) = \sum_j P(X_1 = j / X_0 = i)$$

which is stationarity.

$$P(X_i \in S / X_0 = i) = 1.$$

Theorem 2 : (Chapman-Kolmogorov Equations).

$$P_{ij(m+n)} = \sum_{r=i}^n P_{ir(m)} P_{rj(n)} \text{ Since } P_{m+n} = P_m P_n \text{ and so on } P_n = P^n \text{ the } nth \text{ power of } P.$$

Proof:

$$\begin{aligned} P_{ij(m+n)} &= P(X_{m+n} = j / X_0 = i) \\ &= \sum_r P(X_{m+n} = j, X_m = r / X_0 = i) \\ &= \sum_r P(X_{m+n} = j / X_m = i / X_0 = i) P(X_m = r / X_0 = i) \end{aligned}$$

Using the following in probability rule:

$$P(A \cap B / C) = P(A / B \cap C) P(B / C) \text{ and setting}$$

$$A = \{X_{m+n} = j\}, B = \{X_m = r\}, \text{ and } C = \{X_0 = i\}.$$

Using Markov property yields

$$\begin{aligned} P_{ij(m+n)} &= \sum_r P(X_{m+n} = j / X_m = r) P(X_m = r / X_0 = i) \\ &= \sum_r P_{rj(n)} P_{ir(m)} \\ &= \sum_r P_{ir(m)} P_{rj(n)} \end{aligned}$$

Hence $P_{m+n} = P_m P_n$ and so $P_n = P^n$, the power of P .

2.1 Problem formulation

let $S_i(t)$ ($i = 1, 2, \dots, N, t = 1, 2, \dots, n$) be the of daily closing stock prices in naira of five different selected years of Oando's i -th asset at time t be defined as three state Markov processes in finite states. Let $N \times n$ data matrix associated with $S_i(t)$ be X_{it} . We consider N closing prices over n months, time horizon. For each X_{it} we use the last three closing stock prices of each year (Oct.-Dec.) as row vector which measures the change in stock prices at time t , where $t = 0, 1, 2, \dots, N$ and t is measured in weekly intervals, $t \in \mathbb{N}$. Therefore, the vector is defined such that

$$\theta_{0it} = (\theta_{i01}, \theta_{i02}, \theta_{i03}, \dots, \theta_{i0n}) \quad (1.6)$$

To obtain an estimate of the transition probability as follows

$$P_{ij} = P(X_t = j / X_{t-1} = i), \text{ for } j = 0, 1, 2, 3, \dots, N$$

$$P_{ij} = \begin{cases} P & \text{if } j = 1 + i \\ q = 1 - P & \text{if } j = i - 1 \\ 0 & \text{otherwise} \end{cases}$$

where $k + 1$ is the number of states.

$$\left. \begin{aligned} n_{ij} &= \sum_{i=1}^n P_{ij} \text{ for } i, j = 0, 1, 2, 3 \\ \frac{n_{ij}}{n_i} &\text{ for } i, j = 0, 1, \dots, k \end{aligned} \right\} \quad (1.7)$$

However, for $k = 3$ is an estimate of the transition matrix.

$$\hat{P}_{ij}(\text{Oando})_{2017-2021} = \begin{pmatrix} \hat{P}_{00} & \hat{P}_{01} & \hat{P}_{03} \\ \hat{P}_{10} & \hat{P}_{11} & \hat{P}_{12} \\ \hat{P}_{20} & \hat{P}_{21} & \hat{P}_{22} \end{pmatrix} \quad (1.8)$$

Setting $i, j = 0, 1, 2$ for $k = 3$

$$\hat{P}_{ij}(\text{Oando:price changes}) = (B_{11} \quad B_{12} \quad B_{13}) \begin{pmatrix} \hat{P}_{00} & \hat{P}_{01} & \hat{P}_{02} \\ \hat{P}_{10} & \hat{P}_{11} & \hat{P}_{12} \\ \hat{P}_{20} & \hat{P}_{21} & \hat{P}_{23} \end{pmatrix} = (\phi_{11} \quad \phi_{12} \quad \phi_{13}) \quad (1.9)$$

Assuming X_t has state space and transition probability matrix of (1.9) becomes

$$\hat{P} = \begin{pmatrix} \beta_1 & \phi & \theta \\ 1-\phi & \beta_2 & 1-\alpha \\ 1-\theta & \alpha & \beta_3 \end{pmatrix}. \quad (1.10)$$

Also from where the transition matrices of independent years of Oando Nigeria, PLC is derived. Following the method of [5] The best three prices are chosen in terms of minimum value criterion

$$\left. \begin{aligned} \beta_1 &= \min(\alpha_1, \alpha_2, \dots, \alpha_n) \\ \beta_2 &= \min(\phi_1, \phi_2, \dots, \phi_n) \\ \beta_3 &= \min(\varphi_1, \varphi_2, \dots, \varphi_n) \end{aligned} \right\} \quad (1.11)$$

Thus each minimum value will be used as estimates, in order to determine the value of an economic asset such as stock; consideration should be made to identify some aspect of random variability which is the expected rate of returns and price of the underlying asset, [1]. Hence to determine the expected rate of returns for each stock for the period of trading days the following are defined as

$$\left\{ \frac{1-\phi}{\beta_1}, \frac{1-\theta}{\beta_2} \text{ and } \frac{1-\alpha}{\beta_3} \right\} \quad (1.12)$$

However, the Growth rates of Oando Nigeria PLC were determined by considering the maximum and minimum value of each closing stock prices of associative years denoted as $S_N(t)$, $S_M(t)$ respectively. Therefore, we define Growth-rates, GR_t as:

$$GR_t = \frac{S_M(t) - S_N(t)}{S_M(t)} \times 100\% \quad (1.13).$$

From (1.9) the behavior of stock market, each is categorized as no-change in price, increasing in price and price decrease in near future. Also the impact of percentage increase and decrease of (1.9) were determined.

3 Analysis and results

To demonstrate the closing stock market price performance of Oando investments in finite states using Markov chain model. The daily prices covers from 2017-2021 retrievable from Nigeria Stock Exchange (NSE). From Jan.-Sept. of each year were used to form transition probability matrix while from Oct.-Dec. of each year were used to form a row vectors all for both companies.

Therefore, the 3-Step transition probability matrix and row vector of Onando Nigeria, PLC-2017.

$$P_{ij} - 0_{\text{ando}}_{(2017)} = \begin{pmatrix} 0.3503 & 0.3107 & 0.3390 \\ 0.3398 & 0.3582 & 0.3021 \\ 0.3727 & 0.3239 & 0.3034 \end{pmatrix}, V_{3(2017)} = (6.01 \quad 5.98 \quad 5.94)$$

$$P_{ij} - 0_{\text{ando}}_{(2017-\text{Price Changes})} = (6.01 \quad 5.98 \quad 5.94) \begin{pmatrix} 0.3503 & 0.3107 & 0.3390 \\ 0.3398 & 0.3582 & 0.3021 \\ 0.3727 & 0.3239 & 0.3034 \end{pmatrix} = (4.5591 \quad 4.4144 \quad 4.5328)$$

The 3-Step transition probability matrix and the row vector Onando Nigeria, PLC-2018

$$P_{ij}(\text{Onando})_{(2018)} = \begin{pmatrix} 0.3562 & 0.2762 & 0.3676 \\ 0.2467 & 0.3979 & 0.3554 \\ 0.3576 & 0.3056 & 0.3368 \end{pmatrix}, V_{3(2018)} = (5.01 \quad 3.79 \quad 4.7)$$

$$P_{ij}(\text{Onando})_{(2018-\text{Price Changes})} = (5.01 \quad 3.79 \quad 4.7) \begin{pmatrix} 0.3562 & 0.2762 & 0.3676 \\ 0.2467 & 0.3979 & 0.3554 \\ 0.3576 & 0.3056 & 0.3368 \end{pmatrix} = (4.5591 \quad 4.4144 \quad 4.5328)$$

The 3-Step transition probability matrix and the row vector Onando Nigeria, PLC-2019

$$P_{ij}(\text{Onando})_{(2019)} = \begin{pmatrix} 0.3263 & 0.3338 & 0.3340 \\ 0.2645 & 0.3881 & 0.3474 \\ 0.3846 & 0.3293 & 0.2861 \end{pmatrix}, V_{3(2019)} = (5.99 \quad 5.99 \quad 5.99)$$

$$P_{ij}(\text{Onando})_{(2019-\text{Price Changes})} = (5.99 \quad 5.99 \quad 5.99) \begin{pmatrix} 0.3263 & 0.3338 & 0.3340 \\ 0.2645 & 0.3881 & 0.3474 \\ 0.3846 & 0.3293 & 0.2861 \end{pmatrix} = (5.9547 \quad 5.9900 \quad 5.9900)$$

The 3-Step transition probability matrix and the row vector Onando Nigeria, PLC-2020

$$P_{ij}(\text{Onando})_{(2020)} = \begin{pmatrix} 0.3333 & 0.3333 & 0.3333 \\ 0.4268 & 0.2972 & 0.2760 \\ 0.4923 & 0.3292 & 0.3138 \end{pmatrix}, V_{3(2020)} = (5.45 \quad 4.7 \quad 5.00)$$

$$P_{ij}(\text{Onando})_{(2020-\text{Price Changes})} = (5.45 \quad 4.7 \quad 5.00) \begin{pmatrix} 0.3333 & 0.3333 & 0.3333 \\ 0.4268 & 0.2972 & 0.2760 \\ 0.4923 & 0.3292 & 0.3138 \end{pmatrix} = (5.0495 \quad 5.1029 \quad 5.7993)$$

The 3-Step transition probability matrix and the row vector Onando Nigeria, PLC-2021

$$P_{ij}(\text{Onando})_{(2021)} = \begin{pmatrix} 0.2983 & 0.3616 & 0.3401 \\ 0.3768 & 0.3333 & 0.2899 \\ 0.3246 & 0.3421 & 0.3333 \end{pmatrix}, V_{3(2021)} = (3.31 \quad 3.75 \quad 4.00)$$

$$P_{ij}(\text{Onando})_{(2021-\text{Price Changes})} = (3.31 \quad 3.75 \quad 4.00) \begin{pmatrix} 0.2983 & 0.3616 & 0.3401 \\ 0.3768 & 0.3333 & 0.2899 \\ 0.3246 & 0.3421 & 0.3333 \end{pmatrix} = (3.7038 \quad 3.6567 \quad 3.6905)$$

Company	p_{00} β_1	p_{01} ϕ	p_{02} θ	p_{10} $1-\phi$	p_{11} β_2	p_{12} $1-\alpha$	p_{20} $1-\theta$	p_{21} α	p_{23} β_3
(2017)	0.3503	0.3107	0.3390	0.6893	0.3398	0.6418	0.661	0.3582	0.3034
(2018)	0.3562	0.2762	0.3676	0.7238	0.2467	0.6021	0.6324	0.3979	0.3368
(2019)	0.3263	0.3338	0.334	0.6662	0.2645	0.6119	0.666	0.3881	0.2861
(2020)	0.3333	0.3333	0.3333	0.6667	0.4268	0.7028	0.6667	0.2972	0.3138
(2021)	0.2983	0.3616	0.3401	0.6384	0.3768	0.6667	0.6599	0.3333	0.3333

Table 1: Transition probability matrix of stock market prices of different years

Following the method of [5] gives the following conditions:

$$\beta_1 = \text{Min}(0.3503, 0.3562, 0.3263, 0.3333, 0.2983) = 0.2983$$

$$\beta_2 = \text{Min}(0.33981, 0.2467, 0.2645, 0.4268, 0.3768) = 0.2467$$

$$\beta_3 = \text{Min}(0.30341, 0.3368, 0.2861, 0.3138, 0.3333) = 0.2861$$

Hence we have $\beta_1 = 0.2983$, $\beta_1 = 0.2467$ and $\beta_3 = 0.2861$

Table 2: Expected mean rate of return for each stock

Companies	$\frac{1-\phi}{\beta_1}$	$\frac{1-\theta}{\beta_2}$	$\frac{1-\alpha}{\beta_3}$	Mean
Onando ₍₂₀₁₇₎	2.3108	2.6794	2.2433	2.4112
Onando ₍₂₀₁₈₎	2.4264	2.5634	2.1045	2.3648
Onando ₍₂₀₁₉₎	2.2333	2.6996	2.1388	2.3572
Onando ₍₂₀₂₀₎	2.235	2.7025	2.4565	2.4047
Onando ₍₂₀₂₁₎	2.1401	2.6749	2.3303	2.3818

Table 3: The Growth-rates and summary of stock price changes of Oando Nigeria, PLC

Year	Maximum Stock price	Minimum Stock Price	Growth(%)	Summary of	Stock price	Changes
2017	18.97	5.94	69	5.9769	5.9787	5.9790
2018	7.5	3.14	58	4.5591	4.4144	4.5328
2019	8.48	4.8	43	5.9547	5.9900	5.9900
2020	10.05	4.7	54	5.0495	5.1029	5.7993
2021	6.06	3.751	38	3.7038	3.6507	3.6905

Table 4: Onando transition probability matrix of limiting distribution of various years

Onando ₍₂₀₁₇₎	0.3503	0.3107	0.339
Onando ₍₂₀₁₈₎	0.3562	0.2762	0.3676
Onando ₍₂₀₁₉₎	0.3263	0.3338	0.334
Onando ₍₂₀₂₀₎	0.3333	0.3333	0.3333
Onando ₍₂₀₂₁₎	0.2983	0.3616	0.3401

Table 5: Assessing percentage increase in future price changes in Oando Nigeria, PLC.

Year	% Changes	Future Prices	Variation of price	Changes
2017	0.05	5.9769	0.2988	0.2981
	0.10	5.9787	0.5977	0.5979
	0.15	5.9790	0.8965	0.8985
	0.20		1.1954	1.1958
	0.25		1.4942	1.4948
2018	0.05	4.5591	0.2280	0.2266
	0.10	4.4144	0.45591	0.4533
	0.15	4.5328	0.6839	0.6799

	0.20		0.9118	0.8829	0.9066
	0.25		1.1398	1.1036	1.1332
2019	0.05	5.9547	0.2977	0.2995	0.2995
	0.10	5.9900	0.5955	0.599	0.599
	0.15	5.9900	0.8932	0.8985	0.8985
	0.20		1.1909	1.198	1.198
	0.25		1.4887	1.4975	1.4975
2020	0.05	5.0495	0.2525	0.2551	0.2899
	0.10	5.1029	0.5050	0.5103	0.5799
	0.15	5.7993	0.7574	0.7654	0.8699
	0.20		1.0099	1.0206	1.1599
	0.25		1.2624	1.2757	1.4498
2021	0.05	3.7038	0.1852	0.1825	0.1845
	0.10	3.6507	0.3704	0.3651	0.3691
	0.15	3.6905	0.5556	0.5476	0.5536
	0.20		0.7408	0.7301	0.7381
	0.25		0.9260	0.9127	0.9226

Table 6: Assessing percentage decrease in future price changes in Oando Nigeria, PLC.

Year	% Changes	Future Prices	Variation	of price	Changes
2017	0.25	5.9769	1.4942	1.4947	1.4948
	0.20	5.9787	1.1954	1.1957	1.1958
	0.15	5.9790	0.8965	0.8968	0.8985
	0.10		0.5977	0.5977	0.5979
	0.05		0.2988	0.2989	0.2981
2018	0.25	4.5591	1.1398	1.1036	1.1332
	0.20	4.4144	0.9118	0.8829	0.9066
	0.15	4.5328	0.6839	0.6622	0.6799
	0.10		0.4560	0.4414	0.4533
	0.05		0.2280	0.2207	0.2266
2019	0.25	5.9547	1.4887	1.4975	1.4975
	0.20	5.9900	1.1909	1.198	1.198

	0.15	5.9900	0.8932	0.8985	0.8985
	0.10		0.5955	0.599	0.599
	0.05		0.2977	0.2995	0.2995
2020	0.25	5.0495	1.2624	1.2757	1.4498
	0.20	5.1029	1.0099	1.0206	1.1599
	0.15	5.7993	0.7574	0.7654	0.8699
	0.10		0.5050	0.5103	0.5799
	0.05		0.2525	0.2551	0.2899
2021	0.25	3.7038	0.9260	0.9127	0.9226
	0.20	3.6507	0.7408	0.7301	0.7381
	0.15	3.6905	0.5556	0.5476	0.5536
	0.10		0.3704	0.3651	0.3691
	0.05		0.1852	0.1825	0.1845

4 Discussion of results

It can be seen in Table 1 that the stock quantities were obtained row wise using the method of probability: $1-\phi$, $1-\theta$ and $1-\alpha$. Column 5,7 and 8 respectively. So, by addition of columns: 3 and 5,4 and 8,7 and 9 gives a total probability of 1. This agrees with one of axioms of probability measure which can be used to improve efficiency in term of stock trading. The entire entry stipulates price changes for short- and long-term business plans. It is clear in Tables 2 and 3, the mean rate of returns. Growth rates and future stock price changes are all in indexed in millions of naira all through the trading days which implies that Oando Nigeria, Plc is profit making company.

More so in order to predict the future price of expected returns of investments. Oando (2017): 23% chance of reducing the expected rate of return; 27% chance of increasing its price in future and 22% chance of no change in expected rate of return with mean rate of 24%. Oando (2018): 24% chance of reducing the expected rate of return; 25% chance of increasing its price in the near future and 21% chance of no change in expected rate of return with mean rate of 24%. Oando (2019): 22% chance of reducing the expected rate, 27% chance of increasing its price in the near future and 21% chance of no change in expected rate of return with mean rate of 24%. Oando (2020): 22% chance of reducing its expected rate, 27% chance of increasing its price in the near future and 25% chance of no change in expected rate of return with mean rate of 24%. Oando (2021): 21% chance of reducing the expected rate, 27% chance of increasing its price and 23% chance of no change in expected rate of return with mean rate 24%.

However, in order to predict the future price of entire of investments of different years, we therefore have the following, Oando (2017): Has 35% of reducing its price; 31% chance of increasing its price in the near future; 34% chance of no change in price.

Oando (2019): Has 33% chance of reducing its price; 34% chance of increasing its price and 33% chance of no change in price.

Oando(2020): Has 33% chance of reducing its price 33% chance of increasing its price and 33% Chance of no change in price .

Oando(2021): Has 30% chance of reducing its price ; 36% chance of increasing its price in the near future and 34% chance of no change in price.

The above assessment of Oando Nigeria plc (2017-2021) will provide an insight of these stochastic analysis that will enhancing their investment decision, having assessed the year independently.

Table 4, describes the probability changes of Oando investments which be specific at certain state of time; often converges to a limiting value which is independent of the initial state, hence this formation is a stochastic process.

In Table 5, the future price changes namely “no-change in price, the probability of price increasing in the near future and the probability of reducing in future” were varied by 5%-25% increase to assess the levels of this future price changes. It was discovered that the various levels of percentage changes dominantly indicate an increase over the trading period. This observation leads to good assessments on the part of Oando Nigeria PLC investments since they are been guided properly on the three levels of pricing effects.

However, the future price changes were also varied by 25%-5% decrease. it indicates percentage decrease in various future price changes over the stipulated trading period. In this scenario, it is also a guild to Oando investments over three levels of pricing effects assessment; the benefits of this assessment is to avert severe depletion of capital investments which may endanger profit making throughout the trading period, see Table 6.

6 Conclusion

An application of Markov chain is a mathematical tool for modeling stock price formation; since each finite state communicates for proper management policies. In this paper, stochastic analysis of Markov chain was applied in the closing stock price data of Oando Nigeria plc. The analytical solution of the stochastic analysis showed that the closing stock price of 2017 has the highest mean rate of returns and growth rates respectively :2 .4112 and 69%. Also, stock prices of 2018 have the best probability of price not changing in the near future: 26%. Thus, Oando Nigeria plc is profit making organization, since the growth rates are all indexed in millions of naira.

More so, comparing two or more companies and applying Markov chain in finite and infinite states with be an interesting study.

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A note on exponentiated generalized inverse Pareto distribution

A.A. Ogunde*, O.I. Oseghale†, and O. Fatoki‡

Abstract

The Exponentiated Generalized inverse Pareto (EGIP) distribution is derived using Exponentiated-G generator which introduces additional two new parameters into the inverse Pareto model. Some basic statistical properties of the model are derived which includes: moment and incomplete moment, moment generating function, quantile function, Renyi and q –entropy, mean time to failure, mean residual life, probability weighted moment and order statistics. The model parameter estimated using maximum likelihood estimation method is also presented.

Keywords: stress-strength reliability; quantile function; Renyi and ρ –entropy; mean residual life

1 Introduction

In many real life situations, the standard probability distributions do not provide adequate fit to some lifetime data sets, most especially with those with non-monotone failure. Thus, researchers developed many generators by introducing one or more shape parameters to generate new distributions. The new generated distributions are more flexible and tractable when compare to the classical/ baseline distributions. Some well-known generators can be found in [2]-[13], [15], [17]-[19], [25]-[26], and many others.

In this article the Exponentiated family of distribution is considered to develop a new model with greater flexibility. It has been already used by [1], [20], [21], [23], among many others. The cumulative distribution function (CDF) and probability density function (pdf) of the Exponentiated family of

*Corresponding Author. Department of Statistics, University of Ibadan, Ibadan, Nigeria; E-mail: debiz95@yahoo.com

†Department of Mathematics and Statistics, Joseph Ayo Babalola University, Ikeji Arakeji, Osun State, Nigeria; E-mail: innocentoseghale@gmail.com

‡ Department of statistics, Ogun State Institute of Technology, Igbesa, Ogun State, Nigeria; E-mail: fatoki_olayode@yahoo.com

distributions For a baseline continuous CDF $G(x; \xi)$, [11] defined the Exponentiated generalized (EG) class of distributions by

$$F(x; \xi) = [1 - (1 - G(x; \xi))^a]^b, \quad (1)$$

where $a > 0$ and $b > 0$ are two shape parameters whose role is to control skewness and kurtosis to enable the generation of distributions with heavier lighter tails. The PDF corresponding to (1) is given by

$$f(x; \xi) = abg(x; \xi)(1 - G(x; \xi))^{a-1}[1 - (1 - G(x; \xi))^a]^{b-1}, \quad (2)$$

where, $g(x; \xi)$ is the baseline pdf. Setting $a = 1$, the pdf (1) gives Lehmann Type I class. Also, for $b = 1$, the pdf (1) gives the Lehmann Type II family of distributions. So, the family (1) generalizes both Lehmann Types I and II family of distributions. It is essential to note that the baseline distribution $G(x; \xi)$ is a special case of (1) when $a = b = 1$.

2 Exponentiated generalized inverse Pareto distribution

A random variable X follows an inverse Pareto distribution if its CDF is given according to [23] is

$$F(z) = (\varphi x)^\lambda, \quad x > 0, \quad \lambda, \varphi > 0, \quad 0 < x < \frac{1}{\varphi}. \quad (3)$$

And the associated CDF to (3) is given by

$$f(x) = \lambda \varphi^\lambda x^{\lambda-1}, \quad x > 0, \quad \lambda, \varphi > 0, \quad 0 < x < \frac{1}{\varphi} \quad (4)$$

where, φ is scale and λ is shape parameter.

The CDF and PDF of the EGIP distribution is derived by putting eqn. (3) and eqn. (4) in eqn. (1) and eq. (2) and respectively, given as

$$F(x; a, b, \lambda, \varphi) = [1 - (1 - (\varphi x)^\lambda)^a]^b, \quad (5)$$

and

$$f(x; a, b, \lambda, \varphi) = ab\lambda\varphi^\lambda x^{\lambda-1}(1 - (\varphi x)^\lambda)^{a-1}[1 - (1 - (\varphi x)^\lambda)^a]^{b-1}, \quad (6)$$

where, φ is scale and a, b, λ is shape parameters.

2.1 Some special distributions of EGIP model

Here, we present special distributions to EGEP model as follows:

- For $b = 1$, the EGIP reduces to Generalized Exponentiated Inverse Pareto (GEIP) as a new model.
- For $a = 1$, the EGIP reduces to the Exponentiated Pareto (EP) distribution with
- For $a = 1$ and $\beta = 1$, the EGEP reduces to IP distribution.

2.2 Some basic properties of the exponentiated generalized inverse Pareto distribution

In this section some standard reliability properties of the EGIP have been derived. An expression for the reliability function, hazard, cumulative hazard, and the odd function of the EGIP distribution is respectively, given by

$$R(x; a, b, \lambda, \varphi) = 1 - [1 - (1 - (\varphi x)^\lambda)^a]^b, \quad (7)$$

$$h(x) = \frac{ab\lambda\varphi^\lambda x^{\lambda-1} (1 - (\varphi x)^\lambda)^{a-1} [1 - (1 - (\varphi x)^\lambda)^a]^{b-1}}{1 - [1 - (1 - (\varphi x)^\lambda)^a]^b} \quad (8)$$

$$r(x) = \frac{ab\lambda\varphi^\lambda x^{\lambda-1} (1 - (\varphi x)^\lambda)^{a-1} [1 - (1 - (\varphi x)^\lambda)^a]^{b-1}}{[1 - (1 - (\varphi x)^\lambda)^a]^b} \quad (9)$$

and

$$O(x) = \frac{[1 - (1 - (\varphi x)^\lambda)^a]^b}{1 - [1 - (1 - (\varphi x)^\lambda)^a]^b} \quad (10)$$

The graphs of the distribution function and hazard function of the *EGIP* are given in figure 1 and 2 with various values of the parameters of the distributions.

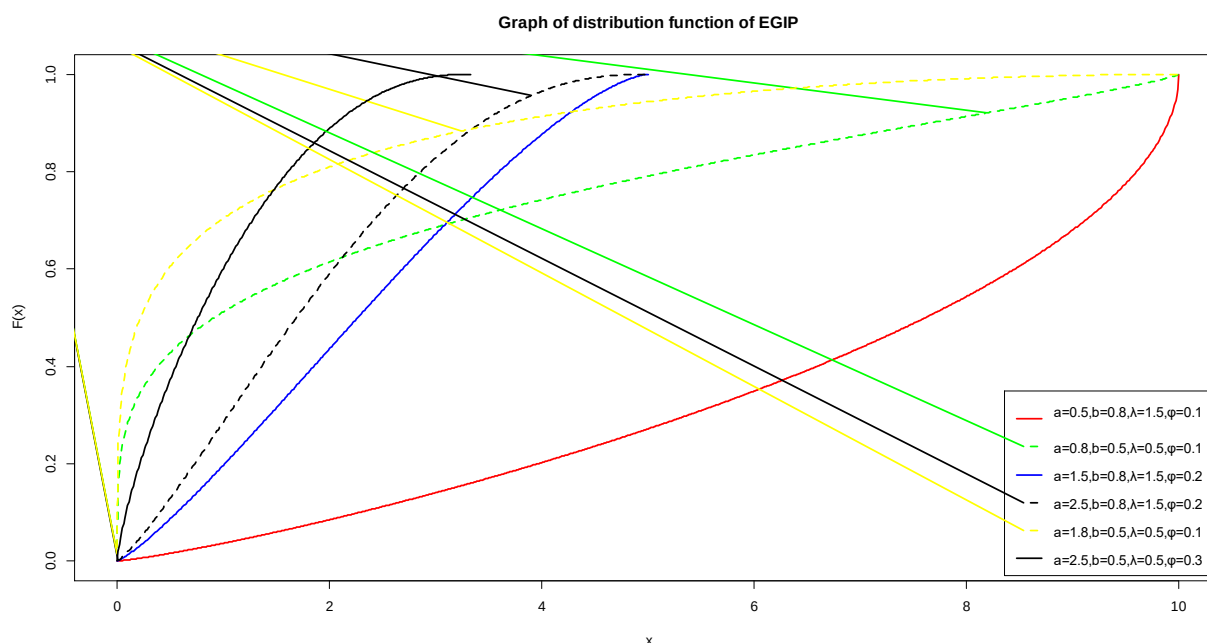


Figure 1. Graph of distribution function of EGIP distribution

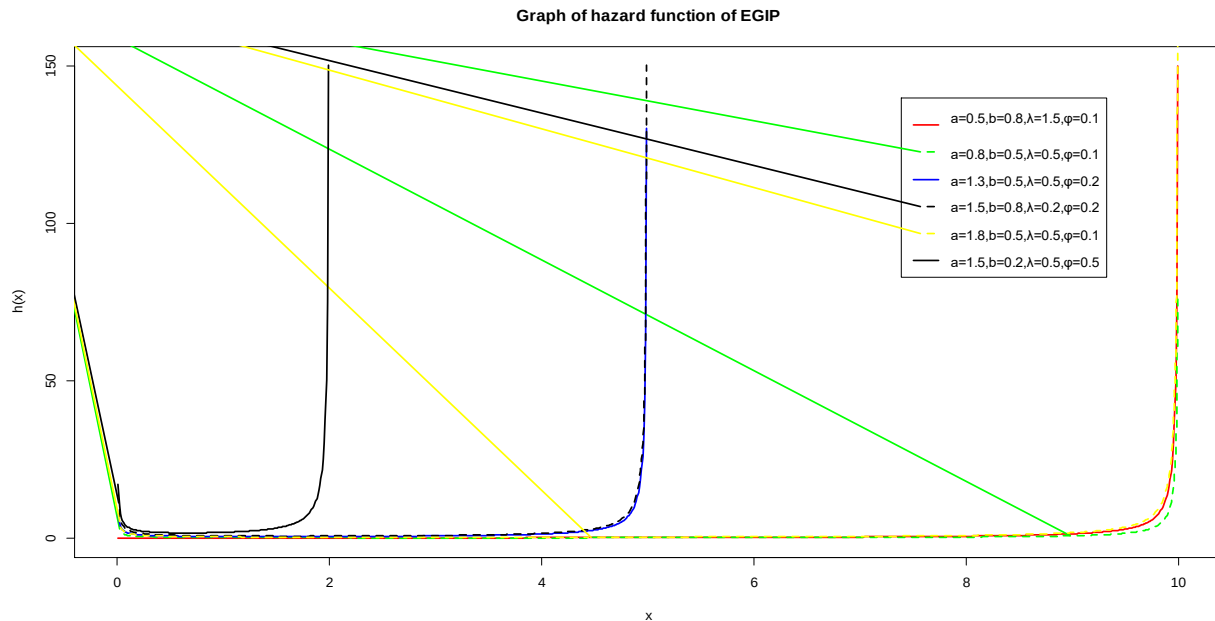


Figure 2. Graph of hazard function of EGIP distribution

2.3 Quantile function and median

In this section the quantile function, median (M) and mode are derived. Where u is Uniform (0,1), the quantile function of the EGIP distribution is obtain by inverting eqn.(5)

$$x_u = \frac{1}{\phi} \left[1 - \left(1 - u^{1/b} \right)^{1/a} \right]^{1/\lambda} \quad (11)$$

median is obtain by taking $u = 0.5$ in eqn. (11) presented as

$$M = \frac{1}{\phi} \left[1 - \left(1 - (0.5)^{1/b} \right)^{1/a} \right]^{1/\lambda} \quad (12)$$

The mode of $EGIP$ distribution can be obtained by solving the following non-linear equation $\frac{\partial}{\partial x} (\log[f(x; a, b, \lambda, \phi)]) = 0$, which is given as

$$\frac{\lambda(b-1)\phi^\lambda x^\lambda (1 - (\phi x)^\lambda)^{a-1}}{[1 - (1 - (\phi x)^\lambda)^a]} + \lambda = \frac{\lambda(a-1)\phi^\lambda x^\lambda}{[1 - (\phi x)^\lambda]} + 1$$

The quantile function can be used to obtain the Bowley's skewness (B) by [14] and Moors's kurtosis (M) by [16]. These measures are given by

$$B = \frac{Q_{0.75} + Q_{0.25} - 2Q_{0.5}}{Q_{0.75} - Q_{0.25}} \text{ and } M = \frac{Q_{0.375} - Q_{0.125} + Q_{0.875} - Q_{0.625}}{Q_{0.75} - Q_{0.25}}$$

3 Moments

The r^{th} moment of $X \sim EGIP(a, b, \lambda, \varphi)$ is found by

$$E(X^r) = \mu'_r = \int_{-\infty}^{\infty} x^r f(x) dx \quad (13)$$

Substitute from Equation (6) into Equation (13), we will obtain the r^{th} moment as

$$\mu'_r = ab\varphi^{-r} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{r}{\lambda} + 1, a(i+1)\right) \quad (14)$$

It should be noted the first moment represent the mean of *EGIP* distribution and is obtained by taking $r = 1$, the second and the third moment is obtained by taking $r=2$ and 3 and are respectively given as

$$\mu'_1 = ab\varphi^{-1} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{1}{\lambda} + 1, a(i+1)\right), \quad (15)$$

$$\mu'_2 = ab\varphi^{-2} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{2}{\lambda} + 1, a(i+1)\right), \quad (16)$$

and

$$\mu'_3 = ab\varphi^{-3} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{3}{\lambda} + 1, a(i+1)\right). \quad (17)$$

Thus the variance of *EGIP* distribution is given by

$$\sigma_X^2 = \frac{ab}{\varphi^2} Z^i B\left(\frac{2}{\lambda} + 1, a(i+1)\right) - \left(\frac{ab}{\varphi} Z^i B\left(\frac{1}{\lambda} + 1, a(i+1)\right)\right)^2 \quad (18)$$

where

$$Z^i = \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i}$$

3.2 The incomplete moment of EGIP distribution

The r^{th} incomplete moment of $X \sim EGIP(a, b, \lambda, \varphi)$ is obtained by

$$\zeta'_r = \int_0^\infty x^r f(x) dx \quad (19)$$

Substitute from Equation (6) into Equation (19), and we will obtain the incomplete moment as

$$\zeta'_r = ab\varphi^{-r} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{r}{\lambda} + 1, a(i+1); \frac{t^{1/\lambda}}{\varphi}\right) \quad (20)$$

3.3 The moment generating function of EGIP distribution

Let $X \sim EGIP(a, b, \lambda, \varphi)$, then the moment generating function of the *EGIP* can be can be obtain using

$$M_X(t) = E(e^{tX}) = \int_{-\infty}^\infty e^{tX} f(x) dx = \sum_{j=0}^\infty \frac{t^j}{j!} \int_{-\infty}^\infty x^j f(x) dx = \sum_{j=0}^\infty \frac{t^j}{j!} \mu'_j \quad (21)$$

Putting eqn. (14) into Eqn. (21), we obtain an expression for the moment generating function for EGIP distribution as

$$M_X(t) = ab\varphi^{-r} \sum_{i=0}^{b-1} \sum_{j=0}^\infty \frac{t^j}{j!} (-1)^i \binom{b-1}{i} B\left(\frac{r}{\lambda} + 1, a(i+1)\right) \quad (22)$$

3.4 Probability-weighted moment (PWM) of EGIP distribution

The PWM of X whose CDF $F(x)$, say $\psi^{v,r}$, is obtained by

$$\psi^{v,r} = E(X^v F^r(x)) = \int_0^\infty x^v F^r(x) f(x) dx. \quad (23)$$

If $X \sim EGIP(a, b, \lambda, \varphi)$, then the PWM $\psi^{v,r}$ is determined by

$$\psi^{v,r} = ab\varphi^{-r} \sum_{i=0}^{b-1} (-1)^i \binom{b(r+1)-1}{i} B\left(\frac{v}{\lambda} + 1, a(i+1)\right) \quad (24)$$

3.5 Mean residual life of EGIP distribution

If $X \sim EGIP(a, b, \lambda, \varphi)$, then the mean residual life is determined by

$$MRL(t) = E[X - t/X > t] = \frac{\int_t^\infty (x - t)f(x)dx}{R(t)} = \frac{\mu'_1 - \int_0^t xf(x)dx}{R(t)} - t \quad (25)$$

The integral $\int_0^t xf(x)dx$ is the incomplete moment of EGIP and μ'_1 is the first moment obtained by taking $r = 1$ in eqn. (20) and $r(t)$ is given in eqn.(7)

$$MRL(t) = \frac{\mu'_1 - ab\varphi^{-r} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{r}{\lambda} + 1, a(i+1); \frac{t^{1/\lambda}}{\varphi}\right)}{1 - [1 - (1 - (\varphi x)^\lambda)^a]^b} - t \quad (25)$$

3.6 Entropy function and ρ –entropy

The entropy function is a measure of uncertainty related to X whose PDF $f(x)$. It plays an important role in computer science, reliability, and others. The Renyi entropy [22] of X, say $I_\rho(X)$, is determined using

$$I_\rho(X) = \frac{1}{1-\rho} \log \int_0^\infty f^\rho(x)dx, \quad \rho \neq 1 \quad (26)$$

If $X \sim EGIP(a, b, \lambda, \varphi)$, then $I_\rho(X)$ is obtained by

$$I_\rho(X) = (ab)^\rho \varphi^{\rho - [\rho(\lambda-1)+1]} \lambda^{\rho-1} \sum_{i=0}^{b-1} (-1)^i \binom{\rho(b-1)}{i} B(P) \quad (27)$$

Where $P = \frac{(\lambda-1)(\rho-1)}{\lambda} + 1, a(i+\rho) - \rho + 1$

Consequently, the q –entropy of X, say $Z_q(X)$, can be obtained using

$$Z_q(X) = \frac{1}{1-\rho} \log(1 - [1 - \rho]I_\rho(X))$$

3.7 Mean time to failure

Assume $X \sim EGIP(a, b, \lambda, \varphi)$, then the mean time to failure of X, say MTTF, is obtained using

$$MTTF = \int_0^\infty xf(x)dx = \mu'_1 \quad (28)$$

Substitute from Equation (20), taking $r = 1$, to obtain

$$MTTF = b\varphi^{-1} \sum_{i=0}^{b-1} (-1)^i \binom{b-1}{i} B\left(\frac{1}{\lambda} + 1, a(i+1)\right) \quad (29)$$

4 Maximum likelihood estimates of the parameters

The maximum likelihood approach is considered in estimating the unknown parameters of the EGIP distribution. Let $\underline{x} = x_1, \dots, x_n$ represent a random sample obtained from the EGIP distribution. The likelihood function $L(\underline{x}; \zeta)$ and the log-likelihood function $\log L(\underline{x}; a, b, \lambda, \varphi) = l(\underline{x}; a, b, \lambda, \varphi)$ corresponding to (30) are respectively given as

$$L(\underline{x}, a, b, \lambda, \varphi) = \prod_{i=1}^n ab\lambda\varphi^\lambda x_i^{\lambda-1} (1 - (\varphi x_i)^\lambda)^{a-1} [1 - (1 - (\varphi x_i)^\lambda)^a]^{b-1} \quad (30)$$

and

$$\begin{aligned} l = & n\log(a) + n\log(b) + n\log(\lambda) + n\lambda\log(\varphi) + (\lambda - 1) \sum_{i=1}^n \log(x_i) \\ & + (a - 1) \sum_{i=1}^n \log(1 - (\varphi x_i)^\lambda) + (b - 1) \sum_{i=1}^n \log[1 - (1 - (\varphi x_i)^\lambda)^a] \end{aligned} \quad (31)$$

The element of the score vector $(\frac{\partial l}{\partial a}, \frac{\partial l}{\partial b}, \frac{\partial l}{\partial \lambda}, \frac{\partial l}{\partial \varphi})$ is given by

$$\frac{\partial l}{\partial a} = \frac{n}{a} + \sum_{i=1}^n \log(1 - (\varphi x_i)^\lambda) - (b - 1) \sum_{i=1}^n \left[\frac{(1 - (\varphi x_i)^\lambda)^a \log(1 - (\varphi x_i)^\lambda)}{[1 - (1 - (\varphi x_i)^\lambda)^a]} \right] \quad (32)$$

$$\frac{\partial l}{\partial b} = \frac{n}{b} + \sum_{i=1}^n \log[1 - (1 - (\varphi x_i)^\lambda)^a] \quad (33)$$

$$\frac{\partial l}{\partial \lambda} = \frac{n}{\lambda} + n\log(\varphi) + \sum_{i=1}^n \log(x_i) + a \sum_{i=1}^n \left[\frac{(\varphi x_i)^\lambda \log(\varphi x_i)}{[1 - (1 - (\varphi x_i)^\lambda)^a][1 - (\varphi x_i)^\lambda]} \right] \quad (34)$$

$$\frac{\partial l}{\partial \varphi} = \frac{n\lambda}{\varphi} + (b - 1)\lambda\varphi^{\lambda-1} \sum_{i=1}^n \left[\frac{x_i^\lambda}{(1 - (\varphi x_i)^\lambda)^a [1 - (\varphi x_i)^\lambda]} \right] - (a - 1)\lambda\varphi^{\lambda-1} \sum_{i=1}^n \frac{x_i^\lambda}{[1 - (\varphi x_i)^\lambda]} \quad (35)$$

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The approximate solution of advanced stochastic time-delay differential equations using block Adams Moulton methods and its application in claims settlement for policyholders satisfaction in the Nigerian insurance industry

C. Chibuisi¹ and B. O. Osu²

Abstract

In this paper, Block Adams Moulton Methods (BAMM) is implemented for the approximate solution of Advanced Stochastic Time-Delay Differential Equations (ASTDDEs) to determine the level of policyholders' satisfaction in settling their claims in Nigerian Insurance Industry. With the help of continuous development of multistep collocation method by matrix inversion techniques, the discrete schemes of the proposed method (BAMM) were derived. The analysis of basic properties of the method such as order and error constant, consistency, zero stability, convergence and region of absolute stability were carried-out and proved satisfactory. The numerical solutions of the method were obtained, computed and presented graphically by solving some advanced stochastic time-delay differential equations using the derived discrete schemes which revealed the effect of advanced stochastic time-delay on the policyholders' claims settlement. The accuracy of the method was examined by comparing the results obtained with other existing methods and was found to give better approximation.

Keywords: advanced stochastic time-delay differential equations; Block Adams Moulton methods; policyholder; claims settlement; insurance industry; Nigeria

1 Introduction

According to [3, 18, 14], claim is an official request made by a policyholder to an insurance industry demanding for a payment based on the terms of the insurance policy. [13, 7, 8], defined claims settlement as corporate policies and industry practices that insurance companies use to validate policyholder payment or reimbursement requests. It has been noticed many-a-times that the claim settlement process for policyholders in Nigerian Insurance Industries takes longer time than expected. This can happen for various reasons such as policyholders not submitting their claims on time, loss of insurance policy

¹ Department of Insurance, University of Jos, Jos, Nigeria; E-mail: chigoziec@unijos.edu.ng

²Corresponding Author. Department of Mathematics, Abia State University, Uturu, Nigeria; E-mail: osu.bright@abiastateuniversity.edu.ng

papers/documents, not following the right pre-authorization claims process for cashless medical treatments and advanced time-delay in exchange of information or documents between organizations, hospitals and insurance companies. In reality, the above stated reasons always result to advanced random/stochastic time-delays between the time the claims occur and the time of settlement of such claims. Advanced stochastic time-delay differential equation (ASTDDE) is a stochastic differential equation where the increment of the process depends not only on current state but also on the advanced/future part of the system being modeled which contains the random/stochastic noise term.

To reveal the applications of SDDEs in applied sciences, economics and engineering, authors such as [6, 19, 2, 17, 9, 4, 1] applied Euler-Maruyama scheme and interpolation techniques such as Hermite, Nordsieck, Newton divided difference and Neville's interpolation to construct continuous split-step schemes of SDDE on a continuous interval $t_0 \leq t \leq t_a$ and evaluation of the delay terms for the numerical solutions of SDDEs but encountered some obstacles as studied by [12]. In order to overcome the advanced random/stochastic time-delays experienced by the policyholders in settling their claims by the insurance companies and the obstacles encountered in the use of interpolation techniques for the evaluation of the delay terms, we applied Block Adams Moulton Methods (BAMM) as a linear multistep collocation method to discretize ASTDDEs on a discrete interval $[t_0, t_a)$ in order to obtain its discrete schemes for its numerical solution.

From [10], advanced stochastic time-delay differential equation (ASTDDE) can be express as

$$\begin{aligned} dy(t) &= \mu(y(t), y(t+\tau), t)dt + \varepsilon(y(t), y(t+\tau), t)d\beta(t) \quad \text{for } t > t_0, \tau > 0 \\ y(t) &= \phi(t), \text{ for } t \leq t_0 \end{aligned} \quad (1)$$

where $\phi(t)$ is the initial function, $y(t)$ is the stochastic process of the current state, t is the time, τ is called the delay, $(t+\tau)$ is the advanced/future time-delay term and $y(t+\tau)$ is the solution of the advanced time-delay term on the drift part of (1). $\beta(t)$ is the Standard Brownian Motion with its differential equivalence as $d\beta(t)$ as the volatility noise term or Wiener process together with solution of the advanced time-delay term as $y(t+\tau)ds(t)$ on the volatility or diffusion part of (1). The drift part of the equation (1) $dy(t) = \mu(y(t), y(t+\tau), t)dt$ is deterministic and takes care of the time the claims occur and the time such claims are settled without any risk involved. The volatility or diffusion part $dy(t) = \varepsilon(y(t), y(t+\tau), t)ds(t)$ is stochastic which takes care of the advanced random/stochastic time-delays between the time the claims occur and the time of settlement of such claims.

2 Derivation of the method

By matrix inversion techniques of the continuous construction of the k -step multistep collocation method formulated by [15], the discrete schemes of Block Adams Moulton Methods (BAMM) for step numbers $k = 2$ and 3 are obtained and presented as;

For $k = 2$ of BAMM

$$\begin{aligned} y_w &= y_{w+1} - \frac{5}{12}af_w - \frac{8}{12}af_{w+1} + \frac{1}{12}af_{w+2} \\ y_{w+2} &= y_{w+1} - \frac{1}{12}af_w + \frac{8}{12}af_{w+1} + \frac{5}{12}af_{w+2} \end{aligned} \quad (2)$$

For $k = 3$ of BAMM

$$y_w = y_{w+2} - \frac{1}{3}af_w - \frac{4}{3}af_{w+1} - \frac{1}{3}af_{w+2}$$

$$y_{w+1} = y_{w+2} + \frac{1}{24}af_w - \frac{13}{24}af_{w+1} - \frac{13}{24}af_{w+2} + \frac{1}{24}af_{w+3}$$

$$y_{w+3} = y_{w+2} + \frac{1}{24}af_w - \frac{5}{24}af_{w+1} + \frac{19}{24}af_{w+2} + \frac{9}{24}af_{w+3} \quad (3)$$

3 Analysis of basic properties of the method

Using the conditions proposed by [11] and [5], the order and error constant, consistency, zero stability, convergence and region of absolute stability of (2) and (3) were analyzed.

3.1 Order and error constant

According to [11], the Linear Multistep Method is said to be of order m if $C_0 = C_1 = 0, \dots, C_m = 0$ but $C_{m+1} \neq 0$ and C_{m+1} is called the error constant.

The order and error constants for (2) are obtained as follows

$$C_0 = C_1 = C_2 = C_3 = \begin{pmatrix} 0 & 0 \end{pmatrix}^T$$

$$C_4 = \begin{pmatrix} -\frac{1}{24} & -\frac{1}{24} \end{pmatrix}^T$$

Hence, (2) has an order $m = 3$ and error constant $\begin{pmatrix} -\frac{1}{24} & -\frac{1}{24} \end{pmatrix}^T$

Using the same techniques, (3) can be presented as follows

$$C_0 = C_1 = C_2 = C_3 = C_4 = \begin{pmatrix} 0 & 0 & 0 \end{pmatrix}^T$$

$$C_5 = \begin{pmatrix} \frac{1}{90} & -\frac{11}{720} & -\frac{19}{720} \end{pmatrix}^T.$$

Therefore, (3) has order $m = 4$ and error constant, $\begin{pmatrix} \frac{1}{90} & -\frac{11}{720} & -\frac{19}{720} \end{pmatrix}^T$.

3.2 Consistency

According to [11], a computational method is said to be consistent if the order e is greater than 1 i.e., $e \geq 1$. Since the order of our proposed method BAMM as analyzed for the discrete schemes (2) and (3) is greater

than 1 i.e. $m \geq 1$, hence, the method is consistent.

3.3 Zero stability analysis

In [5], a computational method is said to be zero stable if the roots $r_s, s=1, 2, 3, \dots, n$ of the first characteristic polynomial $\varphi(e)$ expressed as $\varphi(e) = \det(eZ_2^{(1)} - Z_1^{(1)})$ satisfies $|e_s| \leq 1$ and the roots $|e_s|$ is simple or distinct.

The zero stability for (2) is examined as follows

$$\begin{pmatrix} -1 & 0 \\ -1 & 1 \end{pmatrix} \begin{pmatrix} y_{w+1} \\ y_{w+2} \end{pmatrix} = \begin{pmatrix} 0 & 1 \\ 0 & 0 \end{pmatrix} \begin{pmatrix} y_{w-1} \\ y_w \end{pmatrix} + a \begin{pmatrix} -\frac{2}{3} & \frac{1}{12} \\ \frac{2}{3} & \frac{5}{12} \end{pmatrix} \begin{pmatrix} f_{w+1} \\ f_{w+2} \end{pmatrix} + a \begin{pmatrix} 0 & -\frac{5}{12} \\ 0 & -\frac{1}{12} \end{pmatrix} \begin{pmatrix} f_{w-1} \\ f_w \end{pmatrix},$$

where

$$Z_2^{(1)} = \begin{pmatrix} -1 & 0 \\ -1 & 1 \end{pmatrix}, Z_1^{(1)} = \begin{pmatrix} 0 & 1 \\ 0 & 0 \end{pmatrix}, V_2^{(1)} = \begin{pmatrix} -\frac{2}{3} & \frac{1}{12} \\ \frac{2}{3} & \frac{5}{12} \end{pmatrix} \text{ and } U_2^{(1)} = \begin{pmatrix} 0 & -\frac{5}{12} \\ 0 & -\frac{1}{12} \end{pmatrix}$$

$$\begin{aligned} \varphi(e) &= \det(eZ_2^{(1)} - Z_1^{(1)}) \\ &= |eZ_2^{(1)} - Z_1^{(1)}| = 0 \end{aligned} \tag{4}$$

Now we have,

$$\begin{aligned} \varphi(e) &= \left| e \begin{pmatrix} -1 & 0 \\ -1 & 1 \end{pmatrix} - \begin{pmatrix} 0 & 1 \\ 0 & 0 \end{pmatrix} \right| = \begin{vmatrix} -e & 0 \\ -e & e \end{vmatrix} - \begin{vmatrix} 0 & 1 \\ 0 & 0 \end{vmatrix} \\ \Rightarrow \varphi(e) &= \begin{vmatrix} -e & 1 \\ -e & e \end{vmatrix} \end{aligned}$$

Using Maple (18) software, we obtain

$$\begin{aligned} \varphi(e) &= e^2 + e \\ \Rightarrow e^2 + e &= 0 \\ \Rightarrow e_1 = 1, e_2 = 0. \end{aligned}$$

Since $|e_i| \leq 1, i = 1, 2$, the discrete schemes in (2) is zero stable.

Applying the same approach for (3), we have

$$\begin{pmatrix} 0 & -1 & 0 \\ 1 & -1 & 0 \\ 0 & -1 & 1 \end{pmatrix} \begin{pmatrix} y_{w+1} \\ y_{w+2} \\ y_{w+3} \end{pmatrix} = \begin{pmatrix} 0 & 0 & -1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} y_{w-2} \\ y_{w-1} \\ y_w \end{pmatrix}$$

$$+a \begin{pmatrix} -\frac{4}{3} & \frac{1}{3} & 0 \\ -\frac{13}{24} & -\frac{13}{24} & \frac{1}{24} \\ -\frac{5}{24} & \frac{19}{24} & \frac{3}{8} \end{pmatrix} \begin{pmatrix} f_{w+1} \\ f_{w+2} \\ f_{w+3} \end{pmatrix} + a \begin{pmatrix} 0 & 0 & -\frac{1}{3} \\ 0 & 0 & \frac{1}{24} \\ 0 & 0 & \frac{1}{24} \end{pmatrix} \begin{pmatrix} f_{w-2} \\ f_{w-1} \\ f_w \end{pmatrix}$$

where

$$Z_2^{(2)} = \begin{pmatrix} 0 & -1 & 0 \\ 1 & -1 & 0 \\ 0 & -1 & 1 \end{pmatrix}, Z_1^{(2)} = \begin{pmatrix} 0 & 0 & -1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, V_2^{(2)} = \begin{pmatrix} -\frac{4}{3} & \frac{1}{3} & 0 \\ -\frac{13}{24} & -\frac{13}{24} & \frac{1}{24} \\ -\frac{5}{24} & \frac{19}{24} & \frac{3}{8} \end{pmatrix} \text{ and } U_2^{(2)} = \begin{pmatrix} 0 & 0 & -\frac{1}{3} \\ 0 & 0 & \frac{1}{24} \\ 0 & 0 & \frac{1}{24} \end{pmatrix}$$

$$\begin{aligned} \varphi(e) &= \det(eZ_2^{(2)} - Z_1^{(2)}) \\ &= |eZ_2^{(2)} - Z_1^{(2)}| = 0 \end{aligned} \tag{5}$$

Now we have,

$$\begin{aligned} \varphi(e) &= \left| e \begin{pmatrix} 0 & -1 & 0 \\ 1 & -1 & 0 \\ 0 & -1 & 1 \end{pmatrix} - \begin{pmatrix} 0 & 0 & -1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \right| = \left| \begin{pmatrix} 0 & -e & 0 \\ e & -e & 0 \\ 0 & -e & e \end{pmatrix} - \begin{pmatrix} 0 & 0 & -1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \right| \\ \Rightarrow \varphi(e) &= \begin{vmatrix} 0 & -e & 1 \\ e & -e & 0 \\ 0 & -e & e \end{vmatrix} \end{aligned}$$

The following are obtained using Maple (18) software,

$$\begin{aligned} \varphi(e) &= e^3 - e^2 \\ \Rightarrow e^3 + e^2 &= 0 \\ \Rightarrow e_1 &= 1, e_2 = 0, e_3 = 0. \end{aligned}$$

Since $|e_i| \leq 1$, $i = 1, 2, 3$, the discrete schemes in (3) is zero stable.

3.4 Convergence

Theorem 1: The necessary and sufficient condition for a linear multistep method to be convergent as stated by [5] is that it must be consistent and zero stable. Since the discrete schemes (2) and (3) are both consistent and zero stable, therefore the method is convergent.

3.5 Region of absolute stability

The regions of absolute stability of the numerical methods for ASTDDEs are considered. We considered finding the P - and Q -stability by applying (2) and (3) to the ASTDDEs of this form

$$\begin{aligned} dy(t) &= \mu(y(t) + y(t+\tau))dt + \varepsilon(y(t) + y(t+\tau))d\beta(t) \quad \text{for } t > t_0, \tau > 0 \\ y(t) &= \phi(t), \text{ for } t \leq t_0 \end{aligned} \tag{6}$$

where $\phi(t)$ is the initial function, μ, ε are complex coefficients, $\tau = \eta a, \eta \in \mathbb{Z}^+$, a is the step size and $\eta = \frac{\tau}{a}$, η is a positive integer. Let $H_1 = a\mu$ and $H_2 = a\varepsilon$, then the P -and Q -stability of (2) and (3) for $\eta=1$ are investigated, plotted using Maple 18 and MATLAB and presented in figure 1 to 4 below.

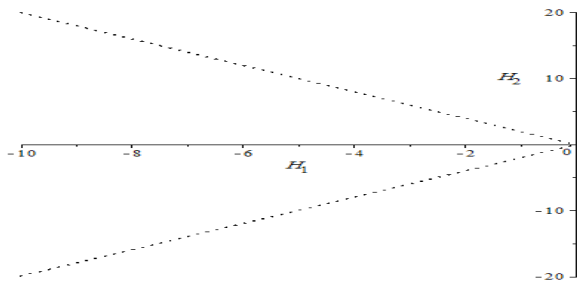


Figure 1. Region of P -stability (BAMM) in (2)

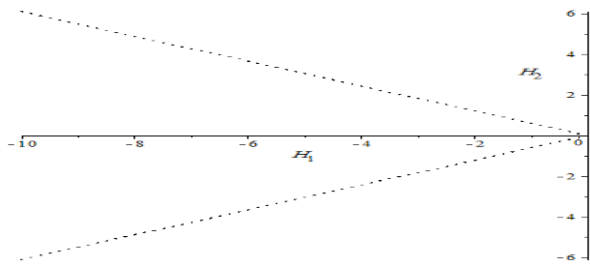


Figure 2. Region of P -stability (BAMM) in (3)

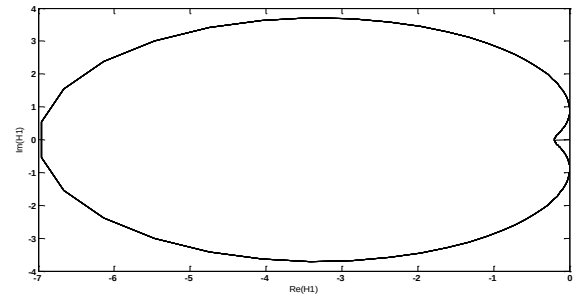


Figure.3. Region of Q -stability (BAMM) in (2)

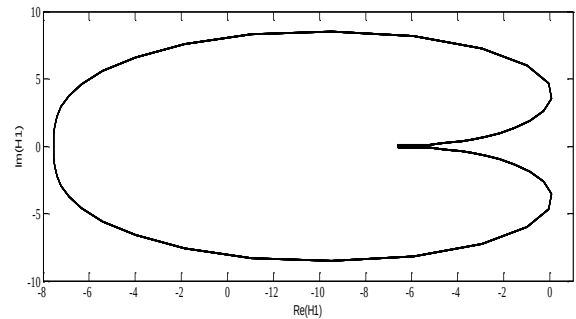


Figure 4. Region of Q -stability (BAMM) in (3)

The P -stability regions in Figs 1 to 2 lie inside the open-ended region while the Q -stability regions in Figs 3 to 4 lie inside the enclosed region.

4 Evaluations of Delay Terms

Here, we shall formulate two accurate and efficient mathematical expressions for the evaluation of the advanced time-delay terms on the drift and the volatility noise term on the diffusion part of the stochastic time-delay differential equations. The advanced time-delay term $(t + \tau)$ shall be evaluated with the accurate and efficient formula of this form

$$\delta_{w+j}(t) = \frac{w}{d} \left((dc + (w + j - \eta - 1)a) \right), d \neq 0 \quad (7)$$

Using normalized Brownian Motion, we formulated an expression to evaluate the volatility noise term $d\beta(t)$ such that the distribution is Gaussian with $N(0,1)$ whose mean μ is 0 and the standard deviation σ is 1. The random process is expressed as

$$\beta(t) = \frac{1}{\sqrt{((w+j-\eta-1)a)\pi}} e^{-\frac{t^2}{(w+j-\eta-1)a}}, \text{ for } t \geq 0 \quad (8)$$

Then by differentiating (8), it gives

$$d\beta(t) = \frac{-2t}{(w+j-\eta-1)a\sqrt{((w+j-\eta-1)a)\pi}} e^{-\frac{t^2}{(w+j-\eta-1)a}}, \text{ for } t \geq 0 \quad (9)$$

where $j \in (-k, k)$, k is a step number, $\eta = \frac{\tau}{a} \in Z$, $\tau = \eta a$; τ is the delay term, $w = 0, 1, 2, \dots, W-1$ and W is the number of solutions in the giving interval which is implemented to evaluate the volatility noise term $d\beta(t)$.

5 Numerical Computations

In this section, the evaluated advanced time-delay term and the volatility noise term using the two expressions (7) and (9) developed above shall be incorporated into some advanced stochastic time-delay differential equations before its evaluation with the discrete schemes (2) and (3) at constant step size $a = 0.01$ to obtain its computational solutions of $dy(t)$.

5.1 Numerical problems

Problem 1

$$dy(t) = \cos(t)((y(y(t)+2))dt + (y(y(t)+2))d\beta(t), \quad 0 \leq t \leq 3$$

$$\alpha(t) = 1, t \geq 0$$

Exact solution $\alpha(t) = 1 + \sin(t)$.

Problem 2

$$dy(t) = -(y(t-1+e^{-t}) + \sin(t-1+e^{-t}) + \cos(t))dt + (y(t-1+e^{-t}) + \sin(t-1+e^{-t}) + \cos(t))d\beta(t),$$

$$0 \leq t \leq 3$$

$$\alpha(t) = \sin(t), t \leq 0$$

Exact Solution $\alpha(t) = \sin(t)$.

6 Results, graphical presentations and discussions

The above problems were solved using the discrete schemes (2) and (3) derived by Block Adams Moulton Methods (BAMM) and the results of the absolute random errors obtained are presented in tables 1 to 2

Table 1: Absolute Random Errors of BAMM for $k = 2$ and 3 using Problem 1

t	K = 2 Absolute Random Error	K = 3 Absolute Random Error
1	0.845719238	0.845719236
2	0.917929971	0.917929971
3	0.154361373	0.154271898
4	0.73881897	0.738732318
5	0.935633477	0.935805002
6	0.250690119	0.251117461
7	0.692001936	0.691574594
8	1.030780292	1.03035295
9	0.460997752	0.459635207
10	0.487584536	0.488442522
11	0.934908259	0.936260796
12	0.462769879	0.464605564
13	0.503583055	0.501747369
14	1.083678941	1.081843256
15	0.75340265	0.751219726
16	0.17474429	0.177026758
17	0.838660557	0.840598537
18	0.618718851	0.620290407
19	0.290043944	0.288472389
20	1.060916364	1.059344809
21	0.989485244	0.990763859
22	0.148704384	0.14812801
23	0.68801341	0.686514681
24	0.746870479	0.743291542
25	0.022305383	0.02588432
26	0.913027428	0.916606365
27	1.098762672	1.106099222
28	0.405121759	0.411231154
29	0.539433381	0.532758559
30	0.873856852	0.866656324

CPU time of BAMM for $k = 2$ is 0.03s and $k = 3$ is 0.10s

Table 2: Absolute Random Errors of BAMM for $k = 2$ and 3 using Problem 2

t	K = 2 Absolute Random Error	K = 3 Absolute Random Error
1	0.837046383	0.836665921
2	0.895889047	0.895889047
3	0.123316526	0.119881874
4	0.783541932	0.782755906
5	0.989876475	0.99335545
6	0.319076026	0.321521156
7	0.613683492	0.610862708
8	0.93800642	0.93556129
9	0.358412665	0.351507123
10	0.604326121	0.606307176
11	1.060231835	1.067356323
12	0.600721595	0.608098621
13	0.360093698	0.352397372
14	0.931089965	0.92371294
15	0.600913805	0.585224284
16	0.329945098	0.339136313
17	0.98484421	1.000672496
18	0.75760374	0.77740328
19	0.127087532	0.146959007
20	0.861093034	0.880892574
21	0.742877411	0.77480406
22	0.145936521	0.119247721
23	1.037968136	1.007150612
24	1.15515455	1.117229542
25	0.447253474	0.409685871
26	0.377751821	0.415676829
27	0.500230288	0.540376758
28	0.261660995	0.217469289
29	1.267095814	1.228416796
30	1.666628252	1.678375518

CPU time of BAMMs for $k = 2$ is 0.07s and $k = 3$ is 0.04s

6.1 Graphical presentation of results

Using Microsoft Excel, the Absolute Random Error Results of BMM for Problem 1 and 2 in Tables 1 and 2 are presented as;

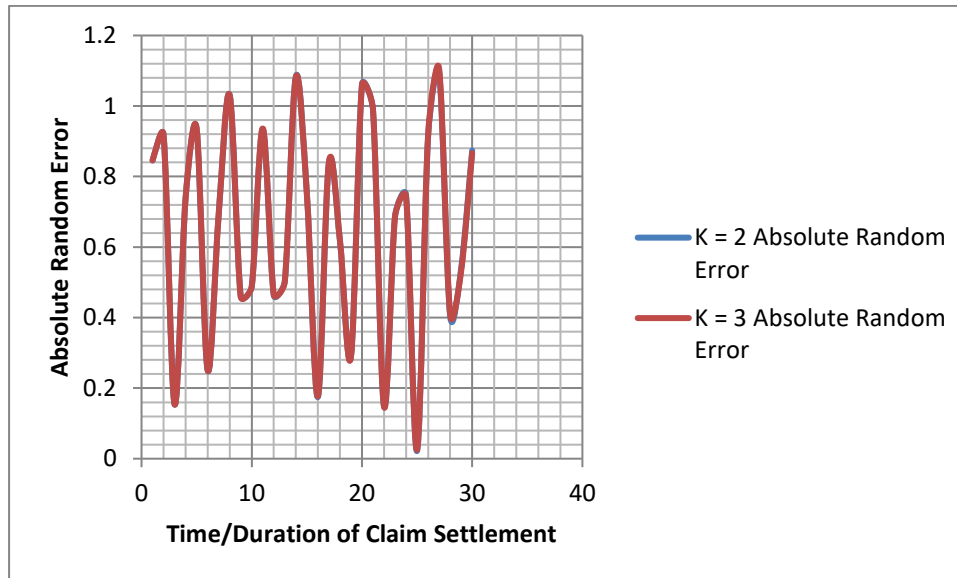


Figure 5. Advanced Stochastic Time-Delay DDEs Absolute Random Error Results for Problem 1

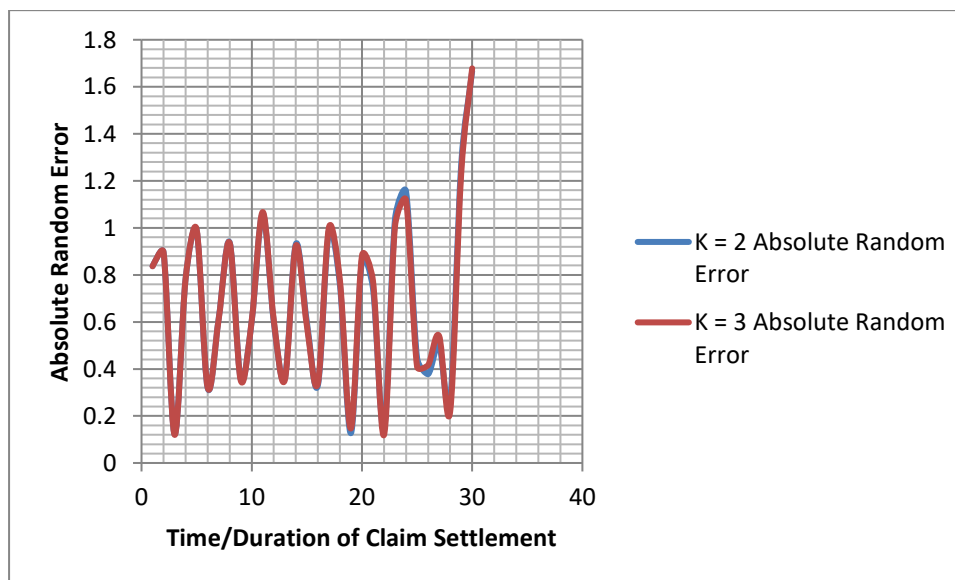


Figure 6. Advanced Stochastic Time-Delay DDEs Absolute Random Error Results for Problem 2

6.2 Comparison of results

In order to determine the accuracy, efficiency and advantage of our method BMM, we compared the absolute maximum errors of our method with other existing methods in [6, 2,16]

Table 3: Comparison between the Maximum Absolute Random Errors (MARE) of our method BMM for $k = 2$ and 3 with $[6, 2, 16]$ for constant step size $a = 0.01$ for Problem 1

Numerical Method	COMPARED MAREs with [6,2,16]
BMM MARE for $k = 2$	1.098762672
BMM MARE for $k = 3$	1.106099222
CSSEMM MARE for $k = 2$	4.76E-02
CSSEMM MARE for $k = 3$	9.17E-02
CSSEMM MARE for $k = 4$	1.62E-01
EMM MARE for $k = 2$	1.84E-02
EMM MARE for $k = 3$	4.04E-03
EMM MARE for $k = 4$	9.73E-04
BSM MARE for $k = 2$	7.04E-01
BSM MARE for $k = 3$	7.04E-01
BSM MARE for $k = 4$	7.04E-01

Table 4: Comparison between the Maximum Absolute Random Errors (MARE) of our method BMM for $k = 2$ and 3 with $[6, 2, 16]$ for constant step size $a = 0.01$ for Problem 2

Numerical Method	COMPARED MAREs with [6,2,16]
HEBMM MARE for $k = 2$	1.666628252
HEBMM MARE for $k = 3$	1.678375518
CSSEMM MARE for $k = 2$	3.18E-02
CSSEMM MARE for $k = 3$	5.90E-02
CSSEMM MARE for $k = 4$	1.37E-01
EMM MARE for $k = 2$	1.09E-01
EMM MARE for $k = 3$	4.91E-02
EMM MARE for $k = 4$	2.44E-02
BSM MARE for $k = 2$	6.96E-01
BSM MARE for $k = 3$	6.96E-01
BSM MARE for $k = 4$	6.96E-01

6.3 Graphical presentation for compared results

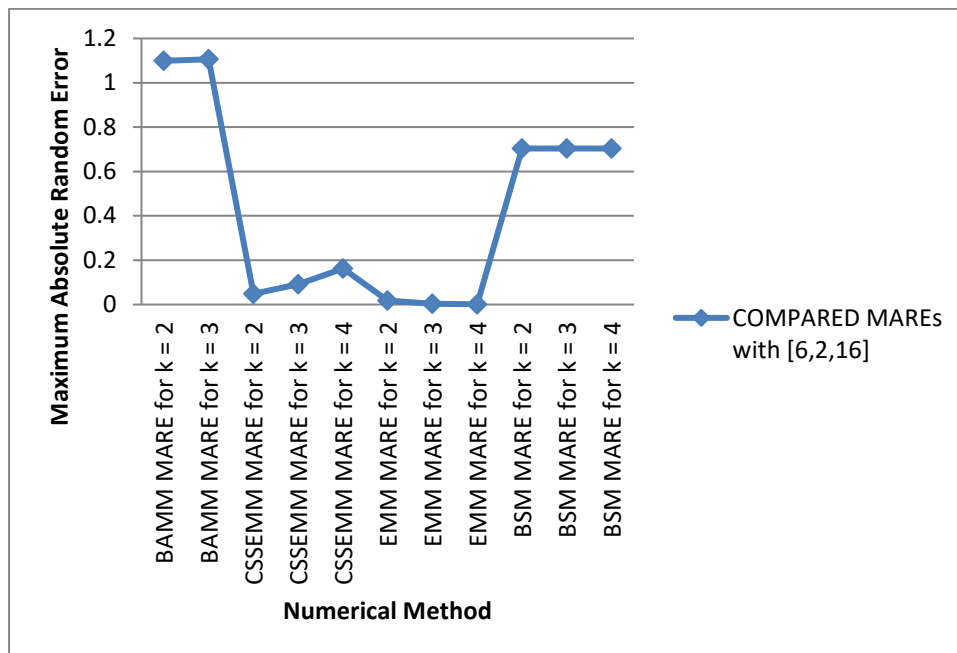


Figure 7. Compared MAREs of BMM with [6, 2, 16] for Problem 1

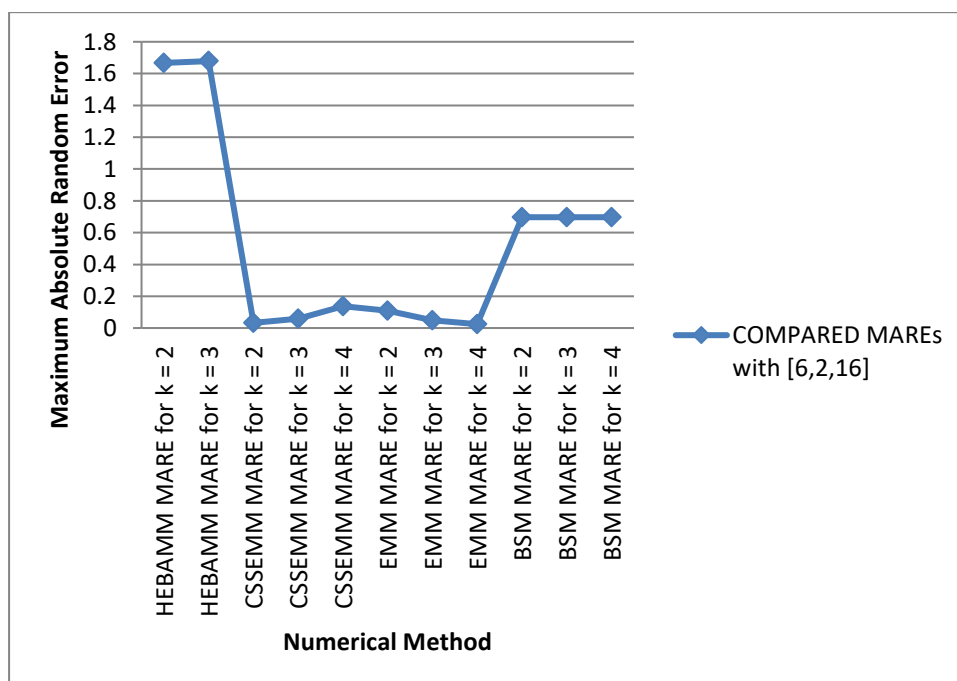


Figure 8. Compared MAREs of BMMs with [6, 2, 16] for Problem 2

7 Conclusion

In this study, we have demonstrated that Block Adams Moulton Methods (BAMM) is suitable for the approximate solution of Advanced Stochastic Time-Delay Differential Equations (ASTDDEs). As shown in tables 1 to 4 and figs. 5 to 8, the numerical results revealed the effect of advanced stochastic time-delay on the policyholders claims settlement by the insurance industry which can lead to policyholders' dissatisfaction and claims dispute between the insurance companies and their customers. The policyholders and the insurance companies have big roles to play for prompt claims settlements by policyholders submitting their claims on time, storing of policyholders data in the cloud to avoid loss of insurance policy papers/documents, following the right pre-authorization claims process for cashless medical treatments, quick response by the organizations in the submission of policyholders claims to the insurance companies and regular supervision and recapitalization of the insurance industries by the government. Also, it was observed in tables 1 to 4 that the discrete schemes of higher step number $k=3$ of BAMM performed slightly better and faster than the lower step number $k=2$ when compared with other existing methods. Further research should be carried-out for step numbers $k=4,5,6...$ on the approximate solutions of ASTDDEs using BAMMs.

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On the transitivity and primitivity of permutation groups of degree $4p$ constructed via wreath products using numerical approach

B. O. Johnson*, S. Hamma†, and M. S. Adamu†

Abstract

Let p be an odd prime number and G a finite permutation group of degree $4p$ generated via wreath products of pairs of permutation groups. In this work, a group concept, namely isomorphism, is applied to discuss the transitivity and primitivity of G using numerical approach. The computational group theory (GAP) is applied to enhance and validate our work.

Keywords: permutation group; primitivity; wreath products; p -groups; isomorphism

1 Introduction

Let C and D be two groups of permutations on non empty sets Ω_1 and Ω_2 , respectively, where $\Omega_1 \cap \Omega_2 = \emptyset$. Let $P = C^{\Omega_2} = \{f: \Omega_2 \rightarrow C\}$. The Wreath product of two permutation groups C and D denoted by $W = CwrD$ is the semi-direct product of P by D , so that, $W = \{(f, d) | f \in P, d \in D\}$, with multiplication in W defined as $(f_1, d_1)(f_2, d_2) = ((f_1, f_2 d_1^{-1}), (d_1, d_2)) \quad \forall f_1, f_2 \in P \text{ and } d_1, d_2 \in D$ is a special form of permutation group as in [11].

When a group G acts on a set Ω , a typical point α is moved by elements of G to various other points. The set of these images is called the orbit of α under G and we denote it by $\alpha^G := \{\alpha^g | g \in G\}$. A group G acting on a set Ω is said to be transitive on Ω if it has one orbit which is equal to the entire set, that is $\alpha^G = \Omega$ for all $\alpha \in \Omega$. Equivalently, G is transitive if for every pair of point $\alpha, \beta \in \Omega$ there exists $g \in G$ such that $\alpha^g = \beta$. If a group is not transitive then it is called intransitive.

A permutation group G acting on a non empty set Ω is called primitive if G acts transitively on Ω and G preserves no non trivial partition of Ω where non-trivial partition means a partition that is not a partition into singleton set or partition into one set Ω . In other words, a group G is said to be primitive on a set Ω if the only sets of imprimitivity are the trivial ones otherwise G is imprimitive on Ω as in [1]. Transitive and Primitive finite permutation groups can be thought of

* Corresponding Author. Department of Mathematics and Statistics, Federal University, Wukari, Taraba State, Nigeria; E-mail: benjohnsonnig@yahoo.com

† Department of Mathematical Science, Abubaka Tafawa Balewa University, Bauchi, Bauchi State, Nigeria.

as the building blocks of finite permutation groups, and questions about finite permutation groups can often be reduced to the primitive case according to [4].

Transitive and primitive permutation groups of special degrees have received much attention in the academic research space. [10] in a paper titled “Wreath Product of Permutation Groups” provided an upgrade to the results of an earlier paper on some finite permutation groups which consist of the basic procedure of computing Wreath product of groups. They also discussed the condition under which the Wreath products of permutation groups are faithful, transitive and primitive. Also some properties of the centre of a group stabilizer and Wreath product of groups were investigated respectively in the work. Transitive and primitive p -subgroup of dihedral groups of degree pq , where p, q are any two distinct odd prime numbers were considered by [7], while more recently, Audu and Hamma in [1] discussed the transitivity and primitivity of all the p -subgroups of dihedral groups of degree at most p^2 using the concept of p -groups. They used the standard program – The Groups, Algorithms and Programming (GAP) to validate their results while Hamma and Aliyu, in [8] worked “On transitive and primitive dihedral groups of degree *at most* 2^r ($r \geq 2$)”. Also, in [9] Hamma and Mohammed discussed the transitivity and primitivity of all the p -subgroups of dihedral groups of degree at most p^3 . They proved theorems and validate them using the Groups, Algorithms and Programming (GAP). Cai and Zhang, in [2] presented “A Note on Primitive Permutation Groups of Prime Power Degree”. Fengler, in his published work explored on “Transitive Permutation Groups of Prime Degree” as in [5].

In this paper, we obtained detailed description of the unique structure of Wreath product groups of degree $4p$ that are not p -groups and discussed their transitive and primitive nature using numerical approach. When the transitive and primitive nature of the Wreath products groups is well understood it facilitates comprehension of the classification of finite simple groups (CFSG) towards a productive review of same.

2 Materials and methods

2.1 p -group

A finite group G is said to be a p -group if its order is a power of p , where p is prime. A subgroup H of a group G is a p -subgroup if it (H) is a p -group.

2.2 Stabilizer

Any subset of G which fixes a specified element α is called the stabilizer of α in G and is denoted by $G_\alpha := \{x \in G \mid \alpha^x = \alpha\}$.

2.3 Orbit

When a group G acts on a set Ω , a typical point α is moved by elements of G to various other points. The set of these images is called the orbit of α under G , and we denote it by $\alpha^G := \{\alpha^x \mid x \in G\}$.

2.4 Dihedral group

Dihedral groups are groups of symmetries of a regular polygon, which consist of rotations and reflections. The group is denoted D_n which symbolizes the symmetry of a regular polygon with n sides (that is, the dihedral group of degree n). Conventionally, we write $D_n = \langle r, f \mid r^n = f^2 = 1, fr = r^{n-1}f = r^{-1}f \rangle$ and we say that D_n is the group generated by the elements r and f subject to the conditions

$r^n = f^2 = 1$; $fr = r^{n-1}f = r^{-1}f$, and the $2n$ distinct elements of D_n are $1, r, r^2, \dots, r^{n-1}, f, rf, r^2f, \dots, r^{n-1}f$.

Here r is a rotation about the centre of the polygon through angle $2\pi/n$ and f is a reflection about an axis of symmetry of the polygon.

2.5 Isomorphism

Two abstract groups $(G, \cdot)$ and $(H, *)$ are said to be isomorphic if there is a bijection $\varphi : G \rightarrow H$ such that $\varphi(xy) = \varphi(x)\varphi(y)$ for all $x, y \in G$. φ is called an Isomorphism.

Theorem 1

Any finite group G is isomorphic to a subgroup of the symmetric group S_n of degree n , where $n = |G|$.

Proof:

Let G act on itself by right multiplication $g^h = gh$ for all $g, h \in G$. If $g^h = g$ then $gh = g$ and so $h = 1$. That is, the kernel of the action is $\{1\}$. The mapping $f: G \rightarrow \text{sym}(G)$ define by $f: g \rightarrow f_g$ where $\alpha f_g = \alpha^g$ for any $\alpha \in G$ is a homomorphism. Then $G/\ker f \cong \text{im } f$. But $\ker f = \{1\}$ and $\text{im } f \leq \text{sym}(G) = S_n$. Accordingly $G \leq S_n$. In general we have that if G acts on Ω with k kernel of the action then $G/k \leq \text{sym}(\Omega)$.

2.7 Transitivity

Let G be a permutation group on Ω , where Ω is a finite set.

We say that G is $\frac{1}{2}$ -transitive if all the orbits have the same size.

Suppose that G has just one orbit Ω . then for all $\alpha \in \Omega$, $\alpha^G = \Omega$ and as such for any $\alpha, \beta \in \Omega$ there exists $g \in G$ such that $\alpha^g = \beta$, and G is said to be transitive on Ω .

The group G is said to be k -fold transitive (or, simply k -transitive) on Ω if, for any sequences $\alpha_1, \alpha_2, \dots, \alpha_k$ such that $\alpha_i \neq \alpha_j$ when $i \neq j$; $\beta_1, \beta_2, \dots, \beta_k$ such that $\beta_i \neq \beta_j$ when $i \neq j$ of k elements of Ω , there exists $g \in G$ such that

$$\alpha_i^g = \beta_i \text{ for } 1 \leq i \leq k.$$

Thus for $k = 2$ we have that for $\alpha_1, \alpha_2, \beta_1, \beta_2$ in Ω with $\alpha_1 \neq \alpha_2, \beta_1 \neq \beta_2$ there exists $g \in G$ such that;

$$\alpha_1^g = \beta_1, \alpha_2^g = \beta_2$$

and we say that G is doubly transitive.

If $k \geq 2$ then k -transitivity implies $(k-1)$ -transitivity.

Let G act on itself by right multiplication. Then, $\Omega = G$. If $\alpha = x, \beta = y$ in Ω and we take $g = x^{-1}y$; then $\alpha^g = x(x^{-1}y) = y = \beta$ and so G is transitive.

Let $H \leq G$ and let G act on right cosets of H in G .

Then G is transitive on $\Omega := (G : H)$. For if $\alpha, \beta \in \Omega$,

then $\alpha = Hx$, $\beta = Hy$ for some $x, y \in G$, and if we take $g := x^{-1}y$ then we have $\alpha^g = (Hx)x^{-1}y = Hy = \beta$.

Lemma 1

Let G be a dihedral group of any order, then G is transitive.

Proof:

For given α_i, α_j as any two vertices of the regular polygon with $i < j$, we readily see that $(\alpha_1\alpha_2 \dots \alpha_i \dots \alpha_j \dots \alpha_n)^{j-i}$ is the rotation about the centre of the polygon through angle $2\pi/n$, (where n is the number of edges of the polygon) which takes α to α_j . As such G is transitive.

2.9 Primitivity

A permutation group G acting on a non empty set Ω is said to be primitive on a set Ω if and only if it preserves the trivial block system otherwise G is imprimitive on Ω . For example, the group $S_3 = \{(1), (12), (13), (23), (123), (132)\}$ is primitive as $\{1,2\}^{(1\ 2\ 3)} = \{2,3\}$ implying that $\Delta^g \neq \Delta$ and $\Delta^g \cap \Delta \neq \emptyset$ for $\Delta = \{1,2\}$.

On the other hand, a subset Δ of Ω is said to be a set of imprimitivity for the action of G on Ω , if for each $g \in G$, either $\Delta^g = \Delta$ or Δ^g and Δ are disjoint. In particular, Ω itself, the 1-element subsets of Ω and the empty set are obviously sets of imprimitivity which are called trivial set of imprimitivity.

The group of symmetry $D_4 = \{(1), (1234), (13)(24), (1432), (13), (24), (12)(34), (14)(23)\}$ of the square with vertices 1,2,3,4 is imprimitive. For take $G_1 = \{(1), (24)\}$.

Let $H = \{(1), (13), (24), (13)(24)\}$ which is a normal subgroup of G . Then H is a group greater than G_1 , but not equal to G .

Theorem 2 [12]

Let G be a transitive permutation group of prime degree on the set Ω . Then G is primitive

Proof

First and foremost, G being transitive, permutes the sets of imprimitivity where all the sets Ω_i have the same order. But $\Omega = \cup |\Omega_i|$, Ω_i being the sets of imprimitivity. As $|\Omega|$ is prime we have that either each $|\Omega_i| = 1$ or Ω or the only sets of imprimitivity. So, G is primitive.

Theorem 3 [12]

Let G be a non-trivial transitive permutation group on Ω . Then G is primitive if and only if $G_\alpha, \alpha \in \Omega$ is a maximal subgroup of G or equivalently G is imprimitive if and only if there is a subgroup H of G properly lying between $G_\alpha (\alpha \in \Omega)$ and G .

Proof

Let the group G be imprimitive and Ψ be a non-trivial subset of the imprimitivity of G .

Let $H = \langle g \in G \mid \Psi^g = \Psi \rangle$

It is obvious H is a proper subgroup of G because $\Psi \subset \Omega$ and G is transitive.

Now choose $\alpha \in \Psi$. If $g \in G$ then $\alpha \in \Psi \cap \Psi^g$ and so $\Psi = \Psi^g$.

Hence $G_\alpha \leq H \leq G$

Since $|\Psi| \neq 1$, choose $\beta \in \Psi$ such that $\beta \neq \alpha$. By transitivity of G , there exists some $h \in G$ with $\alpha^h = \beta$ so that $h \in G_\alpha$. Now $\beta \in \Psi \cap \Psi^h$, so $\Psi = \Psi^h$ and $h \in H - G_\alpha$. Thus $H \neq G_\alpha$

Conversely, suppose that $G_\alpha < H < G$ for some subgroup H .

Let $\Psi = \alpha^H$. Since $H > G_\alpha$, $|\Psi| \neq 1$

Now if $\Psi = \Omega$, then H is transitive on Ω and hence $|\Omega| = |H : G_\alpha|$ showing that $H = G$, a contradiction.

Hence, $\Psi \neq \Omega$

Now we shall show that Ψ is a subset of imprimitivity of G .

Let $g \in G$ and $\beta \in \Psi \cap \Psi^g$ then $\beta = \alpha^h = \alpha^{h^g}$ for some $h, h \in H$.

Hence $\alpha^{h^g h^{-1}} = \alpha$. so $h^g h^{-1} \in G_\alpha < H$

This shows that $g \in H$

Thus $\Psi = \Psi^g$. Hence Ψ is a non-trivial subset of imprimitivity, so G is imprimitive.

Theorem 4 [11]

Let D act on P as $f^d(\delta) = f(\delta d^{-1})$ where $f \in P, d \in D$ and $\delta \in \Delta$.

Let $\mathcal{W}$ be group of all juxtaposed symbols $f d$, with $f \in P, d \in D$ and multiplication given by $(f_1, d_1)(f_2, d_2) = (f_1 f_2 d_1^{-1})(d_1, d_2)$. Then $\mathcal{W}$ is a group called the semi-direct product of P by D with the defined action

Remark 1 [11]

If C and D are finite groups, then the Wreath product $\mathcal{W}$ determined by an action of D on a finite group is of order $|\mathcal{W}| = |C|^{|\Delta|} |D|$

P is a normal subgroup of $\mathcal{W}$ and D is a subgroup of $\mathcal{W}$

The action of $\mathcal{W}$ on $\Gamma \times \Delta$ is given by

$(\alpha, \beta)fd = (\alpha f(\beta), \beta d)$ Where $\alpha \in \Gamma$ and $\beta \in \Delta$

3 Results

Proposition 1

Let G be the Wreath products of permutation groups of degree $4p$. Then G is transitive and imprimitive.

Proof

From the remark, the order of G , that is $|G| = 4p^4$ and $\Omega = \{1, 2, 3, \dots, 4p^4\}$.

That G is transitive follows easily from Lemma 1. Clearly, $|G| = 4p^4 = 2 \cdot 2p^4$ implies that the group G is isomorphic the dihedral group degree $2p^4$ (i.e. $G \cong D_{2p^4}$).

Now, name the vertices of D_{2p^4} as $1, 2, 3, \dots, 2p^4$ and let l be the line of symmetry joining the middle of the vertices 1 and $2p^4$ with the middle of the vertices $\frac{2p^4}{2}$ and $\frac{2p^4+4}{2}$ so that

$$\alpha = (1, 2p^4)(2, 2p^4 - 1)(3, 2p^4 - 2) \dots \left(\frac{2p^4}{2}, \frac{2p^4 + 4}{2}\right)$$

is the reflection in l .

Then $G_{(1)} = D2p^4_{(1)} = \left\{ (1), (2, 2p^4 - 1)(3, 4p^2p^4 - 2) \dots \left(\frac{2p^4}{2}, \frac{2p^4 + 4}{2}\right) \right\}$ is the stabilizer of the point 1 . We readily see that $G_{(1)}$ is a non-identity proper subgroup of G which has

$$H = \left\{ (1), (1, 4p), (2, 4p - 1), (3, 4p - 2), \dots, \left(\frac{2p^4}{2}, \frac{2p^4 + 4}{2}\right), \alpha \right\}$$

as a subgroup properly lying between $D2p^4_{(1)}$ and D_{2p^4} . That is, $D2p^4_{(1)} \leq H \leq D_{2p^4}$. By Theorem 4, D_{2p^4} is imprimitive. Thus, G is transitive and imprimitive.

3.1 Illustrating Example1 (Primitivity of Wreath Product Group of Degree $4p$ where $p = 3$)

Let C be a group of degree 4 and D a group of degree 3 acting on the set $\Omega = \{1, 2, 3, 4\}$ and $\Delta = \{5, 6, 7\}$ respectively. Let $P = C^\Delta = \{f: \Delta \rightarrow C\}$ with $|P| = |C|^{|\Delta|} = 4^3$. We show that the Wreath product $W = C_{wr}D$ of degree $= 12$ of order, $|W| = |C|^{|\Delta|} \times |D| = 4^3 \times 3$ is transitive and imprimitive.

Proof:

We follow the procedure as described in Theorem 4 and Remark 1 to obtain the elements of the Wreath products group, W in cyclic form as:

$$\begin{aligned} W = \{ & (1), (7, 8, 9, 10, 11, 12), (7, 9, 11)(8, 10, 12), (7, 10)(8, 11)(9, 12), (7, 11, 9)(8, 12, 10), \\ & (7, 12, 11, 10, 9, 8), (1, 2, 3, 4, 5, 6), (1, 2, 3, 4, 5, 6)(7, 8, 9, 10, 11, 12), (1, 2, 3, 4, 5, 6)(7, 9, 11)(8, 10, 12), \\ & (1, 2, 3, 4, 5, 6)(7, 10)(8, 11)(9, 12), (1, 2, 3, 4, 5, 6)(7, 11, 9)(8, 12, 10), (1, 2, 3, 4, 5, 6)(7, 12, 11, 10, 9, 8), (1, 3, 5) \\ & (2, 4, 6), (1, 3, 5)(2, 4, 6)(7, 8, 9, 10, 11, 12), (1, 3, 5)(2, 4, 6)(7, 9, 11)(8, 10, 12), (1, 3, 5)(2, 4, 6)(7, 10) \\ & (8, 11)(9, 12), (1, 3, 5)(2, 4, 6)(7, 11, 9)(8, 12, 10), (1, 3, 5)(2, 4, 6)(7, 12, 11, 10, 9, 8), (1, 4)(2, 5)(3, 6), (1, 4)(2, 5) \\ & (3, 6)(7, 8, 9, 10, 11, 12), (1, 4)(2, 5)(3, 6)(7, 9, 11)(8, 10, 12), (1, 4)(2, 5)(3, 6)(7, 10)(8, 11)(9, 12), \\ & (1, 4)(2, 5)(3, 6)(7, 11, 9)(8, 12, 10), (1, 4)(2, 5)(3, 6)(7, 12, 11, 10, 9, 8), (1, 5, 3)(2, 6, 4), \\ & (1, 5, 3)(2, 6, 4)(7, 8, 9, 10, 11, 12), (1, 5, 3)(2, 6, 4)(7, 9, 11)(8, 10, 12), (1, 5, 3)(2, 6, 4)(7, 10)(8, 11)(9, 12), \\ & (1, 5, 3)(2, 6, 4)(7, 11, 9)(8, 12, 10), (1, 5, 3)(2, 6, 4)(7, 12, 11, 10, 9, 8), (1, 6, 5, 4, 3, 2), \\ & (1, 6, 5, 4, 3, 2)(7, 8, 9, 10, 11, 12), (1, 6, 5, 4, 3, 2)(7, 9, 11)(8, 10, 12), (1, 6, 5, 4, 3, 2)(7, 10)(8, 11)(9, 12), \\ & (1, 6, 5, 4, 3, 2)(7, 11, 9)(8, 12, 10), (1, 6, 5, 4, 3, 2)(7, 12, 11, 10, 9, 8), (1, 7)(2, 8)(3, 9)(4, 10)(5, 11)(6, 12), \\ & (1, 7, 2, 8, 3, 9, 4, 10, 5, 11, 6, 12), (1, 7, 3, 9, 5, 11)(2, 8, 4, 10, 6, 12), (1, 7, 4, 10)(2, 8, 5, 11)(3, 9, 6, 12), \\ & (1, 7, 5, 11, 3, 9)(2, 8, 6, 12, 4, 10), (1, 7, 6, 12, 5, 11, 4, 10, 3, 9, 2, 8), (1, 8, 2, 9, 3, 10, 4, 11, 5, 12, 6, 7), \\ & (1, 8, 3, 10, 5, 12)(2, 9, 4, 11, 6, 7), (1, 8, 4, 11)(2, 9, 5, 12)(3, 10, 6, 7), (1, 8, 5, 12, 3, 10)(2, 9, 6, 7, 4, 11), \\ & (1, 8, 6, 7, 5, 12, 4, 11, 3, 10, 2, 9), (1, 8)(2, 9)(3, 10)(4, 11)(5, 12)(6, 7), (1, 9, 3, 11, 5, 7)(2, 10, 4, 12, 6, 8), \\ & (1, 9, 4, 12)(2, 10, 5, 7)(3, 11, 6, 8), (1, 9, 5, 7, 3, 11)(2, 10, 6, 8, 4, 12), (1, 9, 6, 8, 5, 7, 4, 12, 3, 11, 2, 10), (1, 9)(2, 10) \\ & (3, 11)(4, 12)(5, 7)(6, 8), (1, 9, 2, 10, 3, 11, 4, 12, 5, 7, 6, 8), (1, 10, 4, 7)(2, 11, 5, 8)(3, 12, 6, 9), \\ & (1, 10, 5, 8, 3, 12)(2, 11, 6, 9, 4, 7), (1, 10, 6, 9, 5, 8, 4, 7, 3, 12, 2, 11), (1, 10)(2, 11)(3, 12)(4, 7)(5, 8)(6, 9), \\ & (1, 10, 2, 11, 3, 12, 4, 7, 5, 8, 6, 9), (1, 10, 3, 12, 5, 8)(2, 11, 4, 7, 6, 9), (1, 11, 5, 9, 3, 7)(2, 12, 6, 10, 4, 8), \\ & (1, 11, 6, 10, 5, 9, 4, 8, 3, 7, 2, 12), (1, 11)(2, 12)(3, 7)(4, 8)(5, 9)(6, 10), (1, 11, 2, 12, 3, 7, 4, 8, 5, 9, 6, 10), \\ & (1, 11, 3, 7, 5, 9)(2, 12, 4, 8, 6, 10), (1, 11, 4, 8)(2, 12, 5, 9)(3, 7, 6, 10), (1, 12, 6, 11, 5, 10, 4, 9, 3, 8, 2, 7), (1, \\ & 12)(2, 7)(3, 8)(4, 9)(5, 10)(6, 11), (1, 12, 2, 7, 3, 8, 4, 9, 5, 10, 6, 11), (1, 12, 3, 8, 5, 10)(2, 7, 4, 9, 6, 11), (1, 12, 4, 9)(2, \\ & 7, 5, 10)(3, 8, 6, 11), (1, 12, 5, 10, 3, 8)(2, 7, 6, 11, 4, 9) \}. \end{aligned}$$

Now $|\mathcal{W}| = 196$ and $\Omega = \{1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12\}$ is the set of the points of $\mathcal{W}$. It follows from Lemma 1 that $\mathcal{W}$ is transitive as the orbit $\alpha^{\mathcal{W}} = \Omega \ \forall \alpha \in \Omega$.

Also the stabilizer of the point 1 in $\mathcal{W}$ is given by $\mathcal{W}_{\{1\}} = \{(1), (9, 10, 11, 12), (9, 11)(10, 12), (9, 12, 1, 10), (5, 6, 7, 8), (5, 6, 7, 8)(9, 10, 11, 12), (5, 6, 7, 8)(9, 11)(10, 12), (5, 6, 7, 8)(9, 12, 11, 10), (5, 7)(6, 8), (5, 7)(6, 8)(9, 10, 11, 12), (5, 7)(6, 8)(9, 11)(10, 12), (5, 7)(6, 8)(9, 12, 11, 10), (5, 8, 7, 6), (5, 8, 7, 6)(9, 10, 11, 12), (5, 8, 7, 6)(9, 11)(10, 12), (5, 8, 7, 6)(9, 12, 11, 10)\}$ which is obviously a non-identity proper subgroup of $\mathcal{W}$. We readily see from the subgroups of $\mathcal{W}$ that the group $(\mathcal{W})$ has a subgroup $H = \text{Syl}_2(\mathcal{W})$ properly lying between $\mathcal{W}_{\{1\}}$ and $\mathcal{W}$ that is, $\mathcal{W}_{\{1\}} < H < \mathcal{W}$ hence, $\mathcal{W}$ is imprimitive by Theorem 2. Thus, $\mathcal{W}$ is transitive and imprimitive.

3.2 Illustrating Example 2. Primitivity of Wreath Product Group of Degree $4p$ where $p = 5$

Let C be a group of degree 5 and D a group of degree 4 acting on the set $\Omega = \{1, 2, 3, 4, 5\}$ and $\Delta = \{6, 7, 8, 9\}$ respectively. Let $P = C^\Delta = \{f: \Delta \rightarrow C\}$ with $|P| = |C|^{|\Delta|} = 5^4$. Then the Wreath product $\mathcal{W} = C_{\text{wr}}D$ of degree = 20 and order, $|\mathcal{W}| = |C|^{|\Delta|} \times |D| = 2^5 5^4$ is transitive and imprimitive.

Proof:

We follow the procedure as described in Theorem 2 and Remark 1 to obtain the elements of the Wreath products group, $\mathcal{W}$ in cyclic form as:

$\mathcal{W} = \{(1), (11, 12, 13, 14, 15, 16, 17, 18, 19, 20), (11, 13, 15, 17, 19)(12, 14, 16, 18, 20), (11, 14, 17, 20, 13, 16, 19, 12, 15, 18), (11, 15, 19, 13, 17)(12, 16, 20, 14, 18), (11, 16)(12, 17)(13, 18)(14, 19)(15, 20), (11, 17, 13, 19, 15)(12, 18, 14, 20, 16), (11, 18, 15, 12, 19, 16, 13, 20, 17, 14), (11, 19, 17, 15, 13)(12, 20, 18, 16, 14), (11, 20, 19, 18, 17, 16, 15, 14, 13, 12), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 12, 13, 14, 15, 16, 17, 18, 19, 20), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 13, 15, 17, 19)(12, 14, 16, 18, 20), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 14, 17, 20, 13, 16, 19, 12, 15, 18), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 15, 19, 13, 17)(12, 16, 20, 14, 18), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 16)(12, 17)(13, 18)(14, 19)(15, 20), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 17, 13, 19, 15)(12, 18, 14, 20, 16), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 18, 15, 12, 19, 16, 13, 20, 17, 14), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 19, 17, 15, 13)(12, 20, 18, 16, 14), \dots\}.$

Now $|\mathcal{W}| = 2500$ and $\Omega = \{1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20\}$ is the set of the points of $\mathcal{W}$. It follows from Lemma 2.8 that $\mathcal{W}$ is transitive as the orbit $\alpha^{\mathcal{W}} = \Omega \ \forall \alpha \in \Omega$.

Also the stabilizer of the point 1 in $\mathcal{W}$ is $\mathcal{W}_{\{1\}} = \{(1), (11, 12, 13, 14, 15, 16, 17, 18, 19, 20), (11, 13, 15, 17, 19)(12, 14, 16, 18, 20), (11, 14, 17, 20, 13, 16, 19, 12, 15, 18), (11, 15, 19, 13, 17)(12, 16, 20, 14, 18), (11, 16)(12, 17)(13, 18)(14, 19)(15, 20), (11, 17, 13, 19, 15)(12, 18, 14, 20, 16), (11, 18, 15, 12, 19, 16, 13, 20, 17, 14), (11, 19, 17, 15, 13)(12, 20, 18, 16, 14), (11, 20, 19, 18, 17, 16, 15, 14, 13, 12)\}$, which is obviously a non-identity proper subgroup of $\mathcal{W}$. We readily see from the subgroups $\mathcal{W}$ that the group $(\mathcal{W})$ has a subgroup $H = \{(1), (1, 19, 2, 20, 3, 11, 4, 12, 5, 13, 6, 14, 7, 15, 8, 16, 9, 17, 10, 18), (1, 2, 3, 4, 5, 6, 7, 8, 9, 10)(11, 12, 13, 14, 15, 16, 17, 18, 19, 20), (1, 3, 5, 7, 9)(2, 4, 6, 8, 10)(11, 13, 15, 17, 19)(12, 14, 16, 18, 20), (1, 2, 3, 4, 5, 6)(7, 8, 9, 10, 11, 12)\}$

properly lying between $W_{\{1\}}$ and W that is, $W_{\{1\}} < H < W$ clearly, W is imprimitive by theorem 2.11. Thus, W is transitive and imprimitive.

3.3 GAP Result for Wreath Product Group of Degree $4p$, $p = \{3, 5, 7, 11, \dots\}$

We validate our result in this Section by applying a standard programme - the groups, algorithms and programming version 4.11.1 as shown below.

GAP

GAP 4.11.1 of 2021-03-02
<https://www.gap-system.org>
Architecture: x86_64-pc-cygwin-default64-kv7

Configuration: gmp 6.2.0, GASMAN, readline

Loading the library and packages ...

gap>

gap># Wreath Product (of permutation groups C and D) of Degree $4p$ for $p = 3$

gap> C := Group((1,2,3,4));

Group([(1,2,3,4)])

gap> D := Group((5,6,7));

Group([(5,6,7)])

gap> W := WreathProduct(C,D);

Group([(1,2,3,4), (5,6,7,8), (9,10,11,12), (1,5,9)(2,6,10)(3,7,11)(4,8,12)])

gap> Order(W);

192

gap> Elements(W);;

gap> IsTransitive(W);

true

gap> IsPrimitive(W);

false

gap>

gap>

gap># Wreath Product (of permutation groups C and D) of Degree $4p$ for $p = 5$

gap> C := Group((1,2,3,4,5));

Group([(1,2,3,4,5)])

gap> D := Group((6,7,8,9));

Group([(6,7,8,9)])

gap> W := WreathProduct(C,D);

<permutation group of size 2500 with 5 generators>

gap> Order(W);

2500

gap> Elements(W);;

gap> IsTransitive(W);

true

gap> IsPrimitive(W);

false

gap>

gap>

gap># Wreath Product (of permutation groups C and D) of Degree $4p$ for $p = 7$

gap> C := Group((1,2,3,4,5,6,7));


```
Group([ (1,2,3,4,5,6,7) ])
gap> D := Group((8,9,10,11));
Group([ (8,9,10,11) ])
gap> W := WreathProduct(C,D);
<permutation group of size 9604 with 5 generators>
gap> Order(W);
9604
gap> Elements(W);
gap> IsTransitive(W);
true
gap> IsPrimitive(W);
false
gap>
gap>
gap># Wreath Product (of permutation groups C and D) of Degree 4p for p = 11
gap> C := Group((1,2,3,4,5,6,7,8,9,10,11));
Group([ (1,2,3,4,5,6,7,8,9,10,11) ])
gap> D := Group((12,13,14,15));
Group([ (12,13,14,15) ])
gap> W := WreathProduct(C,D);
<permutation group of size 58564 with 5 generators>
gap> Order(W);
58564
gap> Elements(W);
gap> IsTransitive(W);
true
gap> IsPrimitive(W);
false
gap>
gap>
gap># Wreath Product (of permutation groups C and D) of Degree 4p for p = 13
gap> C := Group((1,2,3,4,5,6,7,8,9,10,11,12,13));
Group([ (1,2,3,4,5,6,7,8,9,10,11,12,13) ])
gap> D := Group((14,15,16,17));
Group([ (14,15,16,17) ])

gap> W := WreathProduct(C,D);
<permutation group of size 114244 with 5 generators>
gap> Order(W);
114244
gap> Elements(W);
gap> IsTransitive(W);
true
gap> IsPrimitive(W);
false
gap>
```

4 Conclusion and Recommendation

4.1 Conclusion

This study showed that wreath products group of degree $4p$ where p is an odd prime number is imprimitive.

4.2 Recommendation

This study can be extended by considering for further research, one or a combination of two or more of other theoretic properties such as simplicity, nilpotency, regularity, etc of same algebraic structures.

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Effects of PrEP on the HIV/AIDS dynamics with of immigration of infectives

J.K. Oladejo^{*†} and T.O. Oluyo^{*‡}

Abstract

In this work a six compartmental model is formulated to study the effect of PrEP and the direct inflow of HIV infective immigrants on the spread of HIV/AIDS. The positivity and the equilibrium points were analyzed; the Basic Reproduction number R_0 was generated, the local and global stability of the Disease-Free Equilibrium of the model was established and there exists a feasible region where the model is well posed, a unique Disease-Free Equilibrium point exist in the absence of HIV infective immigrants which is locally and globally asymptotically stable if $R_0 < 1$. Sensitivity index of key parameters revealed that c (number of sexual partners) makes the disease more endemic. Numerical simulation suggests that PrEP reduces the number of HIV infection despite the presence of constant inflow of infective immigrants and rigorous adherence to PrEP and treatment reduces the transmission of HIV/AIDS infection among high-risk population and can slow down the spread of infection.

1 Introduction

Human Immunodeficiency Virus (HIV) infection are spectrum of conditions caused by infection with the HIV. The late symptoms of infection are referred to as Acquired Immune Deficiency Syndrome (AIDS). The emergence of Antiretroviral (ARV), which became available in 1996 made many HIV-positive persons on treatment to have substantially lowered RNA level (viral load) enabling them to live long and healthy lives, through strict adherence to treatment regimens. Evidence does suggest that a low viral load may reduce infectiousness of HIV-positive persons receiving antiretroviral therapy (ART). Globally, 75% of people living with HIV were receiving ART as at December 2021 and 68% were virally suppressed in 2021. [11, 31, 34, 35]

^{*}Department of Pure and Applied Mathematics, Ladoke Akintola University of Technology, PMB 4000, Ogbomoso, Nigeria

[†]Corresponding Author. E-mail: olorundarekike@gmail.com

[‡] E-mail: tooluyo@lautech.edu.ng

Consequently, the incidences of HIV related death have decreased considerably globally by 52% between 2010 and 2021, 650 000 death from HIV-related causes was reported and 1.5million incidences of new infections in 2021.[31] Despite the improvement in the area of treatment of HIV and the declining overall number of new HIV infections globally, across Sub-Sahara Africa the death rate are much higher and has had a major impact on the life expectancy.[14] Majorly, adolescent girls and young women (53%) are being infected at twice the rate of boys and men of same age. It is a home to two third (67%) of all people living with HIV [21].

Moreover, the link between infectious diseases and population mobility must be understood in relation to the different forms, conditions and patterns of migration, which have different influences on the distribution and spread of infectious diseases. For example, in low and middle-income countries, economic migration has played a crucial involvement in the evolution of the HIV/AIDS epidemic [10]. However, research shows that internal and cross-border migrants, particularly male migrant workers are at greater risk of HIV infection and are more likely to spread the disease when they return home. It is well known that the immigrant can either be a susceptible or infective and disease is always persistent with direct immigration of infective individual in the population. [4, 8, 12, 15, 17, 24].

In other to reduce HIV transmission and save lives of people living with HIV and their uninfected sexual partners, WHO recommended that Pre-Exposure Prophylaxis (PrEP) be offered as a prevention choice for people at substantial risk of the HIV infection as part of a combination of prevention approach. PrEP is the use of ARV by HIV negative people at substantial risk to block the acquisition of HIV infection. High quality evidences strongly supports that PrEP is effective with more than 10 randomized controlled studies among range of populations (key population groups) and their sexual partners including serodiscordant heterosexual couples where one partner is infected and other is not, high risk heterosexual couples.[33] Key population groups are people in the population who are at high risk of HIV in all countries and region, they include: men who have sex with men; people who inject drugs; people in prisons and other closed setting; sex workers and their clients; transgender people.[29, 33]. In order to reach the Fast-Track strategy to end the AIDS epidemic by 2030 globally, intervention services need to focus on key population groups and their sexual partners as they account for over 70% of HIV infections globally. In other to avoid the worst-case scenario of half a million-excess death in Sub-Sahara Africa efforts needs to be redoubled, especially among women and girls as they accounted for 50% of new infections in 2020 [31].

Currently, PrEP cannot be accessed through public programs in most countries of Sub-Sahara Africa due to slow public health response. Ideally PrEP should be offered in combination with other preventive methods especially condoms as it prevents other sexually transmitted infections (STI). South Africa became the first African country to approve the use of PrEP followed by Kenya, [28] according to PrEP watch the estimated number of people under the PrEP strategy in some African countries in 2020 is given in the table below.

Table 1: Total number of People using PrEP in some African countries at 2020

African Countries	Estimated number of People
South Africa	530 378
Uganda	269 840
Zimbabwe	89 946
Kenya	256 321
Nigeria	276 671
Zambia	273 779
Togo	100
Senegal	477
Rwanda	17 884

Ref: (<http://www.prepwatch.org/>)

However, in Nigeria about 1.9 million people are living with HIV and one of the countries with the largest HIV epidemic in the world [30]. Currently PrEP is not widely available in Nigeria [23]. There is the need for Nigeria to adopt the WHO recommendation on PrEP and develop guidelines to make it available in the public health sector for people at high risk of the infection. PrEP has the potential to significantly decrease new HIV infection among key populations in Nigeria. It is therefore important to consider the effect of PrEP and its awareness on the HIV dynamics of Nigeria [20]. [1] analyzed the potential impact of antiretroviral chemoprophylaxis on HIV-1 transmission in resource limited settings and concluded that PrEP could prevent 2.7 to 3.2 million new cases of HIV in sub-Saharan Africa over 10 years, if it is targeted to the highest risk groups, and disinhibition could be prevented. Then in 2016 WHO welcomed a plan by the South African ministry of health to provide immediate antiretroviral treatment to all sex workers with HIV and oral PrEP to HIV-negative sex workers to prevent them from acquiring the infection. [28].

Even though in low- and middle-income countries the cost of PrEP might be a major problem, the clinical and cost effectiveness study of antiretroviral therapy (ART) and PrEP for prevention, showed that PrEP can be cost saving if delivered to individuals at increased risk of infection and the cost of the PrEP drug is reduced [13, 27]. Mathematical models of HIV prevention measures were used to estimate the effects of early diagnosis, early treatment, on the HIV epidemic over the next 40 years, as compared with the current situation [1, 13, 16, 19, 24, 25]. Also, attention has been paid to study the effect of migration and immigration of infective [5, 8, 12, 15, 17, 24]. This study attempts to investigate the potential impact of PrEP in a population with direct flow of infective immigrants especially on the high-risk population groups.

In this study a six compartmental deterministic HIV/AIDS transmission model is develop, incorporating treatment for infected individuals and the inclusion of PrEP for HIV-negative persons at substantial risk in a population, which generalizes the model proposed by [24]. The aim is to show that PrEP, can reduce the incidence of the disease, also its potential impact on direct immigration of HIV infective.

The paper is organized as follows: section 2 is the model formulation. In section 3 the rigorous analysis of the model is presented. In section 4 sensitivity analysis, 5 the numerical simulation is carried out to and discussed in detail while section 6 bears the conclusion. The numerical study of the model is computed with the aid of the MAPLE 18 mathematical software.

2 Mathematical model formulation

There is an inflow of new members Λ and Q immigrants where the fraction $0 > \tau < 1$ is infected immigrants. The total population at time t is denoted $N(t)$ is divided into six the susceptible class $S(t)$, Infective class $I(t)$ be members who are infected at primary stage, Pre-AIDS class $P(t)$ be members in the Pre-AIDS stage, AIDS class $A(t)$ be members with full blown Aids, Treated class $T(t)$ are the infected members at various stage undergoing treatment and the class of susceptible members under the PrEP strategy $V(t)$ who are assumed to be at substantial risk of acquiring the disease at time t respectively.

The following assumptions are made in the development of the model:

The population under study is heterogeneous and varies with time; it is divided into six groups: are the Susceptible (S), PrEP strategy (V), Infective (I), Pre-AIDS (P), Treatment (T) and the full blown AIDS (A) classes and $N(t)$ is the total population per unit time.
 $N(t) = S(t) + V(t) + I(t) + P(t) + T(t) + A(t)$.

For this study HIV is considered to be transmitted through sexual intercourse or through infection from infected needle and infected blood. Full blown AIDS are sexually inactive. There is an inflow of new members Λ , who are susceptible and Q , immigrant into the population per time where the fraction $(0 < \tau < 1)$ are infected immigrants.

Table 2

Parameters of the model
S = the susceptible class.
V = the class of susceptible individuals on the PrEP strategy.
I = the infected individuals in the primary stage.
P = the infected individuals in the pre-AIDS stage.
T = the infected individuals who are receiving treatment.
A = the infected individuals with full blown AIDS.
Λ = the constant inflow of susceptible individual into the population.
Q = the inflow of immigrants into the population.
τQ = the fraction of infected immigrants into the population.
$(1 - \tau)Q$ = the fraction of uninfected immigrants into population.
ωS = the fraction of individuals under then PrEP strategy.
c = is the average number of sexual partners in a unit time.
d = the natural mortality rate of individuals in all the classes.
k = fraction of those receiving treatment who develop full blown AIDS.

$\beta_i, i=1,2,3$ is the contact rate.
σ = the rate at which individuals taking PrEP become susceptible.
δ = the rate of movement in the infected class.
α = is the rate of movement in the pre-AIDS class.
θ = is the rate at which the AIDS patients get treatment.
μ = is the disease induced death in AIDS class.
η = fraction of the infective that get treatment.
$\gamma\delta$ = fraction of the pre-AIDS class that develop full blown AIDS.
$\varepsilon\delta$ = fraction of the infective that goes to the pre- AIDS class.

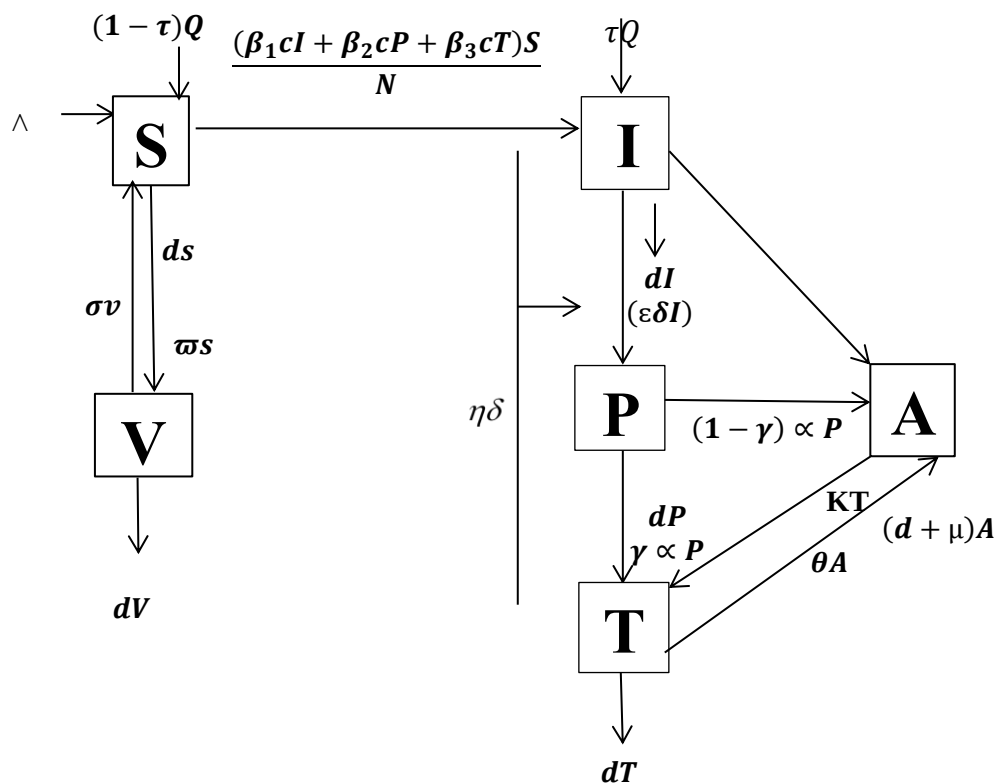


Figure 1

Table 2 and Figure 1 satisfies the model equations as shown below

$$S' = \Lambda + (1 - \tau)Q - \frac{(\beta_1 cI + \beta_2 cP + \beta_3 cT)S}{N} - (\varpi + d)S + \sigma V$$

$$V' = \varpi S - (d + \sigma)V$$

$$I' = \tau Q + \frac{(\beta_1 cI + \beta_2 cP + \beta_3 cT)S}{N} - (\delta + d)I$$

$$P' = \varepsilon \delta I - (\alpha + d)P$$

$$T' = \eta \delta I + \gamma \alpha P + \theta A - (k + d)T$$

$$A' = (1 - \varepsilon - \eta)\delta I + \alpha(1 - \gamma)P + kT - (\theta + \mu + d)A \quad (1)$$

with the initial conditions $S \geq 0, V \geq 0, I \geq 0, P \geq 0, T \geq 0, A \geq 0$ (2)

3 Analysis of model

3.1 Positivity and boundedness of solutions

Lemma 3.1:

Let $S \geq 0, V \geq 0, I \geq 0, P \geq 0, T \geq 0, A \geq 0$. Then the solutions $\{S, V, I, P, T, A\}$ of the system (1) are non-negative for all $t \geq 0$.

Proof: It is necessary to prove that all the solution of the system (1) with positive initial data (2) will remain positive when $t \geq 0$. Considering the first equation

$$\frac{dS}{dt} = \Lambda + (1 - \tau)Q - \frac{(\beta_1 cI + \beta_2 cP + \beta_3 cT)S}{N} - (\varpi + d)S + \sigma V$$

$$\frac{dS}{dt} \geq -(\varpi + d)S$$

Separating the variable $\frac{dS}{dt} \geq -(\varpi + d)S$ (3)

$$\int \frac{1}{S} dS \geq -\int (\varpi + d)dt$$

$$S(t) \geq S_0 e^{-(\varpi + d)t} \geq 0$$

Hence $S(t) \geq 0$.

The same approach can be applied to the other state variables to show that they are non-negative.

Lemma 3.2:

$$\text{Let } \Omega = \left\{ (S, V, I, P, T, A) \in R_+^6 : 0 \leq S + V + I + P + T + A \leq \frac{\Lambda + Q}{d} \right\} \quad (4)$$

is positively invariant for system (1) with non-negative initial conditions (2) in all (R_+^6) .

Proof:

The total population size $N(t) = S(t) + V(t) + I(t) + P(t) + T(t) + A(t)$ this implies that

$$N' = S' + V' + I' + P' + T' + A' \quad (5)$$

$$N' = \Lambda + Q - dN - \mu A$$

$$\text{Let } D = \Lambda + Q. \quad (6)$$

$$\text{Since } \frac{dN}{dt} \leq D - dN.$$

A standard comparison theorem [15, 23] can then be used to show that $N(t) \leq \frac{D}{d} + \left(\frac{N_0 - D}{d} \right) e^{-dt}$

Assuming $N(0) \leq \frac{D}{d}$ we conclude that $N(t) \leq \frac{D}{d}$. Thus, the region Ω is positively invariant.

Hence the system (1) is well posed and epidemiologically meaningful and all the feasible solutions

enter the region $\Omega = \left\{ (S, V, I, P, T, A) \in R^6 : 0 \leq N \leq \frac{\Lambda + Q}{d} \right\}$ with non-negative initial condition. [21]

3.1 Remark

To ensure that the disease-free subspace is invariant, we assume that if the population is free of disease, then the population will remain free of disease. That is, there is no (density independent) immigration of infective. However as long as the infective immigrant are entering the population, the disease-free equilibrium will become unattainable, this is in agreement with [14, 24, 32].

3.2 Existence of equilibrium point for the model

To verify the existence of the equilibrium points, to find the Disease-Free Equilibrium (DFE), it is assumed that there are no infective immigrants by setting $Q=0$ in and the right-hand side of the model equations in (1) is set to zero. At DFE, E_0

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dP}{dt} = \frac{dV}{dt} = \frac{dA}{dt} = \frac{dT}{dt} = 0$$

where $S \neq 0$ and $V \neq 0$ and $I = P = A = T = 0$

Thus,

$$E^0 = (S, V, 0, 0, 0, 0) = \left(\frac{(d + \sigma)\Lambda}{d(\varpi + d + \sigma)}, \frac{\varpi\Lambda}{d(\varpi + d + \sigma)}, 0, 0, 0, 0 \right). \quad (7)$$

$$\text{Also at disease free equilibrium } N = \frac{\Lambda}{d} \quad (8)$$

Similarly, to solve the Endemic Equilibrium Point E^* of the system (1) each of the equations is set to zero to have and substituting $S = S^*, V = V^*, I = I^*, P = P^*, T = T^*, A = A^*$. Endemic Equilibrium Point E^* of the system (1) is obtained as

$$\begin{aligned} P^* &= \frac{\varepsilon \delta I^*}{(\alpha + d)} \\ A^* &= \frac{((\alpha + d)((k + d)(1 - \varepsilon) - d\eta) + \varepsilon\alpha(k + d(1 - \gamma)))\delta I^*}{((\theta + \mu + d)(k + d) - \theta k)(\alpha + d)} \\ T^* &= \frac{((\alpha + d)(\eta(\mu + d) + \theta(1 - \varepsilon)) + \varepsilon\alpha(\gamma(\mu + d) + \theta))\delta I^*}{((\theta + \mu + d)(k + d) - \theta k)(\alpha + d)} \\ V^* &= \frac{(\Lambda + (1 - \tau)Q)q\varpi N}{(R_0(\delta + d)I^* + Nd)(d + \sigma)} \\ S^* &= \frac{(\Lambda + (1 - \tau)Q)qN}{(R_0(\delta + d)I^* + Nd)} \end{aligned} \quad (9)$$

and I^* a quadratic equation:

$$C_2 I^{*2} - C_1 I^* + C_0 = 0 \quad (10)$$

where

$$\begin{aligned} C_2 &= R_0(\delta + d)^2 \\ C_1 &= (Nd - R_0(\Lambda + Q))(\delta + d) \end{aligned}$$

$$C_0 = \tau QNd$$

By Descartes's rule of sign two change of sign means there is at most two real positive root, so there will be positive roots, this implies that $S^*, V^*, I^*, P^*, T^*, A^*$ will have positive value. Therefore, the model (1) has a positive solution.

3.3 Basic reproduction number

The basic reproduction number R_0 is defined as the effective number of secondary cases produced by a typical infected individual during the entire period of infectiousness. The next-generation matrix approach by [27] is employed. Let $x = (S, V, I, P, T, A)^T$.

This is done by taking the largest dominant spectral radius of $R_0 = \left[\frac{\partial F_i(x_0)}{\partial x_i} \right] \left[\frac{\partial V_i(x_0)}{\partial x_i} \right]^{-1}$ where

F_i be the rate at which new infections appear in the compartment i

V_i^{+} be the rate of transfer of individuals into of the compartment i by all other means

V_i^{-} be the rate of transfer of individuals out of the compartment i by all other means

And considering $V_i = V_i^{-} - V_i^{+}$.

Then $F = \left[\frac{\partial F_i(x_0)}{\partial x_i} \right]$ $V = \left[\frac{\partial V_i(x_0)}{\partial x_i} \right]$ at the disease-free equilibrium point gives the following

$$F = \begin{pmatrix} \beta_1 c q & \beta_2 c q & \beta_3 c q & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (11)$$

$$V = \begin{pmatrix} (\delta + d) & 0 & 0 & 0 \\ -\varepsilon \delta & (\alpha + d) & 0 & 0 \\ -\eta \delta & -\gamma \alpha & (k + d) & -\theta \\ -(1 - \varepsilon - \eta) \delta & -\alpha(1 - \gamma) & -k & (\theta + \mu + d) \end{pmatrix} \quad (12)$$

where

$$K_1 = (\delta + d), K_2 = \varepsilon \delta, K_3 = (\alpha + d), K_4 = (d + k), K_5 = (1 - \varepsilon - \eta) \delta, K_6 = (1 - \gamma), \quad (13)$$

$$K_7 = (\theta + \mu + d), K_8 = (\omega + d), K_9 = (d + \sigma), q = \frac{(d + \sigma)}{(\varpi + d + \sigma)}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{K_1} & 0 & 0 & 0 \\ \frac{K_2}{K_1 K_3} & \frac{1}{K_3} & 0 & 0 \\ \frac{K_2 \alpha (\gamma K_7 + \theta K_6) + K_3 (\delta \eta K_7 + \theta K_5)}{K_1 K_3 (K_4 K_7 - k \theta)} & \frac{\alpha (\gamma K_7 + \theta K_6)}{K_3 (K_4 K_7 - k \theta)} & \frac{K_7}{(K_4 K_7 - k \theta)} & \frac{\theta}{(K_4 K_7 - k \theta)} \\ \frac{K_2 \alpha (\gamma k + K_6 K_4) + K_3 (\delta \eta k + K_4 K_5)}{K_1 K_3 (K_4 K_7 - k \theta)} & \frac{\alpha (\gamma k + K_6 K_4)}{K_3 (K_4 K_7 - k \theta)} & \frac{k}{(K_4 K_7 - k \theta)} & \frac{K_4}{(K_4 K_7 - k \theta)} \end{pmatrix}$$

$R_0 = \rho(FV^{-1})$, that is the dominant spectral radius of the largest eigenvalues given by

$$R_0 = \text{Max}[\lambda_1 \lambda_2 \lambda_3 \lambda_4]$$

It follows that the reproduction number R_0 for the model (1) is

$$R_0 = \frac{cq\{\beta_1 K_3(K_4 K_7 - k\theta) + \beta_2 K_2(K_4 K_7 - k\theta) + \beta_3 K_2(\gamma K_7 + \theta K_6) + K_3(\delta \eta K_7 + \theta K_5)\}}{K_1 K_3(K_4 K_7 - k\theta)} \quad (14)$$

3.4 Local stability of DFE

Theorem 3.4: The disease-free Equilibrium of the model (1) in the absence of infective immigrant is locally asymptotically stable if the $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

To determine the local stability of Disease-Free Equilibrium E_0 , we compute the variational matrix of the linearized system (1) corresponding to the equilibrium point E_0 to have the Jacobian matrix given as:

Using the substitution in (14) to have

$$J(E_0) = \begin{pmatrix} -K_8 & \sigma & -\beta_1 cq & -\beta_2 cq & -\beta_3 cq & 0 \\ \varpi & -K_9 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(K_1 - \beta_1 cq) & \beta_2 cq & \beta_3 cq & 0 \\ 0 & 0 & \varepsilon \delta & -K_3 & 0 & 0 \\ 0 & 0 & \eta \delta & \gamma \alpha & -K_4 & \theta \\ 0 & 0 & K_5 & K_6 & k & -K_7 \end{pmatrix} \quad (15)$$

The characteristics equation corresponding with (15)

$$(\lambda^2 b_0 + \lambda b_1 + b_2)(\lambda^4 a_0 + \lambda^3 a_1 + \lambda^2 a_2 + \lambda a_3 + a_4) = 0 \quad (16)$$

where

$$b_0 = 1, b_1 = (K_8 + K_9), b_2 = K_8 K_9 - \omega \sigma,$$

$$a_0 = 1, a_1 = (K_1 + K_3 + K_4 + K_7 - cq\beta_1)$$

$$a_2 = K_3(K_1 + K_4 + K_7) + K_7(K_1 + K_4) + K_1 K_4 - \theta k - cq(\beta_1(K_3 + K_4 + K_7) + K_2 \beta_2 + \eta \delta \beta_3)$$

$$a_3 = K_3(K_1 K_4 - k\theta) + K_7(K_1 + K_4) + K_1(K_4 K_7 - k\theta) - cq((\beta_1(K_3(K_4 + K_7) + K_4 K_7 - k\theta) +$$

$$\beta_2 K_2(K_4 + K_7) + \beta_3(\eta \delta(K_7 + K_3) + \gamma \alpha K_2 + \theta K_5))$$

$$a_4 = K_1 K_3(K_7 K_4 - k\theta)(1 - R_0).$$

By the Routh Hurwitz criteria if $b_1 > 0, b_2 > 0$ then $b_1 b_2 > 0$ for the quadratic equation and if $a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0$ and $a_5 > 0$ then $a_1 a_2 a_3 > a_3^2 + a_1^2 a_4$ for the fourth root equation. On further simplification the roots $b_j > 0$ for $j = 0, 1, 2$ $a_i > 0$ for $i = 0, 1, 2, 3, 4, ..$

So $a_4 > 0$ to have

$$\begin{aligned} K_1 K_3 (K_7 K_4 - k\theta)(1 - R_0) &> 0 \\ (1 - R_0) &> 0 \\ \therefore R_0 &< 1. \end{aligned}$$

It follows that all the eigenvalues $J(E_0)$ have negative real parts this implies that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$.

Thus, disease free equilibrium of the model (1) is locally asymptotically stable if $R_0 < 1$.

3.5 Global stability of disease free equilibrium

To establish the global asymptotic stability (GAS) of the disease-free equilibrium (DFE) state of the model (1) the approach of [7] is employed as follows. The model is written as

$$\frac{dX}{dt} = Y(X, Z) \tag{17}$$

$$\frac{dZ}{dt} = G(X, Z), G(X^*, 0) = 0 \tag{18}$$

where $X = (S, V) \in \mathbb{R}_+^2$ denotes the number of uninfected individuals

$Z = (I, P, T, A) \in \mathbb{R}_+^4$ denotes the number of infected individuals including.

The DFE of the system is denoted $E_0 = (X^*, 0)$. The two conditions H_1 and H_2 that if satisfied will guarantee the global stability

$$H_1: \frac{dX}{dt} = Y(X, 0), X^* \text{ is globally asymptotically stable (GAS)}$$

$$H_2: \frac{dZ}{dt} = G(X, Z) = AY - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0 \text{ for } (X, Z) \in \Omega$$

where $A = D_Z G(X^*, 0)$ is an M-matrix (the off diagonal elements of A are non-negative) and Ω is the region where the model makes biological sense. If the system (1) satisfies the conditions H_1 and H_2 then the following theorem 3.5 holds

Theorem 3.5

The disease-free equilibrium (DFE) point $E_0 = (X^*, 0)$ of the system (1) is globally asymptotically stable provided $R_0 < 1$ then the two conditions H_1 and H_2 are satisfied.

Proof

Let $X = (S, V)$ and $Z = (I, P, T, A)$ to have two vectors valued functions

$$Y(X, Z) = \begin{pmatrix} \Lambda + (1-\tau)Q - \frac{(\beta_1 cI + \beta_2 cP + \beta_3 cT)S}{N} \sigma V - (w+d)S \\ wS - (d+\sigma)V \end{pmatrix} \quad (19)$$

$$G(X, Z) = \begin{pmatrix} \tau Q + \frac{(\beta_1 cI + \beta_2 cP + \beta_3 cT)S}{N} - (\delta+d)I \\ \varepsilon \delta I - (\alpha+d)P \\ \eta \delta I + \gamma \alpha P + \theta A - (k+d)T \\ (1-\varepsilon-\eta)\delta I + (1-\gamma)\alpha P + kT - (\theta+\mu+d)A \end{pmatrix} \quad (20)$$

Considering the reduced system $\frac{dX}{dt} = Y(X, 0)$ from condition H_1

$$Y(X, 0) = \begin{pmatrix} \Lambda + (1-\tau)Q + \sigma V - (w+d)S \\ wS - (d+\sigma)V \end{pmatrix} \quad (21)$$

$X^* = (S^*, V^*) = \left(\frac{\Lambda(d+\sigma)}{d(\omega+d+\sigma)}, \frac{\Lambda\omega}{d(\omega+d+\sigma)} \right)$ is a GAS equilibrium point for the reduced system (21) to show this, the second equation in (21) is solved to obtain $V(t) = \frac{\Lambda\omega}{d(\omega+d+\sigma)} + \left(V_0 - \frac{\Lambda\omega}{d(\omega+d+\sigma)} \right) e^{-(d+\sigma)t}$; it approaches zero as $\frac{\Lambda\omega}{d(\omega+d+\sigma)}$ as $t \rightarrow \infty$. Similarly the solution of the first equation gives $S(t) = \frac{\Lambda(d+\sigma)}{d(\omega+d+\sigma)} + \left(S_0 - \frac{\Lambda(d+\sigma)}{d(\omega+d+\sigma)} \right) e^{-(\omega+d)t}$ which approaches $\frac{\Lambda(d+\sigma)}{d(\omega+d+\sigma)}$ as $t \rightarrow \infty$. This asymptotic dynamic is independent of the initial condition in Ω . Thus, the convergence of the solution of the reduced system in (19) is global in Ω .

To compute $\frac{dZ}{dt} = G(W, Z) = AZ - \hat{G}(W, Z)$, and show $\hat{G}(W, Z) \geq 0$

$$A = D_Z G(X^*, 0) = \begin{pmatrix} \beta_1 c - (\delta+d) & \beta_2 c & \beta_3 c & 0 \\ \varepsilon \delta & -(\alpha+d) & 0 & 0 \\ \eta \delta & \gamma \alpha & -(k+d) & \theta \\ (1-\varepsilon-\eta)\delta & \alpha(1-\gamma) & k & -(\theta+\mu+d) \end{pmatrix} \quad (22)$$

$$\hat{G}(W, Z) = \begin{pmatrix} \beta_1 cI \left(1 - \frac{S}{N}\right) + \beta_2 cP \left(1 - \frac{S}{N}\right) + \beta_3 cT \left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (23)$$

Since $1 > \frac{S}{N}$ it is clear that $\hat{G}(W, Z) \geq 0$. Hence DFE is globally asymptotically stable when $R_0 < 1$.

4 Sensitivity analysis

It is important to perform the sensitivity analysis in order to determine the relative importance of the model parameter to diseases transmission. It is therefore pertinent to know the significance of the different factors responsible for the disease transmission, to determine how best to reduce the mortality and morbidity due to HIV. The normalized forward sensitivity index using the [9]

approach of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. For a differentiable variable function, the sensitivity index is defined as

$$H_m^{R_0} = \frac{m}{R_0} \frac{\partial R_0}{\partial m} \quad (24)$$

where m stands for the different parameter

We compute the numerical sensitivity indices (24) to enable us know which of the parameters have a high impact on R_0 so that it will be the target point of any control or preventive strategy.

5 Numerical simulation and discussion

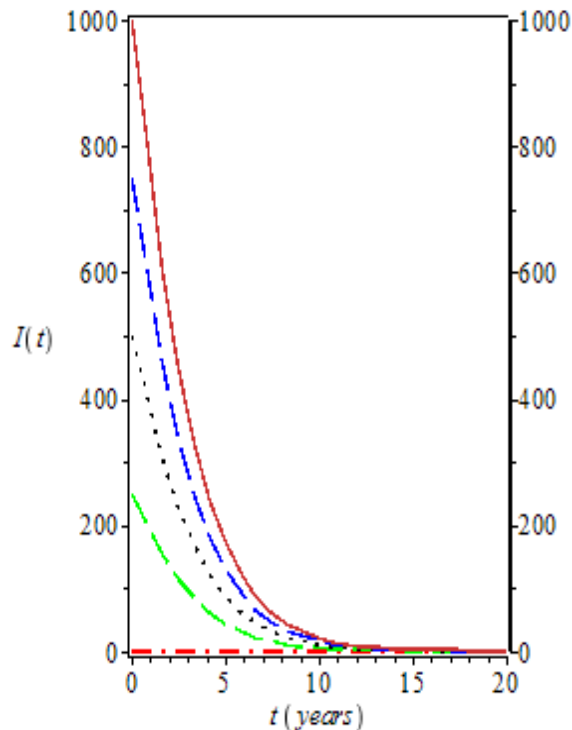


Figure 1: The global stability of the DFE at various initial values

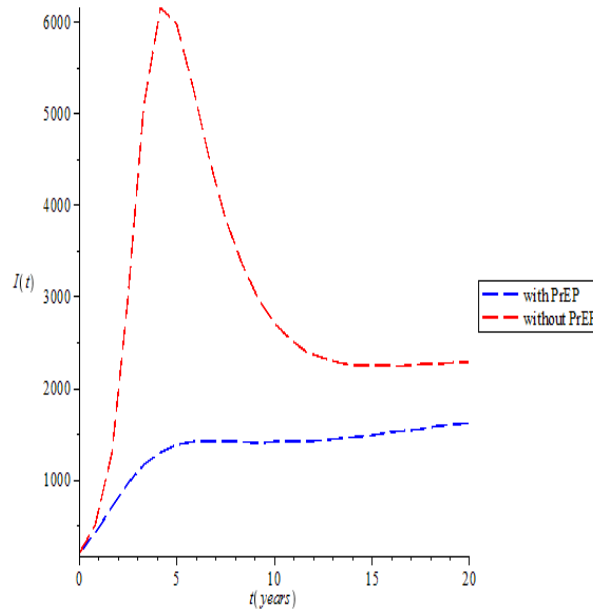


Figure 2 :The effect of PrEP on the Infective class $I(t)$ in the absence of infected immigrants

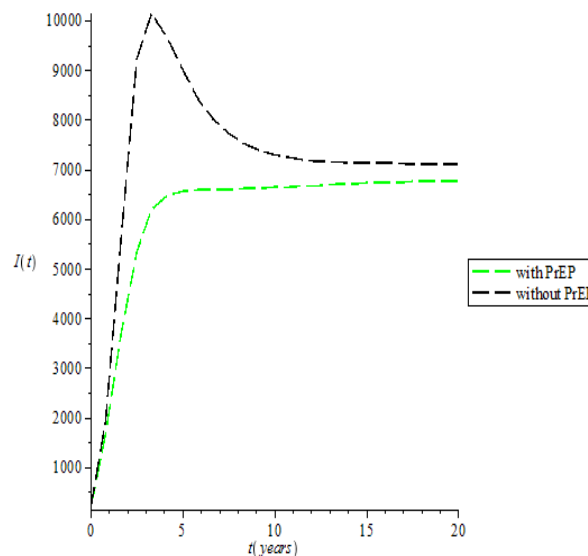


Figure 3 :The effect of PrEP on the Infective class $I(t)$ in the presence of infected immigrants

Figure 1 demonstrates the global stability of the disease-free equilibrium point proved in the theorems 3.5. This implies that regardless of the initial size of the number of infections in the population whenever $R_0 < 1$ the infection will reduce in the population. Figure 2 shows the effect of PrEP on the infective class $I(t)$ in a population with infective immigrant.

Immigration poses a significant risk for disease dissemination; the effect may be substantial in developing countries which normally do not check the complete health status of immigrants. These immigrants place their partners in their home countries and their destination countries at risk of HIV/AIDS. It can be observed from Figure 2 that the infective population increases continuously due to the presence of infected immigrants, meaning that the presence of immigrants increases the total population initially making the infection more endemic and persistent in the population then decreases with time. It is clear that direct inflow of infected immigrants increases the numbers of infective which ultimately increase the prevalence of the disease, to check the spread of disease and prevent it from becoming endemic in the population effective immigration policies such as screening of immigrants should be taken seriously.

Figure 3 also shows that in the population where the PrEP strategy is in place, the peak of epidemic is higher in the figure 1 with presence of infected immigrants than in the absence of infected immigrants but either way it still reduces infection. Thus, PrEP can lead to reduction in the risk of sexual transmission of HIV.

Figure 4 shows the combine effect of PrEP on treatment in the population with or without the inflow of infective immigrants the result shown in Figure 4 suggests that treatment will reduce the disease prevalence in the community, it benefits can be improved when combined with the use of PrEP but their benefit can be compromised if the adherence to usage is poor and illicit sexual behavior is increased. Treatment is important to slow down the progression of HIV to AIDS due to the fact that it reduces the viral load of people living with HIV while PrEP can reduce the risk of being infected by an HIV negative person. Plot result suggest that combination of PrEP and treatment is more effective as treatment keeps people alive by rendering them much less infectious and will be on treatment for life while PrEP prevents people at risk from likely infection. PrEP and treatment can lead to decrease in new cases of infection ultimately to a substantial decline in prevalence of infection.

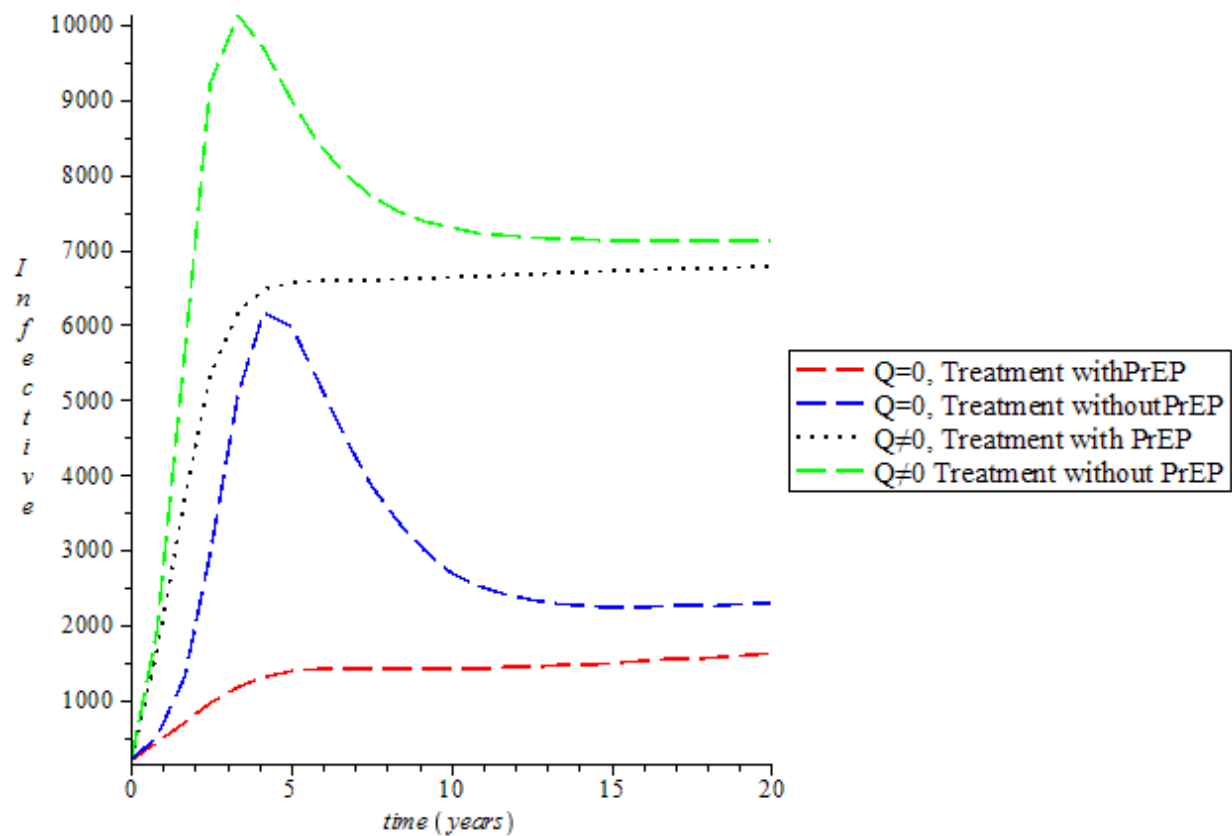


Figure 4: The effect of PrEP on Treatment in the Infective class $I(t)$ with or without immigration

5.1 Result of Sensitivity Index

Sensitivity analysis is used to discover parameters that have a high impact on the basic reproduction number, R_0 and should be the target of any intervention strategies. The parameter which had the greatest influence on the number of new infection caused in the primary infection (basic reproduction number R_0) was determined. Table 5.1 contains the numerical values of these parameters calculated using the approach of [9]. From Table 5.1 it can be seen that the most important parameter contributing to the infection is the number of sexual partners c also influencing the infection is the contact rate β_1 of the infective class, the number of sexual partners c and contact rates of the infective β_1 together interact to cause increased incidence of HIV and the increased prevalence of HIV. This means that while there is any increment in either c , β_1 or any of the parameters with positive values and other parameters kept constant there will be an increase in the value of R_0 . The parameter with negative values means that when these values are increased while other are kept constants there is a decrease in the value R_0 . thus these parameter with positive and negative values have to be the focus or target of intervention strategies as they are crucial to the transmission and prevalence of the HIV/AIDS.

Table 5.1: Sensitivity Indices of R_0

Parameter	Sensitivity values	Value	Source
C	+1	3	[20]
β_1	+0.7562310755	0.15	[20]
ε	+0.1369270468	0.6	[20]
β_2	+0.1274256821	0.05	[20]
β_3	+0.1163432424	0.05	[21]
θ	+0.0922648381	0.1	[21]
σ	+0.0464576074	0.4	[21]
η	+0.0098891756	0.01	[21]
δ	-0.7086120282	0.4	[20]
ϖ	-0.4878048780	0.4	[20]
α	-0.1088394479	0.5	[20]
k	-0.0999801506	0.08	[21]
μ	-0.0904557236	1	[20]
D	-0.0611486848	0.02	[20]

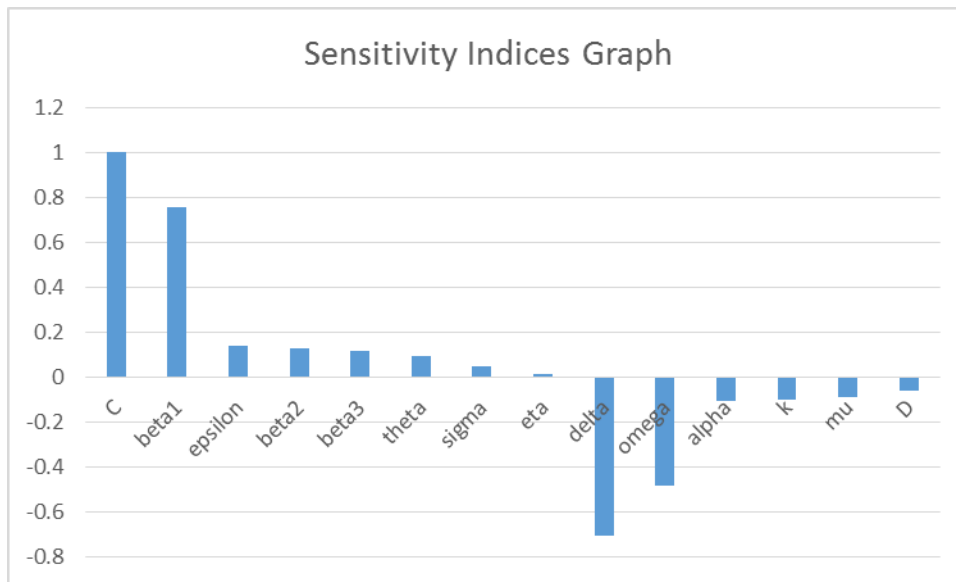


Figure 5.1: The chart of parameters

Generally, the number of sexual partner is the strongest attributor of risk of HIV acquisition; plot result also revealed that the number of new HIV infection is large due to the influence of the

increase in the number of sexual partners. Thus, to reduce the spread of the disease the number of sexual partners as well as unsafe sexual behavior and interaction with infected individuals must be restricted.

6 Conclusion

In this work, a nonlinear deterministic mathematical model was proposed and analyzed to study the effect of PrEP in the dynamics of HIV/AIDs with variable in flow of infective immigrants, and also incorporating treatment at different stage of infection using the standard incidence. Analysis of the model showed that as long as the infective immigrants are entering the population, the disease free equilibrium become unattainable and the existence and uniqueness of Disease Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP) of the model without infective immigrants were proved depending on R_0 . It was established that DFE is locally asymptotically stable if $R_0 < 1$ and for $R_0 > 1$ it is unstable, global stability of the DFE was proved using the Next Generation Operator method [9] and it was established that the effectiveness of PrEP is strict adherence to usage of the medication.

Numerical simulation of the effect of PrEP in the absence of immigration of infective was compared with the model without immigration, it was observed that PrEP reduces the number of new HIV incidences, however, only people who are HIV negative and are at very high risk of HIV infection should take PrEP. Productive campaign messages and awareness about PrEP in various communities needs be created that PrEP is acceptable and effective as an additional choice for the prevention of HIV infection as this in turn strengthens health education regarding HIV infection leading to AIDS. The National Health Care to HIV/AIDS should therefore seek to recommend PrEP to people at risk or have been at risk in the past as recommended by WHO also ensure that they have access to and are supported in the use of PrEP and other prevention measures regardless of social and economic status.

Sensitivity indices of the key parameter revealed that an increase in the number of sexual partner c results in the increase of the incidence of the HIV/AIDS infection. Research results suggest that reduction in the number of sexual partners, change in risky behavior as well as unsafe sexual practices and interactions are highly important for preventing subsequent transmission.

Simulation shows that when there is immigration of infected individuals then the rate of spread of the diseases will be massive as in the case where there is no protection. Effective immigration policies such as screening should be put in place to minimize the potential of infecting others by an already infected individual migrant.

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Numerical simulation of the intracellular dynamics of HBV/Malaria co-infection with immune control and treatment

T.I. Chinebu^{*†}, C.U. Okonkwo^{*}, T. Oyedepo^{*}, O.J. Iyoke^{*}, M.E. Ukwungwu^{*},
N.J. Ikechukwu^{*}, and T.A. Iyanda^{*}

Abstract

In this paper, we proposed an intracellular mathematical model to describe the dynamics of the co-infection of hepatitis B virus (HBV) and malaria co-infection by taking into account the immune response and treatment. We established the wellposedness of the model by proving the existence, positivity and boundedness of solution of the problem. The basic reproduction number of HBV only model in the hepatocyte and malaria only model, both in the hepatocyte and erythrocyte were obtained, and when combined gives the basic reproduction number of the co-infection model. The local stability of the disease free equilibrium is obtained using characteristic equation. We showed that the disease-free equilibrium with treatment effect exists and is asymptotically stable if $R_{0H\pi} < 1$, $R_{0MH\pi_1} < 1$ and $R_{0MR\pi_2} < 1$ and unstable if $R_{0H\pi} > 1$, $R_{0MH\pi_1} > 1$ and $R_{0MR\pi_2} > 1$. Finally, we noticed from the numerical result that when the immune response is large, strong and faster and treatment is effective than the rate at which both infections replicate in the hepatocytes and erythrocytes, HBV only infected hepatocytes and malaria only infected hepatocytes and erythrocytes can be controlled with malaria resurfacing after some time as indicated by the oscillation in the malaria infected erythrocytes. There will be resistance in the co-infected hepatocytes and this may cause liver dysfunction.

Keywords: Hepatitis B virus; Malaria; hepatocytes; erythrocytes; intracellular transmission; stability; immune response; treatment

1 Introduction

Hepatitis B virus (HBV) infection which has become a major global health problem is a noncytotoxic, enveloped virus that causes acute and chronic hepatitis with hepatocellular carcinomas (HCC). HBV is transmitted only through direct contact with an infected person's body

^{*}Department of Applied Sciences, Federal College of Dental Technology and Therapy, Enugu, Nigeria

[†]Corresponding Author

fluid, such as sexual contact, blood to blood transmission and vertical transmission (mother to child). HBV depends upon the host cell for their reproduction but must overcome certain cellular constraints. The objective of a viral particle is to move the genome from an infected cell to an uninfected cell in a replication competent form [47]. For successful elimination of HBV, antiviral cytokines such as type I interferon (INF- α and INF- β) [8] and natural killer cell are respectively released and activated against HBV. Also, CD4⁺ and CD8⁺T cells responses play a central role in the elimination of HBV infection [21]. The T cell response during acute self-limited hepatitis B in people is characterized by a vigorous, polyclonal and multi-specific cytotoxic and helper T cell response. Studies have shown that transferred virus specific cytotoxic T cell can abolish HBV gene expression and replication in the liver without killing the hepatocytes [16]. Also, in acute HBV infection, a vigorous polyclonal cellular immune response of INF- γ is one of the most important mediator.

Malaria is a life threatening mosquito borne blood disease caused by a plasmodium parasite. The infection is initiated when sporozoites are injected with the saliva of a feeding mosquito into the blood [19],[32], and then carried by the circulatory system to the liver which finally invades hepatocyte. The highly motile intracellular parasite in the hepatocyte [37],[43],[29] undergoes an asexual replication, called exocytocytic schizogony which culminates in the production of merozoites that are released into the blood stream. Hypnozoites react several weeks to months or years after primary infection and are responsible for relapses [24]. Merozoites invade erythrocytes [24] and undergo a trophic period in which the parasite enlarges [38],[48]. The early trophozoite is often referred to as ring form because of its morphology. Trophozoite enlargement is accompanied by an active metabolism including the injection of host cytoplasm and proteolysis of hemoglobin into amino acids. The end of the trophic period is manifested by multiple rounds of nuclear divisions without cytokinesis resulting in a schizont [39]. Merozoites bud from the mature schizonts, also called a segmenter [44] and the merozoites are released following rupture of the infected erythrocyte [3]. The blood stage is responsible for the pathology associated with malaria.

There are several geographical areas mainly in Sub Sahara Africa, (SSA), where endemicity of HBV and Malaria overlap despite having different transmission routes [5],[2],[7]. In tropical areas HBV is highly contagious from one infected individual to another and sometimes can present same route of transmission of plasmodium (By Blood-to-Blood Contact, sharing needles and blood transfusions). Therefore, it is not rare that viral infections may be present in the same individual, influencing each other from a clinical perspective.

Co-infection is the process in which a host becomes infected with multiple pathogen species simultaneously [25] it is of vital health importance since there is an interaction and interface of the pathogen species within the host [1]. Both diseases have sessions of high activities in the hepatocyte and their consequences on the erythrocyte (red blood cells) may result to a suppressed immunity in an individual, making them susceptible to the infections; this will also lead to increased mortality and morbidity [2].

Similarity between HBV and malaria biology is the participation of the liver and a key organ part of the immunopathogenesis in both infection [33],[13],[4],[46]. Notably, severe vivax malaria is

associated with remarkable hepatic involvement [13][4],[4],[30], whereas tissue damage is determinant for the presentation of cirrhosis and hepatocellular carcinoma in chronic HBV infections [49],[45],[28]. Counter intuitively, HBV infection has been to lead a distinct systemic inflammatory response in plasmodium infections, results in increasing odds for asymptomatic malaria [4].

Pasquetto et al in [14], described the immunological features of malaria and HBV co-infection where they describe that the hepatic stage infection seems to trigger an early T cell independent cytokine response along with a delayed cytokine response that was simultaneous with the infiltration of T cells. On the other hand, the T cell response appeared earlier in the blood stage than in the hepatic stage infection, possibly because parasitemia was detectable earlier in those individuals and a T cell independent phase was not seen, perhaps reflecting that it was induced primarily by infected hepatocytes. In both cases, however, the generated cytokine ripostes were accompanied with a decline in HBV RNA and DNA in the liver.

Malaria can normally be treated with antimalaria drugs. The type of treatment depends on which kind of malaria is diagnosed, where the patient was infected, the age of patient and how the patient was at the start of treatment [40]. Acute hepatitis B infection does not usually require treatment because about 95% of most adults clear the infection spontaneously [35],[26]. Early antiviral treatment may only be required in fewer than one percent of patients, whose infection are of great severity (fulminant hepatitis) or who are immunocompromised. In patients with fulminant infection, intravenous (IV) feeding may be needed, if the patient has persistent vomiting and can't take oral foods. Ninety percent of patients with acute HBV infection have a favorable course and recover completely [20]. On the other hand, treatment of chronic HBV infection may be necessary to reduce the risk of cirrhosis and liver cancer. Chronically infected individuals with persistently elevated serum alanine aminotransferase, a marker of liver damage and HBV DNA level are candidates for therapy [36]. Thirty to forty percent of patients with chronic hepatitis B respond to *IFN α* treatment. Immune defense is partially active in chronic hepatitis B patients, since the viral load and HBsAg level are lower [23].

Many epidemic models that study the dynamics of cell-to-cell transmission were developed [50],[18],[11]. In other studies, mathematical models, that describe the dynamics of hepatitis B virus infection with cell-to-cell transmission and immune control were proposed by [6],[9],[15]. Also, [10], included treatment and investigated the intracellular delay effect on the stability of the endemically infected steady state. Gurarie et al, [17] developed a new agent-based model that accounts for the essential in – host processes: parasite replication and its regulation by innate and adaptive immunity. Subsequently, [41] proposed within host mathematical model and considered the dynamics of *P. falciparum* malaria from the liver to the blood in human host and then to the mosquito. Orwa et al, [31] present a mathematical model of in – host *P.falciparum* malaria dynamics in the presence of vaccines. Chinebu et al, [12] designed simulation of an intracellular differential equation model of the dynamics of malaria with immune control and treatment which considered the parasite in the liver and blood. However, Scotto et al [34], performed a systemic search of the epidemiology of HBV/Malaria co-infection in particular, their overlapping clinical and histological features and their reciprocal conditioning.

2 Model of HBV and Malaria co-infection

2.1 Model formulation

The formulation of this intracellular co-infection mode, closely follows the epidemiological dynamics of the two diseases in the hepatocytes and erythrocytes i.e., Red Blood Cells (RBCs).

Table 1: Variables with their description

Variables	Description
x	Susceptible hepatocytes
y_H	HBV only infected hypatocytes
z_H	CTL response to HBV
y_M	Malaria (sporozoite) only infected hepatocytes
z_M	CTL response to sporozoite infected hepatocytes
B	Uninfected susceptible erythrocytes (Red blood cells)
y_{HM}	Dually infected hepatocytes with HBV and Malaria (sporozoites)
C	Infected erythrocytes with merozoites
z_{MB}	CTL response to merozoite infected erythrocytes

Table 2: Parameters with their description

Parameters	Description
ψ	Recruitment rate of susceptible hepatocytes
a_1	Natural death rate of hepatocytes
$\frac{\beta a_3}{\mu}$	HBV infected rate of uninfected hepatocytes by free virus
σ	infection rate of uninfected hepatocytes by direct contact of both infected and uninfected hepatocytes
$\frac{\eta b_3}{\Lambda}$	Malaria parasite infection rate of uninfected hepatocytes with sporozoites
$\zeta_1 y_H, \zeta_2 y_M, \zeta_3 y_{HM}$	Proliferation rate of HBV only infected hepatocytes, malaria only infected hepatocytes and HBV-Malaria co-infected hepatocytes respectively
$a_2 y_H z_H$	Rate at which HBV infected hepatocytes are killed by CTL response
$b_1 y_H z_H$	Rate of curing HBV infected hepatocytes by CTL response
$a_5 y_H z_H$	Antigen dependent proliferation (secondary immune response to HBV infected hepatocytes)
$a_3 y_H$	Production rate of free HBV from infected hepatocytes
μv	The removal rate of free virus (natural death)
$\gamma_1, \gamma_2, \gamma_3$	Antigen independent proliferation (primary immune response)
τ	Probability of acquiring Malaria (sporozoites) by HBV infected hepatocytes

$a_6 y_M z_M$	Rate at which Sporozoites infected hepatocytes are killed by CTL response
$a_4 y_M z_M$	Secondary immune response to malaria (sporozoites) infected hepatocytes
ϕ	Recruitment rate of enrythrocytes (RBCs)
k_1	Natural death rate of enrythrocytes
$d_1 C z_{MB}$	Rate of curing merozoite infected enrythrocytes by CTL response
$b_2 y_M z_M$	Rate of curing sporozoites infected hepatocytesby CTL response
ρ	Probability of acquiring HBV by sporozoite infected hepatocytes
$d_2 C z_{MB}$	Killing rate of merozoite infected enrythrocytes by CTL response
d_3	Proliferation rate of infected erythrocytes
$d_4 C z_{MB}$	Secondary immune response to merozoites infected erythrocytes
k_2	Natural death rate of merozoites
k	Rate at which merozoitesare produced per bursting of infected hepatocytes
α	Rate at which uninfected erythrocytes are being infected by merozoites
π	Treatment rate of HBV infected hepatocytes
π_1	Treatment rate of malaria (sporozoite) infected hepatocytes
π_2	Treatment rate of malaria (merozoite) infected erythrocytes
$\omega y_{HM} z_H z_M$	Rate at which co-infected hepatocytes are killed by CTL response

2.2 The model

The variable and parameters that describe the intracellular co-infection dynamics of HBV and malaria are in Table 1 and 2 respectively and the transmission dynamics of the co-infection of HBV and malaria are summarized in the compartmental diagram in figure 1. From the description of the intracellular dynamics of the co-infection in figure 1, we deserve the following system of non linear ordinary differential equation:

$$\left. \begin{aligned}
 \frac{dx}{dt} &= \psi + b_1 y_H z_H + b_2 y_m z_M - a_1 x - \frac{(1 - \pi_1) \eta b_3 x y_M}{\Lambda} - (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H \\
 \frac{dy_H}{dt} &= \zeta_1 y_H + (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H - a_1 y_H - (a_2 + b_1) y_H z_H - (1 - \pi_1) \frac{\tau \eta b_3 y_M}{\Lambda} \\
 \frac{dz_H}{dt} &= \gamma_1 + a_5 y_H z_H - b_4 z_H \\
 \frac{dy_M}{dt} &= \zeta_2 y_M + (1 - \pi_1) \left(\frac{\eta b_3 x y_M}{\Lambda} \right) - (b_2 + a_6) y_H z_M - a_1 y_M - k y_M - \rho (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] y_H \\
 \frac{dz_M}{dt} &= \gamma_2 + a_4 y_M z_M - b_4 z_M \\
 \frac{dy_{HM}}{dt} &= \zeta_3 y_{HM} + \rho (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] y_H + (1 - \pi_1) \left(\frac{\zeta \eta b_3 y_M}{\Lambda} \right) - a_1 y_{HM} - \omega y_{HM} z_H z_M \\
 \frac{dB}{dt} &= \varphi + d_1 C z_{MB} - (1 - \pi_2) \alpha B M - k_1 B \\
 \frac{dM}{dt} &= k C - k_2 M \\
 \frac{dC}{dt} &= (1 - \pi_2) \alpha B M + d_3 C - k_1 C - (d_1 + d_2) z_{MB} \\
 \frac{dz_{MB}}{dt} &= \gamma_3 + d_4 C z_{MB} - b_4 z_{MB}
 \end{aligned} \right\} \quad (2.1)$$

From system (2.1) we have that

$$kC - k_2 M = 0 \Rightarrow M = \frac{kC}{k_2} \quad (2.2)$$

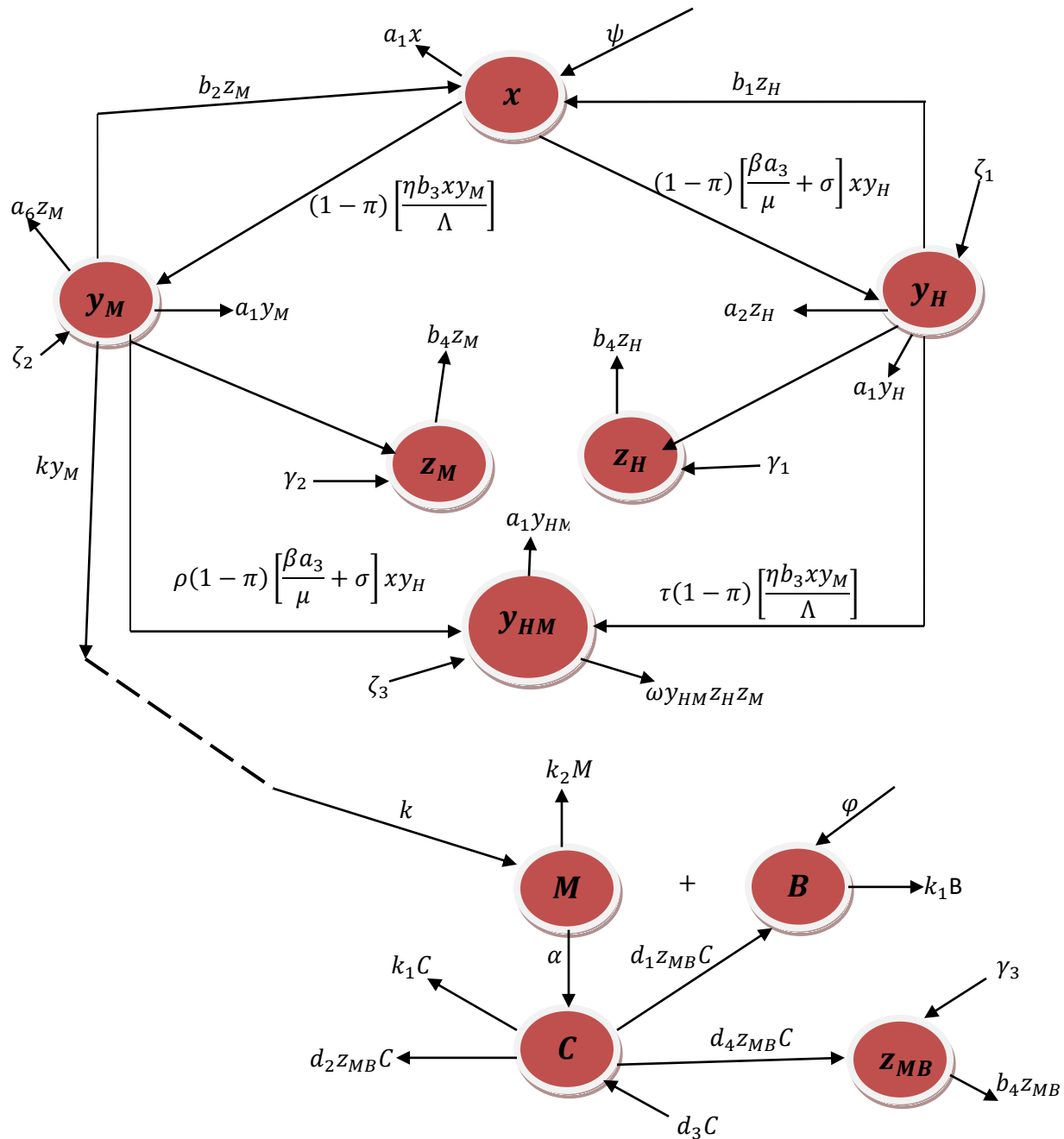


Figure 1: Schematic diagram of the Dynamics of HBV/Malaria Co-infection

Substituting (2.2) into (2.1) we obtain

$$\left. \begin{aligned} \frac{dx}{dt} &= \psi + b_1 y_H z_H + b_2 y_M z_H - a_1 x - (1 - \pi_1) \left(\frac{\tau \eta b_3 x y_M}{\Lambda} \right) - (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H \\ \frac{dy_H}{dt} &= \zeta_1 y_H + (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H - a_1 y_H - (b_1 + a_2) y_M z_H - (1 - \pi_1) \left(\frac{\tau \eta b_3 x y_M}{\Lambda} \right) \\ \frac{dz_H}{dt} &= \gamma_1 + a_5 y_H z_H - b_4 z_H \\ \frac{dy_M}{dt} &= \zeta_2 y_M + (1 - \pi_1) \left(\frac{\tau \eta b_3 x y_M}{\Lambda} \right) - (b_2 + a_6) y_M z_M - a_1 y_M - k_1 y_M - \rho (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] y_H \\ \frac{dZ_M}{dt} &= \gamma_2 + a_4 y_M z_M - b_4 z_M \\ \frac{dy_{HM}}{dt} &= \zeta_3 y_{HM} + \rho (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] y_H + (1 - \pi_1) \left(\frac{\tau \eta b_3 y_M}{\Lambda} \right) - a_1 y_{HM} - \omega y_{HM} z_H z_M \\ \frac{dB}{dt} &= \varphi - (1 - \pi_2) \frac{\alpha k B C}{k_2} - k_1 B + d_1 C z_{MB} \\ \frac{dC}{dt} &= (1 - \pi_2) \frac{\alpha k B C}{k_2} + d_3 C - k_1 C - (d_1 + d_2) C z_{MB} \\ \frac{dz_{MB}}{dt} &= \gamma_3 + d_4 C z_{MB} - b_4 z_{MB} \end{aligned} \right\} \quad (2.3)$$

2.3 Basic frame work

The model sub divides the total population at time t denoted by $N(t)$, into various mutually exclusive compartments depending on their disease status: susceptible (uninfected) hepatocytes to both diseases $x(t)$ HBV infected hepatocytes $y_H(t)$; CTL response to HBV infected hepatocytes; $z_H(t)$; Malaria (sporozoites) infected hepatocytes $y_M(t)$; Dually infected hepatocytes with HBV and Malaria (sporozoites) $y_{HM}(t)$; Susceptible (uninfected) erythrocytes with malaria (merozoites) $B(t)$, infected erythrocytes with malaria (merozoites) $C(t)$; and CTL response to infected erythrocytes with merozoites, $z_{MB}(t)$. The total population is :

$$N(t) = x(t) + y_H(t) + z_H(t) + y_M(t) + z_M(t) + y_{HM}(t) + B(t) + C(t) + z_{MB}(t) \quad (2.4)$$

2.4 Positivity analysis of solution

For system (2.3) to be epidemiologically meaningful, it is important to prove that the feasible solution sets for all time $t \geq 0$. In other words, solution of system (2.3) with positive initial data remain positive for all time t .

Theorem 2.1: The region $\Gamma \in \mathbb{R}_+^9$ is positively invariant with respect to the system of equation (2.3) and a non- negative solution exists for all $0 < t < \infty$.

Proof: if $\pi_1 = \pi_2 = \pi_3 = 0$ and $x(0) = x_0 > 0, y_H(0) = y_{H0} > 0, z_H(0) = z_{H0} > 0, y_M(0) = y_{M0} > 0, z_M(0) = z_{M0} > 0, y_{HM}(0) = y_{HM0} > 0, B(0) = B_0 > 0, C(0) = C_0 > 0$ and $z_{MB}(0) = z_{MB0} > 0$ are the non negative initial conditions, the solution $x(t), y_H(t), z_H(t), y_M(t), z_M(t), y_{HM}(t), B(t), C(t)$, and $z_{MB}(t)$ of system (2.3) are all non negative for $t > 0$ and if not, let there exist a time $\bar{t}$ such that $x(\bar{t}) = 0, x'(\bar{t}) \leq 0$ and $x(t) > 0, y_H(t), z_H(t) > 0, y_H(t) > 0, z_M(t) > 0, y_{HM}(t) > 0, B(t) > 0, C(t) > 0$ and $z_{MB}(t) > 0$ for $0 < t < \bar{t}$. Suppose there exist a time $\hat{t}$ such that $x(\hat{t}) = 0, x'(t) \leq 0, x(t) > 0, y_H(t) > 0, z_H(t) > 0, y_M(t) > 0, z_M(t) > 0, y_{HM}(t) > 0, B(t) > 0, C(t) > 0$, and $z_{MB}(t) > 0$ for $0 < t < \hat{t}$.

This approach was used by Magombedze et al [27]. This can be verified as follows. Suppose for example the variable z_H becomes zero for some time $\bar{t} > 0$ i. e., $z(\bar{t}) = 0$ while other variables are non negative. Then, from the z_H equation we have

$$\begin{aligned} \frac{dz_H(\bar{t})}{dt} &= \gamma_1 + a_5 y_H z_H - b_4 z_H \\ &= \gamma_1 > 0 \end{aligned} \quad (2.5)$$

which is a contradiction. For the latter case, from y_H equation we have

$$\begin{aligned} \frac{dy_H(\hat{t})}{dt} &= \zeta_1 y_H + (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H - a_1 y_H - (b_1 + a_2) y_H z_H - (1 - \pi_1) \frac{\tau \eta b_3 y_M}{\Lambda} \\ &= (1 - \pi_1) \frac{\tau \eta b_3 y_M}{\Lambda} \geq 0 \end{aligned} \quad (2.6)$$

This is true if $y_M(\hat{t}) = 0$ or $y_M(\hat{t}) > 0$ and it follows that the solution $z_H(\hat{t}) \geq 0$ and $y_H(\hat{t}) \geq 0$ are positive at any given time. Similarly, it can be shown that the variables $x(t), y_M(t), z_M(t), y_{HM}(t), B(t), C(t)$ and $z_{MB}(t)$ remain positive for all $t > 0$. Thus the solutions of the system of equation (2.3) remains non negative for all time $t > 0$.

2.5 Mathematical analysis of the HBV and Malaria sub model

2.5.1 HBV-only sub model

We begin by analyzing the HBV only sub model (obtained by setting $y_M = z_M = y_{HM} = B = C = z_{MB} = 0$) in equation (2.3) given by

$$\left. \begin{aligned} \frac{dx}{dt} &= \psi + b_1 y_H z_H - a_1 x - (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H \\ \frac{dy_H}{dt} &= \zeta_1 y_H + (1 - \pi) \left[\frac{\beta a_3}{\mu} + \sigma \right] x y_H - a_1 y_H - (a_2 + b_1) y_H z_H \\ \frac{dz_H}{dt} &= \gamma_1 + a_5 y_H z_H - b_4 z_H \end{aligned} \right\} \quad (2.7)$$

2.5.2 Positivity and boundedness of solutions of HBV-only sub model

To validate our model, we show that all cells and HBV contractions are non-negative and bounded.

Theorem 2.2: All solutions of system (2.7) starting from positive initial value (x_0, y_{H0}, z_{H0}) remain bounded and positive for all $t > 0$. Moreover, we have

$$\begin{aligned} (i) \quad & H(t) \leq H(0) + \frac{\varphi}{a_1} \\ (ii) \quad & z_H(t) \leq z_{H0} + \frac{\gamma_1}{b_4} + \frac{a_5}{a_2 + b_1} \left[\frac{\psi}{a_1} + x_0 + y_{H0} + \Omega_1 \|x\|_\infty + \Omega_2 \|y_H\|_\infty \right], \end{aligned}$$

where $H = x + y_H$ that represents the total hepatocytes $\xi = \min(a_1, a_2)\Omega_1 = \max\left\{0, 1 - \frac{a_1}{b_4}\right\}$ and $\Omega_2 = \max\left\{0, 1 - \frac{a_2}{b_4}\right\}$.

Proof: we first show that any solution starting in the $\mathbb{R}_+^3 = \{(x, y_H, z_H) \in \mathbb{R}_+^3 : x \geq 0, y_H \geq 0, z_H \geq 0\}$ stays in $\mathbb{R}_+^3$. In fact, for $(x(t), y_H(t), z_H(t)) \in \mathbb{R}_+^3$, we have

$$\left. \begin{aligned} \frac{dx}{dt} \Big|_{x=0} &= \psi + b_1 y_H z_H \geq 0 \\ \frac{dy_H}{dt} \Big|_{y_H=0} &= f(x, 0) y_H \geq 0 \\ \frac{dz_H}{dt} \Big|_{z_H=0} &= \gamma_1 \geq 0 \end{aligned} \right\} \quad (2.8)$$

This instantly showed that all solutions of system (2.7) with initial conditions $(x_0, y_{H0}, z_{H0}) \in \mathbb{R}_+^3$ stay in $\mathbb{R}_+^3$. We now show the boundedness of solutions. Let $\frac{dH}{dt} = \psi - a_1 H$. Then,

$$H(t) \leq H(0)e^{-a_1 t} + \frac{\varphi}{a_1} (1 - e^{-a_1 t}) \quad (2.9)$$

Since $0 \leq e^{-a_1 t} \leq 1$ and $1 - e^{-a_1 t} \leq 1$ we have

From the third equation of (2.7), we have that

$$\frac{dz_H}{dt} + b_4 z_H \leq \gamma_1 + a_5 y_H z_H \quad (2.10)$$

Hence

$$\frac{dz_H}{dt} + b_4 z_H \leq \gamma_1 + \frac{a_5}{a_2 + b_1} [\psi - \left(\frac{dx}{dt} + a_1 x \right) - \left(\frac{dy_H}{dt} + a_1 y_H \right)] \quad (2.11)$$

$$\begin{aligned} \frac{dz_H}{dt} e^{b_4 t} - z_{H0} &\leq \left(\frac{\gamma_1}{b_4} + \frac{a_5 \psi}{b_4(a_2 + b_1)} \right) (e^{b_4 t} - 1) - \frac{a_5}{a_2 + b_1} \int_0^t e^{(b_4 - a_1)\theta} \frac{d}{d\theta} (x(\theta) e^{a_1 \theta}) d\theta \\ &\quad - \frac{a_5}{a_2 + b_1} \int_0^t e^{(b_4 - a_1)\theta} \frac{d}{d\theta} (y_H(\theta) e^{a_1 \theta}) d\theta \end{aligned} \quad (2.12)$$

Integrating by parts, we have

$$\left. \begin{aligned} \int_0^t e^{(b_4 - a_1)\theta} \frac{d}{d\theta} (x(\theta) e^{a_1 \theta}) d\theta &= [x(\theta) e^{b_4 \theta}]_0^t - (b_4 - a_1) \int_0^t x(\theta) e^{b_4 \theta} d\theta \\ \int_0^t e^{(b_4 - a_1)\theta} \frac{d}{d\theta} (y_H(\theta) e^{a_1 \theta}) d\theta &= [y_H(\theta) e^{b_4 \theta}]_0^t - (b_4 - a_1) \int_0^t y_H(\theta) e^{b_4 \theta} d\theta \end{aligned} \right\} \quad (2.13)$$

Thus,

$$\begin{aligned} z(t) &\leq \left[\frac{a_5}{a_2 + b_1} (x_0 + y_{H0}) + z_{H0} \right] e^{-b_4 t} + \left(\frac{\gamma_1}{b_4} + \frac{a_5 \psi}{b_4(a_2 + b_1)} \right) (1 - e^{b_4(t)}) \\ &\quad + \frac{a_5}{a_2 + b_1} \left[\int_0^t [(b_4 - a_1)x(\theta) + (b_4 - a_1)y_H(\theta)] e^{b_4(\theta - t)} d\theta - x(t) \right. \\ &\quad \left. - y_H(t) \right] \end{aligned} \quad (2.14)$$

Next is to find an upper bound to this integral and it is done by studying the following cases:

if $b_4 - a_1 \leq 0$ and $b_4 - a_2 \leq 0$, then

$$z_H(t) \leq z_{H0} + \frac{\gamma_1}{b_4} + \frac{a_5}{a_2 + b_1} \left(\frac{\psi}{b_4} + x_0 + y_{H0} \right) \quad (2.15)$$

If $b_4 - a_1 \leq 0$ and $b_4 - a_2 \leq 0$, then

$$z_H(t) \leq z_{H0} + \frac{\gamma_1}{b_4} + \frac{a_5}{a_2 + b_1} \left(\frac{\psi}{b_4} + x_0 + y_{H0} + \left(1 - \frac{a_2}{b_4} \right) \|y_H\|_\infty \right) \quad (2.16)$$

If $b_4 - a_1 \leq 0$ and $b_4 - a_2 \leq 0$, then

$$z_H(t) \leq z_{H0} + \frac{\gamma_1}{b_4} + \frac{a_5}{a_2 + b_1} \left(\frac{\psi}{b_4} + x_0 + y_{H0} + \left(1 - \frac{a_1}{b_4} \right) \|x\|_\infty \right) \quad (2.17)$$

If $b_4 - a_1 \leq 0$ and $b_4 - a_2 \leq 0$, then

$$z_H(t) \leq z_{H0} + \frac{\gamma_1}{b_4} + \frac{a_5}{a_2 + b_1} \left(\frac{\psi}{b_4} + x_0 + y_{H0} + \left(1 - \frac{a_1}{b_4}\right) \|x\|_\infty + \left(1 - \frac{a_2}{b_4}\right) \|y_H\|_\infty \right) \quad (2.18)$$

From (2.15) to (2.18), we can conclude (ii), that is,

$$z_H(t) \leq z_{H0} + \frac{\gamma_1}{b_4} + \frac{a_5}{a_2 + b_1} \left[\frac{\psi}{a_1} + x_0 + y_{H0} + \Omega_1 \|x\|_\infty + \Omega_2 \|y_H\|_\infty \right]$$

2.5.3 Basic reproduction number and stability of equilibrium

The disease free and endemic equilibria are obtained by setting the rates of change or the left hand side of the equations of system (2.7) to zero and also set the infected class to zero and solving the resulting systems at steady state, we have the disease free equilibrium DFE as

$$E_H = \left(\frac{\psi}{a_1}, 0, \frac{\gamma_1}{b_4} \right) \quad (2.19)$$

Then we define the basic reproduction number of system (2.7) as follows

$$R_{0H} = \left[\frac{\psi(1 - \pi)(\beta a_3 + \sigma\mu) + \mu a_1 \zeta_1}{\mu a_1} \right] \left[\frac{b_4}{\gamma_1(a_2 + b_1) + a_1 b_4} \right] \quad (2.20)$$

Which can be rewritten as

$$R_{0H} = \frac{b_4(\psi(1 - \pi)(\mu\sigma + \beta a_3) + \mu a_1 \zeta_1)}{\mu a_1[\gamma_1(a_2 + b_1) + a_1 b_4]} \quad (2.21)$$

Expressing the endemic equilibrium point explicitly in HBV only, the parameters of the model is mathematically tractable. The components of the endemic equilibrium point (x^*, y_H^*, z_H^*) are related through the following equations:

$$\left. \begin{aligned} x^* &= \frac{[(a_2 + b_1)\gamma_1 + a_1(b_4 - a_5 y_H^*) - \mu_1(b_4 - a_5)y_H^*]\mu}{(b_4 - a_5 y_H^*)(1 - \pi)(\beta a_3 + r\mu)} \\ z^* &= \frac{\gamma_1}{(b_4 - a_5 y_H^*)} \\ &\text{and} \\ &+ [(1 - \pi)(\beta a_3 + \sigma\mu)[\gamma_1(b_1 - (a_5 + b_1)) - b_4(a_1 - \zeta_1) - \psi a_5] - \mu a_1 a_5(a_1 - \zeta_1)] y_H^* \\ &+ \varphi b_4(1 - \pi)(\beta a_3 + \sigma\mu) - \gamma_1 a_1 \mu(a_2 + b_1) - \mu a_1^2 b_4 + a_1 b_4 \mu \zeta_1 = 0 \end{aligned} \right\} \quad (2.22)$$

The third equation of (2.22) can be written as

$$P_2 y_H^{*2} + P_1 y_H^* + P_0 = 0 \quad (2.23)$$

were

$$\left. \begin{aligned} P_2 &= a_5(1 - \pi)(\beta a_3 + \sigma\mu)[a_1 - \zeta_1] \\ P_1 &= (1 - \pi)(\beta a_3 + \sigma\mu)[\gamma_1(b_1 - (a_5 + b_1)) - b_4(a_1 - \zeta_1) - \psi a_5] - \mu a_1 a_5(a_1 - \zeta_1) \\ P_0 &= \frac{\psi}{\mu a_1}(1 - \pi)(\beta a_3 + \sigma\mu) + \zeta_1 - \frac{\gamma_1}{b_4}(a_2 + b_1) - a_1 \end{aligned} \right\} \quad (2.24)$$

Also P_0 can be rewritten in the form

$$\frac{b_4(\psi(1 - \pi)(\mu\sigma + \beta a_3) + \mu a_1 \zeta_1)}{\mu a_1[\gamma_1(a_2 + b_1) + a_1 b_4]} - 1 \Rightarrow R_H - 1 \quad (2.25)$$

From system (2.25) we observe that $P_0 > 0$ whenever $R_H > 1$ and $P_0 < 0$ whenever $R_H < 1$.

There will always be stability with respect to perturbations in x, y_H, z_H . The equation $\zeta_1 + \frac{\varphi}{\mu a_1}(1 - \pi)(\beta a_3 + \sigma\mu)$ represents the ability of HBV. ζ_1 Represents the multiplication ability of infected hepatolytes, a_1 represents the death rate of both infected and uninfected hepatolytes, a_3 represents the production ability of HBV from infected hepatocytes, β represents the infectivity ability of HBV, μ represents the death rate of HBV, ψ represents the production ability (recruitment) of uninfected hepatolytes and σ represents infectivity rate of Hepatocytes by free virus, while $1 - \pi$ represents the probability of not effectively treating HBV infection.

Also, the equation $\frac{\gamma_1}{b_4}(a_2 - b_1) - a_1$ represents the ability of the immune system, b_1 represents the ability of CTL to cure HBV infection without destroying the infected hepatolytes (non lytic ability) and a_2 represents the ability of CTL to destroy both the HBV and the infected hepatolytes (lytic ability). Also $\frac{\gamma_1}{b_4}$ is the initial value of CTL while a_1 represents the death rate both infected and uninfected hepatolytes. If $\zeta_1 + \frac{\varphi}{a_1}(1 - \pi)(\beta a_3 + r\mu) < a_1 + \frac{\gamma_1}{b_4}(a_2 + b_1)$ then the immune system is strong enough to control the infection. But if $\zeta_1 + \frac{\varphi}{a_1}(1 - \pi)(\beta a_3 + \sigma\mu) > a_1 + \frac{\gamma_1}{b_4}(a_2 + b_1)$ then, there will always be instability. In other words, HBV can invade the hepatocyte and exist for a long time. Likewise, if $\pi \geq 1$, then the treatment option will be able to control HBV infection and if $\pi < 1$, then the infection will persist and may lead to chronic infection.

2.5.3 Malaria - only sub model

Here we consider the malaria only model (obtained by setting $y_H = z_H = y_{HM} = 0$) in system (2.7) and we have

$$\left. \begin{aligned} \frac{dx}{dt} &= \psi + b_2 y_M z_M - a_1 x - (1 - \pi_1) \frac{\eta b_3 x y_M}{\Lambda} \\ \frac{dy_M}{dt} &= \zeta_2 y_M + (1 - \pi_1) \frac{\eta b_3 x y_M}{\Lambda} - (b_2 + a_6) y_M z_M - a_1 y_M - k y_M \\ \frac{dz_M}{dt} &= \gamma_2 + a_4 y_M z_M - b_4 z_M \\ \frac{dB}{dt} &= \varphi + d_1 C z_{MB} - k_1 B - (1 - \pi_2) \frac{\alpha B k C}{k_2} \\ \frac{dC}{dt} &= (1 - \pi_2) \frac{\alpha k B C}{k_2} + d_3 C - k_1 C - (d_1 + d_2) C z_{MB} \\ \frac{dz_{MB}}{dt} &= \gamma_3 + d_4 C z_{MB} - b_4 z_{MB} \end{aligned} \right\} \quad (2.26)$$

Note that the malarial model is an extension of the model by Chinebu et al, [12], and it is necessary we point out that since the hepatocyte population is free of HBV, that is, there is no HBV infected hepatocyte that can transmit HBV infection to susceptible hepatocytes.

2.5.4 Disease free equilibrium and basic reproduction numbers

The malaria only sub-model (2.26) has a disease free equilibrium (DFE), given by

$$E_M = (x, y_M, z_M, B, C, z_{MB}) = \left(\frac{\psi}{a_1}, 0, \frac{\gamma_2}{b_4}, \frac{\varphi}{k_1}, 0, \frac{\gamma_3}{b_4} \right) \quad (2.27)$$

The basic Reproduction number, that is, the linear stability of E_M can be established using the next generation operator [42] on the system (2.26). We thus have

$$R_{0M} = \left[\frac{\Lambda a_1 \zeta_2 + (1 - \pi_1) \eta b_3 \psi}{\Lambda a_1} \right] \left[\frac{b_4}{\gamma_2 (b_2 + a_6) + b_4 (a_1 + k)} \right] + \left[\frac{d_3 k_1 k_2 + (1 - \pi_2) \alpha k \varphi}{k_1 k_2} \right] \left[\frac{b_4}{\gamma_3 (d_1 + d_2) + b_4 k_1} \right] \quad (2.28)$$

which can further be simplified into

$$R_{0M} = \frac{b_4 (\Lambda a_1 \zeta_2 + (1 - \pi_1) \eta b_3 \psi)}{\Lambda a_1 [\gamma_2 (b_2 + a_6) + b_4 (a_1 + k)]} + \frac{b_4 (d_3 k_1 k_2 + (1 - \pi_2) \alpha k \varphi)}{k_1 k_2 [\gamma_3 (d_1 + d_2) + b_4 k_1]} \quad (2.29)$$

From system (2.29), $\frac{\psi \eta b_3 (1 - \pi_1)}{\Lambda a_1} + \zeta_2$ is the multiplication ability of sporozoite (malaria infection) on the hepatocytes and the ability that the hepatocytes will produce sufficient merozoite that will eventually infect the erythrocytes. $(b_2 + a_6) \frac{\gamma_2}{b_4} - a_1 + k$ is the ability of the immune system in controlling sporozoites in the hepatocytes before merozoite are released to invade the erythrocytes,

$(1 - \pi_3) \frac{\alpha k \varphi}{k_1 k_2} + d_3$ is the multiplication ability of merozoites in the erythrocytes and the probability that the erythrocytes will produce sufficient gametocytes that will be ingested by mosquitoes during blood meal, $(d_1 + d_2) \frac{\gamma_3}{b_4} + k_1$ is the ability of the immune system in controlling the infection at the RBC stage. With this analysis, we have the basic reproduction number R_{0M} as

$$R_{0M} = R_{0M_H} + R_{0M_R} \quad (2.30)$$

where

$$R_{0M_H} = \frac{b_4(\Lambda a_1 \zeta_2 + (1 - \pi_1) \eta b_3 \psi)}{\Lambda a_1 [\gamma_2(b_2 + a_6) + b_4(a_1 + k)]} \quad (2.31)$$

$$R_{0M_R} = \frac{b_4(d_3 k_1 k_2 + (1 - \pi_2) \alpha k \varphi)}{k_1 k_2 [\gamma_3(d_1 + d_2) + b_4 k_1]} \quad (2.32)$$

Therefore, R_{0M_H} describes the expected member of hepatocytes that will be infected by one infectious hepatocyte where as R_{0M_R} describes the average number of infected erythrocytes by one infectious erythrocyte.

2.5.5 Local stability of equilibria

In this section, we describe the local stability of both equilibria of malaria only sub model system (2.26). Note that the Jacobian matrix of (2.26) is given by

$$J := \begin{pmatrix} -a_1 & A & 0 & 0 & 0 & 0 \\ 0 & D & 0 & 0 & 0 & 0 \\ 0 & a_4 \cdot z_M & -b_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k & G & 0 \\ 0 & 0 & 0 & 0 & Q & 0 \\ 0 & 0 & 0 & 0 & d_4 \cdot z_{MB} & -b_4 \end{pmatrix}$$

$$(2.33)$$

where,

$$A = b_2 z_m - (1 - \pi_1) \frac{\eta b_3 x}{\Lambda}, \quad D = \varsigma_1 + \frac{(1 - \pi_1) \eta b_3 X}{\Lambda} - (b_2 + a_6) z_M - a_1 - k$$

$$G = d_1 z_{MB} - (1 - \pi_1) \frac{\alpha k B}{k_2} \text{ and } Q = (1 - \pi) \frac{\alpha k B}{k_2} + d_3 - k_1 - (d_1 + d_2) z_{MB}$$

First, we get the following results.

Theorem 2.3: The infection free equilibrium E_M is locally asymptotically stable if $R_{0M} < 1$ and unstable if $R_{0M} > 1$.

Proof: Evaluating system(2.33) at E_M , we obtain

$$J(E_M) := \begin{pmatrix} -a_1 & A_1 & 0 & 0 & 0 & 0 \\ 0 & D_1 & 0 & 0 & 0 & 0 \\ 0 & \frac{a_4 \gamma_2}{b_4} & -b_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k & G_1 & 0 \\ 0 & 0 & 0 & 0 & Q_1 & 0 \\ 0 & 0 & 0 & 0 & \frac{d_4 \gamma_3}{b_4} & -b_4 \end{pmatrix} \quad (2.34)$$

where

$$A_1 = \frac{b_2 \gamma_2}{b_4} - \frac{(1 - \pi_1) \eta b_3 \psi}{\Lambda a_1}, \quad D_1 = \zeta_2 + \frac{(1 - \pi_1) \eta b_3 \varphi}{\Lambda a_1} - (b_2 + a_6) \frac{\gamma_2}{b_4} - a_1 - k$$

$$G_1 = \frac{d_1 \gamma_3}{b_4} + \frac{(1 - \pi_2) \alpha k \psi}{k_1 k_2} \quad \text{and} \quad Q_1 = \frac{(1 - \pi) \alpha k \psi}{k_1 k_2} + d_3 - k_1 - (d_1 + d_2) \frac{\gamma_3}{b_4}$$

Then the characteristics equation at E_M is given by

$$(\lambda_4 + a_1)(\lambda_5 + D_1)(\lambda_6 + b_4)(\lambda_7 + k)(\lambda_8 + Q_1)(\lambda_9 + b_4) = 0 \quad (35)$$

Hence, the roots of (2.35) are $\lambda_4 = -a_1$, $\lambda_5 = R_{0M_H} - 1$, $\lambda_6 = -b_4$, $\lambda_7 = -k$, $\lambda_8 = R_{0M_R} - 1$ and $\lambda_9 = -b_4$. Clearly λ_4 , λ_6 , λ_7 and λ_9 are negative. However, λ_5 and λ_8 are respectively negative if $R_{0M_H} < 1$ and $R_{0M_R} < 1$ and respectively positive if $R_{0M_H} > 1$ and $R_{0M_R} > 1$. Therefore, E_M is locally asymptotically stable if $R_{0M_H} < 1$ and $R_{0M_R} < 1$ and unstable if $R_{0M_H} > 1$ and $R_{0M_R} > 1$.

Next, we focus on the local stability of the endemic equilibrium E_M^* . This is the point where the disease cannot be totally eradicated but remains in the population of both the hepatocytes and erythrocytes.

Theorem 2.4: The endemic equilibrium E_M^* is locally asymptotically stable if $R_{0M} > 1$ i.e $R_{0MH} > 1$ and $R_{0MR} > 1$.

Proof: To calculate the endemic equilibrium point, we have

$$E_M^* = (X^*, y_m^*, Z_m^*, B^*, C^*, Z_{MB}^*) \neq 0$$

From equation (2.27) we have

$$\left. \begin{aligned} x^* &= \frac{\Lambda[(b_2 + a_6)\gamma_2 + (b_4 - a_4 y_M^*)(a_1 + k - \zeta_2)]}{\eta b_3(1 - \pi_1)(b_4 - a_4 y_M^*)} \\ z_m^* &= \frac{\gamma_2}{b_4 - a_1 y_M^*} \\ B^* &= \frac{k_2[(d_1 + d_2)\gamma_3 + (k_1 - d_3)(b_4 - d_4 C^*)]}{\alpha k(1 - \pi_3)(b_4 - d_4 C^*)} \\ z_{MB}^* &= \frac{\gamma_3}{b_4 - d_4 C^*} \end{aligned} \right\}$$

y_M^* is obtained as

$$\left. \begin{aligned} &\eta b_3 a_4 (1 - \pi_1)(a_1 + k - \zeta_2) y_M^{*2} \\ &+ \left[b_2 b_3 \gamma_3 \eta + \Lambda a_1 a_4 (a_1 + k - \zeta_2) - \psi \eta a_4 b_3 (1 - \pi_1) - \eta b_3 \gamma_2 (1 - \pi_1)(b_4 + a_6) \right. \\ &\quad \left. - \eta b_3 b_4 (1 - \pi_1)(a_1 + k - \zeta_2) \right] y_M^* \\ &\quad + b_4 \psi \eta b_3 (1 - \pi_1) - \Lambda a_1 [\gamma_2 (b_4 + a_6) + b_4 (a_1 + k - \zeta_2)] = 0 \end{aligned} \right\} \quad (2.36)$$

While C^* is obtained as

$$\left. \begin{aligned} &d_4 \alpha k (1 - \pi_2)(k_1 - d_3) C^{*2} \\ &+ \left[d_1 \gamma_3 \alpha k (1 - \pi_2) + k_1 k_2 d_4 (k_1 - d_3) - b_4 \alpha k (1 - \pi_2)(k_1 - d_3) \right. \\ &\quad \left. - k_1 k_2 b_4 (k_1 - d_3) - \varphi \alpha k d_4 (1 - \pi_2) \right. \\ &\quad \left. + \varphi \alpha k b_4 (1 - \pi_2) - k_1 k_2 [\gamma_3 (d_1 + d_2) + b_4 (k_1 - d_3)] \right] C^* = 0 \end{aligned} \right\} \quad (2.37)$$

Equations (2.36) and (2.37) can be rewritten as

$$P_{MH_2} y_M^{*2} + P_{MH_1} y_M^* + P_{MH_0} = 0 \quad (2.38)$$

and

$$P_{MR_2} C_M^{*2} + P_{MR_1} C^* + P_{MR_0} = 0 \quad (2.39)$$

where

$$\left. \begin{aligned} P_{MH_2} &= \eta b_3 a_4 (1 - \pi_1)(a_1 + k - \zeta_2) \\ P_{MH_1} &= b_2 b_3 \gamma_3 \eta + \Lambda a_1 a_4 (a_1 + k - \zeta_2) - \psi \eta a_4 b_3 (1 - \pi_1) - \eta b_3 \gamma_2 (1 - \pi_1)(b_4 + a_6) \\ &\quad - \eta b_3 b_4 (1 - \pi_1)(a_1 + k - \zeta_2) \\ P_{MH_0} &= R_{0MH} - 1 \end{aligned} \right\} \quad (2.40)$$

and

$$\left. \begin{aligned} P_{MR_2} &= d_4 \alpha k (1 - \pi_2) (k_1 - d_3) \\ P_{MR_1} &= d_1 \gamma_3 \alpha k (1 - \pi_2) + k_1 k_2 d_4 (k_1 - d_3) - b_4 \alpha k (1 - \pi_2) (k_1 - d_3) - \alpha k \gamma_3 (1 - \pi_2) (d_1 + d_2) \\ &\quad - k_1 k_2 d_4 (k_1 - d_3) - \varphi \alpha k d_4 (1 - \pi_2) \\ P_{MR_0} &= R_{0MR} - 1 \end{aligned} \right\} \quad (2.41)$$

It is worth noting that the coefficient P_{MH_0} and P_{MR_0} are always positive if and only if $R_{0MH} > 1$ and $R_{0MR} > 1$ respectively and negative if and only if $R_{0MH} < 1$ and $R_{0MR} < 1$ respectively.

2.6 Analysis of the full HBV-Malaria model

Having analysed the dynamics of the two sub-models that is HBV only and malaria only models, The full HBV-malaria model (2.3) is now considered.

2.6.1 Local Stability of the Disease Free Equilibrium

The HBV-malaria model (2.3) has a DFE given by

$$E_{0HM} = (x_0, 0, Z_{H0}, 0, Z_{M0}, 0, B_0, 0, Z_{MB0})$$

where $x_0 = \frac{\psi}{a_1}$, $Z_{H0} = \frac{y_1}{b_4}$, $Z_{M0} = \frac{y_2}{b_4}$, $B_0 = \frac{\varphi}{k_1}$ and $Z_{MB0} = \frac{y_3}{b_4}$

The spectra radii of the next generation matrix FV^{-1} are independent and given in terms of HBV and malaria – only associated reproduction members, in which that of the malaria comprises the Hepatocytes and erythrocytes stages. It is therefore evident that the associated reproduction member for the full HBV – malaria model is given by

$$R_{0HM} = \text{Max}\{R_{0H}, R_{0M}\}, \quad (2.42)$$

where

$$R_{0H} = \frac{b_4(\psi(1 - \pi)(\mu\sigma + \beta a_3) + \mu a_1 \zeta_1)}{\mu a_1[\gamma_1(a_2 + b_1) + a_1 b_4]}$$

$$R_{0M} = \frac{b_4(\Lambda a_1 \zeta_2 + (1 - \pi_1)\eta b_3 \psi)}{\Lambda a_1[\gamma_2(b_2 + a_6) + b_4(a_1 + k)]} + \frac{b_4(d_3 k_1 k_2 + (1 - \pi_2)\alpha k \varphi)}{k_1 k_2[\gamma_3(d_1 + d_2) + b_4 k_1]}$$

R_{0H} Gives the number of secondary HBV infection cases produced by a HBV infectious hepatocytes during its effective infectious period when introduced in a population of mostly HBV susceptible. R_{0M} is the malaria reproduction number in the absence of HBV which gives the number of secondary malaria infections produced by a malaria infectious cell (either hepatocytes and/or erythrocytes) but not infected with HBV, during its infectious period when introduced in a population of malaria susceptible hepatocyte and/or erythrocytes that have no HBV.

A threshold condition for endemicity is given by $R_{0HM} = 1$: The disease dies out if $R_{0HM} < 1$, and becomes endemic if $R_{0HM} > 1$. Then, we can claim the following result.

Theorem 2.5: The disease free equilibrium with treatment effects of HBV – malaria infection exists and is locally asymptotically stable if $R_{0H\pi} < 1$, $R_{0MH\pi_1} < 1$ and $R_{0MR\pi_2} < 1$ and unstable if $R_{0H\pi} > 1$, $R_{0MH\pi_1} > 1$ and $R_{0MR\pi_2} > 1$.

Proof: A partial differentiation of $R_{0H\pi}$, $R_{0MH\pi_1}$ and $R_{0MR\pi_2}$ with respect to π , π_1 and π_2 respectively, and obtain:

$$\begin{aligned}\frac{dR_{0H\pi}}{d\pi} &= -\frac{\psi b_4(\mu\sigma + \beta a_3)}{\mu a_1[\gamma_1(a_2 + b_1) + a_1 b_4]} < 0 \\ \frac{dR_{0MH\pi_1}}{d\pi_1} &= -\frac{b_4 \eta b_3 \psi}{\Lambda a_1[\gamma_2(b_2 + a_6) + b_4(a_1 + k)]} < 0 \\ \frac{dR_{0MR\pi_2}}{d\pi_2} &= -\frac{b_4 \alpha k \varphi}{k_1 k_2[\gamma_3(d_1 + d_2) + b_4 k_1]} < 0\end{aligned}$$

This means that the values of $R_{0H\pi}$, $R_{0MH\pi_1}$ and $R_{0MR\pi_2}$ decrease as π , π_1 and π_2 increase. $R_{0H\pi}, R_{0MH\pi_1}, R_{0MR\pi_2} < 1 < R_{0HM}$. Whenever, the partial derivatives $\frac{dR_{0H\pi}}{d\pi}$, $\frac{dR_{0MH\pi_1}}{d\pi_1}$ and $\frac{dR_{0MR\pi_2}}{d\pi_2}$ are less than zero, it is also evident that treatment is effective as π , π_1 and π_2 approach 1. Therefore, a highly effective drug administered to infected individuals in the asymptomatic stage can lead to effective disease control if $\lim_{\pi, \pi_1, \pi_2 \rightarrow 1} R_{0H\pi}, R_{0MH\pi_1}, R_{0MR\pi_2} < 1$.

3 Numerical results

Numerical simulation of the intracellular co-infection model of HBV and malaria were conducted having the initial conditions for the state variables as: $x = 1$, $y_H = 0.01$, $z_H = 0.01$, $y_M = 0.01$, $z_M = 0.02$, $y_{HM} = 0.01$, $B = 30$, $C = 10$, and $z_{MB} = 0.02$. We use a set of logical parameter values. The basic reproduction number of HBV/malaria co-infection model when control is effective is given as $(R_{0H}, R_{0MH}, R_{0MR}) = (0.3457, 0.0213, 0.9595)$ and when control is not effective is given as $(R_{0H}, R_{0MH}, R_{0MR}) = (4.3333, 1.2041, 22.9791)$. Graphical results are displayed in figures 2 to 7 and all the parameters with their values are shown in table 3 and 4. The simulations of model systems (2.3), (2.7) and (2.26) are performed with time 180 days. The simulations were in the presence of immune response and treatment.

Table 3: Parameter Value

Parameters	Value	Parameters	Value
ψ	3	γ_3	0.25
a_1	0.02	τ	0.00000001
β	1	a_6	0.002
μ	0.58	a_4	0.41
a_3	2	b_4	0.2
σ	0.01	φ	0.83

b_3	0.181	k_1	0.0083
Λ	48	d_1	0.0035
η	0.04	b_2	0.056
a_2	5	ρ	0.00000001
b_1	1	d_4	0.5
a_5	0.1	k_2	48
γ_1	1	k	0.16
γ_2	0.3	α	0.002

Table 4: Model parameter value for control

<i>Parameters</i>	Effective Value	Control	Ineffective Control Value
ζ_1	0.004		0.4
ζ_2	0.0003		0.3
ζ_3	0.001		0.01
d_2	0.24		0.0024
d_3	0.3		0.98
π	0.98		0.75
π_1	0.98		0.05
π_2	0.98		0.05
ω	0.0004		0.4

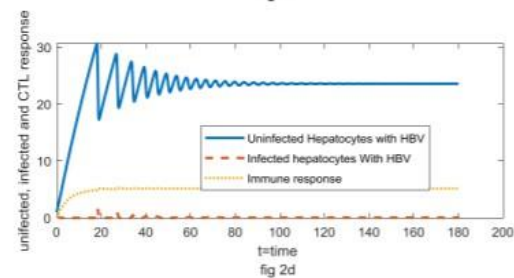
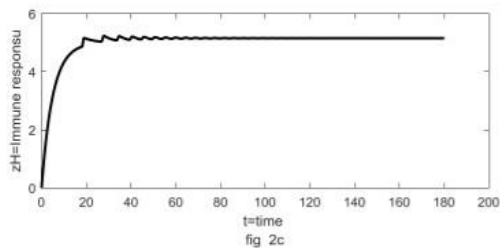
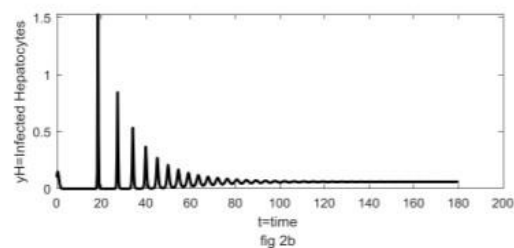
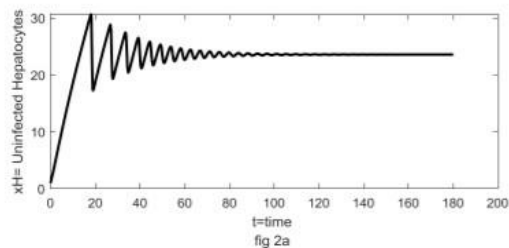
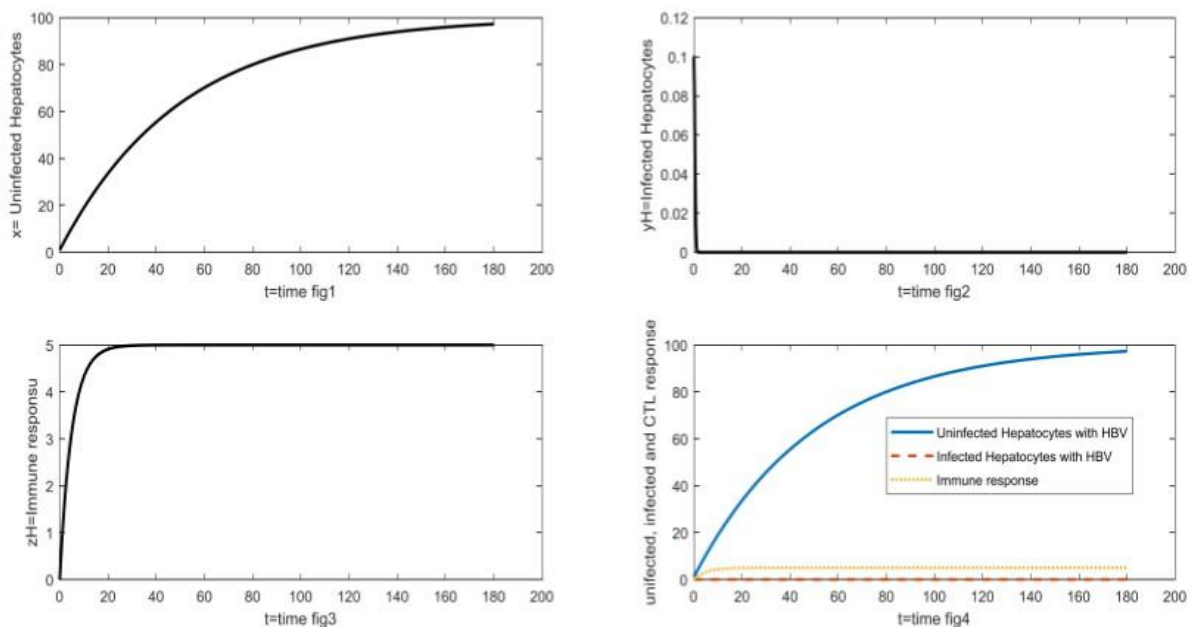
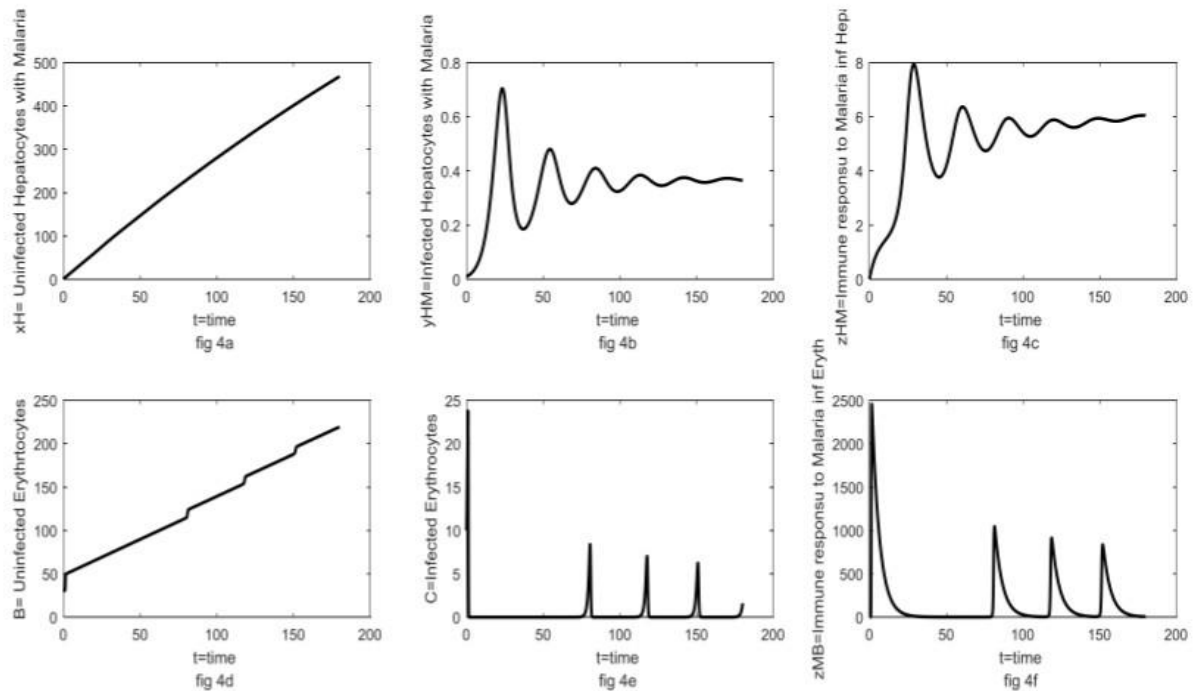


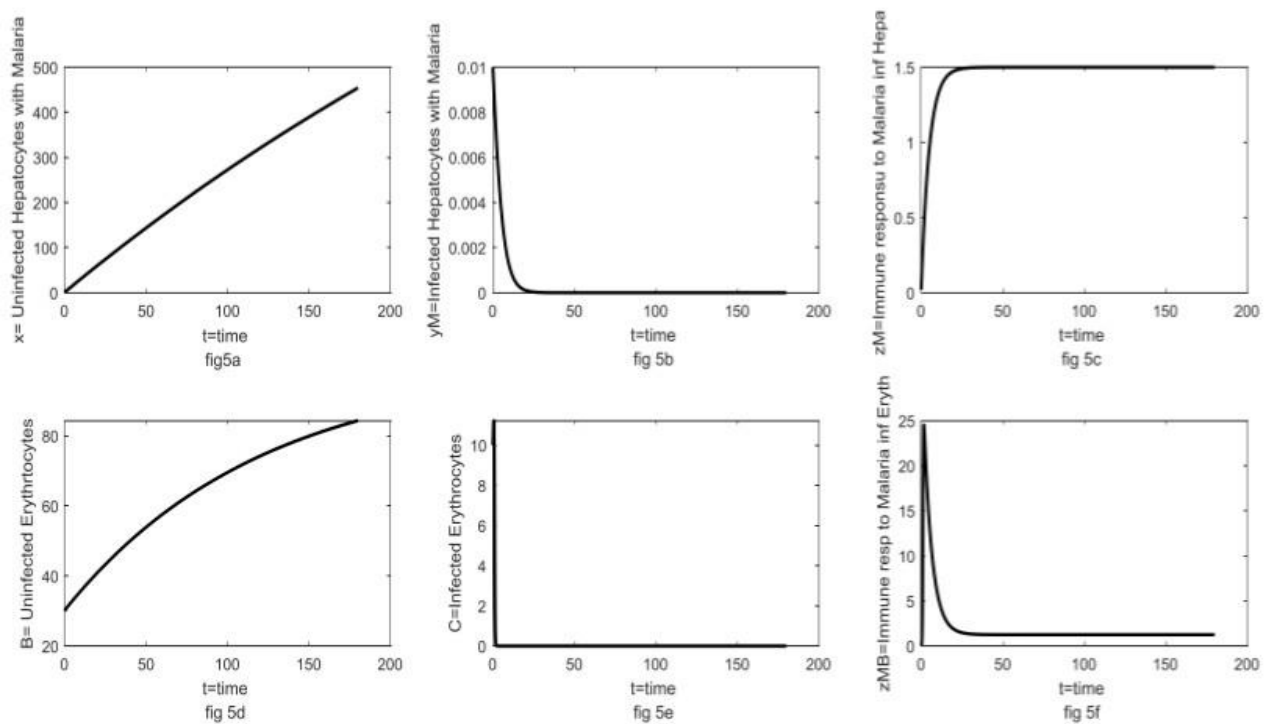
Figure 2(a-d): Simulation of HBV only model of system (2.7) where the proliferation rate of HBV infected hepatocytes is high, immune response is low and treatment rate is not effective.



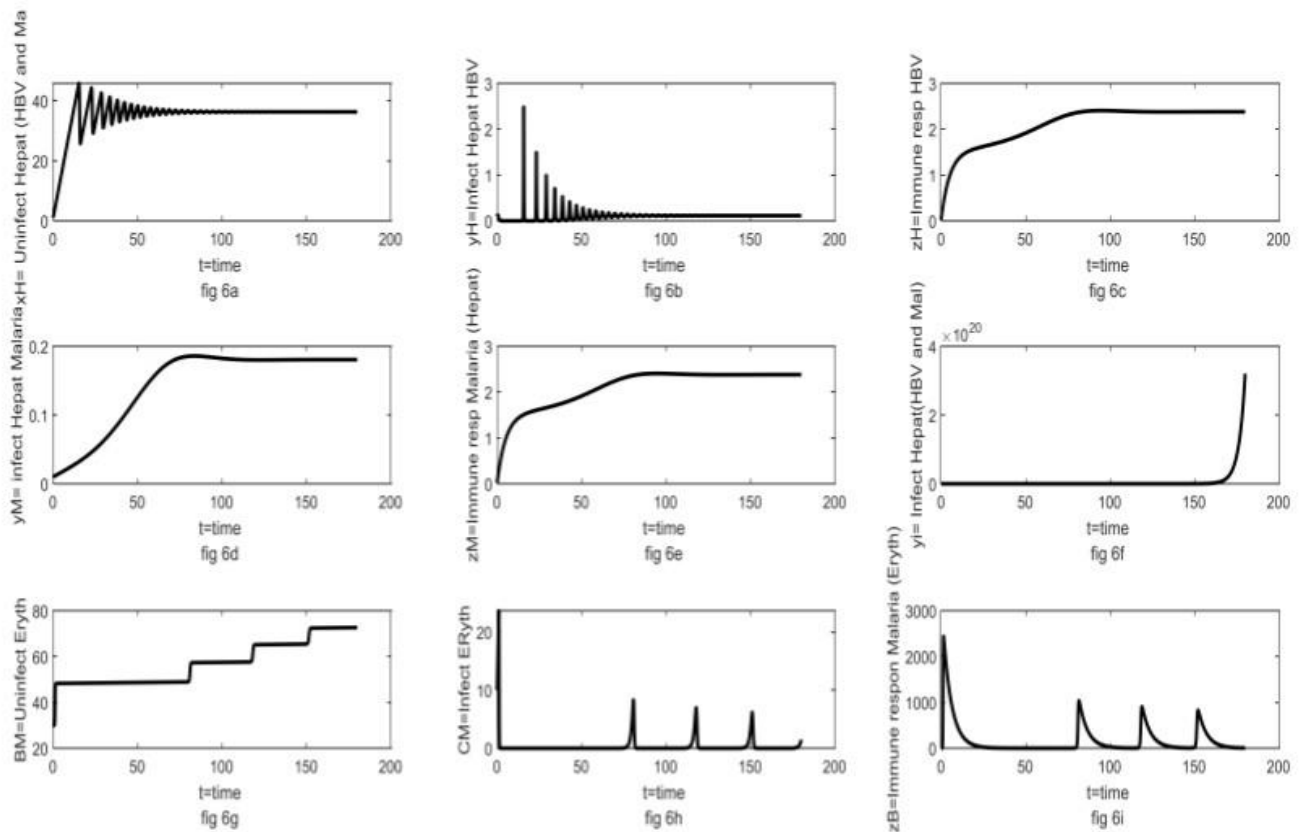
Figures 3(a – d): Simulation of HBV only model of system (2.7) where the proliferation rate of HBV infected hepatocytes is slow and weak, immune response fast and strong, while treatment rate is effective.



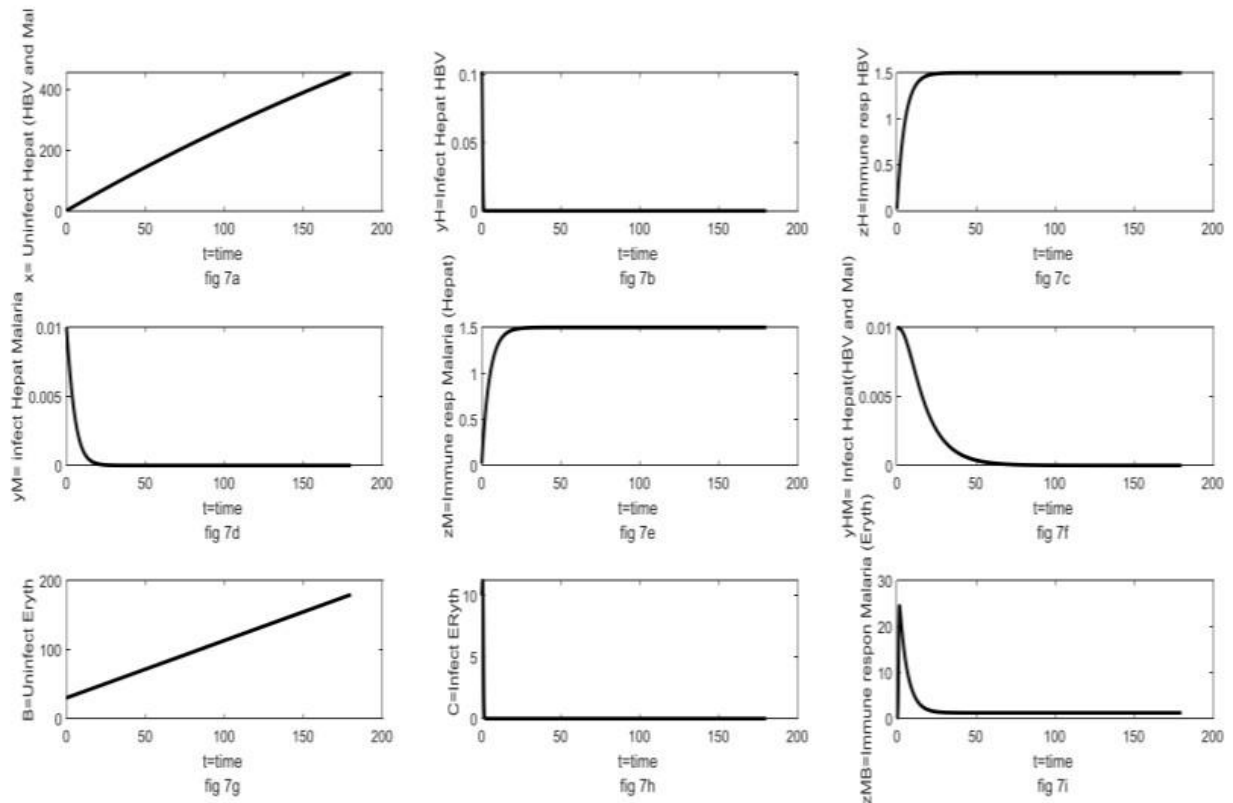
Figures 4(a – f): Simulation of malaria only model of system (2.26) where the proliferation rate of malaria infected hepatocytes and erythrocytes is high and strong, immune response to both infected hepatocytes and erythrocytes is slow and weak with ineffective treatment rate.



Figures 5(a – f): Simulation of malaria only model of system (2.26) where the proliferation rate of malaria infected hepatocytes and erythrocytes is slow and weak, immune response to both infected hepatocytes and erythrocytes is high and strong with effective treatment rate.



Figures 6(a – i): Simulation of HBV-malaria co-infection model of system (2.3) where the proliferation rate of malaria infected hepatocytes, erythrocytes and co-infected hepatocytes is strong, immune response to both infected hepatocytes, erythrocytes and co-infected hepatocytes is weak with ineffective treatment rate.



Figures 7(a – i): Simulation of HBV-malaria co-infection model of system (2.3) where the proliferation rate of malaria infected hepatocytes, erythrocytes and co-infected hepatocytes is weak, immune response to both infected hepatocytes, erythrocytes and co-infected hepatocytes is strong with effective treatment rate.

4 Discussion

From the simulation of HBV only model of system (2.7), we shall notice that HBV infection will persist in the cell population if treatment rate is not effective, immune response is slow and proliferation of HBV infected hepatocytes is high as displayed in figure 2(a-d). We observe the effect of immune response and treatment via the drastic reduction of the infected hepatocytes in figure 3(a-d). As expected, both the uninfected hepatocytes and immune response increases and this imply that with effective treatment and adequate immune response, HBV only infection can be controlled in the liver. Also, it is shown in figures 2 (a-d) that the infected hepatocytes with HBV oscillated for some time before becoming constant continually implying that the treatment and immune response are inadequate and this can lead to chronic HBV.

From the simulation of malaria only model of system (2.26) which was represented in figures 4(a-f), we observe the effect of immune response and treatment both in the hepatocytes and erythrocytes. The simulation results showed that treatment is ineffective within sufficient immune response and this is responsible for the increase in malaria infected hepatocytes and erythrocytes. Figure 5(a-f)

showed that malaria can be controlled in the hepatocytes and erythrocytes, that is, if immune response is fast and strong enough to slow the proliferation rate of both malaria infected hepatocytes and erythrocytes, and with effective treatment stop more cells from being infected. We also noticed that the level of malaria infected hepatocytes and erythrocytes were to a great extent diminished, whereas the uninfected hepatocytes and erythrocytes with malaria and immune responses increases.

Figures 6(a-i) denoted the simulation of HBV-malaria co-infection of model system (3) and it indicates that the rate of HBV-malaria co-infection in the hepatocytes continues to increase. The immune response and treatment control of HBV only infection was inadequate as the infected hepatocytes oscillated before becoming constant. But this is not the same with the immune response and treatment of malaria only infected hepatocytes because they are drastically reduced. This is why Kolawole and Kana [22] stated that individuals with HBV and malaria co-infection tend to have higher level of changes in their disease conditions compared to those with mono-infections. This simply implies that the presence of HBV and malaria co-infection in the hepatocytes and erythrocytes significantly affect the liver function and hematological parameters if not properly controlled in the cell population. Finally, figure 7(a-i) specifies that HBV and malaria co-infection can be controlled via reducing the rate at which hepatocytes are infected by both infections, rate of proliferation of co-infected hepatocytes and rate at which erythrocytes are infected with effectual treatment and fast and strong immune response.

5 Conclusion

In this paper, we considered the immune response and treatment effects hepatitis B Virus and malaria model with intracellular transmission. By the basic reproduction number R_{0H} for HBV in the hepatocytes and R_{0M} for malaria both in the hepatocytes and erythrocytes, which we respectively denoted by R_{0MH} and R_{0MR} were obtained using the next generation matrix.

In the proposed model, the modes of transmission of both infections with their processes were modeled using different functions. Firstly, the existence, positivity and boundedness of solutions of the problem that helps in guaranteeing the wellposedness of the co-infection model were proved. The locally asymptotic stability of both HBV only free and endemic equilibria and malaria only free and endemic equilibria were considered. By analyzing the effect of treatment on both HBV and malaria, we have from theorem 2.5 that the disease free equilibrium with treatment effects of HBV-malaria co-infection exists and is locally asymptotically stable if $R_{0H\pi} < 1$, $R_{0MH\pi_1} < 1$ and $R_{0MR\pi_2} < 1$ and unstable if $R_{0H\pi} > 1$, $R_{0MH\pi_1} > 1$ and $R_{0MR\pi_2} > 1$.

We observe from the analysis that if the cellular immune response for HBV only infection satisfies $\frac{\psi}{\mu a_1}(1 - \pi)(\beta a_3 + \sigma\mu) + \zeta_1 < \frac{\gamma_1}{b_4}(a_2 + b_1) + a_1$, then the immune system and treatment is strong enough to eliminate the infection. But if $\frac{\psi}{\mu a_1}(1 - \pi)(\beta a_3 + \sigma\mu) + \zeta_1 > \frac{\gamma_1}{b_4}(a_2 + b_1) + a_1$, chronic hepatitis appears because the immune system cannot clear the infection. Thus, if β and a_3 are large,

strong fast, then most of the hepatocytes will be infected with HBV and this will be eventually result to massive liver necrosis due to strong immune response.

Similarly, if the cellular immune response for HBV only infection satisfies

$\varsigma_2 + \frac{(1+\pi_1)\eta b_3 \psi}{\Lambda a_1} < (b_2 + a_6) \frac{\gamma_2}{b_4} + a_1 + k$, then the immune response and treatment is capable of stopping the multiplication of sporozoites and subsequently eradicate them from the liver thereby preventing the production of merozoites that can infect the erythrocytes. $\varsigma_2 + \frac{(1+\pi_1)\eta b_3 \psi}{\Lambda a_1} > (b_2 + a_6) \frac{\gamma_2}{b_4} + a_1 + k$, then sufficient merozoite that can infect the erythrocytes would be produced. Also, if $\frac{(1-\pi_2)ak\varphi}{k_1 k_2} + d_3 < k_1 + (d_1 + d_2) \frac{\gamma_3}{b_4}$, the immune response and treatment can control malaria in the erythrocytes. But if $\frac{(1-\pi_2)ak\varphi}{k_1 k_2} + d_3 > k_1 + (d_1 + d_2) \frac{\gamma_3}{b_4}$, then there would be maximum damage caused to the erythrocytes and maximum concentration of merozoites which can be ingested by mosquito during blood meal.

Finally, if $\max \left\{ \frac{\psi}{\mu a_1} (1 - \pi)(\beta a_3 + \sigma \mu) + \zeta_1, \varsigma_2 + \frac{(1+\pi_1)\eta b_3 \psi}{\Lambda a_1} + \frac{(1-\pi_2)ak\varphi}{k_1 k_2} + d_3 \right\} < \max \left\{ \frac{\gamma_1}{b_4} (a_2 + b_1) + a_1, (b_2 + a_6) \frac{\gamma_2}{b_4} + a_1 + k + k_1 + (d_1 + d_2) \frac{\gamma_3}{b_4} \right\}$, then HBV-Malaria co-infection can be eradicated from the hepatocytes and erythrocytes by the combined effect of immune response and treatment. But if otherwise, the HBV-malaria co-infection will persist in the hepatocyte and erythrocyte population thereby causing serious damage to the co-infected individual.

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Method of lines analysis of Dufour and Soret effects on unsteady MHD free convective heat and mass transport flow past a porous infinite plate

U.C. Ugwu^{*†}, A.T. Cole^{*}, A.I. Faruk^{*}, O.A. Adedayo^{*}, F.I. Asonibare^{*},
and M.C. Egbugha[‡]

Abstract

The problem of coupled heat and mass transfer by free convection of a chemically reacting viscous incompressible and electrically conducting fluid past an infinite vertical porous plate under the influence of uniform transverse magnetic field in the presence of diffusion-thermo (Dufour), thermal-diffusion (Soret) and internal heat source or sink is studied. A magnetic field of uniform strength is applied perpendicular to the plate and the fluid is subjected to a normal suction velocity while the heat flux at the plate is constant. The dimensionless governing equations were solved numerically in MATLAB using the Method of Lines (MOL). Some of the results indicating the influence of various thermos-physical parameters of fluid flow, heat and mass transfer characteristics were obtained and presented graphically. The numerical values of skin-friction coefficient and Nusselt number at the plate are derived, discussed numerically for various values of physical parameters and presented through tables. The presence of a generating heat source has effects on the fluid velocity, as well as on temperature.

Keywords: convective flow; heat and mass transfer; heat source; mhd; porous medium; unsteady flows; viscous dissipation.

1 Introduction

Convective heat transfer in fluid occurs in many industrial applications and is an important aspect in the study of heat transfer. If stratification occurs, the fluid temperature is function of distance and convection in such environment exists in lakes, oceans, nuclear reactors where coolant (generally liquid

^{*}Department of Mathematics, Federal University of Technology, Minna, Nigeria

[†] Corresponding Author. E-mail: clement.ugwu@futminna.edu.ng

[‡] Department of Computer Engineering, Ahmadu Bello University, Zaria, Nigeria

metals) is present in magnetic field etc. Cheesewright [1] examined the natural convection along an isothermal vertical surface in non-isothermal surroundings. Chen and Eichhorn [2] studied natural convection along an isothermal vertical plate in thermally analyzed the effect of magnetic field on natural convection in liquid metal (NaK) used as coolant in nuclear reactor. Venkatachala and Nath [17] obtained the non-similarity solution for natural convection in thermally stratified fluid. Uotani [14] experimentally studied the natural convection in thermally stratification for liquid metal (PbBi). Kulkarni et al. [5] investigated the problem of natural convection from an isothermal flat plate suspended in a linearly stratified fluid medium. Ostrach [7] presented the similarity solution of natural convection along vertical isothermal plate.

The study of magnetohydrodynamic (MHD) flows plays an important role in agriculture, engineering and petroleum industries. The problem of free convection under the influence of a magnetic field has attracted the interest of many researchers in view of its applications in geophysics and astrophysics. Ugwu *et al.* [12] investigated the problem of MHD flow of continuous Dusty particle in a Non-newtonian Darcy fluid between parallel plates. Soundalgekar et al. [9] analyzed the problem of free convection effects on Stokes problem for a vertical plate under the action of transversely applied magnetic field. Helmy [4] presented an unsteady two-dimensional laminar free convection flow of an incompressible, electrically conducting (Newtonian or polar) fluid through a porous medium bounded by an infinite vertical plane surface of a constant temperature. Zueco [18] analyzed the hydromagnetic convection past a flat plate. Ugwu *et al* [10] studied the MHD effects on convective flow of dusty viscous fluid. The problem was solved numerically using finite difference method under the influence of magnetic field. The properties of solution and uniqueness of solution was established. The effects of heat and mass transfer on free convective flow of micropolar fluid were studied over an infinite vertical porous plate in the presence of an inclined magnetic field with a constant suction velocity and taking Hall current into account have been discussed by Veera Krishna *et al.* [15] Veera Krishna *et al.* [16] discussed the heat and mass transfer on unsteady, MHD oscillatory flow of second-grade fluid through a porous medium between two vertical plates under the influence of fluctuating heat source/sink, and chemical reaction. Ugwu *et al* [11] studied the effects of MHD flow on convective fluids incorporating viscous dissipation energy though it was a Newtonian fluid. This problem was analysed numerically using method of lines and various fluid parameter and that of the particles were obtained.

In recent times, many researchers have given considerable interest to the study of MHD in the presence of radiation. Mishra *et al.* [6] studied the effect of radiation and non-uniform heat source on the unstable, MHD viscous fluid through a heated plate with time-dependent suction and viscous dissipation. Chiranjeevi *et al.* [3] investigated the problem of the MHD boundary layer flow analysis in the presence of thermal absorption with heat generation and chemical reaction over a plate. Sadiq and Nagarathna [8] studied the heat and mass transport on MHD Free convective flow through a porous medium past and infinite plate with time dependent permeability neglecting viscous dissipation. This was done analytically.

Ugwu *et al.* [13] investigated the unsteady free convective flow of a viscous incompressible electrically conducting fluid over an infinite vertical porous plate under the influence of uniform

transverse magnetic field with time dependent permeability and oscillatory suction embedded with viscous energy dissipation using numerical approach (MOL).

In this paper, we are considering the MOL analysis of Dufour and Soret effects of unsteady MHD free convective fluid of heat and mass transport flow past an infinite porous plate under the influence of transverse magnetic field with time dependent permeability and oscillatory suction embedded with viscous energy dissipation and heat source.

2 Formulation of the problem

We considered the unsteady MHD free convection flow of an incompressible viscous electrically conducting fluid with simultaneous heat and mass transfer over an infinite vertical plate through porous medium with time dependent permeability and oscillatory suction. The y -axis is taken along the plate and x -axis perpendicular to it and u is the velocity along the x -direction.

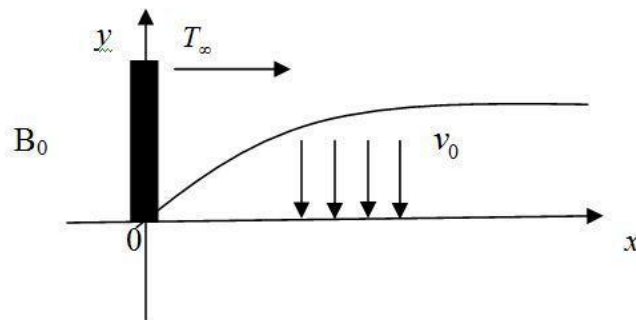


Figure 1: Physical configuration of the problem

The basic assumptions are:

1. All fluid properties are constant.
2. The plate and the fluid are to be at the same temperature and the species concentration is raised or lowered.
3. The magnetic Reynolds number is so small that the induced magnetic field are neglected in comparison to the applied magnetic field.
4. The permeability of the porous medium is

$$k(t) = k(1 + \varepsilon e^{i\omega t}) \quad (1)$$

5. The suction velocity is

$$v(t) = -v_0(1 + \varepsilon e^{i\omega t}) \quad (2)$$

where, v_0 represents the suction / injection velocity at the plate.

6. If the plate is extended to infinite length, then all the physical variables are functions of y and t alone.

The governing equations are given by

$$\frac{\partial u}{\partial y} = 0 \quad (3)$$

$$\frac{\partial u}{\partial t} + v \frac{\partial u}{\partial y} = \nu \frac{\partial^2 u}{\partial y^2} + g\beta(T - T_\infty) + g\beta^*(C - C_\infty) - \frac{\sigma B_0^2}{\rho} u - \frac{\nu}{k(t)} u \quad (4)$$

$$\frac{\partial T}{\partial t} + v \frac{\partial T}{\partial y} = \alpha \frac{\partial^2 T}{\partial y^2} - S_1(T - T_\infty) + \frac{\mu}{\rho c_p} \left(\frac{\partial u}{\partial y} \right)^2 + \frac{D_m K_T}{c_s c_p} \frac{\partial^2 c}{\partial y^2} \quad (5)$$

$$\frac{\partial c}{\partial t} + v \frac{\partial c}{\partial y} = D \frac{\partial^2 c}{\partial y^2} - k_1(C - C_\infty) + \frac{D_m K_T}{T_m} \frac{\partial^2 T}{\partial y^2} \quad (6)$$

Subject to

$$u(y, t) = T(y, t) = C(y, t) = f(t) = 1 + \varepsilon e^{i\omega t} \quad @ y = 0 \quad (7)$$

$$u(y, t) = T(y, t) = C(y, t) = 0 \quad @ y \rightarrow \infty$$

where

u - component of the velocity, t - time, c_p - the specific heat at constant pressure, C - dimensional concentration, D_m - mass diffusivity, g - acceleration due to gravity, k_1 - chemical reaction parameter, α - fluid thermal diffusivity, β - thermal expansion coefficient, β^* - concentration expansion coefficient, μ - coefficient of viscosity, ρ - fluid density, K_T - thermal diffusion ratio, T_m - mean fluid temperature, T_∞ - free stream dimensional temperature, C_∞ - free stream dimensional concentration, σ - electrical conductivity of the fluid, B_0 - the external imposed magnetic field strength, c_s - concentration susceptibility, ν - viscosity, T_w - wall dimensional temperature, C_w - wall dimensional concentration.

From equation (3), it is obvious that the continuity equation is a constant. Also ε is a small value less than unity i.e., $\varepsilon \ll 1$ and ν_0 is a non-zero positive constant, where the negative sign indicates that the suction is towards the plate.

Using the following dimensionless variables, we dimensionalise (3) – (7), taking the Boussinesq's approximation into account in equation (7),

$$u^* = \frac{u}{v_0}; \quad y^* = \frac{v_0 y}{v}; \quad t^* = \frac{v_0^2 t}{v}; \quad v^* = \frac{v}{v_0}; \quad w^* = \frac{vw}{v_0^2}; \quad \theta = \frac{T - T_\infty}{T_w - T_\infty}; \quad \phi = \frac{C - C_\infty}{C_w - C_\infty}; \quad (8)$$

Thus, we obtain,

$$\frac{\partial u}{\partial y} = 0 \quad (9)$$

$$\frac{\partial u}{\partial t} - v_0 (1 + \varepsilon e^{i\omega t}) \frac{\partial u}{\partial y} = \frac{\partial^2 u}{\partial y^2} + Gr\theta + Gm\phi - \left(M^2 + \frac{1}{K} \right) u \quad (10)$$

$$\frac{\partial \theta}{\partial t} - v_0 (1 + \varepsilon e^{i\omega t}) \frac{\partial \theta}{\partial y} = \frac{1}{Pr} \frac{\partial^2 \theta}{\partial y^2} - S\theta + Ec \left(\frac{\partial u}{\partial y} \right)^2 + D_u \frac{\partial^2 \phi}{\partial y^2} \quad (11)$$

$$\frac{\partial \phi}{\partial t} - v_0 (1 + \varepsilon e^{i\omega t}) \frac{\partial \phi}{\partial y} = \frac{1}{Sc} \frac{\partial^2 \phi}{\partial y^2} - Kc\phi + S_r \frac{\partial^2 \theta}{\partial y^2} \quad (12)$$

subject to

$$u(y, t) = \theta(y, t) = \phi(y, t) = 1 + \varepsilon e^{i\omega t} \quad @ y = 0 \quad (13)$$

$$u(y, t) = \theta(y, t) = \phi(y, t) = 0 \quad @ y \rightarrow \infty$$

where,

$M^2 = \frac{\sigma \beta_0^2 v}{\rho v_0^2}$ is the Hartmann number (Magnetic field parameter), $Pr = \frac{v}{\alpha}$ is the Prantl number,

$K = \frac{k}{v^2}$ is the permeability parameter (Porosity or Darcy parameter), $Gm = \frac{vg\beta^*(T_w - T_\infty)}{v_0^3}$ is the

Prandtl number, $Sc = \frac{v}{D}$ is the Schmidt number, $Kc = \frac{k_1 v}{v_0^2}$ is the chemical reaction parameter,

$S = \frac{S_1 v}{v_0^2}$ is the Heat Source parameter, $Gr = \frac{vg\beta(T_w - T_\infty)}{v_0^3}$ is the thermal Grashof number,

$Gm = \frac{vg\beta^*(T_w - T_\infty)}{v_0^3}$ is the mass Grashof number and $Ec = \frac{v_0^2}{Cp(T_w - T_\infty)}$ is the Eckert number,

$D_u = \frac{D_m K_T (C_w - C_\infty)}{vc_s c_p (T_w - T_\infty)}$ is the Dufour number, $S_r = \frac{D_m K_T (T_w - T_\infty)}{vT_m (C_w - C_\infty)}$ is the Soret number.

3 Method of lines

The basic idea of the MOL is to replace the special (boundary value) derivatives in the PDE with algebraic approximations. Once this is done, only the initial value variable, typically time in a physical problem remains. Then, with only one remaining independent variable, we have a system of ODEs

that approximates the original PDE. Any suitable integration algorithm for the initial value ODEs can now be used to compute an approximate numerical solution to the PDE.

Before applying the method of lines to equation (9) – (12) subject to boundary conditions (13), we adopt the approximation below to decouple and linearize equations (9) – (12). In view of the approximation adopted, equations (9) – (12) reduce to;

$$\frac{\partial u}{\partial t} = \nu_0 (1 + \varepsilon e^{i\omega t}) \frac{\partial u}{\partial y} + \frac{\partial^2 u}{\partial y^2} + Gr + Gm - \left(M^2 + \frac{1}{K} \right) u \quad (14)$$

$$\frac{\partial \phi}{\partial t} = \nu_0 (1 + \varepsilon e^{i\omega t}) \frac{\partial \phi}{\partial y} + \frac{1}{Sc} \frac{\partial^2 \phi}{\partial y^2} - Kc\phi + S_r \quad (15)$$

$$\frac{\partial \theta}{\partial t} = \nu_0 (1 + \varepsilon e^{i\omega t}) \frac{\partial \theta}{\partial y} + \frac{1}{Pr} \frac{\partial^2 \theta}{\partial y^2} - S\theta + Ec + D_u \quad (16)$$

Applying the method of lines to equation (14), we discretize the partial derivative in space variable y , to result in approximating system of ODEs in variable t , thus we have;

$$\left(\frac{\partial u}{\partial t} \right)_i = \nu_0 (1 + \varepsilon e^{i\omega t}) \frac{u_{i+1} - u_{i-1}}{2h} + \frac{u_{i+1} - 2u_i + u_{i-1}}{h^2} + Gr + Gm - \left(M^2 + \frac{1}{K} \right) u_i \quad (17)$$

Simplifying the right-hand side of equation (17) gives:

$$\left(\frac{\partial u}{\partial t} \right)_i = \alpha_1 u_{i-1} + \alpha_2 u_i + \alpha_3 u_{i+1} + \alpha_4 \quad (18)$$

where,

$$\alpha_1 = \left(\frac{1}{h^2} - \frac{\nu_0 (1 + \varepsilon e^{i\omega t})}{2h} \right); \alpha_2 = - \left(\frac{2}{h^2} + M^2 + \frac{1}{K} \right); \alpha_3 = \left(\frac{1}{h^2} + \frac{\nu_0 (1 + \varepsilon e^{i\omega t})}{2h} \right); \alpha_4 = Gr + Gm, \quad (19)$$

$i = 1, 2, 3, \dots, N$

Equations (18) – (19) can be solved iteratively using the boundary conditions $u(0, t) = 1 + \varepsilon e^{nt}$ and $u(\alpha, t) = 0$ in equations (13).

For $i = 1, 2, 3, \dots, N$; $u(0, t) = u(y, t) = 1 + \varepsilon e^{iwt}$; & $u(\infty, t) \approx u(N + 1, t) = 0$; in equation (18) can be written as;

$$\begin{aligned}\dot{u}_1 &= \alpha_1 + \alpha_2 u_1 + \alpha_3 u_2 + \alpha_4 \\ \dot{u}_2 &= \alpha_1 u_1 + \alpha_2 u_2 + \alpha_3 u_3 + \alpha_4 \\ \dot{u}_3 &= \alpha_1 u_2 + \alpha_2 u_3 + \alpha_3 u_4 + \alpha_4 \\ \dot{u}_4 &= \alpha_1 u_3 + \alpha_2 u_4 + \alpha_3 u_5 + \alpha_4 \\ \dot{u}_N &= \alpha_1 u_{N-1} + \alpha_2 u_N + \alpha_4\end{aligned}\tag{20}$$

The system in equation (20), in matrix form is given as

$$\begin{bmatrix} \dot{u}_1 \\ \dot{u}_2 \\ \vdots \\ \dot{u}_{N-1} \\ u_N \end{bmatrix} = \begin{bmatrix} \alpha_1 & \alpha_2 & \alpha_3 & 0 & 0 & \dots & 0 & 0 \\ 0 & \alpha_1 & \alpha_2 & \alpha_3 & 0 & \dots & 0 & 0 \\ 0 & 0 & \alpha_1 & \alpha_2 & \alpha_3 & \dots & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & 0 & \dots & \alpha_1 & \alpha_2 \end{bmatrix} \begin{bmatrix} 1 + \varepsilon e^{iwt} \\ u_1 \\ u_2 \\ \vdots \\ u_{N-1} \\ u_N \end{bmatrix} + \begin{bmatrix} \alpha_4 \\ \alpha_4 \\ \alpha_4 \\ \vdots \\ \alpha_4 \\ \alpha_4 \end{bmatrix}\tag{21}$$

where the coefficient of α_1 , α_2 , α_3 and α_4 are given by (19) and $\dot{u}_i = \left(\frac{\partial u}{\partial t} \right)_i$

In a similar way, equation (15) becomes;

$$\left(\frac{\partial \phi}{\partial t} \right)_i = v_0 (1 + \varepsilon e^{iwt}) \frac{\phi_{i+1} - \phi_{i-1}}{2h} + \frac{1}{Sc} \left(\frac{\phi_{i+1} - 2\phi_i + \phi_{i-1}}{h^2} \right) - Kc\phi_i + S_r\tag{22}$$

Simplifying the right-hand side, we obtain;

$$\left(\frac{\partial \phi}{\partial t} \right)_i = \beta_1 \phi_{i-1} + \beta_2 \phi_i + \beta_3 \phi_{i+1} + \beta_4\tag{23}$$

where,

$$\beta_1 = \left(\frac{1}{h^2 Sc} - \frac{\nu_0(1 + \varepsilon e^{i\omega t})}{2h} \right); \beta_2 = -\left(\frac{2}{h^2 Sc} + Kc \right); \beta_3 = \left(\frac{1}{h^2 Sc} + \frac{\nu_0(1 + \varepsilon e^{i\omega t})}{2h} \right); \beta_4 = S_r; \quad (24)$$

Equations (23) – (24) can be solved iteratively using the boundary conditions $\phi(0, t) = 1 + \varepsilon e^{i\omega t}$ and $\phi(\infty, t) = 0$ in equations (13).

For $i = 1, 2, 3, \dots, N$; $\phi(0, t) = 1 + \varepsilon e^{i\omega t}$; & $\phi(\infty, t) \approx \phi(N+1, t) = 0$; in equation (23) can be written in matrix form as;

$$\begin{bmatrix} \dot{\phi}_1 \\ \dot{\phi}_2 \\ \vdots \\ \dot{\phi}_{N-1} \\ \dot{\phi}_N \end{bmatrix} = \begin{bmatrix} \beta_1 & \beta_2 & \beta_3 & 0 & 0 & \dots & 0 & 0 \\ 0 & \beta_1 & \beta_2 & \beta_3 & 0 & \dots & 0 & 0 \\ 0 & 0 & \beta_1 & \beta_2 & \beta_3 & \dots & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & 0 & \dots & \beta_1 & \beta_2 \end{bmatrix} \begin{bmatrix} 1 + \varepsilon e^{i\omega t} \\ \phi_1 \\ \phi_2 \\ \vdots \\ \phi_{N-1} \\ \phi_N \end{bmatrix} + \begin{bmatrix} 0 \\ 0 \\ 0 \\ \vdots \\ 0 \\ 0 \end{bmatrix} \quad (25)$$

where the coefficient β_1 , β_2 , and β_3 are given in equation (24) and $\dot{\phi}_i = \left(\frac{\partial \phi}{\partial t} \right)_i$.

Similarly, equation (16) becomes;

$$\left(\frac{\partial \theta}{\partial t} \right)_i = \left(\frac{1}{h^2 Pr} - \frac{\nu_0(1 + \varepsilon e^{i\omega t})}{2h} \right) \theta_{i-1} - \left(\frac{2}{h^2 Pr} + S \right) \theta_i + \left(\frac{1}{h^2 Pr} + \frac{\nu_0(1 + \varepsilon e^{i\omega t})}{2h} \right) \theta_{i+1} + Ec + D_u \quad (26)$$

$$\left(\frac{\partial \theta}{\partial t} \right)_i = \gamma_1 \theta_{i-1} + \gamma_2 \theta_i + \gamma_3 \theta_{i+1} + \gamma_4 \quad (27)$$

where

$$\gamma_1 = \left(\frac{1}{h^2 Pr} - \frac{\nu_0(1 + \varepsilon e^{i\omega t})}{2h} \right); \gamma_2 = -\left(\frac{2}{h^2 Pr} + S \right); \gamma_3 = \left(\frac{1}{h^2 Pr} + \frac{\nu_0(1 + \varepsilon e^{i\omega t})}{2h} \right); \gamma_4 = Ec + D_u \quad (28)$$

Equations (26) – (28) can be solved iteratively using the boundary conditions $\theta(0, t) = 1 + \varepsilon e^{i\omega t}$ and $\theta(\infty, t) = 0$ in equations (13).

For $i = 1, 2, 3, \dots, N$; $\theta(0, t) = 1 + \varepsilon e^{i\omega t}$; & $\theta(\infty, t) \approx \theta(N+1, t) = 0$; in equation (27) can be written in matrix form as;

$$\begin{bmatrix} \dot{\theta}_1 \\ \dot{\theta}_2 \\ \vdots \\ \dot{\theta}_{N-1} \\ \dot{\theta}_N \end{bmatrix} = \begin{bmatrix} \gamma_1 & \gamma_2 & \gamma_3 & 0 & 0 & \dots & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & \gamma_3 & 0 & \dots & 0 & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & \dots & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & 0 & \dots & \gamma_1 & \gamma_2 \end{bmatrix} \begin{bmatrix} 1 + \varepsilon e^{i\omega t} \\ \theta_1 \\ \theta_2 \\ \vdots \\ \theta_{N-1} \\ \theta_N \end{bmatrix} + \begin{bmatrix} \gamma_4 \\ \gamma_4 \\ \gamma_4 \\ \vdots \\ \gamma_4 \\ \gamma_4 \end{bmatrix} \quad (29)$$

where the coefficient $\gamma_1, \gamma_2, \gamma_3$ and γ_4 are given by the equation (28) and $\dot{\theta}_i = \left(\frac{\partial \theta}{\partial t} \right)_i$.

The three physical quantities at the plate are given as,

$$\text{Skin friction coefficient: } C_f = \frac{\tau' \omega}{\rho u_0 v_0} = \left(\frac{\partial u}{\partial y} \right) \bigg|_{y=0} \quad (30)$$

$$\text{Nusselt Number: } Nu = - \frac{x \left(\frac{\partial T}{\partial y} \right)}{(T_w - T_\infty)} \bigg|_{y=0} \quad (31)$$

$$\text{Sherwood Number: } Sh = - \frac{x \left(\frac{\partial C}{\partial y} \right)}{(C_w - C_\infty)} \bigg|_{y=0} \quad (32)$$

4 Results and discussion

We have considered the effects of some thermo-physical properties of unsteady free convective flow of a viscous incompressible electrically conducting fluid over an infinite vertical porous plate under the influence of uniform transverse magnetic field with time dependent permeability and oscillatory suction in the presence Soret and Dufour parameter numerically using MATLAB simulations. Some graphical and numerical representation are thus given below;

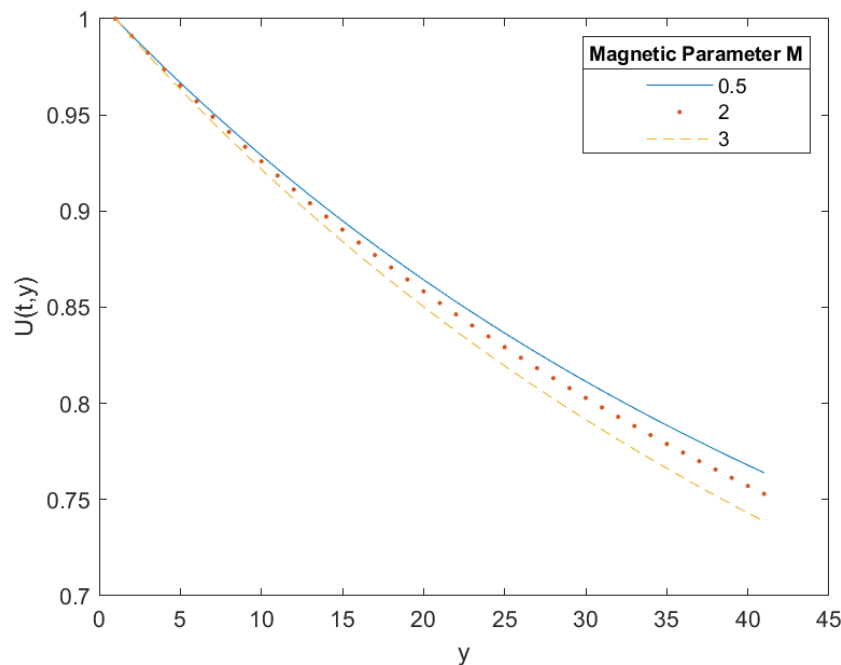


Figure 2: Effects of Magnetic Parameter on the velocity profile.

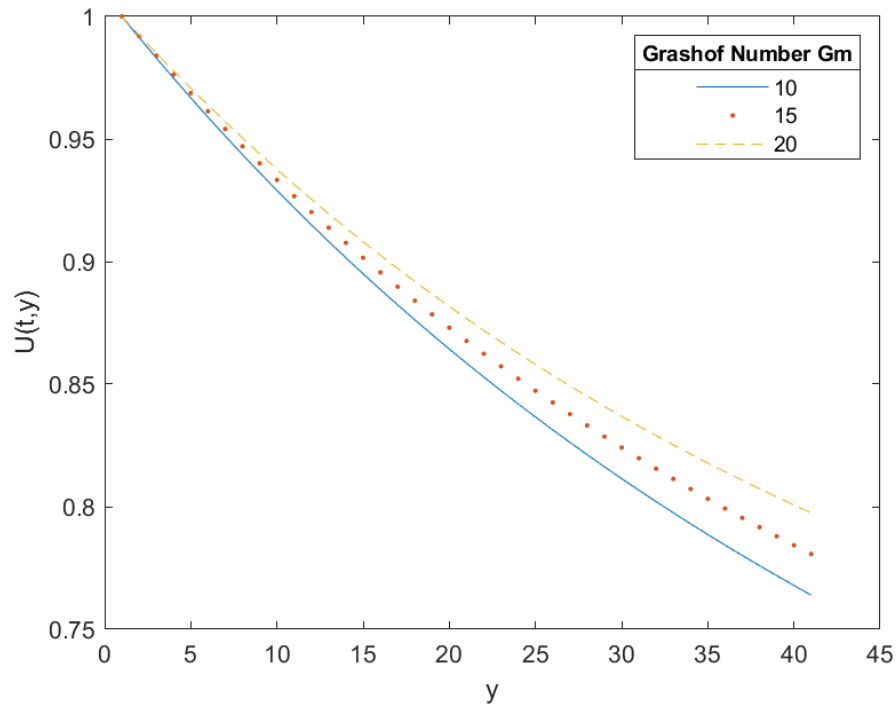


Figure 3: Effects of Mass Grashof Number on the velocity profile

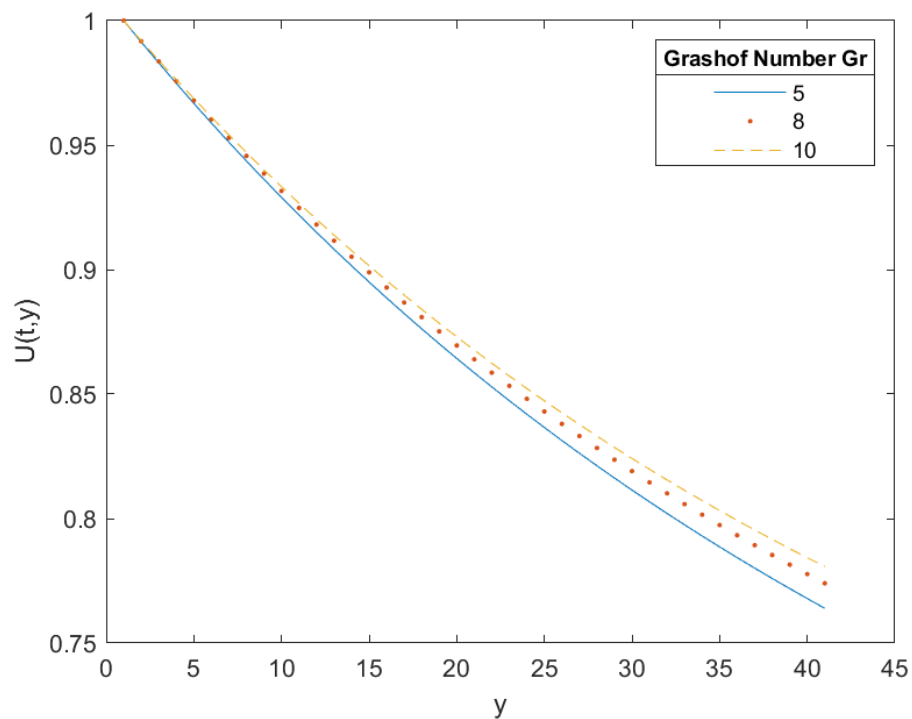


Figure 4: Effects of Thermal Grashof Number on the velocity profile

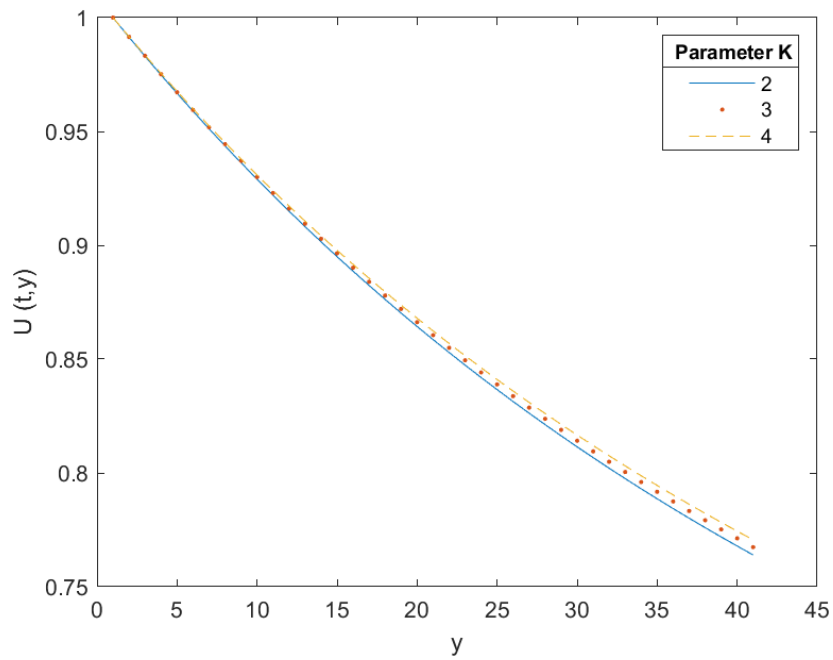


Figure 5: Effects of Permeability (Porosity) on the velocity profile

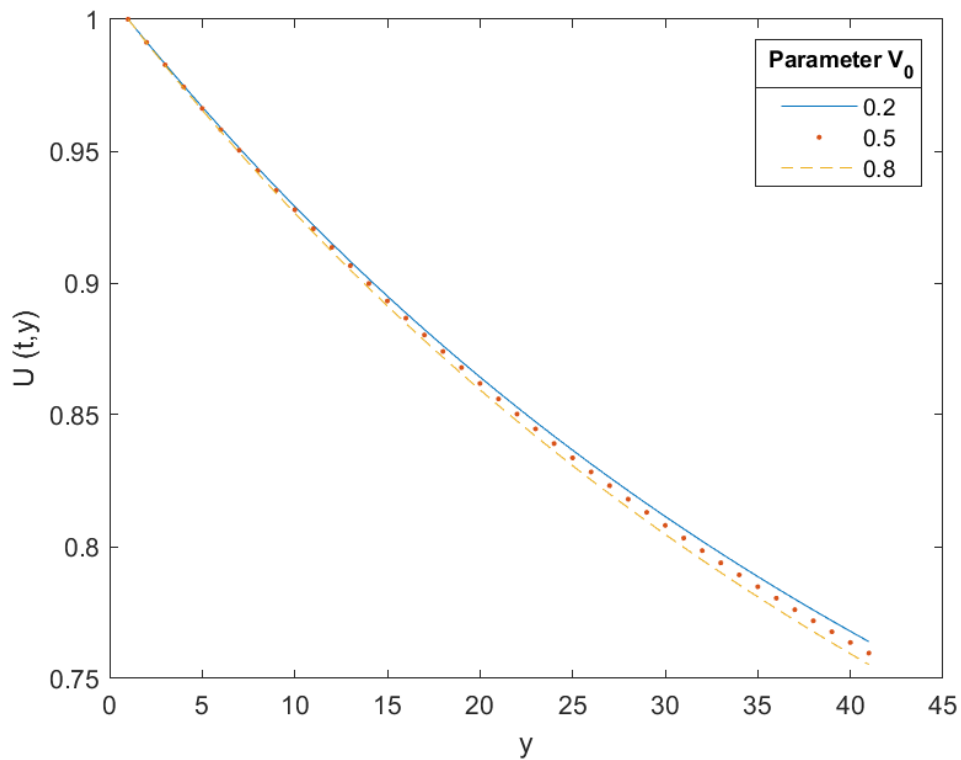


Figure 6: Effects of Parameter V_0 on the velocity profile

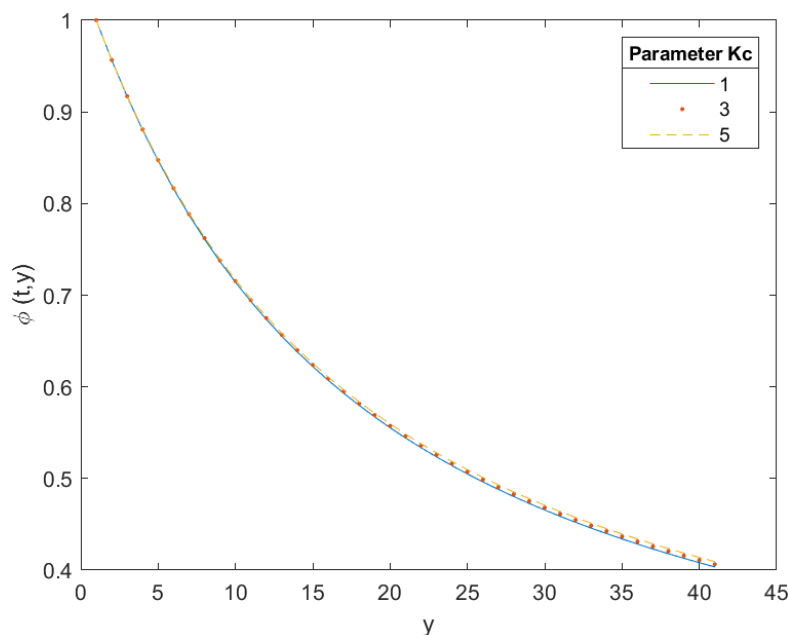


Figure 7: Effects of Parameter Kc on the Concentration profile

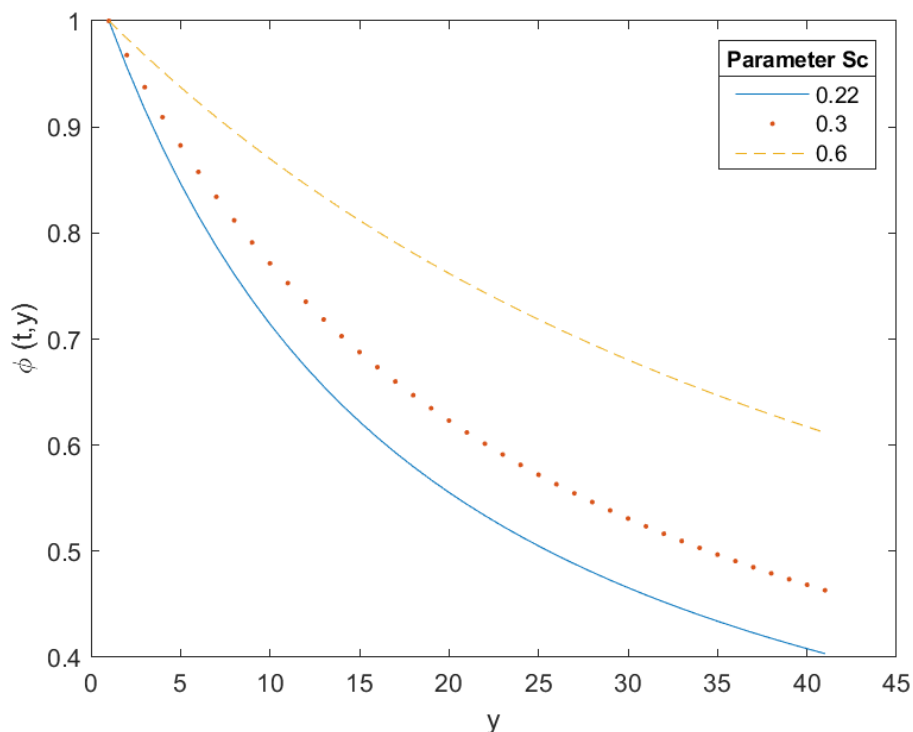


Figure 8: Effects of Schmidt Number, Sc on the Concentration profile

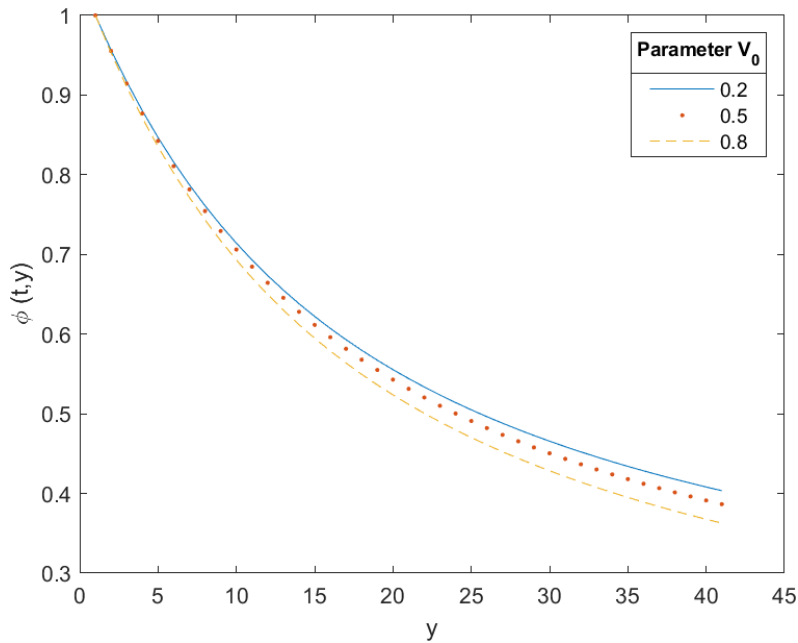


Figure 9: Effects of Parameter V_0 on the Concentration profile

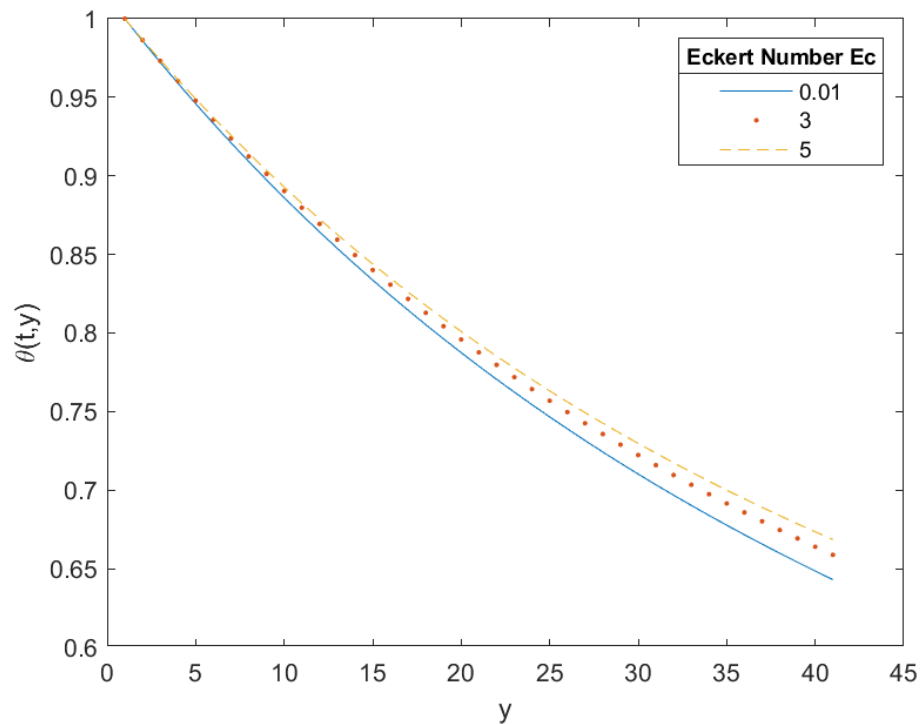


Figure 10: Effects of Eckert Number on the Temperature profile

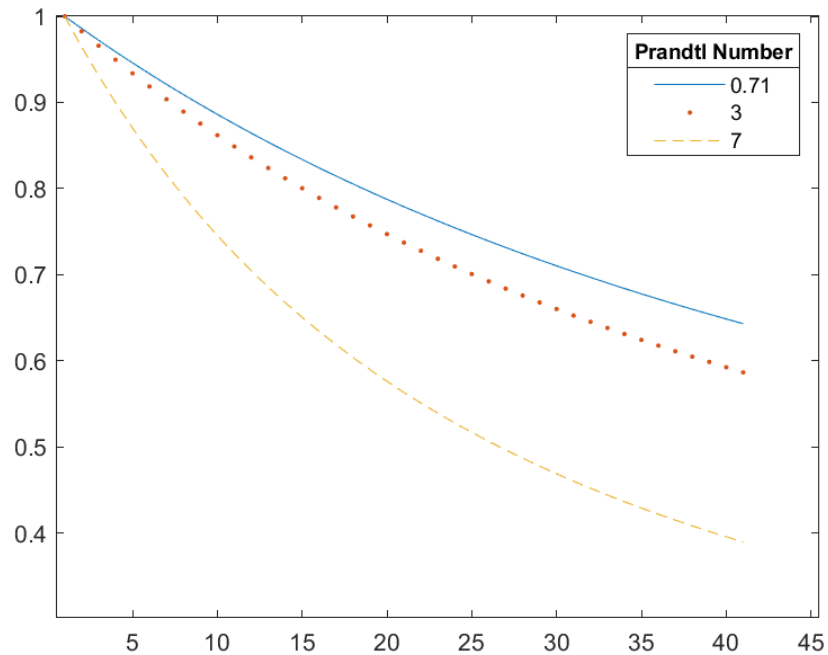


Figure 11: Effects of Prantl number on the Temperature profile

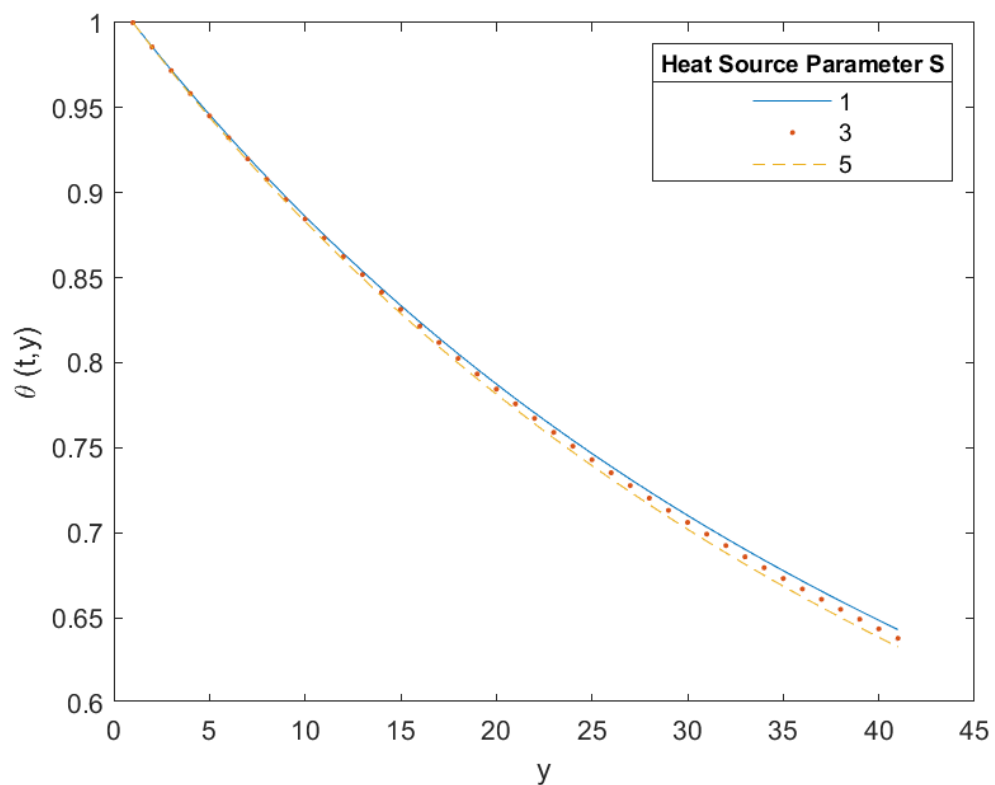


Figure 12: Effects of Heat Source on the Temperature profile

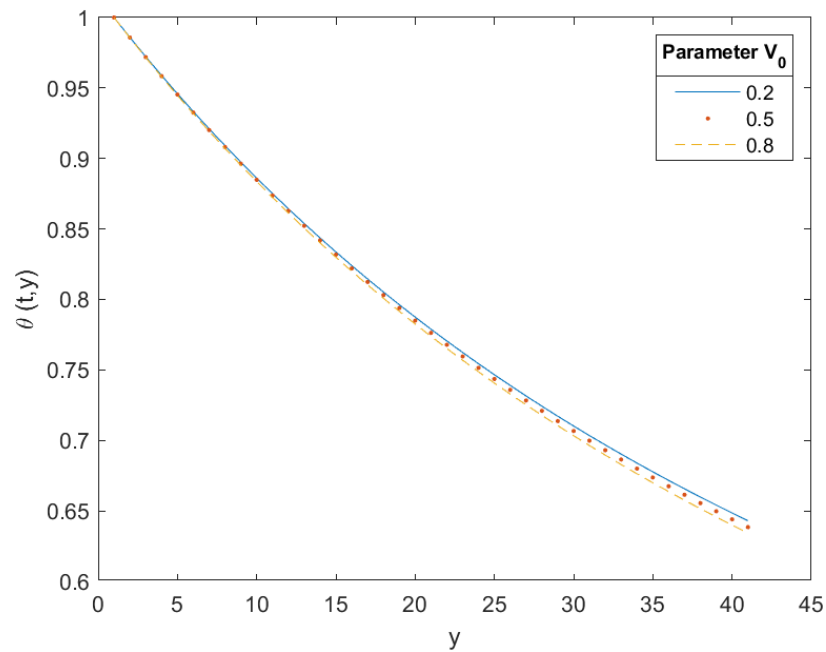


Figure 13: Effects of Parameter V_0 on the Temperature profile

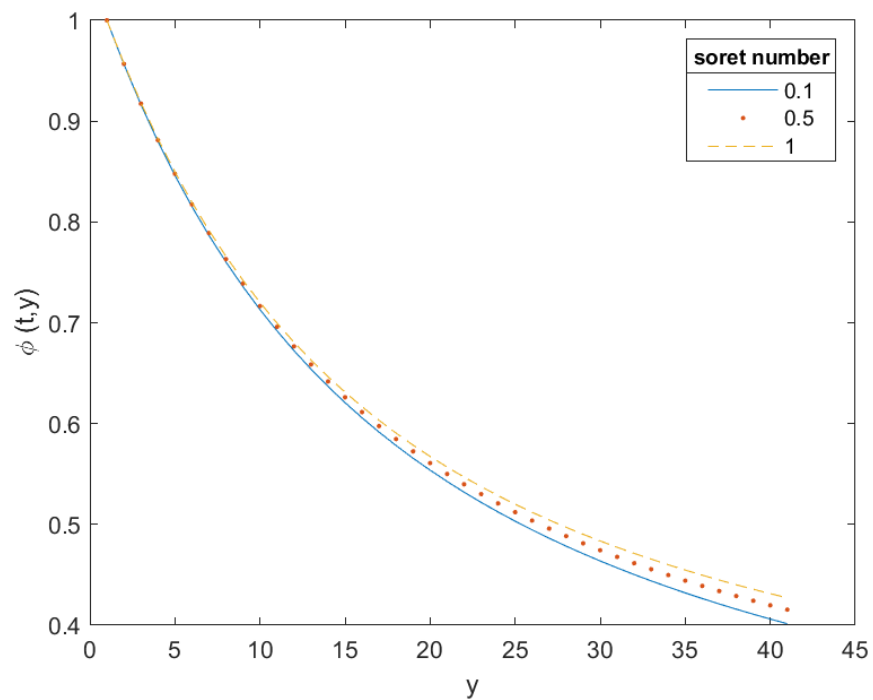


Figure 14: Effects of Soret number on the concentration profile

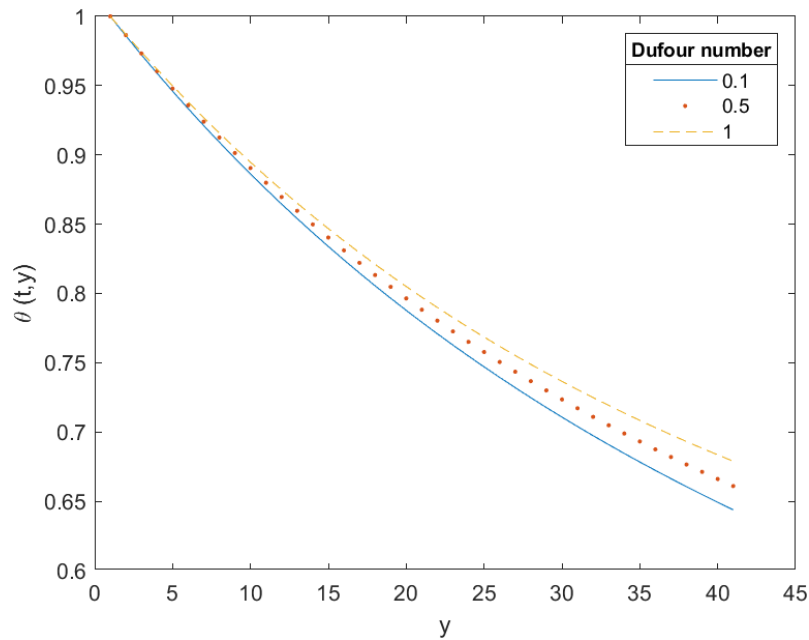


Figure 15: Effects of Dufour number on the temperature profile

Table 1. Computational values for local skin friction $u'(0)$ for different values of M, K, Gm, and Gr where $t := 0.2 : h := 0.1 : v[0] := 0.2 : \varepsilon := 0.001 : S := 1 : Sc := 0.22 : Kc := 1 : Pr := 0.71 : Ec := 0.001 : \omega := \pi/6 : Du := 0.1 : Sr := 0.1 :$

M	K	Gm	Gr	Skin friction (stress)
0.5	2	10	5	12.9134
2				7.6352
3				3.8917
	3			12.1057
	4			10.4123
		15		15.3607
		20		17.5112
			8	13.7213
			10	14.3443

Table 2. Computational values for local Sherwood number (Sh) for different values of Sc , Kc , V_0 , w , and Sr where $t := 0.2 : h := 0.1 : \varepsilon := 0.001 : S := 1 : M := 0.5 : K := 2 : Gr := 5 : Gm := 10 : Pr := 0.71 : Ec := 0.001 : \omega := \pi/6 : Du := 0.1 :$

Sc	Kc	V_0	w	Sr	Sherwood Number (Sh)
0.22	1	0.2	$\pi/6$	0.1	0.5239
0.3					0.6683
0.6					0.9675
	3				0.7551
	5				0.9354
		0.5			0.6816
		0.8			0.8147
			$\pi/4$		0.5254
			$\pi/3$		0.5279
				0.5	0.3917
				1	0.1782

Table 3. Computational values for Nusselt number (Nu) for different values of Pr , S , Ec , Du , V_0 , and w where $t := 0.2 : h := 0.1 : \varepsilon := 0.001 : n := 0.01 : M := 0.5 : K := 2 : Gr := 5 : Gm := 10 : Sc := 0.22 : Kc := 1 : Sr := 0.1 :$

Pr	S	Ec	V_0	Du	Nusselt Number (Sh)
0.71	1	0.01	0.2	0.1	1.1209
3					2.9979
7					4.0122
	2				1.7231
	3				2.0464
		0.5			1.5926
		3			1.9917
			0.5		1.4461
			0.8		1.6022
				0.5	0.9341
				1	0.7533

Table 4. Comparison of Results when $Sc=0$, $Sr=0$ in present study with Basha *et al.* (2019)

Sc	V_0	w	Sherwood Number (Sh)	
			Basha et al. [19]	Present study
0.22	0.2	$\pi/6$	0.044020	0.051062
0.3			0.060238	0.065387
0.6			0.120325	0.196711
	0.5		0.110179	0.189233
	0.8		0.176133	0.242128
		$\pi/4$	0.044236	0.054688
		$\pi/3$	0.044253	0.056167

Figure (2) shows that the magnitude of the velocity component increases with decreasing the intensity of the magnetic field, M . This is due to the Lorentz force the velocity retards continuously throughout the fluid region. Figure (3) shows that the velocity component decreases with decrease in mass Grashof number. Figure (4) shows that the velocity component also increases with increase in thermal Grashof number. Figure (5) depicts that increasing the permeability of the porous medium increases the fluid speed in the entire region. Figure (6) depicts that the velocity component experiences retardation in the flow field with increasing suction parameter. Figure (7) and (8) shows that the Concentration distribution decreases with increase in chemical reaction or Schmidt number respectively, thus causing a retardation in the fluid flow. Also figure (9) shows that decreasing the suction parameter leads to increase in the concentration distribution. These results in the concentration distribution show that the heavier diffusing species have a greater retarding effect on the concentration distribution of the flow field. Figure (10) show that the temperature of the system decreases with a decrease in Eckert number. Figure (11) shows that the temperature of the flow field reduces as the Prantl number increases. This is consistent with the fact that the thermal boundary layer thickness decreases with increasing Prantl number. Figure (12) depicts that with increasing heat source, the temperature of the flow field reduces. This may be as a result of the elastic property of the fluid. It is observed that in figure (13) increasing the suction parameter reduces the temperature of the system. Figure (14) depicts that as Soret number increases the concentration of the fluid increases as well, also figure (15) shows that a decrease in the Dufour number leads to a considerable decrease in the temperature of the system.

The frictional force is a significant phenomenon which characterizes the frictional drag force at the solid surface. From Table 1, it is observed that the ' τ ' increases with the increase in K , Gr , Gm and v_0 , but it is interesting to note that the ' τ ' decreases with the increase in M . From Table 2, It is observed that as Sc , Kc , w and v_0 increases, ' Sh ' also increases at the surface of the plate but decreases with increase in Soret number. From Table 3, we see that all the entries are positive. It is seen that increase in S , Pr , Ec , and v_0 increases Nu at the surface of the plate. Also, Nu decreases with increase in Du . Table 4 represents the comparison of the results with Basha *et al.* (2019).

5 Conclusion

The study has examined the Method of Lines analysis (MOL) on the problem of an unsteady heat and mass transfer flow of an MHD fluid past an infinite porous plate under the influence of Dufour, Soret and other pertinent flow parameters. The following conclusions are drawn from the study; as Soret number (S_r) and Dufour number (D_u) increases, the skin friction coefficient (C_f) increases while the Nusselt number (N_u) decreases. As Soret number (S_r) decreases, the Sherwood number (S_h) increases drastically while decrease in Dufour number has a negligible or no influence on it. As Soret number (S_r) increases, both the velocity $u(y,t)$ profile and the concentration profile $\phi(y,t)$ increase, while there is negligible or no effect on the temperature distribution. As Dufour number (D_u) increases, both the velocity profile $u(y,t)$ and temperature profile $\theta(y,t)$ increase. As the thermal Grashof number (G_r) and modified Grashof number (G_m) increases, the velocity profile $u(y,t)$ increases. As Schmidt number (S_c) and Chemical reaction (K_c) increases, the velocity profile $u(y,t)$ and the concentration profile $\phi(y,t)$ decrease. As Eckert number (E_c) increases, both the velocity profile $u(y,t)$ and temperature profile $\theta(y,t)$ increase. An Increase in the magnetic parameter (M) decreases the velocity profile $u(y,t)$ as a result of a resistive Lorentz force produced by the magnetic field. It has been observed that, in designing a system where high temperature is needed, such as glass production, propulsion system, plasma physics etc., the effects of Soret, Dufour, Eckert number, Grashof number, Chemical reaction parameter, Schmidt number and Magnetic parameter should be considered carefully for optimal performance of such system.

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A two-infection stage model with SIS and SIR dynamics

A.O. Sangotola* and L.J. Sangoyinka†

Abstract

A two-infection stage model is presented in this research work. The dynamics of the model is such that it behaves like a SIS or SIR model depending on the stage treatment is applied. The disease-free equilibrium point and the basic reproduction number are determined. The conditions for the disease-free equilibrium and endemic equilibrium to be globally asymptotically stable are determined. Sensitivity analysis is used to determine the impact of the model parameter on the basic reproduction number. Numerical simulation with suitable approximated parameters using MATLAB is also carried out to give possible dynamics of the model.

Keywords: stability; equilibrium; basic reproduction number; Lyapunov function.

1 Introduction

The dynamic models for infectious disease are mostly based on their compartment structure. The compartment structures for dynamic models are firstly given by Kermack and Mckendrick in 1927 and has been improved over the years [1]. Some of the compartmental models includes: SI, SIS, SIR, SEIR, SEIRS etc. Mathematical models of infectious diseases give insights into disease transmission, progression, stage of infection and helps in mathematical analysis. Blackwood and Childs [3] highlighted several common issues encountered while analyzing basic compartmental models. Melesse and Gumel [9] presented an extensive mathematical analysis of a deterministic model, with a standard incidence function, for the transmission dynamics of a communicable disease with an arbitrary number of distinct infectious stages. The conditions for the global stability of the disease free and endemic equilibrium was determined. Numerical simulation was carried out to illustrate the theoretical results.

* Corresponding Author. Department of Physical Sciences, Bells University of Technology, Ota, Ogun State, Nigeria; E-mail: adekunle4000@hotmail.co.uk , aosangotola@bellsuniversity.edu.ng

†Department of Mathematics and Statistics, The Polytechnic Ibadan, Oyo State, Nigeria; Email: lsangoyinka@gmail.com

Otunuga and Ogunsolu [14] presented a mathematical analysis of the transmission of certain diseases using a stochastic susceptible-exposed-infectious-treated-recovered (SEITR) model with multiple stages of infection and treatment and explore the effects of treatments and external fluctuations in the transmission, treatment and recovery rates. Global stability analysis of the disease free and endemic equilibrium for both the deterministic and stochastic version of the model were presented. Rifanti [16] presented a two-infection stage mathematical method which are chronic stage and acute stage. Both infection stages involve transmissibility. Multistage dynamics of infectious dynamics are also captured in the following literatures: [7, 10, 13, 18].

Here, we consider a two-infection stage model where the first stage is the passive stage and the second stage is the active stage. The model dynamics can be a SIS or SIR dynamics depending on the stage of treatment application. Individuals who recover from the first stage due to early detection and treatment or natural immunity goes back to the susceptible class because the disease has not run its course. Individuals who recover from the second stage moves to the recovered class. This dynamic is explained further under the model formulation.

2 Model formulation

The model is divided into four compartments with the susceptible population denoted by $S(t)$, the first infection stage is denoted by $I_1(t)$, the second infection stage is denoted by $I_2(t)$ while the recovered population denoted by $R(t)$. The susceptible population is increased as a result of migration or birth at rate Λ . The dynamics of the disease is such that there are two infection stages with infectivity in the second stage of infection. There susceptible population is decreased as a result of natural death at rate μ and by interaction with infectious population (I_2) with β as the force of infection. This above interaction causes an increase in the population at the first stage of infection. The first stage of infection is reduced either by natural death, temporary recovery back to the susceptible population at rate σ or allowing the disease to develop into the second stage at rate α . The second infection stage is reduced by natural death, disease induced death at rate δ or by effective treatment at rate γ . The recovered population is increased as a result of recovery from second stage and decreased only by natural death.

The resulting system from the above assumptions is given as

$$\frac{dS}{dt} = \Lambda - \beta SI_2 - \mu S + \sigma I_1 \quad (1)$$

$$\frac{dI_1}{dt} = \beta SI_2 - (\alpha + \mu + \sigma) I_1 \quad (2)$$

$$\frac{dI_2}{dt} = \alpha I_1 - (\mu + \delta + \gamma) I_2 \quad (3)$$

$$\frac{dR}{dt} = \gamma I_2 - \mu R \quad (4)$$

Description of parameters in the model (1) – (4):

Parameter	Meaning
Λ	Recruitment rate
β	Transmission rate
μ	Natural death rate
σ	Recovery rate from stage 1
α	Progression rate to second stage
δ	Disease induced death rate
γ	Recovery rate from stage 2

3 Results and discussions

3.1 Invariant region

We obtain the region where the solution of (1) – (4) is epidemiological feasible.

Theorem 3.1: (Invariant region). The feasible region $\mathcal{R}$ defined by $\{S(t), I_1(t), I_2(t), R(t) \in \mathbb{R}_+^4 : N(0) \leq N(t) \leq \frac{\Lambda}{\mu}\}$ with initial conditions $S(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0$ is positive invariant for system (1) – (4).

Proof: The total population size is given by $N(t) = S(t) + I_1(t) + I_2(t) + R(t)$.

$$\begin{aligned}\frac{dN}{dt} &= \Lambda - \mu N - \delta I_2 \\ \frac{dN_h}{dt} &\leq \Lambda - \mu N\end{aligned}$$

Solving above, we have

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

$$\text{As } t \rightarrow \infty, 0 \leq N(t) \leq \frac{\Lambda}{\mu}, \text{ If } N(0) \leq \frac{\Lambda}{\mu} \text{ then } N(t) \leq \frac{\Lambda}{\mu}. \text{ Hence,}$$

$$N(0) \leq N(t) \leq \frac{\Lambda}{\mu}.$$

Thus, $\mathcal{R}$ is a positive invariant set under the model described by (1) – (4).

3.2 Disease-free equilibrium

The disease-free equilibrium is the point where there is no disease in the population. It lies at the point $\pi_o = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$

3.3 Basic reproduction number

The basic reproductive number is the mean number of secondary infections caused by an infectious individual in an entirely susceptible population. The next generation matrix approach by Driessche and Watmough [6] is applied.

From the infectious stages I_1 and I_2 , we can create a vector $\mathcal{F}$ that represent new infections and $\mathcal{V}$ that represents outflows from the model equations. Hence,

$$\mathcal{F} = \begin{pmatrix} \beta S I_2 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} (\alpha + \mu + \sigma) I_1 \\ (\mu + \delta + \gamma) I_2 - \alpha I_1 \end{pmatrix}.$$

Next, we compute the Jacobian F from $\mathcal{F}$ and V from $\mathcal{V}$ at the disease-free equilibrium point. They are given by

$$F = \begin{pmatrix} 0 & \beta \frac{\Lambda}{\mu} \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} (\alpha + \mu + \sigma) & 0 \\ -\alpha & (\mu + \delta + \gamma) \end{pmatrix}$$

The basic reproduction R_0 is given by $\rho(FV^{-1})$ where ρ is the spectral radius. Thus,

$$R_0 = \frac{\alpha \beta \Lambda}{\mu(\alpha + \mu + \sigma)(\mu + \delta + \gamma)}.$$

3.4 Local stability

Theorem 3.2: (Local stability of disease-free equilibrium). The disease-free equilibrium for the system is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

The equilibrium points of model (1) – (4) is locally asymptotically stable if all the eigenvalues of the Jacobian matrix have negative real parts.

The Jacobian matrix evaluated at the disease-free is given by

$$J(\pi_0) = \begin{pmatrix} -\mu & \sigma & -\frac{\beta \Lambda}{\mu} & 0 \\ 0 & -(\alpha + \mu + \sigma) & \frac{\beta \Lambda}{\mu} & 0 \\ 0 & \alpha & -(\mu + \delta + \gamma) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

There are four roots corresponding to the Jacobian above. Two of the roots are $-\mu$ (twice) while the other roots can be obtained from the characteristic equation of the sub matrix given below.

$$J_1(\pi_0) = \begin{pmatrix} -(\alpha + \mu + \sigma) & \frac{\beta\Lambda}{\mu} \\ \alpha & -(\mu + \delta + \gamma) \end{pmatrix}$$

The remaining eigenvalues are negative if trace of $J_1(\pi_0)$ is negative and determinant of $J_1(\pi_0)$ is positive. The trace of $J_1(\pi_0)$ is negative and the determinant is $(\alpha + \mu + \sigma)(\mu + \delta + \gamma) - \frac{\alpha\beta\Lambda}{\mu}$. The determinant is also equivalent to $(\alpha + \mu + \sigma)(\mu + \delta + \gamma)(1 - R_0)$ and it is positive provided that $R_0 < 1$.

Theorem 3.3: (Existence of endemic equilibrium). The model (1) – (4) has an endemic equilibrium when $R_0 > 1$.

Proof: Let $E_e^* = (S^*, I_1^*, I_2^*, R^*)$ be a non-trivial equilibrium of the model. The model at steady state becomes:

$$\begin{aligned} S^* &= \frac{(\alpha + \mu + \sigma)(\mu + \delta + \gamma)}{\alpha\beta} \\ I_1^* &= \frac{\Lambda}{R_0(\mu + \alpha)} [R_0 - 1] \\ R^* &= \frac{\gamma(\alpha + \mu + \sigma)}{\beta(\mu + \alpha)} [R_0 - 1] \\ I_2^* &= \frac{\mu(\alpha + \mu + \sigma)}{\beta(\mu + \alpha)} [R_0 - 1]. \end{aligned}$$

3.5 Global stability analysis

Theorem 3.4: (Global stability of disease-free equilibrium). The disease-free equilibrium for the system is globally asymptotically stable if $R_0 < 1$.

Proof: Consider the Lyapunov function:

$$\begin{aligned} L &= \alpha I_1 + (\alpha + \mu + \sigma) I_2 \\ \dot{L} &= \alpha \dot{I}_1 + (\alpha + \mu + \sigma) \dot{I}_2 \end{aligned}$$

Substituting and simplifying the expressions for $\dot{I}_1$ and $\dot{I}_2$ gives

$$\begin{aligned} \dot{L} &= \alpha[\beta S I_2 - (\alpha + \mu + \sigma) I_1] + (\alpha + \mu + \sigma)[\alpha I_1 - (\mu + \delta + \gamma) I_2] \\ \dot{L} &= \alpha\beta S I_2 - (\alpha + \mu + \sigma)(\mu + \delta + \gamma) I_2. \end{aligned}$$

Since $S = \frac{\Lambda}{\mu}$ it follows that

$$\dot{L} = (\alpha + \mu + \sigma)(\mu + \delta + \gamma) I_2 [R_0 - 1].$$

Thus $\dot{L} \leq 0$ with equality when $I_2 = 0$. Hence by LaSalle's extension to Lyapunov principle, the limit set for each solution is contained in the largest invariant set for which $I_2 = 0$.

Theorem 3.5: (Global stability of endemic equilibrium). The endemic equilibrium for the system is globally asymptotically stable if $R_0|_{\sigma=0} > 1$.

Proof: Let the associated endemic equilibrium of model (1) – (4) and $R_0|_{\sigma=0} > 1$ exist. Consider the Lyapunov function:

$$\begin{aligned} V &= S - S^{**} - S^{**} \ln \left(\frac{S}{S^{**}} \right) + I_1^{**} - I_1^{**} - \ln \left(\frac{I_1}{I_1^{**}} \right) + \frac{(\alpha + \mu)}{\alpha} \left[I_2^{**} - I_2^{**} - \ln \left(\frac{I_2}{I_2^{**}} \right) \right] \\ \dot{V} &= \dot{S} - \frac{S^{**}}{S} \dot{S} + \dot{I}_1 - \frac{I_1^{**}}{I_1} \dot{I}_1 + \frac{(\alpha + \mu)}{\alpha} \left[\dot{I}_2 - \frac{I_2^{**}}{I_2} \dot{I}_2 \right] \\ \dot{V} &= \Lambda - \beta S I_2 - \mu S - \frac{S^{**}}{S} [\Lambda - \beta S I_2 - \mu S] + \beta S I_2 - (\alpha + \mu) I_1 - \frac{I_1^{**}}{I_1} [\beta S I_2 - (\alpha + \mu) I_1] \\ &\quad + \frac{(\alpha + \mu)}{\alpha} \left[\alpha I_1 - (\mu + \delta + \gamma) I_2 - \frac{I_2^{**}}{I_2} \{ \alpha I_1 - (\mu + \delta + \gamma) I_2 \} \right] \\ \dot{V} &= \Lambda \left(1 - \frac{S^{**}}{S} \right) - \mu S \left(1 - \frac{S^{**}}{S} \right) + \beta S^{**} I_2 - \beta S I_2 \frac{I_1^{**}}{I_1} + (\alpha + \mu) I_1^{**} - \frac{(\alpha + \mu)(\mu + \delta + \gamma) I_2}{\alpha} - (\alpha + \mu) I_1 \frac{I_2^{**}}{I_2} + \\ &\quad \frac{(\alpha + \mu)(\mu + \delta + \gamma) I_2^{**}}{\alpha}. \end{aligned}$$

At the endemic steady state, the following relations obtained from model (1) – (4) hold:

$$\begin{aligned} \Lambda &= \beta S^{**} I_2^{**} + \mu S^{**} \\ (\alpha + \mu) &= \frac{\beta S^{**} I_2^{**}}{I_1^{**}} \\ (\mu + \delta + \gamma) &= \frac{\alpha I_1^{**}}{I_2^{**}}. \end{aligned}$$

Substituting above relations into the Lyapunov function gives:

$$\begin{aligned} \dot{V} &= (\beta S^{**} I_2^{**} + \mu S^{**}) \left(1 - \frac{S^{**}}{S} \right) - \mu S \left(1 - \frac{S^{**}}{S} \right) + \beta S^{**} I_2 - \beta S I_2 \frac{I_1^{**}}{I_1} + \left(\frac{\beta S^{**} I_2^{**}}{I_1^{**}} \right) I_1^{**} - \\ &\quad \left(\frac{\beta S^{**} I_2^{**}}{I_1^{**}} \right) \left(\frac{\alpha I_1^{**}}{I_2^{**}} \right) \left(\frac{I_2}{\alpha} \right) - \left(\frac{\beta S^{**} I_2^{**}}{I_1^{**}} \right) I_1 \frac{I_2^{**}}{I_2} + \left(\frac{\beta S^{**} I_2^{**}}{I_1^{**}} \right) \left(\frac{\alpha I_1^{**}}{I_2^{**}} \right) \left(\frac{I_2^{**}}{\alpha} \right). \end{aligned}$$

Simplifying gives

Further simplification gives

$$\begin{aligned} \dot{V} &= \mu S^{**} \left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right) + 3 \beta S^{**} I_2^{**} - \frac{\beta S^{**2} I_2^{**}}{S} - \beta S I_2 \frac{I_1^{**}}{I_1} - \frac{\beta S^{**} I_1 I_2^{**2}}{I_1^{**} I_2} \\ \dot{V} &= \mu S^{**} \left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right) + \beta S^{**} I_2^{**} \left(3 - \frac{S^{**}}{S} - \frac{S I_1^{**} I_2}{S^{**} I_1 I_2^{**}} - \frac{I_1 I_2^{**}}{I_1^{**} I_2} \right). \end{aligned}$$

Since arithmetic mean is less than or equal to geometric mean,

$$\mu S^{**} \left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right) \leq 0 \text{ and } \left(3 - \frac{S^{**}}{S} - \frac{SI_1^{**}I_2}{S^{**}I_1I_2^{**}} - \frac{I_1I_2^{**}}{I_1^{**}I_2} \right) \leq 0.$$

Thus, $\dot{V} \leq 0$ if and only if $S = S^{**}, I_1 = I_1^{**}, I_2 = I_2^{**}$. Hence, the largest compact invariant subset of the set where $\dot{V} = 0$ is the singleton $(S, I_1, I_2) = (S^{**}, I_1^{**}, I_2^{**})$. It follows by LaSalle's Invariance Principle [8], that every solution to the equations of the model (1) approaches the associated unique endemic equilibria of the model as $t \rightarrow \infty$ for $R_0|_{\sigma=0} > 1$.

3.6 Sensitivity analysis

Sensitivity analysis is used to determine dependencies between input parameters and results of the model. Sensitivity analysis helps to determine how a small change in parameters affects state variables over time. The normalised forward sensitivity index of a variable, w , that depends on a parameter, q , is defined as: $\Gamma_q^w = \frac{\partial w}{\partial q} \times \frac{q}{w}$.

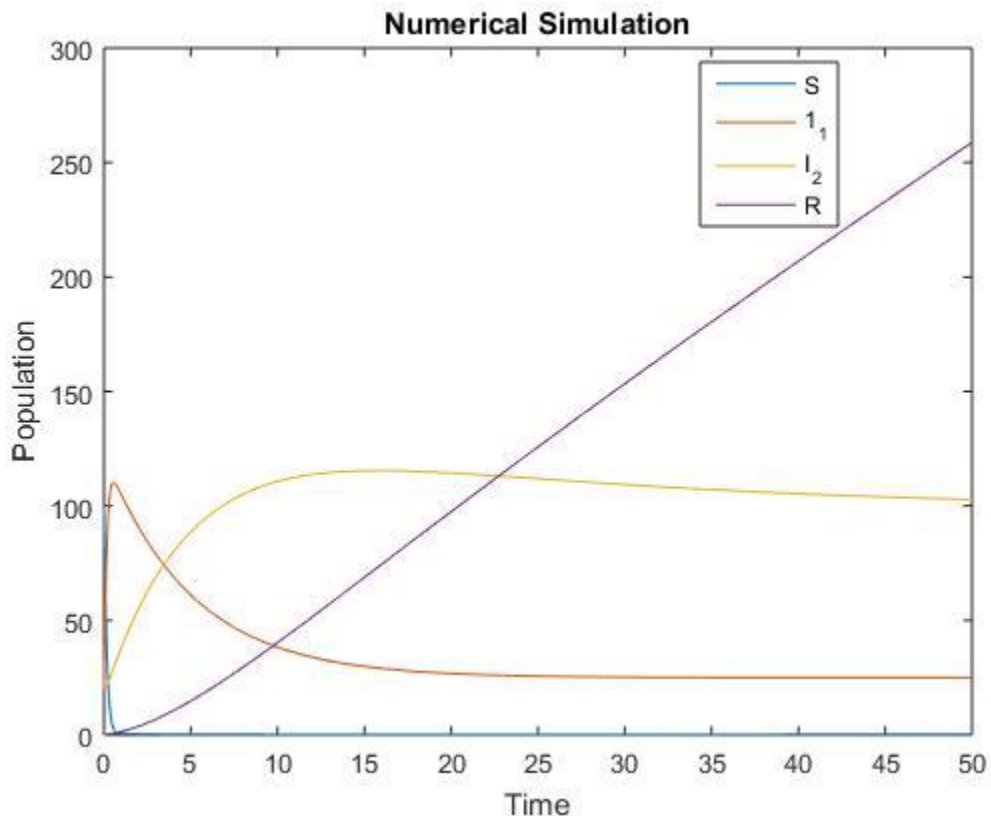
Sensitivity index of the basic reproduction number with respect to the model parameters are computed below:

parameter	$\Gamma_{parameter}^{R_0}$
β	1
Λ	1
α	$\frac{(\mu + \sigma)}{(\alpha + \mu + \sigma)}$
σ	$\frac{-\sigma}{(\alpha + \mu + \sigma)}$
δ	$\frac{-\delta}{(\mu + \delta + \gamma)}$
γ	$\frac{-\gamma}{(\mu + \delta + \gamma)}$
μ	$-\frac{R_0}{\mu} \left[\frac{1}{(\mu + \delta + \gamma)} + \frac{(\alpha + 2\mu + \sigma)}{(\alpha + \mu + \sigma)} \right]$

$\Gamma_{R_0}^d = 1$ revealed that a 10 percent in transmission rate corresponds to 10 percent increase in the basic reproduction number. Sensitivity analysis gives an insight into the required strategy to control the spread of the disease.

3.7 Numerical simulation

The dynamics of the model depends on the input parameters. The input parameters are approximated from most communicable diseases. $\Lambda = 5, \beta = 0.3, \mu = 0.0005, \sigma = 0.01, \delta = 0.0005, \alpha = 0.2, \gamma = 0.05$ with $S(0) = 100, I_1(0) = 20, I_2(0) = 20, R(0) = 0$. The numerical simulation is shown below using MATLAB. The interplay between I_1 and I_2 class will determine whether the susceptible class will have much increase or the recovered class.



4 Conclusion

In this paper, we presented a two-infection stage mathematical model. The basic reproduction number of the model is determined. The disease-free equilibrium point is found to be globally asymptotically stable if $R_0 < 1$. There is an existence of endemic equilibrium provided that $R_0 > 1$. However, the endemic equilibrium is globally asymptotically stable if $R_0|_{\sigma=0} > 1$. Sensitivity analysis shows that the recruitment rate, transmission rate and progression rate from the first infection stage to the second infection stage have a direct proportionality on the basic reproduction number. Numerical simulation with appropriate parameters is carried out to give possible dynamics of the model.

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