On the effect of saturation factor and effective contact rate on the transmission dynamics of Ebola

[1]Loyinmi, C. Adedapo, [2]Shittu, M. Sodiq, & [3]Ijaola, A. Lateef.

[1,2]Department of Mathematics, Tai Solarin University of Education, Ijagun. Ogun State.
[3]Department of Mathematics, Federal University of Agriculture, Abeokuta. Ogun State.
✉: loyinmiac@tasued.edu.ng; + (234) 8056751556

Received: 27:04:2025 **Accepted: 14:05:2025** **Published: 14:05:2025**

Abstract:
In this study we propose a modified SEQIR model to further investigate the dynamics of Ebola virus. A deterministic and stochastic framework was considered to verify the implication(s) of saturation coefficient on the transmission dynamics of Ebola. Necessary qualitative analysis: Existence and Uniqueness, boundedness, local and global stability, at disease - free equilibrium were considered to validate the epidemiological feasibility of the model. The reproduction number was found to be less than unity. Furthermore, the effective contact rate of the disease and saturation coefficient rates were varied within the model to validate the effects of these parameters on the transmission dynamics of the infection and results from numerical simulations using assumed (fitted) values indicate the overall effects of the saturation coefficient rate in phasing out the disease in the long run when moderated.

Keywords: **Ebola virus disease (EVD), saturation effects, stability analysis, stochastic differential equation.**

1. Introduction

The Ebola virus disease (EVD), which was first identified in 1997 and is named after a river in former Zaire is a severe viral hemorrhagic infectious fever. It is a member of the RNA virus family known as filovirus and research has demonstrated that fruit bats in the Pteropodidae family-known to be the virus's natural host-are the conduit for the virus's spread. Personal contact with body tissues, semen or fluids, from infectious individuals dead or alive is the primary method by which the Ebola virus disease is transmitted [1-3]. EVD causes symptoms like the flu initially, but it soon progresses to external and internal bleeding, rash, diarrhoea and vomiting. Once inside the body, the virus begins to attack cells of the immune system, which are liver and blood cells, which are responsible for protecting the body from infection. Those who are infected with the virus become contagious after 21 days during incubation [4]. The virus targets the liver and kidneys, two vital organs, as the fever rises. This results in significant bleeding, tissue destruction, respiratory arrest, shocks, and ultimately death. About 50% to 90% of those infected die between 10 days after acquiring the disease, according to the World Health Organization [5-8]. Due to two distinct Ebola virus strains (Ebola Sudan and Ebola Zaire) that were discovered in those regions, twelve Ebola outbreaks were documented in 2003 in Gabon, Sudan, Uganda and Congo. This nation includes Guinea, Nigeria, Sierra Leone, Senegal, Liberia, Mali, United States, Spain and the United Kingdom reported the biggest outbreaks. Throughout this era, there were 26,724 cases of infections and 11065 deaths [9-13]. Six countries Nigeria, Mali, Senegal, the United Kingdom, Spain, and the United States have been declared free of the Ebola virus illness; nonetheless, there is a recently reported case of Ebola from a country where the disease is widely and actively transmitted [14]. In September 2022, the Uganda Ministry of Health, along with AFRO and WHO, verified the EVD outbreak in Mubende Region. The type of EVD that was confirmed was Sudan virus disease (SVD), and a death case was reported. This case involved a 24-year-old man who lived in a village called Ngabano, Madudu in the District of Mubende. It was seen that the guy had a high temperature, stomach discomfort, diarrhea, and blood in his vomit. There were 142 confirmed cases, 87 recovered cases, and 55 deaths (CFR: 39%) overall [3, 15].

Differential equations have been vital tools in describing the dynamics of infectious diseases [9-20]. and many more dynamical concepts in sciences and engineering. Results from many of this research have positively affected prompt responses to diseases outbreaks [21 - 24].

A significant contribution to the modelling of infectious disease types and transmission was made by Kermack and Mckendrick [22]. This concept was called the SIR model, and several researchers have since expanded it and used it to study a variety of infectious illnesses [16-26].

Researchers are very interested in the mechanics of the transmission of Ebola fever sickness. Astacio and colleagues employed the $SEIR$ and SIR models to mimic two Ebola outbreaks on the one that transpired in Yambuku, Zaire, in 1976 and Kikwit, Zaire, in 1995 [24-25]. The behavior of these models was estimated using the per-capita effective contact rate of a person contracting the disease and the per-capita death rate of infected individuals. The basic reproductive number R_0, determines how infectiously the disease invades a community [9-13, 16-19, 26]. The study asserted that Ebola is not as infectious as previously thought and that the results of their simulations would provide scientists with information that will

enable them to lessen the number of people who may die in the event of future outbreaks. To analyse the stability of the infection-Free Equilibrium State and manage the dynamics of Ebola transmission, some authors created models. They also devised optimum control mechanisms for monkey pox [27-28].

Despite existing literature immensely addressed transmission dynamics and possible control measures to the invasion of the deadly infection, the implications of extensive study on the transmission dynamics of the disease to further propose effective intervention measures to tame the deadly disease cannot be over emphasized. Hence, we present a modified SEQIR to further emphasize the overall implications of the contact rate and saturation coefficient on the transmission dynamics of Ebola virus.

2.0 MATERIALS AND METHODS

2. 1. Model Formulation

To properly understand the dynamics of the Ebola virus infection, the model made some assumptions in the modification of five compartmental classes which are S= Susceptible class, E = Exposed class, Q = Quarantine, I= Infected class and R = recovered compartment, where all classes are equipped with biological parameter.

$$\Psi = \frac{\beta IS}{1+aI}$$

Is the model force of infection, where $\boldsymbol{\beta}$ is the effective contact rate, $\boldsymbol{I}$ is the infection compartment, $\boldsymbol{S}$ is the susceptible class, and $\boldsymbol{a}$ is the saturation factor to lessen the infection severity?

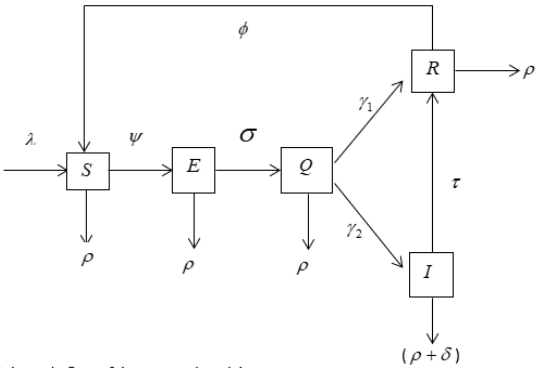

Figure 1: Schematic flow of the proposed model

The following sets of equations are derived from the schematic flow (Figure 1).

$$\begin{aligned}
\frac{dS}{dt} &= \lambda - \rho S - \Psi S + \phi R \\
\frac{dE}{dt} &= \Psi S - \rho E - \sigma E \\
\frac{dQ}{dt} &= \sigma E - Q(\gamma_1 + \gamma_2) - \rho Q \\
\frac{dI}{dt} &= \gamma_2 Q - I(\rho + \delta) - \tau I \\
\frac{dR}{dt} &= \gamma_1 Q + \tau I - R\rho - \phi R
\end{aligned} \qquad (1)$$

$$S(t) \geq 0, E(t) \geq 0, Q(t) \geq 0, I(t) \geq 0, R(t) \geq 0.$$

The following sets of equations are derived from the schematic flow (Figure 1).

Table 1. Parameters values used in the numerical validation

Parameters	Description	Value	Sources
λ	Rate of recruitment	0.09	Assumed
Ψ	Force of infection	0.091196	Assumed
σ	Progression rate from Exposed to quarantine	0.010	Assumed
ρ	Natural death rate	0.0017256	Assumed
γ_1	Progress rate from quarantine to recover	0.05075	Assumed
γ_2	Progression rate from quarantine to infected	0.0087	Assumed
ϕ	Relapse rate	0.2062	Assumed
a	Saturation factor	0 – 1	Calibrated
β	Effective contact rate	0 – 1	Calibrated
τ	Rate of recovery from infected class	0.005	Loyinmi et al., 2022
δ	Disease induced death	0.00074	Loyinmi et al., 2022

2.2 Qualitative Analysis of the proposed model

2.2.1. Existence and Uniqueness of solution

Using the Lipchitz condition, from the systems of equation generated from the schematic diagram.

$$\begin{aligned}
A_1 &= \lambda - \rho S - \Psi S + \varphi R \\
A_2 &= \Psi S - \rho E - \sigma E \\
A_3 &= \sigma E - Q(\gamma_1 + \gamma_2) - \rho Q \\
A_4 &= \gamma_2 Q - I(\rho + \delta) - \tau I \\
A_5 &= \gamma_1 Q + I - R\rho - \phi R
\end{aligned} \qquad (2)$$

Theorem 1: Let K denote the region $0 \leq \chi \leq M$, then the systems of equations (2) possess a unique solution if and only if $\frac{\partial G_i}{\partial b_j}$ are continuous and bounded in K, for $i \neq j$

Proof

$$\left|\frac{\partial A_1}{\partial S}\right| = \left|-(\Psi+\rho)\right| \prec \infty, \left|\frac{\partial A_1}{\partial I}\right| = |0| \prec \infty, \left|\frac{\partial A_1}{\partial R}\right| = |\varphi| \prec \infty, \left|\frac{\partial A_1}{\partial E}\right| = |0| \prec \infty, \left|\frac{\partial A_1}{\partial Q}\right| = |0| \prec \infty,$$

$$\left|\frac{\partial A_2}{\partial S}\right| = \left|-(\Psi)\right| \prec \infty, \left|\frac{\partial A_2}{\partial I}\right| = |0| \prec \infty, \left|\frac{\partial A_2}{\partial R}\right| = |0| \prec \infty, \left|\frac{\partial A_2}{\partial E}\right| = |\rho+\sigma| \prec \infty, \left|\frac{\partial A_2}{\partial Q}\right| = |0| \prec \infty,$$

$$\left|\frac{\partial A_3}{\partial S}\right| = |0| \prec \infty, \left|\frac{\partial A_3}{\partial I}\right| = |\gamma| \prec \infty, \left|\frac{\partial A_3}{\partial R}\right| = |0| \prec \infty, \left|\frac{\partial A_3}{\partial E}\right| = |\sigma| \prec \infty, \left|\frac{\partial A_3}{\partial Q}\right| = \left|-(\gamma_1+\gamma_2+\rho)\right| \prec \infty,$$

$$\left|\frac{\partial A_4}{\partial S}\right| = |0| \prec \infty, \left|\frac{\partial A_4}{\partial I}\right| = \left|-(\rho+\delta+\tau)\right| \prec \infty, \left|\frac{\partial A_4}{\partial R}\right| = |0| \prec \infty, \left|\frac{\partial A_4}{\partial E}\right| = |0| \prec \infty, \left|\frac{\partial A_4}{\partial Q}\right| = |0| \prec \infty,$$

$$\left|\frac{\partial A_5}{\partial S}\right| = |0| \prec \infty, \left|\frac{\partial A_5}{\partial I}\right| = |\tau| \prec \infty, \left|\frac{\partial A_5}{\partial R}\right| = \left|-(\rho+\varphi)\right| \prec \infty, \left|\frac{\partial A_5}{\partial E}\right| = |0| \prec \infty, \left|\frac{\partial A_5}{\partial Q}\right| = |\Psi| \prec \infty,$$

Clearly from the partial derivative above of system (2), the solutions to the systems of equations exist, unique and remain bounded.

2.2.2. Boundedness of solution

Here, we examine and confirm the mathematical and epidemiological soundness of the model. Additionally, that the relevant partitions' solutions are non-negative.

The total population of human compartment.

$$N = S + E + Q + I + R \qquad (3)$$

From the derivatives of sums, we have

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dQ}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \qquad (4)$$

Simplifying (4) we obtain;

$$\frac{dN}{dt} = \lambda - \rho(N) = \lambda - \rho N \rightarrow \frac{dN}{dt} + \rho N = \lambda,$$

$$N(t) = \frac{\lambda_H}{\mu_H} + ce^{-\mu_H t}$$

which at $t \rightarrow \infty$ becomes

$$N(t) \leq \frac{\lambda}{\rho} \qquad (5)$$

From (5) we have demonstrated the epidemiological boundedness of the model.

2.3 Fundamental reproduction number

This number represents the typical number of secondary infections in the E(t) compartment in a fully vulnerable group that were caused by an infected person already in the I(t). The "next generation matrix method" will be used to calculate R_0. Let F represent the matrix of a new infection while V is the matrix of secondary infection.

$$F_1 = \Psi S, \quad F_2 = 0$$

$$\begin{bmatrix} \frac{\partial F_1}{\partial E} & \frac{\partial F_2}{\partial I} \\ \frac{\partial F_2}{\partial E} & \frac{\partial F_2}{\partial I} \end{bmatrix} = \begin{bmatrix} 0 & \beta S \\ 0 & 0 \end{bmatrix}$$

$$V_1 = -(\mu + \theta)E, \quad V_2 = \theta E - (\mu + \delta + \tau)I$$

$$\begin{bmatrix} \frac{\partial V_1}{\partial E} & \frac{\partial V_1}{\partial I} \\ \frac{\partial V_2}{\partial E} & \frac{\partial V_2}{\partial I} \end{bmatrix} = \begin{bmatrix} -(\mu + \theta) & 0 \\ \theta & -(\mu + \delta + \tau) \end{bmatrix}$$

Using $R_0 = \chi(FV^{-1})$, where χ is the spectral radius of FV^{-1},

$$V^{-1} = \frac{Adj(V)}{|V|}, \; Adj(V) = \begin{bmatrix} -(\rho + \delta + \tau) & 0 \\ \sigma & -(\rho + \sigma) \end{bmatrix}, \; |V| = |(\rho + \delta + \tau)(\rho + \sigma)|$$

Hence $V^{-1} = \begin{bmatrix} \frac{-1}{(\rho + \sigma)} & 0 \\ \frac{-\sigma}{(\rho + \delta + \tau)(\rho + \sigma)} & \frac{-1}{(\rho + \delta + \tau)} \end{bmatrix}$,

$$FV^{-1} = \begin{bmatrix} \frac{-\beta\sigma S}{(\rho + \delta + \tau)(\rho + \sigma)} & \frac{-\beta S}{(\rho + \delta + \tau)} \\ 0 & 0 \end{bmatrix}$$

Where k is the Eigenvalue;

$$\left| FV^{-1} - kI \right| = \begin{vmatrix} \frac{-\beta\sigma S}{(\rho + \delta + \tau)(\rho + \sigma)} - k & \frac{-\beta S}{(\rho + \delta + \tau)} \\ 0 & 0 - k \end{vmatrix}$$

The spectral radius of FV^{-1} is $\frac{\beta\sigma S}{(\rho + \delta + \tau)(\rho + \sigma)}$. (6)

Note that $S = \frac{\lambda}{\mu}$, replacing S, the reproduction number become $R_0 = \frac{\beta\sigma\lambda}{\rho(\rho + \delta + \tau)(\rho + \sigma)}$ (7)

2.4. Local Stability Analysis.

Theorem 3: If $R_0 < 1$, then the model is locally asymptotically stable.
Proof

Let.

$$F_1 = \lambda - \rho S - \Psi S + \varphi R, \; F_2 = \Psi S - \rho E - \sigma E, \; F_3 = \sigma E - Q(\gamma_1 + \gamma_2) - \rho Q$$
$$F_4 = \gamma_2 Q - I(\rho + \delta) - \tau I, \; F_4 = \gamma_1 Q + \tau I - \rho R - \phi R$$

Using the Jacobi function as $J(S, E, Q, I, R)$

$$J = \begin{bmatrix} \frac{\partial F_1}{\partial S} & \frac{\partial F_1}{\partial E} & \frac{\partial F_1}{\partial Q} & \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial R} \\ . & . & . & . & . \\ . & . & . & . & . \\ . & . & . & . & . \\ \frac{\partial F_5}{\partial S} & \frac{\partial F_5}{\partial E} & \frac{\partial F_5}{\partial Q} & \frac{\partial F_5}{\partial I} & \frac{\partial F_5}{\partial R} \end{bmatrix} \qquad (8)$$

$$J = \begin{bmatrix} -(\rho + \Psi) & 0 & 0 & \beta S & \phi \\ \Psi & -(\sigma + \rho) & 0 & \beta S & 0 \\ 0 & \sigma & -(\gamma_1 + \gamma_2 + \rho) & 0 & 0 \\ 0 & 0 & \gamma_2 & -(\rho + \delta + \tau) & 0 \\ 0 & 0 & \gamma_1 & \tau & -(\rho + \phi) \end{bmatrix} \qquad (9)$$

At disease free equilibrium

$$J(E^0) = \begin{bmatrix} -\rho & 0 & 0 & \beta S & \phi \\ 0 & -(\sigma + \rho) & 0 & \beta S & 0 \\ 0 & \sigma & -(\gamma_1 + \gamma_2 + \rho) & 0 & 0 \\ 0 & 0 & \gamma_2 & -(\rho + \delta + \tau) & 0 \\ 0 & 0 & \gamma_1 & \tau & -(\rho + \phi) \end{bmatrix} \qquad (10)$$

Solving (10) steadily using Row reduction and Gaussian elimination method we obtain.

$$k_1 = -\rho, \quad k_2 = -(\gamma_1 + \gamma_2 + \rho), \quad k_3 = -(\rho + \delta + \tau),$$
$$k_4 = \left[(\rho + \delta + \tau) - \frac{\sigma\beta S}{(\rho + \sigma)}\right], \quad k_5 = -(\rho + \varphi).$$

From κ_4 it is easy to verify that our reproduction number is less than unity (1), Note that k_4 can be written as;

$$k_4 = \left[(\rho + \delta + \tau) - \frac{\sigma\beta S}{(\rho + \sigma)}\right] \leq -(\rho + \delta + \tau)(1 - R_0) \qquad (11)$$

From (11) it suffices that $K_4 < 0$ if $R_0 < 1$. Therefore, the negativity of all the eigenvalues implies the stability of the system.

2.5 Global stability of D.F.E

$$\begin{aligned}
\frac{dS}{dt} &= \lambda - \rho S + \phi R \\
\frac{dE}{dt} &= \varphi S - \rho E - \sigma E \\
\frac{d\varphi}{dt} &= \sigma E - \varphi(\gamma_1 + \gamma_2) - \rho\varphi \\
\frac{dI}{dt} &= \gamma_2\varphi - I(\rho + \delta) - \tau I \\
\frac{dF}{dt} &= \gamma_1\varphi - \tau I - R(\rho + \varphi)
\end{aligned} \tag{12}$$

Let $X = \{S\}, Y = \{E, \varphi, I\}$.

1. We say the point $E^0 = \{X^0, 0\}$ is stable asymptotically if $R_0 \prec 1$ and the following two conditions are satisfied.
2. $\frac{dX}{dt} = F[X, 0]$, then E^0 is asymptotically stable globally.
3. There exist $G[X, Y] = Dy - G(x, y)\,; G(x, y) \geq 0$ for the first condition.

   $\frac{dS}{dt} = \lambda - \rho S \rightarrow \frac{dS}{dt} + \rho S = \lambda$ which is solvable by integrating factor methods such as;

$$Se^{\rho t} = \int_0^t \lambda . e^{\rho t} dt + c \rightarrow Se^{\rho t} = \frac{\lambda}{\rho} e^{\rho t} + c \rightarrow S(t) = \frac{\lambda}{\rho}$$

as $t \rightarrow \infty$.

The first condition is satisfied. X_0 is globally asymptotically stable.

The Second Condition

$$G(x, y) = \begin{bmatrix} \varphi S - E(\varphi + \sigma) \\ \sigma E - \varphi(\gamma_1 + \gamma_2 + \rho) \\ \gamma_2\varphi - I(\rho + \delta + \tau) \end{bmatrix}$$

$$A(G, x, y) = \begin{bmatrix} \frac{dF_1}{dE} & \frac{dF_1}{dQ} & \frac{dF_1}{dI} \\ \frac{dF_2}{dE} & \frac{dF_2}{dQ} & \frac{dF_2}{dI} \\ \frac{dF_3}{dE} & \frac{dF_3}{dQ} & \frac{dF_3}{dI} \end{bmatrix} = \begin{bmatrix} -(\rho + \sigma) & 0 & \beta S \\ \sigma & -(\gamma_1 + \gamma_2 + \rho) & 0 \\ 0 & \gamma_2 & -(\rho + \delta + \tau) \end{bmatrix}$$

$$= \begin{bmatrix} -(\mu_H + \theta_H) & 0 & BS \\ \theta_H & -(\alpha_1 + \alpha_2 + \mu_H) & 0 \\ 0 & \alpha_2 & -(\mu_H + \delta + \tau) \end{bmatrix} \tag{13}$$

Since $S = N = \frac{\lambda}{\rho}$, this implies $\hat{G}(x, y) = \begin{bmatrix} \beta\frac{\lambda}{\rho}S^0 - S \\ 0 \\ 0 \end{bmatrix}$

Obviously, $\hat{G}(x, y) \geq 0$, Since $S \geq 0$ The Ebola virus can now be completely eradicated from the human population, ensuring global stability.

2.6. Transition probability using stochastic differential equation

The resulting stochastic differential equation has the following form:

$$dX(t) = F(x(t), t)dt + g(x(t), t)dt \tag{14}$$

Where; $F(x(t), t)$ is the drift deterministic part of the model. $g(x(t), t)$ is the diffusion part in which the transition probability is based.

$F(x(t), t) = E\left(\frac{\Delta x}{\Delta \tau}\right)$ and $g(x(t), t) = E\left[\frac{\Delta x.(\Delta x)^T}{\Delta T}\right] = v^{\frac{1}{2}}$,

Since time is constant, $S(t)$ and $I(t)$ are continuous random variable.

Then $\Delta S = S(\Delta t + t) - S(t)$ and $\Delta I = I(\Delta t + t) - I(t)$

Let $X = [x_1, x_2]^T$, where x_1 and x_2 corresponds to $S(t)$ and $I(t)$ of the model.

We can formulate the transition probability from the deterministic part.

$$E(\Delta x) = \sum_{i=1}^{4} p_1 \Delta x_1 + p_2 \Delta x_2 + \ldots p_n \Delta x_n .$$

Expectation

$$E[\Delta x.[\Delta x]] = \sum_{i=1}^{4} p_1 \Delta x_1 .[\Delta x_1]^T + p_2 \Delta x_2 .[\Delta x_2]^T + p_3 \Delta x_3 .[\Delta x_3]^T + \ldots$$

Co-variance.

Table 2: The transition probabilities

Potential Modifications	The likelihood (Probability)	Description (Details)
$\Delta x_1 = [1, 0]^T$	$\lambda S \Delta T$	Emergence of Susceptible
$\Delta x_2 = [-1, +1]^T$	$(\mu_H S + \theta_H E_H + \alpha_2\theta)\Delta T$	Susceptible got infected
$\Delta x_3 = [-1, 0]^T$	$\mu_H S \Delta T$	Natural death of susceptible
$\Delta x_3 = [-1, 0]^T$	$\tau\, l \Delta T$	Susceptible recovered

Substituting the values of P_1, P_2, P_3 and P_4 into the equation

$$E[\Delta x] = P_1 \begin{bmatrix} 1 \\ 0 \end{bmatrix} + P_2 \begin{bmatrix} -1 \\ 1 \end{bmatrix} + P_3 \begin{bmatrix} -1 \\ 0 \end{bmatrix} P_4 \begin{bmatrix} 0 \\ -1 \end{bmatrix} = \begin{bmatrix} P_1 - P_2 + P_3 \\ P_2 - P_4 \end{bmatrix}$$

$$E[\Delta x] = \begin{bmatrix} \lambda S\Delta T - (\rho S + \sigma E + \gamma_2 I - \rho S\Delta T) \\ (\rho S + \sigma E + \gamma_2 I)\Delta T - \tau I\Delta T \end{bmatrix} \quad (15)$$

For the covariance

$$E[\Delta x.[\Delta x]^T] = P_1\begin{bmatrix}1\\0\end{bmatrix}.[1 \quad 0] + P_2\begin{bmatrix}-1\\1\end{bmatrix}.[-1 \quad 1] + P_3\begin{bmatrix}-1\\0\end{bmatrix}.[-1 \quad 0] + P_4\begin{bmatrix}0\\-1\end{bmatrix}.[0 \quad -1]$$

$$E[\Delta x.[\Delta x]^T] = P_1\begin{bmatrix}1 & 0\\0 & 0\end{bmatrix} + P_2\begin{bmatrix}1 & -1\\-1 & 1\end{bmatrix} + P_3\begin{bmatrix}1 & 0\\0 & 0\end{bmatrix} + P_4\begin{bmatrix}0 & 0\\0 & 1\end{bmatrix}$$

$$E[\Delta x.[\Delta x]^T] = \begin{bmatrix}P_1 + P_2 + P_3 & -P_2\\ -P_2 & P_2 + P_4\end{bmatrix}$$

$$E[\Delta x.[\Delta x]^T] = \begin{bmatrix}\lambda S\Delta T + (\rho S + \sigma E + \gamma_2 I)\Delta T + \rho S\Delta T & -(\varphi S + \rho E + \gamma_2 I)\\ -(\varphi S + \sigma E + \gamma_2 I) & (\varphi S + \sigma E + \gamma_2 I)\Delta T + \tau I\Delta T\end{bmatrix}$$

Since $E\left[\frac{\Delta x}{\Delta T}\right] = F(x(t),t)dt$,

$$E\left[\frac{\Delta x.(\Delta x)^T}{\Delta T}\right] = V^{\frac{1}{2}} = g(x(t),t)$$

Now we get $V^{\frac{1}{2}}$, $V^{\frac{1}{2}} = \frac{1}{\beta}\begin{bmatrix}\delta + \sigma & p\\ p & w + \sigma\end{bmatrix}$

Where

$$\sigma = \sqrt{\delta w - p^2} \qquad and \qquad \beta = \sqrt{\delta + w + 2\sigma}$$

$$\begin{aligned}\delta &= (\lambda S + \rho)\Delta T + (\varphi S + \sigma E + \gamma_2 I)\Delta T\\ p &= -(\varphi S + \sigma E + \gamma_2 I)\Delta T\\ w &= (\varphi S + \sigma E + \gamma_2 I)\Delta T + \tau I\Delta T\end{aligned} \quad (16)$$

Therefore,

(17)

$$\theta = \sqrt{[(\lambda S + \mu_H) + (\sigma S + \sigma E + \gamma_2 I)](\varphi S + \sigma E + \gamma_2 I) + \tau I\Delta T(\rho + \lambda) - P^2}$$

$$\theta = \sqrt{\begin{bmatrix}(\lambda S + \rho)(\varphi S + \sigma E + \gamma_2 I) + (\lambda S + \rho)(\tau I) + (\varphi S + \sigma E + \gamma_2 I)\\ (\varphi S + \sigma E + \gamma_2 I) + \tau I(\varphi S + \sigma E + \gamma_2 I) - p^2) - P^2\end{bmatrix}}$$

$$\theta = \sqrt{\begin{bmatrix}(\lambda S + \rho)(\varphi S + \sigma E + \gamma_2 I) + (\varphi S + \sigma E + \gamma_2 I)^2 + (\lambda S + \rho)(\tau I)\\ + \tau I(\varphi S + \sigma E + \gamma_2 I) - (\varphi S + \sigma E + \gamma_2 I)^2\end{bmatrix}}$$

Then.

$$\theta = \sqrt{[(\lambda S + \rho)(\varphi S + \sigma E + \gamma_2 I) + \tau I(\varphi S + \sigma E + \gamma_2 I) + (\lambda S + \rho)\tau I]}$$

$$\beta = \sqrt{\delta + w + 2\sigma}$$

$$\beta = \sqrt{+(\varphi S + \sigma E + \gamma_2 I) + (\varphi S + \sigma E + \gamma_2 I) + \tau I + (\lambda S + \rho) + 2\sigma}$$

$$\beta = \sqrt{(\lambda S + \rho) + \tau I + 2(\varphi S + \sigma E + \gamma_2 I) + 2\sigma}$$

Since $F(x(t),t)$ & $g(x(t),t)$ has been stated clearly then, the stochastic differential equation for the Ebola virus is given thus:

$$dX(t) = F(x(t),t) + g(x(t),t)dw(t)$$

Where $w(t) = w_1(t), w_2(t)$ represent independent Wiener's process for the compartment.

$X(t) = [X_1, X_2]$, Where X_1 and X_2 translate $S(t)$ and $I(t)$. Therefore,

$$\frac{dS}{dt} = \left(S(\lambda - \rho) - (\varphi S + \sigma E + \gamma_2 I)dt\right) + \frac{\delta + \sigma}{\beta}dw_1(t) + \frac{p}{\beta}dw_1(t).$$

$$\frac{dI}{dt} = \left((\varphi S + \sigma E + \gamma_2 I) - \tau I\Delta T\right)dt + \frac{p}{\beta}dw_1(t) + \frac{w + \sigma}{\beta}dw_2(t).$$

Now we define.

$$A(t) = \int_0^t\left(\frac{\delta + \sigma}{\beta}\right)dw_1(t) + \int_0^t\left(\frac{p}{\beta}\right)dw_2(t) = \int_0^t\left(\frac{\delta(S) + \sigma(S)}{\beta(S)}\right)dw_1(S) + \int_0^t\left(\frac{p(S)}{\beta(S)}\right)dw_2(S)$$

Hence,

$$\langle A(t)\rangle = \int\left(\frac{\delta(S) + \sigma(S)}{\beta(S)}\right)^2 dS + \int_0^t\left(\frac{p(S)}{\beta(S)}\right)^2 dS = \int\left(\frac{\delta(S)^2 + 2\delta(S)\sigma(S) + \sigma(S)^2 + p(S)^2}{\beta(S)^2}\right)dS$$

$$= \int[(\lambda S + \rho S) + (\varphi S + \sigma S + \gamma_2 I)]^2 + 2(\lambda S + \rho)(\sigma(S)) - (\rho + \sigma E + \gamma_2 I)^2$$

$$+ \sqrt{(\lambda S + \rho S)(\varphi + \sigma S + \gamma_2 I) + \tau I(\lambda S + \rho S) + (\varphi S + \sigma E + \gamma_2 I)^2}$$

$$+ (\varphi S + \sigma E + \gamma_2 I)^2 + \left(\sqrt{(\lambda S + \rho S) + \tau I + 2(\varphi S + \sigma E + \gamma_2 I) + 2\sigma}\right)^2$$

$$= \int S(\lambda + \rho) + (\rho S + \sigma E + \gamma_2 I)$$

Hence, $A(t) = \int_0^t S(\lambda + \rho) + (\rho S + \sigma E + \gamma_2 I)\ dw(S)$ (18)

As a result,

$$dS(t) = \left(S(\lambda + \rho) - (\rho S + \sigma E + \gamma_2 I)\right)dt + \sqrt{S(\rho + \lambda) + (\rho S + \sigma E + \gamma_2 I)dw(t)}$$

The same process can be applied to the second equation.

$$\frac{dI}{dt} = ((\varphi S + \sigma S + \gamma_2 I) - \tau I)dt + \sqrt{((\rho S + \sigma E + \gamma_2 I) + \tau I)dw(t)}$$

Therefore, the system of stochastic differential equations for the Ebola virus describes how the population's susceptibility and infection vary over time.

Nevertheless, the stochastic differential equation can more accurately represent the framework (system):

$$\frac{dI}{dt} = ((\varphi S + \sigma S + \gamma_2 I) - \tau I)dt + \sqrt{((\rho S + \sigma E + \gamma_2 I) + \tau I)dw(t)}$$

Hence, $S(t) + I(t) = N(t) \succ 0$

3 RESULTS AND DISCUSSION

3.1. Numerical Solution

Solving the sets of differential equations used to model the Ebola virus numerically, we make use of the Finite Difference Scheme (FSD) to decompose model (1) to yield.

$$\begin{aligned}\frac{S_{j+1} - S_j}{h} &= \lambda - (\rho + \Psi)S_k + \varphi R\\ \frac{E_{j+1} - E_j}{h} &= \Psi S - (\rho + \sigma)E_k\\ \frac{Q_{j+1} - Q_j}{h} &= \sigma E - (\gamma_1 + \gamma_2 + \rho)Q_k\\ \frac{I_{j+1} - I_j}{h} &= \gamma_2 Q - (\rho + \delta + \tau)I_k\\ \frac{R_{j+1} - R_j}{h} &= \gamma_1 Q + \tau I - (\rho + \phi)R_k\end{aligned} \quad (19)$$

Equation (19) can alternatively be written as;

$$
\begin{aligned}
S_{j+1} &= S_j + h(\lambda - (\rho + \Psi)S_k + \varphi R) \\
E_{j+1} &= E_j + h(\Psi S - (\rho + \sigma)E_k) \\
Q_{j+1} &= Q_j + h(\sigma E - (\gamma_1 + \gamma_2 + \rho)Q_k) \\
I_{j+1} &= I_j + h(\gamma_2 Q - (\rho + \delta + \tau)I_k) \\
R_{j+1} &= h(\gamma_1 Q + \tau I(\rho + \phi)R_k)
\end{aligned} \tag{20}
$$

Where $j = 0, 1, 2, 3, 4, 5\ldots, h$ is the step size

3.2 Numerical Simulation

To check the overall dynamics of the effect each parameter on the population model, the Matrix Laboratory (MATLAB) was used in simulation along the side Table 2, which contains parameters and their values.

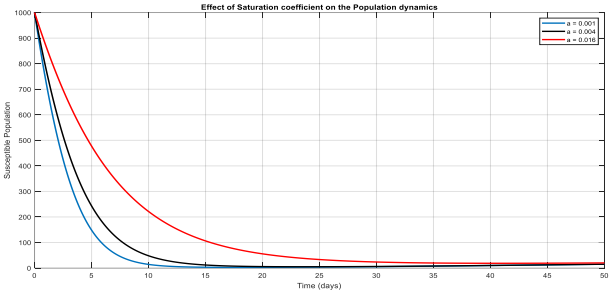

Figure 2: Trajectories solution of saturated coefficient on the susceptible population

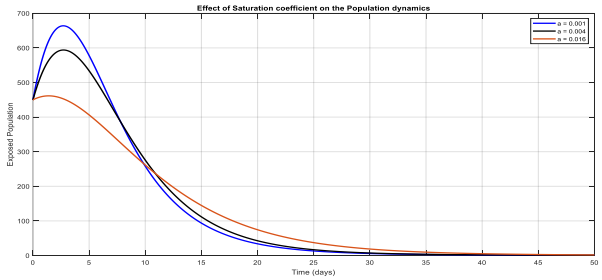

Figure 3: Trajectories solution of saturated coefficient on the exposed population.

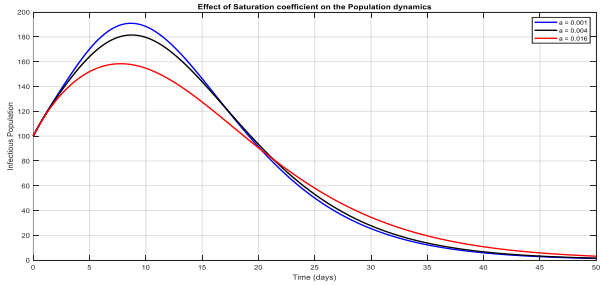

Figure 4: trajectories solution of saturated coefficient on the infected population

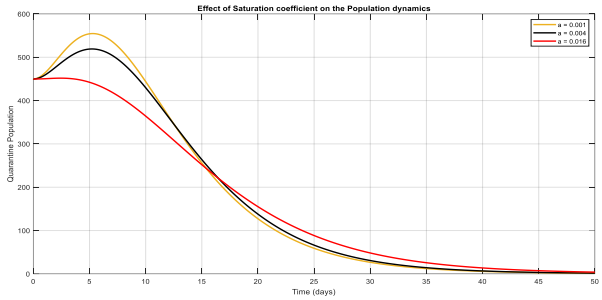

Figure 5: trajectories solution of saturated coefficient on the quarantine population

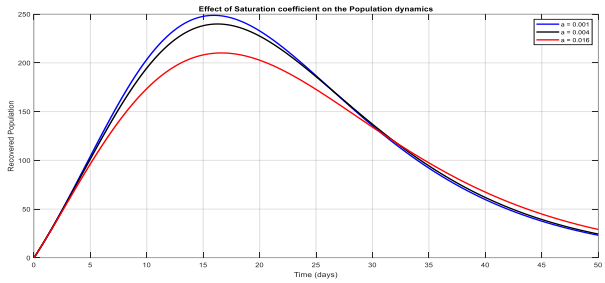

Figure 6: trajectories solution of saturated coefficient on the recovered population

Figure (2-6) demonstrate the effect of saturation on the population density for susceptible, exposed, infected, quarantine and the recovered class respectively. The results indicate that the higher the saturation the faster the infection leaves it host community, which in turn leads to stability on the population density, which indicates that we can have a disease-free equilibrium state in the proposed community if saturation as a form of treatment is well implemented. Government officials, policy makers and health organizations are best informed of the best treatment to implement to finally cure the population of EVD.

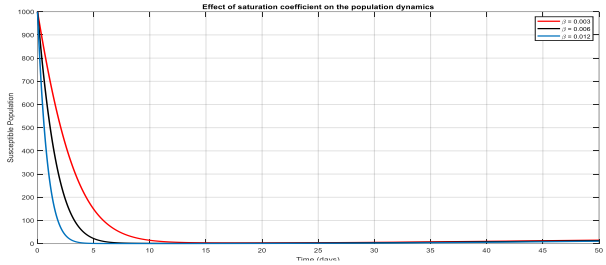

Figure 7: trajectories solution of effective contact rate on the susceptible population

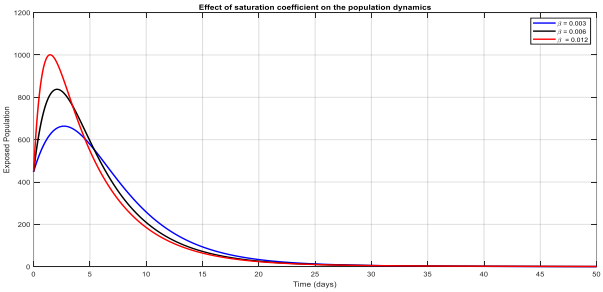

Figure 8: trajectories solution of effective contact rate on the exposed population

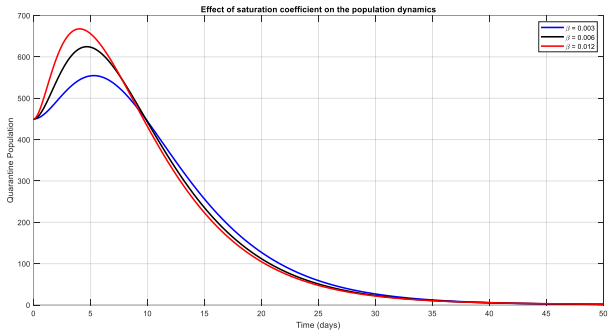

Figure 9: Trajectories solution of effective contact rate on the quarantine population

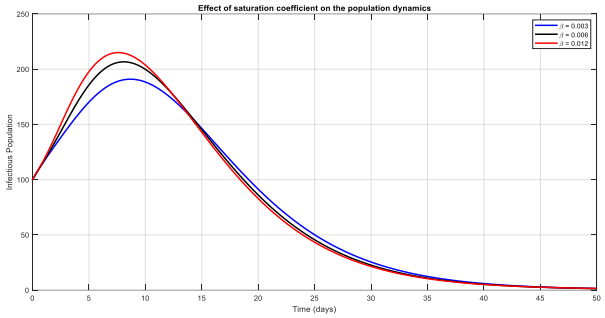

Figure 10: Trajectories solution of effective contact rate on the infectious population

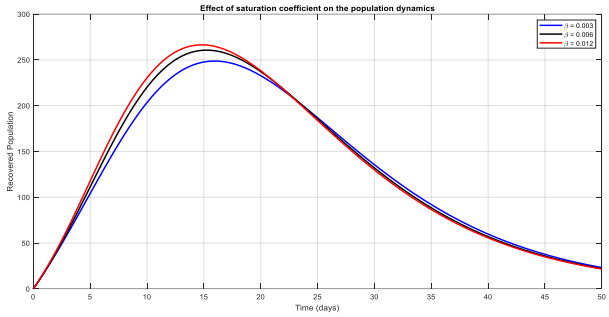

Figure 11 Trajectories solution of effective contact rate on the recovered population.

Figures (7-11) illustrate the effect of contact rate β on all compartments within the proposed model. Results show that the higher the contact rate the more the people get infected, which means that the reproduction number of (EVD) will be greater than unity (1) which later lead to higher mortality in the population. This demonstrates the negative impact the contact rate of the disease could have on the population when not controlled. The infection could become endemic if proper saturation factor as a form of treatment is not considered.

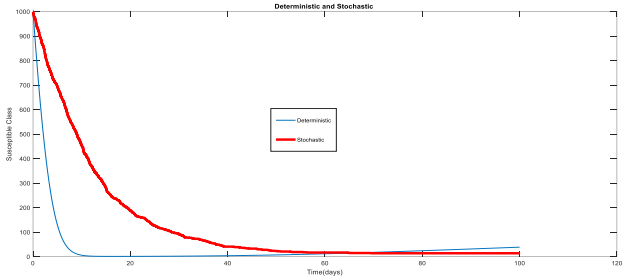

Figure 12: The dynamics of deterministic and stochastic in the susceptible population

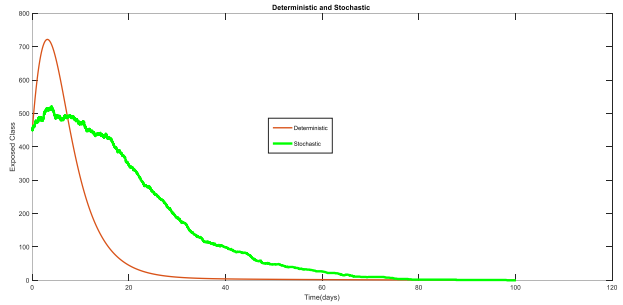

Figure 13: The dynamics of deterministic and stochastic in the exposed population

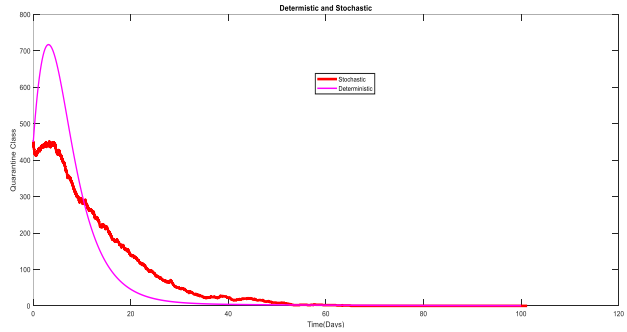

Figure 14: The dynamics of deterministic and stochastic in the quarantine population.

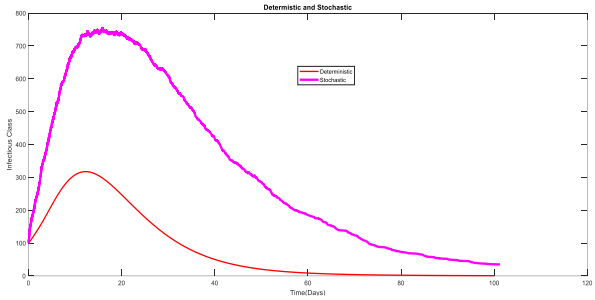

Figure 15: The dynamics of deterministic and stochastic in the infected population.

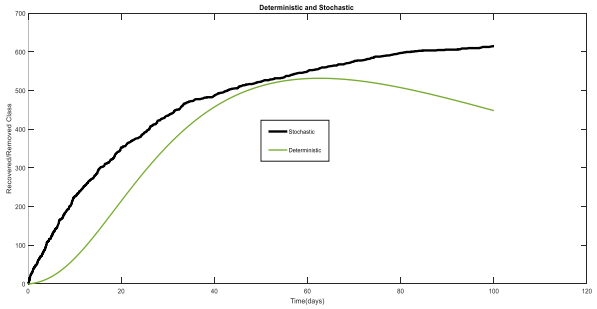

Figure 16: The dynamics of deterministic and stochastic in the removed/recovered population.

Figure (12-16) simply demonstrates the dynamics of stochastic model as an approximation of the deterministic of the model in the long run considering randomness of events in each compartment.

CONCLUSION

This research proposed a modified SEQIR dynamical model to investigate the effect of saturation factors and effective contact rate on the transmission dynamics of Ebola virus and results show that proper control plans can achieve a lower mortality rate in the community. When saturation factor is reduced and contacts among susceptible individuals are impaired, then transmission of the deadly virus is observed to dwindle and get exterminated in the final analysis.

Acknowledgement

Mrs. Adetoun Loyinmi and my co-authors are appreciated for their contributions and moral support.

Conflict of Interest

The authors declare that no funds, grants or other support were received for the preparation of this manuscript. The authors also declare no conflict of interest.

REFERENCES

[1] G.C.E. Mbah, I. S. Onah, O. O. Ahman, O. C., Collins, C. C. Asogwa, and C. Okoye, ''Mathematical modelling approach of the study of Ebola virus disease transmission dynamics in a developing country,'*Afr. J. Infect. Dis*. 17, no. 1, (2023): 10–26.

[2] K. Birmingham, and S. Cooney, ''Ebola: Small, but real progress,'' *Nat. Med*. 8 (2012): https://doi.org/10.1038/nm0402-313

[3] J. O. Agbomola, and A. C. Loyinmi, ''Modelling the impact of some control strategies on the transmission dynamics of Ebola virus in human-bat population: An optimal control analysis,'' *Heliyon*, 8, (2022): https://doi.org/10.1016/j.heliyon.2022.e12121

[4] R. Young, ''Composite wave interactions and the collapse of vacuums in gas dynamics,'' *Journal of* Differential *Equations*, 252 (2012): 5129-5154, https://doi.org/10.1016/j.jde.2012.01.028

[5] S. O. Gbodogbe, ''Harmonizing epidemic dynamics: A fractional calculus approach to optimal control strategies for cholera transmission,'' *Scientific African*, 27 (2025): https://doi.org/10.1016/j.sciaf.2025.e02545

[6] Center for Disease Control and Prevention (CDC), "Ebola Disease Basics" https://www.cdc.gov/vhf/ebola/history/2014-2016-outbreak/index.html.(2024).

[7] G. Chowell, N. W. Hengartner, C. Castillo-Chavez, P. W. Fenimore, and J.M. Hyman, ''The basic reproductive number of Ebola and the effects of public health measures: The cases of Congo and Uganda,'' *J. Theoret. Biol*., 229, no.1 (2004): 119–126. https://doi.org/10.1016/j.jtbi.2004.03.006

[8] J. Breman, P. Piot, K. Johnson, and S. Pattyn, ''The epidemiology of Ebola hemorrhagic fever in Zaire, 1976'' Medicine, Environmental Science, (1978): 85–97. https://api.semanticscholar.org/CorpusID:80009505

[9] J. Agbomola, and A. Loyinmi, 'A mathematical model for the dynamic behavior of Ebola transmission in human-bat population: implication of immediate discharge of recovered individuals. *Preprints*, (2022): https://doi.org/10.21203/rs.3.rs-1399224/v1.

[10] K. O. Idowu, and A. C. Loyinmi, 'Impact of contaminated surfaces on the transmission dynamics of corona virus disease (Covid-19),''*Biomed J. Sci. Tech. Res*., 51 (2023): 42280-90. https://doi.org/10.26717/BJSTR 2023.51008046

[11] O. K. Idowu, and A. C. Loyinmi, 'Qualitative analysis of the transmission dynamics and optimal control of covid-19,'' *EDUCATUM Journal of Science, Mathematics and Technology*, 10: no.1 (2023): 54-70. https://doi.org/10.37134/ejsmt.vol10.1.7.2023

[12] A. C. Loyinmi, and S. O. Gbodogbe, "Epidemiological viability and control of rotavirus: A mathematical modelling approach" FNAS journal of Scientific Innovations, 6, no. 2 (2025): 18-43.

[13] A. C. Loyinmi, T. K. Akinfe, and A. A. Ojo, "Qualitative analysis and dynamical behavior of a Lassa haemorrhagic fever model with exposed rodents and saturated incidence rate,'' *Sci. African*, 14 (2021): e01028. https://doi.org/10.1016/j.sciaf.2021.e01028

[14] S.O. Adewale, I. A. Olopade, G. A. Adeniran, I. T. Mohammed, and S. O. Ajao, ''Mathematical analysis of effects of isolation on Ebola transmission dynamics,'' *Research Journal of Mathematics*. 2 (2015): 2349-5375.

[15] R. T. Abah, A. B. Zhiri, K. Osinubi, and A. Adeniji, 'Mathematical analysis and simulation of Ebola virus disease spread incorporating mitigation measures,' *Franklin Open*. 6 (2024): https://doi.org/10.1016/j.fraope.2023.100066

[16] A.C Loyinmi, and A. L. Ijaola, ''Investigating the effects of some controls measures on the dynamics of diphtheria infection using fractional order model,' *Mathematics and Computational Sciences*, 5, no.4 (2024): 26-47. 10.30511/MCS.2024.2032110.1183

[17] A. C. Loyinmi, and S. O. Gbodogbe, ''Mathematical modelling and control strategies for Nipah virus transmission incorporating Bat – to – pig –to – human pathway,'' *EDUCATUM Journal of Science, Mathematics and Technology*, 11, no.1, (2024): 54-80. https://doi.org/10.37134/ejsmt.vol11.1.7.2024

[18] A. C. Loyinmi, A. S. Ajala, and L. I. Alani, ''Analysis of the effect of vaccination, efficient surveillance and treatment on the transmission dynamics of cholera,'' *Al-Bahir journal for Engineering and Pure Sciences*, 5, (2024): 94 – 107. https://doi.org/10.55810/2313-0083.1070

[19] A. C. Loyinmi, S. O. Gbodogbe, and K. O. Idowu, 'On the interaction of the human immune system with foreign body: mathematical modelling approach,'' *Kathmandu University Journal of Science, Engineering and Technology*, 17, no.2 (2023): 1-17. https://journals.ku.edu.np/kuset/article/view/137

[20] C.E. Madubueze, A. R Kimbir, and T. Aboiyar, "Global stability of Ebola virus disease model with contact tracing and quarantine" *Appl. Math.: Int. J.*, 13, no. 1 (2018).

[21] A. J. Adebisi, M. O. Olayiwola, I. A. Adediran, and A.I. Alaje, ''A novel mathematical model and homotopy pertutbation method analyzing the effects of –saturated incidence and treatment rate on Covid-19 eradication,'' *Iranian Journal of Science*, 48 (2024): 625-636. https://doi.org/10.1007/s40995-024-01608-w

[22] W.O. Kermack, and A. G. McKendrick, 'A contribution to the mathematical theory of epidemics,' *Proc. R. Soc. Lond. Series A*. 115, no. 772, (1927): 700 –721.

[23] A. C. Loyinmi, and A. L. Ijaola, ''Fractional order model of dynamical behavior and qualitative analysis of Anthrax with infected vector and saturation,'' *Int. J of Math. Anal. And Modelling*, 7, no. 2, (2024): 224-264.

[24] C. L. Althaus, ''Estimating the reproduction number of Ebola virus (EBOV) during the 2014 outbreakIn West Africa,'' *PLoS Currents*, https://doi.org/10.1371/currents.outbreaks.91afb5e0f279e7f29e7056095255b288

[25] J. Astacio, D. Briere, M. Guillen, J. Martinez, F. Rodriguez, and N. Valenzuela-Campos, 'Mathematical models to study the outbreaks of Ebola,' Biometrics Unit Technical Reports; Number BU-1365-M, (1996). https://hdl.handle.net/1813/31962

[26] A. C. Loyinmi, 'A fractional order model for Zika virus

transmission dynamics: analysis, control strategies, and simulation insights,' *FNAS journal of scientific Innovations*, 6, no.1 (2024): 84-108.

[27] S. Edward, E. M. Lusekelo, D. M. Ndidi, and E. Simanjilo, ''Mathematical modelling of the transmission dynamics of Ebola virus disease with control strategies,'' *Int. J. Sci.: Basic Appl. Res*. 33, no. 1 (2017): 112–130.

[28] O. Diekmann, and J. A. P. Heesterbeek, ''Mathematical Epidemiology of Infectious Diseases: Model building Analysis and Interpretation,' *John Wiley & Sons*, 5, (2000)