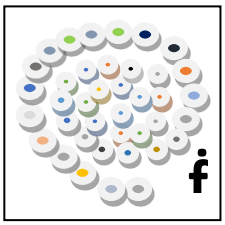


International Journal of Mathematical Analysis and Modelling

(Formerly Journal of the Nigerian Society for Mathematical Biology)

Volume 6, Issue 1 (July), 2023

ISSN (Print): 2682 - 5694
ISSN (Online): 2682 - 5708

International Journal of Mathematical Analysis and Modelling
Volume 6, Issue 1, July 2023, pages 138 – 152

Modelling the transmission dynamics of Ebola infection

F.O Anorue*† and O.R. Okwara*

Abstract

Since not all infectious diseases have been kept under control because of the diversity of pathogens and means of transmission not well known, the adequate control measure to be implemented is impeded or hampered. Working on Ebola disease, we formulated a modified SEIR model to describe the transmission dynamics of the virus. This model considered the environmental contaminant and primates as major factors in the transmission dynamics alongside with infectious humans. Mathematical analysis carried out showed that the disease free-equilibrium is locally and globally asymptotically stable, when the reproduction number $R_0 < 1$ and unstable if $R_0 > 1$. From this analysis and numerical simulations, we conclude that the environmental contaminants and primates should not be neglected in considering the transmission dynamics of Ebola. All these were done to proffer adequate control measures and areas of more considerations at the end of the work.

Keywords: mathematical model; reproduction number; Ebola virus; infection process; equilibrium analysis.

1 Introduction

Infectious diseases are those diseases caused by viruses, bacteria, epiphytes and parasites such as protozoan or worms that have a potential to spread into the population easily. Mathematical models of infectious diseases make predictions about future outbreaks of diseases; suggest intervention measures to be taken in order to control the diseases. The literature on the mathematical models for communicable (infectious) diseases is vast, yet infectious diseases have continued to be the major causes of suffering and mortality in developing countries. Several infectious diseases have different mode of transmission. Diseases such as influenza, measles, rubella and chicken pox that are transmitted by virus and can confer immunity against re-infection, while diseases such as tuberculosis, meningitis and gonorrhoea that are transmitted by bacteria confer no immunity against re-infection.

Diseases such as malaria, yellow fever etc are transmitted by vectors (usually insects). In sexually transmitted diseases with heterosexual transmission, each sex acts as a vector, and the disease is transmitted back and forth between the sexes. The infectious diseases have been a major cause of death and illness globally. In other to salvage the situation, the need for a global perspective that accounts for bio complexity, involving all the interrelated factors that contribute to the evolution and survival of infectious agents, will require the efforts of biologist, ecologists, chemists, epidemiologist, mathematicians, statisticians and atmospheric scientists to shed more lights on how these diseases can be eradicated. Due to the recent outbreak of Ebola Virus Disease, many

*Department of Mathematics, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria.
†Corresponding Author. E-mail Address: favouronyi231@gmail.com

researchers have developed mathematical models to help understand the dynamics of the virus and appropriate intervention techniques to be used to combat the spread of the disease effectively.

The Ebola Virus Disease (EVD) is a highly pathogenic virus that was first identified in 1976 during the two consecutive outbreaks of a hemorrhagic fever simultaneously emerging in various regions of Central Africa. The term "Ebola virus" was introduced by one of the investigation team during the first outbreaks of 1976 as the virus was detected in a village, in the vicinity of the Ebola River of the Democratic Republic of Congo (DRC) and the mortality of EVD is about 50-90%. The EVD is a lethal disease with a high mortality which has caused many deaths. Epidemiologists have investigated the transmission dynamics of this disease and came up with some recommendations for different intervention strategies which have helped to control the spread of this disease. The outbreak was triggered due to the fact that the infected individuals were transported from one country to another, in July 2014.

Ebola virus (EBOV) is a filovirus that belongs to the filoviridae, family that causes a hemorrhagic disease in human and non-human primates. Ebola virus is transmitted to the human population through close contact with the blood, secretions, organs, or other bodily fluids of infected animals that are primates such as fruit bats, chimpanzees, gorillas, monkeys, antelopes and porcupines found ill or dead in the rain forest zones. But since bats are recognized as a major reservoir of many viral infections like measles and mumps, the transmission of Ebola virus is traceable to it as a major reservoir. The transmission of Ebola virus can be spread from person to person by direct contact with the blood and body fluids such as saliva, mucus, vomit, sweat, tears, breast milks, urine and semen of a person who has been affected by the disease according to Leroy(2005). Rafiq *et al* (2020) said that infected individuals do not show symptoms immediately; neither do they start to transmit the virus. This is because the virus undergoes incubation (the period between the infection and the onset of symptoms) which usually ranges from 2-21 days in this case. During the incubation, the virus infects cells, replicates and bursts out of the infected cells, producing Ebola virus glycogen proteins that become prevalent in blood vessels, thus rendering the vessels more permeable. The permeability causes blood vessels to ooze out blood. The natural defence system of the infected person is tampered with the virus, thereby infecting the immune cells, which are channels through which the virus can be transported to other body parts and organs such as the liver, spleen, kidney, and the brain. This could lead to the failure of these organs leading to the death of the person.

Zhi-Qiang Xia *et al* (2013), formed a mathematical model that was used to study and analyze the peak arrival time of the disease and the correlation between related parameters used in estimation and the basic reproduction numberR_0, The model additionally compared the efficiency of different control measures, that includes isolation of the infected and safe burial of the infected individuals that are diseased. Lewnard *et al* (2014), investigated the impact of the Ebola virus disease with limited medical resources. Rachah *et al* (2015), gave a comparison behaviour between two differential mathematical models used in the description of the Ebola virus propagation in West Africa. They investigated the two models in order to improve the prediction and the control of the propagation of the virus and particularly studied the case when the two models generate two similar results. Xia Z.O *et al.* indicates that in order to understand the transmission mechanism of Ebola virus disease in Liberia and search for effective control measure, a mathematical model to study the spread of Ebola virus disease amongst human beings was formed. And based on the fitting method, they performed the parameter estimation and obtained the basic reproduction number in the absence of effective control measures. Based on sensitivity analysis, they demonstrated that only taking single measure cannot control the spread of Ebola virus disease well, which is consistent with the conclusion posed by Khan *et al* (2015), that decrease of incidence at country and country level is attributable to the increasing availability of Ebola virus disease treatment. Considering the fact that in Africa and particularly in the regions that were affected by Ebola outbreak, people live close to

the rain-forests, hunt bats and monkeys and harvest forest tricks for food; Also their traditions and customs which can create a connection with infected persons. He tried to answer questions like; can the consumption of contaminated bush meat, the funeral practices and environmental contamination explain the recurrence and persistence of Ebola virus disease outbreaks in Africa.

From the above reviews, we notice that many mathematical models proposed and developed majorly considered the transmission of the disease to human population, neglecting the source of the infection: the primates (especially the bat family) and also did not consider the contamination in the environment by the bodily fluids released by these primates and infected humans. We propose a generalized model, putting into consideration, the human population, the primate population and the contaminants in other to have and over view of what the transmission process should be.

1.2 Model assumptions

The formulation of the model is guided by the following assumptions:

i. The total population of individuals is not constant.
ii. The populations in compartments of both humans and vectors are non-negative and so are all the parameters involved in the model.
iii. The primates do not recover from the disease and so, naturally die out of the population.
iv. Individuals move to the recovered class from infectious class due to permanent immunity conferred on them via adequate treatment.
v. The primates population can be reduced through human activities.
vi. The human population interact with the primates via various activities carried out by humans within the population of the primates like hunting, research etc which is capable of contaminating the susecptible primates from any infected human.

1.3 Model formulation and analysis

We formulate a deterministic model for the spread dynamics of Ebola virus that considers human population, primate population and contaminants at time t, divided into four classes, three classes and the contaminant compartment respectively. The human population has susceptible class$(\mathrm{S_h})$, exposed class$(\mathrm{E_h})$, infectious class $(\mathrm{I_h})$and the recovered class$(\mathrm{R_h})$. So that the total human population (N_h) at time t is given by:

$$N_h = S_h + E_h + I_h + R_h.$$

The primate population has the susceptible class(S_m), the exposed class $(E_m)$and the infectious class (I_m). So that the total primate population is given by:

$$N_m = S_m + E_m + I_m.$$

Having considered streams of flow from the environmental release and contribution (T. Berge et al 2017, Leah R Johnson 2006, Onyinyechi Anorue and Mark Modebei 2020, then Chun –Hai – Fung 2014), we also have given a compartment for environmental contaminants denoted by (P).

Susceptible class is individuals that are vulnerable to the disease. Individuals are recruited into susceptible class through immigration and birth at a rate b_h and die naturally at a rate μ_h. Through effective contact rate between infectious individuals, infectious primates and the contaminants, the susceptible gets exposed to Ebola virus at a rate $\lambda_1 = \lambda_h\beta_h I_h S_h + \lambda_h\beta_m I_m S_h + \lambda_h \frac{\beta_{\mathrm{vh}}\mathrm{P}}{\mathrm{K+P}} S_h$.

Individuals who are exposed to the virus from the susceptible class get into exposed class at a rateλ_1, become infectious at a rate α_1 also release the virus into the environment at a rate δ_1. They die naturally at a rate μ_h. Rate at which individuals become infectious from the exposed class is given by α_1. Infectious individuals shed the virus into the environment at a rate δ_2. With the infection, the infected die out of the population at a rate 3 and naturally at the rate μ_h . Proportion of the infected undergo treatment through adequate medication and quarantining, obtain permanent immunity moving into the recovered class at a rate$(1 - \alpha_2)$, and also die out of the population naturally at the rate μ_h. The susceptible primates are recruited through immigration and birth at a rate b_m.

Having come into the environment get exposed to the virus through effective contact with the environment and the infected humans and primates at a rate$\lambda_2 = \lambda_m \beta_h I_h S_m + \lambda_m \beta_m I_m S_m + \lambda_m \frac{\beta_{vm} P}{K+P} S_m$ and die out naturally at a rate μ_m. Primates move into the exposed class having contact with the infection at a rateλ_2. When exposed to the virus, the primates become infectious of the disease at a rate α_3, shed this virus into the environment at a rate $\delta_3$and die out of the population at a rate μ_m.

Recruitment into the infectious class is from the exposed primates who have started transmitting the virus at a rateα_3. These primates shed the virus into the environment at a rate $\delta_4$and die out of the population at a rate μ_m. The environment is contaminated through virus shedding that runs from the exposed individuals, infectious individuals, exposed primates and infectious primates at the rate $\delta_1, \delta_2, \delta_3,$ andδ_4 respectively.

1.4 The model equations

From the description of the model in 3.2 above, we derived the following model equations.

$$\frac{dS_h}{dt} = b_h - \lambda_h \beta_h I_h S_h - \lambda_h \beta_m I_m S_h - \frac{\beta_{vh} P}{K+P} S_h - \mu_h S_h \quad \text{(1)}$$

$$\frac{dE_h}{dt} = \lambda_h \beta_h I_h S_h + \lambda_h \beta_m I_m S_h + \frac{\beta_{vh} P}{K+P} S_h - (\alpha_1 + \delta_1 + \mu_h) E_h \quad \text{(2)}$$

$$\frac{dI_h}{dt} = \alpha_1 E_h - (3 + \delta_2 + \mu_h) I_h \quad \text{(3)}$$

$$\frac{dR_h}{dt} = 3(1 - \alpha_2) I_h - \mu_h R_h \quad \text{(4)}$$

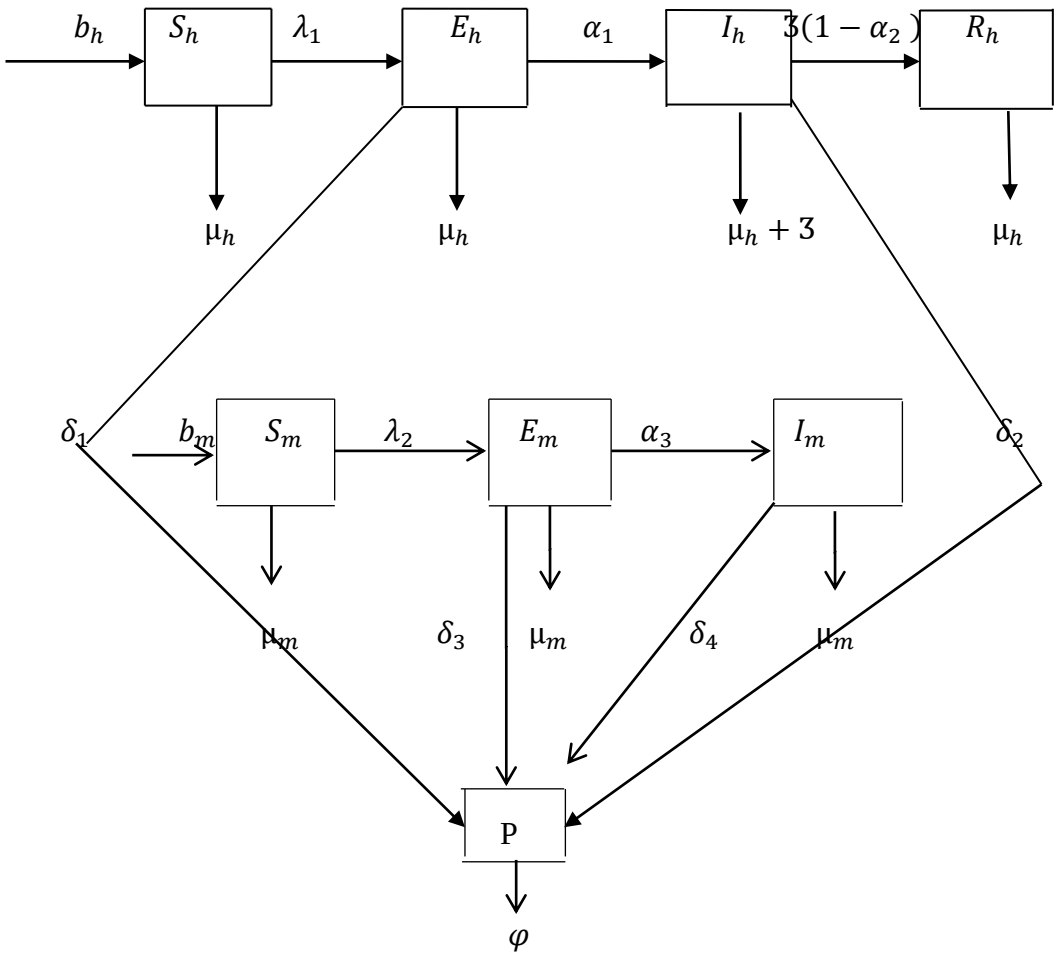
$$\frac{dS_m}{dt} = b_m - \lambda_m \beta_h I_h S_m - \lambda_m \beta_m I_m S_m - \frac{\beta_{vm} P}{K+P} S_m - \mu_m S_m \quad \text{(5)}$$

$$\frac{dE_m}{dt} = \lambda_m \beta_h I_h S_m + \lambda_m \beta_m I_m S_m + \frac{\beta_{vm} P}{K+P} S_m - (\alpha_3 + \delta_3 + \mu_m) E_m \quad \text{(6)}$$

$$\frac{dI_m}{dt} = \alpha_3 E_m - (\delta_4 + \mu_m) I_m \quad \text{(7)}$$

$$\frac{dP}{dt} = \delta_1 E_h + \delta_2 I_h + \delta_3 E_m + \delta_4 I_m - \varphi P \quad \text{(8)}$$

The flow diagram



Variables and Parameters Discreption

S_h	Total number of susceptible individuals
E_h	Total number of exposed individuals
I_h	Total number of infected individuals
R_h	Total number of recovered individuals
S_m	Total number of susceptible primates
E_m	Total number of exposed primates
I_m	Total number of infected primates
P	Amount of Ebola virus concentration in the environment.
b_h	Birth and or immigration rate of humans.
λ_1	Effective contact rate of humans with infections.
α_1	Rate at which the exposed humans become symptomatic infectious.
α_2	Proportion of humans who through adequate treatment recovererd fully from the

	infection.
$(1 - \alpha_2)$	Proportion of humans who died due to the disease.
α_3	Rate at which the exposed primates become symptomatic infectious.
b_m	Birth rate and or immigration rate of primates
λ_2	Effective contact rate of primates with infections.
β_h	The transmission probability of the virus from infected humans to the exposed class.
β_m	The transmission probability of the virus from infected primates to the exposed class.
δ_1	Virus excretion rate from the exposed humans to the environment.
δ_2	Virus excretion rate from the infected humans to the environment.
δ_3	Virus excretion rate from the exposed primates to the environment
δ_4	Virus excretion rate from the infected primates to the environment.
μ_h	Natural death rate of humans.
μ_m	Natural death rate of primates.
$З$	Disease induced death rate of humans
β_V	Contact rate between susceptible primates and Ebola viruses
K	Virus 50% infectious dose capable of causing Ebola desease.
φ	Net decay of the virus.

2 Model analysis and results

2.1 Positivity and boundedness of solutions

Theorem 1. Given the initial condition of the model equations as $S_h(0) > 0, E_h(0) > 0, I_h(0) > 0, R_h(0) > 0, S_m(0) > 0, E_m(0) > 0, I_m(0) > 0, P(0) \geq 0$, then the model equations are non-negative for all $t > 0$.

Proof: The total human population is given by $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$ which satisfies the ordinary differential equation

$$\frac{dN_h}{dt} = b_h - (S_h + E_h + I_h + R_h)\mu_h - ЗI_h - \delta_1 E_h - \delta_2 I_h$$

so that we get

$$\frac{dN_h}{dt} \leq b_h - \mu_h N_h - ЗI_h.$$

Equating the right hand side to zero and solving, we get

$$\frac{dN_h}{dt} \leq \frac{b_h}{(\mu_h + З)} \quad \text{……………………………………………………………} (9)$$

Considering the primate population, we have $N_m(t) = S_m(t) + E_m(t) + I_m(t)$, which satisfies the ordinary differential equation:

$$\frac{dN_h}{dt} = b_h - (S_m + E_m + I_m)\mu_m.$$

This implies

$\frac{dN_h}{dt} \leq b_h - \mu_m N_m$, leading to

$$\frac{dN_h}{dt} \leq \frac{b_h}{\mu_m} \quad \text{……………………………………………………………} (10)$$

For the model equations (1)-(8), defined in the region

$$\Omega = \left\{(S_h, E_h, I_h, R_h, S_m, E_m, I_m, P)\epsilon\Re_+^8 \leq \frac{b_h}{(\mu_h+3)}, \frac{b_h}{\mu_m}\right\} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (11)$$

We conclude that the model equations are positive and bounded.

2.2 Existence and uniqueness of model solutions

Theorem 2. Let $x' = f(x),\ x(t_0) = x_0 \quad \forall\, x = (x_1, x_2, \ldots\ldots\ldots, x_8)$ and $x_0 = (x_{10}, x_{20}, \ldots\ldots\ldots, x_{80})$ be an initial value problem defined in a region R: $|x - x_0| < a$, then:
i). $f(t,x)$ is continuous in R
ii). $|f(t,x)| \leq k$ ie $f(t,x)$ is bounded in R
iii). $f_x = \frac{\partial f}{\partial x}$ is continuos in R
iv). $f_x = \frac{\partial f}{\partial x}$ is bounded in R ie $\left|\frac{\partial f}{\partial x}\right| \leq M$
then there exists at least one solution that is unique in the subinterval $|x - x_0| < \alpha \quad \forall\, \alpha = min\left\{a, \frac{b}{k}\right\}$.

Theorem 3. Let R denote the region defined earlier such that we can get both the region and the bounded solution hold, then there exists a unique solution of the model equations (1)-(8) which is bounded in R.

Proof:
Let

$$f_1 = b_h - \lambda_h\beta_h I_h S_h - \lambda_h\beta_m I_m S_h - \frac{\beta_{vh}P}{K+P}S_h - \mu_h S_h$$
$$f_2 = \lambda_h\beta_h I_h S_h + \lambda_h\beta_m I_m S_h + \frac{\beta_{vh}P}{K+P}S_h - (\alpha_1 + \delta_1 + \mu_h)E_h$$
$$f_3 = \alpha_1 E_h - (3 + \delta_2 + \mu_h)I_h$$
$$f_4 = 3(1 - \alpha_2)I_h - \mu_h R_h$$
$$f_5 = b_m - \lambda_m\beta_h I_h S_m - \lambda_m\beta_m I_m S_m - \frac{\beta_{vm}P}{K+P}S_m - \mu_m S_m$$
$$f_6 = \lambda_m\beta_h I_h S_m + \lambda_m\beta_m I_m S_m + \frac{\beta_{vm}P}{K+P}S_m - (\alpha_3 + \delta_3 + \mu_m)E_m$$
$$f_7 = \alpha_3 E_m - (\delta_4 + \mu_m)I_m$$
$$f_8 = \delta_1 E_h + \delta_2 I_h + \delta_3 E_m + \delta_4 I_m - \varphi P.$$

We show that the $\frac{\partial f_i}{\partial x_j} \quad \forall\, i,j = 1,2,\ldots\ldots,8$ are continuous by solving out the partial derivatives as:

$$\left|\frac{\partial f_1}{\partial S_h}\right| = \left|-\lambda_h\beta_h I_h - \lambda_h\beta_m I_m - \frac{\beta_{vh}P}{K+P} - \mu_h\right| < \infty, \left|\frac{\partial f_1}{\partial I_h}\right| = |-\lambda_h\beta_h S_h| < \infty, \left|\frac{\partial f_1}{\partial I_m}\right| = |-\lambda_h\beta_m S_h| < \infty, \left|\frac{\partial f_1}{\partial P}\right| = \left|-\frac{\beta_{vh}K}{(K+P)^2}S_h\right| < \infty and \left|\frac{\partial f_1}{\partial E_h}\right| = \frac{\partial f_1}{\partial R_h} = \frac{\partial f_1}{\partial E_m} = \frac{\partial f_1}{\partial S_m} = 0 < \infty.$$

$$\left|\frac{\partial f_2}{\partial S_h}\right| = \left|\lambda_h\beta_h I_h + \lambda_h\beta_m I_m + \frac{\beta_{vh}P}{K+P}\right| < \infty, \left|\frac{\partial f_2}{\partial E_h}\right| = |-(\alpha_1 + \delta_1 + \mu_h)| < \infty, \left|\frac{\partial f_2}{\partial I_h}\right| =$$

$|\lambda_h\beta_h S_h| < \infty, \left|\frac{\partial f_2}{\partial I_m}\right| = |\lambda_h\beta_m S_h| < \infty \left|\frac{\partial f_2}{\partial P}\right| = \left|\frac{\beta_{vh}K}{(K+P)^2}S_h\right| < \infty \; and \; \frac{\partial f_2}{\partial R_h} = \frac{\partial f_2}{\partial E_m} = \frac{\partial f_2}{\partial S_m} = |0| < \infty.$

$\left|\frac{\partial f_3}{\partial E_h}\right| = |\alpha_1| < \infty, \left|\frac{\partial f_3}{\partial I_h}\right| = |-(3+\delta_2+\mu_h)| < \infty, \left|\frac{\partial f_3}{\partial S_h}\right| = \left|\frac{\partial f_3}{\partial R_h}\right| = \left|\frac{\partial f_3}{\partial S_m}\right| = \left|\frac{\partial f_3}{\partial E_m}\right| = \left|\frac{\partial f_3}{\partial I_m}\right| = \left|\frac{\partial f_3}{\partial P}\right| = 0 < \infty$

$\left|\frac{\partial f_4}{\partial I_h}\right| = |3(1-\alpha_2)| < \infty, \left|\frac{\partial f_4}{\partial R_h}\right| = |-\mu_h| < \infty, \left|\frac{\partial f_4}{\partial S_h}\right| = \left|\frac{\partial f_4}{\partial E_h}\right| = \left|\frac{\partial f_4}{\partial S_m}\right| = \left|\frac{\partial f_4}{\partial E_m}\right| = \left|\frac{\partial f_4}{\partial I_m}\right| = \left|\frac{\partial f_4}{\partial P}\right| = |0| < \infty$

$\left|\frac{\partial f_5}{\partial S_m}\right| = \left|-\lambda_m\beta_h I_h - \lambda_m\beta_m I_m - \frac{\beta_{vm}P}{K+P} - \mu_m\right| < \infty, \left|\frac{\partial f_5}{\partial I_h}\right| = |-\lambda_m\beta_h S_m| < \infty, \left|\frac{\partial f_5}{\partial I_m}\right| = |-\lambda_m\beta_m S_m| < \infty, \left|\frac{\partial f_5}{\partial P}\right| = \left|-\frac{\beta_{vm}K}{(K+P)^2}S_m\right| < \infty \; and \; \left|\frac{\partial f_5}{\partial S_h}\right| = \left|\frac{\partial f_5}{\partial E_h}\right| = \left|\frac{\partial f_5}{\partial R_h}\right| = \left|\frac{\partial f_5}{\partial E_m}\right| = 0 < \infty$

$\left|\frac{\partial f_6}{\partial S_m}\right| = \left|\lambda_m\beta_h I_h + \lambda_m\beta_m I_m + \frac{\beta_{vm}P}{K+P}\right| < \infty, \left|\frac{\partial f_6}{\partial E_m}\right| = |-(\alpha_3+\delta_3+\mu_m)| < \infty \left|\frac{\partial f_6}{\partial I_m}\right| = |\lambda_m\beta_m S_m| < \infty, \left|\frac{\partial f_6}{\partial P}\right| = \left|\frac{\beta_{vm}K}{(K+P)^2}S_m\right| < \infty, \left|\frac{\partial f_6}{\partial I_h}\right| = |\lambda_m\beta_h S_m| < \infty \; and \; \left|\frac{\partial f_6}{\partial S_h}\right| = \left|\frac{\partial f_6}{\partial E_h}\right| = \left|\frac{\partial f_6}{\partial R_h}\right| = |0| < \infty$

$\left|\frac{\partial f_7}{\partial I_m}\right| = |-(\delta_4+\mu_m)| < \infty \left|\frac{\partial f_7}{\partial E_m}\right| = |\alpha_3| < \infty, \left|\frac{\partial f_7}{\partial I_h}\right| = \left|\frac{\partial f_7}{\partial R_h}\right| = \left|\frac{\partial f_7}{\partial S_h}\right| = \left|\frac{\partial f_7}{\partial E_h}\right| = \left|\frac{\partial f_7}{\partial S_m}\right| = \left|\frac{\partial f_7}{\partial P}\right| = |0|. < \infty$

$\left|\frac{\partial f_8}{\partial E_h}\right| = |\delta_1| < \infty, \left|\frac{\partial f_8}{\partial I_h}\right| = |\delta_2| < \infty, \left|\frac{\partial f_8}{\partial E_m}\right| = |\delta_3| < \infty, \left|\frac{\partial f_8}{\partial I_m}\right| = |\delta_4| < \infty, \left|\frac{\partial f_8}{\partial P}\right| = |-\varphi| < \infty \; and \; \left|\frac{\partial f_8}{\partial R_h}\right| = \left|\frac{\partial f_8}{\partial S_h}\right| = \left|\frac{\partial f_8}{\partial S_m}\right| = 0 < \infty$

All the partial derivatives are continuous and bounded, showing that in the region R, there exists a unique solution of the model equations.

2.3 Disease Free- Equilibrium (DFE)

Let ε_0 denote the disease free-equilibrium for the model equations now represented as $(S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*, P^*)$ at equilibrium state which is generated by equating all the equations to zero and solving in the absence of infection we obtain:

$$\frac{dS_h}{dt} \Rightarrow b_h - \lambda_h\beta_h I_h S_h - \lambda_h\beta_m I_m S_h - \frac{\beta_{vh}P}{K+P}S_h - \mu_h S_h = 0$$
$$\frac{dS_h}{dt} \leq \frac{b_h}{\mu_h} \qquad \text{…….....……..} \quad (12)$$

And

$$\frac{dS_m}{dt} \Rightarrow b_m - \lambda_m\beta_h I_h S_m - \lambda_m\beta_m I_m S_m - \frac{\beta_{vm}P}{K+P}S_m - \mu_m S_m = 0$$
$$\frac{dS_m}{dt} \leq \frac{b_m}{\mu_m} \qquad \text{….....……..…..} \quad (13)$$

$$\text{Then } \left\{\varepsilon_0 = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*, P^*) = \left(\frac{b_h}{\mu_h}, 0,0,0, \frac{b_m}{\mu_m}, 0,0,0\right)\right\} \quad \text{…….……..} \ (14)$$

2.4 Basic reproduction number

The basic reproduction number is the average number of secondary disease delivered by a primary case of an infectious to a whole population that is purely susceptible. This measures the invasion ability of the infectious disease in a population via its transmission route. The disease invades the population when the reproduction number denoted by $R_0 > 1$ but gradually dies out when $R_0 < 1$.

The method of calculating the invasion rate with R_0, uses the spectral radius of the next generation matrix $\rho(FV^{-1})$ where F (non-negative) and V (non-negative) denote the new infection and transition term respectively are calculated using the infectious disease modelling at disease free-equilibrium (12).

Let $f_i = E_h, I_h, E_m, I_m, P$ such that $i = 1,2, \ldots,5$

$$f = \begin{pmatrix} \lambda_h\beta_h I_h S_h + \lambda_h\beta_h I_m S_h + \frac{\beta_{vh}P}{K+P}S_h \\ 0 \\ \lambda_m\beta_h I_h S_m + \lambda_m\beta_m I_m S_m + \frac{\beta_{vm}P}{K+P}S_m \\ 0 \\ 0 \end{pmatrix} \text{ and } v = \begin{pmatrix} (\alpha_1 + \delta_1 + \mu_h)E_h \\ -\alpha_1 E_h + (3 + \delta_2 + \mu_h)I_h \\ (\alpha_3 + \delta_3 + \mu_m)E_m \\ -\alpha_3 E_m + (\delta_4 + \mu_m)I_m \\ -\delta_1 E_h - \delta_2 I_h - \delta_3 E_m - \delta_4 I_m + \varphi P \end{pmatrix}$$

Therefore, the associated matrices F and V for the new infectious terms and the remaining transition terms are computed from the following Jacobian matrices:

$$F = \begin{pmatrix} 0 & \lambda_h\beta_h\frac{b_h}{\mu_h} & 0 & \lambda_h\beta_m\frac{b_h}{\mu_h} & \frac{\beta_{vh}Kb_h}{(K+P)^2\mu_h} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda_m\beta_h\frac{b_m}{\mu_m} & 0 & \lambda_m\beta_m\frac{b_m}{\mu_m} & \frac{\beta_{vm}Kb_m}{(K+P)^2\mu_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (\alpha_1+\delta_1+\mu_h) & 0 & 0 & 0 & 0 \\ -\alpha_1 & (3+\delta_2+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & (\alpha_3+\delta_3+\mu_m) & 0 & 0 \\ 0 & 0 & -\alpha_3 & (\delta_4+\mu_m) & 0 \\ -\delta_1 & -\delta_2 & -\delta_3 & -\delta_4 & \varphi \end{pmatrix}$$

so that,

$$F = \begin{pmatrix} 0 & d_1 & 0 & d_2 & d_3 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & d_4 & 0 & d_5 & d_6 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} h_1 & 0 & 0 & 0 & 0 \\ h_2 & h_3 & 0 & 0 & 0 \\ 0 & 0 & h_4 & 0 & 0 \\ 0 & 0 & h_5 & h_6 & 0 \\ h_7 & h_8 & h_9 & h_{10} & h_{11} \end{pmatrix}$$

where

$$d_1 = \lambda_h\beta_h\frac{b_h}{\mu_h}, d_2 = \lambda_h\beta_m\frac{b_h}{\mu_h}, d_3 = \frac{\beta_{vh}Kb_h}{(K+P)^2\mu_h}, d_4 = \lambda_m\beta_h\frac{b_m}{\mu_m}, d_5 = \lambda_m\beta_m\frac{b_m}{\mu_m}, d_6 = \frac{\beta_{vm}Kb_m}{(K+P)^2\mu_m}$$

$$h_1 = (\alpha_1+\delta_1+\mu_h), h_2 = -\alpha_1, h_3 = (3+\delta_2+\mu_h), h_4 = (\alpha_3+\delta_3+\mu_m), h_5 = -\alpha_3,$$
$$h_6 = (\delta_4+\mu_m), h_7 = -\delta_1, h_8 = -\delta_2, h_9 = -\delta_3, h_{10} = -\delta_4, h_{11} = \varphi$$

$$V^{-1} = \begin{pmatrix} \frac{1}{h_1} & 0 & 0 & 0 & 0 \\ -\frac{h_2}{h_1h_3} & \frac{1}{h_3} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{h_4} & 0 & 0 \\ 0 & 0 & -\frac{h_5}{h_4h_6} & \frac{1}{h_6} & 0 \\ \frac{-h_3h_4h_6h_7+h_2h_4h_6h_8}{h_1h_3h_4h_6h_{11}} & -\frac{h_8}{h_3h_{11}} & \frac{-h_1h_3h_6h_9+h_1h_3h_5h_{10}}{h_1h_3h_4h_6h_{11}} & -\frac{h_{10}}{h_6h_{11}} & \frac{1}{h_{11}} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{(d_3(h_2h_8-h_3h_7))}{h_1h_3h_{11}} - \frac{d_1h_2}{h_1h_3}4 & \frac{h_1}{h_3} - \frac{d_3h_8}{h_3h_{11}} & \frac{(d_3(h_5h_{10}-h_6h_9))}{h_4h_6h_{11}} - \frac{d_2h_5}{h_4h_6} & \frac{d_2}{h_6} - \frac{d_3h_{10}}{h_6h_{11}} & \frac{d_3}{h_{11}} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{(d_6(h_2h_8-h_3h_7))}{h_1h_3h_{11}} - \frac{d_4h_2}{h_1h_3} & \frac{d_4}{h_3} - \frac{d_6h_8}{h_3h_{11}} & \frac{(d_6(h_5h_{10}-h_6h_9))}{h_4h_6h_{11}} - \frac{d_5h_5}{h_4h_6} & \frac{d_5}{h_6} - \frac{d_6h_{10}}{h_6h_{11}} & \frac{d_6}{h_{11}} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$R_0 \Rightarrow FV^{-1} = \frac{\left(d_3(h_2h_8 - h_3h_7)\right)}{h_1h_3h_{11}} - \frac{d_1h_2}{h_1h_3} \quad \ldots\ldots (15)$$

Considering the reproduction number analysis, if $R_0 < 1$, we say that the disease dies out gradually from the population but if $R_0 > 1$, we say there is a case of exponential growth of the disease. The result on the reproduction number will be used in analyzing the stability of the model.

2.5 Local stability analysis

Theorem 4. The disease free-equilibrium of the model equation is locally asymptotically stable if $R_0 < 1$ otherwise, unstable.

Proof:

We consider the partial derivative of the model equations, using the outcome to form the Jacobian matrix near the disease free-equilibrium point. That is

$$J\varepsilon_0 = \begin{pmatrix} -\mu_h & 0 & -\lambda_h\beta_h\frac{b_h}{\mu_h}, & 0 & 0 & 0 & -\lambda_h\beta_m\frac{b_h}{\mu_h}, & -\lambda_h\frac{\beta_{vh}Kb_h}{(K+P)^2\mu_h} \\ 0 & -(\alpha_1+\delta_1+\mu_h) & \lambda_h\beta_h\frac{b_h}{\mu_h} & 0 & 0 & 0 & \lambda_h\beta_m\frac{b_h}{\mu_h} & \lambda_h\frac{\beta_{vh}Kb_h}{(K+P)^2\mu_h} \\ 0 & \alpha_1 & -(3+\delta_2+\mu_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 3(1-\alpha_2) & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & -\lambda_m\beta_h\frac{b_m}{\mu_m} & 0 & -\mu_m & 0 & -\lambda_m\beta_m\frac{b_m}{\mu_m} & -\lambda_m\frac{\beta_{vm}Kb_m}{(K+P)^2\mu_m} \\ 0 & 0 & \lambda_m\beta_h\frac{b_m}{\mu_m} & 0 & 0 & -(\alpha_3+\delta_3+\mu_m) & \lambda_m\beta_m\frac{b_m}{\mu_m} & \lambda_m\frac{\beta_{vm}Kb_m}{(K+P)^2\mu_m} \\ 0 & 0 & 0 & 0 & 0 & -\alpha_3 & -(\delta_4+\mu_m) & 0 \\ 0 & \delta_1 & \delta_2 & 0 & 0 & \delta_3 & \delta_4 & -\varphi \end{pmatrix}$$

so that

$$J\varepsilon_0 = \begin{pmatrix} b_1 & 0 & b_2 & 0 & 0 & 0 & b_3 & b_4 \\ 0 & b_5 & b_6 & 0 & 0 & 0 & b_7 & b_8 \\ 0 & b_9 & b_{10} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & b_{11} & b_{12} & 0 & 0 & 0 & 0 \\ 0 & 0 & b_{13} & 0 & b_{14} & 0 & b_{15} & b_{16} \\ 0 & 0 & b_{17} & 0 & 0 & b_{18} & b_{19} & b_{20} \\ 0 & 0 & 0 & 0 & 0 & b_{21} & b_{22} & 0 \\ 0 & b_{23} & b_{24} & 0 & 0 & b_{25} & b_{26} & b_{27} \end{pmatrix},$$

where $b_1 = -\mu_h, b_2 = -\lambda_h\beta_h\frac{b_h}{\mu_h}, b_3 = -\lambda_h\beta_m\frac{b_h}{\mu_h}, b_4 = -\lambda_h\frac{\beta_{vh}Kb_h}{(K+P)^2\mu_h}, b_5 = -(\alpha_1+\delta_1+\mu_h), b_6 = \lambda_h\beta_h\frac{b_h}{\mu_h}, b_7 = \lambda_h\beta_m\frac{b_h}{\mu_h}, b_8 = \lambda_h\frac{\beta_{vh}Kb_h}{(K+P)^2\mu_h}, b_9 = \alpha_1, b_{10} = -(3+\delta_2+\mu_h), b_{11} = 3(1-\alpha_2), b_{12} = -\mu_h, b_{13} = -\lambda_m\beta_h\frac{b_m}{\mu_m}, b_{14} = -\mu_m, b_{15} = -\lambda_m\beta_m\frac{b_m}{\mu_m}, b_{16} = -\lambda_m\frac{\beta_{vm}Kb_m}{(K+P)^2\mu_m}, b_{17} = \lambda_m\beta_h\frac{b_m}{\mu_m}, b_{18} = -(\alpha_3+\delta_3+\mu_m), b_{19} = \lambda_m\beta_m\frac{b_m}{\mu_m}, b_{20} = \lambda_m\frac{\beta_{vm}Kb_m}{(K+P)^2\mu_m}$, $b_{21} = -\alpha_3, b_{22} = -(\delta_4+\mu_m), b_{23} = \delta_1, b_{24} = \delta_2, b_{25} = \delta_3, b_{26} = \delta_4$ and $b_{27} = -\varphi$.

Then we obtain the eigenvalues as λ_i for $i = 1,2,3, \ldots, 8$. Considering the eigenvalue thus far generated, we have $\lambda_1 = -\mu_h,\ \lambda_2 = -\mu_m, \lambda_3 - \mu_h$….. We conclude from here that if all the eigen values have negative real path, we say that $R_0 < 1$, showing local asymptotic stability, thus ending the poof by showing that the disease will gradually die out at this point.

2.6 Global stability analysis

Theorem 5. We check for global stability of the model by using the Castillo Chavez theorem (Castillo Chavez *et al.* 2002). The model is written in the form

(H1) $\frac{dx}{dt} = F(X, 0)$

(H2) $\frac{dI}{dt} = G(X, I), G(X, 0) = 0,$

where $X \in R^m$ is for the compartment of the uninfected individuals and $I \in R^n$ denotes the number of infected individuals, including the contaminant and $\varepsilon_0 = (X^*, 0)$ denotes the disease free-equilibrium of the system. The system will satisfy (H1) and (H2) before we say that the system is locally asymptomatically stable, and to generate the global stability, then:

$(H1)$ $\frac{dX}{dt} = F(X, 0),\ X^*$ is globally asymptotically stable

and

$(H1) = G(X, I) = AI - \hat{G}(X, I)\ \ , \hat{G}(X, I) \geq 0$ for $(X, I) \in \Omega$.

where $A = \frac{\partial G}{dt}(X^*, 0)$ is an M – matrix (the off diagonal elements of A are non-negative) and is where the model makes biological sense. Then the disease free-equilibrium is G.A.S provided that $R_0 < 1$.

Let $X = (S_h, R_h, S_m)$ and $I = (E_h, I_h, E_m, I_m, P)$

$$F(X, 0) = \begin{pmatrix} b_h - \mu_h S_h \\ \mu_h R_h \\ b_m - \mu_m S_m \end{pmatrix} \text{and}$$

$$G(X, I) = \begin{pmatrix} \lambda_h \beta_h I_h S_h + \lambda_h \beta_m I_m S_h + \lambda_h \frac{\beta_{vh} P}{K + P} S_h - (\alpha_1 + \delta_1 + \mu_h) E_h \\ \alpha_1 E_h - (3 + \delta_2 + \mu_h) I_h \\ \lambda_m \beta_h I_h S_m + \lambda_m \beta_m I_m S_m + \lambda_m \frac{\beta_{vm} P}{K + P} S_m - (\alpha_3 + \delta_3 + \mu_m) E_m \\ \alpha_2 E_m - (\delta_4 + \mu_m) I_m \\ \delta_1 E_h + \delta_2 I_h + \delta_3 E_m + \delta_4 I_m - \varphi P \end{pmatrix}$$

AI

$$= \begin{pmatrix} -(\alpha_1 + \delta_1 + \mu_h) & \lambda_h \beta_h S_h & 0 & \lambda_h \beta_m S_h & \lambda_h \frac{\beta_{vh} P}{K + P} S_h \\ \alpha_1 & -(3 + \delta_2 + \mu_h) & 0 & 0 & 0 \\ 0 & \lambda_m \beta_h S_m & -(\alpha_3 + \delta_3 + \mu_m) & \lambda_m \beta_m S_m & \lambda_m \frac{\beta_{vm} P}{K + P} S_m \\ 0 & 0 & \alpha_2 & -(\delta_3 + \mu_m) & 0 \\ \delta_1 & \delta_2 & \delta_3 & \delta_4 & -\varphi P \end{pmatrix} \begin{pmatrix} E_h \\ I_h \\ E_m \\ I_m \\ P \end{pmatrix}$$

$$\hat{G} = \begin{pmatrix} 0 + \lambda_h \beta_h I_h \left(\frac{b_h}{\mu_h} - S_h\right) + \lambda_h \beta_m I_m \left(\frac{b_h}{\mu_h} - S_h\right) + \lambda_h \frac{\beta_{vh} P}{K+P}\left(\frac{b_h}{\mu_h} - S_h\right) \\ 0 \\ 0 + \lambda_m \beta_h I_h \left(\frac{b_m}{\mu_m} - S_m\right) + \lambda_m \beta_m I_m \left(\frac{b_m}{\mu_m} - S_m\right) + \lambda_m \frac{\beta_{vm} P}{K+P}\left(\frac{b_m}{\mu_m} - S_m\right) \\ 0 \\ 0 \end{pmatrix}$$

Now if $\hat{G}(X, I) = \left(S_h^0 > S_h\right)$ and $(S_m^0 > S_m)$ for $\varepsilon_0 = X^* = \left(\frac{b_h}{\mu_h}, 0,0,0, \frac{b_m}{\mu_m}, 0,0,0\right)$ is the global asymptotic stability of the equlibrium of $\frac{dx}{dt} = F(X, 0)$ which ends the prove.

3 Discussion

In this work, we considered an SEIR modified model of Ebola virus affected by the environmental contaminants and primates, which spread the infection within the human population at any given time t due to interaction rate. We studied the dynamical behaviour of the proposed model by first proving the boundedness and non-negativity of solutions and then well-posedness of the model, then the dynamics is determined by the basic reproduction number R_0 that acts as the determining factor in controlling the infection of Ebola virus. The disease free-equilibrium was analyzed for stability, which showed that the disease free-equilibrium is locally asymptotically stable when $R_0 < 1$, meaning that the disease will gradually die out of the population and it was also revealed that as long as $\hat{G}(X, I) = \left(S_h^0 > S_h\right)$ and $(S_m^0 > S_m)$ for $\varepsilon_0 = X^* = \left(\frac{b_h}{\mu_h}, 0,0,0, \frac{b_m}{\mu_m}, 0,0,0\right)$, we attain a global stability of the model.

4 Conclusion

In this paper, we presented a deterministic model of Ebola transmission bringing into consideration the efficacy of primates and contamination from the environment as one of the contributing factors to the disease spread. The existence and uniqueness of the solutions to the model were proved. We went further to establish the stability of the model at disease free-equilibrium state using the Castillo Chavez theorem. It was shown that for $R_0 < 1$, the model is globally asymptotically stable. With this result, the disease will not strive further in the population.

If the public health workers can educate the masses on the need of constant environmental sanitation and have their domesticated animals (primates especially) well vaccinated, even the wild life conservers toeing the same path, we will have little to contend with as touching other primates beyond human reach and as such, the population will be healthy for human existence.

Our analysis is not exempt of limitations, looking at the individual graph of the eight compartmental subpopulations against time, there arises need for a proper digest as to the proper approach to proffer a lasting check in the transmission of this disease. So would suggest that more work be done from this point as a means of extension on the research work.

References

[1] Althanus, C.I. (2017) Estimating the reproduction number of Ebola virus (EBO) during 2014 outbreak in West Africa. PLOS Curr.

[2] Anorue O.F, and Modebei M.I. (2020). Modeling the effect of education based intervention in The control of Colera. International Journal of Research Iinnovation in Applied Science, 5(12): 2454-6194.

[3] Canacho, A., Kacharski, A.J. and Funks, S. et al. (2014). Potential for large outbreak of Ebola Virus disease. Epidemics, 9: 70-78.

[4] Chowell, G. and Viboud, C. (2015). Controlling Ebola: key roles of Ebola treatment centres. Lancet Infectious Disease, 15: 139-141.

[5] Isaac Chun Hai-Fung (2014). Cholera transmission dynamic models for public health pratitioners. Funf Emerging Themes in Epidemiology, 11:1.

[6] Khan, A., Naveed, M., Dur-e-Ahmed., and Imran, M. (2015) Estimating the basic reproductive ratio for Ebola outbreak in Liberia and Sierra Leone. Infectious Diseases of Poverty, 4: 13.

[7] Lagrand, J., Grais, R. F. and Boelle, P. Y. et al. (2007). Understanding the dynamics of Ebola epidemic. Epidemiology and Infection, 135: 610-616.

[8] Leah R. J. Mathematical Modeling of Cholera : from Bacterial life Histories to human Epidemics June 2006 Dissertation.

[9] LeroyE.M., Kumulungui B., and X. Pourrut et al. (2005). Fruit Bats as reservoirs. Nature, 438: 575-576.

[10] Merler, S., Ajelli, M. and Fumanelli, et al. (2015). Spatiotemporal spread of the 2014 outbreak of Ebola virus disease in Liberia and the effectiveness of non-pharmaceutical interventions: a computational modelling analysis. Lancet Infectious Disease, 15: 204-211.

[11] Mhlanga A. (2019). Dynamical analysis and control in modeling Ebola virus disease. Mhlanga Advances in Difference Equations. https://doi.org/10.1186/s/3662-019-2392-x 458.

[12] Pratik. B. (2019). Major thesis on analysis of prediction models in spread of Ebola virus Disease. Deakin University Australia.

[13] Rachah A.(2015). Mathematical modelling , simulation and optimal control of the 2014 Ebola outbreak in West Africa

[14] RafiqM., Ahmed W., Abbas M., and Baleanu D.(2020). A reliable and competitive mathematical analysis of Ebola epidemic model. Advancesin Difference Equations. 2020:540. https://doi.org/10.1186/s/3662-020-02994-2..

[15] Rhoubari Z., Besbassi, Hattaf H.K., and Yousfi N.(2018) Mathematical modeling of Ebola Virus Disease in Bat Population. Disease Dynamics in Nature and Society, Article ID 5104524. 7. https://doi.org/10.1155/2018/5104524.

[16] Simbiat A.A. (2018) A Mathematical Model of Ebola Virus Disease. Department of Mathematics University of Manitoba. Winnipeg.

[17] TahirM., ShahI.S., ZamenG., & MuhammedsG. (2018). Ebola Virus Epidemic Disease, its modelling and Stability Analysis required abstain strategies. Cogent Biol.4: 1488511. https://doi.org/10.1080/23312025.2018.1488511.

[18] T. Berge, M. Chapwanya, J.M.S Lubuma and Y.A Terefe. (2017). A mathematical model for Ebola epidemic with self protection measures.

[19] Tsanou B., Samuel B., Jean L., Martin L., and Mann M.. Modeling
Ebola virus disease transmission with reservoir in a complex virus life ecology.

[20] Zhi-Qiang Xia, Shi-Fu Wang et al. Modelling the transmission dynamics of Ebola virus diseasein Liberia. www.nature.com.Scientic reports /5:13857/DOI:10.1038/srep 13857.