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Threshold and Sensitivity Analysis of an Ebola Model with Case detection, Vaccination and Environmental Transmission

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Abstract: Ebola hemorrhagic fever, also known as Ebola virus disease (EVD) is a viral infection, usually with high fatality rate whenever there is a breakout. EVD has multiple transmission pathways, ranging from human-human, animal-human, human-environment and environment-human. With these multiple pathways, lots of preventive mechanisms have to be observed whenever there is a break-out so that the number of new cases can be minimized. Of such mechanisms are case detection, environmental sanitation, vaccination and other precautionary motives. Thus, a new mathematical model for EVD was proposed by incorporating case detection at each stage, with environmental contamination and vaccination impact on EVD transmission also considered. The aim of these measures is to check their effectiveness in reducing the number of EVD secondary cases. The model was qualitatively examined by establishing the region of feasible solution, which was found to exist and bounded. The non-negativity of solution for the model system of equation was established by using appropriate theorem. Equilibrium points of the system was analyzed at steady state, which was found to exist as disease free (DFE) and endemic equilibrium (EE). Basic reproduction number (R_0) of the model was computed by using the next generational matrix approach. Local stability analysis of the model was computed to show the region of solution for both the DFE and EE points. The global stability analysis was obtained by constructing a unique and appropriate Lyapunov function. The result shows that the DFE is stable globally (G.A.S) if $R_0 < 1$. Sensitivity analysis of the model was carried out to establish the effect of each parameter of the model on the (R_0). It was discovered from the sensitivity index table that contact rate is the most sensitive parameter in disease transmission because of its large positive value. Numerical simulation for the sensitivity analysis was obtained graphically by varying the values of each parameter. The study concluded that case-detection, quarantining and proper disposal (burial) of dead infective are effective measures in reducing the number of new case(s) of the infection.

Keywords: Threshold, Sensitivity, case-detection, vaccination, numerical simulation

1 Introduction

The Ebola virus disease (EVD) causes an acute and serious illness which is often fatal if left untreated. According to World Health Organization (WHO), EVD first appeared in 1976 in two simultaneous outbreaks in South Sudan and Democratic Republic of Congo (DRC). However, the $2014-2016$ outbreak in West Africa was the largest Ebola outbreak ever, as 28,616 confirmed cases and 11,310 deaths were reported in Guinea, Liberia, and Sierra Leone [6]. It is thought that fruit bats of the Pteropodidae family are natural Ebola virus hosts. Ebola is introduced into the human population through close contact with secretions, the blood, organs or other bodily fluids of infected animals (like fruit bats, chimpanzees, gorillas, monkeys, forest antelope or porcupines). Ebola then spreads through human-to-human transmission via direct contact (through broken skin or mucous membranes) with: (i) Blood or body fluids of a person who is sick with or has died from Ebola, and (ii) Objects that have been contaminated with body fluids (like blood, feces, vomit) from a person sick with Ebola or the body of a person who died from Ebola. The incubation period for this infection is from $2-21$ days before clinical

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symptoms will persist. Health-care workers have frequently been infected while treating patients with suspected or confirmed EVD. This occurs through close contact with patients when infection control precautions are not strictly practiced. Indeed, the nurse that attended to the first confirmed case of EVD in Nigeria died as a result of the disease. This necessitate the need for proper and adequate precautionary measures against this highly infectious disease. Early symptoms of EVD includes (but not limited to) fever, fatigue, headache and sore throat while acute symptoms can include rash, vomiting and diarrhea. Some of the strategies put in place to prevent the spread of EVD include outbreak containment measure like safe and dignified burial of the dead infective, and contact tracing, reducing the risk of wild-life to human transmission by reducing the hunting of carrier animals for food and reducing human-human transmission through regular washing of hand and social distancing. Several researchers have worked extensively on dynamics of transmission of EVD, in order to proffer both curative, preventive and other measures to eradicate EVD. Mathematical modeling has been a viable tool used in studying the dynamics of disease transmission, so as to offer an insight into future prevalence, elimination and control measures that can be adopted. Mathematical modeling simply implies mathematical representation of issues arising on daily basis. This has proven to be an important tool by which physical, biological, ecological and economical problems are solved. By interpreting and representing epidemiological issues as a form of mathematical equations (difference, differential, algebraic etc.), applied mathematicians proffer solution(s) to challenges encountered by biologists and public health workers, by using mathematical model as a potent tool to gain better insight to factors influencing the infectivity of epidemic diseases and provide good mechanism that minimizes the spread of such infections. The properties of these models are investigated qualitatively using the concept of stability analysis while other methods are employed to quantitatively find an approximate solution to the model equation(s), since most non-linear problems do not have exact solution.

Over the years, many mathematical models on EVD have been developed, analyzed and recommendations were drawn to reduce the prevalence of EVD infection. [7] developed a SEIR mathematical model for Ebola spread. Their result indicate that increase in immunity reduces new infections. Also, [16] presented an extended SEIR Ebola epidemic model. They computed the threshold parameter R_0 and used it to established the stability analysis of the model. They numerically solved the model using the non-standard finite difference (NSFD) and compared their result with Runge-Kutta order 4 (RK4). Their result showed that NSFD converges faster than RK4 when large step size is used. Many other models on EVD exist with their peculiar result (check [25], [12] and [15]). [**?**] developed a nonlinear fractional order EVD model with a novel piecewise hybrid technique to study the behaviour of Ebola within a mixed population. Their study investigated the effects of classical Caputo piecewise operator on the behavior of the model, and their findings showcased the advantages of fractional operators over the classical integer order, in enhancing the accuracy of the model to capture the intricate dynamics of the disease. [30] also adopted fractional order model to study the impact of immunization, prompt identification, sanitation and isolation, among others preventive measures, on the dynamics of EVD transmission. They adopted Laplace-Adomian decomposition method to simulate the system of fractional differential equations from their model. [2] discuss the transmission dynamics of EVD with vaccine, condom usage, quarantine, isolation and treatment drug. They concluded that effective implementation of those measures resulted in significant reduction of new EVD cases within 60 days. [4] considered a simple mathematical model for Ebola in Africa by incorporating the indirect transmission route like consumption of contaminated bush meat, environmental contamination and funeral practices for dead infective. Their results show that only one endemic equilibrium exist, which is locally asymptotically stable in the absence of shedding or manipulation of deceased individuals. However, no control measure was introduced to cater for their discovery.

2 Model Formulation

In this work, we proposed an extension to the models of [25] and [15] by including vaccination, exposed and quarantined classes to study the : (i) efficacy of the developed vaccine; (ii) effect of case detection at asymptomatic stage on overall prevalence of EVD within a population; and (iii) impact of the preventive measures (quarantining) on the dynamics of EVD. Total human population $N(t)$ is divided into 8 mutually exclusive groups (otherwise called compartments) denoted as susceptible $(S(t))$, exposed $(E(t))$, quarantined $(Q(t))$, infected $(I(t))$, hospitalized $(H(t))$, Vaccinated $(V(t))$,fully recovered $(R(t))$, deceased $(D(t))$ individuals. Thus,

$$N(t) = S(t)+V(t)+E(t)+Q(t)+I(t)+H(t)+R(t)+D(t) \tag{1}$$

Similarly, the concentration of Ebola virus in the environment is denoted as $P(t)$. The dynamics of EVD is such that human-human and human-environment transmission is possible. Thus, there is multiple mode of spread of the disease within the affected community. Existence of susceptible human interaction with the environment and infected human/animal bring a new case of EVD, this interaction is denoted as β. Generally, recruitment is assumed into the susceptible class at the rate π, and natural mortality occurs across all the classes at the rate μ. Due to diagnosis of EVD status at the I, H and D compartments, a modification parameter denoted

as c is introduced to cater for the reduced rate of new cases of infection from those classes. Further precautions are taken when dealing with the hospitalized and EVD deceased patients at the rate η_1 and η_2 respectively. Thus the force of infection is denoted as χ and given as:

$$\chi = \beta\left[P + c\left(I + \eta_1 H + \eta_2 D\right)\right] \tag{2}$$

The system of equations governing the model is given as:

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \mu S - \alpha S + \omega V - \chi S \\
\frac{dE}{dt} &= \chi S + \rho\chi V - (\mu + \theta_1 + \theta_2)E \\
\frac{dI}{dt} &= \theta_2 E - (\gamma_1 + \mu + \delta_1 + \tau_2)I \\
\frac{dQ}{dt} &= \theta_1 E - (\tau_1 + \mu)Q \\
\frac{dH}{dt} &= \tau_1 Q + \tau_2 I - (\gamma_2 + \mu + \delta_2)H \\
\frac{dR}{dt} &= \gamma_2 H + \gamma_1 I - \mu R \\
\frac{dD}{dt} &= (\mu + \delta_2)H + (\mu + \delta_1)I - \phi D \\
\frac{dV}{dt} &= \alpha S - \rho\chi V - (\mu + \omega)V \\
\frac{dP}{dt} &= \sigma_1 I + \sigma_2 D - \varepsilon P
\end{aligned} \tag{3}$$

The flow diagram for the above system of equation (3) is given below:

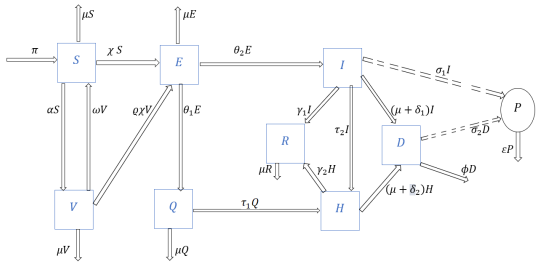

Figure A: Transfer diagram of the model.

It is important to mention some of the assumptions made when formulating this model, such as (i) only individuals in I and D sub-population are assumed to shred the Ebola virus (EV) into the environment. This implies that either the shedding rate of the virus by those at isolation centres $H(t)$ are assumed negligible or are properly disposed, (ii) Only unvaccinated susceptible individuals are recruited into the population and so on.

The parameters in the model and the value used for simulation is presented in table 1 below:

Table 1: Table of Parameter and Values

Symbol	Symbol Meaning	Value	Source
π	Recruitment rate	27	[12]
μ	Natural death	$\frac{1}{365\times 57.7}$	[29]
τ_1	Q-class Hospitalization	0.4	[5]
τ_2	I-class Hospitalization	0.64	[5]
ω	Vaccine waning	0.001	[4]
α	Vaccination rate	0.5	Guess
θ_1	Quarantine rate	0.7	[4]
θ_2	E to I Progression	0.4	[4]
γ_1	I-class Recovery	0.096	Guess
γ_2	H-class Recovery	0.56	Guess
σ_1	I-Class shedding	0.003	[5]
σ_2	D-class shedding	0.003	[5]
η_1	H-class Modification	0.04	Guess
η_2	D-class Modification	0.07	Guess
ε	EV environmental decay	0.75	Guess
c	I-classes Modification	0.0001	[12]
ρ	Vaccine rate failure	0.44	[4]
β	EVD contact rate	0.0003	[8]
ϕ	D-class safe disposal	0.04	[8]
δ_2	H-class EVD-death	0.365	[8]
δ_1	I-class EVD-death	0.4	Guess

3 Basic Properties of the Model

Qualitative analysis of the model shall be discussed in this section. For ease of computation, let $k_1 = \mu + \alpha$, $k_2 = \mu + \omega$, $k_3 = \mu + \theta_2 + \theta_1, k_4 = \mu + \tau_1$, $k_5 = \gamma_1 + \mu + \delta_1 + \tau_2$, $k_6 = \gamma_2 + \mu + \delta_2, k_7 = \mu + \delta_1$, $k_8 = \mu + \delta_2$ in system (3)

3.1 Feasibility Region of the Model

Theorem 1: Suppose $\{S(0), V(0), E(0), I(0), Q(0), H(0), R(0), D(0) \in \mathbb{R}^8_+\}$ denote the initial conditions for all state variables in system (3), and let every other solution set $\{S(t), V(t), E(t), I(t), Q(t), H(t), R(t), D(t)\} \in \Omega$ as $t \to \infty$. Then, the feasible solution which is a positively invariant set of the model is given by:

$$\Omega = \left\{(S, V, E, I, Q, H, R, D) \in \Re^8_+ : N(t) \leq \frac{\pi}{\mu}\right\} \tag{4}$$

Proof: Using equation (1), the dynamics of the model is such that

$$\left.\begin{aligned}
\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dQ}{dt} + \frac{dH}{dt} + \frac{dR}{dt} + \frac{dD}{dt} \\
&= \pi - \mu N - \phi D \\
&\leq \pi - \mu N \\
\frac{dN}{dt} &+ \mu N \leq \pi
\end{aligned}\right\} \tag{5}$$

solving (5) by integrating factor method and applying the initial condition $N(0) = N_0 \geq 0$ to obtain

$$\left.\begin{aligned} N(t) &\leq \frac{\pi}{\mu} + ke^{-\mu t} \\ N(t) &\leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu}\right)e^{-\mu t} \end{aligned}\right\} \quad (6)$$

As $t \to \infty$, it is obvious from the second function of (6) that $N(t) \leq \frac{\pi}{\mu}$. Thus for any time t, $0 \leq N(t) \leq \frac{\pi}{\mu}$, implies the trajectories of the model equation (3) are bounded in the region Ω as given in (4), which complete the proof.

3.2 Non-negativity of Solution

Non-negativity properties of a mathematical model is used to establish the existence of a positive solution for the model at any time t, given a positive initial population. The following theorem shall be used to establish the positivity of solution of (3)

Theorem 2: Suppose the initial population of the system in (3) is

$$\{S(0), V(0), E(0), Q(0), I(0), H(0), R(0), D(0) \geq 0\} \in \Omega$$

Then the solution set $\{S(t), V(t), E(t), Q(t), I(t), H(t), R(t), D(t) \geq 0\}$ for all time t.

Proof: Using each equation in system (3), starting with the first one, it is assumed:

$$\frac{dS}{dt} = \pi - \mu S - \alpha S + \omega V - \chi S$$
$$\geq \pi - (\mu + \alpha)S + \omega V, \quad \beta \in [0,1]$$

Integration and further simplification yields:

$$S(t) \geq S(0)e^{-(\mu+\alpha)t} \geq 0 \quad (7)$$

Since an exponential $e^x \geq 0 \ \forall \ x \in \Re$. Similarly, using the second equation:

$$\frac{dE}{dt} = \chi S + \rho\chi V - (\mu + \theta_1 + \theta_2)E \geq -(\mu + \theta_1 + \theta_2)E$$

$$E(t) \geq E(0)e^{-(\mu+\theta_1+\theta_2)t} \quad (8)$$

Apparently, solving for $V(t), Q(t), I(t), H(t), R(t), D(t)$ in system (3) yield a positive bounded solution for each in set (4). This completes the proof.

3.3 Disease Free Equilibrium

The solution of (3) at steady state such that $\Omega_d = \Omega_d(S_*, V_*, E_*, Q_*, I_*, H_*, R_*, D_*, P_*)$ denotes the disease free equilibrium (DFE) point gives:

$$\Omega_d = \left(\frac{k_2\pi}{k_1k_2 - \omega\alpha}, \frac{\alpha\pi}{k_1k_2 - \omega\alpha}, 0, 0, 0, 0, 0, 0, 0\right) \quad (9)$$

3.4 Computation of Basic Reproduction (R_0)

This important factor in epidemiology established the occurrence of secondary infection. To compute R_0, we adopt the next generational method (NGM), by categorizing the system of equations (3) into two, namely an infected compartments (E, I, H, D, Q, P) and non-infected compartments. F matrix denotes the appearance of new infection while V matrix is the movement in/out of the infected compartments. Reproduction number is obtained from the spectral radius $(\rho(FV^{-1}))$ of the interaction between the two categories. Thus, using NGM approach as adopted by [12] and [19] R_0 for system (3) was computed as follows:

$$F = \begin{pmatrix} 0 & 0 & B_0c & B_0c\eta_1 & B_0c\eta_2 & B_0B_0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

where $B_0 = \beta(S_* + \rho V_*)$

$$V = \begin{pmatrix} k_3 & 0 & 0 & 0 & 0 & 0 \\ \theta_1 & k_4 & 0 & 0 & 0 & 0 \\ \theta_2 & 0 & k_5 & 0 & 0 & 0 \\ 0 & -\tau_1 & -\tau_2 & k_6 & 0 & 0 \\ 0 & 0 & -k_7 & -k_8 & \phi & 0 \\ 0 & 0 & -\sigma_1 & 0 & -\sigma_2 & \varepsilon \end{pmatrix}$$

$$R_0 = \frac{\beta(k_2 + \alpha\varepsilon)[c\varepsilon(\phi\theta_2k_4k_6 + \phi\eta_1A_0 + \eta_2A_1) + A_2]}{\mu(\alpha + \omega + \mu)k_3k_4k_5k_6\phi\varepsilon} \quad (10)$$

where

$$A_0 = k_4\tau_2\theta_2 + k_5\tau_1\theta_1, \ A_1 = k_8A_0 + k_4k_6k_7\theta_2,$$

$$A_2 = \sigma_2A_1 + k_4k_6\sigma_1\phi\theta_2$$

3.5 Local Stability of DFE

Theorem: The disease free equilibrium Ω_d is locally asymptotically stable whenever $R_0 < 1$

Proof: The Jacobian matrix of (2) at Ω_d is locally asymptotically stable if $R_0 < 1$ and unstable otherwise

$$J = \begin{pmatrix} -k_1 & \omega & 0 & 0 & D_1 & D_1\eta_1 & D_1\eta_2 & -\beta S_* \\ \alpha & -k_2 & 0 & 0 & D_0V_* & D_0\eta_1V_* & D_0\eta_2S_* & -\beta\rho S_* \\ 0 & 0 & -k_3 & 0 & B_0c & B_0c\eta_1 & B_0c\eta_2 & B_0 \\ 0 & 0 & \theta_1 & -k_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta_2 & 0 & -k_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -k_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & k_7 & k_8 & -\phi & 0 \\ 0 & 0 & 0 & 0 & \sigma_1 & 0 & \sigma_2 & -\varepsilon \end{pmatrix} \quad (11)$$

Where $D_0 = -\beta c\rho, D_1 = -\beta c S_*$. The characteristics equation of (11) is written in compact form as

$$[(\lambda+k_1)(\lambda+k_2)-\alpha\omega][M-\lambda I_{6\times 6}]=0 \tag{12}$$

where

$$M=\begin{bmatrix} -k_3 & 0 & B_0c & B_0c\eta_1 & B_0c\eta_2 & B_0 \\ \theta_1 & -k_4 & 0 & 0 & 0 & 0 \\ \theta_2 & 0 & -k_5 & 0 & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -k_6 & 0 & 0 \\ 0 & 0 & k_7 & k_8 & -\phi & 0 \\ 0 & 0 & \sigma_1 & 0 & \sigma_2 & -\varepsilon \end{bmatrix} \tag{13}$$

From (12),

$$(\lambda+k_1)(\lambda+k_2)-\alpha\omega=0$$

$$\lambda^2+(k_1+k_2)\lambda_1-\alpha\omega=0 \tag{14}$$

Since all parameters values are non-negative,

$$k_1+k_2>0$$

$$k_1k_2-\alpha\omega=\mu(\alpha+\omega+\mu)>0$$

Thus, by Routh Huthwis criterion, (14) will give two negative eigenvalues.
Obviously $-M=[m_{ij}]$ is a 6×6 matrix such that $m_{ij}\neq 0$, for $i\neq j$, $i,j=1,2,\cdots,6$ and $m_{ii}>0, \forall\, i=1,2,\cdots,6$.

$$z=\left(1,\frac{\theta_1}{k_4},\frac{\theta_2}{k_5},\frac{A_0}{k_4k_5k_6},\frac{A_1}{k_4k_5k_6\phi},\frac{A_2}{k_4k_5k_6\varepsilon\phi}\right)^T \tag{15}$$

Such that

$$Mz=(k_3(1-R_0),0,0,0,0,0)^T \text{ if } R_0<1 \tag{16}$$

and

$$|-Mz|=k_3k_4k_5k_6\varepsilon\phi(1-R_0)>0 \text{ if } R_0<1 \tag{17}$$

Then by matrix theory, M is stable. Hence, the real part of all eigenvalues of $J(\Omega_d)$ are negative provided $R_0<1$ and so Ω_d is locally asymptotically stable for $R_0<1$

3.6 Global Stability of Ω_d

Theorem: The disease free equilibrium Ω_d is globally asymptotically stable if and only if $R_0<1$ and unstable otherwise
Proof: Let L be a Lyapunov function

$$L=v_1E+v_2Q+v_3I+v_4H+v_5D+v_6P \tag{18}$$

where

$$\begin{aligned} v_1&=\frac{R_0[k_1k_2-\alpha\omega]}{(k_2+\varepsilon\alpha)\pi} \\ v_2&=\frac{\beta\tau_1[c\varepsilon(\phi\tau_1+k_8\tau_2)+k_8\sigma_2]}{\varepsilon\phi k_4k_6} \\ v_3&=\frac{\beta}{\varepsilon\phi k_5k_6}[c\varepsilon(\phi k_6+\phi\tau_1)\tau_2+ \\ &\quad (k_6k_7+k_8\tau_2)\tau_2+\sigma_2(k_6k_7+k_8\tau_2)] \\ v_4&=\frac{\beta[\varepsilon(\phi\tau_1+k_8\tau_2)+k_8\tau_2]}{\varepsilon\phi k_6} \\ v_5&=\frac{\beta[c\varepsilon\phi\tau_2+\sigma_2]}{\varepsilon\phi}\quad v_6=\frac{\beta}{\varepsilon} \end{aligned} \tag{19}$$

Differentiating (18) with respect to time

$$\dot{L}_0=v_1\dot{E}+v_2\dot{Q}+v_3\dot{I}+v_4\dot{H}+v_5\dot{D}+v_6\dot{P}$$

$$\left.\begin{aligned} \dot{L}_0&=v_1[\chi(S+\varepsilon V)-k_3E]+v_2[\phi_1E-k_4Q]+ \\ &v_3[\phi_2E-k_5I]+v_4[\tau_1Q+\tau_2I-k_6H]+ \\ &v_5[k_7I+k_8H-\phi D]+v_6[\sigma_1I+\sigma_2D-\varepsilon P] \end{aligned}\right\} \tag{20}$$

Simplifying (20) to get

$$\left.\begin{aligned} \dot{L}_0&=[v_2\theta_1+v_3\theta_2-v_1k_3]E+ \\ &[v_1\beta c(S+\varepsilon V)-v_3k_5+v_4\tau_2+v_5k_7+v_6\sigma_1]I \\ &+[\eta_1v_1\beta c(S+\varepsilon V)-v_4k_6+v_5k_8]H+ \\ &[v_1\beta c\eta_2(S+\varepsilon V)-v_5\phi+v_6\sigma_2]D \\ &+[v_1\beta(S+\varepsilon V)-\varepsilon]P \end{aligned}\right\} \tag{21}$$

Since $S+\varepsilon V\leq\frac{(k_2+\varepsilon\alpha)\pi}{k_1k_2-\alpha\omega}$, (21) becomes

$$\left.\begin{aligned} \dot{L}_0&\leq[v_2\theta_1+v_3\theta_2-v_1k_3]E+ \\ &\left[\frac{\pi v_1\beta c(k_2+\varepsilon\alpha)}{k_1k_2-\alpha\omega}-v_3k_5+v_4\tau_2+v_5k_7+v_6\sigma_1\right]I \\ &+\left[\frac{\eta_1v_1\beta c(k_2+\varepsilon\alpha)\pi}{k_1k_2-\alpha\omega}-v_4k_6+v_5k_8\right]H+ \\ &\left[\frac{\pi v_1\beta c\eta_2(k_2+\varepsilon\alpha)}{k_1k_2-\alpha\omega}-v_5\phi+v_6\sigma_2\right]D \\ &+\left[\frac{\pi v_1\beta(k_2+\varepsilon\alpha)}{k_1k_2-\alpha\omega}-\varepsilon\right]P \end{aligned}\right\} \tag{22}$$

Substitute (19) into (22) to get

$$\dot{L}_0=\beta[\rho+c(I_1+\eta_1H+\eta_2D)](R_0-1) \tag{23}$$

Now $\dot{L}_0<0$ of $R_0<1$ and $\dot{L}_0=0$ of $R_0=1$ or $I_1=H=D=0$. The largest compact invariant set in $\{(S,V,E,Q,I,H,R,D,P)\in\Omega:\ \dot{L}_0=0\}$ is the singleton $\{\Omega_d\}$. Therefore, by Lasalle invariant principle, every solution to system (3) with initial conditions in Ω approaches Ω_d as $t\to\infty$. Thus, since the region Ω is positively-invariant, then the disease free equilibrium Ω_d is globally asymptotically stable in Ω of $R_0<1$

3.7 Existence of Endemic Equilibrium

The solution of (3) steady state such that Ω_e denotes the endemic equilibrium point gives

$$\begin{aligned}
&S^* = \frac{\pi(\rho\chi^* + k_2)}{A^*},\ V^* = \frac{\pi\alpha}{A^*},\ E^* = \frac{\pi\chi^* B^*}{k_3 A^*},\\
&Q^* = \frac{\pi\theta_1\chi^* B^*}{k_3k_4A^*},\ \ I^* = \frac{\pi\theta_2\chi^* B^*}{k_3k_4k_5A^*},\\
&H^* = \frac{\pi A_1\chi^* B^*}{k_3k_4k_5k_6A^*},\ R^* = \frac{\pi\chi^* B^*(\gamma_2 A_0 + ak_4k_6\theta_2\gamma_1)}{k_3k_4k_5k_6A^*}\\
&P^* = \frac{\pi A_3\chi^* B^*}{k_3k_4k_5k_6\varepsilon\phi A^*},\quad D^* = \frac{\pi A_1\chi^* B^*}{k_3k_4k_5k_6\phi A^*}.
\end{aligned} \tag{24}$$

where

$$\chi^* = \beta[P^* + c(I^* + \eta_1 H^* + \eta_2 D^*)] \tag{25}$$

$$A^* = \rho\chi^{*2} + \chi^*(\rho k_1 + k_2) + k_1k_2 - \alpha\omega \tag{26}$$

$$B^* = \rho\chi^* + \rho\alpha + k_2 \tag{27}$$

Substitute (24) into (25) to obtain

$$b_0\chi^{*2} + b_1\chi^* + b_2 = 0 \tag{28}$$

where

$$\begin{aligned}
&b_0 = k_3k_4k_5k_6\rho\varepsilon\phi,\ \ b_1 = k_3k_4k_5k_6\varepsilon\phi(\rho k_1 + k_2) -\\
&\beta\rho c\varepsilon(\phi\theta_2k_4k_6 + \phi\eta_1A_0 + \eta_2A_1) - \beta\rho A_2\\
&b_2 = k_3k_4k_5k_6\varepsilon\phi(k_1k_2 - \alpha\omega)(1 - R_0)
\end{aligned}$$

It is clear that $b_0 > 0$ since all parameters values are non negative and $b_2 < 0$ for $R_0 > 1$. Hence, (3) has a unique positive root by Descarte's rate of sign. This implies that the following result is established.

Lemma 1 The model (3) has a unique positive (endemic) equilibrium if and only if $R_0 > 1$

3.8 Threshold and Sensitivity Analysis of Model Parameters to R_0

Threshold analysis of the basic reproduction number with respect to a parameter in the model gives the trend (upward or downward) to which R_0 will follow when the parameter value either decreases or increases. Most of the time it gives the sign in the deviation of R_0 as the parameter in question changes. It is measured by obtaining the partial derivative of the basic reproduction number with respect to the parameter, that is, $T_{X_i}^{R_0} = \frac{\partial R_0}{\partial X_i}$ where X_i denote the model parameter under consideration. The threshold analysis of each parameter has an increasing effect on R_0 if $\frac{\partial R_0}{\partial X_i} > 0$ and a decreasing effect if $\frac{\partial R_0}{\partial X_i} < 0$.

The result obtained for the threshold analysis of each parameters with respect to the basic reproduction is easy to understand using numerical value rather than the exact expression. For instance, the expression obtained for threshold analysis for the clearance rate of dead infectives (c), shedding rates σ_1 and σ_2 respectively are given as:

$$\begin{aligned}
\frac{\partial R_0}{\partial c} &= \frac{\beta(k_2 + \alpha\varepsilon)[\varepsilon(\phi\theta_2k_4k_6 + \phi\eta_1A_0 + \eta_2A_1)]}{(k_1k_2 - \alpha\omega)k_3k_4k_5k_6\phi\varepsilon}\\
\frac{\partial R_0}{\partial \sigma_1} &= \frac{\beta(k_2 + \alpha\varepsilon)}{(k_1k_2 - \alpha\omega)k_3k_5\phi\varepsilon}\\
\frac{\partial R_0}{\partial \sigma_2} &= \frac{\beta(k_2 + \alpha\varepsilon)(k_5k_6\alpha\varepsilon\theta_2 + k_4\tau_2\theta_2 + k_5k_8)}{(k_1k_2 - \alpha\omega)k_3k_4k_5k_6\phi\varepsilon}
\end{aligned} \tag{29}$$

The sensitivity index of a variable R_0 with respect to a parameter is the ratio of the relative change in R_0 compared to a change in the parameter. It uses the threshold analysis of the basic reproduction number to compute the "'actual" effect expected by the change in the parameter value to the overall effective number. The formula for the normalized forward sensitivity index of R_0 with respect to the parameters for the model system is thus given as:

$$\Upsilon_{X_i}^{R_0} = \frac{\partial R_0}{\partial X_i} \times \frac{X_i}{R_0} \tag{30}$$

Some of the expressions obtained for sensitivity analysis of the model is given as:

$$\begin{aligned}
&\Upsilon_{\sigma_1}^{R_0} = \sigma_1k_4k_6/c\varepsilon\,[\phi\,(k_4k_6\theta_2 + \phi\,(k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,\eta_1 +\\
&(k_5k_6k_7\theta_2 + (k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,k_8\eta_2))] +\\
&k_4k_6\sigma_1\,(k_5k_6k_7\theta_2 + k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,k_8\sigma_2\\
&\Upsilon_{\sigma_2}^{R_0} = (k_5k_6k_7\theta_2 + (k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,k_8)\,\sigma_2/c\varepsilon\,[\phi\,(k_4k_6\theta_2 +\\
&\phi\,(k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,\eta_1 +\ \ (k_5k_6k_7\theta_2 +\\
&(k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,k_8\eta_2))]\\
&+ k_4k_6\sigma_1\,(k_5k_6k_7\theta_2 + k_4\tau_2\theta_2 + k_5\tau_1\theta_1)\,k_8\sigma_2
\end{aligned} \tag{31}$$

Using the parameter value in table 1, the basic reproduction number was obtained as 3.5668. The numerical result obtained for the threshold and sensitivity analysis is given in the Table 2 below:

Table 2: Numerical Result for Threshold and Sensitivity Analysis

Parameter	Threshold Analysis	Sensitivity Value
β	11889.2896	1
η_1	0.006843	0.00007674
δ_1	0.001627	0.366038
θ_1	−0.53787738	−0.1055611
θ_2	0.9416703	0.105604325
δ_2	0.004264	0.095924
τ_1	0.00056175	0.000062998
τ_2	−0.52083268	−0.0934546
α	−0.018890672	−0.002648
σ_1	32.118199989	0.02701439
σ_2	1153.897253	0.970535
γ_1	−1.47320036	−0.0396511
γ_2	−2.7054575	−0.424767
μ	−75112.183776	−0.999918
ω	9.01717998	0.002528

From the table, every parameter with a positive value increases the basic reproduction number while those with negative value decreases R_0.
By varying the value of a parameter, while others are kept constant, the following graphical solutions based on reproduction number R_0 were obtained.

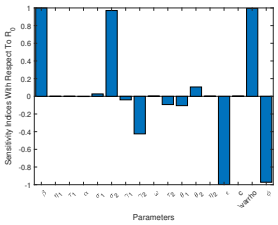

Figure 1: Sensitivity indices of the model parameters

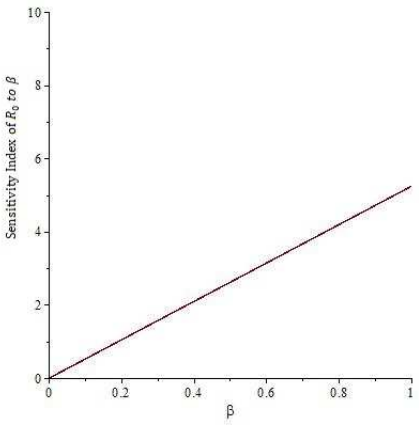

Figure 2: Variation of β to R_0

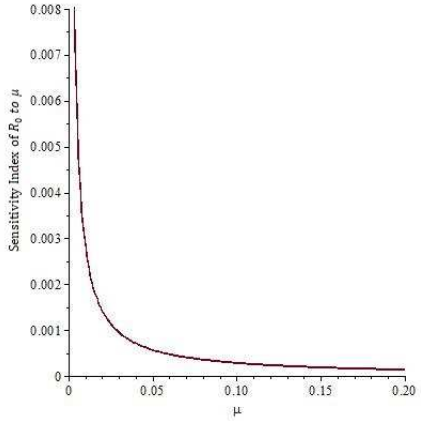

Figure 3: Variation of μ to R_0

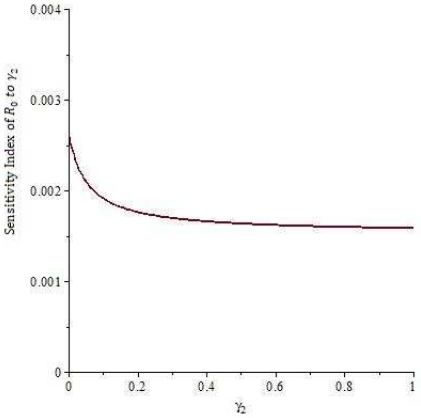

Figure 4: Variation of γ_2 to R_0

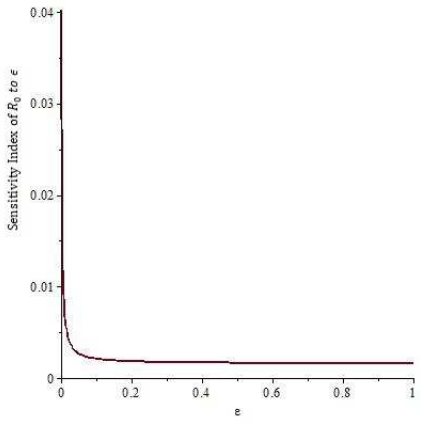

Figure 5: Variation of ε to R_0

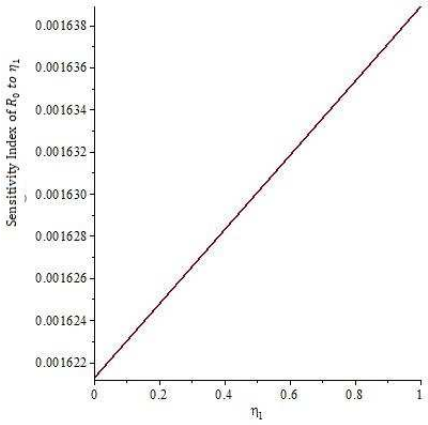

Figure 6: Variation of η_1 to R_0

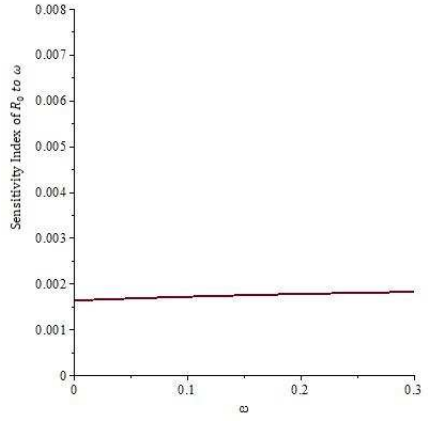

Figure 7: Variation of ω to R_0

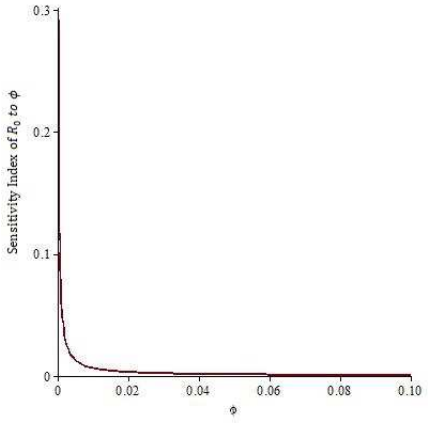

Figure 8: Variation of ϕ to R_0

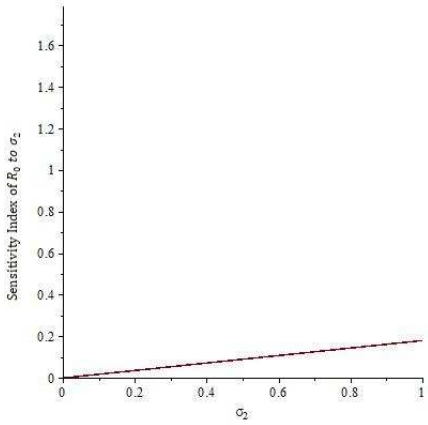

Figure 9: Variation of σ_2 to R_0

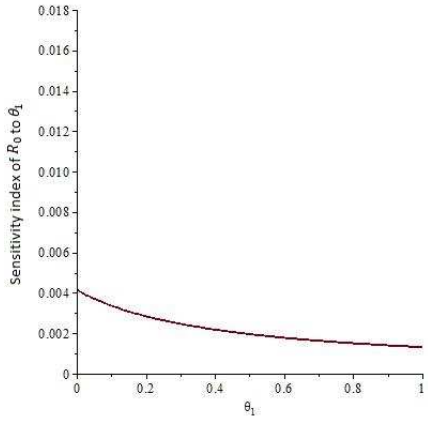

Figure 10: Variation of θ_1 to R_0

4 Numerical Simulation & Discussion of Result

Threshold and sensitivity analyses of a model forms an integral part of qualitative analysis to be done in making the right recommendation on disease eradication from the society. While threshold will pinpoint the direction of flow of a parameter towards disease eradication, sensitivity will give the appropriate effect of such parameter. The graphs in figure $2-10$ depicts the effect of varying a parameter and its effect on the R_0. Figure 2 shows an increase in R_0 as value of contact rate β increases. Indeed, the most sensitive parameter to R_0 is β as can be seen in the graph. The rate of increase in R_0 as β increases slightly is significant. Thus, to lower the spread of EVD within a population, all efforts must be directed towards reducing the contact with an infective individual. This necessitate the need for case detection and quarantining as we presented in our model. Figure 3 depicts the effect of varying μ on the overall R_0. Proper treatment of deceased at burial reduces significantly the number of new cases that may arise. The effect of variation of recovery rate γ_2 on the reproduction number R_0 was graphically presented in figure 4. This shows that hospitalization and treatment of detected cases helps in

reducing the number of new cases. Since there is an improvement in treatment strategy for EVD, it is important for anyone with symptoms to get tested and treated appropriately. It was recommended that treatment for this disease should be subsidized by the government for easy accessibility. Figure 5 is that of variation of EV decay rate (ε) from the environment and its effect on R_0. The curve shows a decline in R_0 as the value ε increases. Thus, proper sanitation and disinfecting the environment contributes immensely to reduction of new cases of EVD. In figure 6, variation of η_1 (modification parameter associated with infected class) was plotted against the reproduction number R_0. The graph obtained has a positive slope, which implies that lack of sensitization will trigger the spread of EVD in the population. It is important to state that, if proper education, awareness and sensitization against the spread of EVD is not well disseminated, then peoples' contact with the infection will increase, thereby trigger the increase in R_0. Effect of vaccine waning rate (ω) against R_0 was depicted in figure 7. It has a slight effect on the reproduction number as can be seen in the graph. In figure 8, the effect of safe disposal (burial) of dead infective was plotted against R_0 and it was observed that it has a downward effect on R_0. In figure 9, shedding rate of D-compartment (σ_2) was plotted against R_0 and the result gives a positive slope curve. This implies that, dead infective gives more Ebola virus (EV) to the community if not properly handled. Variation of θ_1 (quarantine rate) against R_0 is presented in figure 10. The earlier the removal of an infectious/exposed individual from the community, the lower the spread of EVD within the population.
Using $S(0) = 43580, V(0) = 6000, E(0) = 170, Q(0) = 80, I(0) = 50, H(0) = 20, R(0) = 92, D(0) = 8, P(0) = 400$ as the initial conditions for the state variables, together with parameter values in table (1), the dynamics of the model system of equation was plotted at the DFE point to obtain the following graphs

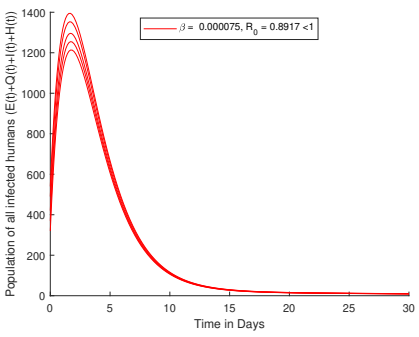

Figure 11: DFE simulation for infected classes

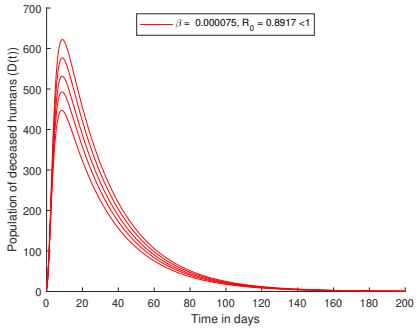

Figure 12: DFE simulation for Deceased class only

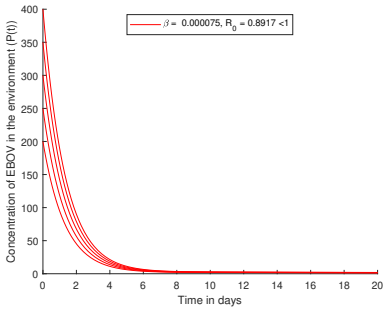

Figure 13: DFE simulation for $P-$ class only

Figure $11-13$ showed that regardless of the initial population, the disease free equilibrium point approaches a common value over a period of time t.

5 Conclusion

In this work, a mathematical model to study the dynamics of EVD transmission was presented for analysis in the presence of some of the preventive measures like case detection, vaccination, quarantining and environmental sanitation. The region of feasible solution for the model was computed. The result showed that the model is bounded for all time t. Basic reproduction number for the model was computed. This is the parameter that determines the number of secondary cases that may arise from the introduction of a single infected individuals into the population of susceptible people. This threshold parameter was used to analyze the sensitivity of model parameters in triggering/reducing the spread of EVD within a certain population. It was observed that reducing contact rate, shedding rate and proper preventive measures can lower the number of new cases of this infection because their reduction reduces R_0 while their increase also increases R_0. The study concluded that case detection at early stage is effective in reducing β (contact

with undetected cases at both asymptomatic/symptomatic stages).

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