A Nonlinear Model for the Analysis of the Impact of a Comprehensive Treatment and Management of Ebola Virus Disease

Din. S. Sarki, Chukwudi Okoye, Sunday I. Onah, Godwin C. E. Mbah

ABSTRACT: This paper addresses the spread of Ebola virus disease in human population by developing a model that considers a wide range of control parameters, including disease surveillance, the availability of isolation centres, improved personal hygiene and the basic biological features of the disease including infectious corpses, using a system ordinary differential equation. The disease free equilibrium of the model is shown to be both locally and globally asymptotically stable provided the associated reproduction number is less than unity. Further analysis of the basic reproduction quantity has indicated the substantial benefit of the deployment of sufficient levels of requisite treatment schedules provided such strategies. We proved the existence of at least one endemic equilibrium point for all $R_E > 1$. In the absence of disease-induced death, we proved that the transcritical bifurcation at $R_E = 1$ is supercritical (forward). Numerical simulations shows that for larger values of the disease-induced death rate, the disease can be effectively contained even in the absence of organised treatment.

Key words. Ebola, epidemic model, reproductive number, bifurcation theory, endemic equilibria, disease-free equilibria, stability analysis.

1. INTRODUCTION

Ebola virus disease (EVD), formerly known as Ebola haemorrhagic fever, is caused by a zoonotic virus first discovered in 1976 in the remote villages of the Democratic Republic of Congo (DRC, formerly Zaire) and Sudan [9]. Ebola is a unique member of the ribonucleic acid virus family that has no known natural reservoir [6]. However, Ebola is notably transmitted into the human population through physical contact with blood, secretions, organs or other bodily liquids of infected animals such as chimpanzees, gorillas, fruit bats, monkeys, forest antelope and porcupines found ill or dead or in the rain forest [9], additionally, such contacts with utensils that have been sufficiently contaminated with such fluids, including unprotected sex with patients who have recently recovered from the disease [10]. Additionally, Ebola in humans is caused by four of five viruses of the genus Ebola virus – Bundibugyo

virus (BDBV), Sudan virus (SUDV), Ta Forest virus (TAFV) and the last, simply called Ebola virus (EBOV, formerly Zaire Ebola virus). The incubation period of Ebola is 2-21 days and the infectious period is 4-10 days [6], [5], [9], and [10]. The onset of Ebola is characterised by severe headaches, malaise, fever, vomiting, bloody diarrhoea, and rash. Severe bleeding and shock are usually followed by death [6]. The Zaire Ebola virus is characterised by haemorrhagic fever and high mortality rate [4]. Diagnosis of Ebola can be difficult, because Ebola is frequently misdiagnosed as typhoid and/or malaria. Currently there is no treatment of Ebola [10] and [7]. Since the first outbreak of the Ebola Virus Disease (EVD) in 1976 in the Democratic Republic of the Congo (DRC) and Sudan [3], more than 20 outbreaks have been reported across eastern and central Africa. The recent incidence, which started in a remote village located in Guékédou in 2013 is the longest and most fatal. As at 11 February, 2015, the total EVD cases stood at 22,859 while deaths were 9,162 deaths [11]. Compared to the 32 years (1976-2012) cumulative sum of past episodes – 2,232 infected people and 1,503 deaths – there are now over ten times the total number of infection cases and over six times the total number of fatalities. In fact, in only around six months, there were 3,774 cases of people infected by EVD and 1,888 deaths in severely-hit countries. In addition to spreading to several West African countries (Senegal, Nigeria and Mali), the EVD has also been detected in other parts of the world, including Spain, Italy, Germany and the United States of America. Further, while the world grappled with the spreading wave of EVD in West Africa, a different strain of the virus was remotely discovered in some villages in Jeera County of the DRC in August 2014.The impact of the recent pandemic, as estimated, is outrageous due to the unprecedented multi-country outbreaks occurring simultaneously across geographical divides. The complexity of the spread has equally been most unprecedented. The death of 488 of the 830 infected health workers and the dimensional transmission through air travel has adversely aggravated its spread and substantially overwhelmed the internal capacity of highest-hit countries for containment. Though all age groups are affected, those most vulnerable to EVD include: the most active segment of the population who bare the heaviest toll, about 20 percent of the infected cases are children, while on per 100,000 populations, women are more affected than men (118 against 115). This is largely due to their frequent exposure to the disease transmission vectors such as vomit or other bodily fluids of an infected family member as well as their roles in certain traditional practices and rituals performed on the deceased. Others at high risk include people in the border regions where infections can be transmitted easily through porous borders; people in capital cities where infected people may go for treatment, particularly those in the slums of these cities; and poor rural areas with inadequate clean water and sanitation facilities. Factors favouring EVD

spread and intervention include the relatively free movement of goods and people and the close community ties across endemic (West African) countries, ignorance or lack of knowledge and preparedness, misdiagnosis by health professionals due to early symptoms resemblance with other endemic diseases such as malaria, cholera and Lassa fever as well as health workers' reluctance to provide care; public's reluctance to engage in contact tracing due to fear of infection, hesitation of infected persons to present themselves for treatment; and risky cultural practices, transfer of corpses over long distances for burials near their ancestors, issues of social, cultural and economic status, the difficulty of coordinating Ebola-related aid and the incapacity of available infrastructure to effectively manage and treat infected patients.

The enormous public health burden inflicted by EVD necessitates the use of mathematical modelling to gain insights into its transmission dynamics and to determine effective control strategies. The present study aims at complementing therapeutic efforts, by developing and qualitatively analysing a robust and comprehensive deterministic model for gaining insights into the impact of an elaborate treatment strategy in controlling EVD in a population. The model allows for the assessment of treatment strategies for each identified disease class. The model will be used to assess the public health (epidemiological) impact of three major treatment strategies, namely: (i) treating asymptomatic individuals, (ii) treating quarantined infected people (quarantine strategy) and (iii) treating EVD infected people who have evade quarantine (quarantine-evasive strategy).

The paper is organised as follows. The model is formulated in Section 2. The Ebola model is analysed in Section 3. The analysis of the model for the stability of the associated disease-free equilibrium is in Section 4, and numerical simulations are carried out in Section 5

2. Model formulation and basic properties.

The paper has distinguished two population types, human and corpse, each subdivided into mutually-exclusive compartments at time *t*. The total human population, denoted by $H(t)$, is comprised of susceptible individuals, denoted by $(S(t))$, individuals asymptotically-infected with EVD, denoted by $(E(t))$, quarantined individuals who are EVD infectious, denoted by $(I_{\mathrm{Q}}(t))$, EVD infectious individuals who have evaded quarantine, denoted by $(I_{\mathrm{N}}(t))$ and individuals who have been effectively treated and have recovered of active EVD, denoted by $(R(t))$. The corpse population, denoted by $C(t)$, on the other hand has been distinguished into non-infectious $C_{\mathrm{N}}(t)$ and infectious $C_{\mathrm{I}}(t)$ compartments. Thus

$H(t) = S(t) + E(t) + I_{\mathrm{Q}}(t) + I_{\mathrm{N}}(t) + R(t)$ while

$C(t) = C_{\mathrm{N}}(t) + C_{\mathrm{I}}(t)$.

We have followed the porous border nature of the majority of the aforementioned countries to basically assume that susceptible individuals are constantly added to the population by recruitment at a rate Π. Further, the dynamics of EVD transmission across the two population types is described as follows

2.1. Transmission by infectious humans

Susceptible individuals become infected with EVD following effective contact with the bodily fluids of infected humans at a rate λ_{H}, given by

$$\lambda_{\mathrm{H}} = \mathrm{E}(1-h\xi)\frac{\beta_3 R + (1-q\sigma\theta)(\beta_1 I_{\mathrm{Q}} + \beta_2 I_{\mathrm{N}})}{H}, \qquad (1)$$

Where E is the effective contact rate for EVD transmission, β_1, β_2 and β_3 are the respective relative effective infectious contact rates from quarantined infectious persons, infectious individuals who have evaded quarantine and infectious persons undergoing (or have completed) treatment. The modification parameters $0 \le \beta_3 < 1, 0 \le q \le 1$ and $0 \le \sigma \le 1$ each models the reduced infection rate due to effective treatment, number of quarantined individuals and surveillance coverage; others, $0 \le \theta < 1, 0 \le \xi < 1$ and $0 \le h \le 1$ each accounts for the availability of isolation centres, rate of public enlightenment and enhanced personal hygiene (due to public enlightenment). Thus we define the force of infection of EVD from infectious humans, λ_{H}, as the sum of the force of infection from quarantined infectious humans, infectious humans who evaded quarantine and treated individuals. Further, we define the probability of disease transmission from quarantined and non-infectious human to susceptible humans, respectively, as I_{Q}/H and I_{N}/H, while that from treated individuals as R/H.

2.1.1. Transmission by infectious corpses

Similarly, susceptible individuals acquire EVD following effective infectious contacts with the bodily fluids of infected corpses at a rate λ_{C}, given by

$$\lambda_{\mathrm{C}} = \mathrm{E}\beta_4(1-h\xi)\frac{C_{\mathrm{I}}}{C} \qquad (2)$$

Following from this we define the force of infection from infectious corpses, λ_{C}, as the effective contact rate that a susceptible human has with an infectious corpse per unit time, E, the probability of disease transmission from an infectious corpse, β_4 and the probability that a corpse is infectious, C_{I}/C. Thus we model the total force of infection of EVD, λ, as the sum of the respective forces of infection from humans and corpses. Finally, natural mortality occurs in all classes at a rate μ, while infectious individuals are liable to disease induced death at a rate δ. Combining all the aforementioned assumptions and definitions, the model for the transformation of Ebola in an extremely characteristic migration prone population is given by the following system of ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - (\mu+\lambda)S + \eta(1-\rho)R,\\
\frac{dE}{dt} &= \lambda S + \eta\rho R - K_1 E,\\
\frac{dI_Q}{dt} &= \alpha\phi_1 E - K_2 I_Q,\\
\frac{dI_N}{dt} &= \alpha\phi_2 E - K_3 I_N, \qquad (1)\\
\frac{dR}{dt} &= \tau_1 E + \tau_2 I_Q + \tau_3 I_N - K_4 R,\\
\frac{dC_N}{dt} &= \mu H - \psi C_N,\\
\frac{dC_I}{dt} &= \delta I_{EVD} - \psi C_I,
\end{aligned}$$

where

$$K_1 = \mu+\tau_1+\alpha\phi, K_2 = \mu+\tau_2+\delta, K_3 = \mu+\tau_3+\delta, K_4 = \mu+\eta.$$

The total compartmental sizes are:

$$H = S+E+I_Q+I_N+R,\ I_{EVD} = I_Q+I_N,\ C = C_N+C_I;$$

representing, respectively the total human population, infectious humans and the number of recorded corpses; with

$$\begin{aligned}
\frac{dH}{dt} &= \Pi - \delta I_{EVD} - \mu H,\\
\frac{dI_{EVD}}{dt} &= \alpha\phi E - (\tau_2 I_Q + \tau_3 I_N) - (\mu+\delta) I_{EVD},\\
\frac{dC}{dt} &= \mu H + \delta I_{EVD} - \psi C
\end{aligned}$$

(2)

and the EVD infection rate from humans and corpses are

$$\lambda_H = E(1-h\xi)\frac{(1-q\sigma\theta)(\beta_1 I_Q + \beta_2 I_N) + \beta_3 R}{H} \quad \text{and}$$

$$\lambda_C = E\beta_4(1-h\xi)\frac{C_I}{C} \quad (3)$$

Respectively. Thus the EVD effective infection rate is the sum

$$\lambda = E(1-h\xi)\left\{\frac{\beta_4 C_I H + C\left[(1-q\sigma\theta)(\beta_1 I_Q + \beta_2 I_N) + \beta_3 R\right]}{CH}\right\} \quad (4)$$

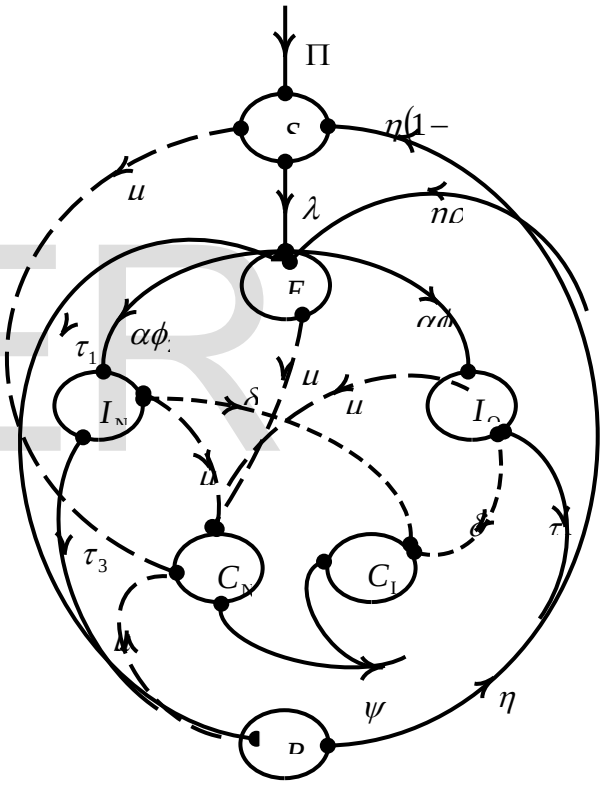

Figure 1: Flow

Table 1: *The state variables for the Ebola model*

Variable	Description
$S(t)$	Number of susceptible humans
$E(t)$	Number of humans exposed to EVD
$I_{\mathrm{EVD}}(t)$	Number of all infectious humans; it comprises of: I_{Q} and I_{N} where:
$I_{\mathrm{Q}}(t)$	Number of quarantined infectious living humans
$I_{\mathrm{N}}(t)$	Number of infectious living humans evading quarantine
$R(t)$	Number of recovered humans
$C_{\mathrm{N}}(t)$	Number non-infectious corpses
$C_{\mathrm{I}}(t)$	Number of infectious corpses

Table 2: *The parameters for the Ebola model*

Parameter	Description	Units
Π	General human recruitment (birth and immigration) rate	$\mathrm{Humans} \times \mathrm{Time}^{-1}$
λ_{H}	EVD infection rate from infectious humans	$\mathrm{Humans} \times \mathrm{Time}^{-1}$
λ_{C}	EVD infection rate from infectious corpses	$\mathrm{Corpse} \times \mathrm{Time}^{-1}$
λ	The force of infection from infectious humans and corpses	Dimensionless
α	Per capita disease maturation (progression) rate of individuals from the exposed (asymptomatic) stage to infectious Ebola stage. $1/\alpha$ is the average duration of the latent period	Time^{-1}
τ_1	Per capita recovery rate for persons from the exposed state to the recovered state. $1/\tau_1$ is the average duration of the asymptomatic period	Time^{-1}
τ_2	Per capita recovery rate for infectious persons from the quarantined state to the recovered state. $1/\tau_2$ is the average duration of the infectious period for quarantined individuals	Time^{-1}
τ_3	Per capita recovery rate for infectious persons from the non-quarantined state to the recovered state. $1/\tau_3$ is the average duration of the infectious period for non-quarantined individuals	Time^{-1}
ϕ_1	Modification rate parameter for progression to quarantine	$\mathrm{Humans} \times \mathrm{Time}^{-1}$
ϕ_2	Modification rate parameter for evasion of quarantine	$\mathrm{Humans} \times \mathrm{Time}^{-1}$
μ	Death removal (and emigration) rate	Time^{-1}
ψ	Per capita burial rate	$\mathrm{Humans} \times \mathrm{Time}^{-1}$
ξ	Rate of public enlightenment	$\mathrm{Humans} \times \mathrm{Time}^{-1}$
H	Enhanced personal hygiene due to public	$\mathrm{Humans} \times \mathrm{Time}^{-1}$

	enlightenment	
σ	Surveillance coverage rate	$\text{Humans} \times \text{Time}^{-1}$
Q	Number of quarantined individuals	$\text{Humans} \times \text{Time}^{-1}$
θ	Availability of isolation centres	$\text{Humans} \times \text{Time}^{-1}$
E	Effective contact rate for EVD of transmission	Dimensionless
β_1	Probability of transmission of infection from quarantined infectious persons to a susceptible person, given that a contact has actually occurred	Dimensionless
β_2	Probability of transmission of infection from non-quarantined infectious persons to a susceptible person , given that a contact has actually occurred	Dimensionless
β_3	Probability of transmission of infection from an infectious person undergoing treatment to a susceptible person, given that a contact has actually occurred	Dimensionless
β_4	Probability of transmission of infection from an infectious corpse to a susceptible person, given that a contact has actually occurred	Dimensionless

The state variables (Table 1) and parameters (Table 2) for the EVD model (Figure 1) satisfy the equations in (1). Further, since the model (1) monitors human populations and corpses recorded, all variables and parameters of the model are strictly positive except the disease induced death rate, d, which is nonnegative. Consider the biologically-feasible region

$$\Omega = \left\{ \left(S, E, I_Q, I_N, R, C_N, C_I \right) \in \Re_+^7 : H \le \Pi/\mu, C \le \Pi/\psi \right\}$$

To ensure that all solutions in Ω remain in Ω for all time (that is, to guarantee the positive invariance of Ω), we recall from (2) and note the following from rates of change of both the total human population and the record of all corpses that:

1. Following [3], whenever $H > \Pi/\mu$, then $dH/dt < 0$. However, following that $\Pi - \mu H$ is a bound of dH/dt, then by recalling the implication of the comparison theorem [1] as used in [3], it can readily be shown that $H(t) \le \Pi/\mu + \left[H(0) - \Pi/\mu \right] e^{-\mu t}$. Obviously, $H(t) \le \Pi/\mu$ if $H(0) \le \Pi/\mu$. Hence we conclude that every solution of the first five equations of the model (1) with initial conditions in Ω remains there for all $t > 0$ (that is the ω-limit sets of the first five equations of (1) are contained in Ω). Thus Ω is positively-invariant and attracting.
2. *Finally, we claim that the phenomenon captured by the last two equations of the system of equation (1) has a unique solution*

that exists and remains in Ω *for all time* $t \geq 0$.

Proof.

We note that the right hand sides of the two equations are continuous with continuous partial derivatives in Ω, implying that they have unique solutions. Next, to show the forward-invariance of Ω, we observe from the equations that if $C_{\mathrm{N}} = 0$, then obviously $dC_{\mathrm{N}}/dt \geq 0$; similarly, if $C_{\mathrm{I}} = 0,$ then it follows that $dC_{\mathrm{I}}/dt \geq 0.$ It is also true that if $H = I_{\mathrm{EVD}} = 0,$ then we have that $dC_{\mathrm{N}}/dt + dC_{\mathrm{I}}/dt = dC/dt < 0.$ Finally, if $C = 0,$ then $dC/dt \geq 0.$ Thus none of the orbits can leave Ω, and a unique solution exists for all time.

We have thus shown that Ω is positively-invariant and attracting. Therefore it is sufficient to consider the dynamics of the flow generated by the system (1) in Ω. Thus the model can be considered as being epidemiologically and mathematically well-posed [3].

3. Disease dynamics and reproductive number

The fundamental challenge concerning any invading infection is the possibility of its spread in a host population. The *basic reproduction number*, R_{E}, which epidemiologically measures the spreading potentiality of an invading disease in a given host population, is an insightful mathematical concept with far reaching significance in epidemic theory [11]. The quantity basically represents the average number of secondary infections most likely to be generated within the infective lifetime of an introduced infectious individual in a previously susceptible population. Such an individual will marginally produce less than a unit infective case when $R_{\mathrm{E}} < 1,$ and more than one if $R_{\mathrm{E}} > 1$ in its entire infectious lifetime in the population.

The next generation operator approach by [12] and highlighted in [2], has been extensively used to obtain the linear stability of Ω and will equally be utilised presently to obtain that for the system (1). Following the notion in [2], the nonzero matrix, *F*, that represents all new infection terms,and M-matrix, *V* representing all worsening terms, are, respectively given by

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mathrm{E}\beta_1(1-h\xi)(1-q\sigma\theta) & \mathrm{E}\beta_2(1-h\xi)(1-q\sigma\theta) & \mathrm{E}\beta_3(1-h\xi) & 0 & \mathrm{E}\beta_4(1-h\xi) \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V=\begin{pmatrix} \mu & 0 & 0 & 0 & -\eta(1-\rho) & 0 & 0 \\ 0 & K_1 & 0 & 0 & -\eta\rho & 0 & 0 \\ 0 & -\alpha\phi_1 & K_2 & 0 & 0 & 0 & 0 \\ 0 & -\alpha\phi_2 & 0 & K_3 & 0 & 0 & 0 \\ 0 & -\tau_1 & -\tau_2 & -\tau_3 & K_4 & 0 & 0 \\ -\mu & -\mu & -(\mu+\delta) & -(\mu+\delta) & -\mu & \psi & 0 \\ 0 & 0 & -\delta & -\delta & 0 & 0 & \psi \end{pmatrix}$$

The EDV reproduction number, denoted by R_{E}, is given by $R_{\mathrm{E}}=\rho\left(FV^{-1}\right)$ where ρ denotes the spectral radius (dominant eigenvalue) is presently obtained as

$$R_{\mathrm{E}}=\mathrm{E}(1-h\xi)\frac{\left\{\begin{array}{l}\psi\beta_3\left[\tau_1K_2K_3+\alpha\left(\tau_3\phi_2K_2+\tau_2\phi_1K_3\right)\right]+\\ \alpha K_4\left[\delta\beta_4\left(\phi_2K_2+\phi_1K_3\right)+\psi(1-q\sigma\theta)\left(\phi_1\beta_2K_3+\phi_2\beta_2K_2\right)\right]\end{array}\right\}}{\psi\left\{K_1K_2K_3K_4-\eta\rho\left[\tau_1K_2K_3+\alpha\left(\tau_3\phi_2K_2+\tau_2\phi_1K_3\right)\right]\right\}} \quad (5)$$

3.1. Existence of disease-free equilibrium (DFE) point

DFE points are steady-state solutions when the disease is considered to be absolutely absent in the population. In [13], the disease related classes comprise of all compartments that are either exposed, infectious or recovered. Here, in addition, we include the class of disease induced death, C_{I}, in the list of diseased classes. To this end therefore, the diseased classes here are $E, I_{\mathrm{Q}}, I_{\mathrm{N}}, R$ and C_{I}. Following [13], our positive orthant in $\Re^7$ by $\Re_+^7$, and the boundary of $\Re_+^7$ by $\partial\Re_+^7$. The positive equilibrium of the human and corpse population values, in the absence of Ebola, for (1) are respectively.

$$H^*=\frac{\Pi}{\mu} \text{ and } C^*=\frac{\Pi}{\psi}. \quad (7)$$

3.2. Local stability of disease free equilibrium (DFE)

In the absence of infection, the model (1) has a constant solution, Ω_0, also referred to (DFE) which, by noting that at the DFE $S^0\approx H^0$ and $C_{\mathrm{N}}^0\approx C^0$, is defined by

$$\Omega_0=\left(\frac{\Pi}{\mu},0,0,0,0,\frac{\Pi}{\psi},0\right). \quad (6)$$

The preceding results follow from [2]

Lemma 3.1. the DFE of the EVD model (1), given by (6), is locally asymptotically stable (LAS) if $R_{\mathrm{E}}<1$ and otherwise if $R_{\mathrm{E}}>1$.

Proof: We employ the Jacobian stability technique of determining the local stability of a system such as (1).The Jacobian matrix of the system (1) at the disease free equilibrium, Ω_0, is given by

$$J(\Omega_0)=\begin{pmatrix} -\mu & 0 & -b_1b_2\beta_1\mathrm{E} & -b_1b_2\beta_2\mathrm{E} & \eta(1-\rho)+b_1\beta_3\mathrm{E} & 0 & -b_1\beta_4\mathrm{E} \\ 0 & -K_1 & b_1b_2\beta_1\mathrm{E} & b_1b_2\beta_2\mathrm{E} & \eta\rho+b_1\beta_3\mathrm{E} & 0 & b_1\beta_4\mathrm{E} \\ 0 & \alpha\phi_1 & -K_2 & 0 & 0 & 0 & 0 \\ 0 & \alpha\phi_2 & 0 & -K_3 & 0 & 0 & 0 \\ 0 & \tau_1 & \tau_2 & \tau_3 & -K_4 & 0 & 0 \\ \mu & \mu & \mu+\delta & \mu+\delta & \mu & -\psi & 0 \\ 0 & 0 & \delta & \delta & 0 & 0 & -\psi \end{pmatrix}$$

where $b_1=1-h\xi,\ b_2=1-q\sigma\theta.$

Thus the eigenvalues of the row transformed Jacobian matrix of system (1) are given by

$$\lambda_1 = -K_1,\ \lambda_2 = -\frac{K_1K_2 - \alpha b_1 b_2 \beta_1 \mathrm{E}}{K_1},$$

$$\lambda_3 = -\frac{K_1K_2K_3 - \alpha b_1 b_2 \mathrm{E}\left(\varphi_1\beta_1K_3 + \varphi_2\beta_2K_2\right)}{K_1K_2 - \alpha\varphi_1 b_1 b_2 \beta_1 \mathrm{E}},$$

$$\lambda_4 = -\psi,$$

$$\lambda_6 = -\mu,\ \lambda_6 = -\frac{K_1K_2K_3K_4 - \left\{\begin{array}{l} b_1\beta_3\mathrm{E}\left[\tau_1K_2K_3 + \alpha\left(\tau_3\varphi_2K_2 + \tau_2\varphi_1K_3\right)\right] + \\ \eta\rho K_2\left(\alpha\varphi_2\tau_3 + \tau_1K_3\right) + \\ \alpha\left[\eta\rho\varphi_1\tau_2K_3 + b_1b_2K_4\mathrm{E}\left(\varphi_1\beta_1K_3 + \varphi_2\beta_2K_2\right)\right] \end{array}\right\}}{K_1K_2K_3 - \alpha b_1 b_2 \mathrm{E}\left(\varphi_1\beta_1K_3 + \varphi_2\beta_2K_2\right)},$$

$$\lambda_7 = -\frac{\psi\left\{K_1K_2K_3K_4 - \eta\rho\left[\tau_1K_2K_3 + \alpha\left(\tau_3\varphi_2K_2 + \tau_2\varphi_1K_3\right)\right]\right\} - b_1\mathrm{E}\left\{\begin{array}{l} \psi\beta_3\left[\tau_1K_2K_3 + \alpha\left(\tau_3\varphi_2K_2 + \tau_2\varphi_1K_3\right)\right] + \\ \alpha K_4\left[\begin{array}{l} \delta\beta_4\left(\varphi_2K_2 + \varphi_1K_3\right) + \\ \psi b_2\left(\varphi_1\beta_1K_3 + \varphi_2\beta_2K_2\right) \end{array}\right] \end{array}\right\}}{K_1K_2K_3K_4 - \left\{\begin{array}{l} \alpha b_1b_2K_4\mathrm{E}\left(\varphi_1\beta_1K_3 + \varphi_2\beta_2K_2\right) + \\ \left(\eta\rho + b_1\beta_3\mathrm{E}\right)\left[\tau_1K_2K_3 + \alpha\left(\tau_3\varphi_2K_2 + \tau_2\varphi_1K_3\right)\right] \end{array}\right\}}$$

It can readily be verified that the first six eigenvalues are negative. The negativity of λ_7 follows from the positivity of numerator from which it can easily be verified that

$$b_1\mathrm{E}\frac{\left\{\psi b_3\left[\tau_1K_2K_3 + \alpha\left(t_2f_1K_3 + t_3f_2K_2\right)\right] + \alpha K_4\left(\psi b_2b_2 + \alpha b_4\right)\left(f_2K_2 + f_1K_3\right)\right\}}{\psi\left\{K_1K_2K_3K_4 - \eta\rho\left[K_2K_3t_1 + \alpha\left(t_3f_2K_2 + t_2f_1K_3\right)\right]\right\}} < 1$$

,

or more precisely that $R_{\mathrm{E}} < 1$, and the result follows.

3.3. Analysis of R_{E}

Following [3], we consider the effect of the proposed three distinct treatment regimes, modelled by τ_1, τ_2 and τ_3, respectively scheduled for E, I_{Q} and I_{N}; on the dynamics of EVD in the community using the threshold quantity R_{E}. It follows from (5) that

$$\lim_{\tau_1\to\infty} R_{\mathrm{E}} = \frac{\mathrm{E}\beta_3\left(1 - h\xi\right)}{K_4 - \eta\rho} > 0 \qquad (7)$$

Similarly,

$$\lim_{\tau_2\to\infty} R_{\mathrm{E}} = \mathrm{E}\left(1 - h\xi\right)\frac{\left\{\psi\beta_3\left[\tau_1K_3 + \alpha\left(\tau_3\phi_2 + \phi_1K_3\right)\right] + \alpha\phi_2K_4\left[\delta\beta_4 + \psi\beta_2\left(1 - q\sigma\theta\right)\right]\right\}}{\psi\left\{K_1K_3K_4 - \eta\rho\left[\tau_1K_3 + \alpha\left(\tau_3\phi_2 + \phi_1K_3\right)\right]\right\}} > 0$$

(8)

and in the same vain

$$\lim_{\tau_3\to\infty} R_{\mathrm{E}} = \mathrm{E}\frac{\left(1 - h\xi\right)\left\{\begin{array}{l} \alpha\psi\phi_1\beta_1K_4\left[1 - h\xi\left(1 - q\sigma\theta\right)^2\right] + \\ \alpha\phi_1\delta\beta_4K_4 + \psi\beta_3\left[\tau_1K_3 + \alpha\left(\tau_2\phi_1 + \phi_2K_2\right)\right] \end{array}\right\}}{\psi\left\{K_1K_2K_4 - \eta\rho\left[\tau_1K_2 + \alpha\left(\tau_2\phi_1 + \phi_2K_2\right)\right]\right\}}$$

(9)

It therefore can be deduced from the foregoing that the deployment of an intensive EDV treatment schedule on individuals in the asymptomatic stage and those in the infectious stage that have either been (or have evaded) quarantine at respectively very high rates, $\tau_1 \to \infty, \tau_2 \to \infty$ and $\tau_3 \to \infty$ has the enormous capacity to effectively contain the disease, provided such an effort would translate into making the respective right hand sides of (7), (8) and (9) less than unity [3].

The partial derivatives of R_E with respect to the treatment parameters (τ_1, τ_2 and τ_3), are giving as

$$\frac{\partial R_E}{d\tau_1} = EK_2K_3 \frac{\begin{Bmatrix} \psi\beta_3(1-h\xi)\{K_1K_2K_3K_4 - \eta\rho[\tau_1K_2K_3 + \alpha(\phi_1\tau_2K_3 + \phi_2\tau_3K_2)]\} + \\ (\eta\rho - K_4)\begin{Bmatrix} \alpha\psi\phi_1\beta_1K_3K_4[1-h\xi(1-q\sigma\theta)^2] + \\ (1-h\xi)\begin{Bmatrix} \alpha K_4[\psi\phi_2\beta_2K_2(1-q\sigma\theta) + \delta\beta_4(\phi_2K_2 + \phi_1K_3)] + \\ \psi\beta_3[\tau_1K_2K_3 + \alpha(\phi_1\tau_2K_3 + \phi_2\tau_3K_2)] \end{Bmatrix} \end{Bmatrix} \end{Bmatrix}}{\psi\{K_1K_2K_3K_4 - \eta\rho[\tau_1K_2K_3 + \alpha(\phi_1\tau_2K_3 + \phi_2\tau_3K_2)]\}^2}, \quad (10)$$

$$\frac{\partial R_E}{d\tau_2} = E \frac{\begin{Bmatrix} (1-h\xi)\{K_1K_2K_3K_4 - \eta\rho[\tau_1K_2K_3 + \alpha(\phi_1\tau_2K_3 + \phi_2\tau_3K_2)]\} \times \\ \{\alpha\phi_2K_4[\delta\beta_4 + \psi\beta_2(1-q\sigma\theta)] + \psi\beta_3[\tau_1K_3 + \alpha(\tau_3\phi_2 + \phi_1K_3)]\} - \\ \{K_1K_3K_4 - \eta\rho[\tau_1K_3 + \alpha(\tau_3\phi_2 + \phi_1K_3)]\} \times \\ \begin{Bmatrix} \alpha\psi\phi_1\beta_1K_3K_4[1-h\xi(1-q\sigma\theta)^2] + \\ (1-h\xi)\begin{Bmatrix} \psi\beta_3[\tau_1K_2K_3 + \alpha(\tau_3\phi_2K_2 + \tau_2\phi_1K_3)] + \\ \alpha K_4[\psi\phi_2\beta_2K_2(1-q\sigma\theta) + \delta\beta_4(\phi_2K_2 + \phi_1K_3)] \end{Bmatrix} \end{Bmatrix} \end{Bmatrix}}{\psi\{K_1K_2K_3K_4 - \eta\rho[\tau_1K_2K_3 + \alpha(\phi_1\tau_2K_3 + \phi_2\tau_3K_2)]\}^2}, \quad (11)$$

and

$$\frac{\partial R_E}{d\tau_3} = E \frac{\begin{Bmatrix} \{K_1K_2K_3K_4 - \eta\rho[\tau_1K_2K_3 + \alpha(\varphi_1\tau_2K_3 + \varphi_2\tau_3K_2)]\} \times \\ \begin{Bmatrix} \alpha\psi\varphi_1\beta_1K_4[1-h\xi(1-q\sigma\theta)^2] + \\ (1-h\xi)[\alpha\varphi_1\delta\beta_4K_4 + \psi\beta_3[\tau_1K_2 + \alpha(\tau_2\varphi_1 + \varphi_2K_2)]] \end{Bmatrix} - \\ \{K_1K_2K_4 - \eta\rho[\tau_1K_2 + \alpha(\tau_2\varphi_1 + \varphi_2K_2)]\} \times \\ \begin{Bmatrix} \alpha\psi\varphi_1\beta_1K_3K_4[1-h\xi(1-q\sigma\theta)^2] + \\ (1-h\xi)\begin{Bmatrix} \psi\beta_3[\tau_1K_2K_3 + \alpha(\tau_3\varphi_2K_2 + \tau_2\varphi_1K_3)] + \\ \alpha K_4[\psi\varphi_2\beta_2K_2(1-q\sigma\theta) + \delta\beta_4(\varphi_2K_2K_4 + \varphi_1K_3)] \end{Bmatrix} \end{Bmatrix} \end{Bmatrix}}{\psi\{K_1K_2K_3K_4 - \eta\rho[\tau_1K_2K_3 + \alpha(\varphi_1\tau_2K_3 + \varphi_2\tau_3K_2)]\}^2}, \quad (12)$$

In an assumptive instance situation where only individuals in the asymptomatic stage receive any form of treatment, that is for $\tau_1 \neq 0$ while $\tau_2 = \tau_3 = 0$, then it will follow from (10) that $\frac{\partial R_E}{\partial \tau_1} < 0$ provided

$$\beta_3 < \Delta_1 = \frac{\alpha K_4(\mu+\delta)(K_4 - \eta\rho)\begin{Bmatrix} \psi\varphi_1\beta_1[1-h\xi(1-q\sigma\theta)^2] + \\ (1-h\xi)[\varphi\delta\beta_4 + \psi\varphi_2\beta_2(1-q\sigma\theta)] \end{Bmatrix}}{\psi(1-h\xi)[(K_1K_2 - \eta\rho\tau_1) - \tau_1(\mu+\delta)^2(K_4 - \eta\rho)]}, \quad (13)$$

Theorem 3.2. We thus infer from the foregoing that the effectiveness of the deployment of all treatment strategies in the asymptomatic stage only would create any desired positive impact provided $\beta_3 < \Delta_1$, so that implying from [3], no particular impact would be felt once $\phi_C = \Delta_1$ while the treatment would have detrimental consequences once $\beta_3 > \Delta_1$.

Similarly, if we consider the peculiar case where $\tau_1 = \tau_3 = 0$ but $\tau_2 \neq 0$, then we will expect from (11) that $\frac{\partial R_E}{\partial \tau_2} < 0$ only if

$$\beta_3 < \Delta_2 = \frac{\left(1-h\xi\right)\left[\left(\mu+\alpha\varphi\right)K_2K_4-\alpha\eta\rho\varphi_1\tau_2\right]\left\{\begin{matrix}\psi\varphi_1\beta_3\left(\mu+\delta\right)+\\ \varphi_2K_4\left[\delta\beta_4+\psi\beta_2\left(1-q\sigma\theta\right)\right]\end{matrix}\right\}+ \left[\alpha\eta\rho\varphi_1-K_4\left(\mu+\alpha\varphi\right)\right]\times \left\{\begin{matrix}\psi\varphi_1\beta_1K_4\left(\mu+\delta\right)\left[1-h\xi\left(1-q\sigma\theta\right)^2\right]+\\ \left(1-h\xi\right)\left[\psi\varphi_1\tau_2\beta_3\left(\mu+\delta\right)+K_4\left(\begin{matrix}\psi\varphi_2\beta_2K_2\left(1-q\sigma\theta\right)+\\ \delta\beta_4\left[\varphi_2K_2+\varphi_1\left(\mu+\delta\right)\right]\end{matrix}\right)\right]\end{matrix}\right\}}{\psi\left(\mu+\delta\right)\left[\left(\mu+\alpha\varphi\right)K_2K_4-\alpha\eta\rho\tau_2\varphi_1\right]^2},$$

(14)

Similarly it will be expected from (12) that $\frac{\partial R_E}{\partial \tau_3} < 0$ if

$$\beta_3 < \Delta_3 = \frac{\left[\left(\mu+\alpha\varphi\right)K_4-\alpha\eta\rho\varphi_2\right]\left\{\begin{matrix}\varphi_1K_3\left[\delta\beta_4\left(1-h\xi\right)+\psi\beta_1\left[1-h\xi\left(1-q\sigma\theta\right)^2\right]\right]+\\ \varphi_2\left(\mu+\delta\right)\left(1-h\xi\right)\left[\delta\beta_4+\psi\beta_2\left(1-q\sigma\theta\right)\right]\end{matrix}\right\} \varphi_1\left[\left(\mu+\alpha\varphi\right)K_3K_4-\alpha\eta\rho\tau_3\varphi_2\right]\left\{\begin{matrix}\delta\beta_4\left(1-h\xi\right)+\\ \psi\beta_1\left[1-h\xi\left(1-q\sigma\theta\right)^2\right]\end{matrix}\right\}}{\psi\varphi_2\left(\mu+\delta\right)^2\left(\mu+\alpha\varphi\right)},$$

(15)

Theorem 3.3. Following from (14) we conclude that the effectiveness of concentrating treatment in the quarantined infectious class would create any desired positive impact only if $\beta_3 < \Delta_2$, no particular impact if $\beta_3 = \Delta_2$ and a detrimental consequences once $\beta_3 > \Delta_2$. From (15) on the other hand we also argue that the effectiveness of concentrating treatment in the pool of infectious individuals who have evaded quarantine would be seen to create the desired positive impact only if $\beta_3 < \Delta_3$, no particular impact if $\beta_3 = \Delta_3$ and a detrimental consequences once $\beta_3 > \Delta_3$.

3.4. Global stability of DFE (Ω_0)

The evaluation of the global stability of a model at its equilibrium removes the restrictions on the initial conditions of the model's variables. This is because at the global asymptotic stability, solutions approach the equilibrium irrespective of the initial conditions. Among the methods of evaluating the global stability of models, we will follow the Castillo-Chavez global stability theorem. For emphasis, we reproduce the statement of the theorem

3.4.1. Castillo-Chavez *et al.*, (2002) theorem for global stability of disease equilibrium

Consider a model system defined by

$$\frac{dX_1}{dt} = F\left(X_1, X_2\right) \quad (16)$$

$$\frac{dX_2}{dt} = G\left(X_1, X_2\right), G\left(X_1, 0\right) = 0 \quad (17)$$

where $X_1 \in \Re^m$ denotes the number of uninfected individuals and $X_2 \in \Re^n$ denotes the number of infected individuals; so that $\Omega_0 = \left(X_1^*, 0\right)$ denotes the disease free equilibrium of the system. Further assume the following conditions:

1. For $\frac{dX_1}{dt} = F(X_1, 0), X_1^*$ is globally asymptotically stable and
2. $G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2), \hat{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$, where the Jacobian

$$A = \frac{dG}{dX_1}(X_1^*, 0) \tag{18}$$

is an M-matrix and Ω is the region where the model is biologically sensible.

Then the DFE $\Omega_0 = (X_1^*, 0)$ is globally asymptotically stable provided $R_E < 1$, where R_E is the basic reproduction number.

Theorem 3.4: *The disease free equilibrium, Ω_0, of the system (1) is globally asymptotically stable (GAS) in Ω if $R_E < 1$.*

Proof: From the foregoing, we rewrite model (1) in the forms (16) and (17) with $X_1 = (S^0, R^0, C_N^0)$ and $X_2 = (E^0, I_Q^0, I_N^0, C_I^0)$ where the component $X_1 \in \Re^3$ denote the uninfected EVD population while the component $X_2 \in \Re^4$ the EVD population. The disease-free equilibrium is $\Omega_0 = (X_1^*, 0)$ where

$$X_1^0 = \left(\frac{\Pi}{\mu}, 0, \frac{\Pi}{\psi}\right)$$

Thus for the global asymptotic stability of X_1^*, it follows from condition (1) that the solutions of the linear differential equations

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{pmatrix} \Pi - \mu S^0 + \eta(1-\rho)R^0 \\ -K_4 R^0 \\ \mu(S^0 + R^0) - \psi C_N^0 \end{pmatrix} \tag{19}$$

are of the form

$$S^0(t) = \frac{\Pi + \eta(1-\rho)R^0}{\mu} - \frac{\Pi + \eta(1-\rho)R^0}{\mu} e^{-\mu t} + S^0(0)e^{-\mu t}, \tag{20}$$

$$R^0(t) = R^0(0)e^{-K_4 t}, \tag{21}$$

$$C_N^0(t) = \frac{\mu(S^0 + R^0)}{\psi} - \frac{\mu(S^0 + R^0)}{\psi} e^{-\psi t} + C_N^0(0)e^{-\psi t}. \tag{22}$$

It follows clearly from (19) that $S^0(t) + R^0(t) \to H^0$ and $C_N^0(t) \to C^0(t)$ as $t \to \infty$ and this is independent of the respective values of $S^0(0)$, $R^0(0)$, and $C_N^0(0)$.. Thus X_1^0 is globally asymptotically stable.

Next, we evaluate the second condition of the stability theorem as follows: the matrix

$$A=\begin{pmatrix} -K_1 & 0 & 0 & 0 \\ \alpha\phi r_1 & -K_2 & 0 & 0 \\ \alpha\phi_2 & 0 & -K_3 & 0 \\ 0 & \delta & \delta & -\psi \end{pmatrix} \quad (23)$$

is obviously an M-matrix. By definition,

$$G=\begin{pmatrix} \mu S^0+\eta\rho R^0-K_1E^0 \\ \alpha\phi_1E^0-K_2I_{\mathrm{Q}}^0 \\ \alpha\phi_2E^0-K_3I_{\mathrm{N}}^0 \\ \delta\left(I_{\mathrm{Q}}^0+I_{\mathrm{N}}^0\right)-\psi C_{\mathrm{I}}^0 \end{pmatrix} \quad (24)$$

then

$$\hat{G}\left(X_1,X_2\right)=AX_2-G\left(X_1,X_2\right)=\begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (25)$$

From the foregoing, we therefore conclude that every solution to the equations of model (1), with initial conditions in Ω approaches Ω_0 as $t\to\infty$, provided $R_{\mathrm{E}}<1$, and the prove follows

3.6. Existence of endemic equilibrium state $\left(\Omega^*\right)$

Endemic equilibrium points are steady-state solutions where the disease persists in the population. Here, we consider the implication of the persistence EVD on the population. The ensuing solution for which EVD is endemic in the population is given by

$$\Omega^*=\left\{\left(S^*,E^*,I_{\mathrm{Q}}^*,I_{\mathrm{N}}^*,R^*,C_{\mathrm{N}}^*,C_{\mathrm{I}}^*\right)\middle|\begin{matrix} S>0,E>0,I_{\mathrm{Q}}>0, \\ I_{\mathrm{N}}>0,R>0,C_{\mathrm{N}}>0,C_{\mathrm{I}}>0 \end{matrix}\right\} \quad (26)$$

with which we will attempt solving the equations in (1) with respect to the force of infection, λ^*, at steady state given as

$$\lambda^*=\mathrm{E}\left(1-h\xi\right)\left\{\frac{\beta_4C_{\mathrm{I}}^*H^*+C^*\left[\begin{matrix}\beta_3R^*+\\ \left(1-q\sigma\theta\right)\left(\beta_1I_{\mathrm{Q}}^*+\beta_2I_{\mathrm{N}}^*\right)\end{matrix}\right]}{C^*H^*}\right\} \quad (27)$$

Simplify each equation (1) when its corresponding derivative is taken as zero and with $\lambda=\lambda^*$, we obtain the following respective expressions:

$$S^*=\frac{a_1\Pi}{a_1\mu+a_2\lambda^*},\ E^*=\frac{\lambda^*\Pi K_2K_3K_4}{a_1\mu+a_2\lambda^*},\ I_{\mathrm{Q}}^*=\frac{\alpha\varphi_1\lambda^*\Pi K_3K_4}{a_1\mu+a_2\lambda^*},$$
$$I_{\mathrm{N}}^*=\frac{\alpha\varphi_2\lambda^*\Pi K_2K_4}{a_1\mu+a_2\lambda^*},$$
$$R^*=\frac{a_{01}\lambda^*\Pi}{a_1\mu+a_2\lambda^*},\ C_{\mathrm{N}}^*=\frac{\mu\Pi\left(a_1+a_3\lambda^*\right)}{\psi\left(a_1\mu+a_2\lambda^*\right)},$$
$$C_{\mathrm{I}}^*=\frac{a_4\delta\lambda^*}{\psi\left(a_1\mu+a_2\lambda^*\right)}. \quad (28)$$

Thus simplifying (27) at (28), gives

$$a_3\left(a_4\delta+\mu a_3\Pi\right)\lambda^{*2}+a_8\lambda^*+a_1a_7=0 \text{ or}$$

$$\lambda^*=\frac{-a_8+\sqrt{a_8^2-4a_1a_3a_7\left(a_4\delta+\mu a_3\Pi\right)}}{2a_3\left(a_4\delta+\mu a_3\Pi\right)} \quad (29)$$

where

$$a_0 = \tau_1 K_2 K_3 + \alpha\left(\varphi_2\tau_3 + \varphi_1\tau_2\right),\ a_1 = K_1K_2K_3K_4 - a_0\eta\rho,$$
$$a_2 = a_1 - a_{01}\eta\left(1-\rho\right),$$
$$a_3 = a_0 + K_4\left[K_2K_3 + \alpha\left(\varphi_1K_3 + \varphi_2K_2\right)\right],$$
$$a_4 = \alpha\Pi K_4\left(\varphi_1K_3 + \varphi_2K_2\right),$$
$$a_5 = a_0\beta_3 + \alpha K_4\left(1-q\sigma\theta\right)\left(\varphi_1\beta_1K_3 + \varphi_2\beta_2K_2\right),$$
$$a_6 = a_3a_4\delta\beta_4 + a_5\left(a_4\delta + \mu a_3\Pi\right),$$
$$a_7 = \mu a_1\Pi - \mathrm{E}\left(1-h\xi\right)\left(\mu a_5\Pi + a_4\delta\beta_4\right),$$
$$a_8 = a_1\left(a_4\delta + 2\mu a_3\Pi\right) - a_6\mathrm{E}\left(1-h\xi\right)$$

Lemma 1: *The EVD model (1) has a unique endemic equilibrium state whenever the effective basic reproduction number,* R_{E}, *is such that* $R_{\mathrm{E}} > 1$, *and no endemic equilibrium otherwise.*

3.6.1. Local stability of the endemic equilibrium

Following the reasons advanced in [3], we also employ the procedure of the centre manifold theory as described in [14] to establish the local asymptotic stability of the endemic equilibrium. To achieve this, we make the following change of variables: let $x_1 = S, x_2 = E, x_3 = I_{\mathrm{Q}}, x_4 = I_{\mathrm{N}}, x_5 = R, x_6 = C_{\mathrm{N}}$ and $x_7 = C_{\mathrm{I}}$ so that $H = x_1 + x_2 + x_3 + x_4 + x_5$, $I_{\mathrm{EVD}} = x_3 + x_4$ and $C = x_6 + x_7$. In addition, by using the notation $X = \left(x_1, x_2, x_3, x_4, x_5, x_6, x_7\right)^T$, the EVD model (1) can be expressed as $\frac{dX}{dt} = \left(f_1, f_2, f_3, f_4, f_5, f_6, f_7\right)^T$ where

$$\frac{dx_1}{dt} = f_1 = \Pi + \eta\left(1-\rho\right)x_5 - \left(\mu+\lambda\right)x_1,$$
$$\frac{dx_2}{dt} = f_2 = \lambda x_1 + \eta\rho x_5 - K_1x_2,$$
$$\frac{dx_3}{dt} = f_3 = \alpha\phi_1x_2 - K_2x_3,$$
$$\frac{dx_4}{dt} = f_4 = \alpha\phi_2x_2 - K_3x_4,$$
$$\frac{dx_5}{dt} = f_5 = \tau_1x_2 + \tau_2x_3 + \tau_3x_4 - K_4x_5,$$
$$\frac{dx_6}{dt} = f_6 = \mu\left(x_1 + x_2 + x_3 + x_4 + x_5\right) - \psi x_6,$$
$$\frac{dx_7}{dt} = f_7 = \delta\left(x_3 + x_4\right) - \psi x_7,$$

(30)

where

$$\lambda = \mathrm{E}\left(1-h\xi\right)\frac{\beta_4x_7\left(x_1+x_2+x_3+x_4+x_5\right) + \left(x_6+x_7\right)\left[\left(1-q\sigma\theta\right)\left(x_3\beta_1 + x_4\beta_2\right) + x_5\beta_3\right]}{\left(x_6+x_7\right)\left(x_1+x_2+x_3+x_4+x_5\right)}$$

The Jacobian of system (30) at the DFE, Ω_0, is given by

$$J\left(\Omega_0\right) = \begin{pmatrix} -\mu & 0 & -\frac{b_1b_2\beta_1\mathrm{ES}^0}{H^0} & -\frac{b_1b_2\beta_2\mathrm{ES}^0}{H^0} & \eta\left(1-\rho\right)+\frac{b_1\beta_3\mathrm{ES}^0}{H^0} & 0 & -\frac{b_1\beta_4\mathrm{ES}^0}{C^0} \\ 0 & -K_1 & \frac{b_1b_2\beta_1\mathrm{ES}^0}{H^0} & \frac{b_1b_2\beta_2\mathrm{ES}^0}{H^0} & \eta\rho+\frac{b_1\beta_3\mathrm{ES}^0}{H^0} & 0 & \frac{b_1\beta_4\mathrm{ES}^0}{C^0} \\ 0 & \alpha\phi_1 & -K_2 & 0 & 0 & 0 & 0 \\ 0 & \alpha\phi_2 & 0 & -K_3 & 0 & 0 & 0 \\ 0 & \tau_1 & \tau_2 & \tau_3 & -K_4 & 0 & 0 \\ \mu & \mu & \mu+\delta & \mu+\delta & \mu & -\psi & 0 \\ 0 & 0 & \delta & \delta & 0 & 0 & -\psi \end{pmatrix}$$

$$J(\Omega_0)=\begin{pmatrix} -\mu & 0 & -b_1b_2\beta_1\mathrm{E} & -b_1b_2\beta_2\mathrm{E} & \eta(1-\rho)+b_1\beta_3\mathrm{E} & 0 & -b_1\beta_4\mathrm{E} \\ 0 & -K_1 & b_1b_2\beta_1\mathrm{E} & b_1b_2\beta_2\mathrm{E} & \eta\rho+b_1\beta_3\mathrm{E} & 0 & b_1\beta_4\mathrm{E} \\ 0 & \alpha\phi_3 & -K_2 & 0 & 0 & 0 & 0 \\ 0 & \alpha\phi_1 & 0 & -K_3 & 0 & 0 & 0 \\ 0 & \tau_1 & \tau_2 & \tau_3 & -K_4 & 0 & 0 \\ \mu & \mu & \mu+\delta & \mu+\delta & \mu & -\psi & 0 \\ 0 & 0 & \delta & \delta & 0 & 0 & -\psi \end{pmatrix}$$

from which we have already shown that

$$R_{\mathrm{E}}=\mathrm{E}(1-h\xi)\frac{\left\{\begin{array}{l}\psi\beta_3\left[\tau_1K_2K_3+\alpha\left(\tau_3\varphi_2K_2+\tau_2\varphi_1K_3\right)\right]+\\ \alpha K_4\left[\begin{array}{l}\delta\beta_4\left(\varphi_2K_2+\varphi_1K_3\right)+\\ \psi\left(1-q\sigma\theta\right)\left(\varphi_1\beta_2K_3+\varphi_2\beta_2K_2\right)\end{array}\right]\end{array}\right\}}{\psi\left\{K_1K_2K_3K_4-\eta\rho\left[\tau_1K_2K_3+\alpha\left(\tau_3\varphi_2K_2+\tau_2\varphi_1K_3\right)\right]\right\}}$$

Now if we consider the case when $R_{\mathrm{E}}=1,$ and further consider $\mathrm{E}=\mathrm{E}^*$ as a bifurcation parameter in view of the inconveniency of using R_{E} as one, then at $R_{\mathrm{E}}=1,$ the value of E^* would be

$$\mathrm{E}^*=\frac{\psi\left\{K_1K_2K_3K_4-\eta\rho\left[\tau_1K_2K_3+\alpha\left(\tau_3\varphi_2K_2+\tau_2\varphi_1K_3\right)\right]\right\}}{(1-h\xi)\left\{\begin{array}{l}\psi\beta_3\left[\tau_1K_2K_3+\alpha\left(\tau_3\varphi_2K_2+\tau_2\varphi_1K_3\right)\right]+\\ \alpha K_4\left[\begin{array}{l}\delta\beta_4\left(\varphi_2K_2+\varphi_1K_3\right)+\\ \psi\left(1-q\sigma\theta\right)\left(\varphi_1\beta_2K_3+\varphi_2\beta_2K_2\right)\end{array}\right]\end{array}\right\}}\quad(32)$$

Eigenvectors of $J(\Omega_0)$ for E near E^*

Supposing, respectively, that V and W are the corresponding left and right eigenvectors associated with the zero eigenvalues of the of the Jacobian (31) at $\mathsf{E}=\mathsf{E}^*$ denoted by J_{E^*}, chosen in such a way that $VJ(\Psi_0)=0$ and $WJ(\Psi_0)=0,$ with $VW=1,$ where $V=(v_1,v_2,v_3,v_4,v_5,v_6,v_7),$ and $W=(w_1,w_2,w_3,w_4,w_5,w_6,w_7)^T$. Then

then it follows that the left eigenvector, $v=[v_1,v_2,v_3,v_4,v_5,v_6,v_7],$ of J_{E^*} is

$$v_1=v_6=0,\ v_3=\frac{\tau_2\left(\eta\rho+b_1\beta_3\mathrm{E}\right)+b_1K_4\mathrm{E}\left(\psi b_2\beta_1+\delta\beta_4\right)}{\psi K_2K_4}v_2,$$

$$v_5=\frac{\eta\rho+b_1\beta_3\mathrm{E}}{K_4}v_2,$$

$$v_4=\frac{\psi\tau_3\left(\eta\rho+b_1\beta_3\mathrm{E}\right)+b_1K_4\mathrm{E}\left(\psi b_2\beta_1+\delta\beta_4\right)}{\psi K_3K_4}v_2,v_7=\frac{b_1\beta_4\mathrm{E}}{\psi}v_2.$$

We note that once $v_2>0,$ then so will v_3,v_4,v_5 and v_7.

Similarly, the right eigenvector, $w=[w_1,w_2,w_3,w_4,w_5,w_6,w_7]^T,$

$$w_1=\frac{\mu a_1\left(\eta(1-\rho)-b_1\beta_3\mathrm{E}\right)-\alpha a_2b_1K_4\mathrm{E}}{\mu K_2K_3K_4}w_2,w_3=\frac{\alpha\varphi_1}{K_2}w_2,$$

$$w_4=\frac{\alpha\varphi_2}{K_3},\ w_5=\frac{a_1}{K_2K_3K_4}w_2,$$

$$w_7=\frac{\alpha\varphi\delta}{\psi K_3}w_2,w_6=\frac{\mu a_1+a_3K_4}{\psi K_2K_3K_4}w_2.$$

Where

$$a_1=\tau_1K_2K_3+\alpha\left(\tau_2\varphi_1K_3+\tau_3\varphi_2K_2\right),$$
$$a_2=\varphi\delta\beta_4K_2+\mu b_2\left(\varphi_1\beta_1K_3+\varphi_2\beta_2K_2\right),$$
$$a_3=\mu K_2K_3+\alpha\left(\mu+\delta\right)\left(\varphi_1K_3+\varphi_2K_2\right)$$

Thus for $w_2 > 0$, it follows from the above that $w_3 > 0,\ w_4 > 0,\ w_5 > 0,\ w_6 > 0$ and $w_7 > 0$. Further it can be verified that $w_1 < 0$.

Following [3], we reproduce [14] for emphasis

Theorem 3.5: *Consider the following general system of ordinary differential equations with a parameter* ϕ

$$\frac{dx}{dt} = f(x,\phi),\ f : \Re^n \times \Re \to \Re \quad \text{and}$$

$$f \in C^2(\Re^n \times \Re), \qquad (33)$$

where0 is an equilibrium point of the system (that is $f(0,\phi) \equiv 0$ *for all* ϕ*) and assume*

A1: $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ *is the linearization matrix of the system (33) around the equilibrium0 with* ϕ *evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;*

A2: *Matrix A has a right eigenvector Wand a left eigenvector V(each corresponding to the zero eigenvalue).*

Let f_k *be the kth component of f and*

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$a = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0).$$

The local dynamics of the system around 0 is totally determined by the signs of a and *b as follows:*

i. $a > 0, b > 0$. *When* $\phi < 0$ *with* $|\phi| << 1, 0$ *is locally asymptotically stable and there exists a positive unstable equilibrium; when* $0 < \phi << 1, 0$ *is unstable and there exists a negative, locally asymptotically stable equilibrium;*

ii. $a < 0, b < 0$. *When* $\phi < 0$ *with* $|\phi| << 1,\ 0$ *is unstable; when* $0 < \phi << 1, 0$ *is a locally asymptotically stable equilibrium, and there exists a positive unstable equilibrium;*

iii. $a > 0, b < 0$. *When* $\phi < 0$ *with* $|\phi| << 1,\ 0$ *is unstable, and there exists a locally asymptotically stable equilibrium stable negative equilibrium; when* $0 < \phi << 1, 0$ *is stable and there exists a positive unstable equilibrium;*

iv. $a < 0, b > 0$. $a < 0, b > 0$. *When* ϕ f *changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $a > 0$ *and* $b > 0$, *then backward bifurcation occurs at* $f = 0$.

Computations of *a* **and** *b:*

Presently, $n = 7$ and with $v_1 = v_6 = 0$, it follows from the above expressions that

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = -\frac{\mu E \beta_1 (1-h\xi)(1-q\sigma\theta)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_4} = -\frac{\mu E \beta_2 (1-h\xi)(1-q\sigma\theta)}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_5} = -\frac{\mu E \beta_3 (1-h\xi)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_7} = -\frac{\psi E \beta_4 (1-h\xi)}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_3^2} = -\frac{2\mu E \beta_1 (1-h\xi)(1-q\sigma\theta)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_4} = -\frac{\mu E (\beta_1+\beta_2)(1-h\xi)(1-q\sigma\theta)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_5} = -\frac{\mu E (1-h\xi)\left[\beta_3+\beta_1(1-q\sigma\theta)\right]}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = -\frac{\mu E \beta_2 (1-h\xi)(1-q\sigma\theta)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_5 \partial x_2} = -\frac{\mu E \beta_3 (1-h\xi)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_4 \partial x_3} = -\frac{\mu E (\beta_1+\beta_2)(1-h\xi)(1-q\sigma\theta)}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_4^2} = -\frac{2\mu E \beta_2 (1-h\xi)(1-q\sigma\theta)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_5^2} = -\frac{2\mu E \beta_3 (1-h\xi)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_4 \partial x_5} = -\frac{\mu E (1-h\xi)\left[\beta_3+\beta_1(1-q\sigma\theta)\right]}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_5 \partial x_3} = -\frac{\mu E (1-h\xi)\left[\beta_3+\beta_1(1-q\sigma\theta)\right]}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_5 \partial x_4} = -\frac{\mu E (1-h\xi)\left[\beta_3+\beta_1(1-q\sigma\theta)\right]}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_6 \partial x_7} = -\frac{\psi^2 E \beta_4 (1-h\xi)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_7^2} = -\frac{2\psi^2 E \beta_4 (1-h\xi)}{\Pi}.$$

From the foregoing, it thus follows that

$$a = \frac{2v_2 E(1-h\xi)}{\mu\Pi}\left\{\begin{array}{l}\psi w_7\beta_4\left[\mu w_1 - 2\psi(w_6+w_7)\right] - \\ \qquad \mu^2(w_2+w_3+w_4+w_5)\\ \left[w_5\beta_3 + (1-q\sigma)(w_3\beta_1+w_4\beta_2)\right]\end{array}\right\}$$

Thus $a < 0$ if

$$\frac{2w_7\psi^2\beta_4(\psi_6+\psi_7)+\mu^2(w_2+w_3+w_4+w_5)\left[\begin{array}{l}w_5\beta_3+\\ (1-q\sigma\theta)(w_3\beta_1+w_4\beta_2)\end{array}\right]}{\mu\psi w_1 w_7\beta_4} > 1$$

For the sign of *b*, it can be shown that the associated non-vanishing partial derivatives of F are

$$\frac{\partial^2 f_1}{\partial x_3 \partial E} = -\frac{\mu\beta_1(1-q\sigma\theta)(1-h\xi)}{\Pi}, \frac{\partial^2 f_1}{\partial x_4 \partial E} = -\frac{\mu\beta_2(1-q\sigma\theta)(1-h\xi)}{\Pi},$$

$$\frac{\partial^2 f_1}{\partial x_5 \partial E} = -\frac{\mu\beta_3(1-h\xi)}{\Pi}, \frac{\partial^2 f_1}{\partial x_7 \partial E} = -\frac{\mu\beta_4(1-h\xi)}{\psi}.$$

From which it follows that

$$b = \frac{v_2(1-h\xi)}{\psi}\left\{\psi(1-q\sigma\theta)(w_3\beta_1+w_4\beta_2)+w_5\beta_3+\mu w_7\right\} > 0$$

Thus, $a < 0$ and $b > 0$. Thus by (by Theorem 2, Item (iv)), we have established the following result (note that this result holds for RH > 1 but close to 1):

Theorem 3.6. The unique endemic equilibrium is LAS for R_E near 1.

In summary, the EVD model (9) has a globally-asymptotically stable DFE whenever $R_E \le 1$, and a unique endemic equilibrium point whenever $R_E > 1$. The unique endemic equilibrium point is LAS at least near $R_E = 1$.

Numerical simulation and discussion of results

For the purpose of model validation and to ensure the compliance of the model to reality, we perform

numerical simulations using the data values in table 3 below

Table 3: *Parameters values*

Parameter	Value	Reference
Π	9863	[7]
α	0.083	[7]
τ_1, τ_2, τ_3	Variable	
ϕ, ϕ_1, ϕ_2	1.201, 0.001, 1.2	
μ	0.00005479	[7]
ψ	0.15	
ξ	0.9	[7]
H	1.0	[7]
σ	0.75	[7]
Q	1.0	[7]
θ	0.65	[7]
δ	0.25	[7]
η	0.065	
ρ	0.07465	
E	0.56	
$\beta_1, \beta_2, \beta_3, \beta_4$	0.9, 0.85, 0.125, 0.25	

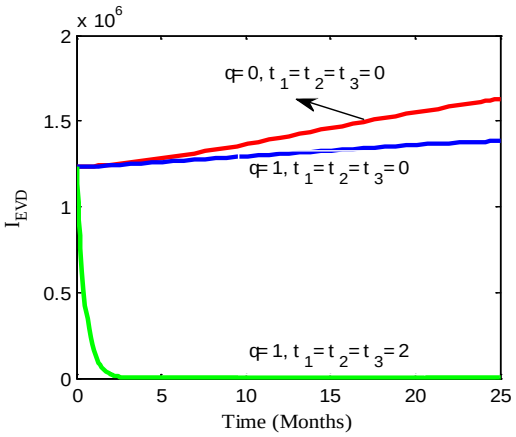

Figure 2a: A plot of cumulative new cases against time for varying levels of the availability of isolation centres and treatments

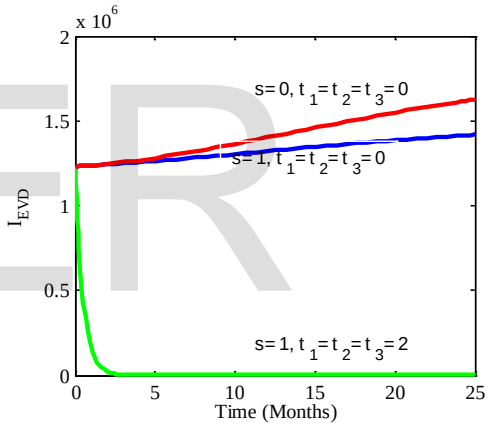

Figure 2b: A plot of cumulative new cases against time for varying surveillance coverage and treatment levels

The availability of isolation centres is seen to suggest extreme EVD transmission scenario. From figure 2a it can be observed the absence of both isolation centres and the requisite treatment strategies would emphatically result in an increasing escalation of the disease at a higher rate than when isolation centres are available but no requisite treatment schedules as

against the total containment prospects of the provision of efficient treatment strategies in the all the isolation centres across the community. A robust and workable disease surveillance and treatment strategy could lead to an impressive containment as shown on figure 2b; while an aggressive surveillance without an accompanying treatment would barely lead to any positive impact; further, the further projects an alarming escalation tendency of the disease in the event of the lack of both surveillance and requisite treatments.

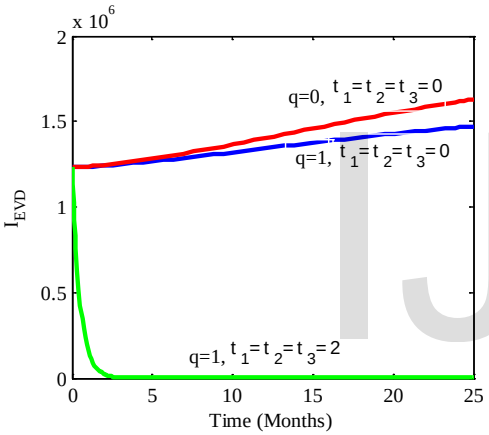

Figure 2c: A plot of cumulative new cases against time for varying percentage of persons quarantined and treatments levels

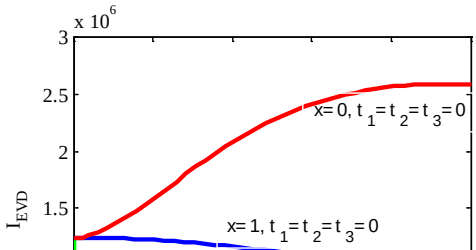

Figure 2d: A plot of cumulative new cases against time for varying rates of public enlightenment campaigns and treatments

Figure 2c on its part portrays the impact of quarantine and treatments on the community. The two extremes are tipped to create marginal impacts on the community while a robust quarantine strategy and implementation in the absence of requisite treatments is barely probable to create any appreciable impact. From figure 2d it readily be anticipated that with rigorous public enlightenment campaigns at maximum levels accompanied by a corresponding intensive treatment levels, the EVD challenge will be absolutely contained. On the other hand, failure to accompany the campaign with requisite treatment would result to a gradual fall of the severity of EVD in the community. This could be as a result of a high public awareness on the transmission and infectiousness of EVD leading to a healthy hygienic consciousness and observance. Additional consequence of the ensuing impact of these campaigns is captured in figure 2e. At the other extreme of the figure is the inherent threat of EVD

escalation in the absence of both public enlightenment campaign as well as requisite treatment.

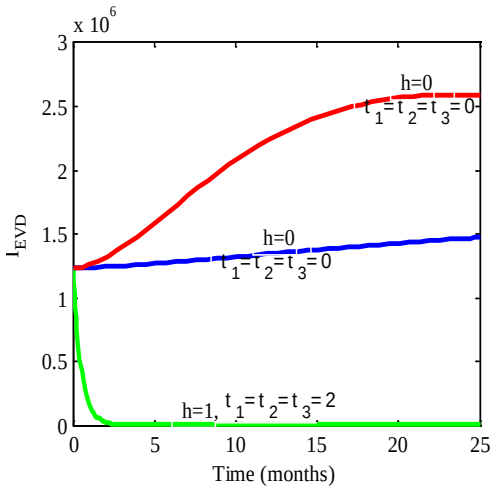

Figure 2e: A plot of cumulative new cases against time for varying levels of personal hygiene and treatments and treatment levels

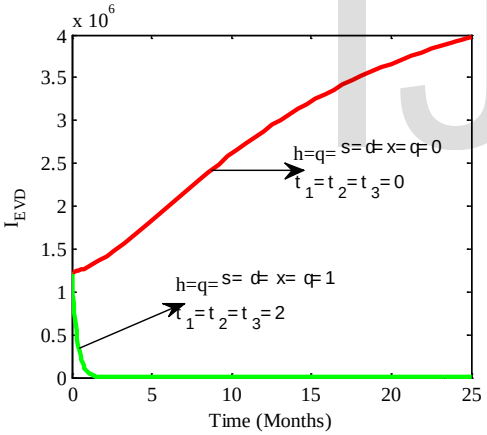

Figure 2f: A plot of cumulative new cases against time for extreme control parameter values and treatment levels

Figure 2e presents the dynamics of the disease in a community for varying levels of hygiene and treatments. It is understood that the disease can completely be contained at simultaneously maximum observable personal hygiene and a sustained marginal treatment programme. It is also noted that a maximal hygiene consciousness in an EVD endemic community has the potentials for substantial containment even in the absence of any form of treatment. The absence of both personal hygiene and treatment however is seen to have gross negative impact level on the community. Figure 2f has summarised the consequences of the extremities of the various control parameters on the dynamics of EVD on the community. We observe the outright escalating possibilities of the disease in the event of the no deployment of requisite control parameters against the potentialities of the maximal deployment control parameters to absolutely eradicate the disease from any challenged community. Finally, figure 2g singles out the direct consequence of disease-induced deaths on the dynamics of the transmission of EVD on any challenged community.

Conclusion

An Ebola outbreak in a human population can be catastrophic. Given the result obtained from the analysis of the model, an uncontrolled transmittable contact between the infected and the susceptible can increase the spread of the disease in the population and cause the disease outbreak to linger for a longer period of time. This implies that timely implementation of the control parameters would go a long way in controlling the spread of the disease in a population ravaged by the Ebola virus disease. Our work revealed that good public enlightenment,

aggressive quarantine system and effective treatment would ultimately reduce the transmittable contacts.

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- Din. Sunday Sarki. Department of Mathematics, School of Social Sciences, Federal College of Education, Pankshin, Plateau State, Nigeria. E-mail: dinsarki@gmail.com
- Chukwudi Okoye. Department of Mathematics, Faculty of Physical sciences, University of Nigeria, Nsukka, Enugu State, Nigeria. E-mail: chukwudi.okoye@unn.edu.ng
- Sunday Ifeanyi Onah. Department of Mathematics, Faculty of Physical sciences, University of Nigeria, Nsukka, Enugu State, Nigeria. E-mail: ifeanyi.onah.pg62687@unn.edu.ng
- Godwin Christopher Ezike Mbah. Department of Mathematics, Faculty of Physical sciences, University of Nigeria, Nsukka, Enugu State, Nigeria. E-mail: godwin.mbah@unn.edu.ng