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Chinwendu E. Madubueze
University of Agriculture, Nigeria

Anande R. Kimbir
University of Agriculture, Nigeria

Terhemen Aboiyar
University of Agriculture, Nigeria

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Global Stability of Ebola Virus Disease Model with Contact Tracing and Quarantine

[*1]Chinwendu E. Madubueze, [2]Anande R. Kimbir and [3]Terhemen Aboiyar

Department of Mathematics/Statistics/Computer Science
University of Agriculture
P.M.B. 2373
Makurdi, Nigeria
[*1]ce.madubueze@gmail.com;
[2] anandekimbir@gmail.com; [3] t_aboiyar@yahoo.co.uk

[*]Corresponding Author

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Abstract

This study considers a deterministic model of Ebola Virus Disease (EVD) incorporating contact tracing and quarantine as interventions. The model analyze the existence and stability of Disease-Free Equilibrium (DFE) and Endemic Equilibrium (EE) states. The local stability of EE is established using centre manifold theorem. The global stability of the two equilibrium states are obtained by constructing the Lyapunov function. Numerical simulations are carried out to examine the impact of contact tracing and quarantine measures on the transmission dynamics of EVD. The result indicates that EVD could be eliminated faster when contact tracing and quarantine measures are implemented together.

Keywords: Ebola virus disease; Contact tracing; Quarantine; Effective reproduction number; Stability analysis; Bifurcation analysis; Lyapunov functions

MSC 2010 No.: 92B05, 92D30, 93D20

1. Introduction

Ebola virus is one of the viruses that causes viral hemorrhagic fevers. It belongs to the family of non-segmented negative-sense single stranded ribonucleic acid (RNA) viruses called the

filoviruses. The natural reservoir for Ebola virus still remains unknown. However, it is speculated that arthropods, rodents and bats could be the hosts (Chowell and Nishiura, 2014; Sullivan et al., 2003). There are five identified subtypes of Ebola virus classified based on their locations of origin namely; Ebola Zaire, Ebola Sudan, Ebola Ivory Coast and Ebola Bundibugyo. All these classifications cause disease in humans except Ebola Reston which causes disease in non-human primates (CDC, 2009; Brooks et al., 2007). There are more than twenty-five epidemics of Ebola since the discovery of the virus in 1976. The 2014 epidemic in West Africa caused by Ebola Zarie was the most deadly because it recorded 28,608 suspected cases and 11,306 deaths (WHO, 2016).

The Ebola virus has an incubation period of usually 4 to 10 days but it can vary from 2 to 21 days. The virus is spread through close contact with the blood, secretions, organs or other bodily fluids of infected animals or an infected person found ill or dead in the population (WHO, 2014; Hauora, 2014). It can also spread through indirect contact with environmental objects contaminated with these fluids in hospital settings such as needles, syringes, beddings and clothing. People remain infectious for as long as their bodily fluids contain the virus.

The signs and symptoms of the Ebola virus disease are usually mistaken for other diseases such as malaria, typhoid fever, influenza and other bacterial infections. The prevention and control of Ebola virus in Africa pose many challenges because the identity and location of the natural reservoir of the virus is still unknown.

There is no licensed medication or vaccine for treatment of Ebola patients other than intensive supportive therapy. However, some drugs and vaccines such as ZMapp and rVSV-ZEBOV, yet to undergo complete clinical trials are used on compassionate grounds. Permanent immunity in patients who recovered from a given subtype and or other subtypes of Ebola virus has not been confirmed (ECDC, 2014). Given this scenario, application of non-pharmaceutical interventions such as contact tracing and quarantine will accelerate the control of the spread of EVD. Contact tracing is defined as the process of identifying, assessing and managing people who have been in contact with an infected person (CDC, 2014). Quarantine, on the other hand, is the restriction of movement of those exposed to a communicable disease for a period of time equivalent to the incubation period (CORE public health functions for BC, 2010).

Several researchers have formulated mathematical models on how to curtail EVD. Legrand et al. (2007) used a modified Susceptible Exposed Infected Recovered (SEIR) stochastic model to study the dynamics of EVD epidemics by subdividing infected individuals into hospitalized, funeral, infectious and symptomatic cases. Transmission was assumed to occur in the community, hospital setting and during funeral if the dead bodies were not properly handled. Recovered individuals were assumed to have permanent immunity. They applied the model to Uganda 2000 and Democratic Republic of Congo 1995 Ebola epidemics. Their result showed that rapid implementation of control measures such as barrier nursing within isolation ward and prompt hospitalization will reduce disease spread within the hospital and the community. Rivers et al. (2014) applied the model of Legrand et al. (2007) to 2014 Liberia Ebola epidemics in order to assess the impact of increasing pharmaceutical interventions on the survival of hospitalized patients.

Okeke et al. (2014) developed deterministic model for Ebola virus disease by separating the health workers from the general population leading to two basic SEIR subpopulation models. Althaus (2014) used a SEIR model to explain the 2014 West African Ebola virus epidemic with an assumption that the epidemic started with a single infected case. House (2014) studied the dynamics of Ebola outbreaks using three stochastic models: the transmission, the new outbreak and the case fatality models.

Atangana and Goufo (2014) developed a SusceptibleInfected-Recovered-Death (SIRD) model to study the spread of EVD in Sub-Saharan African countries such as Liberia, Guinea and Sierra Leone. They assumed that recovered individuals lost immunity and become susceptible again with natural death in Susceptible Infected Recovered (SIR) compartments. Li et al. (2015) proposed a Susceptible-Exposed-Infected-Treatment (SEIT) model of Ebola virus transmission. They assumed that some treated individuals will die of the disease while some will recover and lose immunity. The exposed class was assumed to be infected. Webb et al. (2015) modified an SEIR model for Ebola virus by incorporating contact tracing in the model and applied it to 2014 Sierra Leone, Liberia, and Guinea Ebola epidemics.

This study is motivated by the work of Legrand et al. (2007) and the 2014 Ebola epidemic that ravaged West African countries. It is clear that many mathematical models had been developed to analyze the spread of EVD but none to the best of our knowledge had considered the combined impact of contact tracing and quarantine on EVD. To this end, we consider the role of contact tracing and quarantine as control measures on the transmission dynamics of EVD and examine the global stability of the equilibrium states of the model.

2. The Model Formulation

The model in this paper examines the dynamics of EVD. The total population size denoted by $N(t)$ at time t is divided into four sub-populations namely Susceptible individuals, $S(t)$, Quarantined individuals, $Q(t)$, Infected individuals, $I(t)$, and individuals under treatment, $T(t)$. Susceptible class, $\mathrm{S}(t)$, are individuals who can be infected by Ebola virus following a contact with infected individuals and infected class, $I(t)$, are those infected with Ebola virus in the community and are capable of spreading the disease to susceptible individuals. The Quarantined individuals, $Q(t)$, consists of immigrants from the affected countries, who are coming in or passing through the target population at a rate ε and those from active contact tracing process. The total population size denoted by $N(t)$ at time t is given by

$$N(t) = S(t) + Q(t) + I(t) + T(t).$$

This model concentrates on the long term control of EVD by incorporating vital dynamics. A natural death rate, $\mu > 0$, is assumed in susceptible, infected and treatment sub-populations of the model. A death rate, $\mu_1 > 0$, is assumed for quarantine class since they have a short stay in this class, that is, between 2 and 21 days. The susceptible class is increased in three ways: immigration from non-Ebola affected population at a rate, Λ, treated individuals who recovered but lose immunity at a rate, ϕ, and quarantined individuals who did not develop symptoms after the incubation period of the virus and return to the susceptible class at a rate, σ. Individuals in

the susceptible class may acquire EVD when they come in contact with infected individuals at a rate, $\frac{\beta I}{N}$, where β is defined as the probability that a contact between a susceptible person and an infected person leads to a new infection (Benyah, 2007). In addition, the population of quarantined individuals is increased by recruitment of immigrants from EVD affected population where the disease is endemic at a rate, ε and the population of exposed susceptible individuals identified through contact tracing at a rate, c_1. The population of quarantined individuals that develop symptoms of infection progress to the treatment class at a rate, φ. Contact tracing is carried out on all persons in the treatment class. This is to pull out more infected individuals who are still in the community to the treatment class at a rate, c_2 and those that may have been exposed in the susceptible class to the quarantined class at a rate, c_1 (Hsieh et al., 2010; Mubayi et al., 2010). Infected individuals move to treatment class in two ways. One way is through active contact tracing and the other is through self-presentation to hospitals at a rate, α. Individuals in infected class may die due to EVD at a rate, d_1 (Li et al., 2015).

Furthermore, the following are additional assumptions of the model.

i. The population is homogeneous.
ii. Immigrants from EVD affected populations are quarantined for a period of time equivalent to the incubation period of the disease.
iii. Treatment individuals may become susceptible again when they recover since EVD is not known to confer immunity (Atangana and Goufo, 2014; Kalu et al., 2016).
iv. Individuals who died of the disease are immediately buried, preventing transmission after death (Leward et al., 2014).
v. Exposed class of individuals is ignored since the incubation period of the disease is short.

The above assumptions lead to the flow diagram in Figure 1 while Table 1 gives the parameters of the model.

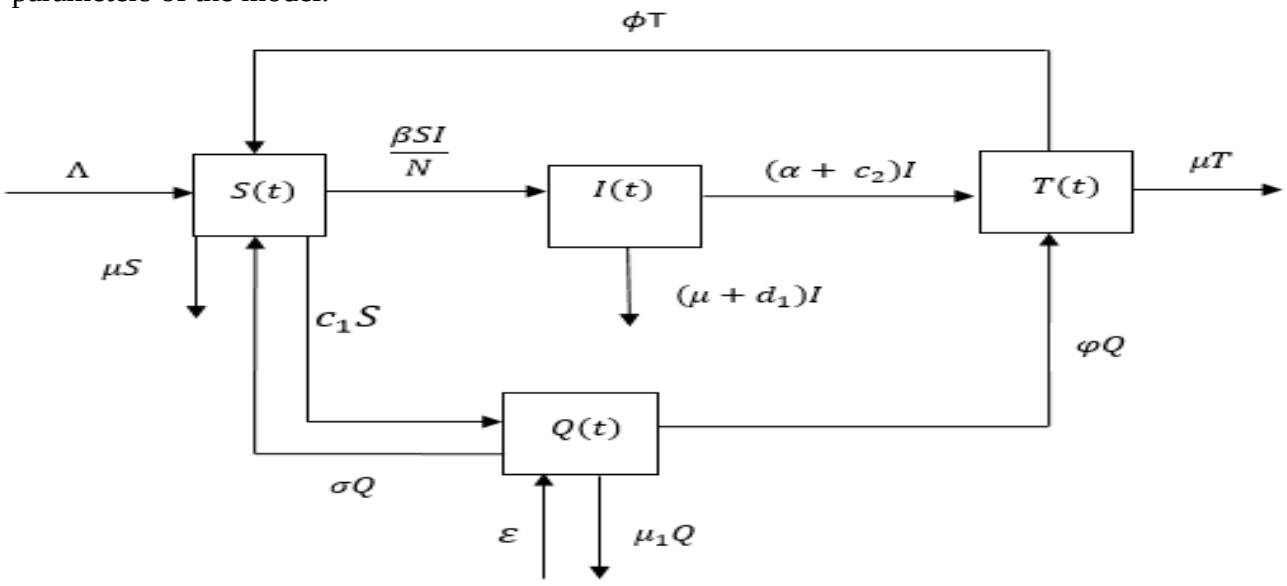

Figure 1. Flow diagram for EVD transmission model with contact tracing and quarantine.

Table 1. Model parameters

Parameter	Description
β	Disease transmission rate
c_2	Contact tracing rate for infected individuals
c_1	Contact tracing rate for exposed susceptible individuals
d_1	Ebola induced death rate for infected class
Λ	Immigration rate from non-Ebola affected populations
σ	Rate at which quarantined individuals who did not develop symptoms of infection return to susceptible class
ϕ	Rate at which individuals under treatment recover and become susceptible again
ε	Immigration rate from Ebola affected populations
μ	Natural death rate for susceptible, infected and treatment classes
μ_1	Death rate for quarantined class for the period of 2 – 21 days
φ	Rate for identifying the infected persons in the quarantined class
α	Rate for identifying the infected persons in the infected class

From the assumptions of the model and the flow diagram in Figure 1, the model equations are presented as follows:

$$\frac{dS}{dt} = \Lambda - \frac{\beta SI}{N} + \sigma Q + \phi T - \mu S - c_1 S, \quad S(0) = S_0, \tag{1}$$

$$\frac{dQ}{dt} = \varepsilon + c_1 S - \sigma Q - \varphi Q - \mu_1 Q, \quad Q(0) = Q_0, \tag{2}$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - (\mu + \alpha + d_1) I - c_2 I, \quad I(0) = I_0, \tag{3}$$

$$\frac{dT}{dt} = \alpha I + \varphi Q + c_2 I - \mu T - \phi T, \quad T(0) = T_0, \tag{4}$$

where, S_0 , Q_0 , I_0 , and T_0 are assumed to be positive.

3. Demographic Parameters

The spread of EVD in Liberia started on 16th of June 2014 as officially reported by WHO with 33 cases and 24 deaths (Althaus, 2014). The whole population of Liberia was estimated at 4,396,554 in the year 2014 (World Bank group, 2016a). We estimate the initial conditions as $S(0) = 4396554 - 33 = 4396521$, $I(0) = 33$, and $T(0) = 33 - 24 = 9$. For $Q(0)$, the initial quarantined individuals, it is computed as $Q(0) = 107 - 33 = 74$ (that is the difference between 30th June, 2014 and 16th June, 2014), since the incubation period of EVD falls within the period. Here, 107 is the number of confirmed cases as at 30th June, 2014 while 33 is the number of confirmed cases as at 16th June, 2014.

We have twelve parameters for model equations (1) – (4). Immigration rate, Λ and natural death rate, μ are estimated from the demographic profile of Liberia. For immigration rate, Λ, we

estimated it from birth rate of Liberia, that is, total number of births per 1000 of a population N in a year. It is estimated at midyear. Liberia birth rate is given as 35 per 1000 for 2014 (World Bank group, 2016b). So, the immigration rate of Liberia is given by $\Lambda = \frac{35}{1000} \times \frac{1}{365} \times 4396554 = 421.59 \approx 422$ individuals per day. In addition, the natural death rate, μ is expressed as total number of deaths per 1000 individual per year estimated in midyear. So, the natural death rate, μ for Liberia for year 2014 is 9 (World Bank group, 2016c; UNICEF, 2016). Thus, $\mu = \frac{9}{1000} \times \frac{1}{365} = 2.465753 \times 10^{-5}\ day^{-1}$. Using the parameter values from Rivers et al. (2014), we have the parameter values given in Table 2.

4. Model Analysis

The model of equations (1) - (4) has a unique solution in the feasible region

$$\Omega = \left\{(S, Q, I, T) \in \mathbb{R}_+^4 : N \leq \frac{\varepsilon+\Lambda}{\mu}\right\}.$$

The details of the proof can be found in (Madubueze et al., 2016). This means that the model is biologically meaningful and epidemiologically well posed in the region, Ω, that is, every solution of the model with initial condition in positive region, Ω, remains or enters at all time (Hethcote, 2000). We now consider the stability analysis of the equations (1) – (4).

4.1. The disease-free equilibrium state and its stability

From Madubueze et al. (2017), the disease – free equilibrium state (DFE), $E_0 = (S_0, Q_0, 0, T_0)$ is given as

$$E_0 = \left[\frac{\Lambda fg+g\sigma\varepsilon+\phi\varphi\varepsilon}{\mu gf+c_1\varphi\mu+gc_1\mu_1}, \frac{\Lambda gc_1+gc_1\varepsilon+g\mu\varepsilon}{\mu gf+c_1\varphi\mu+gc_1\mu_1}, 0, \frac{\Lambda\varphi c_1+\varphi c_1\varepsilon+\mu\varphi\varepsilon}{\mu gf+c_1\varphi\mu+gc_1\mu_1}\right], \tag{5}$$

where

$$N_0 = S_0 + Q_0 + T_0,\ f = \sigma + \varphi + \mu_1, \quad g = \phi + \mu \quad \text{and} \quad h = \mu + \alpha + d_1 + c_2. \tag{6}$$

The effective reproduction number, R_e, is defined as the mean number of new infections generated by a typical infectious individuals introduced in a population where contact tracing and quarantine are adopted as control measures. It is a threshold parameter that governs the spread of disease in a population where control measures are in place. When $R_e < 1$, it means that EVD can be eliminated from the population in the presence of contact tracing and quarantine. However, when $R_e > 1$, it implies that EVD will persist in the population where contact tracing and quarantine are implemented. The effective reproduction number, R_e, is computed using next generation method described by Driessche and Watmough (2002).

This is given as

$$R_e = \frac{\beta S_0}{hN_0} = \frac{\beta}{h}\left[\frac{\Lambda fg+g\sigma\varepsilon+\phi\varphi\varepsilon}{\Lambda fg+g\sigma\varepsilon+\phi\varphi\varepsilon+\Lambda gc_1+gc_1\varepsilon+g\mu\varepsilon+\Lambda\varphi c_1+\varphi c_1\varepsilon+\mu\varphi\varepsilon}\right]. \tag{7}$$

According to Driessche and Watmough (2002), the DFE E_0 is locally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$.

Applying the model equations (1) – (4) to the 2014 Liberia Ebola epidemic gives $R_e = 0.3722121449$. It means that a small inflow of infected immigrants would not generate large outbreaks. Therefore, EVD will be eradicated from the population where contact tracing and quarantine are implemented together (that is, the DFE is stable).

This is comparable to the effective reproduction number by Legrand et al., (2007) for the 2000 Ugandan Ebola epidemic. They estimated $R_e = 0.4$ after implementation of barrier nursing while Rivers et al. (2014) estimated reproduction number, $R_0 = 2.23$ for 2014 Liberia Ebola epidemic after pharmaceutical intervention. Both authors used the same stochastic model and considered either of barrier nursing or pharmaceutical interventions. However, our model is a deterministic model which considered implementation of non-pharmaceutical interventions such as contact tracing and quarantine. From the reproduction numbers aforementioned, it is clear that the implementation of quarantine and contact tracing measures will reduce the spread of EVD faster.

4.2. Endemic equilibrium state E_1

The Endemic Equilibrium (EE) state, E_1, is a steady state solution where the disease persists in the population ($I \neq 0$). It is obtained by setting the derivatives of the model equations (1) – (4) to zero and solving the resultant equations.

For $I \neq 0$, let the force of the infection be

$$\lambda = \frac{\beta I}{N}. \tag{8}$$

Let $E_1 = (S^*, Q^*, I^*, T^*)$ represent any endemic equilibrium of model equations (1) – (4). Then, "solving the resultant equations of model (1) – (4) in terms of $= \frac{\beta I}{N}$, we get"

$$S^* = \frac{h(\Lambda f g+\varepsilon g\sigma+\varepsilon\phi\varphi)}{A\lambda+B},$$

$$Q^* = \frac{\lambda(\varepsilon\mu c_1+\varepsilon\mu\alpha+\varepsilon g\mu+\varepsilon g d_1)+h(\Lambda g c_1+\varepsilon g\mu+\varepsilon g c_1)}{A\lambda+B},$$

$$I^* = \frac{\lambda(\Lambda f g+\varepsilon g\sigma+\varepsilon\phi\varphi)}{A\lambda+B},$$

$$T^* = \frac{\lambda(\Lambda\alpha f+\Lambda f c_2+\varepsilon\sigma\alpha+\varepsilon h\varphi+\varepsilon\sigma c_2)+h(\Lambda\varphi c_1+\varepsilon\mu\varphi+\varepsilon h c_1)}{A\lambda+B},$$

where

$A = f(\alpha\mu + \mu c_2 + \mu g + g d_1)$;
$B = \sigma + \mu^2\varphi + \mu^2\mu_1 + \mu\phi\sigma + \mu\phi\varphi + \mu\phi\mu_1 + \mu\varphi c_1 + \mu c_1\mu_1 + \phi c_1\mu_1$.

Now, $N^* = S^* + Q^* + I^* + T^*$, which gives $N^* = \frac{C\lambda + D}{A\lambda + B}$ with

$C = \Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon + \Lambda\alpha f + \Lambda f c_2 + \alpha\varepsilon\sigma + \varepsilon h\varphi + \sigma\varepsilon c_2 + \varepsilon(\alpha\mu + \mu c_2 + \mu g + g d_1)$,
$D = h(\Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon + \Lambda g c_1 + g c_1\varepsilon + g\mu\varepsilon + \Lambda\varphi c_1 + \varphi c_1\varepsilon + \mu\varphi\varepsilon)$.

Substituting I^* as I and N^* as N into (8) and simplifying gives

$$C\lambda + D(1 - R_e) = 0. \tag{9}$$

It is evident that coefficients C and D are positive.

Hence,

$$\lambda = \frac{D}{C}(R_e - 1) > 0, \qquad \text{whenever} \quad R_e > 1. \tag{10}$$

The component of $E_1 = (S^*, Q^*, I^*, T^*)$ is obtained by substituting the value of λ given in (10) and this gives

$S^* = \frac{Ch(\Lambda f g + \varepsilon g\sigma + \varepsilon\phi\varphi)}{AD(R_e - 1) + BC}$,

$Q^* = \frac{D(\varepsilon\mu c_1 + \varepsilon\mu\alpha + \varepsilon g\mu + \varepsilon g d_1)(R_e - 1) + Ch(\Lambda g c_1 + \varepsilon g\mu + \varepsilon g c_1)}{AD(R_e - 1) + BC}$,

$I^* = \frac{D(\Lambda f g + \varepsilon g\sigma + \varepsilon\phi\varphi)(R_e - 1)}{AD(R_e - 1) + BC}$,

and

$T^* = \frac{D(\Lambda\alpha f + \Lambda f c_2 + \varepsilon\sigma\alpha + \varepsilon h\varphi + \varepsilon\sigma c_2)(R_e - 1) + Ch(\Lambda\varphi c_1 + \varepsilon\mu\varphi + \varepsilon h c_1)}{AD(R_e - 1) + BC}$.

This lead to the following theorem.

Theorem 1.

The model of equations (1) – (4) has one unique (positive) endemic equilibrium, given by E_1, whenever $R_e > 1$.

4.3. Local stability of the endemic equilibrium state

The local stability of endemic equilibrium can be established by finding the eigenvalues of the Jacobian matrix evaluated at the endemic equilibrium state. Sometimes, this approach can be mathematically complicated. The approach of centre manifold theory described by Castillo - Chavez and Song (2004) to investigate the stability of endemic equilibrium near $R_e = 1$ is applied. It is used to examine the existence of backward and forward bifurcation at $R_e = 1$ (Arion et al., 2003; Xue and Wang, 2012). When the bifurcation is forward, it implies that disease free equilibrium is locally asymptotically stable for $R_e < 1$ and there is no disease in the population and also endemic equilibrium is locally asymptotically stable for $R_e > 1$. This means the disease cannot invade the population when $R_e < 1$ and is possible to have global asymptotical stability of the two equilibria. However, backward bifurcation occurs when the endemic equilibrium exists for $R_e < 1$ and disease free equilibrium may exist when $R_e > 1$. This is due to existence of multiple equilibria and re-infection. This happens when a stable endemic equilibrium co – exist with the DFE. The presence of backward bifurcation implies that $R_e < 1$ is not sufficient condition to eradicate the disease that is endemic in the population but is adequate for avoiding an epidemic caused by few infectives introduced initially in the population (Piazza and Wang, 2013). The global asymptotical stability of the two equilibrium states (DFE and EE) may be impossible.

Theorem 2. Centre manifold theory (Castillo - Chavez and Song, 2004).

Consider the following general system of ordinary differential equations with a parameter β:

$$\frac{dx}{dt} = F(x, \beta), \tag{11}$$

$F: R \times R^n \to R$ and $F \in C^2(R^n \, x \, R)$,

where 0 is an equilibrium point for the system (11) for all values of the parameter β, that is $F(0, \beta) \equiv 0$ for all β and

1. $A = D_x F(0,0) = \left[\frac{dF_i}{dx_j}(0,0)\right]$ is the linearization matrix of the system (11) around the equilibrium point 0 with β evaluate at 0.

2. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts.

3. Matrix A has a right eigenvector $\boldsymbol{w}$ and a left eigenvector $\boldsymbol{v}$ corresponding to the zero eigenvalue.

Let f_k be the k^{th} component of F and

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

and also

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

Then, "the local dynamics of the system (11) around the equilibrium point 0 is totally determined by the signs of a and b".

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

ii. $a < 0,\ b < 0$, when $\beta < 0$ with $|\beta| \ll 1,\ 0$ is unstable; when $0 < \beta \ll 1$, asymptotically stable, and there exists a positive unstable equilibrium.

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

iv. $a < 0,\ b > 0$, when $\beta < 0$ changes from negative to positive, 0 changes its stability from stable to unstable. Corresponding to a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if $a < 0$ and $b > 0$, then a forward bifurcation occurs at $\beta = 0$.

Therefore, from (7)

$$R_e = \frac{\beta}{h}\left[\frac{\Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon}{\Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon + \Lambda g c_1 + g c_1\varepsilon + g\mu\varepsilon + \Lambda\varphi c_1 + \varphi c_1\varepsilon + \mu\varphi\varepsilon}\right] = 1.$$

Let $\beta = \beta^*$, where β^* is chosen as the bifurcation parameter that occurs at $R_e = 1$, and solve for β^*. We obtain

$$\beta^* = h\left[\frac{\Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon + \Lambda g c_1 + g c_1\varepsilon + g\mu\varepsilon + \Lambda\varphi c_1 + \varphi c_1\varepsilon + \mu\varphi\varepsilon}{\Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon}\right],$$

so that the DFE, E_0 is locally stable when $\beta < \beta^*$, that is, $R_e < 1$ and unstable when $\beta > \beta^*$, that is, $R_e > 1$.

Also, let $S = x_1,\quad Q = x_2,\ I = x_3,\quad T = x_4$. Then, "the model equations (1) – (4) become"

$$\frac{dx_1}{dt} = f_1 = \Lambda - \frac{\beta^* x_1 x_3}{N} + \sigma x_2 + \phi x_4 - \mu x_1 - c_1 x_1, \tag{12}$$

$$\frac{dx_2}{dt} = f_2 = \varepsilon + c_1 x_1 - f x_2, \tag{13}$$

$$\frac{dx_3}{dt} = f_3 = \frac{\beta^* x_1 x_3}{N} - h x_3, \tag{14}$$

$$\frac{dx_4}{dt} = f_4 = \varphi x_2 + (\alpha + c_2)x_3 - gx_4, \tag{15}$$

where f, g and h are defined in (6) and $N = x_1 + x_2 + x_3 + x_4$.

The Jacobian matrix $J(E_0)$ of model $(1) - (4)$ at the disease-free equilibrium when $\beta = \beta^*$ with $R_e = 1$ is given by

$$J(E_0) = \begin{bmatrix} -(\mu + c_1) & \sigma & -h & \phi \\ c & -f & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \varphi & \alpha + c_2 & -g \end{bmatrix}. \tag{16}$$

The Jacobian matrix $J(E_0)$ in equation (16) has eigenvalue, $\lambda = 0$ as a simple zero eigenvalue and negative eigenvalues using Routh – Hurwitz criteria on the characteristics equation

$$\lambda^3 + P\lambda^2 + Q\lambda + R = 0,$$

where

$$P = g + f + c_1 + \mu,$$

$$Q = fg + c_1 g + \mu g + \mu f + c_1(\varphi + \mu_1),$$

$$R = \mu f g + c_1 \mu_1 g + c_1 \varphi \mu.$$

Applying the centre manifold theory, let $\boldsymbol{w} = (w_1, w_2, w_3, w_4)$ be the right eigenvector associated with the zero eigenvalue**.** This is computed by multiplying the Jacobian matrix $J(E_0)$ (16) with $\boldsymbol{w}$ and equate to zero. We have

$$\left.\begin{aligned} w_2 &= \tfrac{c_1}{f} w_1, \\ w_3 &= \tfrac{\sigma c_1 g + \phi c_1 \varphi - fg(\mu + c_1)}{fgh - \phi(\alpha + c_2)f} w_1, \\ w_4 &= \tfrac{c_1\varphi(gh - \phi(\alpha + c_2)) + c_1(\alpha + c_2)(\sigma g + \phi\varphi) - fg(\alpha + c_2)(c_1 + \mu)}{g(fgh - \phi(\alpha + c_2)f)} w_1. \end{aligned}\right\} \tag{17}$$

Similarly, the left eigenvector of the Jacobian $J(E_0)$ associated with the zero eigenvalue is given by $\boldsymbol{v} = (v_1, v_2, v_3, v_4)$ and it satisfies $\boldsymbol{v}.\boldsymbol{w} = 1$. Transposing Jacobian $J(E_0)$ first and multiplying by $\boldsymbol{v}$ and equate to zero, we have $\boldsymbol{v} = (0,0, v_3, 0)$.

Computation of a and b.

We can only consider when $k = 3$ since $v_1 = v_2 = v_4 = 0$ for $k = 1, \dots, 4$. That is, the function

$$f_3 = \frac{\beta^* x_1 x_3}{N} - hx_3$$

will be used to determine a and b from the system of equations (12) – (15). The associated non-zero partial derivatives of f_3 at DFE E_0 for the model are given by

$$\frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_2 \partial x_3} = -\frac{\beta^* S_0}{N_0^2}\ ,\ \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_3^2} = -\frac{2\beta^* S_0}{N_0^2}\ ,\ \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_1 \partial x_3} = \frac{\beta^*}{N_0} - \frac{\beta^* S_0}{N_0^2}, \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_3 \partial x_4} = -\frac{\beta^* S_0}{N_0^2},$$

with

$$N_0 = S_0 + Q_0 + T_0, \quad \text{since } I_0 = 0.$$

Therefore,

$$a = v_3\left[w_1 w_3 \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_1 \partial x_3} + w_2 w_3 \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_2 \partial x_3} + w_3^2 \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_3^2} + w_3 w_4 \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_3 \partial x_4}\right].$$

Substituting the respective partial derivatives into a and using the properties that $v_3.w_3 = 1$, and $N_0 = \frac{\beta^* S_0}{h}$ since $R_e = 1$, we have, after simplifying, that

$$a = \frac{h}{\beta^* S_0}\left(\beta^* w_1 - h(w_1 + w_2 + 2w_3 + w_4)\right). \tag{18}$$

From (18), we see that $a > 0$, if

$$\beta^* > \frac{h(w_1+w_2+2w_3+w_4)}{w_1},$$

or $a < 0$, if

$$\beta^* < \frac{h(w_1+w_2+2w_3+w_4)}{w_1}.$$

Also, we compute the value of b as

$$b = v_3 w_3 \frac{\partial^2 f_3(E_0,\beta^*)}{\partial x_3 \partial \beta^*}\ .$$

The associated non – zero partial derivative of f_3 at DFE, E_0 is

$$\frac{\partial^2 f_3(0,0)}{\partial x_3 \partial \beta^*} = \frac{S_0}{N_0},$$

so that

$$b = v_3 w_3 \frac{S_0}{N_0} > 0, \qquad \text{since} \qquad v_3 w_3 = 1.$$

Therefore, $b > 0$ and $a < 0$ or $a > 0$ subject to whether

$$\beta^* < \frac{h(w_1+w_2+2w_3+w_4)}{w_1} \quad \text{or} \quad \beta^* > \frac{h(w_1+w_2+2w_3+w_4)}{w_1},$$

where w_2, w_3 and w_4 are defined in (17).

We have the following theorem.

Theorem 3.

The model (1) – (4) exhibits a backward bifurcation at $R_e = 1$ if $\beta^* w_1 > h(w_1 + w_2 + 2w_3 + w_4)$. If $\beta^* < 0$, this implies that there exists unstable negative endemic equilibrium state and when β^* changes from negative to positive, a positive stable endemic equilibrium point exists. Therefore, the endemic equilibrium state, E_1 is locally asymptotically stable for $R_e > 1$, but close to 1 when $\beta^* w_1 < h(w_1 + w_2 + 2w_3 + w_4)$.

Theorem 3 means that the control of the EVD depends on the initial sizes of the sub-population of model equations (1) – (4) when bifurcation is backward. In this case, the EVD may not die out in the population even when $R_e < 1$. However, when bifurcation is forward, it implies that the control of the EVD does not depend on the number of people that are initially infected. This means that EVD will be eradicated in the population when $R_e < 1$ while the disease remains in the population when $R_e > 1$.

4.4. Global stability of equilibrium states

We analyze the global stability of disease-free equilibrium and endemic equilibrium of the model equations (1) – (4) when the bifurcation is forward, since global stability may not exist for backward bifurcation (Garba et al., 2008). The global stability of disease-free equilibrium implies that disease eradication is independent of initial sizes of the subpopulations when $R_e < 1$. Using the theorem by Shuai and Driessche (2013), we state the following theorem.

Theorem 4.

If $R_e < 1$, then the disease-free equilibrium of model (1) – (4) is globally asymptotically stable in Ω.

Proof:

Using matrix – theoretic method by Shuai and Driessche (2013), we have the following Lyapunov function

$$L(t) = \frac{1}{h} I(t). \tag{19}$$

Differentiating $\mathrm{L}(t)$ along solutions of (1) – (4) gives

$$L'(t) = \frac{1}{h}\left(\frac{\beta S}{N} - h\right) I, \tag{20}$$

where $'$ denotes the derivatives with respect to time t. Equation (20) can be written as

$$L'(t) = \left(\frac{S}{N}\frac{N_0}{S_0}\frac{\beta S_0}{hN_0} - 1\right) I. \tag{21}$$

Since $\frac{S}{N} \leq \frac{S_0}{N_0}$, we have

$$L'(t) \leq \left(\frac{\beta S_0}{hN_0} - 1\right) I.$$

Using the definition of R_e in (7), it implies that

$$L'(t) \leq (R_e - 1)I. \tag{22}$$

Thus, if $R_e \leq 1$, then $L'(t) \leq 0$. Also, $L'(t) = 0$ if and only if $S = S_0$, $Q = Q_0$, $T = T_0$, and $I = 0$. Therefore the maximum invariant set in $\{(S, Q, I, T) \in \Omega : L'(t) = 0\}$ is the singleton $\{E_0\}$, where E_0 is the disease free equilibrium state. By LaSalle's invariant principle, every solution to the model equations (1) – (4) with initial conditions in Ω, tends to E_0 as $t \longrightarrow \infty$. Hence, the disease free equilibrium state, E_0 is globally asymptotically stable in Ω if $R_e \leq 1$.

Next, the global stability property of the endemic equilibrium state E_1 is studied for the special case when the disease-induced death rate, $d_1 = 0$ so that $N \to \frac{\Lambda+\varepsilon}{\mu}$ asymptotically. This implies that $\frac{\beta I}{N} \sim \bar{\beta} I$, where $\bar{\beta} = \frac{\beta}{N} = \frac{\beta\mu}{\Lambda+\varepsilon}$ since $N = \frac{\Lambda+\varepsilon}{\mu}$. In the absence of disease-induced death rate $(d_1 = 0)$, the frequency dependent incidence, $\frac{\beta IS}{N}$, changes to density dependent incidence $\bar{\beta} IS$.

We state the following theorem using the approach by (Kaur et al., 2014; Nyerere et al., 2014; Gou and Li, 2011).

Theorem 5.

In the absence of disease-induced death rate, $(d_1 = 0)$, the endemic equilibrium (EE) state. E_1 is globally asymptotically stable in Ω if $R_e > 1$ and the following inequalities are satisfied

$$1 \leq \frac{T^*}{T} \text{ for } 0 < T < T^*,$$

$$1 \leq \frac{Q^*}{Q} \text{ for } 0 < Q < Q^*,$$

$$1 \leq \frac{S^*}{S} \text{ for } 0 < S^* < S,$$

$$b\big((\alpha + c_2)I + \varphi Q\big) > \phi T \frac{S^*}{S},$$

for

$$b = \frac{\phi T^*}{(\alpha + c_2)I^* + \varphi Q^*}.$$

***Proof**:*

The global stability of the EE can be determined by constructing a Lyapunov function $L(t)$ such that

$$L(t) = \left(S - S^* - S^* \ln \frac{S}{S^*}\right) + \left(Q - Q^* - Q^* \ln \frac{Q}{Q^*}\right) + \left(I - I^* - I^* \ln \frac{I}{I^*}\right) + b\left(T - T^* - T^* \ln \frac{T}{T^*}\right),$$

where

$$b = \frac{\phi T^*}{(\alpha + c_2)I^* + \varphi Q^*}. \tag{23}$$

The time derivative of $L(t)$ along the solutions of model equations (1) – (4) is given by

$$\begin{aligned}\frac{dL}{dt} &= \left(1 - \frac{S^*}{S}\right)S' + \left(1 - \frac{Q^*}{Q}\right)Q' + \left(1 - \frac{I^*}{I}\right)I' + b\left(1 - \frac{T^*}{T}\right)T' \\ &= \left(1 - \frac{S^*}{S}\right)\left(\Lambda - \bar{\beta}SI + \phi T + \sigma Q - \mu S - c_1 S\right) + \left(1 - \frac{Q^*}{Q}\right)\left(\varepsilon + c_1 S - (u + \varphi + \sigma)Q\right)\left(1 - \frac{I^*}{I}\right)\left(\bar{\beta}SI - (c_2 + \alpha + d_1 + \mu)I\right) + b\left(1 - \frac{T^*}{T}\right)\big((\alpha + c_2)I + \varphi Q - (\mu + \phi + d_2)T\big).\end{aligned} \tag{24}$$

At the equilibrium state, $E_1 = (S^*, Q^*, I^*, T^*)$, we have the following relations

$$\left.\begin{aligned}\Lambda &= \bar{\beta}S^*I^* + \mu S^* - \phi T^* - \sigma Q^* + c_1 S^* \\ \varepsilon &= (u + \varphi + \sigma)Q^* - c_1 S^* \\ (\alpha + c_2 + d_1 + \mu) &= \bar{\beta}S^* \\ (u + \phi + d_2) &= \frac{(\alpha + c_2)I^*}{T^*} + \frac{\varphi Q^*}{T^*}\end{aligned}\right\}. \tag{25}$$

Substituting (25) into (24), we obtain

$$\frac{dL}{dt} = \left(1 - \frac{S^*}{S}\right)\left(\bar{\beta}S^*I^* + \mu S^* + c_1S^* - \phi T^* - \sigma Q^* - \bar{\beta}SI + \phi T + \sigma Q - \mu S - c_1S\right) + \left(1 - \frac{Q^*}{Q}\right)(c_1S - c_1S^* + (u + \varphi + \sigma)Q^* - (u + \varphi + \sigma)Q) + \left(1 - \frac{I^*}{I}\right)\left(\bar{\beta}SI - \bar{\beta}S^*I\right) + b\left(1 - \frac{T^*}{T}\right)\left((\alpha + c_2)I + \varphi Q - \frac{(\alpha+c_2)I^*T}{T^*} - \frac{\varphi Q^*T}{T^*}\right).$$

This gives

$$\frac{dL}{dt} = \left(1 - \frac{S^*}{S}\right)\left(\bar{\beta}S^*I^*\left(1 - \frac{SI}{S^*I^*}\right) + (c_1 + \mu)S^*\left(1 - \frac{S}{S^*}\right) + \phi T\left(1 - \frac{T^*}{T}\right) + \sigma Q\left(1 - \frac{Q^*}{Q}\right)\right) + \left(1 - \frac{Q^*}{Q}\right)\left((u + \varphi + \sigma)Q^*\left(1 - \frac{Q}{Q^*}\right) + c_1S\left(1 - \frac{S^*}{S}\right)\right) + \left(1 - \frac{I^*}{I}\right)\left(\bar{\beta}S^*I^*\left(\frac{SI}{S^*I^*} - \frac{I}{I^*}\right)\right) + b\left(1 - \frac{T^*}{T}\right)\left((\alpha + c_2)I^*\left(\frac{I}{I^*} - \frac{T}{T^*}\right) + \varphi Q^*\left(\frac{Q}{Q^*} - \frac{T}{T^*}\right)\right). \quad (26)$$

After expansion of (26) and algebraic arrangement, we get

$$\frac{dL}{dt} = (\mu + c_1)S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + (u + \varphi + \sigma)Q^*\left(2 - \frac{Q^*}{Q} - \frac{Q}{Q^*}\right) + \sigma Q\left(1 + \frac{S^*}{S}\frac{Q^*}{Q} - \frac{Q^*}{Q} - \frac{S^*}{S}\right) + c_1S\left(1 + \frac{S^*}{S}\frac{Q^*}{Q} - \frac{Q^*}{Q} - \frac{S^*}{S}\right) - \phi T^*\left(1 + \frac{S^*}{S}\frac{T}{T^*} - \frac{T}{T^*} - \frac{S^*}{S}\right) + \bar{\beta}S^*I^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + b\left((\alpha + c_2)I^*\left(1 + \frac{I}{I^*} - \frac{T}{T^*} - \frac{T^*}{T}\frac{I}{I^*}\right) + \varphi Q^*\left(1 + \frac{Q}{Q^*} - \frac{T}{T^*} - \frac{T^*}{T}\frac{Q}{Q^*}\right)\right). \quad (27)$$

Simplification of (27) with definition of b in (23), we obtain (28)

$$\frac{dL}{dt} = \left(\mu + c_1 + \bar{\beta}I^*\right)S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + (u + \varphi + \sigma)Q^*\left(2 - \frac{Q^*}{Q} - \frac{Q}{Q^*}\right) + (\sigma Q + c_1S)\left(1 + \frac{S^*}{S}\frac{Q^*}{Q} - \frac{Q^*}{Q} - \frac{S^*}{S}\right) + \left(b\left((\alpha + c_2)I + \varphi Q\right) - \phi T\frac{S^*}{S}\right)\left(1 - \frac{T^*}{T}\right). \quad (28)$$

Applying hypothesis of the theorem and the arithmetic – geometric mean inequality, we get

$$2 \le \frac{S^*}{S} + \frac{S}{S^*},\ \ 2 \le \frac{Q^*}{Q} + \frac{Q}{Q^*}, 1 \le \frac{Q^*}{Q} + \frac{S^*}{S} - \frac{S^*}{S}\frac{Q^*}{Q}\ \ and\ \ 1 \le \frac{T^*}{T}.$$

Therefore, $\frac{dL}{dt} \le 0$ for all (S, Q, I, T) in Ω and the equality $\frac{dL}{dt} = 0$ holds only for $S = S^*$, $Q = Q^*$, $I = I^*$, and $T = T^*$. Thus, the endemic equilibrium state E_1 is the only positively invariant set in feasible region $\Omega = \{(S, Q, I, T)\}$. By LaSalle's invariant principle, the endemic equilibrium state, E_1 is globally asymptotically stable in the interior of Ω if $R_e > 1$ and $d_1 = 0$.

5. Numerical Simulation

Numerical Simulations of the model are carried out using the initial conditions, $S(0) = 4396521, Q(0) = 74, I(0) = 33, T(0) = 9$ and parameter values in Table 2 except where specified otherwise.

Table 2: Parameter values of the EVD Model

Parameter	Value $(day)^{-1}$	Source	Parameter	Value $(day)^{-1}$	Source
β	0.160	Rivers et al. (2014)	c_1	0.06	Estimated
			c_2	0.07	,,
α	0.0608	,,	ε	100	,,
φ	0.08333	,,	μ	0.00002465753	,,
ϕ	0.0314862	,,	Λ	422	,,
d_1	0.0301653	,,	μ_1	0.000005	,,
σ	0.047619	,,			

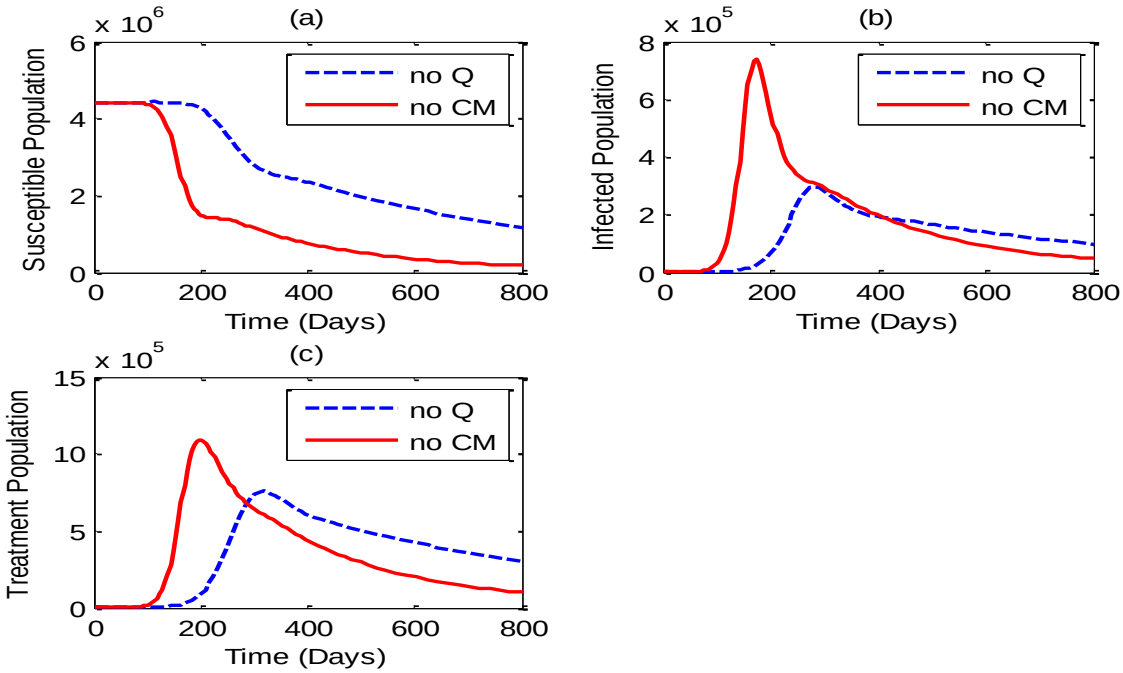

Figure 2. Simulation result showing the absence of quarantine and no control measure on the dynamics of EVD. Without quarantine, $c_1 = \mu_1 = \varepsilon = \sigma = \varphi = 0, Q = 0$ and $R_e = 0.9938508090$. In the case of absence of contact tracing and quarantine measures, $c_1 = c_2 = \mu_1 = \varepsilon = \sigma = \varphi = 0, Q = 0$ and $R_0 = 1.758435805$. Here, Q = Quarantine and CM = control measure.

The Figure 2 considers the absence of control measure on the transmission of EVD. In the absence of quarantine measure (that is only contact tracing measure is implemented), the effective reproduction number, $R_e < 1$ and there is a steady decline in the spread of the EVD because of the presence of contact tracing measure. This implies that EVD will die out in the population even when quarantine measure is not implemented. This happens because of the presence of contact tracing measure. However, when both quarantine and contact tracing measures are absent, the effective reproduction number, $R_e > 1$. This will result to high mortality rate of EVD in the population.

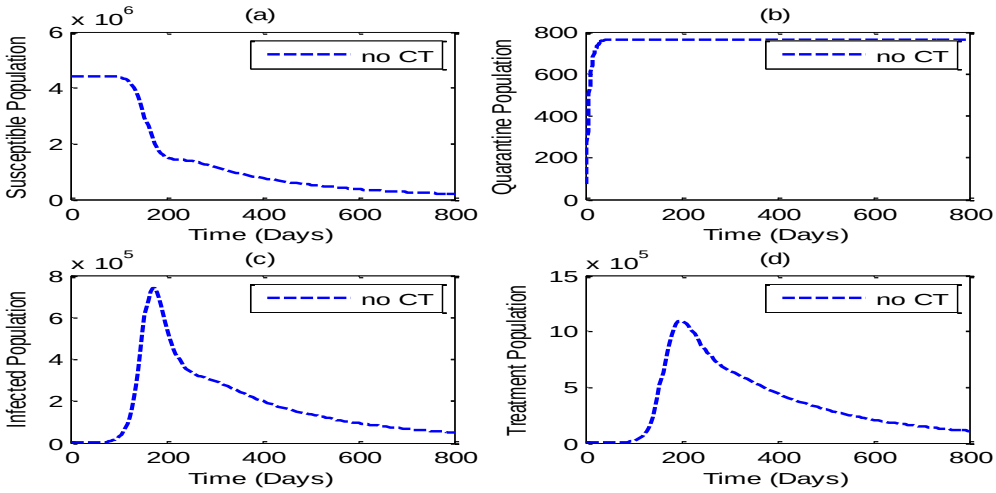

Figure 3. Simulation result showing the absence of contact tracing measure on the dynamics of EVD. Here, $c_1 = c_2 = 0$, $R_e = 1.758204642$ and CT = contact tracing.

The Figure 3 shows the absence of contact tracing in the transmission of EVD. In this case, $R_e > 1$, which behaves similar to when there is no intervention in the population. There is a rapid spread of EVD within first three months of the outbreak which reaches a peak and then begins to decline but not to zero (Figure 3c). This decline could be as a result of behavioral adaptation among individuals within the population. The result indicates that the spread of EVD will gradually decline after several months without contact tracing measure. Nevertheless, the EVD will still remain in the population. This implies that quarantine measure only will not eradicate the disease in the population.

The Figure 4 demonstrates the situations when only infected individuals are traced and when only the susceptible individuals that are exposed are traced in the presence of quarantine intervention. Any of these contact tracing implementation will reduce the number of infected individuals in the population but the reduction will take longer time when only infected individuals are traced. This may be that the infected individuals must have infected people before they are traced making EVD to spread more in the population. This is also shown in their respective effective reproduction number in Figure 4.

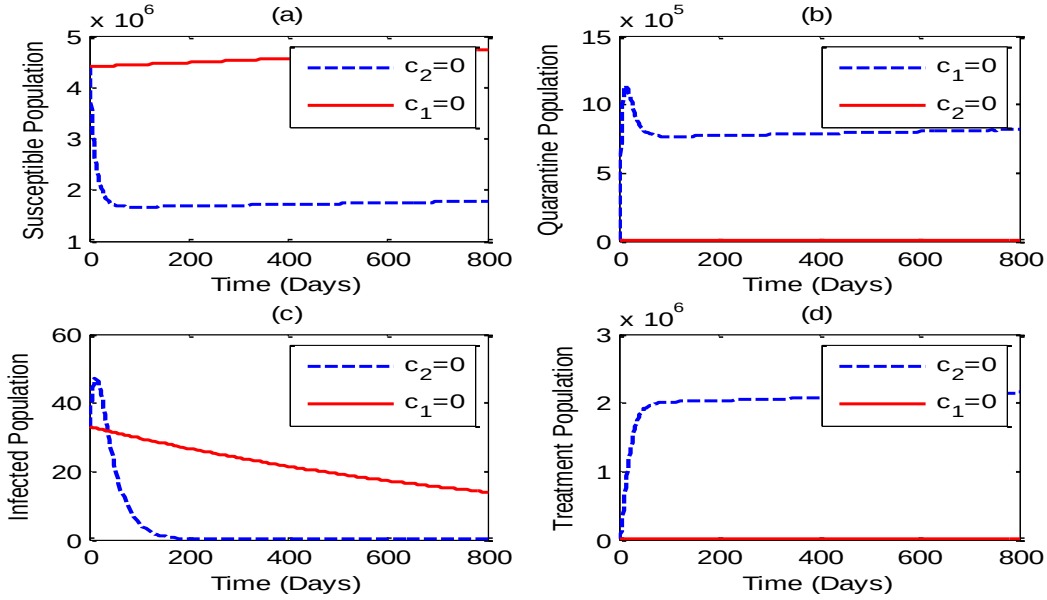

Figure 4. Simulation result showing the effect of contact tracing on dynamics of EVD. Here, when $c_1 = 0$, $R_e = 0.9937$ and $c_2 = 0$, $R_e = 0.6586$.

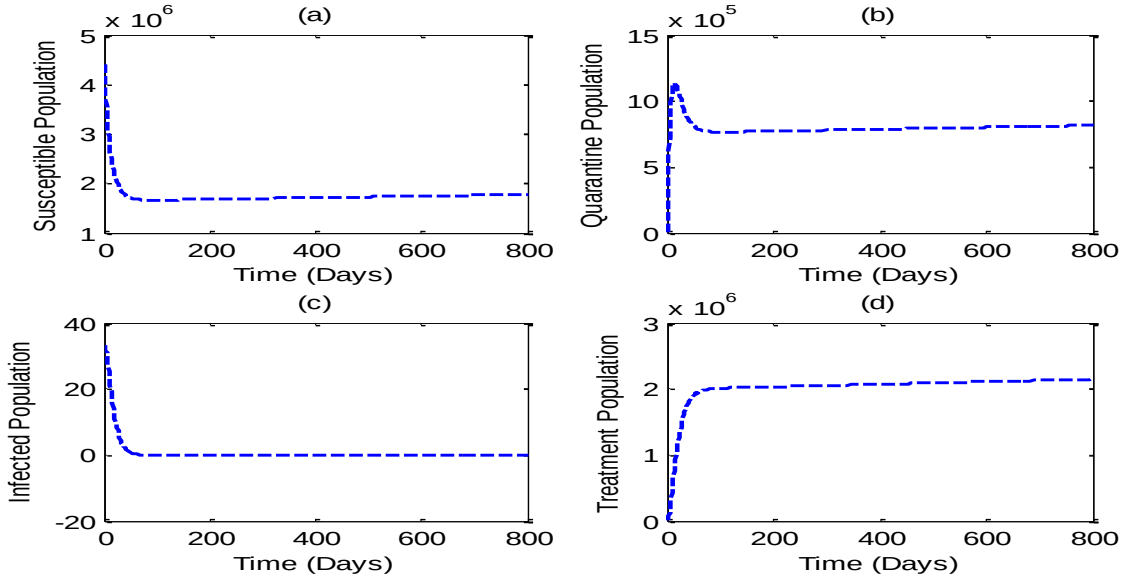

Figure 5. Simulation result showing the dynamics of EVD in the presence of contact tracing and quarantine. Here, $R_e = 0.3722121449$.

The Figure 5 considers when both contact tracing and quarantine are carried out simultaneously. This causes a sharp drop in the number of infected individuals within a very short period of time (Figure 5c). This is comparable with tracing only the susceptible individuals that are exposed (Figure 4c). The implication is that early intervention in terms of contact tracing and quarantine measures are very necessary to limit the spread of EVD.

6. Conclusion

In this study, a deterministic model for the dynamics of EVD is presented and analyzed. The existence and stability of the disease-free and endemic equilibrium states are established and the disease-free equilibrium is shown to be locally asymptotically stable when the effective reproduction number is less than unity. The endemic equilibrium is proved to be locally asymptotically stable when the effective reproduction number is greater than unity but close to unity. This is done using centre manifold theorem by Castillo - Chavez and Song (2004) to prove the existence of forward bifurcation at effective reproduction number equal to unity under certain conditions. With the condition of forward bifurcation, the global stability of the two equilibrium states are established using Lyapunov function approach. In addition, the numerical simulation of the model is carried out to examine the effect of varying some parameters on the transmission of the EVD. The result shows that the combined implementation of contact tracing and quarantine measures has a significant impact in eradicating the EVD in the population.

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