Transactions of the Nigerian Association of Mathematical Physics
Volume 3, (January, 2017), **pp 75 – 82**

Analysis of a Mathematical Model of Ebola Virus Disease with Contact Tracing and Quarantine Measures

C.E. Madubueze, A.R. Kimbir, E.S. Onah and T. Aboiyar

Department of Mathematics/Statistics/Computer Science, University of Agriculture, Makurdi, P.M.B. 2373, Nigeria.

Abstract

Currently there is no approved vaccine or medicine for the treatment of EVD. Thus, non-pharmaceutical interventions such as contact tracing and quarantine are used to control the spread of disease. Contact tracing and quarantine promptly detect new Ebola virus infected persons early before they develop symptoms. These help to prevent secondary transmission of the virus in the community. Several mathematical models have investigated the impact of different interventions on the dynamics of EVD, but none have considered the combined effect of contact tracing and quarantine. Hence, this study considered a deterministic model of Ebola virus disease (EVD) with contact tracing and quarantine as control measures in the dynamics of EVD. The effective reproduction number that governs the spread of the disease is computed using the next generation method. The model is further analyzed for the existence of the disease-free equilibrium state. This is shown to be locally asymptotically stable when the effective reproduction number is less than one and unstable when it is greater than one. Finally, numerical simulation of the model is carried out to determine the impact of model parameters on the dynamics of EVD. Based on the results obtained, it is concluded that EVD can be eradicated when contact tracing and quarantine are implemented together.

Keywords: Ebola virus disease, contact tracing, quarantine, disease free equilibrium, effective reproduction number.

1.0 Introduction

The Ebola virus is highly virulent in humans and non-humans and causes infections that often result in death. It belongs to the family of non-segmented, negative-sense single stranded ribonucleic acid (RNA) viruses called the filoviruses which cause Ebola Virus Disease (EVD) that is endemic in several African countries [1]. There are more than 25 epidemics of Ebola since the discovery of the virus in 1976 with the 2014 epidemic being the most severe. The 2014 epidemic of 24 March 2016, recorded 28,608 cases resulting in the death of 11,306 [2]. The signs and symptoms associated with the Ebola virus are usually mistaken for other diseases such as malaria, typhoid fever, influenza, or various other bacterial infections. This makes it difficult to identify infected persons early [3,4]. The prevention and control of Ebola virus in Africa poses many challenges because the identity and location of the natural reservoir of the virus is still unknown. Currently, there is no medication or vaccine for Ebola patients, rather treatment is intensive supportive therapy. Non-pharmaceutical interventions such as contact tracing and quarantine are used to control the spread of disease especially as there is no vaccine or antiviral drug to treat the disease. Contact tracing and quarantine are used to detect new Ebola virus infected persons early before they develop symptoms and as such prevent secondary transmission of the virus in the community.

Many researchers have developed mathematical models to control the virus. Examples include Arreola *et al.* [5] who studied the effect of quarantine in the spread of Ebola epidemic using a deterministic SIR model. Legrand *et al.*[6] modified a SEIR stochastic model to study the 1995 Democratic Republic of Congo (DRC) and the 2000 Ugandan epidemics of EVD epidemics while Rivers *et al.* [7] assessed the impact of increasing the use of pharmaceutical intervention on improved survival in hospitalized patients. Also, Browine *et al.* [8] modified a SEIR model for Ebola virus by incorporating contact

Corresponding author: C.E. Madubueze, E-mail: ce.madubueze@gmail.com, Tel.: +2348039523389

tracing as a control strategy. Li *et al.* [9] formulated a SEIT model of Ebola virus transmission and applied it to the 2014 Ebola epidemics in Guniea. Although many mathematical models have been used in investigating the impact of different interventions on the dynamics of EVD, none have considered the combined effect of quarantine and contact tracing. These interventions have been proved to be helpful in controlling some diseases specifically Tuberculosis, SARS, and human immunodeficiency virus [10,11,12]. Hence, this study will consider a mathematical model with contact tracing and quarantine as control measures in the dynamics of EVD.

2.0 Model Formulation

We consider the deterministic model of EVD with contact tracing and quarantine. The model is based on the following assumptions.

i. The population is homogeneous.
ii. Immigrants from EVD affected population are quarantined for a period of time equivalent to the incubation period of the virus.
iii. Treated individuals may become susceptible again when they recover since EVD is not known to confer permanent immunity [13,14],
iv. Individuals who died of the disease are immediately buried, preventing transmission after death [15],
v. Exposed class of individuals is ignored since the incubation period of the disease is short.
vi. A natural death rate is assumed in all classes of the model except the quarantined class in which the death rate is assumed a smaller value since quarantined individuals have short stay in the quarantine that is twenty-one days.

Table 1: Model parameters and variables

Variable/Parameter	Description
$S(t)$	Total number of susceptible individuals at time t
$Q(t)$	Total number of quarantined individuals at time t
$I(t)$	Total number of infected individuals at time t
$T(t)$	Total number of treated individuals at time t
β	Disease transmission rate
c_1	Contact tracing rate for susceptible that are exposed individuals
c_2	Contact tracing rate for infected individuals
d_1	Ebola induced death rate for infected class
Λ	Human recruitment rate
σ	Transfer rate from quarantined class to susceptible class after incubation period without developing symptoms
ϕ	Rate at which treated individuals recover and become susceptible again
ε	Immigration rate from Ebola affected area
μ	Natural death rate for susceptible, infected and treated classes
μ_1	Death rate for quarantined class
φ	Treatment rate of quarantined persons
α	Treatment rate of individuals other than the quarantined persons

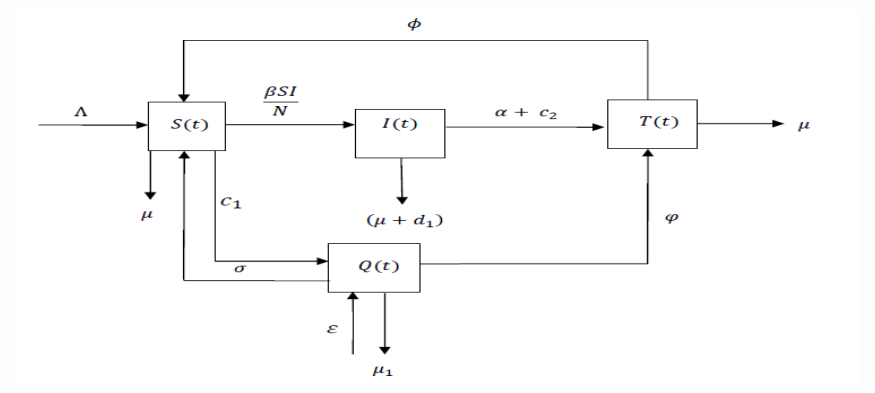

Figure 1: A Flow Diagram for the EVD Model with contact tracing and quarantine.

In view of the assumptions of the model stated above and the flow diagram in Figure 1, the model equations are derived as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta SI}{N} + \sigma Q + \phi T - \mu S - c_1 S \quad , S(0) = S_0 \\
\frac{dQ}{dt} &= \varepsilon + c_1 S - \sigma Q - \varphi Q - \mu_1 Q \quad , Q(0) = Q_0 \\
\frac{dI}{dt} &= \frac{\beta SI}{N} - (\mu + \alpha + d_1) I - c_2 I \quad , I(0) = I_0 \\
\frac{dT}{dt} &= \alpha I + \varphi Q + c_2 I - \mu T - \phi T \quad , T(0) = T_0
\end{aligned} \tag{1}$$

where,S_0, Q_0 , I_0 , and T_0 are assumed to be non –negative.

3.0 Model Analysis

3.1 Invariant Region

In this section, we show that every solution of the model with initial conditions in $\mathbb{R}_+^4$ remains or enters the region Ω at all time t. This is essential in the proof of stability analysis of the model. We state the following lemma.

Lemma. The model (1) has solutions which are contained in the feasible region $\Omega = \left\{(\mathrm{S}, \mathrm{Q}, \mathrm{I}, \mathrm{T}) \in \mathbb{R}_+^4 : \mathrm{N} \leq \frac{\varepsilon+\Lambda}{\mu}\right\}$.

Proof. We prove that the total population of humans at time t, $N(t)$ satisfies the inequality $N(t) \leq \frac{\varepsilon+\Lambda}{\mu}$. Adding the right hand sides of (1)

$$\frac{dN}{dt} = \varepsilon + \Lambda - \mu(S + I + T) - \mu_1 Q - d_1 I,$$

and this gives

$$\frac{dN}{dt} \leq \varepsilon + \Lambda - \mu(S + I + T) - \mu_1 Q. \tag{2}$$

Since $\mu_1 \geq \mu$, (2) can be rewrite as

$$\frac{dN}{dt} + \mu N \leq \varepsilon + \Lambda. \tag{3}$$

Using the method of integrating factor to solve (3) and applying the initial condition $N(0) = N_0$, we get

$$N(t) \leq \frac{\varepsilon+\Lambda}{\mu} + \left[N_0 - \frac{\varepsilon+\Lambda}{\mu}\right] e^{-\mu t}. \tag{4}$$

The population size, $N(t) \to \frac{\varepsilon+\Lambda}{\mu}$, as $t \to \infty$ in (4), which implies that $0 \leq N(t) \leq \frac{\varepsilon+\Lambda}{\mu}$. If $N_0 < \frac{\varepsilon+\Lambda}{\mu}$ then as $t \to \infty$, the trajectories approach $\frac{\varepsilon+\Lambda}{\mu}$; If $N_0 > \frac{\varepsilon+\Lambda}{\mu}$, the solution $N(t)$ decrease to $\frac{\varepsilon+\Lambda}{\mu}$ as $t \to \infty$. In either case the solution approaches $N(t) = \frac{\varepsilon+\Lambda}{\mu}$ as $t \to \infty$.Hence, the feasible solution set of the model (1) enters the region $\Omega = \left\{(\mathrm{S}, \mathrm{Q}, \mathrm{I}, \mathrm{T}) \in \mathbb{R}_+^4 : \mathrm{N} \leq \frac{\varepsilon+\Lambda}{\mu}\right\}$, which is a positively invariant set. According to Hethcote [16], the model (1) is biologically meaningful and epidemiologically well posed in the region Ω. Therefore, it is sufficient to consider the stability analysis of the model (1).

3.2 The Disease-free Equilibrium State and its stability

The disease – free equilibrium state (DFE) ,E_0, is a steady state solution where there is no Ebola virus in the population. This is calculated by setting the right hand side of the model (1) to zero. This gives the following

$$\Lambda - \frac{\beta SI}{N} + \sigma Q + \phi T - \mu S - c_1 S = 0 \tag{5}$$

$$\varepsilon + c_1 S - \sigma Q - \varphi Q - \mu_1 Q = 0 \tag{6a}$$

$$\frac{\beta SI}{N} - (\mu + \alpha + d_1 + c_2) I = 0 \tag{6b}$$

$$(c_2 + \alpha) I + \varphi Q - \mu T - \phi T = 0 \tag{6c}$$

Solving (5) – (8) simultaneously and simplifying, we have

$$E_0 = \left[\frac{\Lambda f g + g\sigma\varepsilon + \phi\varphi\varepsilon}{\mu g f + c_1\varphi\mu + g c_1\mu_1}, \frac{\Lambda g c_1 + g c_1\varepsilon + g\mu\varepsilon}{\mu g f + c_1\varphi\mu + g c_1\mu_1}, 0, \frac{\Lambda\varphi c_1 + \varphi c_1\varepsilon + \mu\varphi\varepsilon}{\mu g f + c_1\varphi\mu + g c_1\mu_1}\right] \tag{6d}$$

where $f = \sigma + \varphi + \mu_1$, $g = \phi + \mu$, $h = \mu + \alpha + d_1 + c_2$

In order to examine the stability of the DFE, we first compute the effective reproduction number, R_e. The effective reproduction number is defined in the presence of a control measure whereas the basic reproduction number denoted by $R_0$is defined in the absence of controls. The basic reproduction number is the average number of secondary infections produced when one infected person is introduced into a host population where everyone is susceptible [17]. R_0determines whether or not an infectious disease will spread in a given population. If $R_0 < 1$, the disease will die out and when $R_0 > 1$, the disease will become endemic in the population. In the same vein, the effective reproduction number, R_e, is defined as the average number of new infections generated by a typical infectious individual introduced in a population where contact tracing and quarantine are introduced as measure controls [18]. 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. R_e is computed using next generation method described by Driessche and Watmough [19]. Based on the notations in [19], the effective reproduction number is given by $R_e = \rho(GU^{-1})$; where ρ is the spectral radius of the matrix GU^{-1}.

From the model equations (1), $F = \frac{\beta SI}{N}$ is the rate of new Ebola virus in compartment I while $V = hI$ is the transfer of individuals in and out of the compartment I by all other means except new infection since we have one infected compartment I.

The associated generation matrices G and U can be found from F and V by taking the partial derivatives of F and V with respect to infected compartment I at DFE E_0. That is $G = \frac{\beta S_0}{N_0}$ is the rate of new infection at DFE E_0, $U = h$ is the remaining transition terms at DFE E_0 and $N_0 = S_0 + Q_0 + T_0$. It follows that the effective reproduction number with contact tracing and quarantine measures is given by

$$R_e = \rho(GU^{-1}) = \frac{\beta S_0}{hN_0} = \frac{\beta}{h}\left[\frac{\Lambda fg + g\sigma\varepsilon + \phi\varphi\varepsilon}{\Lambda fg + g\sigma\varepsilon + \phi\varphi\varepsilon + \Lambda g c_1 + g c_1\varepsilon + g\mu\varepsilon + \Lambda\varphi c_1 + \varphi c_1\varepsilon + \mu\varphi\varepsilon}\right] \quad (7)$$

The effective reproduction number with quarantine only $(c_1 = 0, c_2 = 0)$, R_{eq} is given by

$$R_{eq} = \frac{\beta}{h^*}\left[\frac{\Lambda fg + g\sigma\varepsilon + \phi\varphi\varepsilon}{\Lambda fg + g\sigma\varepsilon + \phi\varphi\varepsilon + g\mu\varepsilon + \mu\varphi\varepsilon}\right] \quad (8)$$

where $h^* = \mu + \alpha + d_1$.

While the basic reproduction number R_0 is computed when there is no control measure in the population. That is when $Q = 0, c_1 = 0, c_2 = 0, \sigma = 0, \mu_1 = 0$ and $\varphi = 0$.

Therefore, $R_0 = \frac{\beta}{h^*}$ (9)

From (7), (8) and (9), this inequality $R_e < R_{eq} < R_0$ may hold. This means that quarantine as a control measure may reduce the spread of the virus in the population but not as much as when it combines with contact tracing measure.

We examine the local stability of DFE E_0 using the linearization method at E_0in the following theorem.

Theorem . The disease–free equilibrium state E_0 of the model is locally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$.

Proof.By linearization method, the Jacobian matrix $J_{E_0}$of the model (1) evaluated at $\boldsymbol{E}_0$ is given as

$$J_{E_0} = \begin{bmatrix} -\mu - c_1 & \sigma & -\frac{\beta S_0}{N_0} & \phi \\ c_1 & -f & 0 & 0 \\ 0 & 0 & \frac{\beta S_0}{N_0} - h & 0 \\ 0 & \varphi & \alpha + c_2 & -g \end{bmatrix} \quad (10)$$

The characteristic equation of the Jacobian matrix J_{E_0} (10) is given as

$$\left(\lambda - \frac{\beta S_0}{N_0} + h\right)(\lambda^3 + A\lambda^2 + B\lambda + C) = 0 \quad (11)$$

where $N_0 = S_0 + Q_0 + T_0$, $\lambda$is an eigenvalue of the Jacobian matrix J_{E_0}

$A = g + f + c_1 + \mu$, B= $fg + c_1 g + \mu g + \mu f + c_1(\varphi + \mu_1)$, C = $\mu fg + c_1\mu_1 g + c_1\varphi\mu$

For the DFE E_0 to be locally asymptotically stable, it means that all the eigenvalues of Jacobian matrix (10) will be negative. One of the eigenvalues of the characteristic equation (11), $\lambda = \frac{\beta S_0}{N_0} - h$ is negative if $\frac{\beta S_0}{hN_0} < 1$ where $R_e = \frac{\beta S_0}{hN_0}$ from the definition of R_e in (7). The other three eigenvalues are found by solving the equation,

$$\lambda^3 + A\lambda^2 + B\lambda + C = 0 \quad (12)$$

Using the Routh – Hurwtiz criteria, all roots of the polynomial (12) have negative real part, if

(i) $A > 0, B > 0, C > 0$, (ii) $AB - C > 0$.

Condition (i) is satisfied. For (ii), we have$AB - C = c_1\mu(\varphi + 2\mu_1) + c_1^2(g + \mu_1 + \varphi) + g\mu(g + \mu) + fg(2\mu + g + 2c_1 + f) + c_1 f(\varphi + \mu + \mu_1) + c_1 g(g + \mu + \varphi) + f\mu(\mu + f)$. Therefore, all the eigenvalues of the matrix (10) are negative when $R_e < 1$. Thus, the disease-free equilibrium E_0 is locally asymptotically stable if $R_e < 1$.

4.0 Numerical Simulation

Numerical Simulations of the model (1) are carried out using a set of initial conditions and parameter values. Some of the parameter values used in the numerical simulations are from the literatures and some are assumed. They are listed in table 2. The fourth order Runge – Kutta method coded in MatLab program language is used to simulate the model (1). The following experiments are considered in this study.

i. The reproduction numbers, R_e, R_{eq}, and R_0,
ii. Relationship between the reproduction numbers, R_e, and R_0 against β,
iii. Infected population against time for the contact tracing rates, c_1, and c_2, for susceptible individualsand infected individuals respectively,

iv.Infected population against time for the contact tracing rates for both infected and susceptible individuals, c_1 and c_2.

Table 2: Parameter values of the EVD Model

Parameter	Value (day)$^{-1}$	Source	Parameter	Value (day)$^{-1}$	Source
$S(0)$	4396521	[20]	d_1	0.0301653	,,
$Q(0)$	74	,,	σ	0.047619	,,
$I(0)$	33	,,	c_1	0.06	Estimated
$T(0)$	9	,,	c_2	0.07	,,
β	0.160	[7]	ε	100	,,
α	0.0608	,,	μ_1	0.000005	,,
φ	0.08333	,,	μ	0.00002465753	[21]
ϕ	0.0314862	,,	Λ	422	[22]

The following numerical experiments are carried out in this study:

Experiment 1. The reproduction numbers, R_e, R_{eq}, and R_0.

Using the parameter values in table 2, wecompute the reproduction numbers,R_e, R_{eq}, and R_0 in equations (7), (8) and (9) respectively. This gives$R_0 = 1.75843$, $R_{eq} = 1.75820$, and $R_e = 0.37221$.

Experiment 2. Relationship between the reproduction numbers, R_e, and R_0 against β.

In this experiment, the relationship between the effective reproduction number, R_e, basic reproduction number, R_0, and disease transmission rate, β is examined. Figure 1 is the graphical representation of the relationship.

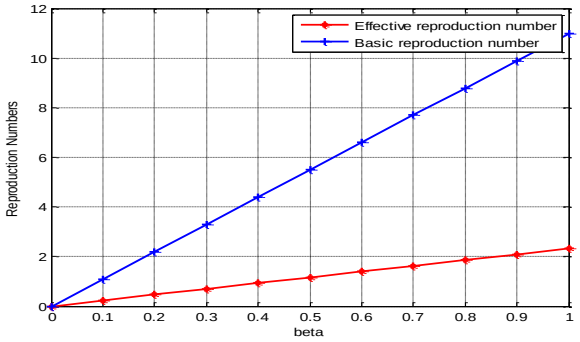

Figure 2: Simulation result showing the relationship between the reproduction numbers R_e and R_0 and disease transmission rate β.

Experiment 3: Infected population against time for the contact tracing rate for susceptible individuals, c_1, and the infected individuals, c_2.

In this experiment, we study the effect of tracing the contact of susceptible individuals that are exposed to EVD and the infected individuals on infected population. The graphsare presented in figures 3(a) and 3(b).

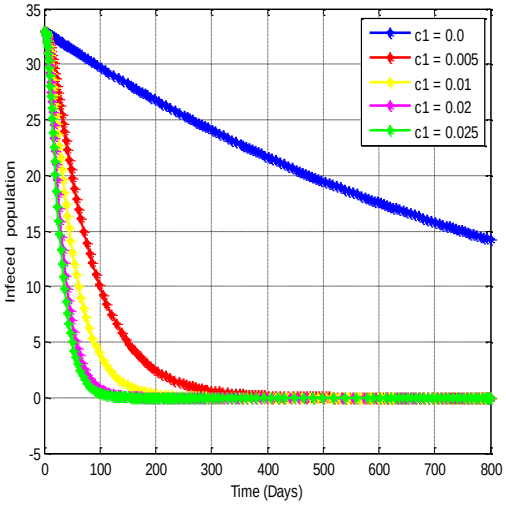

Figure 3(a)

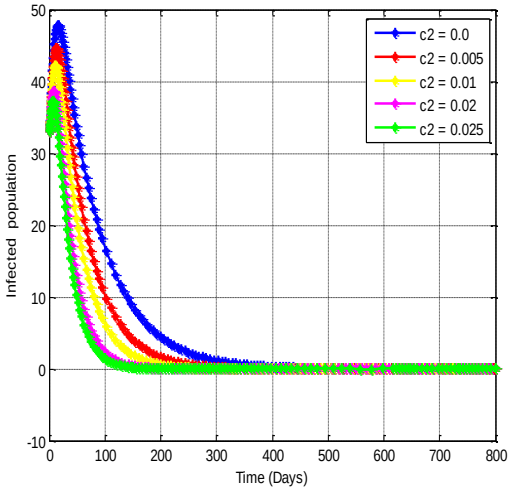

Figure 3(b)

Simulation result showing the effect of contact tracing rates c_1 and $c_2$on infected population.

Experiment 4: Infected population against time for the effect of contact tracing rates for both infected and susceptible individuals, c_1 and c_2.

The effect of varying the contact tracing rates, c_1 and c_2 on the infected population and the different forms of implementing them are considered in this experiment. These different forms include: (i) tracing only the infected individual $(c_1 = 0.0, c_2 = 0.07)$, (ii) tracing only the susceptible individuals that are exposed $(c_1 = 0.06, c_2 = 0.0)$ and (iii) tracing both simultaneously $(c_1 = 0.06, c_2 = 0.07)$. The graphs are presented in figures 4(a) and 4(b).

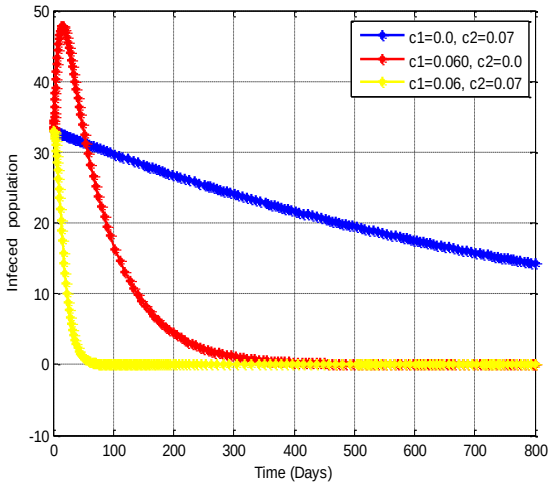

Figure 4(a)

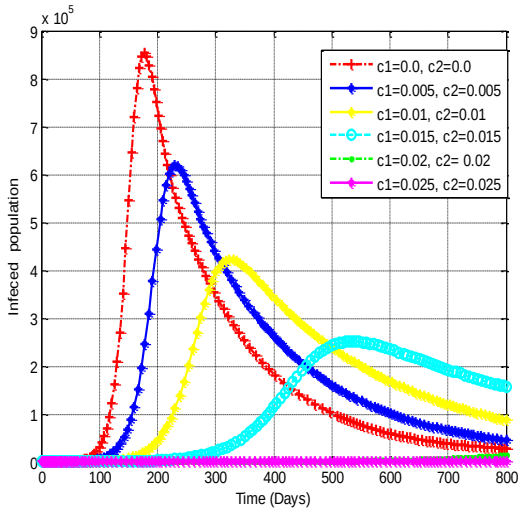

Figure 4(b)

Simulation result showing the effect of contact tracing rates, c_1 and c_2 on infected population.

5.0 Discussion

Experiment 1 shows that $R_e < 1$ and also verifies the inequality that $R_e < R_{eq} < R_0$. The reproduction numbers, R_e, and R_0, in this study are comparable to the reproduction numbers in [6]and [7] for Liberia Ebola epidemics. This implies that the EVD will be eradicated in the population when contact tracing and quarantine measures are implemented together.

Experiment2 considers the reproduction numbers R_e and R_0 against transmission rate β. It is observed that as transmission rate β increase, the reproduction numbers, R_0, and R_e, increase (figure 2) but at the low rate due to the presence of contact tracing and quarantine measures. This means that implementation of quarantine and contact tracing measures reduces the spread of disease faster.

The importance of tracing the susceptible individuals that are exposed is considered in experiment 3. It shows that as the rate of tracing the susceptible individuals that are exposed is increases, the number of infected population is remarkably reduce and this leads to elimination of the disease on time (figure 3(a)).

The effect of tracing the infected individuals is described in figure 3(b). This demonstrates that the number of infected population decreases and tends to zero as time progresses. However, tracing the susceptible individuals that are exposed give a better result than tracing the infected individuals (figures 3(a) and 3(b)). In other words, the disease will be eradicated in the population within a short period of time if susceptible individuals that are exposed are traced immediately and quarantined for a length of time not less than the incubation period of the EVD.

Implementing any of these different forms of contact tracing in experiment 4 will reduce the number of infected individuals in the population but the rate of reduction will take a longer time when only infected individuals are traced (figure 4(a)). In a resource constrained setting, where there is limitation in terms of manpower, money, material and time to trace both infected and susceptible individuals, it may be more beneficiary to trace only the susceptible individuals that are exposed. However, for the disease like EVD that is a point source epidemics, tracing the infected individuals and susceptible individuals that are exposed simultaneously is very crucial in order to limit the spread of EVD. This may translates to early eradication of EVD. Furthermore, figure 4(b)illustrates the effect of active contact tracing of both infected and susceptible individuals on the infected population. It shows a remarkable reduction in the number of infected individuals as the rates of contact tracing increase. This supports the earlier finding (figure 4(a)) that tracing both infected and susceptible individuals that are exposed reduces the number of infected population.

6.0 Conclusion

A deterministic model for the dynamics of EVD is presented and analyzed in this study. The model incorporates contact tracing and quarantine as control measures in order to assess their impact on the EVD dynamics. A feasible region where the

model is epidemiologically and mathematically well posed is shown. A threshold quantity R_e that governs the spread of disease when the control measures are in place is computed using the next generation method by Van den Driessche and Watmough [19]. The impact of the control measures are shown for the reproduction numbers, R_0, R_{eq} and R_e and satisfied the inequality $R_e < R_{eq} < R_0$. The comparison shows that contact tracing and quarantine minimize the number of infected persons in the population and hence reduce the spread of EVD.

Furthermore, the existence and stability of the disease-free state is established when the effective reproduction number is less than unity. Additionally, numerical simulation of the model is carried out to examine the effect of varying certain parameters on the dynamics of the EVD. The result shows that the combined implementation of contact tracing and quarantine measures have most significant impact in eradicating the EVD in the population.

7.0 References

[1] King, J.W., Cunha, B.A., Khan, A.A., Kerkering, T.M., Malik, R. and Talavera, F. (2014). Ebola Virus Infection Clinical Presentation. Medscape Reference: Drugs, Diseases and Procedures. Retrieved on 4th September 2014 from www.emedicine.medscape.com/artcle/216288-overview.

[2] WHO (2016). Ebola Data and Statistics. apps.who.int/gho/data/view.ebola-sitrep.ebola-summary-latest.

[3] WHO (2014). 2014 Ebola Virus Disease (EVD) Outbreak in West Africa, 8th April 2014.

[4] ECDC (2014). Outbreak of Ebola virus disease in West Africa, European Centre for Disease Prevention Control. 8 April 2014. Stockholm.

[5] Arreola, R., McDuffy, D.D., Mejia, M.B. and Oliver, A.I. (1999). The Ebola Virus: Factors Affecting the Dynamics of the Disease. Mathematical and Theoretical Biology Institue. BU-1519-M: 353-374.

[6] Legrand, J., Grais, R.F., Boelle, P.Y., Valleron, A. J. and Flahault, A. (2007). Understanding the Dynamics of Ebola Epidemics. Epidemiology and Infection. **135**(4): 610-621.

[7] Rivers, C. M., Lofgren, E. T., Marathe, M., Eubank, S. and Lewis, B. L. (2014). Modelling the Impact of Interventions on an Epidemic of Ebola in Sierra Leone and Liberia. PLOS Current Outbreaks. Ed 1.

[8] Browine, C., Huo, X., Magal, P., Seydi, M., Seydi, O. and Webb, G. (2015). A Model of the 2014 Epidemic in West Africa with Contact Tracing. PLOS Currents Outbreaks, Ed 1.

[9] Li, Z., Teng, Z., Feng, X., Li, Y., and Zhang, H. (2015). Dynamical Analysis of an SEIT Epidemic Model with Application to Ebola Virus Transmission in Guinea. Computational and Mathematical Methods in Medicine. 2015, article ID 582625, 11 pages.

[10] Mubayi, A., Zaleta, C. K., Martcheva, M. and Castillo-Chavez, C. (2010). A Cost-Based Comparison of Quarantine Strategies for New Emerging Diseases. Mathematical Biosciences and Engineering. **7**(3): 687 - 717.

[11] Hsieh, Y., Wang, Y., Arazoza, H. and Lounes, R. (2010). Modeling Secondary Level of HIV ContactTracing: Its Impact on HIV Intervention in Cuba. BMC Infectious Diseases. **10** (194).

[12] Begun, M., Newall, A.T., Marks, G.B. and Wood, J.G. (2013). Contact Tracing of Tuberculosis: A Systematic Review of Transmission Modelling Studies. PLOS One.**8**(9): e72470.

[13] Atangana, A., and Goufo, E.F.D. (2014). On the Mathematical Analysis of Ebola Hemorrhagic Fever: Deathly Infection Disease in West African Countries. BioMed Research International.**2014**, article ID 261383, 7pp.

[14] Kalu, A.U., Akuagwu, N.A. and Agwu, I.A. (2016). A Mathematical Model for the Control of the Spread of Ebola Virus Disease in West Africa – A Disease-free Equilibrium Approach. British Journal of Mathematical and Computer Science. **12**(5):1-9.

[15] Lewnard, A. J., Ndeffo-Mbah, M.L., Alfaro-Murillo, J., Altice, F.L., Bawo, L., Nyenswah, T.G. and Galvani, A. P. (2014). Dynamics and Control of Ebola Virus Transmission in Montserrado, Liberia: A Mathematical Modelling Analysis. THE LANCET Infectious Diseases.**14**(12):1189 – 1195.

[16] Hethcote, H. W. (2000). The Mathematics of Infectious Diseases. SIAM REVIEW. **42**(4): 599-653.

[17] Benyah, F. (2007). Epidemiological Modeling and Analysis. The 13th Edward A. Bouchet / Abdusa Salam workshop, University of Ghana Legon, Accra. 9 -13th July, 2007.

[18] Shaban, N., and Mofi, H. (2014). Modelling the Impact of Vaccination and Screening on the Dynamics of Human Papillomavirus Infection. International Journal of Mathematical Analysis. 8(**9)**, 441 – 454.

[19] Driessche, P. Van den, and Watmough, J. (2002). Reproduction Numbers and Sub-Thresholds Endemic Equilibrium for Compartmental Models of Disease Transmission. Mathematical Bioscience. **180** (2002): 29 – 48.

[20] Althaus, C.L., Low, N., Musa, E.O., Shuaib, F. and Gsteiger, S. (2015). Ebola Virus Disease Outbreak in Nigeria: Transmission Dynamics and Rapid Control. Epidemics. **11**(2015): 80-84.

[21] World Bank Group (2016). Death Rate for Liberia. http:\\data.world.org\indicator\SP.DYN.CDRT.IN.

[22] UNICEF(2016). At a Glance: Liberia. www.unicef.org\infobycountry\liberia_statistics.html