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EBOLA VIRUS DISEASE SPREAD AND CONTROL IN WEST AFRICA

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Abstract: **This research studies mathematical model for the dynamics of Ebola virus disease incorporating quarantine and public campaign as controls. The model was developed as a system of six (6) non-linear ordinary differential equations. The population is divided into six (6) compartments, namely: Susceptible S(t), Latent L(t), Infectious I(t), Quarantined $Q(t)$ Recovered R(t), and Dead D(t). We obtained and analysed the equilibrium states of the model for stability .The effective Basic Reproduction Number R_{eff} was obtained, which suggests that if $R_{eff} \leq 1$, the Disease free equilibrium state is locally asymptotically stable, meaning that the disease dies out in a population, or if $R_{eff} \geq 1$, the Disease free equilibrium state is unstable meaning that disease persists in a population. The numerical results are clearly displayed, to see the effects of controls on the disease. The control parameters are quarantine (φ), rate of effectiveness of public enlightenment campaign (ξ), treatment rate (τ) and rate of proper burial of infectious dead (ς).**

Keywords: **Ebola, Virus, Disease, Spread, Control, West Africa.**

1. INTRODUCTION

Ebola virus disease (EVD) is an acute viral hemorrhagic fever that is highly contagious, named after a river in the Democratic Republic of the Congo (formerly Zaire) where it was first identified in 1976 (CDC, 2004). It is from a family of RNA (ribonucleic acid) virus called Filovirus.

Ebola virus disease is transmitted by physical contact with secretions, tissues or semen and body fluids from dead or alive infected persons, [CDC,(2003a) and WHO, (2003a)], Nosocomial transmission (transmission from patients within hospital settings) has been typical as patients are often treated by unprepared hospital personnel (there is a need to observe barrier nursing techniques). Individuals exposed to the virus who become infectious do so after a mean incubation period of $1-21$ days (Breman *et al* 1997). Ebola virus disease (EVD) is characterized by initial flu-like symptoms which rapidly progress to vomiting, diarrhoea, rash, and internal and external bleeding. Once inside the body, the virus begins to attack the blood and liver cells (cells of the immune system that normally protect the body against infection). As the fever progresses, the virus destroy vital organs such as the liver and the kidneys, leading to massive bleeding and destruction of internal tissues, shocks and respiratory arrest soon follow, then death (Encarta Encyclopaedia, 2009). Most infected persons die within 10 days of their initial infection (Birmingham and Cooney 2002) , with $50\%-90\%$ mortality (WHO, 2003b).

Twelve outbreaks of Ebola virus disease were reported in Congo, Sudan, Gabon, and Uganda in 2003. CDC & WHO 2003b), with two different strains of the Ebola virus (Ebola-Zaire and the Ebola-Sudan) reported in those regions. An extensive search now reveals the reservoir of the Ebola virus as fruit bat.

Research shows that 2014 – May 2015 witnessed the greatest outbreaks, with reports of outbreaks from nine countries namely, Guinea, Liberia, Sierra Leone, Senegal, Mali, Nigeria,, Spain, United Kingdom and United States with a total of 26,724 cases of infections and 11065 deaths, (WHO, 2015). Six of these countries , Mali, Nigeria, Senegal, Spain, the United Kingdom and the United States who have been declared Ebola virus disease free have previously reported a case or cases imported from a country with widespread and intense transmission (WHO, 2015).

September 20th 2022, the Uganda Ministry of Health, WHO and AFRO, confirmed an outbreak of Ebola virus disease in Mubende District, Uganda, the strain confirmed was Sudan virus disease (SVD) and a fatal case. It was a case of a 24-year-old man, residing in Ngabano village, a sub-county of Madudu in the District of Mubende. On September 11th 2022, it was noticed that he had experienced the following symptoms like high fever, abdominal pain, diarrhoea, and vomiting of blood. On September 17th 2022, samples were collected and of which the laboratory result confirmed he had SVD on September 19th 2022, unfortunately, on that same day, the patient died after five days of being hospitalised. This case has been the fifth case of Ebola outbreak in Uganda. In total,142 cases were confirmed, 87 recovered, and 55 died (CFR: 39%). Additionally, other probable cases were 22 deaths reported which were those who died before their samples were taken (overall CFR: 47%). Also among some healthcare workers, 19 were infected and 7 died. Up to 21 days, about 4000 contacts had been followed up, (**WHO AFRO News**).

9 Ugandan districts had been affected by the outbreak namely, Mubende, Kagadi, Kyegegwa, Masaka, Jinja, Kampala, Wakiso, Bunyangabu, and Kassanda.

Equatorial Guinea's Ministry of Health has confirmed eight more cases of Marburg, bringing the number of confirmed cases to nine since the outbreak of the viral haemorrhagic fever was declared on February 13, 2023.

2. LITERATURE REVIEW

Kermack–Mackendrick (1927) contributed a great deal to modeling infectious disease categories and transmission. They named their model the SIR model.

Few people have shown considerable interest in the transmission dynamics of Ebola fever Disease. For example: (Chowell *et al*, 2004), Althaus (2014), Nishiura (2014). Very few mathematical models have been developed to study its transmission dynamics. Chowell *et al* (2015) reviewed potential mechanisms that could explain the non-exponential growth dynamics of disease spread, and concluded that factors related to the epidemiology of EVD, behaviour changes of the at-risk population, and success of control interventions could be involved alone or in combination. Their observations suggest that quantifying the contribution of each of these factors could help guide expectations of final epidemic size and mitigation efforts in the current and future EVD outbreaks. Astacio *et al* (1996) used the S-1-R and S-E-1-R models, which was a simulation of two Ebola outbreaks: the 1976 outbreak in Yambuku, Zaire and the 1995 outbreak in Kikwit,

Zaire. The dynamics of these models were determined by the per-capita death rate of infected individuals and the per-capita effective contact rate of an individual contracting the disease. The basic reproductive number, *R,* determines the infectiousness of the disease. They claimed that Ebola is not as infectious as previously postulated and that the results of their simulations will equip scientists in future outbreaks with information that may enable them to minimize potential deaths. Zach (2012) created an SIR model of Ebola Fever titled 'A Mathematical look at Ebola Virus' to describe how the Ebola virus could potentially ravage a population. This he did by describing the rate of change of the susceptible population over time by multiplying S(t), I(t) and the constant "a". The population of the susceptible group will be reduced as the infected come into contact with the susceptible.

The population of the infected class changes in two ways. One, individuals leave the susceptible class and join the infected class, adding to the total population of infected people. And two, individuals leave the infected class and join the recovered class, reducing the infected population. Let's say the rate at which the infected join the recovery group is the constant "b". To build a model for the change in population as it changes with time one can multiply "a", S(t) and I(t) and subtract "b" multiplied with I(t). And finally the recovered population can be created. The only factor that changes the population of the recovery group is the addition of the newly recovered infected. This can be shown as the multiplication of "b" with I(t), the infected will die at a certain rate. In the SIR model, the recovery group obtains immunity from the disease after they become infected. In the Ebola-Zaire model the recovered class will not remain immune to the infection.

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He modelled the Ebola-Zaire virus using four separate differential equations. He used a numerical solver to plot the solution and showed the virus wouldn't spread over thousands of people in such a particular manner even though the virus is extremely contagious. Abdulrahman *et al* (2014) developed a model on the stability analysis of Disease-Free Equilibrium State for the transmission dynamics and control of Ebola. They obtained the basic reproduction number and analysed the disease-free equilibrium state for stability. Their finding reveal that once Ebola Fever disease is introduced into a population, the disease morbidity and mortality continue to rise, until high surveillance is put in place to quarantine and treat the infected individuals and proper burial for those that died due to the disease.

Abah *et al* (2015) developed a model to study the transmission dynamics of Ebola Fever disease. They carried out the stability analysis of the disease free equilibrium state of the model and used it to determine the condition for stability of the disease free equilibrium state, which is locally asymptotically stable when τ the treatment rate must be greater than both μ natural death rate and δ_2 disease induced death rate, for the disease free equilibrium state to be locally asymptotically stable and so the disease will die out. If otherwise $\tau < \mu + \delta_2$ that is, if τ treatment rate is less than both μ the natural death rate and δ_2 disease induced death rate, the disease free equilibrium state will be unstable and this could result in an outbreak of Ebola Fever epidemics. Sylvie et al (2022) presented a paper on the global stability analysis of the steady states of a model of EVD with a nonlinear incidence function. They presented through suitably chosen Lyapunov functions the global stability of equilibria. The nonlinear incidence function was chosen to represent the influence of human behaviour on EVD evolution. Data from Liberia and Sierra Leone were used in the fitting process whose results show that the model closely describes the evolution of EVD with human behaviour.

3. MODEL FORMULATION

A mathematical model of the dynamics of Ebola Fever incorporating Quarantine and public campaign as controls was formulated. The population is divided into six (6) compartments, namely: Susceptible S(t), Latent L(t), Infectious I(t), Quarantined $Q(t)$ Recovered R(t), and Dead D(t). The Total population is

$$N(t) = S(t) + L(t) + I(t) + Q(t) + R(t) + D(t).$$

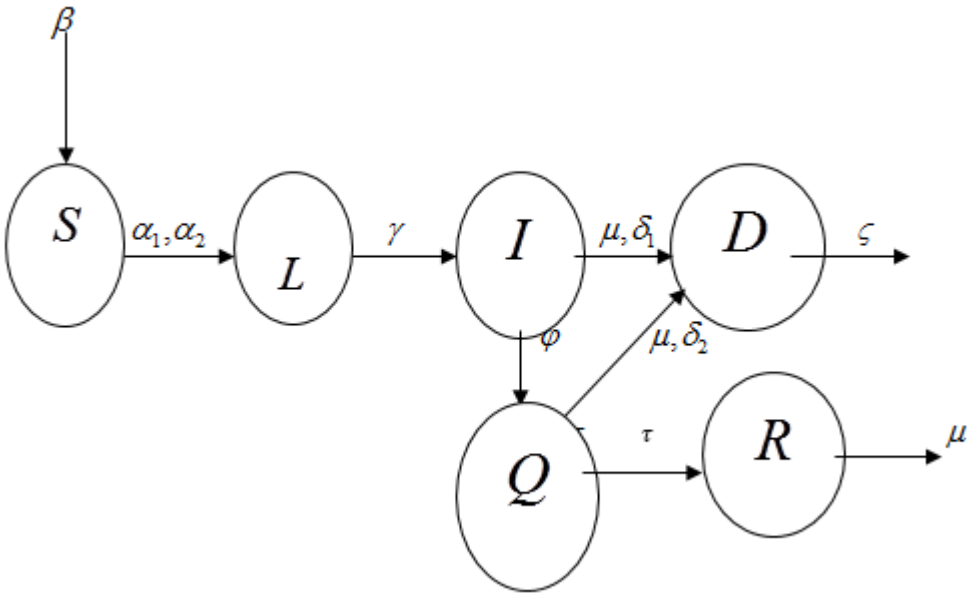

Figure 3.1: Schematic diagram of Ebola Virus Disease transmission and Control model.

Ebola virus disease models are usually comprised of individuals who have not had effective contact with the virus. These individuals are referred to as Susceptible S (t). When susceptible individuals come into contact with infectious individuals, they get infected but do not become infectious immediately, so they move into a class known as Latent L(t).

After the latency period, these individuals become infectious, meaning they can now spread Ebola virus disease. And so they move into a class known as Infectious I(t).

To prevent the spread of the Ebola virus disease, these individuals are isolated into a class known as Quarantine Q (t) for treatment. During the treatment period, some of the individuals in Quarantine recover permanently and now move into a class known as Recovered R(t).

Some individuals who die as a result of Ebola virus disease move from I(t) and Q(t) into a class known as Dead D(t). This class exists because they are capable of spreading Ebola Virus Disease through unsafe burial practices.

$S(t)$ are individuals who have not had effective contact with Ebola virus disease but are prone (susceptible) to Ebola fever through contact with the $I(t)$ and $D(t)$ at the rate α_1 and $\alpha_2(1-\varsigma)$ where α_1 is the effective contact rate between $S(t)$ and $I(t)$, $\alpha_2(1-\varsigma)$ is the effective contact rate between $S(t)$ and $D(t)$. They are generated through a natural birth rate β from $S(t)$, $L(t)$ and $R(t)$ are reduced by a natural death rate μ .

$L(t)$ are individuals who through contact with the $I(t)$ and $D(t)$, got infected at the rate α_1 and $\alpha_2(1-\varsigma)$ where α_1 is the effective contact rate between $S(t)$ and $I(t)$. $\alpha_2(1-\varsigma)$ is the effective contact rate between $S(t)$ and $D(t)$. They are still in incubation period since they have not yet manifested the symptoms of Ebola virus disease. After the twenty one (21) days incubation period, they may become infectious if they do not possess strong immunity to fight off the disease; they then join $I(t)$ at a progression rate γ . They are reduced at a death rate μ .

$I(t)$ are the individuals that are infected with Ebola Fever. They are generated through a progression rate γ from $L(t)$ to $I(t)$. They are reduced due to μ , q and δ_1, where μ is the natural death rate, q is the rate of quarantine and δ_1, the disease induced death rate.

$Q(t)$ are the individuals that are isolated (quarantined) from $I(t)$ through q, where q is the rate of quarantine. They are reduced through τ , μ and δ_2, where τ is the treatment rate, μ is the natural death rate, and δ_2 is disease induced death rate of $Q(t)$.

$R(t)$ are the individuals that have recovered and have acquired permanent immunity through a treatment rate τ . They are reduced by a natural death rate μ .

$D(t)$ is the compartment for those who are dead through infection, and are generated from both classes $I(t)$ and $Q(t)$ respectively through μ , δ_1 and δ_2 respectively, where μ is the natural death rate of $I(t)$ and $Q(t)$ alone. δ_1 and δ_2 , the disease induced death rate of $I(t)$ and $Q(t)$ respectively.

3.1 Assumptions

The following assumptions were made to formulate the model:

1. The mixing of people is homogeneous, meaning that all individuals have equal chance of getting infected if they come in adequate contact with infectious individuals.

2. Those in $S(t)$ get infected through contact with $I(t)$ and $D(t)$.

3. $L(t)$ are infected but not yet infectious, since they get infectious, only when they are symptomatic.

4. The isolation of $I(t)$ to $Q(t)$ cause the spread of Ebola Fever to be very low due to treatment rate τ .

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5. $\delta_2 < \delta_1$ due to the treatment of $Q(t)$ at the rate τ .

6. There is no birth into the $I(t)$ and $Q(t)$ classes because babies of those infected hardly survive the disease. (maternity ward)

7. If Persons in $Q(t)$ recover, they recover permanently due to the treatment rate τ .

8. The dead class $D(t)$ is not the compartment for the total dead, but for the disease induced death from class $I(t)$ and $Q(t)$

3.1.1 Variables

The variables are defined as follows:

$S(t)$ Susceptible class at time t

$L(t)$ Latent class at time t

$I(t)$ Infectious class at time t

$D(t)$ Dead from $I(t)$

$Q(t)$ Quarantined class at time t

$R(t)$ Recovered class at time t

3.1.2 Parameters

The parameters are defined as follows:

β birth rate

μ death rate

δ_1 disease induced death rate of $I(t)$

δ_2 disease induced death rate of Q(t)

α_1 effective contact rate between $I(t)$ and S(t)

$\alpha_2(1-\varsigma)$ effective contact rate between D(t) and S(t)

γ progression rate from L to $I(t)$

φ rate of quarantine

τ treatment rate

ξ the rate of effectiveness of public campaign

ς rate at which the dead is decontaminated and buried

$(1-\xi)$ proportion that ignored public campaign who can still be infected with EBF.

The schematic diagram is described by a system of ordinary differential equations

(3.1) - (3.6):

$$\frac{dS}{dt} = \beta(S+L+R) - \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\varsigma)D}{N}\right)(1-\xi)S - \mu S \quad (3.1)$$

$$\frac{dL}{dt} = \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\varsigma)D}{N}\right)(1-\xi)S - (\gamma+\mu)L \quad (3.2)$$

$$\frac{dI}{dt} = \gamma L - (\varphi+\mu+\delta_1)I \quad (3.3)$$

$$\frac{dQ}{dt} = \varphi I - (\tau+\mu+\delta_2)Q \quad (3.4)$$

$$\frac{dR}{dt} = \tau Q - \mu R \quad (3.5)$$

$$\frac{dD}{dt} = (\mu+\delta_1)I + (\mu+\delta_2)Q - \varsigma D \quad (3.6)$$

Where,

$$N(t) = S(t) + L(t) + I(t) + Q(t) + R(t) \quad (3.7)$$

So that the total population which is changing at the rate $\frac{dN(t)}{dt}$, is given by

$$\frac{dN(t)}{dt} = \beta N - \mu(S+L+R) \quad (3.8)$$

The model (3.1) to (3.8) is epidemiologically and mathematically well posed in the domain Ω with the initial conditions.

Let $\Omega = (S,\ L,\ I,\ Q,\ R,\ D) \in R^6$ (3.9)

$S \geq 0,\quad L \geq 0,\quad I \geq 0,\quad Q \geq 0,\quad R \geq 0,\quad D \geq 0$

$S + L + I + Q + R \leq N$

3.2 Properties of the Model

To analyse the model (3.1) to (3.8) we consider the following theorem

Theorem 3.1: The solutions of the model equations (3.1) to (3.8) are positive for all time $t \geq 0$ provided that the initial conditions are positive.

Proof:

Employing the given assumptions, that all initial conditions are positive, i.e.

$S(0) > 0,\quad L(0) > 0,\quad I(0 > 0,\quad Q(0) > 0,\quad R(0) > 0,\quad$ and $\quad D(0) > 0$

We have by contradiction (Huo, Dang and Li, 2010; Bolarin and Adeboye, 2011) that the solutions of (3.1) to (3.8) are positive if we assume for a contradiction that there exists first time,

$t_1 : S(t_1) = 0$

and $S(t) > 0, \quad L(t) > 0, \quad I(t) > 0, \quad Q(t) > 0, \quad R(t) > 0, \quad D(t) > 0$ (3.10)

$0 < t < t_1$

or there exists

$t_2 : L(t_2) = 0$

and $S(t) > 0, \quad L(t) > 0, \quad I(t) > 0, \quad Q(t) > 0, \quad R(t) > 0, \quad D(t) > 0$ (3.11)

$0 < t < t_2$

or there exists

$t_3 : I(t_3) = 0$

and $S(t) > 0, \quad L(t) > 0, \quad I(t) > 0, \quad Q(t) > 0, \quad R(t) > 0, \quad D(t) > 0$ (3.12)

$0 < t < t_3$

or there exists

$t_4 : Q(t_4) = 0$

and $S(t) > 0, \quad L(t) > 0, \quad I(t) > 0, \quad Q(t) > 0, \quad R(t) > 0, \quad D(t) > 0$ (3.13)

$0 < t < t_4$

or there exists

$t_5 : R(t_5) = 0$

and $S(t) > 0, \quad L(t) > 0, \quad I(t) > 0, \quad Q(t) > 0, \quad R(t) > 0, \quad D(t) > 0$ (3.14)

$0 < t < t_5$

or there exists

$t_6 : D(t_6) = 0$

and $S(t) > 0, \quad L(t) > 0, \quad I(t) > 0, \quad Q(t) > 0, \quad R(t) > 0, \quad D(t) > 0$ (3.15)

$0 < t < t_6$

Now, in the case where

$S(t) = 0$ (3.16)

gives,

$$\frac{dS(t_1)}{dt} = \lim_{t=t_1} \frac{S(t_1) - S(t)}{t_1 - t} < 0 \quad (3.17)$$

and similarly,

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gives

$$\frac{dL(t_2)}{dt} < 0, \quad \frac{dI(t_3)}{dt} < 0, \quad \frac{dQ(t_4)}{dt} < 0, \quad \frac{dR(t_5)}{dt} < 0, \quad \frac{dD(t_6)}{dt} < 0 \tag{3.18}$$

However from equations (3.1) to (3.6) gives,

$$\frac{dS(t_1)}{dt} = \beta(S(t_1) + L + R) - \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\varsigma)D}{N}\right)(1-\xi)S(t_1) - \mu S(t_1) \tag{3.19}$$

i.e.

$$S^i(t_1) = \beta(L + R) > 0 \tag{3.20}$$

Which contradicts (3.17). Therefore, $S(t_1) \neq 0$, and S will remain positive for all t.

Similarly, for the remaining variables gives

$$L^i(t_2) = \left(\frac{\alpha_1 I}{N} + \frac{\alpha_2(1-\varsigma)D}{N}\right)(1-\xi)S > 0 \tag{3.21}$$

$$I^i(t_3) = \gamma L > 0 \tag{3.22}$$

$$Q^i(t_4) = \varphi I > 0 \tag{3.23}$$

$$R^i(t_5) = \tau Q > 0 \tag{3.24}$$

$$D^i = (\mu + \delta_1)I + (\mu + \delta_2)Q > 0 \tag{3.25}$$

These are contradictions of what was supposed for each of the variables, meaning that $L(t_2) \neq 0$, $I(t_3) \neq 0$, $Q(t_4) \neq 0$, $R(t_5) \neq 0$, and $D(t_6) \neq 0$. Hence, S, L, I, Q, R and D remain positive for all t. By this, it is showed that all the solutions of (3.1) to (3.6) are in R^6, provided that the initial conditions are positive. Thus the feasible region is positively-invariant. It is hence, sufficient to consider the dynamics of the model (3.1) to (3.6) in the region Ω.

3.3 The Basic reproduction number R_0 and the effective basic reproduction number, R_{eff}.

One of the most importance concerns about any infectious disease is its ability to invade a population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of disease. These models usually have threshold parameter, known as the basic reproduction number, R_0 such that if $R_0 < 1$, then the disease free equilibrium (DFE) is locally asymptotically stable, and the disease cannot invade a population, but if $R_0 > 1$, then the disease free equilibrium (DFE) is unstable and invasion is always possible The basic reproduction number R_0 is a measure of the potential for disease spread in a population, and it's inarguably (undeniably) 'one of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory' (Heesterbeek and Dietz, 1996). It represents the average number of secondary cases generated by an infected individual if introduced into a susceptible population with no immunity to the disease in the absence of interventions to control the infection, If $R_0 < 1$, then on average, an infected individual produces less than one newly infected individual over the course of the infection period. In that case the infection may die out on the long run. On the other hand, if $R_0 > 1$ each infected individual (primary case) produces, on average more than one new (secondary) infection, the infection will be able to spread in a population. A large value of R_0 may indicate the possibility of major epidemic, Abdulrhaman, (2014).

Similarly, the effective basic reproduction number R_{eff} represent the average number of secondary cases generated by an infected individual if introduced into a susceptible population where control measures are applied, Abdulrhaman, (2014). We now use the approach of Diekmann and Heesterbeek (2000) as analysed by Van de Driessche and Watmough (2002) known as the spectral radius. It is a better and a widely used approach in finding R_0 since it reflects its biological meaning. This approach is employed in finding the effective basic reproduction number, R_{eff} of the system (3.1) to (3.6) which is the spectra radius (ρ) of the next generation matrix, K ,

i.e.

$$R_{eff} = \rho K, \qquad \text{where } K = FV^{-1}$$

F and V are obtained from the Jacobian (3.64), about the disease-free equilibrium. F represents the matrix for the new infection terms and V the matrix for the transition terms. The matrices F and V are formed from the coefficient of the infected classes. The four infected classes are Latent L , Infectious I , Quarantine Q , and the Dead D of the model (3.1) to (3.6).

$$F = \begin{pmatrix} 0 & \frac{S}{N}\alpha_1(1-\xi) & 0 & \frac{S}{N}\alpha_2(1-\xi) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

For easy simplification, let

$$M_1 = \frac{S}{N}\alpha_1(1-\xi) \text{ and } M_2 = \frac{S}{N}\alpha_2(1-\xi)$$

and so the matrix F becomes

$$F = \begin{pmatrix} 0 & M_1 & 0 & M_2 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = -\begin{pmatrix} -(\gamma+\mu) & 0 & 0 & 0 \\ \gamma & (\varphi+\mu+\delta_1) & 0 & 0 \\ 0 & \varphi & -(\tau+\mu+\delta_2) & 0 \\ 0 & \mu+\delta_1 & \mu+\delta_2 & -\varsigma \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & M_1 & 0 & M_2 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \begin{bmatrix} \frac{1}{\gamma+\mu} & 0 & 0 & 0 \\ \frac{\gamma}{(\gamma+\mu)(\varphi+\mu+\delta_1)} & \frac{1}{\varphi+\mu+\delta_1} & 0 & 0 \\ -\frac{\gamma\varphi}{(\gamma+\mu)(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)} & -\frac{\varphi}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)} & \frac{1}{(\tau+\mu+\delta_2)} & 0 \\ \frac{\gamma((\tau+\mu+\delta_2)(\mu+\delta_1)-\varphi(\mu+\delta_2))}{(\gamma+\mu)(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma} & \frac{(\mu+\delta_1)(\tau+\mu+\delta_2)-\varphi(\mu+\delta_2)}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma} & \frac{(\mu+\delta_2)}{(\tau+\mu+\delta_2)\varsigma} & \frac{1}{\varsigma} \end{bmatrix} \tag{3.26}$$

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is given by

$$FV^{-1}=\begin{bmatrix} \frac{\gamma M_1}{(\varphi+\mu+\delta_1)}+\frac{\gamma M_2((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\varphi(\mu+\delta_2))}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma} & M_2((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\frac{\varphi(\mu+\delta_2)}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma}+\frac{M_1}{B}) & \frac{\gamma M_2}{(\varphi+\mu+\delta_1)\varsigma} & \frac{M_2}{\varsigma} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (3.27)$$

We compute the eigenvalues to determine the effective reproduction number R_{eff} by taking the spectral radius (dominant eigenvalue) of the matrix FV^{-1}. This gives (3.27),

$$(J)=\begin{bmatrix} \frac{\gamma M_1}{(\varphi+\mu+\delta_1)}+\frac{\gamma M_2((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\varphi(\mu+\delta_2))}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma}-\lambda & M_2((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\frac{\varphi(\mu+\delta_2)}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma}+\frac{M_1}{B}) & \frac{\gamma M_2}{(\varphi+\mu+\delta_1)\varsigma} & \frac{M_2}{\varsigma} \\ 0 & 0-\lambda & 0 & 0 \\ 0 & 0 & 0-\lambda & 0 \\ 0 & 0 & 0 & 0-\lambda \end{bmatrix}=0 \quad (3.28)$$

Eigenvalues λ_i where $i=1,2,3,4$ were obtained and are given by:

$$\lambda_1=\frac{\gamma\alpha_1(1-\xi)}{(\varphi+\mu+\delta_1)}+\frac{\gamma\alpha_2(1-\xi)((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\varphi(\mu+\delta_2))}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma}$$

Since λ_1 is the dominant eigenvalue it therefore follows that the effective basic reproduction number R_{eff} is given by:

$$\lambda_1=R_{eff}=\frac{\gamma\alpha_1(1-\xi)}{(\varphi+\mu+\delta_1)}+\frac{\gamma\alpha_2(1-\xi)((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\varphi(\mu+\delta_2))}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma}$$

$$R_{eff}=\frac{\gamma\alpha_1(1-\xi)(\tau+\mu+\delta_2)\varsigma+\gamma\alpha_2(1-\xi)((\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)-\varphi(\mu+\delta_2))}{(\varphi+\mu+\delta_1)(\tau+\mu+\delta_2)\varsigma} \quad (3.29)$$

(3.85) is the effective basic reproduction number

The effective basic reproduction number, R_{eff}, was determined by the technique in Van den Driessche P. and Watmough J., (2002) and Lauko I.G., (2006). A reproduction number obtained this way determines the local stability of the disease free equilibrium point which is locally asymptotically stable for $R_{eff}<1$ and unstable for $R_{eff}>1$

4. MODEL ANALYSIS AND RESULTS

The results obtained in this work were tested and confirmed by using numerical methods. Variables and parameters values on Ebola Virus Disease epidemiology and the demographic profile of the population were estimated based on the country concerned.

4.1 Total Population

The total population of Nigeria was estimated at 177,500,000 people in the year 2014 with 4,400,000 for Liberia, 6,300,000 for Sierra Leone, 11,600,000 for Guinea (World Population Data Sheet, 2014).

4.2 Variables and Parameters Values

Ebola fever has three (3) geographically different endemic areas: Guinea, Liberia and Sierra Leone in Western Africa. The values for the Infectious and the Quarantine classes were presented in table (4.1) with specific consideration on the Ebola Fever endemic areas. Also presented in table (4.6) were the values for the parameters.

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Table 4.1: Cases of Ebola virus disease, Quarantine and death in endemic areas: Guinea, Liberia and Sierra Leone

Country	No of Cases in I	No. of cases in Q	No. of deaths D	%No of Cases in I
Guinea	2988	2628	1947	65
Liberia	8745	3143	3746	43
Sierra Leone	10792	8084	3301	31
Total	22525	13855	9004	40

Source: CDC (2014)

Table 4.2: Cases of Ebola virus disease, Quarantine and death in endemic areas: Guinea, Liberia and Sierra Leone

Country	No of Cases in I	No. of cases in R	No. of deaths D	%No of Cases in I
Uganda	142	87	55	39

Source: (WHO, AFRO, News, 2023)

$$\frac{total\ no\ of\ \ deaths}{total\ no\ cases} X100$$

% of **Cases of Ebola Virus in** Guinea = $\frac{1947}{2988} X100 = 65$

4.3 Baseline Parameter Values

The values of the parameters are estimated below and presented in Table (4.6)

Natural birth Rate of Humans (β)

Abdulrhaman, (2014) pointed out that the birth rate of countries varies from 6.85 to 47.60 births per year per 1000 people for the year 2012 (Central Intelligence Agency, 2013).

Tables (4.4) and (4.5) show the birth rate of Ebola Fever endemic countries and for other countries of the world. In Nigeria, the birth rate for 2014 is 39.3 births per 1000 persons.

$$\beta = \frac{birthrate\ per\ 1000}{monthly} = \frac{39.3}{1000} x \frac{1}{12} = \frac{0.0393}{12} = 0.00328$$

Death Removal Rate μ

The death removal rate is generally calculated as the multiplicative inverse of life expectancy at birth. Tables (4.4) and (4.5) show the life expectancy of Ebola Fever endemic countries and for other countries of the world. The life expectancy at birth for the year 2014 estimate is 53.7 births per 1000 persons.

$$\mu = \frac{1}{deathrate} / monthly = \frac{1}{53.7} x \frac{1}{12} = \frac{0.0186}{12} = 0.00156$$

Disease induced death Rate due to I δ_1

$$\frac{total\ no.of\ death}{total\ no.of\ cases} / monthly = \frac{9004}{22525} x \frac{1}{12} = \frac{0.03997}{12} = 0.0333$$

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$$\delta_1 = \frac{90}{100} X\, 0.0333 = 0.03000$$

Disease induced death Rate due to Q δ_2

$$\delta_2 = \frac{10}{100} X\, 0.0333 = 0.0033$$

Progression Rate from L **to** I (γ)

$$\gamma = \frac{1}{21} x \frac{1}{30} = 1.429$$

Table 4.3: Baseline Values for Model Parameters

S/N	Parameter	Baseline Value
1	β	0.0033
2	μ	0.0016
3	δ_1	0.0300
4	δ_2	0.0033
5	α_1	0.9
6	α_2	0.4
7	τ	0-1
8	ξ	0-1
9	ς	0-1
10	γ	1.429
11	φ	0-1

4.3.1 Effects of no control on the population dynamics of Ebola Virus disease transmission.

Figure 4.1 is the graph I(t), R(t) and D(t). There is no recovery because there are no controls such as, quarantine, treatment for infectious population, public enlightenment campaign and proper burial and so the infectious population rises so high as well as the dead curve.

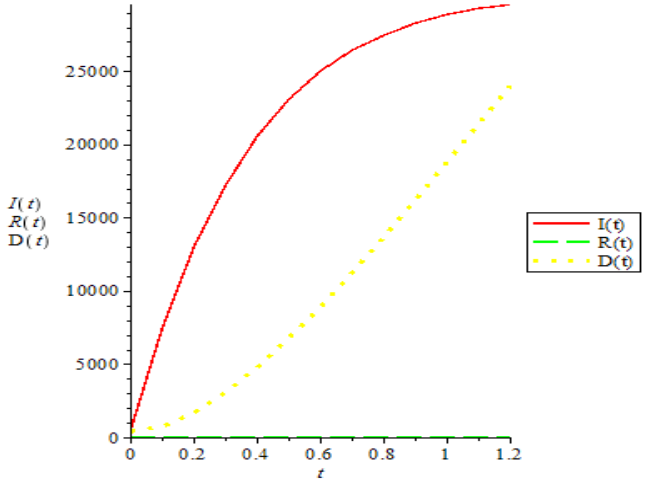

Figure 4.1: Graph of I (t). R (t) and D (t) plotted against time.

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4.3.2 Effects of control on the population dynamics of Ebola virus disease transmission.

We are considering various control parameters such as the $\tau, \varphi, \xi, \varsigma, (0,1)$

These control parameters are varied to see the effect of control measures on the disease. With the four(4) control parameters to be varied as the treatment rate $\tau = \tau_i, where, 0 \le i \le 1$, the rate of quarantine $\varphi = \varphi_i, where, 0 \le i \le 1$, the rate of effectiveness of public campaign $\xi = \xi_i, where, 0 \le i \le 1$, the rate at which the dead is decontaminated and buried $\varsigma = \varsigma_i, where, 0 \le i \le 1$.

Figure 4.2 with only two controls $\varphi = 0, \tau = 0, \xi = 0.25, \varsigma = 0.25$, the graph of the infectious population and the dead curve is gradually decreasing, while the recovery curve is trying to rise.

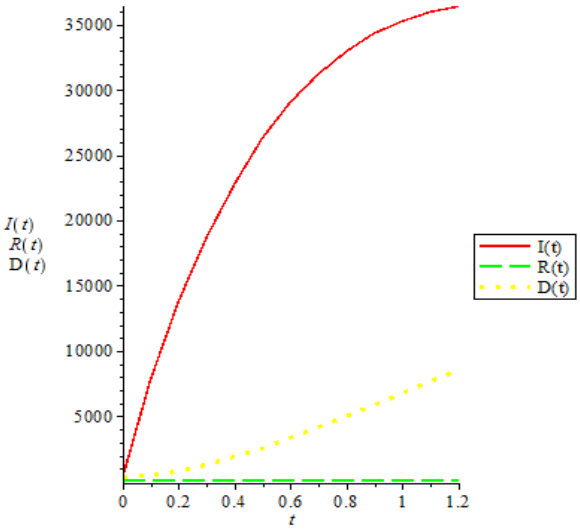

Figure 4.2: Graph of I (t). R (t) and D (t) plotted against time.

Figure 4.3 Graph of I (t). R (t) and D (t) plotted against time. With low controls $\varphi = 0.25, \tau = 0.25, \xi = 0.25, \varsigma = 0.25$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually and the recovery curve is gradually rising.

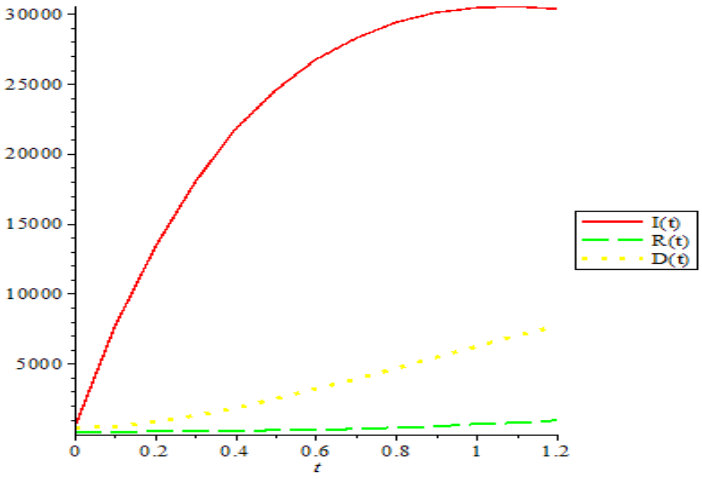

Figure 4.3: Graph of I (t). R (t) and D (t) plotted against time.

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The Figure 4.4 is a graph with controls $\varphi = 0.25, \tau = 0.25, \xi = 0.5, \varsigma = 0.5$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually the while recovery curve is rising fast.

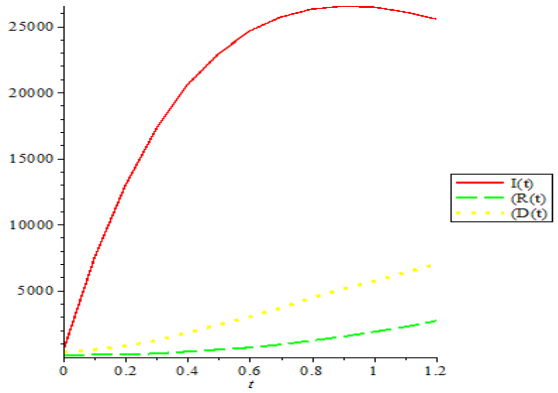

Figure 4.4: Graph of I (t). R (t) and D (t) plotted against time.

Figure 4.5 with control of about $\varphi = 0.25, \tau = 0.25, \xi = 0.5, \varsigma = 0.75$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually and the recovery curve is gradually rising.

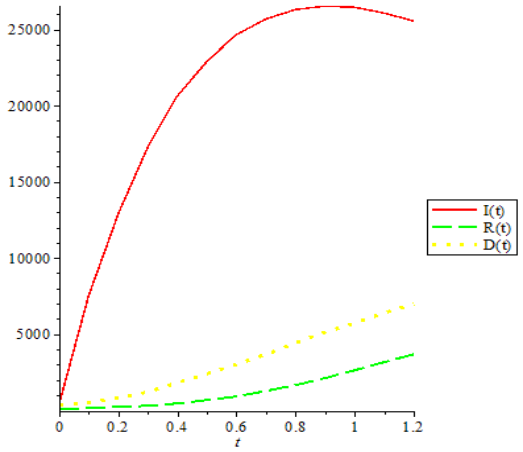

Figure 4.5: Graph of I(t),R() and D(t) plotted against time

Figure 4.6 shows that with controls $\varphi = 0.5, \tau = 0.5, \xi = 0.5, \varsigma = 0.5$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually while the recovery curve is gradually rising.

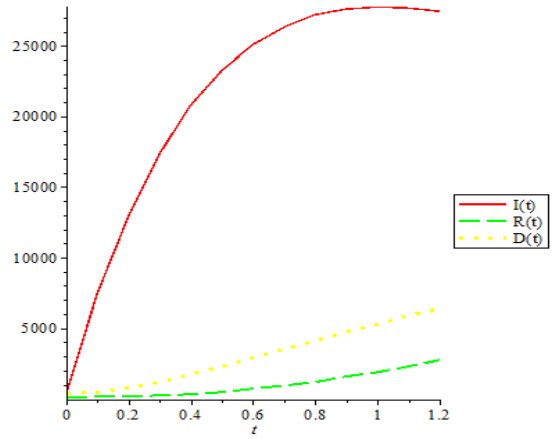

Figure 4.6: Graph of I(t), R(t), D(t) plotted against time.

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Figure 4.7 shows that with controls $\varphi = 0.25, \tau = 0.5, \xi = 0.5, \varsigma = 0.75$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually while the recovery curve is rising high.

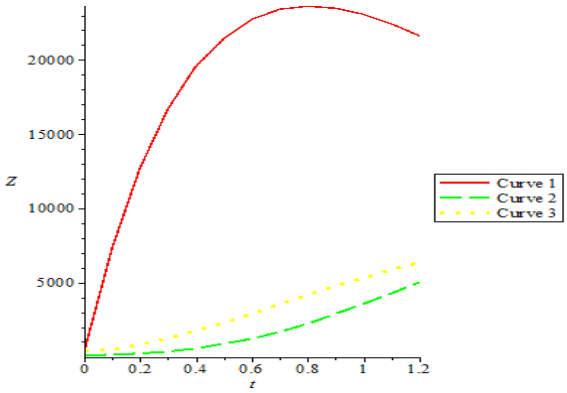

Figure 4.7: Graph of I(t), R(t), D(t) plotted against time.

Figure 4.8 with control of about $\varphi = 0.25, \tau = 0.5, \xi = 0.75, \varsigma = 0.75$, the graph of the infectious population is gradually decreasing as well as the dead curve. The disease is decreasing gradually and the recovery curve is rising and catching up with the dead.

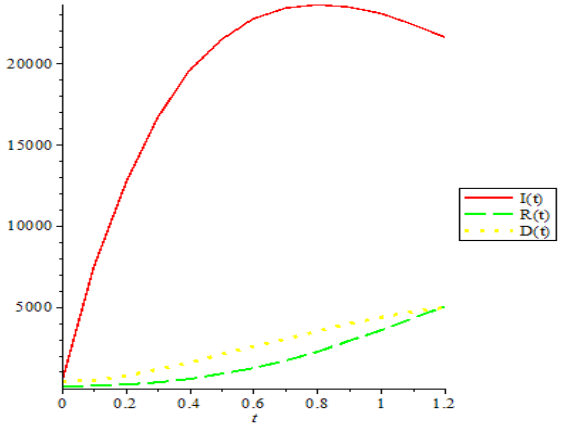

Figure 4.8: Graph of I(t), R(t), D(t) plotted against time.

The Figure 4.9 shows that with controls $\varphi = 0.25, \tau = 0.75, \xi = 0.75, \varsigma = 0.75$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually and the recovery curve is gradually rising

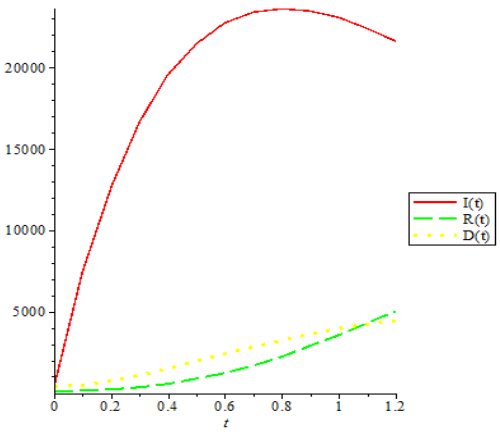

Figure 4.9: Graph of I(t), R(t), D(t) plotted against time.

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Figure 4.10 shows that with controls $\varphi = 0.5, \tau = 0.75, \xi = 0.75, \varsigma = 0.75$, the graph of the infectious population is decreasing and the dead curve is decreasing fast. The disease is decreasing gradually and the recovery curve is rising fast above the dead curve

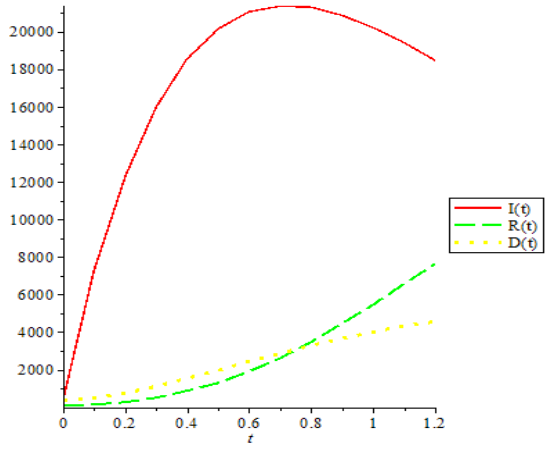

Figure 4.10: Graph of I(t), R(t), D(t) plotted against time.

Figure 4.11 shows that with controls of $\varphi = 0.75, \tau = 0.75, \xi = 0.75, \varsigma = 0.75$, the graph of the quarantine population is decreasing and the graph for the dead is drastically reduced to zero overtime while the recovery graph drastically rose up above the quarantine and dead graph respectively.

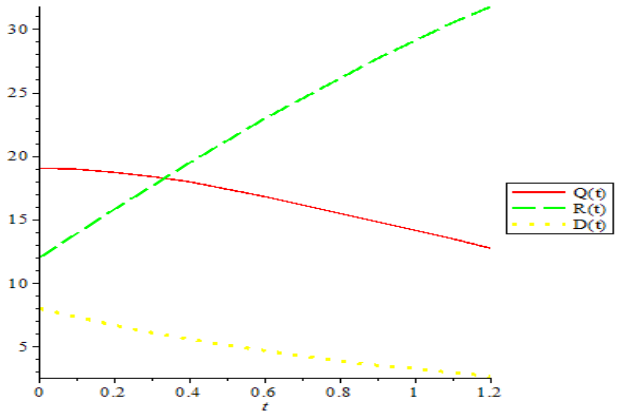

Figure 4.11: Graph of Q(t), R(t) and D(t) against time.

5. CONCLUSION

This research studies mathematical model for the dynamics of Ebola virus disease incorporating quarantine and public campaign as controls. The model was developed as a system of six (6) non-linear ordinary differential equations. The population is divided into six (6) compartments, namely: Susceptible S(t), Latent L(t), Infectious I(t), Quarantined $Q(t)$ Recovered R(t), and Dead D(t). We obtained and analysed the equilibrium states of the model for stability .The effective Basic Reproduction Number R_{eff} was obtained, which suggests that if $R_{eff} \leq 1$, the Disease free equilibrium state is locally asymptotically stable, meaning that the disease dies out in a population, or if $R_{eff} \geq 1$, the Disease free equilibrium state is unstable meaning that disease persists in a population. The analysis of the endemic equilibrium (EE) States of the model were carried out The control parameters are quarantine (φ), rate of effectiveness of public enlightenment campaign (ξ), treatment rate (τ) and rate of proper burial of infectious dead (ς). The numerical results are clearly displayed, to see the effects of controls on the disease.

Figure 4.1 is the graph I(t), R(t) and D(t). There is no recovery because there are no controls such as, quarantine, treatment for infectious population, public enlightenment campaign and proper burial and so the infectious population rises so high as well as the dead curve.

Figure 4.2: with only two controls $\varphi = 0, \tau = 0, \xi = 0.25, \varsigma = 0.25$, the graph of the infectious population and the dead curve is gradually decreasing, while the recovery curve is trying to rise. Figure 4.3: Graph of I (t). R (t) and D (t) plotted against time. With low controls $\varphi = 0.25, \tau = 0.25, \xi = 0.25, \varsigma = 0.25$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually and the recovery curve is gradually rising. The Figure 4.4 is a graph with controls $\varphi = 0.25, \tau = 0.25, \xi = 0.5, \varsigma = 0.5$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually the while recovery curve is rising fast. Figure 4.5: with control of about $\varphi = 0.25, \tau = 0.25, \xi = 0.5, \varsigma = 0.75$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually and the recovery curve is gradually rising. Figure 4.6 shows that with controls $\varphi = 0.5, \tau = 0.5, \xi = 0.5, \varsigma = 0.5$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually while the recovery curve is gradually rising. Figure 4.8: with control of about $\varphi = 0.25, \tau = 0.5, \xi = 0.75, \varsigma = 0.75$, the graph of the infectious population is gradually decreasing as well as the dead curve. The disease is decreasing gradually and the recovery curve is rising and catching up with the dead.

The Figure 4.9 shows that with controls $\varphi = 0.25, \tau = 0.75, \xi = 0.75, \varsigma = 0.75$, the graph of the infectious population and the dead curve is gradually decreasing. The disease is decreasing gradually and the recovery curve is gradually rising. Figure 4.10 shows that with controls $\varphi = 0.5, \tau = 0.75, \xi = 0.75, \varsigma = 0.75$, the graph of the infectious population is decreasing and the dead curve is decreasing fast. The disease is decreasing gradually and the recovery curve is rising fast above the dead curve. Figure 4.11 shows that with controls of Figure 4.11 shows that with controls of $\varphi = 0.75, \tau = 0.75, \xi = 0.75, \varsigma = 0.75$, the graph of the quarantine population is decreasing and the graph for the dead is drastically reduced to zero overtime while the recovery graph drastically rose up above the quarantine and dead graph respectively.

REFERENCES

[1] **ABAH,** Roseline T. and Akinwande Ninuola I. A Mathematical Modeling and Solutions on the Transmission Dynamics of Ebola Virus Disease for a Disease Free Nigeria Abacus, vol. 45, no. 1, 295-310, (2018)

[2] **ABAH,** Roseline T. and Akinwande, Ninuola I. Stability Analysis of the Disease Free Equilibrium State of a Mathematical Model for the Dynamics of EBOLA virus Disease (EVD) Epidemics Abacus, (45), no. 1, 285-294-81, (2018)

[3] Abah, R. T. , Akinwande, N. I., Enagi, I. A.., Kuta, F. A. , Abdulrahaman, S., Somma, S. A.(2015) "Stability analysis of the disease free equilibrium state of a mathematical model of Ebola Fever disease epidemic"

[4] Abdulrhaman, S. (2014) "A mathematical model for the transmission dynamics of Hepatitis B Virus and control of Hepatitis B Virus. Ph.D thesis (unpublished) Department of mathematics and statistics, FUT, Minna.

[5] Akinwande, N.I. (1995) "Local Stability Analysis of Equilibrium State of A Mathematical Model of Yellow Fever Epidemics" J. Nig. Math Soc Vol. 14, pp 73-79

[6] Althaus CL. Estimating the reproduction number of Ebola virus (EBOV) during the 2014 outbreak in West Africa. PLOS Currents Outbreaks. 2014 Sep 2. Edition 1. Wikipedia, "Timeline of reported cases and deaths, Ebola virus epidemic in West Africa," 2014.

[7] Birmingham, K and Cooney, S. Ebola: small, but real progress (news feature). Nature Med 2002;8:313.

[8] Breman, JG, Piot, P, Johnson, KM, et al. The Epidemiology of Ebola Hemorrhagic Fever in Zaire, 1976. Proc Int Colloquium on Ebola Virus Inf held in Antwerp, Belgium, 6 – 8 December 1977.

[9] Castillo-Chavez, C, Velasco-Hernandez, JX and Fridman, S. Modeling Contact Structures in Biology. In: S.A. Levin ed., Frontiers of Theoretical Biology, Lecture Notes in Biomathematics 100, Springer-Veralg,Berlin-Heidelberg-New York 1994:454-91.

[10] Centers for Disease Control (CDC). Ebola Hemorrhagic Fever. (http://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/ebola.htm), accessed on August 24, 2003.

[11] Centers for Disease Control (CDC). Ebola Hemorrhagic Fever: Table Showing Known Cases and Outbreaks, in Chronological Order. (http://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/ebotabl.htm), accessed on August 24, 2003.

[12] Centre for Disease Control (CDC). Interim Guidance for Managing Patients with Suspected Viral Hemorrhagic Fever in U.S.Hospitals (2005).Available athttp://www.cdc.gov

[13] Centers for Disease Control (CDC), 2003 Ebola hemorrhagic feverhttp://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/ebola.htm), accessed on August 24, 2003.

[14] Committee Regarding the 2014 Ebola Outbreak in WestAfrica. Geneva: WHO; 8 Aug 2014. [Accessed 10 Sep 2014].Available from:http://www.who.int/mediacentre/news/statements/2014/ebola-20140808/en/

[15] Chowell, G, Castillo-Chavez, C, Fenimore, PW, et al. Implications of an Uncertainty and Sensitivity analysis for SARS's Basic Reproductive Number for General Public Health Measures (submitted).

[16] Chowell, G, Fenimore, PW, Castillo-Garsow, MA, et al. SARS Outbreaks in Ontario, Hong Kong and Singapore: the role of diagnosis and isolation as a control mechanism. J Theor Biol 2003;24:1-8.

[17] Chowell G, Hengartner NW, Castillo-Chavez C, Fenimore PW, Hyman JM. The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda. J Theor Biol. 2004 Jul 7;229(1):119-26.

[18] Van de Driessche, P. and Watmough, J. (2002): Reproduction numbers and Sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical Bio-sciences, Volume 180, pp: 29-48.

[19] World Health Organization (WHO). Ebola Hemorrhagic Fever. (http://www.who.int/inf-fs/en/fact103.html), accessed on August 24, 2003.

[20] World Health Organization (WHO). Ebola virus disease, West Africa – update on 27 July 2014. Geneva: WHO; 27 Jul 2014.[Accessed 23 Aug 2014]. Available from:http://www.who.int/csr/don/2014_07_27_ebola/en/

[21] World Health Organization (WHO), 2003a.Ebola hemorrhagic fever: disease outbreaks. (http://www.who.int/disease-outbreaknews/disease/A98.4.htm), accessed on October 17, 2003.