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THE DYNAMICS OF AN EBOLA EPIDEMIC MODEL WITH QUARANTINE OF INFECTIVES

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ABSTRACT. The recurrent outbreaks of ebola in Africa present global health challenges. Ebola is a severe, very fatal disease with case fatality rates of up to 90%. In this paper, a theoretical deterministic model for ebola epidemic with quarantine of infectives is proposed and analyzed. The model exhibits two equilibria; the disease free and endemic equilibrium points. The basic reproduction number, R_0, which is the main threshold, is obtained and the stability of the equilibrium points established. Using parameter values drawn from the 2014 West Africa ebola outbreak, a numerical simulation of the model is carried out. It is found that the dynamics of the model are completely determined by R_0 and that a quarantine success rate of at least 70% is sufficient to contain the disease outbreak.

Key words and phrases: Basic reproduction number; Ebola; Endemic equilibrium; Stability; Quarantine.

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1. INTRODUCTION

Ebola is a severe, fatal illness in humans and non-human primates such as monkeys, gorillas and chimpanzees [6, 19]. Ebola first appeared in 1976 in two simultaneous outbreaks in Nzara, Sudan and Yambuku, Democratic Republic of Congo; Yambuku is near River Ebola after which the disease is named [4, 17, 19]. Since then, various outbreaks have been reported in various countries in Africa. The largest and deadliest outbreak occurred in 2014 in the West African countries of Guinea, Liberia, Mali, Nigeria, Senegal and Sierra Leone. It claimed more than 10,000 lives with 6 deaths in Mali, 8 in Nigeria, 2543 in Guinea, 3956 in Sierra Leone and 4809 in Liberia. In fact the 2014 outbreak claimed more lives than all previous outbreaks combined. [19]. The WHO credits enhanced surveillance as well as strengthened preparedness and readiness as the major factors responsible for rapidly bringing the 2017 outbreak in Democratic Republic of Congo under control with only five deaths reported [25].

The exact reservoir of ebola is still unknown although it is believed to be zoonotic with fruit bats of the *pteropodidea* family considered to be the most likely host [2]. The causal agent for ebola is a virus of the family *Filoviridal* and genus ebola virus. The virus is transmitted to humans through close contact with the blood, secretions, organs or other bodily fluids of infected animals. Human to human transmission of ebola results from direct contact (through mucous membranes and broken skin) with blood, secretions, organs and other bodily fluids of infected people and indirect contact with environments contaminated with such fluids [10, 19, 21]. Burial ceremonies such as ones in which mourners have direct contact with the body of the deceased person also play a great role in the disease transmission [16]. Nosocomial transmission (i.e. transmission within a healthy care setting) is responsible for the death of many healthy care givers especially when they do not wear protective gear [6]. Furthermore, it is believed that men who have recovered from the disease can still transmit it through their semen for up to seven weeks after recovery [19].

There is as yet no licensed treatment proven to neutralize the ebola virus but a range of blood, immunological and drug therapies are under development. In fact, in a major trial in Guinea, an experimental ebola vaccine called rVSV-ZEBOV has proven highly protective against the virus [19, 25]. Most infected persons die within ten days of their initial infection [3]. The incubation period, from the time of infection to the onset of symptoms, is 2-21 days [19]. It is believed that ebola can not be transmitted during the incubation period [18]. A person infected with ebola presents the following symptoms: fever, headache, joint and muscle aches, general weakness, diarrhea, vomiting, stomach pains and loss of appetite. Others are cough, sore throat, difficulty breathing and bleeding from all body openings [6, 19]. Good outbreak control relies on applying a package of interventions namely case management, infection prevention and control, surveillance and contact tracing, a good laboratory service, safe burials and social mobilization [19].

As noted in [9], when confronted with a new disease and the risk of an emerging epidemic, mathematics is perhaps not the first Science called upon by policy makers and pathologists and yet to predict the future course of the disease and compare the effectiveness of different control strategies, an epidemiological model informed by biological and economical insights is an essential tool in the arsenal. The 2014 West Africa ebola outbreak attracted worldwide attention in part because it broke out in many countries and claimed an unprecedented number of lives. As a result it has elicited a lot of research, in light of which the study is carried out.

In [7], the authors used a simple $SEIR$ epidemic model and data from two well documented ebola outbreaks in Uganda (2000) and Congo (1975) to estimate the basic reproduction number of ebola. It was found out that a 2-week delay in the implementation of control measures (contact tracing followed by quarantine) resulted in a doubling of the final epidemic size . The same data was used in [18] to develop a stochastic model for the transmission of ebola in different epidemiological settings namely illness in the community, hospitals and traditional burial. It was established that for both epidemic outbreaks, increasing the hospitalization rate reduced the predicted epidemic size. In [8], an $SEIR$ stochastic model was formulated and fit to data of infected cases and deaths in Guinea, Sierra Leone and Liberia while the authors in [11] have studied the short term growth rate of ebola in West Africa using the global epidemic and mobility model to generate stochastic, individual based simulations of epidemic spread worldwide. Deterministic epidemic models in which a compartment of quarantined individuals is included are plenty in literature see [14] and references therein. A mathematical model for the dynamics of ebola virus disease in human populations with quarantine states was considered in [15] while in [20] an SIR model incorporating both direct and indirect transmissions was developed. Further, in [22], a deterministic epidemic model for ebola virus disease incorporating vector population was formulated and analyzed. Using a theory of competitive systems, the authors in [23] analyzed the global stability of an ebola virus disease dynamics model with varying population sizes. The role of personal hygiene of susceptible populations influenced by public enlightenment campaigns was studied in [24]. Finally, a review of West African ebola epidemic dynamics models was carried out in [13]. Clearly literature is abundant on ebola dynamics especially after the catastrophic 2014 outbreak in West Africa that elicited worldwide attention.

In this paper, a deterministic SIQ epidemic model for the dynamics of ebola with quarantine of infectives is formulated and analyzed. Numerical simulations are carried out using data from Liberia, one of the West African countries worst hit by the 2014 outbreak. It is assumed that quarantined individuals are those who are hospitalized and are undergoing treatment in a health facility. The major aim of quarantine is to isolate infectives so as to limit their contact with susceptible thereby reducing the disease transmission rate. Unlike other diseases where an infected individual may recover without treatment, a person infected with ebola can not survive if not hospitalized. Even with hospitalization, not all patients recover and those who recover do not gain immunity. For example, the WHO estimates that the survival rate for the 2014 West African ebola outbreak was about 47%.

This paper is organized as follows; in Section 2, a basic model for the dynamics of ebola with quarantine of infectives is formulated. In Section 3, the equilibrium points and the basic reproduction number are obtained and the local and global stability of the equilibrium points established while in Section 4, numerical simulations of the model are presented and a brief discussion given.

2. MODEL FORMULATION

The total population is divided into three distinct epidemiological classes of individuals who are susceptible, infected and quarantined with sizes denoted by $S(t)$, $I(t)$ and $Q(t)$ respectively. The latent or exposed class has been ignored since ebola can not be transmitted during the latent period [19, 18]. Susceptible individuals get infected through contact with the infected individuals at a rate β. Upon infection, an individual is either quarantined in a health facility where he undergoes treatment or dies from the disease within 10 days after infection [3]. The

quarantined individual may recover without immunity and join the susceptible class again or may die from the disease. It is assumed that only infected individuals are quarantined (or hospitalized) and that infected but unquarantined persons can not recover from the disease. The total population size is variable since there is both recruitment into the susceptible class (through birth and immigration) and natural and disease-induced deaths.

With these assumptions, the basic model for the dynamics of ebola with quarantine of infectives is represented by the following system of ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SI - \mu S + \gamma Q, \\ \frac{dI}{dt} &= \beta SI - (\mu + \alpha + d)I, \\ \frac{dQ}{dt} &= \alpha I - (d + \mu + \gamma)Q. \end{aligned} \tag{2.1}$$

All parameters are positive constants. The constant A is the recruitment rate of susceptible corresponding to birth and immigration, μ is the per capita natural mortality rate, β is the transmission rate, α is the rate at which individuals are quarantined, d is the per capita disease induced death rate and γ is the recovery rate of quarantined individuals.

The total population size $N(t)$ is variable with $N'(t) = A - \mu N - d(I + Q)$. In absence of the disease, the population size approaches the carrying capacity $\frac{A}{\mu}$. The differential equation for $N(t)$ implies that solutions of system (2.1) starting in the positive orthant $\Re^3_+$ either approach, enter or remain in the subset of $\Re^3_+$ defined by $D = \{(S, I, Q)|S \geq 0, I \geq 0, Q \geq 0, S + I + Q \leq \frac{A}{\mu}\}$. Thus it suffices to consider solutions in the region D. Solutions of the initial value problem (IVP) starting in D and defined by system (2.1) exist and are unique on a maximal interval [12]. Since solutions remain bounded in the positively invariant region D, the maximal interval is $[0, \infty)$. Thus the IVP is well posed both mathematically and epidemiologically.

The following theorem on limiting systems [5](pg 33) will be useful in the mathematical analysis of the model.

Lemma 1. *Consider the following two systems: $\frac{dx}{dt} = f(t, x); \frac{dy}{dt} = g(y)$ where $x, y \in R^n$ and f, g are continuous, satisfy the local Lipschitz condition in any compact set $X \in R^n$ and $f(t, x) \to g(x)$ as $t \to \infty$ so that the second system is the limit system of the first system. Let $\phi(t, t_0, x_0)$ and $\varphi(t, t_0, y_0)$ be solutions of these systems respectively. Suppose $e \in X$ is a locally asymptotically stable equilibrium of the limit system and it's attractive region is $W(e) = \{y \in X|\varphi(t, t_0, y) \to e, t \to +\infty\}$. Let $W_\phi \cap W(e) \neq \phi$, then $\lim_{t\to+\infty} \phi(t, t_0, x_0) = e$.*

3. EQUILIBRIUM POINTS AND THEIR STABILITY

The equilibrium points are obtained by setting the derivatives of system (2.1) to zero. Calculations show that the model exhibits two equilibria namely the disease free equilibrium point, $E_0(\frac{A}{\mu}, 0, 0)$ and the endemic equilibrium point $E_1(S^*, I^*, Q^*)$ where $S^* = \frac{\mu+d+\alpha}{\beta}$, $I^* = \frac{\mu+d+\gamma}{\beta}(\frac{A\beta-\mu(\mu+d+\alpha)}{(\mu+d+\alpha)(\mu+d+\gamma)-\alpha\gamma})$ and $Q^* = \frac{\alpha}{\beta}(\frac{A\beta-\mu(\mu+d+\alpha)}{(\mu+d+\alpha)(\mu+d+\gamma)-\alpha\gamma})$.

The basic reproduction number, R_0, defined by [1] as the number of secondary infectives arising out of one infective individual introduced in a disease free population is given by $R_0 = \frac{\beta(\frac{A}{\mu})}{\mu+d+\alpha}$. For convenience, let $k_1 = (\mu+d+\alpha)$ and $k_2 = (\mu+d+\gamma)$. It can be observed that the endemic equilibrium point exists whenever $R_0 > 1$ and $k_1k_2 > \alpha\gamma$.

We proceed to establish the local stability of the equilibria. The local stability of the equilibrium points determines the behavior of the system near the equilibrium points. The Jacobian matrix

at the disease free equilibrium point is

$$\begin{pmatrix} -\mu & -\frac{\beta A}{\mu} & \gamma \\ 0 & \frac{\beta A}{\mu} - k_1 & 0 \\ 0 & \alpha & -k_2 \end{pmatrix}, \tag{3.1}$$

and the eigenvalues are $\lambda_1 = -\mu$, $\lambda_2 = -k_2$ and $\lambda_3 = \frac{\beta A}{\mu} - k_1$. By Routh–Hurwitz criteria, the disease free equilibrium point is locally asymptotically stable provided $R_0 < 1$.
The Jacobian matrix at the endemic equilibrium is

$$\begin{pmatrix} \frac{-\mu(k_1k_2R_0-\alpha\gamma)}{k_1k_2-\alpha\gamma} & -k_1 & \gamma \\ \frac{\mu k_1k_2(R_0-1)}{k_1k_2-\alpha\gamma} & 0 & 0 \\ 0 & \alpha & -k_2 \end{pmatrix}, \tag{3.2}$$

and the characteristic equation is given by

$$\lambda^3 + \lambda^2[\frac{\mu(k_1k_2R_0 - \alpha\gamma)}{k_1k_2 - \alpha\gamma} + k_2] + \lambda[\frac{\mu(k_1k_2R_0 - \alpha\gamma) + \mu k_1^2k_2(R_0 - 1)}{k_1k_2 - \alpha\gamma}] + \mu k_1k_2(R_0 - 1) = 0$$

which is of the form $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$
where $a_1 = \frac{\mu(k_1k_2R_0-\alpha\gamma)}{k_1k_2-\alpha\gamma} + k_2$, $a_2 = \frac{\mu(k_1k_2R_0-\alpha\gamma)+\mu k_1^2k_2(R_0-1)}{k_1k_2-\alpha\gamma}$ and $a_3 = \mu k_1k_2(R_0 - 1)$. Now,

$$a_1a_2 - a_3 = \mu^2(k_1k_2 - \alpha\gamma)(k_1k_2R_0 - \alpha\gamma) + \mu k_2(k_1k_2 - \alpha\gamma)(R_0 - 1)(1 + k_1\alpha\gamma)(k_1k_2 - \alpha\gamma)^2$$

Since the endemic equilibrium exists whenever $k_1k_2 > \alpha\gamma$ and $R_0 > 1$ then $k_1k_2R_0 > \alpha\gamma$. In this case $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1a_2 > a_3$. By Routh-Hurwitz criteria for a characteristic polynomial of degree 3, the endemic equilibrium point is therefore locally asymptotically stable provided $R_0 > 1$.
In the proceeding, the global stability of the equilibria is established. In order to determine the global stability of the disease free equilibrium, consider the Liapunov function $L = I$. It's derivative is

$$\begin{aligned} L' = I' &= [\beta S - (\mu + \alpha + d)]I \\ &= [\beta\frac{A}{\mu} - (\mu + \alpha + d)]I \\ &\leq [\frac{\beta A}{\mu(\mu + \alpha + d)} - 1] \\ &\leq [R_0 - 1] \\ &\leq 0 \end{aligned}$$

whenever $R_0 \leq 1$ since $S \leq \frac{A}{\mu}$. By the Liapunov–LaSalle theorem [12], solutions in D approach the largest positively invariant subset of the set where $L' = 0$ which is the set where $I = 0$. In this set, $Q'(t) = -(\mu+d+\gamma)$, so that $Q \to 0$ as $t \to \infty$. Thus, $S'(t)$ is asymptotically equal to $A - \mu S$ or $S \to \frac{A}{\mu}$. Thus all the solutions in the set where $I = 0$ go to the disease free equilibrium which is therefore globally asymptotically stable.
In order to study the global stability of the endemic equilibrium point, we first note that when $d = 0$, then $N'(t) = A - \mu N$ such that as $t \to \infty$ then $N \to \frac{A}{\mu}$. The limit system for (2.1) is :

$$\begin{aligned} N' &= 0 \\ I' &= [\beta\frac{A}{\mu} - (\mu + \alpha)]I \\ Q' &= \alpha I - (\mu + \gamma)Q \end{aligned} \tag{3.3}$$

with $N = \frac{A}{\mu}$.
Re-writing system (3.3) as $I' = P(I,Q)$ and $Q' = R(I,Q)$, let $H(I,Q) = \frac{1}{I}$ be a Dulac multiplier, then

$$\begin{aligned}\frac{\partial}{\partial I}(HP) + \frac{\partial}{\partial Q}(HR) &= \frac{\partial}{\partial I}[\frac{\beta A}{\mu} - (\mu+\alpha)] + \frac{\partial}{\partial Q}[\alpha - (\mu+\gamma)\frac{Q}{I}] \\ &= -\frac{(\mu+\gamma)}{I} \\ &< 0\end{aligned}$$

so that there are no periodic solutions in the interior of region D. By the Poincare–Bendixson theorem, all solutions starting in the first quadrant region with $I > 0$ and $I + Q \leq \frac{A}{\mu}$ approach (I^*, Q^*) as $t \to \infty$. Thus, E_1 is a globally asymptotically stable equilibrium for the limit system (3.3). By **Lemma 2.1**, all solutions starting in the region $D - \{S, I, Q | I = 0\}$ of the original system (2.1) approach the endemic equilibrium point E_1 as $t \to \infty$. Thus, the endemic equilibrium point E_1 is globally asymptotically stable whenever it exists.
The above results can be summarised in the following theorem:

Theorem 1. *System (2.1) has two equilibria; the disease free and the endemic equilibrium points which are such that;*
(i) When $R_0 < 1$, the disease free equilibrium is the only equilibrium in the interior of D and is globally asymptotically stable.
(ii) When $R_0 > 1$, a unique positive equilibrium, the endemic equilibrium point, also exists in the interior of D and is globally asymptotically stable.

4. NUMERICAL SIMULATIONS

In this section, a numerical simulation of the model is carried out using Matlab's ode45 equation solver and parameter values estimated from the 2014 ebola outbreak in Liberia, West Africa. In [8], the transmission rates for ebola in Guinea, Sierra Leone and Liberia were estimated to be $0.27, 0.45$ and 0.28 respectively while the basic reproduction numbers were respectively $1.51, 2.53$ and 1.59. Similarly, the WHO estimated that the recovery or survival rate, γ, for the 2014 West Africa outbreak of ebola stood at 47% higher than the previous outbreaks [19]. This was attributed to enhanced surveillance as well as increased preparedness and readiness. This means that the death rate,d, from the disease was about 53%. Furthermore, the World Bank estimates the life expectancy for Liberia to be 60.21 years which implies that the per capita natural mortality rate, μ, is approximately 0.00005 per day. The parameters, their description and estimated values are summarized in Table 4.1 and the numerical simulations presented in Figure 4.1.

4.1. **Discussion.** A basic model for ebola with quarantine of infectives has been formulated and analyzed using data from the 2014 outbreak in Liberia, West Africa. The model exhibits two equilibria; the disease free and the endemic equilibrium points. The basic reproduction number, R_0, also called the quarantine basic reproduction number, has been obtained and used to establish the stability of the equilibrium points. From $R_0 = \frac{\beta(\frac{A}{\mu})}{\mu+d+\alpha}$, it can be observed that R_0 is the product of the contact rate β, the number $\frac{A}{\mu}$ of susceptibles at the disease free equilibrium and the average residence time $\frac{1}{\mu+d+\alpha}$ in the infective class. Thus R_0 is the average number of secondary infections in a completely susceptible population when one infective enters the population in the situation where the average infectious period is decreased by the quarantine of some infectives. In absence of quarantine, the basic reproduction number would be $R_0 = \frac{\beta(\frac{A}{\mu})}{\mu+d}$

Table 4.1: Parameter estimates for 2014 outbreak in Liberia, West Africa

Parameter	Description	Value per day
β	transmission rate	0.28
μ	natural mortality rate	0.00005
d	disease induced death rate	0.53
γ	recovery/survival rate	0.47
α	quarantine rate	0.7
A	recruitment rate into susceptibles	0.00005

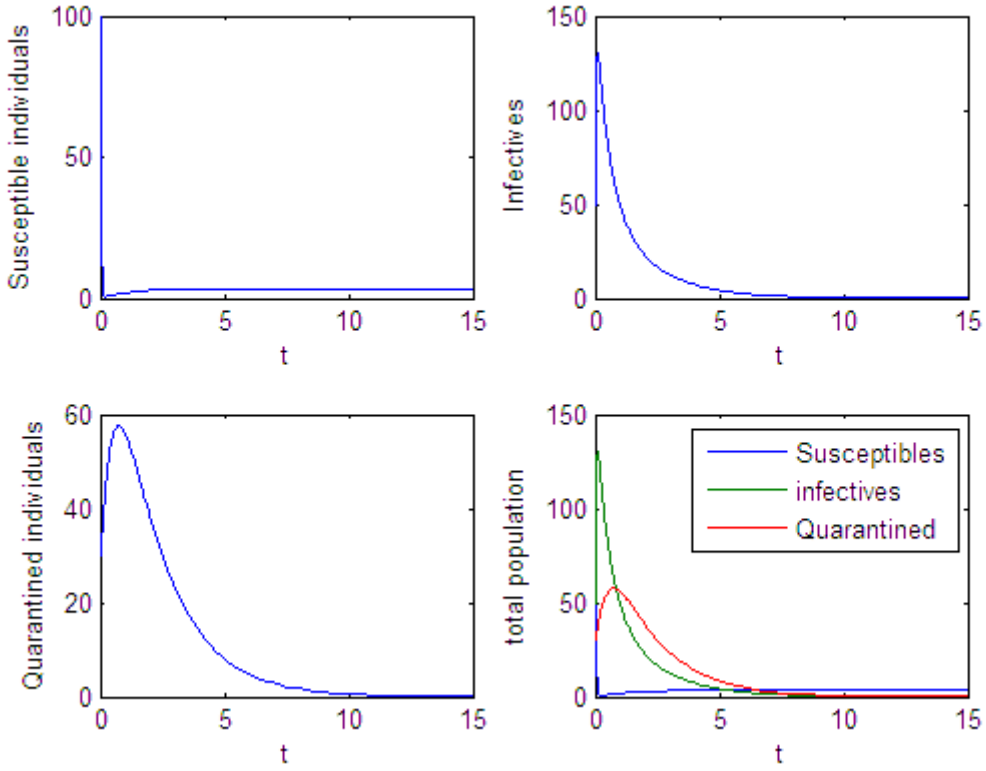

Figure 4.1: Variation with time of population sizes at the disease free equilibrium point with initial values $S = 100$, $I = 50$ and $Q = 30$ and parameter values in the Table 4.1.

with $\alpha = 0$. It is found that when $R_0 < 1$, the disease free equilibrium is globally asymptotically stable and that when $R_0 > 1$ a unique equilibrium point, the endemic equilibrium point, exists and is globally asymptotically stable. Further analysis of R_0 shows that the two dominant parameters during an outbreak of ebola are the transmission rate β and the quarantine rate α of infectives since the other parameters represent natural processes of birth and death. Fighting an ebola outbreak should therefore target to limit the transmission rate and increase the quarantine rate of infected persons. Quarantine of infectives is crucial as it eliminates opportunities for contact between infective and susceptible persons thereby limiting transmission rate. Management of epidemics involves progressively reducing R_0 to a value less than unity. In this case

each infected individual will not spread the disease to others and in the long run the disease will be wiped out. It can be deduced from the expression for R_0 that reducing R_0 to a value less that unity involves reducing the transmission rate β and increasing the quarantine rate α of infectives. From the numerical simulation, it is realized that when the quarantine (or hospitalization) rate of infectives is at least 70%, the disease free equilibrium point is stable and therefore the disease can be eliminated. This call for improved surveillance, preparedness and response. In addition, swift and accurate laboratory testing of samples is essential in rapidly assessing the scope and spread of any ebola outbreak especially in remote parts of Africa.

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