MATHEMATICAL MODELLING OF AN OUTBREAK OF EBOLA VIRUS (EBOV): Predicting the future of Ebola in West Africa.

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ABSTRACT

Ebola virus (EBOV) outbreak is an emergency of international concern and there has been very little work done to predict the spread of the virus in West Africa .The 2014 EBOV outbreak is the largest in the history of mankind. Despite improved control measures, Ebola remains a serious public health risk in African regions where recurrent outbreaks have been observed since the initial epidemic in 1976. In response to the continuing report of new cases of deaths (49.9% of 1914 reported cases between 1st- 31st August 2014) and the effects of control interventions are yet to be determined. Real-time analysis of EBOV could provide helpful information for public health policy in West Africa .In this study we describe 2014 EBOV epidemic using SIR and SEIR Models, fitting the models to the most recent data about reported cases and deaths in Guinea, Sierra Leone and Liberia provided estimates of the basic reproductive numbers R_o of EBOV in absence and presence of control intervention. We offer the most recent example of how tragedy can befall a country. The dynamics of these models are determined by the per-capita death rate of the infected individual and the per-capita effective contact rate of an individual contracting the disease. We computed the basic reproductive number R_O and the effective reproduction number R_e to determine the infectiousness and the dynamics of EBOV. Finally the results of these outbreaks will equip epidemiologist modelling Ebola diseases in future with predictions to enable them minimize potential deaths.

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INTRODUCTION

The mode of transmission of Ebola is complex with multiple variations in the symptoms and its origin is somewhat obscure. There have been outbreaks of Ebola virus disease in central and Eastern African. The latest major outbreak in 2014 occurred in West Africa (Guinea, Sierra Leone and Liberia) with over 4000 reported cases and 2097 deaths (53%) [20]. In this work we considered only the August outbreak of the above named countries.

Ebola is a unique member of the ribonucleic acid virus family that has no known natural reservoir currently, (EBOV) cause a severe form of Viral Haemorrhagic Fever (VHF) with lethality in human ranging from 50%-90% depending on the virus species and strain [18]. The incubation period of Ebola is 2-21 days, and the infectious period is 4-10days. The onset of Ebola is characterized by severe headaches, malaise, fever, vomiting, bloody diarrhea, and rash. Severe bleeding and shock and usually followed by death. The diagnosis of Ebola can be difficult, because Ebola is frequently misdiagnosed as typhoid and malaria. Currently there is no treatment of Ebola [4]. Ebola is transmitted through primary contacts with the bodily fluids of an infected person and health care providers who are in direct contact with such body fluids. Ebola

can also be transmitted through secondary contacts by family members caring for the infected. Finally, Ebola can be transmitted where infection control mechanisms are not practiced. These includes; washing of hands with sanitizer, wearing gloves or as complicated as level four disease control .Airborne spreads has not been proven as a means of transmission. The high virulence in humans and the bio-threat classification as bio-safety level 4/category A, pronounce the complex diversity of the models. There are not adequate animal models for the remaining Ebola virus species Sudan Ebola virus (SEBOV), cote d'lvoire Ebola virus (CIEBOV) and Reston Ebola virus (REBOV) [8].

Dynamic transmission models are increasing in being used to improve our understanding of the epidemiology and the spread of the disease .However there has been no recent comprehensive review of this emerging field. We therefore summarize how mathematical model of improved version can be developed over time. Recent out breaks of Ebola in West Africa began in Guinea in December, 2013 and later spread to Liberia, and Sierra Leone. Historically human behavior has been intractably linked with the spread of infectious diseases [15], understanding the mode of transmission is Key to improving control efforts. The literature of the mathematical modeling of the transmission of Ebola is rather Scant. [3] Conjuncture that the infection process obeys a non – linear saturation – type law. [6] investigated the role of aquatic reservoir, while [9] extended, this model to include hyper infectious state of the virus [14] assented the effect of treatment in a model with carriers. To the best of our knowledge, these studies which none has heretofore articulated quarantine control intervention in West Africa Concurrently do not explicitly consider a deterministic compartmental model with control measures. [17] model optimal intervention strategies by introducing control variables to lower contact rate, increase treatment and accounting for vaccination. While to formulate and analyze a deterministic model for Ebola that includes vaccinating susceptible individuals, quarantine, and treatment of infected individuals. This study is to better understand the mathematical dynamics of a population infected by Ebola when an outbreak occurs. We are using systems of differential equations to model the outbreaks.

DESCRIPTION OF MODEL

The object of this part of the research is to model West African's August, 2014 Ebola Virus disease epidemic outbreak, using the Susceptible – Infections – Recovery (SIR) model. The dynamics of this system happened in two stages: Susceptible to infected, and infected to dead. This suggests a closed system where the susceptible could become infected at some point in time. This model assumes that the initial population is equal to the population that will eventually be infected. The general epidemic model assumes that the people begin susceptible to infections disease may become infected by exposure to an infectious person, becoming immediately infections themselves and after a time t period either recover or die. Recovery constitutes immunity to further infection and they are said to be removed.

The simplest version of this SIR model assumes homogenous mixing and a fixed population size N = S (t) + I (t) + R (t) where S(t), I (t) and R(t) are the numbers of the population who are susceptible, infections and removed at time t. Each contact between susceptible and an infections patient has a probability, P, of leading to transmission and contacts occur at a rate, C per day. Following a model proposed by [12] to explain the frequent rapid rise of Ebola virus disease in West Africa on observed frequently in epidemics such as the London cholera epidemic in 1865. We are able to propose a model that approximates the outbreak reasonably well. The classical system of ordinary differential equations (ODEs) is

$$\frac{dS}{dt} = -\frac{CpSI}{N}$$

$$\frac{dI}{dt} = \frac{CpSI}{N} - \gamma I \quad \textbf{(2.0.1)}$$

$$\frac{dR}{dt} = \gamma I$$

Where C and γ are positive constant and $0 < P \leq I$. If one does not require a separate estimate of P and C one can use $\beta = CP$. The behavior of the system is governed by the first two equations and the number of recovered R can be calculated .The differential

equation for the number of infectious, I (t) can be rewritten then as

$$\frac{dI}{dt} = \beta I (S - \frac{\gamma}{\beta}) \quad \textbf{(2.0.2)}$$

Which leads to a critical value of the susceptible .In order for an epidemic to proceed, the number of the population that are susceptible must be greater than $\frac{\gamma}{\beta}$, and if the united value βN is less than γ, an epidemic out break will not occur. Line arising the systems about steady state (S, I, R) and putting S = S + S and I =I + i leads to

$$\begin{pmatrix} S \\ i \end{pmatrix} \begin{pmatrix} -\beta - \beta s & \\ \beta I & Bs - \gamma \end{pmatrix} \begin{pmatrix} S \\ i \end{pmatrix} \quad \textbf{(2.0.3)}$$

The dominant eigenvalue of the Jacobian at the steady state (S= N, I= 0) gives the growth rate of the epidemic curve, namely $\beta N - \gamma$. The rate of transition from susceptible to infective and of removal from infective is same as the mean field model [12]. Knowing the parameters, it will be easy to compute the solution numerically unfortunately the parameters are rarely known and a fitting has to be model if the epidemic is well described by the model.

The model takes into consideration the number of people infected due to direct contact with an infected individual at time (t) $\beta \frac{S.I}{N}$, where β = CP; P is the probability of successfully getting infected when coming into contact with an infected individual and C is the per capita contact rate. The death rate is denoted by γI, where γ is the per capita death rate. Even though recoveries do occur, we will not return these individuals to the susceptible class since there has never been a person who has recovered from Ebola and contracted the disease again in the same epidemic. The data which we are studying is the number of people that died each day during the August outbreak in West Africa (Guinea, Liberia and Sierra Leone in 2014).

TABLE: 1 Cumulative Ebola Cases and deaths as of the month of August, by country by date reported from the three countries from 22nd March - 31st August, 2014. [19]

Date	Total		Guinea		Liberia		Sierra Leone	
	Cases	Deaths	Cases	Deaths	Cases	Death	Cases	Death
31 Aug 2014	3,707	1,848	771	494	1,698	871	1,216	476
26 Aug 2014	3,069	1,552	648	430	1,378	694	1,026	422
20Aug 2014	2,615	1,427	607	406	1,082	624	910	392
18 Aug 2014	2,473	1,350	579	396	972	576	907	374
16Aug 2014	2,240	1,229	543	394	834	466	848	365
13Aug 2014	2,127	1,145	519	380	786	413	810	348
11 Aug 2014	1,975	1,069	510	377	670	355	783	334
9 Aug 2014	1,848	1,013	506	373	599	323	730	315
6 Aug 2014	1,779	961	495	367	554	294	717	298
4 Aug 2014	1,711	932	495	363	516	282	691	286
1 Aug 2014	1,603	887	487	358	469	255	646	273

Total cases = 3707
Total Death = 1848
Percentage Death = 49.9%

TABLE 2: Cumulative Ebola cases and death by date in the interval of days during the August 2014 outbreak.

Internal	Cases	Death	Percentage
1^{st} -3^{rd}	108	45	41.6%
4^{th} – 5^{th}	68	29	42.6%
6^{th} – 8^{th}	69	52	75.4%
9^{th} – 10^{th}	127	56	44.1%
11^{th} – 12^{th}	152	76	50.0%
13^{th} – 15^{th}	113	84	74.4%
16^{th} – 17^{th}	133	121	90.9% **very high**
18^{th} – 19^{th}	142	72	50.7%
20^{th} – 25^{th}	452	125	27.7%
26^{th} – 31st	632	296	46.4%

Total cases = 1914
Total Death = 956
Percentage Death = 49.9%

The second data is the total number of dead individuals at time (t), which can also be interpreted as the integral of the daily death data. From this we can compute β by solving the second differential equation for small values of t and relating it to the numbers of dead at time t since

$$\frac{dI}{dt} = \beta I \left(\frac{S-\gamma}{\beta}\right) \quad \text{or} \quad \frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I \quad \textbf{(2.0.4)}$$

For small t $\frac{dI}{dt} \approx \beta I - \gamma I$, solving the equation, I (t) = I (0) Exp [(β - γ) t], where I (0) = 1 . Under this conditions, we can assume that I(t) ∝ R $(t + \frac{1}{\gamma})$, where $\frac{1}{\gamma}$ is the average time for an infected individual to die. So Exp [(β-γ) t] = R $(t + \frac{1}{\gamma})$, where K = $\frac{1}{0.49}$ and 49.9% of infected people eventually die.
We have data that represents the total number of dead people at time t, cumulative of β(t), so we fit the data with the curve. We take natural log of the data so that over fit will be a linear fit.

$$(\beta - \gamma)t = \left(\ln \frac{1}{0.49} R\left(t + \frac{1}{\gamma}\right)\right) \quad \textbf{(2.0.5)}$$

$$= \ln \frac{1}{0.49} + \ln R\left(t + \frac{1}{\gamma}\right)$$

The slope of the line which best fits the data is 0.14 this β – γ = 0.14 = 0.14t

Note, $\ln \frac{1}{0.49} = 0$
$0.14t = \ln R\left(t + \frac{1}{\gamma}\right)$
$(\beta - \gamma)\, t = 0.14t$
$(\beta - \gamma)t = 0.14t$ } **(2.0.6)**

$$\beta = 0.14 + \gamma$$

Since the slope of this graph is so sensitive to the number of data points used in the fit, an average of these slopes is used to solve β. This average slope was taken for 10-20 data points. The average slope is 0.118. With this information we are able to calculate a range for the basic reproductive number $R_o = \frac{\beta}{\gamma}$. For our range of γ between $\frac{1}{6}$ and $\frac{1}{31}$, R_o ranges from 1.57 to 6.12. We are now ready to look at the solutions of the system of differential equations.

$$\left.\begin{aligned} \frac{dS}{dt} &= -\frac{\beta SI}{N} \\ \frac{dI}{dt} &= \frac{\beta SI}{N} - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned}\right\} \quad \textbf{(2.0.7)}$$

Now we develop a mean field host – vector – host - model with migration (vital dynamics), appropriate to the transmission characteristics of an Ebola virus. Since we only have numerical solutions, we also only view the graph of the solutions of each of these equations. The solutions plotted contain the number of

susceptible at time t, S (t), the number of infected at time t, I (t), and the number of dead at time t, R (t). We design to relate I (t) and R (t) to plot I (t) to fit $\frac{dR}{dt}$ to the best fit. Recall , $\frac{dR}{dt} = \gamma I$ so the data being considered is fitted by γI plus a shift $\frac{1}{\gamma}$ to account for the average time from infection to death. We first consider how γ varies: $\frac{1}{31} < \gamma < \frac{1}{6}$ we have taken $\gamma = \frac{1}{25}$, $\frac{1}{5}$ and $\frac{1}{31}$ and N (0) = 2000.

The respective graph shows exactly what one expects, since by increasing the period that an infected individual life, $\frac{1}{\gamma}$, there is an increase in the number of dead which the model predicts.

The next variable that we have to take into consideration is N (0). Now taking the range for these variables is attained by taking an educated guess as to who the true susceptible individuals are in the population. These individuals of course are the primary source of the infection which includes family members and public health Worker/ providers or medical staff caring for the infected individuals. This is a reasonable assumption since the only individuals who are at risk are those that held personal contact with the infected individuals. WHO reported more 240 health care providers have developed Ebola in Guinea, Sierra Leone, Liberia and Nigeria and more than 120 have died due to lack of the number of medical staff needed to manage the outbreaks, shortages of protective equipment, or improper use of what is available. The situation is chaotic and the medical response is inadequate, no proven Ebola virus specific treatment exists as of 31st August, 2014. Interventions with as – yet – unknown effects both for treatment and for prevention of Ebola, making medical management of the difficult and that they had limited capacity to safety burry bodies [16]. This work is very important in predicting the future of Ebola in West Africa.

The lowest possible value of N (0) would be about 1000 individuals since only 956 died in this August outbreak. A possible top limit for the greatest values of N(0) could be around 1914-2000, taking into consideration only the population size of families, health care workers and others livelihood in close and personal contact with infected individuals.

The model, for larger values of N (0) over estimates the number of expected individuals that will die. This outcome may give the impression that the model badly represents the data, but in reality this over estimation could be of use to health care – workers who plan for how bad an outbreak may become by knowing statistics about the first 10-20 days of the outbreak.

THE SUSCEPTIBLE – EXPOSED – INFECTIONS – REMOVED MODEL (SEIR)

We will now model the total infections that occurred during the August 2014 Ebola outbreak in Guinea, Liberia and Sierra Leone using modification to the SIR model. In this model, we will differentiate between incubation period and infectious period of the disease. As earlier described S (t) is the number of susceptible individuals at time t. we will refer to the incubation period of the disease as the latent stage this individual has acquired infection but not yet infectious. The number of the latent individual at time t will be donated by E (t). Individuals that are infected with the disease and are suffering the symptoms of Ebola will be classified as infectious individuals. The number of infectious individuals at time t will be denoted by I (t), similarly the number of death individuals at time t will be denoted by R (t). The population studies will be constant population during the outbreak that is the total population at time t will be denoted by N where

$$N = S(t) + E(t) + I(t) + R(t) \quad \textbf{(2.1.0)}$$

A simple system of ordinary differential equations (ODEs) can be used to describe the models.

$$\left.\begin{aligned} \frac{ds}{dt} &= -\beta SI \\ \frac{dE}{dt} &= \beta SI - \alpha E \\ \frac{dI}{dt} &= \alpha E - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned}\right\} \quad \textbf{(2.1.1)}$$

Where α , β, and γ are positive constants. Linearising the system about a steady state (S, E, I) taking S = S + s E = E + e and I = I + i leads to

$$\begin{pmatrix} \dot{s} \\ \dot{e} \\ \dot{i} \end{pmatrix} = \begin{pmatrix} -\beta I & 0 & -\beta S \\ \beta I & -\alpha & \beta S \\ 0 & \alpha & -\gamma \end{pmatrix} \begin{pmatrix} S \\ e \\ i \end{pmatrix} \quad \textbf{(2.1.2)}$$

The dominant eigenvalue of the Jacobian at the steady state (S = N, E = 0, I = 0) gives the growth rate of the epidemic curves.

$$\lambda = \frac{(-\gamma + \alpha) + \sqrt{(\alpha - \gamma)^2 + 4\beta N\alpha}}{2} \quad \textbf{(2.1.3)}$$

Note: The growth of the epidemic is dependent on the rate of transmission from the latent infectious period α. The rise in early epidemic curveλ, is easily calculated during the outbreak. Inferences regarding β based on the value of λ will be highly sensitive to α which is often taken to be the reciprocal of the mean duration of the incubation period. Model of such, implicitly assume that E and I are negative exponentially distributed parameters α and γ respectively. [11] Showed that assumption of the critical community size, the size necessary to sustain endemic transmission, for measles. The over – production or overestimation earlier observed in the S-I –R model of the fade outs that occurred in standard (exponential infectious periods) model was corrected. By allowing the infectious periods be normally distributed in the line with observed infections period distributions. Estimates of infectivity, particularly those based on the early epidemic curve are also highly sensitive to the shape of the survival curve in the exposed and infections compartments. [13]. [7] showed that the latent period for SARs is not exponential [13] showed that non – exponential compartmental sojourn times lead to more realistic model predictions for the SIR model.

Now, to better understand the non – exponential compartmental predictions of Ebola, we choose to design and modify the SIR model as follows.

$$\left.\begin{aligned} \frac{ds}{dt} &= -\frac{\beta S}{N}(I + qE) \\ \frac{dE}{dt} &= \frac{\beta S}{N}(I + qE) - \boldsymbol{\delta}\delta E \\ \frac{dI}{dt} &= \delta E - \gamma I \end{aligned}\right\} \quad \textbf{(2.1.4)}$$

$$\frac{dR}{dt} = \gamma I \quad - \quad - \quad -$$

This model takes into consideration the number of people infected due to direct contact with infected and latent individuals. $\beta S\frac{(I+qE)}{N}$. In this model $\beta = CP$ where P is the probability of successful getting infected when coming into with and infected individual, and C is the per – capita content the parameter $q(0 \leq q \leq I)$ is a weight factor added to the model since it is known that a susceptible individual has a higher chance of getting infected from an infections individuals then infected from an infectious individuals than from a latent individual [4],

The individual in the latent stage eventually shows the symptoms of the disease and pass on to the infections stage. The outbreak began in Guinea in December 2013, but was not dictated until March 2014.The latent stage is denoted by δE, where δ is the per capita infectious rate .Then $\frac{1}{\delta}$ becomes the average time for the latent individual to become infectious, this will be denoted by γI,where γ is the per –capita death rate. Then $\frac{I}{\gamma}$ becomes the average time it takes and individual to die once entered into the infectious stage. As before death and recovery are taken to be the same, since there has not been a case in which a person who survived Ebola contacts the disease again. This shows the number of people who became infected each day during the outbreak in Guinea, Liberia and Sierra Leone, August, 2014. From this data we can now calculate βusing a similar method to the one in the previous model. To do this, we first make assumptions.

Assumption 1: In the beginning of the epidemic
N (t) = S (t)

Assumption 2: Initially, there is a constant number of individuals infected. Those individuals infect other individuals who become latent. It takes $\frac{I}{\delta}$ days for the latent individuals to become infectious. Therefore, for the first $\frac{I}{\delta}$ days, the rate of change of the infectious individuals is 0 (i.e. $\frac{dI}{dt} = 0$).

Assumption 3: In order for an individual to become infectious, they must pass through the latent stage. Thus, the data for the latent stage is the same as the data for the infectious stage, the only difference being that the latent stage data occurred $\frac{I}{\delta}$ days before. Since $\frac{I}{\delta}$ is the average time it takes for a latent individual to become infectious, and the latent stage ranges from 2 to 21 days, we choose $\frac{I}{\delta}$ = 15. Similarly, since $\frac{I}{\gamma}$ is the average time it takes for an infectious individual to die, and of the infectious stage ranges 4 to 10 days, $\frac{I}{\gamma}$ =7. Thus, we then look at the following equations to estimate β:

$$\frac{dE}{dt} = \frac{\beta S}{N}(I + qE) - \delta E$$

$$\Rightarrow \quad \frac{dE}{dt} = \beta(I + qE) - \delta E \quad (*)$$

By the first assumption;

(2.1.5)

$$\Rightarrow \quad \frac{dI}{dt} = \delta E - \gamma I = 0 \Rightarrow I = \delta E/\gamma$$

By the second assumption.

If we substitute I into $*$, then $\frac{dE}{dt}$ =$[\beta(\frac{\delta}{\gamma} - \delta)]E$.

(2.1.6)

The information for $\frac{dE}{dt}$ is given by the daily infection data; the information for E is the cumulative of the infection data. Thus, we have a linear relationship, and we can estimate the slope by doing a linear fit. Using Mathematica and the data for the first 15 days, we obtain the fit where equation of the line is 0.3412 t. Thus, we now have the slope of the best fit line, and $\beta = \frac{0.3412 + \delta}{\frac{\delta}{\gamma} + q} =$ 0.49949,if we Take q = 0.35 and the values of δ *and* γ given above. The choice for q is arbitrary and is picked so that the model best fits the supplies data.

Another important number that needs to be computed is the basic reproductive number, R_O. This number tells us how fast the disease will spread at the beginning of the matrix of the system of equations need to be considered. It is easy to show that the disease – free state is (S,E,I,R) = (N,0,0,0). Once the Jacobian is evaluated at this point, the determine and the trace must both be greater than zero to ensure that the disease – Free State is an unstable fixed point. Once all of this is accomplished, we obtain a value for R_o.

THE BASIC AND THE EFFECTIVE REPRODUCTION RATIO, R_O

The Basic reproduction rate (R_o) is defined as "The average number of persons directly infected by an infectious case during its entire infectious period after entering a totaling susceptible population" [10] R_o is a function of daily infectivity and expected duration of infectivity. The effective reproduction ratio, R_e is the expected number of person directly infected by an infectious case without the assumptions of a fully susceptible population. Two key parameters describing the spread of an infectious and the basic and effective reproduction number R_o and R_e, which are defined as the number of secondary infectious generated by an infected under case in the absence and presence of control intervention. If Re drops below unity, the epidemic eventually stops. Several studies have fitted mathematical models to date from preventing outbreak of the germs Ebola virus [2]. Previous estimate of Ro from outbreaks in Congo (1995) and Uganda (2000) range from 1.3 – 2.7 [5] It will be important to know the reproductive numbers of the current Ebola outbreak in West Africa. This will facilitate making projections and allow comparisons of the effects of control measures in each country. In this study we describe the 2014 EBOV epidemic using SIR and SEIR models. Fitting the models to the most recent data about reported cases and death in Guinea, Sierre Leone and Liberia provided estimates of the reproduction numbers of Ebola in absence and presence of control measures. In the SIR or SEIR model, with constant hazard of transmission between compartment and constant infections, the $R_e = \frac{\beta SI}{\gamma}$ when the entire population is susceptible (S (0) = N), this expression gives the $R_o = \frac{\beta N}{\gamma}$.

A more general expression could be obtained when the infectivity period and the transit time over the infectious period is not necessarily negative exponential

$R_o = \int_o^\infty C(\tau)P(\tau)q(\tau)d\tau$, **(3.0.1)**

Where τ is the time since transmission occurred to an individual and q (τ) is the probability of remaining infectious, C (τ) is the contact rate and P (τ) the probability of transmission per contact, a time period τ from infection. In further work we will use Bayesian inference to estimate the changes in infectivity over the course of Ebola infection.

But for now, as deduced from the system of equations.

$R_o = (\frac{\beta}{\gamma})(I + \frac{q\gamma}{\delta}) = 6.12$ **(3.0.2)**

SUGGESTIONS FOR FUTURE RESEARCH

The models presented in this research used a constant effective rate, β. This is may not be the best model for β since the probability of contracting the Ebola virus varies as the disease becomes more widespread. People are more careful with whom they have contact. Thus the number of contacts decreases as time elapses or as the number of infected increases. Therefore, it makes sense to have β decrease. Another idea for enhancing the model is to consider quarantine. When infected people are isolated, the numbers of contacts that can transmit the disease decrease. This smoothing that could be taken into account in future research with these models. More research needs to be conducted to estimate N, the total population. A good number for N is very important, since as it varies the accuracy of information that may prove helpful in estimating N includes the number of staff members in hospitals, family size, and other data that may help determine the total susceptible at the beginning of an Ebola outbreak.

Research on q is also essential. It is intuitively clear that individuals showing symptoms of the Ebola disease are more infectious than latent individuals who show no symptoms. Therefore, a better value for q would make the model more accurate in predicting the dynamics of a future Ebola outbreak in West Africa.

TABLE 3: Major outbreak in some Africa countries since Ebola was first discovered 1976[19]

Location	Date	Cases	Death	Percentage of death
Democratic Republic of Congo	1976	318	280	88%
Sudan	1976	284	151	53%
Democratic republic of Congo	1999	315	250	79%
Uganda	2000-2001	425	224	53%
Democratic republic of Congo	Dec 2002-April 2005	143	128	90%
Democratic republic of Congo	2007	265	187	71%
Uganda	Dec 2007-June 2008	131	42	33%

Note: Democratic Republic of Congo was Named Zaire when the outbreak occurred

Source: Centres for Disease Control (CDC) and prevention. Ebola was 1st discovered in Zaire in 1976 here the reported cases and death in the biggest Ebola outbreaks since then.

As of 6th September, 2014 another outbreak in the Democratic republic of Congo, where has 59 confirmed cases and 32 deaths is believed to be unrelated to the West Africa outbreak [20].

CONCLUSION

In our model, people are susceptible, infected or recovered. We calculated R_o values from 1.57 – 6.12 meanwhile the range of values for Ebola virus disease is $1.72 \leq R_o \leq 8.60$ R_o earlier computed for the 1976 epidemic in Yambuku in Zaire ranged from $2.6 \leq R_o \leq 5.03$. This is to say our results makes sense since it shows that Ebola patients were infected more in August, 2014 than in the previous months during the outbreak. The England and Wales measles outbreak 1950-68 R_o, ranged 16 – 18, HIV in Hampara 1985-7 ranged 10-11 as earlier reviewed. This study predicts the transmissibility of an agent increases via contact rate as the value of infection control reduces.

Furthermore, this result assumed that the time scale of the outbreak is short and the population size N= 2000 was chosen by design to depict clearly the method of Ebola transmission and therefore making the model more accurate in predicting the dynamics of a future Ebola outbreak. These models are very important because they are put as upper bounds on the number of deaths, and this can help researchers and health care

providers plan for the later part of an outbreak by calculating the parameters from the date at start of the epidemic.

In summary, the number of deaths can also be minimized by lowering the β. This can be achieved by implementing intervention controls such as vaccination, quarantine, washing of hands with sanitizers, and others. We noted, the rate at which the transmission is going, if viable and extremely aggressive intervention control is not employed immediately in the nearest future many will suffer death in West Africa and the neighboring regions. Sure the outbreak will be disastrous. This result will be used to develop programs that will minimize the effective contact rate. This study can be improved / extended in various ways, including understanding the non – linear mode of the transmission, nosocomial pathogens in hospital setting, public health cure intervention policy in reducing effective contact rate in this case by significantly reducing Ebola infection. We must quickly act to eliminate or drastically reduce Ebola virus before it will eliminate us in Africa.

The Key difference between the models presented here and other models of infectious disease is that it predicts with precision the upper bound of deaths at a given time t. This demonstrates the flexibility of mathematical models for an unusual outbreak and shows how mathematical models can respond quickly to a wide variety of challenges in epidemiology. Inaccuracies in the model are to be expected since the parameters dictating the behaviour of the model are obtained from only a few data points. There have been so few amounts of data available. This model's precision is dependent on this limitation. Sensitivity analysis on the transmission dynamics of Ebola in hospitals nosocomially and the endemic equilibrium state of our model will be discussed in a subsequent paper.

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