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# MATHEMATICAL MODELING OF THE SPREAD OFTHE EBOLA VIRUS DISEASE

**C. E. Akpan[1]*, U. M. Yusuf[1], I. M. Saleh[2], B. T. Enoch[1] and M. M. Yalwa[3]**
[1] *Department of Mathematics, Federal University of Lafia, Lafia, Nasarawa State, Nigeria*
[2]*Department of Statistics, Federal University of Lafia, Lafia, Nasarawa State, Nigeria*
[3]*Department of Basic Science, College of Agriculture, Science & Technology, Lafia, Nasarawa State, Nigeria*
***Corresponding email:** collins.ekpan@science.fulafia.edu.ng*

## ABSTRACT
Ebola, a viral and fatal disease with occasional outbreaks on the continent of Africa affects mostly humans and non-human primates, and poses a great health challenge to this part of the globe. The transmission of Ebola Virus can be through direct contact with blood, bodily fluids, or skin of Ebola Virus Disease patients or those who have passed away from the illness. In this paper, we formulate a mathematical model of the transmission of the Ebola virus disease in a population capturing its dynamics to study the impact of healthcare policies on its spread. The model is a four compartment model consisting of Susceptible, Latent, Infectious and Recovery compartments. To gain a good understanding of the model, the formulated model is transformed into difference equations. The basic reproduction number $R_o$ is derived using the next generation matrix method. Further, the disease-free equilibrium of the model is obtained and its stability analysis is carried out. The result shows that the disease-free equilibrium point is locally stable if $R_o < 1$ but may not be asymptotically stable, indicating that the disease will eventually die out. Conversely, if $R_0 > 1$, an endemic equilibrium exists, and the disease will persist at a stable level. Numerical simulations obtained illustrate the efforts of the parameters on the compartment of the model.

**Keywords:** Difference equation method, Ebola, mathematical modeling and reproduction number

## INTRODUCTION
Ebola Virus Disease (EVD) is a deadly disease with occasional outbreaks that occur mostly on the African continent. EVD most commonly affects people and non-human primates (such as monkeys, gorillas and chimpanzees). It is thought that fruit bats of the pteropodid family are natural Ebola virus hosts. Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals. It then spreads through human-to-human transmission via direct contact (through broken skin or mucous membranes). The study by Osemwinyen (2015) carries it that outbreaks of the disease are often traceable to a single index case where an individual has handled the carcass of a gorilla, chimpanzee, or duiker or the bush meat trade (the catching and eating of wild animals, including primates such as gorillas and chimpanzees). Once in the human population, transmission through physical contact with body fluids like blood, secretions, tissues or semen from infected persons becomes highly possible especially within families and hospital personnel with patients. Transmission can also be as a result of indirect contact with surfaces and materials, for example, bedding, clothing, and floor areas, or objects such as syringes, contaminated with the aforementioned fluids (Ngwa & Teboh-ewungkem, 2016).

The Ebola virus was first identified in 1976 near the Ebola River infecting at least 280 people, and there have been several outbreaks of Ebola virus disease (EVD) over the years. However, none of those were as serious as the recent outbreak in West Africa, which started in March 2014 and affected the whole world.On March 23, 2014, the Ministry of Health in Guinea notified the World Health Organization (WHO) of a rapidly evolving outbreak of Ebola virus disease (EVD), now believed to have begun in December 2013. The epidemic spread through West Africa and reached Europe and the United States. As of November 4, 2015, WHO reported more than 28,000 cumulative cases and 11,000 deaths in Guinea, Liberia, and Sierra Leone, where transmission had been most intense (Diane & Njankon, 2019; Li, 2016; Chretien *et al.*, 2015).
The epidemic was more pronounced in these countries due to socioeconomic disadvantage and health system inadequacies bedeviling the countries. The researches of Rachah and Saidi (2021), Nazir *et al.* (2020), Mhlanga (2019) and Berge *et al.* (2017) support this submission as they presented in their works that the survival of the Ebola virus in the environment, due to poor hygienic and sanitary conditions, is probably another source of Ebola infection in many places in Africa. They continued by stating that in Africa, and particularly in the regions that were affected by Ebola outbreaks, people live close to the rain-forests, hunt bats and monkeys, and harvest forest fruits for food. As

part of their tradition and customs, Africans are warmhearted to the extent that even a contagious disease would not stop them from caring for their relatives at home, kissing themselves and shaking hands. Furthermore, during funerals, they wash and dress up their deceased relatives. They share, without proper washing clothes of their deceased relatives. The huge gatherings of people from surrounding villages, towns and cities are most likely among the major factors for the quick spread of the Ebola virus infection in the region.

According to Tae and Young (2015), the recent outbreak started from a 2-year-old boy who was infected by a bat and then it spread through human-to-human transmission via direct contact with bodily fluids of infected people and with surfaces and materials contaminated with these fluids. The Centers for Disease Control and Prevention say that only mammals have shown the ability to spread and become infected with Ebola. That is why health care workers have frequently been infected while treating patients with suspected or confirmed EVD. It has not been proved that Ebola can spread among humans via airborne transmission, although Ebola goes airborne from pigs to monkeys.

Multiple species have been identified, but the recent outbreak was caused by the Zaire species. The Ebola virus is a virological taxon species of the genus Ebola virus. The genus Ebola virus belongs to the virus family Filoviridae (filo virus) alongside with genus Marburg virus and genus Cuevavirus. Genus Ebola virus comprises five distinct species as posited by the World Health Organization (WHO), namely, Bundibugyo Ebola virus (BDBV); Zaire Ebola virus (EBOV); Reston Ebola virus (RESTV); Sudan Ebola virus (SUDV) and Taï Forest Ebola virus (TAFV).

Diagnosis of EVD without a laboratory test can be difficult. Since the symptoms start with fever, severe headache, muscle pain, and fatigue, the onset appears to be similar to that of flu. Progressed symptoms also cause misdiagnosis as malaria or typhoid, diarrhea, vomiting, abdominal pain, and unexplained hemorrhage follow. The delay in laboratory tests for EVD multiplies secondary infections, slows quarantine or isolation, and increases fatality (Tae and Young, 2015). Once infected, the incubation period is anywhere between 2 days and 21 days, but the average is 8-10 days. The average fatality rate is around 50%, and case fatality rates vary from 25 to 90%. Epidemiologists build rings around the virus to stop the spread of Ebola, which starts with the circle of people in direct contact with the patient. All the people in the circle are asked about their own circle of close contacts. With close observation and clear education, such as monitoring the symptoms and avoiding crowded public spaces among others, these rings are usually sufficient to stop the spread of EVD. Isolation is absolutely necessary to bring an end to the spread of Ebola. However, it is not easy to decide whether to quarantine a person or not. Quarantine is a strong control strategy, but is excessive and can be disadvantageous because many quarantined persons may turn out not to be infectious at all. Along the analogous reasoning, the World Health Organization does not recommend any ban on international travel or trade. Closing borders hinders the international community's ability to fight EVD. The World Health Organization and Center for Disease Control and Prevention recommend isolation of the infected persons and self-monitoring of exposed individuals.

The outbreak of Ebola Virus Disease in West Africa happens to be the most severe in recorded history; hence, there is a need to explore the dynamics of this disease through mathematical modeling, in order to control further outbreak of the disease in the World (Olajide, 2020). There are various modeling studies of the EVD epidemic that have been reported using a wide range of quantitative approaches and obtaining analysis of the reproduction number of Ebola outbreak. Household structured epidemic models have also provided some interesting insights of demographic determinants of Ebola epidemic risk. These mathematical models were developed for the largest epidemics reported and involved in original EVD epidemiological data and genomic data, which predicted many more cases than actually occurred, some models, produced more accurate predictions, and others yielded valuable insights. Recently, a system review and meta-analysis of 66 mathematical modeling studies of the EVD epidemic published in the peer-reviewed literature has been applied to assess these key models, data and model performance (Jiang *et al.*, 2017; Diane *et al.*, 2017).

Our study is not left out in the quest to controlling further outbreaks of this deadly disease. Our objectives are to better understand the spread of the Ebola virus, the mathematical dynamics of the disease and its preventative behaviors by creating a mathematical model. We create an epidemiological model with a system of nonlinear differential equations, and the model examines the dynamics of the system analytically and numerically. To see how closely our model describes an outbreak of EVD, we approximate parameter values of the system. Since the model is applied to the recent outbreak in Nigeria, the data set from Nigeria is used to estimate parameter values. The first Ebola case in Nigeria appeared in July 2014, and Nigeria was declared Ebola free in October 2014. Discussions and conclusions follow with the combination of analytical stability analysis and simulation of the model in the last section.

## MATERIALS AND METHODS

In this paper, the mathematical model of Ebola disease by Tae and Young (2015) and the modification is been presented to understand the epidemiology of Ebola disease, the modified model is converted to difference equation method for approximations.

The Ebola Model by Tae and Young (2015) is partitioned into five (5) compartments of total human population (Figure 1). These are susceptible humans at

time t, $S(t)$, latent individuals $L(t)$ who are infected but not infectious, infected and infectious individuals are denoted by $I(t)$ (also known as isolated individuals), $R(t)$ is for recovered number of people from Ebola disease and the individuals who died as a result of the Ebola infection are denoted by $D(t)$.
The number of birth and death rate due to other factors are neglected in Tae and Young Model, they denoted the parameter $\gamma$ to be infectivity rate between $S(t)$ and $D(t)$,$\varphi$ is the rate at which an asymptotic infected individual is isolated and treated before death occurs. $\xi$ and $\tau$ are rates at which people recover and die due to Ebola infection respectively. The rate which $L(t)$ class die of Ebola without confirmation is $\psi$. The transition rates are per capita rates on average. Tae and Young model equations are presented in equation (1) below;

$$\left.\begin{aligned} \frac{ds}{dt} &= -\beta(1-p)S\frac{L}{N} - p(\alpha_L L + \alpha_I I)\frac{S}{N} - \gamma S\frac{D}{N} \\ \frac{dL}{dt} &= -\beta(1-p)S\frac{L}{N} + p(\alpha_L L + \alpha_I I)\frac{S}{N} - \gamma S\frac{D}{N} - \varphi_L - \psi_L \\ \frac{dI}{dt} &= \varphi_L - \xi_I - \tau_I \\ \frac{dR}{dt} &= \xi_I \\ \frac{dD}{dt} &= \psi_L + \tau_I \end{aligned}\right\} \quad (1)$$

And the total human population defined by these classes or compartments is given as;

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

The reproduction number of the Ebola disease obtained by Tae and Young Model is given by

$$R_o = \frac{1}{2}\left[\frac{\beta(1-p)+\alpha p}{\varphi+\psi} + \sqrt{\left(\frac{\beta(1-p)+\alpha p}{\varphi+\psi}\right)^2 + \frac{4p\alpha\varphi}{(\xi+\tau)(\varphi+\psi)}}\right] \quad (2)$$

The authors interpret $R_o$ in terms of Ebola dynamical spread as

$$\frac{\beta(1-p)+\alpha p}{\varphi+\psi} < R_o < \frac{\beta(1-p)\alpha p}{\varphi+\psi} + \sqrt{\frac{p\alpha\varphi}{(\xi+\tau)(\varphi+\psi)}}$$

Where: $\frac{1}{\varphi+\psi}$ is the average time an infected individual stays in class$L(t)$.

Hence, the first sum of $R_o$ is the proportion of $S(t)$ who become infected. The product $(\xi+\tau)(\varphi+\psi)$ indicates individuals entering $I(t)$ after entering$L(t)$.
The $R_o$ is an indicator of how fast the disease & virus spreads or dies out, so the strategies of controlling Ebola is aimed at reducing the reproduction of the disease $R_o$.

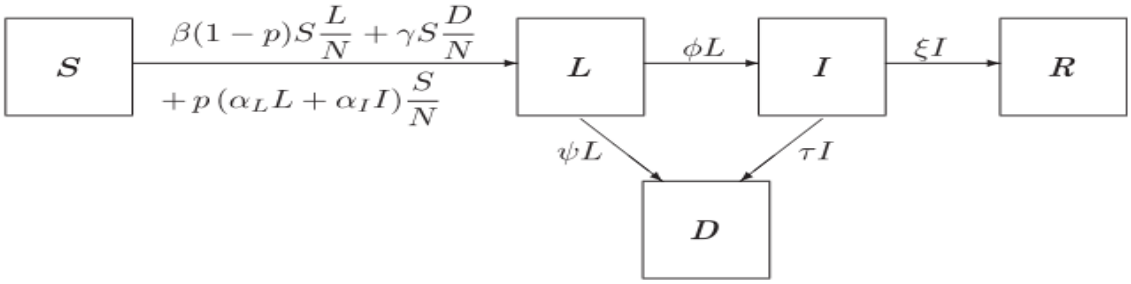


**Figure 1: Model diagram of Ebola virus by Tae and Young (2015)**

**Difference equation**

Many disease outbreaks occur in short well-defined season. It is then important to think of the virus pattern changing from season to season and therefore time is measured discretely with positive integers denoting outbreak seasons. Hence, the approach of describing the disease spread in a population is to write a suitable difference equation governing the spread of disease.
Consider the population growth-equation given as$\frac{dN}{dt} = \frac{N(t+h)-N(t)}{h}$, a linear difference equation for the differential equation of a population growth is obtained as

$\frac{N_{(t+h)}-N(t)}{h} = r\,N(t)$, where $\frac{dN}{dt} = \frac{N(t+h)-N(t)}{h}$

$N_{(t+h)} - N(t) = hrN(t)$,

$N_{t+1} = N_t + rN_t, h = 1$

$N_{t+1} = (1+r)N_t, \; t = 0,1,2,\ldots n$ (3)

Where $r$ is a constant for the growth rate discrete time t.

**Analysis of the Model**

The outbreak of an epidemic results from people who are initially infected with the virus and might have not known but are getting in contact with other people in the society. As the infected individuals keep getting contact with others, the more others are likely to be infected with the virus. Therefore, we introduce the following parameters$b$ to represent the recruitment rate into susceptible class $S(t)$ of the human population, such that $b \propto \mu N$ where μ is the natural death rate of human population. The recruitment rate $b$ is due to both birth rate and immigration of people into Ebola affected areas or regions.

Since the deterministic model solution in influence depends on the value of its parameters, we introduce ω to represent the number of people who die due to Ebola virus and exempted the $D(t)$ compartment from Tae and Young Model. We assumed that the recovery rate of Ebola infected individuals is permanent which implies that recovered persons are susceptible again to the virus.

The diagram depicting the modification is shown in Figure 2, Tables 1 – 2.

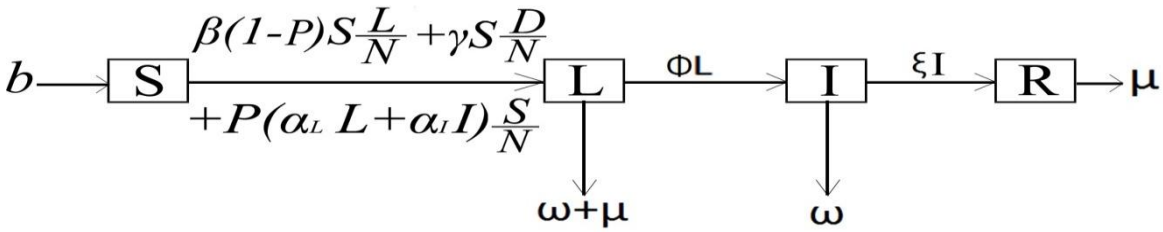


**Figure 2: Model diagram of Ebola virus**

The modified model equations are given by;

$$\left.\begin{aligned}
\frac{dS}{dt} &= b - \beta(1-p)S\frac{L}{N} - p(\alpha_L L + \alpha_I I)\frac{S}{N} - \gamma S\frac{D}{N} \\
\frac{dL}{dt} &= \beta(1-p)S\frac{L}{N} + \gamma S\frac{D}{N} + p(\alpha_L L + \alpha_I I)\frac{S}{N} - (\omega+\mu)L - \phi L \\
\frac{dI}{dt} &= \phi L - (\omega+\xi)I \\
\frac{dR}{dt} &= \xi I - \mu R
\end{aligned}\right\} \quad (4)$$

And $N(t) = S(t) + L(t) + I(t) + R(t)$ (5)

Where $\beta = p\beta * c\beta$ (probability of getting infected and per capita contact rule)

**Table 1: Model parameters and description**

| Parameter | Description |
|---|---|
| $b$ | Recruitment rate of human into $S(t)$ |
| $\beta$ | Transmission rate of Ebola |
| $p$ | Proportion of susceptible health workers |
| $\alpha_L$ | Probability of health workers getting infected in $L(t)$ |
| $\alpha_I$ | Probability of health workers getting infected in $I(t)$ |
| $\varphi$ | Isolated and Infectious individuals in treatment |
| $\omega$ | Individuals who die due to Ebola infection |
| $\xi$ | Individuals who recovered from Ebola infection |
| $\mu$ | Natural death rate of human population |

**Table 2: Model variables and description**

| State Variables | Description |
|---|---|
| S(t) | Susceptible human to Ebola disease at time t |
| L(t) | Latent individuals who are infected but infectious at time t |
| I(t) | Infected and Infectious individuals who are isolated for treatment at time t |
| R(t) | Individual who recovered from Ebola disease at time t |

**Conversion of model equations to difference equations**

In this section, we convert the modified model (4) into difference equations in order to obtain the approximate solution of the model. The conversion results are given below.

$$S_{t+1} = S_t + hb - h\beta(1-p)L_t\frac{S_t}{N} - hp(\alpha_L L_t + \alpha_I I_t)\frac{S_t}{N} - h\mu S_t$$

$$L_{t+1} = L_t + h\beta(1-p)L_t\frac{S_t}{N} + hp(\alpha_L L_t + \alpha_I I_t)\frac{S_t}{N} - h(\mu+\omega+\varphi)ht$$

$$I_{t+1} = I_t + h\varphi L_t - (\xi+\omega)hI_t$$

$$R_{t+1} = R_t + h\xi I_t - h\mu R_t$$

At $t = 0, 1, 2 \ldots n$ and where $h$ is the step size of the discretize time in which $h \geq 0$.

**Reproduction number of the modified model**

To obtain the reproduction number, $R_o$ of the disease model, we applied the next generation matrix method. We derived two matrices from the infectious classes of the model, according to the next generation matrix method; matrix $A_1$ stands for appearance of new infections in the population class $L$ and $I$, and matrix $A_2$ stands for the transfer of individuals between the partial derivatives of the right-hand side of the model equation (4) at disease free equilibrium point $E_o = \left(\frac{b}{\mu}, 0,0,0\right)$

$$A_1 = \begin{bmatrix} \dfrac{b\beta(1-p)+\alpha_L p}{\mu N} & \alpha_I P \\ \varphi & 0 \end{bmatrix}, \qquad A_2 = \begin{bmatrix} \mu+\omega+\varphi & 0 \\ 0 & \xi+\omega \end{bmatrix}$$

Next, we obtain $|A_1 A_2^{-1} - \lambda I| = 0$ (Carley Helmitton)

$$A_2^{-1} = \begin{bmatrix} \dfrac{1}{\mu+\omega+\varphi} & 0 \\ 0 & \dfrac{1}{\xi+\omega} \end{bmatrix}$$

$$A_1 A_2^{-1} = \begin{bmatrix} \dfrac{b\beta(1-p)+\alpha_L p}{\mu N} & \alpha_I P \\ \varphi & 0 \end{bmatrix} \begin{bmatrix} \dfrac{1}{\mu+\omega+\varphi} & 0 \\ 0 & \dfrac{1}{\xi+\omega} \end{bmatrix}$$

$$A_1 A_2^{-1} = \begin{bmatrix} \dfrac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N} & \dfrac{\alpha_I P}{\xi+\omega} \\ \dfrac{\varphi}{\mu+\omega+\varphi} & 0 \end{bmatrix}$$

$$\therefore, \qquad |A_1 A_2^{-1} - \lambda I| = 0 \rightarrow \begin{bmatrix} \dfrac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N} - \lambda & \dfrac{\alpha_I P}{\xi+\omega} \\ \dfrac{\varphi}{\mu+\omega+\varphi} & -\lambda \end{bmatrix} = 0$$

$$\lambda^2 - \left(\frac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N}\right)\lambda - \frac{\alpha_I \varphi p}{(\varphi+\mu+\omega)(\xi+\omega)} = 0 \qquad (6)$$

The equation (6) is a quadratic equation in terms of $\lambda$.

$$\therefore, \qquad \lambda = \frac{-b \pm \sqrt{b^2-4ac}}{2a}$$

$$a = 1, \qquad b = -\left(\frac{\beta(1-p)+\alpha_L p}{\mu+\omega+\varphi}\right) \text{ and } c = \frac{-\alpha_I p}{(\varphi+\mu+\omega)(\xi+\omega)}$$

$$\lambda = \frac{1}{2}\left[\frac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N} \pm \sqrt{\left(\frac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N}\right)^2 - \frac{4p\varphi\alpha_I}{(\varphi+\mu+\omega)(\xi+\omega)}}\right]$$

Since the reproduction number should not be negative, we omit $(-)$ of$(\pm)$.

$$\therefore, \qquad \lambda_1 = \frac{1}{2}\left[\frac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N} + \sqrt{\left(\frac{b\beta(1-p)+\alpha_L p\mu N}{(\mu+\omega+\varphi)\mu N}\right)^2 - \frac{4p\varphi\alpha_I}{(\varphi+\mu+\omega)(\xi+\omega)}}\right]$$

Where, $\lambda_1 = R_o > 1$ which is asymptotically unstable at endemic equilibrium.

**Local stabilty of the model**

To establish the stability analysis of the modified Ebola model, we verify the stability condition in the following theorems.

**Theorem:** The disease-free equilibrium point $E_o$ is locally and asymptotically stable if the reproduction number of the modified model $R_o < 1$ and unstable if the$R_o > 1$.

**Proof:** The Jacobian matrix of the modified Ebola model is obtained and is given as;

$$J_{E_o} = \begin{bmatrix} -\mu & \frac{-\beta(1-p)b-p\alpha_L b}{\mu N t_o} & \frac{-\alpha_I pb}{\mu N t_o} & 0 \\ 0 & \frac{\beta(1-p)b+p\alpha_L b}{\mu N t_o} - (\mu+\omega+\varphi) & \frac{\alpha_I pb}{\mu N t_o} & 0 \\ 0 & \varphi & -(\xi+\omega) & 0 \\ 0 & 0 & \xi & -\mu \end{bmatrix} \qquad (7)$$

The matrix (7) is obtained by differentiating the RHS of equation (4) at the disease-free equilibrium point $E_o = (S(t_o), L(t_o), I(t_o), R(t_o)) = \left(\frac{b}{\mu}, 0,0,0\right)$

The matrix (7) has the eigenvalues $e_{1=}e_2 = -\mu$
The remaining eigen values can be obtained by calculating the determinant

$$det\begin{bmatrix} \frac{\beta(1-p)b + p\alpha_L b}{\mu N t_o} - (\mu + \omega + \varphi) & \frac{\alpha_I pb}{\mu N t_o} \\ \varphi & -(\xi + \omega) \end{bmatrix}$$

By Carley Hemitton theorem, this implies that,

$$det\left(\begin{bmatrix} \frac{\beta(1-p)b + p\alpha_L b}{\mu N t_o} - (\mu + \omega + \varphi) & \frac{\alpha_I pb}{\mu N t_o} \\ \varphi & -(\xi + \omega) \end{bmatrix} - Ie\right) = 0$$

$$e^2 - \left(\frac{\beta(1-p)b+p\alpha_L b}{\mu N t_o} - (\mu + \omega + \varphi) - (\xi + \omega)\right)e - \left[\left(\frac{\beta(1-p)b+\alpha_L pb}{\mu N t_o} - (\mu + \omega + \varphi)\right) \times (\xi + \omega) + \frac{\alpha_I pb}{\mu N t_o}\varphi\right] = 0 \quad (8)$$

Equation (8) is a quadratic equation in terms of independent variable 'e' (eigenvalues) to be determined. If the $e_3$ and $e_4$ obtained from (8) are negative, it implies that the $R_o < 1$ otherwise $R_o > 1$. Thus, the modified Ebola model (4) is locally stable if $R_o < 1$.

**Discretized Ebola model parameters estimation**

The simulation of the discretized model (5) is carried out using Maple 17 with our conservative estimates and the reported data of confirmed cases of Ebola in West Africa by WHO as recorded in the Ebola mathematical model of Rachah and Torres (2015) and Ebola virus transmission model in Sierra Leone by Li *et al.* (2020). The parameter values derived from the mentioned authors worked for our model simulation are shown in Table 3.

**Table 3: Modified Ebola model parameters**

| S/N | State Variables and Parameters | Value | Reference |
|---|---|---|---|
| 1 | N | 6348350 (Sierra Leone, 2020) | Li *et al.* (2020) |
| 2 | $S(0)$ | 460 | Amira *et al.* (2015) |
| 3 | L(0) | 100 | Estimated |
| 4 | $I(0)$ | 12 | Amira *et al.* (2015) |
| 5 | $R(0)$ | 0 | Amira *et al.* (2015) |
| 6 | $b$ | 30 | Estimated |
| 7 | $\beta$ | 0.09162 [0.1173,1.1247] | Amira *et al.* (2015) |
| 8 | $p$ | 0.1674 [0.0623,1.0713] | Li *et al.* (2020) |
| 9 | $\varepsilon$ | 0.0075 [0.005, 0.01] | Amira *et al.* (2015) |
| 10 | $\varphi$ | 0.023 | Li *et al.* (2020) |
| 11 | $\alpha_L$ | 0.0623 [0.0623,1.0713] | Li *et al.* (2020) |
| 12 | $\alpha_I$ | 0.1560[0.0623,1.0713] | Li *et al.* (2020) |
| 13 | $\omega$ | 0.0062 [0.00455, 1.1318] | Li *et al.* (2020) |
| 14 | $\mu$ | 0.0000315 | Estimated for Sierra Leone (2020) |

**Discretized model simulation in maple**

The discretized model (3) simulation is carried out using Maple 17 with the state variables, initial values and the parameters estimation as shown in Table 3.

**RESULTS AND DISCUSSION**

The numerical values of the discretized Ebola model generated using Maple 17 is presented in Table 4.

**Table 4: Impacts of Ebola epidemics**

| Step size $(h)$ | $t$ (months) | Susceptible human Class $S(h)$ | Latent human class $L(h)$ | Infectious human class $I(h)$ | Recovered human class $R(h)$ |
|---|---|---|---|---|---|
| 0 | 0 | 460.0000 | 100.000 | 12.0000 | 0.0000 |
| 0.1 | 1 | 460.0010 | 100.010 | 12.0100 | 0.0001 |
| 0.2 | 2 | 462.9985 | 99.7077 | 12.0656 | 0.0090 |
| 0.3 | 3 | 468.9954 | 99.1250 | 12.3590 | 0.0271 |
| 0.4 | 4 | 477.9908 | 98.2559 | 12.8736 | 0.0549 |
| 0.5 | 5 | 489.9845 | 97.1073 | 13.6012 | 0.0935 |
| 0.6 | 6 | 504.9765 | 95.6883 | 14.5316 | 0.1445 |
| 0.7 | 7 | 522.9665 | 94.01045 | 15.6530 | 0.2099 |

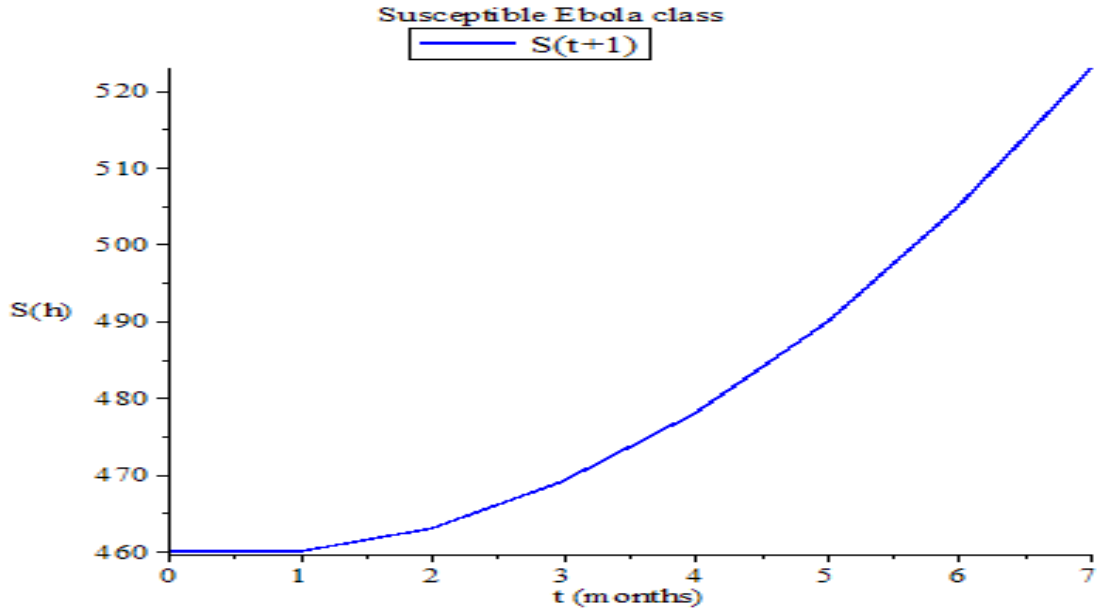


**Figure 3: Susceptible human class impacts of the Ebola epidemics in seven months**

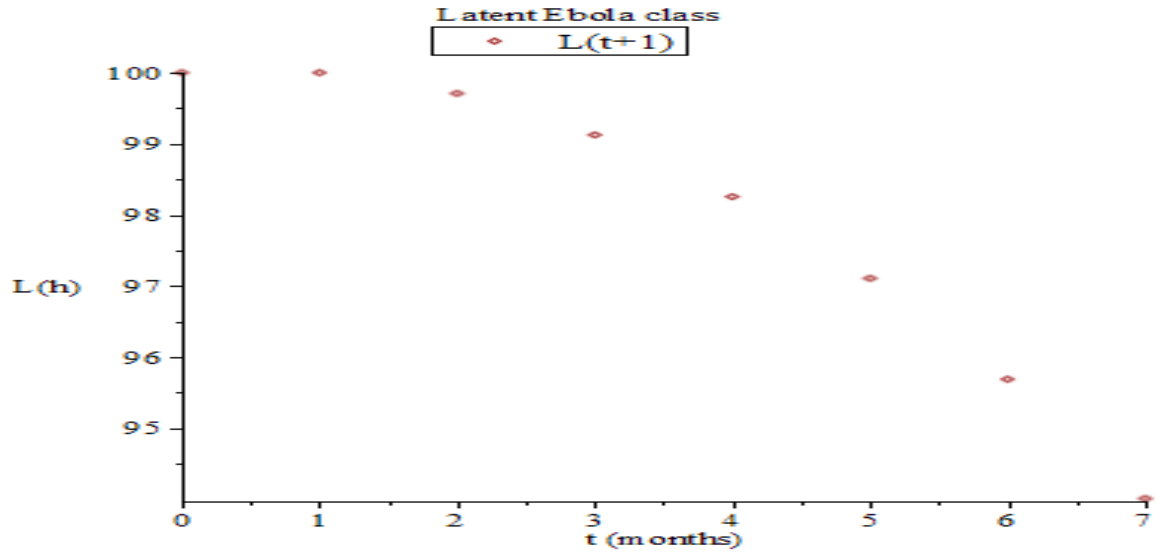


**Figure 4: Latent human class impacts of the Ebola epidemics in seven months**

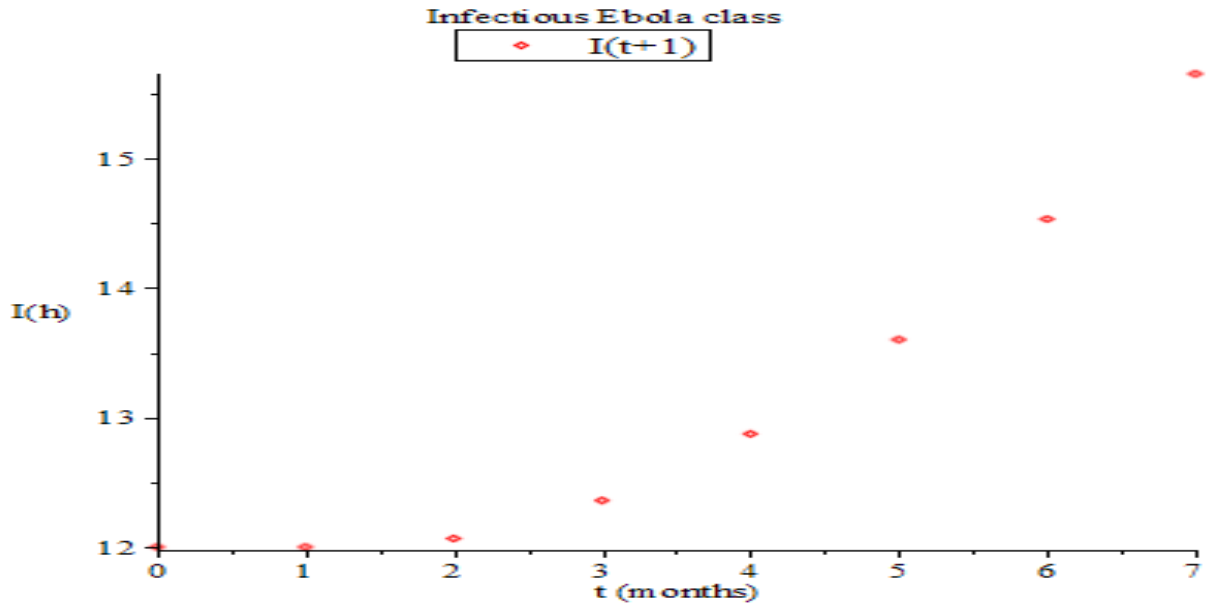


**Figure 5: Infectious human class impacts of the Ebola epidemics in seven months**

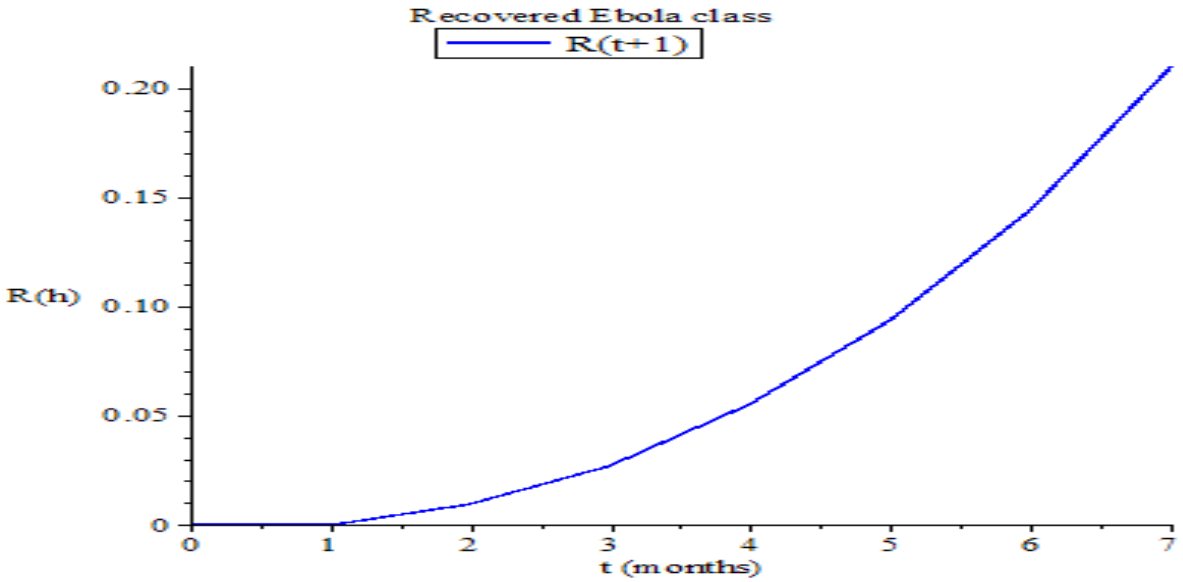


**Figure 6: Recovered human class impacts of the Ebola epidemics in seven months**

**Discussion of numerical results**

**Experiment 1:**

From Figure 1, more people in the area of Ebola outbreak will become susceptible to the epidemics as time increases due to lack of unawareness and proper enforcement of policy that may restrict people's movement in the region.

**Experiment 2:**

The Figure 2 above gives an indication that the initial number of people who are not infectious but can spread disease will gradually reduce if people become more aware and there is enforcement of policy that can restrict people's movement in the region.

**Experiment 3:**

From Figure 3, the quarantine or Isolation Health Centers for people who are exposed to the disease and the infectious individuals will have more people as policies are been enforced to ensure no further spread of the disease in the region.

**Experiment 4:**

The Figure 4 above is an indication that if proper medical attention is available and administered to the Latent and Infectious people at the quarantine or isolation centers, there is likelihood of people recovering from the disease though at some minimal rate as shown in Table 4.

## CONCUSION

In this paper, a deterministic Ebola model by Tae and Young, 2015 was modified. The modification model has additional parameter $b$ which stands for the rate of travelers and new born children in the region where the Ebola epidemics is suspected, $\mu$ denoted the natural death rate of people in the region and the parameter $\omega$ stands for the death rate of people who die as a result of Ebola virus. The modified model stability analysis shows that, the model is stable if the reproduction number,$R_0$ is less than one ($R_0 < 1$) and is unstable if$R_0 > 1$. We further discretized the modified model in order to simulate the impacts of the diseases in the case of an outbreak in West Africa region. The numerical results obtained with the situated graphs shows that, if there is no proper awareness about the outbreak of the disease and enforcement of policies to curtail the disease, there will be a drastic impact of the disease in the region. And in the event that there are policies and their enforcement are in place, there is good argument by our numerical simulation that, the rate at which people will get contact with the exposed individuals to the disease (Latent class) will reduce and the infectious people under medical observation may likely recover.

**Conflict of interest:** The authors declare no conflict of interest.

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