

Mathematical Model of Transmission Dynamic of Ebola Virus Disease

Samuel Yohanna¹, M. M Adamu², A. D Hina³, Okai J. O⁴, Adejoh Jeremiah⁵

^{1,2,4,5}Abubakar Tafawa Balewa University, Bauchi, Nigeria

³Gombe State University, Gombe, Nigeria

jerry4complex@gmail.com

Article Info:

Submitted:	Revised:	Accepted:	Published:
Mar 29, 2025	Apr 13, 2025	Apr 25, 2025	Apr 30, 2025

Abstract

This study investigates the impact of treatment and vaccination on the transmission dynamics of Ebola virus disease (EVD) within human populations, as well as the effects of environmental factors on vector populations. We formulated a system of ordinary differential equations (ODEs) to model these dynamics and applied the method of linearized stability analysis to solve the equations. The stability analysis revealed that the disease-free equilibrium (DFE) states of the models remain stable when certain parameters—specifically, the treatment rate in the human population and the recovery rate in the vector population—are appropriately adjusted. Numerical simulations demonstrated that achieving a disease-free equilibrium state requires simultaneous treatment and vaccination of the population. The findings highlight the necessity of integrated intervention strategies to effectively control EVD transmission, contributing valuable insights for public health policy and future research on infectious disease management.

Keywords: Ebola Virus Disease; Treatment Dynamics; Vaccination Strategy; Ordinary Differential Equations; Disease-Free Equilibrium.

INTRODUCTION

Ebola virus disease also known as Ebola hemorrhagic fever is a rare and deadly disease caused by infection with one of the Ebola virus species. Ebola virus causes disease both in human and non – human primates. The Ebola virus have five identified species namely, Zaire Ebola virus (EBOV), Sudan Ebola Virus (SUDV), Bundibugy Ebola Virus (BDBV), Reston Ebola Virus (RESTV) and Tai Forest (Cote Divore) Ebola Virus (TUFV).

Four of these viruses (except RESTV) are known to cause Ebola virus disease in humans. In 1976, Ebola virus was first discovered near the Ebolk River in what what is now known as democratic republic of Congo, (Khan et al, 1999). Since then, there are been several outbreaks in Africa.

Human beings can get Ebola virus through direct contact (through broken skin or mucus membrane in the eyes, nose or mouth) with blood or body fluids of a person who has died from Ebola disease. Ebola virus disease (EVD) is a disease caused by Ebola virus which belongs to the Filoviridae virus family, transmitted by infected bats and from persons – to – person via direct contact with infected bodily fluid, secretions, blood contaminated environment/surface and organs, (Agustio et al 2017); Saeed et al, 2015, (WHO, 2016). In these research which we intend to extend Augusto et al, 2017) by examine the effect of the treatment of vaccination in the human population as well as effect of environmental factors on this vectors population. one can equally get the disease through objects (such as syringes and needles) that have been contaminated with the virus from a person sick with Ebola disease or the body of a person who die from Ebola disease (Center of Disease Control CDC, 2016). The 2014 Ebola disease outbreak in West Africa is related with EBOV virus. This outbreak is the largest. one in the history of disease with multiple countries affected (WHO Ebola Team Response, 2014), (Rivers et al, 2014). In their work stated that the outbreak began 23, 2014. The outbreak led to wild spread and intense transmission in West Africa countries like Guinea, Liberia and Sierra Leone, as well as slight cases in give other countries namely, Nigeria, Senegal, Mali, Spain and USA, (Marisa et al, 2014). This outbreak is considered the deadliest because it records 18, 608 suspected cases and 11, 306 deaths, (WHO, 2016). Form this, one can see that the fatality rate (0.3952), of the disease is very high and thus needed urgent scientific and research attention. In generally this viral disease has high case fatality rate which in past outbreaks varied between 25% and 90%

with an average of 50%. However, it is known that the Zaire species, that was responsible for the West Africa outbreaks carrier a higher death or fatality rate (Ahman Et al 2000).

Ebola virus has been identifies in Sativa, breast milk, stool and tears of acutely ill patients. Even when the symptoms of the Ebola disease were no longer detectable in a patients blood, the virus was still found present in breast milk. It was equally shown in some research was equally shown in some research works that the virus can be present in semen 40 days after the virus symptoms began (Ahman et al, 2021). For these reasons, people are encouraged to practice abstinence from both breastfeeding and unprotected sex immediately after recovering from Ebola virus disease (Bausoh et al, 2007), (Ahman et al, 2021). When infected with Ebola disease, the symptoms of this disease may appear as from between two to twenty one days. If an individual has no symptoms he will not be infectious.

On the issue of hos to the virus, it is speculated that orthropods, rodents and bats are host for Ebola virus (Chowell and Nishitara, 2014m Suvivan et al, 2003). Direct transmission on form reservoirs or secondary infected animals may occur. Bush animals may transfer the virus to humans when infected and humans comes in contact with their infected and infectious body fluids or when their meat is eaten by humans uncooked or under cooked. In this situation, the virus then spreads in the human population through human – to – human transmission. Airborne spread of the disease has not been detected or discovered in Ebola out breaks, (Washington state department of health, 2018). A study carried out in December, 2016 showed that the use of the vaccine of RVSV Ebola is 70% to 100% effective in protecting person against infection f the (2015), two main strategies to stop the spread of Ebola are to diagnose infectious individuals.

1.1 MODEL FORMULATION

1.2 OPERATIONAL DEFINITION OF TERMS

1. **Susceptible Individuals:** Are individuals who can be infected but have not yet contacted with the virus.
2. **Exposed Individuals:** Are individual who have contacted with the virus but not yet affected
3. **Infections Individuals:** Are individual that have contacted with the virus and can be transmit the disease to a susceptible individual

4. **Treatment Individual:** Are those individual that are treated
5. **Recovered Individual:** Are those individual that are recovered.
6. **Deceased Individuals:** Are those individual that died as a result of Ebola virus.
7. **Vaccination Individual:** are those individual that are vaccinated
8. **Immunity Individual:** Are those individual that developed permanent immunity to the Ebola virus after vaccination
9. **Susceptible Vectors:** Are those vector that can be infected but have not yet contacted with the virus
10. **Exposed Vectors:** Are those vector that have contacted with the virus but not yet affected.
11. **Infections Vectors:** Are those vector that have contacted with the virus and can be transmit the disease to human or vector.
12. **Decease Vectors:** Are those vector that died as a result of Ebola virus
13. **Recovered Vectors:** Are vector that recovered due to environmental factors.

2.0 METHODOLOGY

This section presents the existing and modified Ebola virus models and discusses the method to be used in studying the modified model. This section presents the existing and modified Ebola virus models and discusses the method to use in studying the modified model

Table 1

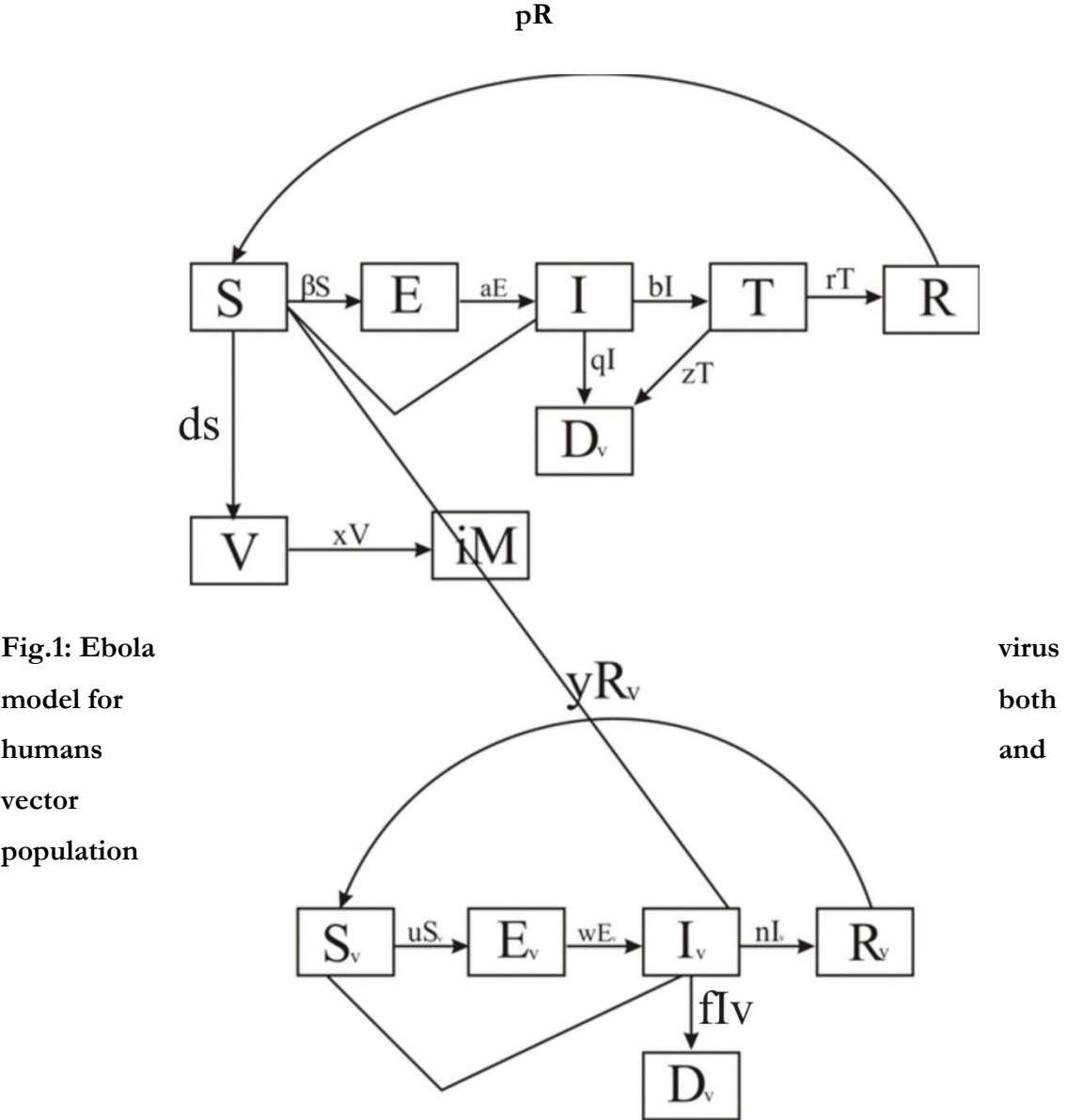
PARAMETERS/VARIABLES	DESCRIPTION
$S(t)$	The number of susceptible individual at time t .
$E(t)$	The number of exposed individual at time t .
$I(t)$	The number of Infectious individuals at time t .
$R(t)$	The number of Recovered individual at

	time t .
$D(t)$	The number of Deceased individual at time t .
$V(t)$	The number of vaccinated individual at time t ,
$Im(t)$	The number of Immune individual at time t .
$Sv(t)$	The number of susceptible vector at time t .
$Iv(t)$	The number of infectious vector at time t .
$Rv(t)$	The number of recovered vector at time t .
$Dv(t)$	The number Deceased vector at time t .
b	Transfer rate of individuals from susceptible compartment to exposed compartment
A	Transfer rate of individuals from exposed compartment to infectious compartment
b	Transfer rate of individuals form infectious compartment to treatment compartment
R	Transfer rate of individuals from treatment compartment to recovered compartment
P	Transfer rate of individuals from recovered compartment susceptible compartment
Q	Transfer rate of individuals from infectious compartment to deceased compartment

d	Transfer rate of individuals form susceptible compartment to vaccination compartment
X	Transfer rate of individuals form vaccination compartment to permanent immunity compartment
m	Transfer rate of concentration of Ebola virus in the environment compartment to susceptible vector compartment
u	Transfer rate of vector form susceptible compartment to the exposed compartment
w	Transfer rate of vector form exposed compartment to infectious compartment
n	Transfer rate of vector from infectious compartment to recovered compartment
Y	Transfer rate of vector form recovered compartment to susceptible compartment
f	Transfer rate of vector from infectious compartment to deceased compartment

**EBOLA VIRUS MODEL DIAGRAM
AND VECTOR POPULATION**

FOR BOTH HUMAN



2.2 EBOLA VIRUS MODEL FOR HUMAN AND VECTOR POPULATION

$$\begin{aligned} \frac{dS}{dt} &= hr - S(\beta \\ &+ d) \end{aligned} \quad (23)$$

$$\begin{aligned} \frac{dE}{dt} &= \beta S \\ &- aE \end{aligned} \quad (24)$$

$$\begin{aligned} \frac{dI}{dt} &= aE - I(b \\ &+ q) \end{aligned} \quad (25)$$

$$\begin{aligned} \frac{dT}{dt} &= bI - T(r + \\ &z) \end{aligned} \quad (26)$$

$$\begin{aligned} \frac{dR}{dt} &= rT \\ &- hR \end{aligned} \quad (27)$$

$$\begin{aligned} \frac{dv}{dt} &= dS \\ &- xV \end{aligned} \quad (28)$$

$$\begin{aligned} \frac{dIm}{dt} &= xV \end{aligned} \quad (29)$$

$$\begin{aligned} \frac{dD}{dt} &= qI \\ &+ zT \end{aligned} \quad (30)$$

$$\begin{aligned} \frac{dSv}{dt} &= yRv \\ &- tSv \end{aligned} \quad (31)$$

$$\begin{aligned} \frac{dEv}{dt} &= usy \\ &- wEv \end{aligned} \quad (32)$$

$$\begin{aligned} \frac{dIv}{dt} &= wEb \\ &- fIv \end{aligned} \quad (33)$$

$$\begin{aligned} \frac{dDv}{dt} &= fIv \end{aligned} \quad (34)$$

$$\begin{aligned} \frac{dRv}{dt} &= nIv \\ &- yRv \end{aligned} \quad (35)$$

2.3 MODEL ANALYSIS

The disease – free equilibrium for human population compartment are obtained by setting models equation to zero i.e

$$\begin{aligned} \frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = \frac{dD}{dt} = \frac{dV}{dt} = \frac{dIm}{dt} \\ = 0 \end{aligned} \quad (36)$$

Since $N = S + E + I + T + R + D + v + Im$ and N is constant (we assumed that the outbreak has a short time scale).

The system has four infected states, E, I, T and D and four uninfected states, S, R, V and Im . Although there are eight states in the model. It is eight dimensional as the total population size is constant.

At the infection free state

$$\begin{aligned} E &= I = T = R \\ &= 0 \end{aligned} \quad (37)$$

(Journal o the royal society, 5, Nov. 2009)

The linearization of equation, (23), (27), (28) and (29) is closed

We have the linear system

$$\begin{aligned} E^1 &= \beta S \\ &- aE \end{aligned} \quad (38)$$

$$\begin{aligned} I^1 &= aE - I(b \\ &+ q) \end{aligned} \quad (39)$$

$$\begin{aligned} T^1 &= bI - T(r \\ &+ z) \end{aligned} \quad (40)$$

$$\begin{aligned} D^1 &= qI \\ &+ zT \end{aligned} \quad (41)$$

$K_L = -T W^{-1}$ (The next generation matrix with the large domain which is same with disease – free equilibrium)

Where, K_L = Disease – free equilibrium

T = is the transmission part describing the production of new infection

$-W^{-1}$ = The transition part, describing the change of state, (including removal by death or acquisition of immunity).

From the first three equation (38), (39) and (40) we have

$$T = \begin{pmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \end{pmatrix}, \quad -W^{-1} = \begin{pmatrix} -a & 0 & 0 \\ 0 & -(b+q) & 0 \\ 0 & 0 & -(r+z) \end{pmatrix}, \quad W = \begin{pmatrix} \frac{1}{a} & 0 & 0 \\ 0 & \frac{1}{b+q} & 0 \\ 0 & 0 & \frac{1}{r+z} \end{pmatrix}$$

$$K_L = TW^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ a & 0 & 0 \\ 0 & b & 0 \end{pmatrix} \times \begin{pmatrix} \frac{1}{a} & 0 & 0 \\ 0 & \frac{1}{b+q} & 0 \\ 0 & 0 & \frac{1}{r+z} \end{pmatrix}$$

$$K_L = \begin{pmatrix} 0 & \frac{a}{b+q} & 0 \\ 0 & 0 & \frac{b}{r+z} \\ 0 & 0 & 0 \end{pmatrix}$$

The dimensional eigen value of this matrix is equal to R_o , where

$$R_o = \frac{ab}{(r+z)(b+q)} \quad (42)$$

2.4 THE DISEASE-FREE EQUILIBRIUM FOR VECTOR POPULATION ARE ALSO OBTAINED BY SETTING MODELS EQUATIONS TO ZERO I.E

$$\frac{dSv}{dt} = \frac{dEv}{dt} = \frac{dIv}{dt} = \frac{dDv}{dt} = \frac{dRv}{dt} = 0 \quad (43)$$

Since $Nv = Sv + Ev + Iv + Dv + Rv$ and Nv is constant (we assumed that the outbreak has a short time scale).

The system has three (3) infected states Ev , Iv and Dv and two (2) uninfected states, Sv and Rv .

Although there are five (5) states in the model. It is the five dimensional as the total population size is constant

At the infected – free state

$$Ev = Iv = Dv = Rv = 0 \quad (44)$$

The linearization of equations (18) is closed

We have the linear system

$$\begin{aligned} Elv \\ = tSv \\ - wEv \end{aligned} \tag{45}$$

$$\begin{aligned} Iv^1 \\ = wEv - Iv (f \\ + n) \end{aligned} \tag{46}$$

$$\begin{aligned} R^1v \\ = nIv \\ - yRv \end{aligned} \tag{47}$$

$$\begin{aligned} D^1v \\ = fIv \end{aligned} \tag{48}$$

$K_L - Tw^{-1}$ (The next generation matrix with large domain which is same with disease – free equilibrium)

Where

K_L = Disease – free equilibrium

T = is the transmission port describing the production of new infection.

$-W^{-1}$ = The transition part, describing the change of state, (including removal by death or acquisition of immunity).

From the First three equations (45), (46) and (47) we have

$$T = \begin{pmatrix} 0 & 0 & 0 \\ w & 0 & 0 \\ 0 & n & 0 \end{pmatrix}, \quad -W^{-1} = \begin{pmatrix} -w & 0 & 0 \\ 0 & -(f+n) & 0 \\ 0 & 0 & -y \end{pmatrix}$$

$$K_L = -TW^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ w & 0 & 0 \\ 0 & n & 0 \end{pmatrix} \times \begin{pmatrix} \frac{1}{w} & 0 & 0 \\ 0 & \frac{1}{f+n} & 0 \\ 0 & 0 & \frac{1}{y} \end{pmatrix}$$

$$K_L = \begin{pmatrix} 0 & \frac{w}{f+n} & 0 \\ 0 & 0 & \frac{n}{y} \\ 0 & 0 & 0 \end{pmatrix}$$

The dimensional eigen value of this matric is equal to R_0 , where

$$\begin{aligned} R_0 &= \frac{w}{f+n} \times \frac{n}{y} \\ &= \frac{wn}{(f+n)y} \end{aligned} \tag{49}$$

2.5 NUMERICAL SIMULATION FOR HUMAN POPULATION HYPOTHETICAL DATA

We assumed fixed values for b , a , q , z and vary the treatment rate, then substitute in to equation (42) we have.

Table: 2

a	b	q	r	K_L
2	3	0.1	5	0.0968
2	3	0.1	6	0.0806
2	3	0.1	7	0.0691
2	3	0.1	8	0.0605
2	3	0.1	9	0.0538

2.6 NUMERICAL SUMILATION FOR VECTOR POPULATION USING HYPOTHETICAL DATA.

We assumed fixed values of w , n , f and vary the recovered rate, then substitute into equation (49), we have

Table: 3

w	n	f	y	K_L
2	3	0.1	4	0.0915
2	3	0.1	5	0.01225
2	3	0.1	6	0.0087
2	3	0.1	7	0.0064
2	3	0.1	8	0.0043

SECTION FOUR (3)

3.0 RESULT AND DISCUSSION

Based on our numerical simulation shows that the disease dynamics can be control by both treatment and vaccination. However it is observed form our numerical simulation that with

fixed values of b , a , and q rate and increase the treatment rate r the disease-free equilibrium state is easily realized. In human population while in non-primate (Vector) with fixed values of w , n , and f rate and increase the recovered rate y the disease-free equilibrium state is easily realized. To control this transmission of the virus unachieved a disease-free equilibrium state, we need to simultaneously treat and vaccinate the population.

SECTION FIVE (4)

5.0 SUMMARY

Mathematical model for Ebola virus transmission and control was developed examined the effect of treatment and vaccination and on the human population. The first chapter deals mainly with the introduction of the disease and the biological transmission of Ebola virus disease. Chapter two (2) is literature review based on the work of various author references and equation was made. Also chapter three (3) deals with the various methods used to determine the basic reproduction number of the disease-free equilibrium and we discuss the stability point of the disease-free equilibrium. The chapter four (4) is the result of the models and discussion of the stability point based on numerical simulation.

6.0 CONCLUSTON

Based on our numerical simulation we conclude that a fixed vaccination rate and increasing value of the treatment rate the disease free equilibrium state is easily realized.

REFERENCES

- Agusto, F. B., Teboh-Ewungkem, M. I., & Gumel, A. B. (2015). Mathematical Assessment of the Effect of Traditional Beliefs and Customs on the Transmission Dynamics of the 2014 Ebola Outbreaks. *BMC Medicine*, 13(1), 1–17.
- Ahman, Q. O., & Mbah, G. C. E. (2020a). Modelling Novel Coronavirus (COVID-19) with the Relevance of Strict Movement Restriction in Italy. *Academic Journal of Statistics and Mathematics*, 6(8), 1–15.
- Ahman, Q. O., Omale, D., Asogwa, C. C., Nnaji, D. U., & Mbah, G. C. E. (2020). Transmission Dynamics of Ebola Virus Disease with Vaccine, Condom Use, Quarantine, Isolation, and Treatment Drug. *African Journal of Infectious Diseases (AJID)*, 15(1), 10–23.
- Ahman, Q. O., Omale, D., Atokolo, W., Nnaji, D. U., Ugwu, S. C., & Mbah, G. C. E. (2020). Application of Fractional Calculus to the Dynamics of Ebola Disease Combining Vaccine, Condom, Quarantine, Isolation, and Treatment Drugs as Measures. *Academic Journal of Statistics and Mathematics*, 6(11), 1–17.
- Allthaus, C. L. (2014). Estimating the Reproduction Number of Ebola Virus (EBOV) During the 2014 Outbreak in West Africa. *PLOS Currents Outbreaks*, 1, 18–36.
- Atokolo, W., Akpa, J., Daniel, M. A., Olayemi, K. S., & Mbah, G. C. E. (2020). Modelling the Impact of Optimal Control Strategies on the Dynamics of Zika Virus Disease Using the Sterile Insect Technology. *Journal of Advances in Mathematics and Computer Sciences*, 35(8), 13–33.
- Bausch, D. G., Towner, J. S., Dowell, S. F., Kaducu, F., Lukwiya, M., Sanchez, A., & Rollin, P. E. (2007). Assessment of the Risk of Ebola Virus Transmission from Bodily Fluids and Vomits. *The Journal of Infectious Diseases*, 196(S2), S142–S147.
- Bhunu, C. P. (2015). Assessing the Potential of Pre-Exposure Vaccination and Chemoprophylaxis in the Control of Lymphatic Filariasis. *Applied Mathematics and Computation*, 250, 571–579.
- Bolarin, G., & Adeboye, K. R. (2011). On the Use of Delay Differential Equation in Modelling the Rate of HIV/AIDS Infection in Nigeria. *The Journal of Mathematical Association of Nigeria (Abacus)*, 38(20), 76–86.
- Castillo-Chavez, C., & Song, B. (2004). Dynamical Models of Tuberculosis and Their Applications. *Mathematical Biosciences and Engineering*, 1(2), 361–404.
- CDC. (2015). Identify, Isolate, Inform: Emergency Department Evaluation and Management for Patients Under Investigation (PUIs) for Ebola Virus Disease (EVD). Internet: <http://www.cdc.gov/vhf/ebola/healthcare-us/emergency-services/emergencydepartments>.
- Chowell, G., & Nishiura, H. (2015). Transmission Dynamics and Control of Ebola Virus Disease (EVD): A Review. *BMC Medicine*, 12(1), 196.
- Didigwu, N. E., Mbah, G. C. E., & Bassey, E. B. (2019). Optimal Control Analysis Model of Ebola Virus Infection: Impact of Socio-Economic Status. *International Journal of Applied Science and Mathematics*, 6(6), 2394–2894.
- Gomes, M. F. C., Pastorey, Piontti, A., Rossi, L., Chao, D., Longini, I., Halloran, M. E., & Vespignani, A. (2014). Assessing the International Spreading Risk Associated with the 2014 West African Ebola Outbreak. *PLOS Currents Outbreaks*.

- Heesterbeek, J. A. P. (2014). The Role of Mathematical Models in Infectious Disease Epidemiology. *Acta Biotheoretica*, 50(3), 189–204.
- Khan, A. S., Tshioko, F. K., Heymann, D. L., Le Guenno, B., & Nabeth, P. (1999). The Re-Emergence of Ebola Hemorrhagic Fever, Democratic Republic of the Congo, 1995. *Commission de Lutte Contre les Epidémies à Kikwit. The Journal of Infectious Diseases*, 179(1), S76–S86.
- Lassalle, J. P. (1976). *The Stability of Dynamical Systems*. Regional Conference Series in Applied Mathematics. SIAM.
- Legrand, J., Grais, R. F., Boelle, P. Y., Valleron, A. J., & Flahault, A. (2007). Understanding the Dynamics of Ebola Epidemics. *Epidemiology and Infection*, 135(4), 610–621.
- Madubueze, C. E., Kimbir, A. R., & Aboiyar, T. (2018). Global Stability of Ebola Virus Disease Model with Contact Tracing and Quarantine. *Applications and Applied Mathematics: An International Journal (AAM)*, 13(1), 382–403.
- Eisenberg, M. C., Eisenberg, J. N. S., Silva, J. P. D., Wells, E. V., Cherng, S., Kao, Y.-H., & Meza, R. (2014). Modelling Surveillance and Interventions in the 2014 Ebola Epidemic.
- Onah, I. S., Collins, O. C., Madueme, P. G. U., & Mbah, G. C. E. (2020). Dynamical System Analysis and Optimal Control Measures of Lassa Fever Disease Model. *International Journal of Mathematics and Mathematical Sciences*, 2020, 1–18.
- Rivers, C. M., Lofgren, E. T., Marathe, M., Eubank, S., & Lewis, B. L. (2014). Modeling the Impact of Interventions on an Epidemic of Ebola in Sierra Leone and Liberia. *PLOS Currents*, 6(2014), 777–794.
- Saeed, R. J. M., Neger, O., Samaneh, B., Sepideh, J. M., & Seyehahmad, S. (2015). Ebola Viral Disease: A Literature Review. *Asian Journal of Tropical Diseases*, 210(4), 18–42.
- Sullivan, N., Yang, Z., & Nabel, G. J. (2003). Ebola Virus Pathogenesis; Implications for Vaccine and Therapies. *Journal of Virology*, 77(18), 9733–9737.
- Unaegbu, E. N., Onah, I. S., & Oyesanya, M. O. (2021). A Fractional Order HIV/AIDS Model Using Caputo-Fabrizio Operator. *African Journal of Infectious Diseases*, 15(2), 1–18.
- Washington State Department of Health. (2018). DOH 420–126. Last revised: March.
- World Health Organization. (2016). Ebola Data and Statistics.
- World Health Organization. (2015). Ebola Response Phase 3: Framework for Achieving and Sustaining a Resilient Zero. Geneva, Switzerland: WHO.
- World Health Organization. (2014). Ebola Virus Disease in West Africa – The First 9 Months of the Epidemic and Forward Projections. *New England Journal of Medicine*, 371(16), 1481–1495.