Int. J. Adv. Appl. Math. and Mech. 4(3) (2017) 14 – 21 (ISSN: 2347-2529)

Journal homepage: www.ijaamm.com

IJAAMM

International Journal of Advances in Applied Mathematics and Mechanics

Modeling approach to investigate the dynamics of Zika virus fever: A neglected disease in Africa

Research Article

Saul C. Mpeshe [a, *], **Nkuba Nyerere** [b], **Stephano Sanga** [a]

[a] *Department of Mathematics, University of Iringa, P.o. Box 200, Iringa, Tanzania*

[b] *Department of Mathematics and Biometry, Sokoine University of Agriculture, P.O. Box 3000, Morogoro, Tanzania*

Received 09 Septamber 2016; accepted (in revised version) 28 December 2016

Abstract: A simple SIR model of Zika virus fever (ZIKVF) dynamics has been presented in order to compute the basic reproduction number $\mathscr{R}_0$ and investigate the relative effect of each parameter in $\mathscr{R}_0$ through sensitivity analysis. Sensitivity indices indicated that $\mathscr{R}_0$ is most sensitive to the natural death rate of *Aedes* mosquitoes, and least sensitive to the recovery rate of human. This means that increasing the natural death rate of mosquitoes and the recovery rate of human will reduce $\mathscr{R}_0$ and hence, control of the disease is possible. Numerical simulation shows that ZIKVF if introduced in a system can persist because $\mathscr{R}_0 = 7.03 > 1$. Further studies are required in order to quantify the effect of ZIKVF in the society and the countermeasures to combat the disease.

MSC: 92B05 • 92D30 • 92C60 • 93D05 • 93D20

Keywords: Zika virus • zika virus fever • basic reproduction number • Metzler matrix • sensitivity analysis

1. Introduction

Zika virus fever (ZIKVF) is a viral disease caused by Zika virus (ZIKV), a member of the family *Flaviridae* and the genus *Flavivirus* [1]. Common symptoms of infection with the virus include mild headaches, maculopapular rash, fever, malaise, conjunctivitis, and joint pains [2]. Zika fever has been a relatively mild disease of limited scope, with only one in five persons developing symptoms, with no fatalities, but its true potential as a viral agent of disease is unknown [3]. To-date no specific medical countermeasures available for ZIKVF and medical countermeasure development challenges has been addressed in [4].

ZIKV is primarily transmitted to humans through the bite of infected *Aedes* species mosquitoes, including *Aedes aegypti* and *Aedes albopictus*, the most important vectors for transmitting DENV and CHIKV globally. ZIKV was first isolated from rhesus monkey in Zika Forest of Uganda in 1947 and from the mosquito *Aedes africanus* in 1948 [5]. ZIKV has also been isolated in *Aedes aegypti* [6, 7] and *Aedes albopictus* was identified as the primary vector for ZIKV transmission in the Gabon outbreak of 2007 [8].

Further studies has indicated other potential mode of transmission such as sexual intercourse when infected person involve in unsafe sex with another person [9, 10], blood transfusions activities [11, 12], and perinatal transmission [13].

The first human isolate of ZIKVF in Africa is that of 1952 in Uganda and Tanzania [14], followed by that of 1954 in Nigeria [15]. Other studies showing human isolate of ZIKV includes [16], [17], and [18]. Evidence of human infection with ZIKV has been reported from other African countries such as Central African Republic, Egypt, Gabon and Sierra Leone. In Asia includes India, Indonesia, Malaysia, the Philippines, Thailand, and Vietnam [3]. The disease was

* Corresponding author.

E-mail address: saul.mpeshe@tumaini.ac.tz (Saul C. Mpeshe).

primarily considered to be of Africa and Asia until 2007 when an outbreak occur in Yap island, Federated State of Micronesia [2, 3]. The current reported outbreak of ZIKV is that of Nov 2015 in Cape Verde where 17 out of 64 serum samples tested positive for ZIKV [19].

The second and largest outbreak outside Africa and Asia is that of 2013-2014 in French Polynesia where about 28,000 (11% of the population) case were consulted for ZIKVF [20–22]. The recent outbreak outside Africa and Asia is that of 2015 - to date in America where there has been a rapid regional spread in South America, Latin America, and Caribbean. The history of ZIKV worldwide is well summarized in [19].

ZIKV spreads throughout the areas of the world where DENV infection is endemic [23], and many of the outbreaks in Pacific islands occurred concurrently with outbreaks of DENV and CHIKV [24–26]. The current outbreak of ZIKV in Cape Verde is showing that ZIKV is now returning to its place of origin. The presence of *Aedes* mosquito species in Africa which has caused several outbreaks of dengue fever in Africa including the 2007 outbreak in Gabon, 2010 outbreak in Kenya and 2013 outbreak in the United Republic of Tanzania suggest the possibility of re-emergence of ZIKV in Africa.

The potential transmission of ZIKV to human beings is evident. While ZIKVF is currently endemic in some parts of the world, a little is known by many people in Africa. Little or no effort is being made to understand the dynamics of ZIKV infection to human, the modes of transmission and possible ways of prevention or control mechanisms. That is to say, the disease has been neglected in Africa. Modeling the dynamics of ZIKVF will help bring awareness to people and have a better understanding of the transmission dynamics of ZIKVF, the risk associated to the disease, and measures to control the disease. Therefore, in this article we propose a simple model that investigates the dynamics of ZIKVF in Africa. Mathematical models uses a set of mathematical equations derived from a theoretical framework to help our understanding in population dynamics and epidemiology as in [27, 29, 30] and [31]

2. Material and methods

2.1. Model formulation

The model considers only *Aedes* mosquitoes and human population with no disease-dependent death rate for human. Due to vertical transmission in *Aedes*, we include a recruitment of infected mosquitoes to the infected mosquito compartment. The population for *Aedes* mosquitoes consists of susceptible adults (S_a) and infectious adults (I_a). The human population consists of susceptible humans (S_h), infectious humans (I_h), and recovered humans (R_h). We do not consider other modes of transmission such as sexual, blood transfusion, and perinatal due to insufficient information. Table 1 shows the model parameters and their description as they have been used in this work.

Table 1. Parameters used in the model formulation and their description.

Parameter	Description
b_a	daily birth rate of *Aedes* mosquitoes
b_h	daily birth rate in human
μ_a	natural death rate of *Aedes* mosquitoes
μ_h	natural death rate of human
γ_h	recovery rate in human
α_h	rate at which the recovered human become susceptible
λ_{ah}	Adequate contact rate: *Aedes* to humane
λ_{ha}	Adequate contact rate: humans to *Aedes*
p	vertical transmission rate in *Aedes* mosquitoes

The mode of transmission of ZIKV from vector to host and host to vector is shown in the model flowchart shown by Fig. 1. Using the parameters in Table 1 and the model flow diagram in Fig. 1, an SIR model is derived on the basis of the explanations above using first order nonlinear ordinary differential equations as follows:

Aedes Mosquito

$$\frac{dS_a}{dt} = b_a(1-p)N_a - \mu_a S_a - \lambda_{ha}\frac{I_h}{N_h}S_a, \tag{1a}$$

$$\frac{dI_a}{dt} = b_a p N_a + \lambda_{ha}\frac{I_h}{N_h}S_a - \mu_a I_a, \tag{1b}$$

$$\frac{dN_a}{dt} = (b_a - \mu_a)N_a. \tag{1c}$$

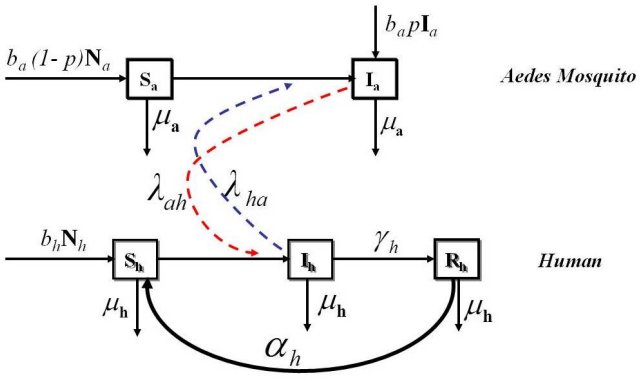

Fig. 1. Flow diagram for the ZIKVF model.

Humans

$$\frac{dS_h}{dt} = b_h N_h + \alpha_h R_h - \mu_h S_h - \lambda_{ah}\frac{I_a}{N_a}S_h, \tag{2a}$$

$$\frac{dI_h}{dt} = \lambda_{ah}\frac{I_a}{N_a}S_h - (\mu_h + \gamma_h)I_h, \tag{2b}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - (\mu_h + \alpha)R_h, \tag{2c}$$

$$\frac{dN_h}{dt} = (b_h - \mu_h)N_h. \tag{2d}$$

In terms of proportions the model can be written as

$$\frac{ds_a}{dt} = b_a(1-p) - \mu_a s_a - \lambda_{ha} s_a i_h, \tag{3a}$$

$$\frac{di_a}{dt} = b_a p + \lambda_{ha} s_a i_h - \mu_a i_a, \tag{3b}$$

$$\frac{ds_h}{dt} = b_h + \alpha_h r_h - \mu_h s_h - \lambda_{ah} s_h i_a, \tag{3c}$$

$$\frac{di_h}{dt} = \lambda_{ah} s_h i_a - (\mu_h + \gamma_h)i_h, \tag{3d}$$

$$\frac{dr_h}{dt} = \gamma_h i_h - (\mu_h + \alpha_h)r_h, \tag{3e}$$

$$\tag{3f}$$

To determine whether the model is well-posed epidemiologically and mathematically, we need to investigate the feasibility of the model solution. We write the system in compact form as:

$$\frac{dX}{dt} = M(x)X + F \tag{4}$$

where $X = (S_a, I_a, S_h, I_h, R_h)^T$, $M(x)$ is a 5 by 5 matrix and F is a column matrix.

Further computations gives,

$$M(x) = \begin{bmatrix} -(\mu_a + \lambda_{ha} i_h) & 0 & 0 & 0 & 0 \\ \lambda_{ha} i_h & -\mu_a & 0 & 0 & 0 \\ 0 & 0 & -(\mu_h + \lambda_{ah} i_m) & 0 & \alpha_h \\ 0 & 0 & \lambda_{ah} i_m & -(\mu_h + \gamma_h) & 0 \\ 0 & 0 & 0 & \gamma_h & -(\mu_h + \alpha_h) \end{bmatrix} \tag{5}$$

and

$$F = (b_a(1-p), b_a p, b_h, 0, 0)^T \geq 0 \tag{6}$$

Observe that the matrix $M(x)$ is a Metzler matrix for all $\mathbb{R}^5_+$. Therefore, the model system is positively invariant in $\mathbb{R}^5_+$, and F is Lipschitz continuous. Thus, the feasible region for the model system is the set

$$\mathscr{D} = \{(S_a, I_a, S_h, I_h, R_h) \geq 0 \in \mathbb{R}^5_+\} \tag{7}$$

That is, the solution remain in the feasible region $\mathscr{D}$ if it starts in this region. The disease free equilibrium for the model is $E^0 = (\frac{b_a}{\mu_a}, 0, \frac{b_h}{\mu_h}, 0, 0, 0)$ and the endemic equilibrium is $E^* = (s_a^*, i_a^*, s_h^*, i_h^*, r_h^*)$.

2.2. The basic reproduction number

The basic reproduction number $\mathscr{R}_0$ is computed using the method of next generation matrix as outlined by [32]. Considering the equations

$$\frac{di_a}{dt} = b_a p + \lambda_{ha} s_a i_h - \mu_a i_a, \tag{8a}$$

$$\frac{di_h}{dt} = \lambda_{ah} s_h i_a - (\mu_h + \gamma_h) i_h, \tag{8b}$$

$$\tag{8c}$$

the resulting next generation matrix is

$$\mathscr{F}_i = \begin{bmatrix} \lambda_{ha} s_a i_h \\ \lambda_{ah} s_h i_a \end{bmatrix}, \tag{9}$$

and

$$\mathscr{V}_i = \begin{bmatrix} -b_a p + \mu_a i_a \\ (\mu_h + \gamma_h) i_h \end{bmatrix}, \tag{10}$$

which gives

$$F = \begin{bmatrix} 0 & \lambda_{ha} s_a \\ \lambda_{ah} s_h & 0 \end{bmatrix}, \tag{11}$$

and

$$V = \begin{bmatrix} \mu_a & 0 \\ 0 & (\mu_h + \gamma_h) \end{bmatrix}. \tag{12}$$

Therefore,

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_a} & 0 \\ 0 & \frac{1}{(\mu_h+\gamma_h)} \end{bmatrix}, \tag{13}$$

and

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\lambda_{ha} s_a}{\mu_a} \\ \frac{\lambda_{ah} s_h}{(\mu_h+\gamma_h)} & 0 \end{bmatrix}. \tag{14}$$

Thus, the basic reproduction number which is the spectral radius of FV^{-1} is given by

$$\mathscr{R}_0 = \sqrt{\frac{b_a b_h \lambda_{ha} \lambda_{ah}}{\mu_a^2 \mu_h (\mu_h + \gamma_h)}}. \tag{15}$$

The quantities in the basic reproduction number can be explained as follows: $\frac{b_a}{\mu_a}$ is the mean number of births over lifespan of *Aedes* mosquitoes, $\frac{b_h}{\mu_h}$ is the mean number of births over lifespan of human, $\frac{\lambda_{ha}}{(\mu_h+\gamma_h)}$ is the mean number of *Aedes* mosquitoes infected by human during its infective period, and $\frac{\lambda_{ah}}{\mu_a}$ is the mean number of human infected by *Aedes* during its life span.

2.3. Sensitivity analysis of $\mathscr{R}_0$

In order to determine how best we can reduce the transmission of ZIKVF, it is necessary to study the relative importance of different factors responsible for its transmission and prevalence through sensitivity indices. Sensitivity indices quantify how the basic reproduction number $\mathscr{R}_0$ changes in response to the small shifts in the value of a parameter. Generally, the initial disease transmission is directly related to the, $\mathscr{R}_0$, and thus, sensitivity values can be used to judge which parameters are important to measure accurately and where variation in parameters will translate into variation in $\mathscr{R}_0$ [33].

Since $\mathscr{R}_0$ is given explicitly by the Eq. (15), we derive an analytical expression for its sensitivity to each parameter using the normalized forward sensitivity index as in [28]. Hence, the sensitivity index of $\mathscr{R}_0$ with respect to birth rate of *Aedes* mosquito b_a is given by

$$\Upsilon_{b_a}^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial b_a} \times \frac{b_a}{\mathscr{R}_0} = \frac{1}{2} \tag{16}$$

which does not depend on any parameter. Similarly, the sensitivity index of $\mathscr{R}_0$ with respect to birth rate of human b_h is given by

$$\Upsilon_{b_h}^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial b_h} \times \frac{b_h}{\mathscr{R}_0} = \frac{1}{2} \tag{17}$$

which does not depend on any parameter as well.

Proceeding in a similar manner, we compute and evaluate the sensitivity indices of $\mathscr{R}_0$ using the parameter values given in Table 2. The resulting sensitivity indices for the parameters in $\mathscr{R}_0$ are shown in Table 3. For almost all parameters, the sign of the sensitivity indices of $\mathscr{R}_0$ (i.e., whether $\mathscr{R}_0$ increases or decreases when a parameter increases) agrees with an intuitive expectation from the model parameters.

Table 2. Parameters used in the model.

Parameter	Range	Value	Reference
b_a	[1/200 1/100] per day	1/100 per day	[33]
b_h	N/A	36.39/1000/365 per day	[34]
μ_a	[1/30 1/60] per day	1/60	[33]
μ_h	N/A	8/1000/365 per day	[34]
α_h	N/A	0.100 per day	assumed
γ_h	[1/2 1/7] per day	1/5 per day	[2]
p	[0.01 0.10] per day	0.05	[33]
λ_{ah}	N/A	0.750 per day	[35]
λ_{ha}	N/A	0.375 per day	[35]

Table 3. Sensitivity indices of $\mathscr{R}_0$.

	Parameter	Sensitivity Index
1	b_a	0.5000
2	b_h	0.5000
3	μ_a	-1.0000
4	μ_h	-0.5000
5	γ_h	-0.4999
6	λ_{ha}	+0.5000
7	λ_{ah}	+0.5000

2.4. Numerical simulations

In this section, numerical simulation are carried out using parameter values given in Table 2. Due to lack of data, the value of α_h (rate at which the recovered human become susceptible) has been assumed for the purpose of illustration only. The initial values used in simulations are $s_h = 0.8$, $i_h = 0.2$, $r_h = 0$, $s_a = 0.8$, and $i_a = 0.1$. Fig. 2 show the graph of the variation of each compartment in ZIKVF model with respect to time. Numerical simulation help to study the persistence of the disease when introduced in a closed or isolated system.

3. Discussion

A simple model to investigate the dynamics of ZIKVF has been established and analysed. In this model the basic reproduction number $\mathscr{R}_0$ has been computed and the relative effect of changes of parameter values to $\mathscr{R}_0$ has been established through sensitivity analysis. The values of sensitivity indices and their sign are given in Table 3. The positive sign of the sensitivity indices indicates that $\mathscr{R}_0$ increases when a particular parameter increases, while the negative sign of the sensitivity index indicates that $\mathscr{R}_0$ decreases when a particular parameter increases and viceversa. The results of sensitivity analysis show that the natural death rate of mosquitoes, μ_a, is the most sensitive parameter, while, the recovery rate, γ_h is the least sensitive parameter.

The negative sign of the sensitivity index of $\mathscr{R}_0$ with respect to μ_a implies for instance that a 10% increase in the natural death rate of mosquitoes leads to approximately a 10% decrease in $\mathscr{R}_0$. Similarly, the negative sign of the sensitivity index of $\mathscr{R}_0$ with respect to γ_h implies for instance that a 10% increase in the natural death rate of mosquitoes

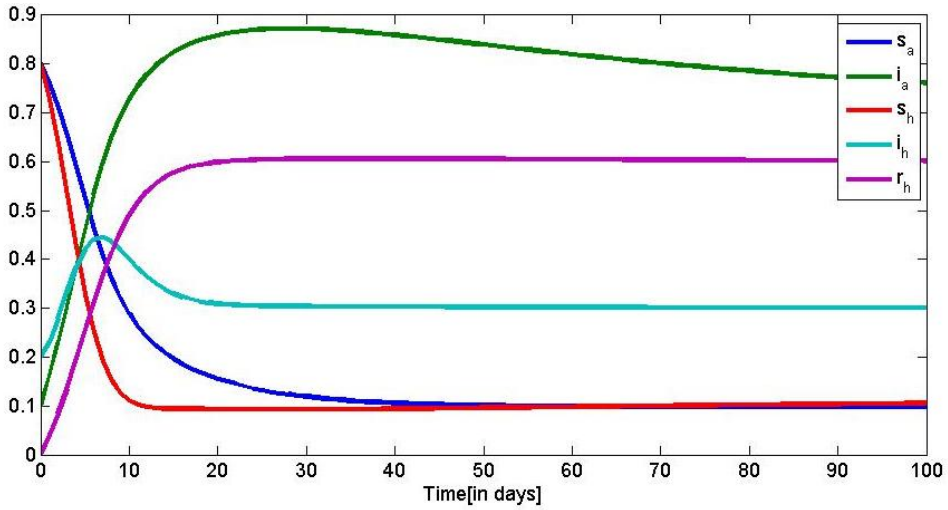

Fig. 2. Graphs for each compartment in ZIKVF model.

leads to approximately a 4.999% decrease in $\mathscr{R}_0$. Indeed, an increase in μ_a shortens the lifespan of mosquitoes, thus, a large number of infected mosquitoes die before becoming infectious and thereby reducing $\mathscr{R}_0$. Also, the increase in recovery rate of human shortens the infectious period of human, thus, a large number of human recover before infecting other people.

To analyze the variation of susceptible, infected and recovered with respect to time in the two populations numerical simulations have been performed. The results of numerical simulation are shown in Fig. 2. With the given parameter values, the values of $\mathscr{R}_0 = 7.03$ which is greater than unity, thus, the disease is likely to persist even when the system is closed or isolated. From the graph we see that the population of susceptible human reduces and remains constant after 10 days which is likely to be the incubation period of ZIKVF. After 20 days the graphs for s_h, i_h and r_h, remains constant after switching. This means that the rate of infection, rate of recovery, and rate of becoming susceptible after recovery is now constant. Therefore, increasing the recovery rate while reducing the infection rate at this stage will help to control the disease.

4. Conclusion

ZIKVF continues to be a disease of potential threat and burden to human all over the world. It has been established that the basic reproduction number $\mathscr{R}_0 = 7.03$ which is greater than unity suggesting the persistence of the disease when an outbreak occurs. Sensitivity analysis of $\mathscr{R}_0$ with respect to each parameter involved has been established indicating that $\mathscr{R}_0$ is most sensitive to the natural death rate of mosquitoes, μ_a, and least sensitive to the recovery rate, γ_h. These results suggest the control measures which will reduce the population of *Aedes* mosquitoes such as the application of insecticides to increase death rate of mosquitoes and also increase recovery rate of human through treatment and other control measures.

A number of challenges remain for the control and prevention of ZIKVF. Knowledge regarding how the virus is transmitted among mosquitoes and human, the transmission of ZIKV through blood transfusion, sexual intercourse and perinatal, and the role of vertebrates in propagating the virus must be answered to predict and control future outbreaks of ZIKVF. Though the study used a simple model, the current analysis enables us to gain valuable insights and remains however an important step in theoretically analyzing the disease.

Acknowledgments

The authors would like to thank the all people who contributed in the completion of this article.

Conflict of interest

The author declares no conflict of interest

References

[1] D. J. Gubler, G. Kuno, L. Markoff, Flaviviruses, In: Knipe, K., Howley, P. M.(Eds.), Fields Virology 2, 5th ed., Lippincott Williams and Wilkins Publishers, Philadelphia, USA (2007) 1153–1252.

[2] M.R. Duffy, T.H. Chen, W.T. Hancock, A.M. Powers, J.L. Kool, R.S. Lanciotti, M. Pretrick, M. Marfel, S. Holzbauer, C. Dubray, L. Guillaumot, A.Griggs, M.Bel, A.J. Lambert, J.Laven, O.Kosoy, A.Panella, B.J. Biggerstaff, M. Fischer, E.B. Hayes, Zika Virus Outbreak on Yap Island, Federated States of Micronesia, New Engl. J. of Med. 360(24) (2009) 2536–2543.

[3] E.B. Hayes, Zika Virus Outside Africa, Emerg. Infect. Dis. 15(9) (2009) 1347–1350.

[4] R.W. Malone, J. Homan, M.V. Callahan, J. Glasspool-Malone, L. Damodaran, A.D.B. Scheider, Zika Virus: Medical Countermeasure Development Challenges, PLoS Negl. Tro. Dis. 10(3) 2016 .

[5] G.W.A. Dick, S.F. Kitchen, A.J. Haddow, Zika virus. I. Isolations and serological specificity, Trans. R. Soc. Trop. Med. Hyg. 46(5) (1952) 509–520.

[6] J.P. Boorman, J.S. Porterfield, A simple technique for infection of mosquitoes with viruses; transmission of Zika virus, Trans. R. Soc. Trop. Med. Hyg. 50(3) (1956) 238–242.

[7] N.J. Marchette, R.Garcia, A.Rudnick, Isolation of Zika virus from Aedes aegypti mosquitoes in Malaysia, Am. J. Trop. Med. Hyg. 18 (1969) 411–415.

[8] G. Grard, M. Caron, I.M. Mombo, D. Nkoghe, S. Mboui Ondo, Zika virus in Gabon (Central Africa)–2007: a new threat from Aedes albopictus ? PLoS Negl. Trop. Dis. 8(2) 2014.

[9] D. Musso, C. Roche, E. Robin, T. Nhan, A. Teissier, V.M. Cao-Lormeau, Potential sexual transmission of Zika virus, Emerg. Infect. Dis. 21(2) (2015) 359–361.

[10] B.D. Foy, K.C. Kobylinski, J.L. C. Foy, B.J. Blitvich, A. Travassos Da Rosa, A.D. Haddow, R.S. Lanciotti, R.B. Tesh, Probable NonâĂŞVector-borne Transmission of Zika Virus, Colorado, USA, Emerg. Infect. Dis. 17(5) (2011) 880–882.

[11] D. Musso, T. Nhan, E. Robin, C. Roche, D. Bierlaire, K. Zisou, A. Shan Yan, V.M. Cao-Lormeau, J. Broult, Potential for Zika virus transmission through blood transfusion demonstrated during an outbreak in French Polynesia, November 2013 to February 2014, Eurosurveillance 19(14) 2014.

[12] G. Marano, S. Pupella, S. Vaglio, G.M. Liumbruno, G. Grazzini, Zika virus and the never-ending story of emerging pathogens and transfusion medicine, Blood Transfus 2015 (2015) 1–6.

[13] M.Besnard, S.Lastere, A.Teissier, V.Cao-Lormeau, D.Musso, Evidence of perinatal transmission of Zika virus, French Polynesia, December 2013 and February 2014, Euro. Surveill. 19 (2014) 20751.

[14] K.C. Smithburn, Neutralizing antibodies against certain recently isolated viruses in the sera of human beings residing in East Africa, J. Immunol. 69 (1952) 223–234.

[15] F.N. Macnamara, Zika virus: a report on three cases of human infection during an epidemic of jaundice in Nigeria, Trans. R. Soc. Trop. Med. Hyg. 48 (1954) 139–145.

[16] W.G.C. Bearcroft, Zika virus infection experimentally induced in a human volunteer, Trans. R. Soc. Trop. Med. Hyg. 50(5) (1956) 442–448.

[17] D.I. Simpson, Zika virus infection in man, Trans. R. Soc. Trop. Med. Hyg. 58 (1964) 335–338.

[18] A.H. Fagbami, Zika virus infections in Nigeria: virological and seroepidemiological investigations in Oyo State, J. Hyg. Lond. 83 (1979) 213–219.

[19] D. Musso, D.J. Gubler, Zika virus, Clin. Microbiol. Rev. 29 (2016) 487–524.

[20] V.M. Cao-Lormeau, C. Roche, A. Teissier, E. Robin, A.L. Berry, Zika virus, French polynesia, South pacific, 2013, Emerg. Infect. Dis. 20 (2014) 1085–1086.

[21] D. Musso, Zika Virus Transmission from French Polynesia to Brazil, Emerg. Infect. Dis. 21 (2015) 1887.

[22] D. Musso, E.J. Nilles, V.M. Cao-Lormeau, Rapid spread of emerging Zika virus in the Pacific area, Clin. Microbiol. Infect. 20 (2014) 595–596.

[23] J.J. Waggoner, B.A. Pinsky, Zika virus: diagnostics for an emerging pandemic threat, J. Clin. Microbiol. 54 (2016) 860–867.

[24] D. Musso, V.M. Cao-Lormeau, D.J. Gubler, Zika virus: following the path of dengue and chikungunya? Lancet 386 (2015) 243–244.

[25] A. Roth, A. Mercier, C. Lepers, D. Hoy, S. Duituturaga, E. Benyon, L. Guillaumot, Y. Souares, Concurrent outbreaks of dengue, chikungunya and Zika virus infectionsâĂŤan unprecedented epidemic wave of mosquito-borne viruses in the Pacific 2012–2014, Euro. Surveill. 19 (2014) 20929.

[26] M. Dupont-Rouzeyrol, O. O'Connor, E. Calvez, M. Daures, M. John, J.P. Grangeon, A.C. Gourinat, Co-infection with Zika and dengue viruses in 2 patients, New Caledonia, 2014, Emerg. Infect. Dis. 21 (2015) 381–382.

[27] J.P. Mpele, Y. Nkansah-Gyekye, O.D. Makinde, A model of a fishery resource in the presence of water hyacinth: the case of Lake Victoria, Int. J. Adv. Appl. Math. and Mech. 2(1) (2014) 34–41.

[28] S. C. Mpeshe, H. Heikki and J. M. Tchuenche, A Mathematical Model of Rift Valley Fever with Human Host, Acta Biotheor, 59(3-4)(2011) 231-250.

[29] G.M. Mlay, L. Luboobi, D. Kuznetsov, F. Shahada, Optimal treatment and vaccination control strategies for the

dynamics of pulmonary tuberclosis, Int. J. Adv. Appl. Math. and Mech. 2(3) (2015) 196–207.
[30] X. Zhou, Q. Sun, Stability of a fractional-order HBV infection model, Int. J. Adv. Appl. Math. and Mech. 2(2) (2016) 1–6.
[31] M.A. Khan, A. Walid, S. Islam, I. Khan, S. Shafie, T. Gul, Stability analysis of an SEIR epidemic model with non-linear saturated incidence and temporary immunity, Int. J. Adv. Appl. Math. and Mech. 2(3) (2015) 1–14.
[32] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002) 29–48.
[33] S.C. Mpeshe, L.S. Luboobi, Y. Nkansah-Gyekye, Modeling the Impact of Climate Change on the Dynamics of Rift Valley Fever, Comput. and Math. Methods in Med. 2014 (2014) 1–12.
[34] CIA, The World Fact Book, (2016), Retrived on 30th May 2016.
[35] M. Derouich, A. Boutayel, E.H. Twizell, A model of Dengue, BioMed. Eng. Online 2(4)(2003) 1–10.