Modeling and Sensitivity Analysis of Dynamical Transmission of Lassa Fever

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ABSTRACT

Lassa fever is an acute viral disease which occurs across West Africa regions. In view of this a non-linear deterministic model was considered to study dynamics spread of Lassa fever. Existence and uniqueness of the model were determined and the basic reproduction number of the model was computed using nextgeneration matrix, and sensitivity analysis of the model was performed and shows that the most sensitive parameters to the dynamical spread of Lassa fever are human birth rate β_{H} , followed by average number of female partners acquired by susceptible male c_2 , and transmission rate from sexual interaction between resulting infected male and susceptible female η_2 have the highest values. It concludes that average number of female partners acquired by susceptible male and sexual transmission rate between the infected male and susceptible female should be reduced.

Keywords: Existence and Uniqueness, disease free equilibrium, basic reproduction numbers and sensitivity analysis

INTRODUCTION

Lassa fever is an acute viral disease which occurs across West Africa regions. The disease was discovered in 1969 when two missionary nurses died in Nigeria. The virus was named after the town in Nigeria where it first occurred. The disease is a member of the virus family *Arenaviridae*, is a single-stranded RNA virus and is zoonotic in the sense that human become infected after contact with infected animals.

Lassa fever is known to be endemic in parts of west Africa such as Benin, Ghana, Sierra Leone, Liberia, Mali, Guinea and Nigeria; however, as well as other neighboring countries are also at risk of this disease. The reservoir, or host, of Lassa virus is a rodent known as the "multimammate rat". When mastomys rats are infected, this rodent is able to excrete the virus in urine and faeces for an extended period of time or for the rest of its life. Mastomys rodents breed frequently, produce large numbers of offspring, and are numerous in the savannas and forests of west, central, and east Africa. In addition, Mastomys readily colonize human homes and areas where food is stored. All of these factors contribute to the relatively efficient transmission of Lassa virus from infected rodents humans. to Sexual transmission of Lassa virus is another case, Lassa fever occurs in all age groups and both sexes. Persons who are at greatest risk are those living in rural areas are usually where Mastomys found, especially in communities with poor sanitation or crowded living conditions. Health workers are at risk when caring for Lassa fever patients in the absence of proper barrier nursing and infection prevention and control practices (Disease outbreak news, 27 May, 2017).

Signs and symptoms of Lassa fever mostly occur within 1-3 weeks when the patient comes in contact with the virus. Approximately 80% of Lassa fever virus infections have mild symptoms and undiagnosed. Mild symptoms include slight fever, general malaise, weakness and headache. About 20% of infected individuals, however, disease may progress to more serious symptoms including hemorrhaging (in gums, eyes, or nose, as examples), respiratory distress, repeated vomiting, facial swelling, pain in the chest, abdomen. back. and and shock. Neurological problems have also been described, including hearing loss, tremors, and encephalitis. Death may occur within two weeks after symptom onset due to multi-organ failure. The most common complication of Lassa fever is deafness. Various degrees of deafness occur in approximately one-third of infections, and in many cases hearing loss is permanent. As far as is known, severity of the disease does not affect this complication: deafness may develop in mild as well as in severe cases. Onuorah et al (2016), developed a Lassa fever model using the sex structure approach. Their model represents the transmission dynamics of the Lassa fever disease using a set of ordinary differential Susceptible individuals, equations. male/female can be infected via interaction with the active Reservoir (Mastomys Natelensis), and via sexual interaction with opposite sex. Incorporate the rate at which virus is shedding into the environment into Onuorah at al. (2016) work the following model is developed. Omoloye et al (2020) investigates the application of DTM to solve dynamic transmission of Lassa fever model in a population. The mathematical model was formulated using first order differential equation. Firstly, existence and uniqueness of the solution was determined to establish that the model is mathematically well posed for the application of DTM. Numerically, simulations were conducted to compare the results obtained by DTM and that of fourthorder Runge-Kutta method.

MODEL FORMULATION

The model is constructed by dividing the total human population N_{H} into four mutually exclusive sub-populations, Susceptible Male $S_{1}(t)$, Infected Male $I_{1}(t)$ and Susceptible Female $S_{2}(t)$, Infected Female $I_{2}(t)$. Also, the total Natural Reservoir or host population denoted by N_{R} is divided into dormant Reservoir host $R_{1}(t)$, active Reservoir host $R_{2}(t)$ and V(t) denote the population of Lassa virus in the environment.

Model has the following basic assumption, humans are only recruiting into the susceptible class through birth rate, and susceptible individuals, male or female can be infected through interaction with the active reservoir and sexual interaction with opposite sex and the rate at which virus is shedding into the environment. The force of infection is given by a standard incidence as $(c_1\eta_1(1-\varepsilon\tau)I_2 + \eta_3R_2 + \eta_5V)$

$$\frac{\eta_1(1-\varepsilon \iota)I_2+\eta_3K_2+\eta}{N_H}$$

Where N_H is the total population and the constants, $\eta_1, \eta_2, \eta_3, \eta_4$ and η_5 is the transmission rate result from sexual interaction between either through susceptible female and infected male or susceptible male and infected female.

The model are described by the following differential equations

$$\frac{dS_{1}}{dt} = \beta_{H}\theta N_{H} + \gamma I_{1} - \frac{(c_{1}\eta_{1}(1-\varepsilon\tau)I_{2} + \eta_{3}R_{2} + \eta_{5}V)S_{1}}{N_{H}} - \mu_{1}S_{1}$$

$$\frac{dI_{1}}{dt} = \frac{(c_{1}\eta_{1}(1-\varepsilon\tau)I_{2} + \eta_{3}R_{2} + \eta_{5}V)S_{1}}{N_{H}} - (\mu_{1} + \delta_{1} + \gamma)I_{1}$$

$$\frac{dS_{2}}{dt} = \beta_{H}(1-\theta)N_{H} + \gamma I_{2} - \frac{(c_{2}\eta_{2}(1-\varepsilon\tau)I_{1} + \eta_{4}R_{2} + \eta_{6}V)S_{2}}{N_{H}} - \mu_{1}S_{2}$$

$$\frac{dI_{2}}{dt} = \frac{(c_{2}\eta_{2}(1-\varepsilon\tau)I_{1} + \eta_{4}R_{2} + \eta_{6}V)S_{2}}{N_{H}} - (\mu_{1} + \delta_{1} + \gamma)I_{2}$$

$$\frac{dR_{1}}{dt} = \beta_{R}N_{R} - (\sigma + \mu_{2} + \delta_{2})R_{1}$$

$$\frac{dR_{2}}{dt} = \sigma R_{1} - (\mu_{2} + \delta_{2})R_{2}$$

$$\frac{dV}{dt} = e_{3}R_{2} + e_{2}I_{2} + e_{1}I_{1} - \phi V$$
(1)

Parameter	Description
$\beta_{\scriptscriptstyle H}$	Natural birth rate for human population
β_{R}	Natural birth rate for host population
θ	Fraction of male birth $0 < \theta < 1$
η_1	Transmission rate resulting from sexual interaction between infected female and susceptible male
η_2	Transmission rate resulting from sexual interaction between infected male and susceptible female
η_3	Transmission rate resulting from interaction between active virus Reservoir and susceptible male
$\eta_{_4}$	Transmission rate resulting from interaction between active virus Reservoir and susceptible female
η_5	Transmission rate resulting from interaction between susceptible male with lassa virus in the environment
η_6	Transmission rate resulting from interaction between susceptible female with lassa virus in the environment
<i>c</i> ₁	Average number of male partners acquired by a susceptible female
<i>c</i> ₂	Average number of female partners acquired by a susceptible male
μ_1	Natural death rate for human
μ_2	Natural death rate for human
γ	Recovery rate of infected human
σ	Progression rate from dormant to active reservoir host
δ_1	Death rate of human due to infection
δ_2	Death rate of virus reservoir due to pesticide application
ε	Efficacy of condom
τ	Compliance of condom
<i>e</i> ₁	Discharging rate of lassa virus into the environment from infected male
<i>e</i> ₂	Discharging rate of lassa virus into the environment from infected female
<i>e</i> ₃	Discharging rate of lassa virus into the environment from active Reservoir
ϕ	The death rate of lassa virus

Table 1: Parameters used in the model

EXISTENCE AND UNIQUENESS OF SOLUTION

Theorem 1 Let

$$\begin{aligned} x_1' &= f_1(x_1, x_2, \dots, x_n, t), x_1(t_0) = x_{10} \\ x_2' &= f_2(x_1, x_2, \dots, x_n, t), x_2(t_0) = x_{20} \\ x_3' &= f_3(x_1, x_2, \dots, x_n, t), x_3(t_0) = x_{30} \\ \vdots \\ x_n' &= f_n(x_1, x_2, \dots, x_n, t), x_n(t_0) = x_{n0} (2) \end{aligned}$$

Suppose *D* is the region in (n+1)dimensional space (one dimension for *t* and *n* dimensions for the vector *x*). If the partial derivatives $\frac{\partial f_i}{\partial x_j}$ where i, j = 1, 2, ..., n are

continuous in

$$D = \{(x,t) : |t - t_0| \le a, |x - x_0| \le b\}, \text{ Then}$$

there is a constant $\delta > 0$ such that there exists a unique continuous vector solution

 $\underline{x} = [x_1(t), x_2(t), x_3(t), \dots, x_n(t)]$ in the interval $|t - t_0| \le \delta.$ Let:

$$\begin{split} f_{1} &= \frac{dS_{1}}{dt} = \beta_{H} \theta N_{H} + \gamma I_{1} - \frac{(c_{1}\eta_{1}(1 - \varepsilon \tau)I_{2} + \eta_{3}R_{2} + \eta_{5}V)S_{1}}{N_{H}} - \mu_{1}S_{1} \\ f_{2} &= \frac{dI_{1}}{dt} = \frac{(c_{1}\eta_{1}(1 - \varepsilon \tau)I_{2} + \eta_{3}R_{2} + \eta_{5}V)S_{1}}{N_{H}} - (\mu_{1} + \delta_{1} + \gamma)I_{1} \\ f_{3} &= \frac{dS_{2}}{dt} = \beta_{H}(1 - \theta)N_{H} + \gamma I_{2} - \frac{(c_{2}\eta_{2}(1 - \varepsilon \tau)I_{1} + \eta_{4}R_{2} + \eta_{6}V)S_{2}}{N_{H}} - \mu_{1}S_{2} \\ f_{4} &= \frac{dI_{2}}{dt} = \frac{(c_{2}\eta_{2}(1 - \varepsilon \tau)I_{1} + \eta_{4}R_{2} + \eta_{6}V)S_{2}}{N_{H}} - (\mu_{1} + \delta_{1} + \gamma)I_{2} \\ f_{5} &= \frac{dR_{1}}{dt} = \beta_{R}N_{R} - (\sigma + \mu_{2} + \delta_{2})R_{1} \\ f_{7} &= \frac{dR_{2}}{dt} = \sigma R_{1} - (\mu_{2} + \delta_{2})R_{2} \\ f_{8} &= \frac{dV}{dt} = e_{3}R_{2} + e_{2}I_{2} + e_{1}I_{1} - \phi V \\ \hline \begin{pmatrix} \mathbf{3} \\ \\ \\ \\ \\ D = [(S_{1},I_{1},S_{2},I_{2},R_{1},R_{2},V):[S_{1} - S_{10}] \leq a_{1}|I_{1} - I_{10}| \leq b_{1}|S_{2} - S_{20}| \leq c_{1}|I_{2} - I_{20}| \leq d_{1}|R_{1} - R_{10}| \leq e_{1}|R_{2} - R_{2}| \\ \end{cases}$$

The above equation (2.3) has a

unique solution. Proof Partial derivative was evaluated at the origin thus: $\frac{\partial f_1}{\partial S_1}\Big|_{(0,0,0,0,0,0,0)} = -\mu_1, \frac{\partial f_2}{\partial S_1}\Big|_{(0,0,0,0,0,0,0)} = 0,$ $\frac{\partial f_3}{\partial S_1}\Big|_{(0,0,0,0,0,0,0)} = 0, \frac{\partial f_1}{\partial S_1}\Big|_{(0,0,0,0,0,0,0)} = 0,$ $\frac{\partial f_1}{\partial I_1}\Big|_{(0,0,0,0,0,0)} = \gamma,$ ∂f_2 $\frac{\partial f_2}{\partial I_1}\Big|_{(0,0,0,0,0,0,0)} = -(\mu_1 + \delta_1 + \gamma),$ $\frac{\partial f_3}{\partial I_1}\bigg|_{(0,0,0,0,0,0,0)} = 0, \ \frac{\partial f_7}{\partial I_1}\bigg|_{(0,0,0,0,0,0,0)} = e_1,$ $\frac{\partial f_1}{\partial S_2}\Big|_{(0,0,0,0,0,0,0)} = 0, \ \frac{\partial f_2}{\partial S_2}\Big|_{(0,0,0,0,0,0,0)} = 0,$ $\frac{\partial f_3}{\partial S_2}\Big|_{(0,0,0,0,0,0,0)} = -\mu_2, \frac{\partial f_4}{\partial S_2}\Big|_{(0,0,0,0,0,0,0)} = 0,$ $\frac{\partial f_1}{\partial I_2}\bigg|_{(0,0,0,0,0,0,0)} = 0, \left.\frac{\partial f_3}{\partial I_2}\right|_{(0,0,0,0,0,0,0)} = \gamma,$ $\frac{\partial f_4}{\partial I_2}\Big|_{(0,0,0,0,0,0,0)} = -(\mu_1 + \delta_1 + \gamma),$ $\left. \frac{\partial f_7}{\partial I_2} \right|_{(0,0,0,0,0,0)} = e_2,$ $\frac{\partial f_1}{\partial R_1}\Big|_{(0,0,0,0,0,0,0)} = 0, \ \frac{\partial f_2}{\partial R_1}\Big|_{(0,0,0,0,0,0,0)} = 0,$ $\frac{\partial f_5}{\partial R_1}\Big|_{(0,0,0,0,0,0,0)} = -(\sigma + \mu_2 + \delta_2),$ $\frac{\partial f_6}{\partial R_1}\Big|_{(0,0,0,0,0,0)} = \sigma,$ $\frac{\partial f_1}{\partial R_2}\Big|_{(0,0,0,0,0,0,0)} = 0, \frac{\partial f_2}{\partial R_2}\Big|_{(0,0,0,0,0,0,0)} = 0,$ $\frac{\partial f_6}{\partial R_2}\Big|_{(0,0,0,0,0,0,0)} = -(\mu_2 + \delta_2),$ $\frac{\partial f_7}{\partial R_2}\bigg|_{(0,0,0,0,0,0)} = e_3,$

$$\frac{\partial f_1}{\partial V}\Big|_{(0,0,0,0,0,0,0)} = 0, \ \frac{\partial f_2}{\partial V}\Big|_{(0,0,0,0,0,0,0)} = 0,$$
$$\frac{\partial f_3}{\partial V}\Big|_{(0,0,0,0,0,0,0)} = 0, \ \frac{\partial f_7}{\partial V}\Big|_{(0,0,0,0,0,0,0)} = -\phi,$$
$$\frac{\left|\frac{\partial f_i}{\partial x_i}\right|, i.j = 1, 2, \dots 7$$

Since $|Cx_j|$ are continuous and bounded. Hence, follow Derrick and Grossman of theorem 1 above, equation (3) has a unique solution and the model (1) is mathematically well posed.

DISEASE FREE EQUILIBRIUM POINT

At steady-state solution of the system (1) is obtained by setting

$$\frac{dS_1}{dt} = \frac{dI_1}{dt} = \frac{dS_2}{dt} = \frac{dI_2}{dt} = \frac{dR_1}{dt} = \frac{dR_2}{dt} = \frac{dV}{dt} = 0$$

Let E_0 denote the disease free equilibrium state of the model (1)

At disease free

$$E_0 = (S_1, I_1, S_2, I_2, R_1, R_2, V) = \left(\frac{\beta_H \theta N_H}{\mu_1}, 0, \frac{\beta_H (1-\theta) N_H}{\mu_1}, 0, 0, 0, 0\right)$$

BASIC REPRODUCTION NUMBER

The basic reproduction number is the number of secondary cases of infection emanating from a single infection source[Diekmann, O., Hesterbeek, J. A. P. and Metz, J. A. J.(1990], using next Generation matrix method to obtain the basic reproduction number. The matrices F (new infection terms) and V (other remaining transfer terms) are given as;

$$\begin{split} R_{0} &= \rho(FV^{-1}), \\ R_{0} &= \frac{1}{2} \begin{bmatrix} \frac{\eta_{5}\beta_{\mu}\theta_{e_{1}}}{\phi\mu_{i}(\mu_{i}+\delta_{i}+\gamma)} + \frac{\eta_{e}\beta_{\mu}(1-\theta)e_{1}}{\phi\mu_{i}(\mu_{i}+\delta_{i}+\gamma)} \\ &+ \sqrt{\left[\frac{\eta_{5}\beta_{\mu}\theta_{e_{1}}}{\phi\mu_{i}(\mu_{i}+\delta_{i}+\gamma)}\right]^{2} + \left(\frac{\eta_{e}\beta_{\mu}(1-\theta)e_{1}}{\phi\mu_{i}(\mu_{i}+\delta_{i}+\gamma)}\right)^{2} - \frac{2\eta_{5}\eta_{e}\beta_{\mu}^{2}\theta(1-\theta)e_{1}^{2}}{\phi^{2}\mu_{i}^{2}(\mu_{i}+\delta_{i}+\gamma)^{2}} + \frac{4c_{2}^{2}\eta_{2}^{2}(1-\varepsilon\tau)^{2}\beta_{\mu}^{2}(1-\theta)}{\mu_{i}^{2}(\mu_{i}+\delta_{i}+\gamma)^{2}} \end{bmatrix}} \end{split}$$

GLOBAL STABILITY OF DISEASE FREE EQUILIBRIUM

Global stability of epidemiological model is the most important and makes the model predictable as it guarantees that the model is independent of the initial size of the population this is achieved through (Castilo- Chavez et al 2002).

Theorem 2

The disease free equilibrium of the model (1) is Globally Asymptotically stable

(GAS) if
$$R_0 < 1$$

Proof

In establish the global stability of the disease free equilibrium, the two conditions (H1) and (H2) as in Castilo- Chavez et al 2002) must be satisfied for $R_0 < 1$ the model in (1) write in the form

$$\frac{dX_1}{dt} = F(X_1, X_2)$$

$$\frac{dX_2}{dt} = G(X_1, X_2); G(X_1, 0)$$

Where $X_1 = (x, z, v, w,)$ and $X_2 = (y, u, p)$

Component of $X_1 \in \mathbb{R}^4$ denoting uninfected population and the component of $X_2 \in \mathbb{R}^3$ denoting infected population.

From

$$E_{0} = (X_{1}^{*}, 0), X_{1}^{*} = \left(\frac{\beta_{H} \theta N_{H}}{\mu_{1}}, 0 \frac{\beta_{H} (1 - \theta) N_{H}}{\mu_{1}}, 0, 0, 0, 0\right)$$
(4)

Now, the first component is globally asymptotically stability of X_1^* , obtain

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{bmatrix} \beta_H \theta N_H - \mu_1 x \\ \beta_H (1 - \theta) N_H - \mu_1 z \\ 0 \\ 0 \end{bmatrix} (5)$$

From above equation (4), solve first order differential equation that is

$$\frac{dx}{dt} + \mu_1 x = \beta_H \theta N_H \tag{6}$$

If integrating Factor $I.F = e^{\mu_i t}$, multiplying (6) by I.F obtain

$$e^{\mu_{t}t}\left(\frac{dx}{dt} + \mu_{1}x = \beta_{H}\theta N_{H}\right) \Rightarrow e^{\mu_{t}t}\frac{dx}{dt} + \mu_{1}xe^{\mu_{t}t} = \beta_{H}\theta N_{H}e^{\mu_{t}t}$$
(7)

Write equation (7) as total differential

$$\frac{d}{dt} \left(x e^{\mu_l t} \right) = \beta_H \theta N_H e^{\mu_l t} \tag{8}$$

Integrate both sides of (8), obtain

$$xe^{\mu_{1}t} = \frac{\beta_{H}\theta N_{H}e^{\mu_{1}t}}{\mu_{1}} + C$$
 divide through by

 $e^{\mu_1 t}$ obtain

$$x(t) = \frac{\beta_H \theta N_H}{\mu_1} + C e^{-\mu_1 t}$$
(9)

Applying
$$x(0) = 0$$
, obtain

$$x(t) = \frac{\beta_H \theta N_H}{\mu_1} - \frac{\beta_H \theta N_H}{\mu_1} e^{-\mu_1 t}$$
(10)

Collect like term

$$x(t) = \frac{\beta_H \theta N_H}{\mu_1} \left(1 - e^{-\mu_1 t} \right)$$
(11)

Taking the limit as $t \to 0$, it gives $x(t) = \frac{\beta_H \theta N_H}{2}$

$$\mu_1 \tag{12}$$

Invariably,

$$z(t) = \frac{\beta_H (1-\theta) N_H}{\mu_1}$$
(13)

Hence,

DFE

$$X_{1}^{*} = \left(\frac{\beta_{H}\theta N_{H}}{\mu_{1}}, 0, \frac{\beta_{H}(1-\theta)N_{H}}{\mu_{1}}, 0, 0, 0, 0\right)_{\text{is}}$$

globally asymptotically stable. For second conditions that is $G(X_1, X_2) = AX_2 - G(X_1, X_2)$ It gives

$$A = \begin{pmatrix} -(\mu_1 + \delta_1 + \gamma) & \frac{nx}{N_H} \\ \frac{mz}{N_H} & -(\mu_1 + \delta_1 + \gamma) \\ 0 & 0 \end{pmatrix}$$

Clearly, this is an M-matrix

$$G(X_1, X_2) = \begin{pmatrix} \frac{(nu + \eta_3 w + \eta_5 p)x}{N_H} & -(\mu_1 + \delta_1 + \gamma) \\ \frac{(my + \eta_4 w + \eta_6 p)z}{N_H} & -(\mu_1 + \delta_1 + \gamma) \\ e_3 w - \phi p & e_2 + e_1 \end{pmatrix}$$

Then, $\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2) \ge 0$

 $\hat{G}(X_1, X_2) \ge 0$ Since all the parameters are assumed to be non-negative,

This clearly $\hat{G}(X_1, X_2) \ge 0$ hence, this end proof.

SENSITIVITY ANALYSIS

Table 2: Sensitivity indices of parameters for the Lassa model	reproduction number R_0 to
Paramotor	Sonsitivity indiana

r al allietel	Sensitivity mulces		
$eta_{\scriptscriptstyle H}$	+1.00000001		
<i>C</i> ₂	+0.9735166		
$\eta_{_2}$	+0.9735166		
e_1	+0.0264834		
${\eta}_{6}$	+0.01052437		
η_5	+0.01595903		
heta	-1.46010237		
μ_1	-1.00179856		
γ	-0.98021583		
τ	- 0.08555899		
ε	-0.08465362		
δ_1	-0.01798561		
ϕ	-0.0264834		

Sensitivity analysis is to assess the contribution and the relative impact of each of the parameters on basic reproduction number. The normalized forward sensitivity of the basic reproduction number with respect to all parameters tabulated in Table 3 is computed. The index allows us to measure the relative changes in a variable when a parameter changes.

The sensitivity index may be alternatively defined using partial derivatives. The normalized sensitivity index of R_0 that depends differentially on a parameter p is $y_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$

defined

RESULT OF NUMERICAL SIMULATION

by

Numerical simulation of the model was carried out by the help of MAPLE 18 software using the Runge-Kuta method of order four (4) together with parameter value given in the table 4.

Table 3.	Table of	parameters	and	their	values.

Parameter	Values	References
$\beta_{\scriptscriptstyle H}$	0.038	CIA(2015)
β_R	0.56	Estimated
η_1	0.6	Estimated
η_2	0.5	Estimated
η_3	0.5	Estimated
$\eta_{_4}$	0.5	Estimated
η_5	0.5	Estimated
ϕ	0.001	Estimated
τ	0-1	Abdulrahaman(2014)
σ	0.7	Estimated
δ_1	0.2	Estimated
δ_2	0.3	Estimated
μ_1	0.02	Estimated
μ_2	0.6	Estimated
<i>e</i> ₁	0.01	Estimated
Е	0.8	Garba&Gumel(2010)
<i>e</i> ₂	0.7	Estimated
<i>e</i> ₃	0.9	Estimated
<i>C</i> ₁	2.0	Williams et al (1996)
C_{2}	3.0	Williams et al (1996)







Fig 2: Show behaviours of Infected male

DISCUSSION

In figure 1 as the average number of female partner acquired increases. susceptible male population increases as a result of birth rate and recovery rate of infected male but later decreases as a result of increasing in female partner acquired. In figure 2, infected male population decreases but later increases a little bit before finally decreases, this attributed to increasing in number of female partners acquired. Also, in figure 3, initially susceptible female population increases but later decreases as a result of sexual transmission with infected male.

Finally, in figure 4, infected female population decreases and increases as a result of sexual transmission.







CONCLUSION

mathematical Α model was developed and analyzed to gain more insight into the effect of transmission on dynamical spread of Lassa fever. Mathematically, the model consists of seven - nonlinear system of differential equation. It shows that there exists a domain D where the model is mathematically and epidemiologically well posed. The basic reproduction number was determined, disease free equilibrium point was obtained and global stability of disease free equilibrium point is stable. Also from the table 3 above, the sensitivity analysis of the model reveals that the most sensitive parameters to the basic reproduction number R_0

 \mathbf{K}_0 are human birth rate, followed by average number of female partners acquired

by susceptible male C_2 and transmission rate resulting from sexual interaction between

infected male and susceptible female η_2 have the highest values. It concludes that the average number of female partners acquired by susceptible male and sexual transmission rate between the infected male and susceptible female should be reduced.

Acknowledgement: None

Conflict of Interest: None

Source of Funding: None

REFERENCES

- 1. Anderson, R. M. and May R. M. (1991). Infectious Diseases of Humans: Dynamics and Control. Oxford: Oxford University Press.
- Bawa, M., Abdulrahaman, S., Jimoh, O.R., & Adabara, N.U. (2013). *Stability analysis* of the disease-free equilibrium state of Lassa fever disease. Journal of Science and Technology, Mathematics and Education (JOSTMED), 9(2), 115 – 123.
- 3. Castill- Chavez C., Feng Z., & Huang W. (2002). On the computation of R_0 and its role on global stability.
- Centre for Disease Control. Imported Lassa fever. Morbidity Mortal Weekly Reports, 53(38), 894-897
- 5. Central Intelligence Agency (2015). World fact book for the year 2014. Retrieved on 20 February 2016 from http://www.cia/library/publication/the-world fact book/geos/ni.htm.
- Driessche, V. P., & Wathmough, J. (2005). Reproductive Number and Sub-Threshold Endemic Eqilibria for Compartment Modelling of Disease Transmission. Mathematics Bioscience, 180, 29-48.
- Eze, K. C., Salami T. A. T, Eze I. C., Pogoson, A. E., Omodia, N., & Ugochukwu, M. (2010). *High Lassa Fever Activity in Northern Part of Edo State Nigeria: Re Analysis of Confirmatory Test Result*, African Journal of Health Sciences,16(34), 52-56.
- Fisher-Hoch, S.P., Tomori, O., Nasidi, A., Perez-Oronoz, G.I., Fakile, Y., & Hutwagner, L. (1995). *Review of cases of* nosocomial Lassa fever in Nigeria: the high price of poor medical practice. Biomedical Journal, 311, 857–869.

- Garba M. S., Gumel, A.B., & Abubakar, M.R. (2008), *Backward Bifurcation in Dengue transmission dynamics*. Mathematic al Bioscience Doi:10.1016/j.mbs.2008.05.00 2.
- 10. Garba, S. M., & Gumel, A. B. (2010). Mathematical Recipe for HIV Elimination in Nigeria. Journal of the Nigeria Mathematical Society, 29, 51-112 **Mathematics** International Journal of and Statistics Studies Vol.4, No.1, pp.30-49, February 2016 Published by European Centre for Research Training and Development UK (www.eajournals.org)
- 11. Günther, S., Weisner, B., Roth, A., Grewing, T., Asper, M., Drosten, C., Emmerich, P., Petersen, J., Wilczek, M., & Schmitz, H.(2001). Lassa Fever Encephalopathy: Lassa Virus in Cerebrospinal Fluid but Not in Serum. The Journal of Infectious Diseases, 184(3), 345–349. doi:10.1086/322033.2001. 11443561
- Hethcote, H.W. (1978). An Immunization Model for Heterogeneous Population. Theoretical Population Biology 14(1978), 338-349
- 13. Lakshmikantham, V., Leela, S., & Martynyuk, A. A. (1999). *Stability Analysis of Non-linear systems*, 164. New York and Basel: Marcel Dekker, Inc.
- Okuonghae, D. & Okuonghae, R. (2006). A Mathematical model for Lassa fever. Journal of the Nigerian Association of Mathematical Physics, 10,457-464.
- 15. Ogbu,O. E., Ajuluchukwu, C. J.,& Uneke, C.J. (2007). *Lassa fever in West Africa subregion: an Overview*. Journal of Vector Borne Diseases 44, 1-11.
- Ogabi, C.O., Olusa, T.V., & Madufor, M.A. (2012). Controlling Lassa Fever in Northern Part of Edo State, Nigeria using SIR Model. New Science Journal 5(12), 115-121.
- 17. Omilabu, S.A, Badaru, S.O., Okokhere, P., Asogun, D., Drosten, C., & Emmerich, P. (2005). Lassa fever, Nigeria, 2003 and 2004. Emerging Infectious Diseases, 11,1642–4.
- Onuorah, M.O., Ojo, M.S., Usman, D.J., Ademu, A.(2016) Basic Reproductive Number for the Spread and Control of Lassa fever, International Journal of Mathematics Trends and Technology (IJMTT). Vol. 30(1):1-7

- 19. Omoloye, M.A., Yusuff, M. I. & Emiola, O.K.S. Application of differential transformation solving method for dynamical transmission of Lassa fever model. World Academy of Science. Engineering and Technology International Journal of Physical and Mathematical Sciences Vol: 14, No: 11, 2020, Pp: 151-154.
- Onuorah, M.O, Akinwande, N.I,Nasir, M.O and Ojo, M.S, Sensitivity analysis of lassa fever model, International Journal of Mathematics and Statistics studies. Vol.4, No.1, Pp30-49,February 2016
- 21. Promed-mail. (2006). Lassa *fever Liberia* (02). Number 20061001.2812. Retrieved

on July 8, 2013 from http://www.promedma il.chip.org/pipermail/promed/003770.html

- 22. Richmond, J. K., & Deborah, J. B. (2000). Lassa Fever Epidemiology, Clinical features, and Social Consequences. Biomedical Journal, 327, 1271-1275.
- 23. Tara, K. H. (2004). *Virology notes in Lassa fever*. Retrieved on March 10, 2012 from www.taraharper.com/v lass.html World Health Organisation. (2004).

How to cite this article: Omoloye MA, Sanusi AO, Sanusi IO et.al. Modeling and sensitivity analysis of dynamical transmission of lassa fever. *International Journal of Research and Review*. 2021; 8(10): 531-539. DOI: https://doi. org/10.52403/ijrr.20211067
