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Research article

Mathematical model formulation and analysis of the transmission dynamics of Lassa fever

K.O. Achema[1]*, D. J. Yayaha[2], W.T. Ademosu[1]

[1]Department of Mathematics, Joseph Sarwuan Tarka University, Makurdi, Benue State, Nigeria.
[2]Department of Mathematics and Statistics, Confluence University of Science and Technology, Osara, Kogi State, Nigeria.

*Corresponding Author: K.O. Achema. Email: achema.kenneth@uam.edu.ng

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Abstract: In 1969, two missionary nurses died due to Lassa fever infection, which led to the identification of the Lassa virus (LASV) in Nigeria. Infections from the Lassa virus are about 80% asymptomatic, but severe cases normally result in multi-organ failure or death. This accounts for about 15% of the hospitalized cases. Different scientific strategies to eradicate the disease have yielded minimal results. In this study, a mathematical model to study the transmission dynamics of Lassa fever is formulated and analyzed. The model has five compartments. The human population is compartmentalized into three sub-populations, while the rodent population is compartmentalized into two sub-populations. The model has two equilibrium states, namely, the disease-free equilibrium (DFE) and the disease endemic equilibrium (DEE). The stability analysis of the DFE revealed that it is locally asymptotically stable when the basic reproduction number (R_0) is less than one and unstable otherwise. The sensitivity analysis on the model reproduction number revealed that the infection transmission rates from human-to-human, rodent-to-human, and from human-to-rodent are the causes of the disease persistence in the human population. The Hopf-bifurcation analysis of the model using the transmission rate from both rodents and humans to humans as the bifurcation parameter shows the stability point of the model at $\alpha_h = 0.025$. The numerical analysis result perfectly aligns with the model's qualitative results obtained.

Mathematics Subject Classification: 00X00, 00G00, 00D00
Keywords: Mathematical model; Lassa fever; reproduction number; analysis.

1 Introduction

After two missionary nurses died from Lassa fever in 1969, the Lassa virus (LASV) was discovered, which led to the initial identification of Lassa fever in Nigeria. The disease is caused by a zoonotic virus that is present in Mastomys/multimammate rodents and is spread from person to person by contact with infected rodents. It is now endemic in Nigeria and poses a serious threat to public health because, although many infections are mild, it can spread and, in certain situations, cause serious illness. About 80% of infections

are mild or asymptomatic, but in severe cases, it can cause multi-organ failure, bleeding, and death, with a case fatality rate of 1-15% among hospitalized patients. The disease can also spread person-to-person through contact with blood and other body fluids of infected individuals. The causative agent of Lassa virus is a single-stranded RNA virus from the Arenaviridae family [11].

Close contact with the blood, urine, feces, saliva, or other bodily fluids of an infected Lassa fever patient can spread the disease. Contact with the urine, feces, or saliva of infected rats can infect humans. This can occur by inhaling dust from dirty surfaces where infected rats are present, touching contaminated objects or surfaces, or consuming or drinking contaminated food or water. Lassa fever symptoms include the following. Fever, general weakness and malaise, headache, sore throat, bleeding from the gums or other body tissues, difficulty breathing, nausea, chest pain, facial swelling, neurological impairment, and circulatory failure are some of the causes. It leads to complicated problems like; Sensorineural deafness is a common sequela, with varying incidence rates reported. Lassa fever is particularly dangerous during pregnancy, with very high rates of maternal and fetal death, especially in the third trimester [9].

A study conducted by [7] about the modelling dynamics of Lassa fever in Nigeria. They presented deterministic dynamic model to study its transmission behavior in the population. To mimic the disease biological history, they divide the population in to two groups: humans and rodents. The results shows that if $R_0 < 1$ then the system is stable, otherwise unstable. The model findings were performed using the non-linear least square method on cumulative report cases in Nigeria. Another study investigates by [3] about a non-autonomous system of non-linear ordinary differential equations that capture the dynamics of Lassa fever transmission and seasonal variation in the Mastomys rodent birth. They evaluate LF disease intervention strategies by predicting optional intervention best fit in controlling the disease in the population using the electricity of the equilibria prevalence. A mathematical model of the transmission dynamics of Lassa fever infection with in two different but complementary hosts was presented [4]. The model includes a death infectious human compartment that can affect vulnerable individual. According to the study's findings, the best way to control secondary transmission dynamics is to establish more Lassa fever diagnostic centers and use burial practices.

Lassa fever continues to threaten global health, though there are ongoing efforts to reduce the incidence and burden of the disease. Here, a deterministic mathematical model is developed to study the transmission dynamics of the disease with the impacts of the proposed control strategies. The compartmental model comprises a set of coupled ordinary differential equations (ODE) representing human-to-human and rodent-to-human pathways. The basic reproduction number was derived using the Next-generation matrix approach. Furthermore, the Lassa fever epidemic scenario was formulated as an optimal control problem while Pontryagin's Maximum Principle was adopted to obtain the associated optimality system. The resulting system was solved numerically and simulated for different epidemic scenarios. Findings from our simulations show that optimal combinations of early hospitalization, public health education, timely detection cum treatment, and pest control strategies would result in a remarkable reduction of the disease spread. Moreover, conscientious implementation of these proposed control measures could lead to eradication of the epidemic within 100 days [6,8].

A study by [1] investigates about the effect of treatment compliance on the dynamics and control of Lassa fever. The model validity was examined and established using ample mathematics theorems. The equilibria and a threshold for disease eradication were derived. The stability was analyzed and the necessary and sufficient conditions for the equilibria of the model to be stable both locally and globally were derived. Further, sensitivity analysis was carried out to investigate the relative contributions of various parameters to Lassa fever spread and management. Numerical simulation was later conducted via a logical parameter value from the literature to visualize the effect of parameters perturbations on the dynamics of the disease. Results from the study revealed that Lassa fever eradication is a function of total compliance to treatment procedures.

Despite continuous efforts by the Nigerian Centre for Disease Control (NCDC) and international partners to curb Lassa fever, outbreaks persist annually, affecting both rural and urban populations. The disease's persistence indicates that current control measures are insufficient to stop its spread. The intricacy of Lassa fever dynamics, including the part played by rodent reservoirs, asymptomatic human carriers, and potential environmental contamination, requires a strong mathematical framework. Knowing how these elements work together will help identify the main forces behind transmission and aid in the development of efficient control plans suited to the Nigerian environment.

The organisation of this paper is as follows. The model is formulated in Section 2. The analyses of the formulated model and its numerical simulation are carried out in Section 3, while the results of the model analyses and simulations are discussed in Section 4. The main conclusions from this study are summarized in Section 5.

2 Model Formulation and Analysis

2.1 Assumptions of the model equations

The following assumptions are valid for the model formulation.

H_1 Both symptomatic and asymptomatic humans are classified as infected humans.

H_2 Recovered human may still become susceptible.

2.2 Model variables and parameters

The model variables and parameters are given as Table 1.

2.3 Model flow diagram

In order to formulate the model equations the following flow diagram is appropriate.

Variable	Interpretation
S	Susceptible human population
I	Infected human population
R	Recovered human population
M	Susceptible rodents
J	Infected rodents
Parameter	**Interpretation**
Λ_h	Recruitment rate of humans
γ	Immunity warning rate of humans
ϵ	Recovered rate of infected humans
μ_h	Natural death rate of humans
δ_h	Disease induced death rate of humans
α_h	Transmission probability from humans to rodents
α_r	Transmission probability from rodents and human
Λ_r	Recruitment rate of rodent through birth
μ_r	Natural death rate of rodents

Table 1: Description of the model variables and parameters

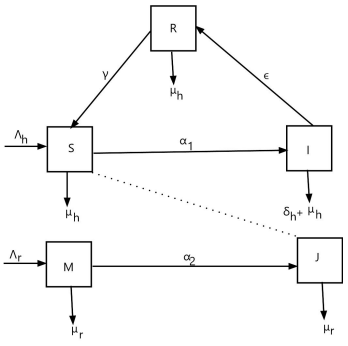

Figure 1: Model flow diagram

2.4 Model equations

Keeping track of the above assumptions, variables, and parameters descriptions, the formulated model equations are given by

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda_h + \gamma R - \alpha_1 S - \mu_h S, \\
\frac{dI}{dt} &= \alpha_1 S - (\epsilon + \mu_h + \delta_h) I, \\
\frac{dR}{dt} &= \epsilon I - (\mu_h + \gamma) R, \\
\frac{dM}{dt} &= \Lambda_r - (\alpha_2 + \mu_r) M, \\
\frac{dJ}{dt} &= \alpha_2 M - \mu_r J,
\end{aligned} \tag{2.1}$$

where $\alpha_1 = \frac{\alpha_r J + \alpha_h I}{N_h}$ and $\alpha_2 = \frac{\alpha_r I}{N_r}$,

Subject to the following initial conditions $S(0) > 0,\ I(0) \geq 0, R(0) \geq 0,\quad M(0) > 0, J(0) \geq 0.$

3 Analysis of the model equations

In this section, the researcher considers the following under the analysis of the model; Disease-free equilibrium, basic reproduction number, local stability and sensitivity analysis of Lassa fever.

3.1 Disease-free equilibrium (DFE)

Disease-free equilibrium is a state at which disease no longer exist in a compartment.

To compute DFE state we let

$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dM}{dt} = \frac{dJ}{dt} = 0$

Thus,

$$\begin{aligned}
&\Lambda_h + \gamma R - \alpha_1 S - \mu_h S = 0 \\
&\alpha_1 S - (\epsilon + \mu_h + \delta_h) I = 0 \\
&\epsilon I - (\mu_h + \gamma) R = 0 \\
&\Lambda_r - (\alpha_2 + \mu_r) M = 0 \\
&\alpha_2 M - \mu_r J = 0
\end{aligned} \tag{3.1}$$

At DFE $I = J = 0$, we have,

$$\epsilon I - (\mu_h + \gamma) R = 0$$

$R = 0$ hence, I = 0. Substituting in the appropriate places to get

$$S_0 = \frac{\Lambda_h}{\mu_h}, \quad M_0 = \frac{\Lambda_r}{\mu_r}$$

Therefore, the DFE is

$$\varepsilon_0 = (\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_r}{\mu_r}, 0)$$

3.1.1 Disease Endemic Equilibrium (DEE)

The disease endemic equilibrium is obtained by setting the left hand side of the equation (2.1) to zero and solved for each of the state variable. Thus, the disease endemic equilibrium becomes

$$\varepsilon_1 = (S^*, I^*, R^*, M^*, J^*),$$

where

$$\begin{aligned}
S^* &= \frac{\Lambda_h(\gamma + \mu_h)}{(\alpha_1^* + \mu_h)(\gamma + \mu_h - \epsilon\gamma\alpha_1^*)}, \\
I^* &= \frac{\Lambda_h(\gamma + \mu_h - \epsilon\gamma\alpha_1^*) + \gamma\epsilon\Lambda^h\alpha_1^*}{(\gamma + \mu_h - \epsilon\gamma\alpha_1^*)(\alpha_1^* + \mu_h)}, \\
R^* &= \frac{\epsilon\Lambda_h\alpha_1^*}{\gamma + \mu_h - \epsilon\gamma\alpha_1^*}, \\
M^* &= \frac{\Lambda_r}{\alpha_2^* + \mu_r}, \\
J^* &= \frac{\alpha_2^* M}{\mu_r}.
\end{aligned}$$

3.2 Basic reproduction number

The basic reproduction number, R_0, is defined as the average number of new infections generated by a single infected individual in a completely susceptible population. Basic reproductive number is used to determine the transmission potential of a disease. Taken the infected classes and using the next generation technique to calculate the basic reproduction number by the next-generation matrix approach [5]

Considering the infected class of the model to be I and J. We now obtain

$$f = \begin{pmatrix} \alpha_1 S \\ \alpha_2 M \end{pmatrix} \quad \text{and} \quad v = \begin{pmatrix} (\epsilon + \mu_h + \delta_h)I \\ \mu_r J \end{pmatrix}.$$

But $\alpha_1 = \frac{\alpha_r J + \alpha_h I}{N_h}$ and $\alpha_2 = \frac{\alpha_r I}{N_r}$ where $N_h = S + I + R$ and $N_r = M + J$. Then, $f_1 = \frac{\alpha_r J + \alpha_h I}{N_h} S$ and $f_2 = \frac{\alpha_r I}{N_r} M$, $v_1 = (\epsilon + \mu_h + \delta_h)I$ and $v_2 = \mu_r J$.

Now obtaining the Jacobian matrix of F and V with respect to I and J, we use

$$\partial f(I,J) = F = \begin{pmatrix} \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial J} \\ \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial I} \end{pmatrix} \quad \text{and} \quad \partial v(I,J) = V = \begin{pmatrix} \frac{\partial v_1}{\partial I} & \frac{\partial v_1}{\partial J} \\ \frac{\partial v_2}{\partial I} & \frac{\partial v_2}{\partial I} \end{pmatrix}$$

Thus, F and V matrices are obtained as:

$$F = \begin{pmatrix} \frac{\alpha_h \mu_h}{\Lambda_h} S & \frac{\alpha_r \mu_h}{\Lambda_h} S \\ \alpha_r & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \epsilon + \mu_h + \delta_h & 0 \\ 0 & \mu_r \end{pmatrix}$$

Thus,

$$F(\varepsilon_0) = \begin{pmatrix} \alpha_h & \alpha_r \\ \alpha_r & 0 \end{pmatrix}$$

and the inverse of V is given by

$$V^{-1} = \begin{pmatrix} \frac{1}{(\epsilon+\mu_h+\delta_h)} & 0 \\ 0 & \frac{1}{\mu_r} \end{pmatrix}$$

Therefore, $F(\varepsilon_0)V^{-1} = \begin{pmatrix} \alpha_h & \alpha_r \\ \alpha_r & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\epsilon+\mu_h+\delta_h)} & 0 \\ 0 & \frac{1}{\mu_r} \end{pmatrix}$

$$F(\varepsilon_0)V^{-1} = \begin{pmatrix} \frac{\alpha_h}{(\epsilon+\mu_h+\delta_h)} & \frac{\alpha_r}{\mu_r} \\ \frac{\alpha_r}{(\epsilon+\mu_h+\delta_h)} & 0 \end{pmatrix} \tag{3.2}$$

Or

$$F(\varepsilon_0)V^{-1} = \begin{pmatrix} K_1 & K_2 \\ K_3 & 0 \end{pmatrix} \tag{3.3}$$

where $K_1 = \frac{\alpha_h}{(\epsilon+\mu_h+\delta_h)}$, $K_2 = \frac{\alpha_r}{\mu_r}$ and $K_3 = \frac{\alpha_r}{(\epsilon+\mu_h+\delta_h)}$

Therefore, the characteristic equation corresponds to Eq. (3.4) is given by

$$\lambda^2 - K_1\lambda - K_2K_3 = 0 \tag{3.4}$$

Thus,

$\lambda_1 = \frac{K_1 + \sqrt{K_1^2 + 4K_2K_3}}{2}$ and $\lambda_2 = \frac{K_1 - \sqrt{K_1^2 + 4K_2K_3}}{2}$

Therefore, λ_1 is the dominant eigenvalue.

Hence, $\lambda_1 = R_0 = \frac{K_1 + \sqrt{K_1^2 + 4K_2K_3}}{2}$

Substituting the values of K_1, K_2 and K_3 we have

$$R_0 = \frac{1}{2}\left(\frac{\alpha_h}{\delta_h + \mu_h + \epsilon} + \sqrt{\frac{\alpha_h^2}{(\delta_h + \mu_h + \epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h + \mu_h + \epsilon)}} \right). \tag{3.5}$$

Equation(3.6) contains both human and vector parameters which represent, the average number of humans infected by an infected animal; and the average number of animals infected by an infected humans.

3.3 Sensitivity analysis

Sensitivity analysis is used to determine the impact of the model parameters on the model's basic reproduction number (R_0) [10]. The normalized forward sensitivity index of R_0 with respect to a parameter ζ (that is, model parameters) is given by

$$\Gamma_{\zeta}^{R_0} = \frac{\partial R_0}{\partial \zeta} \times \frac{\zeta}{R_0}.$$

The sensitivity indices for the parameters of the model are calculated as follows:

$$\Gamma_{\alpha_h}^{R_0} = \frac{\alpha_h}{(\delta_h + \mu_h + \epsilon)\sqrt{\frac{\alpha_h^2 + \frac{4\alpha_r^2(\delta_h+\mu_h+\epsilon)}{\mu_r}}{(\delta_h+\mu_h+\epsilon)^2}}}$$

$$\Gamma_{\epsilon}^{R_0} = \frac{\epsilon\left(\frac{-\frac{2\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^3} - \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)^2}}{2\sqrt{\frac{\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)}}} - \frac{\alpha_h}{(\delta_h+\mu_h+\epsilon)^2} \right)}{\frac{\alpha_h}{\delta_h+\mu_h+\epsilon} + \sqrt{\frac{\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)}}}$$

$$\Gamma_{\delta_h}^{R_0} = \frac{\delta_h\left(\frac{-\frac{2\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^3} - \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)^2}}{2\sqrt{\frac{\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)}}} - \frac{\alpha_h}{(\delta_h+\mu_h+\epsilon)^2} \right)}{\frac{\alpha_h}{\delta_h+\mu_h+\epsilon} + \sqrt{\frac{\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)}}}$$

$$\Gamma_{\mu_h}^{R_0} = \frac{\mu_h\left(\frac{-\frac{2\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^3} - \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)^2}}{2\sqrt{\frac{\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)}}} - \frac{\alpha_h}{(\delta_h+\mu_h+\epsilon)^2} \right)}{\frac{\alpha_h}{\delta_h+\mu_h+\epsilon} + \sqrt{\frac{\alpha_h^2}{(\delta_h+\mu_h+\epsilon)^2} + \frac{4\alpha_r^2}{\mu_r(\delta_h+\mu_h+\epsilon)}}}$$

$$\Gamma_{\alpha_r}^{R_0} = \frac{4\alpha_r^2}{\mu_r\left(\frac{\alpha_h^2}{\delta_h+\mu_h+\epsilon} + \alpha_h\sqrt{\frac{\alpha_h^2+\frac{4\alpha_r^2(\delta_h+\mu_h+\epsilon)}{\mu_r}}{(\delta_h+\mu_h+\epsilon)^2}} + \frac{4\alpha_r^2}{\mu_r}\right)}$$

$$\Gamma_{\mu_r}^{R_0} = -\frac{2\alpha_r^2}{\mu_r\left(\frac{\alpha_h^2}{\delta_h+\mu_h+\epsilon} + \alpha_h\sqrt{\frac{\alpha_h^2+\frac{4\alpha_r^2(\delta_h+\mu_h+\epsilon)}{\mu_r}}{(\delta_h+\mu_h+\epsilon)^2}} + \frac{4\alpha_r^2}{\mu_r}\right)}$$

3.4 Local stability

From the model equations, we have

$$\begin{aligned} f_1 &= \Lambda_h + \gamma R - \alpha_1 S - \mu_h S, \\ f_2 &= \alpha_1 S - (\epsilon I + \mu_h + \delta_h) I, \\ f_3 &= \epsilon I - (\mu_h + \gamma) R \\ f_4 &= \Lambda_r - (\alpha_2 + \mu_r) M \\ f_5 &= \alpha_2 M - \mu_r J \end{aligned} \tag{3.6}$$

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial M} & \frac{\partial f_1}{\partial J} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial S} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial S} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial S} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial S} \end{pmatrix}.$$

Therefore,

$$J(\varepsilon_0) = \begin{pmatrix} -\mu_h & 0 & \gamma & 0 & -\alpha_r \\ 0 & -(\epsilon+\mu_h+\delta_h) & 0 & 0 & \alpha_r \\ 0 & \epsilon & -(\mu_h+\gamma) & 0 & 0 \\ 0 & 0 & 0 & -\mu_r & -\alpha_r \\ 0 & 0 & 0 & 0 & -(\mu_h+\alpha_r) \end{pmatrix}.$$

Or

$$J(\varepsilon_0) = \begin{pmatrix} -\mu_h & 0 & \gamma & 0 & -\alpha_r \\ 0 & -K_1 & 0 & 0 & \alpha_r \\ 0 & \epsilon & -K_2 & 0 & 0 \\ 0 & 0 & 0 & -\mu_r & -\alpha_r \\ 0 & 0 & 0 & 0 & -(\mu_h+\alpha_r) \end{pmatrix}.$$

Where $K_1 = (\epsilon + \mu_h + \delta_h)$ and $K_2 = (\mu_h + \gamma)$

It is now sufficient to show that all the eigenvalues of $J(\varepsilon_0)$ are negative.

Hence, the first three eigenvalues are; $-\mu_h, -\mu_r$ and $-(\mu_h + \alpha_r)$. Other eigenvalues are obtained from the sub-matrix G which is written as;

$$G = \begin{pmatrix} -K_1 & 0 \\ \epsilon & -K_2 \end{pmatrix}.$$

According to Routh-Hurwitz condition, all the matrix G are real and negative if;

1. Trace (G) < 0
2. Determinant (G) > 0

We show that,

$$Trace(G) = -(K_1 + K_2) < 0$$

and

$$Det(G) = K_1K_2(1 - R_0) > 0$$

Therefore, all the eigenvalues of the Jacobian matrix G are real and negative if $\{R_h, R_r\} \in R_0 < 1$. The Lassa fever disease-free equilibrium is locally asymptotically stable and unstable if otherwise.

3.5 Global asymptotic stability

The global asymptotic stability analysis of the model is carried out by using the Lyapunov function:

$$\begin{aligned} L(S, I, R, M, J) &= S - S^* - S^* ln\frac{S}{S^*} + I - I^* - I^* ln\frac{I}{I^*} \\ &= R - R^* - R^* ln\frac{R}{R^*} + M - M^* - M^* ln\frac{M}{M^*} \\ &= J - J^* - J^* ln\frac{J}{J^*}, \end{aligned}$$

where $(S^*, I^*, R^*, M^*, J^*)$ is the endemic equilibrium of the model. The time derivative of L is:

$$\begin{aligned} \frac{dL}{dt} &= \left(1 - \frac{S}{S^*}\right)\frac{dS}{dt} + \left(1 - \frac{I}{I^*}\right)\frac{dI}{dt} + \left(1 - \frac{R}{R^*}\right)\frac{dR}{dt} \\ &\quad + \left(1 - \frac{M}{M^*}\right)\frac{dM}{dt} + \left(1 - \frac{J}{J^*}\right)\frac{dJ}{dt}. \end{aligned}$$

After substitution and simplification of the model equations, we obtain

$$\frac{dL}{dt} = -\mu_h(S - S^*)^2 - (\epsilon + \mu_h + \delta_h)(I - I^*)^2 - (\gamma + \mu_h)(R - R^*)^2 - \mu_r(M - M^*)^2.$$

Since the value of $\frac{dL}{dt}$ is negative (i.e. $\frac{dL}{dt} < 0$), the Lyapunov function is decreasing along the trajectories of the model system.

The largest invariant set in $\{(S, I, R, M, J) : \frac{dL}{dt} = 0\}$ is the endemic equilibrium of the model. By LaSalle's invariance principle, the endemic equilibrium is globally asymptotically stable (GAS) provided the $\frac{dL}{dt} < 0$.

3.6 Hopf-bifurcation analysis

To analyse the Hopf-bifurcation of the model equations accurately, we need to find the critical value of the bifurcation parameter (α_h) and the corresponding eigenvalues. The characteristic equation of model (1) is given by

$$\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0,$$

where $a_i (i = 1, 2, 3, 4, 5)$ represents the model parameters while the λ represents roots of the model equations.

Assuming the system undergoes Hopf bifurcation at $\alpha_h = \alpha_h^c$, we have a pair of purely imaginary eigenvalues:

$$\lambda_{1,2} = \pm i\Omega_0$$

The equilibrium or critical value α_h^c and Ω_0 satisfy:

$$-\Omega_0^2 + a_2^c + i\Omega_0(a_1^c\Omega_0^2 - a_3^c) = 0,$$

where a_i^c are evaluated at $\alpha_h = \alpha_h^c$.

Solving for Ω_0 and α_h^c we get

$$\Omega_0^2 = \frac{a_3^c}{a_1^c}$$

The explicit expressions for α_h^c and Ω_0 are complex depending on the model parameters values.

4 Results and Discussion

Here, numerical simulations for the system of equations of the model are presented and discussed. The solutions are obtained by using MATLAB and Mathematica.

4.1 Numerical Simulations

In this section, the numerical simulation of the model is presented. All the parameter values, source and their sensitivity indices for the model compartment are displayed. The table below shows the parameter

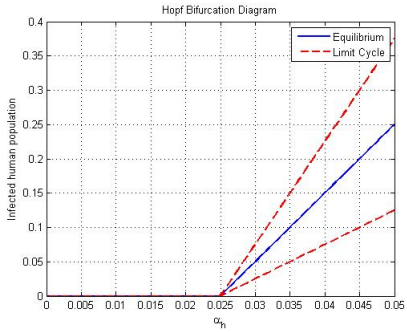

Figure 2: Bifurcation diagram

values, source and sensitivity index of the compartmental model.

Parameter	Parameter value	Source	Sensitivity index
Λ_h	10	[1]	-
γ	0.0150	Assumed	-
ϵ	0.1000	Assumed	-0.0767240
μ_h	0.0003	[7]	-0.0002302
δ_h	0.6	Assumed	-0.4603440
α_h	0.0100	Assumed	+0.07459680
α_r	0.0200	Assumed	+0.9254030
Λ_r	0.1000	[7]	-
μ_r	0.0627	[7]	-0.4627020

Table 2: Description of the model variables and parameters

5 Conclusions

In Figure 2, a limit cycle occurs at $\alpha_h = 0.025$, which represents the bifurcation parameter. The sudden change in this parameter, which represents the transmission rate of infection from human to human, can disrupt the system stability. Figure 3 shows the infected human population over time at varying α_1, a parameter representing both the transmission rate of infection from rodents and humans to humans. The higher the transmission rate, the higher the infected human population grows. However, a steady-state equilibrium is achieved after 60 days of the disease outbreak. In Figure 4, the infected population is studied over time at varying parameter γ, the vaccine waning rate. The infection thrives when the vaccine waning rate is higher. However, a steady stable equilibrium is achieved after 40 days. Figure 5 is a plot of human population over time at $\alpha_1 = 0.5$ (representing infection transmission rate from both rodents and humans to humans). The recovered population grew higher than the susceptible and infected populations, which were later contained. A similar figure is plotted in Figure 6 with $\alpha_1 = 0.9$. In this case, the infected population outgrows the susceptible population, but the three sub populations coexist. Figure 7 is also similar to

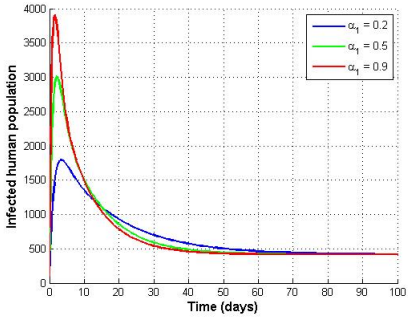

Figure 3: Infected human population over time

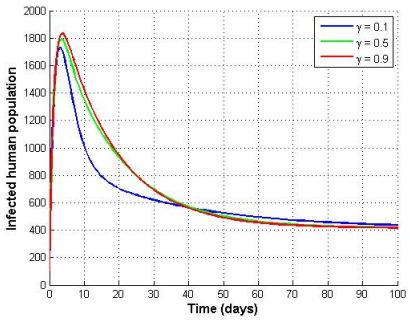

Figure 4: Infected human population over time at varying parameter values of γ

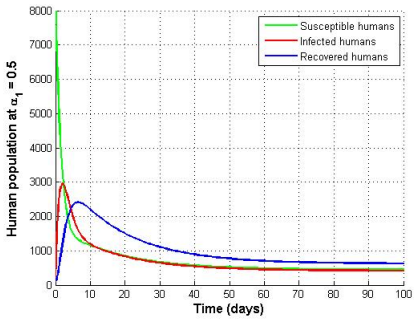

Figure 5: Infected human population over time at $\alpha_1 = 0.5$

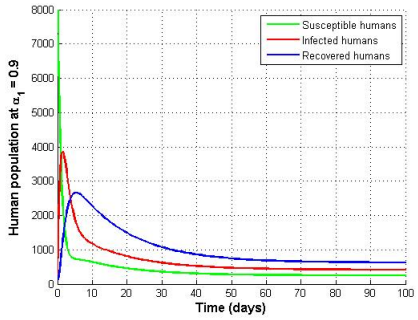

Figure 6: Infected human population over time at $\alpha_1 = 0.9$

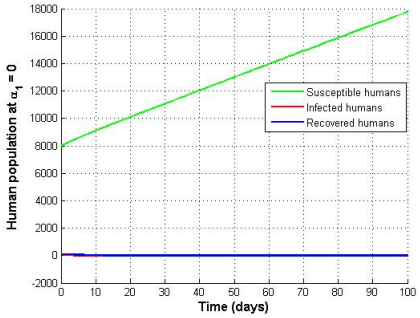

Figure 7: Infected human population over time at $\alpha_1 = 0$

Figures 5 and 6 but with $\alpha_1 = 0$ (i.e., absence of infection). The susceptible population grows without bound, and the infected and recovered populations maintain zero population since there is no infection. This figure demonstrates the computed equilibrium value of the disease-free equilibrium (DFE) of the human population component.

A mathematical model to study the dynamics of Lassa fever is formulated and analysed for possible control strategies. Various analyses such as the disease equilibrium states, stability, sensitivity of parameters' values of the basic reproduction number, Hopf-bifurcation, and simulation were carefully considered. Disease reduction or elimination depends on the disease threshold value, that is, the basic reproduction number. It was revealed that the disease will die out when $R_0 < 1$ and persist when $R_0 > 1$. Bifurcation analysis is required whenever $R_0 = 1$. The two parameters (α_h and α_r) which represent the transmission rates of Lassa virus infection from human and rodent to human, respectively, are critical for disease elimination strategies.

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