ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

Formulation and Qualitative Analysis of a Mathematical Model for Lassa Fever Transmission Dynamics with DOTS as Control Measure

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Abstract
This study develops a compartmental mathematical model to describe the transmission of Lassa fever among human and rodent populations. The model incorporates the Direct Observation therapy strategy as control measure. We derive the basic reproduction number R_0using the next generation matrix and analyze the stability of disease-free and endemic equilibria. The results provide theoretical thresholds crucial for understanding disease persistence and eradication dynamics.

Keywords: DOTS, stability, reproduction number

Introduction

Lassa fever (LF) is a zoonotic disease spread by infected rodents known as multimammate rats (Ojo *et al*, 2021; Adewale *et al*, 2009). Lassa fever is an acute viral haemorrhagic illness caused by the Lassa virus, belonging to the category of viral haemorrhagic fever. Notably, individuals infected by the virus may not display immediate symptoms, with manifestations typically emerging after an incubation period of up to 21 days (Andrew *et al*, 2013; Sambo & Madubueze, 2020**)**

Symptom onset is characterized by fever, general weakness, headache, vomiting, and muscle pain. Severe cases frequently progress to mucosal and gastrointestinal hemorrhaging. (Andrew *et al.,* 2013; Akindokun *et al.,* 2024; Musa *et al*, 2020**).** The case fatality rate (CFR) following infection is approximately 1%, with mortality typically occurring within 14 days of symptom onset. For those who survive, some may endure hearing loss, though this impairment may gradually improve within three months (Andrew *et al.,* 2013; Akindokun *et al.,* 2024; Olupitan *et al*, 2024**).**

Understanding the intricacies of LF transmission and its varied clinical presentation is vital for devising effective control and prevention strategies. The role of multimammate rats as vectors underscores the importance of addressing not only the human population but also implementing measures to curb the rodent population, thereby mitigating the risk of transmission and enhancing overall public health (Mayowa *et al.,* 2021; Adesola *et al*, 2024**).**

The primary transmission pathway involves human contact with the urine or faeces of infected multimammate mice. This zoonotic transfer marks the initial link in the chain of human infection. As the disease progresses, direct human-to-human contact becomes another avenue for its spread, adding a layer of complexity to containment efforts. The diagnosis of this ailment based solely on symptoms is a formidable challenge due to the diverse and often non-specific nature of its clinical presentation. Consequently, the confirmation of infection relies on sophisticated laboratory testing
methods, including the identification of the virus's ribonucleic acid (RNA), the presence of antibodies specific to the virus, or the isolation of the virus itself through cell culture techniques. It's worth noting that the symptoms of this disease share similarities with other prevalent illnesses including Ebola virus disease, malaria, typhoid, and yellow fever, further underscoring the importance of accurate and timely diagnostic procedures (Al-Mustapha *et al*, 2024; Okolo *et al*, 2020).

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Model Foemulation

Motivated by the work of Ojo *et al* (2021), we hope to formulate our model to incorporate DOTS. This would be done by introducing the treatment parameter (T_h) to the model proposed by Ojo *et al* (2021) and the incorporation of direct observation therapy strategy (DOTS).

We present below a seven compartmental deterministic systems of nonlinear ordinary differential equations, to study the transmission dynamics of Lassa fever in Nigeria:

The model comprises of two interacting population of human and rodent at time t denoted by $N_h(t)$ and $N_r(t)$ respectively. The human population is further divided into five compartments at any time t; Susceptible $S_h(t)$, Exposed $E_h(t)$, infectious $I_h(t)$, Treated $T_h(t)$ and Recovered $R_h(t)$ human compartments. On the other hand, the rat population is divided into two compartments at any time t; Susceptible $S_r(t)$ and Infectious $I_r(t)$ rat compartments. Hence we have that:

$N_h(t) = S_h(t) + E_h(t) + I_h(t) + T_h(t) + R_h(t)$ and

$N_r(t) = S_r(t) + I_r(t)$

The compartmental model which shows the mode of transmission of lassa fever between the two interacting populations is shown in the figure below:

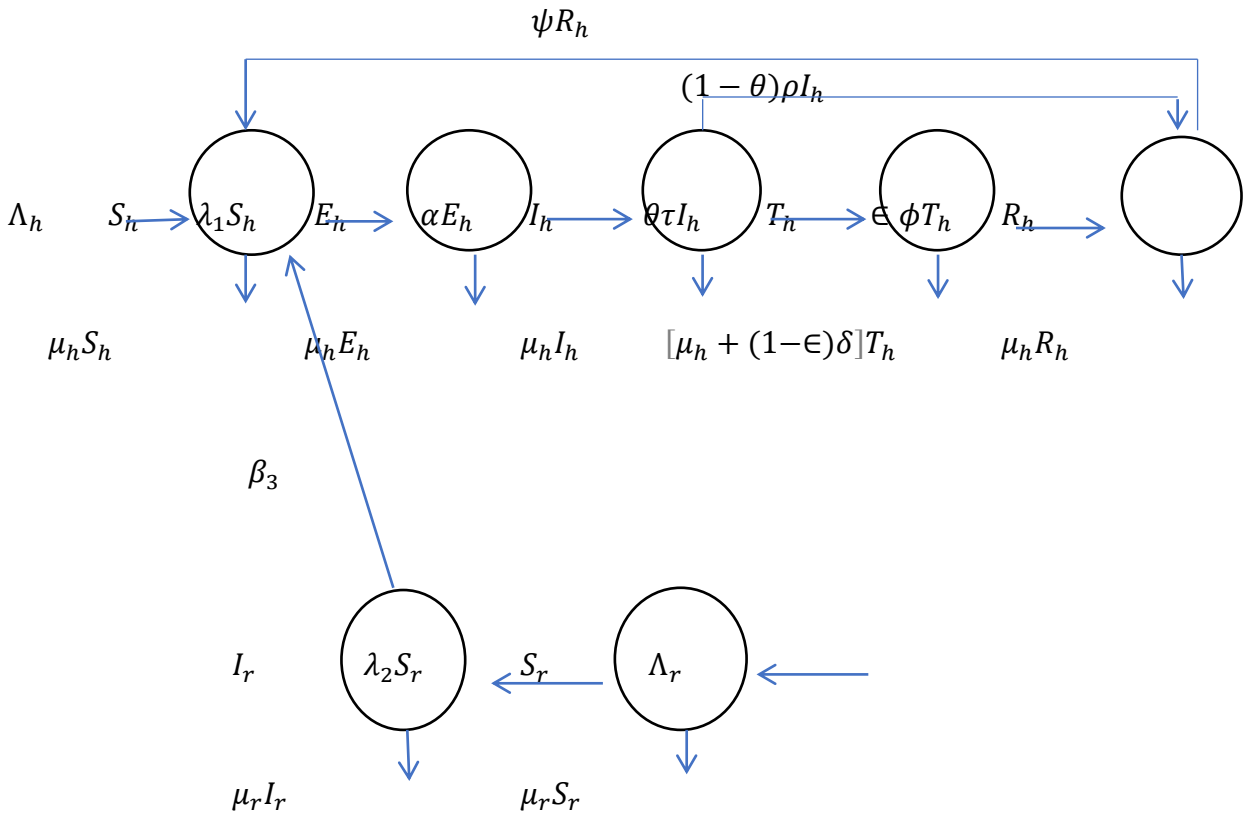

Figure 1: Compartmental model which shows the mode of transmission of lassa fever

Where;

$$\lambda_1 = \frac{\beta_1 I_h + (1-\eta)\beta_2 T_h + \beta_3 I_r}{N_h}$$

$$\lambda_2 = \frac{\beta_4 I_r}{N_r}$$

ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

The state variable and parameters used for the transmission model are described below state variable and parameters used for the transmission model are described in the following tables:

Table 1: Description of State Variables of Our Model

Parameters	Discriptions
S_h(t)	Number of humans susceptible to lassa fever at time t
E_h(t) T	Number of humans exposed to lassa infection at time t
I_h(t)	Number of infectious humans with lassa fever infection at time t
T_h(t)	Number of treated humans at time t
R_h(t)	Number of recovered humans at time t
S_r(t)	Number of rats susceptible to lassa fever at time t
I_r(t)	Number of recovered humans at time t
N_h(t)	Total number of human populations at a time
N_r(t)	Total number of rat populations at a time

Table 2: Description of Our Model Parameters

Parameters	Discriptions
Λ_h	Recruitment rate of susceptible humans
Λ_r	Recruitment rate of susceptible rats
μ_h	Per capita natural mortality rate of humans
μ_r	Per capita natural mortality rate of rats
δ	Per capita disease – induced mortality rate of humans
β_1	Probability that a contact with an infectious human result in transmission of disease to the susceptible human
β_2	Probability that a contact with a treated human result in transmission of disease to the susceptible human
β_3	Probability that a contact with the contaminated environment results in transmission of disease to the susceptible human
β_4	Rodent to rodent transmission
$\in$	DOTS efficacy

ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

$\boldsymbol{\eta}$	Reduction in infectiousness
ρ	Per capita spontaneous recovery rate due to natural immunity
ϕ	Per capita rate of recovery due to DOTS
ψ	Per capita loss of immunity by recovered humans
α	Per capita latent period in humans
θ	Fraction of infected humans who seek treatment under DOTS
τ	Per capita progression rate from the infectious human to the treatment compartment

From the model compartmental model in figure 1, we obtain a seven – dimensional system of ordinary differential equations which describe the progress of the disease as:

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \psi R_h - (\lambda_h + \mu_h)S_h \\
\frac{dE_h}{dt} &= \lambda_1 S_h - (\alpha + \mu_h)E_h \\
\frac{dI_h}{dt} &= \alpha E_h - [\theta\tau + (1-\theta)\rho + \mu_h]I_h \\
\frac{dT_h}{dt} &= \theta\tau I_h - [\epsilon\phi + (1-\phi)\delta + \mu_h]T_h \\
\frac{dR_h}{dt} &= (1-\theta)\rho I_h + \epsilon\phi T_h - (\psi + \mu_h)R_h \\
\frac{dS_r}{dt} &= \Lambda_r - (\lambda_2 + \mu_r)S_r \\
\frac{dI_r}{dt} &= \lambda_2 S_r - \mu_r I_r
\end{aligned}\right\} \quad (1)$$

with the initial conditions:
$S_h(0) = 0, E_h(0) = 0, \quad I_h(0) = 0, \quad T_h(0) = 0 \quad R_h(0) = 0$
$S_r(0) = 0, I_r(0) = 0$

Analysis of The Model
Invariant Region
Lemma1: The model system (1) has solutions which are contained in the feasible region $D = D_h \times D_r$, where;

$$D_h = \left[(S_h, E_h, I_h, T_h, R_h) \in R_+^5 : S_h > 0, E_h \geq 0, I_h \geq 0, T_h \geq 0, R_h \geq 0, N_h \leq \frac{\Lambda_h}{\mu_h}\right] \quad (2)$$

$$D_r = \left[(S_r, I_r) \in R_+^2 : S_r > 0, I_r > 0, N_r \leq \frac{\Lambda_r}{\mu_r}\right] \quad (3)$$

ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

Proof:
Let $D = [S_h, E_h, I_h, T_h, R_h, S_r I_r] \in R_+^7$ be any solution set of the model (3.14) with non-negative initial conditions. From the differential equations (3.14), we have

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - (1-\epsilon)\delta T_h \quad (4)$$

In the absence of the disease ($\delta = 0$)

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h \quad (5)$$

Solving equation (3.16), yieds

$$N_h \leq \frac{\Lambda_h}{\mu_h} + (N_h(0) - \frac{\Lambda_h}{\mu_h})e^{-\mu_h t} \quad (6)$$

Hence at $t \to \infty$, equation (3.17) simplifies to
$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}$.

Thus all the feasible solution of the human population are in the region:

$$D_h = \left[(S_h, E_h, I_h, T_h, R_h) \in R_+^5 : N_h(t) \leq \frac{\Lambda_h}{\mu_h}\right]$$

Similarly;

$$D_r = \left[(S_r, I_r) \in R_+^2 : N_r(t) \leq \frac{\Lambda_r}{\mu_r}\right]$$

Therefore the feasible region for the model system (3.14) is given by;

$$D_h = \left[\begin{array}{c} (S_h, E_h, I_h, T_h, R_h, S_r, I_r) \in R_+^7 : \\ S_h > 0, E_h \geq 0, I_h \geq 0, T_h \geq O, R_h \geq 0, S_r > 0 \\ I_r > 0, N_h \leq \frac{\Lambda_h}{\mu_h}, N_r \leq \frac{\Lambda_r}{\mu_r} \end{array}\right]$$

Hence the feasible region is biologically meaningful and mathematically well-posed in D.

Positivity of Solutions
It is imperative to prove that all solutions of the system (1) with positive initial data will remain positive for all time $t > 0$. This will be verified by the lemma below:
Lemma 2 $If\ [S_h(0), E_h(0), I_h(0), T_h(0), R_h(0), S_r(0), I_r(0)] \epsilon D$, then the solution set $S_h(t), E_h(t), I_h(t), T_h(t), R_h(t), S_r(t), I_r(t)$ of the model solution (1) is positive for all $t > 0$.

Proof;
From the first equation of (3.14), we have;
$\frac{dS_h}{dt} = \Lambda_h + \mu_h R_h - \left(\frac{\beta_1 I_h + (1-\eta)\beta_2 T_h + \beta_3 I_r}{N_h}\right) S_h$
Since $\Lambda_h + \mu_h R_h \geq 0$, then;

ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

$\frac{dS_h}{dt} \geq -\left(\frac{\beta_1 I_h+(1-\eta)\beta_2 T_h+\beta_3 I_r+\mu_h}{N_h}\right)$
(6)

Solving equation (6) at $t = 0$ gives

$S_h(t) \geq S_h(0)e^{-(\int \omega dt+\mu_h t)} \geq 0$ since $\mu_h e^x > 0\ \forall x \in \mathbb{R}$
Where $\omega = \frac{\beta_1 I_h+(1-\eta)\beta_2 T_h+\beta_3 I_r}{N_r}$
Using the approach, from the second equation of (1) we have,

$\frac{dE_h}{dt} = \left(\frac{\beta_1 I_h+(1-\eta)\beta_2 T_h+\beta_3 I_r}{N_h}\right) S_h - (\alpha+\mu_h)E_h.$
Since $\left(\frac{\beta_1 I_h+(1-\eta)\beta_2 T_h+\beta_3 I_r}{N_h}\right) S_h > 0$
$\therefore \frac{dE_h}{dt} \geq -(\alpha+\mu_h)E_h$ (7)

Solving equation (3.19) at t=0

$E_h(t) = E_h(0)e^{-(\alpha+\mu_h)t} \geq 0.$

Similarly, from the third equation of (1), we have,

$\frac{dI_h}{dt} = \alpha E_h - (\theta\tau + (1-\theta)\rho + \mu_h)I_h.$

This implies

$\frac{dI_h}{dt} \geq -(\theta\tau + (1-\theta)\rho + \mu_h)I_h$ (8)

Solving equation (3.21) when t=o gives

$I_h(t) = I_h(0)e^{-(\theta\tau+(1-\theta)\rho+\mu_h)t} > 0.$

Hence it can be shown from the above procedure that the remaining equations of the system (3.14) are positive $\forall t \geq 0$.

Existence of Disease-Free Equilibrium (DFE) points
The disease-free equilibrium points are steady state solutions of the system (1) when the right-hand side is equal to zero. That is:

$$\left.\begin{aligned}
\Lambda_h + \psi R_h - \left(\frac{\beta_1 I_h + (1-\eta)\beta_2 T_h + \beta_3 I_r}{N_h} + \mu_h\right) S_h = 0 \\
\left(\frac{\beta_1 I_h + (1-\eta)\beta_2 T_h + \beta_3 I_r}{N_h}\right) S_h - (\alpha+\mu_h)E_h = 0 \\
\alpha_h E_h - (\theta\tau + (1-\theta)\rho + \mu_h)I_h = 0 \\
\theta\tau I_h - (\epsilon\emptyset + (1-\epsilon)\delta + \mu_h)T_h = 0 \\
(1-\theta)\rho I_h + \epsilon\emptyset T_h - (\psi+\mu_h)R_h = 0 \\
\Lambda_r - \frac{\beta_4 I_r S_r}{N_r} - \mu_r S_r = 0 \\
\frac{\beta_4 I_r S_r}{N_r} - \mu_r + I_r = 0
\end{aligned}\right\} \quad (9)$$

ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

Therefore, the corresponding disease-free equilibrium point is given as:
$E_0 = (S_h^*,\ E_h^*,\ I_h^*,\ T_h^*,\ R_h^*,\ S_r^*,\ I_r^*) = \left(\frac{\Lambda_h}{\mu_h},\ 0,\ 0,\ 0,\ 0,\ \frac{\Lambda_r}{\mu_r},\ 0\right)$

Basic Reproduction Number R_0

The basic reproduction number, denoted by R_0, is a fundamental threshold parameter utilized to analyze the qualitative behavior of epidemiological models. Pioneered by Diekmann et al. (1990), R_0 is defined as the expected number of secondary infections generated by a single typical infectious individual introduced into a completely susceptible population. Structurally, it serves as a critical threshold that dictates whether a pathogen will persist or vanish from a given population (Musa *et al*, 2019). In this study, we employ the next-generation matrix operator framework formalized by van den Driessche and Watmough in 2002 (see also Diekmann and Heesterbeek, 2000) to analytically derive R_0 by isolating the disease-transmitting (infected) compartments of system (1). That is:

$$E_h = \lambda_1 S_h - (\alpha + \mu_h)E_h$$

$$I_h = \alpha E_h - [\theta\tau + (1-\theta)\rho + \mu_h]I_h \qquad (10)$$

$$T_h = \theta\tau I_h - [\epsilon\phi + (1-\theta)\delta + \mu_h]T_h$$

$$I_r = \lambda_2 S_r - \mu_r I_r$$

Let $\boldsymbol{x} = \{E_h^{**},\ I_h^{**},\ T_h^{**},\ I_r^{**}\}^T$, therefore the model (3.21) can be written as;
$\dot{\boldsymbol{x}} = F(x) - V(x)$, where F and V are defined as:

$$F = \begin{pmatrix} \frac{S_h}{N_h}(\beta_1 I_h + (1-\eta)\beta_2 I_h \\ 0 \\ 0 \\ \frac{S_r}{N_r}\beta_4 I_r \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} (\alpha_h + \mu_h)E_h \\ [\theta\tau + (1-\theta)\rho + \mu_h]I_h - \alpha_h E_h \\ [\epsilon\varphi + (1-\theta)\partial + \mu_h]T_h - \theta\tau I_h \\ \mu_R I_r \end{pmatrix}$$

The Jacobian matrix of F and V at the disease – free equilibrium point, E_0 gives:

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_3\Lambda_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta_1 S_h}{\mu_r} & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \alpha + \mu_h & 0 & 0 & 0 \\ -\alpha & \theta\tau(1-\theta)\rho + \mu_h) & 0 & 0 \\ 0 & -\theta\tau & \epsilon\phi + (1-\theta)\delta + \mu_h & 0 \\ 0 & 0 & 0 & \mu_r \end{pmatrix}$$

ISSN:2584-2706 DOI: https://doi.org/10.5281/zenodo.22031054

The inverse of the Jacobian matrix V denoted by V^{-1} gives:

$$\begin{pmatrix} \frac{1}{\alpha+\mu_h} & 0 & 0 & 0 \\ \frac{\alpha}{(\alpha+\mu_h)[\theta\tau+(1-\theta)\rho+\mu_h]} & \frac{1}{\theta\tau+(1-\theta)\rho+\mu_h} & 0 & 0 \\ \frac{\alpha\theta\tau}{(\alpha+\mu_h)[\theta\tau+(1-\theta)\rho+\mu_h][\epsilon\phi+(1-\theta)\delta+\mu_h]} & \frac{\theta\tau}{[\theta\tau+(1-\theta)\rho+\mu_h][\epsilon\phi+(1-\phi)\delta+\mu_h]} & \frac{1}{(\epsilon\theta+(1-\phi)\delta\mu_h+\mu_h)} & 0 \\ 0 & 0 & 0 & \frac{1}{\mu_r} \end{pmatrix}$$

Therefore, the product of the Jacobian matrix F and the inverse of V denoted by FV^{-1} yields;

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & a \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ c & d & 0 & 0 \end{pmatrix}$$

where:

$$a = \frac{\lambda_1\Lambda_h}{\mu_h\mu_r}, \quad c = \frac{\lambda_2\Lambda_r\alpha}{\mu_r(\alpha+\mu_h)[\theta\tau(1-\theta)\rho+\mu_h)]}, \quad d = \frac{\lambda_2\Lambda_r}{\mu_r[\theta\tau+(1-\theta)\rho+\mu_h]}$$

So that the spectral radius (dominant eigenvalues) of the matrix FV^{-1} is calculated from the characteristic equation; $|FV^{-1} - \lambda I| = 0$ where I is a 4×4 identity matrix: That is:

$$\begin{vmatrix} -\lambda & 0 & 0 & a \\ 0 & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & 0 \\ c & d & 0 & -\lambda \end{vmatrix} = 0 \qquad (11)$$

Solving for $\lambda_i, i = 1, 2, 3, 4$, in equation (3.23) and substituting for a, c, d,. we have;

$$\lambda_i = \begin{pmatrix} 0 \\ 0 \\ -\sqrt{\dfrac{\lambda_1\lambda_2\Lambda_h\Lambda_r\alpha}{\mu_h\mu_r^2(\alpha+\mu_h)(\theta\tau+(1-\theta)\rho+\mu_h)}} \\ \sqrt{\dfrac{\lambda_1\lambda_2\Lambda_h\Lambda_r\alpha}{\mu_h\mu_r^2(\alpha+\mu_h)(\theta\tau+(1-\theta)\rho+\mu_h)}} \end{pmatrix}$$

But $R_0 = \rho(FV^{-1})$,

Hence,

$$R_0 = \sqrt{\frac{\lambda_1\lambda_2\Lambda_h\Lambda_r\alpha}{\mu_h\mu_r^2(\alpha+\mu_h)(\theta\tau+(1-\theta)\rho+\mu_h)}}$$

(3.24)

3.2.5 Local Stability Analysis of the Disease – free Equilibrium Point

Theorem 3.3.1: The disease – free equilibrium point for system (3.14) is locally asymptotically stable if $R_0 < 1$

Proof: The Jacobian matrix of the system (1) evaluated at the disease – free equilibrium point E_0 denoted by $J_0[E_0]$ is given as:

$$\begin{pmatrix} -\mu_h & 0 & -\beta_1 & -(1-\eta)\beta_2 & \psi & 0 & -\beta_3 \\ 0 & -(\alpha_h+\mu_h) & \beta_1 & (1-\eta)\beta_2 & 0 & 0 & \beta_3 \\ 0 & \alpha & -(\Theta\tau+(1-\theta)\rho+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta\tau & -(\epsilon\psi+(1-\phi)+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & (1-\theta)\rho & \epsilon\varphi & -(\psi+\mu_h) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_r & -\beta_4 \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_4-\mu_r \end{pmatrix}$$

We need to show that all the eigenvalues of $J_0[E_0]$ have negative real parts. The sixth column contains only the diagonal term, $-\mu_r$, which form the first eigenvalue. Therefore, $J_1[E_0]$ yields;

$$\begin{pmatrix} -\mu_h & 0 & -\beta_1 & -(1-\eta)\beta_2 & \psi & -\beta_3 \\ 0 & -(\alpha_h+\mu_h) & \beta_1 & (1-\eta)\beta_2 & 0 & \beta_3 \\ 0 & \alpha & -(\Theta\tau+(1-\theta)\rho+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & \theta\tau & -(\epsilon\psi+(1-\phi)+\mu_h) & 0 & 0 \\ 0 & 0 & (1-\theta)\rho & \epsilon\varphi & -(\psi+\mu_h) & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_4-\mu_r \end{pmatrix}$$

Similarly, the first column of $J_1[E_0]$ contains only the diagonal term, $-\mu_h$, which forms a negative eigenvalue. Hence;

$$J_2[E_0]$$

$$= \begin{pmatrix} -(\alpha_h + \mu_h) & \beta_1 & (1-\eta)\beta_2 & 0 & \beta_3 \\ \alpha & -(\Theta\tau + (1-\theta)\rho + \mu_h) & 0 & 0 & 0 \\ 0 & \theta\tau & -(\epsilon\psi + (1-\phi) + \mu_h) & 0 & 0 \\ 0 & (1-\theta)\rho & \epsilon\varphi & -(\psi + \mu_h) & 0 \\ 0 & 0 & 0 & 0 & \beta_4 - \mu_r \end{pmatrix}$$

In the same way, the fourth column of $J_2[E_0]$ contains only the diagonal term, $-(\psi + \mu_h)$, which represents a negative eigenvalue. Therefore

$$J_3[E_0] = \begin{pmatrix} -(\alpha_h + \mu_h) & \beta_1 & (1-\eta)\beta_2 & \beta_3 \\ \alpha & -(\Theta\tau + (1-\theta)\rho + \mu_h) & 0 & 0 \\ 0 & \theta\tau & -(\epsilon\psi + (1-\phi) + \mu_h) & 0 \\ 0 & 0 & 0 & \beta_4 - \mu_r \end{pmatrix}$$

We need to show that all the eigenvalues of $J_0[E_0]$ have negative real parts. The sixth column contains only the diagonal term, $-\mu_h$, which form the first eigenvalue. Therefore, $J_1[E_0]$ yields;

$$J_1 = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & \varepsilon_h & 0 \\ 0 & -(\delta_h + \phi_h) & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & -(\tau_h + \mu_h + \delta_h) & 0 & 0 & 0 \\ 0 & 0 & \delta_h & -(1+\alpha_h) & 0 & 0 \\ 0 & 0 & 0 & \tau_h & -(\mu_h + \varepsilon_h) & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_r \end{bmatrix}$$

Similarly, the first column of $J_1[E_0]$ contains only the diagonal term, $-\mu_h$, which forms a negative eigenvalue. Hence;

$$J_2 = \begin{bmatrix} -(\delta_h + \phi_h) & 0 & 0 & 0 & 0 \\ \sigma_h & -(\tau_h + \mu_h + \delta_h) & 0 & 0 & 0 \\ 0 & \delta_h & -(1+\alpha_h) & 0 & 0 \\ 0 & 0 & \tau_h & -(\mu_h + \varepsilon_h) & 0 \\ 0 & 0 & 0 & 0 & -\mu_r \end{bmatrix}$$

In the same way, the fourth column of J_2 contains only the diagonal elements $-(\mu_h + \varepsilon_h)$, which represents a negative eigenvalue. Therefore

$$J_3 = \begin{bmatrix} -(\delta_h + \phi_h) & 0 & 0 & 0 \\ \sigma_h & -(\tau_h + \mu_h + \delta_h) & 0 & 0 \\ 0 & \delta_h & -(1+\alpha_h) & 0 \\ 0 & 0 & 0 & -\mu_r \end{bmatrix}$$

Similarly, the second column of J_3 contains only the diagonal element $-(\tau_h + \mu_h + \delta_h)$, which represents a negative eigenvalue. Therefore

$$J_4 = \begin{bmatrix} -(\delta_h + \phi_h) & 0 & 0 \\ 0 & -(1+\alpha_h) & 0 \\ 0 & 0 & -\mu_r \end{bmatrix}$$

The remaining eigenvalues are obtained from the sub – matrix J_4. Obviously, the eigenvalues of the matrix $J_4[E_0]$ are the roots of the characteristic equation:

$$|J_4 - \lambda \mathrm{I}| = 0$$

That is;

$$\left(\begin{bmatrix} -(\delta_h + \phi_h) & 0 & 0 \\ 0 & -(1+\alpha_h) & 0 \\ 0 & 0 & -\mu_r \end{bmatrix} - \begin{pmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{pmatrix} \right) = 0$$

Let $D_1 = (\delta_h + \emptyset_h)$, $D_2 = -(1+\alpha_h)$ and $\mathrm{D}_3 = \mu_r$,

$$\left(\begin{bmatrix} D_1 & 0 & 0 \\ 0 & D_2 & 0 \\ 0 & 0 & D_3 \end{bmatrix} - \begin{pmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{pmatrix} \right) = 0$$

Implies

$$\begin{bmatrix} D_1 - \lambda & 0 & 0 \\ 0 & D_2 - \lambda & 0 \\ 0 & 0 & D_3 - \lambda \end{bmatrix} = 0$$

$$(D_1-\lambda)\left[(D_2-\lambda)(D_3-\lambda)-0\right]=0$$
$$(D_1-\lambda)\left[D_2D_3-\lambda D_2-\lambda D_3+\lambda^2\right]=0$$
$$(D_1-\lambda)(\lambda^2-\lambda D_2-\lambda D_3+\lambda^2)=0$$
$$D_1\lambda^2-\lambda D_1D_2-\lambda D_1D_3+D_1D_2D_3-\lambda^3+\lambda^2D_2+\lambda^2D_3-\lambda D_2D_3=0$$
$$-\lambda(D_1D_2+D_1D_3+D_2D_3)+\lambda^2(D_1+D_2+D_3)-\lambda^3=0$$
$$-\lambda^3+\lambda^2(D_1+D_2+D_3)-\lambda(D_1D_2+D_1D_3+D_2D_3)=0$$

Let $A_1=-1$
$A_2=(D_1+D_2+D_3)$
$A_3=-(D_1D_2+D_1D_3+D_2D_3)$
$A_3\lambda^3+A_2\lambda^2+A_1\lambda=0$

To prove that all roots of equation (3.14) have negative real parts, we employ the Routh – Hourwitz criterion. The Routh-Hurwitz criteriom according Ojo *et al* (2021) for a real algebraic equation as show below
$a_nx^n+a_{n-1}x^{n-1}+\cdots+a_1x+a_0=0$
states that, given $a_n>0,$ all roots of (3.25) have negative real part if and only if

$$H_0=a_n$$

$$H_1=a_{n-1}$$

$$H_2=\begin{vmatrix}a_{n-1} & a_n\\ a_{n-3} & a_{n-2}\end{vmatrix}$$

$$H_3=\begin{vmatrix}a_{n-1} & a_0 & 0\\ a_{n-3} & a_{n-2} & a_{n-1}\\ a_{n-5} & a_{n-4} & a_{n-3}\end{vmatrix}$$

$$\vdots$$

$$H_n=\begin{vmatrix}a_{n-1} & \dots & 0\\ \vdots & \ddots & \vdots\\ 0 & \dots & a_0\end{vmatrix}$$

are all positive, with $a_j=0$ for $j<1$. This is true if and only if either all even-numbered H_i or all odd-numbered H_i are positive (liénard–chipart criterion).
Hence, using the Routh – Hurwitz criterion, we show that all the roots of polynomial (3.25) have negative real parts if and only if the coefficients $A_i,\ i=0,1,2,3\,4$ are positive and matrices $H_i>0,$ for $i=0,1,2,3,4,5.$
In terms of R_0, further simplification of A_1 yields;

$A_1=-(D_1D_2+D_1D_3+D_2D_3)[1-R_0^2]$
The expression for R_0^2 in terms of A_{irs} can be written as;

$R_0^2 = \frac{(D_1D_2+D_1D_3+D_2D_3)}{(D_1D_2+D_1D_3+D_2D_3)}$
$R_0^2 = 0$
$R_0^2 < 0$

From the foregoing, we have been able to show that all the eigenvalues of the Jacobian matrix are all negative real parts. We also show that $R_h, R_r \in R_0 < 1$.
It must be noted that $-(\mu_r - \beta_r)$ can also be re-written as

$$-\mu_r(1-\frac{\beta_r}{\mu_r}) = -\mu_r(1-R_r)$$

where $R_r = \frac{\beta_r}{\mu_r}$ .and $R_r \in R_0 < 1$

Also, $-(\mu_h - \beta_h) = -\mu_h(1-\frac{\beta_h}{\mu_h}) = -\mu_h(1-R_h)$,

where $R_h = \frac{\beta_h}{\mu_h}$ .and $R_h \in R_0 < 1$

Thus, all the eigenvalues of the Jacobian matrix in (3.25) are all negative real parts, that is $(R_h, R_r) \in R_0 < 1$. This shows that the lassa fever free equilibrium is locally asymptotically stable.
From an epidemiological perspective going from the analysis above, it implies that the spread of lassa fever can be effectively controlled in the population when $R_0 < 1$, However, if $R_0 > 1$ and by Descartes' rule of signs (Musa, 2019), we observe that there is a positive eigenvalue which indicates that the disease – free equilibrium point will be unstable.

Conclusion

In this study, we developed, analysed a deterministic model to describe the transmission dynamics of Lassa fever in Nigeria. Transmission of Lassa fever requires interaction between two-interacting hosts (namely human and rodent population), thus we sub-divided the human population into susceptible, exposed, infectious, treated and recovered humans, while the rodent population was subdivided into a susceptible and infectious rodents. The local and global stability of the model was investigated using the reproduction number which was obtained by using the Jacobian matrix. The result shows that the Lassa fever-free equilibrium R_0 is locally and globally asymptotically stable if $R_0 < 1$ and unstable otherwise.
From an epidemiological perspective going from the analysis above, it implies that the spread of Lassa fever can be effectively controlled in the population when $R_0 < 1$.

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