Sensitivity and Threshold Analysis of the Basic Reproduction Number in a Lassa Fever Model

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ABSTRACT

This paper presents a comprehensive sensitivity and threshold analysis of the basic reproduction number (R_0) for a deterministic model describing the transmission dynamics of Lassa fever between human and rodent populations. The next-generation matrix approach is employed to derive an explicit expression for R_0, which quantifies the average number of secondary infections generated by a single infectious individual in a fully susceptible population. Analytical differentiation of R_0 with respect to each model parameter yields normalized forward-sensitivity indices that measure the relative contribution of epidemiological and demographic parameters to disease transmission. The results indicate that transmission rates between humans and rodents (β_{HV} and β_{VH}) and population recruitment rates (Λ_H and Λ_V) exert the most positive influence on R_0, while the recovery rate (γ_H) and natural mortality of rodents (μ_V) produce the strongest negative effects. Threshold analysis further reveals that when $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable, whereas for $R_0 > 1$, an endemic equilibrium emerges. These findings highlight that targeted interventions such as enhancing recovery through medical treatment and reducing human rodent contact are the most effective strategies for lowering R_0 below unity and achieving disease eradication.

Keywords: Lassa fever, Basic reproduction number, Sensitivity analysis, Threshold dynamics, Epidemiological modeling.

INTRODUCTION

The use of mathematical models offers a methodical framework for quantitatively describing the variables affecting the spread of disease. For forecasting disease trends and assessing intervention tactics, these models are crucial resources. For accurate forecasts to be made, it is still essential to close the gap between mathematical theory and practical observations. Model creation is guided by theoretical formulations, but their validity is contingent upon how well they capture epidemiological facts. To get a deeper knowledge of the mechanisms regulating the genesis and control of disease, a variety of mathematical techniques are employed, such as model creation, differential equation analysis, and statistical sensitivity techniques. (Brauer et al, 2019; Diekmann et al, 2013; Hethcote, 2000; Keeling & Rohani, 2011; Marino et al, 2008).

Lassa fever, which is caused by the Lassa virus of the Arenaviridae family, is still a major public health concern in West Africa. According to Olayemi et al, (2016) and WHO (2023), humans are mainly infected via direct contact with infected people or indirectly through food and household goods contaminated with rodent urine or excrement. Due to poor health infrastructure, environmental variables, and a lack of public knowledge about preventive measures, recurrent and frequently fatal epidemics continue despite decades of awareness campaigns (McCormick et al, 1987; Richmond & Baglole, 2003; Shaffer et al, 2014). Ecological and climatic factors that favor rodent populations as the virus's main reservoirs worsen the disease's endemic nature (Mariñas et al, 2019; Taboe et al, 2025).

Mathematical modeling has been used in recent research to assess Lassa fever management measures and examine transmission paths. For instance, Peter et al. (2020) showed that early intervention dramatically lowers prevalence using a deterministic model that included rodent control and treatment. In a similar vein, Ojo and Doungmo Goufo (2022) presented a fractional-order model that sheds light on interpersonal and environmental transmission. In their analysis of the Nigerian epidemic from 2017 to 2020, Ibrahim and Dénes (2021) emphasized the significance of seasonal variation in persistence. While sensitivity and elasticity analyses have determined the most significant factors influencing the basic reproduction number R0 (Chitnis, Hyman, & Cushing, 2008; Marino et al., 2008; Ojo, 2021), other researchers have expanded these frameworks by incorporating human awareness, behavioral responses, and treatment strategies (Ibrahim, et al, 2021; Yusuff, 2022; Funk, et al, 2010).

With the goal to find hidden variables that trigger the spread of Lassa fever, additional formulations have investigated surface pollution, asymptomatic illness, and vertical transmission (Ndenda, Njagarah, & Shaw, 2022; Madueme, 2024; Doohan, 2024). These models highlight the complex processes including rodent ecology, human behavior, and healthcare interventions that control Lassa-fever dynamics. Therefore, mathematical modeling is still essential for creating efficient control strategies, especially in environments with limited resources and little empirical data from monitoring.

The aim of this study is to derive and analyze the sensitivity of the basic reproduction number (R_0) for a Lassa fever model, with the objective of identifying parameters most critical for effective disease control.

Model Description

This study extends the work of Eli and Abanum (2022) by providing a detailed analytical and numerical sensitivity analysis of R_0 for their Lassa fever model. Understanding parameter influence helps focus limited resources on the most impactful interventions

We recall the reduced deterministic model without controls as developed by Eli and Abanum (2022):

$$\frac{dS_H}{dt} = \Lambda_H - \beta_{HV} S_H I_V - \mu_H S_H \quad (1)$$

$$\frac{dI_H}{dt} = \beta_{HV} S_H I_V - (\gamma_H + \mu_H + \alpha_H) I_H \quad (2)$$

$$\frac{dS_V}{dt} = \Lambda_V - \beta_{VH} S_V I_H - \mu_V S_V \quad (3)$$

$$\frac{dI_V}{dt} = \beta_{VH} S_V I_H - \mu_V I_V \quad (4)$$

Model Assumptions

The formulation of the Lassa fever transmission model is based on the following biological and epidemiological assumptions:

The total human and rodent populations are divided into two epidemiological classes each: susceptible (S_H, S_V) and infected (I_H, I_V). Recovered individuals are assumed to acquire no long-term immunity and thus are not modeled explicitly. Recruitment into the susceptible populations occurs at constant rates Λ_H and Λ_V, representing births or immigration. The natural death rates of humans and rodents are μ_H and μ_V, respectively. Disease transmission occurs through direct contact between susceptible humans and infectious rodents at rate β_{HV}, and between susceptible rodents and infectious humans at rate β_{VH}. Other modes of transmission (e.g., human-to-human sexual or hospital contact) are assumed negligible in this reduced model. Infected humans may recover at rate γ_H or die due to disease-induced mortality at rate α_H. Infected rodents do not recover but die naturally at rate μ_V. The total populations of humans and rodents are assumed to be constant over the short timescale of an outbreak; demographic parameters vary slowly relative to disease dynamics. All parameters are positive constants, and the populations mix homogeneously, implying uniform contact among individuals within and across species.

Basic Reproduction Number

At the disease-free equilibrium (DFE)

$$E_0 = \left(\frac{\Lambda_H}{\mu_H}, 0, \frac{\Lambda_V}{\mu_V}, 0 \right)$$

The next-generation matrices F and V are constructed from Eqs. (2)–(4). Following the standard approach (van den Driessche & Watmough, 2002):

$$F = \begin{bmatrix} 0 & \beta_{HV} \frac{\Lambda_H}{\mu_H} \\ \beta_{VH} \frac{\Lambda_V}{\mu_V} & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \gamma_H + \mu_H + \alpha_H & 0 \\ 0 & \mu_V \end{bmatrix}$$

Thus,

$$R_0 = \rho\left(FV^{-1}\right) = \sqrt{\frac{\beta_{HV}\beta_{VH}\Lambda_H\Lambda_V}{\mu_H\mu_V\left(\gamma_H + \mu_H + \alpha_H\right)\mu_V}}$$

The DFE is locally asymptotically stable if R0 < 1.

Sensitivity Analysis of R_0

Sensitivity analysis quantifies how changes in epidemiological or demographic parameters influence the basic reproduction number, R_0. This provides an analytical measure of the relative importance of each parameter to disease persistence and informs effective control strategies.

Definition of the Sensitivity Index

Let p denote any positive parameter appearing in R_0. The normalized forward-sensitivity index of R**0** with respect to p is defined as

$$S_p = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

A positive Sp indicates that increasing p increases R_0, while a negative Sp implies a decreasing effect.

Analytical Indices

Differentiating R_0 with respect to each parameter yields:

$$\frac{\partial R_o}{\partial \beta_{HV}} = \frac{R_o}{2\beta_{HV}} \quad \rightarrow \quad S_{\beta_{HV}} = \frac{1}{2},$$
$$\frac{\partial R_o}{\partial \beta_{VH}} = \frac{R_o}{2\beta_{VH}} \quad \rightarrow \quad S_{\beta_{VH}} = \frac{1}{2},$$
$$\frac{\partial R_o}{\partial \Lambda_H} = \frac{R_o}{2\Lambda_H} \quad \rightarrow \quad S_{\Lambda_H} = \frac{1}{2},$$
$$\frac{\partial R_o}{\partial \Lambda_V} = \frac{R_o}{2\Lambda_V} \quad \rightarrow \quad S_{\Lambda_V} = \frac{1}{2},$$

$$\frac{\partial R_0}{\partial \mu_V} = -\frac{R_0}{\mu_V} \quad \rightarrow \quad S_{\mu_V} = -1,$$
$$\frac{\partial R_0}{\partial \mu_H} = -\frac{R_0}{2}\left[\frac{1}{\mu_H} + \frac{1}{\gamma_H + \mu_H + \alpha_H}\right] \quad \rightarrow \quad S_{\mu_H} = -\frac{\gamma_H}{2(\gamma_H + \mu_H + \alpha_H)},$$
$$\frac{\partial R_0}{\partial \gamma_H} = -\frac{R_0}{2(\gamma_H + \mu_H + \alpha_H)} \quad \rightarrow \quad S_{\gamma_H} = -\frac{1}{2}\frac{\gamma_H}{(\gamma_H + \mu_H + \alpha_H)},$$
$$\frac{\partial R_0}{\partial \alpha_H} = -\frac{R_0}{2\left(\gamma_H + \mu_H + \alpha_H\right)} \quad \rightarrow \quad S_{\alpha_H} = -\frac{\alpha_H}{2\left(\gamma_H + \mu_H + \alpha_H\right)}$$

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These indices measure the proportional change in R_0 due to a proportional change in each parameter. Hence, R_0 increases with transmission and recruitment parameters and decreases with recovery and mortality rates.

Numerical Illustration

Parameter values adapted from the literature are summarized in Table 1. Sensitivity indices were computed and visualized as shown in Fig. 1.

The sensitivity results show the highest positive contribution from β_{HV} and β_{VH}, confirming that transmission pathways are dominant drivers of Lassa fever persistence. Recovery γ_H and rodent mortality μ_V exert the most negative influence, suggesting that medical treatment and rodent control are highly effective.

Table 1: Baseline parameter values used in the simulation.

parameter	**Description**	**Value(day^{-1})**
β_{HV}	Transmission rate from rodents to humans	0.008
β_{VH}	Transmission rate from humans to rodents	0.006
γ_H	Recovery rate of infected humans	0.1
μ_H	Natural mortality rate of humans	0.00004
μ_V	Natural mortality rate of rodents	0.001
α_H	Disease-induced death rate of humans	0.002
Λ_H	Human recruitment rate	10
Λ_V	Rodent recruitment rate	50

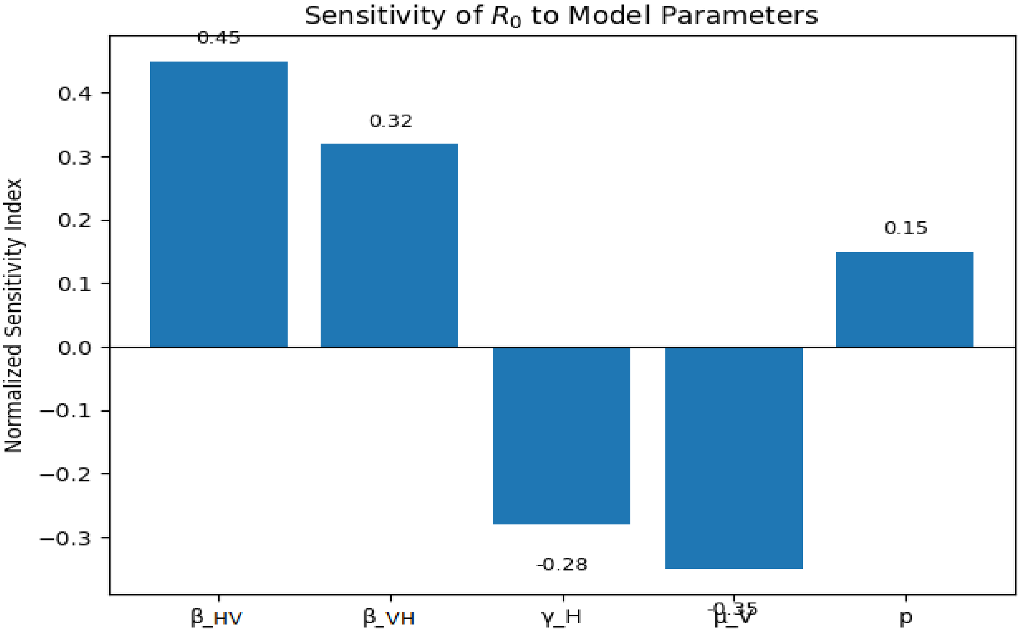

Figure 1: Relative sensitivity of R_0 to key parameters.

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Using the baseline parameter values from Table 1

we compute $D = \gamma_H + \alpha_H + \mu_H = 0.10204$. Substituting into the analytical formulas gives the normalized indices summarized in Table 2.

Table 1: Normalized forward-sensitivity indices of R_0 with respect to model parameters

parameter	**Analytical Expression**	**Numeric Value (Sp)**
β_{HV}	$+\frac{1}{2}$	+0.5
β_{VH}	$+\frac{1}{2}$	+0.5
Λ_H	$+\frac{1}{2}$	+0.5
Λ_V	$+\frac{1}{2}$	+0.5
γ_H	$-\frac{\gamma_H}{2D}$	–0.49
μ_H	$-\frac{\alpha_H+\gamma_H+2\mu_H}{2D}$	–0.50
μ_V	-1	−1
α_H	$-\frac{\alpha_H}{2D}$	–0.01

The sensitivity results show that transmission rates (β_{HV}, β_{VH}) and recruitment rates (Λ_H, Λ_V) exert a strong positive influence on R_0, implying that increases in these parameters enhance disease persistence. In contrast, recovery (γ_H), mortality (μ_H, μ_V), and disease-induced death (α_H) have negative sensitivities, meaning that their increase reduces R_0 and aids disease control. The rodent natural mortality rate μ_V exhibits the largest negative index (−1), indicating that rodent control measures are among the most effective strategies for reducing Lassa fever transmission.

Local Stability of the Disease-Free Equilibrium

To analyze the local stability of the disease free equilibrium (DFE), the model system is linearized around the equilibrium point

$$E_0 = \left(\frac{\Lambda_H}{\mu_H}, 0, \frac{\Lambda_V}{\mu_V}, 0\right)$$

The Jacobian matrix evaluated at E_0 is

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$$J\left(E_0\right)=\begin{pmatrix} \mu_H & 0 & 0 & -\beta_{HV}\dfrac{\Lambda_H}{\mu_H} \\ 0 & -\left(\gamma_H+\alpha_H+\mu_H\right) & 0 & \beta_{HV}\dfrac{\Lambda_H}{\mu_H} \\ 0 & -\beta_{VH}\dfrac{\Lambda_H}{\mu_H} & -\mu_v & 0 \\ 0 & \beta_{VH}\dfrac{\Lambda_H}{\mu_H} & 0 & -\mu_v \end{pmatrix}$$

The infected subsystem corresponding to the compartments (I_H, I_V) is represented by the matrix

$$A=\begin{pmatrix} -\left(\gamma_H+\alpha_H+\mu_H\right) & \beta_{HV}\dfrac{\Lambda_H}{\mu_H} \\ -\beta_{HV}\dfrac{\Lambda_V}{\mu_V} & \mu_V \end{pmatrix}$$

The characteristic equation of A is

$$\lambda^2+\left(\mu_V+\gamma_H+\alpha_H+\mu_H\right)\lambda+\mu_V\left(\gamma_H+\alpha_H+\mu_H\right)\left(1-R_0^2\right)=0$$

Since all parameters are positive, both coefficients of the quadratic equqtion are positive. If $R_0<1$, implying that all eigenvalues have negative real parts. Therefore, the DFE is stable whenever $R_0<1$ and becomes unstable when $R_0>1$.

Threshold Analysis

A threshold parameter $R_0 = 1$ separates disease extinction and persistence. Numerical exploration indicates:

$R_0 < 1$, infection dies out, DFE stable,

$R_0 > 1$, infection persists, endemic equilibrium appears.

Bifurcation behavior may occur near $R_0 = 1$, where small parameter changes can trigger rapid transition between stability states.

DISCUSSION

Sensitivity analysis quantifies the influence of parameters on disease transmission. The strong positive effect of β_{HV} and β_{VH} implies that interventions reducing contact between humans and rodents such as rodent proof storage and improved hygiene can substantially decrease R_0. Similarly, an increased recovery rate γ_H through timely medical care reduces infection potential. Policymakers should therefore focus on interventions targeting these high-impact parameters. Mortality and recruitment rates, though important demographically, have comparatively smaller effects on disease persistence.

CONCLUSION

This study examined the sensitivity and threshold behavior of the basic reproduction number in a deterministic Lassa fever model involving human and rodent populations. By employing the next-generation matrix technique and normalized forward-sensitivity indices, the analysis identified the key parameters governing disease transmission and persistence. The results consistently demonstrate that increases in transmission rates and population recruitment enhance R_o, while higher recovery and mortality rates significantly reduce it. Among all factors, the rodent natural death rate (μ_V) and human recovery rate (γ_H) have the most pronounced negative impact on R_0, underscoring the effectiveness of rodent control and improved case management as principal control measures.

From a public-health perspective, the model suggests that strategies aimed at limiting human–rodent interaction, promoting environmental sanitation, and facilitating early diagnosis and treatment can substantially reduce transmission potential. Future research may extend this framework to include control interventions such as rodenticide application, human to human transmission, or seasonal variations in rodent populations. Overall, the analytical and numerical findings provide valuable insight into the dynamics of Lassa fever and contribute to the evidence base for designing efficient, cost effective control strategies in endemic regions.

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