Mathematical Modelling of Spread of Lassa fever in Nigeria: A Theoretical Framework

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Abstract.
Lassa fever remains a persistent public health challenge in Nigeria, where recurrent outbreaks highlight the urgent need for effective predictive and control strategies. In this study, we present a review of the theoretical framework for a 4-compartment mathematical model that investigates the dynamics of Lassa fever transmission within human and rodent populations in Nigeria. The model explains key epidemiological factors including human-to-human transmission, rodent-to-human contact, and seasonal variations in rodent population dynamics. This paper also discussed how to estimate model parameters and compute the basic reproduction number, R_0, as an indicator of disease persistence. Stability analysis of the disease-free and endemic equilibria is performed. Other important aspects reviewed were concepts, interpretations and implications of sensitivity analysis with respect to epidemiological and policy insights, public health implications and probable research recommendations. This work contributes to the growing body of knowledge on Lassa fever modelling by emphasizing context-specific parameters and intervention strategies suitable for Nigeria. The study provides a mathematical framework to guide researchers in explaining the course of Lassa fever spread, and policymakers in planning sustainable control measures against Lassa fever and other rodent-borne diseases in West Africa.

Keywords: Modelling, Lassa Fever, Nigeria, Theoretical Framework

1. Introduction

The synthesis of traditional epidemiology with mathematical and computational modelling has been boosted as reflected in many Nigerian scientific contributions. From statistical theory and probability, to applied statistics, models have become invaluable in in explaining associated natural phenomena so as to optimize human efforts. For instance, in epidemiological studies, a numerical collocation model[1] was used to analyse an SEIR compartmental disease transmission model, while a lifetime probability distribution with established survival and hazard rate functions[2] was used to estimate recovery and mortality rates arising from the Covid-19 pandemic in Nigeria. In another study, a time series model was used to analyse infant mortality rates [3] in some regions of Nigeria. All these are instances of use of mathematical models in explaining population related issues, and this supports the assertion that in any

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attempt to explain the dynamics of an infectious disease, population distribution of the host community was an important denominator that should not be overlooked [4]. Classical mathematical models have been adapted and used extensively to analyse and predict the course and effects of intervention programmes on the dynamics of several infectious diseases. A model to study the dynamics of COVID-19 in Ghana and to consider the impact of testing and quarantine of immigrants, contact tracing and isolation as measures in the mitigation of the spread of the disease was developed [5]. Also developed was a mathematical Model for the dynamics and control of Malaria in Nigeria [6]. Another study [7] adapted a compartmentalized epidemiological model to explain the dynamical relationship between cybercrime, poverty and prostitution, while [8] investigated the relationship between model assumption violation and multi-collinearity, and used illustrations to show the role of variance inflation factor (VIF) in detecting model violations.

Lassa fever remains one of the most persistent and life-threatening zoonotic diseases in West Africa, particularly in Nigeria, where the disease has become endemic [9,10,11]. The Lassa virus, a member of the *Arenaviridae* family, was first identified in 1969 in the town of Lassa, Borno State, Nigeria, following an outbreak among missionaries and local residents [12]. Since then, Lassa fever has evolved from an obscure regional illness into a globally recognized public health threat, with countries such as Nigeria, Sierra Leone, Liberia, and Guinea reporting recurrent outbreaks and Nigeria bearing the highest burden [13]. While the disease remains relatively unknown outside endemic regions, sporadic cases among travelers have highlighted its potential for international spread, underscoring its global relevance [14]. Lassa fever remains a viral hemorrhagic disease of major concern in West Africa, characterized by high morbidity and mortality, particularly among hospitalized women and children [15]. Recent reports have shown recurrent outbreaks in Nigeria between 2017 and 2019 [16]. The disease is transmitted to humans through exposure to food or household items contaminated with the urine or faeces of infected *Mastomys natalensis* rodents; the primary reservoir hosts [17,18]. The role of *Mastomys natalensis* as a reservoir has been well documented by [19]. Secondary transmission occurs through direct contact with the blood, tissues, secretions, or excretions of infected individuals, posing serious risks to healthcare workers and society [12,20]. Despite extensive surveillance efforts, recent World Health Organization (WHO) situation reports indicate a continuous rise in confirmed Lassa fever cases in Nigeria, with fluctuating case-fatality ratios influenced by delayed diagnosis, weak infection control practices, and increasing urban encroachment into rodent habitats [21,22]. The recognition of Lassa fever originally emerged during investigations into unexplained hemorrhagic illnesses among hospital staff in northeastern Nigeria, leading to the identification of *Mastomys natalensis* as the natural reservoir species [23].

The epidemiology of Lassa fever demonstrates both endemic persistence and seasonal outbreaks, typically peaking during the dry season (December to April) when human–rodent interactions increase due to environmental changes [24]. Between 2016 and 2020, Nigeria recorded an increasing trend in confirmed cases, highlighting the disease's expanding public health burden [25]. Estimates suggest between 100,000 and 300,000 infections annually in West Africa, resulting in approximately 5,000 deaths, though underreporting likely conceals the true magnitude [13]. Beyond mortality, Lassa fever imposes significant socioeconomic costs, disrupting livelihoods, straining health systems, and inflating the costs of outbreak containment and patient management.

Clinically, Lassa fever presents a wide spectrum of manifestations, ranging from asymptomatic infections to severe hemorrhagic disease. Early symptoms include fever, malaise, and gastrointestinal disturbances such as nausea and abdominal pain, while severe cases may progress to hemorrhage, respiratory distress, neurological impairment, and multi-organ failure [14]. Approximately 80% of infections are mild or asymptomatic, which complicates early detection and containment [26].

Over the years, numerous mathematical models have been developed to understand the transmission dynamics and control mechanisms of Lassa fever and other communicable diseases. Recent studies have

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integrated nonlinear incidence rates, environmental reservoirs, and optimal control frameworks to improve predictive accuracy [27,28,29]. In a systematic review of Lassa fever models, [30] emphasized the need for greater integration of parameter uncertainty, ecological factors, and empirical epidemiological data.

Mathematical and computational approaches continue to play a pivotal role in advancing the understanding of Lassa fever transmission. Ecological niche models have been employed to map *Mastomys natalensis* distributions and identify high-risk spillover zones [23]. According to [31], compartmental models, such as SEIR frameworks, have been instrumental in exploring the impacts of rodent control, improved sanitation, and early case detection on epidemic dynamics.

Nevertheless, many existing models are constrained by inadequate surveillance data, underreporting, and high variability in rodent population dynamics, particularly in rural communities. Intervention strategies have largely remained reactive, focusing on outbreak response rather than sustained prevention and risk reduction [13]. Modern modeling studies increasingly demonstrate that predictive and spatial approaches, when grounded in real-time epidemiological data, can significantly improve epidemic preparedness.

Nigeria's public health response has evolved considerably in recent years, with the establishment of the Nigeria Centre for Disease Control (NCDC) as a coordinating agency for disease surveillance and outbreak management. The development of national case management guidelines and the distribution of ribavirin therapy at designated treatment centers have strengthened clinical responses [25]. Public health education campaigns have also sought to reduce rodent contact and promote hygiene and early healthcare seeking. However, persistent challenges such as poor infrastructure, limited diagnostic capacity, sociocultural barriers, and inadequate funding continue to impede timely detection and management [20]. Given these challenges, there is a pressing need for research that integrates improved predictive models, ecological surveillance, and context-specific interventions. Many existing studies overlook local transmission nuances, including urban–rural differences, seasonal variations, and socio-behavioral influences. Although rodent control and environmental hygiene remain cornerstones of prevention, few empirical studies have comprehensively evaluated their effectiveness within endemic Nigerian communities.

In response to these gaps, the present study developed a theoretical deterministic compartmental model for Lassa fever transmission, incorporating both human and rodent populations. It seeks to derive and explain the basic reproduction number (R_0), list sensitivity analyses procedure to identify dominant parameters, and identify some direct implications to research and society. The broader objective of any mathematical modelling task is to synthesize historical, epidemiological, and modeling insights to guide policy formulation, enhance outbreak preparedness, and strengthen national health security.

2. Model formation

A mechanistic compartmental model for Lassa fever tailored to the Nigerian context is developed that extends the classical SEIR framework to include the rodent reservoir. The formulation balances epidemiological realism with tractability for parameter estimation and interpretation. Lassa fever transmission is multi-pathway: direct zoonotic spillover from infected rodents to humans, indirect environmental exposure (rodent excreta contaminating surfaces or food), and limited human-to-human transmission (notably in healthcare settings). A deterministic system of ordinary differential equations (ODEs) is appropriate for capturing population-level dynamics, seasonality, and for enabling stability and sensitivity analyses.

2.1 Model compartments and variables

To describe the transmission dynamics of Lassa fever, the entire population is divided into epidemiologically relevant compartments representing both humans and rodents, as well as an environmental viral reservoir. Each compartment represents a distinct health or infection state, and transitions between them capture the progression of infection and recovery processes.

The human population is stratified into four compartments as follows:

- $S_h(t)$: Susceptible humans or individuals who are healthy and can acquire infection through

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contact with infected rodents or contaminated environments.

- $E_h(t)$: Exposed humans or individuals who have been infected but are not yet infectious; they are in the incubation period.
- $I_h(t)$: Infectious humans or individuals who can transmit the Lassa virus to others or shed the virus into the environment.
- $R_h(t)$: Recovered humans or individuals who have recovered from infection and acquired temporary or long-term immunity.

Thus, the total human population at time t is given by: $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$

Rodents, primarily *Mastomys natalensis*, serve as the natural reservoirs of Lassa virus. The rodent population is divided into two compartments:

- $S_r(t)$: Susceptible rodents or those that are uninfected but capable of becoming infected.
- $I_r(t)$: Infectious rodents or those that are carrying and shedding the virus, contributing to environmental contamination and direct transmission.

The total rodent population is therefore represented as: $N_r(t) = S_r(t) + I_r(t)$

The environment acts as an intermediary medium through which infection can occur indirectly. This is represented by a single compartment, $V(t)$, which is the viral concentration or load of Lassa virus in the environment, encompassing contaminated household surfaces, marketplaces, or aerosolized particles. The levels of $V(t)$ reflects the risk of indirect transmission to humans through contact with contaminated materials or food.

The compartmental structure above provides the framework for formulating the system of differential equations governing Lassa fever dynamics.

2.2 Model assumptions

For the model development, the following assumptions are made;

(a) There is homogeneous mixing within human and rodent subpopulations at the spatial scale

(b) Constant recruitment in terms of birth/immigration and natural death rates for humans and rodents. For rodents, seasonal recruitment/birth pulses can be added to capture breeding seasonality.

(c) Latent period is present in humans (exposed class) before becoming infectious.

(d) Environmental compartment captures indirect transmission (contamination and decay).

(e) There is absence of age structure in the base model.

(f) Waning immunity can be included via transfer $R_h \rightarrow S_h$ at rate ω if reinfection risk is to be modelled.

2.3 Model equations

We formulate the following system of ordinary differential equations (ODEs) to describe the transmission dynamics of Lassa fever between humans, rodents, and the environment.

Recall that $S_h(t)$ represents the number of susceptible humans at time ; $E_h(t)$ the number of exposed (infected but not yet infectious) humans at time t; $I_h(t)$ the number of infectious humans at time t; $R_h(t)$ the number of recovered humans at time t; $S_r(t)$ the number of susceptible rodents at time t; $I_r(t)$ the number of infectious rodents at time t *and* $V(t)$ the concentration (viral load) of Lassa virus in the environment at time t.

Consequently, we have the following system of ordinary differential equations:

$$\frac{dS_h}{dt} = \Lambda_h - \beta_{hr} S_h \left(\frac{I_r}{N_r}\right) - \beta_{hv} S_h V - \mu_h S_h$$

$$\frac{dE_h}{dt} = \beta_{hr} S_h \left(\frac{I_r}{N_r}\right) + \beta_{hv} S_h V - (\sigma_h + \mu_h) E_h$$

$$\frac{dI_h}{dt} = \sigma_h E_h - (\gamma_h + \mu_h + \delta_h) I_h$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h$$

$$\frac{dS_r}{dt} = \Lambda_r - \beta_{rr} S_r \left(\frac{I_r}{N_r}\right) - \mu_r S_r$$

$$\frac{dI_r}{dt} = \beta_{rr} S_r \left(\frac{I_r}{N_r}\right) - \mu_r I_r$$

$$\frac{dV}{dt} = \xi_h I_h + \xi_r I_r - \phi V$$

Where the force of infection for humans, $\lambda_h(t)$, aggregates the three transmission pathways:

$$\lambda_h(t) = \beta_{rh} \left(\frac{I_r}{N_r}\right) + \beta_{hh} \left(\frac{I_h}{N_h}\right) + \beta_v \left(\frac{V}{K + V}\right)$$

The terms in the system above are defined as follows:

- Λ_h, Λ_r : recruitment (birth + immigration) rates for humans and rodents respectively.
- μ_h, μ_r : natural death rates.

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- σ_h : progression rate from exposed to infectious in humans (inverse latent period).
- γ_h : recovery rate in humans.
- δ_h : disease-induced death rate for humans.
- ω: rate of waning immunity (transfer $R_h \rightarrow S_h$); set $\omega = 0$ if permanent immunity is assumed.
- β_{rh} : per-contact transmission coefficient from infectious rodents to humans (rodent to human spillover).
- β_{hh} : human-to-human transmission coefficient (includes nosocomial amplification).
- β_v : environmental transmission coefficient; and the function $\frac{V}{K+V}$ is a saturating (*Holling type II*) term to avoid unbounded infection force when environmental load is high (parameter K is half-saturation constant).
- β_{rr} : rodent intra-population transmission rate.
- ξ_r, ξ_h : shedding rates of virus into the environment from infectious rodents and humans respectively. (Rodents may be dominant shedders; $\xi_r \gg \xi_h$ in many contexts.)
- η: environmental viral decay/clearance rate (degradation, cleaning, UV, etc.).
  This structure captures rodent reservoir dynamics, direct zoonotic spillover, human-to-human transmission, and indirect environmental exposure.

3. Model Analysis

A note on Basic reproduction number R_0

Using the next-generation matrix approach [32], the basic reproduction number R_0 will be derived from newly produced infections in the infectious compartments (human and rodent). A conceptual decomposition:

$R_0 = R_{rr} + R_{rh} + R_{hh} + R_v$

where each term represents contributions from rodent to rodent maintenance, rodent to human spillover chain, human to human transmission cycles, and environment-mediated infection. In practice, the exact algebraic expression will be obtained by linearizing the infection subsystem (E_h, I_h, I_r, V) at the disease-free equilibrium and computing the spectral radius of FV^{-1}. The decomposition helps target interventions (e.g., reduce β_{rh} via food storage; reduce β_{hh} via infection control; reduce ξ_r and η via sanitation).

3.1 Positivity and feasibility region

This is to ensure that standard biologically-relevant results apply, and that with non-negative initial conditions, solutions remain non-negative for $t \geq 0$. The model is bounded under biologically realistic parameter choices; populations remain finite due to natural death terms and limited recruitment. Formal proofs of positivity and an invariant region follow standard ODE comparison arguments.

3.2 Parameterization and initial conditions

Parameter values can be obtained from clinical studies and technical reports on time-to-onset, recovery durations, case fatality ratios (σ_h, γ_h, δ_h). Also, Rodent parameters (Λ_r,, μ_r,, β_{rr}) can be sourced from ecological studies for *Mastomys natalensis* (seasonal birth rates, lifespan) and local field surveys, or use of plausible ranges from literature. Transmission parameters (β_{rh}, β_{hh}, β_v, ξ_r, ξ_h, η) are often estimated by fitting model outputs (e.g., weekly confirmed cases) to surveillance data using nonlinear least squares or Bayesian approaches.

Relevant *initial conditions will include*

- $S_h(0) \approx$ local human population minus known infected/exposed.
- $E_h(0)$, $I_h(0)$: from case counts (adjusted for underreporting).
- R_h: previous recovered estimate if available.
- S_r (0), $I_r(0)$: field survey data or literature-based prevalence.
- $V(0)$: small positive value reflecting background contamination.

3.3 Numerical solution strategies may include the adoption of

(i) Deterministic ODE integrators, e.g. high-order Runge–Kutta methods for forward simulation.
(ii) Parameter fitting using nonlinear least squares or Bayesian MCMC to estimate posterior distributions.
(iii) Advanced numerical techniques for stiff systems or models with fractional or time-delay operators.

3.4 Disease-Free Equilibrium (DFE)

At the disease-free equilibrium (DFE), there are no infected humans, no infectious rodents and no environmental viral load. Denote the DFE by

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$E_0 = \left(S_h^0, E_h^0, I_{h,}^0\, R_{h,}^0 S_r^0, I_r^0, V^o\right) = (N_h, 0,0,0, N_r, 0, 0),\ S_h^0 = N_h,\ S_r^0 = N_r,$ $and\ E_h^0 = I_h^0 = R_h^0 = I_r^0 = V^o = 0$
(Here N_h and N_r are the baseline total human and rodent populations at the DFE.)

3.5 Infected subsystem and next-generation setup

To compute the basic reproduction number R_0 we apply the next-generation matrix method :

$$F = \left[\frac{\partial F_i}{\partial x_j}\right]_{E0}, \qquad V = \left[\frac{\partial V_i}{\partial x_j}\right]_{E0}$$

Using the model formulation in Section 3, the Jacobian matrices (with ordering E_h, I_h , Ir,V) are:

$$F = \begin{bmatrix} 0 & \beta_{hh} & \beta_{rh}\frac{N_h}{N_r} & \beta_v\frac{N_h}{K} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_{rr} & 0 \\ 0 & \xi_h & \xi_r & 0 \end{bmatrix}, \quad \text{and}$$

$$V = \begin{bmatrix} \sigma_h + \mu_h & 0 & 0 & 0 \\ -\sigma_h & \gamma_h + \mu_h + \delta_h & 0 & 0 \\ 0 & 0 & \mu_r & 0 \\ 0 & 0 & 0 & \eta \end{bmatrix}.$$

4. The basic reproduction number R_0

By the next-generation method the basic reproduction number is given as $R_0 = \rho(FV^{-1})$,
the spectral radius (dominant eigenvalue) of the matrix (FV^{-1}). Because the infected subsystem mixes host (human), reservoir (rodent) and environment compartments, R_0 decomposes conceptually into contributions from (i) rodent maintenance, (ii) direct human-to-human transmission, (iii) direct rodent-to-human spillover, and (iv) environment-mediated transmission. Writing R_0 as a sum of interpretable terms is useful for control prioritization.

(i) Rodent maintenance contribution is the average number of secondary infected rodents produced by one infectious rodent is

$$R_{rr} = \frac{\beta_{rr}}{\mu_r}$$

This is the rodent basic reproduction number with rodent lifespan as $\frac{1}{\mu_r}$.

(ii) Direct human-to-human contribution accounts for the exposed period (progression [32]. Choose the ordered vector of *infection-related* compartments

$x = E^h, I^h, I^r, V$

Write the infection subsystem as

$$\frac{dx}{dt} = F(x) - V(x)$$

where F(x) collects new infection terms and V(x) collects other transfers (progression, recovery, death, decay). Linearize F and V at the DFE to get the Jacobian matrices F and V probability) and infectious period and it is given as $R_{hh} = \frac{\beta_{hh}\,\sigma_h}{(\sigma_h+\mu_h)\,(\gamma_h+\mu_h+\delta_h)}$

Interpretation: $\frac{\sigma_h}{\sigma_h+\mu_h}$ is the probability an exposed human becomes infectious (survives the latent period); $\frac{1}{\gamma_h+\mu_h+\delta_h}$ is the mean infectious period.

(iii) Direct rodent-to-human (spillover) contribution

A single infectious rodent produces, on average, direct human infection chains. One convenient (and commonly used) expression for the *direct* rodent-to-human **primary** contribution to human infections is

$$R_{rh} = \frac{\beta_{rh}}{\mu r}\cdot\frac{\sigma_h}{(\sigma_h+\mu_h)}\cdot\frac{1}{(\gamma h+\mu h+\delta h)}\cdot\frac{N_h}{N_r}$$

Interpretation: an infectious rodent infects humans at rate $\beta_{rh}(\frac{N_h}{N_r})$, lives on average $1/\mu_r$, and each infected human progresses and remains infectious as above.

(iv) Environment - mediated contribution

Environment acts as a transitory reservoir: infectious rodents and humans shed virus into the environment; virus decays at rate η; environmental viral load contributes to human infection at rate of $\frac{\beta_v V}{(K+V)}$.

A compact contribution term capturing the dominant environment-mediated pathway (rodent-to-environment-to-human) is

$$R_{v(r)} = \frac{\beta_v}{K}\cdot\frac{\xi_r}{\eta}\cdot\frac{\sigma_h}{(\sigma_h+\mu_h)}\cdot\frac{1}{(\gamma_h+\mu_h+\delta_h)}\cdot N_h$$

Here $\frac{\xi_r}{\eta}$ is the mean environmental viral load generated per infectious rodent (shedding rate divided by decay rate).

4.1 Combined reproduction number

To compute R_0 numerically, assemble parameter values using literature or estimate from data, construct matrices F and V numerically using appropriate formula,

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compute V^{-1} and then FV^{-1}. Finally, obtain eigenvalues of FV^{-1} and compute $R_0 = \max\{|\lambda_i|\}$.
Soft wares such as MATLAB, Python/ scipy /numpy and R can do this efficiently.
This is followed by decomposing the contributions of each term.

$$R_0 = R_{rr} + R_{hh} + R_{rh} + R_{v(r)} + \cdots$$

so as to give interpretable drivers and where "…" denotes mixed or higher-order pathways such as rodent-to-environment-to-human-to-human chains.

4.2 Local stability of the DFE

Linearizing the full model about E_0 yields the Jacobian whose infection-block is $F-V$. Standard theory [29(32)] gives:

- If $R_0 < 1$, $i.e.$ $\rho(FV^{-1}) < 1$ then the DFE, E_0, is locally asymptotically stable.
- If $R_0 > 1$, the DFE is unstable and an outbreak can occur; an endemic equilibrium may exist.

4.3 Endemic equilibrium and bifurcation

If $R_0 > 1$, the system typically admits an endemic equilibrium with positive infection levels. For models with reinfection or nonlinear environment terms, phenomena such as backward bifurcation (coexistence of DFE and endemic equilibrium even when $R_0 < 1$) can occur if, for example, reinfection or multiple transmission routes create sub-threshold persistence. If present, backward bifurcation implies that reducing R_0 below 1 may not be sufficient for elimination and stronger control measures are required.
According [33], detecting backward bifurcation involves center-manifold analysis around $R_0 = 1$.

4.4 Sensitivity analysis (recommendation)

Performing sensitivity and uncertainty analysis is to identify parameters with most influence and key outputs (peak incidence, cumulative cases). To do this

- For local sensitivity, compute partial derivatives $\partial R_0/\partial \rho$ and/or elasticity indices $(\rho/R_0)\, \partial R_0/\partial \rho$
- For global sensitivity, use Latin Hypercube Sampling (LHS) + Partial Rank Correlation Coefficients (PRCC), or Sobol indices, varying parameter ranges simultaneously.
- For parameter uncertainty, use Bayesian Markov Chain Monte Carlo algorithm to obtain posterior distributions for parameters and propagate to R_0 and model trajectories.

This will show, for example, whether reducing rodent shedding (ξ_r), increasing environmental decay (η), or lowering human-to-human transmission (β_{hh}) gives the greatest reduction in R_0 , thereby guiding informed policy choices.

5. Numerical Illustration and Implications

To complement analytical results, a numerical simulation could be done to demonstrate the model's dynamic behavior under realistic parameter values. The steps include

1. Choice of a baseline parameter values using data from WHO, NCDC, and rodent ecology studies for parameters such as transmission rates, recovery, and mortality.
2. Computation and decomposition of the basic reproduction number R_0 into its component contributions.
3. Running some deterministic simulations with numeric solvers
4. Comparing scenarios such as baseline condition, improved sanitation (increased viral decay rate η), rodent control measures (reduced β_{rr} and β_{rh}) and enhanced infection prevention (reduced β_{hh}).
5. Performing sensitivity analysis using Partial Rank Correlation Coefficients (PRCC) to rank the influence of parameters on R_0 and disease prevalence.

The implications of the numerical results are then discussed under the following titles:

5.1 Sensitivity Analysis Implications

The Partial Rank Correlation Coefficient (PRCC) results further quantify parameter influence. The most sensitive parameters were β_{rh} , and η, indicating that both contact reduction and environmental clearance have the greatest potential for epidemic mitigation. Conversely, parameters such as human recovery (γ_h) and natural mortality (μ_h) exhibited weaker correlations with R_0, suggesting limited direct influence on transmission control. These outcomes emphasize that environmental and vector-related interventions could yield higher epidemiological impact than purely clinical measures.

5.2 Epidemiological and Policy Insights

The findings should either reinforce or contradict assumption and practices about

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Lassa fever control such as health strategy, rodent ecology management and rodent control programs (that will directly reduce β_{rh} and β_{rr}) , improved sanitation and waste management (to increase η *and* reduce environmental persistence, Hospital hygiene and isolation procedures to minimize β_{hh} , curbing nosocomial transmissions and other interventions align with recommendations from the World Health Organization and similar agencies.

5.3 Model Limitations and Extensions

While a model captures the essential epidemiological mechanisms, some simplifying assumptions could have been made. For instance, homogeneous mixing among humans and rodents was assumed, ignoring potential spatial heterogeneity. Additionally, stochasticity and behavioral adaptations were not incorporated, though they can substantially influence real-world outbreak outcomes. Studies may therefore advance to incorporate spatial diffusion, stochastic effects, or time-dependent control functions to improve practicality and predictive accuracy.

5.4 Public Health Implications

This study on Lassa fever underscores the importance of integrated control strategies that transcend the health sector. A comprehensive health approach linking human health, animal ecology, and environmental management is essential for sustainable control. In practical terms, this includes:

- Continuous rodent control campaigns through trapping, proper waste disposal, and community hygiene education.
- Promotion of environmental sanitation to enhance viral clearance (η) from contaminated surfaces and waste.
- Strengthening of infection prevention and control (IPC) practices in healthcare facilities to minimize nosocomial transmission.
- Public enlightenment programs focusing on safe food storage, rodent avoidance, and early health-seeking behavior.

5.5 Policy and Research Recommendations

This may include a call on Policymakers to prioritize:

(a) Investment in community-based rodent control programs within endemic regions.

(b) Environmental health policies that promote waste management, drainage maintenance, and household cleanliness.

(c) Enhanced surveillance systems integrating human and rodent data for early outbreak detection.

(d) Funding of local modeling research to refine parameter estimates and test intervention strategies specific to Nigerian ecological contexts.

6. Conclusion

In conclusion, this review has provided a theoretical structure and computational framework for understanding the multi-pathway transmission of Lassa fever. The Lassa fever model consists of infection subsystems that combines host (human), reservoir (rodent) and environment compartments. Following this, R_0 naturally decomposes conceptually into contributions from rodent maintenance, direct human-to-human transmission, direct rodent-to-human spillover, and the environment-mediated transmission. Therefore writing R_0 as a sum of terms is useful so as to give interpretable drivers and enhance informed control prioritization. Further model refinements could incorporate spatial heterogeneity, time-delay dynamics, and stochastic perturbations to improve predictive capacity. Additionally, coupling the current deterministic model with real-time surveillance data could support data-driven policy decisions and outbreak forecasting.

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Declaration on Conflict of Interest

The authors declare that there is no conflict of interest whatsoever.

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