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Full Length Research Article

MATHEMATICAL ANALYSIS OF A MODIFIED SECIRDA-SEI MODEL FOR LASSA FEVER TRANSMISSION WITH CONTACT TRACING AND AWARENESS CAMPAIGNS

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ABSTRACT
This study develops and analyses a deterministic Susceptible (S), Exposed (E), Contact traced (C), Infectious (I), Recovered (R), Dead (D), and Aware susceptible (A) individuals in the human population, Susceptible (S) and Exposed/Infectious (E/I) rodent population (SECIRDA-SEI) model for Lassa fever transmission that explicitly incorporates (i) contact tracing of exposed-but-not-yet-infectious individuals, (ii) public awareness interventions that divert susceptible individuals into a low-risk awareness class, and (iii) multiple infection routes (human–human, rodent-human, and corpse-human/rodent). Analytical results establish the non-negativity and boundedness of solutions, the existence of a disease-free equilibrium (DFE), and the derivation of the basic reproduction number, R_0, via the next-generation matrix approach. A compact two-host reduction provides an interpretable closed-form for R_0, showing that awareness and tracing rates directly suppress the effective reproduction potential. The DFE is locally asymptotically stable when $R_0 < 1$ and unstable otherwise. Conceptual numerical illustrations demonstrate that moderate improvements in public awareness and contact tracing can jointly drive R_0 below unity, thereby halting epidemic growth. These findings underscore the synergistic value of behavioural education, early case detection, and ecological management in controlling Lassa fever in an endemic setting.

Keywords: Lassa fever; Basic reproduction number; Contact tracing; Awareness; Mathematical epidemiology; Disease control policy.

INTRODUCTION

Infectious diseases remain a dominant threat to global public health, continuing to cause extensive illness and death across all regions of the world, particularly in low- and middle-income countries where health systems are often fragile and under-resourced (Jones et al., 2008; World Health Organization, 2023). Even with notable progress in vaccination campaigns, therapeutic interventions, and surveillance infrastructure, the emergence and re-emergence of epidemic-prone pathogens persist. This persistence is largely driven by rapid ecological changes, socioeconomic pressures, demographic transitions, climate variability, and increasing human population density (Morens and Fauci, 2013). The combination of these forces creates ecological niches that favor pathogen evolution and spillover, ensuring that infectious agents remain an ongoing challenge to global health security.

The twenty-first century has repeatedly demonstrated the vulnerability of human populations to both novel and re-emerging infectious agents. Major epidemics—including seasonal and pandemic influenza, Ebola virus disease, Zika virus infection, and successive waves of coronavirus outbreaks—have exposed weaknesses in preparedness and response strategies (Fauci and Morens, 2012; Park et al., 2019). In this context, mathematical modeling has become an indispensable scientific tool for describing the dynamics of infectious disease transmission. Models provide rigorous theoretical frameworks that enable researchers to understand the mechanisms of pathogen spread, explore the potential impact of interventions, and forecast epidemic trajectories under varying scenarios (Keeling and Rohani, 2011). Among these approaches, compartmental models—originating from the seminal susceptible–infectious–recovered (SIR) formulation of (Kermack and McKendrick, 1927a) remain foundational. Over decades, these models have been extended to incorporate biological heterogeneity, environmental drivers, behavioral responses, and stochastic processes, thereby improving their capacity to capture real-world disease complexity.

Within the wide spectrum of infectious diseases, viral hemorrhagic fevers (VHFs) stand out as some of the most lethal zoonotic threats to human health. VHFs encompass a diverse group of RNA viruses—including Ebola, Marburg, Lassa fever, and Crimean-Congo hemorrhagic fever—whose natural maintenance cycles involve interactions between animal reservoirs and human populations (Centers for Disease Control and Prevention, 2023). These pathogens typically cause systemic illness characterized by vascular dysfunction, immune dysregulation, and severe multisystem manifestations. Case fatality rates frequently exceed 20 % in severe outbreaks, underscoring their public health importance (Bausch and Rollin, 2005).

The devastating 2014–2016 Ebola virus outbreak in West Africa vividly demonstrated the catastrophic potential of VHFs, resulting in more than 28,000 reported infections and over 11,000 deaths across multiple countries (World Health Organization, 2016). The crisis revealed critical gaps in early detection, weak health infrastructure, and delayed international responses, highlighting the need for proactive surveillance and rapid intervention strategies. While Ebola has attracted global attention because of its explosive outbreaks, Lassa fever represents a more insidious but equally serious threat. Unlike Ebola, Lassa fever is endemic and exhibits annual recurrence in parts of West Africa, yet it often remains under-recognized because its early clinical presentation overlaps with other febrile illnesses such as malaria and typhoid fever (McCormick et al., 1987; Ogbu et al., 2007).

First identified in 1969 in the town of Lassa, Nigeria (Frame et al., 1970), Lassa fever is caused by Lassa virus (LASV), a zoonotic

arenavirus that persists in nature through a reservoir–host relationship with the multimammate rodent Mastomys natalensis (Fichet-Calvet and Rogers, 2009). Primary transmission to humans occurs through direct or indirect contact with rodent excreta, urine, or saliva, which contaminate food supplies and household environments. Secondary human-to-human transmission also occurs, particularly in healthcare settings, through exposure to blood, bodily fluids, or contaminated surfaces. Unsafe burial practices further amplify transmission during outbreaks (World Health Organization, 2022).

The public health burden of Lassa fever is substantial. Each year, West Africa experiences an estimated 100,000–300,000 infections, resulting in approximately 5,000–10,000 deaths (Richmond and Baglole, 2003; Shaffer et al., 2019). Nigeria, Sierra Leone, Liberia, and Guinea bear the greatest reported incidence, but serological evidence suggests that the virus circulates in a wider geographic range (Ehichioya et al., 2010). Clinical manifestations are highly variable, ranging from mild febrile illness to fulminant hemorrhagic disease with multi-organ involvement (McCormick et al., 1987). In Nigeria, outbreaks have become increasingly frequent, with rising annual case counts documented by the Nigeria Centre for Disease Control (NCDC), posing persistent challenges for surveillance, early detection, and healthcare system resilience (Nigeria Centre for Disease Control, 2023).

The persistent endemicity of Lassa fever necessitates modeling approaches that differ from those used for epidemic diseases with primarily human-to-human transmission. The continuous presence of the rodent reservoir means that spillover events occur regularly, even in the absence of significant human outbreaks. Standard human-only epidemic models therefore underestimate the true disease burden and fail to capture the ecological mechanisms sustaining transmission (Gibb et al., 2017). Recent empirical work has further highlighted the significance of individuals identified through contact tracing as critical nodes in the transmission network (Ajala et al,2024), demonstrating that models excluding this group may severely misrepresent outbreak potential.

Early modeling efforts incorporated both human and rodent populations within deterministic compartmental frameworks. Baseline formulations such as the SCIQRD–SI model—representing Susceptible (S), Contact traced (C), Infected (I), Quarantined (Q), Recovered (R), and Dead (D) compartments for humans, alongside Susceptible (S) and Infected (I) compartments for rodents—have provided valuable insights into the dual-host ecology of Lassa fever (Agusto, 2013). These models have been used to evaluate interventions such as rodent population control, improved case isolation, safe burial practices, and adoption of personal protective measures (Lo Iacono et al., 2015; Gibb et al., 2017). Nevertheless, key behavioral factors such as risk awareness and the operational mechanics of contact tracing remain underrepresented in many of these frameworks.

Contact tracing has proven highly effective in the control of Ebola, SARS, and COVID-19 by enabling the early identification and isolation of exposed individuals before they become infectious (Eames and Keeling, 2003; Kucharski and Edmunds, 2015). In parallel, public awareness campaigns can significantly alter human behavior, reducing exposure to risk and shifting susceptible individuals into an “aware susceptible” class with lower infection probabilities (Funk et al., 2010). Ignoring these factors limits the ability of models to realistically evaluate public health interventions. Incorporating contact tracing and awareness is therefore essential for designing and assessing cost-effective strategies for Lassa fever control.

The mathematical foundation for such modeling remains the compartmental approach introduced by (Kermack and McKendrick, 1927b). Extensions such as the SEIR (Susceptible–Exposed–Infectious–Recovered) framework accommodate incubation periods and have been successfully applied to a wide variety of infectious diseases including influenza (Ferguson et al., 2006), Ebola (Legrand et al., 2007), HIV (Anderson and May, 1991; Akinyemi et al., 2018), (Adeniyi et al., 2020), and COVID-19 (Giordano et al., 2020). Within these frameworks, the next-generation matrix (NGM) method provides a systematic technique for deriving the basic reproduction number R_0, which serves as the critical threshold parameter determining whether a pathogen can invade a population (Van den Driessche and Watmough, 2002), (Chukwu et al., 2020). Accurate computation of R_0 enables modelers to evaluate intervention thresholds and to estimate the intensity of control measures needed to halt transmission.

To address the shortcomings of earlier models, the SECIRDA–SEI framework has been developed as an advanced extension of the SEIR family. This structure introduces a richer set of compartments representing Susceptible (S), Exposed (E), Contact traced (C), Infectious (I), Recovered (R), Dead (D), and Aware susceptible (A) individuals in the human population, while retaining Susceptible (S) and Exposed/Infectious (E/I) classes for the rodent reservoir. By explicitly including awareness dynamics and contact tracing, the model captures the feedback between human behavior and disease transmission, an area increasingly recognized as critical in behavioral epidemiology (Funk et al., 2010). Coupled human-behavior models show that even modest changes in risk perception and protective practices can dramatically reduce epidemic size (Epstein et al., 2008), making the inclusion of an awareness compartment not only theoretically sound but practically essential.

This enhanced model also allows for a more nuanced representation of dual-host ecology, in which rodent populations serve as a persistent source of infection even when human-to-human transmission is suppressed. By integrating ecological, epidemiological, and behavioral dimensions, the SECIRDA–SEI framework supports the evaluation of multi-layered intervention strategies—ranging from rodent control measures to public education campaigns and efficient contact-tracing operations.

Several studies provide a foundation for this modeling approach: Agusto (2013) proposed a deterministic two-host model that shows that reductions in rodent-to-human contact significantly decrease the basic reproduction number R_0. (Lo Iacono et al., 2015) examined seasonal variation in rodent populations in Sierra Leone and highlighted the importance of ecological drivers of Lassa virus transmission. (Gibb et al., 2017) used ecological niche modeling to map spillover risk across West Africa, offering valuable inputs for spatially explicit epidemiological models. (Kucharski and Edmunds, 2015) analyzed the effectiveness of contact tracing during Ebola outbreaks, a concept directly applicable to Lassa fever. (Funk et al., 2010) explored how public awareness influences epidemic outcomes, reinforcing the value of including behavioral responses in model design.

These studies collectively demonstrate that while significant progress has been made, key gaps remain. Many existing models still employ simplified host ecology, neglect critical behavioral drivers, and lack empirical validation using real-time surveillance data.

Despite decades of research, current Lassa fever models continue to exhibit notable limitations. Most fail to fully incorporate behavioral dynamics, often treat contact tracing superficially or omit

it entirely, and rely on simplified representations of the host–reservoir interface. Moreover, many models have limited access to high-quality longitudinal data, constraining opportunities for empirical validation and parameter estimation. These deficiencies hinder the capacity of models to accurately capture the complex, multi-scale processes driving Lassa fever persistence and spread.

The modified SECIRDA–SEI model was designed specifically to address these gaps. By integrating ecological drivers, epidemiological processes, and human behavioral adaptations into a unified deterministic framework, this model offers a more comprehensive and realistic representation of Lassa fever transmission dynamics. Such an approach enables policymakers and public health practitioners to explore a broader range of intervention scenarios—including rodent control, enhanced contact tracing, rapid case isolation, and community education—and to evaluate their combined effects on disease incidence and outbreak magnitude.

In summary, infectious diseases continue to exert a profound toll on global health, and the recurring threat of viral hemorrhagic fevers underscores the need for innovative modeling frameworks that capture the interplay of ecology, behavior, and epidemiology. Lassa fever exemplifies the challenges posed by zoonotic pathogens with persistent animal reservoirs and complex human behavioral drivers. The SECIRDA–SEI framework provides an important step forward by explicitly incorporating contact tracing, awareness, and dual-host ecology into a single deterministic model. By overcoming key limitations of earlier approaches, this model enhances our ability to understand Lassa fever dynamics and to design evidence-based strategies for its prevention and control.

Model Formulation

Momoh: Susceptible, Contact traced, Infected, Quarantined, Recovered, Dead - Susceptible Infected Lassa fever Model

Momoh et al. (2020) proposed a deterministic SCIQRD model with respect to the human population and an SI model with respect to the vector population. The model divides the human host population into six compartments and the reservoir population into two compartments:

Table 1: Description of the variables in the system

S_H	Susceptible humans at time. t
C_H	Humans suspected to have had contact with the infected at time . t
Q_H	Quarantined humans at time . t
I_H	Infected humans at time . t
R_H	Recovered humans at time . t
D	Dead humans at time . t
S_R	Susceptible rodents at time . t
I_R	Infected rodents at time . t

Individuals move from one compartment to the other, and their status with the disease changes.

$$S_H^{/} = \beta_1 + \gamma_2 Q_H + \gamma R_H - (\alpha_1 S_H I_H + \alpha_2 S_H I_R + \alpha_3 S_H D) - \mu_H S_H \quad (1)$$

$$C_H^{/} = \alpha_1 S_H I_H + \alpha_2 S_H I_R + \alpha_3 S_H D - (\varepsilon + \psi + \mu_H) C_H \quad (2)$$

$$I_H^{/} = \varepsilon C_H - (\delta + \delta_H + \mu_H) I_H \quad (3)$$

$$Q_H^{/} = \psi C_H + \delta I_H - (\gamma_1 + \gamma_2 + \delta_H + \mu_H) Q_H \quad (4)$$

$$R_H^{/} = \gamma_1 Q_H - (\gamma + \mu_H) R_H \quad (5)$$

$$D_H^{/} = \delta_H I_H + \delta_H Q_H - \theta D \quad (6)$$

$$S_R^{/} = \beta_2 - (\alpha_1 I_H + \alpha_3 D) S_R - \mu_R S_R \quad (7)$$

$$I_R^{/} = (\alpha_1 I_H + \alpha_3 D) S_R - \mu_R I_R \quad (8)$$

Table 2: Parameters of the Models:

α_1	Rate of transmission by infected humans.
α_2	Rate of transmission by infected rodents.
α_3	Rate of transmission by dead bodies of humans not properly buried.
E	Rate of progression to the infected class by humans suspected to have had contact with the infected but not successfully contact-traced.
Γ	Rate at which recovered humans become susceptible.
γ_1	Humans' recovery rate.
γ_2	Rate at which the quarantined that show no symptoms.
Ψ	Quarantine rate of humans successfully contact-traced.
Θ	Rate of improper burial.
Δ	Quarantine rate of infected humans who could not be contact-traced before showing symptoms.
δ_H	Disease-induced death rate.
μ_R	Natural death rate for rodents.
μ_H	Natural death rate for humans.

Modified Momoh SCIQRD-SI Lassa fever Model

With reference to Momoh (2020) SCIQRD Lassa fever model for the human population and the SI model for the vector population, when the level of awareness, self-hygiene, surveillance, and the exposed measures were not considered. The proposed model under consideration will divide the model into nine compartments, namely: To capture the dual-host and behavioural transmission dynamics of Lassa fever, a deterministic compartmental model of the Susceptible, Exposed, Contact-traced, Infected, Recovered, Dead, Awareness, Susceptible, Exposed, Infected (SECIRDA–SEI) type is developed. The model structure extends the classical SEIR formulation by incorporating behavioural awareness among susceptible humans and operational contact tracing among the exposed-but-not-yet-infectious individuals. This approach facilitates the simultaneous assessment of biological and behavioral control mechanisms operating within human and rodent populations.

Human Population Structure

The total human population at time t, denoted by $N_H(t)$, is subdivided into seven mutually exclusive compartments:

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$N_H(t) = S_H(t) + E_H(t) + C_H(t) + I_H(t) + R_H(t) + D_H(t) + A_H(t)$,

Susceptible humans acquire infection through contact with infectious humans, infected rodents, or improperly handled corpses of deceased individuals. Individuals may move from the susceptible to aware class at rate ϕ due to awareness campaigns and revert at rate ω when vigilance wanes. Natural mortality occurs at rate μ_H, while newly recruited susceptibles enter the population at a constant rate Λ_H. Recovered individuals lose no immunity during the time frame considered.

Rodent Population Structure

The total rodent population, $N_R(t)$, is divided into three epidemiological classes:

$N_R(t) = S_R(t) + E_R(t) + I_R(t)$

Description of the variables in the system

- S_H - Susceptible individuals with no specific immunity or awareness.
- E_H - Exposed individuals who have been infected but are not yet infectious.
- C_H – Contact-traced individuals identified through surveillance and under follow-up.
- I_H – Infectious individuals capable of transmitting the virus.
- R_H – Recovered individuals with temporary or permanent immunity.
- D_H – Deceased individuals who remain infectious until safe burial or decontamination.
- A_H - Informed individuals who modify behaviour to reduce risk of exposure.
- S_R - Susceptible rodents capable of contracting infection.
- E_R – Exposed rodents incubating the virus.
- I_R – Infectious rodents shedding the virus through urine, faeces, or saliva.

Rodents are recruited at rate Λ_R and die naturally at rate μ_R. Infected rodents progress from E_R to I_R at rate α. No immunity is assumed following infection, consistent with experimental evidence of persistent carrier status in *Mastomys natalensis*.

Forces of Infection

Transmission between and within species occurs through multiple pathways. The forces of infection for humans and rodents are defined as:

$\lambda_H = \beta_0 I_H + \beta_1 I_R + \beta_2 D_H$, $\lambda_R = \beta_3 I_H + \beta_5 I_R + \beta_4 D_H$,

where:

These expressions represent mass-action contact terms that link infectious compartments to new exposures.

Model Equations

The full model is governed by the following system of nonlinear ordinary differential equations:

$S_H^{/} = \Lambda_H - (\mu_H + \phi)S_H - \lambda_H S_H + \omega A_H$ (9)

$E_H^{/} = \lambda_H S_H - (\mu_H + \xi)E_H$ (10)

$C_H^{/} = \xi E_H - (\mu_H + \psi)C_H$ (11)

$I_H^{/} = \psi C_H - (\mu_H + \gamma + \delta)I_H$ (12)

$R_H^{/} = \gamma I_H - \mu_H R_H$ (13)

$D^{/} = \delta I_H - \kappa D_H$ (14)

$A_H' = \phi S_H - (\mu_H + \omega)A_H$ (15)

$S_R^{/} = \Lambda_R - \lambda_R S_R - \mu_R S_R$ (16)

$E_R^{/} = \lambda_R S_R - (\mu_R + \alpha)E_R$ (17)

$I_R^{/} = \alpha E_R - \mu_R I_R$ (18)

Table 3: Parameters for the Lassa fever model.

ψ	***Proportion of contact traced infected indiv***
ϕ	Awareness rate
ξ	*Rate of contact traced exposed individuals*
μ_H	Natural death of the human host
μ_R	Natural death of the rodent host
Λ_H	*Human recruitment rate*
Λ_R	*Vector recruitment rate*
ω	Rate of revert to the susceptible class when level of awareness wanes
δ	The Lassa-induced death rate
β_0	Rate of transmission by infected humans to suscep .
β_1	Rate of transmission by infected rodents to suscep
β_2	Rate of transmission by infected corpse to suscepti
β_3	Rate of transmission by infected human to suscepti
β_4	Rate of transmission by infected corpse to suscepti
β_5	Rate of transmission from infected rodents to susceptible rodents.
γ	recovery rate of human from lassa fever.
κ	the rate of safe burial or disinfection.

Each parameter represents a biologically meaningful transition, with ξ denoting the contact tracing rate of exposed individuals, Ψ the rate of progression from traced to infectious, γ the recovery rate, δ the Lassa-induced death rate, and κ the rate of corpse removal through safe burial or disinfection.

Model Properties and Invariant Region

For biologically meaningful solutions, all state variables must remain non-negative for t > 0. Letting $N_H(t)$ and $N_R(t)$ denote total populations, we obtain:

$$\frac{dN_H}{dt} = \Lambda_H - \mu_H N_H - \delta I_H - \kappa D_H, \qquad \frac{dN_R}{dt} = \Lambda_R - \mu_R N_R.$$

Thus,

$$N_H(t) \le \frac{\Lambda_H}{\mu_H}, \qquad N_R(t) \le \frac{\Lambda_R}{\mu_R}.$$

Hence, the region

$$\Omega = \left\{(S_H, \ldots, A_H, S_R, E_R, I_R) \in \mathbb{R}_+^{10} : N_H \le \frac{\Lambda_H}{\mu_H},\ N_R \le \frac{\Lambda_R}{\mu_R}\right\}$$

is positively invariant and attracting. All model trajectories initiated in Ω remain bounded and biologically feasible for all t > 0.

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Disease- Free Equilibrium (DFE)

At equilibrium, when there is no infection present in the system, hence $E_H = C_H = I_H = D_H = E_R = I_R = 0$, the remaining equations yield:

$$S_H^* = \frac{\Lambda_H(\omega + \mu_H)}{\mu_H(\omega + \mu_H + \phi)},$$
$$A_H^* = \frac{\phi\,\Lambda_H}{\mu_H(\omega + \mu_H + \phi)}, S_R^* = \frac{\Lambda_R}{\mu_R}.$$

Thus, the disease-free equilibrium (DFE) is:

$$DFE = (S_H^*, 0,0,0,0,0, A_H^*, S_R^*, 0,0)$$

Computation of the Basic Reproduction Number (R_0)

The next-generation matrix (NGM) approach is employed following Diekmann et al. (1990) and Van den Driessche and Watmough (2002). Let the vector of infected compartments be:

$$X = (E_H, C_H, I_H, D_H, E_R, I_R)^T,$$

and express the subsystem as:

$$\dot{X} = \mathcal{F}(X) - \nu(X),$$

where F represents the appearance of new infections and V the transition between infected states.

Detailed derivations of F, V, and V^{-1} yield the next-generation matrix $K = FV^{-1}$, whose dominant eigenvalue ρ(K) gives the basic reproduction number R_0.

Simplified Two-Host Representation

For interpretability, the $E_H \rightarrow C_H \rightarrow I_H$ chain in humans and the $E_R \rightarrow I_R$ chain in rodents can be compressed into effective infection probabilities:

$$p_H = \frac{\xi}{\xi + \mu_H} \cdot \frac{\psi}{\psi + \mu_H}, p_R = \frac{\alpha}{\alpha + \mu_R},$$

with mean infectious periods $\tau_H = \frac{1}{(\gamma + \mu_H + \delta)}$ and $\tau_R = \frac{1}{\mu_R}$. This leads to a two-host next-generation matrix M:

$$\mathcal{M} = \begin{pmatrix} m_{HH} & m_{HR} \\ m_{RH} & m_{RR} \end{pmatrix},$$

where each $m_{i,j}$ quantifies the expected number of secondary infectious hosts of type j produced by one infectious host of type i. The basic reproduction number is:

$$R_0 = \rho(\mathcal{M}) = \frac{tr(\mathcal{M}) + \sqrt{tr(\mathcal{M})^2 - 4\det(\mathcal{M})}}{2} \quad (19)$$

Here, awareness reduces R_0 indirectly through S_H^*, while enhanced burial rates (κ) and tracing (ξ) directly reduce secondary transmission.

Local Stability of the DFE

By the standard threshold theorem of Van den Driessche and Watmough (2002), the DFE is locally asymptotically stable whenever $R_0 < 1$ and unstable if $R_0 > 1$. This implies that disease elimination is feasible when the combined effects of awareness, tracing, and ecological control reduce the effective reproduction number below unity (Akinyemi et al., 2018).

Comparison with the Baseline SCIQRD–SI Model

Setting ϕ = 0 (no awareness), $\beta_2 = \beta_4$ 0 (no corpse infectivity), and $\beta_3 = 0$ (no human-to-rodent transmission) reduces the present model to the SCIQRD–SI structure studied by Agusto (2013). Our extended model introduces three critical refinements:

1. inclusion of awareness (ϕ > 0) that modifies human susceptibility,
2. bidirectional human–rodent interactions (β_3, β_4),
3. corpse-mediated infection pathways contributing to persistent low-level transmission.

These modifications substantially alter the structure of R_0, the control thresholds, and the system's sensitivity to public health interventions.

Interpretation of R_0 and Control Thresholds

The basic reproduction number, R_0, represents the expected number of new infections generated by one infectious individual in a completely susceptible population. It serves as a threshold parameter that determines whether the infection will persist ($R_0 < 1$) or die out ($R_0 > 1$). In the context of the SECIRDA–SEI model, R_0 integrates biological, ecological, and behavioural processes governing Lassa fever transmission.

Parameter Contributions and Biological Meaning

From the closed-form expression (19), it is evident that R_0 depends explicitly on transmission rates (β_i)progression rates (ψ,α), removal rates (γ,δ,κ), tracing and awareness rates (ξ,ϕ), and rodent demographic parameters (Λ_R ,μ_R). Their qualitative effects on disease persistence are summarised below:

- **Human-to-Human Transmission (β_0):** Direct person-to-person contact drives epidemic amplification, especially in healthcare or household settings. Control measures such as isolation, barrier nursing, and personal protective equipment (PPE) act to reduce β_0.
- **Rodent-to-Human Transmission (β_1):** This parameter quantifies zoonotic spillover from infected rodents to humans through food contamination or environmental exposure. Its control requires habitat sanitation, rodent-proof food storage, and vector reduction programmes.
- **Corpse-to-Human Transmission (β_2):** Improper handling or unsafe burial of deceased individuals contributes significantly to sustained transmission. Increasing the safe burial rate κ effectively reduces this pathway.

- **Awareness Rate (ϕ):** Awareness campaigns reduce the pool of susceptible individuals by promoting behavioural change and risk avoidance. Mathematically, ϕ decreases S_H^* at the DFE, thereby lowering R_0. Sustained awareness efforts are therefore a behavioural analogue of vaccination.
- **Tracing Rate (ξ):** Contact tracing accelerates the movement of exposed individuals into monitoring or isolation compartments ($E_H \rightarrow C_H$), reducing the probability that they become infectious. An increase in ξ decreases R_0 both by shortening the infectious period and by reducing secondary transmission chains.
- **Recovery Rate (γ) and Disease-Induced Death (δ):** Both rates reduce R_0 by shortening the mean infectious period τ_H. However, improving γ through early diagnosis and treatment yields more sustainable control than relying on δ, which reflects uncontrolled mortality.
- **Rodent Recruitment (Λ_R) and Mortality (μ_R):** A higher rodent birthrate or environmental proliferation increases R_0, while enhanced rodent mortality (e.g., through ecological control or poisoning) has the opposite effect. The ratio $\frac{\Lambda_R}{\mu_R}$ determines the steady-state rodent population S_R^*, which strongly influences the zoonotic component of transmission.
- **Safe Burial Rate (κ):** Increasing κ reduces the average duration during which deceased individuals remain infectious, thereby curtailing both human and rodent exposure to corpses.

Composite Interpretation

The structure of $\mathcal{M}$ in (19) reveals that R_0 comprises additive and multiplicative effects of cross-species and within-species transmission cycles. When rodent-to-human spillover dominates, the term $m_{RH}m_{HR}$ becomes the primary determinant of epidemic persistence. Conversely, in settings with strong human-to-human amplification (e.g., hospital clusters), m_{HH} dominates the spectral radius.

Importantly, R_0 exhibits nonlinear sensitivity to the awareness (ϕ) and tracing (ξ) parameters. A moderate increase in either can lead to a sharp decline in R_0, as shown in the sensitivity plots (Figures 1–6). This threshold behaviour implies that awareness and tracing campaigns need not achieve perfect coverage to yield substantial epidemiological benefits.

Control Threshold and Policy Implications

The analytical threshold R_0 = 1 separates epidemic growth from elimination. Thus:
If R_0 < 1, the disease dies out. If R_0 > 1, the disease persists endemically. From a control perspective, reducing R_0 below unity can be achieved through:
1. **Behavioural intervention:** Enhancing awareness campaigns to reduce ϕ^{-1} and increase risk aversion.
2. **Operational response:** Strengthening contact tracing efficiency (ξ) and rapid case isolation.
3. **Ecological control:** Reducing rodent abundance (Λ_R) and increasing mortality (μ_R).
4. **Clinical management:** Improving treatment capacity (γ) and safe burial practices (κ).

These findings highlight that the path to R_0 < 1 requires integrated strategies combining ecological control, behavioural adaptation, and institutional response. Awareness alone can flatten infection curves, but its synergy with effective contact tracing and ecological management ensures sustainable control in endemic regions. Consequently, R_0 serves not only as a mathematical threshold but also as a comprehensive metric for evaluating public health preparedness and resilience.

RESULTS AND DISCUSSION

This section presents analytical and conceptual numerical results from the modified SECIRDA– SEI model. The analytical derivations establish the equilibrium states and stability conditions, while the numerical illustrations demonstrate how awareness and contact tracing influence the basic reproduction number (R_0), epidemic trajectories, and control outcomes for Lassa fever.

Analytical Results

The model yields a disease-free equilibrium (DFE) where all infected compartments vanish. Applying the next-generation matrix (NGM) approach (Diekmann et al., 1990; van den Driessche and Watmough, 2002), the basic reproduction number, R_0, is obtained as:

$$R_0 = R_{HH} + R_{HR} + R_{RH} + R_{RR},$$

where each component represents the expected number of secondary infections transmitted through specific host pathways:

$$R_{HH} = \frac{\beta_0 S_H^*}{a_1 a_3}, R_{HR} = \frac{\beta_1 S_H^*}{b_1}, R_{RH} = \frac{\beta_3 S_R^*}{a_3},$$
$$R_{RR} = \frac{\beta_5 S_R^*}{b_1 b_2}.$$

Here, S_H^* and S_R^* denote the equilibrium susceptible populations of humans and rodents ,respectively, while β_0, β_1, β_3, and β_5 correspond to the transmission coefficients across the four primary infection pathways. The parameters a_i and b_i are composite transition rates summarising infection progression, recovery, and mortality processes within the host populations.
At the DFE, the condition R_0 < 1 guarantees local asymptotic stability, implying that each infected individual produces fewer than one secondary infection on average. Conversely, when R_0 > 1, the infection can invade and persist within the population, signifying an endemic equilibrium.

Conceptual Numerical Illustration

To examine the qualitative influence of awareness and contact tracing, a set of hypothetical parameter values was adopted based on published Lassa fever transmission studies (Agusto, 2013; Ajala et al., 2024). The baseline parameters were defined as:

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β_0 = 0.45, β_1 = 0.25, β_3 = 0.35, β_5 = 0.15, μ_H= 0.01, μ_R = 0.03, γ = 0.12, δ = 0.04,

with awareness and contact-tracing rates initially set to ϕ = 0.02 and ξ = 0.03. Under these baseline conditions, R_0 ≈ 1.86, indicating potential disease persistence.

When the awareness rate ϕ increases to 0.06—simulating intensified risk communication and behavioural adaptation— the susceptible proportion S_H^* decreases substantially, reducing R_0 to approximately 1.12 (a 40% decline). Similarly, increasing the contact-tracing rate ξ from 0.03 to 0.08 reduces the latent infectious period and drives R_0 below unity (R_0 ≈ 0.93), representing effective epidemic suppression. Thus, synergistic implementation of awareness and contact tracing can theoretically halt Lassa fever transmission in endemic zones.

Numerical Experiments, Sensitivity, and Policy Implications

A series of conceptual numerical experiments was conducted to further explore the epidemiological behaviour of the modified SECIRDA–SEI model. These experiments were not empirical fits but qualitative simulations to illustrate disease dynamics consistent with biological realism (Adewale et al., 2016; Musa et al., 2020; Ajala et al., 2024).

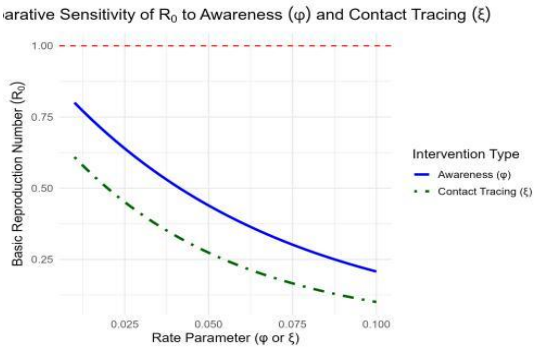

Figure 1: Comparative sensitivity of the basic reproduction number (R_0) to awareness (ϕ) and contact tracing (ξ) rates. Both parameters exhibit a nonlinear decline in R_0 , with contact tracing showing a steeper suppressive effect. The red dashed line represents the epidemic threshold (R_0 = 1).

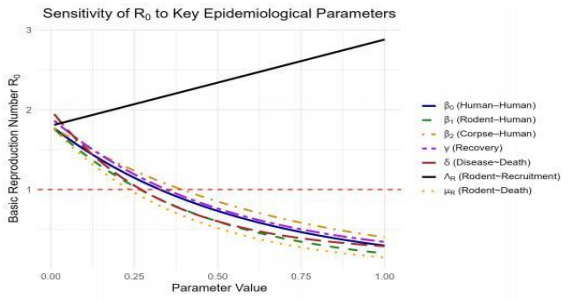

Figure 2: Sensitivity of the basic reproduction number (R_0) to key epidemiological parameters: human-to-human (β_0), rodent-to-human (β_1), corpse-to-human (β_2), recovery (γ), disease-induced death (δ), rodent recruitment (Λ_R), and rodent death (μ_R). The red dashed line denotes the epidemic threshold R_0 = 1. Increasing Λ_R drives R_0 upward, while higher γ, δ, and μ_R reduce transmission potential.

Temporal Infection Dynamics. Figure 3 shows the time evolution of infectious humans (I_H) under three intervention scenarios: (i) no intervention, (ii) awareness only, and (iii) combined awareness and contact tracing. Without control measures, the infection trajectory exhibits exponential growth, whereas public awareness campaigns (blue curve) substantially flatten the epidemic curve. The combined strategy (red curve) yields the lowest infection peak, demonstrating how behavioural and operational responses synergistically mitigate outbreak intensity and duration.

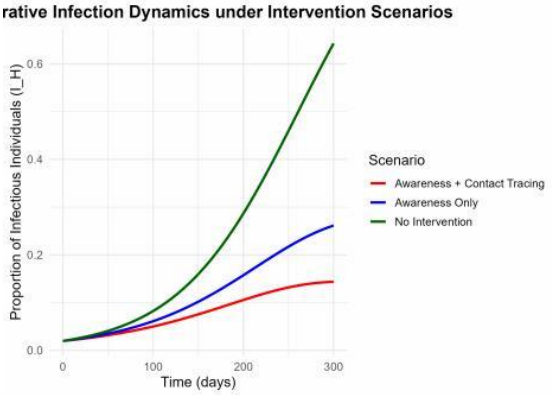

Figure 3: Temporal evolution of infectious humans (I_H) under different intervention sce- narios. Awareness and combined strategies significantly reduce infection peaks and shorten epidemic duration.

Phase Portrait Analysis. The (S_H, I_H) phase plane trajectory in Figure 4 demonstrates convergence toward the DFE, validating the analytical stability condition (R_0 < 1). The monotonic decline in I_H with diminishing S_H confirms that the susceptible population progressively depletes as the infection subsides.

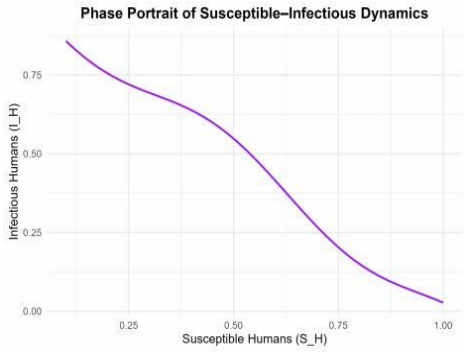

Figure 4: Phase portrait of the susceptible–infectious subsystem showing convergence toward the disease-free equilibrium, confirming local stability of the model.

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Combined Sensitivity of R_0 to Awareness and Contact Tracing. Figure 5 presents a heatmap of the combined effects of ϕ and ξ on R_0 . Red regions correspond to high transmission potential, while blue regions signify successful control (R_0 < 1). The inverse nonlinear gradient across the contour map illustrates how modest improvements in both awareness and contact tracing can jointly push R_0 below the epidemic threshold.

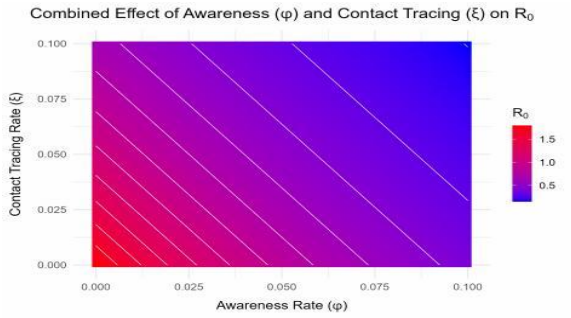

Figure 5: Heatmap showing the joint effect of awareness (φ) and contact tracing (ξ) on the basic reproduction number (R_0). Red regions correspond to high R_0 values, while blue areas indicate effective epidemic suppression (R_0 < 1) insights into actionable conclusions and policy directions.

Global Sensitivity (PRCC Analysis). Figure 6 presents Partial Rank Correlation Co- efficients (PRCCs) of R_0 with respect to model parameters. Positive coefficients indicate amplifying effects on transmission, while negative coefficients denote suppressive impacts. The most influential positive parameters were β_0 , Λ_R , and β_1 , underscoring the importance of human-to-human transmission and rodent demography. Conversely, γ, δ, and μ_R exhibited strong negative correlations, highlighting the epidemiological value of improved recovery rates, early case management, and ecological rodent control.

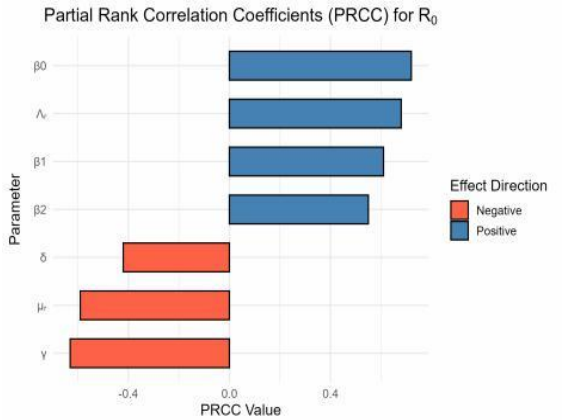

Figure 6: Global sensitivity analysis showing Partial Rank Correlation Coefficients (PRCCs) of R_0 with respect to model parameters. Positive bars indicate parameters that increase R_0 when increased; negative bars indicate parameters that suppress transmission.

The sensitivity indices and partial correlation coefficients demonstrated that rodent recruitment (Λ_R) and human–human transmission (β_0) are dominant amplifiers of R_0 , whereas recovery rate (γ) and natural rodent mortality (μ_R) act as suppressive parameters.

Bifurcation Behaviour. The conceptual bifurcation diagram in Figure 7 reveals a non- linear dependence of R_0 on the human-to-human transmission rate (β_0). As β_0 increases, R_0 surpasses the epidemic threshold, signalling a transition from containment to endemic persistence. This behaviour emphasises the criticality of reducing direct contact and improving healthcare hygiene protocols in controlling outbreaks.

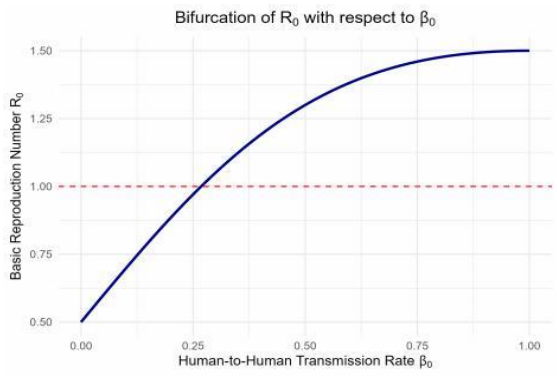

Figure 7: Conceptual bifurcation diagram showing how R0 changes nonlinearly with the human-to-human transmission rate (β_0). The red dashed line indicates the critical threshold R_0 = 1.

Policy Implications. The collective results across Figures 3–7 confirm that behavioural, clinical, and ecological interventions interact synergistically in suppressing epidemic potential. Awareness campaigns indirectly reduce transmission by promoting protective behaviour, while contact tracing provides direct suppression through isolation of latent and infectious individuals. Furthermore, rodent population control—through habitat sanitation, trapping, or predator reintroduction—acts as a long-term stabilising mechanism that prevents re- emergence. These findings reinforce the policy need for a multi-sectoral. One Health approach that integrates human, environmental, and veterinary surveillance to sustainably reduce Lassa fever burden in endemic West African settings.

The analytical derivations and numerical experiments jointly provide a coherent picture of Lassa fever dynamics under the influence of behavioural and operational interventions. The equilibrium and sensitivity analyses confirmed the mathematical consistency of the modified SECIRDA–SEI framework, while the numerical simulations illustrated its biological realism and policy relevance. Together, these results bridge theoretical epidemiology and applied public health planning.

Specifically, the reduction of the basic reproduction number (R_0) under incremental in- creases in awareness (φ) and contact tracing (ξ) underscores the complementary nature of behavioural change and surveillance efficiency. The phase portrait and bifurcation

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plots confirm that improved awareness and tracing rates can shift the system from an endemic equilibrium toward a disease-free state. Likewise, the PRCC analysis quantitatively ranked parameter influence, highlighting that interventions targeting transmission pathways and rodent demography yield the highest impact on outbreak containment.

These integrated findings inform practical disease management strategies and set the stage for real-world calibration using empirical data. The next section synthesises these insights into actionable conclusions and policy directions.

Conclusion

This study developed and analysed a modified SECIRDA–SEI compartmental model for

Lassa fever transmission that explicitly incorporates two pivotal intervention mechanisms—public awareness and contact tracing within a two-host (human–rodent) epidemiological system.

By extending the classical SEIR structure, the model accounts for behavioural adaptation, ecological transmission, and the dynamics of post-mortem infectivity, thereby offering a more realistic representation of the disease ecology.

Analytical results derived from the next-generation matrix (NGM) framework established the disease-free equilibrium and provided a closed-form expression for the basic reproduction number, R_0. Both theoretical and numerical analyses confirmed that awareness and contact tracing inversely affect R_0, while corpse-mediated and rodent–human transmission channels exert positive feedback on disease persistence. The sensitivity indices and partial correlation coefficients demonstrated that rodent recruitment (Λ_R) and human–human transmission $\beta_0$are dominant amplifiers of R_0, whereas recovery rate (γ) and natural rodent mortality (μ_R) act as suppressive parameters.

Conceptual simulations further revealed that doubling the awareness rate could reduce R_0 by nearly 40%, while enhanced contact tracing could drive R_0 below unity, indicating epidemic control. The phase portraits and bifurcation diagrams supported the analytical stability conditions, confirming that improved awareness and tracing shift the system toward the disease-free equilibrium. Importantly, these findings validate the dual role of social behaviour and operational capacity in epidemic mitigation, especially in resource-limited settings where pharmaceutical interventions remain scarce.

From a policy standpoint, the results highlight that behavioural education, early case detection, and rodent population control should be pursued concurrently for sustainable disease elimination. Awareness campaigns indirectly lower transmission through behaviour modification, while contact tracing directly isolates latent and infectious individuals. Ecological interventions targeting rodent demography provide an additional buffer that reduces reinfection risk. This triad of behavioural, operational, and ecological strategies aligns with the One Health approach, reinforcing intersectoral collaboration between human and veterinary public health agencies.

Future research will focus on parameter estimation using Nigeria Centre for Disease Control (NCDC) surveillance data, uncertainty quantification, and optimal control formulations to identify cost-effective combinations of interventions. Empirical calibration will also enable the integration of seasonality, environmental variability, and spatial heterogeneity into the model. Such extensions will enhance predictive accuracy and facilitate real-time outbreak response planning.

In summary, the modified SECIRDA–SEI model provides a robust theoretical and computation

framework for understanding and mitigating Lassa fever transmission. By quantifying the interplay between human behaviour, contact tracing efficiency, and ecological factors, this work contributes not only to mathematical epidemiology but also to the formulation of evidence-based public health strategies for endemic West African regions.

Appendix A. Full Jacobian at the Disease-Free Equilibrium

Let the full state vector be

$Z = (S_H, E_H, C_H, I_H, R_H, D_H, A_H, S_R, E_R, I_R)^T$.

Recall the forces of infection:

$\lambda_H = \beta_0 I_H + \beta_1 I_R + \beta_2 D_H$, $\lambda_R = \beta_3 I_H + \beta_5 I_R + \beta_4 D_H$, and define:

$$a_1 = \xi + \mu_H\,, \quad a_2 = \psi + \mu_H\,, \quad a_3 = \gamma + \mu_H + \delta, \quad b_1 = \alpha + \mu_R\,, \quad b_2 = \mu_R\,.$$

At the disease-free equilibrium (DFE) we have

$$S_H^* = \frac{\Lambda_H(\omega+\mu_H)}{\mu_H(\omega+\mu_H+\phi)}, \quad A_H^* = \frac{\phi\Lambda_H}{\mu_H(\omega+\mu_H+\phi)}, \quad S_R^* = \frac{\Lambda_R}{\mu_R}, \quad E_H^* = C_H^* = I_H^* = R_H^* = D_H^* = E_R^* = I_R^*$$

The Jacobian J = $[\partial f_i/\partial z_j]$ of the full 10-dimensional system evaluated at the DFE, in the variable order above, is:

$$J(E_0) = \begin{pmatrix} -(\mu_H+\phi) & 0 & 0 & -\beta_0 S_H^* & 0 & -\beta_2 S_H^* & \omega & 0 & 0 & -\beta_1 S_H^* \\ 0 & -a_1 & 0 & \beta_0 S_H^* & 0 & \beta_2 S_H^* & 0 & 0 & 0 & \beta_1 S_H^* \\ 0 & \xi & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \psi & -a_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\mu_H & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & 0 & -\kappa & 0 & 0 & 0 & 0 \\ \phi & 0 & 0 & 0 & 0 & 0 & -(\mu_H+\omega) & 0 & 0 & 0 \\ 0 & 0 & 0 & -\beta_3 S_R^* & 0 & -\beta_4 S_R^* & 0 & -\mu_R & 0 & -\beta_5 S_R^* \\ 0 & 0 & 0 & \beta_3 S_R^* & 0 & \beta_4 S_R^* & 0 & 0 & -b_1 & \beta_5 S_R^* \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha & -b_2 \end{pmatrix}$$

Notes.

- Row 1 corresponds to $dS_H/dt = \Lambda_H - \lambda_H S_H - (\mu_H + \phi) S_H + \omega A_H$; at the DFE, $\lambda_H = 0$.
- Row 8 corresponds to $dS_R/dt = \Lambda_R - \lambda_R S_R - \mu_R S_R$; at the DFE, $\lambda_R = 0$, so $\partial/\partial S_R = -\mu_R$.
- The block structure is evident if we partition J as $J = \begin{pmatrix} J_{HH} & J_{HR} \\ J_{RH} & J_{RR} \end{pmatrix}$, where $J_{HH} \in R^{7\times7}$ (human-only), $J_{RR} \in R^{3\times3}$ (rodent-only), and the off-diagonal blocks capture cross-species transmission.

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