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Full Length Research Article

OPTIMIZING LASSA FEVER OUTBREAK CONTROL: A COMPARATIVE STUDY ON THE EFFICACY OF CONTACT TRACING INTEGRATION IN EPIDEMIOLOGICAL MODELS

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ABSTRACT
Lassa fever is a contagious viral haemorrhagic disease that remains a major public health concern in West Africa, especially in Nigeria. This paper develops a mathematical epidemiological model that captures the dynamics of Lassa fever spread and estimates the effectiveness of the contact tracing strategy in controlling this disease. The model includes the human-to-rodent interactions and human-to-human transmission. There are two comparative modeling frames: one with contact tracing mechanisms and the other without, which enables assessment of the epidemiological impact. The qualitative properties of the models, such as positivity and boundedness, were analyzed analytically, and the results showed that the models were well-posed. The elimination or persistence of a disease depends on the basic reproduction number (R0), which is calculated by the next-generation matrix method. The findings reveal that the successful application of contact tracing can significantly reduce reproduction numbers and limit the spread of the infection through early recognition and isolation of infected people. Further comparative analysis reveals that models using contact tracing have lower infection prevalence and better results in controlling the outbreak. The results show the relevance of timely contact response and proper surveillance in curbing the spread of Lassa fever. The research will make a useful contribution to policymakers and health officials in society by informing the development of evidence-based interventions and helping prevent the outbreak of Lassa fever.

Keywords: Lassa fever model, Contact tracing, Mathematical epidemiological modeling, Next-generation matrix, Reproduction number

INTRODUCTION

Lassa fever is a viral haemorrhagic illness endemic in several West African countries, with Nigeria bearing the highest burden of the disease (Ajala *et al*, 2025). First identified in the town of Lassa, Nigeria, in 1969, the Lassa virus causes Lassa fever and belongs to the Arenavirus family. The virus is primarily transmitted to humans through contact with food or household items contaminated with the urine or faeces of infected rodents, particularly the multimammate rat (*Mastomys natalensis*). Human-to-human transmission can also occur through direct contact with the blood, urine, faeces, or other bodily fluids of infected individuals, particularly in healthcare settings (Richmond & Baglole, 2003; Grace *et al*., 2021).

Given the zoonotic nature of Lassa fever and its potential for human-to-human transmission, the disease poses significant public health challenges in endemic regions. The World Health Organization (WHO) estimates that between 100,000 and 300,000 infections occur annually, resulting in approximately 5,000 deaths (WHO, 2020). However, these numbers likely underestimate the true burden of the disease due to underreporting and the asymptomatic nature of many infections. Despite its high morbidity and mortality rates, Lassa fever remains a neglected tropical disease, with limited resources dedicated to its control and prevention (Dan-Nwafor *et al*., 2019; Njuguna *et al*., 2022; Aloke *et al*., 2023).

One of the critical epidemiological metrics used to assess the transmission potential of infectious diseases like Lassa fever is the basic reproduction number (R_0). R_0 represents the average number of secondary infections generated by a single infected individual in a completely susceptible population. When R_0 is greater than 1, an outbreak is likely to continue spreading; when it is less than 1, the outbreak will eventually subside. Controlling the spread of Lassa fever, therefore, requires interventions that effectively reduce R_0 to below 1, thereby interrupting transmission and preventing further spread. The spread of the disease across Nigeria is shown in Figure 1 below, while Figure 2 illustrates interactions within the human population (McKendrick *et al*., 2023).

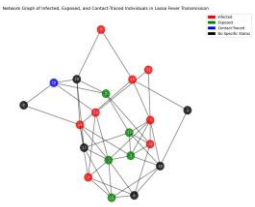

Figure 1: Lassa fever outbreak in Nigeria. **Figure 2**: Schematic Lassa fever transmission

LITERATURE REVIEW

Contact tracing is a cornerstone of public health efforts to control the spread of infectious diseases. It involves identifying individuals who have been in close contact with an infected person and taking appropriate measures, such as testing, isolation, and monitoring, to prevent further transmission. The effectiveness of contact tracing in controlling outbreaks has been demonstrated in various contexts, including Ebola, SARS, and COVID-19 (Hellewell *et al*., 2020; Kucharski *et al*., 2020; Hossain *et al*., 2022). In these cases, rapid and comprehensive contact tracing was critical to containing the outbreaks and reducing the

reproduction number to levels the healthcare system could manage.

Kucharski *et al*. (2020). Ajayi *et al*. (2020) have shown that contact tracing, when effectively implemented, can significantly reduce the reproduction number of infectious diseases by identifying and isolating secondary cases before they can infect others. This is particularly important in the early stages of an outbreak, when the potential for widespread transmission is highest. However, the effectiveness of contact tracing in reducing R_0 is influenced by several factors, including the timeliness of case detection, the speed and coverage of the tracing process, and the compliance of the contacts with isolation and monitoring recommendations (Thomas-Craig *et al*. 2021; Pozo-Martin *et al*., 2023; Leung *et al*., 2024).

Dynamic Modeling of Lassa Fever Transmission

Dynamic modeling is a powerful tool for understanding the transmission dynamics of infectious diseases and evaluating the potential impact of different control strategies. Models can incorporate a range of factors, including transmission rates, contact patterns, and intervention strategies, to simulate the course of an outbreak and predict the effectiveness of various public health measures (Adeniyi *et al*., 2020, 2022, 2023; Metilelu et al., 2022; Oke *et al*., 2023; Adewara & Ajala, 2016). In the case of Lassa fever, dynamic models have been used to explore how different interventions, such as rodent control, vaccination, and contact tracing, might influence the disease's reproduction number and overall transmission dynamics (Althaus *et al*., 2014; Andraud *et al*., 2019; Aloke *et al*., 2023).

One of the key challenges in modelling the transmission of Lassa fever is accounting for the complexity of the disease's epidemiology. Lassa fever transmission involves interactions between human populations, rodent reservoirs, and environmental factors, making it difficult to predict the course of an outbreak and the potential impact of different interventions. Moreover, the asymptomatic nature of many Lassa fever infections complicates efforts to identify and isolate cases, as asymptomatic individuals may still contribute to virus transmission (Suleiman *et al*., 2020; McKendrick *et al*., 2023).

Despite these challenges, several studies have highlighted the potential of contact tracing to reduce R_0 and control Lassa fever outbreaks. Althaus *et al*. (2014) used a stochastic model to assess the impact of various control strategies on the reproduction number of Lassa fever in West Africa. Their findings suggested that contact tracing, when combined with other measures such as improved case detection and isolation, could significantly reduce R_0, thereby helping to contain outbreaks more effectively. Similarly, Andraud *et al.* (2019) applied a compartmental model to simulate Lassa fever outbreaks. They found that incorporating contact tracing significantly reduced the reproduction number, particularly when implemented early in an outbreak.

Empirical Review of Lassa Fever Outbreak

The basic reproduction number, R_0, is a critical parameter in the modeling of infectious diseases, including Lassa fever. It provides a measure of the potential spread of an infection in a population. An R_0 greater than 1 suggests that the infection will likely spread, while an R_0 less than 1 indicates that the infection will eventually die out. Various factors influence R_0, including the virus's transmissibility, the duration of infectiousness, and the population's susceptibility.

Anderson and May's (1991) seminal work on the mathematical modeling of infectious diseases laid the groundwork for understanding the dynamics of R_0 across various contexts, including emerging viral diseases such as Lassa fever. In the context of Lassa fever, studies by Leirs *et al*. (1996) and Fichet-Calvet and Rogers (2009) highlighted the role of environmental and behavioral factors in rodent-to-human transmission, further emphasizing the complexity of calculating and interpreting R_0 in dynamic epidemiological models.

Contact tracing is a key public health intervention used to identify and isolate individuals who have been exposed to an infectious disease, thereby preventing further transmission. The effectiveness of contact tracing in controlling outbreaks has been well documented in various infectious diseases, including Ebola, SARS, and, more recently, COVID-19 (Hellewell *et al*., 2020; Kucharski *et al*., 2020). However, its specific impact on the reproduction number of Lassa fever has been studied less extensively.

The World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) have emphasized contact tracing as an essential tool in outbreak response strategies for Lassa fever, particularly in areas with high transmission rates. Studies by Ilori *et al.* (2019) and Ajayi *et al.* (2020) in Nigeria have demonstrated that timely and effective contact tracing can significantly reduce the spread of Lassa fever. However, challenges such as resource limitations and community resistance often hinder its implementation.

Dynamic models of infectious disease transmission incorporate factors such as contact patterns, transmission rates, and intervention strategies, such as contact tracing, to predict the course of an outbreak and evaluate the potential impact of different control measures. The Susceptible-Infectious-Recovered (SIR) model and its derivatives are commonly used frameworks for such analyses (Kermack & McKendrick, 1927).

Several studies have applied dynamic modeling techniques to Lassa fever, exploring how different interventions might influence the disease's trajectory. A study by Andraud *et al.* (2019) utilized a compartmental model to simulate Lassa fever outbreaks in endemic regions. Their findings indicated that incorporating contact tracing into the model reduced the reproduction number, thereby highlighting its potential effectiveness as a control measure. However, the study also noted the importance of rapid implementation and high coverage rates for contact tracing to achieve its full impact.

While the theoretical benefits of contact tracing are well-established, practical challenges often limit its effectiveness in real-world settings. Factors such as delayed case identification, incomplete contact tracing, and low community engagement can reduce the overall impact of this intervention on the reproduction number (Kucharski *et al*., 2020). In the context of Lassa fever, additional challenges include the asymptomatic nature of many infections, the rural and often inaccessible settings where outbreaks occur, and the presence of other concurrent health issues that strain local public health resources

(Richmond & Baglole, 2003).

Moreover, the effectiveness of contact tracing in reducing R_0 is also contingent on the accuracy and timeliness of data collection, as well as the ability to model the complex interactions between human and rodent populations that drive Lassa fever transmission (Suleiman *et al.*, 2020). As such, dynamic models must account for these uncertainties and incorporate sensitivity analyses to provide robust estimates of the potential impact of contact tracing on Lassa fever's reproduction number. Aloke *et al*. (2023) evaluated the current understanding of Lassa fever (LF), focusing on the transmission, pathogenicity, and treatment options for the Lassa virus (LASV), and examined empirical research on LASV, emphasizing its genetic diversity, which complicates vaccine and therapeutic development due to the virus's ability to evade the immune system. Their analysis indicated that LASV nucleoprotein (NP) is a promising therapeutic target because NP inhibitors could effectively impede viral replication. They suggested the need for comprehensive research into LASV's biochemistry and genetics, improved preventive measures, and the development of novel vaccines. They concluded that targeted approaches and innovative strategies are essential for effective LF management and control.

None of the literature reviewed included contact-traced individuals who were not infected in their model formulations. Also, previous studies did not consider the level of awareness in the progression of infection among humans. Hence, taking into account the numerous dynamic modelling studies conducted on Lassa fever, the current study has identified potential gaps and focuses on addressing them in the existing literature. Given the critical role of R_0 in determining the course of an outbreak and the potential impact of contact tracing on this metric, this study aims to investigate the effectiveness of contact tracing in dynamic modelling of Lassa fever transmission. Specifically, the study will explore how different levels of contact tracing coverage and timeliness influence the reproduction number and overall outbreak dynamics. By developing and analyzing dynamic models that incorporate contact tracing as a key intervention, the study seeks to provide insights into the optimal strategies for controlling Lassa fever and reducing its public health impact.

Dynamic modeling offers a valuable framework for exploring the potential of contact tracing to reduce R_0 and control Lassa fever outbreaks. This study will contribute to the existing body of knowledge by providing a detailed analysis of how contact tracing can be optimized to enhance its effectiveness in reducing the reproduction number and controlling the spread of Lassa fever. Moreover, this study would be beneficial to public health authorities, healthcare workers, communities in endemic regions, and policymakers. Public health agencies benefit from improved models for managing outbreaks, allowing for more effective resource allocation and containment measures. Healthcare workers gain from earlier detection and isolation of cases, reducing their exposure and improving patient outcomes. Communities in West Africa, particularly those most affected by Lassa fever, stand to benefit from reduced transmission and fatalities, enhancing overall public health. Policymakers and governments can implement more efficient public health strategies based on the study's recommendations, thereby optimizing resource use and mitigating the economic burden of outbreaks. Additionally, global health organizations and researchers gain valuable insights into disease modelling and control that can be applied to other viral haemorrhagic fevers and future pandemics, making this research broadly impactful across health systems and academic fields.

MATERIALS AND METHODS

The study uses a deterministic model to analyze the dynamics of Lassa fever transmission, accounting for transmission via the multimammate rat. The model equations are divided into two categories: those without contract tracing and those with it. The material used for this research is secondary data available on the Nigeria Center for Disease Control website.

Assumption of the models

i) Individuals in the population are equally likely to interact with one another.

ii) Rate of interaction is constant in some cases.

iii) Individuals from each compartment die at a per capita death rate μ.

iv) The rodent vectors do not die from the Lassa fever virus.

v) Rodent vector is born susceptible.

vi) The parameters of the models are all strictly positive.

vii) The model assumes all individuals that make up the population are equally likely to become infected if they come in close contact with an infectious person.

viii) Creating awareness on how susceptible individuals could protect themselves is produced by a saturating function that counts on the density of the infected population given $A = \frac{a_0 I}{a_1 + a_2 I}$. This saturating function was used in Adeniyi *et al*. (2020), where *α0, α1, and α2 are the rates* of information growth.

ix) It is not clear from the literature whether Lassa fever confers permanent immunity on recovered humans, so there is no permanent immunity.

x) The transmission rate among rodents is ignored.

xi) The vaccinated individual moves to the recovery compartment.

xii) Vaccinated individuals are completely protected.

xiii) The transmission rate from human to rodent and rodent to human is not the same.

Model Equation with Contract Tracing

The overall human and multimammate rat at any given time 't' are given by $N_H(t)$ & $N_R(t)$. Furthermore, $N_H(t)$ & $N_R(t)$ can be further broken down into $N_H(t) = S_H(t) + E_H(t) + C_H(t)+I_H(t)+R_H(t)$ and $N_R(t) = S_R(t)+E_R(t)+IR(t)$ respectively. Furthermore, $N_H(t)$ & $N_R(t)$ can be further broken down into $N_H(t) = S_H(t) + E_H(t) + I_H(t) + R_H(t)$ and $N_R(t) = S_R(t)+E_R(t)+I_R(t)$, respectively, where $S_H(t)$, $E_H(t)$, $I_H(t)$, $R_H(t)$, $S_R(t)$, $E_R(t)$, $I_R(t)$ denote susceptible human, exposed human, contact traced human, infected human, recovered human, susceptible rat, exposed rat, and infected rat classes, respectively.

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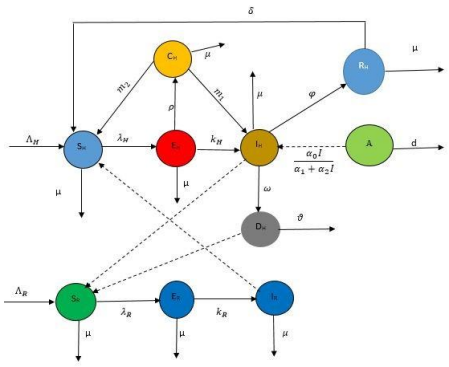

Figure 3: The epidemiological flow for the Lassa fever model with contact tracing

$$\begin{cases} S'_{H*}(t) = \Lambda_H - \mu_H S_H - (\beta_0 I_H + \beta_1 I_R + \beta_3 D_H) S_H + m_2 C_H + \delta R_H . \\ E'_{H.}(t) = \beta_0 I_H S_H + \beta_1 I_R S_H + \beta_3 D_H S_H - (\mu_H + k_H + \rho) E_H \\ C'_H(t) = E_H \rho - C_H(m_1 + m_2 + \mu_H) I_H \\ I'_H(t) = C_H m_1 + k_H E_H - (\mu_H + \varphi + \omega) I_H \\ R'_H(t) = \varphi I_H - (\mu_H + \delta) R_H \\ D'_H(t) = \omega I_H - \vartheta D_H \\ A'_H(t) = \frac{\alpha_0 I_H}{\alpha_1 + \alpha_2 I_H} - dA_H . \\ S_R(t) = \Lambda_H - (\beta_2 I_H + \beta_3 D_H) S_R - \mu_R S_R \\ E'_R(t) = \beta_2 I_H S_R + \beta_3 D_H S_R - (k_R + \mu_R) E_R \\ I'_R(t) = k_R E_R - \mu_R I_R \end{cases} \quad (1)$$

where the initial conditions are: $\{S_H(0) \geq 0, E_H \geq 0, C_H \geq 0, I_H \geq 0, R_H \geq 0, D_H \geq 0, A_H \geq 0, S_R \geq 0, E_R \; \mathfrak{R}^{10}_+$, the set of solutions $\{S_H, E_H, C_H, I_H, R_H, D_H, A_H, S_R, E_R, I_R\}$.

Model Equation without Contract Tracing

The overall populations of human and multimammate rat at any given time 't' are given by
$N_H(t)$ & $N_R(t)$. Furthermore, $N_H(t)$ & $N_R(t)$ can be further broken down into
$N_H(t) = S_{H*}(t) + E_{H*}(t) + I_{H*}(t) + R_{H*}(t)$ and $N_R(t) = S_{R*}(t) + E_{R*}(t) + I_{R*}(t)$ respectively, where

$S_H(t)$, $E_H(t)$, $I_H(t)$, $R_H(t)$, $S_R(t)$, $E_R(t)$, *and* $IR(t)$ *denote the susceptible human, exposed human, infected human, recovered human, susceptible rat, exposed rat,* and infected rat classes, respectively.

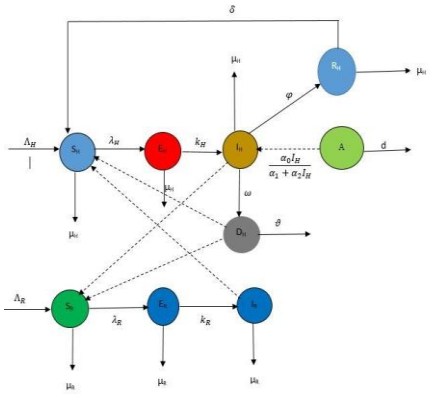

Figure 4: The epidemiological flow for the Lassa fever model without contact tracing

$$\begin{cases} S'_{H*}(t) = \Lambda_H - \mu_H S_{H*} - (\beta_0 I_{H*} + \beta_1 I_{R*} + \beta_3 D_{H*}) S_{H*} + \delta R_{H*} \\ E'_{H.}(t) = \beta_0 I_{H*} S_{H*} + \beta_1 I_{R*} S_{H*} + \beta_3 D_{H*} S_{H*} - (\mu_{H*} + k_H) E_{H*} \\ I'_{H*}(t) = k_H E_{H*} - (\mu + \varphi + \omega) I_{H*} \\ R'_{H_*}(t) = \varphi I_{H*} - (\mu_H + \delta) R_{H*} \\ D'_{H*}(t) = \omega I_{H*} - \vartheta D_{H*} \\ A'_{H*}(t) = \frac{\alpha_0 I_{H*}}{\alpha_1 + \alpha_2 I_{H*}} - dA_{H*} \\ S_{R*}(t) = \Lambda_H - (\beta_2 I_{H*} + \beta_3 D_{H*}) S_{R*} - \mu_R S_{R*} \\ E'_{R*}(t) = \beta_2 I_{H*} S_{R*} + \beta_3 D_{H*} S_{R*} - (k_R + \mu_R) E_{R*} \\ I'_{R*}(t) = k_R E_{R*} - \mu_R I_{R*} \end{cases} \quad (2)$$

where the initial conditions are:
$\{S_{H*} \geq 0, E_{H*} \geq 0, I_{H*} \geq 0, R_{H*} \geq 0, D_{H*} \geq 0, A_{H*} \geq 0, S_{R*} \geq 0, E_{R*} \geq 0, IR_* \geq 0\} \in \mathfrak{R}^9_+$, the solution set $\{SH*, EH*, IH*, RH*, DH*, AH*, SR*, ER*, IR*\}$.

It is also assumed that all the parameters in both models are non-negative. The description of the variables and parameters is given in Tables 1 and 2 below:

Table 1: Description of the variables used in the models

Variable	Description
S_H (t)	Susceptible human at time "t".
E_H (t)	Exposed humans at time "t".
C_H (t)	Contact-traced human at time "t". Infectious Humans
I_H (t)	at time "t".
D_H (t)	Dead Humans at time "t".
R_H (t)	Recovered Humans at time "t".
A_H (t)	Level of Awareness of the Disease at time "t". Susceptible Rodents at
S_R (t)	time "t".
E_R (t)	Exposed Rodents at time "t". Infectious
I_R (t)	Rodents at time "t".

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Table 2: Description of the parameters used in the models

Parameter	Description
k_H	Rate of progression from exposed to infected class.
k_R	Rate of progression of the exposed rodents to the infected class.
P	Proportion of contact-traced exposed individuals.
m_1	Proportion of contact-traced individuals infected with the virus.
m_2	Proportion of contact-traced individuals who are not infected and returned to S_H.
Δ	Loss of immunity
Ω	Death of a human due to Lassa fever.
μ_R	Natural death of the rodent host
Θ	Burial rate of Lassa fever-induced corpse.
Φ	Recovery rate of humans from Lassa fever.
μ	Natural death of the human host
D	Rate at which information degenerates
α_0	Information growth rate.
α_1	Half saturation point
α_2	Saturation constant of information.
β_0	Rate of transmission by infected humans to susceptible humans
β_1	Transmission rate from infected rodents to susceptible humans.
β_2	Transmission rate from infected humans to susceptible rodents.
β_3	Transmission rate of Lassa fever-induced dead bodies.
Λ_H	Human recruitment rate
Λ_R	Vector recruitment rate

Model Analysis

In this section, the emphasis is on the stability analysis of the model, considering the positivity and boundedness of solutions, the model's steady states, and their stability.

Positivity and boundedness of solutions

In this part, we show that the model systems (1 & 2) are epidemiologically well-posed and realistic if all variables in systems (1 & 2) are non-negative for all time t. Thus, we show this through the Lemma stated below:

Lemma 1. The solution $S_H(t)$, $E_H(t)$, $C_H(t)$, $I_H(t)$, $R_H(t)$, $D_H(t)$, $A_H(t)$, $S_R(t)$, $E_R(t)$, $I_H(t)$ of system (1) with initial conditions $S_H(0) > 0$, $E_H(0) \geq 0$, $C_H(0) \geq 0$, $I_H(0) \geq 0$, $R_H(0) \geq 0$, $D_H(0) \geq 0$, $A_H(0) \geq 0$, $S_R(0) > 0$, $E_R(0) \geq 0$, & $I_R(0) \geq 0$ and $S_{H*}(t)$, $E_{H*}(t)$, $I_{H*}(t)$, $R_{H*}(t)$, $D_{H*}(t)$, $A_{H*}(t)$, $S_{R*}(t)$, $E_{R*}(t)$, $I_{H*}(t)$ of system (2) with initial conditions $S_{H*}(0) > 0$, $E_{H*}(0) \geq 0$, $I_{H*}(0) \geq 0$, $R(0)_{H*} \geq 0$, $D_{H*}(0) \geq 0$, $A_{H*}(0) \geq 0$, $S_{R*}(0) > 0$, $E_{R*}(0) \geq 0$, & $I_{R*}(0) \geq 0$ are positive for all $t > 0$.

Proof: The total population of humans and rodents in Eqn. 1 is given as:

$$N_H = S_H(t) + E_H(t) + C_H(t) + I_H(t) + R_H(t); N_R = S_R(t) + E_R(t) + I_R(t)$$

$$\frac{\delta N_H}{\delta t} = \frac{\delta S_H}{\delta t} + \frac{\delta E_H}{\delta t} + \frac{\delta C_H}{\delta t} + \frac{\delta I_H}{\delta t} + \frac{\delta R_H}{\delta t}; \frac{\delta N_R}{\delta t} = \frac{\delta S_R}{\delta t} + \frac{\delta E_R}{\delta t} + \frac{\delta I_R}{\delta t}$$

$$\frac{\delta N_H}{\delta t} = \Lambda_H - \mu_H N_H - \omega I_H; \frac{\delta N_R}{\delta t} = \Lambda_R - \mu_R N_R$$

When infection does not exist, then $\omega I_H = 0$, we have:

$$\frac{\delta N_H}{\delta t} \leq \Lambda_H - \mu_H N_H - \omega I_H; \frac{\delta N_R}{\delta t} \leq \Lambda_R - \mu_R N_R$$

Integrating the above equation using the integrating factor $e^{\int \mu_H dt} = e^{\mu_H t}$ for human population and $e^{\int \mu_R dt} = e^{\mu_R t}$ for rodent population gives:

$$e^{\mu_H t}\frac{\delta N_H}{\delta t} + \mu_H N_H e^{\mu_H t} = \Lambda_H e^{\mu_H t};\ e^{\mu_R t}\frac{\delta N_R}{\delta t} + \mu_R N_R e^{\mu_R t} = \Lambda_R e^{\mu_R t}$$

$$\int \frac{d}{dt}(N_H e^{\mu_H t}) \leq \int (\Lambda_H\ e^{\mu_H t})dt; \int \frac{d}{dt}(N_R e^{\mu_R t}) \leq \int (\Lambda_R\ e^{\mu_R t})dt$$

By integrating the above gives:

$$(N_H e^{\mu_H t}) \leq \frac{\Lambda_H}{\mu_H}(e^{\mu_H t}) + C_1; (N_R e^{\mu_R t}) \leq \frac{\Lambda_R}{\mu_R}(e^{\mu_R t}) + C_2;$$

$$N_H(t) \leq \frac{\Lambda_H}{\mu_H} + C_1 e^{-\mu_H t}; N_R(t) \leq \frac{\Lambda_R}{\mu_R} + C_2 e^{-\mu_R t};$$

$$\lim_{t\to\infty} N_H(t) \leq \lim_{t\to\infty}\left(\frac{\Lambda_H}{\mu_H} + C_1 e^{-\mu_H t}\right); \lim_{t\to\infty} N_R(t) \leq \lim_{t\to\infty}\left(\frac{\Lambda_R}{\mu_R} + C_2 e^{-\mu_R t}\right);$$

Same principle applies to equation (2) hence, the model (Equations 1 & 2) will be analyzed in the region:

$$\Omega_1 = \left\{\left(S_H, E_H, C_H, I_H, R_H, D_H, A_H, S_R, E_R, I_R \in R_+^{10}: N_H(t) \leq \frac{\Lambda_H}{\mu_H}, A_H(t) \leq \frac{\alpha_0\Lambda_H}{d(\alpha_1\mu_H + \alpha_2\Lambda_H)}, N_R(t) \leq \frac{\Lambda_R}{\mu_R}\right)\right\}$$

$$\Omega_2 = \{(S_H, E_H, I_H, R_H, D_H, A_H, S_H, E_H, I_H \in R_+^9: N_{H*}(t) \leq \frac{\Lambda_H}{\mu_H}, A_H(t) \leq \frac{\alpha_0\Lambda_R}{d(\alpha_1\mu_R + \alpha_2\Lambda_R)}, N_{R*}(t) \leq \frac{\Lambda_R}{\mu_R}\right)\}$$

Thus, in these regions, systems (Equations 1 and 2) are mathematically and epidemiologically well posed.

Equilibrium point of Model 1

Considering equation (1) which is non-linear, the existence of all the equilibriums of the model can be investigated by making the rate of change with respect to time 't' of all the dynamical variables equal zero, that is:

$$\frac{\delta S_H}{\delta t} = \frac{\delta E_H}{\delta t} = \frac{\delta C_H}{\delta t} = \frac{\delta I_H}{\delta t} = \frac{\delta R_H}{\delta t} = \frac{\delta D_H}{\delta t} = \frac{\delta A_H}{\delta t} = \frac{\delta S_R}{\delta t} = \frac{\delta E_R}{\delta t} = \frac{\delta I_R}{\delta t} = 0$$

$$\Lambda_H - (\mu_H + \lambda_H)S_H + m_2 C_H + \delta R_H = 0 \quad (3)$$

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$$\lambda_H S_H - A_0 E_H = 0 \quad (4)$$
$$\rho E_H - A_l C_H = 0 \quad (5)$$
$$m_1 C_H + k_H E_H - A_2 I_H = 0 \quad (6)$$
$$\varphi I_H - A_3 R_H = 0 \quad (7)$$
$$\omega I_H - \vartheta D_H = 0 \quad (8)$$
$$\frac{a_0 I_H}{a_1 + a_2 I_H} - d A_H = 0 \quad (9)$$
$$\Lambda_R - (\mu_R + \lambda_R) S_R = 0 \quad (10)$$
$$\lambda_R S_R = A_4 E_R = 0 \quad (11)$$
$$k_R E_R - \mu_R I_R = 0 \quad (12)$$

The equilibrium points of equations (1) can be obtain by solving the set of algebraic equations (3)-(12) simultaneously, thus gives:

$$\begin{cases} S_H = \frac{A_0 A_1 A_2 A_3 \Lambda_H}{A_7 \lambda_H + A_8} \\ E_H = \frac{A_1 A_2 A_3 \Lambda_H \lambda_H}{A_7 \lambda_H + A_8} \\ C_H = \frac{\rho A_2 A_3 \Lambda_H \lambda_H}{A_7 \lambda_H + A_8} \\ I_H = \frac{A_5 A_3 \Lambda_H \lambda_H}{A_7 \lambda_H + A_8} \\ R_H = \frac{\varphi A_5 A_3 \Lambda_H \lambda_H}{A_7 \lambda_H + A_8} \\ D_H = \frac{\omega A_5 A_3 \Lambda_H \lambda_H}{\vartheta (A_7 \lambda_H + A_8)} \\ A_H = \frac{\alpha_0 A_5 A_3 \Lambda_H \lambda_H}{d(\alpha_1 [A_7 \lambda_H + A_8] + \alpha_2 A_5 A_3 \Lambda_H \lambda_H)} \\ S_R = \frac{\Lambda_R}{\mu_R + \lambda_R} \\ E_R = \frac{\Lambda_R \lambda_R}{A_4 (\mu_R + \lambda_R)} \\ I_R = \frac{K_R \Lambda_R \lambda_R}{A_4 \mu_R (\mu_R + \lambda_R)} \end{cases} \quad (13)$$

where:

$A_0 = \mu_H + \rho + k_H$; $A_1 = m_1 + m_2 + \mu_H$; $A_2 = \mu_H + \varphi + \omega$; $A_3 = \mu + \delta$; $A_4 = k_R + \mu_R$;

$A5 = \rho m1 + A1kH$

In order to obtain the unique equilibrium points, we recall that the force of infection for rodents is given as:

$$\lambda_R = \beta_2 I_{II} + \beta_3 D_H \quad (14)$$

The solution of Eq.(14) for λ_R gives:

Case I

$$\lambda_R = 0 \text{ or } \lambda_R = \frac{A_9 k_6}{A_7 (k_6 + 2A_8 A_{12})} \quad (15)$$

Likewise, the force of infection for humans is given as:

$$\lambda_H = \beta_0 I_H + \beta_1 I_R + \beta_3 D_H \quad (16)$$

The solution of Eq.(16) for λ_H gives:

Case II

$$\lambda_H = 0 \; or \; \lambda_H = \frac{k_4 + \sqrt{k_4^2 + 4A_8^2 \mu_R (R_0^2 - 1) A_7 A_{12}}}{2A_7 A_{12}} \quad (17)$$

From solutions (15 & 17) the following cases arise:

When $\lambda_H = 0$ & $\lambda_R = 0$, the Lassa fever free equilibrium (LFFE) is established, denoted by L_0 which when substituted in the general equilibrium point, gives: $L_e = \left(\frac{\Lambda_H}{\mu_H}, 0,0,0,0,0, \frac{\Lambda_R}{\mu_R}, 0,0\right)$. This is when there is no Lassa fever infection and there is no need for medication.

When $\lambda_H = \frac{k_4 + \sqrt{k_4^2 + 4A_8^2 \mu_R (R_0^2 - 1) A_7 A_{12}}}{2A_7 A_{12}}$ & $\lambda_R = \frac{A_9 k_6}{A_7 (k_6 + 2A_8 A_{12})}$ The Lassa fever endemic equilibrium (LFEE), denoted by L^* is represented by: $L^*(S_H^*, E_H^*, C_H^*, I_H^*, R_H^*, D_H^*, A_H^*, S_R^*, E_R^*, I_R^*)$.

Hence, the following were obtained after substitutions were made:

$$\begin{cases} S_H^* = \frac{2A_0 A_1 A_2 A_3 A_{12} \Lambda_H}{2A_8 A_{12} + k_6}. \\ E_H^* = \frac{A_1 A_2 A_3 \Lambda_H k_6}{A_7 [2A_8 A_{12} + k_6]}. \\ C_H^* = \frac{\rho A_2 A_3 \Lambda_H k_6}{A_7 [2A_8 A_{12} + k_6]}. \\ I_H^* = \frac{A_3 A_5 \Lambda_H k_6}{A_7 [2A_8 A_{12} + k_6]}. \\ R_H^* = \frac{\varphi A_3 A_5 \Lambda_H k_6}{A_7 [2A_8 A_{12} + k_6]}. \\ D_H^* = \frac{\omega A_3 A_5 \Lambda_H k_6}{A_7 \vartheta [2A_8 A_{12} + k_6]}. \\ A_H^* = \frac{\alpha_0 A_3 A_5 \Lambda_H k_6}{2d[A_7 \alpha_1 (k_6 + 2A_8 A_{12}) + \alpha_2 A_3 A_5 \Lambda_H k_6]}. \\ S_R^* = \frac{\Lambda_R A_7 [2A_8 A_{12} + k_6]}{A_7 \mu_R [2A_8 A_{12} + k_6] + A_9 k_6}. \\ E_R^* = \frac{\Lambda_R A_9 k_6}{A_4 A_7 \mu_R [2A_8 A_{12} + k_6] + A_4 A_9 k_6)}. \\ I_R^* = \frac{K_R \Lambda_R A_9 k_6}{A_4 \mu_R [A_7 \mu_R (2A_8 A_{12} + k_6) + A_9 k_6]}. \end{cases} \quad (18)$$

Therefore, L^* above represents the presence of the Lassa fever virus in the population. To justify the above as an endemic equilibrium point, it is important to ensure that all components are positive. Thus, this is only possible if and only if $\lambda_H > 0$ and $\lambda_R > 0$. Epidemiologically, the presence of LFEE indicates that at least one of the model's infected classes is non-empty, meaning that Lassa fever is in circulation in the population.

The Reproduction Number for the Model

The expected number of infections an individual in the population will generate when the entire population is susceptible to infection can be obtained using the next-generation matrix. The expected number of secondary infections produced by an infected individual in a population of susceptible individuals is given by: $G = FV^{-1}$. $R_0 = \rho(FV^{-1})$ of the system of equations (2), where $\rho(G)$ denotes the spectral radius.

Definition 3.1: The *spectral radius* of matrix G is defined as the maximum of the absolute values of the eigenvalues of G:
$\rho(G) = sup\{|\lambda| : \lambda \epsilon \sigma(G)\}$, where $\sigma(G)$ denotes the set of eigenvalues of G.

The Reproduction Number For Model 1

The reproduction number, R_o, indicates the potential for an infection to spread or decline in Nigeria and how rapidly it can be transmitted. The higher the R_o, the more likely the disease is to become an epidemic. So, we have the following expression:

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta_0 \Lambda_H}{\mu_H} & \frac{\beta_3 \Lambda_H}{\mu_H} & 0 & \frac{\beta_1 \Lambda_H}{\mu_H} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 \Lambda_R}{\mu_R} & \frac{\beta_3 \Lambda_R}{\mu_R} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}; \; V$$
$$= \begin{pmatrix} A_0 & 0 & 0 & 0 & 0 & 0 \\ -\rho & A_1 & 0 & 0 & 0 & 0 \\ -k_H & -m_1 & A_2 & 0 & 0 & 0 \\ 0 & 0 & -\omega & \vartheta & 0 & 0 \\ 0 & 0 & 0 & 0 & A_4 & 0 \\ 0 & 0 & 0 & 0 & -k_R & \mu_R \end{pmatrix}$$

Using Maple (2016) software, the inverse of the above matrix (V^{-1}) is obtained as:

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$$V^{-1} = \begin{pmatrix} \frac{1}{A_0} & 0 & 0 & 0 & 0 & 0 \\ \frac{\rho}{A_0A_1} & \frac{1}{A_1} & 0 & 0 & 0 & 0 \\ \frac{\rho m_1 + A_1k_H}{A_0A_1A_2} & \frac{m_1}{A_1A_2} & \frac{1}{A_2} & 0 & 0 & 0 \\ \frac{\omega(\rho m_1 + A_1k_H)}{\vartheta A_0A_1A_2} & \frac{\omega m_1}{\vartheta A_1A_2} & \frac{\omega}{\vartheta A_2} & \frac{1}{\vartheta} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{A_4} & 0 \\ 0 & 0 & 0 & 0 & \frac{k_R}{A_4\mu_R} & \frac{1}{\mu_R} \end{pmatrix}$$

Hence, the product of $G = FV^{-1}$ gives the reproduction number, where F and V are the rates of new infections and transitions near the equilibrium. The dominant eigenvalue of G is the basic reproduction number denoted by R_0, and it is obtained as $|G - \lambda I| = 0$ where I is the identity matrix.

$$R_0 = \frac{\frac{\Lambda_H A_5(\vartheta\beta_0+\omega\beta_3)}{\mu_H\vartheta A_0A_1A_2} + \sqrt{\left(\frac{\Lambda_H A_5(\vartheta\beta_0+\omega\beta_3)}{\mu_H\vartheta A_0A_1A_2}\right)^2 + 4\left[\left(\frac{\Lambda_R A_5(\vartheta\beta_2+\omega\beta_3)}{\mu_R\vartheta A_0A_1A_2}\right)\left(\frac{\Lambda_H\beta_1k_R}{A_4\mu_R\mu_H}\right)\right]}}{2} \quad (19)$$

For $R_0 < 1$, it implies that $R_{0(\mathrm{hh})} + \sqrt{R_{0(\mathrm{hh})}^2 + 4R_{0(\mathrm{hr})}R_{0(\mathrm{rh})}}$ must be less than 2 where:

$R_{0(hh)} = \frac{\Lambda_H A_5(\vartheta\beta_0+\omega\beta_3)}{\mu_H\vartheta A_0A_1A_2}, R_{0(hr)} = \frac{\Lambda_R A_5(\vartheta\beta_2+\omega\beta_3)}{\mu_R\vartheta A_0A_1A_2}$ *and* $R_{0(rh)} = \frac{\Lambda_H\beta_1k_R}{A_4\mu_R\mu_H}$ represent the reproduction number of human to human, human to rodent and rodent to human respectively. Hence, the reproduction number in (19) can be rewritten as: given as;

$$\lambda_R = \beta_2 I_H + \beta_3 D_H$$

Thus gives:

case 3: $\lambda_R = 0$ & $\lambda_R = \frac{\lambda_9 k_6^*}{A_7^*(k_6^*+2A_8^*A_{12}^*)}$, *Also,* in order to obtain the unique equilibrium points, we recall the force of infection for human is given as: $\lambda H = \beta 0 IH + \beta 1 IR + \beta 3 DH$.

Thus it is established that:

$$\lambda_H = 0 \text{ or } \lambda_H = \frac{k_4^* + \sqrt{k_4^{*(2)} + 4A_8^{*(2)}\mu_R(R_0^2-1)A_7^*A_{i2}^*}}{2.A_7^*A_{i2}^*} \quad (22)$$

From solution (22) the following case arises:

Case 4: $\lambda_H = 0$ or $\lambda_H = \frac{k_4^* + \sqrt{k_4^{*(2)} + 4A_8^{*(2)}\mu_R(R_0^2-1)A_7^*A_{i2}^*}}{2.A_7^*A_{i2}^*}$

In cases 3 & 4:

When $\lambda_H = 0$ & $\lambda_R = 0$, the Lassa fever free equilibrium (LFFE), denoted by L_0, gives:

$L_e = \left(\frac{\Lambda_H}{\mu_H}, 0,0,0,0,0, \frac{\Lambda_R}{\mu_R}, 0,0\right)$

This is when there is no Lassa fever infection and no need for medication.

When $\lambda_H = \frac{k_4^* + \sqrt{k_4^{*(2)} + 4A_8^{*(2)}\mu_R(R_0^2-1)A_7^*A_{i2}^*}}{2.A_7^*A_{i2}^*}$. & $\lambda_R = \frac{\lambda_9 k_6^*}{A_7^*(k_6^*+2A_8^*A_{12}^*)}$, the Lassa fever endemic equilibrium (LFEE), denoted by L^* is represented by: $L^*(S_{H*}^*, E_{H*}^*, I_{H*}^*, R_{H*}^*, D_{H*}^*, A_{H*}^*, S_{R*}^*, E_{R*}^*, I_{R*}^*)$.

Hence, the following were obtained after the substitution was made:

$$R_0 = \frac{R_{0(hh)} + \sqrt{R_{0(hh)}^2 + 4R_{0(hr)}R_{0(rh)}}}{2}$$

Equilibrium Point of the Model 2

Considering equation (2) which is non-linear, the existence of all the equilibriums of the model can be investigated by making the rate of change with respect to time 't' of all the dynamical variables equal zero, that is:

$$\frac{\delta S_{H*}}{\delta t} = \frac{\delta E_{H*}}{\delta t} = \frac{\delta I_{H*}}{\delta t} = \frac{\delta R_{H*}}{\delta t} = \frac{\delta D_{H*}}{\delta t} = \frac{\delta A_{H*}}{\delta t} = \frac{\delta S_{R*}}{\delta t} = \frac{\delta E_{R*}}{\delta t} = \frac{\delta I_{R*}}{\delta t} = 0$$

The equilibrium points of equations (2) can be obtained by equating Eq. (2) to zero and solving the set of algebraic equations simultaneously, which gives:

$$\begin{cases} S_{H*} = \frac{(\mu_H+k_H)\mu_H A_2A_3\Lambda_H}{A_7\lambda_H+A_8} \\ E_{H*} = \frac{\mu_H A_2A_3\Lambda_H\lambda_H}{A_7\lambda_H+A_8} \\ I_{H*} = \frac{\mu_H k_H A_3\Lambda_H\lambda_H}{A_7\lambda_H+A_8} \\ R_{H*} = \frac{\mu_H k_H\varphi A_3\Lambda_H\lambda_H}{A_7\lambda_H+A_8} \\ D_{H*} = \frac{\omega\mu_H k_H A_3\Lambda_H\lambda_H}{\vartheta(A_7\lambda_H+A_8)} \\ A_{H*} = \frac{\alpha_0\mu_H k_H A_3\Lambda_H\lambda_H}{d(\alpha_1[A_7\lambda_H+A_8]+\alpha_2\mu_H k_H A_3\Lambda_H\lambda_H)} \\ S_{R*} = \frac{\Lambda_R}{\mu_R+\lambda_R} \\ E_{R*} = \frac{\Lambda_R\lambda_R}{A_4(\mu_R+\lambda_R)} \\ I_{R*} = \frac{K_R\Lambda_R\lambda_R}{A_4\mu_R(\mu_R+\lambda_R)} \end{cases} \quad (20)$$

In order to obtain the unique equilibrium points, we recall that the force of infection for rodents is

$$\lambda_R = 0 \text{ or } \lambda_R = \frac{\lambda_9 k_6^*}{A_7^*(k_6^*+2A_8^*A_{12}^*)} \quad (21)$$

From solution (21), the following case arises:

$$\begin{cases} S_{H*}^* = \frac{2(\mu_H+k_H)\mu_H A_2A_3A_{12}^*\Lambda_H}{2A_8^*A_{12}^*+k_6^*} \\ E_{H*}^* = \frac{\mu_H A_2A_3k_6^*\Lambda_H}{A_7^*[2A_8^*A_{12}^*+k_6^*]} \\ I_{H*}^* = \frac{\mu_H k_H A_3k_6^*\Lambda_H}{A_7^*[2A_8^*A_{12}^*+k_6^*]} \\ R_{H*}^* = \frac{\varphi\mu_H k_H A_3k_6^*\Lambda_H}{A_7^*[2A_8^*A_{12}^*+k_6^*]} \\ D_{H*}^* = \frac{\omega\mu_H k_H A_3k_6^*\Lambda_H}{A_7^*[2A_8^*A_{12}^*+k_6^*]} \\ A_{H*}^* = \frac{\alpha_0\varphi\mu_H k_H A_3k_6^*\Lambda_H}{2d[A_7^*\alpha_1(k_6^*+2A_8^*A_{12}^*+k_6^*)+\alpha_2\mu_H k_H A_3k_6^*\Lambda_H]} \\ S_{R*}^* = \frac{A_7^*[2A_8^*A_{12}^*+k_6^*]\Lambda_R}{A_7^*[2A_8^*A_{12}^*+k_6^*]\mu_R+A_9^*k_6^*} \\ E_{R*}^* = \frac{k_6^*A_9^*\Lambda_R}{A_4(A_7^*[2A_8^*A_{12}^*+k_6^*]\mu_R+A_9^*k_6^*])} \\ I_{R*}^* = \frac{k_6^*A_9^*k_R\Lambda_R}{A_4\mu_R[A_7^*[2A_8^*A_{12}^*+k_6^*]\mu_R+A_9^*k_6^*]} \end{cases} \quad (23)$$

Therefore, L^* above represents the presence of the Lassa fever virus in the population. To justify the above as an endemic equilibrium point, it is important to ensure that all components are positive. Thus, this is only possible if and only if $\lambda_H > 0$ and $\lambda_R > 0$.

Epidemiologically, the presence of LFEE indicates that at least one of the model's infected classes is non-empty, meaning that Lassa fever is in circulation in the population.

where: $A_0 = \mu_H + \rho + k_H$; $A_1 = m_1 + m_2 + \mu_H$; $A_2 = \mu_H + \varphi + \omega$; $A_3 = \mu_H + \delta$; $A_4 = k_R + \mu_R$;

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$A_5 = \rho m_1 + A_1 k_H$: $A_6^* = \frac{A_2(\mu_H + k_H)}{k_H}$; $A_7^* = A_2 A_3(\mu_H + k_H)\mu_H - \delta\varphi\mu_H k_H$;
$A_8^* = A_2 A_3 \mu_H^2(\mu_H + k_H)$; $A_9^* = \frac{A_3\mu_H k_H \Lambda_H(\vartheta\beta_2 + \omega\beta_3)}{\vartheta}$; $A_{10}^* = \frac{A_3\mu_H k_H \Lambda_H(\vartheta\beta_0 + \omega\beta_3)}{\vartheta}$;
$A_{11}^* = \frac{\beta_1 k_R \Lambda_R}{A_4\mu_R}$; $A_{12}^* = A_9^* + A_7^*\mu_R$; $k_4^* = (R_0(h) - 1)A_8^* A_{12}^* + [R_0(hr)R_0(rh) - 1]\, A_7^* A_8^* \mu_R$
$k_5^* = A_8^* A_{10}^* \mu_R + A_8^* A_9^* A_{11}^* - A_8^{*(2)}\mu_R$; $K_6^* = K_4^* + \sqrt{k_4^{*(2)} + 4A_8^{*(2)}\mu_R(R_0^2 - 1)A_7^* A_{12}^*}$

The Reproduction Number For Model 2

The reproduction number for model 2 can be obtained as follows:

Hence, the product of $G = FV^{-1}$ gives the reproduction number, where F and V are the rates of new infections and transitions near the equilibrium. The dominant eigenvalue of G is the basic reproduction number denoted by R_0, and it is obtained as $|G - \lambda I| = 0$ where I is the identity matrix.

$$F = \begin{pmatrix} 0 & \frac{\beta_0\Lambda_H}{\mu_H} & \frac{\beta_3\Lambda_H}{\mu_H} & 0 & \frac{\beta_1\Lambda_H}{\mu_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2\Lambda_R}{\mu_R} & \frac{\beta_3\Lambda_R}{\mu_R} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}; \; V = \begin{pmatrix} \mu_H + k_H & 0 & 0 & 0 & 0 \\ -k_H & A_2 & 0 & 0 & 0 \\ 0 & -\omega & \vartheta & 0 & 0 \\ 0 & 0 & 0 & A_4 & 0 \\ 0 & 0 & 0 & -k_R & \mu_R \end{pmatrix}$$

Using Maple (2016) software, the inverse of the above matrix (V^{-1}) is obtained as:

$$V^{-1} = \begin{pmatrix} \frac{1}{(\mu_H + k_H)} & 0 & 0 & 0 & 0 \\ \frac{k_H}{(\mu_H + k_H)A_2} & \frac{1}{A_2} & 0 & 0 & 0 \\ \frac{\omega k_H}{\vartheta(\mu_H + k_H)A_2} & \frac{\omega}{\vartheta A_2} & \frac{1}{\vartheta} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{A_4} & 0 \\ 0 & 0 & 0 & \frac{k_R}{A_4\mu_R} & \frac{1}{\mu_R} \end{pmatrix}$$

Hence, the product of $G = FV^{-1}$ gives the reproduction number, where F and V are the rates of new infectices and transitions newr the equilibrium. The dominant eigenvalue of G is the basic reproduction number devoted by R_0, and it is obtained as $|G - \lambda/| = 0$ where 1 is the identity matrix.

$$R_0 = \frac{\frac{\Lambda_H k_H(\vartheta\beta_0 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)} + \sqrt{\left(\frac{\Lambda_H k_H(\vartheta\beta_0 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)}\right)^2 + 4\left[\left(\frac{\Lambda_R k_H(\vartheta\beta_2 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)}\right)\left(\frac{\Lambda_H\beta_1 k_R}{A_4\mu_R\mu_H}\right)\right]}}{2} \quad (24)$$

For $R_0 < 1$, it implies that $R_{0(\mathrm{hh})} + \sqrt{R_{0(\mathrm{hh})}^2 + 4R_{0(\mathrm{hr})}R_{0(\mathrm{rh})}}$ must be less than 2 where:

$R_{0(hh)} = \frac{\Lambda_H k_H(\vartheta\beta_0 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)}$, $R_{0(hr)} = \frac{\Lambda_R k_H(\vartheta\beta_2 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)}$ *and* $R_{0(rh)} = \frac{\Lambda_H\beta_1 k_R}{A_4\mu_R\mu_H}$ represent the reproduction number of human to human, human to rodent and rodent to human respectively. Hence, the reproduction number in (24) can be rewritten as:

$$R_0 = \frac{\Lambda_R k_H(\vartheta\beta_2 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)} + \left(\frac{\Lambda_R k_H(\vartheta\beta_2 + \omega\beta_3)}{\mu_H\vartheta A_2(\mu_H + k_H)}\right)\left(\frac{\Lambda_H\beta_1 k_R}{A_4\mu_R\mu_H}\right)$$

Parameters Estimation and Data Presentation

In this section, mathematical models are used to estimate some parameters from the available data.

i) The natural mortality rate of humans, denoted by μ_H, can be obtained using the expression:

$$\hat{\mu_H} = \frac{1}{\mu_o}, \quad (25)$$

where μ_0 = 60.48 years, indicating the average life expectancy in Nigeria.

ii) Similarly, the death rate of rodents, μ_R, can be determined as:

$$\hat{\mu_R} = \frac{1}{\mu_o^*}, \quad (26)$$

where μ^*_0 = 1 year, the lifespan of rodents.

iii) The progression rate from the exposed class to the infected class, k_H, is approximated by:

$$\hat{k_H} = \frac{\sum_{t=1}^{n} I_H(t)}{\sum_{t=1}^{n}(E_H(t) - \mu_H E_H(t))}, \quad (27)$$

with the associated confidence interval:

$$\hat{k_H} \pm 1.96\sqrt{\hat{k_H}(1 - \hat{k_H})}.$$

iv) The recovery rate φ of infected humans is given as:

$$\hat{\varphi} = \frac{\sum_{t=1}^{n} R_H(t)}{\sum_{t=1}^{n}(I_H(t) - \mu_H I_H(t))}, \quad (28)$$

with the associated confidence interval:

$$\hat{\varphi} \pm 1.96\sqrt{\frac{\hat{\varphi}(1 - \hat{\varphi})}{I_H(t)}}.$$

v) The Lassa fever-induced mortality rate among infected individuals, ω, can be expressed as:

$$\hat{\omega} = \frac{\sum_{t=1}^{n} D_H(t)}{\sum_{t=1}^{n}(I_H(t) - \mu_H I_H(t))}, \quad (29)$$

$$\hat{\omega} \pm 1.96\sqrt{\frac{\hat{\omega}(1 - \hat{\omega})}{I_H(t)}}.$$

with the associated confidence interval:

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Here, D_H (t) denotes the number of Lassa fever-related deaths at time t in weeks, for t = 1, 2, . . . , n.

vi) The ρ of a contact-traced human exposed to the disease is given by:

$$\hat{\rho} = \frac{\sum_{t=1}^{n} C_H(t)}{\sum_{t=1}^{n} (E_H(t) - \mu_H E_H(t))}, \quad (30)$$

with the associated confidence interval:

$$\hat{\rho} \pm 1.96\sqrt{\frac{\hat{\rho}(1-\hat{\rho})}{E_H(t)}}.$$

vii) The m_1 contact-traced human who became infectious is given by:

$$\hat{m_1} = \frac{\sum_{t=1}^{n} I_t}{\sum_{t=1}^{n} (C_H(t) - \mu_H C_H(t))}, \quad (31)$$

with confidence interval:

$$\hat{m_1} \pm 1.96\sqrt{\frac{\hat{m_1}(1-\hat{m_1})}{C_H(t)}}.$$

viii) The contact-traced humans who were uninfected and moved to the susceptible class, m_2, is determined as:

$$\hat{m}_2 = 1 - \left(\frac{\sum_{t=1}^{n} I_t}{\sum_{t=1}^{n} (C_H(t) - \mu_H C_H(t))}\right). \quad (32)$$

The results generated by the system of ordinary differential equations (ODEs) using the optimal parameters found by the generic algorithm (GA) after several iterations, and other parameters that were fixed for the model. The differential equations (DEs were solved numerically using Runge Kutta ODE45 for the first week, 2021 with the following initial conditions $S_H(0) = 6000, C_H(0) = 7, E_H(0) = 109, I_H(0) = 9, D_H(0) = 2, R_H(0) = 7, A = 0.01, S_R(0) = 500, E_R(0) = 5, I_R(0) = 2$ used. The principal objective was to demonstrate the model's ability to fit the reported number of Lassa fever cases in Nigeria. The parameter values obtained by GA are shown in Table 3, and Figure 5 illustrates the estimated cumulative number of Lass fever cases and the model fit.

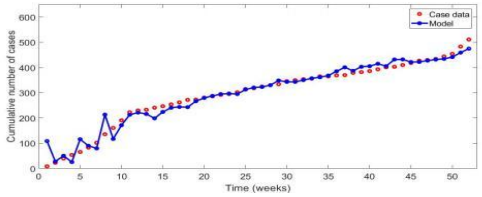

Figure 5: Data fitting of the model in Nigeria.

Table 3: Symbols, values, and units of the parameters used in the model

Parameter	Estimate	Unit	Baseline	Reference
k_H	0.109	Per week	(0,1)	Estimated from the data
ρ	0.646	Per week	(0,1)	Estimated from the data
m_1	0.006	Per week	(0,1)	Estimated from the data
m_2	0.994	Per week	(0,1)	Estimated from the data
ω	0.2	Per week	(0,1)	Estimated from the data
k_R	0.042319	Per week	(0,1)	Fitted
β_1	0.008295	Per week	(0,1)	Fitted
β_2	0.010502	Per week	(0,1)	Fitted
β_3	0.001562	Per week	(0,1)	Fitted
β_0	0.003609	Per week	(0,1)	Fitted
δ	0.3278	Per week	(0,1)	Ojo et al (2022)
μ_R	0.0192	Per week	(0.0357,0.0384)	Demographic Data (2022)
ϑ	0.5	Per week	(0,1)	Momoh et.al(2020)
μ_H	0.0003	Per week	(0.000192,0.0192)	Demographic Data
D	0.975	Variable	(0,1)	Adeniyi et.al
α_0	0.025	Variable	(0,1)	Adeniyi et.al(2022)
α_1	0.9495	Variable	(0,1)	Adeniyi et.al(2022)
α_2	0.0263	Variable	(0,1)	Adeniyi et.al(2022)
φ	0.845	Per week	(0,1)	Estimated from the data
S_H	6000	Person per week	(500,226960000)	Demographic Data (2022)
S_R	500	Person per week	(200,30000)	Assumed

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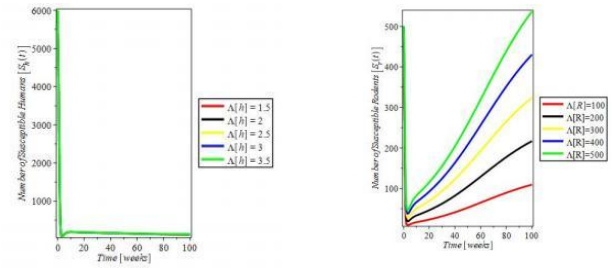
Analysis of the Basic Reproduction Number R_o

Contact tracing has a significant effect on the reproduction number, as shown in Table 4 and Figure 6, reducing the spread of the virus by 0.313. The spread of the virus from human to human is highest, while rodent-to-human transmission is lowest.

Table 4: Reproduction Number (with & without contact tracing) for Models 1 & 2

Mode of transmission	Model 1 with contact tracing	Model 2 without contact tracing
R_h	3.551890730	23.69886167
R_{hr}	0.7840189091	5.231116918
R_{rh}	4.329789087	4.329789087
R_0	2.635626538	6.807972870

Figure 6(a) illustrates the dynamics of the number of vulnerable humans over time, where Λ_H represents the recruitment rate of humans. Initially, the susceptible population declines steeply, and this decline accelerates as ΛH increases. This trend suggests that while higher recruitment rates initially increase the pool of susceptible individuals, the susceptible population quickly stabilizes, likely due to infection dynamics or interventions. Over time, the system reaches a steady state where the number of susceptible humans remains relatively constant, indicating a balance between human recruitment and disease transmission. This high- lights that recruitment rates influence the initial spread but have minimal long-term impact once equilibrium is achieved while figure 6(b) displays the dynamics of the number of vulnerable rodents over time, where Λ_R represents the recruitment rate of rodents. Initially, there is a steep decline in the susceptible population, which occurs more rapidly as Λ_R increases. This trend suggests that while higher recruitment rates initially lead to a larger pool of susceptible rodents, the susceptible population quickly stabilizes, likely due to infection dynamics or interventions. Over time, the system reaches a steady state where the number of susceptible rodents remains relatively constant, indicating a balance between rodents recruitment and disease transmission. This highlights that recruitment rates influence the initial spread but have minimal long-term impact once equilibrium is achieved.



(a) **(b)**

Figure 6: (a) Effect of variation of (Λ_H) on susceptible human. (b) Effect of variation of (Λ_R) on susceptible rodent.

In Figure 7 (a & b), the gap between the two curves widens as Λ_H increases. This suggests that contact tracing is especially effective in managing outbreaks when the population grows rapidly, thereby increasing the susceptible pool. The graph demonstrates how the human recruitment rate (Λ_H) influences the reproduction number of Lassa fever. While a higher Λ_H increases R_0, effective contact tracing mitigates this impact by curtailing the spread of infection, underscoring its importance as a control measure in growing populations. At the same time, figure 7 (c & d) below shows a sensitivity analysis of R_0 with respect to changes in ΛR, the transmission rate of Lassa fever due to recruitment of multimammate rats. The analysis is conducted under two scenarios: with contact tracing (red line) and without contact tracing (blue line). The reproduction number R_0 is lower in the contact tracing scenario (red line) than in the scenario without contact tracing (blue line) for any given value of ΛR. This implies that contact tracing effectively reduces the spread of the disease by lowering R_0. Even as Λ_R increases, the presence of contact tracing keeps R0 at a lower level than in the scenario without contact tracing. When Λ_R is low (near 0), the difference in R_0 between the two scenarios is small. However, as Λ_R increases, the gap widens, showing that contact tracing becomes more beneficial in reducing R_0

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When the recruitment rate of the multimammate rate is higher.

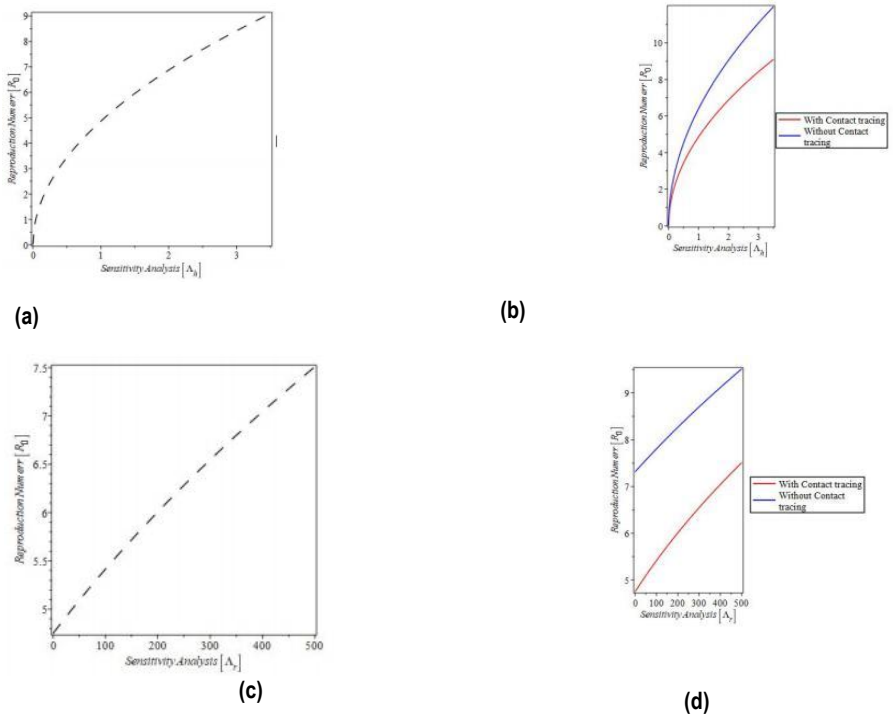

Figure 7: (a & b) Effect of variation of Λ_H on R_0. (c & d) Effect of variation of Λ_R on R_0.

Figure 8(a) below illustrates how increasing the person-to-person transmission rate β_0 affects the susceptible human population over time. A higher β_0 value leads to a more significant reduction in the susceptible population as more people get infected due to increased transmission among humans, while a lower β_0 values result in fewer infections among humans.

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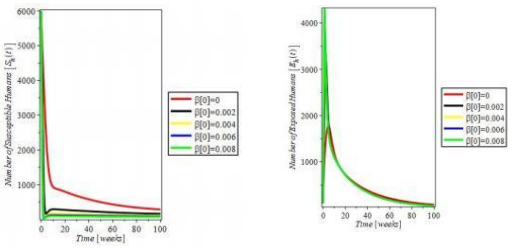
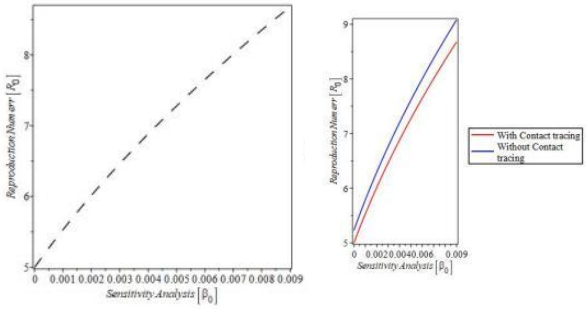
allowing a larger portion of the population to remain susceptible throughout the 100 weeks. This trend highlights that human-to-human transmission plays a crucial role in reducing the susceptible human population. As β_0 increases, the risk of infection among humans grows, resulting in a steeper decline in susceptibility within the population. In Figure 8(b), as β0 increases, the peak number of exposed individuals slightly rises, and the decline becomes more gradual. For instance, the green line (highest $\beta_0 = 0.008$) maintains a larger exposed population than the lower β_0 values (such as the red or black lines). All curves show a rapid decline in the number of exposed humans within the first few weeks. This suggests that many exposed individuals progress to the infectious state quickly, thereby reducing the exposed population.



(a) **(b)**

Figure 8: (a) Effect of variation of β_0 on susceptible human. (b) Effect of variation in β0 on exposed humans.

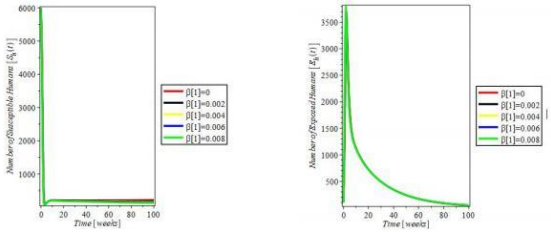
In Figure 9, as β0 increases, R0 also increases in both scenarios (with and without contact tracing). This indicates that a higher human-to-human transmission rate leads to a higher R_0, meaning the disease spreads more readily among humans. Increasing β_0 (human-to-human transmission rate) without interventions such as contact tracing leads to a higher reproduction number, making the outbreak harder to control. Implementing contact tracing helps reduce R_0 at all transmission levels, indicating it is an effective measure to slow down the spread of Lassa fever by limiting the number of secondary infections caused by each infected individual.



(a) **(b)**

Figure 9: Effect of variation of β_0 on R_0.

In Figure 10(a) below, for all values of β_1, there is an initial rapid decline in the number of susceptible humans. This sharp drop suggests that at the beginning of the time period, many susceptible individuals are exposed to the infection, primarily through interactions with infected rodents. After the initial decline, the number of vulnerable humans stabilizes at a much lower level over time. This stabilization occurs at varying levels depending on the value of β_1. Higher β_1 values (such as 0.006 and 0.008, shown by the blue and green lines) lead to a smaller long- term susceptible population, indicating a higher rate of infections due to increased rodent-to- human transmission while Lower β_1 values (such as 0.002 or 0) result in a higher long-term susceptible population, as fewer humans are infected when rodent-to-human transmission is limited or absent. Likewise, in Figure 10(b), all curves start with a high number of exposed individuals, close to the upper limit of 3500. This high initial exposure level likely reflects the early phase of an outbreak, when many individuals are first exposed to the virus. After the initial phase, the number of exposed humans declines gradually over time, approaching near-zero by the end of the 100-week period. This indicates that, over time, the number of newly exposed individuals decreases as the epidemic runs its course as the exposed human progresses to the infected class.



(a) **(b)**

Figure 10: (a) Effect of variation of β_1 on susceptible human. (b) Effect of variation of β_1 on exposed human.

Figure 11 depicts the relationship between the reproduction number (R_0) and the trans- mission rate of Lassa fever from rodents to humans (β_1) under two scenarios: with contact tracing (red line) and without contact tracing (blue line). The graph indicates that contact tracing effectively reduces the reproduction number by identifying and isolating infected indi- viduals, thereby interrupting transmission chains. The gap between the two curves increases as β_1 grows, showing that contact tracing has a more significant relative impact when transmission rates are higher. In conclusion, the graph underscores the importance of contact tracing as an intervention to mitigate Lassa fever's spread. It also emphasizes the critical role of controlling the rodent-to-human transmission rate (β_1) to reduce the outbreak's severity.

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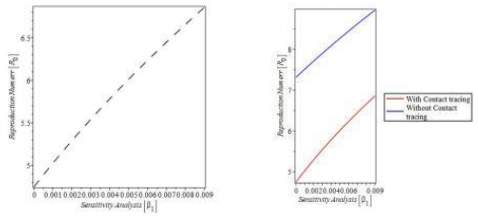
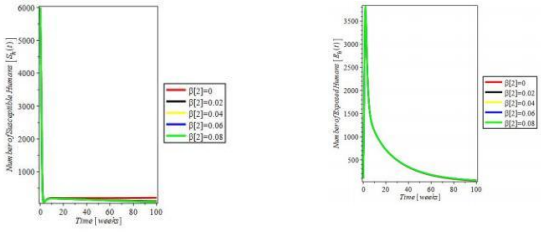


(a) **(b)**

Figure 11: Effect of variation of β_1 on R_0.

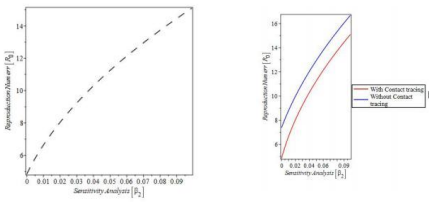
In the figure 12(a) below, for all values of β_2, there is an initial rapid decline in the number of susceptible humans. This sharp drop suggests that, at the beginning of the time period, many susceptible individuals are exposed to the infection, primarily due to interactions with infected rodents. After the initial decline, the number of vulnerable humans stabilizes at a much lower level over time. This stabilization occurs at varying levels depending on the value of β_2. Higher β_2 values (such as 0.006 and 0.008, shown by the blue and green lines) lead to a smaller long-term susceptible population, indicating a higher rate of infections due to increased human-to-rodent transmission while Lower β_2 values (such as 0.002 or 0) result in a higher long-term susceptible population, as fewer humans are infected when human-to-rodent transmission is limited or absent. Likewise in figure 4.15(b) all curves start with a high number of exposed individuals, close to the upper limit of 3500. This high initial exposure level likely represents the early phase of an outbreak when many individuals are initially exposed to the virus. After the initial phase, the number of exposed humans declines gradually over time, approaching near-zero by the end of the 100-week period. This indicates that, over time, the number of newly exposed individuals decreases as the epidemic runs its course as the exposed human progresses to the infected class.



(a) **(b)**

Figure 12: (a) Effect of variation of β_2 susceptible human. (b) Effect of variation of β_2 exposed human

The figure 13 below shows a sensitivity analysis of the basic reproduction number R_0 in relation to changes in β_2, the transmission rate of lassa fever from infected humans to susceptible rodents. The analysis is conducted under two scenarios: with contact tracing (red line) and without contact tracing (blue line). The reproduction number R_0 is lower in the contact tracing scenario (red line) compared to the scenario without contact tracing (blue line) for any given value of β_2. This implies that contact tracing effectively reduces the spread of the disease by lowering R_0. Even as β_2 increases, the presence of contact tracing keeps R0 lower than in the scenario without it. When β_2 is low (near 0), the difference in R_0 between the two scenarios is small. However, as β_2 increases, the gap widens, indicating that contact tracing becomes more beneficial in reducing R0 when the transmission rate of Lassa fever from infectious humans to vulnerable rodents is higher.



(a) **(b)**

Figure 13: Effect of variation of β_2 on R_0.

In Figure 14(a), in the first few weeks, there is a steep decline in the number of vulnerable rodents across all values of β_3. This initial drop likely corresponds to an outbreak or a high-transmission phase in which many susceptible rodents become infected. After the initial decline, the number of vulnerable rodents begins to increase over time, eventually approaching steadily, reaching 500 as the weeks progress. This suggests a period during which susceptible rodents transition to the exposed class. Higher β_3 values (e.g., 0.006 and 0.008) result in a faster increase in the susceptible rodent population over time. This may seem counterintuitive, but it could imply that high transmission from dead bodies leads to a quicker transition of susceptible rodents to the exposed class, while Lower β3 values (e.g., 0 and 0.002) lead to a slower increase in the number of susceptible rodents. This suggests that when transmission from dead bodies is minimal or absent, the infection may persist longer among the rodent population, thereby delaying the transition of susceptible rodents to the exposed class or replenishment of susceptibles. Also, Figure 14(b) below shows an initial peak in the number of infectious rodents for all values of β_3 within the first few weeks, reaching a maximum of about 26 infected rodents. This likely corresponds to an initial outbreak or peak in infection due to high transmission among the rodents. Following the initial peak, the number of infected rodents steadily declines across all values of β_3, approaching zero by around 90-100 weeks. This indicates that the infection rate decreases over time, leading to near eradication within the rodent population as the weeks progress. The different values of β_3 (ranging from 0 to 0.008) do not show a significant difference in the early stages of infection, as all lines peak at nearly the same level. However, higher values of β_3 (such as 0.006 and 0.008, shown by the blue and green lines) slightly extend the infection duration, resulting in a slower decline than lower values. This suggests that, while dead bodies contribute to transmission, the overall impact is relatively small in this model.

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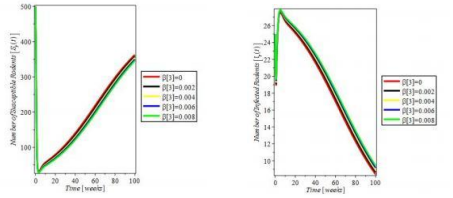
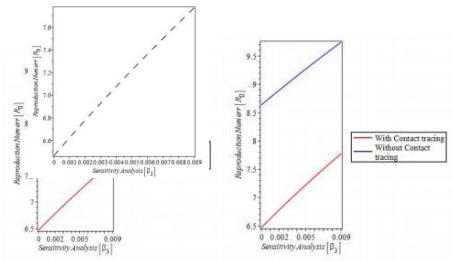


(a) **(b)**

Figure 14: (a) Effect of variation of β_3 on susceptible rodent. (b) Effect of variation of β_3 on infected rodent.

Figure 15 below shows a sensitivity analysis of R_0 with respect to changes in β3, the transmission rate of Lassa fever from contact with dead bodies. The analysis is conducted under two scenarios: with contact tracing (red line) and without contact tracing (blue line). The reproduction number R_0 is lower in the contact tracing scenario (red line) than in the scenario without contact tracing (blue line) for any given value of β3. This implies that contact tracing effectively reduces the spread of the disease by lowering R_0. Even as β_3 increases,

The presence of contact tracing keeps R0 lower than in the scenario without it. When β_3 is low (near 0), the difference in R_0 between the two scenarios is small. However, as β_3 increases, the gap widens, indicating that contact tracing becomes more beneficial in reducing R0 when the transmission rate from dead bodies is higher.



a) **(b)**

Figure 15: Effect of variation of β_3 on R_0.

Figure 16(a) indicates that as the progression rate (k_H) from the exposed class to the infectious class increases, the number of exposed humans (E_H) decreases more rapidly over time, suggesting that faster progression reduces the duration of exposure in the population. while figure 16 b) shows that as the progression rate (k_H) from the exposed class to the infectious class increases, the number of infectious humans (I_H) initially peaks higher but then declines more rapidly over time, indicating a faster progression leads to quicker infection dynamics and eventual reduction in the infected population.

Figure 16(c) shows that, as the progression rate from the exposed class to the infectious class (k_R) increases, the number of exposed rodents (E_R) decreases more rapidly over time, indicating a faster transition from exposed class to infected class and thereby reducing the exposed rodent population while figure 16(d) illustrates that, as the progression rate from the exposed.

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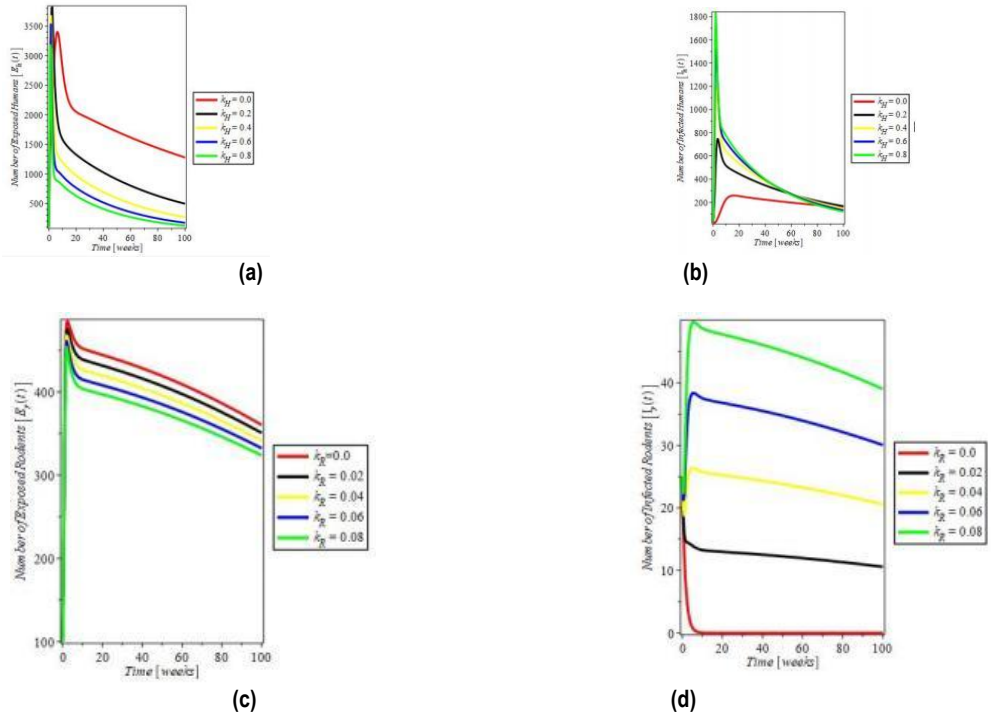
class to the infected class (k_R) increases, the peak number of infected rodents (k_R) rises but then declines faster over time, indicating a quicker depletion of the infected rodents, suggesting the impact of rodent control.



(a) (b)

(c) (d)

Figure 16: (a) Sensitivity analysis of k_H on exposed human. (b) Sensitivity analysis of k_H on infected human. (c) Sensitivity analysis of k_R on exposed rodent. (d) Sensitivity analysis of k_R on infected rodent.

Conclusion

Comparative analysis of Lassa fever transmission models highlights the transformative impact of integrating contact tracing into outbreak management strategies. Models incorporating contact tracing consistently outperform those that do not, demonstrating superior capabilities to reduce transmission rates, identify and isolate exposed individuals earlier, and optimize the allocation of healthcare resources. By leveraging real-time data on contact networks, these models offer a more precise understanding of the disease's spread and enhance the effectiveness of containment measures. Furthermore, integrating contact tracing into epidemiological models not only curtails the spread of Lassa fever but also builds public trust in health interventions. This trust is critical in ensuring compliance with containment measures, ultimately leading to more successful outbreak management. The findings underscore the importance of adopting advanced, data-driven approaches to disease modeling, particularly in regions prone to recurrent outbreaks of Lassa fever and other infectious diseases. In conclusion, the strategic incorporation of contact tracing into Lassa fever models is essential for optimizing containment efforts and improving public health outcomes. This approach should be prioritized in developing future disease management protocols, with the potential to reduce the burden of Lassa fever in affected regions significantly. The study on the effectiveness of contact tracing in managing Lassa fever outbreaks offers critical insights for public health policies, particularly in West Africa. A key takeaway is the demonstrated value of incorporating contact tracing into epidemiological models, which significantly improves the early detection and isolation of infected individuals. Policymakers should prioritize integrating contact tracing systems into outbreak response strategies to manage disease transmission better and reduce secondary infections. Additionally, the study emphasizes the need for efficient allocation of healthcare resources. In optimizing contact tracing, governments can ensure that resources such as personnel, medical supplies, and facilities are used more effectively. This is particularly important in resource-constrained regions, where swift and targeted interventions can prevent further strain on healthcare systems. Another vital policy implication is the role of public trust in the success of outbreak management. The study shows that integrating contact tracing not only enhances control measures but also increases public confidence in health interventions. Policymakers should, therefore, focus on clear and transparent communication strategies to ensure that communities are informed and cooperative with contact tracing efforts. Finally, the findings suggest that the benefits of contact tracing extend beyond Lassa fever, making it a valuable tool for managing future outbreaks of similar infectious diseases. Governments should develop comprehensive preparedness plans that incorporate contact tracing to quickly address the emergence of viral haemorrhagic fevers and other public health threats. By doing so, they can improve outbreak control, safeguard public health, and strengthen health systems in vulnerable regions.

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