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MATHEMATICAL MODELLING OF THE IMPACT OF REDUCED INFECTION ON LASSA HAEMORRHAGIC FEVER TRANSMISSION DYNAMICS: A THEORETICAL STUDY

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Abstract: A novel deterministic model for the transmission dynamics of Lassa Haemorrhagic Fever (LHF) is designed and deployed to qualitatively assess the role of the impact of reduced relative infection of the human-human mode of transmission compared to the rodent-human mode of transmission on the population population dynamics for Lassa Haemorrhagic Fever disease burden. The model is shown to undergo the backward bifurcation phenomenon due to the presence of the parameter which accounts for the rate at which temporary immunity wanes. A unique threshold for the rate at which temporary immunity wanes was also obtained. A special case of the model showed that the disease-free equilibrium was globally asymptotically stable in the absence of the rate at which temporary immunity wanes.

Keywords: Backward bifurcation, Lassa Haemorrhagic Fever, *Mastomys natalensis*, Rodents, Stability

Introduction

Lassa Haemorrhagic Fever (LHF) is an animal-borne acute viral haemorrhagic disease [4, 14]. This malady was first described in the 1950s however, the causative virus was not known until 1969 when it was discovered in a town called Lassa in Nigeria [4, 10]. Lassa Haemorrhagic Fever is predominantly endemic in Benin Republic, Ghana, Guinea, Liberia, Mali, Sierra Leone, and Nigeria, all in West Africa [14], where an estimated 100,000 to 300,000 infections, with approximately 5,000 (about 1%-2%) fatalities occur annually [4, 9, 14]. Non-standardization of surveillance for cases of the disease is not uniformly carried thus rendering these estimates crude [4]. Annually, in certain areas in Liberia and Sierra Leone, 10% to 16% of hospitalized individuals are confirmed cases of Lassa Haemorrhagic Fever, which is indicative of the tremendous impact of the disease burden on the human population residing in this vast region [4].

The animal vector or reservoir for the Lassa virus is a multimammate rodent, *Mastomys natalensis*, which is extensively distributed in the West African region [4]. These rodents, once infected are able to excrete the virus in their urine for an extended period of time, probably for its lifetime [4]. *Mastomys* rats, known to produce large numbers of offspring due to their high frequency of breeding, are many in the forests and savannas of west, central and east Africa [4]. These rats readily invade and colonize human communities especially homes and areas reserved for food storage. These aforementioned factors contribute efficiently to the spread of Lassa virus to human beings from infected rats [4]. Transmission of Lassa virus to humans arises from consumption of food or water

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contaminated by faeces or urine of infected *M. natalensis*, or consumption of infected rats [9]. The virus can also be spread from humans to humans (though rare and in small-scale outbreaks) mostly in households and hospital environments via direct contact with blood or bodily secretions of infected persons [9, 13, 14]. Infections caused by rats are responsible for more than 95% of Lassa cases [13]. Epidemiological evidence supporting airborne spread of Lassa Haemorrhagic Fever between individuals does not exist [14].

Mathematical models have been used in understanding the dynamics of many emerging and reemerging diseases [6]. Many authors have improved the literature having developed and deployed mathematical models for investigating Lassa Haemorrhagic Fever disease dynamics with the purpose of answering distinct questions. The impact of contributions from regular contact with *M. natalensis* and infectious contact those suffering from the disease [9], subdividing the rodent population into infant and adult classes [2], seasonal variation in the births of the rodents was captured in [1], the effects of socio-economic conditions [5], rodent control [9] and optimal control analysis [11]. It is evident, from the foregoing, that the several mathematical models have been developed to analyze Lassa Haemorrhagic Fever infection but none has looked at the possibility

Table 1: Description of state variables of model (8)

Variables	Description
S_h	Susceptible human population
E_h	Human population exposed to Lassa Haemorrhagic Fever
I_a	Human population asymptomatically infected with Lassa Haemorrhagic Fever
I_s	Human population symptomatically infected with Lassa Haemorrhagic Fever
R_h	Human population recovered from Lassa Haemorrhagic Fever
S_r	Susceptible rodent (*M. natalensis*) population
I_r	Infected rodent (*M. natalensis*) population

Table 2: Description of parameters of model (8)

Parameters	Description
Λ_h	Human recruitment rate
μ_h	Natural death rate of humans
β_hh	Transmission rate of infected humans
μ_0	Disease-induced mortality rate of humans
θ_0	Rate at which temporary immunity wanes
θ_h	Rate at which the exposed class progresses to the infectious classes
ζ	Proportion of exposed individuals who become asymptomatic, with $0 < \zeta < 1$
ϕ_a	Recovery rate of the I_a class due to immunity
ϕ_s	Treatment rate for the I_s class
θ_s	Relative rate of infectiousness of infectious humans, with $0 < \theta_s < 1$
β_R	Transmission rate between *M. natalensis* and humans
η_r	Growth rate of *M. natalensis*
K_0	Carrying capacity of the environment for the *M. natalensis* population
β_r	Transmission rate of infected *M. natalensis*
μ_r	Natural *M. natalensis* mortality

of the impact of reduced infection on the burden of the disease in the population in the presence of waning temporary immunity of individuals both recovered and treated for the disease, to the best of the authors' knowledge. The objective of this paper is to mathematically model the impact of reduced infection on the burden of the disease in the population in the presence of waning temporary immunity of individuals both recovered and treated for the disease, analyze the model.

In this study, in Section 2, a novel mathematical model is formulated to investigate the effect of reduced infection on the dynamics of Lassa Haemorrhagic Fever at population level. In Section 3, important thresholds governing the disease dynamics are obtained and the local and global asymptotic stabilities of equilibria are established while Section 4 concludes the paper.

Model Formulation

The total human population at time *t*, denoted by $N_h(t)$, is split into the mutually exclusive compartments of susceptible to infections $(S_h(t))$, exposed to LHF $(E_h(t))$, asymptomatically infected with LHF $(I_a(t))$, symptomatically infected with LHF $(I_s(t))$ and recovered from LHF $(R_h(t))$, individuals so that

$$N_h(t) = S_h(t) + E_h(t) + I_a(t) + I_s(t) + R_h(t).$$

Similarly, the entire *Mastomys natalensis* population in the environment at time *t*, given by $N_r(t)$, is broken down into the mutually exclusive compartments of susceptible rodents $(S_r(t))$ and infectious rodents $(I_r(t))$, where

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

The wholly susceptible human population, $S_h(t)$, is generated by the recruitment of individuals (assumed susceptible) into the population at a rate Λ_h. This class is further populated by individuals from the recovered class, R_h, who lose their disease-acquired immunity at the rate θ_0. The population of susceptible individuals is reduced due to Lassa Haemorrhagic Fever infection at the rate λ_r. The population is further reduced by natural mortality at a rate μ_h. Thus

$$S_h^{'} = \Lambda_h - \lambda_r S_h + \theta_0 R_h - \mu_h S_h. \qquad (1)$$

The exposed human population, $E_h(t)$, is populated by the movement of individuals from susceptible individuals infected with Lassa Haemorrhagic Fever infection at the rate λ_r. The population is further reduced by individuals who move into the population of humans asymptomatically infected with LHF at the rate θ_h and natural death rate μ_h. Thus

$$E_h^{'} = \lambda_r S_h - (\theta_h + \mu_h) E_h. \qquad (2)$$

The asymptomatically infected human population, $I_a(t)$, is populated by the movement of individuals from a fraction of individuals exposed to Lassa Haemorrhagic Fever infection at the rate $\zeta\theta_h$. The population is further reduced by individuals who recover from LHF due to natural immunity at the rate ϕ_a and natural death rate μ_h. Thus

$$I_a^{'} = \zeta\theta_h E_h - (\phi_a + \mu_h) I_h. \qquad (3)$$

The symptomatically infected human population, $I_s(t)$, is populated by the movement a fraction of individuals exposed to Lassa Haemorrhagic Fever infection at the rate $(1-\zeta)\theta_h$. The population is reduced by individuals who recover after treatment for LHF at the rate ϕ_s. The symptomatically infected human population further reduced by individuals who suffer disese-induced mortality at the rate μ_0 and natural death rate μ_h. Thus

$$I_s^{'} = (1-\zeta)\theta_h E_h - (\phi_s + \mu_0 + \mu_h)I_s. \quad (4)$$

The recovered human population, $R_h(t)$, is populated by the movement of the population of asymptomatically infected individuals who recovered from Lassa Haemorrhagic Fever infection due to natural immunity at the rate ϕ_a. The recovered population is further increased by symptomatic individuals successfully treated for LHF infection at the rate ϕ_s. The population is reduced by individuals who lose their temporary immunity from LHF at the rate θ_0. The recovered human population further is reduced by individuals who succumb to natural death at the rate μ_h. Thus

$$R_h^{'} = \phi_a I_a + \phi_s I_s - (\theta_0 + \mu_h)R_h. \quad (5)$$

The susceptible *M. natalensis* population, $S_r(t)$, is populated by a logistic growth term at the rate $\eta_r N_r(1-\frac{N_r}{K_0})$, where η_r is the growth rate of *M. natalensis* and K_0 is the carrying capacity of the rodent population. The population is reduced by rodents who pick up the virus responsible for the disease at from other infectious rodents at the rate β_r. The susceptible *M. natalensis* population is reduced by rodents who die naturally at the rate μ_r. Thus

$$S_r^{'} = \eta_r N_r\left(1-\frac{N_r}{K_0}\right) - \beta_r I_r S_r - \mu_r S_r. \quad (6)$$

The infectious *M. natalensis* population, $I_r(t)$, is populated by susceptible *M. natalensis* who become infected at the rate β_r. The infectious *M. natalensis* population is reduced by rodents who die naturally at the rate μ_r. Thus

$$I_r^{'} = \beta_r I_r S_r - \mu_r I_r. \quad (7)$$

On the basis of the foregoing, the LHF model is the following deterministic system of seven non-linear ordinary differential equations (the state variables and parameters of the model are tabulated in Table 1 and Table 2 respectively):

$$\begin{aligned} S_h^{'} &= \Lambda_h - \lambda_r S_h + \theta_0 R_h - \mu_h S_h, \\ E_h^{'} &= \lambda_r S_h - (\theta_h + \mu_h)E_h, \\ I_a^{'} &= \zeta\theta_h E_h - (\phi_a + \mu_h)I_h, \\ I_s^{'} &= (1-\zeta)\theta_h E_h - (\phi_s + \mu_0 + \mu_h)I_s, \\ R_h^{'} &= \phi_a I_a + \phi_s I_s - (\theta_0 + \mu_h)R_h, \\ S_r^{'} &= \eta_r N_r(1-\tfrac{N_r}{K_0}) - \beta_r I_r S_r - \mu_r S_r, \\ I_r^{'} &= \beta_r I_r S_r - \mu_r I_r. \end{aligned} \quad (8)$$

where the force of infection associated with LHF is given below:

$$\lambda_r = \beta_R I_r + \theta_s\beta_h(I_a + I_s), \quad (9)$$

where

$$\beta_R I_r \quad (10)$$

is the *M. natalensis* to human contribution to the force of infection associated with LHF while

$$\theta_s\beta_h(I_a + I_s) \quad (11)$$

is the human to human contribution to the force of infection associated with LHF.

Positivity and Boundedness of Solutions

Theorem 1. Let the initial data for the Lassa Haemorrhagic Fever model (8) be given as $S_h(0) > 0, E_h(0) > 0, I_a(0) > 0, I_s(0) > 0, R_h(0) > 0, S_r(0) > 0$ and $I_r(0) > 0$. Then the orbits $(S_h(t), E_h(t), I_a(t), I_s(t), R_h(t), S_r(t)$ and $I_r(t))$ of the model with positive initial conditions, will continue to be positive for all time $t > 0$.

Proof: Let $t_1 = \sup\{t > 0: S_h(0) > 0,\ E_h(0) > 0,\ I_a(0) > 0,\ I_s(0) > 0,\ R_h(0) > 0,\ S_r(0) > 0,\ I_r(0) > 0\}$.

Consider the first equation of model (8), given below as

$$\frac{dS_h(t)}{dt} = \Lambda_h + \theta_0 R_h - (\lambda_r + \mu_h)S_h(t) \quad (12)$$

$$\geq \Lambda_h - (\lambda_r + \mu_h)S_h(t), \quad (13)$$

which can be re-expressed as

$$\frac{d}{dt}\left[S_h(t)exp\left\{\mu_h t + \int_0^t \lambda_r(\tau)\, d\tau\right\}\right]$$

$$\geq \Lambda_h exp\left\{\mu_h t + \int_0^t \lambda_r(\tau)\, d\tau\right\}. \quad (14)$$

$$S_h(t_1)\exp\{\mu_h t_1 + \int_0^{t_1} \lambda_r(\tau)\quad d\tau\} - S_h(0)$$

$$\geq \int_0^{t_1} \Lambda_h \left[\exp\{\mu_h y + \int_0^y \lambda_r(\tau)d\tau\}\right]dy, \quad (15)$$

So that,

$$S_h(t_1) \geq S_h(0)\exp\ [-\mu_h t_1 - \int_0^{t_1} \lambda_r(\tau)d\tau] + [\exp\{-\mu_h t_1 - \int_0^{t_1} \lambda_r(\tau)d\tau\}] \times \int_0^{t_1} \Lambda_h \left[\exp\{\mu_h y + \int_0^y \lambda_r(\tau)d\tau\}\right]dy > 0. \quad (16)$$

Hence $S_h(t) > 0,\ \forall\ t > 0$.

Similarly, it can be shown that other state variables in (8) are positive, i.e., $E_h(t) > 0,\ I_a(t) > 0,\ I_s(t) > 0,\ R_h(t) > 0,\ S_r(t) > 0,\ I_r(t) > 0,\ \forall\ t > 0$.

Thus, we have established positivity for all the state variables in model (8) for all time.

We proceed to establish the boundedness of solutions to the model (8).

Theorem 2. Let $(S_h(t), E_h(t), I_a(t), I_s(t), R_h(t), S_r(t), I_r(t))$ be trajectories of the system with initial conditions and the biological feasible region given by the set $\mathcal{D}_o = \mathcal{D}_h \times \mathcal{D}_r \times \subset \mathbb{R}_+^5 \times \mathbb{R}_+^2$, where:

$$\mathcal{D}_h = \{(S_h, E_h, I_a, I_s, R_h) \in \mathbb{R}_+^5 : N_h \leq \frac{\Lambda_h}{\mu_h}\}$$

$$\mathcal{D}_r = \{(S_h, I_r) \in \mathbb{R}_+^2 : N_r \leq \frac{K_0(\eta_r - \mu_r)}{\eta_r}\}$$

is positively-invariant and attracts the entire positive trajectories of the model (8).

Proof: Adding up the right flank of the vector field for the human population in (8), yields

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \mu_0 I_s. \quad (17)$$

From (8), it ensues that $\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$. Hence, $\frac{dN_h}{dt} \leq 0$ if $N_h(t) \geq \frac{\Lambda_h}{\mu_h}$. Employing a standard comparison theorem [8], we prove that $N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t})$. In particular, if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, thus $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ for every $t > 0$. Hence, the set $\mathcal{D}_h$ is positively invariant. Moreover, if $N_h(0) > \frac{\Lambda_h}{\mu_h}$, then either the orbits enters the domain $\mathcal{D}_h$ in finite time or $N_h(t)$ asymptotically approaches $\frac{\Lambda_h}{\mu_h}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_h$ attracts every trajectory in $\mathbb{R}_+^5$.

Similarly, adding up the right flank of the vector field for the *M. natalensis* population in , yields

$$\frac{dN_r}{dt} = \eta_r N_r \left(1 - \frac{N_r}{K_0}\right) - \mu_r N_r. \quad (18)$$

From (18),, which follows that $\frac{N_r(t)}{K_0(\eta_r - \mu_r) - \eta_r N_r(t)} - \frac{N_r(0)}{K_0(\eta_r - \mu_r) - \eta_r N_r(0)} = e^{(\eta_r - \mu_r)t}$. But $N_r(0) = N_r(K_0) = 0$. Hence, $\frac{N_r(t)}{K_0(\eta_r - \mu_r) - \eta_r N_r(t)} = e^{(\eta_r - \mu_r)t}$. Therefore, $N_r(t) = \frac{K_0(\eta_r - \mu_r)}{\eta_r + e^{-(\eta_r - \mu_r)t}}$. Therefore $N_r(t) = \frac{K_0(\eta_r - \mu_r)}{\eta_r}$ as $t \to \infty$. Hence, $N_r(t)$ is bounded by $\frac{K_0(\eta_r - \mu_r)}{\eta_r}\ \forall\ time,\ t$. Thus, the domain $\mathcal{D}_r$ attracts every trajectory in $\mathbb{R}_+^2$.

Therefore, it is sufficient to study the dynamics of the flows generated by the model system (8) in $\mathcal{D}_o$. We conclude, therefore, that the model (8) is together mathematically and epidemiologically well-posed.

Mathematical Analysis of the Model

Local Asymptotic Stability (LAS) of the Disease-free Equilibrium (DFE)

The model system has a disease-free equilibrium (DFE) given by

$$\begin{aligned}\mathcal{E}_0 &= (S_h^*, E_h^*, I_a^*, I_s^*, R_h^*, S_r^*, I_r^*) \\ &= \left(\frac{\Lambda_h}{\mu_h}, 0,0,0,0, \frac{K_0(\eta_r-\mu_r)}{\eta_r}, 0\right) \end{aligned} \quad (19)$$

The linear stability of $\mathcal{E}_0$ is established by deploying the next-generation operator method on the model system (8). Deploying specific notations as espoused by, it ensues that matrices F and V, respectively, for the fresh infection terms and the other transition terms, are given by

$$F = \begin{bmatrix} 0 & \frac{\theta_s\beta_h\Lambda_h}{\mu_h} & \frac{\theta_s\beta_h\Lambda_h}{\mu_h} & \frac{\beta_r\Lambda_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_r K_0(\eta_r-\mu_r)}{\mu_r} \end{bmatrix};$$

$$V = \begin{bmatrix} (\theta_h+\mu_h) & 0 & 0 & 0 \\ -\zeta\theta_h & (\phi_a+\mu_h) & 0 & 0 \\ (1-\zeta)\theta_h & 0 & (\phi_s+\mu_0+\mu_h) & 0 \\ 0 & 0 & 0 & \mu_r \end{bmatrix}$$

where F and V are the matrix of fresh infections and transition terms, respectively. It ensues that *effective reproduction number*, denoted by $\mathcal{R}_L = \rho(FV^{-1})$, is

$$\mathcal{R}_L = \frac{\theta_h\theta_s\beta_h\Lambda_h[\zeta(\phi_s+\mu_0+\mu_h)+(1-\zeta)(\phi_a+\mu_h)]}{\mu_h(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h))} \quad (20)$$

where $\rho(FV^{-1})$ is the spectral radius belonging to the matrix FV^{-1}. Consequently, the result below stems from the conclusion of Theorem 2 in [12].

Lemma 1: The DFE $\mathcal{E}_0$ of (8) is locally asymptotically stable on the condition that $\mathcal{R}_{HS} < 1$ and unstable on the condition that $\mathcal{R}_L > 1$.

The threshold quantity,$\mathcal{R}_L$, is the effective reproduction number of the disease [12]. It is a measure of the mean number of secondary Lassa Haemorrhagic Fever infections generated by a typical infected human in a completely susceptible population or at the DFE [12]. The epidemiological connotation of Lemma 3.1 implies that whenever $\mathcal{R}_L$ is less than one, Lassa Haemorrhagic Fever can be annihilated from the population if the initial sizes of the classes of the model system are in the basin of attraction of the disease-free equilibrium $\mathcal{E}_0$. Thus, a little influx of Lassa Haemorrhagic Fever-infected humans into the population will not provoke enormous Lassa Haemorrhagic Fever outbreaks, with the resultant effect of the disease dying out over time.

Analysis of the Effective Reproduction Number, $\mathcal{R}_L$

Using the threshold parameter, $\mathcal{R}_L$, we wish to determine the effect of the rate at which the exposed class progresses to the infectious classes (θ_h), the relative rate of infectiousness of humans (θ_s), the recovery rate of the asymptomatic class due to immunity (ϕ_a), the treatment rate for the symptomatic class (ϕ_s) and the relative rate of infectiousness of infectious humans (θ_s) on the control of Lassa Haemorrhagic Fever in the population.

Calculating the partial derivatives of $\mathcal{R}_L$ with respect to the parameter under scrutiny (θ_h) further exposes the consequence of this parameter on Lassa Haemorrhagic Fever regulation among the populace. This implies

$$\frac{\partial\mathcal{R}_L}{\partial\theta_h} = \frac{\theta_s\beta_h\Lambda_h((1-\zeta)\phi_a+\zeta(\phi_s+\mu_0)+\mu_h)}{\mu_h(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)} > 0 \quad (21)$$

Apparently, it follows from (21) that the partial derivative is positive, unconditionally.

Thus, the rate at which the exposed class progresses to the infectious classes will exert a positive consequence in increasing the burden of Lassa Haemorrhagic Fever among the populace, regardless of the rates of the other parameters in the expression on the right flank of (21).

Calculating the partial derivatives of $\mathcal{R}_L$ with respect to the parameter under scrutiny (θ_s) further shows the effect of this parameter on Lassa Haemorrhagic Fever control among the populace. This implies

$$\frac{\partial \mathcal{R}_L}{\partial \theta_s} = \frac{\theta_h \beta_h \Lambda_h((1-\zeta)\phi_a+\zeta(\phi_s+\mu_0)+\mu_h)}{\mu_h(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)} > 0 \qquad (22)$$

Consequently, it follows from (22) that the partial derivative is positive, unconditionally. Thus, the relative rate of infectiousness of infectious humans will have a positive influence in increasing the burden of Lassa Haemorrhagic Fever among the populace, regardless of the rates of the other parameters in the expression on the right flank of (22).

Calculating the partial derivatives of $\mathcal{R}_L$ with respect to the parameter under scrutiny (ϕ_a) further shows the effect of this parameter on Lassa Haemorrhagic Fever control among the populace. This implies

$$\frac{\partial \mathcal{R}_L}{\partial \phi_a} = -\frac{\theta_h \theta_s \beta_h \Lambda_h(\phi_s+\mu_0+\mu_h)}{\mu_h(\theta_h+\mu_h)(\phi_a+\mu_h)^2(\phi_s+\mu_0+\mu_h)} < 0 \qquad (23)$$

Consequently, it follows from (23) that the partial derivative is negative, unconditionally. Thus, the revocery rate of the asymptomatic class due to immunity will exert a positive influence in reducing the burden of Lassa Haemorrhagic Fever among the populace, regardless of the rates of the other parameters in the expression on the right flank of (23).

Calculating the partial derivatives of $\mathcal{R}_L$ with respect to the parameter under scrutiny (ϕ_s) further shows the effect of this parameter on Lassa Haemorrhagic Fever control among the populace. This implies

$$\frac{\partial \mathcal{R}_L}{\partial \phi_s} = -\frac{\theta_h \theta_s \beta_h \Lambda_h(1-\zeta)(\phi_a+\mu_h)}{\mu_h(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)^2} < 0 \qquad (24)$$

Consequently, it follows from (24) that the partial derivative is negative, unconditionally. Thus, the treatment rate for the symptomatic class will exert a positive influence in reducing the burden of Lassa Haemorrhagic Fever among the populace, regardless of the rates of the other parameters in the expression on the right flank of (24).

It is obvious from that

$$\lim_{\phi_s \to \infty} \mathcal{R}_L = 0 \qquad (25)$$

From (25), a near complete annihilation of Lassa Haemorrhagic Fever is feasible. In this situation, an effective strategy will be to pay serious attention to treatment regimen for infected humans.

Lemma 2: The treatment rate (ϕ_s) for the symptomatic infectious class will exert a positive influence in decreasing the Lassa Haemorrhagic Fever burden in a populace, regardless of the rates of the other parameters that constitute the effective reproduction number.

Endemic Equilibrium Point (EEP)

Let the endemic equilibrium point, $\mathcal{E}_L^*$, of the system (8) is defined by

$$\mathcal{E}_L^* = (S_h^{**},\ E_h^{**},\ I_a^{**},\ I_s^{**},\ R_h^{**},\ S_r^{**},\ I_r^{**}) \qquad (26)$$

where

$$\begin{aligned} S_h^{**} &= \frac{(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)I_s^{**}}{\theta_h(\theta_r+\theta_m\theta_s\beta_h I_s^{**})(1-\zeta)(\phi_a+\mu_h)}, \\ E_h^{**} &= \frac{(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)I_s^{**}}{\theta_h(1-\zeta)(\phi_a+\mu_h)}, \\ I_a^{**} &= \frac{\zeta(\phi_s+\mu_0+\mu_h)I_s^{**}}{(1-\zeta)(\phi_a+\mu_h)}, \\ R_h^{**} &= \frac{[\phi_a\zeta(\phi_s+\mu_0+\mu_h)+\phi_s(1-\zeta)(\phi_a+\mu_h)]I_s^{**}}{(\theta_0+\mu_h)(1-\zeta)(\phi_a+\mu_h)}, \\ S_r^{**} &= \frac{\mu_r}{\beta_r}, \\ I_r^{**} &= \frac{\beta_r K_0(\eta_r-\mu_r)-\eta_r\mu_r}{\eta_r\beta_r}. \end{aligned} \quad (27)$$

The force of infection is:

$$\lambda_r^{**} = \theta_r + \theta_m\theta_s\beta_h I_s^{**}. \quad (28)$$

where

$$\theta_m = \frac{\zeta(\phi_s+\mu_0+\mu_h)+(1-\zeta)(\phi_a+\mu_h)}{(1-\zeta)(\phi_a+\mu_h)}, \quad (29)$$

$$\theta_r = \frac{\beta_R[\beta_r K_0(\eta_r-\mu_r)-\eta_r\mu_r]}{\eta_r\beta_r}, \quad (30)$$

Substituting (29) and (30) into (27) and after several algebraic manipulations and simplifications, it is shown that the EEP associated with the system (8) satisfies the polynomial (expressed as a function of I_s^{**})

$$(\mathcal{M}_1-\mathcal{M}_2)(I_s^{**})^2+(\mathcal{M}_3-\mathcal{M}_4)I_s^{**}-\mathcal{M}_5=0. \quad (31)$$

where,

$$\begin{aligned} \mathcal{M}_1 &= (1-\zeta)(\theta_0+\mu_h)(\theta_h+\mu_h)(\phi_a+\mu_h)^2(\phi_s+\mu_0+\mu_h), \\ \mathcal{M}_2 &= \theta_0\theta_m\theta_s\beta_h[\phi_a\zeta(\phi_s+\mu_0+\mu_h)+\phi_s(1-\zeta)(\phi_a+\mu_h)], \\ \mathcal{M}_3 &= (\theta_0+\mu_h)(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)\left[\frac{\beta_R(\beta_r K_0(\eta_r-\mu_r)-\eta_r\mu_r)+\beta_r\eta_r\mu_r}{\eta_r\beta_r}\right], \\ \mathcal{M}_4 &= \theta_0(\phi_a\zeta(\phi_s+\mu_0+\mu_h)+\phi_s(1-\zeta)(\phi_a+\mu_h)) \\ &\quad +\theta_s\beta_h\Lambda_h(\theta_0+\mu_h)(\zeta(\phi_s+\mu_0+\mu_h)+(1-\zeta)(\phi_a+\mu_h)), \\ \mathcal{M}_5 &= (1-\zeta)\Lambda_h(\theta_0+\mu_h)(\phi_a+\mu_h)[\frac{\beta_R(\beta_r K_0(\eta_r-\mu_r)-\eta_r\mu_r)}{\eta_r\beta_r}]. \end{aligned} \quad (32)$$

The above result is captured in the theorem below.

Theorem 1. The model system (8) has:

1. two endemic equilibria on the assumption that $\mathcal{M}_1 > \mathcal{M}_2$, $\mathcal{M}_3 < \mathcal{M}_4$, $\mathcal{M}_5 < 0$ or $\mathcal{M}_1 < \mathcal{M}_2$, $\mathcal{M}_3 > \mathcal{M}_4$, $\mathcal{M}_5 > 0$ and $\mathcal{R}_L > 1$,
2. one unique endemic equilibrium on the assumption that $\mathcal{M}_1 > \mathcal{M}_2$, $\mathcal{M}_3 > \mathcal{M}_4$, $\mathcal{M}_5 > 0$ or $\mathcal{M}_1 > \mathcal{M}_2$, $\mathcal{M}_3 < \mathcal{M}_4$, $\mathcal{M}_5 > 0$ or $\mathcal{M}_1 < \mathcal{M}_2$, $\mathcal{M}_3 > \mathcal{M}_4$, $\mathcal{M}_5 < 0$ or $\mathcal{M}_1 < \mathcal{M}_2$, $\mathcal{M}_3 < \mathcal{M}_4$, $\mathcal{M}_5 < 0$ and $\mathcal{R}_L > 1$,
3. nil endemic steady state otherwise, whenever $\mathcal{R}_L < 1$.

It is significant to mention, at this juncture, that item (1) of Theorem 3.1 (above) is indicative of the presence of backward bifurcation in the model (8). The backward bifurcation phenomenon is pronounced as a consequence of the the co-existence of an infection-free state as well as an endemic steady state that are both stable at whatever time the corresponding reproduction number is less than one. This, therefore, implies that the standard condition required for disease control ($\mathcal{R}_L < 1$) is not any more sufficient for effectively regulating Lassa Haemorrhagic Fever among the populace, although it remains a necessary condition. In such a scenario, effective strategies for Lassa Haemorrhagic Fever control will now have to be based on the basic conditions of different compartments of the model system under consideration. We observe that the EEP of the model possesses a unique endemic equilibrium point when $\mathcal{R}_L > 1$ (and does not have an EEP whenever $\mathcal{R}_L < 1$, and hence no possibility of a backward bifurcation when $\mathcal{R}_L < 1$).

Backward Bifurcation Analysis

Theorem 2. The model (8) experiences backward bifurcation at $\mathcal{R}_L = 1$ whenever $\theta_0 > \theta_0^c$, with θ_0^c defined as

$$\theta_0^c = \frac{\theta_s \beta_h \Lambda_h}{\mu_h}(\omega_3 + \omega_4) > 0, \quad (32)$$

Proof: We employ the ensuing alteration of variables. Let $S_h = x_1$, $E_h = x_2$, $I_a = x_3$, $I_s = x_4$, $R_h = x_5$, $S_r = x_6$, and $I_r = x_7$, so that $N_h = x_1 + x_2 + x_3 + x_4 + x_5$ and $N_r = x_6 + x_7$; hence the model (8) is re-written as

$$\begin{aligned}
\dot{x}_1 &\equiv f_1 = \Lambda_h + \theta_0 x_5 - (\beta_R x_7 + \theta_s \beta_h (x_3 + x_4)) - \mu_h x_1,\\
\dot{x}_2 &\equiv f_2 = (\beta_R x_7 + \theta_s \beta_h (x_3 + x_4)) - (\theta_h + \mu_h) x_2,\\
\dot{x}_3 &\equiv f_3 = \zeta \theta_h x_2 - (\phi_a + \mu_h) x_3,\\
\dot{x}_4 &\equiv f_4 = (1-\zeta)\theta_h x_2 - (\phi_s + \mu_0 + \mu_h) x_4,\\
\dot{x}_5 &\equiv f_5 = \phi_a x_3 + \phi_s x_4 - (\theta_0 + \mu_h) x_5,\\
\dot{x}_6 &\equiv f_6 = \eta_r (x_6 + x_7)\left(1 - \tfrac{(x_6 + x_7)}{K_0}\right) - \beta_r x_6 x_7 - \mu_r x_6,\\
\dot{x}_7 &\equiv f_7 = \beta_r x_6 x_7 - \mu_r x_7.
\end{aligned} \quad (33)$$

The Jacobian for the system at the DFE is given by

$$J_{\beta_h^*=} = \begin{bmatrix}
-\mu_h & 0 & -\frac{\theta_s \beta_h \Lambda_h}{\mu_h} & -\frac{\theta_s \beta_h \Lambda_h}{\mu_h} & \theta_0 & 0 & -\frac{\beta_R \Lambda_h}{\mu_h} \\
0 & -m_1 & \frac{\theta_s \beta_h \Lambda_h}{\mu_h} & \frac{\theta_s \beta_h \Lambda_h}{\mu_h} & 0 & 0 & \frac{\beta_R \Lambda_h}{\mu_h} \\
0 & \zeta\theta_h & -m_2 & 0 & 0 & 0 & 0 \\
0 & (1-\zeta)\theta_h & 0 & -m_3 & 0 & 0 & 0 \\
0 & 0 & \phi_a & \phi_s & -m_4 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -(\eta_r - \mu_r) & -(\eta_r - \mu_r)\left(\frac{\beta_r K_0}{\eta_r} + 2\right) + \eta_r \\
0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_r K_0}{\eta_r} - \mu_r
\end{bmatrix} \quad 34$$

where $m_1 = \theta_h + \mu_h$, $m_2 = \phi_a + \mu_h$, $m_3 = \phi_s + \mu_0 + \mu_h$ and $m_4 = \theta_0 + \mu_h$.

Consider the case when $\mathcal{R}_L = 1$. Working out the value for $\beta_h = \beta_h^*$ from $\mathcal{R}_L = 1$ gives

$$\beta_h = \beta_h^* = \frac{\mu_h(\theta_h + \mu_h)(\phi_a + \mu_h)(\phi_s + \mu_0 + \mu_h))}{\theta_h \theta_s \Lambda_h [\zeta(\phi_s + \mu_0 + \mu_h) + (1-\zeta)(\phi_a + \mu_h)]} \quad (35)$$

Matrix $J_{\beta_h^*}$ possesses a right eigenvector given by $\mathbf{w} = (\omega_1, \omega_2, \ldots, \omega_7)^T$, such that

$$\begin{aligned}
\omega_1 &= -\frac{\theta_s \beta_h \Lambda_h (\omega_3 + \omega_4)}{\mu_h^2} + \frac{\theta_0 \omega_5}{\mu_h},\ \omega_2 = \omega_2 > 0,\\
\omega_3 &= \frac{\zeta \theta_h \omega_2}{\phi_a + \omega_S + \mu_h},\\
\omega_4 &= \frac{\theta_h [(1-\zeta)(\phi_a + \mu_h)]\omega_2}{(\phi_a + \mu_h)(\phi_s + \mu_0 + \mu_h)},\\
\omega_5 &= \frac{\theta_h [\phi_a \zeta(\phi_s + \mu_0 + \mu_h) + \phi_s (1-\zeta)(\phi_a + \mu_h)]\omega_2}{(\theta_0 + \mu_h)(\phi_a + \mu_h)(\phi_s + \mu_0 + \mu_h)},\\
\omega_6 &= \omega_7 = 0,
\end{aligned} \quad (36)$$

In addition, $J_{\beta_h^*}$ possesses a left eigenvector $\mathbf{v} = (\nu_1, \nu_2, \ldots, \nu_7)$ satisfying $\mathbf{v}.\mathbf{w} = \mathbf{1}$, with

$$\begin{aligned}
\nu_1 &= 0,\ \nu_2 = \nu_2 > 0,\\
\nu_3 &= \frac{\theta_s \beta_h \Lambda_h \nu_2}{\mu_h (\phi_a + \mu_h)},\\
\nu_4 &= \frac{\theta_s \beta_h \Lambda_h \nu_2}{\mu_h (\phi_s + \mu_0 + \mu_h)},\\
\nu_5 &= \nu_6 = 0,\\
\nu_7 &= -\frac{\eta_r \beta_R \Lambda_h \nu_2}{\mu_h [\beta_r K_0 (\eta_r - \mu_r) - \eta_r \mu_r]}.
\end{aligned} \quad (37)$$

Applying the *Center Manifold Theory* as espoused by [3], we calculate the related non-zero partial derivatives of the right flanks of the transformed system (33), *(appraised in the absence of infection with* $\beta_h = \beta_h^*$*)* that the related bifurcation coefficients, a and b, are given by

$$a = \sum_{k,i,j=1}^{n} \nu_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \text{and}$$

$$b = \textstyle\sum_{k,i=1}^{n} \nu_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0), \quad (38)$$

The related non-zero partial derivatives for bifurcation coefficient a for the model system (8) (or (33)) are:

$$\begin{aligned}\frac{\partial^2 f_2}{\partial x_1 \partial x_3} &= \theta_s\beta_h = \frac{\partial^2 f_2}{\partial x_3 \partial x_1},\\ \frac{\partial^2 f_2}{\partial x_1 \partial x_4} &= \theta_s\beta_h = \frac{\partial^2 f_2}{\partial x_4 \partial x_1},\\ \frac{\partial^2 f_2}{\partial x_1 \partial x_7} &= \beta_r = \frac{\partial^2 f_2}{\partial x_7 \partial x_1},\\ \frac{\partial^2 f_7}{\partial x_6 \partial x_7} &= \beta_r = \frac{\partial^2 f_7}{\partial x_7 \partial x_6}.\end{aligned} \tag{39}$$

It ensues from the above expressions, (after several algebraic calculations), that

$$\begin{aligned}a &= v_2 \sum_{i,j=1}^{7} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + v_7 \sum_{i,j=1}^{7} w_i w_j \frac{\partial^2 f_7}{\partial x_i \partial x_j}\\ &= 2\theta_s\beta_h v_2 \omega_1(\omega_3+\omega_4)\\ &= 2\theta_s\beta_h v_2(\omega_3+\omega_4)\left(-\frac{\theta_s\beta_h\Lambda_h(\omega_3+\omega_4)}{\mu_h^2}+\frac{\theta_0\omega_5}{\mu_h}\right).\end{aligned} \tag{40}$$

The related non-zero partial derivative for bifurcation coefficient b for the model system (8) (or (33)) is:

$$\begin{aligned}\frac{\partial^2 f_2}{\partial x_3 \partial \beta_h^*} &= \frac{\theta_s\beta_h\Lambda_h}{\mu_h},\\ \frac{\partial^2 f_2}{\partial x_4 \partial \beta_h^*} &= \frac{\theta_s\beta_h\Lambda_h}{\mu_h}.\end{aligned} \tag{41}$$

It ensues also from that

$$\begin{aligned}b &= v_2 \sum_{i=1}^{7} w_i \frac{\partial^2 f_2}{\partial x_i \partial \beta_h^*},\\ &= \frac{\theta_s\beta_h\Lambda_h}{\mu_h} v_2(\omega_3+\omega_4).\end{aligned} \tag{42}$$

Obviously $b > 0$ for all biologically reasonable parameter values. Thus, backward bifurcation appearers if and only if the rate at which temporary immunity wanes (θ_0), is large enough such that $a > 0$. This, therefore, implies that if the the rate at which temporaray immunity wanes is less than the quantity, (θ_0^c), the effective reproduction number then becomes a necessary and sufficient tool for promoting control measures that will lead to disease eradication.

Consequent upon the results obtained in Theorem 3.2 above, we claim the following result.

Theorem 3. (Non-existence of backward bifurcation) The model (8) (or (33)) does not experience backward bifurcation in the direction $\mathcal{R}_L = 1$, whenever $\theta_0 = 0$.

Proof: Consider the distinctive case of the model (8) with negligible rate at which temporaray immunity wanes (i.e., $\theta_0 = 0$). Then the backward bifurcation coefficient, a, in (41) reduces to:

$$a = -\frac{2\theta_s^2\beta_h^2\Lambda_h}{\mu_h^2} v_2(\omega_3+\omega_4)^2 < 0. \tag{43}$$

Thus, this study has confirmed that the existence of the rate at which temporary immunity wanes causes backward bifurcation in the epidemic dynamics of Lassa Haemorrhagic Fever.

Global Asymptotic Stability (GAS) of DFE

Consider the distinctive case of the model (8) with $\theta_0 = 0$ (i.e., removing the parameter that causes backward bifurcation as discussed above). In this case, the model reduces to

$$\begin{aligned}S_h^{'} &= \Lambda_h - \lambda_r S_h - \mu_h S_h,\\ E_h^{'} &= \lambda_r S_h - (\theta_h+\mu_h)E_h,\\ I_a^{'} &= \zeta\theta_h E_h - (\phi_a+\mu_h)I_h,\\ I_s^{'} &= (1-\zeta)\theta_h E_h - (\phi_s+\mu_0+\mu_h)I_s,\\ R_h^{'} &= \phi_a I_a + \phi_s I_s - \mu_h R_h,\\ S_r^{'} &= \eta_r N_r(1-\frac{N_r}{K_0}) - \beta_r I_r S_r - \mu_r S_r,\\ I_r^{'} &= \beta_r I_r S_r - \mu_r I_r.\end{aligned} \tag{44}$$

We claim the following result.

Theorem 4. The DFE of the model (8), without the rate at which temporary immunity wanes (i.e., $\theta_0 = 0$) is GAS in $\mathcal{D}_o$ if $\mathcal{R}_L \leq 1$ and unstable on the condition that $\mathcal{R}_L > 1$.

Proof: Consider the following Lyapunov function

$$\mathcal{U} = \mathcal{K}_1 E_h + \mathcal{K}_2 I_a + \mathcal{K}_3 I_s + \mathcal{K}_4 I_r, \tag{45}$$

where

$$\mathcal{K}_1 = \frac{\theta_h[\zeta(\phi_s++\mu_h)+(1-\zeta)(\phi_a+\mu_h)]}{\mu_h(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)}, \quad \mathcal{K}_2 = \frac{1}{\phi_a+\mu_h}, \quad (46)$$
$$\mathcal{K}_3 = \frac{1}{\phi_s+\mu_0+\mu_h} \quad \text{and} \quad \mathcal{K}_4 = \frac{1}{\mu_r},$$

with Lyapunov derivatives (where a dot represents a time derivative)

$$\dot{\mathcal{U}} = \mathcal{K}_1 \dot{E}_h + K_2 \dot{I}_a + \mathcal{K}_3 \dot{I}_s + \mathcal{K}_4 \dot{I}_r. \quad (47)$$

Substituting the right hand side of model (45) into (48) gives

$$\begin{aligned}\dot{\mathcal{U}} &= \mathcal{K}_1\lambda_r S_h \\ &+[\mathcal{K}_2\zeta\theta_h + \mathcal{K}_3(1-\zeta)\theta_h - \mathcal{K}_1(\theta_h+\mu_h)]E_h \\ &-[\mathcal{K}_2(\phi_a+\mu_h)]I_a \\ &-[\mathcal{K}_3(\phi_s+\mu_0+\mu_h)]I_a \\ &+[\mathcal{K}_4(\beta_r S_r I_r - \mu_r)]I_r, \\ &= (I_a+I_s)\theta_h\theta_s\beta_h S_h[\tfrac{\zeta(\phi_s+\mu_h)+(1-\zeta)(\phi_a+\mu_h)}{(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)}](\mathcal{R}_L-1) \\ &+\left(\tfrac{\beta_R S_r}{\mu_r}\left[\tfrac{\zeta(\phi_s+\mu_h)+(1-\zeta)(\phi_a+\mu_h)}{(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)}\right]-1\right)I_r. \end{aligned} \quad (48)$$

Substituting $(N_r - S_r)$ for I_r in (48), we obtain

$$\begin{aligned}\dot{\mathcal{U}} &= (I_a+I_s)\theta_h\theta_s\beta_h S_h[\tfrac{\zeta(\phi_s+\mu_h)+(1-\zeta)(\phi_a+\mu_h)}{(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)}](\mathcal{R}_L-1) \\ &+\left(\tfrac{\beta_R S_r}{\mu_r}\left[\tfrac{\zeta(\phi_s+\mu_h)+(1-\zeta)(\phi_a+\mu_h)}{(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)}\right]-1\right)(N_r-S_r). \end{aligned} \quad (49)$$

At DFE, $S_h \le \Lambda_h/\mu_h$ and $S_r \le N_r$. Hence

$$\therefore \dot{\mathcal{U}} \le (I_a+I_s)\tfrac{\theta_h\theta_s\beta_h\Lambda_h}{\mu_h}[\tfrac{\zeta(\phi_s+\mu_h)+(1-\zeta)(\phi_a+\mu_h)}{(\theta_h+\mu_h)(\phi_a+\mu_h)(\phi_s+\mu_0+\mu_h)}](\mathcal{R}_L-1).$$

Hence, $\dot{\mathcal{U}} \le 0$ whenever $\mathcal{R}_L \le 1$ with $\dot{\mathcal{U}} = 0$ if and only if $I_a = I_s = 0$. Hence, $\mathcal{U}$ represents a Lyapunov function in $\mathcal{D}_o$. Therefore, it ensues from *LaSalle's Invariance Principle* [8] that:

$$(E_h(t), I_a(t), I_s(t), I_r(t)) \to (0,0,0,0) \quad \text{as} \quad t \to \infty.$$

Consequently, every orbit of the equations of the model (44), with $\theta_0 = 0$, approaches the DFE of the model (44), as $t \to \infty$ for $\mathcal{R}_L \le 1$. This result shows that in a population *where there is recovery from and treatment for LHF cases*, on the condition that there is negligible temporary immunity, that is, $\theta_0 = 0$, the DFE will be globally asymptotically stable (GAS) whenever $\mathcal{R}_L \le 1$. Hence, Lassa Haemorrhagic Fever can be annihilated from the population whenever $\mathcal{R}_L \le 1$, irrespective of the initial sizes of the sub-populations.

Conclusions

A mathematical model which investigates, theoretically, the impact of reduced infection on the transmission dynamics of Lassa Haemorrhagic Fever burden in a given population was developed in this work. The computation of the effective reproduction number, $\mathcal{R}_L$, in the process of investigating the disease-free equilibrium of the model, established the local asymptotic stability of the DFE whenever $\mathcal{R}_L < 1$. Further analysis of the effective reproduction number showed that the treatment rate for the symptomatic infectious class will exert a positive influence in reducing the Lassa Haemorrhagic Fever burden in the theoretical population under investigation, regardless of the rates of the other parameters that constitute the effective reproduction number. Also, the disease-free equilibrium of the model was shown to be globally asymptotically stable a population *where there is recovery from and treatment for Lassa Haemorrhagic Fever cases*, on the condition that there is negligible temporary immunity whenever $\mathcal{R}_L < 1$.

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