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Full Length Research Article

MATHEMATICAL MODEL FOR THE TRANSMISSION DYNAMICS OF LASSA FEVER WITH CONTROL

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ABSTRACT
In this work, we proposed a mathematical model for transmission dynamics of Lassa fever by incorporating isolation and treatment as control strategies. The disease free equilibrium state of the model was obtained, and used to derive the basic reproduction number, R_0 using the next generation method. We further proved that the disease free equilibrium state is locally asymptotically stable whenever the reproduction number is less than unity. The numerical simulations revealed that the infection transmission rate constitute an essential parameter for an epidemic to occur, thus efforts should geared at bringing the infection transmission parameter to the lowest level to ensure eradication. The simulations further revealed that combined effective treatment and isolation of infected individuals are vital to eradicating the disease transmission.

Keywords: Bounded Solution, Basic Reproduction Number, Lassa Fever, Local Stability

1. INTRODUCTION

Lassa fever is a Viral Haemorrhagic Fever (VHF) caused by Lassa virus (LV). Lassa virus is an ambisense RNA virus in the Arenaviridae family and is the etiological agent of Lassa fever (Cashman *et al*, 2017) and transmitted by rats. The virus has been known since the 1950s, but the virus was not identified until 1969, when two missionary nurses died from it in the town of Lassa in Borno, Nigeria (Richmond and Baglole 2003). Lassa fever is a zoonotic disease, i.e. it can be transmitted from an infected animal to a human. Multimammate rats of the genus *Mastomys* is the reservoir host of the virus (Walker *et al*, 1975). It is associated with acute and potentially fatal haemorrhagic illness caused by Lassa virus (Hallam *et al*, 2018).

Due to the fact that certain varieties of *mastomys* often live in human homes, the virus is easily transmitted to humans. Most transmissions of the infection are from rodent to human and to a lesser extent from person-to-person. Infectious rodents continue to shed the virus throughout their lifetime and do not show clinical symptoms but excrete the virus through their urine, saliva, respiratory secretion and exposed blood vessels. Transmission from rodent to humans occurs via direct contact with infected urine, faeces, and saliva of the rodents; via contact with excretion or secretion-infected materials; or via ingestion of excretion-contaminated food (Hallam *et al*, 2018). Human-to-human transmission is possible through direct contact with infected blood or bodily fluid, urine, faeces or any other secretions from the body of an infected person. Sexual transmissions have also been reported (WHO, 2017).

Various mathematical studies have been carried out on mathematical modeling of Lassa fever transmission dynamics without any control strategies (see, for instance, James *et al,* 2015a; Onuorah *et al,* 2016; Obabiyi *et al*, 2017; Faniran, 2017; Usman and Adamu, 2018). Others assess various control strategies (see, instance, James *et al,* 2015b; Adewale *et al,* 2016; Abdulhamid and Hassan, 2018). Akinpelu and Akinwade (2018) presented a deterministic model, which focus on isolation of those with symptoms. The present study extends the Akinpelu and Akinwade (2018) study by incorporating isolation and treatment of individuals with Lassa infection. Further, this study allows the possibility of transmission of Lassa infection from human to human (only rodents to humans were considered in Akinpelu and Akinwade, 2018).

The paper is organized as follows. The mathematical model is formulated in Section 2 and analysed in Section 3. Numerical simulations and discussion of results are carried out in Section 4. The conclusive remarks are passed in section 5.

2. Model formulation

This model subdivides the human population $N_H(t)$ into four compartments of susceptible $S_H(t)$, latent (exposed) $L_H(t)$, infectious $I_H(t)$ and isolated $I_S(t)$ humans. So that;
$N_H(t) = S_H(t) + L_H(t) + I_H(t) + I_S(t)$
Also, the model subdivides the rodent population into susceptible $S_R(t)$ and infectious $I_R(t)$ rodents. So that;
$N_R = S_R(t) + I_R(t)$

2.1. Basic Assumptions

Here, we modify and extend the work by Akinpelu and Akinwande (2018). Specifically:

(i) The dynamics of the susceptible, $S_R(t)$ rodents, infected, $R_R(t)$ rodents, isolation of infected humans $I_H(t)$ are captured in our model.
(ii) We include human to human transmission and rodent to human transmission
(iii) We allow treatment of both infected and isolated compartment at the rate α_1 and α_2 respectively.
(iv) We also assume that those that treated could be re-infected and are factored back into the susceptible class for lack of permanent immunity.

The variables and parameters used in the model are defined in Table 1

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Table 1: Variables and Parameters used in the model and their description

Variable/Parameter	Description
$S_H(t)$	The number of susceptible hosts at time t
$L_H(t)$	The number of latent hosts at time t
$I_H(t)$	The number of infectious human hosts at time t
$I_S(t)$	The number of isolated human hosts at time t
$S_R(t)$	The number of susceptible rodent vectors at time t
$I_R(t)$	The number of infectious rodent vectors at time t
α_1	Infected treatment rate
α_2	Isolated treatment rate
β_1	Contact rate of rodents
β_2	Contact rate of humans
γ	Progression rate to infectious class
δ	Death rate due to disease in the human population
θ_1	Isolation rate of exposed humans
θ_2	Isolation rate of infected humans
μ_H	Natural death rate of humans
μ_R	Natural death rate of rodents
π_H	Recruitment rate of humans
π_R	Recruitment rate of rodents

From the above assumptions, definition of variables and parameters, the interactions and flow in the different compartments are as depicted in the schematic flow diagram below

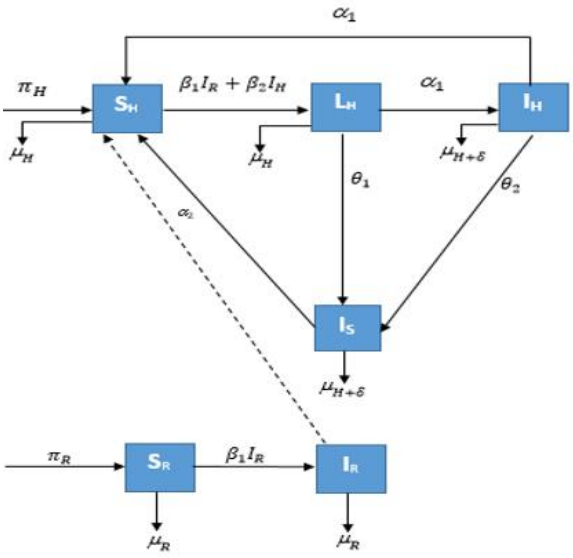

Figure 1: Schematic description of the mathematical model

2.1.1. Susceptible, Exposed, Infected and Isolated Humans

The population of susceptible humans S_H, are recruited (by birth) at the rate π_H. and treatment of the infected and isolated individuals (at the rate α_1 and α_2). It is reduced by infection, following contact with infected rodent (at the rate β_1) and infected human (at the rate β_2). The susceptible population is further reduced by natural death (at the rate μ_H). Putting all these definitions together leads to the following expression for the rate of change of the susceptible human population.

$$\frac{ds_H}{dt} = \pi_H - (\beta_1 I_R + \beta_2 I_H)S_H + \alpha_1 I_H + \alpha_2 I_S - \mu_H S_H$$

The population of Latent humans L_H is generated following infection (at the rates β_1 and β_2). They are decreased as a result of progression into the infectious class (at the rate γ), isolation (at the rate θ_1) and natural death (at the rate μ_H), so that

$$\frac{dL_H}{dt} = (\beta_1 I_R + \beta_2 I_H)S_H - \gamma L_H - \theta_1 L_H - \mu_H L_H$$

Infectious humans I_H are generated as a result of progression into the infected class from the latent class (at the rate γ). It is diminished by treatment (at the rate α_1), isolation (at the rate θ_2), disease-induced death (at the rate δ) and natural death (at the rate μ_H), so that

$$\frac{dI_H}{dt} = \gamma L_H - \alpha_1 I_H - \theta_2 I_H - \mu_H I_H - \delta I_H$$

The population of Isolated humans I_S are generated (at the rates θ_1 and θ_2). It is diminished by treatment (at the rate α_2), disease-induced death (at the rate δ) and natural death (at the rate μ_H), so that

$$\frac{dI_S}{dt} = \theta_1 L_H + \theta_2 I_H - \alpha_2 I_S - \delta I_S - \mu_H I_S$$

2.1.2. Susceptible and Infected Rodents

The population of susceptible rodents S_R are recruited (at the rate π_R). It is reduced by infection, following contact with infected rodents (at the rate β_1) and by natural death (at the rate μ_R). Thus,

$$\frac{\mathrm{d}S_R}{\mathrm{dt}} = \pi_R - \beta_1 S_R I_R - \mu_R S_R$$

The population of infected rodents is increased by infection of the susceptible rodents (at the rate β_1) and are depleted as a result of natural death (at the rate μ_R), so that

$$\frac{dI_R}{\mathrm{dt}} = \beta_1 S_R I_R - \mu_R I_R$$

2.2. Model Equations

The above assumptions and formulations leads to the following system of ordinary differential equations:

$$\frac{dS_H}{dt} = \pi_H - (\beta_1 I_R + \beta_2 I_H)S_H + \alpha_1 I_H + \alpha_2 I_S - \mu_H S_H \quad (1)$$

$$\frac{dL_H}{dt} = (\beta_1 I_R + \beta_2 I_H)S_H - \gamma L_H - \theta_1 L_H - \mu_H L_H \quad (2)$$

$$\frac{dI_H}{dt} = \gamma L_H - \alpha_1 I_H - \theta_2 I_H - \mu_H I_H - \delta I_H \quad (3)$$

$$\frac{dI_S}{dt} = \theta_1 L_H + \theta_2 I_H - \alpha_2 I_S - \delta I_S - \mu_H I_S \quad (4)$$

$$\frac{\mathrm{d}S_R}{\mathrm{dt}} = \pi_R - \beta_1 I_R S_R - \mu_R S_R \quad (5)$$

$$\frac{dI_R}{\mathrm{dt}} = \beta_1 S_R I_R - \mu_R I_R \quad (6)$$

$$N_H(t) = S_H(t) + L_H(t) + I_H(t) + I_S(t) \quad (7)$$

$$N_R = S_R(t) + I_R(t) \quad (8)$$

2.3. Basic Properties

Since the model (1) – (6) monitors both human and rodent population, it is assumed that all the variables and parameters of the model are non-negative for $t \geq 0$.

We will study the invariant region. Here we show that the solution for the model equations (1) – (6) are uniformly – bounded in the subset $\Omega \subset \mathbb{R}_+^6$.

Lemma 1:
The feasible region Ω is positively invariant and attracting with respect to the system (1) – (6).

Proof
We shall split the system (1) – (6) into two parts, namely the human component (N_H) and the rodent component (N_R), given respectively by
$N_H = S_H + L_H + I_H + I_S$
and
$N_R = S_R + I_R$
Consider the feasible region
$\Omega = \Omega_H \cup \Omega_R \in \mathbb{R}_+^4 \times \mathbb{R}_+^2$
with
$\Omega_H = \left\{(S_H, L_H, I_H, I_S) \in \mathbb{R}_+^4 : N_H \leq \frac{\pi_H}{\mu_H}\right\}$
and
$\Omega_R = \left\{(S_R, I_R) \in \mathbb{R}_+^2 : N_R \leq \frac{\pi_R}{\mu_R}\right\}$
The rate of change of the human population N_H obtained by adding the first four equations in the model (1) – (6) is given as

$$\frac{dN_H}{dt} = \pi_H - \mu_H(S_H + L_H + I_H + I_S) - \delta(I_H + I_S)$$

$$= \pi_H - \mu_H N_H - \delta(I_H + I_S) \quad (9)$$

When there is no infection, it is clear from (8) that

$$\frac{dN_H}{dt} \leq \pi_H - \mu_H N_H$$

$$\frac{dN_H}{dt} + \mu_H N_H \leq \Pi_H \quad (10)$$

Integrating (9) with respect to t where the integrating factor, $IF = e^{\int \mu_H dt} = e^{\mu_H t}$
We have

$$e^{\mu_H t} N_H = \int \pi_H e^{\mu_H t} dt + C$$

$$\Rightarrow e^{\mu_H t} N_H \leq \frac{\pi_H}{\mu_H} e^{\mu_H t} + C$$

$$N_H(t) \leq \frac{\pi_H}{\mu_H} + Ce^{-\mu_H t}$$

At $t = 0$

$$C = N(0) - \frac{\pi_H}{\mu_H}$$

$$\therefore N_H(t) \leq \frac{\pi_H}{\mu_H} + \left(N(0) - \frac{\pi_H}{\mu_H}\right) e^{-\mu_H t}$$

$$N_H(t) \leq N(0)e^{-\mu_H t} + \frac{\pi_H}{\mu_H}(1 - e^{-\mu_H t}) \quad (11)$$

Similarly, the rate of change of the rodent population N_R obtained by adding the last two equations in the model (1) – (6) is given as

$$\frac{dN_R}{dt} = \pi_R - \mu_R(S_R + I_R)$$

$$= \pi_R - \mu_R N_R \quad (12)$$

When there is no infection, it is clear from (11) that

$$\frac{dN_R}{dt} \leq \pi_R - \mu_R N_R$$

$$\frac{dN_R}{dt} + \mu_R N_R \leq \pi_R \quad (13)$$

Integrating (12) with respect to t where the integrating factor, $IF = e^{\int \mu_R dt} = e^{\mu_R t}$
We have

$$e^{\mu_R t} N_R = \int \pi_R e^{\mu_R t} dt + C$$

$$\Rightarrow e^{\mu_R t} N_R \leq \frac{\pi_R}{\mu_R} e^{\mu_R t} + C$$

$$N_R(t) \leq \frac{\pi_R}{\mu_R} + Ce^{-\mu_R t}$$

At $t = 0$

$$C = N(0) - \frac{\pi_R}{\mu_R}$$

$$\therefore N_R(t) \leq \frac{\pi_R}{\mu_R} + \left(N(0) - \frac{\pi_R}{\mu_R}\right) e^{-\mu_R t}$$

$$N_R(t) \leq N(0)e^{-\mu_R t} + \frac{\pi_R}{\mu_R}(1 - e^{-\mu_R t}) \quad (14)$$

Applying the theorem of differential inequality (Birkhof and Rota, 1982) on equations (11) and (14), we obtain
$0 \leq N_H(t) \leq \frac{\pi_H}{\mu_H}$ and $0 \leq N_R(t) \leq \frac{\pi_R}{\mu_R}$ $as\ t \to \infty$
In particular,
$N_H(t) \leq \frac{\pi_H}{\mu_H}$ and $N_R(t) \leq \frac{\pi_R}{\mu_R}$ if $N_H(0) \leq \frac{\pi_H}{\mu_H}$ and $N_R(0) \leq \frac{\pi_R}{\mu_R}$ respectively. it follows that all possible solution of the system (1) – (6) will enter the region $\Omega = \Omega_H \cup \Omega_R \in \mathbb{R}_+^4 \times \mathbb{R}_+^2$

that is,

$$\Omega = \left\{(S_H, L_H, I_H, I_S, S_R, I_R) \in \mathbb{R}_+^6 : N_H \leq \frac{\pi_H}{\mu_H}, N_R(t) \leq \frac{\pi_R}{\mu_R}\right\}$$

Thus, every solution of the model (1) – (6) with initial condition in Ω remains there for $t > 0$. It follows that Ω is positive invariant. Hence it is sufficient to consider the dynamics of the flow generated by (1) – (6) in Ω. In this region, the model can be considered as being epidemiologically and mathematically well posed.

3. MODEL ANALYSIS

The model (1) – (6) has a disease-free equilibrium (DFE) given by

$$\boldsymbol{E_0} = (S_H^*, L_H^*, I_H^*, I_S^*, S_R^*, I_R^*) = \left(\frac{\pi_H}{\mu_H}, 0,0,0, \frac{\pi_R}{\mu_R}, 0\right) \quad (15)$$

We will use the disease-free equilibrium state and the next generation operator method to compute the basic reproduction number R_0.

3.1. Basic Reproduction Number R0

The basic reproduction number or reproductive number of an infectious disease is the average number of secondary infections when one infected individual is introduced into a host population where everyone is susceptible (Diekmann *et al*, 1990; Diekmann *et al, 2010*). We use the next generation matrix approach to compute the Basic Reproduction Number R_0.

The basic reproduction number R_0 is the spectral radius of the product matrix FV^{-1}. That is, $R_0 = \rho(FV^{-1})$

The associated non-negative matrix F, for the new infective terms and the non-singular M-matrix, V, for the remaining transfer terms at the DFE are respectively given by

$$F = \begin{pmatrix} 0 & \beta_2 \frac{\pi_H}{\mu_H} & \beta_1 \frac{\pi_H}{\mu_H} \\ \gamma & 0 & 0 \\ 0 & 0 & \beta_1 \frac{\pi_R}{\mu_R} \end{pmatrix} \tag{16}$$

and

$$V = \begin{pmatrix} (\gamma + \theta_1 + \mu_H) & 0 & 0 \\ 0 & (\alpha_1 + \theta_2 + \mu_H + \delta) & 0 \\ 0 & 0 & \mu_R \end{pmatrix} \tag{17}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\gamma+\theta_1+\mu_H)} & 0 & 0 \\ 0 & \frac{1}{(\alpha_1+\theta_2+\mu_H+\delta)} & 0 \\ 0 & 0 & \frac{1}{\mu_R} \end{pmatrix} \tag{18}$$

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta_2\pi_H}{\mu_H(\alpha_1+\theta_2+\mu_H+\delta)} & \frac{\beta_1\pi_H}{\mu_H\mu_R} \\ \frac{\gamma}{(\gamma+\theta_1+\mu_H)} & 0 & 0 \\ 0 & 0 & \frac{\beta_1\pi_R}{\mu_R^2} \end{pmatrix} \tag{19}$$

It follows that the basic reproduction number, denoted by R_0, is given by (where ρ denotes the spectral radius)

$$R_0 = \sqrt{\frac{\gamma\beta_2\pi_H}{\mu_H(\gamma+\theta_1+\mu_H)(\alpha_1+\theta_2+\mu_H+\delta)}} \tag{20}$$

3.2. Local Stability Of Disease Free Equilibrium (Dfe) State

We investigate the local stability of the disease free (DFE) state by evaluating the associated Jacobian of equations (1) – (6) at the DFE state. The Jacobian matrix J for the system (1) – (6), evaluated at the disease-free equilibrium, $\mathcal{E}_0$ is given by

$$J\mathcal{E}_0 = \begin{vmatrix} -\mu_H & 0 & \beta_2\frac{\pi_H}{\mu_H}+\alpha 1 & \alpha 2 & 0 & -\beta_1\frac{\pi_H}{\mu_H} \\ 0 & -(\gamma+\theta_1+\mu_H) & \beta_3\frac{\pi_H}{\mu_H} & 0 & 0 & \beta_1\frac{\pi_H}{\mu_H} \\ 0 & \gamma & -(\alpha_1+\theta_2+\mu_H+\delta) & 0 & 0 & 0 \\ 0 & \theta_1 & \theta_2 & -(\alpha_2+\delta+\mu_H) & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_R & -\beta_1\frac{\pi_R}{\mu_R} \\ 0 & 0 & 0 & 0 & 0 & \beta_1\frac{\pi_R}{\mu_R}-\mu_R \end{vmatrix} \tag{21}$$

The disease-free equilibrium is locally asymptotically stable if and only if all of the eigenvalues of the Jacobian matrix J have negative real part (Benyah, 2007). The eigenvalues can be determined by solving the characteristic equation $|J - \lambda I| = 0$

The eigenvalues are given by

$$\begin{aligned} (-\mu_H-\lambda)\left(\left(\frac{B_1\pi_R}{\mu_R}-\mu_R\right)-\lambda\right)(-\mu_R & \\ -\lambda)(-(\alpha_2+\delta+\mu_H) & \\ -\lambda)\Big[(-(\gamma+\theta_1+\mu_H) & \\ -\lambda)\left(-(\alpha_1+\theta_2+\delta+\mu_H)-\lambda\right) & \\ -\left(\frac{\gamma\beta_2\pi_H}{\mu_H}\right)\Big] = 0 & \end{aligned}$$

$$\begin{aligned} (-\mu_H-\lambda)\left(\left(\frac{B_1\pi_R}{\mu_R}-\mu_R\right)-\lambda\right)(-\mu_R & \\ -\lambda)(-(\alpha_2+\delta+\mu_H) & \\ -\lambda)\Big[\big[(\gamma+\theta_1+\mu_H)(\alpha_1+\theta_2+\delta & \\ +\mu_H)+\lambda(\gamma+\theta_1+\mu_H) & \\ +(\alpha_1+\theta_2+\delta+\mu_H)\lambda+\lambda^2\big] & \\ -\left(\frac{\gamma\beta_2\pi_H}{\mu_H}\right)\Big] = 0 & \end{aligned}$$

that is

$$(-\mu_H-\lambda)\left(\left(-\left(\mu_R-\frac{B_1\pi_R}{\mu_R}\right)-\lambda\right)\right)(-\mu_R-\lambda)(-(\alpha_2+\delta+\mu_H)-\lambda)\left[\left[\lambda^2+\lambda\left[\gamma+\theta_1+\mu_H+\alpha_1+\theta_2+\delta+\mu_H\right]+\left((\gamma+\theta_1+\mu_H)(\alpha_1+\theta_2+\delta+\mu_H)-\left(\frac{\gamma\beta_2\pi_H}{\mu_H}\right)\right)\right]\right] = 0 \tag{22}$$

Then

$$-\mu_H = \lambda, \qquad \lambda = -\left(\mu_R - \frac{B_1\pi_R}{\mu_R}\right), \qquad \lambda = -\mu_R, \qquad \lambda = -(\alpha_2+\delta+\mu_H)$$

and

$$\left[\lambda^2+\lambda\left[\gamma+\theta_1+\mu_H+\alpha_1+\theta_2+\delta+\mu_H\right]+\left((\gamma+\theta_1+\mu_H)(\alpha_1+\theta_2+\delta+\mu_H)-\left(\frac{\gamma\beta_2\pi_H}{\mu_H}\right)\right)\right] = 0 \tag{23}$$

Obviously, four eigenvalues are negative. Now equation (23) is the characteristic equation of the sub matrix J_1, where

$$J_1 = \begin{pmatrix} -(\gamma + \theta_1 + \mu_H) & \frac{\beta_2 \pi_H}{\mu_H} \\ \gamma & -(\alpha_1 + \theta_2 + \delta + \mu_H) \end{pmatrix} \quad (24)$$

If the trace of $J_1 < 0$ and the $\det(J_1) > 0$ then the eigenvalues are negative
The trace of

$$J_1 = -(\gamma + \theta_1 + \mu_H + \alpha_1 + \theta_2 + \delta + \mu_H) < 0 \quad (25)$$

and

$$det(J_1) = (\gamma + \theta_1 + \mu_H)(\alpha_1 + \theta_2 + \delta + \mu_H) - \frac{\gamma \beta_2 \pi_H}{\mu_H} > 0 \quad (26)$$

That is,

$$1 - \frac{\gamma \beta_2 \pi_H}{\mu_H(\gamma + \theta_1 + \mu_H)(\alpha_1 + \theta_2 + \delta + \mu_H)} > 0 \quad (27)$$

$1 - R_0 > 0$ if $R_0 < 1$

Thus we proved the following lemma

Lemma 2: The DFEs of the model (1) – (6), given ε_0, locally asymptotically stable (LAS) if $R_0 < 1$ and ε_0 is unstable if $R_0 > 1$.

4. Numerical Simulations

Numerical simulations for the model (1) - (6) are carried out, using the parameters in Table 2, unless otherwise stated, to illustrate some of the analytical results established in this study.
The numerical simulations were conducted using the Runge-Kuta method (RK4) embedded in MATLAB.

4.1. Baseline Parameter Values

We show a baseline table for the parameters used in this model. The sources are also stated.

Table 2: Baseline Parameter values for equations (1) – (6)

Parameters	Baseline value	Reference
α_1	0.18	(Richmond and Bagole, 2003)
α_2	0.18	(Richmond and Bagole, 2003)
β_1	0.43	(Abdulhamid & Hussaini, 2018)
β_2	0.4	(Abdulhamid & Hussaini, 2018)
γ	0.7869	(Richmond & Bagole, 2003)
δ	0.1133	(NCDC, 2018)
θ_1	0.5	(Onuorah *et al.* 2016)
θ_2	0.5	Assumed
μ_H	0.018182	(Abdulhamid & Hussaini, 2018)
μ_R	0.1858	(Ogabi, 2012)
π_H	0.038	(Abdulhamid & Hussaini, 2018)
π_R	0.05	(Ogabi, 2012)

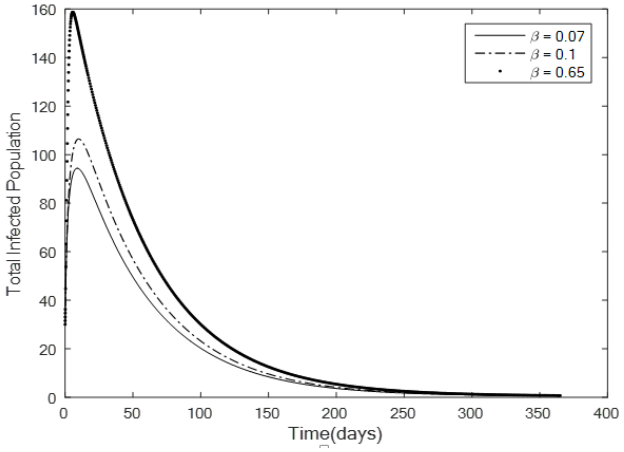

Figure 3: Graph of infected individuals with Lassa virus without human to human transmission and controls

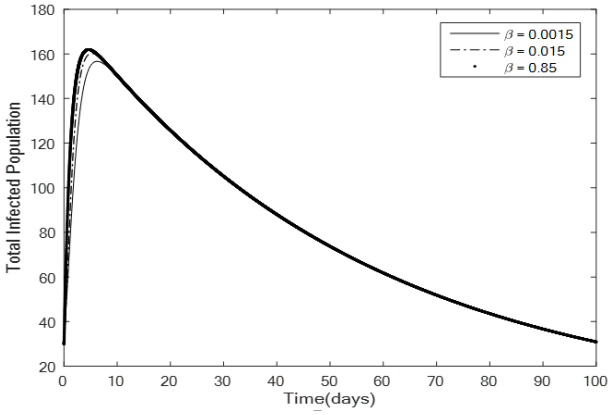

Figure 4: Graph of infected individuals varying human to human transmission rate without control $R_0 = 0.02080$ for $\beta = 0.0015$, $R_0 = 0.05210$ for $\beta = 0.015$ and $R_0 = 2.95295$ for $\beta = 0.85$

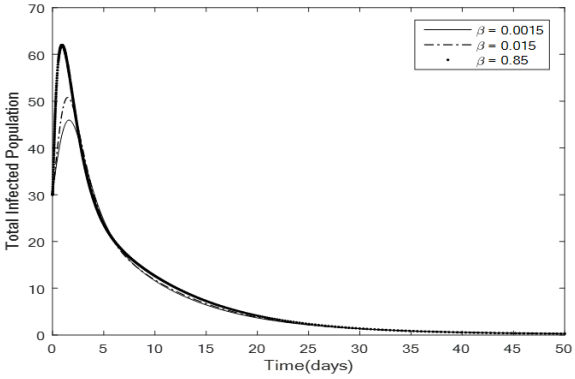

Figure 5: Graph of infected individuals varying human to human transmission with control $(\alpha = 0.18; \theta = 0.5)$. $R_0 = 5.25476\,x\,10^{-4}$ for $\beta = 0.0015$, $R_0 = 5.21000\,x\,10^{-3}$ for $\beta = 0.015$, $R_0 = 0.29515$ for $\beta = 0.85$.

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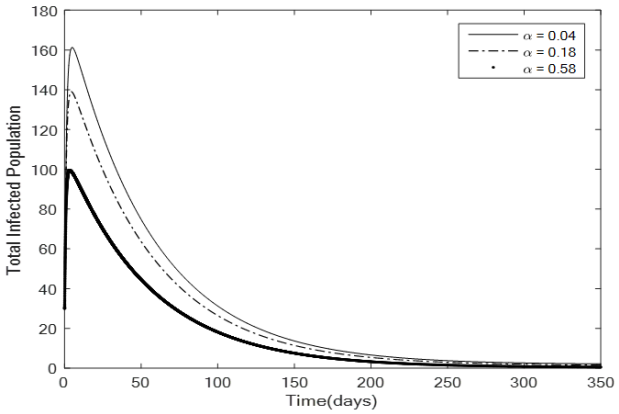

Figure 6: Graph of infected individuals varying treatment rate without isolation ($\theta = 0$) $R_0 = 1.11054$ for $\alpha = 0.04$, $R_0 = 1.00120$ for $\alpha = 0.18$ and $R_0 = 1.07166$ for $\alpha = 0.58$

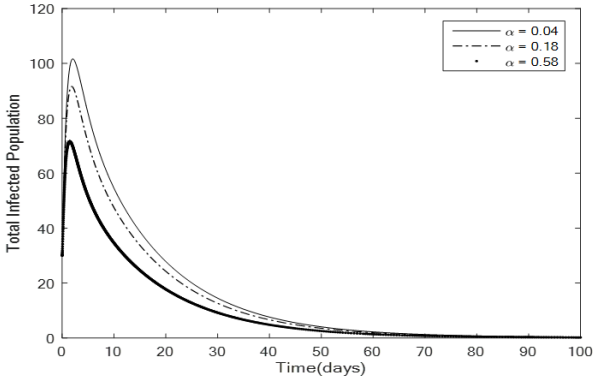

Figure 7: Graph of infected individuals varying treatment in the presence of isolation ($\theta = 0.18$) $R_0 = 0.86641$ for $\alpha = 0.04$, $R_0 = 0.78814$ for $\alpha = 0.18$ and $R_0 = 0.64503$ for $\alpha = 0.58$

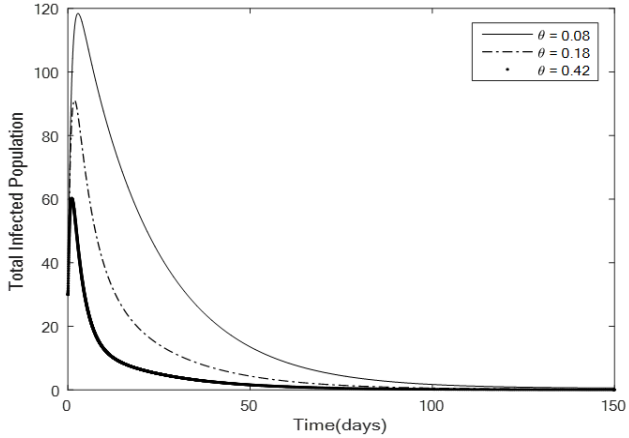

Figure 8: Effect of isolation on infected individuals with treatment $R_0 = 0.94238$ for $\theta = 0.08$, $R_0 = 0.86795$ for $\alpha = 0.18$ and $R_0 = 0.74312$ for $\beta = 0.42$

4.2. DISCUSSION OF RESULTS

This section discusses the analytical and numerical results of the study. The qualitative analysis of the model solution is bounded and the local as well as global stability of the disease free equilibrium (DFE) state was established. The Lassa fever model has a locally asymptotically stable (LAS) disease-free equilibrium (DFE) whenever the basic reproduction number, R_0, is less than unity. The basic reproduction number, given by $R_0 = \sqrt{\frac{\gamma\beta_2\pi_H}{\mu_H(\gamma+\theta_1+\mu_H)(\alpha_1+\theta_2+\mu_H+\delta)}}$ determines whether the disease can spread or not. The numerical results as presented in Figures 3 to 8 of section 4 are discussed as follows:

Figure 3 shows increasing prevalence of Lassa fever with increasing rodent infection transmission rates ($\beta_1 = 0.07, 0.01, 0.65$) without human to human transmission and in the absence of treatment and isolation. With the basic reproduction number, $R_0 < 1$ in each case, shows convergence of the solution profile to the disease free equilibrium (DFE). Thus for an effective preventive strategy, effort should be geared at reducing the infection transmission rate.

In figure 4, we considered rodents to human and human to human transmission without controls. By keeping the rodent infection transmission fixed and varying human to human transmission rates ($\beta_2 = 0.0015; 0.015; 0.85$) with the basic reproduction number, $R_0 < 1$ in each case, shows convergence of the solution profile to the disease free equilibrium (DFE).

Figure 5 exhibits a decreasing prevalence of Lassa fever infection with increasing infection transmission rates ($\beta_2 = 0.0015; 0.015; 0.85$). This Figure reveals that with treatment ($\alpha = 0.18$) and isolation ($\theta = 0.5$) of the infected humans, the disease can be eradicated in the shortest possible time.

Figure 6 shows decreasing prevalence of Lassa fever infection with increasing treatment rates ($\alpha = 0.04, 0.18, 0.58$) without isolation. The stronger the treatment rate, the shorter it takes for the eradication of the diseases. The implication of this result is that effective treatment of infected humans can be useful intervention for control and eradication of Lassa fever

Figure 7 also reveals a decreasing prevalence of Lassa fever infection in humans with increasing treatment rates ($\alpha = 0.04, 0.18, 0.58$) and isolation rate ($\theta = 0.18$). This Figure further shows that with a combination of treatment and isolation of infected humans the disease can be eradicated within a shorter period than only treatment of infected humans.

Figure 8 shows a decreasing prevalence of Lassa fever infection with increasing isolation rate ($\theta = 0.08, 0.18, 0.42$) and treatment. Thus a combination of isolation and treatment of infected individuals as a control strategy is effective for control and eradication of Lassa fever.

5. Conclusion

In this work, we developed a mathematical model to study the transmission dynamics of Lassa fever. This study modified and extended the model by Akinpelu and Akinwande (2018) by incorporating isolation and treatment as control strategies. The disease free equilibrium states were obtained and the basic reproduction number R_0 was computed using the next generation method. The result of the model analysis further shows that the

disease free equilibrium state was locally asymptotically stable if the reproductive number, $R_0 < 1$ and unstable if $R_0 > 1$.

Also, we conducted numerical simulations using Runge-Kutta 4th order method which is embedded in MATLAB. Numerical simulations of the model revealed that the infection transmission rates (rodent to human and human to human) constitutes an essential key to preventive strategies against pandemics. Numerical simulation further shows that effective treatment of infected human is a good control strategy for Lassa fever infection but combining effective isolation and treatment of infected humans is crucial as a control measure against any pandemics.

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