

**MATHEMATICAL MODEL FOR LASSA FEVER  
TRANSMISSION DYNAMICS WITH VARIABLE  
HUMAN AND RESERVOIR POPULATION**

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**Abstract:** A new mathematical model for Lassa fever is presented with focus on two populations: Humans and rodents. Maximum principle theorem is used to establish the positivity and the boundedness results of solutions. Conditions are derived for existence of disease free and endemic equilibria, and stabilities analysed. A threshold parameter  $R_0(a_i)$  exists and the disease can persist if and only if  $R_0(a_i)$  exceeds 1. Finally, numerical simulations of the model using a set of reasonable parameter values are carried out to investigate the effectiveness of diagnostic factors. This study suggests that early diagnostic (early treatment) of infected humans, maintaining hygienic environment, use of new needle when taking injection and control of the rodent carrying the virus are the best strategies against the spread of the disease.

**Key Words:** mathematical model, rodent, stability, reproduction number

## 1. Introduction

Lassa fever is an acute viral zoonotic illness caused by Lassa virus, an arenavirus know to be responsible for a severe haemorrhagic fever characterised by fever, muscle aches, sore throat, nausea, vomiting, chest and abdominal pain [4]. The virus exhibits persistent asymptomatic infection with profuse urinary virus ex-

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cretion in *Mastomys natalensis*, ubiquitous and highly commensal rodent host [15, 16]. Infection peak between January and May during the dry season - but cases are seen year round [23]. The incubation period of Lassa virus haemorrhagic fever is between 3-21 days [12]. According to estimation Lassa virus is responsible for 100,000-300,000 infections and approximately 5000 deaths annually [5].

Infected rodents remain carriers throughout their life and do not show clinical symptoms but excrete the virus through the urine, saliva, respiratory secretion and exposed blood vessel through micro and macro trauma [15]. Transmission to man is through faecal oral route or respiratory tract by inhaling contaminated air containing the virus or when infected blood touches bruised skin or by sexual intercourse. Person-to-person transmission of Lassa fever can also occur through contaminated medical equipment, such as reused needles [8].

The major and most common lesion of Lassa fever in humans occurs in the liver [6, 10, 25, 26]. Ribavirin the antiviral drug is effective in the treatment of Lassa fever but only if administered early in the course of illness [13]. Interestingly, when infected human recovered from illness they have permanent immunity (they are not susceptible again). The highest incidence of Lassa fever appears to be in area of eastern sierra Leone, south-eastern Guinea, northern Liberia, central and southern Nigeria [3,9,10 16,]. In spite of the great progress made in recent years in the understanding of the life cycle of arenaviruses, including Lassa virus, and the new insight gained into the pathogenesis and molecular epidemiology of Lassa fever, as well as the development of the state-of-the-art technologies for the diagnosing this life threatening disease, Lassa fever has been reported in Burkina faso, Ivory coast, Ghana, Senegal, Gambia and Mali [12]. The notion that the Lassa virus is endemic in larger areas of west Africa was further supported by the results of investigation of an imported case of Lassa fever in Germany in 2000 [11, 22].

To put this research into proper perspective, we briefly give an account of some existing literatures on mathematical study of Lassa fever. Okuonghae D and Okuonghae R [19] discussed a mathematical model for the transmission of Lassa fever. Steady states of their model were examined for epidemic and endemic situation. The results of their model show that the interim control of the rodents carrying the virus and some isolation policy on the infected individuals are the best strategies against the spread of the disease.

Bawa et al [1] developed a mathematical model for Lassa fever transmission dynamics in two interacting population. They obtained the basic reproduction number and stability of the disease free equilibrium was established. The results of their work suggest that every effort must be put in place by all concerned to

prevent the virus infection by reducing reproduction number.

James et al [14] formulated a mathematical model for Lassa fever transmission dynamics. They obtained the basic reproduction number which can be used to control the transmission dynamics of the disease and conditions for local stability of the disease free equilibrium was established.

Onuorah, Akinwande et al [20] developed a mathematical model for Lassa fever as a six dimensional system of nonlinear ordinary differential equation with rigorous analyses. The results of their analysis and numerical simulation show the effects of the control parameters on the various compartments of the model and conclude that if the basic reproduction number is low the disease will still continue to spread. So, they suggest that further study of the endemic equilibrium have to be done which will indicate if reducing  $R_0$  is not enough to ensure the containment of the virus. Lassa fever model presented in this study incorporates diagnostic factor,  $\gamma(a_i)\alpha_1(a_i)E_h(t, a_i)$  (since early diagnostic enables treatment) which are critical for prediction and the need of extensions to enhance their predictive power for decision support; and a new way which subdivides rodent compartment called susceptible, exposed and infectious rodent with discrete age structure denoted by  $S_r(t, e_j)$ ,  $E_r(t, e_j)$  and  $I_r(t, e_j)$  respectively. This is done based on the assumption that susceptible rodent becomes exposed and then progress to infectious stage when they share unprotected storage of garbage, food stuff and water with infectious rodents or from inhalation of aerosols from urine and faeces. (See [1,14,19,20] etc for existing models to verify our contributions).

## 2. Model Formulation

We present a new mathematical model to study the transmission and spread of Lassa fever in two interacting population of humans and rodents. The model subdivides the total human population size at time  $t$  and discrete age  $a_i$  denoted by  $N_h(t, a_i)$  with  $i = 0, 1, 2, \dots, L$  and  $a_L$  is the maximum age of humans in the population, into susceptible humans  $S_h(t, a_i)$ , exposed humans  $E_h(t, a_i)$ , infected humans  $I_h(t, a_i)$  and recovered humans  $R_h(t, a_i)$ . Hence we have  $N_h(t, a_i) = S_h(t, a_i) + E_h(t, a_i) + I_h(t, a_i) + R_h(t, a_i)$ . A loss of individuals is as a result of infection

$$\left( \frac{\rho(a_i)\sigma_1(e_j)I_r(t, e_j) + \eta(a_i)\sigma_2(a_i)I_h(t, a_i) + \theta(e_j) + \kappa(a_i)}{N_h(t, a_i)} \right) S_h(t, a_i)$$

and natural death  $\mu_h(a_i)S_h(t, a_i)$ . The exposed human gain individuals through infection

$$\left( \frac{\rho(a_i)\sigma_1(e_j)I_r(t, e_j) + \eta(a_i)\sigma_2(a_i)I_h(t, a_i) + \theta(e_j) + \kappa(a_i)}{N_h(t, a_i)} \right) S_h(t, a_i)$$

and loses individual when they become infected  $\epsilon_h(a_i)E_h(t, a_i)$  and to natural death  $\mu_h(a_i) \times E_h(t, a_i)$ . The infected human  $I_h(t, a_i)$  gain individuals when exposed individuals becomes infected and loses individual when they die  $\mu_h(a_i)I_h(t, a_i)$  and disease induced death  $\delta_h(a_i)I_h(t, a_i)$ . After some time, exposed and infected human recovers and moves to the recovered class  $R_h(t, a_i)$ . However recovered human has permanent immunity and never go back to susceptible class again. A loss of individuals is as a result of natural death  $\mu_h R_h(t, a_i)$ . It is assumed that the new birth of susceptible human  $S_h(t, a_i)$  are susceptible,  $\theta(e_j) \propto \frac{C_v}{K_v}$  and  $\theta(e_j)$  is generated from urine and faeces of infectious rodents, where  $C_v$  is the amount of virus in air and  $K_v$  is the saturation of virus in air. Similarly,  $\kappa(a_i)$  is generated from blood of infectious individuals,  $\kappa(a_i) \propto \frac{A_v}{S_v}$ , where  $A_v$  is the amount of virus in needle and  $S_v$  is the saturation of virus in needle.

In similar manner, we subdivides the total rodent population size at time  $t$  and discrete age  $e_j$  denoted by  $N_r(t, e_j)$  with  $j = 0, 1, 2, \dots, T$  and  $e_T$  is the maximum age of rodents in the population, into susceptible rodents  $S_r(t, e_j)$ , exposed rodents  $E_r(t, e_j)$  and infected rodents  $I_r(t, e_j)$ . Hence we have  $N_r(t, e_j) = S_r(t, e_j) + E_r(t, e_j) + I_r(t, e_j)$ . Susceptible rodent class  $S_r(t, e_j)$  gain more individual into rodent population by input rate  $\Lambda_r(e_j)$ , while it loses rodents through natural death  $\mu_r(e_j)S_r(t, e_j)$ , hunting  $\delta_r(e_j)S_r(t, e_j)$  and infection

$$\left( \frac{\beta(e_j)\sigma_1(e_j)I_r(t, e_j) + \theta(e_j)}{N_r(t, e_j)} \right) S_r(t, e_j).$$

Transmission of Lassa virus to susceptible rodents occurs when they share unprotected storage of garbage, food stuff and water with infected rodents or from inhalation of aerosols from urine. When a susceptible rodent interacts with infectious rodent, the virus enters the rodent with probability  $\beta(e_j)$  and therefore the susceptible go to the exposed class  $E_r(t, e_j)$ . The exposed rodent then becomes infectious and enters the class  $I_r(t, e_i)$  after a given time. It is assumed that the recruitment rate of rodent is greater than rodent's number of death at initial time ( $\Lambda_r(e_j) \geq \mu_r(e_j)N_r(0, e_j)$ ). In this study, it is assumed that individuals who recovered from Lassa fever will never go back to susceptible class again (they remain recovered for life). The assumptions above suggest that Lassa virus can only be transmitted from: (i)human to human (ii)rodent

to human (iii) rodent to rodent, and as a result, we have the following system of nonlinear ordinary differential equations.

$$\begin{aligned} & \frac{dS_h(t, a_i)}{dt} \\ = & \Lambda_h(a_i) - \sum_{i=0}^L \sum_{j=0}^T \left( \frac{\rho(a_i)\sigma_1(e_j)I_r(t, e_j) + \eta(a_i)\sigma_2(a_i)I_h(t, a_i) + \theta(e_j) + \kappa(a_i)}{N_h(t, a_i)} \right) \\ & \times S_h(t, a_i) - \mu_h(a_i)S_h(t, a_i) \quad (2.1) \end{aligned}$$

$$\begin{aligned} & \frac{dE_h(t, a_i)}{dt} \\ = & \sum_{i=0}^L \sum_{j=0}^T \left( \frac{\rho(a_i)\sigma_1(e_j)I_r(t, e_j) + \eta(a_i)\sigma_2(a_i)I_h(t, a_i) + \theta(e_j) + \kappa(a_i)}{N_h} \right) \\ & \times S_h(t, a_i) - (\gamma(a_i)\alpha_1(a_i) + \epsilon_h(a_i) + \mu_h(a_i))E_h(t, a_i) \quad (2.2) \end{aligned}$$

$$\frac{dI_h(t, a_i)}{dt} = \sum_{i=0}^L \epsilon_h(a_i)E_h(t, a_i) - (\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))I_h(t, a_i) \quad (2.3)$$

$$\begin{aligned} & \frac{dR_h(t, a_i)}{dt} \\ = & \sum_{i=0}^L \gamma(a_i)\alpha_1(a_i)E_h(t, a_i) + \psi(a_i)\alpha_2(a_i)I_h(t, a_i) - \mu_h(a_i)R_h(t, a_i) \quad (2.4) \end{aligned}$$

$$\begin{aligned} \frac{dS_r(t, e_j)}{dt} = & \Lambda_r(e_j) - \sum_{j=0}^T \left( \frac{\beta(e_j)\sigma_1(e_j)I_r(t, e_j) + \theta(e_j)}{N_r(t, e_j)} \right) S_r(t, e_j) \\ & - (\mu_r(e_j) + \delta_r(e_j))S_r(t, e_j) \quad (2.5) \end{aligned}$$

$$\begin{aligned} \frac{dE_r(t, e_j)}{dt} = & \sum_{j=0}^T \left( \frac{\beta(e_j)\sigma_1(e_j)I_r(t, e_j) + \theta(e_j)}{N_r(t, e_j)} \right) S_r(t, e_j) \\ & - (\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j))E_r(t, e_j) \quad (2.6) \end{aligned}$$

$$\frac{dI_r(t, e_j)}{dt} = \sum_{j=0}^T \epsilon_r(e_j)E_r(t, e_j) - (\mu_r(e_j) + \delta_r(e_j))I_r(t, e_j) \quad (2.7)$$

## 2.1. Existence and Positivity of Solution

Since our model monitors changes in the human and rodent populations and the parameters are assumed to be nonnegative for all  $t \geq 0$ , therefore the system of equation (2.1)-(2.7) will be analyzed in a feasible region  $\mathcal{R}$  of biological interest.

**Theorem 2.1.** *The feasible region  $\mathcal{R}$  defined by*

$$\{S_h(t, a_i), E_h(t, a_i), I_h(t, a_i), R_h(t, a_i), S_r(t, e_j), E_r(t, e_j), I_r(t, e_j) \in \mathcal{R}^7 :$$

$$N_h(0, a_i) \leq N_h(t, a_i) \leq \sum_{i=0}^L \frac{\Lambda_h(t, a_i)}{\mu_h(a_i)}, N_r(0, e_j) \leq N_r(t, e_j) \leq \sum_{j=0}^T \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)} \}$$

with initial conditions  $S_h(0, a_i) \geq 0, E_h(0, a_i) \geq 0, I_h(0, a_i) \geq 0, R_h(0, a_i) \geq 0, S_r(0, e_j) \geq 0, E_r(0, e_j) \geq 0, I_r(0, e_j) \geq 0$  is positive invariant for system (2.1)-(2.7).

*Proof.* If the total population size is given by  $N_h(t, a_i) = S_h(t, a_i) + E_h(t, a_i) + I_h(t, a_i) + R_h(t, a_i)$  and the total size of rodent population is

$$N_r(t, e_j) = S_r(t, e_j) + E_r(t, e_j) + I_r(t, e_j).$$

Then one see from (2.1)-(2.7) that

$$\frac{dN_h(t, a_i)}{dt} \leq \Lambda_h(a_i) - \sum_{i=0}^L \mu_h(a_i) N_h(t, a_i) \quad (2.8)$$

$$\frac{dN_r(t, e_j)}{dt} \leq \Lambda_r(e_j) - \sum_{j=0}^T (\mu_r(e_j) + \delta_r(e_j)) N_r(t, e_j) \quad (2.9)$$

solving the differential inequalities (2.8) and (2.9) one after the other gives

$$N_h(t, a_i) e^{\mu_h(a_i)t} \leq \sum_{i=0}^L N_h(0, a_i) + \frac{\Lambda_h(a_i)}{\mu_h(a_i)} e^{\mu_h(a_i)t} - \frac{\Lambda_h(a_i)}{\mu_h(a_i)}$$

so that

$$N_h(t, a_i) \leq \sum_{i=0}^L N_h(0, a_i) e^{-\mu_h(a_i)t} + \frac{\Lambda_h(a_i)}{\mu_h(a_i)} - \frac{\Lambda_h(a_i)}{\mu_h(a_i)} e^{-\mu_h(a_i)t}$$

this implies

$$N_h(t, a_i) \leq \sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)} (1 - e^{-\mu_h(a_i)t}) + N_h(0, a_i)e^{-\mu_h(a_i)t}$$

and

$$N_r(t, e_j)e^{(\mu_r(e_j)+\delta_r(e_j))t} \leq \sum_{j=0}^T N_r(0, e_j) + \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)} e^{(\mu_r(e_j)+\delta_r(e_j))t} - \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)}$$

so that

$$N_r(t, e_j) \leq \sum_{j=0}^T N_r(0, e_j)e^{-(\mu_r(e_j)+\delta_r(e_j))t} + \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)} - \frac{\Lambda_r(e_j)}{\mu_r(e_j)} e^{-(\mu_r(e_j)+\delta_r(e_j))t}$$

this implies

$$N_r(t, e_j) \leq \sum_{j=0}^T \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)} (1 - e^{-(\mu_r(e_j)+\delta_r(e_j))t}) + N_r(0, e_j)e^{-(\mu_r(e_j)+\delta_r(e_j))t}$$

Taking the limits as  $t \rightarrow \infty$  gives

$$N_h(t, a_i) \leq \sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)}$$

and

$$N_r(t, e_j) \leq \sum_{j=0}^T \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)}.$$

Thus the following feasible region

$$\mathcal{R} = \{S_h(t, a_i), E_h(t, a_i), I_h(t, a_i), R_h(t, a_i), S_r(t, e_j), E_r(t, e_j), I_r(t, e_j) \in \mathcal{R}^7 :$$

$$N_h(t, a_i) \leq \sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)}, N_r(t, e_j) \leq \sum_{j=0}^T \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)}. \quad \square$$

**Theorem 2.2.** Assuming that  $N_h^2(t, a_i)$  and  $N_h^1(t, a_i)$  are super-solution and sub-solution for human and  $N_r^2(t, e_j)$  and  $N_r^1(t, e_j)$  for rodent respectively and are twice differentiable. Then, the solutions  $S_h(t, a_i)$ ,  $E_h(t, a_i)$ ,  $I_h(t, a_i)$ ,  $R_h(t, a_i)$ ,  $S_r(t, e_j)$ ,  $E_r(t, e_j)$ ,  $I_r(t, e_j)$  of system (2.1)- (2.7) with non-negative initial conditions in  $\mathcal{R}$ , remain non-negative in  $\mathcal{R}$  and there is no explosion of the population for all  $t > 0$ .

*Proof.* Adding (2.1)-(2.4) gives

$$\frac{dN_h(t, a_i)}{dt} = \Lambda_h(a_i) - \sum_{i=0}^L \mu_h N_h(t, a_i) - \delta_h I_h(t, a_i) \quad (2.10)$$

Let  $N_h^2(t, a_i)$  be a super-solution and  $N_h^1(t, a_i)$  be a sub-solution, then

$$\begin{aligned} \frac{dN_h^2(t, a_i)}{dt} &> \Lambda_h(a_i) - \sum_{i=0}^L \mu_h N_h^2(t, a_i) - \delta_h I_h(t, a_i) \\ \frac{dN_h^1(t, a_i)}{dt} &< \Lambda_h(a_i) - \sum_{i=0}^L \mu_h N_h^1(t, a_i) - \delta_h I_h(t, a_i) \end{aligned} \quad (2.11)$$

$$-\frac{dN_h^2(t, a_i)}{dt} < -\Lambda_h(a_i) + \sum_{i=0}^L \mu_h(a_i) N_h^2(t, a_i) + \delta_h I_h(t, a_i) \quad (2.12)$$

combining (2.11) and (2.12) gives

$$\begin{aligned} \frac{d}{dt}(N_h^1(t, a_i) - N_h^2(t, a_i)) &< \sum_{i=0}^L \mu_h N_h^2(t, a_i) - \mu_h N_h^1(t, a_i) \\ &< \sum_{i=0}^L -\mu_h(N_h^1(t, a_i) - N_h^2(t, a_i)). \end{aligned} \quad (2.13)$$

Let there exists a function  $z$  which satisfies the inequality (2.13). Then such a function  $z$  is set as  $e^{-(c+1)}(N_h^2(t, a_i) - N_h^1(t, a_i))$  where  $c$  is yet to be determined.



Since  $N_h^1(t, a_i)$  and  $N_h^2(t, a_i)$  are bounded functions, it is rather easy to obtain the constant  $c$ . From the Lipschitz condition of

$$f(N_h(t, a_i)) = \Lambda_h(a_i) - \sum_{i=0}^L \mu_h(a_i)N_h(t, a_i) - \delta_h(a_i)I_h(t, a_i),$$

we have

$$|f(N_h^2(t, a_i) - f(N_h^1(t, a_i))| \leq c|N_h^2(t, a_i) - N_h^1(t, a_i)|.$$

Now suppose  $N^2(t, a_i)$  and  $N_h^1(t, a_i)$  are twice continuously differentiable, then the inequality:  $\frac{d^2}{dt^2} \{e^{-(c+1)t}(N_h^2(t, a_i) - N_h^1(t, a_i))\} < (c + 1 - \mu_h(a_i))z'$  holds whenever  $z$  is positive. A little bit of algebra proves that if  $z$  is negative, it follows that

$$\frac{d^2z}{dt^2} < -z. \tag{2.14}$$

A minimum of function  $z$  satisfy  $\frac{d^2z}{dt^2} > 0$ . Inequality (2.14) proves that this minimum cannot be reached for a negative value of  $z$ . it follows that  $N_h^2(t, a_i) - N_h^1(t, a_i)$  is a positive function. Let us apply the result to feasible region.

Assume that the initial population is less than  $\sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)}$  i.e  $N_h(0, a_i) \leq$

$N_h(t, a_i) \leq \sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)}$  and let  $N_h(t, a_i)$  be the solution. Function  $N_h(0, a_i) \equiv 0$

is a sub-solution and function  $N_h(t, a_i) \equiv \sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)}$  is a super-solution. Solu-

tion  $N_h(t, a_i)$  can be considered both as a sub-solution and super-solution. An application of maximum principle [27] to the couples  $(N_h^1(t, a_i), N_h^2(t, a_i)) = (N(0, a_i), N_h(t, a_i))$  and  $(N_h^1(t, a_i), N_h^2(t, a_i)) = (N_h(t, a_i), \frac{\Lambda_h(a_i)}{\mu_h(a_i)})$  shows that

$$N_h(0, a_i) \leq N_h(t, a_i) \leq \sum_{i=0}^L \frac{\Lambda_h(a_i)}{\mu_h(a_i)}.$$

Similarly, adding (2.5)-(2.7) gives

$$\frac{dN_r(t, e_j)}{dt} = \Lambda_r(e_j) - \sum_{j=0}^T (\mu_r(e_j) + \delta_r(e_j))N_r(t, e_j)$$

then,  $N_r(0, e_j) \leq N_r(t, e_j) \leq \sum_{j=0}^T \frac{\Lambda_r}{\mu_r + \delta_r}$  holds using similar argument. This

shows that our system of equation (2.1)-(2.7) is well-posed and there is no explosion of the population, hence the proof of our theorem.  $\square$

### 3. Equilibrium and Stability Analysis

In this section, equilibrium and stability analysis of the model are discussed. When modelling infectious diseases, the most important issue that arises is whether the disease spread could attain pandemic level or it could be wiped out. To have a better understanding of the disease, equilibrium and stability analysis is performed.

#### 3.1. Stability of the Disease Free Equilibrium

Disease-free equilibrium points is a steady-state solution where there is no Lassa fever infection. Solving the system of equation (2.1)-(2.7) in the absence of disease, we obtain the following equilibrium point,

$$\pi_0 = \left( \frac{\Lambda_h(a_i)}{\mu_h(a_i)}, 0, 0, 0, \frac{\Lambda_r(e_j)}{\mu_r(e_j) + \delta_r(e_j)}, 0, 0 \right) \quad (3.1)$$

We obtain a basic reproduction number  $R_0$  by expressing (2.1)-(2.7) as the difference between the rate of new infections in each infected compartments denoted by  $F$  and the rate of transfer between each infected compartments denoted by  $G$  (see [7,18, 24]):

$$\begin{bmatrix} \frac{dE_h(t, a_i)}{dt} \\ \frac{dI_h(t, a_i)}{dt} \\ \frac{dE_r(t, e_j)}{dt} \\ \frac{dI_r(t, e_j)}{dt} \end{bmatrix} = F - G = \begin{bmatrix} \sum_{i=0}^L \sum_{j=0}^T \left( \frac{\rho(a_i)\sigma_1(e_j)I_r(t, e_j) + \eta(a_i)\sigma_2(a_i)I_h(t, a_i) + \theta(e_j) + \kappa(a_i)}{N_h(t, a_i)} \right) S_h(t, a_i) \\ 0 \\ \sum_{j=0}^T \left( \frac{\beta(e_j)\sigma_1(e_j)I_r(t, e_j) + \theta(e_j)}{N_r(t, e_j)} \right) S_r(t, e_j) \\ 0 \end{bmatrix}$$

$$- \begin{bmatrix} \sum_{i=0}^L (\epsilon_h(a_i) + \gamma(a_i)\alpha_1(a_i) + \mu_h(a_i))E_h(t, a_i) \\ \sum_{i=0}^L -\epsilon_h(a_i)E_h(t, a_i) + (\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))I_h(t, a_i) \\ \sum_{j=0}^T (\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j))E_r(t, e_j) \\ \sum_{j=0}^T -\epsilon_r(e_j)E_r(t, e_j) + (\mu_r(e_j) + \delta_r(e_j))I_r(t, e_j) \end{bmatrix}.$$

The Jacobian matrices  $J_F$  and  $J_G$  of  $F$  and  $G$  are found about  $\pi_0$

$$T = \begin{bmatrix} V_{11} & \sum_{i=0}^L \frac{\eta(a_i)\sigma_2(a_i)}{\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i)} & V_{13} & \sum_{i=0}^L \sum_{j=0}^T \frac{\rho(a_i)\sigma_1(e_j)}{\mu_r(e_j) + \delta_r(e_j)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & V_{33} & \sum_{j=0}^T \frac{\beta(e_j)\sigma_1(e_j)}{\mu_r(e_j) + \delta_r(e_j)} \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

where,

$$T = J_F J_G^{-1}$$

$$V_{11} = \sum_{i=0}^L \frac{\eta(a_i)\sigma_2(a_i)\epsilon_h(a_i)}{(\gamma(a_i)\alpha_1(a_i) + \epsilon_h(a_i) + \mu_h(a_i))(\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))}$$

$$V_{13} = \sum_{i=0}^L \sum_{j=0}^T \frac{\rho(a_i)\epsilon_r(e_j)\sigma_1(e_j)}{(\mu_r(e_j) + \epsilon_r(e_j) + \delta_r(e_j))(\mu_r(e_j) + \delta_r(e_j))}$$

$$V_{33} = \sum_{j=0}^T \frac{\epsilon_r(e_j)\beta(e_j)\sigma_1(e_j)}{(\mu_r(e_j) + \epsilon_r(e_j) + \delta_r(e_j))(\mu_r(e_j) + \delta_r(e_j))}$$

$R_0$  is the maximum eigenvalue of  $T$

$$R_0(a) =$$

$$\sum_{i=0}^L \frac{\eta(a_i)\sigma_2(a_i)\epsilon_h(a_i)}{(\gamma(a_i)\alpha_1(a_i) + \epsilon_h(a_i) + \mu_h(a_i))(\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))}. \quad (3.2)$$

**Theorem 3.1.** *The disease-free equilibrium for the system (2.1)-(2.7) is locally asymptotically stable if  $R_0(a_i) < 1$  and unstable if  $R_0(a_i) > 1$ .*

*Proof.* The Jacobian of system (2.1)-(2.7) evaluated at the disease free equilibrium point is

$$J_1(\pi_0) = \begin{bmatrix} -\mu_h(a_i) & 0 & \sum_{i=0}^L -\eta(a_i)\sigma_2(a_i) & 0 & 0 & 0 & -E \\ 0 & A & \sum_{i=0}^L \eta(a_i)\sigma_2(a_i) & 0 & 0 & 0 & E \\ 0 & \epsilon_h(a_i) & C & 0 & 0 & 0 & 0 \\ 0 & \gamma(a_i)\alpha_1(a_i) & \psi(a_i)\alpha_2(a_i) & -\mu_h(a_i) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & D & 0 & -H \\ 0 & 0 & 0 & 0 & 0 & B & H \\ 0 & 0 & 0 & 0 & 0 & \epsilon_r(e_j) & D \end{bmatrix},$$

where

$$A = -\epsilon_h(a_i) - \gamma(a_i)\alpha_1(a_i) - \mu_h(a_i), \quad B = -\epsilon_r(e_j) - \mu_r(e_j) - \delta_r(e_j),$$

$$C = -\psi(a_i)\alpha_2(a_i) - \mu_h(a_i) - \delta_h(a_i), \quad D = -\mu_r(e_j) - \delta_r(e_j),$$

$$E = \sum_{i=0}^L \sum_{j=0}^T \rho(a_i)\sigma_1(e_j), \quad H = \sum_{j=0}^T \beta(e_j)\sigma_1(e_j).$$

The first, fourth and fifth columns have diagonal entries, therefore the diagonal entries  $-\mu_h(a_i)$  twice and  $-\mu_r(e_j) - \delta_r(e_j)$  are three of the eigenvalues of the Jacobian matrix i.e  $\lambda_1 = -\mu_h(a_i)$ ,  $\lambda_2 = -\mu_h(a_i)$  and  $\lambda_3 = -\mu_r(e_j) - \delta_r(e_j)$ . Thus we find the remaining eigenvalues by excluding the corresponding columns and rows. The remaining eigenvalues are obtained from the submatrix

$$J_2(\pi_0) = \begin{bmatrix} A & \sum_{i=0}^L \eta(a_i)\sigma_2(a_i) & 0 & \sum_{i=0}^L \sum_{j=0}^T \rho(a_i)\sigma_1(e_j) \\ \epsilon_h(a_i) & C & 0 & 0 \\ 0 & 0 & B & \sum_{j=0}^T \beta(e_j)\sigma_1(e_j) \\ 0 & 0 & \epsilon_r(e_j) & D \end{bmatrix}$$

The eigenvalues of the matrix  $J_2(\pi_0)$  are the roots of the characteristic equation

$$\begin{aligned}
 & [(\epsilon_h(a_i) + \gamma(a_i)\alpha_1(a_i) + \mu_h(a_i) + \lambda)(\psi(a_i)\alpha_2(a_i) + \delta_h(a_i) + \mu_h(a_i) + \lambda) \\
 & - \eta(a_i)\epsilon_h(a_i)\sigma_2(a_i)][(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j) + \lambda)(\mu_r(e_j) + \delta_r(e_j) + \lambda) \\
 & - \beta(e_j)\epsilon_r(e_j)\sigma_1(e_j)] = 0 \quad (3.3)
 \end{aligned}$$

This implies that

$$\begin{aligned}
 & [(\epsilon_h(a_i) + \gamma(a_i)\alpha_1(a_i) + \mu_h(a_i) + \lambda)(\psi(a_i)\alpha_2(a_i) + \delta_h(a_i) + \mu_h(a_i) + \lambda) \\
 & - \eta(a_i)\epsilon_h(a_i)\sigma_2(a_i)] = 0 \quad (3.4)
 \end{aligned}$$

or

$$[(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j) + \lambda)(\mu_r(e_j) + \delta_r(e_j) + \lambda) - \beta(e_j)\epsilon_r(e_j)\sigma_1(e_j)] = 0. \quad (3.5)$$

Let

$$b_1 = \epsilon_h(a_i) + \gamma(a_i)\alpha_1(a_i) + \mu_h(a_i), b_2 = \psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i)$$

then (3.4) becomes

$$c_2\lambda^2 + c_1\lambda + c_0 = 0, \quad (3.6)$$

where

$$\left. \begin{aligned}
 c_2 &= 1 \\
 c_1 &= b_1 + b_2 \\
 c_0 &= b_1b_2 - \frac{\eta(a_i)\sigma_2(a_i)\epsilon_h(a_i)}{(\epsilon_h(a_i) + \gamma(a_i)\alpha_1(a_i) + \mu_h(a_i))(\psi(a_i)\alpha_2(a_i) + \delta_h(a_i) + \mu_h(a_i))}
 \end{aligned} \right\} \quad (3.7)$$

Further simplification of  $c_0$  in terms of  $R_0(a)$  gives

$$c_0 = b_1b_2(1 - R_0(a)) \quad (3.8)$$

The Routh-Hurwitz criterion follows since all the roots of the polynomial (3.6) have negative real part if and only if the coefficient  $c_i$  are positive and matrices  $H_i > 0$  for  $i = 0,1,2$ . From (3.7) it is shown that  $c_1 > 0$  and  $c_2 > 0$  since all  $b'_i$ 's are positive. Moreover, if  $R_0(a) < 1$  it is follows from (3.8) that  $c_0 > 0$ . Also the Hurwitz matrices for the polynomial (3.6) are found to be positive. That is,

$$H_1 = c_1, c_2 > 0, \quad H_2 = \begin{vmatrix} c_0 & 0 \\ c_1 & 1 \end{vmatrix} > 0$$

Therefore, all the eigenvalues of the Jacobian matrices  $J(\pi_0)$  have negative real part when  $R_0(a) < 1$  and the disease free equilibrium point is locally

asymptotically stable. However, when  $R_0(a) > 1$ , we see that  $c_0 < 0$  and by Descartes' rule of signs [17] there is exactly one sign change in the  $c_2, c_1, c_0$  of coefficient of the polynomial (3.6). So, there is one eigenvalue with the positive real part and the disease free equilibrium point is unstable. At this juncture, we can now infer from the equation (3.8) that there exists a  $c_0^*$  such that  $c_0^* = b_1 b_2 (1 - R_0(a_i))$  and since  $R_0(a)$  majorizes  $R_0(a_i)$  the system (2.1)-(2.7) is locally asymptotically stable if  $R_0(a_i) < 1$  and unstable if  $R_0(a_i) > 1$ .  $\square$

### 3.2. Existence of Endemic Equilibrium

We shall use the theorem below to show that the developed model (2.1)-(2.7) has an endemic equilibrium point  $E_e$ . The endemic equilibrium point is a positive steady state solution when the disease persists in the population.

**Theorem 3.2.** *Lassa fever model (2.1)-(2.7) has no endemic equilibrium when  $R_0(a) < 1$  and a unique endemic equilibrium exist when  $R_0(a) > 1$ .*

*Proof.* Let  $E_e = (S_h^*, E_h^*, I_h^*, R_h^*, S_r^*, E_r^*, I_r^*)$  be a non-trivial equilibrium of system (2.1)-(2.7) i.e all component of  $E_e$  are positive. At the steady state, we gets

$$S_h^*(a_i) = \frac{I_h^*(a_i) \Lambda_h(a_i) (\psi(a_i) \alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i)) (\gamma(a_i) \alpha_1(a_i) + \mu_h(a_i) + \epsilon_h(a_i))}{\sum_{i=0}^L \sum_{j=0}^T \mu_h(a_i) \epsilon_h(a_i) U}$$

$$E_h^*(a_i) = \frac{I_h^*(a_i) (\psi(a_i) \alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))}{\sum_{i=0}^L \epsilon_h(a_i)}$$

$$R_h^*(a_i) = \frac{\sum_{i=0}^L (\psi(a_i) \alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i)) \gamma(a_i) \alpha_1(a_i) I_h^*(a_i)}{\mu_h(a_i) \sum_{i=0}^L \epsilon_h(a_i)} + \frac{\psi(a_i) \alpha_2(a_i) I_h^*(a_i)}{\mu_h(a_i)}$$

$$S_r^*(e_j) = \frac{\Lambda_r^2(e_j)}{\sum_{j=0}^T [(\beta(e_j)\sigma_1(e_j)I_r^*(e_j) + \theta(e_j)) + \Lambda_r(e_j)](\mu_r(e_j) + \delta_r(e_j))}$$

$$E_r^*(e_j) = \sum_{j=0}^T \frac{(\beta(e_j)\sigma_1(e_j)I_r^*(e_j) + \theta(e_j))\Lambda_r(e_j)}{(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j))[(\beta(e_j)\sigma_1(e_j)I_r^*(e_j) + \theta(e_j)) + \Lambda_r(e_j)]}$$

$$I_r^*(e_j) = \Lambda_r(e_j)\epsilon_r(e_j)\beta(e_j)\sigma_1(e_j) - (\theta(e_j) + \Lambda_r(e_j))(\mu_r(e_j) + \delta_r(e_j))(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j)) + P,$$

where

$$U = [\rho(a_i)\sigma_1^2(e_i)\Lambda_r(e_j)\epsilon_r(e_j)\beta(e_j) - (\theta(e_j) + \Lambda_r(e_j))\rho(e_j)\sigma_1(e_j)(\mu_r(e_j) + \delta_r(e_j)) \times (\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j)) + \rho(a_i)\sigma_1(e_j)P + \eta(a_i)\sigma_2(a_i)I_h^*(a_i) + \theta(e_j) + \kappa(a_i)]$$

$$P = \sqrt{\frac{[(\theta(e_j) + \Lambda_r(e_j))(\mu_r(e_j) + \delta_r(e_j))(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j)) - \Lambda_r(e_j)\epsilon_r(e_j)\beta(e_j)\sigma_1(e_j)]^2 + M}{2\beta(e_j)\sigma_1(e_j)(\mu_r(e_j) + \delta_r(e_j))(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j))}}$$

$$M = 4\beta(e_j)\sigma_1(e_j)\Lambda_r(e_j)\epsilon_r(e_j)\theta(e_j)(\mu_r(e_j) + \delta_r(e_j))(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j))$$

and  $I_h^*(a_i)$  is a positive solution of a equation given by

$$K_1(I_h^*)^2 + K_2I_h^* + K_3 = 0 \tag{3.9}$$

where

$$K_1 = \eta(a_i)\sigma_2(a_i)\mu_h(a_i)$$

$$K_2 = \rho(a_i)\sigma_1(e_j)\epsilon_r(e_j)\beta(e_j)\mu_h(a_i)\Lambda_r(e_j) + \mu_h(a_i)\rho(a_i)\sigma_1(e_j)(\theta(e_j) + \Lambda_r(e_j)) \times (\mu_r(e_j) + \delta_r(e_j)) \times (\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j)) + \mu_h(a_i)\rho(a_i)\sigma_1(e_j)P + \mu_h(a_i)(\theta(e_j) + \kappa(a_i)) + \Lambda_h(a_i)\mu_h(a_i)(1 - R_0(a))$$

$$K_3 = d_1 - (d_2 + d_3),$$

where

$$d_1 =$$

$$\frac{\mu_h(a_i)\epsilon_h(a_i)\Lambda_h(a_i)\rho(a_i)\sigma_1(e_j)(\theta(e_j) + \Lambda_r(e_j))(\mu_r(e_j) + \delta_r(e_j))(\epsilon_r(e_j) + \mu_r(e_j) + \delta_r(e_j))}{(\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))(\gamma(a_i)\alpha_1(a_i) + \mu_h(a_i) + \epsilon_h(a_i))}$$

$$d_2 = \frac{\Lambda_h(a_i)\rho(a_i)\sigma_1^2(e_j)\Lambda_r(e_j)\epsilon_r(e_j)\beta(e_j)\mu_h(a_i)\epsilon_h(a_i) + \rho(a_i)\sigma_1(e_j)\mu_h(a_i)\epsilon_h(a_i)\Lambda_h(a_i)P}{(\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))(\gamma(a_i)\alpha_1(a_i) + \mu_h(a_i) + \epsilon_h(a_i))}$$

$$d_3 = \frac{\Lambda_h(a_i)\epsilon_h(a_i)\mu_h(a_i)(\theta(e_j) + \kappa(a_i))}{(\psi(a_i)\alpha_2(a_i) + \mu_h(a_i) + \delta_h(a_i))(\gamma(a_i)\alpha_1(a_i) + \mu_h(a_i) + \epsilon_h(a_i))}.$$

It is clearly seen that  $K_1 > 0$ . When  $R_0(a) < 1$ , one sees that  $K_2 > 0$ . However, when  $R_0(a) > 1$ , then  $K_2 < 0$  and endemic equilibrium exists. Finally,  $K_3 < 0$  if  $d_1 < (d_2 + d_3)$ .  $\square$

#### 4. Numerical Results

The model (2.1)-(2.7) is simulated using the parameters in Table 2 to illustrate some of the theoretical results established in this study (some of these parameters are taken from [4,5,20] and by considering initial conditions  $S_h(0) = 100$ ,  $E_h(0) = 10$ ,  $I_h(0) = 5$ ,  $R_h(0) = 0$ ,  $S_r(0) = 1000$ ,  $I_r(0) = 30$ . The numerical simulations are conducted using Maple 17 software and the results are given in Figure 1-10 to illustrate the system's behaviour for different values of model's parameters. Figure 1 shows the behaviour of the susceptible human population and it is observed that the susceptible human population drops as a result of infection by infected rodent, infected human and inhalation of aerosol produced when rodent urinate. The magnitudes of the exposed human population in figure 2 decreases when they progress to infected class or when they moves to recovered class as a result of early diagnosis. The decrease in the number of infectious human population in figure 3 contribute to the increase in the number of recovered human population in figure 4. In figure 7 and 8, the effect of the recovery rate of the infectious human sub-population on the dynamical behaviour of the system was investigated. It is noticed that as the value of parameter  $\psi$  increases, the number of the infectious human decreases with time. Conversely, decreasing value of the recovery rate increases the number of the infectious human in the population. Since early diagnosis enables treatment, we investigated the effect of the recovery rate of the exposed human sub-population on the dynamical behaviour of the system. It is observed that as the value of parameter  $\gamma$  increases the number of the exposed human decreases with time. Conversely, decreasing value of the recovery rate increases the number of the exposed human in the population. See figure 9 and 10.



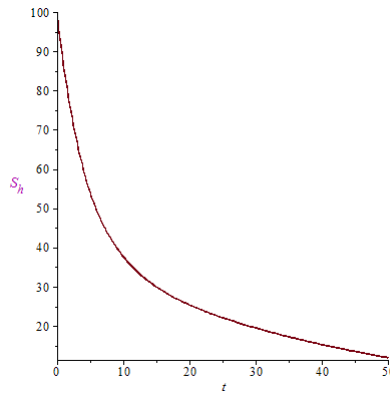


Figure 1: The behaviour of susceptible human when  $R_0(a_i) < 1$ ,  $\sigma_1(e_j) = 0.8$ ,  $\sigma_2(e_j) = 0.56$ , and  $\theta(e_j) = 0.022$

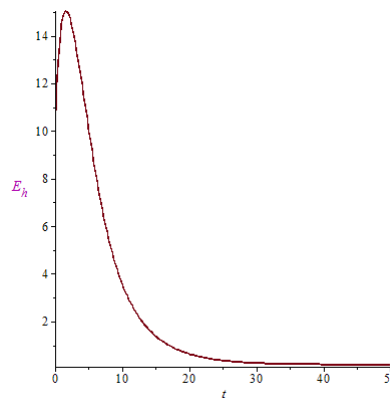


Figure 2: The behaviour of exposed human when  $R_0(a_i) < 1$ ,  $\sigma_1(e_j) = 0.8$ ,  $\sigma_2(a_i) = 0.56$ , and  $\theta(e_j) = 0.022$

Figure 5 shows the behaviour of the susceptible reservoir host and it is observed that the magnitude of susceptible reservoir decreases as a result of infection by infected reservoir or hunting. Similarly, the magnitude of exposed reservoir decreases when they progress infectious class or hunting as show in figure 6. Figure 7 shows the behaviour of infectious reservoir and it is noticed that the magnitude of infectious reservoir as of hunting and natural death.

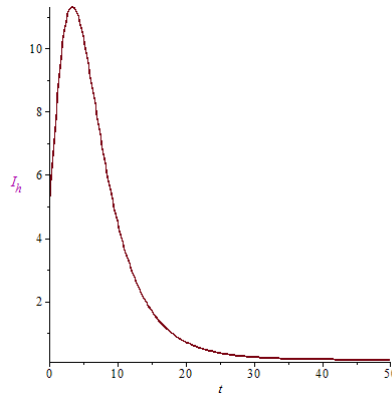


Figure 3: The behaviour of infected human when  $R_0(a_I) < 1$ ,  $\sigma_1 = 0.8$ ,  $\sigma_2 = 0.56$ , and  $\theta = 0.022$

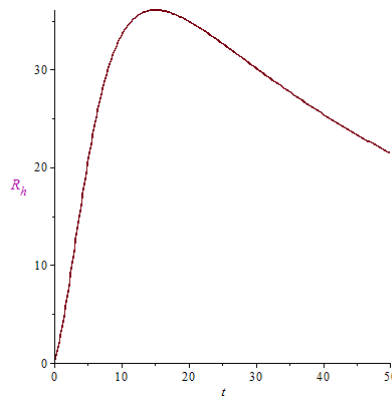


Figure 4: The behaviour of recovered human when  $R_0(a_i) < 1$ ,  $\sigma_1(e_j) = 0.8$ , and  $\theta(e_j) = 0.022$

## 5. Conclusion

We have highlighted a compartmental modelling approach for two different populations: human and rodent with rigorous analyses. In contrast to models for the spread of Lassa fever in [1, 14, 19, 20], the model presented in this study considers the human and rodent populations as a dynamical variable with discrete age structure. It is assumed that individuals infected with Lassa virus are treated early with antiviral drug. The expression for the reproduction number  $R_0(a_i)$  are given in terms of the model parameters. The analytical

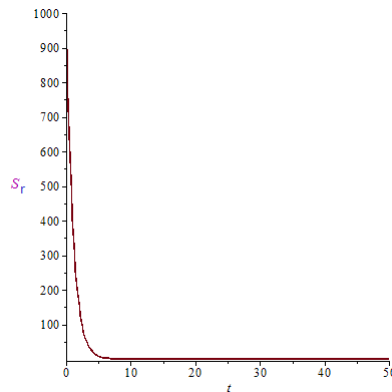


Figure 5: The behaviour of susceptible reservoir host when  $R_0(a_i) < 1$ ,  $\sigma_1(e_j) = 0.8$ , and  $\theta(e_j) = 0.022$

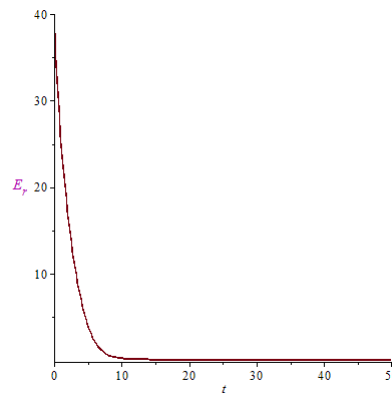


Figure 6: The behaviour of exposed reservoir host when  $R_0(a_i) < 1$ ,  $\sigma_1(e_j) = 0.8$ , and  $\theta(e_j) = 0.022$

results show that the spread of Lassa fever can be effectively controlled in the population if the associated intervention strategies can make  $R_0(a_i)$  less than unity and provided the associated disease-induced mortality is zero, but if  $R_0(a_i) > 1$  or  $R_0(a_i) > 1$  the disease will persist in the population. The results of simulations reveal that increase in recovery rate contributes to decrease in the number of exposed and infectious human in the population and increase in the number of recovered human. This study suggests that early diagnostic of infected humans, maintaining hygienic environment, use of new needle when taking injection and interim control of the rodent carrying the virus are the

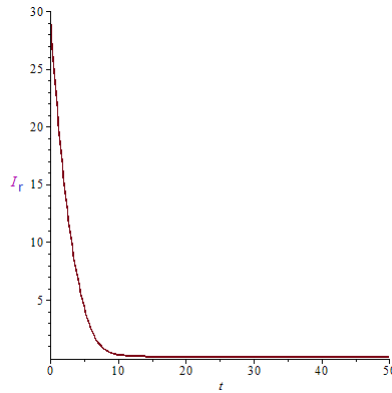


Figure 7: The behaviour of infected reservoir host when  $R_0(a_i) < 1$ ,  $\sigma_1(e_j) = 0.8$ , and  $\theta(e_j) = 0.022$

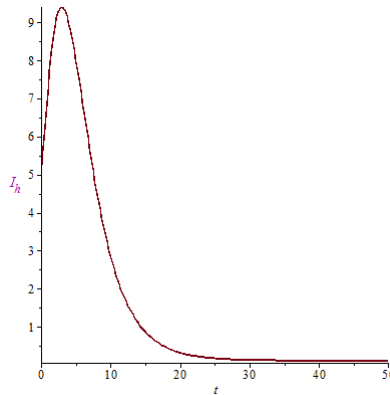


Figure 8: The behaviour of the infectious human when varying  $\psi(e_j) = 1.5$

best strategies against the spread of the disease.

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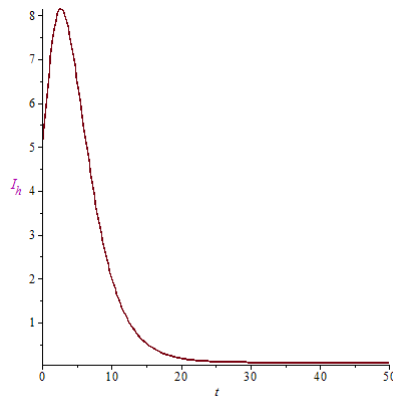


Figure 9: The behaviour of the infectious human when varying  $\psi(a_i) = 2.1$

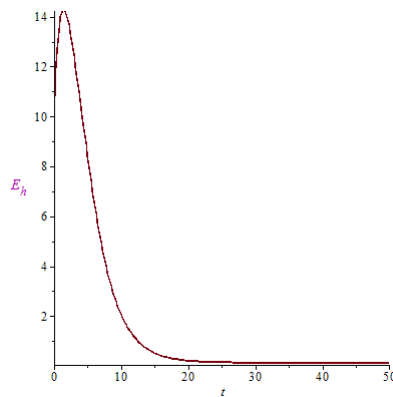


Figure 10: The behaviour of the exposed human when varying  $\gamma(a_i) = 0.6$

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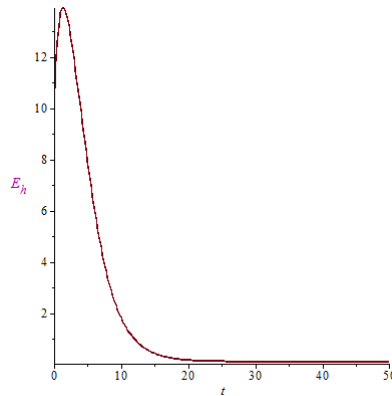


Figure 11: The behaviour of the exposed human when varying  $\gamma(a_i) = 0.75$

Table 1: Description of the state variables

State variables	Definition
$S_h(t, a_i)$	Number of susceptible humans
$E_h(t, a_i)$	Number of exposed humans
$I_h(t, a_i)$	Number of infected humans
$R_h(t, a_i)$	Number of recovered humans
$S_r(t, e_j)$	Number of susceptible reservoirs
$E_r(t, e_j)$	Number of exposed reservoirs
$I_r(t, e_j)$	Number of infected reservoirs

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Table 2: Description of the model parameters

Definition	Parameter	Value
Recruitment term of humans	$\Lambda_h(a_i)$	0.038
Effective transmission rate in susceptible humans by infected rodents	$\rho(a_i)$	0.6
Interacting rate of reservoirs	$\sigma_1(e_j)$	0.8
Effective transmission rate in susceptible humans by infected humans	$\eta(a_i)$	0.6
Interacting rate of humans	$\sigma_2(a_i)$	0.56
Treatment rate of exposed humans	$\alpha_1(a_i)$	0.05
Treatment rate of infected humans	$\alpha_2(a_i)$	0.9
Effective contact rate between Lassa virus and humans or reservoirs	$\theta(e_j)$	0.022
Rate of inoculation	$\kappa(a_i)$	0.018
Progression rate of humans from the exposed state to the infectious state	$\epsilon_h(a_i)$	0.85
Diagnostic factor of exposed humans	$\gamma(a_i)$	0.9
proportion of effective treatment of infected humans	$\psi(a_i)$	0.45
Natural death rate of humans	$\mu_h(a_i)$	0.02
Progression rate of reservoirs from the exposed state to infectious state	$\epsilon_r(e_j)$	0.85
Effective transmission rate in susceptible reservoirs by infected reservoirs	$\beta(e_j)$	0.75
Disease induced death rate of humans	$\delta_h(e_j)$	0.2
Mortality of reservoirs due to hunting	$\delta_r(e_j)$	0.3
Natural death rate reservoirs	$\mu_r(e_j)$	0.6
Recruitment term of reservoirs	$\Lambda_r(e_j)$	0.56

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