# Basic Reproductive Number for the Spread and Control of Lassa fever

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***Abstract* —** *A Mathematical Model was developed for the spread and control of Lassa Lever. The model incorporates two control parameters, the use of condom to control human to human transmission via sexual contact with opposite sex and the use of Rodenticide to reduce both the dormant and active Rat populations. Existence and stability were analysed for disease free equilibrium. Key to our analysis is the definition of a basic reproductive number* $(R_0)$, *which is the number of secondary infections that one infective individual would create over the duration of the infectious period provided that everyone else is susceptible. It is known that when* $R_0 \leq 1$ *the disease dies out, and when* $R_0 > 1$ *the disease persists in the population.*

**Keywords** — *Stability, Equilibrium, Susceptible, Condom, Control.*

## I. INTRODUCTION

Lassa fever is an acute viral Hemorrhagic fever (VHF) first isolated in a town called Lassa in the Yedseram River Valley in the present Borno State of Northern Nigeria in 1969 [1]. The first victim is Laura Wine, 65 year old female nurse who works at Lassa mission hospital [2]. Lassa fever is endemic in Nigeria, Liberia, Sierra Leone, Guinea, and other West African countries, affecting about 2 – 3 million persons with 5000 - 10,000 fatalities annually [3]. Since its initial discovery in Lassa-Nigeria, rural and nosocomial outbreaks of Lassa fever have occurred repeatedly in other parts of Nigeria: Jos, Onitsha, Zonkwa, Ekpoma [4].

[5] reported outbreaks in some cities of west African countries of Sierra Leone, Liberia, Guinea. In Côte d'Ivoire, Ghana, Togo and Benin, no outbreak has ever been recorded, though isolated cases show evidence of viral circulation [6]. Lassa fever therefore appears to have 2 geographically separate endemic areas: the Mano River region (Guinea, Sierra Leone, Liberia) in the West, and Nigeria in the East. However, Some Lassa fever cases have been "imported" into the U.S. and U.K. through travelers who acquired the disease elsewhere [1].

Lassa fever is a zoonotic disease, i.e. it can be transmitted from infected animal to a human. The natural Reservoir of the Lassa virus is Multimammate Rat species known as Mastomys Natalencesis [7]. Because certain varieties of *Mastomys* often live in human homes, the virus is easily transmitted to humans. Transmission occurs via direct contact with rat urine, faces, and saliva; via contact with excretion- or secretion-infected materials; or via ingestion of excretion-contaminated food. Victims can also become infected via skin breaks, and via mucous membranes from aerosol transmission from dust-borne particles. In some areas, the rodents are used as a food source, thus providing additional exposure to the infected rat blood, as well as allowing ingestion of potentially contaminated meat. [8] stated that Health workers become infected usually from contact with rodent saliva or contamination of needles.

Unlike other arena viruses, Lassa virus can be fairly easily transmitted from human to human the [9]. [10] stated that humans can contact the disease from other humans via aerosol transmission (coughing), or from direct contact with infected human blood, urine, or semen. Lassa virus has been isolated from semen 6 weeks after acute illness; thus the virus can be transmitted to sexual partners by convalescent men,[1].

The symptoms of Lassa fever develop about 21 days after the infection with acute illness involving multi organs. Specific symptoms include fever, facial swelling, muscle fatigue, vomiting, cough, meningitis, and hypertension. In some patients neurological problems, including hearing loss which may be transient or permanent, tremors, and encephalitis, have been described the [11]

### A. *Mathematical Models of Infectious Diseases.*

Mathematical Modeling is a tool which has been used to study the mechanisms by which diseases spread, it is also used to predict the future course of an outbreak and to evaluate strategies to control an epidemic [12].

The first scientist who systematically tried to quantify causes of death was [13]. The bills he studied were listings of numbers and causes of deaths published weekly. Graunt's analysis of causes of death is considered the beginning of the "theory of competing risk" which according to [12] is "a theory that is now well established among modern epidemiologists".

The earliest account of mathematical modeling of spread of disease was carried out in 1766 by Daniel Bernoulli. Trained as a physician, Bernoulli created a mathematical model to defend the practice of

inoculation against smallpox [14]. The calculations from this model showed that universal inoculation against smallpox would increase the life expectancy from 26 years 7 months to 29 years 9 months [15].

Daniel Bernoulli's work preceded our modern understanding of germ theory, it was followed by [16] model on measles but it was not until the research of [17] into the spread of malaria, that modern theoretical epidemiology began. This was soon followed by the work of [18] whose paper a contribution to Mthematical Theory of Epidemics was published in 1927. A simple deterministic (compartmental) model was formulated in this paper. The model was successful in predicting the behavior of outbreaks very similar to that observed in many recorded epidemics [19].

Lassa fever is a viral Hemorrhagic fever, alongside with Ebola, Marburg, and Dengue etc. Currently scholarly article in Mathematical Model of Lassa fever is rather scant. Articles on mathematical model for transmission of other Hemorrhagic fever like Ebola fever, Yellow fever, Dengue fever, abound in the literature: [20], [21],[22] and [23]. For Lassa fever model see; [24], [25] and [26]

In formulating the model, we assume a homogenous mixing of the human and Reservoir host population such that there are equal chances of transmitting the virus when there is a contact between susceptible human and active Reservoir host. We represent the transmission dynamics of the Lassa fever disease using a set of ordinary differential equations. The total human population at time $t$ denoted by $N_H(t)$ is sub-divided into four (4) mutually exclusive sub-populations of Susceptible Male $S_1(t)$, Infected Male $I_1(t)$, Susceptible Female $S_2(t)$, Infected Female $I_2(t)$, such that $N_H(t) = S_1(t) + I_1(t) + S_2(t) + I_2(t)$. Similarly, the total Natural Reservoir host population at time $t$, denoted by $N_R(t)$ is sub-divided into dormant Reservoir host $R_1(t)$, active Reservoir host $R_2(t)$, such that $N_R(t) = R_1(t) + R_2(t)$.

## B. *Parameters of the Model*

| | |
|---|---|
| $\beta_H$ | The natural Birth rate of human population |
| $\beta_R$ | The natural birth rate of vectors |
| $\theta$ | The proportion of human birth that is male $0 < \theta < 1$ |
| $\alpha_1$ | The rate of transmission resulting from sexual interaction between infected female and susceptible male |
| $\alpha_2$ | The rate of transmission resulting from sexual interaction between infected male and susceptible female |
| $\alpha_3$ | The rate of transmission resulting from interaction between active virus Reservoir and susceptible male |
| $\alpha_4$ | The rate of transmission resulting from interaction between active virus Reservoir and susceptible female |
| $c_1$ | Average number of male partners acquired by a susceptible female |
| $c_2$ | Average number of female partners acquired by a susceptible male |
| $\mu_1$ | Natural death rate of human population |
| $\mu_2$ | Natural death rate of Reservoir population |
| $\gamma$ | Recovery rate of infected human |
| $\sigma$ | Progression rate from dormant to active Reservoir host |
| $\delta_1$ | Death rate of human population due to infection |
| $\delta_2$ | Death rate of virus Reservoir population due to application of pesticide |
| $\varepsilon$ | Efficacy of condom |
| $\tau$ | Compliance of condom usage |

$$\frac{dS_1}{dt} = \beta_H \theta N_H + \gamma I_1 - \frac{(c_2\alpha_1(1-\varepsilon\tau)I_2 + \alpha_3 R_2)S_1}{N_H} - \mu_1 S_1 \quad (1)$$

$$\frac{dI_1}{dt} = \frac{(c_2\alpha_1(1-\varepsilon\tau)I_2 + \alpha_3 R_2)S_1}{N_H} - (\mu_1 + \delta_1 + \gamma)I_1 \quad (2)$$

$$\frac{dS_2}{dt} = \beta_H (1-\theta) N_H + \gamma I_2 - \frac{(c_1\alpha_2(1-\varepsilon\tau)I_1 + \alpha_4 R_2)S_2}{N_H} - \mu_1 S_2 \quad (3)$$

$$\frac{dI_2}{dt} = \frac{(c_1\alpha_2(1-\varepsilon\tau)I_1 + \alpha_4 R_2)S_2}{N_H} - (\mu_1 + \delta_1 + \gamma)I_2 \quad (4)$$

$$\frac{dR_1}{dt} = \beta_R N_R - (\sigma + \mu_2 + \delta_2)R_1 \quad (5)$$

$$\frac{dR_2}{dt} = \sigma R_1 - (\mu_2 + \delta_2)R_2 \quad (6)$$

## C. *Derivation of the Model Equation*

The model has six compartments namely, Susceptible Male $S_1(t)$, Infected Male $I_1(t)$, Susceptible Female $S_2(t)$, Infected Female $I_2(t)$, dormant Reservoir host $R_1(t)$, active Reservoir host $R_2(t)$. The various compartments were generated as explained below.

The Susceptible Male population $S_1(t)$ is generated through male birth, at the rate $\beta_H \theta N_H$ where $0 < \theta < 1$. It is increased by the number of The Dormant Reservoir $R_1(t)$ population is generated by the natural birth rate of active virus Reservoir host denoted by $\beta_R N_R$, and is reduced by progression to

active reservoir population due to maturity at the rate $\sigma$, natural death at the rate $\mu_2$ and death due to application of pesticide at the rate $\delta_2$.

The Active Reservoir population $R_2(t)$ is generated by the progression of dormant Reservoir population to active Reservoir population due to maturity. It is decreased by natural death at the rate $\mu_2$ and death due to application pesticide at the rat $\delta_2$.

males who recovered from the infection at the rate $\gamma$, it is on the other hand reduced by natural death at the rate $\mu_1$, interaction with infected female $I_2$, and active Reservoir host $R_2$, which results to the force of infection $\frac{(c_2\alpha_1(1-\varepsilon\tau)I_2+\alpha_3R_2)S_1}{N_H}$, where $c_2$ is the average sexual partners acquired by Susceptible male $S_1$, $\alpha_1$ is the transmission rate when there is interaction between $I_2$ and $S_1$, the factor $(1-\varepsilon\tau)$ is the effect of condom usage by $S_1$. $\alpha_3$ is the transmission rate when there is interaction between $R_2$ and $S_1$. The Infected Male population $I_1(t)$ is generated by sexual interaction with infected female $I_2(t)$ and interaction with active Reservoir host $R_2(t)$ i.e. $\frac{(c_2\alpha_1(1-\varepsilon\tau)I_2+\alpha_3R_2)S_1}{N_H}$, it is decreased by natural death at the rate $\mu_1$, death due to infection at the rate $\delta_1$ and recovery of infected male $I_1$ as a result of treatment at the rate $\gamma$.

The Susceptible Female $S_2(t)$ population is generated through female birth, at the rate $\beta_H(1-\theta)N_H$ where $0<\theta<1$. It is increased by recovered infected female at the rate $\gamma$, it is on the other hand reduced by natural death at the $\mu_1$ and interaction with infected male $I_1$ and active Reservoir host $R_2$, which results to the force of infection $\frac{(c_1\alpha_2(1-\varepsilon\tau)I_1+\alpha_4R_2)S_2}{N_H}$, where $c_1$ is the average sexual partners acquired by Susceptible female $S_2$, $\alpha_2$ is the transmission rate when there is interaction between $I_1$ and $S_2$, the factor $(1-\varepsilon\tau)$ is the effect of condom usage by $S_2$. $\alpha_4$ is the transmission rate when there is interaction between $R_2$ and $S_2$.

The Infected Female $I_2(t)$ population is generated by sexual interaction with infected male $I_1$ and interaction with active reservoir $R_2$ i.e. $\frac{(c_1\alpha_2(1-\varepsilon\tau)I_1+\alpha_4R_2)S_2}{N_H}$, it is decreased by natural death at the rate $\mu_1$, death due to infection at the rate $\delta_1$ and recovery of infected female $I_2$ as a result of treatment at the rate $\gamma$. The Dormant Reservoir $R_1(t)$ population is generated by the natural birth rate of active virus Reservoir host denoted by $\beta_R N_R$, and is reduced by progression to active reservoir population due to maturity at the rate $\sigma$, natural death at the rate $\mu_2$ and death due to application of pesticide at the rate $\delta_2$.

The Active Reservoir population $R_2(t)$ is generated by the progression of dormant Reservoir population to active Reservoir population due to maturity. It is decreased by natural death at the rate $\mu_2$ and death due to application pesticide at the rat $\delta_2$.

The total human population size is given by;

$$N_H = S_1 + I_1 + S_2 + I_2 \quad (7)$$

The total Reservoir population size is given by

$$N_R = R_1 + R_2 \quad (8)$$

By adding equations (1) to (4), we have;

$$\frac{dN_H}{dt} = \beta_H N_H - \mu_1 N_H - \delta_1(I_1 + I_2) \quad (9)$$

By adding equations (5) to (6), we have;

$$\frac{dN_R}{dt} = \beta_R N_R - (\mu_2 + \delta_2) \quad (10)$$

## II. Basic Properties of the Model

In this section, the basic dynamical features of the model equations (1) to (6) will be explored.

**Theorem 1** The closed set

$$D=\left\{(S_1,I_1,S_2,I_2,R_1,R_2)\in\Re_+^6 : S_1+I_1+S_2+I_2\le\frac{\beta_H}{\mu_1 N_H}; R_1+R_2\le\frac{\beta_R}{(\mu_2+\delta_2)}\right\}$$

Is positively-invariant and attracting with respect to the basic model equations (1) to (6)

**Proof**

From equations (7), to (10);

$\frac{dN_H}{dt} \le \beta_H - \mu_1 N_H$,

$\frac{dN_R}{dt} \le \beta_R - (\mu_2 + \delta) N_R$.

It follows that $\frac{dN_H}{dt} < 0$ and $\frac{dN_R}{dt} < 0$ if $N_H(t) > \frac{\beta_H}{\mu_1}$ and $N_R(t) > \frac{\beta_R}{\mu_2}$ respectively.

Thus a standard comparison theorem as in [27] can be used to show

that $N_H(t) \le N_H(0)e^{\mu_1(t)} + \frac{\beta_H}{\mu_1}\left(1 - e^{-\mu_1(t)}\right)$

and $N_R(t) \le N_R(0)e^{\mu_2(t)} + \frac{\beta_r}{\mu_2+\delta_2}\left(1 - e^{(-\mu_2+\delta_2)(t)}\right)$.

In particular $N_H(t) \le \frac{\beta_H}{\mu_1}$ and $N_R(t) \le \frac{\beta_R}{\mu_2+\delta_2}$ if $N_H(0) \le \frac{\beta_H}{\mu_1}$ and $N_R(0) \le \frac{\beta_R}{\mu_2+\delta_2}$ respectively.

Thus $D$ is positively-invariant. Further, if $N_H(0) > \frac{\beta_H}{\mu_1}$, and $N_R(0) > \frac{\beta_R}{\mu_2+\delta_2}$, then either

the solution enters $D$ in finite time or $N_H(t)$ approaches $\frac{\beta_H}{\mu_1}$, and $N_R(t)$ approaches $\frac{\beta_R}{\mu_2+\delta_2}$, and the infected variables $I_1 + I_2$ approaches 0. Hence $D$ is attracting, that is all solutions in $\Re_+^6$ eventually enters $D$. Thus in $D$, the basic model equations (1) to (6) is well posed epidemiologically and mathematically according to [28]. Hence it is sufficient to study the dynamics of the basic model equations (1) to (6)

**Theorem 2**

All the solution of the model equation (1) to (6) are positive for all time $t \geq 0$ provided that the initial conditions are positive.

**Proof**

Under the assumption that all initial conditions are positive, i.e
$S_1(0) > 0, I_1(0) > 0, S_2(0) > 0, I_2(0) > 0, R_1(0) > 0,$ and $R_2(0) > 0$
We have by contradiction, that the solution of (1) to (6) are positive if we assume for a contradiction that there exists first time,
$t_1 : S_1(t_1) = 0$ and
$S_1(t) > 0, I_1(t) > 0, S_2(t) > 0, I_2(t) > 0, R_1(t) > 0, R_2(t) > 0,$

$0 < t < t_1$ (11)
or there exists
$t_2 : I_1(t_2) = 0$ and
$S_1(t) > 0, I_1(t) > 0, S_2(t) > 0, I_2(t) > 0, R_1(t) > 0, R_2(t) > 0,$
$0 < t < t_2$ (12)
or there exists,
$t_3 : S_2(t_3) = 0$ and
$S_1(t) > 0, I_1(t) > 0, S_2(t) > 0, I_2(t) > 0, R_1(t) > 0, R_2(t) > 0,$
$0 < t < t_3$ (13)
or there exists,
$t_4 : I_2(t_4) = 0$ and
$S_1(t) > 0, I_1(t) > 0, S_2(t) > 0, I_2(t) > 0, R_1(t) > 0, R_2(t) > 0,$
$0 < t < t_4$ (14)
$t_5 : R_1(t_5) = 0$ and
$S_1(t) > 0, I_1(t) > 0, S_2(t) > 0, I_2(t) > 0, R_1(t) > 0, R_2(t) > 0,$
$0 < t < t_5$ (15)
$t_6 : R_2(t_6) = 0$ and
$S_1(t) > 0, I_1(t) > 0, S_2(t) > 0, I_2(t) > 0, R_1(t) > 0, R_2(t) > 0,$
$0 < t < t_6$ (16)
Now with the case where
$S_1(t_1) = 0$ (17)
we have;

$$\frac{dS_1(t_1)}{dt} = \lim_{t \to t_1} \frac{S_1(t) - S_1(t_1)}{t - t_1} < 0 \quad (18)$$

similarly, we have

$$\frac{dI_1(t_2)}{dt} < 0, \frac{dS_2(t_3)}{dt} < 0, \frac{dI_2(t_4)}{dt} < 0, \frac{dR_1(t_5)}{dt} < 0, \frac{dR_2(t_6)}{dt} < 0. \quad (19)$$

However, from equations (9) to (14) and (19). we have

$$S_1'(t_1) = \beta_H \theta N_H + \gamma I_1 - \frac{(c_2\alpha_1(1-\varepsilon\tau)I_2 + \alpha_3 R_2)S_1(t_1)}{N_H} - \mu_1 S_1(t_1) \quad (20)$$

i.e

$$S_1'(t_1) = \beta_H \theta N_H + \gamma I_1 > 0 \quad (21)$$

which contradicts (16) therefore,
$S_1'(t_1) \neq 0$
and
$S_1$, will remain positive for all $t$
Similarly, for the remaining variables, we have

$$I_1'(t_2) = \frac{(c_2\alpha_1(1-\varepsilon\tau)I_2 + \alpha_3 R_2)S_1}{N_H} > 0 \quad (22)$$

$$S_2'(t_3) = \beta_h(1-\theta)N_H + \gamma I_2 > 0 \quad (23)$$

$$I_2'(t_4) = \frac{(c_1\alpha_2(1-\varepsilon\tau)I_1 + \alpha_3 R_2)S_2}{N_H} > 0 \quad (24)$$

$$R_1'(t_5) = \beta_R N_R > 0 \quad (25)$$

$$R_2'(t_6) = \sigma R_1 > 0 \quad (26)$$

These are contradictions of what was supposed for each of the variables, meaning that

$$I_1(t_2) \neq 0, S_2(t_3) \neq 0, I_2(t_4) \neq 0, R_1(t_5) \neq 0, R_2(t_6) \neq 0. \quad (27)$$

Hence $I_1, S_2, I_2, R_1$, and $R_2$ remains positive for all $t$
By this we have shown that all the solutions of (3.1) to (3.6) are positive, provided that the initial conditions are positive.

## III. ANALYSIS OF THE MODEL AND RESULTS

### A. *Disease Free Equilibrium*

At equilibrium states, the rate of change of the state variables with respect to time is zero, i.e,

$$\frac{dS_1}{dt} = \frac{dI_1}{dt} = \frac{dS_2}{dt} = \frac{dI_2}{dt} = \frac{dR_1}{dt} = \frac{dR_2}{dt} = 0$$

We define disease compartments as the Infected male, Infected female compartments that is $I_1$ and $I_2$. We let $(S_1, I_1, S_2, I_2, R_1, R_2) = (x, y, z, u, v, w)$ at disease free equilibrium, equations the right hand side of our model equation (1) to (6) to zero and solving with the above change of variable, we have our DFE

$$E_0 = (x, y, z, u, v, w) = \left(\frac{\beta_H \theta N_H}{\mu_1}, 0, \frac{\beta_H(1-\theta)N_H}{\mu_1}, 0, 0, 0\right) \quad (28)$$

### B. *Stability of Equilibrium*

The notion of stability of equilibrium is of considerable theoretical and practical importance, and has been widely discussed in the literature [27], [29].

### 1) Local Stability of Disease Free Equilibrium $E_0$

For us to analyse our disease free equilibrium for stability we first obtain the Jacobian matrix at disease and basic reproductive number of our model.

At DFE, the Jacobian matrix is

$$J_{E_0} = \begin{bmatrix} -\mu_1 & \gamma & 0 & \frac{px}{N_H} & 0 & \frac{\alpha_3 x}{N_H} \\ 0 & -A_1 & 0 & \frac{px}{N_H} & 0 & \frac{\alpha_3 x}{N_H} \\ 0 & \frac{qz}{N_H} & -\mu_1 & 0 & 0 & \frac{\alpha_4 z}{N_H} \\ 0 & \frac{qz}{N_H} & 0 & -A_1 & 0 & \frac{\alpha_4 z}{N_H} \\ 0 & 0 & 0 & 0 & -A_2 & 0 \\ 0 & 0 & 0 & 0 & \sigma & -(\mu_2+\delta_2) \end{bmatrix} \quad (29)$$

where

$P = c_2\alpha_1(1-\varepsilon\tau)$ , $q = c_1\alpha_2(1-\varepsilon\tau)$ ,

$A_1 = (\mu_1+\delta_1+\gamma)$ and $A_2 = (\mu_2+\delta_1+\gamma)$

### 2) *Basic Reproductive Number* $(R_0)$

One of the most important concerns about any infectious disease is its ability to invade a population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of the disease. These models usually have a threshold parameter, known as the basic reproductive number $R_0$ such that when $R_0 < 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $R_0 > 1$ ,then the DFE is unstable and invasion is always possible see [28].

We define the basic reproductive number $R_0$ as the number of secondary infections that one infective individual would create over the duration of the infectious period provided that everyone else is susceptible. We use the next generation matrix approach as described by [30] to derive our Basic Reproductive Number diseases.

Here, the basic reproductive number $R_0$ is the spectral radius of the product matrix $FV^{-1}$, i.e. $R_0 = \rho(FV^{-1})$

Our model has two Infective compartments namely the Infective male $I_1$, and Infected female $I_2$. It follows that the matrices $F$ and $V$ for the new infective terms and remaining transfer terms respectively are given below:

.

$$F = \begin{bmatrix} 0 & \frac{px}{N_H} \\ \frac{qz}{N_H} & 0 \end{bmatrix} \qquad V = \begin{bmatrix} A_1 & 0 \\ 0 & A_1 \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & \frac{px}{N_H A_1} \\ \frac{qz}{N_H A_1} & 0 \end{bmatrix} \quad (30)$$

The spectral radius of (30) is given by

$$\rho(FV^{-1}) = \sqrt{\frac{px.qz}{(N_H A_1)^2}}$$

Substituting the values of $x$, $z$ at equilibrium, the values of $A_1$, $p$ and $q$ gives

$$R_0 = \sqrt{\frac{(c_2\alpha_1(1-\varepsilon\tau)\beta_H\theta)\times(c_1\alpha_2(1-\varepsilon\tau)\beta_H(1-\theta))}{\mu_1((\mu_1+\delta_1+\gamma))^2}} \quad (31)$$

**THEOREM 3**: The disease free equilibrium of the model equations (1) to (6) is locally asymptotically stable (LAS) if $0 < R_0 < 1$

**Proof**

Though the local stability of DFE when $R_0 < 1$ is a direct consequence of theorem 2 in [30] which need not be proved, however we confirm the correctness of the theorem by using the standard linearization technique. We transform (29) presented above to an upper triangular matrix using elementary row reduction method to have;

$$J_{E_0} = \begin{bmatrix} -\mu_1 & \gamma & 0 & \frac{px}{N_H} & 0 & \frac{\alpha_3 x}{N_H} \\ 0 & -A_1 & 0 & \frac{px}{N_H} & 0 & \frac{\alpha_3 x}{N_H} \\ 0 & 0 & -\mu_1 & 0 & 0 & A_4 \\ 0 & 0 & 0 & -A_1+A_3 & 0 & A_4 \\ 0 & 0 & 0 & 0 & -A_{2v} & 0 \\ 0 & 0 & 0 & 0 & \sigma & -(\mu_2+\delta_2) \end{bmatrix} \quad (32)$$

where

$$A_3 = \frac{qzpx}{A_1(N_H)^2} + \gamma \,, \quad A_4 = \frac{qzpx}{A_1(N_H)^2}\gamma + \frac{\alpha_3}{N_H} \,,$$

Thus the characteristics equation of the upper triangular Jacobian matrix (32) is given by;

$$J_{E_0} = \begin{bmatrix} -(\mu_1+\lambda) & \gamma & 0 & \frac{px}{N_H} & 0 & \frac{\alpha_3 x}{N_H} \\ 0 & -(A_1+\lambda) & 0 & \frac{px}{N_H} & 0 & \frac{\alpha_3 x}{N_H} \\ 0 & 0 & -(\mu_1+\lambda) & 0 & 0 & A_4 \\ 0 & 0 & 0 & -(A_1-A_3+\lambda) & 0 & A_4 \\ 0 & 0 & 0 & 0 & -(A_1+\lambda) & 0 \\ 0 & 0 & 0 & 0 & \sigma & -(\mu_2+\delta_2+\lambda) \end{bmatrix} \quad (33)$$

Equating the product of the diagonal of an upper triangular Jacobian matrix to zero gives the eigen-values of the matrix, therefore the eigen values of (33) are;

$\lambda_1 = \lambda_3 = -\mu_1, \lambda_2 = -A_1,$

$\lambda_4 = -(\mu_1+\delta_1+\gamma) + \frac{qzpx}{(N_H)^2(\mu_1+\delta_1+\gamma)}$, $\lambda_5 = -A_2$,

$\lambda_6 = (\mu_2+\delta_2)$

For local stability of disease free equilibrium Routh-Hurwitz criteria requires that all eigen values have negative real part. Since all the eigen-values of (33) are negative, i.e

$\lambda_i < 0$ for $i = 1,2,3..6$ implies $R_1 < 1$, hence the disease free equilibrium DFE is locally asymptotically stable if $R_1 < 1$

### 3) Global Stability of Disease Free Equilibrium

Global stability of epidemiological model is necessary and makes the model predictable as it guarantees that the model is independent of the initial size of the population. Global asymptotic stability (GAS) of an epidemiological model can be established by constructing appropriate Lyapunov function [22]. However, to establish the GAS of our model, we adapt the method used in [32], see appendix C.

**THEOREM 3**. The disease free equilibrium of the model equations (1) to (6) is Globally Asymptotically stable (GAS) if $R_0 < 1$.

**Proof**

To establish the global stability of the disease free equilibrium, the two conditions (H1) and (H2) as in [32] must be satisfied for $R_0 < 1$. We write the model equations (1) to (6) in the form

$$\frac{dX_1}{dt} = F(X_1, X_2)$$

$$\frac{dX_2}{dt} = G(X_1, X_2); G(X_1, 0)$$

where $X_1 = (x, z, v, w)$ and $X_2 = (y, u)$

With the components of $X_1 \varepsilon R^4$, denoting uninfected population and the components of $X_2 \varepsilon R^2$ denoting the infected population.

From (28)

$$E_0 = (X_1^*, 0),\ X_1^* = \left(\frac{\beta_H \theta N_H}{\mu_1}, 0, \frac{\beta_{hH}(1-\theta)N_H}{\mu_1}, 0, 0, 0\right) \quad (34)$$

Now for the first component, that is globally asymptotically stability of $X_1^*$, we have

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{bmatrix} \beta_H \theta N_H - \mu_1 x \\ \beta_H (1-\theta) N_H - \mu_1 z \\ 0 \\ 0 \end{bmatrix} \quad (35)$$

From equation (18) we solve the first equation i.e.

$$\frac{dx}{dt} = \beta_R \theta N_H - \mu_1 x$$

Using integrating factor (IF)

we have

$$\frac{dx}{dt} + \mu_1 x = \beta_R \theta N_H \quad (36)$$

which give our IF as

$$IF = e^{\int \mu_1 dt} = e^{\mu_1 t} \quad (37)$$

Multiplying both sides of (36) by our IF, we have

$$e^{\mu_1 t}\frac{dx}{dt} + e^{\mu_1 t}\mu_1 x = e^{\mu_1 t}\beta_R \theta N_H \quad (38)$$

Integrating both sides of (38), we have

$$e^{\mu_1 t} x + C_1 = \frac{\beta_R \theta N_H}{\mu_1} e^{\mu_1 t} + C_2 \quad (39)$$

$$e^{\mu_1 t} x = \frac{\beta_R \theta N_H}{\mu_1} e^{\mu_1 t} + C_2 - C_1 \quad (40)$$

$$e^{\mu_1 t} x = \frac{\beta_R \theta N_H}{\mu_1} e^{\mu_1 t} + C \quad (41)$$

Multiplying both sides of (41) by $e^{-\mu_1 t}$ we have

$$x(t) = \frac{\beta_R \theta N_H}{\mu_1} + Ce^{-\mu_1 t} \quad (42)$$

where

$C = C_2 - C_1$

For $x(0) = 0$

we have that

$$C = -\frac{\beta_R \theta N_H}{\mu_1}^{-\mu_1 t} \quad (43)$$

Substituting (43) above into (42), we have:

$$x(t) = \frac{\beta_H \theta N_H}{\mu_1} - \frac{\beta_H \theta N_H e^{-\mu_1 t}}{\mu_1} + x(0)e^{-\mu_1 t} \quad (44)$$

Similarly,

$$z(t) = \frac{\beta_H (1-\theta) N_H}{\mu_1} - \frac{\beta_H (1-\theta) N_H e^{-\mu_1 t}}{\mu_1} + z(0)e^{-\mu_1 t} \quad (45)$$

we have $x(t) + z(t) \to 1$ as $t \to \infty$

Regardless of the value of $x(0)$, and $z(0)$

Thus $X_1^* = \left(\frac{\beta_H \theta N_H}{\mu_1}, 0, \frac{\beta_{hH}(1-\theta)N_H}{\mu_1}, 0, 0, 0\right)$ is globally asymptotically stable.

Next for the 2nd condition, that is

$G(X_1, X_2) = AX_2 - G(X_1, X_2)$, we have that

$$A = \begin{bmatrix} -(\mu_1 + \delta_1 + \gamma) & \frac{px}{N_H} \\ \frac{qz}{N_H} & -(\mu_1 + \delta_1 + \gamma) \end{bmatrix} \quad (46)$$

This is clearly an M-matrix (the off diagonal elements of A are non-negative

$$G(X_1, X_2) = \begin{bmatrix} \frac{(pu + \alpha_3 w)x}{N_H} & -(\mu_1 + \delta_1 + \gamma) \\ \frac{(qy + \alpha_4 w)z}{N_H} & -(\mu_1 + \delta_1 + \gamma) \end{bmatrix} \quad (47)$$

then,

$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2) \geq 0 =$

i.e

$\hat{G}(X_1, X_2) \geq 0$

Since all the parameters are assumed to be non-negative, it is obvious that

$\hat{G}(X_1, X_2) \geq 0$, and this completes the proof.

## C. *Conclusion*

In this research work, a Mathematical Model with standard incidence is developed and analysed to study the transmission and control of Lassa Fever. Mathematically we modelled Lassa Fever as a 6 – dimensional system of non – linear ordinary differential equation. We first show that there exist a domain $D$ where our model is Mathematically and Epidemiologically well posed The Model incorporates two control parameters, condom efficacy $(\varepsilon)$ and compliance $(\tau)$ and $\delta_2$ which is the rate at which both the dormant and active vector are killed due to the use of Rodenticide/Rat poison. The Disease Free quilibrium points of the model were obtained, and analysed for stability. We obtained an important threshold parameter Basic Reproductive Number $R_0$, it is known that when $R_0 \leq 1$ the disease dies out, and when $R_0 > 1$ the disease persists in the population.

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