MATHEMATICAL MODEL OF THE TRANSMISSION DYNAMICS OF LASSAFEVER WITH SEPARATION OF INFECTED INDIVIDUAL AND TREATMENT AS CONTROL MEASURES

ABSTRACT

A non–linear deterministic mathematical model is formulated and analysed to study the controllability oflassa fever incorporating separationof infected individuals and treatment measures. The model assumes that humans susceptible acquired the Infection via interaction with the infected rodent populations at a constant rate and also the model assumes that treatment is only given to separated human population. The existence, uniqueness and positivity of the model's solution have been carried out and the results shows that the solution exist and is unique.Again, the disease – free equilibrium state was obtained and analysed. We obtained an important threshold parameter called the effective reproduction number $\mathcal{R}_{eff}$ using the next generation method. If $R_{eff} < 1$ the disease-free equilibrium exists and is locally and globally asymptotically stable, implying that Lassa fever can be controlled and eradicated within the population in a finite time and if the $\mathcal{R}_{eff} > 1$, the disease invade and become endemic in the population.

Keywords: Lassa fever, Mathematical Model, Separation of Infected Human, Treatment, Existence

1.0 INTRODUCTION

Lassa Fever (LF), technically known as Lassa Hemorrhagic Fever (LHF) is a deadly infectious illness to man caused by a Lassa Virus (LASV) or Lassa Hemorrhagic Fever Virus (LASHFV) from a carrier "multimam-mate rat" (Genus name *Mastomysnatalensis)*.These kind of rat (multimam-mate rat) are found in abundant in the sub-saharan part of Africa and infected rodent with the virus serves as a reservoir or host to the lassa virus (LASV) and transport it within the region in West Africa and some areas beyond. According to [1], Lassa fever is an acute viral infection associated with a wide spectrum of disease manifestations, which range from mild to hemorrhagic fever characterized by multiorgan failure.

Lassa fever virus is mainly a zoonosis (a disease that is animal-borne or transmitted to humans from animals),specifically an African rat, also called the natal multimammate rodent (*Mastomysnatalensis*) serves as a host or reservoir of the virus. Once the rat has become a carrier, it will excrete the virus throughout the rest of its lifetime through feces and urine creating ample opportunity for **exposure** ***[2].***The multimammate rat can quickly produce a large number of offspring, tends to colonize human settlements increasing the risk of rodent-human contact, and is found throughout the west, central and eastern parts of the African continent*[2]*. The virus is probably transmitted by contact with the **faeces** or urine of animals accessing grain stores in residences*Werner et al* ***[3].***Transmission or Infection of Lassa virus to humans typically occurs by direct or indirect exposure to animal excrement through the respiratory or gastrointestinal tracts or eating contaminated food, touching soiled objects, or exposure to open cuts or sores. Inhalation of tiny particles of infectious material (aerosol) is believed to be the most significant means of exposure. It is possible to acquire the infection through broken skin or mucous membranes that are directly exposed to infectious material Because Mastomys rodents often live in and around homes and scavenge on leftover human food items or poorly stored food. Mastomys rodents are sometimes consumed as a food source and infection may occur when rodents are caught and prepared. Direct contact with infected rodents is not the only way in which people are infected; person-to-person transmission may occur after exposure to virus in the blood, tissue, secretions, or excretions of a Lassa virus-infected \individual, presenting a disease risk for healthcare workers (called

nosocomial transmission) where proper personal protective equipment (PPE) is not available or not used. Lassa virus may be spread in contaminated medical equipment, such as reused needles. Also, during sexual intercourse, the virus can be transmitted because the virus is present in urine for between three and nine weeks after infection, and it can be transmitted in semen for up to three months after becoming infected**[4] and "Lassa fever"[5].**Casual contact (including skin- to-skin contact without exchange of body fluids) does not spread Lassa virus. Finally, No study has proven presence of lassa virus in the breast milk, but the high level of viremia suggests it may be possible**,[6].**Above all, individuals who are at a higher risk of contracting the infection are those who live in rural areas where Mastromys are discovered, and where sanitation is not prevalent.

Lassa fever is endemic in parts of west Africa including Sierra Leone, Liberia, Guinea and Nigeria; however, other neighboring countries are also at risk, as the animal vector for Lassa virus, the "multimammate rat" (*Mastomysnatalensis*) is distributed throughout the region. In 2009, the first case from Mali was reported in a traveler living in southern Mali; Ghana reported its first cases in late 2011. Separation cases have also been reported in Côte d'Ivoire and Burkina Faso and there is serologic evidence of Lassa virus infection in Togo and Benin. The number of Lassa virus infections per year in west Africa is estimated at 100,000 to 300,000, with approximately 5,000 deaths. Unfortunately, such estimates are crude, because surveillance for cases of the disease is not uniformly performed. In some areas of Sierra Leone and Liberia, it is known that 10%-16% of people admitted to hospitals every year have Lassa fever, which indicates the serious impact of the disease on the population of this region**.In this paper, a non – linear deterministic mathematical model of Lassa fever shall be formulated to study the impact of Transmission Dynamics on Lassa Fever Incorporating separation and Treatment as a Control Measures. We wish to show that our results both analytical and numerical with the control measurescan reduce the spread of Lassa fever to an optimal level in infinite time.**

1.1 **The Specific Objective of the Study**

The specific objective of these study are to:

1. To formulate and analyse a mathematical model on the controllability of lassa fever incorporating isolation and treatment measures in terms of the reproduction number.
2. To determine the stability of the equilibrium points.
3. To understand how isolation and treatment can reduce mortality rate among the infected individuals.
4. To contribute on how isolation can reduce the force of infection rate among the unaffected individuals.

1.2 MODEL FORMULATION AND ANALYSIS

1.3 Assumptions of the model

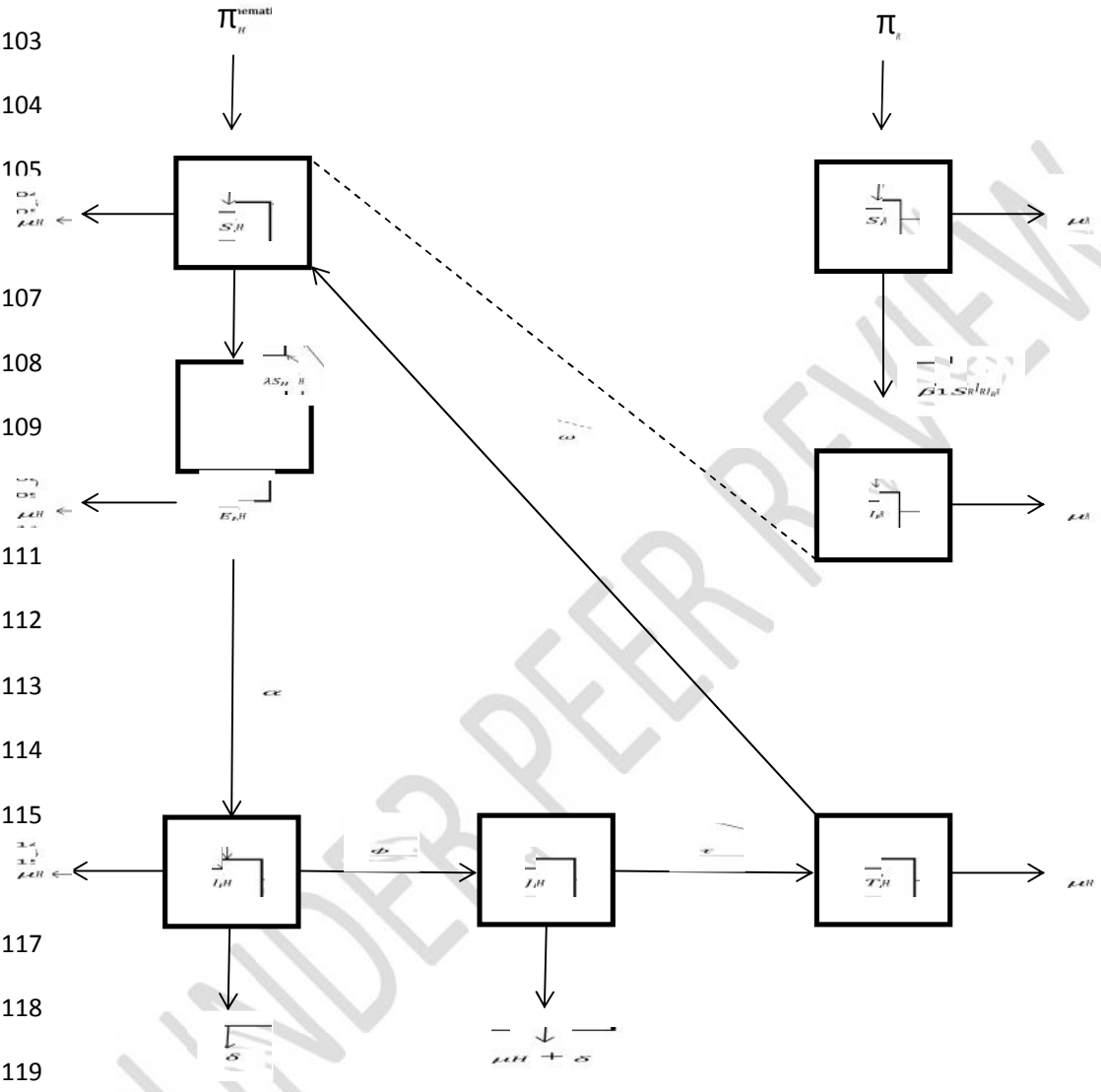
i. The total human and rodent populations are given by $N_H = S_H + E_H + I_H + J_H + T_H$ and $N_R = S_R + I_R$ respectively.
ii. We assumed that treatment is only given to separate human population.
iii. Recruitment into the susceptible population is either by birth or immigration.
iv. Members of the infected human population can as well move to the susceptible human population via treatment.

v. Infection is acquired via interaction unlike the direct contact in the existing model.

Table 1: State Variables and Parameters of the Model

Variables/Parameters	Description
S_H	Susceptible human at time t
E_H	Exposed human at time t
I_H	Infected human at time t
J_H	separated human at time t
T_H	Treated human at time t
S_R	Susceptible rodent at time t
I_R	Infected rodent at time t
π_H	Recruitment rate into the susceptible human
π_R	Recruitment rate into the susceptible rodent
μ_H	Natural death rate in human
μ_R	Natural death rate in rodent
p_2	**Effective contact between infected human and susceptible human**
β_1	**Effective contact between infected rats and either susceptible human or susceptible rodent**
σ_1	Progression rate to active Lassa fever
α	Separation rate
ϕ	Treatment rate
ψ	Recovery rate
ω	Human disease induced death
δ	Probability of getting Lassa fever
β	
N_H	Total population of human
N_R	Total population of rodent
λ	Force of infection

Figure 1: Schematic diagram of our Model



1.4 The model equations

From the above assumptions and the schematic diagram, the model will be governed by the following non – linear differential equation;

$$\frac{dS_H}{dt} = \pi_H + \omega T_H - \lambda S_H - \mu_H S_H \qquad 1$$

$$\frac{dE_H}{dt} = \lambda S_H - (\mu_H + \alpha)E_H \qquad 2$$

$$\frac{dI_H}{dt} = \alpha E_H - (\mu_H + \delta + \phi)I_H \qquad 3$$

$$\frac{dJ_H}{dt} = \phi I_H - (\mu_H + \delta + \tau)J_H \quad 4$$

$$\frac{dT_H}{dt} = \tau J_H - (\mu_H + \omega)T_H \quad 5$$

$$\frac{dS_R}{dt} = \pi_R - \beta_1 S_R I_R - \mu_R S_R \quad 6$$

$$\frac{dS_R}{dt} = \beta_1 S_R I_R - \mu_R I_R \quad 7$$

where

$$\lambda = \beta_1 I_R - \beta_2 I_H \quad 8$$

With the initial conditions $S_H(0) = S_H^0, E_H(0) = E_H^0, I_H(0) = I_H^0, J_H(0) = J_H^0, T_H(0) = T_H^0, S_R(0) = S_R^0, I_R(0) = I_R^0$ and $N(0) = N_0$. The force of infection $\lambda = \beta_1 I_R - \beta_2 I_H$, where β_1 and β_2 are the effective contact between infected rats and either susceptible humans or susceptible rodents and effective contact between infected human and susceptible human respectively with,

$$N_H(t) = S_H(t) + E_H(t) + I_H(t) + J_H(t) + T_H(t) \quad 9$$

Where $N_H(t)$ denotes the total human population at a given time with its time derivative given by;

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dJ_H}{dt} + \frac{dT_H}{dt} \quad 10$$

Plugging (1) – (5) into (10) gives

$$\frac{dN_H}{dt} = \pi_H - (I_H + J_H)\delta - \mu_H N_H \quad 11$$

Also,

$$N_R(t) = S_R(t) + I_R(t) \quad 12$$

Where $N_R(t)$ denotes the total rodents population at a given time with its time derivative given by;

$$\frac{dN_R}{dt} = \frac{dS_R}{dt} + \frac{dI_R}{dt} \quad 13$$

Substituting (6) and (7) into (13) gives

$$\frac{dN_R}{dt} = \pi_R - \mu_R N_R \quad 14$$

1.5 The invariant region (Region of biological interest)

As the system (1) – (5) monitors human population, all related state variables and parameters are assumed to be non – negative for all $t \geq 0$. Therefore, the above system is dissipative in the proper subset $\Gamma \subset \mathbb{R}_+^5$. Thus, we state and prove the following results:

Lemma 1: The solutions of the system (1) – (7) are feasible for all $t > 0$ if they enter the invariant region $\Gamma = (S_H, E_H, I_H, J_H, T_H, S_R, I_R)$.

Proof 1:

Let $\Gamma = (S_H, E_H, I_H, J_H, T_H, S_R, I_R)$ be any solution of the system (1) – (7), with non – negative initial conditions. From equation (11), we see that in the absence of Lassa fever $(I_H = J_H = 0)$, we obtained;

$$\frac{dN_H}{dt} \leq \pi_H - \mu_H N_H \qquad 15$$

Rearranging (15) gives

$$\frac{dN_H}{dt} + \mu_H N_H \leq \pi_H \qquad 16$$

Solving (16) using the method of integrating factor (IF) we compute the IF as follows:

$$IF = e^{\int \mu_H dt} = e^{\mu_H t} \qquad 17$$

Multiplying both sides of (16) by (17) yields

$$e^{\mu_H t}\frac{dN_H}{dt} + \mu_H N_H e^{\mu_H t} \leq \pi_H e^{\mu_H t}$$

That is,

$$\frac{d}{dt}(\mu_H N_H e^{\mu_H t}) \leq \pi_H e^{\mu_H t} \qquad 18$$

Integrating both sides of (18) gives

$$N_H(t)e^{\mu_H t} \leq \frac{\pi_H}{\mu_H} e^{\mu_H t} + \psi \qquad 19$$

Where ψ is a constant of integration.

This means

$$N_H(t) \leq \frac{\pi_H}{\mu_H} + \psi e^{-\mu_H t} \qquad 20$$

Applying the initial condition: $N_H(0) = N_H^0$, we obtain;

$$N_H^0 - \frac{\pi_H}{\mu_H} \leq \psi \qquad 21$$

Substituting (21) into (20) we have

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

Applying Birkhoff and Rota's theorem on differential inequality (Birkhoff and Rota, 1982), we have $0 \leq N_H(t) \leq \frac{\pi_H}{\mu_H}$, as $t \to \infty$.

The total population approaches $K = \frac{\pi_H}{\mu_H}$, as $t \to \infty$. which is commonly known as the carrying capacity. Therefore, the feasible solutions set of the extended model (1) – (5) enters the region below

$$\left\{\Gamma = \Gamma = (S_H, E_H, I_H, J_H, T_H) \in \mathbb{R}_+^5 : S_H > 0, E_H \geq 0, I_H \geq 0, J_H \geq 0, T_H \geq 0, N_H \leq \frac{\pi_H}{\mu_H}\right\}.$$

Thus in this region our model is biologically feasible. Here whenever $N > \frac{\pi_H}{\mu_H}$ then $\frac{dN_H}{dt} < 0$ which means the population reduces asymptotically to the carrying capacity and whenever $N \leq \frac{\pi_H}{\mu_H}$ every solution with initial condition in Γ remains in that region for $t > 0$, so the model is well posed in Γ. Therefore, the region Γ is positively – invariant (i.e. solutions remain positive for all time.) and the model is well posed and biologically meaningful and this ends the proof of the Lemma 1.

1.6 Existence of disease free equilibrium state (ε_0) of the model.

Here, we compute the model disease free equilibrium state by setting the time – derivatives on the right hand sides of the model system (1) – (9) to zero such that

$$\frac{dS_H}{dt} = \frac{dE_H}{dt} = \frac{dI_H}{dt} = \frac{dJ_H}{dt} = \frac{dT_H}{dt} = \frac{dS_R}{dt} = \frac{dI_R}{dt} = 0 \qquad 22$$

So that we now have

$$0 = \pi_H + \omega T_H - (\beta_1 I_R + \beta_2 I_H) S_H - \mu_H S_H \quad 23$$

$$0 = (\beta_1 I_R + \beta_2 I_H) S_H - (\mu_H + \alpha) E_H \quad 24$$

$$0 = \alpha E_H - (\mu_H + \delta + \phi) I_H \quad 25$$

$$0 = \phi I_H - (\mu_H + \delta + \tau) J_H \quad 26$$

$$0 = \tau J_H - (\mu_H + \omega) T_H \quad 27$$

$$0 = \pi_R - \beta_1 S_R I_R - \mu_R S_R \quad 28$$

$$0 = \beta_1 S_R I_R - \mu_R I_R \quad 29$$

Recall that, the disease free equilibrium state of the model (1) – (9) is scenario where there is no disease in the system which implies that

$$E_H = I_H = J_H = T_H = I_R = 0$$

Plugging (31) into (1) – (9) and solving accordingly we obtain

$$\varepsilon_0 = (S_H^0, E_H^0, I_H^0, J_H^0, T_H^0, S_R^0, I_R^0) = \left(\frac{\pi_H}{\mu_H}, 0, 0, 0, 0, \frac{\pi_R}{\mu_R}, 0\right) \quad 30$$

1.7 Effective reproduction number ($\mathcal{R}_{eff}$)

$$\mathcal{R}_0 = \rho(FV^{-1})$$

where $\rho(FV^{-1})$ is the spectral radius of next generation matrix.

We calculate the basic reproduction number using the next generation operator method on the system (1) – (9) as follows;

The vector F_i of the rates of the new infection in compartment E_H, I_H, J_H, T_H and I_R is given by

$$F_i = \begin{pmatrix} (\beta_1 I_R + \beta_2 I_H) S_H \\ 0 \\ 0 \\ 0 \\ \beta_1 I_R S_R \end{pmatrix} \quad 31$$

Also, the remaining transfer terms in compartment E_H, I_H, J_H, T_H and I_R is given by

$$V_i = \begin{pmatrix} (\mu_H + \alpha) E_H \\ (\mu_H + \delta + \phi) I_H - \alpha E_H \\ (\mu_H + \delta + \tau) J_H - \phi I_H \\ (\mu_H + \omega) E_H - \tau J_H \\ \mu_H I_R \end{pmatrix} \quad 32$$

The matrix of partial derivative of F_i at the disease free equilibrium state at $\mathcal{E}_0 = (S_H^0, 0,0,0,0, S_R^0, 0)$ is given by

$$F(\mathcal{E}_0) = \begin{pmatrix} 0 & \frac{\beta_2 \pi_H}{\mu_H} & 0 & 0 & \frac{\beta_1 \pi_H}{\mu_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_1 \pi_R}{\mu_R} \end{pmatrix} \quad 33$$

Rewriting (33) yields

$$F(\mathcal{E}_0) = \begin{pmatrix} 0 & F_{12} & 0 & 0 & F_{15} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & F_{55} \end{pmatrix} \quad 34$$

where;

$$F_{12} = \frac{\beta_2 \pi_H}{\mu_H}$$

$$F_{15} = \frac{\beta_1 \pi_H}{\mu_H} \quad 35$$

$$F_{55} = \frac{\beta_1 \pi_R}{\mu_R}$$

Also, the matrix of the partial derivatives of V_i at the disease free equilibrium state $\mathcal{E}_0 = (S_H^0, 0,0,0,0, S_R^0, 0)$ is given by

$$V(\mathcal{E}_0) = \begin{pmatrix} (\mu_H + \alpha) & 0 & 0 & 0 & 0 \\ -\alpha & (\mu_H + \delta + \phi) & 0 & 0 & 0 \\ 0 & -\phi & (\mu_H + \delta + \tau) & 0 & 0 \\ 0 & 0 & -\tau & (\mu_H + \omega) & 0 \\ 0 & 0 & 0 & 0 & \mu_R \end{pmatrix} \quad (36)$$

Equation (36) can also be written as;

$$V(\mathcal{E}_0) = \begin{pmatrix} V_{11} & 0 & 0 & 0 & 0 \\ -V_{21} & V_{22} & 0 & 0 & 0 \\ 0 & -V_{32} & V_{33} & 0 & 0 \\ 0 & 0 & -V_{43} & V_{44} & 0 \\ 0 & 0 & 0 & 0 & V_{55} \end{pmatrix} \quad (37)$$

where;

$$\left.\begin{array}{ll} V_{11} = \mu_H + \alpha & V_{55} = \mu_R \\ V_{22} = \mu_H + \delta + \phi & V_{21} = \alpha \\ V_{33} = \mu_H + \delta + \tau & V_{32} = \phi \\ V_{44} = \mu_H + \omega & V_{43} = \tau \end{array}\right\} \quad (38)$$

Computing the inverse of (37) gives

$$V^{-1} = \begin{pmatrix} \frac{1}{V_{11}} & 0 & 0 & 0 & 0 \\ \frac{V_{21}}{V_{11}V_{22}} & \frac{1}{V_{22}} & 0 & 0 & 0 \\ 0 & \frac{V_{32}}{V_{22}V_{33}} & \frac{1}{V_{11}} & 0 & 0 \\ \frac{V_{21}V_{32}V_{43}}{V_{11}V_{22}V_{33}V_{44}} & \frac{V_{43}}{V_{22}V_{44}} & \frac{V_{43}}{V_{33}V_{44}} & \frac{1}{V_{44}} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{V_{55}} \end{pmatrix} \quad (39)$$

Rewriting (39) we have;

$$V^{-1} = \begin{pmatrix} A_{11} & 0 & 0 & 0 & 0 \\ A_{21} & A_{22} & 0 & 0 & 0 \\ 0 & A_{32} & A_{33} & 0 & 0 \\ A_{41} & A_{42} & A_{43} & A_{44} & 0 \\ 0 & 0 & 0 & 0 & A_{55} \end{pmatrix} \quad (40)$$

where;

$$A_{11} = \frac{1}{(\mu_H+\alpha)} \quad (41)$$

$$A_{21} = \frac{\alpha}{(\mu_H+\alpha)(\mu_H+\delta+\phi)} \quad (42)$$

$$A_{22} = \frac{\phi}{(\mu_H+\delta+\phi)} \quad (43)$$

$$A_{32} = \frac{\phi}{(\mu_H+\delta+\phi)(\mu_H+\delta+\tau)} \quad (44)$$

$$A_{33} = \frac{1}{(\mu_H+\delta+\tau)} \quad (45)$$

$$A_{41} = \frac{\alpha\phi\tau}{(\mu_H+\alpha)(\mu_H+\delta+\phi)(\mu_H+\delta+\tau)(\mu_H+\omega)} \quad 46$$

$$A_{42} = \frac{\tau}{(\mu_H+\delta+\phi)(\mu_H+\omega)} \quad 47$$

$$A_{43} = \frac{\tau}{(\mu_H+\delta+\phi)(\mu_H+\omega)} 48$$

$$A_{44} = \frac{1}{(\mu_H+\omega)} \quad 49$$

$$A_{42} = \frac{1}{\mu_R} \quad 50$$

To compute FV^{-1} we use (41) and (43) so that;

$$FV^{-1} = \begin{pmatrix} 0 & F_{12} & 0 & 0 & F_{15} \\ 0 & 0 & 0 & 0 & 0 \\ \vdots & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & F_{55} \end{pmatrix} \begin{pmatrix} A_{11} & 0 & 0 & 0 & 0 \\ A_{21} & A_{22} & 0 & 0 & 0 \\ 0 & A_{32} & A_{33} & 0 & 0 \\ A_{41} & A_{42} & A_{43} & A_{44} & 0 \\ 0 & 0 & 0 & 0 & A_{55} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & F_{11}A_{12} & 0 & 0 & F_{11}F_{15} \\ 0 & F_{12}A_{12} & 0 & 0 & F_{12}A_{15} \\ \vdots & 0 & 0 & 0 & 0 \\ 0 & F_{41}A_{12} & 0 & 0 & F_{41}A_{15} \\ 0 & 0 & 0 & 0 & F_{55}A_{55} \end{pmatrix} \quad 51$$

It follows that the effective reproduction number $\mathcal{R}_{eff}$ is computed by taking the spectral radius (dominant eigenvalue) of the matrix FV^{-1} using the characteristics equation **36 and 42**

$$det(FV^{-1} - \lambda_E I) = 0 \quad 52$$

or

$$\begin{vmatrix} -\lambda & F_{11}A_{12} & 0 & 0 & F_{11}F_{15} \\ 0 & F_{12}A_{12} - \lambda & 0 & 0 & F_{12}A_{15} \\ 0 & 0 & -\lambda & 0 & 0 \\ 0 & F_{41}A_{12} & 0 & -\lambda & F_{41}A_{15} \\ 0 & 0 & 0 & 0 & F_{55}A_{55} - \lambda \end{vmatrix} = 0 \quad 53$$

Evaluating (56) accordingly gives;

$$\lambda_1 = \lambda_3 = \lambda_4 = 054$$

and

$$\lambda_2, \lambda_5 = max\left(\frac{\beta_2\alpha\pi_H}{\mu_H(\mu_H+\alpha)(\mu_H+\delta+\phi)}, \frac{\beta_1\pi_R}{\mu_R}\right) 55$$

Therefore, the largest (dominant) eigenvalue also known as the effective reproduction denoted by $\mathcal{R}_{eff}$ is given by

$$\mathcal{R}_{eff} = \frac{\beta_2\alpha\pi_H}{\mu_H(\mu_H+\alpha)(\mu_H+\delta+\phi)} 56$$

with

$\frac{1}{\mu_H}, \frac{1}{(\mu_H+\alpha)}$ and $\frac{1}{(\mu_H+\delta+\phi)}$ which refers to per capital human mortality,

Biological Interpretation 1: The biological meaning of the parameter components of the effective reproduction number are as follows:

$\left(\frac{\pi_H}{\mu_H}\right)$: The carrying capacity for human population.

$\left(\frac{\alpha}{\mu_H+\alpha}\right)$: The proportion of individuals from the exposed human that becomes infectious.

$\left(\frac{\beta_2}{\mu_H+\delta+\phi}\right)$: The average number of susceptible human infected by a **single human infectious**.

1.8 Numerical Simulations

Here, we carryout numerical simulation of the model (1) – (9) using the set ofreasonable parameters and initial values given in Table 2 and 3 whose sources are mainly from [24] as well as assumed values based on the literature of the disease in order to have more realistic simulation results

Table 2: Parameters Values for Numerical and Sensitivity Analysis

Parameters	Values	Sources
eters	2000	[18]
	500	[18]
	0.2	[18]
	0.2	Estimated
	0.003	[18]
	0.2	Estimated
	0.75	Assumed
	0.02	[18]
	0.02	[18]
	0.1	Assumed
	0.54	Assumed

Table 3: Parameters Values for Numerical and Sensitivity Analysis

Variables	Values	Sources
ria bles	10000	[19]

	3000		Assumed
	2000		[19]
	1500		[19] Assumed
	600		[19]
	200	[19]	
	125		[19]

Table 4: Sensitivity Indices of $\mathcal{R}_{eff}$

S/N	Parameter	Sensitivity Index	Sign
1		1.0000000000	+
2		1.0000000000	+
3		0.8695652173	+
4		0.3125000000	−
5		0.6250000000	−
6		1.9320652180	−

1.9 Numerical Results

In this sub – section, we presents the numerical results of the above experiment as follows:

2.1 Simulation results showing the trends of the state variables of the Lassa fever model with separation rate for the infected class.

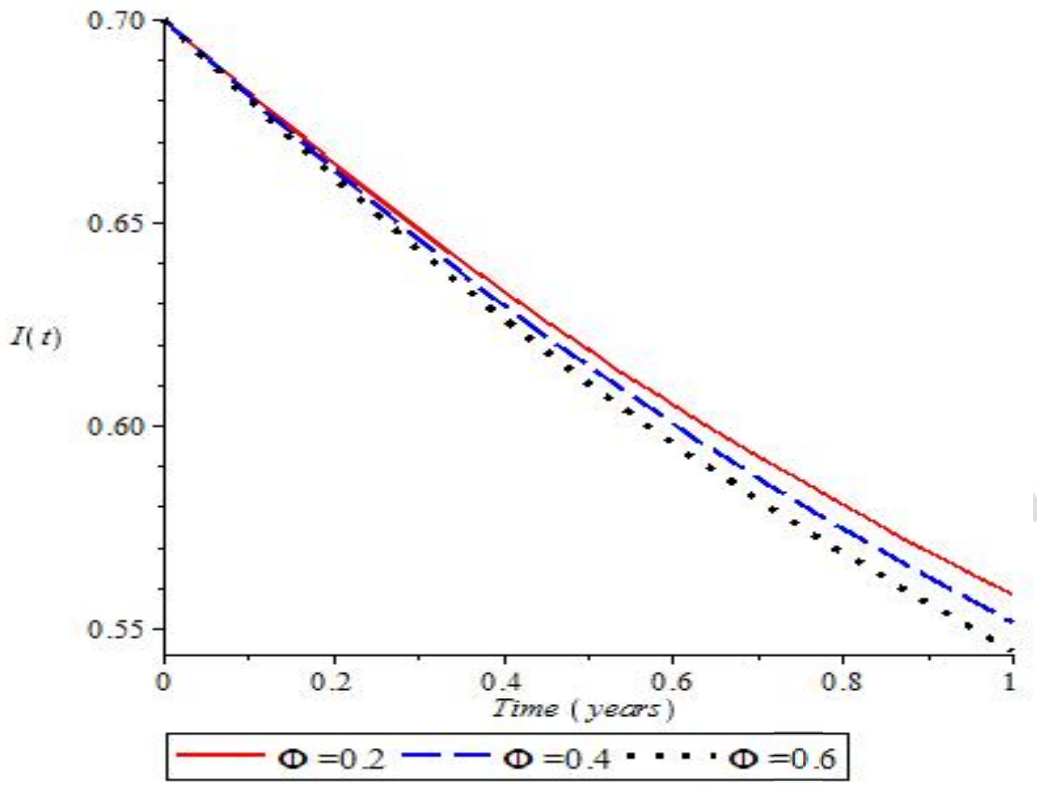


Figure 2: Simulation results showing the effect of separation rate ϕ on infected humans.

2.2 Simulation results showing the trends of the state variables of the Lassa fever model with recovery rate due to treatment for; infected humans and treated humans.

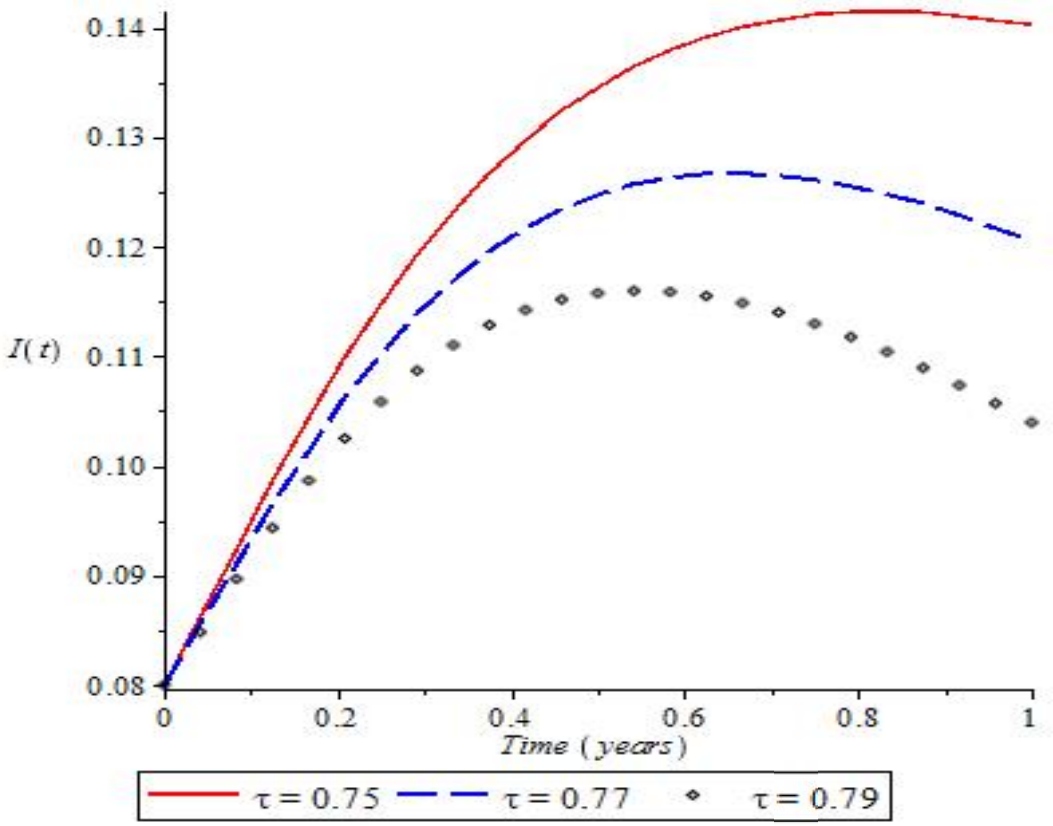


Figure 4: Simulation results showing the impact of treatment rate τ on infected individuals.

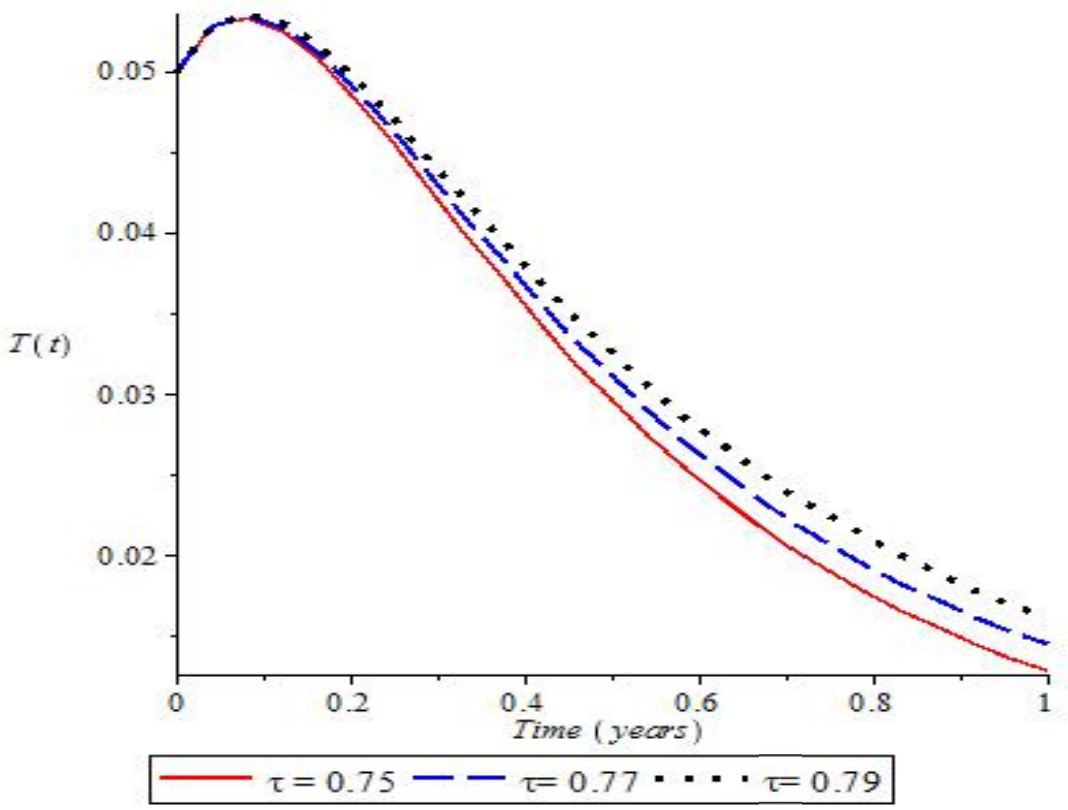


Figure 5: Simulation results showing the effect of treatment rate τ on treated humans.

2.0 Conclusion

Nigeria is endemic to Lassa Fever and has being rated as one of thecountry inWest Africa with the high transmission rate of Lassa fever,but we should not panic as theimpact ofthis paperwill highly contribute in curbing Lassa fever since our Reproduction number is less than one ($\mathcal{R}_{eff} < 1$), which simply implies that Lassa Fever can beeradicatedfrom the country.Therefore we conclude thatsince our$\mathcal{R}_{eff} < 1$ isless than one the disease will surely die out in infinite time.

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