

Modelling Lassa fever Dynamics Incorporating Quarantine Rate

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Abstract:-In this article a Lassa fever dynamics control that incorporates quarantine class is proposed. The population is sub-divided into two sub population namely the human and rodent class. The human population was sub-divided into four sub-classes: susceptible, $S_H(t)$, infected, $I_H(t)$, quarantine, $Q_H(t)$ and recovered, $R_H(t)$ humans while the rodent class is sub-divided into susceptible, $S_R(t)$ and infected, $I_R(t)$ rodents. The Disease Free Equilibrium (DFE) was analysed and investigated using stability theory of differential equations. The sufficient condition for disease free equilibrium was checked using Jacobian matrix approach. It was shown that the introduced quarantine parameter helps in controlling and eradication of Lassa fever virus in the population with respect to time. Numerical simulations were also carried out to investigate the influence of key parameters on the spread of the disease, especially the quarantine parameter to support the analytical conclusion and illustrate possible behavioural scenario of the model.

Keywords: Lassa Fever, Quarantine Rate, Local Stability, Disease Free Equilibrium, Disease Endemic Equilibrium

I. INTRODUCTION

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 Multi-mammate Rat species known as *Mastomys natalensis* [1, 2]. 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.

Okuonghae and Okuonghae [2] developed a Susceptible Infected Susceptible (SIS) model for the transmission of Lassa fever disease. The model investigated the equilibrium states and examined them for endemic and epidemic situations. The model further calculated the basic reproductive number and gave conditions for disease outbreak.

In similar vein, a Susceptible Infected Recovered (SIR) model for controlling Lassa fever transmission in northern part of Edo state, Nigeria was developed. The model advocated for

health policies that will keep the basic reproductive number, R_0 below 1, thereby keeping the transmission of the disease under control [3, 4].

The Lassa fever model developed by [5] is a major shift from the first two papers cited. The researchers divided the human population into susceptible, S_H and the infected, I_H humans while the reservoir population was divided into infant, R_H and adult, Areservoir. The virus (generated from urine and faeces) in the environment was represented by V . The major parameters of their model are, H_b per capital birth rate of Human, R_b per capital birth rate of the reservoir, R per capital natural death rate of Human, H per capital death rate of the reservoir, H Lassa fever induced death rate, R mortality death of the reservoir due to hunting, effective contact rate for human, and effective contact rate between reservoir and human, recovery rate of infected human and progression rate from infant to adult reservoir. The model recommended that efforts should be made to keep the basic reproductive number below unity to ensure that the virus is contained [5, 6, 7].

It is against this framework that this research is aimed at modelling Lassa fever dynamics control incorporating quarantine. In this research, we introduce new variable called the quarantine class. By introducing the quarantine class, we succeeded in making the basic reproductive number below unity suggested by [5].

II. MODEL FORMULATION

Lassa fever models usually encompassed individuals who have not come into contact with the virus known as susceptible humans $S_H(t)$. The susceptible rodents $S_R(t)$ become at the rate ϕ and infectious rodent infects human at the rate ρ when they come into direct contact with the infected rodent, their urine, excreta or eating food contaminated by the infectious rodent's saliva. The infected human are treated at the rate δ and some moved to the quarantine human class $Q_H(t)$ at the rate α_2 . Those who are not aware of the treatment will be removed from the population through death at the rate α_3 . While the quarantine human class return to the susceptible human class at the rate γ_1 . The existence of region where the model is epidemiologically feasible is established. Stability analysis of the disease free equilibrium is investigated through the

reproduction number obtained using the next generation operator approach.

In this model, individuals are recruited into the susceptible population of human at the rate π and susceptible population of rodent at the rate η . The infection spread at the rate k , where

k and is the probability of getting Lassa fever, c is the contact rate. Both human and rodent die naturally at the rate μ_1 and μ_2 respectively. The total population of human and Rodent are given by $N_H(t) = S_H(t) + I_H(t) + Q_H(t) + R_H(t)$ and $N_R(t) = S_R(t) + I_R(t)$ respectively.

$N(t) = N_H(t) + N_R(t) =$ Total population size at time t .

The table below shows the variables and parameters used in the new model.

Table1: Model variables and parameters

Variable	Description
$S_H(t)$	Number of susceptible human at time t
$S_R(t)$	Number of susceptible rodent at time t
$I_H(t)$	Number of infected human at time t
$I_R(t)$	Number of infected rodent at time t
$R_H(t)$	Number of Recovered human at time t
$Q_H(t)$	Number of quarantine at time t

Parameter	Description
β	Probability of getting Lassa fever infection
π	Recruitment rate into susceptible humans
μ_1	Natural death rate human
μ_2	Natural death rate rodent
α_1	Progression rate to active Lassa fever
α_2	Quarantine rate
α_3	Treatment rate
ω	Disease induced death rate of infected humans rate
ρ	Rate at which rodent infect humans
ϕ	Rate at which susceptible rodents become infected
η	Recruitment rate into susceptible rodent
c	Contact rate
γ_1	The rate at which susceptible removed humans become susceptible again

The following diagram describes the dynamics of infection, and will be useful in the formulation of model equations.

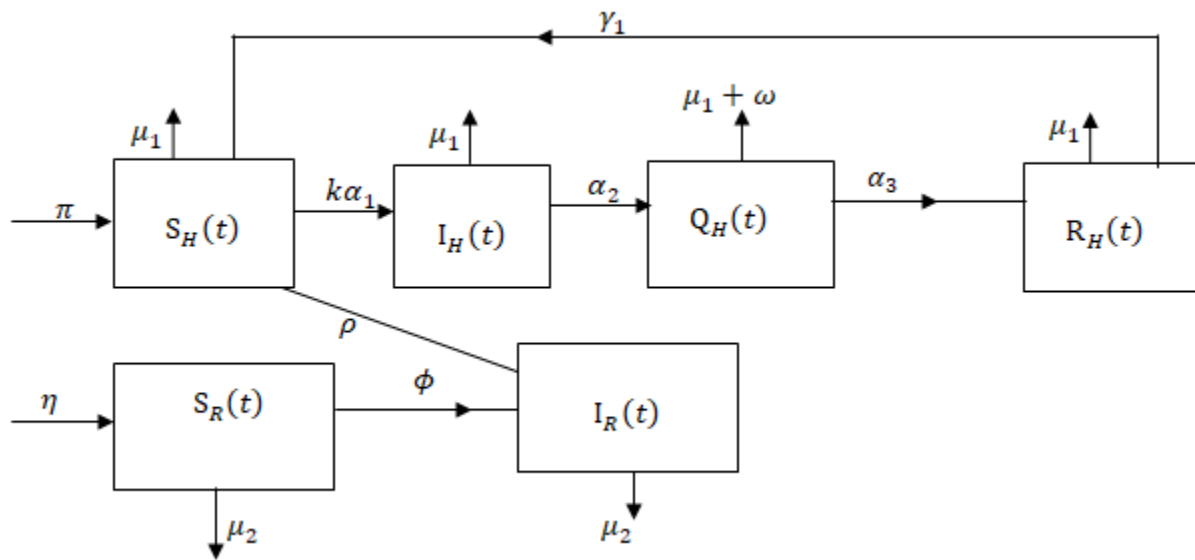


Figure 1: Flow diagram of the dynamics Lassa Fever

III. ASSUMPTIONS OF THE MODEL

The model assumptions include the following:

1. Assume that some people are not aware of the presence of Lassa fever.
2. We assume that treatment is given to only infected individuals.
3. That all individuals have equal chance of being infected if they come in contact with infectious rodents.
4. We assume that some infected humans move to the quarantine class before moving to the recovered class, this is due to the fact that disease is contagious.
5. That all the recruits are neither immune nor infected.

6. Assume that Lassa fever can be contacted through urine, saliva and excreta from the infectious rodents.
7. That despite the public health education campaigns, some people are still ignorant of the disease.
8. We assume that the quarantine humans recovered.

IV. THE MODEL EQUATIONS

From the assumptions and the flow diagram above, the following model equations are derived.

For the Human Populations:

$$\frac{dS_H}{dt} = \pi + \rho I_R + \gamma_1 R_H - k\alpha_1 S_H - \mu_1 S_H \quad (1)$$

$$\frac{dI_H}{dt} = k\alpha_1 S_H - (\mu_1 + \alpha_2 + \omega) I_H \quad (2)$$

$$\frac{dQ_H}{dt} = \alpha_2 I_H - (\mu_1 + \alpha_3) Q_H \quad (3)$$

$$\frac{dR_H}{dt} = \alpha_3 Q_H - (\mu_1 + \gamma_1) R_H \quad (4)$$

For the Rodent Populations:

$$\frac{dS_R}{dt} = \eta - (\mu_2 + \phi) S_R \quad (5)$$

$$\frac{dI_R}{dt} = \phi S_R - (\mu_2 + \rho) I_R \quad (6)$$

With initial conditions

$$S_H(t) > 0, I_H(t) > 0, Q_R(t) > 0, R_H(t) = 0, S_R(t) > 0, I_R(t) > 0. \text{ The force of the infection } k = \frac{\beta c I}{N}$$

$$\left. \begin{aligned} N_H(t) &= S_H(t) + I_H(t) + Q_R(t) + R_H(t) \\ N_H(t) &= S_R(t) + I_R(t) \end{aligned} \right\} \quad (7)$$

$$N(t) = N_H(t) + N_R(t) \quad (8)$$

V. MATHEMATICAL ANALYSIS OF THE MODEL

Existence of the Disease Free Equilibrium (DFE), E_f

In the absence of the disease, it implies that $I_H(t) = 0, Q_R(t) = 0, R_H(t) = 0, I_R(t) = 0$.

Therefore the above system of equations is reduced to

$$\frac{dS_H}{dt} = \pi + \mu_1 S_H \quad (9)$$

$$\frac{dS_R}{dt} = \eta - (\mu_2 + \phi) S_H \quad (10)$$

hence letting equation (9) and (10) to zero and solving them simultaneously, we get

$$S_H = \frac{\pi}{\mu_1}, S_R = \frac{\pi}{\mu_2 + \phi},$$

hence,

$$E_f = (S_H, I_H, Q_H, R_R, S_R, I_R) = \left(\frac{\pi}{\mu_1}, 0, 0, 0, \frac{\pi}{\mu_2 + \phi}, 0 \right) \quad (11)$$

Existence of the Endemic Equilibrium (EE) State

Solving the system (1) – (7) simultaneously, we get

$$S_H^* = \frac{N(\mu_1 + \alpha_2 + \omega)}{\beta(\mu_2 + \phi)\alpha_1} \quad (12)$$

$$I_H^* = \frac{-1}{(\mu_1 + \alpha_2 + \omega)(\mu_2 + \phi)(\rho + \mu_2)\beta c \alpha_1 \alpha_3}$$

$$\left\{ \begin{aligned} &-\pi(\mu_2 + \phi)(\rho + \mu_2)\beta c \alpha_1 \alpha_3 - \\ &\rho \alpha_3 \phi \beta c \alpha_1 (\mu_1 + \alpha_2 + \omega)(\mu_2 + \phi)(\rho + \mu_2)\beta c \alpha_1 \alpha_3 \mu_1 \\ &+(\mu_2 + \phi)(\rho + \mu_2)(\mu_1 + \gamma_1)\beta c \alpha_1 \gamma_1 \end{aligned} \right\} \quad (13)$$

$$I_H^* = 0 \quad (14)$$

$$Q_H^* = \frac{-1}{(\mu_1 + \alpha_2 + \omega)(\mu_2 + \phi)(\rho + \mu_2)\beta c \alpha_1 \alpha_3}$$

$$\alpha_2 \left[\begin{aligned} &-\pi(\mu_2 + \phi)(\rho + \mu_2)\beta c \alpha_1 \alpha_3 - \rho \alpha_3 \phi \beta c \alpha_1 + \\ &(\mu_1 + \alpha_2 + \omega)(\mu_2 + \phi)(\rho + \mu_2)\beta c \alpha_1 \alpha_3 \mu_1 \\ &+(\mu_2 + \phi)(\rho + \mu_2)(\mu_1 + \gamma_1)\beta c \alpha_1 \gamma_1 \end{aligned} \right] \quad (15)$$

$$Q_H^* = 0 \quad (16)$$

$$R_H^* = \frac{(\mu_1 + \gamma_1)}{\alpha_3} \quad (17)$$

$$S_R^* = \frac{\eta}{(\mu_2 + \phi)} \quad (18)$$

$$I_R^* = \frac{\eta \phi}{(\mu_2 + \phi)(\rho + \mu_2)} \quad (19)$$

VI. COMPUTATION OF THE BASIC REPRODUCTIVE NUMBER (R_0) OF THE MODEL

The Basic Reproductive number (R_0) is define as the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone else is susceptible. $R_0 = 1$ is a threshold below which the generation of secondary cases is insufficient to maintain the infection in human community. If $R_0 < 1$, the number of infected individuals will decrease from generation to next and the disease dies out and if $R_0 > 1$ the number of infected individuals will increase from generation to the next and the disease will persist [10]

We first rearranged the model equations (1) – (7) beginning with the infective classes to obtain the following equations below:

$$\frac{dI_H}{dt} = k\alpha_1 S_H - (\mu_1 + \alpha_2 + \omega) I_H \quad (20)$$

$$\frac{dI_R}{dt} = \phi S_R - (\mu_2 + \rho) I_H \quad (21)$$

$$\frac{dQ_H}{dt} = \alpha_2 I_H - (\mu_1 + \alpha_3) Q_H \quad (22)$$

$$\frac{dR_H}{dt} = \alpha_3 Q_H - (\mu_1 + \gamma_1) R_H \quad (23)$$

$$\frac{dS_R}{dt} = \eta - (\mu_2 + \phi) S_H \quad (24)$$

$$\frac{dS_H}{dt} = \pi + \rho I_R + \gamma_1 R_H - k\alpha_1 S_H - \mu_1 S_H \quad (25)$$

To compute the basic reproductive number (R_0) of the model (1) – (7), we employ the next generation method as applied in Diekmann *et al.* [10]. From equations (1) – (7), using their approached we have that

$$\mathcal{F}_i = \begin{pmatrix} k\alpha_1 S_H \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{26}$$

and

$$\mathcal{V}_i = \begin{pmatrix} (\mu_1 + \alpha_2 + \omega)I_H \\ -\phi S_R + (\mu_2 + \rho)I_H \\ -\alpha_2 I_H + (\mu_1 + \alpha_3)Q_H \\ -\alpha_3 Q_H + (\mu_1 + \gamma_1)R_H \\ -\eta + (\mu_2 + \phi)S_H \\ -\pi - \rho I_R - \gamma_1 R_H + k\alpha_1 S_H + \mu_1 S_H \end{pmatrix} \tag{27}$$

where, \mathcal{F}_i and \mathcal{V}_i are the rate of appearances of new infections in compartment i and the transfer of individuals into and out of compartment i by all other means respectively. Using the linearization method, the associated matrices at disease-free equilibrium (E_0) and after taking partial derivatives as defined by

$$\mathcal{DF}_i(E_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix} \text{ and } \mathcal{DV}_i(E_0) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix}$$

where F is nonnegative and V is a non-singular matrix, in which both are the $m \times m$ matrices defined by

$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(E_f) \right]$ and $V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(E_f) \right]$, with $1 \leq i, j \leq m$ and m is the number of infected classes. In particular $m = 3$, we have

$$FV^{-1} = \begin{pmatrix} \frac{\beta c \alpha_1 \pi}{\mu_1 N(\mu_1 + \alpha_2 + \omega)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{28}$$

$$|FV^{-1} - \lambda I| = \begin{vmatrix} \frac{\beta c \alpha_1 \pi}{\mu_1 N(\mu_1 + \alpha_2 + \omega)} - \lambda & 0 & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} = 0 \tag{29}$$

And characteristics polynomial of equation (29) is given as

$$\lambda^3 + \frac{\beta c \alpha_1 \pi}{\mu_1 N(\mu_1 + \alpha_2 + \omega)} \lambda^2 = 0 \tag{30}$$

and the eigenvalues is given by

$$\lambda_1 = 0, \lambda_2 = 0, \lambda_3 = \frac{\beta c \alpha_1 \pi}{\mu_1 N(\mu_1 + \alpha_2 + \omega)} \tag{31}$$

The most positive eigenvalues being the λ_2 is the Basic Reproduction Number (R_0)

Hence, we have

$$R_0 = \frac{\beta c \alpha_1 \pi}{\mu_1 N(\mu_1 + \alpha_2 + \omega)} \tag{32}$$

VII. STABILITY ANALYSIS OF DISEASE FREE EQUILIBRIUM STATE(E_0)

To study the behaviour of the system (1) – (7) around the disease-free equilibrium state

$E_f = \left(\frac{\pi}{\mu_1}, 0, 0, 0, \frac{\pi}{\mu_2 + \phi}, 0 \right)$, we resort to the linearized stability approach.

Let

$$f_1 = \pi + \rho I_R + \gamma_1 R_H - k\alpha_1 S_H - \mu_1 S_H \tag{33}$$

$$f_2 = k\alpha_1 S_H - (\mu_1 + \alpha_2 + \omega)I_H \tag{34}$$

$$f_3 = \alpha_2 I_H - (\mu_1 + \alpha_3)Q_H \tag{35}$$

$$f_4 = \alpha_3 Q_H - (\mu_1 + \gamma_1)R_H \tag{36}$$

$$f_5 = \eta - (\mu_2 + \phi)S_H \tag{37}$$

$$f_6 = \phi S_R - (\mu_2 + \rho)I_H \tag{38}$$

The Jacobian (J_{E_f}) is given by

$$(J_{E_f}) = \begin{pmatrix} -\mu_1 & \rho - \frac{\beta c \alpha_1 \pi}{\mu_1 N} & 0 & 0 & 0 & \gamma_1 \\ 0 & -(\mu_1 + \alpha_2 + \omega) + \frac{\beta c \alpha_1 \pi}{\mu_1 N} & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 & -(\mu_1 + \alpha_3) & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\mu_2 + \phi) & 0 & 0 \\ 0 & 0 & 0 & \phi & -(\mu_2 + \rho) & 0 \\ 0 & 0 & \alpha_3 & 0 & 0 & -(\mu_1 + \gamma_1) \end{pmatrix} \tag{39}$$

Rewriting the matrix (39), we get

$$(J_{E_f}) = \begin{pmatrix} -\mu_1 & \rho - \frac{\beta c \alpha_1 \pi}{\mu_1 N} & 0 & 0 & 0 & \gamma_1 \\ 0 & -A + \frac{\beta c \alpha_1 \pi}{\mu_1 N} & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 & -B & 0 & 0 & 0 \\ 0 & 0 & 0 & -D & 0 & 0 \\ 0 & 0 & 0 & \phi & -E & 0 \\ 0 & 0 & \alpha_3 & 0 & 0 & -F \end{pmatrix} \tag{40}$$

The determinant and the trace of matrix (J_{E_f}) represented by equation (40) above is given

$$Det(J_{E_f}) = \frac{(AN - \beta c \alpha_1 \pi)DF\mu_1 BE}{N} \tag{41}$$

$$Trace(J_{E_f}) = -\left(\mu_1 + A - \frac{\beta c \alpha_1 \pi}{\mu_1 N} + B + D + E + F \right) \tag{42}$$

where,

$$\begin{cases} A = (\mu_1 + \alpha_2 + \omega), B = (\mu_1 + \alpha_3) \\ D = (\mu_2 + \phi), E = (\mu_2 + \rho), F = (\mu_1 + \gamma_1) \end{cases} \tag{43}$$

Theorem 1

The disease free equilibrium state $E_f = (\frac{\pi}{\mu_1}, 0, 0, 0, -\frac{\pi}{\mu_2+\phi}, 0)$ of the model (33) – (38) is locally asymptotically stable if $R_0 < 1$ otherwise E_f is unstable.

Proof:

The Jacobian matrix of the system (33) – (38) is given by equation (40) above. If the Jacobian matrix is evaluated at the disease-free equilibrium state, then the required criteria for stable equilibrium are that the Determinant of the Jacobian (J_{E_f}) is positive and the Trace of the Jacobian (J_{E_f}) is negative.

From the determinant of the Jacobian matrix above given in equation (41) we have that

$$Det(J_{E_f}) = \frac{(AN - \beta c \pi \alpha_1) DF \mu_1 BE}{N} \tag{44}$$

$$Det(J_{E_f}) = \frac{\{((\mu_1 + \alpha_2 + \omega)N - \beta c \pi \alpha_1)(\mu_2 + \phi)(\mu_1 + \gamma_1)(\mu_2 + \rho)\mu_1\}}{N}$$

or

$$Det(J_{E_f}) = \frac{((\mu_1 + \alpha_2 + \omega)N \mu_1 - \beta c \pi \alpha_1 \mu_1)\{(\mu_2 + \phi)(\mu_1 + \gamma_1)(\mu_2 + \rho)\mu_1\}}{N} \tag{45}$$

Expressing the determinant in terms of R_0 , notice that

$$\mu_1 N (\mu_1 + \alpha_2 + \omega) = \frac{\beta c \alpha_1 \pi}{R_0} \tag{46}$$

using equation (46) into (45) we get

$$Det(J_{E_f}) = \frac{(\frac{\beta c \alpha_1 \pi}{R_0} - \beta c \pi \alpha_1 \mu_1)\{(\mu_2 + \phi)(\mu_1 + \gamma_1)(\mu_2 + \rho)\mu_1\}}{N} > 0, \text{ if } R_0 < 1 \tag{47}$$

Similarly, the Trace of the Jacobian Matrix (J_{E_f}) is given by

$$Trace(J_{E_f}) = - \left(\begin{array}{c} \mu_1 + (\mu_1 + \alpha_2 + \omega) \\ -\frac{\beta c \alpha_1 \pi}{\mu_1 N} + (\mu_1 + \alpha_3) + (\mu_2 + \phi) \\ +(\mu_2 + \rho) + (\mu_1 + \gamma_1) \end{array} \right) \tag{48}$$

Expressing the trace in terms of R_0 and substituting into (48) we get

$$Trace(J_{E_f}) = - \left(\begin{array}{c} \mu_1 + (\mu_1 + \alpha_2 + \omega) \\ -\mu_1 N (\mu_1 + \alpha_2 + \omega) R_0 + (\mu_1 + \alpha_3) \\ +(\mu_2 + \phi) + (\mu_2 + \rho) + (\mu_1 + \gamma_1) \end{array} \right) < 0, \text{ if } R_0 < 1 \tag{49}$$

Since $det(J_{E_0}) > 0$ and $Trace(J_{E_0}) < 0$ under the prescribed threshold conditions, then the disease free equilibrium (E_0) is locally asymptotically stable.

VIII. RESULTS

Numerical Experiments of the Model

The age-structured deterministic model (1)–(6) was solved numerically using Runge-Kutta-Fehlberg 4-5th order method and implemented using Maple 17 Software (Maplesoft, Waterloo Maple Inc, 2012). The model equations were first transformed into proportions, thus reducing the model equations to six differential equations. The parameters used in the implementation of the model are shown in Table 2 below. Parameters were chosen in consonance with the threshold values obtained in the stability analysis of the disease free equilibrium state of the model. We used the estimated values of the parameters used by [5, 7, 8] in the numerical experiments.

List of Numerical Experiments

- (1) The effect of treatment when on the infected population when the quarantine rate is constant
- (2) The effect of quarantine rate on the infected population when contact rate is constant.
- (3) The effect of quarantine rate on the infected population with treatment rate when contact rate is constant
- (4) The effect of quarantine rate on the recovered population contact rate is constant

Graphical Presentation of Results

Experiment 1: The effect of treatment when on the infected population when the contact rate is constant

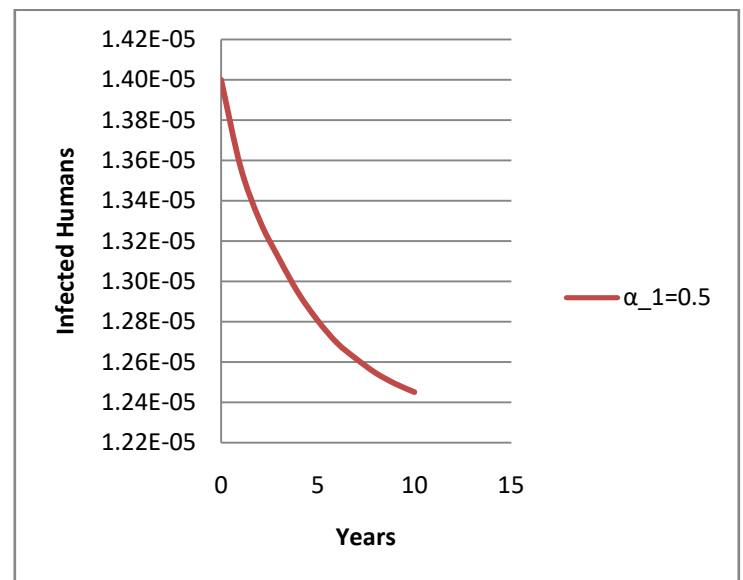


Figure 2 Graph showing the effect treatment when on the infected population at low and high ($\alpha_1 = 0.03$, $\alpha_1 = 0.0$, $c = 0.00018$) when the contact rate is constant

Experiment 2: The effect of quarantine rate on the infected population when contact rate is constant

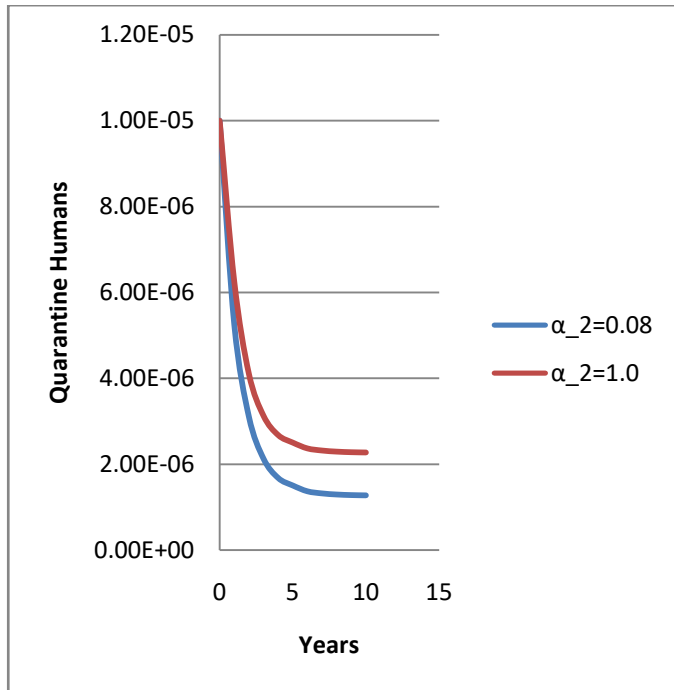


Figure 3 Graph showing the effect quarantine on the infected population when the quarantine rate is constant ($\alpha_2 = 0.1.0$, $\alpha_2 = 0.008$, $c = 0.00018$)

Experiment 3: The effect of quarantine rate and treatment rate on the infected population when contact rate is constant

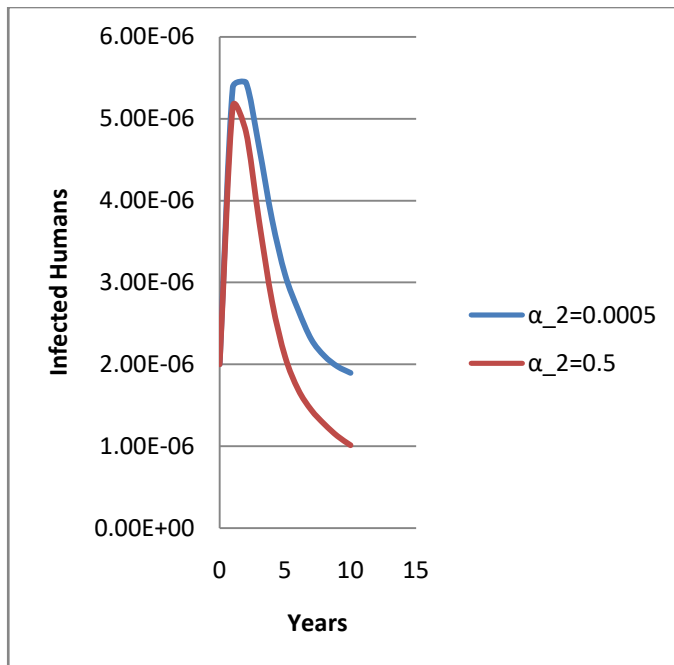


Figure 4 Graph showing the effect quarantine and treatment rate when the quarantine rate is constant ($\alpha_2 = 0.0005$, $\alpha_2 = 0.5$, $c = 0.00018$)

Experiment 4: The effect of quarantine rate on the recovered population contact rate is constant

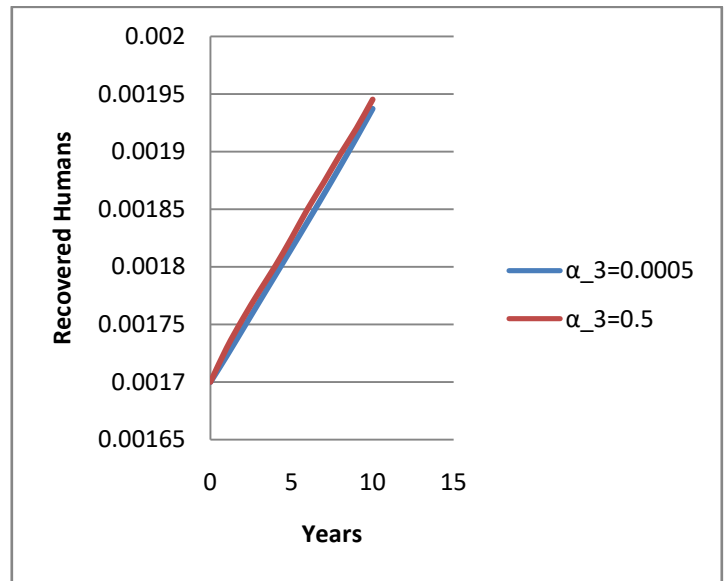


Figure 5: Graph showing the effect quarantine rate on recovered population when the quarantine rate is constant ($\alpha_3 = 0.0005$, $\alpha_3 = 0.5$, $c = 0.00018$)

IX. DISCUSSION OF RESULTS

Figure 2 shows that, infected human population decreased initially due to quarantine rate and it shows more impact when the quarantine rate is increased despite the increased in the contact rate, but the infected population increase with respect to time when less people are treated and quarantined and more people get infected. Figure 3 shows that, treated human population was zero initially and increased to a certain point with respect to time due to the awareness programs and treated rate and then drop as treated human recovered which lead to the increment in susceptible population of human. Figure 4 shows that the infected human population increase initially and drop drastically with respect to time due to high quarantine rate and death rate due to the disease. Figure 5 shows the effect of treatment rate on the quarantine population, it is observed from the graph that increment in treatment rate lead to the increment in the recovered population.

Our results agree with [8, 9] results which asserted that as time decreases, infected human increases which decrease the rate at which people are susceptible to the disease. But treated human increases slightly and decrease as time increases which lead to the increase in the susceptible population of human. The susceptible rodent decreases as the infected rodent increases while removed human increase slightly because many people are still ignorant of the treatment. This demand for more awareness programs even in the rural areas in order to reduce disease induced death rate. In contrast, the introduction of the new compartment called the quarantine class plays an important role in the controlling the disease, this is as a result of the mode of transmission of the disease.

X. CONCLUSION

In this paper, we developed a new mathematical model which incorporated some important factors that plays significant role in the control of Lassa fever. These factors are: disease induced death rate and the quarantine parameter. The introduced quarantine parameter helps in controlling and eradication of Lassa fever virus with respect to time. We obtained the basic reproduction numbers, R_0 . Our analysis reveals that the disease can be control if the basic reproduction number, R_0 is less than one regardless of the initial population profile. Thus, every effort must be put in place by all concerned to prevent the virus infection by reducing R_0 strictly less than unity. Finally, there is need for further research work on the effects of various control strategy such as quarantine, vaccination, personal hygiene dynamics of Lassa fever disease as well as stability analysis for endemic equilibrium.

REFERENCES

- [1] Fisher –Hosh, S. P., Tomori, O., Nasidi A, Perez-Oronoz, G. I., Fakile Y., Hutwagner, L., & McCormick, J. B. (1995). Review of Cases of nosocomial Lassa fever Nigeria: the High Price of Poor Medical Practice. Pp 857-9
- [2] Okuonghae, D. & Okuonghae, A I. (2006). A Mathematical Model for Lassa fever. *Journal of National Association of mathematical Physics*, 10:457-464.
- [3] Ogabi, C.O., Olusa, T.V. & Madufor, M. A. (2012). Controlling Lassa fever Transmission in Northern Part of Edo State, Nigeria Using SIR Model. *New York Science Journal*, 5(12):3-8
- [4] Bawa, G. S., Abia, E. E., Omage, J. J., Hassan, M. R., Abdul, S. B. (2013). *Nutritive value of Dolichos lablab (Lablab purpureus CV. Rongai forage Cut at Different Stages of Growth on Performance of Weaned rabbits. Nigeria J. Anim. Sci., 15: Pp 23-36*
- [5] Omale, D. & Edibo T. E., (2016). Mathematical Models for Lassa fever Transmission with Control Strategies. *Computing, Information Systems, Development Informatics & Allied Research Journal Vol. 6 No. 4.*
- [6] McCormick, J. B., Webb, P.A., Krebs. J. W., Johnson. K. M, Smith, E. S. (1987). A Prospective Study of the Epidemiology and Ecology of Lassa fever. *J Infect Dis., 155:437- 444.*
- [7] Centers for Disease Control and Prevention, (CDC) (2015). "Lassa Fever Surveillance-United States, 1981–2008". *MMWR. Morbidity and mortality weekly report* 60 (21): 689–93. PMID 21637182.
- [8] Olaniyi, S. & Obabiyi, O. S. (2013). Mathematical Model for Malaria Transmission Tynamics in Human and Mosquito Populations with Nonlinear Forces of Infection. *International journal of pure and applied Mathematics*, 88(1):125-156
- [9] Onuorah, M. O., Ojo, M. S., Usman, D. J., & Ademu, A. (2016). Basic Reproductive Number for the Spread and Control of Lassa fever. *International Journal of Mathematics Trends and Technology (IJMTT)*
- [10] Diekmann, O., Heesterbeek, J.A.P. & Metz. J.A.J, (1990). On the Definition and the Computation of the Basic Reproduction Number, in *Models of Infectious Diseases in Heterogeneous Populations. J. Math. Biol., Volume 28, PP: 365-382.*