MATHEMATICAL ANALYSIS OF LASSA FEVER MODEL WITH ISOLATION

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ABSTRACT

In this paper, a mathematical model with isolation of infected individuals for the transmission of Lassa fever is developed and analyzed. We obtained the basic reproduction number R_o *which is the average number of new secondary infection generated by a single infected individual/rat during infectious period. The analysis shows that the disease free equilibrium is locally and globally asymptotically stable whenever the threshold quantity* R_o *is less than unity i.e.* $R_o < 1$. *The endemic equilibrium of the model exists under certain condition. The numerical analysis carried out using MAPLE 17 software. The result shows that the isolation of infected individuals reduces the dynamical spread of Lassa as there will be less interaction with the infected individual in the society, the result also shows that treatment of infected-isolated individuals gives a better result which means that government should intensify effort in isolation and the treatment of isolated-infected individuals in order to control the spread of the disease.*

Keywords: Lassa, Reproduction Number, Equilibrium points and Stability

INTRODUCTION

Lassa fever is a viral disease that is majorly caused by Lassa virus. It was first discovered in 1961 in the Lassa town of Borno State, Nigeria [3]. Endemic situation of Lassa virus was reported in some cities of West Africa of countries of Sierra Leone, Liberia, Guinea and Nigeria [11, 12]. In Cote d'Ivoire, Ghana, Togo and Benin the outbreak of Lassa fever has never been reported, though the isolation strategy revealed the evidence of viral circulation [7].

The carrier of Lassa Virus is a small rodent (rat), the Multimammate rat of the genus Mastomys. The transmission occurs when an individual come in contact directly with the blood, urine, feaces of rats and other body secretions of an infected person [1, 7, 14]. Since the rodents lives in an environment very close to human, it aids transmission from the rodent to human of the virus through direct contact. Furthermore, contact with the virus may also occur when an individual absorbs particles in the air containing Lassa virus from an infected person [2]. The symptoms of Lassa fever begin to show in an individual after being infected between one and three weeks. Those symptoms include facial, muscle fatigue, vomiting, cough, meningitis and hypertension. The presence of Lassa virus may result into neurological problems including loss of hearing which may be transient or permanent, tremors and

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encephalitis [13, 15]. The disease is mild or has no observable symptoms in up to 80% of people infected, but 20% develop a severe multisystem diseases. Even after recovery, the virus remains in body fluids for long periods of time [7, 8].

For SIR model (Susceptible, Infected, Recovered) and SEIR model (Susceptible, Exposed, Infected, Recovered), individuals that are treated or given vaccine and recovered cannot return to susceptible because of the permanent immunity they have in their system. Many researchers have worked on mathematical modeling of Lassa fever outbreak in the presence some factors that may enhance its outbreak. Bawa et. al [1] worked on Stability analysis of the disease free equilibrium state for Lassa fever disease, they developed a deterministic model for Lassa fever in a population with vital dynamics. Their analysis revealed that the disease can be control if the basic reproduction number R0 is less than one regardless of the initial population profile. James et. al [9] developed a mathematical model of Lassa fever disease dynamics using a set of ordinary differential equations. They discovered that the zero equilibrium state is stable when the birth rate of the human population is less than the death rate and same when the birth rate of the mastomysnatalensis (reservoir) is less than the total death rates.

In this paper, we presented a new six compartmental model with isolation of infected individual for the dynamical spread of Lassa to check the treatments of infected-isolated individuals and infected without isolation individuals.

MODEL FORMULATION

The population size $N_h(t)$ of human is sub–divided into sub–classes of individuals who are Susceptible $S_h(t)$, Exposed E_h(t), Infected $I_h(t)$, and Isolation $J_h(t)$, So that;

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + J_h(t) \tag{1}$$

Also, the population size $N_R(t)$ of the rodents is sub–divided into susceptible rodents $S_R(t)$ and Infectious rodents $I_R(t)$. So that;

$$N_R(t) = S_R(t) + I_R(t) \tag{2}$$

The susceptible population is increased by the recruitment of individuals into the population (either by birth or immigration at the rate π_h). The population decreases by the newly borne infected individuals that move to infected class. The population also decreases by infection following a contact with infectious rodents and human (at the rate β_1 *and* β_2) and natural death (at the rate μ_h). Thus;

$$S_h' = (1-\rho)\pi_h - (\beta_1 I_R + \beta_2 I_h)S_h - \mu_h S_h \tag{3}$$

The population of the exposed class consists of newly infected individuals following a contact with the infected rodents and human (at the rate β_1 *and* β_2). The class increases due to the treatment of the infected and isolated individuals (at the rate U_1 *and* U_2). The exposed population declines due to progression to infectious class (at the rate κ_h) and natural death (at the rate μ_h).Thus;

$$E_h' = (\beta_1 I_R + \beta_2 I_h)S_h - (\kappa_h + \mu_h)E_h + U_1 I_h + U_2 J_h \quad (4)$$

The population of infected individual increases by newly borne infected individual (at the rate ρ) and progression from exposed class (at the rate κ_h). The population declines due to treatment (at the rate U_1), those that are isolated (at the rate σ), natural death (at the rate μ_h) and disease induced death (at the rate δ_h). Thus;

$$I_h' = \rho\pi_h + \kappa_h E_h - (\mu_h + \sigma + \delta_h + U_1)I_h \quad (5)$$

The population of the isolated individual increases by those that are infected but isolated (at rate σ). The population decreases due to natural death (at the rate μ_h), death due to the disease (at the rate δ_h) and the treatment (at the rate U_2). Then,

$$J_h' = \sigma I_h - (\mu_h + \delta_h + U_2)J_h \quad (6)$$

Susceptible rodents (S_R) are generated at a constants rate π_R (recruitment rate) and acquire infection following effective contact with infected rodent (at a rate β_1). The rodents suffer death (at the rate μ_R) Hence,

$$S_R' = \pi_R - \beta_1 I_R S_R - \mu_R S_R \quad (7)$$

The infected class of the rodents has newly infected rodents and reduces by the death (at the rate μ_R). Hence;

$$I_R' = \beta_1 I_R S_R - \mu_R I_R \quad (8)$$

In summary, combining the above formulations and assumptions together, we have the following system of differential equations. The definitions of variables and parameters used are given in tables 1 and 2.

$$\left.\begin{aligned} S_h' &= (1-\rho)\pi_h - (\beta_1 I_R + \beta_2 I_h)S_h - \mu_h S_h \\ E_h' &= (\beta_1 I_R + \beta_2 I_h)S_h - (\kappa_h + \mu_h)E_h + U_1 I_h + U_2 J_h \\ I_h' &= \rho\pi_h + \kappa_h E_h - (\mu_h + \sigma + \delta_h + U_1)I_h \\ J_h' &= \sigma I_h - (\mu_h + \delta_h + U_2)J_h \\ S_R' &= \pi_R - \beta_1 I_R S_R - \mu_R S_R \\ I_R' &= \beta_1 I_R S_R - \mu_R I_R \end{aligned}\right\} \quad (9)$$

ANALYSIS OF THE MODEL

Lemma 1: The closed set $D = D_h \times D_R \subset R_+^6$ is positive invariant for the model equation (9) with non-negative initial condition in R_+^6

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Proof: Consider the biologically-feasible $D = D_h \times D_R \subset R_+^6$ with

$$D_h = \{(S_h, E_h, I_h, J_h) \in R_+^4 :: N_h \le \frac{(1-\rho)\pi_h}{\mu_h}\} \text{and } D_R = \{(S_R, I_R) \in R_+^2 :: N_R \le \frac{\pi_R}{\mu_R}\}$$

We shall show that D is positive invariance (i.e all solution in D remain in D for all time t>0). The rate of change of the total population of human and rodents by adding gives;

$\frac{dN}{dt} = \pi_h - \mu N_h - \delta I_h$ and $\frac{dN}{dt} = \pi_R - \mu N_R$

Where $N_h = S_h + E_h + I_h + J_h$ and $N_R = S_R + I_R$

A standard comparison theorem [10] can be used to show that

$$N_h(t) \le N_h(0)e^{-\mu t} + \frac{(1-\rho)\pi_h}{\mu_h}(1-e^{-\mu t}) \text{ and } N_R(t) \le N_R(0)e^{-\mu t} + \frac{\pi_R}{\mu_R}(1-e^{-\mu_R t}).$$

In particular $N_h(t) \le \frac{(1-\rho)\pi_h}{\mu_h}$ and $N_R(t) \le \frac{\pi_R}{\mu_R}$, if $N_h(0) \le \frac{(1-\rho)\pi_h}{\mu_h}$ and $N_R(0) \le \frac{\pi_R}{\mu_R}$ respectively. Therefore, all solution of the model with initial condition in D remains there for t>0. This implies that D is positively-invariant, in this region the model can be considered as been epidemiologically and mathematically well posed.

Disease Free Equilibrium point

At steady state, $S_h' = E_h' = I_h' = J_h' = S_h' = I_R' = 0,$

Let E_o denotes the disease free equilibrium state of the model equation (9).

Then, at disease free $E_h = I_h = J_h = I_R = 0,$

$$\therefore E_o = (S_h, E_h, I_h, J_h, S_R, I_R,) = \left(\frac{(1-\rho)\pi_h}{\mu_h}, 0, 0, 0, \frac{\pi_R}{\mu_R}, 0\right) \quad (10)$$

Basic Reproduction Number

The basic reproduction number is the number of secondary cases of infection emanating from a single infection source [5]. Next Generation matrix method [6] is used to obtain the basic reproduction number. The matrices F (new infection terms) and V (other remaining transfer terms) are given as;

$$F = \begin{bmatrix} 0 & \frac{\beta_2(1-\rho)\pi_h}{\mu_h} & 0 & \frac{\beta_1(1-\rho)\pi_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_1\pi_R}{\mu_R} \end{bmatrix}$$

And

$$V = \begin{bmatrix} K_1 & -U_1 & -U_2 & 0 \\ -\kappa_h & K_2 & 0 & 0 \\ 0 & -\sigma_h & K_3 & 0 \\ 0 & 0 & 0 & K_4 \end{bmatrix}$$

Where; $K_1 = \kappa_h + \mu_h,\ K_2 = \sigma + \delta_h + U_1,\ K_3 = \mu_h + \delta_h + U_2,\ K_4 = \mu_R$

Then, the basic reproduction number denoted by R_o is given by $R_o = \rho\left(FV^{-1}\right)$

$$\therefore R_o = \max\left[\frac{\beta_1\pi_R}{\mu_R{}^2}, \frac{\beta_2(-1+\rho)\pi_h K_3\kappa_h}{\mu_h(\sigma\kappa_h U_2 - K_3K_2K_1 - K_3\kappa_h U_1)}\right]$$

The threshold quantity R_o is the basic reproduction number of the model equation above, which is the average number of new case of an infection caused by one typical infected rat/ human in a population consisting of susceptible only.

Local Stability of Disease Free Equilibrium Point

Theorem 1: The disease free equilibrium of the modeled equation (9) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: To determine the local stability of E_0, the following Jacobian matrix is computed corresponding to equilibrium point E_0. Considering the stability of the disease free equilibrium at the critical point ($\frac{(1-\rho)\pi_h}{\mu_h}$, 0, 0, 0, $\frac{\pi_R}{\mu_R}$, 0). We have

$$J_L = \begin{pmatrix} -\mu_h - \lambda & 0 & \frac{-\beta_2(1-\rho)\pi_h}{\mu_h} & 0 & 0 & \frac{-\beta_1(1-\rho)\pi_h}{\mu_h} \\ 0 & -c_1 - \lambda & k_2 & U_2 & 0 & 0 \\ 0 & \kappa_h & -c_3 - \lambda & 0 & 0 & 0 \\ 0 & 0 & \sigma_h & -k \\ & & & c_4 - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_R - \lambda & \frac{-\beta_1\pi_R}{\mu_R} \\ 0 & 0 & 0 & 0 & 0 & -c_5 - \lambda \end{pmatrix}$$

$$c_1 = \kappa_h + \mu_h,\ c_2 = \frac{\beta_2(1-\rho)\pi_h}{\mu_h},\ c_3 = \sigma + \mu_h + \delta + U_1, c_4 = \mu_h + \delta + U_2 \text{ and } c_5 = \frac{\beta_1\pi_R}{\mu_R} - \mu_R$$

The eigen values are $\lambda = -\mu_h, -\mu_R, -c_5$ and the remaining matrix is given by;

$$\begin{vmatrix} -c_1 - \lambda & k_2 & U_2 \\ \kappa_h & -c_3 - \lambda & 0 \\ 0 & \sigma_h & -c_4 - \lambda \end{vmatrix} = 0 \qquad (11)$$

The characteristics polynomial of (11) is given by

$$A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 = 0 \qquad (12)$$

Where

$$A_3 = 1$$
$$A_2 = c_4 + c_3 + c_1$$
$$A_1 = c_1c_3 + c_1c_4 - c_2\kappa_h + c_3c_4 \qquad (13)$$
$$A_0 = [1 - R_0](\sigma_h\kappa_h U_2 - c_1c_3c_4 + c_4U_1\kappa_h)\mu_h$$

Then the Routh Hurwitz criterion will be employed to determine the nature of other roots, which states that all the roots of the polynomial will have negative real parts if and only if all the coefficients A_i (i=0, 1, 2, 3) are all positive and that the matrices T_i (i=1, 2, 3) are all

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positive. Clearly, from (13) above $A_3 > 0, A_2 > 0, A_1 > 0 \, and \, A_0 > 0$ if $R_0 < 1$. Also, the Hurwitz matrix T_i are all positive which are given below;

$$T_1 = A_2 > 0, T_2 = \begin{vmatrix} A_2 & A_3 \\ A_0 & A_1 \end{vmatrix} > 0, T_3 = \begin{vmatrix} A_2 & A_3 & 0 \\ A_0 & A_1 & A_2 \\ 0 & 0 & A_0 \end{vmatrix} > 0$$

Therefore, all the eigen-values of the polynomial (12) are negative which shows that the disease free equilibrium is locally asymptotically stable.

Global stability of the Disease Free Equilibrium

Theorem 2: The disease free equilibrium of model given by (9) is globally asymptotically stable if $R_o<1$.

Proof:

We will use comparison theorem [10] to prove the global stability. The rate of change of variables representing the infected components of equation (9) can be re-written as;

$$\begin{pmatrix} E_h' \\ I_h' \\ J_h' \\ I_R' \end{pmatrix} = (F - V) \begin{pmatrix} E_h \\ I_h \\ J_h \\ I_R \end{pmatrix} - F_i \begin{pmatrix} E_h \\ I_h \\ J_h \\ I_R \end{pmatrix}$$

Where;

$$\begin{pmatrix} E_h' \\ I_h' \\ J_h' \\ I_R' \end{pmatrix} = (F - V) \begin{pmatrix} -\kappa_h - \mu_h - \beta_2 S_h + U_1 + U_2 + \beta_1 S_h \\ \kappa_h - \sigma_h - \mu_h - \delta - U_1 \\ \sigma_h - \mu_h - \delta - U_2 \\ \beta_1 S_h - \mu_R \end{pmatrix} - F_i \begin{pmatrix} E_h \\ I_h \\ J_h \\ I_R \end{pmatrix} \quad (14)$$

Then,

$$\begin{pmatrix} E_h' \\ I_h' \\ J_h' \\ I_R' \end{pmatrix} \leq (F - V) \begin{pmatrix} -\kappa_h - \mu_h - \beta_2 S_h + U_1 + U_2 + \beta_1 S_h \\ \kappa_h - \sigma_h - \mu_h - \delta - U_1 \\ \sigma_h - \mu_h - \delta - U_2 \\ \beta_1 S_h - \mu_R \end{pmatrix}$$

All the eigen values of the matrix F – V have negative real parts. It follows that the linearized differential inequality system above is stable whenever $R_o < 1$.Consequently, by comparison theorem [10] we have that $E_h = I_h = J_h = I_R = 0, \rightarrow (0,0,0,0,0)$ as $t \rightarrow \infty$. Substituting $E_h = I_h = J_h = I_R = 0$ into (1) we have that $S_h(t) \rightarrow S_h(0), and \, S_R(t) \rightarrow S_R(0)$ as $t \rightarrow \infty$. Hence, we have a positive invariant region. it follows that disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

Existence of the Endemic Equilibrium

Theorem 3: The model (9) has a unique endemic equilibrium when the basic reproduction number exceeds unity (i.e. $R_o > 1$)

Proof: Let the endemic equilibrium ε_1^{**} of the model be given be $\varepsilon_1^{**} = (S_h^{**}, E_h^{**}, I_h^{**}, J_h^{**}, S_R^{**}, I_R^{**})$. Then, by setting the model equations to zero, the results are as follow;

$$S_h^{**} = \frac{(1-\rho)\pi_h \mu_R}{I_h^{**}\mu_R\beta_2 + \pi_R\beta_1 - \mu_R^2 + \mu_R\mu_h}$$

$$E_h^{**} = \frac{\pi_R(1-\rho)(U_2+\mu_h+\delta_h)\left[\beta_2 I_h^{**}\mu_R + (\beta_1\pi_R - \mu_R^2)\right]}{(I_h^{**}\mu_R\beta_2 + \pi_R\beta_1 - \mu_R^2 + \mu_R\mu_h)(\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)} + \frac{I_h^{**}(\sigma U_2 + U_1U_2 + U_1\mu_h + U_1\delta_h)}{(\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)}$$

$$K_h^{**} = \frac{\sigma I_h^{**}}{\mu_h+\delta_h+U_2}$$

$$S_R^{**} = \frac{\pi_R}{\beta_1 I_R^{**} + \mu_R}$$

$$I_R^{**} = \frac{\beta_1\pi_R - \mu_R^2}{\mu_R\beta_1}$$

and I_h^{**} is given by the solution of the quadratic equation;

$$AI_h^{**2} + BI_h^{**} + C = 0$$

Where;

$$A = \mu_R\beta_2\left[\kappa_h(\sigma U_2 + U_1U_2 + U_1\mu_h + U_1\delta_h) - (\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)(\mu_h+\sigma+\delta_h+U_1)\right]$$

$$\begin{aligned} B = {} & \kappa_h(\sigma U_2 + U_1U_2 + U_1\mu_R + U_1\delta_R)(\pi_R\beta_1 - \mu_R^2) - \\ & (\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)\left[(\mu_h+\sigma+\delta_h+U_1)(\pi_R\beta_1 - \mu_R^2) + \mu_R\beta_2\rho\pi_h\right] \\ & + (R_0-1)(\mu_R\mu_h)\left[(\mu_h+\sigma+\delta_h+U_1)(\mu_h+\delta_h+U_2)(\kappa_h+\mu_h) - \kappa_h(\sigma U_2 + U_1(U_2+\mu_h+\delta_h))\right] \end{aligned}$$

$$C = \pi_h\kappa_h(1-\rho)(\pi_R\beta_1 - \mu_R^2)(U_2+\mu_R+\delta_h) + \rho\pi_h(\pi_R\beta_1 - \mu_R^2 + \mu_R\mu_h)(\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)$$

The coefficient of I_h^{**2} is positive (i.e. A >0) if;

$$\kappa_h(\sigma U_2 + U_1U_2 + U_1\mu_h + U_1\delta_h) > (\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)(\mu_h+\sigma+\delta_h+U_1)$$

And the constant C is positive. Moreover, B is negative whenever $R_o > 1$, if also;

$\kappa_h(\sigma U_2 + U_1U_2 + U_1\mu_h + U_1\delta_h) > (\mu_h+\delta_h+U_2)(\kappa_h+\mu_h)(\mu_h+\sigma+\delta_h+U_1)$, Then, there exists positive roots. Therefore, there exists an endemic equilibrium whenever $R_o > 1$

NUMERICAL SIMULATION

Numerical simulation of the model was carried out by the help of MAPLE 17 software using differential transformation method. The parameters values are as given in the table 2.

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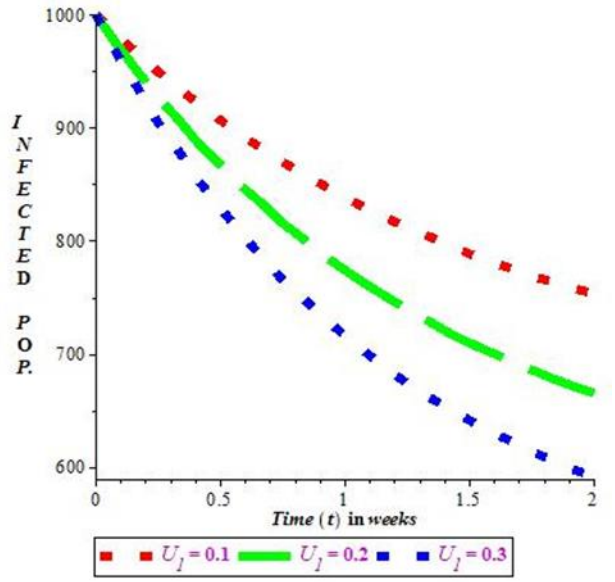


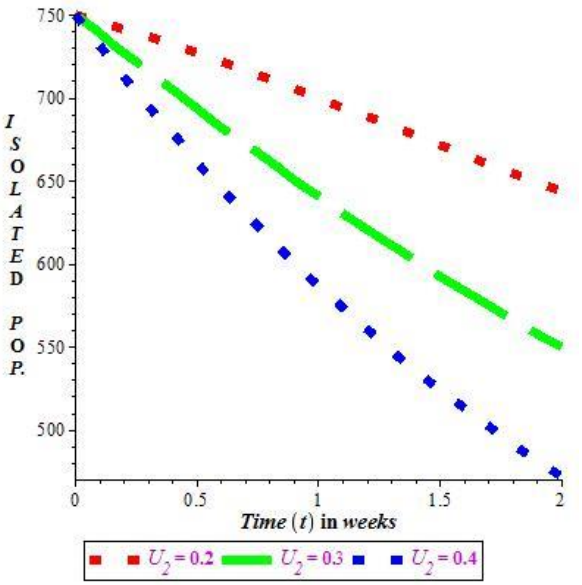
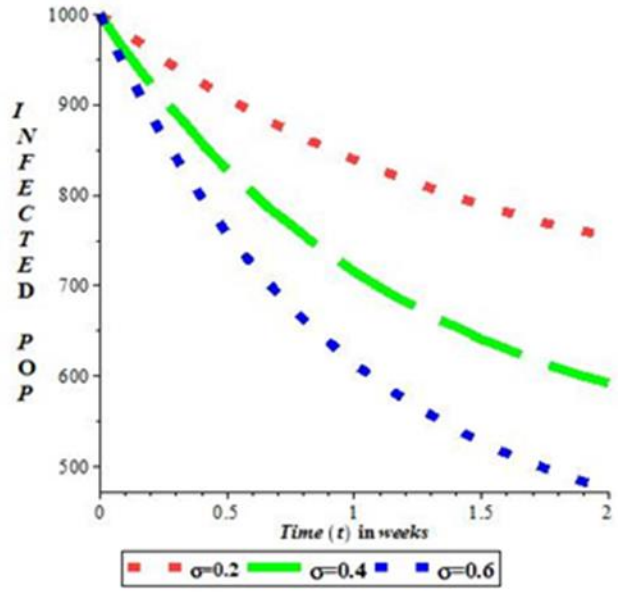


Fig.1



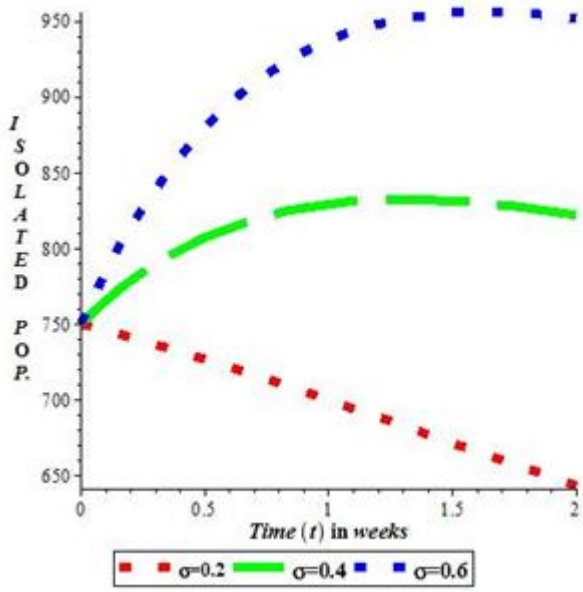


Fig.2

DISCUSSION OF RESULTS AND CONCLUSION

A six (6) new compartmental model was formulated to gain insight into the effect of isolation of infected individuals and treatment of infected individuals in the dynamical spread of Lassa fever due to effective contact of human and infected rats. The positivity of solution shows that the model is mathematically and epidemiologically well posed. Basic reproduction number ‘ R0 ’ which determines whether Lassa disease dies out or spread was calculated using next generation matrix method, the result shows that, disease dies out whenever the threshold R0 < 1 but spreads when it exceeds unity i.e. R0 >1 . The global stability of disease free equilibrium was analyzed using comparism method [10].

Numerical simulation of the model was carried out by MAPLE (17) software . Figures 1 and 2 of the numerical simulation showed that, isolation of infected individuals would reduce the dynamical spread of Lassa fever in the society. Moreover, this would help the public not to have interaction with infected individuals and would enable infected-isolated individuals to receive proper and adequate treatment.

Figures 3 and 4 showed the pronounced effect of treatment on infected and infected-isolated individuals. Results showed that treatment of infected-isolated individuals yield a better result compared to infected individuals. When the treatment rate of infected individuals is 0.3, we have about 580 infected whereas the infected-isolated individuals have reduced to 550.

In conclusion, isolation of infected individuals should be targeted as one of the control measures by government and policy health makers in the dynamical control of Lassa fever in the society.

Table 1. Table of variables and Description

Variable	*Description*
$S_h(t)$	Susceptible Individuals
$E_h(t)$	Exposed Individuals
$I_h(t)$	Infected Individuals
$J_h(t)$	Isolated Individuals
$S_R(t)$	Susceptible Rodents (Rats)
$I_R(t)$	Infected Rodents

Table 2. Table of parameters and their values

Parameter	*Description*	*Values*	*References*
ρ	Rate of infection at birth	0.2	Estimated
π_h	Recruitment rate of human	2000	Estimated
β_1	Contact rate of rat	0.2	Estimated
β_2	Contact rate of human	0.2	Estimated
μ_h	Natural death rate of human	0.02	CIA(2015)
δ_h	Death due to disease of human	0.1	Estimated
κ_h	Progression rate	0.003	Estimated
σ	Isolation rate	0.2	Estimated
U_1	Infected treatment rate	0.1	Estimated
U_2	Isolated treatment rate	0.2	Estimated
π_R	Recruitment rate of rat	500	Estimated
μ_R	Natural death rate of rat	0.02	Estimated

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