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A MATHEMATICAL MODEL ON THE TRANSMISSION DYNAMICS OF LASSA FEVER WITH VARIABLE HUMAN AND RODENT POPULATION

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Abstract

This work presents a mathematical model on the transmission dynamics of Lassa fever in a two interacting human and rodent population. The model was subdivided into seven compartments, five for humans and two for rodents (rats). The boundedness and positivity of our results were established. We showed conditions for disease-free equilibrium and endemic equilibrium. The basic reproduction number was calculated using the next generation approach. We established the local and global stability of the model by using the Jacobian approach and comparison theorem respectively, and showed that they are both stable when the reproduction number of both human and rodent is less than one ($R_0 < 1$). Finally we carried out numerical simulation of the model using a set of reasonable parameter values to see the effectiveness of rodent control, early diagnostic approach, treatment and drug adherence on reducing drastically Lassa fever from the population. The study showed that early diagnosis and treatment, reduction of rodent population, complete and adequate drug adherence and maintaining hygienic environment are the best strategies against the spread of Lassa fever disease.

***Keywords*:** Lassa fever, Basic Reproduction number, Stability analysis, Equilibrium, Simulation.

1.0 Introduction

Lassa Fever is an acute arena viral haemorrhagic fever caused by Lassa virus, an arenavirus known to be responsible for severe haemorrhagic fever characterised by fever, muscle aches, sore throat, nausea, vomiting, chest and abdominal pain [1]. It was first found in a town called Lassa in the Yedseram River Valley in the present Borno State of Northern Nigeria in 1969 [2]. Lassa virus is a single-stranded RNA virus belonging to the virus family Arenaviridae. It is a zoonotic disease that is primarily transmitted to humans from contact with infected animals, usually a rodent of the genus Mastomys known widely as rat [3].

The Mastomys rat which is the carrier of Lassa fever do not show clinical symptoms of Lassa fever, but can remain carriers for life, but transmits the virus to humans and other primates through the shed of their urine, saliva, respiratory secretion and exposed blood vessel through micro and macro trauma [4]. There can also be cases of secondary infection through inhalation or ingestion. Person-to-person transmission of Lassa fever can also occur through contaminated medical equipment, such as reused needles. Victims can also be infected through skin breaks and through mucous membranes from aerosol transmission from dust-borne particles [5]. In some areas, especially in Africa rodents serve as food source, and thereby providing additional exposure to the infectious rat blood, as well as ingestion of potentially contaminated meat. Lassa fever is is knwon to be endemic in Benin, Ghana, Liberia, Mali, Burkina faso, Gambia, Sierra Lone, and Nigeria, it also exists in other African countries as well [6].

The symptoms of Lassa fever usually occur 2-21 days after the infection, it is characterised by gradual show of fever, general weakness, facial swelling, vomiting, cough and sometimes meningitis and hypertension. In some areas, it can include neurological problems, hearing loss which may be transient or permanent [7]. Death occurs within the first 14 days

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of onset in fatal cases [3]. It is reported that 1 out of each 5 infections results in severe disease cases, where the virus affects several viral organs such as the liver, spleen and kidneys. The disease however, when confirmed in a community, requires prompt isolation of affected patients, good infection prevention and control practices, also rigorous contact tracing can stop outbreaks [3].

2.0 Literature Review

It is also vital to look at various works on mathematical model of Lassa fever transmission dynamics, as regards to different areas of approach. Omale and Edibo [8] developed a model, but in their model they neglected the latency/exposed period in the successive trends of infection progression in both humans and the animals and considered reinfection after recovery from the treatment intervention in the humans. They considered linear incidence transmission in the animals and the force of infection in the model did not account for animal to human transmission. Bawa et al [5] developed a mathematical model for Lassa fever where they divided the human population into susceptible humans S_H and the infectious humans I_H and the reservoir population into infant I_R and the adult reservoir A_R and represented the virus in the environment by V. They explained that the virus compartment is generated from the urine and faeces of infected human and adult reservoirs. They recommend that effort should be made to keep the basic reproduction number below 1. Okuonghae D and Okuonghae R [9] discussed a mathematical model for the transmission of Lassa fever. Steady states of their model were examined for epidemic and endemic situation. The results of their model show that the interim control of the rodents carrying the virus and some isolation policy on the infected individuals is the best strategies against the spread of the disease. Onuorah et al [10] developed Lassa fever model using the sex structure approach. Their model represented the transmission dynamics of the Lassa fever disease using a set of ordinary differential equations. The total human population at time t denoted by $N_H(t)$ was sub-divided into four mutually exclusive sub-populations. Their model had the following assumptions: Susceptible individuals, male/female can be infected through interaction with the active reservoir (Mastomys Natelensis), and through sexual interaction with opposite sex. Two major controls were considered, the use of condom to reduce contact through sexual interaction and the use of pesticide/rat poison to kill the natural reservoir.

In this work we developed a mathematical model for the transmission dynamics of Lassa fever taking into consideration the latent period of humans after interaction with infected rodents. we have both the human compartment and the rodent compartment, we considered early diagnosis of the disease, drug adherence and control of the rodent, as to see their effects in controlling the spread of Lassa fever in this section, we developed a Mathematical model describing the transmission dynamics of Lassa fever. The definitions and representations of the model parameters and state variables are highlighted; flow diagram describing the dynamics of the disease was shown, basic assumptions also highlighted in this section.

3.0 Model Formulation

3.1 Description of the Model

The model presented here is of two compartments, the human and rodent compartment. The total human population denoted by N_h, is sub-divided into Susceptible human S_h, Exposed humans E_h, Infectious humans I_h, Treated T_h and Recovered humans R_h so that we now have that $N_h = S_h + E_h + I_h + T_h + R_h$. while total rodent population denoted by N_r is sub-divided into Susceptible rodents S_r and Infectious rodents I_r so that we now have that $N_r = S_r + I_r$.

The susceptible humans are generated by the recruitment of humans into the population by either birth or immigration at a rate Λ_h. The population of susceptible humans decreases as a result of interaction with either infectious humans or infectious rodents and thereby progressing to the exposed class at a rate λ_h. The exposed population decreases either by early diagnosis leading to treatment and recovery or progressing to infectious class at the rate of ρ. The infectious population reduces by treatment at the rate σ or by disease induced date rate δ or by recovery rate which is very small α. The treated humans reduce by a recovery rate γ, while the recovered humans reduce by loosing immunity at a rate Ψ. It is also worthy to note that either of the populations reduces by natural death rate μ.

Similarly, the susceptible rodents are generated by birth rate denoted by Λ_r. The population of the susceptible rodents reduces by progression to infectious class at the rate of λ_r as a result of interaction with the infectious human. The infectious rodent remains infectious in all its lifetime but can be reduced by natural death rate as well as the susceptible rodents. These variables and parameters are illustrated further in the tables below.

Table I: Description of parameters

Λ_h	Recruitment rate of humans into the population, either by birth or immigration.
λ_h	Rate of progression from susceptible humans to exposed humans due to interaction with infectious humans and infectious rodents.
ρ	Rate of progression from Exposed humans to Infectious humans.
e	Rate of early diagnosis of the disease.
μ	Natural death rate.
τ	Rate of progression from Exposed humans to Treated humans.
δ	Disease induced death rate.
σ	Rate of progression from Infectious human to Treated human.
α	Rate of progression from Infectious human to Recovered human.
γ	Rate of progression from Treated humans to Recovered humans.
d	Rate of compliance to drug intake.
Ψ	Rate at which Recovered humans loose immunity and progress to Susceptible class.
Λ_r	Recruitment rate of rodents into the population.
λ_r	Rate of progression from Susceptible rodents to infectious rodents due to interaction with the Infectious human.
c	Rate of rodent control in the environment.

3.2 Assumptions of the Model

In order to formulate the model equations, the following assumptions were made:

- Susceptible human population is infected by Lassa through interaction between the infectious rodent and infectious human.
- Susceptible rodent become infectious when they come in contact with the infectious humans.
- All infectious humans who get treated completely do not die due to the disease but recovers.
- When one is not completely treated, the symptoms may hide for sometime, but resurfaces after sometime.
- We neglected the interaction between infectious rodent and susceptible rodents.
- Recovered humans can loose their immunity and thereby becomes susceptible again.
- There is human to human spread of the disease.

3.3 Model Equations

From the model description above and the flow diagram, we derive the following model equations:

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \Psi_h - \lambda_h S_h - \mu S_h \\
\frac{dE_h}{dt} &= \lambda_h S_h - (\mu + \rho(1-e) + \tau)E_h \\
\frac{dI_h}{dt} &= \rho(1-e)E_h - (\mu + \delta + \alpha + \sigma)I_h \\
\frac{dT_h}{dt} &= \tau e E_h + \sigma I_h - (\mu + \gamma(1-d))T_h \\
\frac{dR_h}{dt} &= \alpha I_h + \gamma(1-d)T_h - (\mu + \varphi)R_h \\
\frac{dS_r}{dt} &= \Lambda_r + (\mu + \lambda_r(1-c))S_r \\
\frac{dI_r}{dt} &= \lambda_r(1-c)S_r - \mu I_r
\end{aligned}\right\} \quad (1)$$

Subject to the following nonnegative initial conditions:
$S_h(0) > 0$, $E_h(0) \geq 0$, $I_h(0) \geq 0$, $T_h(0) \geq 0$, $R_h(0) \geq 0$ and
$S_r(0) > 0$, $I_r(0) \geq 0$.
with

$$\left.\begin{aligned}
S_h(0) + E_h(0) + I_h(0) + T_h(0) + R_h(0) &\leq N_h(0) \\
S_r(0) + I_r(0) &\leq N_r(0)
\end{aligned}\right\} \quad (2)$$

where

$$\lambda_h = \emptyset I_r + \beta I_h \quad (3)$$

and

$$\lambda_r = \theta I_h \tag{4}$$

3.4 Model Analysis

In this section, we carryout the model analysis by showing that all feasible solutions of the model system are uniformly bounded in a proper subset of Ω. Thus the feasible region which is given as

$$\Omega = \{S_h,\ E_h,\ I_h,\ T_h,\ R_h, S_r,\ I_r\} \in \mathbb{R}_+^7 : N_h \leq \frac{\Lambda_h}{\mu}, N_r \leq \frac{\Lambda_r}{\mu} \tag{5}$$

is considered. In doing this, we take the derivative of the total human and rodent population and with proper simplification, we will have

$$\frac{dN_h}{dt}\Lambda_h - \mu N_h \tag{6}$$

$$\frac{dN_h}{dt}\Lambda_h - \mu N_h \tag{7}$$

Applying the theorem of differential inequality by Birkoff and Rota [11] in (6) and (7), we have

$$\left.\begin{aligned} N_h &\leq \frac{\Lambda_h}{\mu}(1 - e^{-\mu t}) + N_h(0)e^{-\mu t} \\ N_r &\leq \frac{\Lambda_r}{\mu}(1 - e^{-\mu t}) + N_r(0)e^{-\mu t} \end{aligned}\right\} \tag{8}$$

at $t = 0$, $N_h \leq N_h(0)$

$t = \infty$, $N_h \leq \frac{\Lambda_h}{\mu}$

Since, $N_h \geq 0$, then $0 \leq N_h \leq \frac{\Lambda_h}{\mu}$

Therefore, $0 \leq N_h \leq \frac{\Lambda_h}{\mu}$ and $0 \leq N_r \leq \frac{\Lambda_r}{\mu}$ as $t \to \infty$.

This means or implies that $\frac{\Lambda_h}{\mu}$ and $\frac{\Lambda_r}{\mu}$ are the upper bounds for the human population and the rodent population respectively. Therefore the solutions of the model equations of (1) enter the feasible region Ω which is a positively invariant set. Thus the system described by the system of equations (1) is both mathematically meaningful and epidemiologically well-posed.

3.5 Model Disease-free Equilibrium

Disease-free equilibrium denoted as DFE is a steady state solution where there is no Lassa fever infection in the population. It is gotten by solving the system of equation (1) in the absence of disease, that is where $E_h = I_h = T_h = R_h = I_r = 0$, which is given as:

$$E_0 = (S_h^0,\ E_h^0,\ I_h^0,\ T_h^0,\ R_h^0,\ S_r^0,\ I_r^0) = (\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, \frac{\Lambda_r}{\mu}, 0) \tag{9}$$

3.6 Model Endemic Equilibrium

Endemic equilibrium denoted by EE is a steady state where there is a presence of Lassa fever infection in the population. This is also gotten by solving the system of equation (1) where there is disease.

$$S_h = \frac{\Lambda_h}{\varphi k_5} \tag{10}$$

$$E_h = \frac{\lambda_h \Lambda_h}{\varphi k_3 k_5} \tag{11}$$

$$I_h = \frac{\rho(1-e)\lambda_h \Lambda_h}{\varphi k_2 k_3 k_5} \tag{12}$$

$$T_h = \frac{\tau e \lambda_h \Lambda_h}{\rho k_3 k_4 k_5} + \frac{\sigma\rho(1-e)\lambda_h \Lambda_h}{\varphi k_2 k_3 k_4 k_5} \tag{13}$$

$$R_h = \frac{(\lambda_h + \mu)\Lambda_h}{\varphi^2 k_5} - \frac{\Lambda_h}{\varphi} \tag{14}$$

$$S_r = \frac{\Lambda_r}{\mu(\mu + \lambda_r(1-c))} \tag{15}$$

$$I_r = \frac{\lambda_r(1-c)\Lambda_r}{\mu(\mu + \lambda_r(1-c))} \tag{16}$$

From above the Endemic Equilibrium points is given as

$$E^* = (S_h^*, E_h^*, I_h^*, T_h^*, R_h^*, S_r^*, I_r^*) = (\frac{\Lambda_h}{\varphi k_5}, \frac{\lambda_h \Lambda_h}{\varphi k_3 k_5}, \frac{\rho(1-e)\lambda_h \Lambda_h}{\varphi k_2 k_3 k_5}, \frac{\tau e \lambda_h \Lambda_h}{\rho k_3 k_4 k_5} + \frac{\sigma\rho(1-e)\lambda_h \Lambda_h}{\varphi k_2 k_3 k_4 k_5}, \frac{(\lambda_h + \mu)\Lambda_h}{\varphi^2 k_5} - \frac{\Lambda_h}{\varphi}, \frac{\Lambda_r}{\mu(\mu + \lambda_r(1-c))}, \frac{\lambda_r(1-c)\Lambda_r}{\mu(\mu + \lambda_r(1-c))}) \tag{17}$$

Where

$k_1 = \mu + \varphi,\ \ k_2 = \mu + \delta + \alpha + \sigma,\ \ k_3 = \mu + \rho(1-e) + \tau e,\ \ k_4 = \mu + \gamma(1-d),\ \ k_5 = \frac{\lambda_r \tau \mu}{\varphi} - \frac{\alpha\rho\lambda_r(1-e)}{k_1 k_2 k_3} - \frac{\gamma\tau e(1-d)\lambda_h}{k_4^2} - \frac{\gamma\rho\sigma\lambda_h(1-c)(1-d)}{k_2 k_3 k_4^2}$

3.7 Basic Reproduction Number (R_0)

The basic reproduction number is a parameter used to determine how long a disease will prevail in a particular population. If the basic reproduction number is less than one, it implies that an infected individual produces an average less than an infected person, and by calculation, it means that with time the disease will die out from the population. Diekmann et al [12] defined the basic reproduction number, R_0 as the number of secondary infections that one infectious individual will create over the duration of the infectious period, provided that everyone is susceptible.

We computed $R_0$of the model using the next generation matrix [12], [13]. It is defined as the largest eigenvalue or spectral radius of the characteristic equation $|FV^{-1} - \lambda I| = 0$, where λ is an eigenvalue associated with FV^{-1}, evaluating F and V at disease free equilibrium (DFE), we have:

$$f_i = \begin{bmatrix} \lambda_h S_h \\ 0 \\ 0 \\ \lambda_r(1-c)S_r \end{bmatrix} \qquad v_i = \begin{bmatrix} (\mu + \rho(1-e) + \tau e)E_h \\ (\mu + \delta + \alpha + \sigma)I_h - \rho(1-e)E_h \\ (\mu + \gamma(1-d)T_h - \tau e E_h + \sigma I_h \\ \mu I_r \end{bmatrix}$$

In obtaining associated matrices F and V for the new infections and the remaining transition terms, we form a Jacobian matrix evaluated at $E_h, I_h, T_h,$ and I_r, to obtain

$$F = \begin{bmatrix} 0 & \beta S_h & 0 & \emptyset S_h \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \theta S_r & 0 & 0 \end{bmatrix} \qquad V = \begin{bmatrix} \mu + \rho(1-e) + \tau e & 0 & 0 & 0 \\ -\rho(1-e)\ \mu + \delta + \alpha + \sigma & 0 & 0 & \\ -\tau e & \sigma & \mu + \gamma(1-d) & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \qquad (18)$$

By computing the value of V^{-1} and evaluating the characteristic polynomial equation $|FV^{-1} - \lambda I| = 0$ and also evaluating at DFE. we obtain the basic reproduction number for the model as:

$$R_0 = \frac{\beta\Lambda_h\rho(1-e)}{\mu(\mu+\rho(1-e)+\tau e)(\mu+\delta+\alpha+\sigma)} + \frac{\theta\Lambda_r(1-c)}{\mu^2} \qquad (19)$$

Which can be written as

$$R_0 = R_{0h} + R_{0r} \qquad (20)$$

Where $R_{0h} = \frac{\beta\Lambda_h\rho(1-e)}{\mu(\mu+\rho(1-e)+\tau e)(\mu+\delta+\alpha+\sigma)}$ and $R_{0r} = \frac{\theta\Lambda_r(1-c)}{\mu^2}$

R_{0h} is the basic reproduction number for humans, while

R_{0r} is the basic reproduction number for rodents.

4.0 Stability Analysis

4.1 Local Stability of Disease-free Equilibrium

Theorem 4.1: The disease-free equilibrium for the system (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The Jacobian of systems (1) for humans and for rodents are evaluated at the disease free equilibrium point. The local stability of E_0 is determined based on the signs of the eigenvalues of these Jacobians. The equilibrium E_0 is locally stable if the real part of these eigenvalues is all negative. At the steady state of the model, the Jacobian matrix is given by

$$J_1(E_0) = \begin{bmatrix} \beta I_h + \emptyset I_r & 0 & \beta S_h & 0 & \varphi \\ \beta I_h & -(\mu + \rho(1-e) + \tau e & \beta S_h & 0 & 0 \\ 0 & \rho(1-e) & -(\mu + \delta + \alpha + \sigma) & 0 & 0 \\ 0 & \tau e & \sigma & -(\mu + \gamma(1-d)) & 0 \\ 0 & 0 & \alpha & \gamma(1-d) & -(\mu + \varphi) \end{bmatrix} \qquad (21)$$

To get the eigenvalues, we solve the Jacobian using $|J_1(E_0) - \lambda I| = 0$ we now have that the eigenvalues are as follows

$\lambda_1 = 0$
$\lambda_2 = -(\mu + \varphi)$
$\lambda_3 = -(\mu + \gamma(1-d))$
For λ_4 and λ_5
we have

$$\begin{vmatrix} -d-\lambda & \beta S_h \\ \rho(1-e) & -f-\lambda \end{vmatrix} = 0 \quad (22)$$

where
$d = \mu + \delta + \alpha + \sigma$ and $f = \mu = \rho(1-e) + \tau e$
Solving (21), we have

$$\lambda^2 + \lambda(d+f) + df - \beta S_h \rho(1-e) = 0 \quad (23)$$

The values of λ can only be negative, if the coefficient of λ and the constant is positive.
Since the parameters are all positive, we then just have to show that the constant part, which is $df - \beta S_h \rho(1-e)$ is positive
From (22), we will have

$$-(\beta S_h \rho(1-e) - df) = -df\left(\frac{\beta S_h \rho(1-e)}{df} - 1\right) = -df\left(\frac{\beta \Lambda_h \rho(1-e)}{\mu(\mu+\rho(1-e)+\tau e)(\mu+\delta+\alpha+\sigma)} - 1\right)$$

where $\frac{\beta \Lambda_h \rho(1-e)}{\mu(\mu+\rho(1-e)+\tau e)(\mu+\delta+\alpha+\sigma)}$ is the R_0 for human
Which implies that the constant will be positive for $R_0 < 1$, which agrees with theorem 4.1.
We also computed the Jacobian for the rodents and found the eigenvalues to be
$\lambda_6 = 0$ and
$\lambda_7 = -\mu$
From above its observed that all the eigenvalues are either zero or negative, which implies that the disease free equilibrium is locally asymptomatically stable if $R_0 < 1$.

4.2 Global Stability of Disease-free Equilibrium

Theorem 4.2: The DFE of the model is globally asymptotically stable if $R_{0h} < 1$, $R_0 < 1$ and unstable if $R_0 > 1$, $R_0 > 1$.
Proof: We will establish theorem 4.2 by using the comparison theorem as used by Usman et al [14]. Applying the theorem, we compare the rate of change of the infectious compartments of the rodent as well as humans in the population, as shown in the equations below:

$$\begin{bmatrix} E_h \\ I_h \\ T_h \\ I_r \end{bmatrix} = (F-V) \begin{bmatrix} E_h \\ I_h \\ T_h \\ I_r \end{bmatrix} - S_h^0 A \begin{bmatrix} E_h \\ I_h \\ T_h \\ I_r \end{bmatrix} - S_r^0 \mathrm{B} \begin{bmatrix} E_h \\ I_h \\ T_h \\ I_r \end{bmatrix} \quad (24)$$

Where F and V are defined earlier in (18), while S_h^0 and S_r^0 are populations of the humans and rodents at DFE, and A and B are nonzero matrices. Now (24) can be written as

$$\begin{bmatrix} E_h \\ I_h \\ T_h \\ I_r \end{bmatrix} \leq (F-V) \begin{bmatrix} E_h \\ I_h \\ T_h \\ I_r \end{bmatrix} \quad (25)$$

Therefore, the matrix $(F-V)$ was obtained as

$$(F-V) = \begin{bmatrix} -(\mu+\rho(1-e)+\tau e) & \beta S_h & 0 & 0 \\ \rho(1-e) & -(\mu+\delta+\alpha+\sigma) & 0 & 0 \\ \tau e & \sigma & -(\mu+\gamma(1-d)) & 0 \\ 0 & \theta(1-c)S_r & 0 & -\mu \end{bmatrix} \quad (26)$$

Evaluating the characteristic polynomial equation $|(F-V) - \lambda I| = 0$, we obtain the following values of λ as the eigenvalues:
$\lambda_1 = -\mu, \lambda_2 = -(\mu + \gamma(1-d)), \lambda_3 = -(1-R_{0h})(1-R_{0r}), \lambda_4 = (\mu + \rho(1-e) + \tau e)(\mu + \delta + \alpha + \sigma)(R_{0h}R_{0r} - 1)$.

It is clear from above, that $\lambda_{1,2,3,4} < 0$, if R_{0h},$R_{0r} < 1$. Meaning that all the eigenvalues of (26) have negative real part, implying that the model (1) is said to be globally stable. But we can completely complete the proof of theorem 4.2 by constructing a lyapunov function, which is not presented here.

5.0 Numerical Simulation

In this section, we present the numerical analysis of the model using parameter values in the table below. The simulations were carried out using MATLAB codes encoded with ode45 and values of parameters gotten from existing literature on Lassa fever and few of them were assumed.

Table 2: Parameter values and source

Parameter	Value	Source
Λ_h	0.03	[15]
Λ_r	0.56	[15]
μ_h	0.02	[15]
μ_r	0.6	[15]
θ	0.52	[8]
α	0.0002	Assumed
ρ	0.85	[15]
e	0.85	[15]
τ	0.05	[15]
γ	0.75	[8]
$\emptyset$	0.022	[15]
d	0.45	[15]
β	0.6	[15]
δ	0.2	[15]
σ	0.9	[15]
c	0.2	Assumed
φ	0.75	[10]

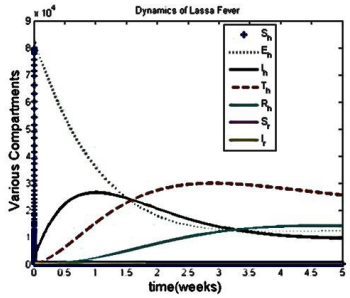

Figure 2: Graphical illustration of the general dynamics of Lassa fever in both human and rodent compartments.

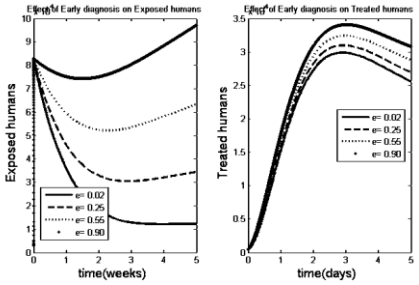

Figure 3: Graphical illustration of the effect of early diagnosis on the exposed and treated humans.

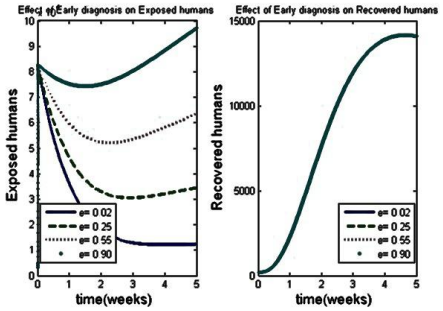

Figure 4: Graphical illustration showing the effect of early diagnosis on exposed and recovered human population.

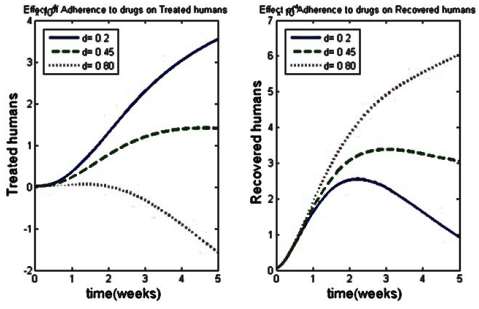

Figure 5: Graphical illustration showing the effect of adherence to drugs on treated and recovered human population.

5.0 Discussion of Results

In this work, we developed a mathematical model on the dynamics of Lassa fever transmission. In developing the model we divided the human population into five compartments ie Susceptible, Exposed, Infectious and Recovered class, and the rodent population into two compartments ie Susceptible and Infectious class. Recovery from the disease according to the model is as a result of treatment which can be from early diagnosis or after been infected and infectious, the recovered population can become susceptible after losing immunity. We computed the basic reproduction number for both humans and the rodents. Disease free and endemic equilibria were investigated and their stability analysed respectively.

It was established that the disease-free equilibrium was locally and globally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.

We carried out numerical analysis on our model, to investigate the effect of our control strategies on the system. Figure 2 showed the general dynamics of the Lassa fever disease, and it is evident that the susceptible humans almost went to extinction as a result of the interaction between infectious rodents and susceptible humans and between infectious humans and susceptible humans. Showing that without our control measures, the susceptible humans will all become infectious. Figure 3 shows the effect of early diagnosis on the exposed humans and treated humans, and from the graph, it can be seen that early diagnosis increases treatment of the disease, and it also showed evidently in figure 4 that it increases exponentially the recovery rate of humans from the disease. Figure 5 shows the effect of adherence to drug intake on the treated class and the recovered class. It is evidently shown that complete adherence to drugs increases recovery rate of humans from the disease at a very high rate. Figure 6 shows that control of rodents in the population by good hygiene and use of pesticides reduces the rate of infection on humans and also reduces infectious rodents in the population. Lastly figure 7, shows that with complete adherence to drug, early diagnosis and reduction of rodents in the population, the susceptible population increases showing, that the three control measures are the most effective way of preventing spread of Lassa fever disease.

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