EFFECTS OF QUARANTINE ON TRANSMISSION DYNAMICS OF LASSA FEVER

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ABSTRACT

In this paper, a mathematical model of Lassa fever is formulated. The model includes quarantine as a control strategy and allows re-infection. The model is shown to be well-posed. The disease free equilibrium is shown to be locally asymptotically stable whenever the basic reproduction number is less than unity and unstable otherwise. Numerical simulations have been used to show the impact of the control measure.

Keywords: Quarantine, re-infection, immunity, reproduction number, stability.

INTRODUCTION

Lassa fever, a viral hemorrhagic fever transmitted by rats, is endemic in West Africa (Carey et al., 1972; Frase, 1974; Monath et al., 1973; Monath et al., 1974). After an incubation period of 6 to 21 days, an acute illness with multi-organ involvement develops. Non-specific symptoms include fever, facial swelling, and muscle fatigue, as well as conjunctivitis and mucosal bleeding. Its symptoms include muscle pain, ulcers of the mucous membranes, headache, internal bleeding and inflammation of the throat. It also causes the destruction of internal tissues; lungs, heart and kidney failure (WHO, 2016). Furthermore, it is an infectious, often fatal, viral disease marked by high fever, which accounts for up to one-third of deaths in hospitals within the affected regions and 10% to 16% of total cases (CDC, 2014; CDC, 2015). It kills approximately 5,000 people per year (Richmond and Baglole, 2003).

The recognized human arenavirus infection history in Africa began in 1969 with the death of two medical missionaries mysteriously and the near-fatal illness of a third (Buckley et al., 1970; Frame et al., 1970; Frame, 1975). An arenavirus which was isolated from two of these patients is given the name of Lassa virus after the town of Lassa, Nigeria, where the disease, known as Lassa fever occurred.

Lassa virus is transmitted from animals; specifically it spreads to humans from a rodent known as natal multimammate mouse (Mastomys natalensis) or African rat. This is probably the most common mouse in equatorial Africa, ubiquitous in human households and eaten as a delicacy in some areas (Richmond and Baglole, 2003). Infection in the rodent population is in a persistent asymptomatic state. The virus is probably transmitted by contact with the feces or urine of animals accessing grain stores in residences (Richmond and Baglole, 2003). The possibility that Lassa virus could be used as a biological weapon has raised the profile of the need for greater understanding of Lassa fever and for more effective control and treatment programmes (Richmond and Baglole, 2003). Because of its high case fatality rate, ability to spread easily by human-to-human contact, and potential for aerosol release, Lassa virus is classified as a Bio-safety Level 4 (BSL4) and NIAID Bio-defense category A agent. The potential use of Lassa virus as a biological weapon directed against civilian or military targets necessitates the development of counter-threat measures, such as diagnostic assays, vaccines and therapeutics. Moreover, the impact of the disease in endemic regions of West Africa is immense, and therefore means to diagnose, treat and prevent this viral hemorrhagic fever will provide a significant public health benefit (CDC, 2015; WHO 2016). Re-infection occurs in Lassa fever as strengthened by (Richmond and Baglole, 2003). Many mathematical models have been designed and used to assess the effect of preventive measures on the spread of Lassa virus in a given community. This study extends the works of (James et al., 2015a; James et al., 2015b; Lo Iacono et al., 2015) by inter alia,

[i.] Incorporating environmental contribution to the transmission which is not considered in (James et al., 2015a; Lo Iacono et al., 2015);
[ii.] The environment is considered to be saturated;
[iii.] Incorporating quarantine as control measure (James et al., 2015b);
Model Formulation

The total human population, \( N_H(t) \), is divided into susceptible individuals \( S_H(t) \), asymptomatic individuals \( E_H(t) \), symptomatic individuals \( I_H(t) \) and individuals in quarantine receiving treatment \( Q(t) \), so that:

\[
N_H(t) = S_H(t) + E_H(t) + I_H(t) + Q(t). 
\]

Whereas, the total population of rodents, at time \( t \) denoted by \( N_R(t) \), is divided into two compartments for susceptible rodents and infected rodents, such that:

\[
N_R(t) = S_R(t) + I_R(t). 
\]

The susceptible population with risk of Lassa virus infection \( S_R(t) \) is generated by recruitment of humans at a constant rate \( \Pi \) (all humans recruited into the population are assumed to be at risk of Lassa-infection), infected individuals recover at a rate \( \gamma_1 \) and quarantine individuals recover at a rate \( \tau_2 \). The population is decreased by infection at a rate \( \lambda_H \), moving from the susceptible class to exposed class at a rate \( \lambda_H \) and natural death at a rate \( \mu_H \). Thus, 

\[
\frac{dS_H}{dt} = \Pi + \tau_1 I_H + \tau_2 Q - \lambda_H S_H - \mu_H S_H 
\]

The population of asymptomatic humans \( E_H(t) \) is generated by Lassa infection at the rate \( \lambda_H \). It is reduced by the development of clinical symptoms of Lassa at a rate \( \gamma_1 \) and natural death at the rate \( \mu_H \). Thus, 

\[
\frac{dE_H}{dt} = \lambda_H S_H - (\gamma_1 + \mu_H) E_H 
\]

The population of symptomatic individuals is increased at the rate \( \gamma_1 \) and diminished by recovery at the rate \( \tau_1 \), quarantine at the rate \( \gamma_2 \), natural death at the rate \( \mu_H \) and death induced by the disease at a rate \( \delta \). Thus, 

\[
\frac{dI_H}{dt} = \gamma_1 E_H - (\tau_1 + \gamma_2 + \delta + \mu_H) I_H. 
\]

The population of quarantine individuals is generated as a result of quarantining the symptomatic individuals at the rate \( \gamma_2 \) and diminished by recovery of individuals in the quarantine at the rate \( \tau_2 \), death induced by the disease \( \delta \) and natural death \( \mu_H \). Thus, 

\[
\frac{dQ}{dt} = \gamma_2 I_H - (\tau_2 + \delta + \mu_H) Q 
\]

The population of pathogens \( P \) in the environment is generated as a result of shedding from the infected rodents at a rate \( \alpha \). It is diminished by natural death of the pathogens, so that 

\[
\frac{dP}{dt} = \alpha I_R - \mu_P P 
\]

The population of susceptible rodents \( S_R \) is assumed to follow a logistic growth rate \( b_L(1 - \frac{N_R}{K}) \), where \( b_L \) is the maximum rate of growth of rodents and \( K > N_R \) is the carrying capacity (which is related to availability of food and space). Thus this shows that the growth of rodents is density dependent. The rodents population \( S_R \) decreased by Lassa fever infection and natural death at the rates \( \lambda_R \) and \( \mu_R \), respectively. Therefore, 

\[
\frac{dS_R}{dt} = b_L(1 - \frac{N_R}{K}) - \lambda_R S_R - \mu_R S_R 
\]

The population of infected rodents \( I_R \) is generated following the infection of susceptible rodents at the rate \( \lambda_R \) and decreased only because of natural death. Hence, 

\[
\frac{dI_R}{dt} = \lambda_R S_R - \mu_R I_R. 
\]

The model for the Lassa fever is described by the following system of differential equations while the flow diagram of the model is shown in Figure 1. The parameters and associated variables are presented in tables 1 and 2.
Some of the main assumptions made in the formulation of the model are as follows;

[i.] Homogeneous mixing of the human and rodents populations such that there are equal chances of transmitting the virus. Transmission patterns which are possible includes: rodent-to-rodent, rodent-to-human, human-to-human, human-to-rodent, environment-to-human and rodent contaminate the environment (Lo lacono et al., 2015);

[ii.] Successful treatment against Lassa fever does not guarantee permanent immunity against Lassa re-infection (Richmond and Baglole, 2003);

[iii.] Natural recovery is possible (Ajayi, 2014);

[iv.] Infected humans can transmit the disease via human-rodent infection (Lo lacono et al., 2015);
The virus does not kill the vector (i.e. they die naturally (James et al., 2015a); The model (1) extends the works in (James et al., 2015a; James et al., 2015b; Lo lacono et al., 2015) by opportuna,

[i.] incorporating environmental contribution to the transmission;

[ii.] The environment is considered to be saturated;

[iii.] incorporating quarantine as control measure (James et al., 2015b);

(iv.) The contribution of individuals is considered negligible;

[v.] The population of the reservoir (rodents) is divided into susceptible and infected classes;

[vi.] Recovered individuals have temporary immunity (Richmond and Baglole, 2003);

[vii.] Latency period is incorporated while it was neglected in (James et al., 2015a, James et al., 2015b);

[viii.] Using a logistic rate for susceptible rodents (constant rate was used in (James et al., 2015a, James et al., 2015b);

[ix.] Using incidence rate for both human (constant rate was used in (James et al., 2015a) and rodents population (constant rate was used in (James et al., 2015a, James et al., 2015b)));

2.1 Basic properties of the model.

Here, we first prove that a solution to the initial-value problem of system (1) exists and in fact, the solution is unique.

Theorem 2.1

Let $(S_{t0},E_{t0},I_{t0},Q_0,P_0,S_{R0},I_{R0}) \in \mathbb{R}$ be given. There exist, $t_0$ and continuously differentiable functions $(S_t(t), E_t(t), I_t(t), Q(t), P(t), S_R(t), I_R(t))$ such that the ordered heptads $(S_t(t), E_t(t), I_t(t), Q(t), P(t), S_R(t), I_R(t))$ satisfies model (1) and $(S_{t0}(t), E_{t0}(t), I_{t0}(t), Q(t), P(t), S_{R0}(t), I_{R0}(t)(0) = (S_{t0}, E_{t0}, I_{t0}, Q_0, P_0, S_{R0}, I_{R0})$.

Proof

The Classical Picard-Lindelof theorem will be utilized to prove the result. Since the system of ordinary differential equations is autonomous, it is enough to show that the function $f: \mathbb{R}^7 \rightarrow \mathbb{R}^7$ is defined by

$$f(y) = \begin{bmatrix}
\Pi + \tau_1 y_2 + \tau_2 y_4 - \lambda_H y_1 - \mu_H y_1 \\
\lambda_H y_1 - (y_1 + \mu_H) y_2 \\
y_1 y_2 - (y_1 + y_2 + \delta + \mu_H) y_3 \\
y_2 y_3 - (y_2 + \delta + \mu_H) y_4 \\
\alpha y_7 - \mu y_7 \\
\beta_1 (1 - \frac{N}{K}) - \lambda_R y_6 - \mu_R y_6 \\
\lambda_H y_6 - \mu_R y_7
\end{bmatrix}$$

where, $\lambda_H = \beta_H y_2 + \beta_E y_3 / (c + y_3) + \beta_R y_7$, and $\lambda_R = \beta_R y_7 + \beta_{RR} y_3$ is locally Lipschitz in its $y$ argument. In fact, it is enough to show that the Jacobian matrix

$\nabla f(y) = \begin{bmatrix}
A & B \\
C & D
\end{bmatrix}$

where,

$$A = \begin{bmatrix}
-\lambda_H + \mu_H & 0 & \tau_1 - \beta_H y_1 \\
\lambda_H & -(y_1 + \mu_H) & \beta_H y_1 \\
0 & y_1 & -(y_1 + y_2 + \delta + \mu_H)
\end{bmatrix},
B = \begin{bmatrix}
\frac{\beta_E y_3}{(c + y_3)^2} & 0 & \beta_R y_1 \\
0 & \frac{\beta_E y_3}{(c + y_3)^2} & 0 \\
0 & 0 & 0
\end{bmatrix},
C = \begin{bmatrix}
0 & 0 & 0 \\
0 & 0 & 0 \\
0 & 0 & \beta_{RR} y_6
\end{bmatrix},
D = \begin{bmatrix}
0 & 0 & 0 \\
-\mu_p & 0 & 0 \\
0 & -\frac{\beta_1}{K} + \lambda_R + \mu_R & -\frac{\beta_1}{K} + \beta_R y_6 \\
0 & 0 & \lambda_R - \mu_R + \beta_{RR} y_6
\end{bmatrix}$$

is linear in $y$ and therefore locally bounded for every $y \in \mathbb{R}^7$ so $f$ is locally Lipschitz in $y$. By the Picard-Lindelof Theorem, there exists a unique solution $y(t)$, to the ordinary differential equation $y'(t) = f(y(t))$ with initial value $y(0) = y_0$ on $[0, t_0]$ for some time $t_0 > 0$. Moreover, for positive initial data it can be shown that solutions remain positive as long as they exist. A lucky by product of the result above is that the obtained solutions are also bounded.

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Now on the interval $[0, t^*]$ such that the solution exists on $[0, t^*]$.

\[ T^* = \sup \{ t \in [0, t^*] : S_H(s), E_H(s), I_H(s), Q(s), P(s), S_R(s), I_R(s) > 0, \forall s \in [0, t^*) \} \]

Now on the interval $[0, T^*]$ we can estimate the population values knowing that all constants in the system are positive. Using this and the positivity of solutions on $[0, T^*]$, we can place lower bounds on $\frac{dS_H}{dt}$ and $\frac{dS_R}{dt}$ since

\[ \frac{dS_H}{dt} = \lambda R S_H(1 - \frac{N_R}{K}) - \mu H S_H - \mu S_H \leq \Pi + \tau I_H + \gamma I_H \]

\[ \frac{dS_R}{dt} = b_2(1 - \frac{N_R}{K}) - \lambda R S_R - \mu H S_R \leq b_2(1 - \frac{N_R}{K}) \]

\[ S_H(t) \leq c_1(1 + t), \quad \text{where the constant } c_1 \text{ satisfies } c_1 \geq \max \{ \Pi, K \}, \quad K = S_H(0) + \frac{\tau I_H(0)}{(1 + \gamma I_H + \delta + \mu H)} + \frac{\tau Q(0)}{(1 + \gamma I_H + \delta + \mu H)} \]

Similarly, we can place upper bound on $\frac{dS_H}{dt}$ and $\frac{dS_R}{dt}$ so that

\[ \frac{dS_H}{dt} = \Pi + \tau I_H + \gamma I_H - \lambda R S_H - \mu H S_H \leq \Pi + \tau I_H + \gamma I_H \]

\[ S_H(t) \leq c_1(1 + t), \quad \text{where the constant } c_2 \text{ satisfies } c_2 \geq \max \{ b_2, S_H(0) \} \]

Now, we can estimate the population values knowing that all constants in the system are positive. Using this and the positivity of solutions on $[0, T^*]$, we can place lower bounds on $\frac{dS_H}{dt}$ and $\frac{dS_R}{dt}$ since

\[ \frac{dS_H}{dt} = \lambda R S_H(1 - \frac{N_R}{K}) - \mu H S_H - \mu S_H \leq \Pi + \tau I_H + \gamma I_H \]

\[ \frac{dS_R}{dt} = b_2(1 - \frac{N_R}{K}) - \lambda R S_R - \mu H S_R \leq b_2(1 - \frac{N_R}{K}) \]

There is a bound on $S_H$ and $S_R$, simplifying after substituting $\lambda_H$ and $\lambda_R$ we have,

\[ \frac{d(E_H + I_H + P + I_R)}{dt} \leq c_3(1 + t)I_H + (c_4(1 + t) + \alpha)I_H + \gamma I_H + \beta EH P \]

where the constants $c_3$ and $c_4$ are, respectively, ($c_3 R_H + c_4 R_H$) and ($c_5 R_H + c_6 R_H$) also ($\Pi + \alpha > P$), $c_5$ satisfies $c_5 \geq \max \{ c_3, \alpha \}$ and $c_6$ satisfies $c_6 \geq \max \{ c_3, c_5 \}$. Consequently, the inequality becomes

\[ \frac{d(E_H + I_H + P + I_R)}{dt} \leq c_6(1 + t)(E_H + I_H + P + I_R) \]

for $t \in [0, t^*]$ where $c_6 > 0$ depends on $c_6 > 0$, $E_H0, I_H0, P0, S_R0$ and $I_R0$ only. Since $E_H(t)$ is positive, we can place an upper bound on $I_H(t)$, $P(t)$, $I_R(t)$

\[ \frac{dE_H}{dt} \leq c_7 e^{t^2} \]

\[ \frac{dI_H}{dt} \leq c_7 e^{t^2} \]

\[ \frac{dP}{dt} \leq c_7 e^{t^2} \]

Moreover, since $I_H(t)$, $P(t)$ and $I_R(t)$ are positive it follows that $E_H(t)$ is as well, hence

\[ \frac{dE_H}{dt} \leq c_7 e^{t^2} \]

Furthermore, adding the first four equations gives:

\[ \frac{dN_H}{dt} = \Pi - \mu H N_H - \delta(I_H + Q) \]

Then, $\Pi - \mu H N_H - \delta(I_H + Q) \leq \frac{dN_H}{dt} \leq \Pi - \mu H N_H$ so that
Substituting \( \lambda \) and the bound on \( I_H(t) \), \( P(t) \), \( I_R(t) \) that is, \( e^{\epsilon^2} \), it becomes

\[
\frac{dS_H}{dt} \geq -C_0(1 + e^{\epsilon^2})S_H
\]

for \( t \in [0, T^*] \) where \( C_0 \) satisfies \( C_0 \geq \max \{ \mu_H, C_{10} \} \) and \( C_R \geq \max \{ \beta_H, \beta_{EH}, \beta_R \} \), so

\[
\frac{dS_R}{dt} + C_0(1 + e^{\epsilon^2})S_H \geq 0. \text{ It is known that } \frac{d}{dt}(S_H(t) + e^{c_0(1+e^{\epsilon^2})dt}) \geq 0
\]

hence for \( t \in [0, T^*] \), \( S_H(t) > S_H(0)e^{-c_0(1+e^{\epsilon^2})dt} \)

Now for \( S_R(t) \)

\[
\frac{dS_R}{dt} = b_L \left( \frac{1 - N_R}{K} \right) - \lambda_R S_R - \mu_R S_R \geq -b_L \left( \frac{S_R + I_R}{K} \right) - \lambda_R S_R - \mu_R S_R
\]

\[
\geq -b_L \left( \frac{S_R}{K} \right) - b_L \left( \frac{I_R}{K} \right) - \lambda_R S_R - \mu_R S_R
\]

Substituting \( \lambda_R \) with the bounds established earlier on \( I_H \) and \( I_R \), gives

\[
\frac{dS_R}{dt} \geq -C_{12}(1 + e^{\epsilon^2})S_R
\]

where \( C_{12} \) satisfies \( C_{12} \geq \max \{ C_{10}, C_{11} \} \), and \( C_{10} = \left( \frac{b_H}{K} + \mu_R \right) \), \( C_{11} = C_2 \beta_R + C_{10} \beta_{HR} \)

hence, \( \frac{dS_R}{dt} + C_{12}(1 + e^{\epsilon^2})S_R \geq 0 \) and it follows that

\[
\frac{d}{dt}(S_R(t) + e^{c_{12}(1+e^{\epsilon^2})dt}) \geq 0 \text{ hence for } t \in [0, T^*]
\]

\[
S_R(t) > S_R(0)e^{-c_{12}(1+e^{\epsilon^2})dt}
\]

Thus the values of \( S_H, E_H, I_H, Q, P, S_R \) and \( I_R \) remain strictly positive for all \( [0, T^*] \), including at time \( T^* \). By continuity, there must exist a \( T^* \) such that \( S_H, E_H, I_H, Q, P, S_R \) and \( I_R \) are still positive.

This contradicts the definition of \( T^* \)

\[
(T^* = \sup \{ t \in [0,t_0] : S_H(s), E_H(s), I_H(s), Q(s), P(s), S_R(s), I_R(s) > 0, \forall s \in [0,t_0] \})
\]

and shows that the model (1) is strictly positive on the entire interval \([0,t^*]\). Furthermore, on the same interval, all of the functions remain bounded, so the interval of existence can be extended further. In fact, the bounds on \( S_H, E_H, I_H, Q, P, S_R \) and \( I_R \) derived above hold on a compact time interval. Thus, the time interval may be extended on which the solution exists to \([0, t_0] \) for any \( t_0 > 0 \) and from the above argument, the solutions remain bounded and positive on \([0, t_0]\).

With this, a general idea that the model is sound was obtained and can stay with certainty that it remains biologically valid as long as it begins with biologically-reasonable (i.e., positive) initial data. Following (Hethcote, 2000), the model is mathematically well-posed and epidemiologically realistic, since all the variables remain nonnegative for all \( t > 0 \). Hence, it is sufficient to consider the dynamics of the model (1) in \( D \).

**Lemma 2.3.**

The following biologically feasible region of the model equation (1) 

\[
D = (S_H, E_H, I_H, Q, P, S_R, I_R) \in \mathbb{R}^{7+} : S_H + E_H + I_H + Q \leq \frac{\mu_H}{\mu_H} : S_R + I_R \leq \frac{p_H}{p_R}
\]

is positively invariant and attracting.

**Proof.** It follows from the fact that

\[
\frac{dN_H(t)}{dt} = \Pi - \mu_H N_H(t) \quad \text{and} \quad \frac{dN_R(t)}{dt} = b_L - \left( \frac{b_H}{K} + \mu_R \right) N_R(t) \geq b_L - \mu_R N_R(t)
\]

so that \( \frac{dN_H(t)}{dt} < 0 \) and \( \frac{dN_R(t)}{dt} < 0 \) if \( N_H(t) > \frac{\mu_H}{\mu_R} \) and \( N_R(t) > \frac{p_H}{p_R} \).

Thus, a standard comparison theorem as in (Lakhsmikantham et al., 2015) can be used to show that

\[
N_H(t) \leq N_H(0)e^{-\mu_H t} + \frac{\mu_H}{\mu_R} \left( 1 - e^{-\mu_R t} \right) \quad \text{and} \quad N_R(t) \leq N_R(0)e^{-\mu_R t} + \frac{p_R}{p_H} \left( 1 - e^{-\mu_H t} \right).
\]

In particular, \( N_H(t) \leq \frac{\mu_H}{\mu_R} \) and \( N_R(t) \leq \frac{p_R}{p_H} \) respectively. Thus, \( D \) is positively-invariant.

Furthermore, if \( N_H(t) > \frac{\mu_H}{\mu_R} \) and \( N_R(t) > \frac{p_R}{p_H} \) then either the solution enters \( D \) infinite time, or \( N_H(t) \) approaches \( \frac{\mu_H}{\mu_R} \) and \( N_R(t) \) approaches \( \frac{p_R}{p_H} \) and the infected variables approaches zero. Here \( D \) is attracting (i.e. all solutions in \( \mathbb{R}^{7+} \) eventually approach, enter or stay in \( D \)). Hence the model (1) is epidemiologically well-posed in \( D \) as in (Hethcote, 2000).
Analysis of the model

It is instructive, however, to analyze system (1) first of all. This is done below.

Local asymptotic stability of disease-free equilibrium (DFE)

The human-rodent model (1) has a DFE, obtained by setting the right-hand sides of the equations in the model to zero, given by

\[ \epsilon_{0L} = (S_H^*, E_H^*, I_H^*, Q^*, P^*, S_R^*, I_R^*) = \left( \frac{\mu_L}{\mu_H}, 0, 0, 0, 0, \beta_{RH} \right) \]

The linear stability of \( \epsilon_{0L} \) will be investigated using the next generation operator method on the system (1). The matrices \( F \) (for the new infection terms) and \( V \) (for the remaining transition terms) associated with the model are given, respectively, by

\[
F = \begin{pmatrix}
0 & \beta_{RH}^* & 0 & \beta_{EH}^* \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & \mu_R^*
\end{pmatrix},
V = \begin{pmatrix}
k_1 & 0 & 0 & 0 \\
-\gamma_1 & k_2 & 0 & 0 \\
0 & -\gamma_2 & k_3 & 0 \\
0 & 0 & \mu_p & -\alpha \\
0 & 0 & 0 & \mu_R
\end{pmatrix}
\]

The threshold quantity \( R_0 \) is the basic reproduction number for Lassa fever (Anderson and May, 1982; Anderson and May 1991; Hethcote, 2000). It represents the average number of secondary cases that one infectious human (or rodent) would generate over the duration of the infectious period if introduced into a completely susceptible human (rodent) population.

**Lemma 3.1**

The DFE \( \epsilon_{0L} \) of system (1) is locally asymptotically stable (LAS) if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

**Simulations**

The numerical simulation was conducted using ODE15s in-built in MATLAB where averted cases were computed for \( R_0 < 1 \) and \( R_0 > 1 \), also four graphs were obtained; graph of infected humans with and without quarantine when \( R_0 = 0.8764 \) as depicted in Figure 2; graph of averted cases when \( R_0 < 1 \) and \( \text{AVERTION}=4.7128 \times 10^3 \) labeled Figure 3. Moreover, graph of infected humans with and without quarantine when \( R_0 = 3.1481 \) labelled Figure 4 and graph of averted cases, \( \text{AVERTION}=4.7128 \times 10^3 \) label Figure 5. The parameter values in table 4 were used to get the figures below.
Table 4: Description of parameters of the model

| Parameter | Interpretation                                                                                   | Range/Baseline value          | Reference                                      |
|-----------|-------------------------------------------------------------------------------------------------|-------------------------------|-----------------------------------------------|
| \( \Pi \) | Recruitment rate for humans                                                                      | 64,787,478                    | (Richmond and Baglole, 2003)                   |
| \( \mu_H, \mu_R, \mu_P \) | Natural death rates of humans, rodents and pathogens respectively                                | 0.018182, 0.1858, 16248.088819 | (CIA, 2008; Oliff, 2003; Stephenson, 1984)     |
| \( \beta_R, \beta_{RH}, \beta_{EH}, \beta_RH \) | Transmission rates from infected rodents to susceptible rodents, rodents to susceptible humans, ingesting pathogens from the contaminated environment and air to susceptible humans, infected humans to susceptible humans and from humans to susceptible rodents, respectively, | 0.43, 0.43, Estimate, 0.4, Estimate. Dimensionless | (Elizabeth et al., 2014; Elizabeth et al., 2014; Estimate; Elizabeth et al., 2014; Estimate;) |
| \( \alpha \) | Rates of shedding from rodent to environment                                                      | \( 10^3 - 10^5(\text{TCID})_{50/ml} \) | (McCormick, 1987)                             |
| \( \kappa_c \) | Concentration of the pathogens in the contaminated environment and air                          | \( 10^3 - 10^5(\text{TCID})_{50/ml} \) | (McCormick, 1987)                             |
| \( \tau_1 \) | Re-infection rate of humans from infected                                                        | \([0.01, 0.18]\)               | (Richmond and Baglole, 2003)                   |
| \( \tau_2 \) | Re-infection rate of humans from isolated humans                                                 | \([0.01, 0.18]\)               | (Richmond and Baglole, 2003)                   |
| \( \gamma_1 \) | Progression rate of exposed humans to infected class                                             | 0.7869                        | (Richmond and Baglole, 2003)                   |
| \( \gamma_2 \) | Progression rate of infected humans to isolated class                                            | \([0.05, 0.08]\)               | (McCormick, 1987)                             |
| \( K \) | Carrying capacity for rodents                                                                     | 946                           | (McCormick, 1987)                             |
| \( \delta \) | Disease-induced death rate for humans                                                             | \([0.0452, 0.1133]\)          | (NCDC, 2018)                                  |
| \( b_L \) | Maximum rate of growth of rodent population                                                      | \(1.502 \text{ per head per 28 days}\) | (Oliff, 1953)                                 |
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Figure 2. Graph of infected humans with and without intervention when $R_0 < 1$ at $\beta_H = 4 \times 10^{-11}$

Figure 3. Graph of averted cases, $\text{AVERTION} = 4.7128 \times 10^9$

Figure 4. Graph of infected humans with and without quarantine when $R_0 > 1$ at $\beta_H = 4 \times 10^{-14}$
CONCLUSION

In this work, a mathematical model is developed and analyzed to study the transmission and control of Lassa fever. Mathematically we modeled Lassa fever as a 7-dimensional system of non-linear ordinary differential equation. We first show that there exists a domain where our model is well posed mathematically and epidemiologically. The model incorporates quarantine and re-infection parameters. The DFE point of the model is obtained and analyzed for stability. We obtained an important threshold parameter called basic reproductive number \( R_0 \).

Numerical simulations, using the parameter values in table 4 show that the associated reproduction number \( R_0 < 1 \), and decrease when quarantine is implemented as shown in Figure 2. Thus, the outlook of the effective control of Lassa virus is greatly enhanced if a control strategy based on using quarantine of the infected and infectious human is implemented, which shows the averted cases in Figure 3. But when \( R_0 > 1 \), the Figure 4 and Figure 5 shows the infected humans with and without quarantine when \( R_0 > 1 \) at \( \beta_H = 4 \times 10^{-14} \), and the averted cases respectively.

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