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THEORETICAL PERSPECTIVES ON THE WITHIN-HUMAN HOST MODEL FOR LASSA FEVER

CHINEDU VICTOR OBASI[1,*], GODWIN CHRISTOPHER MBAH[2]

[1]Department of Mathematics, Alvan Ikoku Federal College of Education, Owerri, Imo State, Nigeria

[2]Department of Mathematics, University of Nigeria, Nsukka, Enugu State, Nigeria

Abstract: In this paper, we theoretically analyzed the within-human host model for Lassa fever earlier proposed by Obasi and Mbah, so as to understand the dynamics of Lassa fever transmission at the human population level. The model has locally asymptotically stable disease-free equilibrium whenever the associated reproduction number is less than unity. This model will undergo the phenomenon of backward bifurcation where the stable disease-free co-exists with a stable endemic equilibrium, when the associated reproduction number is less than unity. This implies that bringing down the reproduction number to below unity is not enough to eradicate Lassa fever disease within human population. It is also shown that the model has a globally-asymptotically stable disease-free equilibrium whenever the associated reproduction number is less than unity. The reproduction number, $R_{WH} < 1$, which is an important parameter in the control of Lassa fever infection, has been calculated using the next generation method. We have also shown that the endemic equilibrium point exists for $R_{WH} > 1$ and has been noted that this endemic equilibrium is unique and locally asymptotically stable based on Lyaponuv Function. However, this work has thrown up important parameters that could be gathered by the relevant government agencies for better understanding of the burden of Lassa fever disease in the human population.

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*Corresponding author

E-mail address: obasi1212@gmail.com

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1. INTRODUCTION

Lassa fever (LF) is a deadly epidemic disease which threaten public health security and was discovered in Nigeria in 1969. LF is a viral hemorrhagic fever caused by the arenavirus and transmitted primarily from rodents (multimammate rats) to humans, human to human and aerosol transmission [2, 4, & 6]. The Arenaviruses are a family of viruses whose members are generally associated with rodent-transmitted diseases in humans. The incubation period is 5-21 days in a susceptible host and treatment is done using ribavirin drug which is effective when started within the first 6 days of illness. An estimated 300,000-500,000 infections per year with 5000 deaths have been reported [6]. The spread of infection diseases has always been of concerns and a threat to public health epidemiologists [3].

The goal of public health epidemiologists is first to understand the dynamics of LF, then to predict its course, and finally to develop ways of controlling it. This goal however will be absolutely achieved through mathematical modeling. By mathematical model we mean a mathematical representation of a system that can be used to explore its behaviour. Mathematical modeling is a significant and powerful tool that can be employed in analyzing the spread and control of infectious diseases such as LF. Many studies have been carried out to model the transmission dynamics of LF disease in humans and rodents [3, 4, & 5]. Many of these studies focuses only on the transmission of the disease in human and the rodent populations but recently, Obasi and Mbah [3] formulated a coupled deterministic system of differential equations accounting for all the three known LF transmission routes. Thus, it is instructive to carry out modeling studies to analyze the transmission dynamic properties of the uncoupled model at the within-human host level. To the best of our knowledge, there are no theoretical models that examine the uncoupled transmission dynamics of LF analytically. Our goal is to theoretically analyze the within-human host model for LF earlier proposed by Obasi and Mbah [3] so as to understand the dynamics of LF transmission at the human population level. The paper is organized as follows: the model is given in section 2 and analyzed in sections 3 and 4. Section 5 provides concluding remarks.

2. WITHIN-HUMAN HOST MODEL

According to Obasi and Mbah [3], the model sub-divides the total human population at time t, namely; S denoting the number of susceptible individuals, E the exposed non-infectious

individuals, I denoting the number of infectious individuals (infected and diagnosed or symptomatic) and R, denoting the number of recovered individuals. The susceptible human population increases due to recruitment at Λ, a loss of immunity from the recovered class at a rate ψ and recovered humans without immunity at a rate $(1-v)$. The susceptible human population reduces as a result of a contact with an infectious rodent and a sexual contact with infected humans, aerosols and a natural death at a rate μ. The exposed human population increases the transmission routes and reduces due to a transition of individuals from the class of exposed to the infected class at a rate of κ and a natural death at a rate of μ. The infected humans may recover with temporary immunity at a rate v and progress to recovered class while the remaining proportion recovers without immunity and become susceptible at a rate $(1-v)$, while ϕ is the proportion of humans who recovered spontaneously. The infected human population reduces by natural death and induced death at rates μ and δ respectively. The recovered human population reduces by natural death at a rate μ and loss of immunity at a rate ψ. Another aspect of the transmission route is the opportunistic airborne transmission-infectious that naturally cause disease by small airborne particles (aerosol) that contain microorganisms. The modified version of Wells-Riley equation is used to describe airborne transmission route in this Lassa fever model. The exponent represents the degree of exposure to infection and $\left(1-e^{-rt}\right)$ is the probability of a single susceptible being infected. Note that t is the exposure time while r is the exposure rate, which is given as $r=\frac{q}{Q}$. Here q is the number of doses of airborne infection while Q is the volume flow rate of fresh or disinfected air on airborne infection. Thus, putting the above formulations and assumptions together gives the following within-human host model, given by system of ordinary differential equations below. A full description of the variables and parameters to be used in the model are in Table (1) and Table (2) respectively.

$$\begin{cases} \dfrac{dS}{dt} = \Lambda + \phi(1-v)I + \psi R - \beta_1 \sigma I_r S - \beta_2 \varepsilon IS - \eta\left(1-e^{-rt}\right)S - \mu S \\ \dfrac{dE}{dt} = \beta_1 \sigma I_r S + \beta_2 \varepsilon IS + \eta\left(1-e^{-rt}\right)S - (\kappa+\mu)E \\ \dfrac{dI}{dt} = \kappa E - (\phi+\delta+\mu)I \\ \dfrac{dR}{dt} = \phi v I - (\psi+\mu)R \end{cases} \tag{1}$$

Table 1: Description of the state variables of the model

Variable	Description
S	Number of Susceptible humans
E	Number of Exposed humans
I	Number of Infectious humans
R	Number of Recovered humans
I_r	Number of Infected rodents

Table 2: Description of the parameters of the basic Lassa fever model

Parameters	Description
Λ	Recruitment level of humans
δ	Per capita Lassa-induced death rate
ψ	Recovered human loss of immunity
ϕ	Spontaneous individual recovery
ν	Fraction of recovered humans without immunity
β_1	Transmission rate per contact by an infectious rodent
β_2	Transmission rate per contact by an infective through sexual activity
η	Relative infectiousness of individuals with aerosol
μ	Natural mortality rate for humans
κ	Progression rate of human from exposed to infected
σ	Contact rate of rodent per human per unit time
ε	Relative human-to-human transmissibility of infected humans
r	Exposure rate to aerosol

3. QUALITATIVE PROPERTIES OF THE MODEL

For the model (1) to be meaningful, it is important to prove that all its state variables are non-negative for all time (t). In other words, the solutions of the model (1) with positive initial data will remain positive for all $t \geq 0$. Suppose $S_h(0) \geq 0$. The first equation of system (1) can be written as:

$$\frac{d}{dt}\left[S(t)\eta(t)\right]=\Lambda\eta(t), \tag{2}$$

where

$$\eta(t)=\exp\left(\int_0^t\left[\lambda(S)+\mu\right]dS\right)>0$$

is the integrating factor. Hence, integrating this last relation with respect to *t*, we have

$$S(t)\eta(t)-S(0)=\int_0^t\Lambda\eta(S)dS,$$

so that the division of both side by $\eta(t)$ yields

$$S(t)=\left[S(0)+\int_0^t\Lambda\eta(S)dS\right]\times\eta^{-1}(t)>0.$$

The same arguments can be used to show that other state variables are positive for all $t>0$. The dynamics of model (1) is a dynamical system in the biological feasible compact set:

$$\Gamma:=\left\{(S,E,I,R)\in\mathbb{R}_+^4:N\le\frac{\Lambda}{\eta\left(1-e^{-rt}\right)+\mu}\right\} \tag{3}$$

4. ASYMPTOTIC STABILITY OF DISEASE-FREE EQUILIBRIUM (DFE)

The disease-free equilibrium of the model (1) is given by

$$\xi_0=\left(S^*,E^*,I^*,R^*\right)=\left(\frac{\Lambda}{\eta\left(1-e^{-rt}\right)+\mu},0,0,0\right)$$

The linear stability of ξ_0 can be established using the next generation operator method on the system (1). Using the notations in [3, 5], it follows that *F* and *V*, which stands for the new infection terms and remaining transition terms, respectively, are given by

$$F=\begin{pmatrix}0 & \beta_2\varepsilon S\\ 0 & 0\end{pmatrix};V=\begin{pmatrix}(\kappa+\mu) & 0\\ -\kappa & (\phi+\delta+\mu)\end{pmatrix}$$

It follows that the dynamics is completely determined by the reproduction number, R_{WH}, is given by

$$R_{WH}=\frac{\beta_2\varepsilon\kappa\Lambda}{(\kappa+\mu)(\phi+\delta+\mu)\left(\eta\left(1-e^{-rt}\right)+\mu\right)} \tag{4}$$

The result below follows from theorem 2 in [3].

Lemma 2: The DFE of the within-human host model, given by ξ_0, is locally asymptotically stable (LAS) if $R_{WH} < 1$, and unstable if $R_{WH} > 1$.

Hence, by Theorem 2 in [3], the DFE is locally asymptotically stable (LAS) whenever $R_{WH} < 1$ but unstable if $R_{WH} > 1$.Therefore, we do not need to show the LAS of the DFE by the method of linearization of the system (at the DFE). Calculating R_{WH} using the method of the next generation matrix approach in [3] automatically proves the LAS of the DFE. Epidemiologically, this implies that Lassa fever will be eliminated from the population whenever $R_{WH} < 1$ if the initial size of the sub-populations are in the basin of attraction of the DFE i.e. a small influx of Lassa fever infectious individuals into the community will not generate a large Lassa fever outbreak and the disease dies out in time.

5. BACKWARD BIFURCATION ANALYSIS

It is instructive to characterize the type of bifurcation the model (1) may undergo. This will go a long way in determining factors that could hinder efforts in tackling Lassa fever in the human population. We claim the following result.

Theorem 1: The model (1) exhibits backward bifurcation at $R_{WH} = 1$ whenever a bifurcation coefficient, denoted by a (and given below) is positive.

Proof

Let $\xi_1 = \left(S^{**}, E^{**}, I^{**}, R^{**}\right)$

represents any arbitrary endemic equilibrium of the model (1) (that is, an equilibrium in which at least one of the infected components is non-zero). The existence of backward bifurcation will be explored using Centre Manifold Theory [1]. To apply this theory, it is convenient to carry out the following change of variables.

Let $S = x_1, E = x_2, I = x_3, R = x_4$. Further, by using the vector notation $X = \left(x_1, x_2, x_3, x_4\right)^T$, the model (1) can be written in the form $\frac{dX}{dt} = F(X)$, with $F = (f_1, f_2, f_3, f_4)^T$, as follows

$$\begin{aligned}
\dot{x}_1 &\equiv f_1 = \Lambda + \phi(1-\nu)x_3 + \psi x_4 - \beta_1\sigma I_r x_1 - \beta_2\varepsilon x_3 x_1 - \eta\left(1-e^{-rt}\right)x_1 - \mu x_1 \\
\dot{x}_2 &\equiv f_2 = \beta_1\sigma I_r x_1 + \beta_2\varepsilon x_3 x_1 + \eta\left(1-e^{-rt}\right)x_1 - (\kappa+\mu)x_2 \\
\dot{x}_3 &\equiv f_3 = \kappa x_2 - (\phi+\delta+\mu)x_3 \\
\dot{x}_4 &\equiv f_4 = \phi\nu x_3 - (\psi+\mu)x_4
\end{aligned} \tag{5}$$

where

$$G_1 = \phi(1-\nu) - \beta_2\varepsilon S^*, G_2 = \beta_2\varepsilon S^*, G_3 = \phi+\delta+\mu, G_4 = \eta\left(1-e^{-rt}\right)+\mu$$

and force of infection given by

$$\lambda = \beta_1\sigma I_r^* + \beta_2\varepsilon I^* + \eta\left(1-e^{-rt}\right)$$

Let us choose $\beta_2 = \beta^*$ as a bifurcation parameter. Solving for $\beta_2 = \beta^*$ from $R_{WH} = 1$ gives

$$\beta_2 = \beta^* = \frac{(\kappa+\mu)G_3G_4}{\varepsilon\kappa\Lambda}$$

The Jacobian of the transformed system (3), evaluated at the DFE with $\beta_2 = \beta^*$, is given by

$$J^* = J(\xi_0)|_{\beta_2=\beta^*} = \begin{pmatrix} -G_4 & 0 & G_1 & \psi \\ \eta\left(1-e^{-rt}\right) & -(\kappa+\mu) & G_2 & 0 \\ 0 & \kappa & -G_3 & 0 \\ 0 & 0 & \phi\nu & -(\psi+\mu) \end{pmatrix} \tag{6}$$

The matrix J^* has a right eigenvector given by $w = (\omega_1, \omega_2, \omega_3, \omega_4)^T$, where

$$\begin{aligned}
\omega_1 &= \frac{G_1\omega_3 + \psi\omega_4}{G_4} \\
\omega_2 &= \omega_2 > 0 \\
\omega_3 &= \frac{\kappa\omega_2}{G_3} > 0 \\
\omega_4 &= \frac{\phi\nu\kappa}{(\psi+\mu)G_3}\omega_2 > 0
\end{aligned} \tag{7}$$

Furthermore, the matrix J^* has a left eigenvector $v = (\nu_1, \nu_2, \nu_3, \nu_4)^T$, satisfying $w \bullet v = 1$, with

$$\begin{aligned}
\nu_1 &= \frac{\eta\left(1-e^{-rt}\right)}{G_4}\nu_2 > 0 \\
\nu_2 &= \nu_2 > 0 \\
\nu_3 &= \frac{\kappa+\mu}{\kappa}\nu_2 > 0 \\
\nu_4 &= \frac{\psi+\mu}{\psi}\nu_2 > 0
\end{aligned} \tag{8}$$

It follows from Theorem 3 in [1], by computing the associated non-zero partial derivatives of $F(X)$ (evaluated at the DFE), that the associated bifurcation coefficients, a and b, defined by

$$a = \sum_{k,i,j=1}^{4} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$
$$and \tag{9}$$
$$b = \sum_{k,i=1}^{4} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0)$$

are computed to be

$$a = 2\varepsilon\beta^* v_2 \left(\frac{G_1\omega_3 + \psi\omega_4}{G_4}\right)\left(\frac{\kappa\omega_2}{G_3}\right)\left(1 - \frac{\eta\left(1-e^{-rt}\right)}{G_4}\right) > 0 \tag{10}$$

and

$$b = \varepsilon v_2 \left(\frac{G_1\omega_3 + \psi\omega_4}{G_4}\right)x_3^* - v_1\omega_2\left(\frac{\kappa\omega_2}{G_3}\right)x_1^* \tag{11}$$

In summary, the Centre Manifold Theorem [1] states that, at a bifurcation point, the system (or model) undergoes a backward bifurcation if the bifurcation coefficients satisfies $a > 0$ and $b > 0$. Thus, the within-human host model will exhibit backward bifurcation. The epidemiological implication of this result is that bringing down the reproduction number to below unity is not enough to eradicate Lassa fever disease within human population.

6. GLOBAL ASYMPTOTIC STABILITY: SPECIAL CASE $\sigma = \eta = 0$.

Consider the model (1) with $\sigma = \eta = 0$. We claim the following:

Theorem 2: The DFE of the model (1) with $\sigma = \eta = 0$ is globally-asymptotically stable (GAS) whenever $R_{WH} < 1$.

Proof. Consider the model (1) with $\sigma = \eta = 0$. Further, consider the following linear Lyaponuv function

$$F = \kappa E(t) + (\kappa + \mu) I(t)$$

with Lyaponuv derivative (where a dot represents differentiation with respect to *t*

$$\begin{aligned}
\dot{F} &= \kappa\dot{E}(t) + (\kappa+\mu)\dot{I}(t) \\
&= \kappa\left(\lambda S - (\kappa+\mu)E\right) + (\kappa+\mu)\left(\kappa E - (\phi+\delta+\mu)I\right) \\
&= (\kappa+\mu)(\phi+\delta+\mu)\left[\frac{\beta_2\varepsilon\kappa S}{(\kappa+\mu)(\phi+\delta+\mu)} - 1\right]I \\
\dot{F} &\le (\kappa+\mu)(\phi+\delta+\mu)\left[R_{WH} - 1\right]I
\end{aligned} \tag{12}$$

Hence, $\dot{F} \le 0$ if $R_{WH} \le 1$ with $\dot{F} = 0$ if and only $I = 0$. Therefore F is a Lyaponuv function in Ω and it follows Salle's Invariance Principle [1], that every solution to the equations in (1) (with $\sigma = \eta = 0$) with initial conditions in Ω converges to ξ_0 as $t \to \infty$. i.e., $(E(t), I(t), R(t)) \to (0,0,0)$ as $t \to \infty$. Substituting $E = I = R = 0$ into the first equation of (1) gives $S(t) \to \frac{\Lambda}{\eta\left(1-e^{-rt}\right)+\mu_h}$ as $t \to \infty, R_{WH} \le 1$, so the DFE point is globally asymptotically stable for $R_{WH} < 1$.

7. EXISTENCE AND STABILITY OF THE DISEASE ENDEMIC EQUILIBRIUM

To establish the existence of the disease endemic equilibrium of the model (1), let

$\xi_1 = \left(S^{**}, E^{**}, I^{**}, R^{**}\right)$

Represents any arbitrary disease endemic equilibrium of the model (1). The equations in (1) are solved in terms of the force of infection at steady state to give

$$S^{**} = \frac{-(\phi+\delta+\mu_h)(\psi+\mu_h)\lambda^{**}\Lambda}{(\psi+\mu)(1-\nu)\phi\kappa\lambda^{**} + \lambda^{**}\psi\phi\nu\kappa - (\phi+\delta+\mu)(\psi+\mu)(\lambda^{**}+\mu)(\kappa+\mu)}\left[\frac{(\kappa+\mu)}{\lambda^{**}}\right]$$

$$E^{**} = \frac{-(\phi+\delta+\mu_h)(\psi+\mu_h)\lambda^{**}\Lambda}{(\psi+\mu)(1-\nu)\phi\kappa\lambda^{**} + \lambda^{**}\psi\phi\nu\kappa - (\phi+\delta+\mu)(\psi+\mu)(\lambda^{**}+\mu)(\kappa+\mu)}$$

$$I^{**} = \frac{-(\phi+\delta+\mu_h)(\psi+\mu_h)\lambda^{**}\Lambda}{(\psi+\mu)(1-\nu)\phi\kappa\lambda^{**} + \lambda^{**}\psi\phi\nu\kappa - (\phi+\delta+\mu)(\psi+\mu)(\lambda^{**}+\mu)(\kappa+\mu)}\left[\frac{\kappa}{(\phi+\delta+\mu)}\right]$$

$$R^{**} = \frac{-(\phi+\delta+\mu_h)(\psi+\mu_h)\lambda^{**}\Lambda}{(\psi+\mu)(1-\nu)\phi\kappa\lambda^{**} + \lambda^{**}\psi\phi\nu\kappa - (\phi+\delta+\mu)(\psi+\mu)(\lambda^{**}+\mu)(\kappa+\mu)}\left[\frac{\kappa\phi\nu}{(\phi+\delta+\mu)(\psi+\mu)}\right]$$

Note that the disease force of infection at steady state, λ^{**} is expressed as

$$\lambda^{**} = \beta_1\sigma I^{**} + \beta_2\varepsilon I^{**} + \eta\left(1-e^{-rt}\right)$$

Substituting the expressions above gives $a_0\lambda^{**} + b_0 = 0$, where

$$\begin{aligned} a_0 &= (\psi+\mu)(1-\nu)\phi\kappa + \psi\phi\nu\kappa - (\phi+\delta+\mu)(\psi+\mu)(\kappa+\mu) \\ b_0 &= \beta_2\varepsilon\kappa\Lambda(\psi+\mu) - \mu(\phi+\delta+\mu)(\psi+\mu)(\kappa+\mu) \\ &= (\psi+\mu)\left[\beta_2\varepsilon\kappa\Lambda - \mu(\phi+\delta+\mu)(\kappa+\mu)\right] \\ &= \mu(\phi+\delta+\mu)(\psi+\mu)(\kappa+\mu)(R_{WH}-1) \end{aligned} \qquad (13)$$

The coefficient a_0 is always positive, the coefficient b_0 is positive (negative) if R_{WH} is greater than (less than) unity. Furthermore, there is no negative endemic equilibrium if $b_0 \le 0$. If $b_0 > 0$, then there is a unique endemic equilibrium. This result is summarized below.

Lemma 3: The model (1) has a unique positive endemic equilibrium when $\sigma = \eta = 0$ whenever $R_{WH} > 1$.

8. LOCAL ASYMPTOTIC STABILITY OF THE DISEASE ENDEMIC EQUILIBRIUM

The local stability of the disease endemic equilibrium point can be discussed by examining the linearized form of the system (1) at the given steady state ξ_1. This is done by computing the Jacobian matrix of the model (1). At the disease endemic equilibrium point, the Jacobian of the system (1) is

$$J(\xi_1) = \begin{pmatrix} P & 0 & A & \psi \\ \lambda & -B & C & 0 \\ 0 & \kappa & -D & 0 \\ 0 & 0 & \phi\nu & -E \end{pmatrix} \tag{14}$$

where

$$A = \phi(1-\nu) - \beta_2 \varepsilon S^{**}, B = \kappa + \mu, C = \beta_2 \varepsilon S^{**}, D = \phi + \delta + \mu, E = \psi + \mu, P = -\left(\lambda^{**} + \mu\right)$$

The characteristic polynomial, associated with the local stability of $J(\xi_1)$, is

$$A_4 \ell^4 + A_3 \ell^3 + A_2 \ell^2 + A_1 \ell + A_0 = 0,$$

where

$$\begin{aligned} A_4 &= 1 \\ A_3 &= E + D + B - P \\ A_2 &= BD + BE - BP - C\kappa + DE - DP - EP - A\kappa \\ A_1 &= BDE - BDP - BEP - CE\kappa + CP\kappa - DEP - AE\kappa - \phi\nu\kappa\psi \\ A_0 &= CEP\kappa - BDEP \end{aligned} \tag{15}$$

and ℓ_i represents the eigenvalues of the Jacobian. The local stability of the disease endemic equilibrium is tied to the roots of (15). The disease endemic equilibrium point is locally asymptotically stable when the polynomial in (15) have negative real roots.

9. CONCLUDING REMARKS

In this paper, we theoretically analyzed the within-human host model for Lassa fever earlier proposed by Obasi and Mbah [3] so as to understand the dynamics of Lassa fever transmission at the human population level. The model has locally asymptotically stable disease-free equilibrium

whenever the associated reproduction number is less than unity. This model will undergo the phenomenon of backward bifurcation. The epidemiological implication of this result is that bringing down the reproduction number to below unity is not enough to eradicate Lassa fever disease within human population. It is also shown that the model has a globally-asymptotically stable disease-free equilibrium whenever the associated reproduction number is less than unity. The reproduction number, $R_{WH} < 1$, which is an important parameter in the control of Lassa fever infection, has been calculated using the next generation method. We have also shown that the endemic equilibrium point exists for $R_{WH} > 1$ and has been noted that this endemic equilibrium is unique and locally asymptotically stable based on Lyaponuv Function. However, this work has thrown up important parameters (for example σ and η) that could be gathered by the relevant government agencies for better understanding of the burden of Lassa fever disease in the human population. The analysis suggests that Lassa fever disease can be eradicated when the basic reproduction number is less than unity. We therefore advocate for health policies that will keep the basic reproduction number below one, thereby keeping the occurrence of Lasa fever under control.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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