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A Mathematical Model for the Control of Lassa Fever.

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Abstract

A mathematical model for the control of laser fever disease was developed. The research shows that lassa fever disease can be controlled by reducing the transmission rate and using control strategies that can lead to a reduction in the basic reproduction number. The numerical imulation shows the effect of the control parameter on the various classes of the model. The results also show that the infected human population increases at the initial stage because of the migration from susceptible human class and get to the peak after a certain period of time. However, the infected human population begins to drop due to the high disease-induced death, recovery and migration to the Treated class. Similarly, the treated human population increases at the initial stage due to the migration from the infected class and later decreases due to migration to the treated class. The treated and recovered human populations increase at the initial stage but later beg into decrease because of the effective prevention and control strategies and the efficacy of the vaccine.

1. INTRODUTION

Lassa fever is an infectious disease that is transmitted from animal to human and human to human. Mas-tomys (atalensis), a rat species is the animal host of lassa fever. People contract lassa fever has a result of poor hygienic conditions, inadequate or ineffective prevention measures and contact between susceptible persons and infected food, animal or human [1]. Lassa fever is an acute viral haemorrhagic fever illness that is known to be endemic in many West African countries including Nigeria according to World Health Organization 2017. The current outbreak of the disease in Nigeria started in December 2016and a total of 501 suspected cases including 104 deaths have been reported as at 9June, 2017. Out of this number, 189 have been further classified, 175 laboratory-confirmed including 59 deaths and 14probable cases (all dead). The current Lassa fever outbreak has been reported in 17 Nigerian states and each has at least one confirmed case. As at 9 June, 2017, the outbreak was still active in 9 states. Prevention of Lassa fever relies on promoting good community hygiene to discourage rodents from entering homes. In health-care settings, staff should always apply standard infection prevention and control precautions when caring for patients, regardless of their presumed diagnosis [2]. The incubation period of lassa fever is between six to twenty-one days. The disease is endemic in West African countries like Sierra loane, Ghana and Nigeria. [3] proposed a model of lassa fever disease dynamics. The equilibrium states were obtained and were analysed for stability. It was discovered that the zero equilibrium state of the disease is stable when the birth rate of the human population is less than the death rate and when the birth rate of the mastomysnatalensis (reservoir) is less than the total death rates. [4] formulated a six-dimensional ordinary differential equation modeling the transmission of lassa virus between humans and reservoir with control strategies. The stability analysis of the disease free equilibrium was obtained and the basic reproduction number using the next generation operator approach. The model results shows that the disease free equilibrium is locally a symptotically stable at and unstable at. The existence of the endemic equilibrium was determined. The numerical simulations was carried out and the possible nature of the model. [5] developed an SIR model for the control of lassa fever in Northern part of Edo State. The results of the analysis showed that for the disease to be eradicated from the endemic area, the transmission rate must be very low when compared with the recovery rate. The paper advocated adequate health education, low cost housings chemetoreduce over-population in some places and good health policy. Adequate equilibrium for the diagnosis and treatment of lassa fever must be provided. Lassa fever patients must be isolated and vaccinated to increase the rate of recovery. [6] developed a mathematical model for the spread and control of lassa fever. The six compartmental model incorporates two control parameters, the used of condom to control human to human transmission via sexual contact with opposite sex and the use of Rodenticide to reduce both dormant and active rat population. The disease free equilibrium points of the model were obtained and the analysis was carried out to determine its stability.

2. THEORETICALANALYSIS

2.1 Table1: Model Variables

Variable	Description
$S_h(t)$	Susceptible Human Population
$I_h(t)$	Infected Population
$T_h(t)$	Treated Human Population
$R_h(t)$	Recovered Human Population
$S_v(t)$	Susceptible Vector Population
$I_v(t)$	Infected Vector Population

2.2 Model Parameters

Parameter Description

α_h Natural Birth Rate of Human Population

μ_h Natural Death Rate of Human Population

ω_h Rate of Loss of Immunity of Recovered Population

γ_1 Progression Rate from Susceptible Human to Infected Human Population

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β_h	Contracting Rate for Susceptible Human Population as a Result of Interaction with the Infected Human Po
β_v	Contracting rate for Susceptible Human Population as a Result of Interaction with Infected Vector Population
δ_h	Disease-Induced Death Rate of Human Population
μ_v	Natural Death Rate of Vector Population
γ_2	Rate of Progression from Infected Human to Treated Human Population
α_v	Birth Rate of Vector Population
δ_v	Disease-Induced Death Rate of Vector Population
ω_v	Rate of Progression from Susceptible Vector to Induced Vector Population
γ_3	Recovery Rate of Treated Human Population
γ_4	Rate of Recovery by Natural Immunity
β	Treatment Factor

2.3 Model Equation

$$\frac{dS_h}{dt} = \alpha_h + \varpi_h R_h - \mu_h S_h - \gamma_1 S_h \quad (2.1)$$

$$\frac{dI_h}{dt} = \gamma_1 S_h - \gamma_2 I_h - \delta_h I_h - \mu_h I_h - \gamma_4 I_h + \beta_v S_h \quad (2.2)$$

$$\frac{dT_h}{dt} = \gamma_2 I_h - \eta\gamma_3 T_h - (1-\eta)\delta_h T_h - \mu_h T_h \quad (2.3)$$

$$\frac{dR_h}{dt} = \eta\gamma_3 T_h + \gamma_4 I_h - \omega_h T_h - \mu_h T_h \quad (2.4)$$

$$\frac{dS_y}{dt} = \alpha_v - \omega_v S_v - \mu_v S_v \quad (2.5)$$

$$\frac{dI_y}{dt} = \omega_v S_v - \mu_v I_v - \delta_v I_v \quad (2.6)$$

Were the infection rate β_V is due to the interaction between susceptible humans and infected humans and susceptible humans and infected rodents and it is defined as

$\beta_V = \beta_h I_h S_h + \beta_V I_V S$

2.4 Derivation of the Model Equations

The model is made up of six classes namely: susceptible human population $S_h(t)$, infected human population $I_h(t)$, treated human population $T_h(t)$, recovered human population $R_h(t)$, susceptible vector population $S_V(t)$ and infected human population $I_V(t)$.

The susceptible human population $S_h(t)$ is generated through natural birth rate of human population α_h and it is increased by the number of recovered human population due to the rate of loss of immunity of recovered population at the rate ω_h. This population is reduced by the natural death rate of human populationμ_h and the progression rate from susceptible to infected human population at the rate γ_1.

The infected human population $I_h(t)$ is generated by the progression from susceptible human population to infected human population at the rate γ_1 also it is increased by the contact rate for susceptible human population as a result of interaction with infected vector population at the rate β_V and is reduced by the rate of progression from infected to treated human population at the rate γ_2 and disease–induced death rate of human population δ_h.It is also decreased by natural death and natural immunity at the rate μ_h and γ_4 respectively.

The treated human population T_h (t) is generated by the rate of progression from infected to treated human population at the rate γ_2 and it is reduced by recovery rate of treated human population at the rate $\eta\gamma_3$. It is further reduced by disease–induced and natural death at the rate (1-η) δ_h and μ_h respectively.

The recovered human population $R_h(t)$ is generated by the recovery rate of treated human population $\eta\gamma_3$ and rate of recovery by natural immunity γ_4,but it is decreased by the rate of loss of immunity of recovered population at the rate ω_h and natural death rate of human population at the rate μ_h.

The susceptible vector population $S_V(t)$ is generated by the birth rate of vector population α_V and reduced by the rate of progression from susceptible to infected vector population at the rate ω_V and it is also decreased by the natural death rate of vector population μ_V.

The infected vector population $I_V(t)$ is generated by the rate of progression from susceptible to infected vector population at the rate ω_V and decreased by the natural death rate of vector population μ_V. It is also reduced by the disease–induced death rate of vector population at the rate δ_V.

Basic Properties of the Model

Theorem 1: The closed set

Proof:

$\frac{dN_h}{dt} \leq \alpha_h - \mu_h N_h$. $\frac{dN_v}{dt} \leq \alpha_v - (\mu_h + \delta_h)N_v$. It follows that $\frac{dN_h}{dt} \leq 0$and $\frac{dN_v}{dt} \leq 0$ if $N_h(t) > \frac{\alpha_h}{\mu_h}$ and $N_h(t) > \frac{\alpha_v}{\mu_v}$ respectively. Thus, a standard comparison theorem as in [7] can be used to show that $N_h(t) \leq N_h(0)e^{\mu_h(t)} + \frac{\alpha_h}{\mu_h}(1 - e^{\mu_h(t)})$ and $N_v(t) \leq N_v(0)e^{\mu_v(t)} + \frac{\alpha_v}{\mu_v + \delta_v}(1 - e^{(\mu_v + \delta_v)(t)})$. In particular $N_h(t) \leq \frac{\alpha_h}{\mu_h}$ and $N_v(t) \leq \frac{\alpha_v}{\mu_v + \delta_v}$ if $N_h(0) \leq \frac{\alpha_h}{\mu_h}$and $N_v(0) \leq + \frac{\alpha_v}{\mu_v + \delta_v}$. Thus, D is positively-invariant. Further, if $N_h(0) > \frac{\alpha_h}{\mu_h}$and $N_v(0) > \frac{\alpha_v}{\mu_v + \delta_v}$, then either the solution enters D in the finite time or $N_h(t)$ approaches $\frac{\alpha_h}{\mu_h}$ and $N_v(t)$ approaches $\frac{\alpha_v}{\mu_v + \delta_v}$, and the infected variable $I_h + T_h$ approaches 0. Hence, D is attracting, i.e all solutions in $\mathfrak{R}_+^6$ eventually enters D. Thus in D, the basic model equation (2.1) to (2.6) is well defined epidemiologically and mathematically according to [8]. Hence, it is sufficient to study the dynamics of the model equation (2.1) to (2.6)

2.5 Basic Reproductive Number (R_0)

These are the number of secondary infections produced by an infection in a population that is totally susceptible. It can be measured by counting the number of secondary cases following the transmission of the disease into a susceptible population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of the disease. These models usually have athreshold parameter, known as the basic reproductive number R_0 such that when $R_0 < 1$then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $R_0 > 1$,then the DFE is unstable and invasion is always possible. The basic reproductive number R_0 is the spectral radius of the product matrix [9]FV^{-1}, i.e, $R_0 = \rho(FV^{-1})$

The model has two infective compartments namely the Infected population I_h, and the Treated population T_h. It follows that the matrices F and V for the new infective terms and remaining transfer terms respectively are given below:

$$F = \begin{bmatrix} \beta_h S_h^2 & 0 \\ 0 & \eta\delta_h \end{bmatrix} \quad V = \begin{bmatrix} (\gamma_2 + \delta_h + \mu_h + \gamma_4) & 0 \\ -\gamma_2 & (\eta\gamma_3 + \mu_h + \gamma_4) \end{bmatrix} \tag{2.7}$$

$$F^{-1} = \begin{bmatrix} \beta_h S_h^2 & 0 \\ 0 & \eta\delta_h \end{bmatrix} \begin{bmatrix} \frac{1}{(\gamma_2 + \delta_h + \mu_h + \gamma_4)} & 0 \\ \frac{-\gamma_2}{(\gamma_2 + \delta_h + \mu_h + \gamma_4)(\delta_h + \mu_h)} & \eta\gamma_3 + \frac{1}{(\mu_h + \gamma_4)} \end{bmatrix} \tag{2.8}$$

Then

$$FV^{-1} = \begin{bmatrix} \frac{\beta_h S_h^2}{(\gamma_2 + \delta_h + \mu_h + \gamma_4)} & 0 \\ \frac{-\gamma_2}{(\gamma_2 + \delta_h + \mu_h + \gamma_4)(\delta_h + \mu_h)} & \eta\gamma_3 + \frac{1}{(\mu_h + \gamma_4)} \end{bmatrix} \tag{2.9}$$

Theorem2: All the solution of the model equations (2.1) to (2.6) are positive for all time $t \geq 0$provided that the initial conditions are positive.

Proof:

Under the assumption that all initial conditions are positive, i.e.

$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0$.

By contradictions, we have that the solution of (2.1) to (2.6) are positive if we assume for contradiction that there exists an initial time, t_1:$S_h(t_1) = 0$ and

$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0$,
$0<t<t_1$
or there exists

t_2:$I_h(t_2)=0$ad

$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0$,
$0<t<t_2$
or there exists
3:$T_h(t_3)=0$and
$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0$,
$0<t<t_3$
or there exists
t_4:$R_h(t_4)=0$and
$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0$,
$0<t<t_4$
or there exists

t_5:$S_v(t_5)=0$and
$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0,$
$0<t<t_5$
or there exists
t_6:$I_v(t_6)=0$and
$S_h(0)>0, I_h(0)>0, T_h(0)>0, R_h(0)>0, S_v(0)>0, I_v(0)>0,$
$0<t<t_6$
Now, the case where

$$S_h(t_1)=0 \tag{2.10}$$

We have;

$$\frac{dS_h(t_1)}{dt}=\lim_{t\to t_1} S_h \frac{(t)-S_h}{t-t_1}(t_1)<0$$

Similarly, we have

$$\frac{dI_h(t_2)}{dt}<\frac{dT_h(t_3)}{dt}<0,\ \frac{dR_h(t_4)}{dt}<0$$

$$\frac{dS_y(t_5)}{dt}<0\ \frac{dI_y(t_6)}{dt}<0$$

However, from the model equation we have that

$$S_h^v(t_1)=\alpha_h+\omega_h R_h-\mu_u S_h(t_1)-\gamma_1 S_h(t_1)$$

i.e.

$$S_h^v(t_1)=\alpha_h+\omega_h R_h>0$$

Which contradicts equation (2.10) therefore,

$$S_h^v(t_1)=0$$

The S_h will remain positive for all t. Similarly, for the remaining variables, we have

$$S_h^v(t_2)=\gamma_1 S_h+\beta_v\ IS_h^v>0$$

$$T_h^v(t_3)=\ \gamma_2 I_h>0$$

$$R_h^v(t_4)=\eta\gamma_3 T_h+\ \gamma_4 I_h>0$$

$$S_v^v(t_5)=\alpha_v>0$$

$I_v^v(t_6)=\omega_v S_v>0$. This contradictions affect the variables that

$$I^o I_h(t_2)=0,\ {}^\circ T_h(t_3)=0,\ {}^\circ R_h(t_4)=0,\ {}^\circ S_v(t_5)=0,\ {}^\circ I_h(t_6)=0$$

Hence I_h, T_h, R_h, S_v, I_v remains positive for all t. By this we have shown that all the solution of the model equation are positive, provided that the initial conditions are positive.

3 EXPERIMENTALWORK

In this section, we discuss the results of the model and the effect of the control strategies that are being employed to control the spread of laser fever disease in endemic area.

Table 1:Table of Values

Parameters	Values	Source
Δ_V	0.3	[1]
A_h	0.0000215	[11]
A_V	0.05	[5]
Ω_V	0.06	[4]
μ_h	0.0000548	[11]
μ_V	0.04	[4]
γ_1	0.52	[4]
B_h	0.52	[4]
B_V	0.00005	[11]
Δ_h	0.01	[4]
$\gamma_{3,4}$	0.52	Assumed

4 RESULTS AND DISCUSSION

Figure 1showsthatthe Susceptible human population decreases with time because of the high rate of infection. The Susceptible human population then move to the Infected class.

Figure 2 shows that the Infected human population increases at the initial stage because of the migration from susceptible human class and get to the peak after a certain period of time. However, the infected human population begins to drop due to the high disease-induced death, recovery and migration to the Treated class.

Figure 3 shows that the Treated human population increases due to the migration from the Infected class, where the Infected class get treated. However, the Treated class then decreases with time due to treatment and migrate to the Recovery class

Figure 4 shows that the Recovery human population increases due to the migration from the Treated class. Whereby the Infected population recovered from the infection and moves back to the Susceptible human population

Figure 5 shows that the Susceptible vector population decreases due to human killing them or by natural death.

Figure 6 shows the Infected vector population decreases due to not able to infect new ones and will eventually die out or been killed by human as time increases.

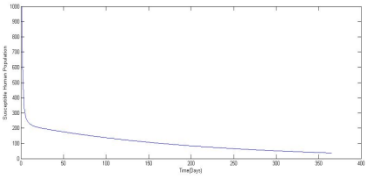

Figure 1: Graph of Susceptible Human Population against Time

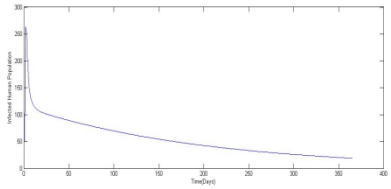

Figure 2:Graph of Infected Human Population against Time

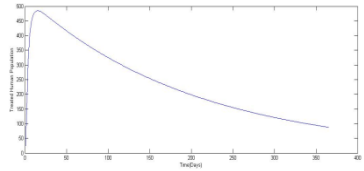

Figure 3:Graph of Treated Human Population against Time

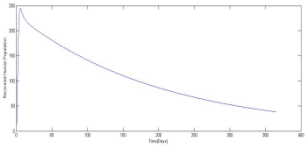

Figure 4:Graph of Recovered Human Population against Time

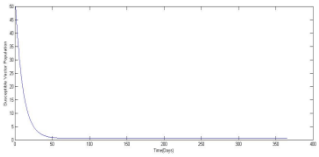

Figure 5: Graph of Susceptible Vector Population against Time

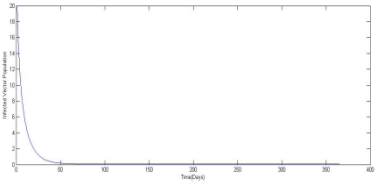

Figure 6: Graph of Infected Vector Population against Time

5 CONCLUSION

A mathematical model for the control of lassa fever has been developed. The numerical simulation of the model shows that after an initial rise in the number of susceptible, infected, treated recovered human populations due to some certain factors, there are significant reduction in the number of these classes of human population.

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