

## Modelling and optimal control analysis of Lassa fever disease

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### ABSTRACT

Lassa fever is a severe hemorrhagic viral infection whose agents belong to *Mastomys natalensis*. Generally, humans contract Lassa virus through exposure to food or household products that have been contaminated with the excreta of the infected rodents. Lassa fever is endemic in some West African countries including Nigeria. A basic model is proposed to examine the transmission of the disease. The proposed model is subjected to qualitative study via the theory of differential equations and the threshold quantity that denotes the dominant eigenvalue was derived using next-generation matrix approach. The basic model is further extended to an optimal control model with four controls namely, the fumigation of the environment with pesticide, the use of condom to prevent human to human transmission during sexual activities, early treatment and the use of indoor residual spray. The theory of optimal control was explored to establish the necessary conditions for curtailing the transmission of Lassa fever. Numerical simulation was conducted and the results showed that if the Lassa fever transmission and spread were to be reduced significantly in the endemic region, all the control measures must be taken with all seriousness.

### 1. Introduction

Lassa fever is a rat-spreading, haemorrhagic disease. The disease originated in Nigeria [1]. Although Lassa fever has been in Nigeria in the 1950s, it only became known in 1969 when it claimed the lives of two nurses at Lassa town, one of the towns in today's Borno State [2]. Lassa fever is confined to West Africa and has become a serious health challenge in the region with significant morbidity and mortality recorded each year [3]. The eradication of Lassa fever from the West African sub-region has become challenging because recovery from the disease cannot be predicted with certainty as the virus can remain in the bodily fluids of humans (e.g. semen) after recovery [4].

Lassa fever cases are predominant in Nigeria, Liberia, Sierra-Leone and Guinea. Nevertheless, other neighbouring countries also stand the risk of contracting the disease as the agents of Lassa fever, *Mastomys natalensis* are distributed throughout the neighbouring countries. In certain regions in Liberia and Sierra- Leone, it is reported that about

10%–16% of all hospitalization each year is due to Lassa fever, confirming the health implication of Lassa fever on the population of these countries [5]. Human-to-human transmission and laboratory spread of the disease may also emanate from the inadequate prevention and control strategies, particularly in the health care system [6]. The incidences of Lassa fever are strongest in the dry season, despite the breeding of multimammate rodents' reservoir during the rainy season [7–11].

The incubation period of Lassa fever ranges from 6 to 21 days. For most Lassa fever infection, (about 80%), symptoms are usually mild and undetectable. The mild signs include moderate fever, general pain and fatigue, and headache. Nevertheless, the disease can progress to severe symptoms in 20% of people infected with the disease. The infection can grow to acute symptoms, including respiratory disorder, incessant vomiting, severe pains in the back, chest and abdomen, facial disfiguration and shock [12].

Several mathematical models have been used to analyse physical,

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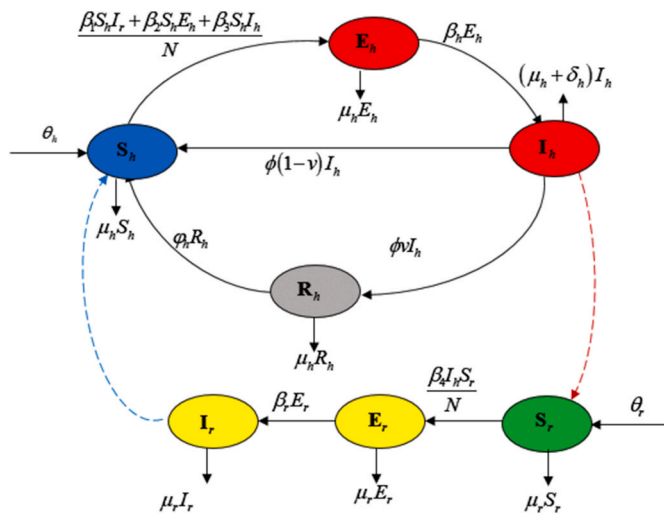


Fig. 1. Pictorial representation of the model.

**Table 1**  
Detailed definition of variables and parameters.

Variable	Description
$S_h(t)$	Susceptible human
$E_h(t)$	Exposed human
$I_h(t)$	Infected human
$R_h(t)$	Recovered human
$S_r(t)$	Susceptible rat
$E_r(t)$	Exposed rat
$I_r(t)$	Infected rat
Parameter	Description
$\theta_h$	Recruitment rate of susceptible humans
$\theta_r$	Recruitment rate of susceptible rat
$\alpha_h$	Rate of progression of human from the exposed class to the infected class.
$\mu_h$	Natural death rate of humans.
$\delta_h$	The disease-induced death rate for humans
$\tau$	Constant rate due to treatment.
$\phi$	Natural recovery rate for individuals
$v$	Human recovery rate with limited immunity
$(1 - v)$	Proportion of infected human with Lassa fever with limited immunity becoming susceptible to Lassa fever.
$\theta$	Constant rate of using the indoor residual spray
$\beta_1$	Probability of disease transmission per contact by an infectious rat
$\beta_2$	Rate of spread of the disease via sexual activity through asymptomatic infected human
$\beta_3$	Rate of spread of the disease via sexual activity through symptomatic infected human
$\beta_4$	Rate of spread of the disease per contact by an infectious human.
$\beta_r$	Movement rate of exposed rat to infected rat
$\mu_r$	Natural mortality rate for rat.

biological and many other complex systems dynamics. Different studies have been conducted using the mathematical models applied to epidemiology, which includes the following [13–19]. Given this, few studies have attempted to use the mathematical modelling techniques to study the dynamics of Lassa virus [20–29]. The application of optimal control theory to disease modelling offers useful knowledge about how control steps can be implemented. Numerous infectious diseases have been contained through vaccination, diagnosis, public education, and so on [30]. Since the implementation of optimal control theory in disease modelling, a considerable number of infectious disease studies have been conducted using the concept of optimal control theory [31–36].

We have identified that till now, only one study have attempted to study optimal control of Lassa fever disease dynamics using

mathematical modelling approach. [37] developed a deterministic model of Lassa fever using *SIR* and *SI* approach for the human and rat populations respectively. Lassa fever can also be transmitted from person to person through contact with an infected person’s blood, urine, saliva, throat secretion or semen [38]. This study bridges the gap by considering a deterministic model, incorporating exposed human compartment and exposed rat compartment. The following controls are considered. The use of condom during sex as control to prevent human to human transmission, fumigating the environment with pesticide, early treatment and the use of indoor residual spray which was not considered in their work. In view of the above, we developed a deterministic mathematical model to analyse the dynamics of Lassa fever disease with optimal control strategies and the effects of such controls. The paper is organised as follows; section two deals with the formulation of the model and the mathematical analysis of the optimal control problem, section three presents the numerical simulation and the results. Sections four and five are the discussion of results and conclusion respectively.

## 2. Materials and methods

In this section, we propose a Lassa fever model which comprises two groups; the human population and rat population. The human population is divided into four compartments; namely, the susceptible  $S_h$ , the exposed  $E_h$ , the infected  $I_h$ , and the recovered  $R_h$  individuals. Similarly, the rat population is divided into the following compartments;  $S_r, E_r, I_r$  which respectively represents the susceptible rat, the exposed rat population and the infected rat population. The human and rat population are function of time  $t$ , that is,  $S_h(t), E_h(t), I_h(t), R_h(t)$  and  $S_r(t), E_r(t), I_r(t)$  respectively.  $\theta_h$  represents recruitment rate of humans which is by birth or immigration.  $\theta_r$  is the rate of recruitment for the rat,  $\alpha_h$  represents the rate of progression of humans from the exposed class to the infected class.  $\mu_h$  represents the natural death rate of humans. The disease-induced death rate by humans is represented by  $\delta_h$ ,  $\tau$  is the constant rate due to treatment. There is a natural recovery rate for individuals denoted by  $\phi$ . We assume that the infected human may recover with limited immunity at the rate  $v$  by migrating to the recovery class whereas, the proportion of infected individuals with limited immunity become susceptible to Lassa fever at the rate  $(1 - v)$ .  $\theta$  is the constant rate of using indoor residual spray,  $\beta_1$  represents the probability of disease transmission per contact by an infectious rat,  $\beta_2$  is the rate of spread of the disease via sexual activity through asymptomatic infected human,  $\beta_3$  is the rate of spread of the disease via sexual activity through symptomatic infected human,  $\beta_4$  is the rate of spread of the disease per contact by an infectious human. The infected rat population is increased as a result of movement from exposed class at the rate  $\beta_r$  but is reduced by the natural mortality rate at  $\mu_r$ . From the above descriptions, we have the following system of differential equations in equation (1) while the pictorial representation of the model is displayed in Fig. 1. The associated model’s variables and parameters are described in Table 1.

$$\begin{aligned}
 \frac{dS_h}{dt} &= \theta_h + (1 - v)\phi I_h + \varphi_h R_h - \left( \frac{\beta_1 S_h I_r + \beta_2 S_h E_r + \beta_3 S_h I_h}{N_h} \right) - \mu_h S_h, \\
 \frac{dE_h}{dt} &= \left( \frac{\beta_1 S_h I_r + \beta_2 S_h E_r + \beta_3 S_h I_h}{N_h} \right) - \beta_h E_h - \mu_h E_h, \\
 \frac{dI_h}{dt} &= \beta_h E_h - (\mu_h + \delta_h) I_h - \phi I_h, \\
 \frac{dR_h}{dt} &= v\phi I_h - (\varphi_h + \mu_h) R_h, \\
 \frac{dS_r}{dt} &= \theta_r - \frac{\beta_4 I_h S_r}{N_h} - \mu_r S_r, \\
 \frac{dE_r}{dt} &= \frac{\beta_4 I_h S_r}{N_h} - (\beta_r + \mu_r) E_r,
 \end{aligned}
 \tag{1}$$

**Table 2**

Parameters of the model and values.

$S_h = 100, E_h = 20, I_h = 10, R_h = 5, S_r = 1000, E_r = 10, I_r = 20$

Parameter	Value	Source	Parameter	Value	Source
$\beta_h$	0.01	Assumed	$\beta_4$	0.025	[12]
$\mu_r$	0.0038	[26]	$\phi$	0.05	Assumed
$\mu_h$	0.003465	[26]	$\tau$	0.2	Assumed
$\theta_r$	0.00001	[12]	$\nu$	0.23	Assumed
$\theta_h$	1.2	Assumed	$\theta$	0.75	Assumed
$\beta_1$	0.0182	[25]	$\delta_h$	0.00019231	[13]
$\beta_2$	0.083	[25]	$\varphi_h$	0.00385	[25]
$\beta_3$	0.024	[25]			

**2.1. The model analysis**

Normalizing system (1), we obtain,

$$\begin{aligned}
 S'_h(t) &= \theta_h + (\phi + \tau)(1 - \nu)I_h + \varphi_h R_h - \beta_1 S_h I_r - \beta_2 S_h E_h - \beta_3 S_h I_h - \mu_h S_h, \\
 E'_h(t) &= \beta_1 S_h I_r + \beta_2 S_h E_h + \beta_3 S_h I_h - \beta_h E_h - \mu_h E_h, \\
 I'_h(t) &= \beta_h E_h - (\mu_h + \delta_h)I_h - \phi I, \\
 R'_h(t) &= (\phi + \tau)I_h \nu - \varphi_h R_h - \mu_h R_h, \\
 S'_r(t) &= \theta_r - \beta_4 I_h S_r - \mu_r S_r - \theta S_r, \\
 E'_r(t) &= \beta_4 I_h S_r - (\beta_r + \mu_r)E_r, \\
 I'_r(t) &= \beta_r E_r - \mu_r I_r.
 \end{aligned}
 \tag{2}$$

For the human population,

$N_h = S_h + E_h + I_h + R_h$ , the differential equation is given as

$$\frac{dN_h}{dt} = \theta_h - \delta_h I_h - \mu_h N_h.$$

Also, for the rat population,

$N_r = S_r + E_r + R_r$ , and the corresponding differential equation is given as

$$\frac{dN_r}{dt} = \theta_r - \mu_r N_r$$

**Theorem 1.**

Let  $(S_h, E_h, I_h, R_h, S_r, E_r, I_r)$  be the solution of (1) with the initial conditions in a biologically feasible region.

$\Gamma = \Gamma_h \times \Gamma_r$  with

$$\Gamma_h = \left\{ S_h, E_h, I_h, R_h \in R_+^4 : N_h \leq \frac{\theta_h}{\mu_h} \right\} \text{ and}$$

$$\Gamma_r = \left\{ S_r, E_r, R_r \in R_+^3 : N_r \leq \frac{\theta_r}{\mu_r} \right\}$$

Then  $\Gamma$  is non-negative invariant.

**Proof.**

Following the approach of [22], we have that,

$$0 \leq N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\theta_h}{\mu_h}(1 - e^{-\mu_h t}), \tag{3}$$

also

$$N_r(t) \leq N_r(0)e^{-\mu_r t} + \frac{\theta_r}{\mu_r}(1 - e^{-\mu_r t}). \tag{4}$$

Hence, the set  $\Gamma$  is positive invariant and for  $t \rightarrow \infty, 0 \leq$

$N_h(t) \leq \frac{\theta_h}{\mu_h}$  and  $N_r(t) \leq \frac{\theta_r}{\mu_r}$ . Therefore,  $\Gamma$  is an attracting set.

**2.2. Lassa fever free equilibrium state**

Setting the right-hand side of (2) to zero i.e.,

$$S'_h = E'_h = I'_h = R'_h = S'_r = E'_r = I'_r = 0$$

Thus, the Lassa free equilibrium state is given as

$$P_0 = (S_h, E_h, I_h, R_h, S_r, E_r, I_r) = \left( \frac{\theta_h}{\mu_h}, 0, 0, 0, \frac{\theta_r}{\mu_r}, 0, 0 \right). \tag{5}$$

**2.3. The basic reproduction number**

The tendency for Lassa fever to spread becomes higher if an individual is infected with Lassa fever in the population. The threshold for disease transmissibility in epidemiology is called the basic reproduction number. This threshold shall be computed for our model to make predictions regarding the outbreak or otherwise of Lassa fever in the population. Following the approach of [25], we obtain,

$$F = \begin{pmatrix} \frac{\beta_2 \theta_h}{\mu_h} & \frac{\beta_3 \theta_h}{\mu_h} & 0 & \frac{\beta_1 \theta_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_4 \theta_r}{\mu_r} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \beta_h + \mu_h & 0 & 0 & 0 \\ -\beta_h & \mu_h + \beta_h + \varphi & 0 & 0 \\ 0 & 0 & \mu_r + \beta_r & 0 \\ 0 & 0 & -\beta_r & \mu_r \end{pmatrix},$$

$$V^{-1} = \begin{pmatrix} \frac{1}{a_1} & 0 & 0 & 0 \\ \frac{\beta_h}{a_1 a_2} & \frac{1}{a_2} & 0 & 0 \\ 0 & 0 & \frac{1}{a_3} & 0 \\ 0 & 0 & \frac{\beta_r}{a_3 \mu_r} & \frac{1}{\mu_r} \end{pmatrix}.$$

where

$$\begin{aligned}
 a_1 &= \beta_h + \mu_h \\
 a_2 &= \mu_h + \beta_h + \varphi \\
 a_3 &= \mu_r + \beta_r
 \end{aligned}$$

Thus, the basic reproduction number is given as,

$$R_0 = \rho FV^{-1} = \frac{\beta_h \theta_h}{\mu_r \mu_h a_1 a_2} + \sqrt{\frac{\beta_1 \beta_2 \beta_3 \theta_h + \beta_r \theta_r \beta_4}{\mu_h a_1 a_2 a_3}}. \tag{6}$$

**2.4. Extension of the model into optimal control**

We further extend the model in (1) by incorporating four control variables namely; fumigating the environment with pesticide, the use of condom, early treatment and the use of indoor residual spray  $u_1(t), u_2(t)$ ,

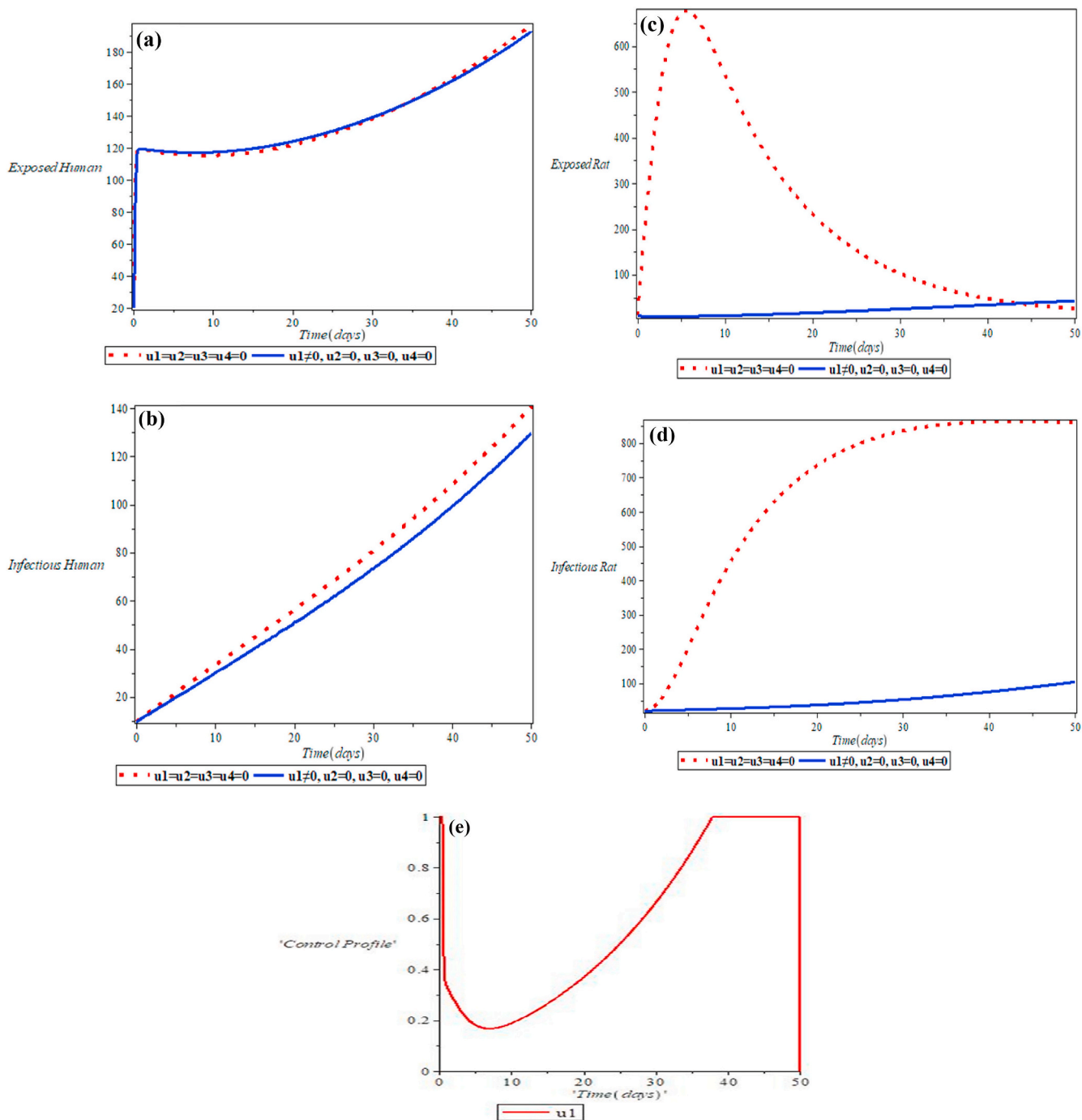


Fig. 2. Simulation showing the effect of fumigating the environment on exposed human, infected human, exposed rat and infected rat population.

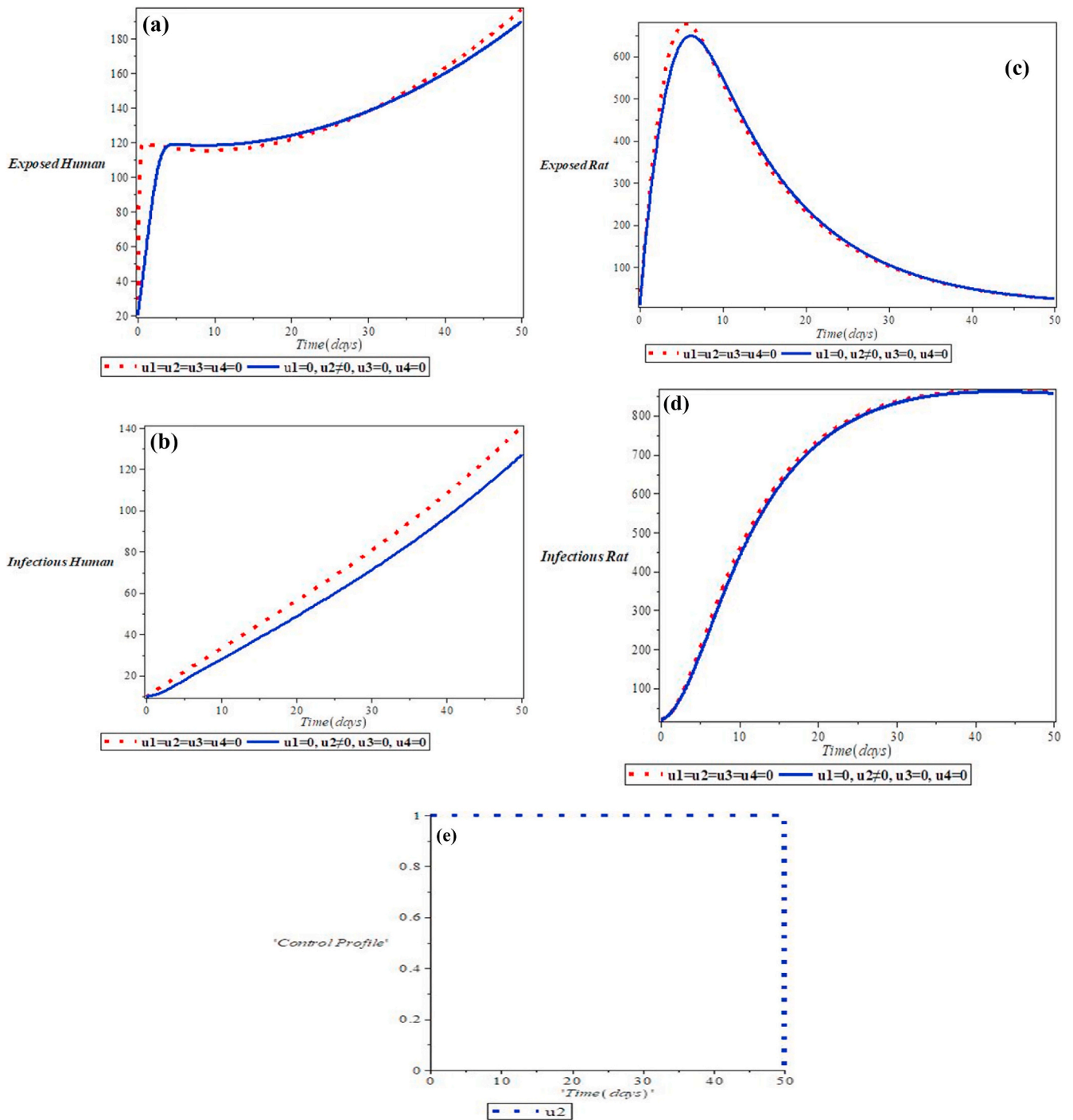


Fig. 3. Effect of the use of condom on exposed human, infected human, exposed rat and infected rat population.

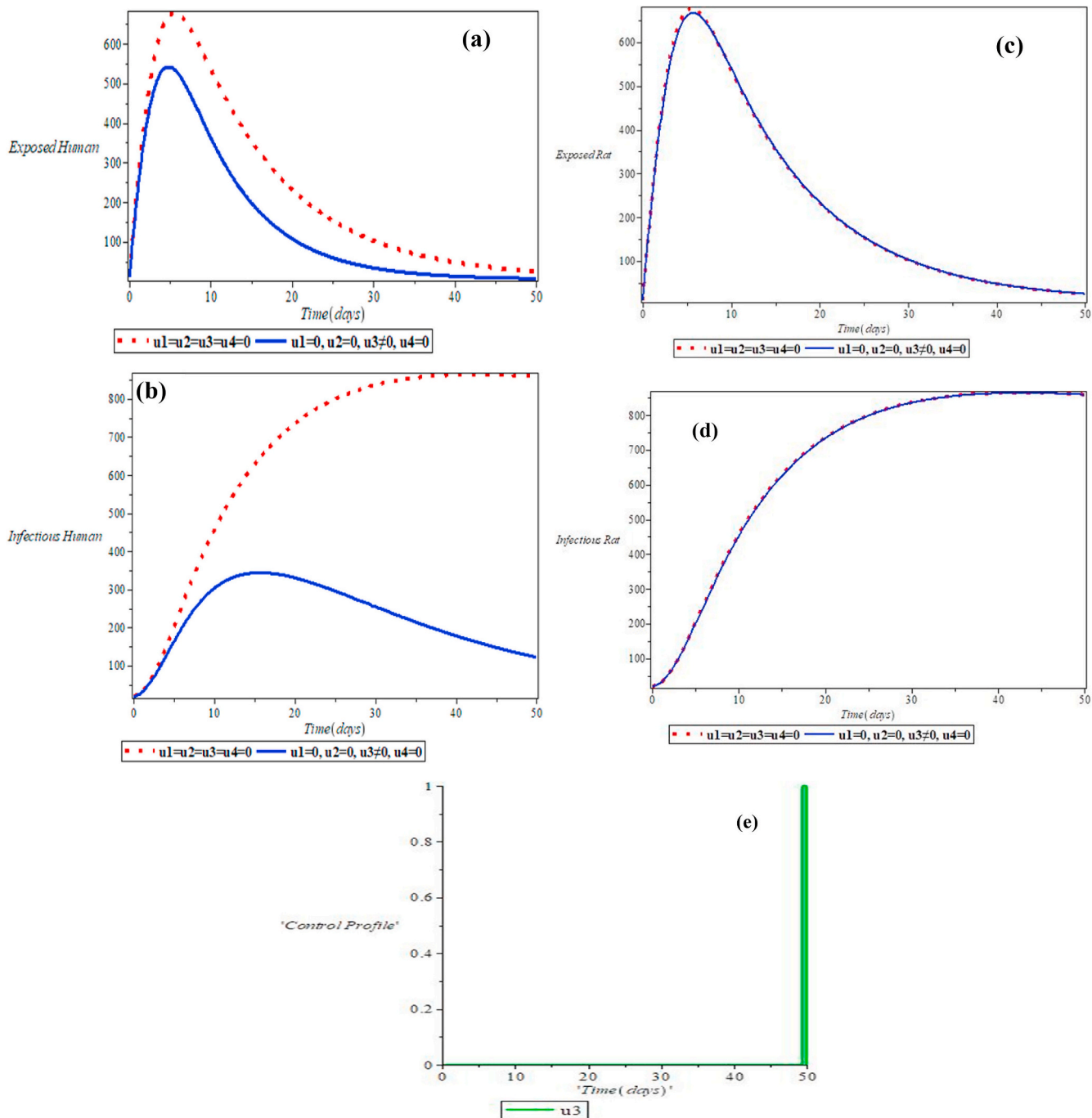


Fig. 4. Effect of early treatment on exposed human, infected human, exposed rat and infected rat population.

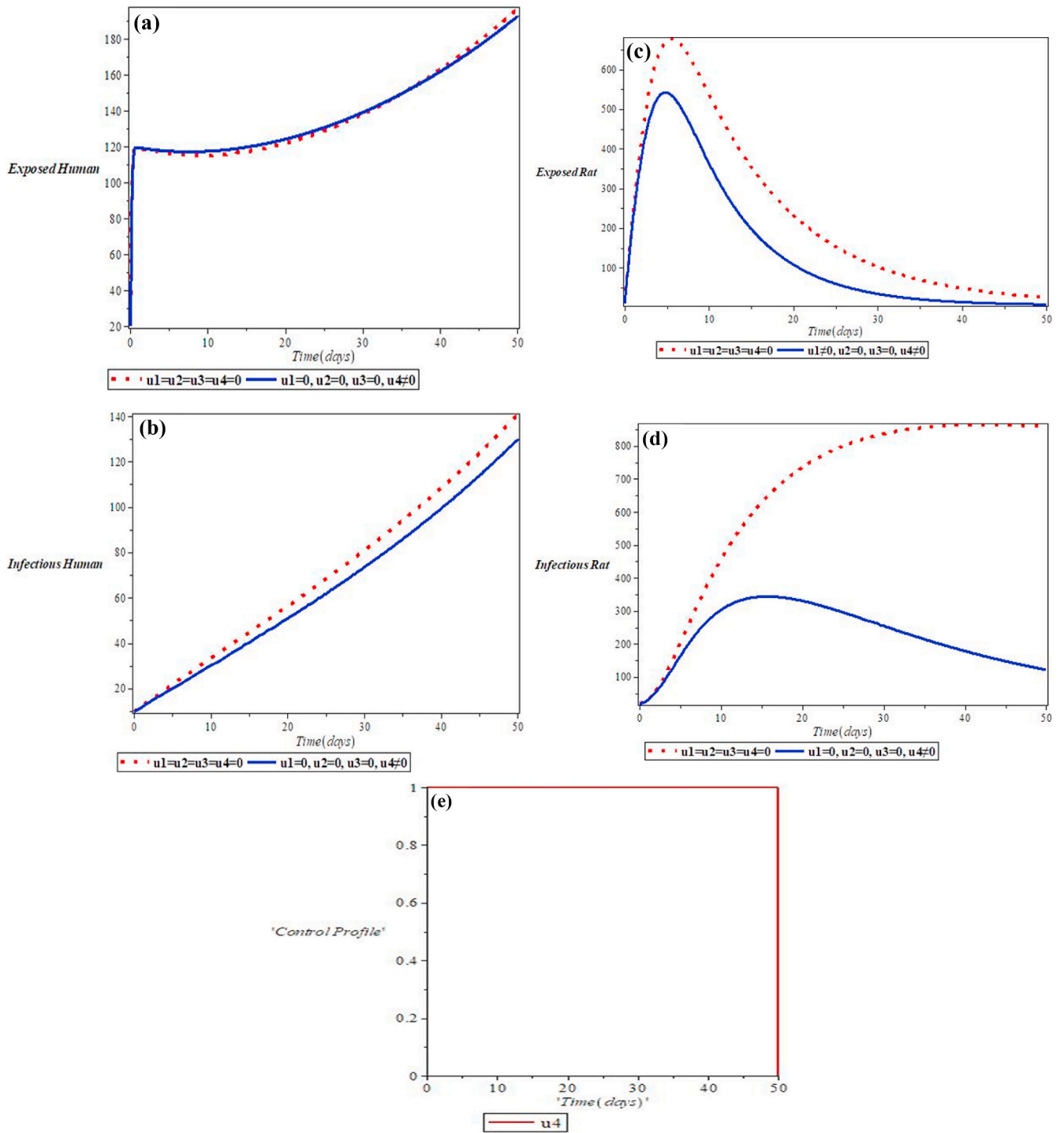


Fig. 5. Effect of the use of indoor residual spray on exposed human, infected human, exposed rat and infected rat population.

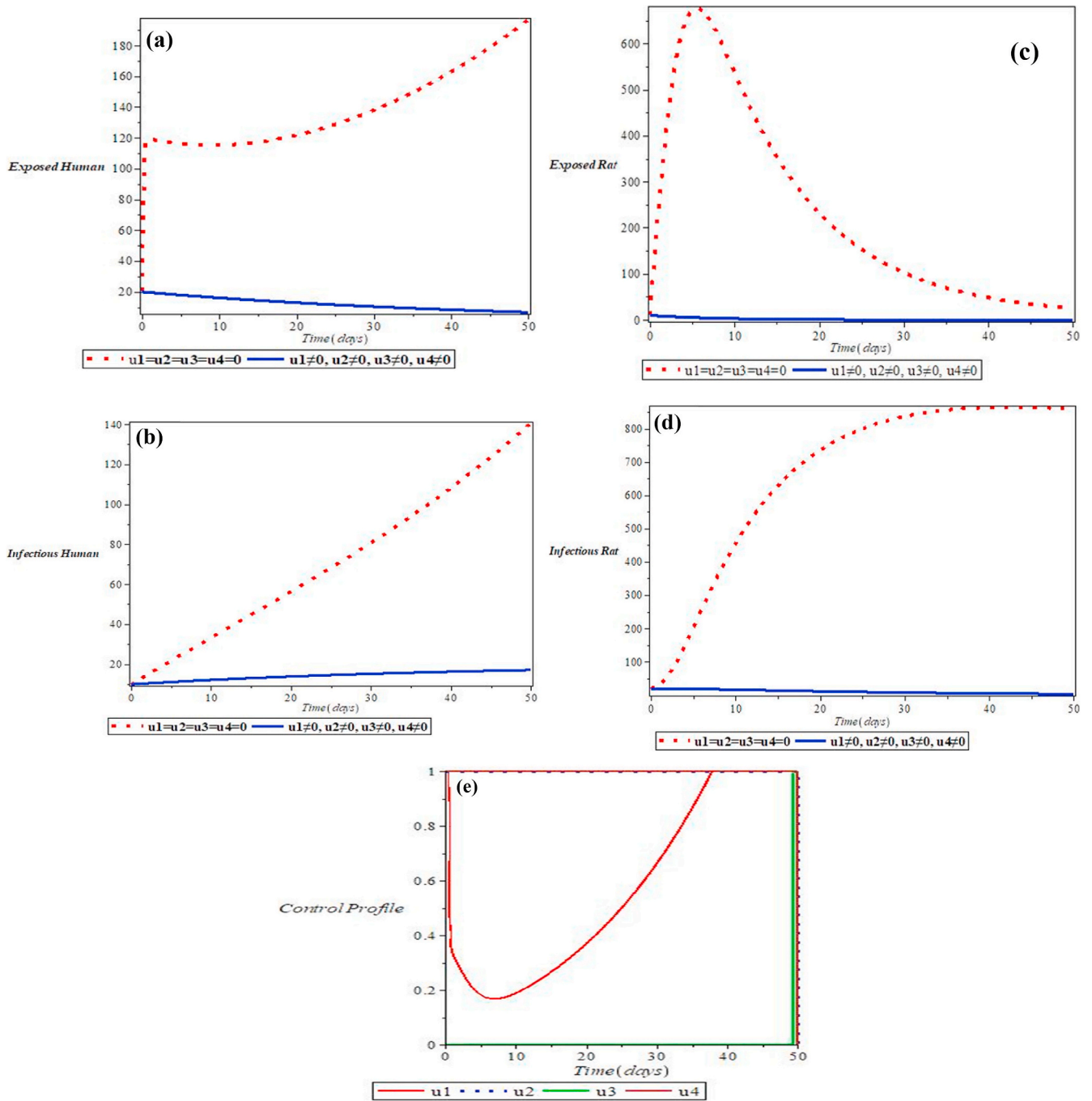


Fig. 6. Effect of the application of all the controls on exposed human, infected human, exposed rat and infected rat population.

$u_3(t)$ ,  $u_4(t)$  respectively. The number of susceptible humans is reduced by

$$\beta_1 S_h I_r (1 - u_1) - \beta_2 S_h E_h (1 - u_2) - \beta_3 S_h I_h (1 - u_2)$$

Due to contact with the infected rat and as a result of sexual contact with asymptomatic and symptomatic infected human. The infected human recover through drug administration at a rate  $\tau u_3$ . The

susceptible rat population is risen by the daily recruitment rate  $\theta_r$  and reduces by

$$\beta_4 I_h S_r (1 - u_1)$$

Upon contact with the infected human.  $\theta u_4(t)$  represent the control measure by indoor residual spray. By incorporating the above description into (2) we obtain the following differential equations



$$\begin{aligned} \frac{dS_h}{dt} &= \theta_h + (\phi + \tau u_3)(1 - \nu)I_h + \varphi_h R_h - \beta_1 S_h I_r(1 - u_1) - \beta_2 S_h E_h(1 - u_2) - \beta_3 S_h I_h(1 - u_2) - \mu_h S_h, \\ \frac{dE_h}{dt} &= \beta_1 S_h I_r(1 - u_1) + \beta_2 S_h E_h(1 - u_2) + \beta_3 S_h I_h(1 - u_2) - (\beta_h + \mu_h)E_h, \\ \frac{dI_h}{dt} &= \beta_h E_h - (\phi + \tau u_3)I_h - (\mu_h + \delta_h)I_h, \\ \frac{dR_h}{dt} &= (\phi + \tau u_3)\nu I_h - (\varphi_h + \mu_h)R_h, \\ \frac{dS_r}{dt} &= \theta_r - \beta_4 I_h S_r(1 - u_1) - \mu_r S_r - \theta u_4 S_r, \\ \frac{dE_r}{dt} &= \beta_4 I_h S_r(1 - u_1) - \beta_r E_r - \mu_r E_r - \theta u_4 E_r, \\ \frac{dI_r}{dt} &= \beta_r E_r - \mu_r I_r - \theta u_4 I_r. \end{aligned}$$

The objective function is define as

$$u_1^* = \max \left\{ 0, \min \left( 1, \frac{\beta_1 S_h I_h (\lambda_2 - \lambda_1) + \beta_4 I_h S_r (\lambda_6 - \lambda_5)}{K_1} \right) \right\},$$

$$J(u_1, u_2, u_3, u_4) = \int_0^t \left( B_1 E_h(t) + B_2 I_h(t) + B_3 N_r(t) + K_1 \frac{u_1^2(t)}{2} + K_2 \frac{u_2^2(t)}{2} + K_3 \frac{u_3^2(t)}{2} + K_4 \frac{u_4^2(t)}{2} \right) dt$$

Hamiltonian,  $H$  is defined as

$$u_2^* = \max \left\{ 0, \min \left( 1, \frac{\beta_2 S_h E_h (\lambda_2 - \lambda_1) + \beta_3 I_h S_h (\lambda_2 - \lambda_1)}{K_2} \right) \right\},$$

$$\begin{aligned} H(u_1, u_2, u_3, u_4) &= \left( B_1 E_h + B_2 I_h + B_3 N_r + K_1 \frac{u_1^2}{2} + K_2 \frac{u_2^2}{2} + K_3 \frac{u_3^2}{2} + K_4 \frac{u_4^2}{2} \right) + \lambda_1 (\theta_h + \tau u_3(1 - \nu)I_h + \varphi_h R_h - \beta_1 S_h I_r(1 - u_1) - \beta_2 S_h E_h(1 - u_2) - \beta_3 S_h I_h(1 - u_2) \\ &- \mu_h S_h) + \lambda_2 (\beta_1 S_h I_r(1 - u_1) + \beta_2 S_h E_h(1 - u_2) + \beta_3 S_h I_h(1 - u_2) - \beta_h E_h - \mu_h E_h) + \lambda_3 (\beta_h E_h - (\phi + \tau u_3)I_h - \mu_h I_h - \delta_h I_h) + \lambda_4 (\tau \nu u_3 I_h - (\varphi_h + \mu_h)R_h) \\ &+ \lambda_5 (\theta_r - \beta_4 I_h S_r(1 - u_1) - \mu_r S_r - \theta u_4 S_r) + \lambda_6 (\beta_4 I_h S_r(1 - u_1) - \beta_r E_r - \mu_r E_r - \theta u_4 E_r) + \lambda_7 (\beta_r E_r - \mu_r I_r - \theta u_4 I_r) \end{aligned}$$

**Theorem 2.**

There exists an optimal control with the corresponding solution  $(S_h, E_h, I_h, R_h, S_r, E_r, I_r)$  corresponding to the state equations in (2) and the adjoint variables  $\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t), \lambda_6(t), \lambda_7(t)$  such that,

$$\begin{aligned} -\dot{\lambda}_1 &= -[\beta_1 I_r(1 - u_1) + \beta_2 E_h(1 - u_2) + \beta_3 I_h(1 - u_2)](\lambda_1 - \lambda_2) + \lambda_1 \mu_h, \\ -\dot{\lambda}_2 &= \beta_2 S_h(1 - u_2)(\lambda_1 - \lambda_2) + \beta_h(\lambda_1 - \lambda_2) + \mu_h \lambda_2 - B_1, \\ -\dot{\lambda}_3 &= (\phi + \tau u_3)[(1 - \nu)\lambda_1 + \nu(\lambda_3 - \lambda_4)] + \beta_3 S_h(1 - u_2)(\lambda_1 - \lambda_2) + (\mu_h + \delta_h)\lambda_3 - B_2, \\ -\dot{\lambda}_4 &= \varphi_h(\lambda_1 - \lambda_4) + \mu_h \lambda_4, \\ -\dot{\lambda}_5 &= \beta_4 I_h(1 - u_1)(\lambda_5 - \lambda_6) + (\mu_r + \theta u_4)\lambda_5 - B_3, \\ -\dot{\lambda}_6 &= \beta_r(\lambda_6 - \lambda_7) + (\mu_r + \theta u_4)\lambda_6 - B_3, \\ -\dot{\lambda}_7 &= \beta_1 S_h(1 - u_1)(\lambda_1 - \lambda_2) + (\mu_r + \theta u_4)\lambda_7 - B_3 \end{aligned}$$

Such that,

$$u_3^* = \max \left\{ 0, \min \left( 1, \frac{\nu(\phi + \tau)(\lambda_3 - \lambda_4)I_h + (\nu - 1)(\phi + \tau)I_h \lambda_1}{K_3} \right) \right\},$$

$$u_4^* = \max \left\{ 0, \min \left( 1, \frac{\theta(S_r \lambda_5 + E_r \lambda_6 - I_r \lambda_7)}{K_4} \right) \right\},$$

**3. Results**

We perform the numerical simulations by using the values in the Table 2. We solve the optimal control system which is made up of the state and adjoint equations. The optimality system is solved numerically via forward-backwards sweep method. We also use the following values for the weight factors.

$$B_1 = 1, B_2 = 1.5, B_3 = 1.5, K_1 = 0.2, K_2 = 0.2, K_3 = 0.15, K_4 = 0.5$$

To examine the effect of the control interventions, we considered the

following strategies to see the effect of the best control strategies on exposed rat, infected rat, exposed human and infected human population.

1. Fumigating the environment with pesticide  $u_1$
2. The use of condom  $u_2$
3. Early treatment as control intervention  $u_3$
4. The use of indoor residual spray  $u_4$
5. Applying all the control strategies that is, fumigating the environment with pesticide, use of condom, early treatment and the use of indoor residual spray  $u_1(t)$ ,  $u_2(t)$ ,  $u_3(t)$ ,  $u_4(t)$  respectively.

#### 4. Discussion of results

##### 4.1. Effects of fumigating the environment with pesticide $u_1$

We use fumigation of the environment with pesticide as the only control strategy ( $u_1 \neq 0, u_2, u_3, u_4 = 0$ ) to minimise the objective functional  $J$ , while the other controls  $u_2, u_3$  and  $u_4$  are set to zero. In Fig. 2(a)-(e). It is observed that the strategy does not have any effect on the exposed and infected humans but has a greater impact on the exposed and infected rats.

##### 4.2. Effects of condom usage $u_2$

We apply the use of condom as the only control strategy that is, ( $u_2 \neq 0, u_1, u_3, u_4 = 0$ ) to minimise the spread of Lassa fever, while other controls  $u_1, u_3$  and  $u_4$  are made to be zero. In Fig. 3, we observe that the strategy does not have any effect on the exposed and infected rats but has a significant effect on the exposed and infected humans as the use of condom can prevent human to human transmission during sexual activities. The number of individuals in the exposed and infected classes reduces considerably in Fig. 3 (a)–(e).

##### 4.3. Effects of early treatment as control intervention $u_3$

We apply early treatment as the only control strategy ( $u_3 \neq 0, u_1, u_2, u_4 = 0$ ) to check the transmission of Lassa fever, while the other interventions  $u_1, u_2$  and  $u_4$  are set to zero. In Fig. 4 (a)–(e). It is observed that the strategy does not influence the exposed and the infected rat populations but has a significant effect on the exposed and infected humans as their populations decrease significantly.

##### 4.4. Effects of the use of indoor residual spray $u_4$

We apply indoor residual spray as the only control strategy ( $u_4 \neq 0, u_1, u_2, u_3 = 0$ ) to curtail the Lassa fever spread, while other controls  $u_1, u_2$  and  $u_3$  are set to zero in Fig. 5 (a)–(e). The strategy does not have any effect on humans but has a significant effect on the exposed and infected rats. The indoor residual spray controls the rats but does not have a side effect on humans.

##### 4.5. The combination of all four controls

Fumigation of the environment with pesticide, use of condom, early treatment and use of indoor residual spray  $u_1(t)$ ,  $u_2(t)$ ,  $u_3(t)$ ,  $u_4(t)$  respectively. In Fig. 6 (a)–(e). It is clearly shown that the application of all the four controls yields better results i.e. there is a drastic decrease in the number of exposed humans, infected humans, exposed rats and infected rats. Therefore, Lassa fever can be eradicated in any given population with time if all the suggested controls are adequately applied.

#### 5. Conclusion

In this study, a model for the dynamics of Lassa fever was proposed.

The solutions of the model were first of all tested for the basic properties of positivity and boundedness and were found to be positive and bounded at all time. A qualitative analysis was then carried out by deriving the equilibria points, computing the reproduction number and investigating the existence and stability of equilibria. By using the Pontryagin's maximum principle, we formulated the optimal control problem by analysing the conditions for the optimal control of the disease spread. The optimized system was solved numerically and the numerical simulations were carried out to illustrate the analytical results. The numerical simulation results showed that optimal level was attained in the control and containment of Lassa fever when fumigation of the environment with pesticide, use of condom, early treatment and use of indoor residual spray were combined. Therefore, the fight against the spread of Lassa fever disease requires a multifaceted approach. We have not considered the stability aspect in this work and other control measures like personal hygiene, this gives space for future research. The findings obtained in this work can be a valuable reference for the Lassa fever Local National Control Program and the basis for the preparation and design of the best intervention strategies to eradicate Lassa fever disease.

#### Author contribution statement

Olumuyiwa J. Peter<sup>a</sup>, conceived the idea, Olumuyiwa J. Peter<sup>a</sup>, Adesoye I. Abioye<sup>a</sup> and Festus A. Oguntolu<sup>b</sup>, did the writing-original draft, Abdullaziz G. Zakari<sup>e</sup>, Titilayo A. Owolabi<sup>c</sup>, and Timilehin Gideon Shaba<sup>a</sup> Review & Editing, Olumuyiwa J. Peter<sup>a</sup> formulated the model, Olumuyiwa J. Peter<sup>a</sup>, Adesoye I. Abioye<sup>a</sup> and Titilayo A. Owolabi<sup>c</sup> wrote the coding aspect.

#### Ethical statement

We wish to confirm that we have adhered to the ethics for publication according to the requirements of Elsevier and COPE.

#### Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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