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Effect of Rainfall on the Transmission Model of Leptospirosis

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ABSTRACT

In this paper, a dynamical model that describes the transmission of leptospirosis is proposed and analyzed. The human population is divided into three compartments depending on disease status, susceptible, infected and recovered compartment. The rat population is divided into two compartments, susceptible and infected compartment. We take into account the effect of rainfall in both dry and rainy seasons for our model. The stability theory of differential equations was determined by using the Routh – Hurwitz criteria. The stability conditions both disease free equilibrium and endemic equilibrium are found and their stabilities are investigated, which depended on the basic reproductive number. If $\Re_0 < 1$, the disease free equilibrium point is local asymptotically stable that mean, the disease will die out. But if $\Re_0 > 1$, there exist the endemic equilibrium, which is local asymptotically stable that there will be leptospirosis outbreak. The numerical simulations are presented to support the analytic results of the model.

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INTRODUCTION

Leptospirosis is a health problem in tropical and subtropical regions. Leptospirosis is an infectious disease caused by pathogenic bacteria called *Leptospira*, that are transmitted directly or indirectly from animals to humans by contact with the urine or blood of infected animals or by contact with a *Leptospira*-contaminated environment. *Leptospira* come into body through cuts or abrasions of skin, or mucous membranes (nose, mouth, eyes) and sometimes through waterlogged skin via the moist soil or water which is contaminated with the waste products of infected animals. The population at risk is the people who have the occupation which contact animals, soil and water, such as: mine workers, sewer workers, fish workers, veterinarians, slaughterhouse workers, agriculture workers and the disease has also been associated with the people who swimming, wading, kayaking, and rafting in contaminated lakes and rivers. In humans, leptospirosis can cause a wide range of symptoms, including: high fever, headache, chills, muscle aches vomiting, jaundice (yellow skin and eyes), red eyes, abdominal pain, diarrhea, rash. Symptom of leptospirosis may last from a few days to 3 weeks or longer (CDC,2014). Lau C.L. *et al*. (2010) said that heavy rainfall flooding and also natural disasters increase the risk of leptospirosis by bringing bacteria and their animal hosts into closer contact with humans.

Many model which consist the system of differential equations have been proposed to represent the dynamics of both human and vector population. In 2010, Zaman G. considered the real data to studied the dynamical behavior and role of optimal control theory of leptospirosis disease. In 2012, Zaman G. *et al*. presented the dynamical interaction including local and global stability of leptospirosis infected vector and human population. Khan M.A. *et al*. (2014) presented the optimal control problem applied to a dynamical leptospirosis infected vector and human population by using multiple control variables. Jantraporn Suksawat and Surapol Naowarat. (2014) proposed and analyzed SIR model for Conjunctivitis which take into account the effect of rainfall on hot and cool season. In this paper, we consider effect of rainfall on the dynamical transmission model of leptospirosis. In section 2, we formulate the propose model. In section 3, we analyze the model by stability theory of differential equations, to determine disease free and endemic equilibrium, derive the basic reproductive number and investigate the stability of the model. In section 4, we present the numerical simulations to support the

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analytic results. Finally, we conclude our study in section 5.

Model Formulation:

In this study, we formulated the mathematical model of leptospirosis with SIR (Susceptible-Infected-Recovered) model for human population and SI (Susceptible-Infected) model for vector (rat) population. We assume that $S_h(t)$ represents number of susceptible human at time t; $I_h(t)$ represents number of infected human at time t; $R_h(t)$ represents number of recovered human at time t. For rat population, $S_v(t)$ represents number of susceptible rat at time t; $I_v(t)$ represents number of infected rat at time t. The total population of human and rat are constant denoted by $N_h(t)$ and $N_v(t)$. Thus, $N_h(t) = S_h(t) + I_h(t) + R_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$. The diagram which showed the interaction of both human population and rat population are presented in Fig.1.

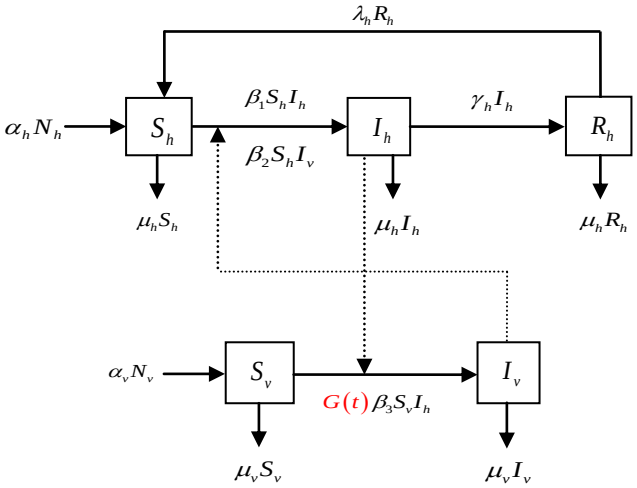

Fig.1: The diagram for the dynamical transmission model of leptospirosis.

The definitions of variables and parameters are given as follows:

α_h is the birth rate of human population,

α_v is the birth rate of rat population,

μ_h is the natural mortality rate of human population,

μ_v is the natural mortality rate of rat population,

β_1 is the rate of direct transmission from infected human,

β_2 is the rate of transmission from infected rat,

β_3 is the rate of the disease carrying of susceptible rat per host per unit time,

λ_h is the rate of the immune human become susceptible again,

γ_h is the recovered rate for human from the infections, $G(t)$ is the rainfall in the particular season.

The dynamical transmission model of leptospirosis are described by the follow system of five non-linear differential equations as follows:

$$\frac{dS_h}{dt} = \alpha_h N_h - \mu_h S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \lambda_h R_h \quad (1)$$

$$\frac{dI_h}{dt} = \beta_2 S_h I_v + \beta_1 S_h I_h - \mu_h I_h - \gamma_h I_h \quad (2)$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h - \lambda_h R_h \quad (3)$$

$$\frac{dS_v}{dt} = \alpha_v N_v - \mu_v S_v - G(t)\beta_3 S_v I_h \quad (4)$$

$$\frac{dI_v}{dt} = G(t)\beta_3 S_v I_h - \mu_v I_v \quad (5)$$

Model Analysis:

Equilibrium Points:

We first determine the equilibrium points. The system has two equilibrium points; disease free equilibrium points and endemic equilibrium points. By setting the right hand side of equations (1) – (5) to zero. We found two equilibrium points.

1. Disease Free Equilibrium Points (E_0):

In the case of disease free and we take into account dry season, that is $I_h = 0$, $I_v = 0$. Thus, we found $E_0(S_h,I_h,R_h,S_v,I_v) = (N_h,0,0,N_v,0)$.

2. Endemic Equilibrium Points (E_1):

In the case the disease is presented and we take into account rainy season, that is $I_h \neq 0$, $I_v \neq 0$. We obtained

$$S_h^* = \frac{\left(\lambda_h\gamma_h I_h^* + \alpha_h N_h(\mu_h+\lambda_h)\right)\left(\mu_v(\mu_v+\beta_3 G(t)I_h^*)\right)}{(\mu_h+\lambda_h)\left(\mu_h\mu_v(\mu_v+\beta_3 G(t)I_h^*)+\beta_2\alpha_v N_v\beta_3 G(t)I_h^*+\beta_1\mu_v I_h^*(\mu_v+\beta_3 G(t)I_h^*)\right)} \quad (6)$$

$$I_h^* = \frac{-A_2+\sqrt{A_2^2-4A_1A_3}}{2A_1} \quad (7)$$

$$R_h^* = \frac{\gamma_h I_h^*}{\mu_h+\lambda_h} \quad (8)$$

$$S_v^* = \frac{\alpha_v N_v}{\mu_v+G(t)\beta_3 I_h^*} \quad (9)$$

$$I_v^* = \frac{\alpha_v N_v\beta_3 G(t)I_h^*}{\mu_v\left(\mu_v+\beta_3 G(t)I_h^*\right)} \quad (10)$$

Where:

$A_1 = \lambda_h\gamma_h G(t)\beta_1\beta_3\mu_v - G(t)\beta_1\beta_3\mu_h\mu_h\mu_v - G(t)\beta_1\beta_3\mu_v\gamma_h\mu_h - G(t)\beta_1\beta_3\mu_v\mu_h\lambda_h - G(t)\beta_1\beta_3\mu_v\gamma_h\lambda_h,$

$A_2 = \lambda_h\gamma_h\mu_v\mu_v\beta_1 + \alpha_h N_h\mu_h G(t)\beta_1\beta_3\mu_v + \alpha_h N_h\lambda_h G(t)\beta_1\beta_3\mu_v + G(t)\beta_2\beta_3\alpha_v N_v\lambda_h\gamma_h - \mu_h\mu_v\mu_h\mu_h G(t)\beta_3 - \mu_h\mu_h G(t)\beta_2\beta_3\alpha_v N_v$
$- \beta_1\mu_v\mu_v\mu_h\mu_h - \mu_h\mu_v\gamma_h\mu_h G(t)\beta_3 - G(t)\beta_2\beta_3\alpha_v N_v\gamma_h\mu_h - \beta_1\mu_v\mu_v\gamma_h\mu_h - \mu_h\mu_v\mu_h\lambda_h G(t)\beta_3 - G(t)\beta_2\beta_3\alpha_v N_v\mu_h\lambda_h$
$- \beta_1\mu_v\mu_v\mu_h\lambda_h - \mu_h\mu_v G(t)\beta_3\gamma_h\lambda_h - G(t)\beta_2\beta_3\alpha_v N_v\gamma_h\lambda_h - \beta_1\mu_v\mu_v\gamma_h\lambda_h,$

$A_3 = \alpha_h N_h\mu_h\mu_v\mu_v\beta_1 + \alpha_h N_h\lambda_h\mu_v\mu_v\beta_1 + \beta_2 G(t)\beta_3\alpha_v N_v\mu_h\alpha_h N_h + \beta_2 G(t)\beta_3\alpha_v N_v\lambda_h\alpha_h N_h - \mu_h\mu_v\mu_v\mu_h\mu_h - \mu_h\mu_v\mu_v\gamma_h\mu_h$
$- \mu_h\mu_v\mu_v\mu_h\lambda_h - \mu_h\mu_v\mu_v\gamma_h\lambda_h.$

Thus the endemic equilibrium points become $E_1\left(S_h^*, I_h^*, R_h^*, S_v^*, I_v^*\right)$.

Basic Reproductive Number ($\Re_0$):

The basic reproductive number is obtained by the characteristic equation at the disease free state $E_0(S_h,I_h,R_h,S_v,I_v) = (N_h,0,0,N_v,0)$. From the characteristic equation we considered the quadratic equation $\lambda^2 + B_1\lambda + B_2$.

Where:

$B_1 = \mu_v + \mu_h + \gamma_h - \beta_1 N_h,$

$B_2 = \left(\mu_h\mu_v + \gamma_h\mu_v - \beta_1 N_h\mu_v - G(t)\beta_2\beta_3 N_h N_v\right).$

For the conditions of the Routh-Hurwitz criteria (Allen, 2006) which the values of $B_1 > 0$ and $B_2 > 0$ that mean:

$\mu_v + \mu_h + \gamma_h - \beta_1 N_h > 0$

$\mu_h\mu_v + \gamma_h\mu_v - \beta_1 N_h\mu_v - G(t)\beta_2\beta_3 N_h N_v > 0$

Thus,

$\mu_v(\mu_h + \gamma_h - \beta_1 N_h) > G(t)\beta_2\beta_3 N_h N_v$

$1 > \frac{G(t)\beta_2\beta_3 N_h N_v}{\mu_v(\mu_h+\gamma_h-\beta_1 N_h)}$

$1 > R_0$

We obtaind the basic reproductive number as shown,

$\Re_0 = \sqrt{R_0},$

$\Re_0 = \sqrt{\frac{G(t)\beta_2\beta_3 N_h N_v}{\mu_v(\mu_h+\gamma_h-\beta_1 N_h)}}.$

Stability Analysis:

In this section, The stability analysis of an equilibrium point; disease free equilibrium points and endemic equilibrium points is determinded from the Jacobian matrix of the system.

For the disease free state (E_0):

The Jacobian matrix at E_0 is given by

$$J_0 = \begin{bmatrix} -\mu_h & -\beta_1 N_h & \lambda_h & 0 & -\beta_2 N_h \\ 0 & \beta_1 N_h - \mu_h - \gamma_h & 0 & 0 & \beta_2 N_h \\ 0 & \gamma_h & -\mu_h - \lambda_h & 0 & 0 \\ 0 & -G(t)\beta_3 N_v & 0 & -\mu_v & 0 \\ 0 & G(t)\beta_3 N_v & 0 & 0 & -\mu_v \end{bmatrix} \quad (11)$$

The eigenvalues of Jacobian matrix J_0 at the disease free state E_0 are obtained by solving $det\left(J_0 - \lambda I\right) = 0$ and the characteristic equation that we obtained is given below:

$$\left(\lambda+\mu_h\right)\left(\lambda+\mu_v\right)\left(\lambda+\mu_h+\lambda_h\right)\left(\lambda^2+B_1\lambda+B_2\right) = 0 \quad (12)$$

Where:

$B_1 = \mu_v + \mu_h + \gamma_h - \beta_1 N_h,$

$B_2 = \left(\mu_h\mu_v + \gamma_h\mu_v - \beta_1 N_h\mu_v - G(t)\beta_2\beta_3 N_h N_v\right).$

From the characteristic equation, we can see that $\lambda_1 = -\mu_h < 0, \lambda_2 = -\mu_v < 0, \lambda_3 = -\left(\mu_h + \lambda_h\right) < 0$.

The other two are the solution of quadratic equation $\lambda^2 + B_1\lambda + B_2$. The two eigenvalues of $\lambda^2 + B_1\lambda + B_2$ will have negative real parts if they satisfy the Routh-Hurwitz criteria. Thus, the disease free state (E_0) is locally asymptotical stable for $\Re_0 < 1$ where $\lambda^2 + B_1\lambda + B_2$ satisfies the following two conditions below:

1. $B_1 > 0,$
2. $B_2 > 0.$

For the disease endemic state (E_1):

In the same manner for the disease endemic state, the Jacobian matrix at E_1 is given by

$$J_1 = \begin{bmatrix} -\mu_h - \beta_2 I_v^* - \beta_1 I_h^* & -\beta_1 S_h^* & \lambda_h & 0 & -\beta_2 S_h^* \\ \beta_2 I_v^* + \beta_1 I_h^* & \beta_1 S_h^* - \mu_h - \gamma_h & 0 & 0 & \beta_2 S_h^* \\ 0 & \gamma_h & -\mu_h - \lambda_h & 0 & 0 \\ 0 & -G(t)\beta_3 S_v^* & 0 & -\mu_v - G(t)\beta_3 I_h^* & 0 \\ 0 & G(t)\beta_3 S_v^* & 0 & G(t)\beta_3 I_h^* & -\mu_v \end{bmatrix} \quad (13)$$

Where $S_h^*, I_h^*, R_h^*, S_v^*, I_v^*$ are given by equation (6) – (9) and the characteristic equation of Jacobian matrix at E_1, becomes equation (14):

$$\lambda^5 + C_1\lambda^4 + C_2\lambda^3 + C_3\lambda^2 + C_4\lambda + C_5 = 0. \quad (14)$$

Where:

$C_1 = \mu_v - K - N - J - M$,

$C_2 = JN + MN - \mu_v N + KN + MJ - \mu_v J + JK - \mu_v M + MK - \mu_v K - ed + c(a+b)$,

$C_3 = edf - MJN + \mu_v JN - JKN + \mu_v MN - MKN + \mu_v KN + edN + \mu_v MJ - MJK + \mu_v KJ + edJ + \mu_v KM + edM - c(a+b)J + c(a+b)\mu_v + ed(a+b) - (a+b)\lambda_h - c(a+b)N$,

$C_4 = -edfJ + edfM - MJN\mu_v + MJKN - KJN\mu_v - edJN - KMN\mu_v - edMN - KMJ\mu_v - edMJ - (a+b)c\mu_v J + (a+b)cJN - (a+b)edJ - (a+b)cN\mu_v - (a+b)edf - (a+b)edN - (a+b)\lambda_h\mu_v + (a+b)\lambda_h N$,

$C_5 = KMJN\mu_v + edMJN + edfJM + c(a+b)JN\mu_v + edfJ(a+b) + edJN(a+b) + (a+b)\lambda_h\gamma_h\mu_v N$,

$a = \beta_2 I_v^*$, $b = \beta_1 I_h^*$, $c = \beta_1 S_h^*$, $d = \beta_2 S_h^*$, $e = G(t)\beta_3 S_v^*$, $f = G(t)\beta_3 I_h^*$,

$M = -\mu_h - a - b$, $K = c - \mu_h - \gamma_h$, $J = -\mu_h - d - \lambda_h$, $N = -\mu_v - f$.

The five eigenvalues of $\lambda^5 + C_1\lambda^4 + C_2\lambda^3 + C_3\lambda^2 + C_4\lambda + C_5 = 0$ will have negative real parts if they satisfy the Routh-Hurwitz criteria. Thus, the disease endemic state (E_1) is locally asymptotical stable for $\Re_0 > 1$ when $\lambda^5 + C_1\lambda^4 + C_2\lambda^3 + C_3\lambda^2 + C_4\lambda + C_5 = 0$ satisfies the following conditions:

1. $C_1 > 0, C_2 > 0, C_3 > 0, C_4 > 0, C_5 > 0$,
2. $C_1C_2C_3 > C_3^2 + C_1^2C_4$,
3. $(C_1C_4 - C_5)(C_1C_2C_3 - C_3^2 - C_1^2C_4) > C_5(C_1C_2 - C_3)^2 + C_1C_5^2$.

Numerical Results:

In this section, we present the numerical simulation of the proposed model. The parameter values that we used in the numerical simulations are given in Table 1.

Stability of disease free state:

From the values of parameters in Table 1, we obtained the eigenvalues and basic reproductive number are: $\lambda_1 = -0.00004215, \lambda_2 = -0.00182648, \lambda_3 = -0.00281993, \lambda_4 = -0.026712, \lambda_5 = -0.00182327, \Re_0 = 5.849 \times 10^{-7}$ Since all of eigenvalues are to be negative and the basic reproductive number is to be less than one, the disease free equilibrium will be local asymptotically stable, as is demonstrated in Fig. 2.

Table 1: Parameter values used in the numerical simulations at disease free state.

Notation	Parameter description	Value
N_h	Total number of human	1,000
N_v	Total number of rat	500
α_h	Birth rate of human population	4.21×10^{-5} day^{-1}
α_v	Birth rate of rat population	1.83×10^{-3} day^{-1}
μ_h	Natural mortality rate of human population	4.21×10^{-5} day^{-1}
μ_v	Natural mortality rate of rat population	1.83×10^{-3} day^{-1}
β_1	Rate of direct transmission from infected human	4×10^{-5}
β_2	Rate of transmission from infected rat	4×10^{-5}
β_3	Rate of the disease carrying of susceptible rat per host per unit time	4×10^{-5}

λ_h	Rate of the immune human become susceptible again	1/360 day^{-1}
γ_h	Recovered rate for human from the infections	1/15 day^{-1}
$G(t)$	Rainfall in dry season	0.0001

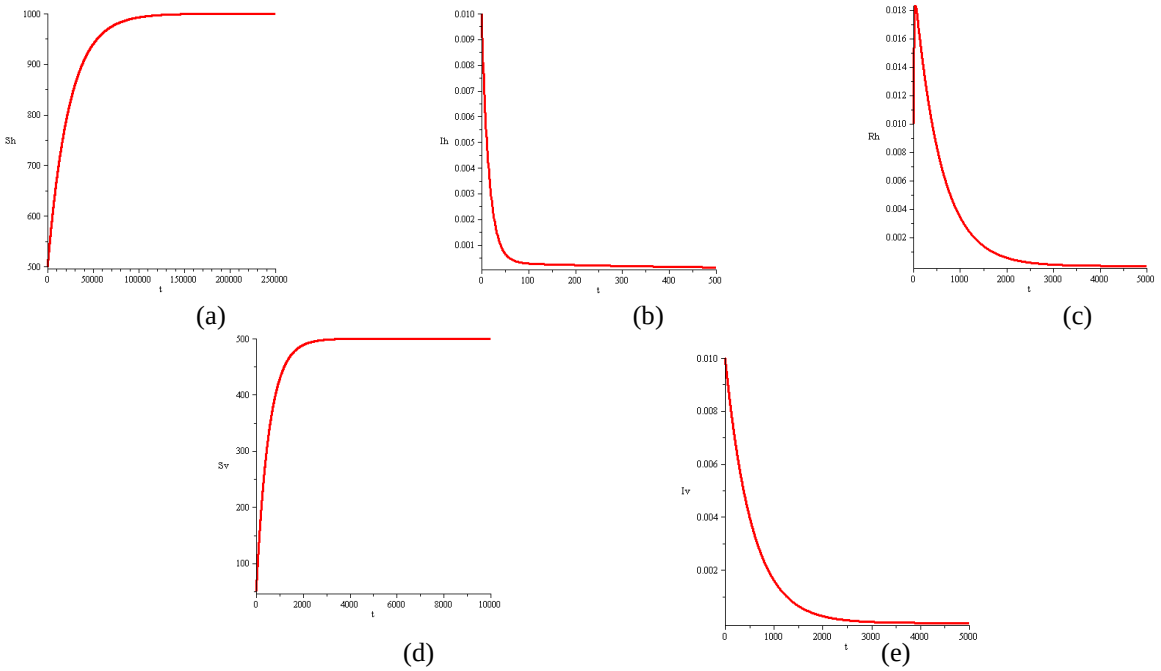

Fig. 2: Time series of (a) Susceptible human (S_h), (b) infected human (I_h), (c) recovered human (R_h), (d) Susceptible rat (S_v) and (e) infected rat (I_v). We see fractions of populations approach to the disease free state $E_0(1000,0,0,500,0)$.

Stability of disease endemic state:

We change the value of the rainfall in rainy season ; $G(t)=0.60$ and keep the other values of parameter to be those given in Table 1. We obtained the eigenvalues and basic reproductive number are: $\lambda_1=-0.03946254, \lambda_2=-0.00281992, \lambda_3=-0.00182648, \lambda_4=-0.00004214, \lambda_5=-0.01092724, \Re_0=4.9197$

Since all of eigenvalues are to be negative and the basic reproductive number is to be greater than one, the endemic equilibrium will be local asymptotically stable, as is demonstrated in Fig. 3.

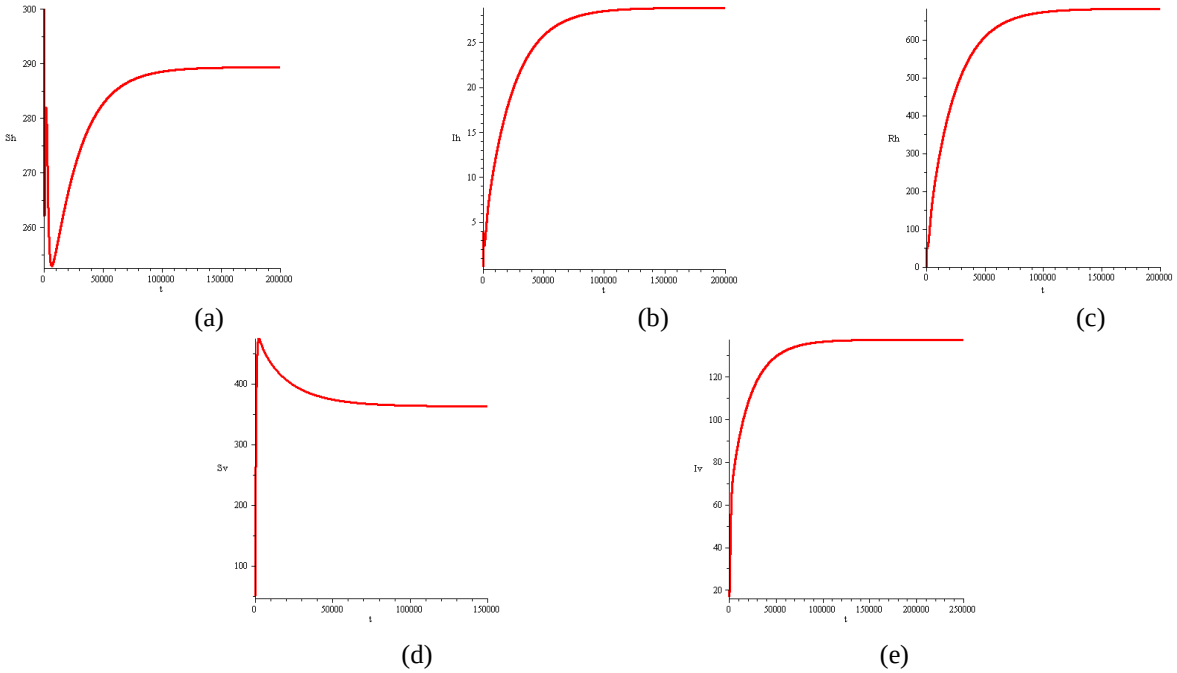

Fig. 3: Time series of (a) Susceptible human (S_h), (b) infected human (I_h), (c) recovered human (R_h),

(d) Susceptible rat (S_v) and (e) infected rat (I_v). We see fractions of populations approach to disease endemic state E_1(289.35,28.76,679.89,362.95,137.05).

Conclusion:

From the model, the basic reproductive number is $\Re_0 = \sqrt{\frac{G(t)\beta_2\beta_3 N_h N_v}{\mu_v(\mu_h+\gamma_h-\beta_1 N_h)}}$. The basic reproductive number is the threshold condition for investigating the stability of the solutions of model (Anderson R.M. and May R.M., 1991). If $R_0 < 1$, the disease free equilibrium point is local asymptotically stable as shown in Fig. 2, that is disease will die out. If $R_0 > 1$, the endemic equilibrium point is local asymptotically stable as shown in Fig. 3, that is there will be leptospirosis outbreak. Our simulated results showed that $\Re_0$ will increase when the rainfall increase. We found that the values of $\Re_0$ were 5.849×10^{-7}, 4.9197 when G(t) = 0.0001, 0.60, respectively. It seen that the infected human will decrease when the rainfall decreases. Thus, the susceptible human have less change of being infected with *Leptospira* when they do not come in contact with contaminated water.

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