International Journal of Mathematics and
Computer Science, **16**(2021), no. 3, 949–961

Analytical and Numerical Solutions of Leptospirosis Model

Abhineshwary Bhalraj, Amirah Azmi, Mohd Hafiz Mohd

School of Mathematical Sciences
Universiti Sains Malaysia
11800 USM, Penang, Malaysia

email: abhineshwary695@yahoo.com, amirahazmi@usm.my,
mohdhafizmohd@usm.my

(Received February 14, 2021, Revised March 4, 2021,
Accepted May 13, 2021)

Abstract

Leptospirosis is a zoonosis occurring worldwide, carried by rodents which causes death in humans. Outbreaks of Leptospirosis can occur following heavy rain and flooding. The Susceptible-Infected-Recovered (SIR) model is employed in this paper in order to better understand the mechanisms of disease transmission. The factors that affect the transmission dynamics are analyzed. Disease free and endemic equilibrium points are determined from the proposed model and the local stability analysis for both of the equilibrium points is conducted. In addition, bifurcation analysis and numerical solutions of the model are conducted. A good accord is observed between the theoretical findings and the numerical simulations. Based on the research outcome, the parameter of natural death rate of rat population helps us to introduce the way to control the outbreak of Leptospirosis disease besides the basic reproduction number which acts as a key parameter in Leptospirosis epidemiology.

Key words and phrases: Leptospirosis, SIR model, Basic reproduction number, Bifurcation analysis.
AMS (MOS) Subject Classifications: 92B05, 92C60.
ISSN 1814-0432, 2021, http://ijmcs.future-in-tech.net

1 Introduction

Over the years, mathematics has been used to understand the modes of transmission of diseases in the world [1]. There are many diseases that are widely spread in this world such as Dengue, Hantavirus and Leptospirosis. Diseases that are spread from person to person are known as infectious disease whereas diseases that are generally caused by genetic or environmental factors are identified as noninfectious diseases [2]. According to Thayaparan et al. [3], Leptospirosis is a type of animal bacterial disease causing illness and even death in humans. It is caused by infection with bacteria (Leptospira interrogans). Leptospira are spread through animal hosts urine, which can get into water or soil and causing them to become contaminated. The animal hosts that carry Leptospira are not harmed themselves [4]. Humans can catch the disease if they have cuts on their skin (or through their mucosa of nose, eyes, and mouth) that come into contact with urine-contaminated water, food or soil [5]. When people exposed to contaminated water, such as floodwaters, outbreaks of Leptospirosis typically happen [6]. The transmission from an individual to another individual is very rare [7]. The sources of bacteria are generally cattle, pigs, sheep, goats, dogs, horses, mice, raccoons and rats [8].

Mathematical models have become a fundamental tool in suggesting appropriate strategies to eradicate or at least minimize Leptospirosis disease and in predicting future outbreak of it [9, 10]. Many models based on differential equations have been studied by numerous authors to describe the dynamical systems of both human and rat population. Kongnuy [11] discussed a mathematical model for the transmission of Leptospirosis disease between human and vector population. It is shown from the numerical results that Leptospirosis disease can be controlled by decreasing the transmission rate of Leptospirosis from an infected vector to a susceptible vector and human. Khan et al. [12] studied the dynamical behavior of Leptospirosis disease by considering saturated incidence rate. Incidence rate here means that susceptible population decreased by following the effective contact with the infected individuals and vectors. The simulation results of the proposed model suggested that the rate at which the infection force saturates must equal to 0.83 in order to reduce the risk of Leptospirosis disease transmission. El-Shahed [7] developed a fractional order model for the spread of Leptospirosis. From the numerical results, he concluded that the time taken to converge to the disease free equilibrium increases with lower values of fractional derivative. To gain better understanding of the transmission dynamics model proposed

by El-Shahed [7], we examine different outcomes of species interactions by employing the bifurcation analysis in this paper. In add, we verify our simulation findings and determine the factors that affect the transmission dynamics of Leptospirosis by analyzing the bifurcation analysis.

The rest of the paper is constructed as follows. In Section 2 we briefly discuss the mathematical model formulation of Leptospirosis disease. We identify the equilibrium points and examine the local stability analysis of the equilibrium points obtained in Section 3. In Section 4, we conduct the analytical result on transcritical bifurcation of Leptospirosis model. In Sections 5 and 6, we present the bifurcation analysis and numerical simulations respectively to validate the mathematical analysis of the model. Finally, we conclude our work.

2 Mathematical Model

The SIR model has been used to describe the transmission dynamics of Leptospirosis. The SIR model has both human and vector populations. The vector in this study is rats. The human population is categorized into three epidemiological subclasses; susceptible, infected and recovered denoted by S_h, I_h and R_h respectively. The rat population comprises of two classes that are susceptible S_r and infected I_r only with the assumption that the rat never recovers from infection. The total population for humans is denoted by N_h, with $N_h = S_h + I_h + R_h$ and the total population for rats is denoted by N_r, with $N_r = S_r + I_r$. We assume that the total human and rat populations are constant.

Figure 1 above shows the compartment diagram of SIR Leptospirosis model. A refers to recruitment rate of human population, μ_h is the natural death rate of human population, θ_h represents the rate of immune individuals become susceptible again, γ_h refers to recovery rate of human, B refers to recruitment rate of rat population, γ_r is the natural death rate of rat population, β_h represents the transmission rate of Leptospirosis from an infected rat to a susceptible human, β_r refers to transmission rate of Leptospirosis from an infected rat to a susceptible rat, δ_h refers to the rate of infected human dies from the disease and δ_r refers to the rate of infected rat dies due to disease.

We assume that all the parameters stated in Fig. 1 are non-negative. The transmission of Leptospirosis disease which consists of five nonlinear ordinary

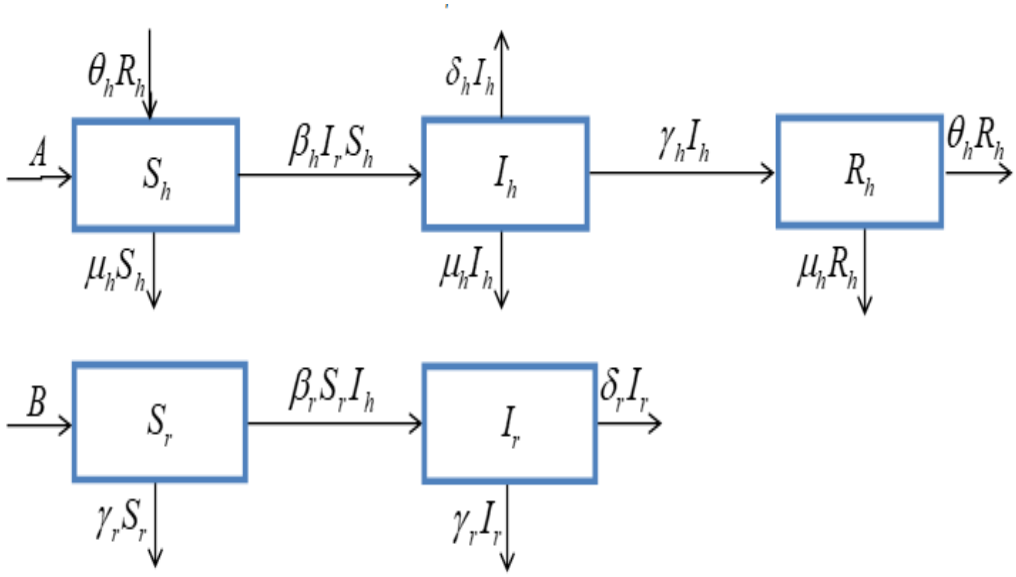

Figure 1: The flow diagram of human and rat interaction

differential equations as described in El-Shahed [7] is given by

$$\frac{dS_h}{dt} = A - \mu_h S_h - \beta_h I_r S_h + \theta_h R_h, \qquad S_h(0) \geq 0, \tag{2.1}$$

$$\frac{dI_h}{dt} = \beta_h I_r S_h - (\mu_h + \delta_h + \gamma_h) I_h, \qquad I_h(0) \geq 0, \tag{2.2}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - (\mu_h + \theta_h) R_h, \qquad R_h(0) \geq 0, \tag{2.3}$$

$$\frac{dS_r}{dt} = B - \gamma_r S_r - \beta_r S_r I_h, \qquad S_r(0) \geq 0, \tag{2.4}$$

$$\frac{dI_r}{dt} = \beta_r S_r I_h - (\gamma_r + \delta_r) I_r, \qquad I_r(0) \geq 0. \tag{2.5}$$

3 Equilibrium Points and Local Stability Analysis

To obtain the equilibrium points, we set the left hand side of equations (2.1) to (2.5) equal to zero. Then, we solve them simultaneously. Thus, our model has

(i) disease free equilibrium, $E_0 = (S_h,\, I_h,\, R_h,\, S_r,\, I_r) = \Big(\dfrac{A}{\mu_h}, 0, 0, \dfrac{B}{\gamma_r}, 0\Big)$ and
(ii) endemic equilibrium, $E_1 = (S_h,\, I_h,\, R_h,\, S_r,\, I_r)$ where

$$S_h=\frac{(\mu_h+\delta_h+\gamma_h)(\delta_r+\gamma_r)(A\beta_r(\mu_h+\theta_h)+(\mu_h^2+\theta_h\delta_h+\mu_h(\theta_h+\delta_h+\gamma_h)\gamma_r)}{(\beta_r(B\beta_h(\mu_h^2+\theta_h\delta_h+\mu_h(\delta_h+\theta_h+\gamma_h)+\mu_h(\mu_h+\theta_h)(\mu_h+\delta_h+\gamma_h)(\delta_r+\gamma_r))},$$
$$I_h=\frac{(\mu_h+\delta_h+\gamma_h)(\mu_h+\theta_h)(\delta_r+\gamma_r)\gamma_r\mu_h(R_0-1)}{(\beta_r(B\beta_h(\mu_h^2+\theta_h\delta_h+\mu_h(\delta_h+\theta_h+\gamma_h)+\mu_h(\mu_h+\theta_h)(\mu_h+\delta_h+\gamma_h)(\delta_r+\gamma_r))},$$
$$R_h=\frac{(\mu_h+\delta_h+\gamma_h)(\delta_r+\gamma_r)\gamma_r\gamma_h\mu_h(R_0-1)}{(B\beta_h(\mu_h^2+\theta_h\delta_h+\mu_h(\delta_h+\theta_h+\gamma_h)+\mu_h(\mu_h+\theta_h)(\mu_h+\delta_h+\gamma_h)(\delta_r+\gamma_r))},$$
$$S_r=\frac{(B\beta_h(\mu_h^2+\theta_h\delta_h+\mu_h(\delta_h+\theta_h+\gamma_h)+\mu_h(\mu_h+\theta_h)(\mu_h+\delta_h+\gamma_h)(\delta_r+\gamma_r))}{(\beta_h(A\beta_r(\mu_h+\theta_h)+(\mu_h^2+\theta_h\delta_h+\mu_h(\theta_h+\delta_h+\gamma_h))\gamma_r))},$$
$$I_r=\frac{(\mu_h+\delta_h+\gamma_h)(\mu_h+\theta_h)(\delta_r+\gamma_r)\gamma_r\mu_h(R_0-1)}{(\beta_h(\delta_r+\gamma_r)(A\beta_r(\mu_h+\theta_h)+(\mu_h^2+\theta_h\delta_h+\mu_h(\theta_h+\delta_h+\gamma_h))\gamma_r))}.$$

The basic reproduction number of our model, R_0, is calculated by using constant term of the characteristic polynomial method [13]. It is given by

$$R_0 = \frac{A\beta_h B\beta_r}{\mu_h(\mu_h+\delta_h+\gamma_h)\gamma_r(\gamma_r+\delta_r)}. \tag{3.6}$$

In the next part of this section, local stability analysis of disease free equilibrium point and endemic equilibrium point is examined from the Jacobian matrix of our model. By taking the partial derivatives with respect to S_h, I_h, R_h, S_r, I_r, the Jacobian matrix, J of our model is given below:

$$J=\begin{bmatrix} -\mu_h-\beta_h I_r & 0 & \theta_h & 0 & -\beta_h S_h \\ \beta_h I_r & -\mu_h-\delta_h-\gamma_h & 0 & 0 & \beta_h S_h \\ 0 & \gamma_h & -\mu_h-\theta_h & 0 & 0 \\ 0 & -\beta_r S_r & 0 & -\gamma_r-\beta_r I_h & 0 \\ 0 & \beta_r S_r & 0 & \beta_r I_h & -\delta_r-\gamma_r \end{bmatrix}.$$

Theorem 3.1. *The disease free equilibrium point, E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ [7].*

Proof. The Jacobian matrix, J is then evaluated at the disease free equilibrium, E_0. By performing elementary row operations, the characteristic equation of the Jacobian matrix, J at E_0 is

$$(-\mu_h-\theta_h-\lambda)(\mu_h+\lambda)(\gamma_r+\lambda)(\lambda^2+a_1\lambda+a_0)=0, \tag{3.7}$$

where $a_1 = (\mu_h+\delta_h+\gamma_h)+(\gamma_r+\delta_r)$, $a_0 = (\mu_h+\delta_h+\gamma_h)(\gamma_r+\delta_r)(1-R_0)$.

Clearly, the three roots of the characteristic equation (3.7) are $-(\mu_h + \theta_h)$, $-\mu_h$ and $-\gamma_r$ have negative real parts. The other two roots can be obtained from the second order polynomial of the characteristic equation (3.7). The Routh-Hurwitz criteria for ordinary differential equation is satisfied as $a_1 > 0$, $a_0 > 0$ if $R_0 < 1$. Thus, all the eigenvalues of the characteristic equation (3.7) have negative real parts. Therefore, our model at E_0 is locally asymptotically stable if $R_0 < 1$. □

Theorem 3.2. *The endemic equilibrium point, E_1 is locally asymptotically stable if $R_0 > 1$ and unstable if $R_0 < 1$ [7].*

Proof. The Jacobian matrix, J is then evaluated at the endemic equilibrium, E_1. By performing elementary row operations, the characteristic equation of the Jacobian matrix, J at E_1 is

$$\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0, \tag{3.8}$$

where $a_1 = (3\mu_h + \theta_h + \delta_h + \gamma_h + \delta_r + 2\gamma_r + \beta_r I_h^* + \beta_h I_r^*)$,

$a_2 = (3\mu_h^2 + 2\mu_h\theta_h + 2\mu_h\delta_h + \theta_h\delta_h + 3\mu_h\delta_r + \lambda_h\delta_r + \delta_h\delta_r + 2\mu_h\gamma_h + \theta_h\gamma_h + \delta_r\gamma_h + 6\mu_h\gamma_r + 2\theta_h\gamma_r + 2\delta_h\gamma_r + \delta_r\gamma_r + 2\gamma_h\gamma_r + \gamma_r^2 + 3\mu_h\beta_r I_h^* + \beta_r\theta_h I_h^* + \beta_r\delta_h I_h^* + \beta_r\delta_r I_h^* + \beta_r\gamma_h I_h^* + \{\beta_r\gamma_r I_h^* - \beta_h\beta_r S_h^* S_r^*\} + 2\mu_h\beta_h I_r^* + \beta_h\theta_h I_r^* + \beta_h\delta_h I_r^* + \beta_h\delta_r I_r^* + \beta_h\gamma_h I_r^* + 2\beta_h\gamma_r I_r^* + \beta_h\beta_r I_h^* I_r^*)$,

$a_3 = (\mu_h^3 + \mu_h^2\theta_h + \mu_h^2\delta_h + \mu_h\theta_h\delta_h + 3\mu_h^2\delta_r + 2\mu_h\theta_h\delta_r + 2\mu_h\delta_h\delta_r + \lambda_h\delta_h\delta_r + \mu_h^2\gamma_h + \mu_h\theta_h\gamma_h + 2\mu_h\delta_r\gamma_h + \theta_h\delta_h\gamma_h + 6\mu_h^2\gamma_r + 4\mu_h\theta_h\gamma_r + 4\mu_h\delta_h\gamma_r + 2\theta_h\delta_h\gamma_r + 3\mu_h\delta_r\gamma_r + \theta_h\delta_r\gamma_r + \delta_h\delta_h\gamma_r + 4\mu_h\gamma_h\gamma_r + 2\theta_h\gamma_h\gamma_r + \delta_r\gamma_h\gamma_r + 3\mu_h\gamma_r^2 + \theta_h\gamma_r^2 + \delta_h\gamma_r^2 + \gamma_h\gamma_r^2 + 3\mu_h^2\beta_r I_h^* + 2\mu_h\beta_r\theta_h I_h^* + 2\mu_h\beta_r\delta_h I_h^* + \beta_r\theta_h\delta_h I_h^* + 3\mu_h\beta_r\delta_r I_h^* + \beta_r\theta_h\delta_h I_h^* + \beta_r\delta_h\delta_r I_h^* + 2\mu_h\beta_r\gamma_h I_h^* + \beta_r\theta_h\gamma_h I_h^* + \beta_r\delta_r\gamma_h I_h^* + 3\mu_h\beta_r\gamma_r I_h^* + \beta_r\theta_h\gamma_r I_h^* + \beta_r\delta_h\gamma_r I_h^* + \{\beta_r\gamma_h\gamma_r I_h^* - 2\mu_h\beta_h\beta_r S_h^* S_r^* - \beta_h\beta_r\theta_h S_h^* S_r^* - \beta_h\beta_r\gamma_r S_h^* S_r^*\} + \mu_h^2\beta_h I_r^* + \mu_h\beta_h\theta_h I_r^* + \mu_h\beta_h\delta_h I_r^* + \beta_h\theta_h\delta_r I_r^* + 2\mu_h\beta_h\delta_r I_r^* + \beta_h\theta_h\delta_r I_r^* + \beta_h\delta_h\delta_r I_r^* + \mu_h\beta_h\gamma_h I_r^* + \beta_h\delta_r\gamma_h I_r^* + 4\mu_h\beta_h\gamma_r I_r^* + 2\beta_h\theta_h\gamma_r I_r^* + 2\beta_h\delta_h\gamma_r I_r^* + \beta_h\delta_r\gamma_r I_r^* + 2\beta_h\gamma_h\gamma_r I_r^* + \beta_h\gamma_r^2 I_r^* + 2\mu_h\beta_h\beta_r I_h^* I_r^* + \beta_h\beta_r\theta_h I_h^* I_r^* + \beta_h\beta_r\delta_h I_h^* I_r^* + \beta_h\beta_r\delta_r I_h^* I_r^* + \beta_h\beta_r\gamma_h I_h^* I_r^* + \beta_h\beta_r\gamma_r I_h^* I_r^*)$,

$a_4 = (\mu_h^3\delta_r + \mu_h^2\theta_h\delta_r + \mu_h^2\delta_h\delta_r + \mu_h\theta_h\delta_h\delta_r + \mu_h^2\delta_r\gamma_h + \mu_h\theta_h\delta_r\gamma_h + 2\mu_h^3\gamma_r + 2\mu_h^2\lambda_h\gamma_r + 2\mu_h^2\delta_h\gamma_r + 2\mu_h\theta_h\delta_h\gamma_r + 3\mu_h^2\delta_r\gamma_r + 2\mu_h\theta_h\delta_r\gamma_r + 2\mu_h\delta_h\delta_r\gamma_r + \theta_h\delta_h\delta_r\gamma_r + 2\mu_h^2\gamma_h\gamma_r + 2\mu_h\theta_h\gamma_h\gamma_r + 2\mu_h\delta_r\gamma_h\gamma_r + \theta_h\delta_r\gamma_h\gamma_r + 3\mu_h^2\gamma_r^2 + 2\mu_h\theta_h\gamma_r^2 + 2\mu_h\delta_h\gamma_r^2 + \theta_h\delta_h\gamma_r^2 + 2\mu_h\gamma_h\gamma_r^2 + \theta_h\gamma_h\gamma_r^2 + \mu_h^3\beta_r I_h^* + \mu_h^2\beta_r\theta_h I_h^* + \mu_h^2\beta_r\delta_h I_h^* + \mu_h\beta_r\theta_h\delta_h I_h^* + 3\mu_h^2\beta_r\delta_r I_h^* + 2\mu_h\beta_r\theta_h\delta_r I_h^* + 2\mu_h\beta_r\delta_h\delta_r I_h^* + \beta_r\theta_h\delta_h\delta_r I_h^* + \mu_h^2\beta_r\gamma_h I_h^* + \mu_h\beta_r\theta_h\gamma_h I_h^* + 2\mu_h\beta_r\delta_r\gamma_h I_h^* + \beta_r\theta_h\delta_r\gamma_h I_h^* + 3\mu_h^2\beta_r\gamma_r I_h^* + 2\mu_h\beta_r\theta_h\gamma_r I_h* + 2\mu_h\beta_r\delta_h\gamma_r I_h^* + \beta_r\theta_h\delta_h\gamma_r I_h^* +$

$2\mu_h\beta_r\gamma_h\gamma_r I_h^* + \{\beta_r\theta_h\gamma_h\gamma_r I_h^* - \mu_h^2\beta_h\beta_r S_h^* S_r^* - \mu_h\beta_h\beta_r\theta_h S_h^* S_r^* - 2\mu_h\beta_h\beta_r\gamma_r S_h^* S_r^* - \beta_h\beta_r\theta_h\gamma_r S_h^* S_r^*\} + \mu_h^2\beta_h\delta_r I_r^* + \mu_h\beta_h\theta_h\delta_r I_r^* + \mu_h\beta_h\delta_h\delta_r I_r^* + \beta_h\theta_h\delta_h\delta_r I_r^* + \mu_h\beta_h\delta_r\gamma_h I_r^* + 2\mu_h^2\beta_h\gamma_r I_r^* + 2\mu_h\beta_h\theta_h\gamma_r I_r^* + 2\mu_h\beta_h\delta_h\gamma_r I_r^* + 2\beta_h\theta_h\delta_h\gamma_r I_r^* + 2\mu_h\beta_h\delta_r\gamma_r I_r^* + \beta_h\theta_h\delta_r\gamma_r I_r^* + \beta_h\delta_h\delta_r\gamma_r I_r^* + 2\mu_h\beta_h\gamma_h\gamma_r I_r^* + \beta_h\delta_r\gamma_h\gamma_r I_r^* + 2\mu_h\beta_h\gamma_r^2 I_r^* + \beta_h\theta_h\gamma_r^2 I_r^* + \beta_h\delta_h\gamma_r^2 I_r^* + \beta_h\gamma_h\gamma_r^2 I_r^* + \mu_h^2\beta_h\beta_r I_h^* I_r^* + \mu_h\beta_h\beta_r\theta_h I_h^* I_r^* + \mu_h\beta_h\beta_r\delta_h I_h^* I_r^* + \beta_h\beta_r\theta_h\delta_h I_h^* I_r^* + 2\mu_h\beta_h\beta_r\delta_r I_h^* I_r^* + \beta_h\beta_r\theta_h\delta_r I_h^* I_r^* + \beta_h\beta_r\delta_h\delta_r I_h^* I_r^* + \mu_h\beta_h\beta_r\gamma_h I_h^* I_r^* + \beta_h\beta_r\delta_r\gamma_h I_h^* I_r^* + 2\mu_h\beta_h\beta_r\gamma_r I_h^* I_r^* + \beta_h\beta_r\theta_h\gamma_r I_h^* I_r^* + \beta_h\beta_r\delta_h\gamma_r I_h^* I_r^* + \beta_h\beta_r\gamma_h\gamma_r I_h^* I_r^*),$

$a_5 = (\mu_h^3\delta_r\gamma_r + \mu_h^2\theta_h\delta_r\gamma_r + \mu_h^2\delta_h\delta_r\gamma_r + \mu_h\theta_h\delta_h\delta_r\gamma_r + \mu_h^2\delta_r\gamma_h\gamma_r + \mu_h\theta_h\delta_r\gamma_h\gamma_r + \mu_h^3\gamma_r^2 + \mu_h^2\theta_h\gamma_r^2 + \mu_h^2\delta_h\gamma_r^2 + \mu_h\theta_h\delta_h\gamma_r^2 + \mu_h^2\gamma_h\gamma_r^2 + \mu_h\theta_h\gamma_h\gamma_r^2 + \mu_h^3\beta_r\delta_r I_h^* + \mu_h^2\beta_r\theta_h\delta_r I_h^* + \mu_h^2\beta_r\delta_h\delta_r I_h^* + \mu_h\beta_r\theta_h\delta_h\delta_r I_h^* + \mu_h^2\beta_r\delta_r\gamma_h I_h^* + \mu_h\beta_r\theta_h\delta_r\gamma_h I_h^* + \mu_h^3\beta_r\gamma_r I_h^* + \mu_h^2\beta_r\theta_h\gamma_r I_h^* + \mu_h^2\beta_r\delta_h\gamma_r I_h^* + \mu_h\beta_r\theta_h\delta_h\gamma_r I_h^* + \mu_h^2\beta_r\gamma_h\gamma_r I_h^* + \{\mu_h\beta_r\theta_h\gamma_h\gamma_r I_h^* - \mu_h^2\beta_h\beta_r\gamma_r S_h^* S_r^* - \mu_h\beta_h\beta_r\theta_h\gamma_r S_h^* S_r^*\} + \mu_h^2\beta_h\delta_r\gamma_r I_r^* + \mu_h\beta_h\theta_h\delta_r\gamma_r I_r^* + \mu_h\beta_h\delta_h\delta_r\gamma_r I_r^* + \beta_h\theta_h\delta_h\delta_r\gamma_r I_r^* + \mu_h\beta_h\delta_r\gamma_h\gamma_r I_r^* + \mu_h^2\beta_h\gamma_r^2 I_r^* + \mu_h\beta_h\theta_h\gamma_r^2 I_r^* + \mu_h\beta_h\delta_h\gamma_r^2 I_r^* + \beta_h\theta_h\delta_h\gamma_r^2 I_r^* + \mu_h\beta_h\gamma_h\gamma_r^2 I_r^* + \mu_h^2\beta_h\beta_r\delta_r I_h^* I_r^* + \mu_h\beta_h\beta_r\theta_h\delta_r I_h^* I_r^* + \mu_h\beta_h\beta_r\delta_h\delta_r I_h^* I_r^* + \beta_h\beta_r\theta_h\delta_h\delta_r I_h^* I_r^* + \mu_h\beta_h\beta_r\delta_r\gamma_h I_h^* I_r^* + \mu_h^2\beta_h\beta_r\gamma_r I_h^* I_r^* + \mu_h\beta_h\beta_r\theta_h\gamma_r I_h^* I_r^* + \mu_h\beta_h\beta_r\delta_h\gamma_r I_h^* I_r^* + \beta_h\beta_r\theta_h\delta_h\gamma_r I_h^* I_r^* + \mu_h\beta_h\beta_r\gamma_h\gamma_r I_h^* I_r^*).$

The Routh-Hurwitz criteria for ordinary differential equation is satisfied as $a_1 > 0$, $a_2 > 0$, $a_3 > 0$, $a_4 > 0$, $a_5 > 0$, $a_1a_2a_3 > a_3^2 + a_1^2a_4$, $(a_1a_4 - a_5)(a_1a_2a_3 - a_3^2 - a_1^2a_4) > a_5(a_1a_2 - a_3)^2 + a_1a_5^2$ if $R_0 > 1$ and the terms under braces are positive. Thus, all the eigenvalues of the characteristic equation (3.8) have negative real parts. Therefore, our model at E_1 is locally asymptotically stable if $R_0 > 1$ and the terms under braces are positive. □

4 Analytical Result on Transcritical Bifurcation

Since we obtain only two equilibrium points as stated in the earlier section, thus we can only observe transcritical bifurcation of our model. So, we will focus on the existence of transcritical bifurcation of the proposed model by determining the bifurcation point.

Noting that, we consider the natural death rate of rat population, γ_r as the bifurcation parameter. We can derive the transcritical bifurcation point,γ_{r_T} from the basic reproduction number in equation (3.6). Then,

$$\gamma_{r_T} = \frac{-\mu_h^2\delta_r R_0 - \mu_h\delta_h\delta_r R_0 - \mu_h\delta_r\gamma_h R_0 \pm \sqrt{\mu_h}\sqrt{\mu_h + \delta_h + \gamma_h}\sqrt{R_0}\sqrt{4A\beta_h B\beta_r + \mu_h^2\delta_r^2 R_0 + \mu_h\delta_h\delta_r^2 R_0 + \mu_h\delta_r^2\gamma_h R_0}}{2(\mu_h^2 R_0 + \mu_h\delta_h R_0 + \mu_h\gamma_h R_0)}. \quad (4.9)$$

As mentioned earlier, we only consider non-negative parameter values to ensure that our proposed model is locally asymptotically stable. Thus, by substituting the value for each parameter; $A = 1.6$, $B = 1.2$, $\mu_h = 0.034$, $\beta_h = 0.0098$, $\gamma_h = 0.007$, $\delta_h = 0.0000001$, $\beta_r = 0.0078$, $\delta_r = 0.0094$ and $R_0 = 1$ into equation (4.9), transcritical bifurcation occurs when $\gamma_{r_T} = 0.3198$.

5 Bifurcation Analysis

Bifurcation analysis of our model is carried out by using XPPAUT. The effect of varying the parameter values on the dynamical behavior of our model around each equilibrium point is analyzed. Noting that the basic reproduction number, R_0 is the function of the parameter γ_r has significant epidemiological meaning. Therefore, we consider the natural death rate of rat population, γ_r as the bifurcation parameter to present the bifurcation diagram by simulations. To illustrate these, the parameter values that will be used are $A = 1.6$, $B = 1.2$, $\mu_h = 0.034$, $\beta_h = 0.0098$, $\gamma_h = 0.007$, $\delta_h = 0.0000001$, $\beta_r = 0.0078$, $\theta_h = 0.00067$, $\delta_r = 0.0094$ and $\gamma_r = 0.17$, the same parameter values that will be used in Fig. 3 (b). These values were used by El-Shahed [7].

In particular, Fig. 2 shows the bifurcation diagram of the infected human, I_h with the natural death rate of rat population when $\gamma_r = 0.17$. The red color lines denote stable steady states while the black color lines denote unstable steady states.

In Fig. 2, when infected human equal to zero, the horizontal line shows the disease free equilibrium whereas when infected human is not equal to zero, the slanting line shows the endemic equilibrium. In general, any value of γ_r will give us the same threshold value for natural death rate of rat population, which determine the dynamical behaviors of the Leptospirosis model. The green color point is the threshold value, γ_{r_T} corresponding to a transcritical bifurcation point. In the previous section, we have computed this transcritical bifurcation point analytically and it is given in the equation (4.9). By comparing our analytical calculation in equation (4.9) with numerical continuation results in Fig. 2, we can observe that the threshold values are in good agreement between the two findings in Section 4 and Fig. 2.

To the left of γ_{r_T} in Fig. 2, it shows that the unstable disease free equilibrium (black line) and stable endemic equilibrium (red line) exist, which results in persistence of rat populations and leads to stable coexistence of

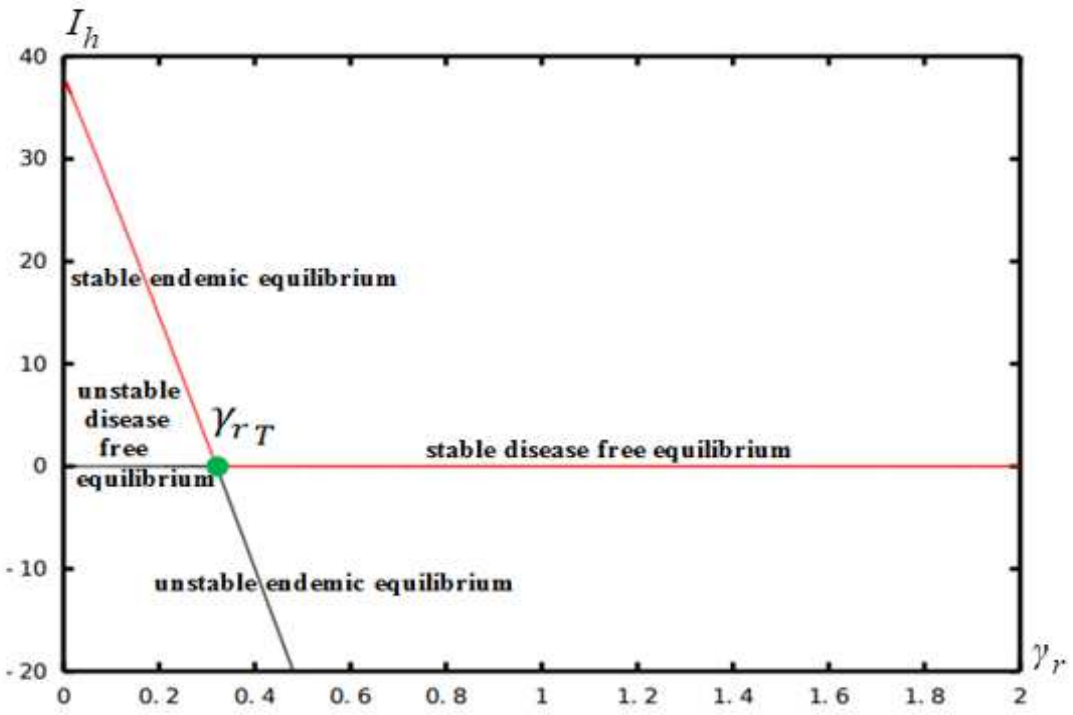

Figure 2: One parameter bifurcation diagram of the infected human with natural death rate of rat population when $\gamma_r = 0.17$

rat and human populations. Consequently, to the right of γ_{r_T} in Fig. 2, it shows that the unstable endemic equilibrium (black line) and stable disease free equilibrium (red line) exist, which leads to the extinction of rat populations. At γ_{r_T}, transcritical bifurcation occurs, and there is an exchange of stability between two steady states; in particular, the unstable disease free (or endemic) steady state exchanges stability with the stable disease free (or endemic) steady state.

6 Numerical Simulations

In this section, we present the numerical solution of our model. The given model is solved numerically by using the built-in function, called NDSolve in MATHEMATICA. The parameter values used in the numerical solution are $A = 1.6$, $B = 1.2$, $\mu_h = 0.034$, $\beta_h = 0.0098$, $\gamma_h = 0.007$, $\delta_h = 0.0000001$, $\beta_r = 0.0078$, $\theta_h = 0.00067$, $\delta_r = 0.0094$ and $\gamma_r = 0.17$ varies. The initial

conditions $S_h(0) = 100$, $I_h(0) = 20$, $R_h(0) = 30$, $S_r(0) = 50$ and $I_r(0) = 10$ are used in Fig. 3 (a) and (b).

In particular, Fig. 3 (a) represents the dynamical behavior of human and rat population when $\gamma_r = 0.417$ whereas Fig. 3 (b) represents the dynamical behavior of human and rat population when $\gamma_r = 0.17$. For each figure, the red color curve represents the population of susceptible human, the blue color curve depicts the population of infected human, the green color curve representing the recovered human population, the purple color curve shows the class of susceptible rat and the yellow color curve represents the population of infected rat.

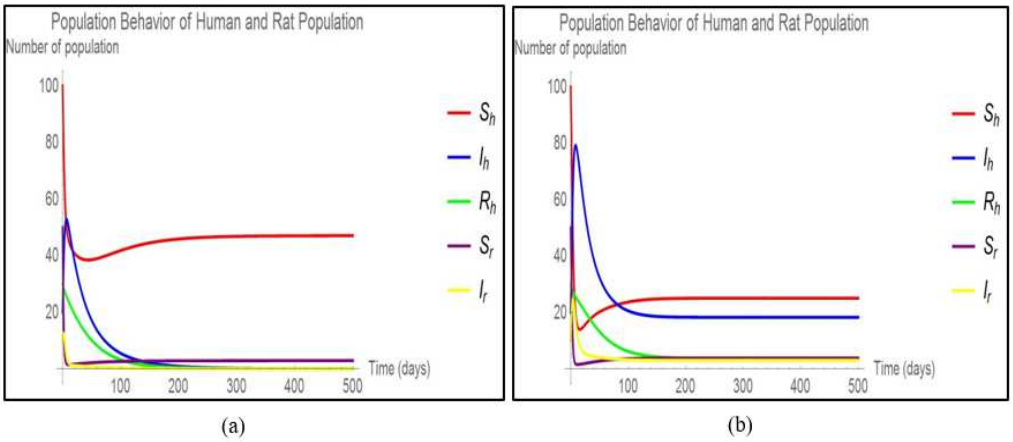

Figure 3: Population behavior of human and rat population with time (days) for (a) $R_0 = 0.5921$ (determined from (3.6)) when $\gamma_r = 0.417$ (b) $R_0 = 3.4521$ (determined from (3.6)) when $\gamma_r = 0.17$

In Fig. 3 (a), it can be seen that when $\gamma_r (= 0.417)$ is high, there is a gradual decrease in the infected rat, I_r population. This causes a decrease in infected human, I_h population and eventually be eradicated from Leptospirosis epidemic model. Recovered human, R_h population will decline gradually over the 500 days period. The results vividly demonstrates that the Leptospirosis disease will die off with high γ_r. At the same time, when γ_r is quite high, this will cause the susceptible rat, S_r and susceptible human, S_h populations to decrease. When $R_0 < 1$, the graph of the numerical solution in Fig. 3 (a) converges to $E_0 = (47.0588, 0, 0, 2, 8777, 0)$. Based from Fig. 2, when $\gamma_r = 0.417$, it can be seen that the disease free equilibrium is stable.

By decreasing γ_r from 0.417 to 0.17 and maintaining the other parameters

values, we can observe (by comparing Fig. 3 (a) and Fig. 3 (b)) that there is difference in the values for both human and rat populations over the 500 days period. When the value of γ_r decreases, the steady value of infected human, recovered human, susceptible rat and infected rat populations increases while the steady value of susceptible human decreases. When $R_0 > 1$, we can see that the solution converges to $E_1 = (25.073, 18.2924, 3.69331, 3.83778, 3.05227)$ where all species coexist in the ecosystem. Based from Fig. 2, when $\gamma_r = 0.17$, it can be observed that the endemic equilibrium is stable.

7 Conclusion

In this paper, a system of nonlinear ordinary differential equations to describe the behavior of the Leptospirosis model in human and rat populations is studied. The outbreak of the Leptospirosis infection has two states namely disease free equilibrium and endemic equilibrium. We used an alternative approach, constant term of the characteristic polynomial method to find the basic reproduction number, R_0. If $R_0 < 1$, then the disease will disappear in community but if $R_0 > 1$, then the disease will persist in community as shown in Fig. 3 (a) and Fig. 3 (b). Local stability analysis of each equilibrium point shows that our proposed model is locally asymptotically stable. Since we obtained only two equilibrium points as stated in Section 3, thus we can only observe transcritical bifurcation of the proposed model. We also conduct the analytical result on transcritical bifurcation of our model by determining the transcritical bifurcation point; $\gamma_{r_T} = 0.3198$.

In addition, we show the bifurcation analysis and time series plot of the proposed model. Our work demonstrates the occurrence of transcritical bifurcations as the mechanism that can mediate the coexistence of human and rat populations. From a biological viewpoint, transcritical bifurcation corresponds to an invasion of a species in a biological system. This bifurcation may result in the observations of different species presence-absence as some biologically-relevant parameters change. When natural death rate of rat population, $\gamma_{r_T} < 0.3198$, unstable disease free equilibrium and stable endemic equilibrium exist whereas when $\gamma_{r_T} > 0.3198$, stable disease free equilibrium and unstable endemic equilibrium exist. At $\gamma_{r_T} = 0.3198$, transcritical bifurcation occurs, where there is an exchange of stability between two steady states. From the numerical results of our model, when $R_0 < 1$, our numerical solutions approach the disease free equilibrium but when our numerical solutions converge to the endemic equilibrium. Thus, our numerical result

shows a good agreement with the analytical result.

With this, we can also observe (by comparing Fig. 3 (a) and Fig. 3 (b)) that the time to converge to the disease free equilibrium is longer than the time to converge to the endemic equilibrium because the amount of time before the disease disappears is longer than that of the endemic state. This reflects what would happen in the real world.

To conclude, it is common that R_0 is used to measure the transmission of Leptospirosis infection but in our study here, the parameter γ_r helps us to introduce the way to control the outbreak of Leptospirosis disease.

Acknowledgment. The authors gratefully acknowledge the School of Mathematical Sciences, Universiti Sains Malaysia for the facilities used in this study.

References

[1] M. Keeling, One parameter (almost) does it all, The mathematics of diseases, (2001), https://plus.maths.org/content/mathematics-diseases

[2] M. J. Keeling, P. Rohani, Modeling Infectious Diseases in Humans and Animals, Princeton University Press, United Kingdom, (2008), 1–3.

[3] S. Thayaparan, I. Robertson, A. Fairuz, S. Lela, M. T. Abdullah, Leptospirosis, an emerging zoonotic disease in Malaysia, Malays. J. Pathol., **35,** no. 2, (2013), 123–132.

[4] J. K. Lim, V. A. Murugaiyah, A. Ramli, H. Abdul Rahman, N. Mohamed, N. Shamsudin, J. C. Tan, A Case Study: Leptospirosis In Malaysia, WebmedCentral Infectious Diseases, **2,** no. 12, (2011).

[5] A. Bhalraj, A. Azmi, Mathematical Modelling of the Spread of Leptospirosis, Proceedings of the International Conference on Mathematical Sciences and Technology 2018, AIP Conference Proceedings 2184, (2018), 060031-1 060031-10.

[6] W. Triampo, D. Baowan, I. M. Tang, N. Nuttavut, J. Wong-Ekkabut, G. Doungchawee, A Simple Deterministic Model for the Spread of Leptospirosis in Thailand, Int. J. Biol. Med. Sci., **2,** no. 1, (2007), 22–26.

[7] M. El-Shahed, Fractional Order Model for the Spread of Leptospirosis, Int. J. Math. Anal., **8,** no. 54, (2014), 2651–2667.

[8] S. H. Goh, R. Ismail, S. F. Lau, P. A. Megat Abdul Rani, T. B. Mohd Mohidin, F. Daud, A. R. Bahaman, S. Khairani-Bejo, R. Radzi, K. H. Khor, Risk Factors and Prediction of Leptospiral Seropositivity Among Dogs and Dog Handlers in Malaysia, Int. J. Environ. Res. Public Health, **16,** no. 9, (2019), 1–11.

[9] M. A. Khan, S. Ullah, S. Kumar, A Robust Study on 2019-nCoV Outbreaks Through Non-Singular Derivative, Eur. Phys. J. Plus, **136,** no. 168, (2021), 1–20.

[10] M. F. Khan, H. Alrabaiah, S. Ullah, M. A. Khan, M. Farooq, M. Mamat, M. I. Asjad, A New Fractional Model for Vector-Host Disease with Saturated Treatment Function via Singular and Non-Singular Operators, Alex. Eng. J., **60,** no. 1, (2020), 629–645.

[11] R. Kongnuy, Local Stability of Equilibria: Leptospirosis, Int. J. Math. Comput. Sci., **6,** no. 6, (2012), 625–629.

[12] M. A. Khan, S. F. Saddiq, S. Islam, I. Khan, S. Shafie, Dynamic Behavior of Leptospirosis Disease with Saturated Incidence Rate, Int. J. Appl. Comput. Math., **2,** no. 4, (2016), 435–452.

[13] J. Li, D. Blakeley, R. J. Smith, The Failure of R_0, Comput. Math. Methods Med., **2011,** no. 1, (2011), 1–17.