

Research Article

A Mathematical Model Analysis for the Transmission Dynamics of Leptospirosis Disease in Human and Rodent Populations

Habtamu Ayalew Engida ¹, David Mwangi Theuri,² Duncan Gathungu ², John Gachohi,³ and Haileyesus Tessema Alemneh⁴

¹Pan African University for Basic Science, Technology and Innovation (PAUSTI)/JKUAT, Kenya

²Department of Mathematics, Jomo Kenyatta University of Agriculture and Technology, Kenya

³Department of Public Health, Jomo Kenyatta University of Agriculture and Technology, Kenya

⁴Department of Applied Mathematics, University of Gondar, Ethiopia

Correspondence should be addressed to Habtamu Ayalew Engida; hayalew21@gmail.com

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This work is aimed at formulating and analyzing a compartmental mathematical model to investigate the impact of rodent-born leptospirosis on the human population by considering a load of pathogenic agents of the disease in an environment and the incidence rate of human infection due to the interaction between infected rodents and the environment. Firstly, the basic properties of the model, the equilibria points, and their stability analysis are studied. We also found the basic reproduction number (R_0) of the model using the next-generation matrix approach. From the stability analysis, we obtained that the disease-free equilibrium (DFE) is globally asymptotically stable if $R_0 < 1$ and unstable otherwise. The local stability of endemic equilibrium is performed using the phenomenon of the center manifold theory, and the model exhibits forward bifurcation. The most sensitive parameters on the model outcome are also identified using the normalized forward sensitivity index. Finally, numerical simulations of the model are performed to show the stability behavior of endemic equilibrium and the varying effect of the human transmission rates, human recovery rate, and the mortality rate rodents on the model dynamics. The model is simulated using the forward fourth-order Runge-Kutta method, and the results are presented graphically. From graphical stability analysis, we observed that all trajectories of the model solutions evolve towards the unique endemic equilibrium over time when $R_0 > 1$. Our numerical results revealed that decreasing the transmission rates and increasing the rate of recovery and reduction of the rodent population using appropriate intervention mechanisms have a significant role in reducing the spread of disease infection in the population.

1. Introduction

Leptospirosis is an infectious bacterial disease caused by a pathogenic spirochete bacteria called *Leptospira interrogans*, and it occurs throughout the world but is most common in tropical and subtropical regions, especially in developing countries including South-East Asia countries and sub-Saharan Africa [1–3]. It is one of the major direct zoonosis diseases worldwide that affects humans and animals [4, 5]. Recent studies reported that more than one million human cases with an estimated 60,000 deaths occur worldwide each year due to the *Leptospira interrogans* [1, 4].

Human infection occurs through direct contact with infected animal reservoir urine, tissues, or other body fluid, or more commonly by contact with infected animal (reservoirs) urine-contaminated environment [1, 6, 7]. Person-to-person transmission of the disease occurs very rarely. Globally, the main animal reservoirs of human leptospirosis are rodents (rats and mice), especially in urban slum environments [8–10]. The high concentration of *Leptospira* bacteria shed in slum environments mainly occurs due to the high prevalence of infection in rat population [6].

The incubation period of leptospirosis disease is typically 5 to 14 days [1, 11]. The symptoms and signs of the disease

are nonfixed and may be confused with other diseases' symptoms (dengue, hantavirus, malaria, melioidosis, influenza, etc.) due to the initially nonspecific presentation. Consequently, its cases are sometimes underrecognized [11]. In humans, leptospirosis illness can be demonstrated by two stages. The primary (an acute) stage is illustrated by mild illness with nonspecific signs like higher fever, headache, and conjunctival suffusion [1, 12], while syndromes in the second (severe illness or immune) stage of leptospirosis include jaundice, kidney failure, haemorrhage (especially pulmonary), meningitis, cardiac arrhythmias, respiratory insufficiency, and hemodynamic collapse [1, 11].

Human vaccine against leptospirosis is not widely practiced and accessible only in a few developed countries which protect only against the serovar in the vaccine, and regular boosting is required [11]. However, prevention and control interventions can be implemented to avoid the risk of leptospirosis infections (especially for people whose working environment exposes them to the risk of infections), to minimize the acquisition of the infection, or to reduce the human infections in infected population. Preventive measures include personal protective equipment (PPE) (wearing rubber boots, waterproof overalls/dressings to cover wounds or skin, goggles, and rubber gloves) and personal hygiene. Environmental modifications such as draining wet areas or improvements in urban slum environment to reduce the concentration of pathogenic spirochete leptospires from the environment [1, 12].

Patients with early leptospirosis infection (mild illness) can be treated through antibiotic doxycycline, ampicillin or amoxicillin, azithromycin, or clarithromycin. Patients with severe illness can be treated through IV penicillin and ceftriaxone drugs [1, 11].

Recently, a mathematical model has become an important tool in underlying mechanisms of disease transmission and spread and has been used to predict outcomes of diseases in communities, help to explain key factors in the disease transmission process, suggest effective control and preventive measures, and provide an estimate for the seriousness and potential scale of the epidemic [13]. In particular, deterministic mathematical models have been used to study the dynamics of infectious diseases within human and vector hosts in the population by dividing the individuals into different stages and making assumptions about the nature and time rate of transfer from one compartment to another [14].

A few researchers have been investigated a two-strain deterministic mathematical model, one strain for the human population and another strain for vectors (or animal population), for the transmission process of leptospirosis disease in various forms to demonstrate the dynamical spread of the disease between the two population groups.

Pimpunchat et al. [15] proposed the susceptible, infected, removed (SIR) model of leptospirosis which consists of both human and vector host populations. In their model, the human population is divided into three compartmental, susceptible, infected, and recovered humans, whereas the vector population was divided into two classes, susceptible and infected vectors. They performed numerical solutions to analyze and examine the behavior of the disease.

Likewise, another compartmental mathematical model was developed by incorporating exposed cases in both human and vector populations for transmission dynamics of leptospirosis disease in [16]. They illustrated numerical simulations of the formulated model by using the well-known numerical method, the Runge-Kutta of fourth-order in the MATLAB program. Authors in [17] presented a compartmental mathematical model for the dynamic behavior of the leptospirosis disease with saturated incidence. They described the model using nonlinear ordinary differential equations by subdividing the host population of humans into three classes, susceptible individuals, infected individuals, and recovered individuals, and also by subdividing vectors (animal) population into two compartments, susceptible and infected vectors. They investigated the stability analysis of the disease-free and endemic equilibrium by using the basic reproduction number. From the results of stability analysis, they obtained that the disease-free equilibrium is stable both locally and globally whenever the basic reproduction number is less than one. Similarly, authors in [18] investigated a mathematical model of the leptospirosis disease using a compartmental approach. However, in view of above models, the contraction of *Leptospira interrogans* in environment for transmission had not been incorporated.

Although the human infection of the disease occurs in multiple different ways, most of the infections acquired directly or indirectly from environment (soil or water) contaminated with *Leptospira* especially in urban slum areas [7–9].

Motivated from [5, 17], this study examines a two-strain model, SEIRS model for the human population and another SIRS model for rodent (vector) population taking account the concentration leptospires population in environment and the transmission rates between susceptible individuals and the load of the pathogen in the environment and between susceptible rodents and infected rodents for the dynamics behavior of leptospirosis disease. Thus, in this work, we present two host populations (namely, humans and rodents) and the pathogenic population with its two growth controls (namely, the natural death rate of *Leptospira* and carrying capacity controls). The rest of this work is organized as follows. The leptospirosis model is described and formulated in Section 2. Section 3 is concerned with the basic properties of the model, local and global stability of disease-free equilibrium, the phenomena of bifurcation analysis, and local stability of endemic equilibrium. Section 4 is devoted to the sensitivity analysis for the basic reproduction number (R_0) of the model to each of the parameter values. The numerical simulations and discussions are presented in Section 5. Our conclusions are given in Section 6.

2. Model Formulation

Using system of nonlinear differential equations, we build up a compartmental mathematical model for the transmission process of leptospirosis epidemic. The model includes human, vector (rodent), and bacterial populations. The total human population denoted by $N_h(t)$ is subdivided into four compartments: $S_h(t)$, $E_h(t)$, $I_h(t)$, and $R_h(t)$. Thus,

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t). \quad (1)$$

The total rodent population denoted by $N_v(t)$ is subgrouped into three groups: $S_v(t)$, $I_v(t)$, and $R_v(t)$ with

$$N_v(t) = S_v(t) + I_v(t) + R_v(t). \quad (2)$$

Moreover, the state variables of the model are described in Table 1.

In the formulation of the model, the following assumptions are considered:

- (i) Each parameter of the model cannot be negative
- (ii) We assume that susceptible human and rodent populations increase at constant rates are given by recruitment of the individuals Λ and Π , respectively
- (iii) We assume that susceptible humans can be infected in two different ways: through either direct contact with infected rodents urine or contact with contaminated environments
- (iv) Assume there is a homogeneous mixing between human and rodent populations

The susceptible individuals can acquire infection with the incidence rate $\lambda_h = \lambda_{hB} + \lambda_{hv}$ with $\lambda_{hB} = \beta_2 B_l / (\kappa + B_l)$ and $\lambda_{hv} = \beta_1 I_v$. The susceptible rodents can acquire infection with the force of infection $\beta_3 I_h$. The population B_l increases its size in contaminated environment from the release of bacteria by infected humans and rodents with the rate of τ_1 and τ_2 , respectively. Further, description of the parameters of the model is summarized in Table 2.

From the flowchart diagram in Figure 1, we obtained the following set of differential equations for the compartmental model of the leptospirosis disease.

$$\left\{ \begin{array}{l} \frac{dS_h}{dt} = \Lambda + \gamma R_h - (\lambda_h + \mu) S_h, \\ \frac{dE_h}{dt} = \lambda_h S_h - (\theta + \mu) E_h, \\ \frac{dI_h}{dt} = \theta E_h - (\alpha + \delta + \mu) I_h, \\ \frac{dR_h}{dt} = \delta I_h - (\gamma + \mu) R_h, \\ \frac{dS_v}{dt} = \Pi + \rho R_v - (\beta_3 I_h + \mu_v) S_v, \\ \frac{dI_v}{dt} = \beta_3 I_h S_v - (\sigma + \mu_v) I_v, \\ \frac{dR_v}{dt} = \sigma I_v - (\rho + \mu_v) R_v, \\ \frac{dB_l}{dt} = \tau_1 I_h + \tau_2 I_v - \mu_b B_l, \end{array} \right. \quad (3)$$

where $\lambda_h = \beta_2 B_l / (\kappa + B_l) + \beta_1 I_v$. With the initial

$$\begin{aligned} S_h(0) &\geq 0, \\ E_h(0) &\geq 0, \\ I_h(0) &\geq 0, \\ R_h(0) &\geq 0, \\ S_v(0) &\geq 0, \\ I_v(0) &\geq 0, \\ R_v(0) &\geq 0, \\ B_l(0) &\geq 0. \end{aligned} \quad (4)$$

3. Model Analysis

3.1. Positivity of Solutions

Theorem 1. Let $\Omega_l(0) = (S_h(0), E_h(0), I_h(0), R_h(0), S_v(0), I_v(0), R_v(0), B_l(0)) \in R_+^8 \cup \{0\}$ be the initial condition for equation (3). Then, the set of solutions $\{S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), I_v(t), R_v(t), B_l(t)\}$ of equation (3) is nonnegative for all $t \geq 0$.

Proof. Given that a set of nonnegative initial conditions $S_h(0), E_h(0), I_h(0), R_h(0), S_v(0), I_v(0), R_v(0), B_l(0)$, consider equation (3). Let $t_1 = \sup \{t > 0 : S_h(t_0) > 0, E_h(t_0) > 0, I_h(t_0) > 0, R_h(t_0) > 0, S_v(t_0) > 0, I_v(t_0) > 0, R_v(t_0) > 0, B_l(t_0) > 0, \forall t_0 \in [0, t]\}$. From the first equation of the system (3), it follows that

$$\frac{dS_h}{dt} + (\lambda_h(t) + \mu) S_h = \Lambda + \gamma R_h(t), \quad (5)$$

which can be rewritten as

$$\frac{d}{dt} \left[S_h(t) e^{\mu t + \int_0^t \lambda_h(\tau) d\tau} \right] = (\Lambda + \gamma R_h(t)) e^{\mu t + \int_0^t \lambda_h(\tau) d\tau}. \quad (6)$$

Thus,

$$S_h(t_1) e^{\mu t_1 + \int_0^{t_1} \lambda_h(\tau) d\tau} - S_h(0) = \int_0^{t_1} (\Lambda + \gamma R_h(t)) \left[e^{\mu z + \int_0^z \lambda_h(\tau) d\tau} \right] dz. \quad (7)$$

Therefore,

$$\begin{aligned} S_h(t_1) &= S_h(0) e^{-\mu t_1 - \int_0^{t_1} \lambda_h(\tau) d\tau} + \left[e^{-\mu t_1 - \int_0^{t_1} \lambda_h(\tau) d\tau} \right] \\ &\times \int_0^{t_1} \left[e^{\mu z + \int_0^z \lambda_h(\tau) d\tau} (\Lambda + \gamma R_h(z)) \right] dz \geq 0. \end{aligned} \quad (8)$$

TABLE 1: Description of variables of the leptospirosis disease model.

Variable	Description
$S_h(t)$	The susceptible individuals; individuals who are healthy but can be infected
$E_h(t)$	Latently infected individuals; individuals having the disease but not showing the symptoms
$I_h(t)$	The infectious individuals that are showing symptoms of the disease and can transmit disease to others
$R_h(t)$	Those that have recovered from the disease and have got temporary immunity
$S_v(t)$	Susceptible rodents
$I_v(t)$	Infected rodents
$R_v(t)$	Rodents that are recovered from the disease
B_l	Leptospirosis causing bacterial population

TABLE 2: Description of parameters of the leptospirosis disease model.

Parameter	Description
Λ	The recruitment rate of susceptible humans
Π	The recruitment rate of susceptible rodents
β_1	The transmission coefficient of the disease from infected rodents to susceptible humans
β_2	The transmission rate of infection from environment to human
β_3	The transmission rate of infection from infectious individuals to susceptible rodents
γ	The disease waning immunity for humans
μ	The natural death rate of the human population
μ_v	The natural death rate of rodent population
μ_b	The natural death rate of leptospirosis causing bacteria population
θ	The rate of the exposed human move to infected class
α	The death rate due to the disease infection
δ	The recovery rate from symptomatic infectious
ρ	The disease waning immunity for rodents
σ	Rate of recovery from leptospirosis rodent infection
κ	The concentration of pathogenic population in environment
τ_1	The rate of increase of bacteria by I_h
τ_2	The rate of increase of bacteria by I_v

Similarly, the fifth equation of the system (3) yields that

$$S_v(t_1) = S_l(0)e^{-\mu_l t_1 - \int_0^{t_1} \lambda_l(\tau) d\tau} + \left[e^{-\mu_l t_1 - \int_0^{t_1} \lambda_l(\tau) d\tau} \right] \times \int_0^{t_1} \left[e^{\mu_l z + \int_0^z \lambda_l(\tau) d\tau} (\Pi + \rho R_v(z)) \right] dz \geq 0. \quad (9)$$

Next, from the second equation of model (3), it follows that

$$\frac{dE_h}{dt} \geq -(\theta + \mu)E_h. \quad (10)$$

Applying the comparison theorem and variation of formula for (10) yields

$$E_h(t) \geq E_h(0)e^{-(\theta + \mu)t} \geq 0, \forall t \geq 0. \quad (11)$$

Similarly,

$$\begin{cases} I_h(t) \geq I_h(0)e^{-(\alpha + \delta + \mu)t} \geq 0, \forall t \geq 0, \\ R_h(t) \geq R_h(0)e^{-(\gamma + \mu)t} \geq 0, \forall t \geq 0, \\ I_v(t) \geq I_v(0)e^{-(\sigma + \mu_v)t} \geq 0, \forall t \geq 0, \\ R_v(t) \geq R_v(0)e^{-(\rho + \mu_v)t} \geq 0, \forall t \geq 0, \\ B_l(t) \geq B_l(0)e^{-(\mu_b)t} \geq 0, \forall t \geq 0. \end{cases} \quad (12)$$

Therefore, all solutions of the system (3) remain nonnegative for all nonnegative initial conditions. \square

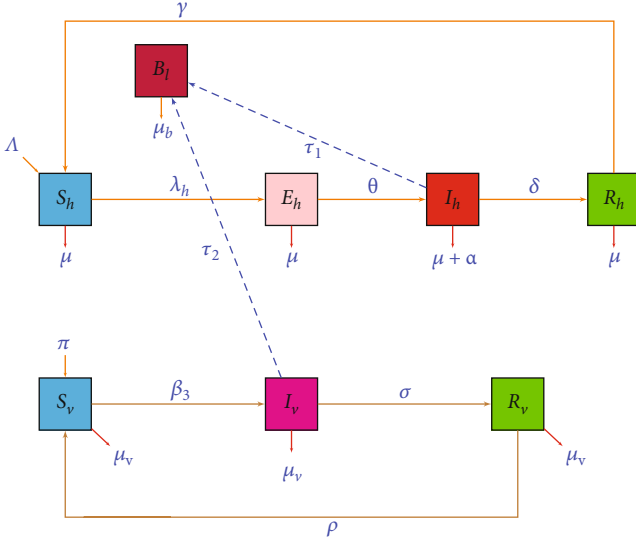


FIGURE 1: Compartmental flow diagram of leptospirosis disease transmission.

3.2. Invariant Region

Theorem 2. All solutions of the leptospirosis model equation (3) remain in $\Omega_l = \{(S_h, E_h, I_h, R_h, S_v, I_v, R_v, B_l) \in \mathbb{R}_+^8 : 0 \leq N_h(t) \leq \Lambda/\mu, 0 \leq N_v(t) \leq \Pi/\mu_v, 0 \leq B_l(t) \leq \tau^*/\mu_{bl}((\Lambda/\mu) + (\Pi/\mu_v))\}$.

Proof. From equation (1), we get

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt}. \quad (13)$$

The first four equations of the system (3) and equation (13) give rise to

$$\frac{dN_h}{dt} = \Lambda - \mu N_h - \alpha I_h. \quad (14)$$

Since the parameter, α , and the state variable I_h are non-negative in (14), we have

$$\frac{dN_h}{dt} \leq \Lambda - \mu N_h. \quad (15)$$

Let $N_h^*(t)$ be the solution of the ODE $dN_h/dt = \Lambda - \mu N_h$ with an initial condition. Then,

$$\frac{dN_h^*}{dt} = \Lambda - \mu N_h^* \text{ with } N_h^*(0) = N_{h0} \forall t \geq 0. \quad (16)$$

Thus, equation (16) has a unique solution. By applying separation of variable on the differential equation (16), we get

$$\frac{dN_h^*}{\Lambda - \mu N_h^*} = dt. \quad (17)$$

Integrating (17) on both sides yields

$$N_h^*(t) = N_h^*(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}). \quad (18)$$

We now apply the comparison theorem [19] on the differential equation (15) and then $N_h(t) \leq N_h^*(t)$. Thus,

$$N_h(t) \leq N_h^*(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}). \quad (19)$$

As $t \rightarrow \infty$, the population size $N_h \rightarrow \Lambda/\mu$. Hence,

$$N_h(t) \leq \frac{\Lambda}{\mu} \text{ for } 0 \leq N_h^*(0) \leq \frac{\Lambda}{\mu}, \forall t \geq 0. \quad (20)$$

On the other hand, if $N_h^*(0) > \Lambda/\mu$, the solution decrease to Λ/μ as $t \rightarrow \infty$.

Also, the last equation of (3) and equations (20) and (23) imply that

$$\frac{dB_l}{dt} \leq \tau^* \left(\frac{\Lambda}{\mu} + \frac{\Pi}{\mu_v} \right) - \mu_b B_l, \quad (21)$$

where $\tau^* = \max\{\tau_1, \tau_2\}$. Equation (21) yields

$$B_l(t) \leq B_l(0)e^{-\mu_b t} + \frac{\tau^*}{\mu_{bl}} \left(\frac{\Lambda}{\mu} + \frac{\Pi}{\mu_v} \right) (1 - e^{-\mu_b t}) \forall t \geq 0. \quad (22)$$

Hence,

$$\Omega_h = \left\{ (S_h, E_h, I_h, R_h, B_l) \in \mathbb{R}_+^5 : 0 \leq N_h(t) \leq \frac{\Lambda}{\mu}, 0 \leq B_l \leq \frac{\tau^*}{\mu_{bl}} \left(\frac{\Lambda}{\mu} + \frac{\Pi}{\mu_v} \right) \right\}, \quad (23)$$

is positively invariant region.

Finally, the 5th equation of (3) implies that

$$\frac{dN_v}{dt} \leq \Pi - \mu N_v, \text{ with } N_v(0) = N_{v0}. \quad (24)$$

Then, equation (24) has a unique solution. Similarly, applying the comparison theorem and integrating the differential equation (21) yield

$$N_v(t) = N_v(0)e^{-\mu_v t} + \frac{\Pi}{\mu_v}(1 - e^{-\mu_v t}). \quad (25)$$

Therefore,

$$0 \leq N_v(t) \leq \frac{\Pi}{\mu_v} \text{ for } 0 \leq N_v(0) \leq \frac{\Pi}{\mu_v}, \quad (26)$$

$$\Omega_v = \left\{ (S_v, I_v, R_v) \in \mathbb{R}_+^3 : 0 \leq N_v(t) \leq \frac{\Pi}{\mu_v} \right\}.$$

Therefore, the biologically feasible region for (3) is given by

$$\Omega_I = \Omega_h \times \Omega_v \subset R_+^5 \times R_+^3. \quad (27)$$

Therefore, every solution of the differential equation model with initial conditions in Ω_I remains in $\Omega_I \forall t \geq 0$. Thus, the region Ω_I is positively invariant with respect to the system (3). \square

3.3. Disease-Free Equilibrium. The disease-free equilibrium (DFE) of the model (3) only exists in the absence of infections. We compute DFE by setting left-hand side of the system (3) equal to zero. Since $E_h^* = I_h^* = R_h^* = I_v^* = R_v^* = B_l^* = 0$ at DFE, we have

$$\begin{cases} \Lambda + \gamma R_h^* - \left(\frac{\beta_1 B_l^*}{\kappa + B_l^*} + \beta_2 I_v^* + \mu \right) S_h^* = 0, \\ \Pi + \rho R_v^* - (\beta_3 I_l^* + \mu_v) S_v^* = 0. \end{cases} \quad (28)$$

Thus, the system (28) yields $S_h^* = \Lambda/\mu$ and $S_v^* = \Pi/\mu_v$. Therefore, the DFE point of the model (3) is given by

$$\varepsilon_{0I}^* = (S_h^*, 0, 0, 0, S_v^*, 0, 0, 0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, \frac{\Pi}{\mu_v}, 0, 0, 0 \right). \quad (29)$$

To compute the basic reproduction number for our model (3), we follow the method presented in [20]. Considering only the infective compartments $W = (E_h, I_h, I_v, B_l)$ in (3), then we have $dW/dt = F(t) - V(t)$,

$$F(t) = \begin{bmatrix} \lambda_h S_h \\ 0 \\ \beta_3 S_v I_h \\ 0 \end{bmatrix}, \quad (30)$$

$$V(t) = \begin{bmatrix} \varepsilon_1 E_h \\ -\theta E_h + \varepsilon_2 I_h \\ \varepsilon_4 I_v \\ -\tau_1 I_h - \tau_2 I_v + \mu_{bl} B_l \end{bmatrix},$$

where

$$\begin{aligned} \lambda_h &= \frac{\beta_2 B_l}{\kappa + B_l} + \beta_1 I_v, \\ \varepsilon_1 &= \theta + \mu, \\ \varepsilon_2 &= \alpha + \delta + \mu, \end{aligned} \quad (31)$$

The Jacobian matrices of $F(t)$ and of $V(t)$ at ε_{0I}^* are, respectively, given by

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Pi}{\mu \kappa} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_3 \Pi}{\mu_v} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad (32)$$

$$V = \begin{bmatrix} \varepsilon_1 & 0 & 0 & 0 \\ -\theta & \varepsilon_2 & 0 & 0 \\ 0 & 0 & \varepsilon_3 & 0 \\ 0 & -\tau_1 & -\tau_2 & \mu_b \end{bmatrix}.$$

Therefore, the next-generation matrix of the model is given by

$$3G = FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Lambda}{\mu \kappa} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_3 \Pi}{\mu_v} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\varepsilon_1} & 0 & 0 & 0 \\ \frac{\theta}{\varepsilon_1 \varepsilon_2} & \frac{1}{\varepsilon_2} & 0 & 0 \\ 0 & 0 & \frac{1}{\varepsilon_4} & 0 \\ \frac{\theta \tau_1}{\varepsilon_1 \varepsilon_2 \mu_b} & \frac{\tau_1}{\varepsilon_2 \mu_b} & \frac{\tau_2}{\varepsilon_4 \mu_b} & \frac{1}{\mu_b} \end{bmatrix}$$

$$= \begin{bmatrix} y_1 & y_2 & y_3 & y_4 \\ 0 & 0 & 0 & 0 \\ y_5 & y_6 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad (33)$$

where

$$\begin{aligned} y_1 &= \frac{\beta_2 \Lambda \theta \tau_1}{\kappa \mu \mu_b \varepsilon_1 \varepsilon_2}, \\ y_2 &= \frac{\beta_2 \Lambda \tau_1}{\kappa \mu \mu_b \varepsilon_2}, \\ y_3 &= \frac{\Lambda (\beta_2 \tau_2 + \beta_1 \kappa \mu_b)}{\kappa \mu \mu_b \varepsilon_4}, \\ y_4 &= \frac{\beta_2 \Lambda}{\kappa \mu \mu_b}, \\ y_5 &= \frac{\beta_3 \Pi \theta}{\mu_v \varepsilon_1 \varepsilon_2}, \\ y_6 &= \frac{\beta_3 \Pi}{\mu_v \varepsilon_2}. \end{aligned} \quad (34)$$

Considering $|G - \lambda I| = 0$, we have

$$\begin{vmatrix} y_1 - \lambda & y_2 & y_3 & y_4 \\ 0 & -\lambda & 0 & 0 \\ y_5 & y_6 & -\lambda & 0 \\ 0 & 0 & 0 & -\lambda \end{vmatrix} = 0 \implies \lambda^2(\lambda^2 - \lambda y_1 - y_3 y_5) = 0. \quad (35)$$

Thus,

$$\begin{aligned} \lambda_1 &= \lambda_2 = 0, \\ \lambda_3 &= \frac{1}{2} \left(y_1 - \sqrt{y_1^2 + 4y_3 y_5} \right) < 0, \\ \lambda_4 &= \frac{1}{2} \left(y_1 + \sqrt{y_1^2 + 4y_3 y_5} \right) > 0, \end{aligned} \quad (36)$$

are the eigenvalues of the matrix G. Therefore, the basic reproduction number of the model is given by

$$R_0 = \rho(G) = \frac{1}{2} \left(\frac{\beta_2 \Lambda \theta \tau_1}{\kappa \mu \mu_b \varepsilon_1 \varepsilon_2} + \sqrt{\left(\frac{\beta_2 \Lambda \theta \tau_1}{\kappa \mu \mu_b \varepsilon_1 \varepsilon_2} \right)^2 + 4 \frac{\beta_3 \Pi \Lambda \theta (\beta_2 \tau_2 + \beta_1 \kappa \mu_b)}{\kappa \mu \mu_v \mu_b \varepsilon_1 \varepsilon_2 \varepsilon_4}} \right). \quad (37)$$

The basic reproduction number R_0 can be written in terms of the basic reproduction numbers corresponding to the vector transmission R_v , human transmission R_h , and environment-to-human transmission R_b as

$$R_0 = \frac{1}{2} \left[R_b + \sqrt{R_b^2 + 4R_{hv}} \right], \quad (38)$$

where $R_b = \beta_2 \Lambda \theta \tau_1 / \kappa \mu \mu_b \varepsilon_1 \varepsilon_2$ and $R_{hv} = R_h + R_v$ with $R_h = \beta_3 \beta_2 \Pi \Lambda \theta \tau_2 / \kappa \mu \mu_v \mu_b \varepsilon_1 \varepsilon_2 \varepsilon_4$ and $R_v = \beta_3 \beta_1 \Pi \Lambda \theta / \mu \mu_v \varepsilon_1 \varepsilon_2 \varepsilon_4$.

3.4. Local Stability of the Disease-Free Equilibrium

Theorem 3. *The disease-free equilibrium point, ε_{0l}^* , is locally asymptotically stable whenever $R_0 < 1$ and unstable otherwise.*

Proof. The Jacobian matrices of the model (3) at DFE ε_{0l}^* is given by

$$J(\varepsilon_{0l}^*) = \begin{bmatrix} -\mu & 0 & 0 & \gamma & 0 & \frac{-\beta_1 \Lambda}{\mu} & 0 & \frac{-\beta_2 \Lambda}{\mu \kappa} \\ 0 & -\varepsilon_1 & 0 & 0 & 0 & \frac{\beta_1 \Lambda}{\mu} & 0 & \frac{\beta_2 \Lambda}{\mu \kappa} \\ 0 & \theta & -\varepsilon_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & -\varepsilon_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{-\beta_3 \Pi}{\mu_v} & 0 & -\mu_v & 0 & \rho & 0 \\ 0 & 0 & \frac{\beta_3 \Pi}{\mu_v} & 0 & 0 & -\varepsilon_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma & -\varepsilon_5 & 0 \\ 0 & 0 & \tau_1 & 0 & 0 & \tau_2 & 0 & -\mu_b \end{bmatrix}, \quad (39)$$

where $\varepsilon_1 = \theta + \mu$, $\varepsilon_2 = \alpha + \delta + \mu$, $\varepsilon_3 = \gamma + \mu$, $\varepsilon_4 = \sigma + \mu_v$, and $\varepsilon_5 = \rho + \mu_v$.

Expanding the determinant of the characteristic equation $|J(\varepsilon_{0l}^*) - \lambda I| = 0$ by the first, fourth, fifth, and seventh columns, respectively, we obtain four of the eigenvalues of $J(\varepsilon_{0l}^*)$: $\lambda_1 = -\mu$, $\lambda_2 = -(\gamma + \mu)$, $\lambda_3 = -\mu_v$, and $\lambda_4 = -(\rho + \mu_v)$. The other four eigenvalues will be determined from the following polynomial equation:

$$\begin{vmatrix} -\varepsilon_1 - \lambda & 0 & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Lambda}{\mu \kappa} \\ \theta & -\varepsilon_2 - \lambda & 0 & 0 \\ 0 & \frac{\beta_3 \Pi}{\mu_v} & -\varepsilon_4 - \lambda & 0 \\ 0 & \tau_1 & \tau_2 & -\mu_b - \lambda \end{vmatrix} = 0, \quad (40)$$

$$\implies P_4(\lambda) = \lambda^4 + A_1 \lambda^3 + A_2 \lambda^2 + A_3 \lambda + A_4 = 0,$$

where

$$A_1 = \varepsilon_1 + \varepsilon_2 + \varepsilon_4 + \mu_b,$$

$$A_2 = \varepsilon_1 \mu_b + \varepsilon_1 \varepsilon_2 + \varepsilon_1 \varepsilon_4 + \varepsilon_2 \mu_b + \varepsilon_4 \mu_b + \varepsilon_2 \varepsilon_4,$$

$$\begin{aligned} A_3 &= \varepsilon_1 \varepsilon_2 \mu_b + \varepsilon_1 \varepsilon_4 \mu_b + \varepsilon_1 \varepsilon_2 \varepsilon_4 + \varepsilon_2 \varepsilon_4 \mu_b - \frac{\beta_2 \Lambda \tau_1 \theta}{\mu \kappa} - \frac{\beta_1 \beta_3 \Lambda \Pi \theta}{\mu \mu_v} \\ &= \varepsilon_4 \mu_b (\varepsilon_1 + \varepsilon_2) + \varepsilon_1 \varepsilon_2 \mu_b \left(1 - \frac{\beta_2 \Lambda \theta \tau_1}{\kappa \mu \mu_b \varepsilon_1 \varepsilon_2} \right) + \varepsilon_1 \varepsilon_2 \varepsilon_4 \left(1 - \frac{\beta_1 \beta_3 \Lambda \Pi \theta}{\mu \mu_v \varepsilon_1 \varepsilon_2 \varepsilon_4} \right) \\ &= \varepsilon_4 \mu_b (\varepsilon_1 + \varepsilon_2) + \varepsilon_1 \varepsilon_2 \mu_b (1 - R_b) + \varepsilon_1 \varepsilon_2 \varepsilon_4 (1 - R_v), \end{aligned}$$

$$\begin{aligned} A_4 &= \varepsilon_1 \varepsilon_2 \varepsilon_4 \mu_b - \left(\frac{\beta_2 \Lambda \theta \tau_1 \varepsilon_4}{\mu \kappa} + \frac{\beta_1 \beta_3 \Lambda \Pi \theta \mu_b}{\mu \mu_v} + \frac{\beta_2 \beta_3 \Lambda \Pi \theta \tau_2}{\kappa \mu \mu_v} \right) \\ &= \varepsilon_1 \varepsilon_2 \varepsilon_4 \mu_b - \left(\frac{\beta_2 \Lambda \theta \tau_1 \varepsilon_4}{\mu \kappa} + \frac{\beta_3 \Pi \Lambda \theta (\beta_2 \tau_2 + \beta_1 \kappa \mu_b)}{\kappa \mu \mu_v} \right) \\ &= \varepsilon_1 \varepsilon_2 \varepsilon_4 \mu_b \left[1 - \left(\frac{\beta_2 \Lambda \theta \tau_1 \varepsilon_4}{\mu \kappa} + \frac{\beta_3 \Pi \Lambda \theta (\beta_2 \tau_2 + \beta_1 \kappa \mu_b)}{\kappa \mu \mu_v} \right) \right] \\ &= \varepsilon_1 \varepsilon_2 \varepsilon_4 \mu_b [1 - (R_b + R_{hv})]. \end{aligned} \quad (41)$$

Since all the model parameters are positive, the above four eigenvalues of the matrix $J(\varepsilon_{0l}^*)$ are negative real numbers. To ensure that all the remaining four eigenvalues of the matrix have negative real parts, the Routh-Hurwitz stability criterion (see Theorem 5.1 in [21]) requires $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, $A_4 > 0$, $A_1A_2 - A_3 > 0$, and $A_1A_2A_3 - (A_3^2 + A_1^2A_4) > 0$. Among these, $A_1 > 0$ and $A_2 > 0$ are obvious.

Similarly,

$$\begin{aligned} A_1A_2 - A_3 &= A_2(\varepsilon_4 + \mu_b) + \varepsilon_2^2(\varepsilon_1 + \varepsilon_4 + \mu_b) \\ &\quad + \varepsilon_1(\varepsilon_1\mu_b + \varepsilon_1\varepsilon_2 + \varepsilon_2\mu_b + \varepsilon_1\varepsilon_4 + \varepsilon_2\varepsilon_4) \quad (42) \\ &\quad + \frac{\beta_2\Lambda\tau_1\theta}{\mu\kappa} + \frac{\beta_1\beta_3\Lambda\Pi\theta}{\mu\mu_\nu} > 0. \end{aligned}$$

The other three inequalities hold when $R_0 > 1$; the details are described as follows.

Since $R_b < R_0$, $1 - R_b > 0$ for $R_0 < 1$. Similarly, $1 - R_\nu = (1 - R_0)[1 - R_b + R_0] + R_b + R_h > 0$ for $R_0 < 1$. Thus,

$$A_3 = \varepsilon_4\mu_b(\varepsilon_1 + \varepsilon_2) + \varepsilon_1\varepsilon_2\mu_b(1 - R_b) + \varepsilon_1\varepsilon_2\varepsilon_4(1 - R_\nu) > 0 \text{ if } R_0 < 1. \quad (43)$$

Next,

$$A_4 = \varepsilon_1\varepsilon_2\varepsilon_4\mu_b[1 - (R_b + R_{hv})] = \varepsilon_1\varepsilon_2\varepsilon_4\mu_b(1 - R_0)[1 - R_b + R_0] > 0 \text{ if } R_0 < 1. \quad (44)$$

Finally, we need to show that $A_1A_2A_3 - (A_3^2 + A_1^2A_4) > 0$. Let

$$\begin{aligned} \phi_0 &= \varepsilon_1\mu_b, \\ \phi_1 &= \varepsilon_1\varepsilon_2, \\ \phi_2 &= \varepsilon_1\varepsilon_4, \\ \phi_3 &= \varepsilon_2\mu_b, \\ \phi_4 &= \varepsilon_4\mu_b, \\ \phi_5 &= \varepsilon_2\varepsilon_4, \\ \psi_0 &= \varepsilon_4\mu_b(\varepsilon_1 + \varepsilon_2), \\ \psi_1 &= \varepsilon_1\varepsilon_2\mu_b(1 - R_b), \\ \psi_3 &= \varepsilon_1\varepsilon_2\varepsilon_4(1 - R_\nu). \end{aligned} \quad (45)$$

Then, $A_2 = \phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5$, and $A_3 = \psi_0 + \psi_1 + \psi_2$.

Now,

$$\begin{aligned} A_1A_2A_3 &= \varepsilon_1[\psi_0(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_1(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_2(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5)] \\ &\quad + \varepsilon_2[\psi_0(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_1(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_2(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5)] \\ &\quad + \varepsilon_4[\psi_0(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_1(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_2(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5)] \\ &\quad + \mu_b[\psi_0(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_1(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) \\ &\quad + \psi_2(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5)], \end{aligned}$$

$$\begin{aligned} A_3^2 + A_1^2A_4 &= (\psi_0 + \psi_1 + \psi_2)^2 + (\varepsilon_1 + \varepsilon_2 + \varepsilon_4 + \mu_b)^2\varepsilon_1\varepsilon_2\varepsilon_4\mu_b[1 - R_b - R_{hv}] \\ &= \psi_0^2 + \psi_1^2 + \psi_2^2 + 2(\psi_0\psi_1 + \psi_0\psi_2 + \psi_1\psi_2) \\ &\quad + [\varepsilon_1^2 + \varepsilon_2^2 + \varepsilon_4^2 + \mu_b^2 + 2(\varepsilon_1\varepsilon_2 + \varepsilon_1\varepsilon_4 + \varepsilon_1\mu_b + \varepsilon_2\varepsilon_4 \\ &\quad + \varepsilon_2\mu_b + \varepsilon_4\mu_b)]\varepsilon_1\varepsilon_2\varepsilon_4\mu_b[1 - R_b - R_{hv}]. \end{aligned} \quad (46)$$

After manipulating the parameters algebraically, we obtain

$$A_1A_2A_3 - (A_3^2 + A_1^2A_4) = \Omega_0 + \Omega_1, \quad (47)$$

where

$$\begin{aligned} \Omega_0 &= \varepsilon_1[\psi_0(\phi_0 + \phi_1 + \phi_2 + \phi_4) + \psi_1(\phi_0 + \phi_1 + \phi_4) + \psi_2(\phi_1 + \phi_2 + \phi_4)] \\ &\quad + \varepsilon_2[\psi_1(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4) + \psi_2(\phi_0 + \phi_1 + \phi_2 + \phi_4 + \phi_5)] \\ &\quad + \varepsilon_4[\psi_0(\phi_1 + \phi_2 + \phi_4) + \psi_2(\phi_1 + \phi_2 + \phi_4 + \phi_5)] \\ &\quad + \mu_b[\psi_0(\phi_2 + \phi_3 + \phi_4 + \phi_5) + \psi_1(\phi_0 + \phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5) > 0], \\ \Omega_1 &= \varepsilon_1[\phi_3R_b(A_3 + \psi_0) + \phi_5A_3R_\nu] \\ &\quad + [(\varepsilon_1^2 + \varepsilon_2^2 + A_2)\phi_1\phi_4(1 + R_h) + \phi_4(\phi_5^2 + \phi_0^2 + \varepsilon_2A_2) \\ &\quad + (\varepsilon_4^2 + \mu_b^2 + A_2)\phi_1\phi_4(R_b + R_{hv}) > 0]. \end{aligned} \quad (48)$$

Clearly, $\Omega_0 > 0$ and $\Omega_1 > 0$.

Therefore, $A_1A_2A_3 - (A_3^2 + A_1^2A_4) > 0$ if $R_0 < 1$. This completes the proof. \square

3.5. Global Stability of the Disease-Free Equilibrium. The method illustrated in [22, 23] is used to investigate the global asymptotic stability (GAS) of DFE point of the model. Firstly, the model (3) must be written in the form:

$$\begin{cases} \frac{dX}{dt} = F(X, Y), \\ \frac{dY}{dt} = G(X, Y), G(X, 0) = 0. \end{cases} \quad (49)$$

where $X = (S_h, R_h, S_\nu, R_\nu)$ represents the number of uninfected individuals and $Y = (E_h, I_h, I_\nu, B_l)$ denotes the number of infected individuals. Let X^* be the disease-free equilibrium of the system $dX/dt = F(X, 0)$. Then, $X^* = (\Lambda/\mu, 0, \Pi/\mu_\nu, 0)$. The DFE point of the model $\varepsilon_{0l}^* = (X^*, 0) = (\Lambda/\mu, 0, 0, 0, \Pi/\mu_\nu, 0, 0, 0)$ is guaranteed to be GAS if $R_0 < 1$ (which

is locally asymptotically stable (LAS)), and the following two conditions H_1 and H_2 hold:

H_1 : For $dX/dt = F(X, 0)$, X^* is globally asymptotically stable.

H_2 : $G(X, Y) = AY - G^*(X, Y)$, $G^*(X, Y) \geq 0$, $\forall (X, Y) \in \Omega_\Gamma$, where $A = \partial G(\varepsilon_{0l}^*)/\partial Y$ is an M -matrix and Ω_Γ is the region where the model makes biological sense. If the model equation (3) satisfies the above two conditions, then the following theorem holds.

Theorem 4. *The disease-free equilibrium point, $\varepsilon_{0l}^* = (\Lambda/\mu, 0, 0, 0, \Pi/\mu_v, 0, 0, 0)$, is GAS for the model (3) provided that $R_0 < 1$ (LAS) and conditions H_1 and H_2 hold.*

Proof. We only need to show that the conditions H_1 and H_2 hold when $R_0 < 1$. Considering the system (3), we have

$$F(X, 0) = \begin{bmatrix} S_h'(t) \\ R_h'(t) \\ S_v'(t) \\ R_v'(t) \end{bmatrix} = \begin{bmatrix} \Lambda + \gamma R_h - \mu S_h \\ -(\gamma + \mu)R_h \\ \Pi + \rho R_v - \mu_v S_v \\ -(\rho + \mu_v)R_v \end{bmatrix}, X^* = \left(\frac{\Lambda}{\mu}, 0, \frac{\Pi}{\mu_v}, 0 \right). \quad (50)$$

Note that the second and the fourth equations of the system (50) are first-order linear ODEs, and their solutions are given by, respectively,

$$R_h(t) = R_{h,0} e^{-(\gamma+\mu)t}, \quad (51)$$

$$R_v(t) = R_{v,0} e^{-(\rho+\mu_v)t}. \quad (52)$$

Comparing equation (51) and the first equation of the system (50) yields

$$S_h'(t) = \Lambda + \gamma R_{v,0} e^{-(\rho+\mu_v)t} - \mu S_h(t). \quad (53)$$

Solving the first-order linear ODE (53), its solution is found as

$$S_h(t) = \frac{\Lambda}{\mu} + S_{h,0} e^{-\mu t} - R_{h,0} e^{-(\gamma+\mu)t}. \quad (54)$$

Similarly,

$$S_v'(t) = \Pi + \rho R_{v,0} e^{-(\rho+\mu_v)t} - \mu_v S_v(t), \quad (55)$$

and its solution is given by

$$S_v(t) = \frac{\Pi}{\mu_v} + S_{v,0} e^{-\mu_v t} - R_{v,0} e^{-(\rho+\mu_v)t}. \quad (56)$$

Now, suppose that the time, $t \rightarrow \infty$, we need to show that $X \rightarrow X^*$. Obviously, $R_h(t) \rightarrow 0$, $R_v(t) \rightarrow 0$ as $t \rightarrow \infty$. Thus, $S_h(t) \rightarrow \Lambda/\mu$, $S_v(t) \rightarrow \Pi/\mu_v$ as $t \rightarrow \infty$, regardless of the values of $S_h(0)$, $R_h(0)$ and $S_v(0)$, $R_v(0)$, respectively. Thus, all points with respect to this condition

converge at $X_0^* = (\Lambda/\mu, 0, \Pi/\mu_v, 0)$. Hence, $X^* = (\Lambda/\mu, 0, \Pi/\mu_v, 0)$ is GAS.

Next, we have

$$G(X, Y) = \begin{bmatrix} G_1(X, Y) \\ G_2(X, Y) \\ G_3(X, Y) \\ G_4(X, Y) \end{bmatrix} = \begin{bmatrix} \left(\frac{\beta_2 B_l}{\kappa + B_l} + \beta_1 I_v \right) S_h - (\theta + \mu) E_h \\ \theta E_h - (\alpha + \delta + \mu) I_h \\ \beta_3 S_v I_h - (\sigma + \mu_v) I_v \\ \tau_1 I_h + \tau_2 I_v - \mu_b B_l \end{bmatrix}. \quad (57)$$

We can then obtain

$$A = \frac{\partial G(X^*, 0)}{\partial Y} = \begin{bmatrix} -(\theta + \mu) & 0 & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Lambda}{\kappa \mu} \\ \theta & -(\alpha + \delta + \mu) & 0 & 0 \\ 0 & \frac{\beta_3 \Pi}{\mu_v} & -(\sigma + \mu_v) & 0 \\ 0 & \tau_1 & \tau_2 & -\mu_b \end{bmatrix}, \quad (58)$$

which is clearly an M -matrix

$$G^*(X, Y) = AY - G(X, Y) = \begin{bmatrix} \beta_1 I_v \left(\frac{\Lambda}{\mu} - S_h \right) + \frac{\beta_2 B_l}{\kappa} \left(\frac{\Lambda}{\mu} - \frac{\kappa S_h}{\kappa + B_l} \right) \\ 0 \\ \beta_3 I_h \left(\frac{\Pi}{\mu_v} - S_v \right) \\ 0 \end{bmatrix}. \quad (59)$$

Since all parameters are nonnegative, $0 \leq (\kappa S_h / (\kappa + B_l)) \leq S_h \leq \Lambda/\mu$ ($(\kappa / (\kappa + B_l)) \leq 1$), $0 \leq S_v \leq \Pi/\mu_v$. It follows that $(\Lambda/\mu - S_h) \geq 0$, $((\Lambda/\mu) - (\kappa S_h / (\kappa + B_l))) \geq 0$, and $(\Pi/\mu_v - S_v) \geq 0$.

Hence, $G^*(X, Y) \geq 0 \forall (X, Y) \in \Gamma$. Therefore, the disease-free equilibrium point, $\varepsilon_{0l}^* = (\Lambda/\mu, 0, 0, 0, \Pi/\mu_v, 0, 0, 0)$, of the model (3) is globally asymptotically stable. This completes the proof. \square

Remark 5. The model (3) does not exhibit a backward bifurcation at $R_0 = 1$ when $R_0 < 1$, since DFE is the only stable (or positive) equilibrium point for $R_0 < 1$ in the Theorem 4.

The backward bifurcation has been observed in the leptospirosis epidemic model [16].

3.6. Endemic Equilibrium (EE). The EE point of our model is computed by setting the system of differential equations in (3) to zero. Thus, it is obtained by $dS_h^*/dt = dE_h^*/dt = dI_h^*/dt = dR_h^*/dt = dS_v^*/dt = dI_v^*/dt = dR_v^*/dt = dB_l^*/dt = 0$, which is given by $E_l^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, I_v^*, R_v^*, B_l^*)$, where all disease states are considered to be positive, and thus, I_h^* must be greater than zero for all the other states to be positive.

Now, by setting the system of equations in (3) to zero at steady state, we have

$$\left\{ \begin{array}{l} \frac{dS_h^*}{dt} = \Lambda + \gamma R_h^* - \left(\beta_2 \frac{B_l^*}{\kappa + B_l^*} + \beta_1 I_v^* + \mu \right) S_h^* = 0, \\ \frac{dE_h^*}{dt} = \left(\beta_2 \frac{B_l^*}{\kappa + B_l^*} + \beta_1 I_v^* \right) S_h^* - (\theta + \mu) E_h^* = 0, \\ \frac{dI_h^*}{dt} = \theta E_h^* - (\alpha + \delta + \mu) I_h^* = 0, \\ \frac{dR_h^*}{dt} = \delta I_h^* - (\gamma + \mu) R_h^* = 0, \\ \frac{dS_v^*}{dt} = \Pi + \rho R_v^* - (\beta_3 I_h^* + \mu_v) S_v^* = 0, \\ \frac{dI_v^*}{dt} = \beta_3 I_h^* S_v^* - (\sigma + \mu_v) I_v^* = 0, \\ \frac{dR_v^*}{dt} = \sigma I_v^* - (\rho + \mu_v) R_v^* = 0, \\ \frac{dB_l^*}{dt} = \tau_1 I_h^* + \tau_2 I_v^* - \mu_b B_l^* = 0. \end{array} \right. \quad (60)$$

Due to the complexity of the model system (3), all other state variables at the steady state are expressed in terms of

the steady state of I_h which is denoted as I_h^* . Accordingly, we found as

$$E_l^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, I_v^*, R_v^*, B_l^*), = \left(\frac{\Lambda + \gamma R_h^*}{\lambda^* + \mu}, \frac{\varepsilon_2}{\theta} I_h^*, I_h^*, \frac{\delta}{\varepsilon_3} I_h^*, \frac{\Pi + \rho R_v^*}{\beta_3 I_h^* + \mu_v}, \frac{\beta_3 \Pi \varepsilon_5 I_h^*}{\mu_v (\varepsilon_4 \varepsilon_5 + \beta_3 \varepsilon_6 I_h^*)}, \frac{\beta_3 \Pi \sigma I_h^*}{\mu_v (\varepsilon_4 \varepsilon_5 + \beta_3 \varepsilon_6 I_h^*)}, \frac{\tau_1 I_h^* + \tau_2 I_v^*}{\mu_b} \right), \quad (61)$$

where

$$\begin{aligned} \varepsilon_1 &= \theta + \mu, \\ \varepsilon_2 &= \alpha + \delta + \mu, \\ \varepsilon_3 &= \gamma + \mu, \\ \varepsilon_4 &= \sigma + \mu_v, \\ \varepsilon_5 &= \rho + \mu_v, \\ \varepsilon_6 &= \sigma + \rho + \mu_v. \end{aligned} \quad (62)$$

By combining the equations of (61) and the first and second equations of the model system (3), we obtained a septic (or heptic) polynomial equation for I_h^* which is described as follows:

$$P(I_h^*) = I_h^* \left(A(I_h^*)^6 + B(I_h^*)^5 + C(I_h^*)^4 + D(I_h^*)^3 + E(I_h^*)^2 + F I_h^* + G \right) = 0, \quad (63)$$

where

$$A = -[\Psi_3 \Psi_7 \eta_4 + \Psi_7^2 (\theta + \mu) (\alpha + \delta + \mu) (\gamma + \mu) \mu] < 0 \text{ always,}$$

$$B = -\eta_4 (\Psi_2 \Psi_7 + \Psi_3 \Psi_6) + \Psi_7 \eta_3 (\theta + \mu) (\alpha + \delta + \mu) (\gamma + \mu) \mu \\ \cdot [\eta_3^2 \kappa \mu_b (R_b - 1) + \eta_3 \tau_1 (\sigma + \mu_v) (\rho + \mu_v) \mu_v (R_v - 1) \\ - \eta_3 (\eta_2 \tau_1 + \eta_1 \tau_2) - \Psi_6] < 0 \text{ if } R_0 < 1,$$

$$C = -\eta_4 (\Psi_1 \Psi_7 + \Psi_2 \Psi_6 + \Psi_3 \Psi_5) + \Psi_7 \Omega_2 \\ - \Psi_7 \Psi_5 (\theta + \mu) (\alpha + \delta + \mu) (\gamma + \mu) \mu + \Psi_6 \Omega_3 \\ = -\eta_4 (\Psi_1 \Psi_7 + \Psi_2 \Psi_6 + \Psi_3 \Psi_5) + \Psi_6 \Omega_3 \\ + (\gamma + \mu) (\theta + \mu) (\alpha + \delta + \mu) \mu [\eta_2 \eta_3 \kappa \mu_b 2 (R_b - 1) \\ + (\sigma + \mu_v) (\rho + \mu_v) \mu_v (R_v - 2) (\eta_3 \kappa \mu_b + \eta_2 \tau_1 + \eta_1 \tau_2)] < 0 \text{ if } R_0 < 1,$$

$$D = -\eta_4 (\Psi_1 \Psi_6 + \Psi_2 \Psi_5 + \Psi_3 \Psi_4) \\ + \Psi_7 [\Omega_1 - \Psi_4 (\theta + \mu) (\alpha + \delta + \mu) (\gamma + \mu) \mu] + \Psi_6 \Omega_2 + \Psi_5 \Omega_3,$$

$$E = -\eta_4 (\Psi_1 \Psi_5 + \Psi_2 \Psi_4) + \Psi_6 \Omega_1 + \Psi_5 \Omega_2 + \Psi_4 \Omega_3,$$

$$F = -\eta_4 \Psi_1 \Psi_4 + \Psi_5 \Omega_1 + \Psi_4 \Omega_2, \quad G = \Psi_4 \Omega_1 < 0 \text{ if } R_0 < 1, \quad (64)$$

with

$$\eta_1 = \Pi\beta_3(\rho + \mu_v),$$

$$\eta_2 = \mu_v(\sigma + \mu_v)(\rho + \mu_v),$$

$$\eta_3 = \beta_3\mu_v(\sigma + \rho + \mu_v),$$

$$\eta_4 = \theta\gamma(\alpha + \mu) + \mu(\alpha + \delta + \mu)(\theta + \gamma + \mu),$$

$$\Psi_1 = \eta_2(\beta_2(\eta_2\tau_1 + \eta_1\tau_2) + \beta_1\kappa\eta_1\mu_b),$$

$$\Psi_2 = (\eta_2\tau_1 + \eta_1\tau_2)(\beta_2\eta_3 + \beta_1\eta_1) + \eta_3(\beta_2\eta_2\tau_1 + \beta_1\kappa\eta_1\mu_b),$$

$$\Psi_3 = \eta_3\tau_1(\beta_2\eta_3 + \beta_1\eta_1),$$

$$\Psi_4 = \kappa\eta_2^2\mu_b,$$

$$\Psi_5 = 2\kappa\eta_2\eta_3\mu_b + \eta_2(\eta_2\tau_1 + \eta_1\tau_2),$$

$$\Psi_6 = \eta_3(\kappa\eta_3\mu_b + 2\eta_2\tau_1 + \eta_1\tau_2),$$

$$\Psi_7 = \eta_3^2\tau_1,$$

$$\begin{aligned}\Omega_1 &= (\gamma + \mu)[\Psi_1\Lambda\theta - \Psi_4(\theta + \mu)(\alpha + \delta + \mu)\mu] \\ &= \eta_2\kappa\mu_b\mu_v\mu(\theta + \mu)(\alpha + \delta + \mu)(\gamma + \mu) \\ &\quad \cdot (\sigma + \mu_v)(\rho + \mu_v)(R_0 - 1)(R_0 - R_b + 1),\end{aligned}$$

$$\begin{aligned}\Omega_2 &= (\gamma + \mu)[\Psi_2\Lambda\theta - \Psi_5(\theta + \mu)(\alpha + \delta + \mu)\mu] \\ &= (\gamma + \mu)(\theta + \mu)(\alpha + \delta + \mu)\mu[\eta_2\eta_3\kappa\mu_b(2R_b - 1) \\ &\quad + (\sigma + \mu_v)(\rho + \mu_v)\mu_v(R_v - 1)(\eta_3\kappa\mu_b + \eta_2\tau_1 + \eta_1\tau_2)],\end{aligned}$$

$$\begin{aligned}\Omega_3 &= (\gamma + \mu)[\Psi_3\Lambda\theta - \Psi_6(\theta + \mu)(\alpha + \delta + \mu)\mu] \\ &= \eta_3(\theta + \mu)(\alpha + \delta + \mu)\mu[\eta_3^2\kappa\mu_b(R_b - 1) \\ &\quad + \eta_3\tau_1(\sigma + \mu_v)(\rho + \mu_v)\mu_v(R_v - 1) - (\eta_2\tau_1 + \eta_1\tau_2)].\end{aligned}\tag{65}$$

3.7. Local Stability of the Endemic Equilibrium

Theorem 6. *The endemic equilibrium E_l^* of the model (3) is locally asymptotically stable for $R_0 > 1$ (but near 1) (based on Theorem 4.1 by [24]).*

Proof. Now, consider the system of equations (3). To investigate the nature of the bifurcation, we use the method introduced in Theorem 4.1 by [24]. To apply this method, the following simplification and change of variables are made first of all. Let $S_h = x_1$, $E_h = x_2$, $I_h = x_3$, $R_h = x_4$, $S_v = x_5$, $I_v = x_6$, $R_v = x_7$, and $B_l = x_8$.

By using vector notation,

$$X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T.\tag{66}$$

The model system (3) can be written as

$$\frac{dX}{dt} = F(X),\tag{67}$$

where $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)^T$.

The following system of differential equations is established:

$$\left\{\begin{aligned}\frac{dx_1}{dt} &= \Lambda + \gamma x_4 - \left(\frac{\beta_2 x_8}{\kappa + x_8} + \beta_1 x_6 + \mu\right)x_1 = f_1, \\ \frac{dx_2}{dt} &= \left(\frac{\beta_2 x_8}{\kappa + x_8} + \beta_1 x_6 + \mu\right)x_1 - (\theta + \mu)x_2 = f_2, \\ \frac{dx_3}{dt} &= \theta x_2 - (\alpha + \delta + \mu)x_3 = f_3, \\ \frac{dx_4}{dt} &= \delta x_3 - (\gamma + \mu)x_4 = f_4, \\ \frac{dx_5}{dt} &= \Pi + \rho x_7 - (\beta_3 x_3 + \mu_v)x_5 = f_5, \\ \frac{dx_6}{dt} &= \beta_3 x_3 x_5 - (\sigma + \mu_v)x_6 = f_6, \\ \frac{dx_7}{dt} &= \sigma x_6 - (\rho + \mu_v)x_7 = f_7, \\ \frac{dx_8}{dt} &= \tau_1 x_3 + \tau_2 x_6 - \mu_b x_8 = f_8.\end{aligned}\right.\tag{68}$$

Choosing β_1^* as bifurcation parameter and solving for β_1^* from equation (37) when $R_0 = 1$, we obtained as

$$\beta_1^* = \frac{\kappa\mu_b\mu_v\varepsilon_1\varepsilon_2\varepsilon_4 - \beta_3\Pi\Lambda\theta(\tau_1\kappa\mu_b + \beta_2\Pi\tau_2)}{\beta_3\Pi\Lambda\theta\kappa\mu_b}.\tag{69}$$

Considering $\beta_1 = \beta_1^*$, the Jacobian of the system (68) evaluated at $(\varepsilon_0^l, \beta_1^*)$ is given by

$$J(\varepsilon_0^l, \beta_1^*) = \begin{bmatrix} -\mu & 0 & 0 & \gamma & 0 & -J_1 & 0 & -J_2 \\ 0 & -J_3 & 0 & 0 & 0 & J_1 & 0 & J_2 \\ 0 & \theta & -J_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & -J_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & -J_6 & 0 & -\mu_v & 0 & \rho & 0 \\ 0 & 0 & -J_6 & 0 & 0 & -J_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma & 0 & 0 \\ 0 & 0 & \tau_1 & 0 & 0 & \tau_2 & 0 & -\mu_b \end{bmatrix},\tag{70}$$

where $J_1 = \beta_1^* \Lambda / \mu$, $J_2 = \beta_2 \Lambda / \kappa \mu$, $J_3 = \theta + \mu$, $J_4 = \alpha + \delta + \mu$, $J_5 = \gamma + \mu$, $J_6 = \beta_3 \Pi / \mu_v$, $J_7 = \sigma + \mu_v$, and $J_8 = \rho + \mu_v$.

The characteristic polynomial equation of the matrix $J(\varepsilon_{0l}^*, \beta_1^*)$ is given by

$$P^*(\lambda) = \lambda(\lambda + \mu)(\lambda + \mu_v)(\lambda + \gamma + \mu)(\lambda + \rho + \mu_v) \cdot [\lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3] = 0, \quad (71)$$

where

$$\begin{aligned} A_1 &= (\theta + \mu) + (\alpha + \delta + \mu) + (\sigma + \mu_v) + \mu_b, \\ A_2 &= (\theta + \mu)(\mu_b + \alpha + \delta + \mu + \sigma + \mu_v) \\ &\quad + \mu_b(\alpha + \delta + \mu + \sigma + \mu_v) + (\alpha + \delta + \mu)(\sigma + \mu_v), \\ A_3 &= J_7 \mu_b (J_3 + J_4) + J_3 J_4 \mu_b (1 - R_b) + J_3 J_4 J_7 (1 - R_v), \\ &= (\sigma + \mu_v) \mu_b (\theta + 2\mu + \alpha + \delta) + (\theta + \mu)(\alpha + \delta + \mu) \mu_b R_v \\ &\quad + (\theta + \mu)(\alpha + \delta + \mu)(\sigma + \mu_v) \mu_b R_b. \end{aligned} \quad (72)$$

Thus, $\lambda_1 = 0$, $\lambda_2 = -\mu$, $\lambda_3 = -\mu_v$, $\lambda_4 = -(\gamma + \mu)$, and $\lambda_5 = -(\rho + \mu_v)$ are the five eigenvalues of $J(\varepsilon_{0l}^*, \beta_1^*)$. The remaining three eigenvalues are the zeros of the polynomial equation $\lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3 = 0$, since $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, $A_1 A_2 - A_3 > 0$ for $R_0 = 1$ (see the proof of theorem (3.3.3)). We conclude that zero is a simple eigenvalue of $J(\varepsilon_{0l}^*, \beta_1^*)$ and all other eigenvalues of $J(\varepsilon_{0l}^*, \beta_1^*)$ have negative real numbers or real parts. This is an indication that the center manifold theorem is applicable. We first compute the left and right eigenvectors of the Jacobian matrix $J(\varepsilon_{0l}^*, \beta_1^*)$. let $Y = (y_1, y_2, y_3, y_4, y_5, y_6, y_7, y_8)^T$ be the right eigenvector of $J(\varepsilon_{0l}^*, \beta_1^*)$ corresponding to $\lambda = 0$. Then, Y is computed as

$$\begin{bmatrix} -\mu & 0 & 0 & \gamma & 0 & -J_1 & 0 & -J_2 \\ 0 & -J_3 & 0 & 0 & 0 & J_1 & 0 & J_2 \\ 0 & \theta & -J_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & -J_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & -J_6 & 0 & -\mu_v & 0 & \rho & 0 \\ 0 & 0 & -J_6 & 0 & 0 & -J_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma & 0 & 0 \\ 0 & 0 & \tau_1 & 0 & 0 & \tau_2 & 0 & -\mu_b \end{bmatrix} \begin{bmatrix} y_1 \\ y_2 \\ y_3 \\ y_4 \\ y_5 \\ y_6 \\ y_7 \\ y_8 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}. \quad (73)$$

Then, equation (73) can be expressed as

$$\begin{aligned} -\mu y_1 - \gamma y_4 - J_1 y_6 - J_2 y_8 &= 0, \\ -J_6 y_3 - \mu_v y_5 + \rho y_7 &= 0, \\ -J_3 y_2 + J_1 y_6 + J_2 y_8 &= 0, \\ J_6 y_3 - J_7 y_6 &= 0, \\ \theta y_2 - J_4 y_3 &= 0, \\ \rho y_6 - J_8 y_7 &= 0, \\ \delta y_3 - J_5 y_4 &= 0, \\ \tau_1 y_3 + \tau_2 y_6 - \mu_b y_8 &= 0. \end{aligned} \quad (74)$$

The components of Y can be obtained from (74) as

$$\left\{ \begin{aligned} y_1 &= -y_3 \frac{[\theta \gamma (\alpha + \mu) + \mu (\alpha + \delta + \mu) (\theta + \gamma + \mu)]}{\theta \mu (\gamma + \mu)} < 0, \\ y_2 &= y_3 \left(\frac{\alpha + \delta + \mu}{\theta} \right) > 0, \\ y_3 &= y_3 > 0, \\ y_4 &= y_3 \left(\frac{\delta}{\gamma + \mu} \right) > 0, \\ y_5 &= y_3 \left(\frac{\beta_3 \Pi \mu_v (\sigma + \rho + \mu_v)}{\mu_v (\sigma + \mu_v) (\rho + \mu_v)} \right) > 0, \\ y_6 &= y_3 \left(\frac{\beta_3 \Pi}{\mu_v (\sigma + \mu_v)} \right) > 0, \\ y_7 &= y_3 \left(\frac{\beta_3 \Pi \sigma}{\mu_v (\sigma + \mu_v) (\rho + \mu_v)} \right) > 0, \\ y_8 &= y_3 \left(\frac{\mu_v \tau_1 (\sigma + \mu_v) + \beta_3 \Pi (\sigma + \mu_v) \tau_2}{\mu_v \mu_b (\sigma + \mu_v)} \right) > 0. \end{aligned} \right. \quad (75)$$

Next, the left eigenvector $Z = (z_1, z_2, z_3, z_4, z_5, z_6, z_7, z_8)^T$ of the Jacobian matrix $J(\varepsilon_{0l}^*, \beta_1^*)$ corresponding to $\lambda = 0$ is computed as

$$\begin{bmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -J_3 & \theta & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -J_4 & \delta & -J_6 & J_6 & 0 & \tau_1 \\ \gamma & 0 & 0 & -J_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_v & 0 & 0 & 0 \\ -J_1 & J_1 & 0 & 0 & 0 & -J_4 & \sigma & \tau_2 \\ 0 & 0 & 0 & 0 & \rho & 0 & -J_8 & 0 \\ -J_2 & J_2 & 0 & 0 & 0 & 0 & 0 & -\mu_b \end{bmatrix} \begin{bmatrix} z_1 \\ z_2 \\ z_3 \\ z_4 \\ z_5 \\ z_6 \\ z_7 \\ z_8 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}. \quad (76)$$

Thus, Z can be found as

$$Z = \left(0, z_2, z_2 \frac{(\theta + \mu)}{\theta}, 0, 0, z_2 \frac{\mu_\nu(\mu\mu_b\kappa(\theta + \mu)(\alpha + \delta + \mu) - \beta_2\Lambda\theta\tau_1)}{\beta_3\Pi\mu\mu_b\kappa\theta}, 0, z_2 \frac{\beta_2\Lambda}{\mu\kappa} \right), \quad (77)$$

where $z_2 > 0$ and it is calculated to ensure that the eigenvectors satisfy the condition $Z \cdot Y = 1$, for $R_0 = 1$.

The local stability near the bifurcation point $\beta_1 = \beta_1^*$ is determined by the signs of two associated constants a and b . Based on the center manifold theorem 4.1 in [24], we compute the coefficients a and b , as

$$a = \sum_{k,i,j=1}^n z_k y_i y_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (\varepsilon_{0l}^*, \beta_1^*), \quad (78)$$

$$b = \sum_{k,i=1}^n z_k y_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_1} (\varepsilon_{0l}^*, \beta_1^*).$$

Since the first, fourth, fifth, and seventh components of Z are zero, we do not need the derivatives of f_1, f_4, f_5 , and f_7 . The derivatives of f_2, f_3, f_6 , and f_8 are $\partial^2 f_2 / \partial x_1 \partial x_6 = \partial^2 f_2 / \partial x_6 \partial x_1 = \beta_1$, $\partial^2 f_2 / \partial x_1 \partial x_8 = \partial^2 f_2 / \partial x_8 \partial x_1 = \beta_2 / \kappa$, $(\partial^2 f_2 / \partial x_6 \partial \beta_1^*) (\varepsilon_{0l}^*, \beta_1^*) = \Lambda / \mu$, and $(\partial^2 f_2 / \partial x_8^2) (\varepsilon_{0l}^*, \beta_1^*) = -2\beta_2 \Lambda / \kappa^2 \mu$, and any other second order partial derivative of f_k is zero, $\forall k = 1, 2, 3, 4, 5, 6, 7, 8$.

It follows that

$$a = \sum_{k,i,j=1}^n z_k y_i y_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (\varepsilon_{0l}^*, \beta_1^*)$$

$$= z_2 y_1 y_6 \frac{\partial^2 f_2}{\partial x_1 \partial x_6} (\varepsilon_{0l}^*, \beta_1^*) + z_2 y_1 y_8 \frac{\partial^2 f_2}{\partial x_1 \partial x_8} (\varepsilon_{0l}^*, \beta_1^*) + z_2 y_8^2 \frac{\partial^2 f_2}{\partial x_8^2} (\varepsilon_{0l}^*, \beta_1^*). \quad (79)$$

Substituting the values for partial derivatives and y_1, y_8 , we obtain

$$a = -z_2 y_3^2 \left[\beta_3 \beta_1 \Pi \sigma \Phi + \frac{\beta_2 \Phi [\mu_\nu \tau_1 (\sigma + \mu_\nu) + \beta_3 \Pi \tau_2]}{\kappa \mu_b} \right. \\ \left. + \frac{\beta_2 \Lambda}{\kappa^2 \mu} \left(\frac{\mu_\nu \tau_1 (\sigma + \mu_\nu) + \beta_3 \Pi \tau_2}{\mu_\nu \mu_b (\sigma + \mu_\nu)} \right)^2 \right] < 0. \quad (80)$$

where $\Phi = [\theta\gamma(\alpha + \mu) + \mu(\alpha + \delta + \mu)(\theta + \gamma + \mu)] / \theta\mu_\nu\mu(\gamma + \mu)(\sigma + \mu_\nu)$ and

$$b = z_2 y_6 \frac{\partial^2 f_2}{\partial x_6 \partial \beta_1^*} (\varepsilon_{0l}^*, \beta_1^*) = z_2 y_3 \left(\frac{\beta_3 \Pi \Lambda}{\mu_\nu \mu (\sigma + \mu_\nu)} \right) > 0 \text{ (always)}. \quad (81)$$

Since $a < 0$ and $b > 0$ and with the help of Theorem 4.1 by Castillo-Chavez and Song in [24], it is confirmed that the system follows a forward bifurcation at $R_0 = 1$ (see Figure 2), and hence, the endemic equilibrium is locally asymptotically stable for $R_0 > 1$, but sufficiently close to 1

The epidemiological implication of this shows that the leptospirosis disease can be eradicated in infected population as long as when $R_0 < 1$. The condition $R_0 < 1$ is necessary and sufficient for disease elimination for a model that undergoes the forward bifurcation [25]. \square

4. Sensitivity Analysis

In this section, we investigated the sensitivity of the parameters for the basic reproduction of the model using the idea presented in [26–28]. It is important to carry out the sensitivity of the basic reproduction number R_0 for its parameters. This will give parameters with a high impact on the leptospirosis model (3) and therefore allow to target on control measures to reduce the transmission of the disease. To measure the sensitivity index of R_0 to a given parameter β , we use the following relation:

$$\Omega_\beta^{R_0} = \left(\frac{\partial R_0}{\partial \beta} \right) \left(\frac{\beta}{R_0} \right). \quad (82)$$

An analytical expression for the sensitivity index of each parameter involved in R_0 is derived as follows:

$$\Omega_{\beta_1}^{R_0} = \frac{R_\nu}{R_0(2R_0 - R_b)} > 0 (\because R_0 > R_b),$$

$$\Omega_{\beta_2}^{R_0} = \frac{R_b(2R_0 - R_b) + R_b^2 + 2R_\nu}{2R_0(2R_0 - R_b)} > 0,$$

$$\Omega_\Lambda^{R_0} = \frac{R_b(2R_0 - R_b) + R_b^2 + 2R_{h\nu}}{2R_0(2R_0 - R_b)} > 0,$$

$$\Omega_\Pi^{R_0} = \frac{R_{h\nu}}{R_0(2R_0 - R_b)} > 0,$$

$$\Omega_\mu^{R_0} = - \left(\frac{[R_b(2R_0 - R_b) + R_b^2 + 2R_{h\nu}][\varepsilon_1 \varepsilon_2 + \mu(\varepsilon_1 + \varepsilon_2)]}{2R_0(2R_0 - R_b)} \right) < 0,$$

$$\Omega_{\beta_3}^{R_0} = \frac{R_{h\nu}}{R_0(2R_0 - R_b)} > 0,$$

$$\Omega_\theta^{R_0} = \frac{\mu [R_b(2R_0 - R_b) + R_b^2 + 2R_{h\nu}]}{2R_0(2R_0 - R_b)} > 0,$$

$$\Omega_{\mu_\nu}^{R_0} = - \frac{(\varepsilon_4 + \mu_\nu) R_{h\nu}}{R_0(2R_0 - R_b)} < 0, \quad (83)$$

$$\Omega_\alpha^{R_0} = - \frac{\alpha}{\varepsilon_2} \left(\frac{R_b(2R_0 - R_b) + R_b^2 + 2R_{h\nu}}{2R_0(2R_0 - R_b)} \right) < 0,$$

$$\Omega_{\tau_1}^{R_0} = \frac{R_b(2R_0 - R_b) + R_b^2}{2R_0(2R_0 - R_b)} > 0,$$

$$\Omega_\delta^{R_0} = - \frac{\delta}{\varepsilon_2} \left(\frac{R_b(2R_0 - R_b) + R_b^2 + 2R_{h\nu}}{2R_0(2R_0 - R_b)} \right) < 0,$$

$$\Omega_\sigma^{R_0} = - \left(\frac{\sigma R_{h\nu}}{\varepsilon_4 R_0(2R_0 - R_b)} \right) < 0,$$

$$\Omega_{\tau_2}^{R_0} = \frac{R_h}{R_0(2R_0 - R_b)} > 0,$$

$$\Omega_x^{R_0} = - \frac{R_b R_0 + R_h}{R_0(2R_0 - R_b)} < 0,$$

$$\Omega_{\mu_b}^{R_0} = - \frac{R_b R_0 + R_h}{R_0(2R_0 - R_b)} < 0.$$

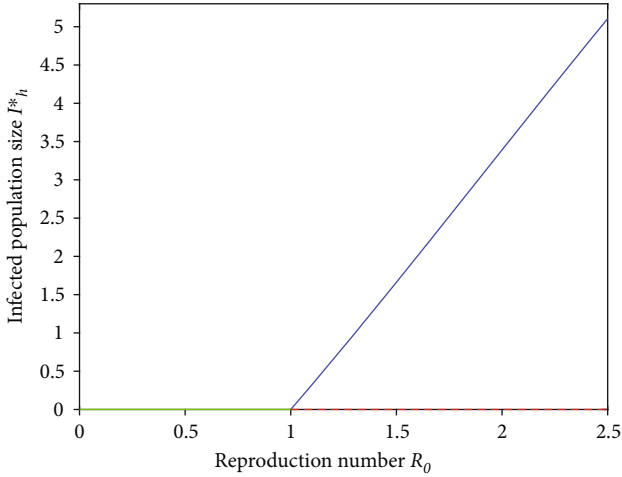


FIGURE 2: Forward bifurcation.

The values of the sensitivity indices of R_0 to each of its parameter values are computed and listed in Table 3 in decreasing order of sensitivity indices from the most to the least sensitive parameter. We used an algorithm Markov Chain Monte Carlo (MCMC) to fit parameter values of the model. The parameter values were used from previously published papers, and others were estimated as shown in Table 3.

The negative and positive signs of sensitivity index values in Table 3 represent the negative and positive (respectively) influence of parameters on R_0 . Thus, from the table, it can be observed that R_0 increases when the values of $\Lambda, \Pi, \beta_1, \beta_2, \beta_3, \theta, \tau_1, \tau_2$ are increase and vice versa. On the other hand, increasing the values of $\mu, \mu_v, \mu_b, \sigma, \delta, \alpha, \kappa$ decreases R_0 and vice versa. From the results of sensitivity analysis, the parameters $\Lambda, \Pi, \beta_1, \beta_3, \mu, \mu_v, \delta$ are the most influencing on R_0 . Further, decreasing Λ by 10% decreases R_0 by 5.54%. Increasing Π or β_3 by 10% increases R_0 by 4.47%. In contrast, increasing δ by 10% decreases R_0 by 3.2%. Similarly, decreasing (or increasing) μ and μ_v by a given percentage increases (or decreases, respectively) R_0 by half of the percentage. Epidemiological implications of this is that preventive and control efforts should be targeted on parameters $\Lambda, \Pi, \beta_1, \beta_3, \mu_v, \delta$ to reduce the burden of the disease in infected population since it is not reasonable biologically or ethically to use the mortality rate of humans as a control measure. In particular, reducing the total number of rodent population can minimize the transmission of disease, since μ_v is the reduction rate of rodents and it has a highly negative influence on R_0 . Further, the effect of the most sensitive parameters on disease dynamics behavior is illustrated graphically in the next section.

5. Numerical Simulations and Discussions

In this section, we perform some numerical results for the leptospirosis model (3) to demonstrate the results of the qualitative analysis of the model that has been already performed in the previous sections. To do this, we used the fourth-order Runge-Kutta method in the MATLAB program. The parameter values used in the simulations are

TABLE 3: Sensitivity indices of R_0 to each of the parameter values.

Parameter	Value	Sensitivity index
Λ	0.27	0.5421
μ_v	0.0029	-0.0306
μ	0.0009	-0.0058
Π	2	0.4579
β_3	0.0007	0.4579
σ	0.064	-0.438
β_1	0.00033	0.4167
δ	0.072	-0.3457
α	0.04	-0.1921
β_2	0.0815	0.5
κ	10000	-0.1254
τ_1	0.06	0.0842
τ_2	0.2	0.0412
μ_b	0.05	-0.1254
θ	0.092	0.0004879

given in Table 4. These parameter values have taken from previously published papers, and others are estimated as shown in Table 4.

The initial conditions are chosen as $(S_h(0), E_h(0), I_h(0), R_h(0), S_v(0), I_v(0), R_v(0), B_m(0)) = (270, 20, 10, 0, 510, 10, 0, 100)$. Using equation (63), parameter values of the Table 4 and the initial conditions, we found that $R_0 \approx 2.8552 > 1$, a unique positive endemic equilibrium which given by $E_l^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, I_v^*, R_v^*, B_l^*) \approx (47.668, 6.5229, 5.3154, 4.2571, 628.6425, 34.9632, 26.0494, 146.2314)$ with unique positive value $I_h^* \approx 5.3154$; the other six roots of the polynomial equation (63) are obtained as $I_0 = 0, I_1 \approx -9658.1994, I_2 \approx -6509.1639, I_3 \approx -54.7671, I_4 \approx -47.8513, I_5 \approx -47.2545$.

From graphical illustration of stability analysis of EE, we observed that all trajectories of the model solutions eventually move towards the steady-state E_l^* as shown the in Figures 3(a)–3(i) since E_l^* the unique positive EE is locally asymptotically stable for $R_0 > 1$. This shows that the disease persists in population because $R_0 = 2.8552 > 1$. On the other hand, if changing the values of $\beta_1, \beta_2, \beta_3, \mu_v$, and δ into 0.0001, 0.0515, 0.0003, 0.0035, and 0.092, respectively, then it yields $R_0 \approx 0.932 < 1$. In this case, the model has the DFE which is globally asymptotically stable, and hence, E_l^* is unstable. Thus, all trajectories of the model solutions tend to the DFE over time as shown in Figures 4(a)–4(f). Biologically, the implication is that the disease will die out from the community over time, while the susceptible humans and susceptible rodents eventually approach their maximum value for this particular study. Therefore, we should reduce the value of R_0 as much as possible to eradicate the disease rapidly.

5.1. Effect of the Transmission Rates of Humans and Rodents (β_1 and β_3) on Dynamics Behavior of the Disease. The transmission rates β_1 and β_3 play a significant role on expansion

TABLE 4: Description of parameter values used in the simulations.

Parameter	Description	Value	Unit	Source
Λ	The recruitment rate of susceptible humans	$\mu \times N_0$	Humans day ⁻¹	Assumed
Π	The recruitment rate of susceptible rodents	2	Rodents day ⁻¹	[5]
β_1	The transmission coefficient between S_h and I_v	0.00033	(Rodents day) ⁻¹	Assumed
β_2	The transmission coefficient between S_h and B_b	0.0815	Day ⁻¹	Assumed
β_3	The transmission coefficient between S_v and I_h	0.0007	(Humans day) ⁻¹	Assumed
μ	The natural death rate of susceptible humans	0.0009	Day ⁻¹	[29]; [5]
α	The disease-related death rate of humans	0.04	Day ⁻¹	Assumed
θ	The rate at which latently infected individual transfer to infectious	0.092	Day ⁻¹	[30]
γ	Leptospirosis disease waning immunity from R_h	0.089	Day ⁻¹	[31]
δ	The rate at which infectious individual transfer to human recovery class	0.072	Day ⁻¹	Assumed
σ	The rate of recovery from infectious rodent	0.064	Day ⁻¹	Assumed
ρ	Leptospirosis disease waning immunity from R_v	0.083	Day ⁻¹	Assumed
μ_v	The natural death rate of the rodents	0.0029	Day ⁻¹	[29]
μ_b	The natural death rate of the bacteria	0.05	Day ⁻¹	[6]
τ_1	The rate of increase of bacterial population due to I_h	0.06	No.of pathogen $\frac{\text{}}{\text{humans day}}$	Assumed
τ_2	The rate of increase of bacterial population due to I_v	0.2	No.of pathogen $\frac{\text{}}{\text{rodents day}}$	Assumed
κ	The concentration of pathogenic population	10000	No.of pathogen	Assumed

of the disease transmission by increasing the incidence rates of humans and rodents in the infected population. In this section, we illustrate the sensitivity of the model (3) in human, rodent, and bacterial populations using different values of the parameters. In Figures 5(a) and 5(b), it can be seen that decreasing the human transmission rate (β_1) increases the sizes of susceptible humans and susceptible rodents (eventually) and vice versa. On the other hand, reducing this parameter reduces the sizes of infected individuals, infected rodents, recovered rodents, and bacterial population from the beginning and eventually as shown Figures 5(c)–5(f), respectively. This was performed by taking the values of β_1 as $\beta_1 = 0.00017$, $\beta_1 = 0.00023$, $\beta_1 = 0.00033$, $\beta_1 = 0.0005$, and $\beta_1 = 0.0008$. In the same manner, we observed the effects for the rodent transmission rate β_3 by setting its values as $\beta_3 = 0.0005$, $\beta_3 = 0.0006$, $\beta_3 = 0.0007$, $\beta_3 = 0.00085$, and $\beta_3 = 0.00099$ as shown in Figure 6. A decrease (or increase) in the value of human transmission rate β_3 will cause decrease (or increase, respectively) in the size of the infected individuals, infected rodents, and recovered rodents as indicated in Figures 6(b), 6(d), and 6(e), respectively. Similarly, in Figure 6(f), it can be seen that decreasing β_3 decreases the growth bacterial population in an environment with time as they are directly proportional. However, decreasing (or increasing) the human transmission rate (β_3) increases (or decreases, respectively) the size of the number of susceptible humans and susceptible rodents as shown in Figures 6(a) and 6(a), respectively. In view of the simulations of the model in Figures 5 and 6, the smaller parameter values (as possible) will result in the smaller R_0 value and will decrease the incidence rates of human and rodent infections. This shows that

reducing the expansion of human infection depends on the reduction of the transmission rates β_1 and β_3 on susceptible individuals and susceptible rodents. Therefore, by minimizing the values of β_1 and β_3 significantly, the number of infected humans, infected rodents, and the growth of leptospires in the environment can be reduced and could finally vanish. This can be achieved by treating infected individuals (using treatment control), avoiding contact with the contaminated environment (soil or water), or by reducing infected rodents as well as the total number of rodent population.

5.2. Effect of the Human Recovery Rate (δ) on Dynamics Behavior of the Disease. The infectious individuals can be reduced by increasing the value of the human recovery rate (δ) using control interventions (treatment of disease infection). In this section, we simulate the model to observe the effect of δ in human, rodent, and bacterial populations by varying the value δ . From Figure 7, we observed that δ is directly proportional with susceptible and recovered individuals as revealed in Figures 7(a) and 7(c). Moreover, it can be seen in Figures 7(b), 7(e), and 7(f) that the population of infected humans and infected rodents and population of bacterial increase (or decrease) from the beginning and eventually as δ decreases (or increases, respectively), whereas the size of susceptible rodents increases (or decreases) from the beginning and eventually as δ increases (or decreases, respectively) with time as shown in Figure 7(d). From an epidemiological perspective, human leptospirosis infection can be minimized by reducing the number of infected individuals significantly using treatment control effort on infected individuals.

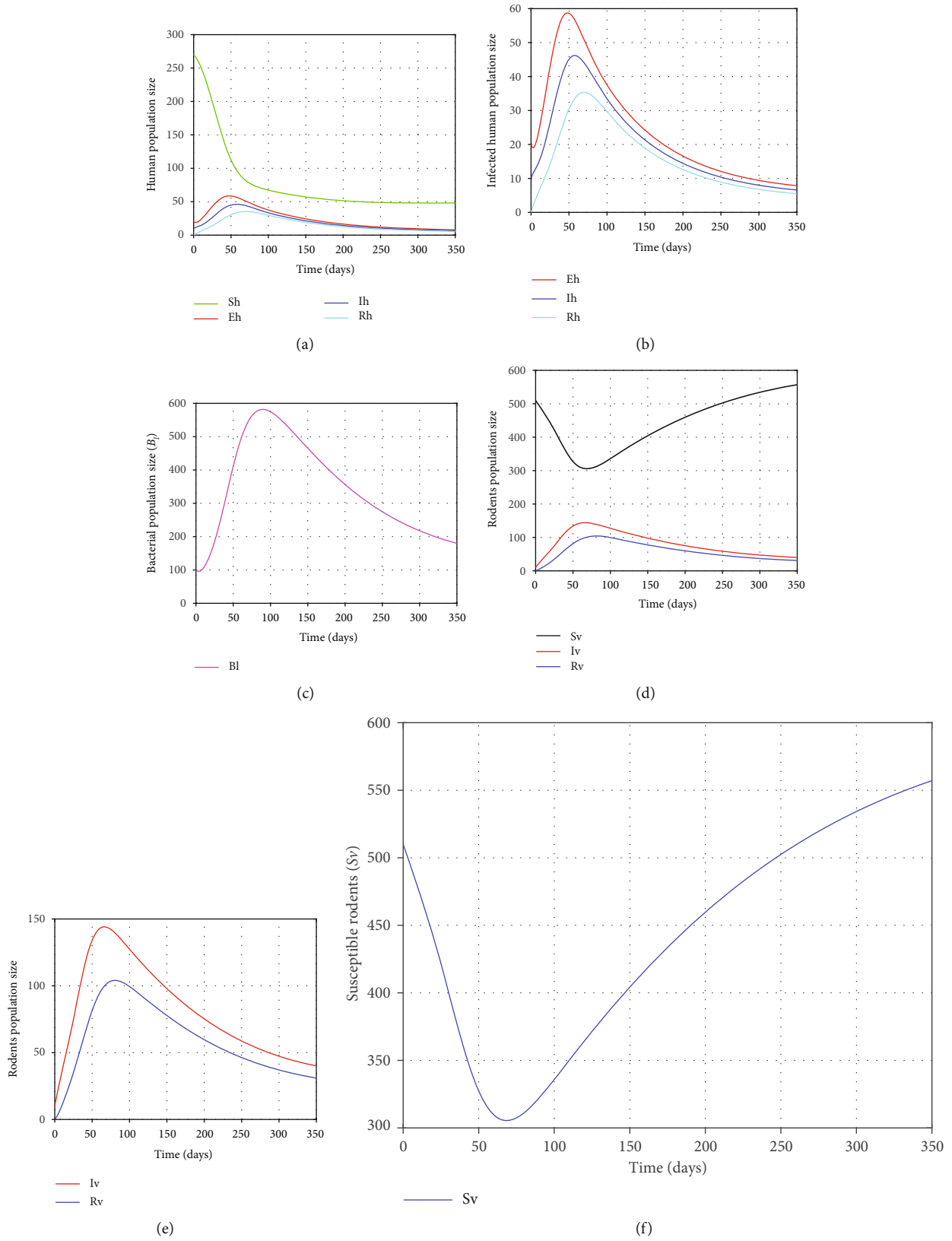


FIGURE 3: Continued.

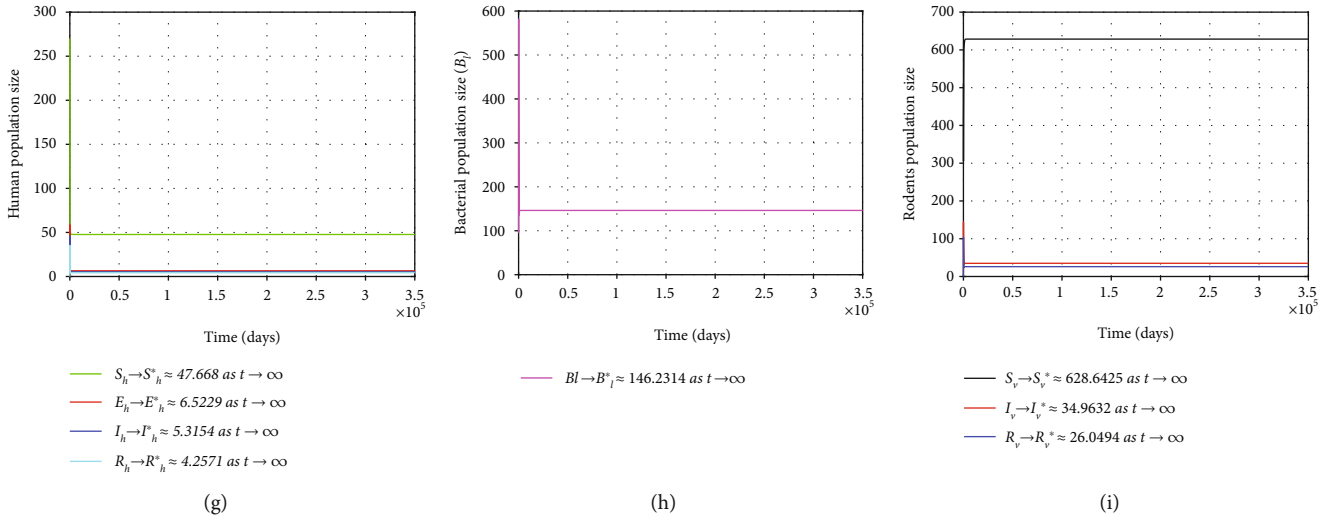


FIGURE 3: Plots showing the stability behavior of $EE_1^* = (47.668, 6.5229, 5.3154, 4.2571, 628.6425, 34.9632, 26.0494, 146.2314)$ of the model (3) as t varies.

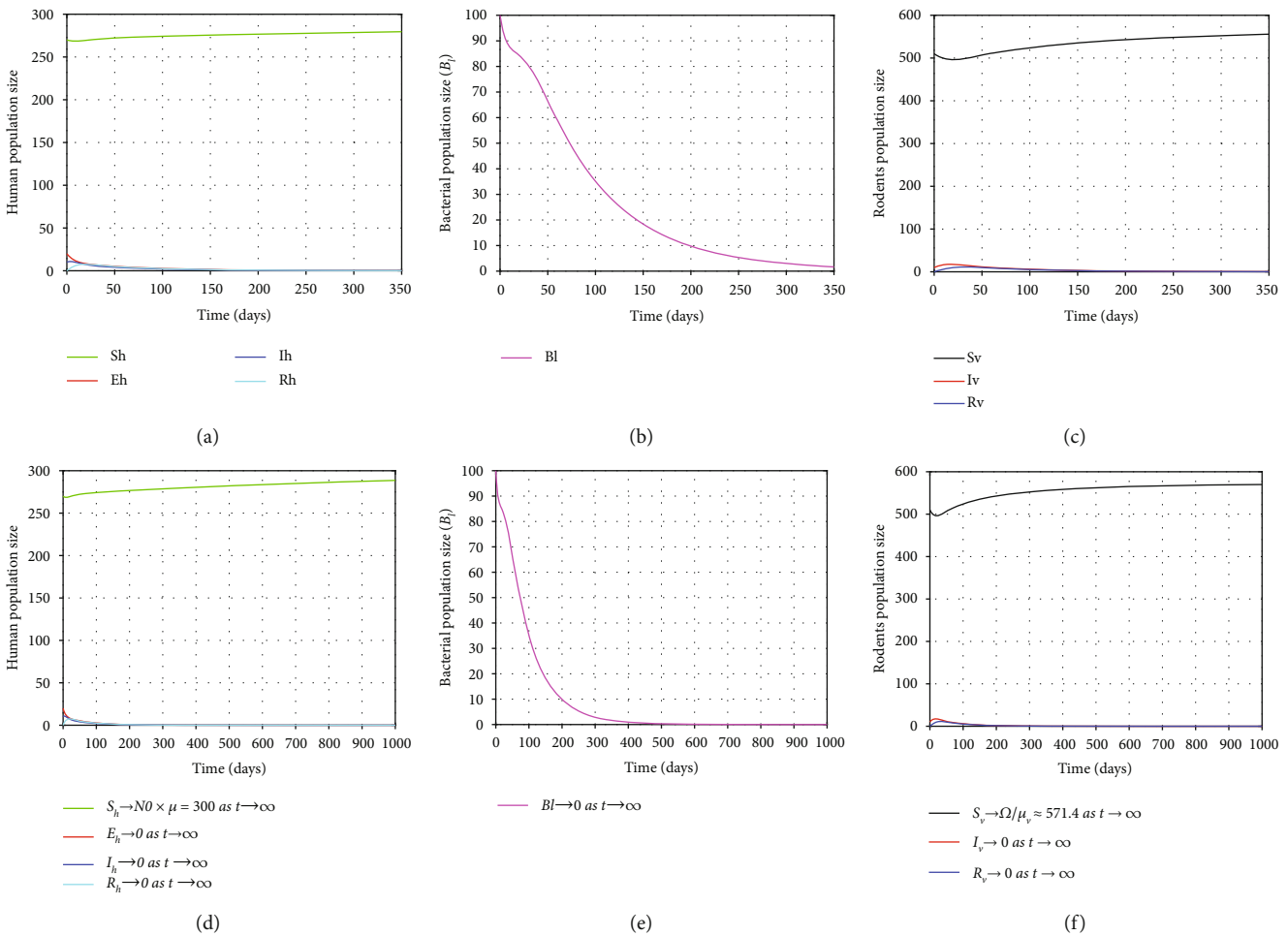


FIGURE 4: Plots showing the stability behavior of $DFE_0^* = (300, 0, 0, 0, 571.4, 0, 0, 0)$ of the model (3) as time changes.

5.3. *Effect of the Mortality Rate of Rodents (μ_v).* From Figures 5(b), 6(c), and 7(d), increasing the number of susceptible rodents depends on the reduction of the parameters

$\beta_1, \beta_3,$ and δ on susceptible humans, susceptible rodents, and infected individuals, respectively. However, reducing these parameters may cause an increase in the total number

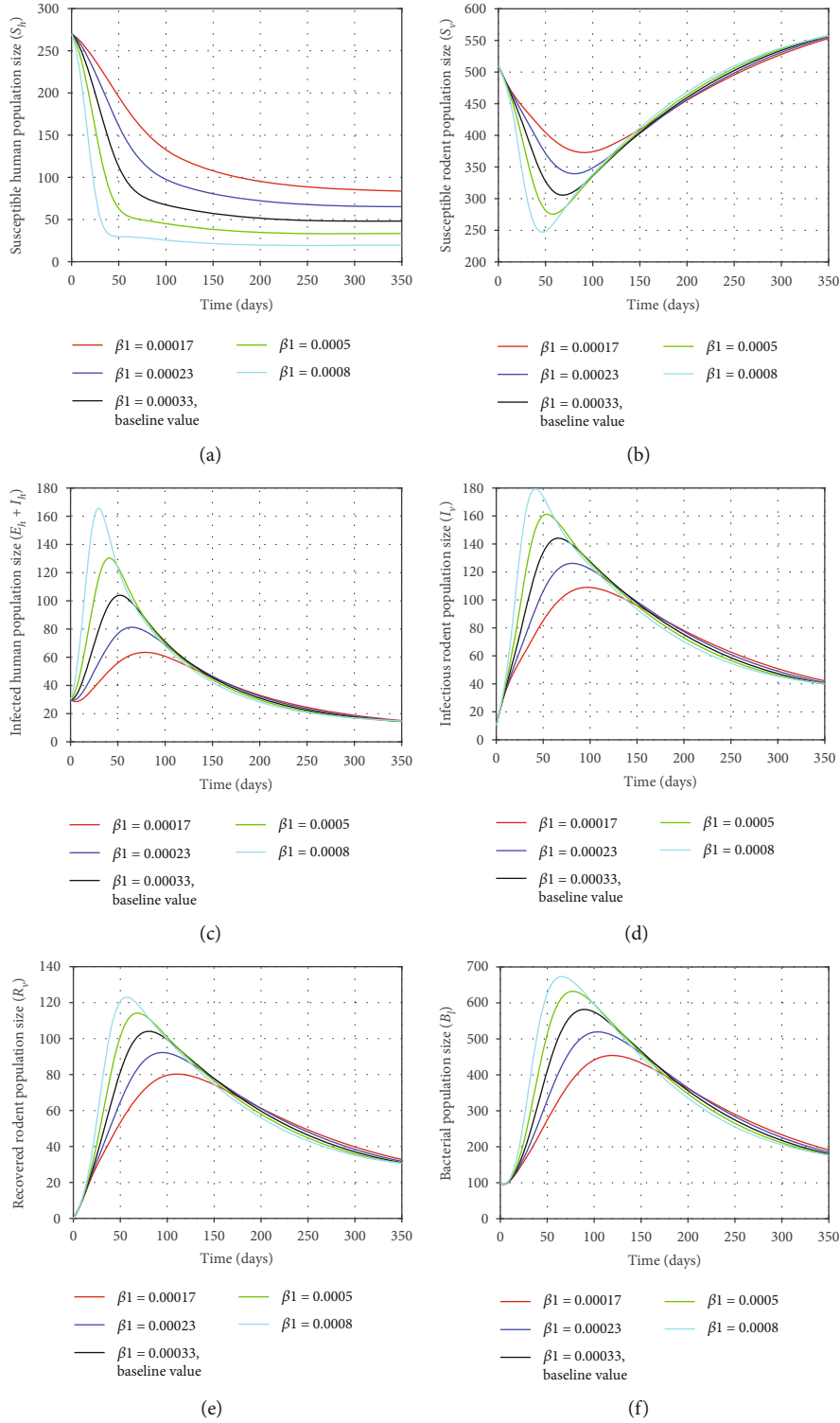


FIGURE 5: Plots showing the sensitivity of model (3) using different values of β_1 .

of rodent population slowly (as can be seen in the figures). Thus, the control mechanism for growth of the rodent population (for instance, rodenticide) should be implemented to reduce the total number of rodent population. The effect of mortality rate of rodents μ_v in human, rodent, and bacterial population is illustrated in this section by using the different values of μ_v as $\mu_v = 0.001$,

$\mu_v = 0.0029$, $\mu_v = 0.0059$, $\mu_v = 0.009$, and $\mu_v = 0.014$. It can be observed, from Figures 8(b)–8(f), that the number of infected individuals, the total number of rodents ($S_v + I_v + R_v$), and the size of bacterial population decrease as μ_v increases. Conversely, decreasing the mortality rate of rodents decreases the number of susceptible humans as shown in Figure 8(a).

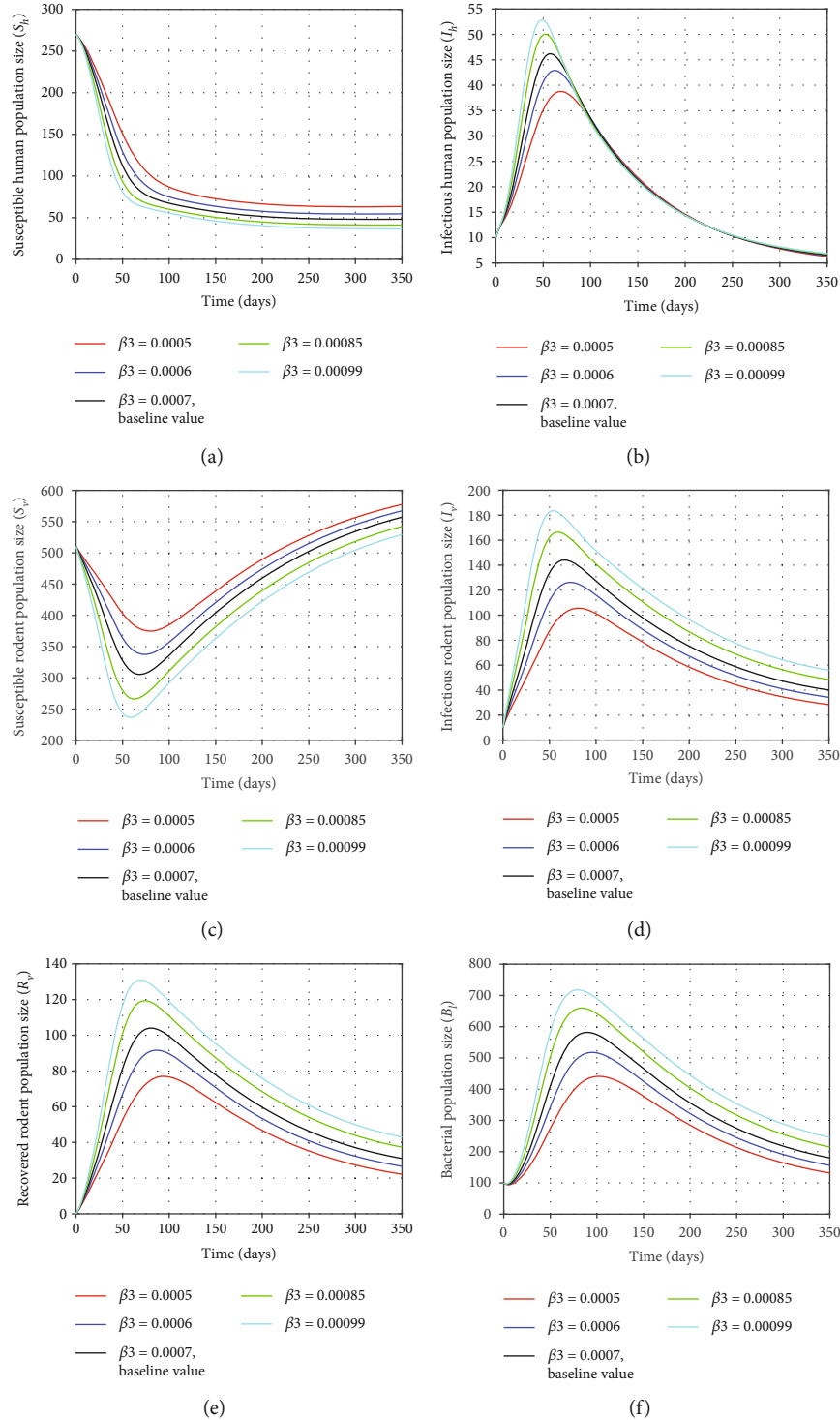


FIGURE 6: Plots showing the sensitivity of model (3) using different values of β_3 .

Therefore, controlling the major direct zoonotic infections in a human population depends on the reduction of animal reservoirs, as in the case of leptospirosis infection. Reduction of the rodent population may also reduce the number of human infections as well as the concentration of *Leptospira interrogans* in the environment. Thus, the rodent born leptospirosis

infections of humans can be controlled by implementing rodents' reduction mechanism. Further, since $R_0 \approx 2.8552 > 1$, all trajectories of the model solutions eventually move towards the unique positive E_l^* . Thus, it should be intensified to use optimal efforts on interventions to eliminate the disease in the infected population as much as possible.

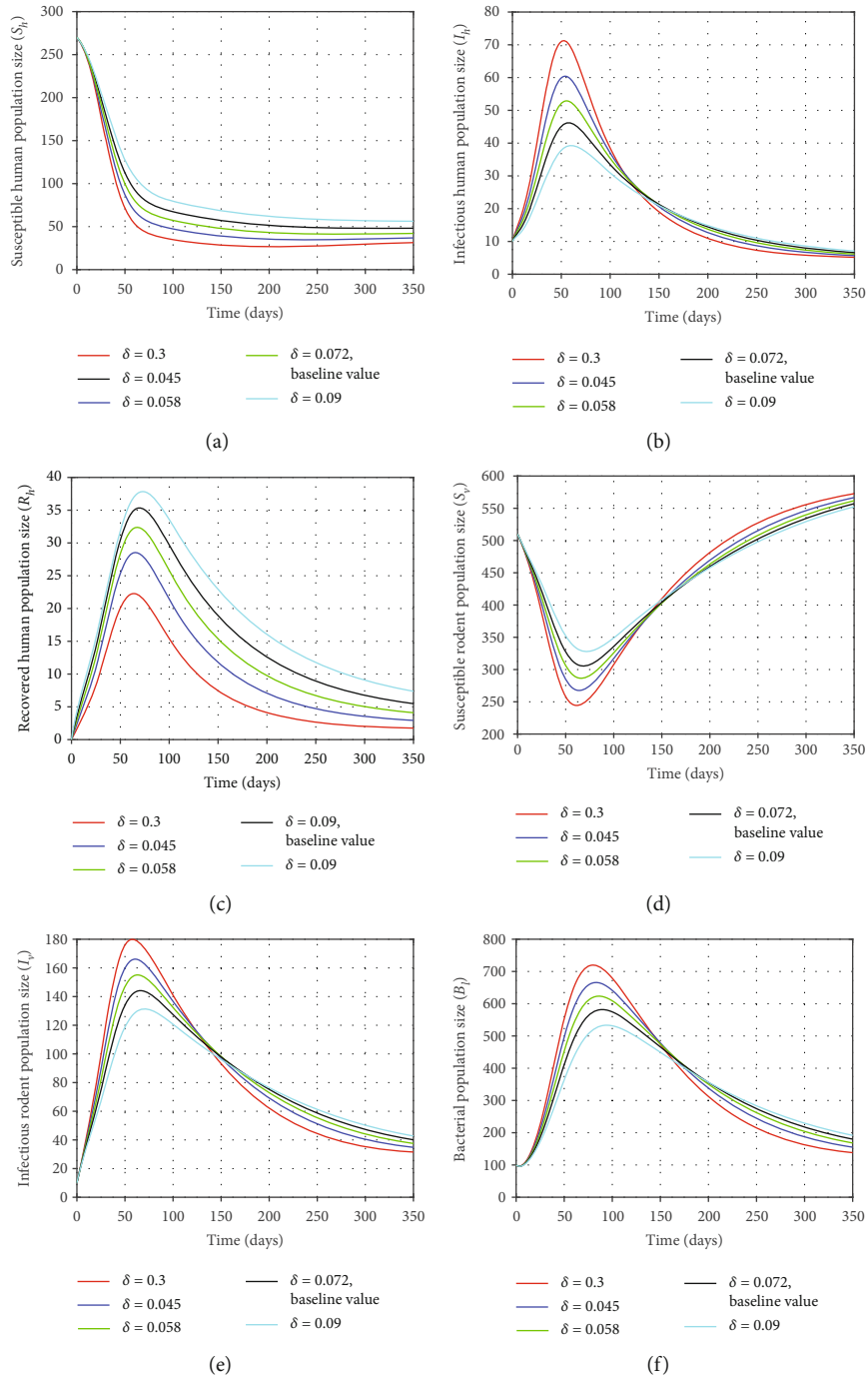


FIGURE 7: Plots showing the sensitivity of model (3) using different values of δ .

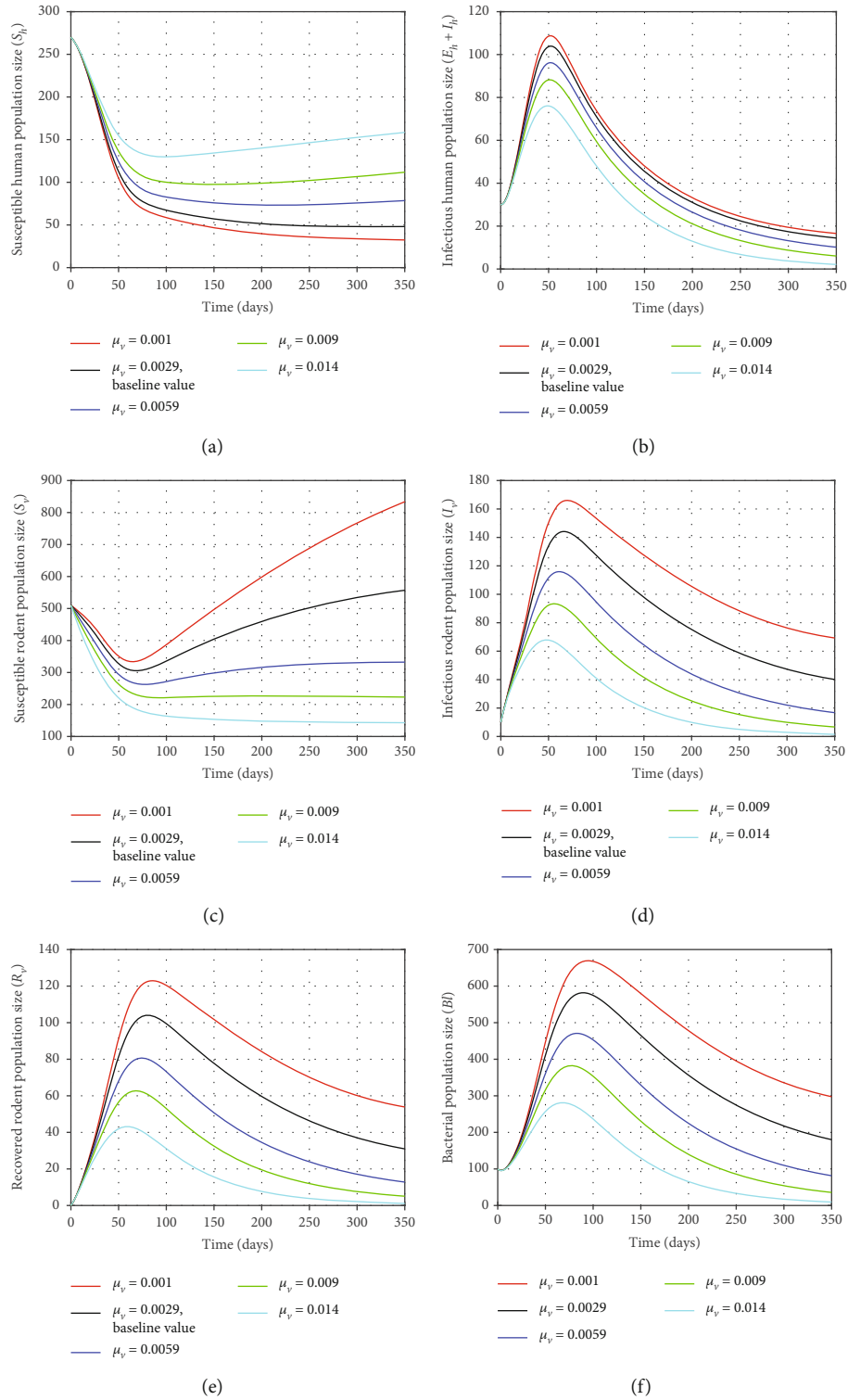


FIGURE 8: Plots showing the sensitivity of model (3) using different values of μ_v .

6. Conclusion

In this work, we presented and analyzed a deterministic mathematical model for the transmission process of leptospirosis disease, which includes human, vector (rodent), and bacterial populations. Firstly, the qualitative properties of the model are studied. Based on the next-generation

matrix approach, the basic reproduction number (R_0) of the formulated model is computed to perform sensitivity analysis of the model parameters. From the stability analysis of the model, we found that the disease-free equilibrium (DFE) is globally asymptotically stable if $R_0 < 1$ and unstable otherwise. The local stability of an endemic equilibrium is performed based on the general center manifold theory,

and EE is locally asymptotically stable when $R_0 < 1$. The model exhibits forward bifurcation. This shows that the leptospirosis disease can be eradicated in infected population as long as when $R_0 < 1$. The most sensitive parameters for R_0 are Λ , Π , μ , β_1 , β_3 , μ_v , and δ , among other parameters. This illustrates that minimizing the number of infected individuals depends on the reduction of β_1 on susceptible individuals and the increase of recovery rate δ . This shows that minimizing the human infection depends on the reduction of β_3 on susceptible vectors and the increase of mortality rate of rodents μ_v . Epidemiological implications of this illustrate that preventive and control efforts should be targeted on the parameters β_1 , β_3 , and μ_v to reduce the burden of the disease in human populations since it is not reasonable ethically or biologically to use the death rate of humans as a control measure. Finally, the model is simulated to show the changing effects of the most influencing parameters on the disease expansion as well as the stability behaviors of the steady states. From graphical stability behavior of EE, all trajectories of the model solutions eventually move to the unique EE for $R_0 \approx 2.8552 > 1$. In this case, the disease persists in the population. On the other hand, all trajectories of the model solutions gradually move towards the DFE for $R_0 < 1$. The biological implication of this shows that the disease will die from a community as long as when the basic reproduction number of the model is less than unity. Therefore, the value of R_0 should be reduced as much as possible to eradicate the disease rapidly in an infected population. Moreover, our numerical results of the model demonstrate that reducing the transmission of human leptospirosis infection can be achieved by implementing the presentation and control interventions. Thus, our study recommends the following intervention efforts to minimize the risk factors and control the disease infection that should be implemented in human, rodent, and bacterial populations in an infected community:

- (i) Reduce the rodent population using rodenticide or resource reduction. Implementation of this intervention also reduces the shedding rate of leptospire into the environment
- (ii) Prevention interventions on susceptible humans. Avoid contact with infected rodents and rodents urine-contaminated environment (floodwater, lakes, rivers, or soil) using an appropriate personal protective equipment (PPE) such as wearing rubber boots, waterproof dressings to cover wounds or skin, goggles, and rubber gloves as well as maintaining good personal hygiene. These practices will minimize the acquisition of the infection in the community
- (iii) Use the treatment control on infected individuals. Although the leptospirosis infection shows nonfixed symptoms and signs initially, its clinical manifestation can be detected by microbiological laboratory tests. This control mechanism can minimize the expansion of the disease by treating infected individuals through antibiotics that are recommended

for treatment of the disease in the infected population following clinical rules

- (iv) Increase mortality rate of *Leptospira interrogans* in environment (especially, urban slum environment) using appropriate environmental modifications

These clinical interpretations (interventions) can be implemented by heightening the awareness of public health-care sectors on the spread and impacts of the disease on human health and by providing information for infected populations to use an effort to minimize the risk factors of the disease through intensifying successive education (or training) on the disease. However, to illustrate the effectiveness and cost-effectiveness of these intervention efforts an optimal control intervention should be studied.

Abbreviations

WHO: World Health Organization
 CDC: Centers for Disease Control and Prevention
 DFE: Disease-free equilibrium
 EE: Endemic equilibrium
 ODE: Ordinary differential equation
 SIR: Susceptible-infected-removed
 SEIR: Susceptible-exposed-infected-removed.

Data Availability

The numerical data used to support the findings of this study have been taken from previously published articles and cited in the Table 4 of this paper. These published articles are also cited at relevant places within the text as references.

Conflicts of Interest

The authors declare no conflict of interest.

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