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Stability analysis of leptospirosis compartmental model with impact of contaminated environment

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Abstract. The main objective of this article is to analyse the stability properties of a model involving humans, animals and contaminated environment. As a first step, the model is formulated, and its biological well-posedness is proved. Then the basic reproduction number is derived using the next generation matrix (NGM) method. The local and global asymptotic stability of the system at the disease free and endemic equilibrium points are also established. Finally, numerical simulations to illustrate the validity of the theoretical results are performed.

Keywords: leptospirosis, free-living pathogen, next-generation matrix method, basic reproduction number, stability analysis.

1 Introduction

Infectious diseases and the casualties caused by their rapid spread pose a global threat, especially to the poor and resource deficient countries. The aftermath of the rapid spread of infectious diseases are beyond the damages in public health as they can significantly affect the day to day life and global economy.

Leptospirosis is a notable infectious disease, which is zoonotic in nature and predominantly prevalent in the tropical and subtropical regions. The disease caused by the bacteria *Leptospira* affects both humans and animals alike. Leptospires enter into humans either directly through contact with infected animals (rats, livestock, dogs, pigs, cats etc.) or

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indirectly through the soil and water contaminated with the urine, tissues or other bodily fluids of infected animals. Leptospirosis can cause severe health problems in both humans and animals. Interested readers may go through the monographs [9, 28] and references therein for further details. The above discussion underlines the need to understand the transmission dynamics of leptospirosis, forecast potential outbreaks and develop effective control strategies.

Mathematical modelling is an effective tool for describing and analysing complex real-world phenomena, providing insights into their current dynamics and aiding in the prediction of their future dynamics. Differential equations models are widely used across various fields to describe systems in which time changes are of significant interest. In particular, they are widely used in the studies of population dynamics, epidemiology, climate modelling, cancer research [1], ecological systems such as food chains [17], predator–prey interactions [4] etc.

Infectious diseases are primarily studied using compartmental differential equations models, which subdivide populations into compartments based on the disease status. The flow of population among compartments are described by system of differential equations. The theory of compartmental differential equations models in the context of infectious diseases originated from the pioneering work of Kermack and Mckendrick in 1927 [11]. Formulating and analysing the compartmental models provide valuable insights into the transmission dynamics of the disease spread, which can aid in predicting potential outbreaks, formulating public policies, assessing impact of interventions and effective disease control.

There is abundant literature on compartmental models [11], which studied the transmission dynamics of leptospirosis. Rodents being considered as primary reservoir of leptospires [28], a lot of works have been done focusing the infection in rodents and the spreading in humans and rodents. Some of the works worth mentioning are [7, 16, 27]. Pimpunchat et al. proposed a compartmental model to investigate the dynamical behaviour of leptospirosis [24]. The article [5] studied the leptospirosis spread in the endemic states of India and performed sensitivity analysis to show that the meteorological factors triggering the incidence rate are rainfall, fishing, climate, agriculture etc. Based on their study, preventive strategies to reduce the spread of leptospirosis were also suggested. For more works, readers can refer the monographs [6, 13, 26] and references therein.

The article [13] presented a compartmental model involving the presence of leptospira bacteria in the environment to study leptospirosis and discussed how the exposure of living organisms to these bacteria significantly facilitates the transmission of the disease. Studies indicate that leptospires in a free-living state may persist in wet soil or surface water from a few weeks to almost a year even during dry season and it reproduces in contaminated environment [13, 29]. In fact, contaminated environment – particularly surface water and waterlogged soil – acts as a secondary reservoir for free-living leptospires. Heavy rainfall and flood create favourable conditions for leptospires to survive and reproduce [13]. Hence outbreaks of leptospirosis often tend to follow up after rainy season. Occupation in fields like agriculture, animal husbandry, veterinary health care, waste management etc., which requires close contact with animals or exposure to contaminated environment, elevates the risk for contracting leptospirosis [10].

Although earlier studies have considered the influence of contaminated environment in the disease spread, it still has not been completely explored. In [16], Holt developed a system of ordinary differential equations to study the interaction between rodents and leptospira bacteria alone. An age structured SIR model involving cattles and free-living bacteria is considered in [6]. Though Triampo et al. [27] proposed a more accurate model with humans, vectors and bacteria, the direct transmission from bacteria to humans is not explored. In [7], Baca-Carrasco et al. put forward a SI model incorporating the direct transmission from bacteria to humans. But recovered class is a key component in studying disease dynamics whose inclusion is necessary for far more accurate and complete representation. In [3], the authors have proposed and analysed stability properties of a fractional-order compartmental model for leptospirosis, including recovered class in humans and considering the environmental effects. Stability analysis of a simple ODE model to study the dynamics of leptospirosis by incorporating recovered classes in both human and vector populations, along with the contaminated environment, is given in [15].

Stability analysis of dynamical systems studies its long-time behaviour by analysing the impact due to small perturbations near equilibrium points. Such a study in epidemic models eventually investigates whether the disease will die out, persist or continue to spread in the population. For this reason, many researchers have performed stability analysis for compartmental models, which are often formulated to provide a general framework for a class of diseases with similar nature of spread or to govern the spread of some specific disease. These are some of the works on stability analysis of epidemic models: [2, 14, 18, 24, 25].

To the best of our knowledge, no ODE compartmental model for leptospirosis with the environmental compartment considered the global stability at the endemic equilibrium point. This motivated the authors to develop a leptospirosis model following the ideas in [13] and analyse the stability at both disease-free and endemic equilibrium points.

Novelties of the work. In the formulated model, we have considered human and animal populations, each categorised into susceptible, infected and recovered classes, along with free-living leptospira population, giving significant attention to the direct and indirect transmission of the disease. We have considered logistic growth for the free-living leptospira in the environment, providing more accurate and realistic representation of the growth limited by the carrying capacity of environment. Though some earlier studies considered the transmission of leptospirosis under the influence of free-living leptospires in contaminated environment, our model is expected to provide a more comprehensive understanding of the transmission of leptospirosis due to the novelties of our model.

Difficulties of the work. The method of Lyapunov functions is widely used to perform global asymptotic stability for dynamical systems. But the absence of a general procedure for constructing Lyapunov functions makes it a challenging task in higher dimensions. The Li–Muldowney geometrical approach of autonomous system [19] and a general criterion for proving global asymptotic stability of equilibria for nonlinear autonomous systems has been proposed in [20]. To our knowledge, no previous studies have applied this method in five or higher dimensions. This article presents the first exploration of the method to investigate the global stability at the endemic equilibrium in five dimensions.

2 Model description

In this section, we formulate an ODE compartmental model for leptospirosis transmission dynamics with a special focus on contaminated environment. While formulating a mathematical model to study an infectious disease dynamics, one needs to make some assumptions regarding population characteristics, disease status etc. Leptospirosis, being an endemic in many countries, requires the consideration of its long-term dynamics, leading to incorporation of birth and death rates into the model. Leptospirosis is primarily transmitted through infected animals and contaminated environment [13]. Deaths due to leptospirosis are relatively small, transmission from mother to offspring does not occur or is rare [27] and offers only temporary immunity after recovery [13]. Also, all newborns are considered non-immunized [27], and we assume constant human (N_h) and animal (N_a) populations to reduce complexity of the model without significantly compromising its accuracy [24]. In the absence of shedding from infected animals, the growth of leptospira bacteria is not enough to maintain itself in the environment due to its decay [8]. In light of the above discussions, the following key assumptions were adopted to formulate the model.

(A1) Both human and animal populations are assumed to be constant.
(A2) Every newborn is susceptible.
(A3) Recovered humans and animals loss immunity over time.
(A4) Humans cannot transmit the disease neither directly nor indirectly.
(A5) Vertical transmission of the disease is absent.
(A6) There is no disease related death.
(A7) Pathogen population cannot maintain itself through growth in the contaminated environment. So $\mu_L > \mu_g$. For that, we refer to Table 1.

Based on the disease status, both human and animal population are subdivided into 3 disjoint compartments. Let S_h, I_h and R_h denote susceptible, infected and recovered humans, respectively. S_a, I_a and R_a denote their counterparts in animal population. In addition to this, a compartment L representing free-living leptospires in the environment is also incorporated, which holds greater importance in disease spread as contaminated environment is a secondary reservoir for the leptospires. If $N_h(t)$ and $N_a(t)$ denote the

Table 1. Parameters with units used for the model.

Notation	Parameter description	Notation	Parameter description
μ_h	Birth (death) rate of humans	μ_a	Birth (death) rate of animals
β_{ha}	Transmission rate from animal to human	β_{aa}	Transmission rate from animal to animal
β_{hL}	Transmission rate from contaminated environment to human	β_{aL}	Transmission rate from contaminated environment to animal
γ_h	Recovery rate of humans	γ_a	Recovery rate of animals
ν_h	Rate of loss of immunity for humans	ν_a	Rate of loss of immunity for animals
μ_L	Pathogen decay rate	μ_g	Pathogen growth rate
ω_1	Pathogen shedding rate	c	Per capita carrying capacity

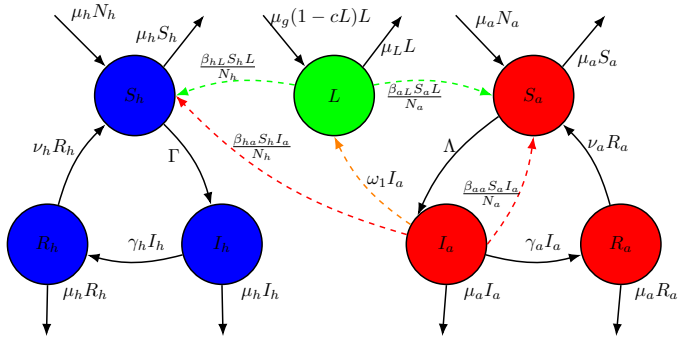


Figure 1. Flow chart of leptospirosis spread.

total human and animal populations at time t, we have

$$N_h(t) = S_h(t) + I_h(t) + R_h(t) \quad \text{and} \quad N_a(t) = S_a(t) + I_a(t) + R_a(t) \quad \forall t.$$

Based on (A1)–(A7), an ODE compartmental model of coupled nonlinear autonomous system is formulated as shown below:

$$\begin{aligned}
\frac{\mathrm{d}S_h}{\mathrm{d}t} &= \mu_h N_h - \frac{\beta_{ha} I_a}{N_h} S_h - \frac{\beta_{hL} L}{N_h} S_h - \mu_h S_h + \nu_h R_h,\\
\frac{\mathrm{d}I_h}{\mathrm{d}t} &= \frac{\beta_{ha} I_a}{N_h} S_h + \frac{\beta_{hL} L}{N_h} S_h - \mu_h I_h - \gamma_h I_h,\\
\frac{\mathrm{d}R_h}{\mathrm{d}t} &= \gamma_h I_h - \nu_h R_h - \mu_h R_h,\\
\frac{\mathrm{d}S_a}{\mathrm{d}t} &= \mu_a N_a - \frac{\beta_{aa} I_a}{N_a} S_a - \frac{\beta_{aL} L}{N_a} S_a - \mu_a S_a + \nu_a R_a,\\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \frac{\beta_{aa} I_a}{N_a} S_a + \frac{\beta_{aL} L}{N_a} S_a - \mu_a I_a - \gamma_a I_a,\\
\frac{\mathrm{d}R_a}{\mathrm{d}t} &= \gamma_a I_a - \nu_a R_a - \mu_a R_a,\\
\frac{\mathrm{d}L}{\mathrm{d}t} &= \omega_1 I_a - \mu_L L + \mu_g(1-cL)L
\end{aligned} \tag{1}$$

subject to the initial conditions

$$\begin{aligned}
&S_h(0) > 0, \qquad I_h(0) > 0, \qquad R_h(0) > 0,\\
&S_a(0) > 0, \qquad I_a(0) > 0, \qquad R_a(0) > 0, \qquad L(0) > 0.
\end{aligned}$$

It may be noted that all the parameters appearing in model (1) are assumed to be positive and are given in Table 1.

A schematic diagram of the transmission dynamics of the leptospirosis across the compartments of the model (1) is given in Fig. 1, where

$$\Gamma = \frac{\beta_{ha} S_h I_a}{N_h} + \frac{\beta_{hL} S_h L}{N_h}, \qquad \Lambda = \frac{\beta_{aa} S_a I_a}{N_a} + \frac{\beta_{aL} S_a L}{N_a}.$$

2.1 Biological well-posedness of the model

We now proceed to prove the biological well-posedness of (1) to ensure the existence and uniqueness of a non-negative bounded solution. Throughout this article, let $\mathbb{R}$ denote the real line with norm $|\cdot|$ and $\mathbb{R}^n$, $n \geqslant 2$, denote the Euclidean space with Euclidean norm $\|\cdot\|$. Additionally, let $\mathbb{R}^n_+$ denote the set of all points in $\mathbb{R}^n$ with non-negative coordinates.

Lemma 1 [Non-negativity]. *Let* $(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t)) \in \mathbb{R}^7$ *be the solution to model* (1) *with the initial condition* $(S_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0), L(0))$ *in* $\mathbb{R}^7_+$. *Then* $(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t))$ *remains non-negative for all* $t \geqslant 0$.

Proof. Using the positivity of parameters, from the first equation of system (1) we obtain the following inequality [14]:

$$\frac{\mathrm{d}S_h}{\mathrm{d}t} \geqslant -\left(\frac{\beta_{ha} I_a}{N_h} + \frac{\beta_{hL} L}{N_h} + \mu_h\right) S_h.$$

A direct integration of the above yields

$$S_h(t) \geqslant S_h(0) \exp\left\{-\int_0^t \left(\frac{\beta_{ha} I_a(\tau)}{N_h} + \frac{\beta_{hL} L(\tau)}{N_h} + \mu_h\right) \mathrm{d}\tau\right\} \geqslant 0.$$

By similar calculations, we obtain

$$S_a(t) \geqslant S_a(0) \exp\left\{-\int_0^t \left(\frac{\beta_{aa} I_a(\tau)}{N_a} + \frac{\beta_{aL} L(\tau)}{N_a} + \mu_a\right) \mathrm{d}\tau\right\} \geqslant 0,$$

$$I_h(t) \geqslant I_h(0)\mathrm{e}^{-(\mu_h+\gamma_h)t} \geqslant 0, \qquad I_a(t) \geqslant I_a(0)\mathrm{e}^{-(\mu_a+\gamma_a)t} \geqslant 0,$$

$$R_h(t) \geqslant R_h(0)\mathrm{e}^{-(\mu_h+\nu_h)t} \geqslant 0, \qquad R_a(t) \geqslant R_a(0)\mathrm{e}^{-(\mu_a+\nu_a)t} \geqslant 0$$

for all $t \geqslant 0$. Now, it remains to prove the non-negativity of $L(t)$. If possible, let $L(t) < 0$ for some $t > 0$. Then, by intermediate value theorem, $L(s) = 0$ for some $s < t$. Then, at $t = s$, $\mathrm{d}L/\mathrm{d}t = \omega_1 I_a$. Since L(t) is decreasing at $t = s$, $\mathrm{d}L/\mathrm{d}t$ is negative at $t = s$. But $\omega_1 I_a$ is always non-negative, a contradiction. Hence $(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t)) \in \mathbb{R}^7_+$ for all $t \geqslant 0$. □

Lemma 2 [Boundedness]. *The solution* $(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t))$ *in* $\mathbb{R}^7_+$ *to model* (1) *with the initial condition* $(S_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0), L(0))$ *in* $\mathbb{R}^7_+$ *is uniformly bounded.*

Proof. From Lemma 1 and assumption (A1) it follows that

$$\left|S_h(t)\right|^2 + \left|I_h(t)\right|^2 + \left|R_h(t)\right|^2 \leqslant 3N_h^2, \tag{2}$$

$$\left|S_a(t)\right|^2 + \left|I_a(t)\right|^2 + \left|R_a(t)\right|^2 \leqslant 3N_a^2. \tag{3}$$

Since $0 \leqslant I_a \leqslant N_a$, $\mathrm{d}L/\mathrm{d}t \leqslant \omega_1 N_a - (\mu_L - \mu_g)L - \mu_g c L^2$. Thus if M is the unique positive zero of the quadratic polynomial $\omega_1 N_a - (\mu_L - \mu_g)L - \mu_g c L^2$, it follows that $L(t) \leqslant M$ for all $t \geqslant 0$. This fact, together with (2) and (3), gives

$$\begin{aligned}&\big\|\big(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t)\big)\big\|\\&\quad\leqslant \big[3N_h^2 + 3N_a^2 + M^2\big]^{1/2}.\end{aligned}$$ □

Let

$$\begin{aligned}\Omega = \big\{&\big(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t)\big) \in \mathbb{R}^7:\\&S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t) \geqslant 0,\\&S_h(t) + I_h(t) + R_h(t) = N_h,\ S_a(t) + I_a(t) + R_a(t) = N_a,\\&L(t) \leqslant M\big\}.\end{aligned}$$

From Lemmas 1 and 2 it follows that Ω is a positively invariant set. We establish the existence of a unique solution for (1) in Ω in the following theorem.

Theorem 1 [Existence and uniqueness]. *Given an initial condition* $(S_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0), L(0))$ *in* Ω*, for all* $t \geqslant 0$*, model* (1) *admits a unique solution* $(S_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t), L(t))$ *in* Ω*.*

Proof. Model (1) can be rewritten in the matrix form as $\dot{X} = f(X)$, where

$$X(t) = \begin{pmatrix} S_h(t)\\ I_h(t)\\ R_h(t)\\ S_a(t)\\ I_a(t)\\ R_a(t)\\ L(t)\end{pmatrix}, \qquad f(X) = \begin{pmatrix} \mu_h N_h - \frac{\beta_{ha} I_a}{N_h} S_h - \frac{\beta_{hL} L}{N_h} S_h - \mu_h S_h + \nu_h R_h\\ \frac{\beta_{ha} I_a}{N_h} S_h + \frac{\beta_{hL} L}{N_h} S_h - \mu_h I_h - \gamma_h I_h\\ \gamma_h I_h - \nu_h R_h - \mu_h R_h\\ \mu_a N_a - \frac{\beta_{aa} I_a}{N_a} S_a - \frac{\beta_{aL} L}{N_a} S_a - \mu_a S_a + \nu_a R_a\\ \frac{\beta_{aa} I_a}{N_a} S_a + \frac{\beta_{aL} L}{N_a} S_a - \mu_a I_a - \gamma_a I_a\\ \gamma_a I_a - \nu_a R_a - \mu_a R_a\\ \omega_1 I_a - \mu_L L + \mu_g (1 - cL) L.\end{pmatrix}.$$

Ω is a compact subset of $\mathbb{R}^7_+$, and $f \in C^1(\Omega)$ is globally Lipschitz on Ω. Also, $X_0 = X(0) \in \Omega$. Thus it follows that system (1) with the initial condition X_0 has a unique solution in Ω; see [23, pp. 188–189, Thm. 3]. □

3 Equilibrium points

For an epidemic model, there are two kind of equilibrium points associated with it: disease-free equilibrium (DFE) and endemic equilibrium (EE), whose definitions are given in [26]. Model (1) is said to be at equilibrium if all the time derivatives in (1) are zero simultaneously. Hence, equating (1) to zero and substituting $I_h = I_a = L = 0$, we obtain the disease-free equilibrium of model (1) as

$$E_0 = \big(S_h^0, I_h^0, R_h^0, S_a^0, I_a^0, R_a^0, L^0\big) = (N_h, 0, 0, N_a, 0, 0, 0).$$

Equating (1) to zero and assuming $(I_h, I_a, L) \neq (0,0,0)$, we obtain the endemic equilibrium (EE) as $E_* = (S_h^*, I_h^*, R_h^*, S_a^*, I_a^*, R_a^*, L^*)$, where

$$
\begin{aligned}
S_h^* &= \frac{2c\mu_g N_h(\nu_h\gamma_h N_h\phi + \theta N_h\mu_h(\nu_h+\mu_h))}{\theta(\nu_h+\mu_h)(2c\mu_g\beta_{ha}I_a^* + \beta_{hL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*}) + 2c\mu_g\mu_h N_h)},\\
I_h^* &= \frac{N_h(\beta_{hL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*}) + 2c\mu_g\beta_{ha}I_a^*)}{\theta},\\
R_h^* &= \frac{\gamma_h N_h(\beta_{hL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*}) + 2c\mu_g\beta_{ha}I_a^*)}{(\nu_h+\mu_h)\theta},\\
S_a^* &= \frac{2c\mu_g N_a(\mu_a+\gamma_a)I_a^*}{2c\mu_g\beta_{aa}I_a^* + \beta_{aL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*})},\\
R_a^* &= \frac{\gamma_a I_a^*}{\mu_a+\nu_a}, \qquad L^* = \frac{\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*}}{2c\mu_g}
\end{aligned}
$$

with

$$
\alpha = \mu_g - \mu_L, \qquad k = 1 + \frac{\gamma_h}{\nu_h+\mu_h}, \qquad \phi = \beta_{hL}\big(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*}\big) + 2c\mu_g\beta_{ha}I_a^*,
$$

$$
\theta = 2c\mu_g N_h(\gamma_h+\mu_h) + 2c\mu_g k\beta_{ha}I_a^* + \beta_{hL}k\big(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*}\big).
$$

The roots of the polynomial $\omega_1 I_a^* + (\mu_g - \mu_L)L - c\mu_g L^2$ are $(\alpha + \sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*})/(2c\mu_g)$ and $(\alpha - \sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*})/(2c\mu_g)$. We have chosen L^* to be the positive zero of the polynomial since E_* is a point in Ω.

Lemma 3. *The endemic equilibrium point E_* of system* (1) *is unique.*

Proof. To establish that E_* is unique, it suffices to identify an I_a^* in $[0, N_a]$, which uniquely solves the equation

$$
\frac{2c\mu_g N_a(\mu_a+\gamma_a)I_a^*}{2c\mu_g\beta_{aa}I_a^* + \beta_{aL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a^*})} + I_a^* + \frac{\gamma_a I_a^*}{\mu_a+\nu_a} = N_a.
$$

Let

$$
f(I_a) = \frac{2c\mu_g N_a(\mu_a+\gamma_a)I_a}{2c\mu_g\beta_{aa}I_a + \beta_{aL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a})} + I_a + \frac{\gamma_a I_a}{\mu_a+\nu_a} - N_a.
$$

Then f is continuous on $[0, N_a]$, $f(0) < 0$ and $f(N_a) > 0$. Then, by the intermediate value theorem, it follows that there exist at least one I_a, say $I_a^* \in (0, N_a)$, such that $f(I_a^*) = 0$. Now,

$$
f'(I_a) = \frac{2\beta_{aL}c\mu_g N_a(\mu_a+\gamma_a)[\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a} - \frac{4c\mu_g\omega_1 I_a}{2\sqrt{\alpha^2+4c\mu_g\omega_1 I_a}}]}{[2c\mu_g\beta_{aa}I_a + \beta_{aL}(\alpha+\sqrt{\alpha^2+4c\mu_g\omega_1 I_a})]^2} + \frac{\gamma_a}{\mu_a+\nu_a} + 1
$$

for all $I_a \in (0, N_a)$. Since

$$\sqrt{\alpha^2+4c\mu_g\omega_1 I_a} - \frac{4c\mu_g\omega_1 I_a}{2\sqrt{\alpha^2+4c\mu_g\omega_1 I_a}} = \frac{\alpha^2+4c\mu_g\omega_1 I_a}{2} + \frac{\alpha^2}{2\sqrt{\alpha^2+4c\mu_g\omega_1 I_a}} > 0,$$

it follows that $f'(I_a) > 0$, f is strictly increasing on $[0, N_a]$, which ensure the uniqueness of I_a^*. □

4 Basic reproduction number

Basic reproduction number, usually denoted by R_0, is an important concept in epidemiology that predicts whether an infection will spread in a population. The epidemiological definition of R_0 is that it is the average number of secondary infections produced by single infectious host introduced into a totally susceptible population [8]. We employ the next-generation matrix (NGM) method proposed by Diekmann, Heesterbeek and Metz to calculate the basic reproduction number for model (1). In the next-generation matrix method, R_0 is defined as the spectral radius of the next-generation matrix. A detailed theory on R_0 can be found in [8, 21].

In (1), the infected compartments are I_h, I_a and L. Transmissions occurring in the infected compartments are rewritten as $F - V$, where F represents the rate of appearance of new infection in the compartment, and V is the net outcome (difference between incoming infected individuals and outgoing infected individuals) of the remaining transmissions like recovery, disease progression, death, birth etc. in the compartment. Here

$$F = \begin{bmatrix} \frac{\beta_{ha}S_hI_a}{N_h} + \frac{\beta_{hL}S_hL}{N_h} \\ \frac{\beta_{aa}S_aI_a}{N_a} + \frac{\beta_{aL}S_aL}{N_a} \\ \omega_1 I_a + \mu_g(1-cL)L \end{bmatrix}, \qquad V = \begin{bmatrix} (\gamma_h+\mu_h)I_h \\ (\gamma_a+\mu_a)I_a \\ \mu_L L \end{bmatrix}.$$

If J_F and J_V denote the Jacobian matrices associated with F and V, respectively,

$$J_F(E_0) = \begin{bmatrix} 0 & \beta_{ha} & \beta_{hL} \\ 0 & \beta_{aa} & \beta_{aL} \\ 0 & \omega_1 & \mu_g \end{bmatrix}, \qquad J_V(E_0) = \begin{bmatrix} \mu_h+\gamma_h & 0 & 0 \\ 0 & \mu_a+\gamma_a & 0 \\ 0 & 0 & \mu_L \end{bmatrix}.$$

The next-generation matrix is then given by

$$J_F(E_0)J_V^{-1}(E_0) = \begin{bmatrix} 0 & \frac{\beta_{ha}}{\gamma_a+\mu_a} & \frac{\beta_{hL}}{\mu_L} \\ 0 & \frac{\beta_{aa}}{\gamma_a+\mu_a} & \frac{\beta_{aL}}{\mu_L} \\ 0 & \frac{\omega_1}{\gamma_a+\mu_a} & \frac{\mu_g}{\mu_L} \end{bmatrix}$$

and

$$R_0 = \frac{\beta_{aa}\mu_L+\mu_g(\mu_a+\gamma_a)+\sqrt{[\beta_{aa}\mu_L-\mu_g(\mu_a+\gamma_a)]^2+4\omega_1\beta_{aL}\mu_L(\mu_a+\gamma_a)}}{2\mu_L(\mu_a+\gamma_a)}.$$

5 Stability analysis at disease free equilibrium point

Stability of solutions is crucial in determining biological significance to the system since solutions that shows large changes to small perturbations are often unreasonable and meaningless. The definitions of local and global asymptotic stability for an equilibrium point are given in [12, 21]. Since (A1) holds, replacing S_h by $N_h - I_h - R_h$ and S_a by $N_a - I_a - R_a$, the 7-dimensional system (1) can be reduced to an equivalent 5-dimensional system given by

$$\begin{aligned}
\frac{\mathrm{d}I_h}{\mathrm{d}t} &= \frac{\beta_{ha} I_a (N_h - I_h - R_h)}{N_h} + \frac{\beta_{hL} L (N_h - I_h - R_h)}{N_h} - \mu_h I_h - \gamma_h I_h,\\
\frac{\mathrm{d}R_h}{\mathrm{d}t} &= \gamma_h I_h - \nu_h R_h - \mu_h R_h,\\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \frac{\beta_{aa} I_a (N_a - I_a - R_a)}{N_a} + \frac{\beta_{aL} L (N_a - I_a - R_a)}{N_a} - \mu_a I_a - \gamma_a I_a,\\
\frac{\mathrm{d}R_a}{\mathrm{d}t} &= \gamma_a I_a - \nu_a R_a - \mu_a R_a,\\
\frac{\mathrm{d}L}{\mathrm{d}t} &= \omega_1 I_a - \mu_L L + \mu_g (1 - cL) L
\end{aligned} \tag{4}$$

subject to the initial conditions

$$I_h(0) > 0, \;\; R_h(0) > 0, \;\; I_a(0) > 0, \;\; R_a(0) > 0, \;\; L(0) > 0.$$

The DFE and EE for the reduced system (4) are $E_0' = (0, 0, 0, 0, 0)$ and $E_*' = (I_h^*, R_h^*, I_a^*, R_a^*, L^*)$, respectively. Now, it suffices to establish the local and global stability at E_0' and E_*' of (4). The positively invariant region for (4) is given by

$$\begin{aligned}
\Omega' = \big\{ \big(I_h(t), R_h(t), I_a(t), R_a(t), L(t)\big) \in \mathbb{R}^5 \colon\; & I_h(t), R_h(t), I_a(t), R_a(t), L(t) \geqslant 0,\\
& I_h(t) + R_h(t) \leqslant N_h, \; I_a(t) + R_a(t) \leqslant N_a, \; L(t) \leqslant M \big\}.
\end{aligned}$$

Lemma 4. *Ω' is a compact absorbing subset of $\mathbb{R}_+^5$ under system* (4)*.*

Proof. Since Ω is a closed and bounded subset of $\mathbb{R}_+^5$, which is positively invariant under system (1), Ω' is compact and positively invariant under system (4). When initial conditions are in Ω', the positive invariance implies Ω' is an absorbing set. □

5.1 Local stability of disease free equilibrium point

The DFE point E_0 of (1) is locally asymptotically stable if all the eigenvalues of the Jacobian of the reduced system (4) evaluated at E_0' have negative real part [21]. This is established in the following theorem.

Theorem 2. *The DFE E_0' of* (4) *is locally asymptotically stable if $R_0 < 1$ and unstable otherwise.*

Proof. The Jacobian matrix of reduced system (4) evaluated at E_0' is given by

$$J(E_0') = \begin{bmatrix} -\mu_h - \gamma_h & 0 & \beta_{ha} & 0 & \beta_{hL} \\ \gamma_h & -\nu_h - \mu_h & 0 & 0 & 0 \\ 0 & 0 & \beta_{aa} - \mu_a - \gamma_a & 0 & \beta_{aL} \\ 0 & 0 & \gamma_a & -\mu_a - \nu_a & 0 \\ 0 & 0 & \omega_1 & 0 & \mu_g - \mu_L \end{bmatrix}.$$

The characteristic equation of $J(E_0')$ is

$$(-\mu_h - \gamma_h - \lambda)(-\nu_h - \mu_h - \lambda)(-\mu_a - \nu_a - \lambda)\big(\lambda^2 + A_1\lambda + A_2\big) = 0, \tag{5}$$

where

$$\begin{aligned} A_1 &= \mu_a + \gamma_a - \beta_{aa} + \mu_L - \mu_g, \\ A_2 &= (\beta_{aa} - \mu_a - \gamma_a)(\mu_g - \mu_L) - \omega_1\beta_{aL}. \end{aligned}$$

The eigenvalues $-\mu_h - \gamma_h$, $-\nu_h - \mu_h$, $-\mu_a - \nu_a$ are clearly negative. By Routh–Hurwitz criterion [21], the remaining eigenvalues of (5) have negative real parts if and only if $A_1 > 0$ and $A_2 > 0$. For that, it suffices to show that

$$(\beta_{aa} - \mu_a - \gamma_a)(\mu_g - \mu_L) - \omega_1\beta_{aL} > 0 \quad \text{if } R_0 < 1. \tag{6}$$

For this implies that $(\beta_{aa} - \mu_a - \gamma_a)(\mu_g - \mu_L)$ is positive, which, together with assumption (A7), gives $\beta_{aa} < \mu_a + \gamma_a$, and hence $(\mu_a + \gamma_a - \beta_{aa} + \mu_L - \mu_g) > 0$. Let $R_0 < 1$. Then we have

$$\begin{aligned} &\mu_L\big[\beta_{aa} - 2(\mu_a + \gamma_a)\big] + \mu_g(\mu_a + \gamma_a) \\ &\quad < -\sqrt{\big[\beta_{aa}\mu_L - \mu_g(\mu_a + \gamma_a)\big]^2 + 4\omega_1\beta_{aL}\mu_L(\mu_a + \gamma_a)}, \end{aligned}$$

which in turn yields the inequality

$$\begin{aligned} &\big[\big(\beta_{aa} - 2(\mu_a + \gamma_a)\big)\mu_L + \mu_g(\mu_a + \gamma_a)\big]^2 \\ &\quad > \big[\beta_{aa}\mu_L - \mu_g(\mu_a + \gamma_a)\big]^2 + 4\omega_1\beta_{aL}\mu_L(\mu_a + \gamma_a). \end{aligned}$$

Rearranging the terms in the above yields

$$\begin{aligned} &4\mu_L(\mu_a + \gamma_a)\big\{(\mu_L - \mu_g)(\mu_a + \gamma_a) + \beta_{aa}(\mu_g - \mu_L)\big\} \\ &\quad > 4\mu_L(\mu_a + \gamma_a)\omega_1\beta_{aL}. \end{aligned} \tag{7}$$

Using the fact that $4\mu_L(\mu_a + \gamma_a)$ is positive, from (7) we obtain

$$(\mu_L - \mu_g)(\mu_a + \gamma_a - \beta_{aa}) > \omega_1\beta_{aL}.$$

Thus $(\mu_g - \mu_L)(\beta_{aa} - \mu_a - \gamma_a) - \omega_1\beta_{aL} > 0$. Hence, if $R_0 < 1$, E_0' is locally asymptotically stable. Now, for $R_0 \geqslant 1$, we have

$$\begin{aligned} &\mu_L[\beta_{aa} - 2(\mu_a + \gamma_a)] + \mu_g(\mu_a + \gamma_a) \\ &\quad \geqslant -\sqrt{[\beta_{aa}\mu_L - \mu_g(\mu_a + \gamma_a)]^2 + 4\omega_1\beta_{aL}\mu_L(\mu_a + \gamma_a)}, \end{aligned}$$

which yields

$$\begin{aligned}&\big[\big(\beta_{aa}-2(\mu_a+\gamma_a)\big)\mu_L+\mu_g(\mu_a+\gamma_a)\big]^2\\&\quad\leqslant\big[\beta_{aa}\mu_L-\mu_g(\mu_a+\gamma_a)\big]^2+4\omega_1\beta_{aL}\mu_L(\mu_a+\gamma_a).\end{aligned}$$

Rearranging the terms and taking out the positive quantity $4\mu_L(\mu_a+\gamma_a)$, we obtain $(\mu_g-\mu_L)(\beta_{aa}-\mu_a-\gamma_a)-\omega_1\beta_{aL}\leqslant 0$. Hence, if $R_0\geqslant 1$, E_0' is asymptotically unstable. □

5.2 Global stability of disease free equilibrium point

The global stability at E_0' is established using Theorem 9.2 in [12, p. 403]. The method is summarized as follows: let $X=(I_h,I_a,L)^{\mathrm{T}}$ and $Y=(R_h,R_a)^{\mathrm{T}}$ denote the infected and uninfected compartments of (4), respectively. Then system (4) can be rewritten as

$$\frac{\mathrm{d}X}{\mathrm{d}t}=-AX-\bar{F}(X,Y),\qquad \frac{\mathrm{d}Y}{\mathrm{d}t}=G(X,Y),$$

where

$$A=\begin{bmatrix}\mu_h+\gamma_h & -\beta_{ha} & -\beta_{hL}\\ 0 & \gamma_a+\mu_a-\beta_{aa} & -\beta_{aL}\\ 0 & -\omega_1 & \mu_L-\mu_g\end{bmatrix},\qquad G(X,Y)=\begin{bmatrix}\gamma_hI_h-\nu_hR_h-\mu_hR_h\\ \gamma_aI_a-\nu_aR_a-\mu_aR_a\end{bmatrix}$$

and

$$\bar{F}(X,Y)=\begin{bmatrix}\beta_{ha}I_a(1-\frac{(N_h-I_h-R_h)}{N_h})+\beta_{hL}L(1-\frac{(N_h-I_h-R_h)}{N_h})\\ \beta_{aa}I_a(1-\frac{(N_a-I_a-R_a)}{N_a})+\beta_{aL}L(1-\frac{(N_a-I_a-R_a)}{N_a})\\ c\mu_gL^2\end{bmatrix}.$$

If the matrix A is a non-singular M-matrix and $\bar{F}(X,Y)\geqslant 0$ for all $(X,Y)\in\Omega'$, then DFE E_0' is globally asymptotically stable.

Remark 1. (See [12].) A matrix A is an M-matrix if it can be expressed in the form $A=sI-B$, where $B\geqslant 0$ (B is a non-negative matrix) and $s\geqslant\rho(B)$ with $\rho(B)$ being the spectral radius of B.

Theorem 3. *The DFE E_0' of* (4) *is globally asymptotically stable if $R_0<1$ and unstable otherwise.*

Proof. We have $\det(A)=(\mu_h+\gamma_h)[(\gamma_a+\mu_a-\beta_{aa})(\mu_L-\mu_g)-\omega_1\beta_{aL}]$. Equation (6) shows that A is non-singular. Now, A can be rewritten as $A=sI-B$, where

$$B=\begin{bmatrix}-\mu_h-\gamma_h+s & \beta_{ha} & \beta_{hL}\\ 0 & \beta_{aa}-\gamma_a-\mu_a+s & \beta_{aL}\\ 0 & \omega_1 & \mu_g-\mu_L+s\end{bmatrix}.$$

Choosing $s \geqslant \{\mu_h + \gamma_h, |\xi|\}$, where

$$\xi = \frac{1}{2}\big(\mu_L - \mu_g - \beta_{aa} + \gamma_a + \mu_a \pm \sqrt{(\mu_L-\mu_g-\beta_{aa}+\gamma_a+\mu_a)^2 - 4(\mu_L-\mu_g)(\gamma_a+\mu_a-\beta_{aa}) + 4\omega_1\beta_{aL}}\big),$$

gives $s \geqslant \rho(B)$. This, together with $B \geqslant 0$ (from the choice of s), proves that A is an M-matrix. By Lemma 1, $\bar{F}(X,Y) \geqslant 0$ for all $(X,Y) \in \Omega'$. Hence, by Theorem 9.2 in [12, p. 403], $X(t) = (I_h(t), I_a(t), L(t))^{\mathrm{T}} \to (0,0,0)^{\mathrm{T}}$ as $t \to \infty$. Solving $\mathrm{d}R_h/\mathrm{d}t = \gamma_h I_h - \mu_h R_h - \nu_h R_h$, we obtain

$$R_h(t) = \mathrm{e}^{-(\mu_h+\nu_h)t}R_h(0) + \mathrm{e}^{-(\mu_h+\nu_h)t}\int_0^t \gamma_h I_h(\tau)e^{(\mu_h+\nu_h)\tau}\,\mathrm{d}\tau,$$

which tends to zero as $t \to \infty$ due to the boundedness of $I_h(t)$ established in Lemma 2. In a similar manner, solving $\mathrm{d}R_a/\mathrm{d}t$, we can show that $R_a(t) \to 0$ as $t \to \infty$. Hence $Y(t) = (R_h(t), R_a(t))^{\mathrm{T}} \to (0,0)^{\mathrm{T}}$ as $t \to \infty$. □

6 Stability analysis at endemic equilibrium point

Here we establish the local and global asymptotic stability at the endemic equilibrium (EE) point E'_* of system (4).

6.1 Local stability of endemic equilibrium point

Theorem 4. *If* $R_0 > 1$ *and*

$$\text{(C1)}\quad \left(-\frac{\beta_{aa}}{N_a}(N_a - 2I_a^* - R_a^*) + \beta_{aL}\frac{L^*}{N_a} + \mu_a + \gamma_a\right)(\mu_L - \mu_g + 2c\mu_g L^*) > \omega_1\beta_{aL},$$

then the endemic equilibrium E'_* *of* (4) *is locally asymptotically stable.*

Proof. The Jacobian matrix $J(E'_*)$ of system (4) evaluated at E'_* is given by

$$J(E'_*) = \begin{bmatrix} J_{11} & -\frac{\beta_{ha}I_a^*}{N_h} - \frac{\beta_{hL}L^*}{N_h} & J_{13} & 0 & \frac{\beta_{hL}(N_h - I_h^* - R_h^*)}{N_h} \\ \gamma_h & -\nu_h - \mu_h & 0 & 0 & 0 \\ 0 & 0 & J_{33} & -\frac{\beta_{aa}I_a^*}{N_a} - \frac{\beta_{aL}L^*}{N_a} & \frac{\beta_{aL}(N_a - I_a^* - R_a^*)}{N_a} \\ 0 & 0 & \gamma_a & -\mu_a - \nu_a & 0 \\ 0 & 0 & \omega_1 & 0 & \mu_g - \mu_L - 2c\mu_g L^* \end{bmatrix},$$

where

$$J_{11} = -\frac{\beta_{ha}I_a^*}{N_h} - \frac{\beta_{hL}L^*}{N_h} - \mu_h - \gamma_h, \qquad J_{13} = \frac{\beta_{ha}(N_h - I_h^* - R_h^*)}{N_h},$$
$$J_{33} = \frac{\beta_{aa}(N_a - 2I_a^* - R_a^*)}{N_a} - \frac{\beta_{aL}L^*}{N_a} - \mu_a - \gamma_a.$$

The characteristic equation of $J(E'_*)$ is obtained as

$$\left[(J_{11}-\lambda)(-\nu_h-\mu_h-\lambda)+\gamma_h\left(\frac{\beta_{ha}I_a^*}{N_h}+\frac{\beta_{hL}L^*}{N_h}\right)\right] \times\left[\lambda^3+A_1\lambda^2+A_2\lambda+A_3\right]=0,$$

where

$$\begin{aligned}
A_1 &= -J_{33}+2c\mu_gL^*+\mu_a+\nu_a+\mu_L-\mu_g,\\
A_2 &= -J_{33}(\mu_a+\nu_a)+\gamma_a(\frac{\beta_{aa}I_a^*}{N_a}+\frac{\beta_{aL}L^*}{N_a})+J_{33}\left(\mu_g-\mu_L-2c\mu_gL^*\right)\\
&\quad-\frac{\omega_1\beta_{aL}(N_a-I_a^*-R_a^*)}{N_a}-(\mu_a+\nu_a)(\mu_g-\mu_L-2c\mu_gL^*),\\
A_3 &= \left[J_{33}(\mu_a+\nu_a)-\gamma_a\left(\frac{\beta_{aa}I_a^*}{N_a}+\frac{\beta_{aL}L^*}{N_a}\right)\right](\mu_g-\mu_L-2c\mu_gL^*)\\
&\quad-\omega_1\beta_{aL}\frac{(N_a-I_a^*-R_a^*)}{N_a}(\mu_a+\nu_a).
\end{aligned}$$

By Lemmas 1 and 2, EE point $(I_h^*,R_h^*,I_a^*,R_a^*,L^*)$ is in Ω'. The quadratic factor of the characteristic equation has positive coefficients. Hence the two eigenvalues have negative real parts. By Routh–Hurwitz criterion [21], the remaining eigenvalues have negative real parts if $A_1,A_2,A_3>0$ and $A_1A_2-A_3>0$. Since $\mu_L>\mu_g,\omega_1\beta_{aL}>0$ and (C1) holds,

$$-J_{33}=-\frac{\beta_{aa}}{N_a}(N_a-2I_a^*-R_a^*)+\frac{\beta_{aL}L^*}{N_a}+\mu_a+\gamma_a>0. \tag{8}$$

Now, from (8) and (A7) it is immediate that $A_1>0$. (C1) along with (8) give

$$\begin{aligned}
A_2 &= -J_{33}(\mu_a+\nu_a)+\gamma_a\left(\frac{\beta_{aa}I_a^*}{N_a}+\frac{\beta_{aL}L^*}{N_a}\right)+(\mu_a+\nu_a)(\mu_L-\mu_g+2c\mu_gL^*)\\
&\quad+\left\{\left[-\frac{\beta_{aa}}{N_a}(N_a-2I_a^*-R_a^*)+\frac{\beta_{aL}L^*}{N_a}+\mu_a+\gamma_a\right](\mu_L-\mu_g+2c\mu_gL^*)\right.\\
&\quad\left.-\omega_1\beta_{aL}\right\}+\frac{\omega_1\beta_{aL}}{N_a}(I_a^*+R_a^*)>0,\\
A_3 &= \left\{\left[-\frac{\beta_{aa}}{N_a}(N_a-2I_a^*-R_a^*)+\frac{\beta_{aL}L^*}{N_a}+\mu_a+\gamma_a\right][\mu_L-\mu_g+2c\mu_gL^*]-\omega_1\beta_{aL}\right\}\\
&\quad\times(\mu_a+\nu_a)+\gamma_a\left(\frac{\beta_{aa}I_a^*}{N_a}+\frac{\beta_{aL}L^*}{N_a}\right)(\mu_L-\mu_g+2c\mu_gL^*)\\
&\quad+\frac{\omega_1\beta_{aL}}{N_a}(I_a^*+R_a^*)(\mu_a+\nu_a)>0,
\end{aligned}$$

and

$$
\begin{aligned}
&A_1A_2 - A_3\\
&\quad= \left[\left\{\left(-\frac{\beta_{aa}}{N_a}(N_a-2I_a^*-R_a^*)+\frac{\beta_{aL}L^*}{N_a}+\mu_a+\gamma_a\right)(\mu_L-\mu_g+2c\mu_gL^*)-\omega_1\beta_{aL}\right\}\right.\\
&\qquad\left.+(\mu_a+\nu_a)^2\right](\mu_L-\mu_g+2c\mu_gL^*)+\gamma_a\left(\frac{\beta_{aa}I_a^*}{N_a}+\frac{\beta_{aL}L^*}{N_a}\right)(\mu_a+\nu_a)\\
&\qquad+\left[(\mu_a+\nu_a)(\mu_L-\mu_g+2c\mu_gL^*)+\frac{\omega_1\beta_{aL}(I_a^*+R_a^*)}{N_a}-J_{33}(\mu_a+\nu_a)\right]\\
&\qquad\times(\mu_L-\mu_g+2c\mu_gL^*)-J_{33}\left[A_2+(\mu_a+\nu_a)^2\right]>0. \qquad \square
\end{aligned}
$$

6.2 Global stability of endemic equilibrium point

The global stability of the EE point E'_* is established using the Li–Muldowney geometric approach [19, 20]. Consider the system

$$\dot{x} = f(x), \quad x \in D, \tag{9}$$

where $f : D \subseteq \mathbb{R}^n \to \mathbb{R}^n$ is a continuously differentiable function. Let $x(t, x_0)$ be the solution of system (9) passing through the initial value $x(0, x_0) = x_0 \in D$. For system (9), we assume the following hypotheses.

(H1) D is simply connected.
(H2) There exists a compact absorbing set $K \subseteq D$.
(H3) x^* is a unique equilibrium of (9), which satisfies $f(x^*) = 0$.

The solution x^* will be globally asymptotically stable if it is locally asymptotically stable and all the trajectories in D converges to the unique equilibrium x^*. For that, it suffices to rule out the existence of non constant periodic solution for (9).

Let $P(x)$ be a non-singular $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function on D for which P_f is the directional derivative of P along f, and $\|P^{-1}(x)\|$ is uniformly bounded. We define

$$B = P_f P^{-1} + PJ^{[2]}P^{-1}, \tag{10}$$

where $J^{[2]}$ is the second additive compound matrix of the Jacobian J of (9). Let $\mu(B)$ denote the Lozinski measure of the matrix B on $\mathbb{R}^n$ w.r.t. norm $\|\cdot\|$ defined by $\mu(B) = \lim_{x\to 0}(|I + Bx| - 1)/x$. If $\bar{q} = \limsup_{t\to\infty} \sup_{x_0\in K}(1/t)\int_0^t \mu(B(x(s, x_0))\,\mathrm{d}t < 0$, there won't be a non-constant periodic solutions for (9). This ensures that no orbit results in a simple rectifiable curve in D, invariant for (4). The following lemma provides a sufficient condition for precluding the existence of non-constant periodic solution.

Lemma 5. (*See* [20].) *Suppose that conditions* (H1)–(H2) *are satisfied. Then there are no non-constant periodic solutions for* (4) *if the following condition holds*:

(C2) *There exist functions* $h_i(t)$ $(i = 1, 2, \ldots, n)$*, a large enough* $T_1 > 0$ *and some positive numbers* $\alpha_1, \alpha_2, \ldots, \alpha_n$ *such that for all* $t \geqslant T_1$ *and for all* $x_0 \in K$*,*

$$b_{ii}(t) + \sum_{i \neq j} \frac{\alpha_j}{\alpha_i} |b_{ij}| \leqslant h_i(t),$$

where $\limsup_{t\to\infty}(1/t)\int_0^t h_i(t)\,\mathrm{d}t < \delta_i < 0$ *for some* δ_i*, for* $i = 1, 2, \ldots, n$*. Here* $b_{ij}(t)$ *represent entries of matrix* $B(x(t, x_0))$ *in* (10)*.*

The above condition is a Bendixson criterion robust under C^1 local perturbation of f at all non-equilibrium non-wandering points for (9) and the global asymptotic stability at the endemic equilibrium point follows. The global stability of the EE point E'_* of (4) is proved using Theorem 5 and is stated as Theorem 6.

Theorem 5. (*See* [20].) *Suppose that conditions* (H1)–(H3) *are satisfied. Then the unique equilibrium* x^* *of* (9) *is globally asymptotically stable in* D *if* (C2) *holds.*

Theorem 6. *If* $\beta_{aa} \leqslant \nu_a$*,* $\gamma_h \leqslant \gamma_a$ *and* $\gamma_a \leqslant \nu_a + \nu_h$ *for* $R_0 > 1$*, then under assumptions* (H1)–(H3)*, model* (4) *is globally asymptotically stable at the endemic equilibrium* E'_* *and unstable otherwise.*

Proof. For system (4), choose $D = \mathbb{R}^5_+$ and $K = \Omega'$. Then K is a compact absorbing subset of the simply connected set D. By Lemma 3, E'_* is unique. Hence assumptions (H1)–(H3) are verified. Here the fourth additive compound matrix $J^{[4]}$ (of the Jacobian J of (4)) of order 5×5 is used instead of $J^{[2]}$ [22] and is given by

$$J^{[4]} = \begin{bmatrix} A_{11} & 0 & \frac{-\beta_{aL}(N_a - I_a - R_a)}{N_a} & 0 & \frac{-\beta_{hL}(N_h - I_h - R_h)}{N_h} \\ 0 & A_{22} & \frac{-\beta_{aa} I_a}{N_a} - \frac{\beta_{aL} L}{N_a} & 0 & 0 \\ -\omega_1 & \gamma_a & A_{33} & 0 & \frac{-\beta_{ha}(N_h - I_h - R_h)}{N_h} \\ 0 & 0 & 0 & A_{44} & \frac{-\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} \\ 0 & 0 & 0 & \gamma_h & A_{55} \end{bmatrix},$$

where

$$\begin{aligned}
A_{11} &= -\frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} - \gamma_h - \mu_h - \nu_h - \mu_h - \frac{\beta_{aL} L}{N_a} - \mu_a - \gamma_a \\
&\quad + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} - \nu_a - \mu_a, \\
A_{22} &= -\frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} - \gamma_h - \mu_h - \nu_h - \mu_h - \frac{\beta_{aL} L}{N_a} - \mu_a - \gamma_a \\
&\quad + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} + \mu_g - \mu_L - 2c\mu_g L, \\
A_{33} &= -\frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} - \gamma_h - \mu_h - \nu_h - \mu_h - \mu_a - \nu_a + \mu_g - \mu_L - 2c\mu_g L, \\
A_{44} &= -\frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} - \gamma_h - \mu_h + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} - \frac{\beta_{aL} L}{N_a} - \mu_a - \gamma_a \\
&\quad - \mu_a - \nu_a + \mu_g - \mu_L - 2c\mu_g L,
\end{aligned}$$

$$A_{55} = -\nu_h - \mu_h + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} - \frac{\beta_{aL}L}{N_a} - \mu_a - \gamma_a - \mu_a - \nu_a + \mu_g - \mu_L - 2c\mu_g L.$$

Now, choose a non-singular, continuously differentiable 5×5 matrix function P as

$$P(S_h, I_h, R_h, S_a, I_a, R_a, L) = \operatorname{diag}(L, I_a, I_a, I_h, I_h).$$

The time derivative of P along the direction of f is $P_f = \operatorname{diag}(\dot{L}, \dot{I}_a, \dot{I}_a, \dot{I}_h, \dot{I}_h)$. Then we have

$$B = P_f P^{-1} + P J^{[4]} P^{-1} + \begin{bmatrix} A_{11} + \frac{\dot{L}}{L} & 0 & \frac{-L}{I_a}\frac{\beta_{aL}(N_a - I_a - R_a)}{N_a} & 0 & \frac{-L}{I_h}\frac{\beta_{hL}(N_h - I_h - R_h)}{N_h} \\ 0 & A_{22} + \frac{\dot{I}_a}{I_a} & \frac{-\beta_{aa}I_a}{N_a} - \frac{\beta_{aL}L}{N_a} & 0 & 0 \\ \frac{-I_a}{L}\omega_1 & \gamma_a & A_{33} + \frac{\dot{I}_a}{I_a} & 0 & \frac{-I_a}{I_h}\frac{\beta_{ha}(N_h - I_h - R_h)}{N_h} \\ 0 & 0 & 0 & A_{44} + \frac{\dot{I}_h}{I_h} & \frac{-\beta_{ha}I_a}{N_h} - \frac{\beta_{hL}L}{N_h} \\ 0 & 0 & 0 & \gamma_h & A_{55} + \frac{\dot{I}_h}{I_h} \end{bmatrix}.$$

Now, taking $\alpha_i = 1$ for $i = 1, 2, 3, 4, 5$, we will define

$$h_i(t) = b_{ii} + \sum_{j=1,\, j\neq i}^{5} |b_{ij}| \quad \text{for } i = 1, 2, 3, 4, 5. \tag{11}$$

We have to show that

$$\limsup_{t\to\infty} \frac{1}{t} \int_0^t h_i(t)\, \mathrm{d}t < \delta_i < 0 \quad \text{for some } \delta_i, \text{ for } i = 1, 2, 3, 4, 5.$$

The fact that $(I_h, R_h, I_a, R_a, L) \in \Omega'$, Lemmas 1 and 2 will be used from this point onwards wherever necessary. Using (11), we obtain

$$h_1(t) = \frac{\dot{L}}{L} + A_{11} + \frac{L}{I_a}\frac{\beta_{aL}(N_a - I_a - R_a)}{N_a} + \frac{L}{I_h}\frac{\beta_{hL}(N_h - I_h - R_h)}{N_h}.$$

Since

$$-\frac{\beta_{aa}}{N_a}I_a - \frac{\beta_{aL}}{N_a}L \leqslant 0, \quad -\frac{\beta_{ha}}{N_h}I_a - \frac{\beta_{hL}}{N_h}L \leqslant 0 \quad \text{and} \quad \frac{I_a}{I_h}\frac{\beta_{ha}}{N_h}(N_h - I_h - R_h) \geqslant 0,$$

we obtain an upper bound for $h_1(t)$ by

$$\begin{aligned} h_1(t) &\leqslant \frac{\dot{L}}{L} + \left[\frac{I_a}{I_h}\frac{\beta_{ha}}{N_h}(N_h - I_h - R_h) + \frac{L}{I_h}\frac{\beta_{hL}}{N_h}(N_h - I_h - R_h) - \gamma_h - \mu_h\right] \\ &\quad + \left[\frac{\beta_{aa}}{N_a}(N_a - I_a - R_a) + \frac{L}{I_a}\frac{\beta_{aL}(N_a - I_a - R_a)}{N_a} - \gamma_a - \mu_a\right] \\ &\quad - (\mu_h + \nu_h + \mu_a + \nu_a) \\ &\leqslant \frac{\dot{L}}{L} + \frac{\dot{I}_h}{I_h} + \frac{\dot{I}_a}{I_a} - (\mu_h + \nu_h + \nu_a + \mu_a). \end{aligned}$$

Now,

$$h_2(t) = \frac{\dot{I}_a}{I_a} - \frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} - \nu_h - \mu_h - \gamma_h - \mu_h - \frac{\beta_{aL} L}{N_a} - \mu_a - \gamma_a + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} + \mu_g - \mu_L - 2c\mu_g L + \frac{\beta_{aa} I_a}{N_a} + \frac{\beta_{aL} L}{N_a}. \tag{12}$$

Equation (12) and the conditions

$$-\frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} \leqslant 0, \tag{13}$$
$$\mu_g - \mu_L - 2c\mu_g L \leqslant 0 \quad \text{and} \quad \frac{L}{I_a}\beta_{aL}\frac{(N_a - I_a - R_a)}{N_a} \geqslant 0$$

yield that

$$h_2(t) \leqslant \frac{2\dot{I}_a}{I_a} - (\mu_h + \nu_h + \mu_h + \gamma_h).$$

In a similar manner, we obtain

$$h_3(t) = \frac{I_a}{L}\omega_1 + \gamma_a + \frac{\dot{I}_a}{I_a} + A_{33} + \frac{I_a}{I_h}\frac{\beta_{ha}(N_h - I_h - R_h)}{N_h}.$$

Since $\gamma_a \leqslant \nu_a + \nu_h$ by assumption, using $-c\mu_g L \leqslant 0$ and (13),

$$h_3(t) \leqslant \frac{\dot{I}_a}{I_a} + \frac{\dot{L}}{L} + \frac{\dot{I}_h}{I_h} - (\mu_h + \mu_a).$$

From (11) and assumption that $\beta_{aa} \leqslant \nu_a$ it follows that

$$\begin{aligned} h_4(t) &= \frac{\dot{I}_h}{I_h} + \left(\frac{\beta_{ha} I_a}{N_h} + \frac{\beta_{hL} L}{N_h}\right) - \frac{\beta_{ha} I_a}{N_h} - \frac{\beta_{hL} L}{N_h} - \mu_h - \gamma_h \\ &\quad + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} - \frac{\beta_{aL} L}{N_a} - \mu_a - \gamma_a - \mu_a - \nu_a \\ &\quad + \mu_g - \mu_L - 2c\mu_g L \\ &\leqslant \frac{\dot{I}_h}{I_h} - (\mu_h + \gamma_h + \mu_h + \nu_h + \mu_a + \mu_L - \mu_g). \end{aligned}$$

Again using Eq. (11),

$$h_5(t) = \frac{\dot{I}_h}{I_h} + \gamma_h - \nu_h - \mu_h + \frac{\beta_{aa}(N_a - 2I_a - R_a)}{N_a} - \frac{\beta_{aL} L}{N_a} - \mu_a - \gamma_a - \mu_a - \nu_a + \mu_g - \mu_L - 2c\mu_g L. \tag{14}$$

Equation (14), together with the assumptions $\gamma_h \leqslant \gamma_a$ and $\beta_{aa} \leqslant \nu_a$, gives

$$h_5(t) \leqslant \frac{\dot{I}_h}{I_h} - (\mu_a + \mu_h + \nu_h + \mu_a + \mu_L - \mu_g).$$

By integrating the function $h_1(t)$ and taking its limit supremum as $t \to \infty$, we obtain

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t h_1(t)\,\mathrm{d}t \leqslant \lim_{t\to\infty} \frac{1}{t}\left[\ln\frac{L(t)}{L(0)} + \ln\frac{I_a(t)}{I_a(0)} + \ln\frac{I_h(t)}{I_h(0)}\right] - (\mu_h+\nu_h+\nu_a+\mu_a)$$
$$< -(\mu_h+\nu_h+\nu_a+\mu_a) < 0.$$

Similarly, we have

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t h_2(t)\,\mathrm{d}t \leqslant \lim_{t\to\infty} \frac{2}{t}\ln\frac{I_a(t)}{I_a(0)} - (\mu_h+\nu_h+\mu_h+\gamma_h) < 0,$$

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t h_3(t)\,\mathrm{d}t \leqslant \lim_{t\to\infty} \frac{1}{t}\left[\ln\frac{I_a(t)}{I_a(0)} + \ln\frac{L(t)}{L(0)} + \ln\frac{I_h(t)}{I_h(0)}\right] - (\mu_h+\mu_a) < 0,$$

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t h_4(t)\,\mathrm{d}t \leqslant \lim_{t\to\infty} \frac{1}{t}\ln\frac{I_h(t)}{I_h(0)} - (\mu_h+\gamma_h+\mu_h+\nu_h+\mu_a+\mu_L-\mu_g) < 0,$$

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t h_5(t)\,\mathrm{d}t \leqslant \lim_{t\to\infty} \frac{1}{t}\ln\frac{I_h(t)}{I_h(0)} - (\mu_a+\mu_h+\nu_h+\mu_a+\mu_L-\mu_g) < 0.$$

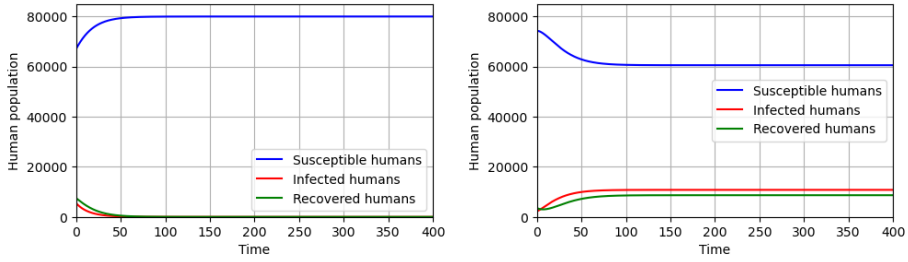
Hence the EE point E'_* of (4) is globally asymptotically stable. □

7 Numerical simulations

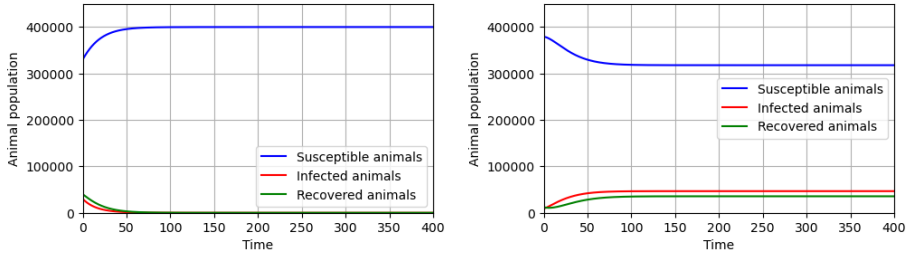
This final part validates the theoretical results obtained through numerical simulations carried out in SageMath. The parameter values corresponding to $R_0 \approx 0.9366 < 1$ and $R_0 \approx 8.3487 > 1$ are given in Table 2.

Table 2. Parameter values for DFE and EE.

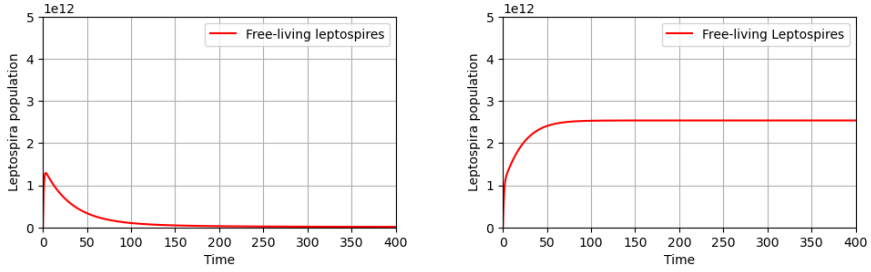
Symbol	Values for $R_0 < 1$	Values for $R_0 > 1$	Reference
N_h	80000	80000	assumed
N_a	400000	400000	assumed
β_{hL}	4×10^{-12}	4×10^{-10}	assumed
β_{aL}	1.5×10^{-11}	1.5×10^{-9}	assumed
β_{ha}	9.633×10^{-5}	9.633×10^{-5}	[5]
β_{aa}	10^{-4}	10^{-4}	[6]
μ_h	3.9×10^{-5}	3.9×10^{-5}	[13]
μ_a	9.13242×10^{-4}	9.13242×10^{-4}	[13]
ν_h	0.089	0.089	[15]
ν_a	0.083	0.083	[15]
γ_h	0.0714285714	0.0714285714	[13]
γ_a	0.064	0.064	[15]
ω_1	7×10^7	7×10^7	[10]
μ_L	0.02381	0.02381	[13]
μ_g	0.005	0.005	assumed
c	10^{-10}	10^{-10}	assumed



(a) Human population dynamics



(b) Animal population dynamics



(c) Leptospira population dynamics

Figure 2. Behaviour of solutions of model (1) when $R_0 < 1$ and $R_0 > 1$

Figure 2 shows the behaviour of solution of model (1) for $R_0 < 1$ and $R_0 > 1$. It can be seen that the solution tends to DFE $(80000, 0, 0, 400000, 0, 0, 0)$ when $R_0 < 1$, and the solution tends to EE $(60200, 11000, 8800, 314400, 48000, 36600, 2.573 \cdot 10^{12})$ when $R_0 > 1$.

It is evident from R_0 that the parameters β_{aL}, β_{aa}, ω_1, μ_g and μ_L influence the leptospirosis transmission dynamics. Figure 3 indicates that an increase in the values of β_{aL}, β_{aa}, ω_1 and μ_g leads to greater disease spread, whereas increasing μ_L reduces the disease spread.

Finally, the leptospirosis model (1) is fitted to the reported case data from Kerala, India, spanning from January 2021 to December 2022, using the parameter values listed in Table 2. The reported case data used for fitting is taken from the website of Directorate of Health Services, Government of Kerala [30] and listed in Table 3. Figure 4 shows the actual and predicted cumulative number of leptospirosis cases in Kerala over this period.

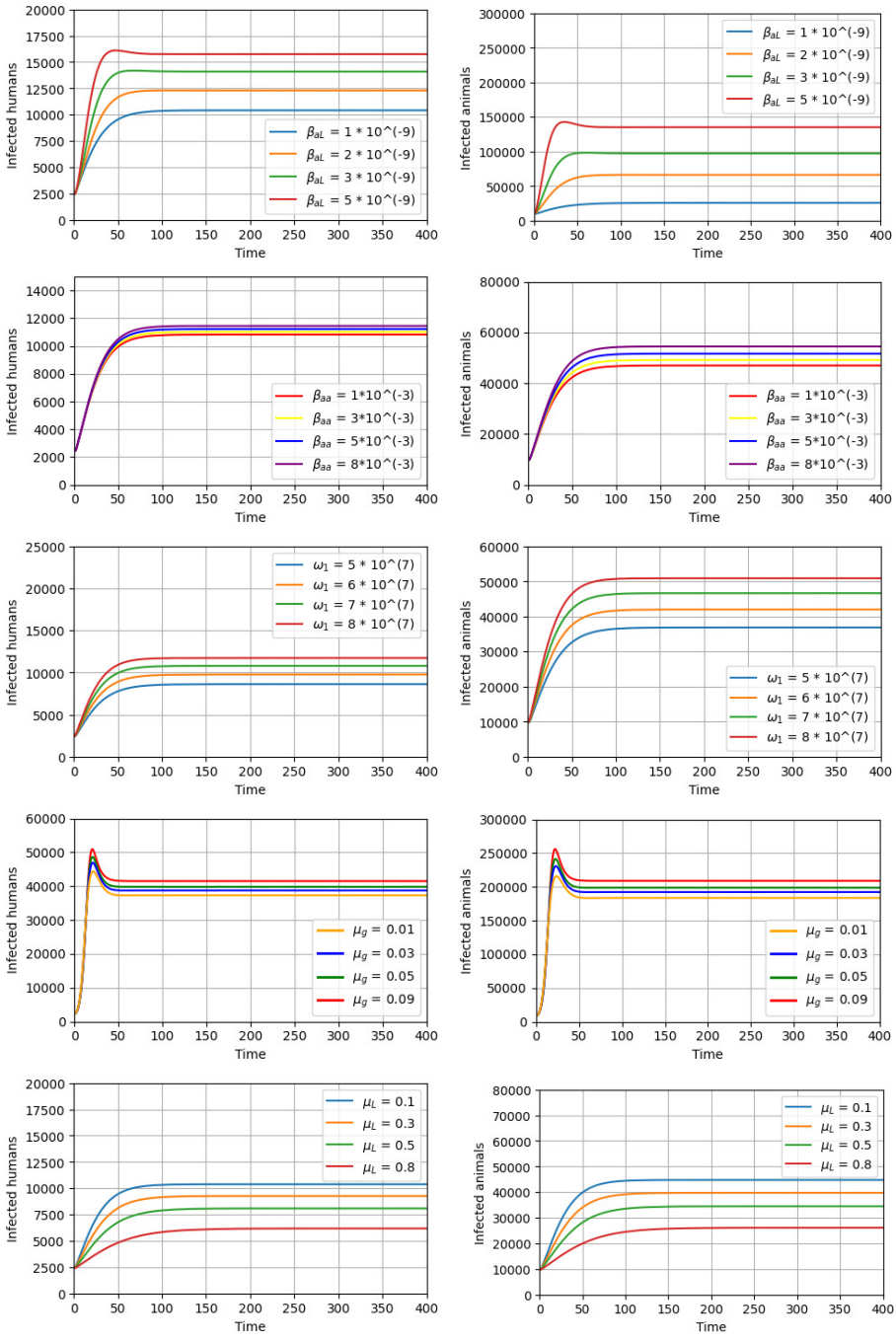


Figure 3. Dynamics of leptospirosis for varying values of β_{aL}, β_{aa}, ω_1, μ_g, and μ_L.

Table 3. Monthly leptospirosis cases in 2021 and 2022.

Cases	Month											
	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
2021	75	111	57	42	40	132	152	181	179	210	285	281
2022	107	109	123	123	167	235	305	377	274	233	226	203

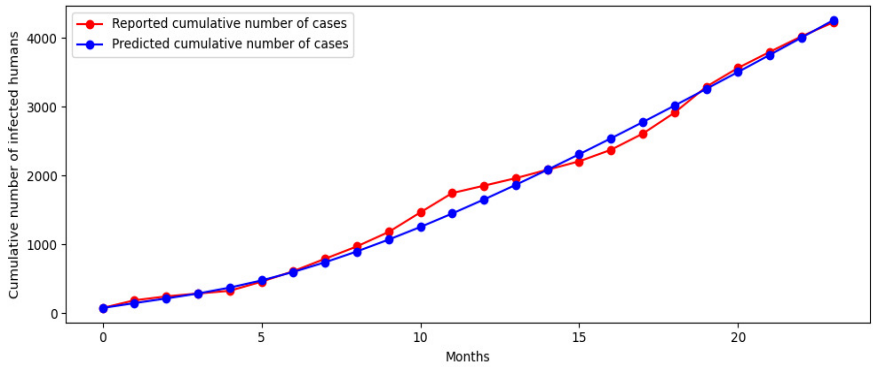


Figure 4. Cumulative number of leptospirosis cases in Kerala from January 2021 to December 2022: predicted vs. actual data.

8 Conclusion

Leptospirosis is already an endemic in several countries, and the frequent occurrence of floods along with recurrent climate changes contaminate the environment thereby facilitating the spread. Hence the presence of free-living leptospira in the environment requires far more attention than it currently receives. In this article, we studied the significance of free-living leptospires in the leptospirosis transmission by formulating a biologically well-posed model involving humans, animals and contaminated environment. The basic reproduction number derived for model (1) shows that the parameters β_{aa}, β_{aL}, ω_1 and μ_g positively affect the value of R_0, whereas the parameter μ_L negatively influences R_0. For better understanding of the long-term behaviour of the disease nature, the local and global asymptotic stabilities at the disease-free and endemic equilibrium points are also established. Finally, the numerical simulations are performed to support the theoretical results obtained, and model (1) is fitted to the reported case data from Kerala, India.

Our study suggest that to reduce the impact of the disease, the presence of free-living leptospires in environment, transmission between environment and animals, as well as between animals themselves and the shedding of leptospira must be decreased considerably. Targeted rodent control, use of protective gear in contaminated environment, improving sanitation and reducing exposure to leptospira can effectively reduce the transmission of leptospirosis. As a next step, we plan to carry out sensitivity analysis for the formulated model to identify the parameters having major impact on the leptospirosis spread and formulate an optimal control problem to derive necessary optimality conditions to reduce the leptospirosis disease transmission.

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Conflicts of interest. The authors declare no conflicts of interest.

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