



Research article

Dynamic analysis and optimal control of leptospirosis based on Caputo fractional derivative

Ling Zhang, Xuewen Tan*, Jia Li and Fan Yang

School of Mathematics and Computer Science, Yunnan Minzu University, Kunming 650031, China

* **Correspondence:** Email: tanxw0910@ymu.edu.cn.

Abstract: Caputo fractional derivative solves the fractional initial value problem in Riemann-Liouville (R-L) fractional calculus. The definition of a Caputo-type derivative is in the same form as the definition of an integral differential equation, including the restriction of the value of the integral derivative to the value of the unknown function at the endpoint $t = a$. Therefore, this paper introduced the Caputo fractional derivative (CFD) to establish the transmission model of leptospirosis. First, to ensure that the model had a particular significance, we proved the dynamic properties of the model, such as nonnegative, boundedness, and stability of the equilibrium point. Second, according to the existence mode and genetic characteristics of pathogenic bacteria of leptospirosis, and from the perspective of score optimal control, we put forward measures such as wearing protective clothing, hospitalization, and cleaning the environment to prevent and control the spread of the disease. According to the proposed control measures, a control model of leptospirosis was established, and a forward-backward scanning algorithm (FB algorithm) was introduced to optimize the control function. Three different disease control strategies were proposed. Finally, the numerical simulation of different fractional orders used the fde12 (based on Adams–Bashforth–Moulton scheme) solver. The three optimized strategies, A, B, and C, were compared and analyzed. The results showed that the optimized control strategy could shorten the transmission time of the disease by about 80 days. Therefore, the above methods contributed to the study of leptospirosis and the World Health Organization.

Keywords: leptospirosis; Caputo fractional derivative; optimal control; numerical analysis

1. Introduction

Throughout human history, the emergence of infectious diseases has not only affected the health and quality of life of many people but also claimed many lives. At the same time, it also significantly impacts the economy, healthcare system, and social stability and poses a severe threat to public health. Therefore, preventing and effectively controlling infectious diseases can reduce the economic losses

caused by diseases and have great significance for protecting personal health, improving public health, and maintaining social stability. Leptospirosis is a zoonotic disease. According to the World Health Organization (WHO), the incidence of endemic human leptospirosis is estimated at 5 per 100,000 per year, and the incidence of epidemic human leptospirosis is 14 per 100,000 per year [1]. However, recent estimates suggest that there are 1.03 million cases of leptospirosis each year and that most leptospirosis cases and deaths occur in tropical regions. Men aged 20–29 years have the highest incidence, while older men aged 50–59 years have the highest estimated mortality [2]. According to studies, the symptoms of infected people after infection are varied; there are many sporadic clinical manifestations, from asymptomatic or flu, fever, to various organ failure, and death [3–6]. Because leptospirosis is an acute febrile disease, usually characterized initially by fever which can be confused with diseases such as dengue fever, malaria, and typhoid through carelessness, coupled with the rapid progression of the disease and poor sanitation, leptospirosis has spread in many developing countries [4, 5]. The reproduction of this pathogenic bacteria occurs mainly in the renal tubules of mammals [3, 5, 7]. The bacteria is excreted into the environment through urine, and humans become infected through contact with an infected animal's urine (or other bodily fluids other than saliva) or contact by water, soil, or food contaminated by an infected animal [7]. As a result, leptospirosis is more likely to infect people with frequent contact with animals, contaminated water, or soil. Examples include slaughterhouse workers, veterinarians, hunters, sewage treatment workers, farmers, construction workers, aquaculturists, etc. [7–10]. Outbreaks of leptospirosis are often associated with natural disasters, with flooding proving to be one of the most critical drivers of infection on islands and in Asia, along with and potential risks to rodent exposure and hygiene [11]. Therefore, studying the spread and control of leptospirosis is of great practical significance to safeguard people's lives and health and maintain social stability and order.

Mathematical models are often built to analyze the characteristics of disease transmission. For example, Guo and Li established a differential equation system to simulate the transmission process of the novel coronavirus. They established a zoning model to explore the competitive transmission characteristics of the Omicron and the Delta strains, which provided scientific suggestions for controlling the spread of the epidemic [12, 13]. However, in recent years, fractional calculus has been used more and more in biological and medical modeling than traditional integer models. For example, Zhang et al. discussed the stability of integer-order and fractional-order systems in depth and intuitively demonstrated many advantages of fractional-order systems through time response [14]. Tuan proposed a mathematical model of Caputo's fractional derivative transmission of COVID-19, solving the system and numerical simulation of worldwide transmission based on obtained reproduction numbers indicating that the epidemic is continuing [15]. Using Matlab, Almeida proposed a model based on Caputo fractional derivatives to simulate a chickenpox outbreak among schoolchildren in Shenzhen, China [16]. Therefore, fractional models are more accurate in capturing the dynamic characteristics of biological systems. Introducing fractional differential equations in studying infectious disease models provides a novel and accurate mathematical tool for studying infectious disease transmission.

Regarding the mathematical model of leptospirosis, Mukdasai uses the advantages of random number sets supervised neural networks to simulate a fractional leptospirosis model numerically [17]. Ullah and Khan developed a fractional model of leptospirosis with the Atangana-Baleanu (AB) derivative, and the results show that fractional order plays a vital role in better understanding the dynamics of the disease [18]. Okosun et al. has studied control measures, vaccination, and treatment to control the spread of leptospirosis and advocates that policymakers should work on vaccination and treatment

regimes that will better help control the epidemic [19]. Baca-Carrasco et al. developed a susceptible and infected model and proposed several intervention techniques to control the disease, suggesting that rodents and other animals that serve as bacterial hosts and are bacteria-free in the environment can infect humans [20]. Khan et al. analyzed leptospirosis with saturation incidence [21]. Alalhareth et al. used fractional derivatives to analyze the transmission dynamics of leptospirosis with environmental effects and bifurcations [22]. Ngoma et al. investigated a fractional model of the transmission dynamics of leptospirosis in the environmental sub-compartment [23]. Qu et al. analyzed the dynamics of leptospirosis in the context of piece smart classical global and classical fractional operators [24].

In the above studies on leptospirosis, different scholars considered different influencing factors and established different mathematical models to analyze the transmission dynamics of leptospirosis. Few scholars have proposed control measures to optimize disease control. Therefore, based on previous studies, this paper mainly made the following contributions: (1) The Caputo fractional differential (CFD) equation model with good disease fitting effect was introduced to study the transmission characteristics of leptospirosis. Considering the order mismatch between the two sides of the model, fractional order parameters are introduced on the right side. (2) To make the fractional-order model established by us meaningful, we analyze its dynamic characteristics including non-negative, bounded, local, and global stability of local equilibrium point (EEP) and disease-free equilibrium point (DFEP). (3) To control the spread of leptospirosis, we proposed three control functions, developed three different control strategies, and adopted the forward-backward scanning algorithm to optimize the control functions. Finally, numerical simulation is carried out using the fde12 solver. The influence of different fractions on disease transmission and the effect of different control strategies on disease control were analyzed.

The structure of this paper is as follows: The second section gives some theorems of fractional calculus. The third part constructs a fractional model of leptospirosis according to the existence mode and transmission characteristics of leptospirosis. The fourth part proves the fractional order model's non-negative, boundedness, and stability. Section 5 establishes the optimal control model of leptospirosis according to the three control functions we proposed, and the forward and backward scan algorithm is introduced to optimize the control function. The sixth part is the numerical simulation results using the fde12 solver. The seventh part is the conclusion.

2. Database description

This section introduces some theorems and properties of fractional calculus [25].

Definition 2.1. [26] When $\alpha > 0$, the left Riemann-Liouville fractional integral is

$${}_t D_t^{-\alpha} g(t) = \frac{1}{\Gamma(\alpha)} \int_{t_0}^t (t-s)^{\alpha-1} g(s) ds,$$

or

$${}_t I_t^\alpha g(t) = \frac{1}{\Gamma(\alpha)} \int_{t_0}^t (t-s)^{\alpha-1} g(s) ds.$$

$\Gamma(\cdot)$ is the gamma function.

Definition 2.2. When $\alpha \in (n-1, n]$, the left CFD is

$${}_t^C D_t^\alpha g(t) = {}_t I_t^{n-\alpha} \frac{d^n}{dt^n} g(t) = \frac{1}{\Gamma(n-\alpha)} \int_{t_0}^t (t-s)^{n-\alpha-1} g^{(n)}(s) ds,$$

where n is a positive integer.

Definition 2.3. The Laplace transform of $g(t)$ is $G(s)$, and when $\alpha \in (n - 1, n]$ (n is a positive integer), the Laplace transform of the left CFD of $g(t)$ is

$$\mathcal{L}\{{}^C D^\alpha g(t), s\} = s^\alpha G(s) - \sum_{i=0}^{n-1} s^{\alpha-i-1} g^{(i)}(0).$$

Definition 2.4. Two-argument Mittag-Leffler function is

$$E_{l,r}(z) = \sum_{n=0}^{\infty} \frac{z^n}{\Gamma(ln + r)}, \quad l > 0, r > 0.$$

Satisfy the following properties [27]:

$$E_{l,r}(z) = zE_{l,l+r}(z) + \frac{1}{\Gamma(r)}. \quad (2.1)$$

Definition 2.5. The Laplace transform of $t^{r-1}E_{l,r}(\pm\lambda t^l)$ is

$$\mathcal{L}\{t^{r-1}E_{l,r}(\pm\lambda t^l)\} = \frac{s^{l-r}}{s^l \mp \lambda}. \quad (2.2)$$

Definition 2.6. (Routh–Hurwitz stability criterion) The closed-loop characteristic equation of the known system is

$$D(s) = a_n s^n + a_{n-1} s^{n-1} + \dots + a_1 s + a_0 = 0. \quad (2.3)$$

Suppose all coefficients in the above equation are real numbers and $a_n > 0$. In that case, the necessary condition for system stability is that all coefficients of the system characteristic equation are positive numbers.

3. Fractional order model

Considering the existence mode and transmission characteristics of leptospirosis, inspired by article [18], we choose the following mathematical model as the basis model and then introduce the CFD to study the transmission of leptospirosis. The model consists of a human host and a susceptible vector. The human host is divided into three compartments: susceptible population $S_h(t)$, infected population $I_h(t)$, and recovering population $R_h(t)$. The medium consists of two compartments of susceptible animal $S_v(t)$ and infected animal $I_v(t)$. Thus, the following differential equation form is obtained:

$$\begin{cases} \frac{dS_h}{dt} = b_1 - \beta_1 S_h I_h - \beta_2 S_h I_v + \lambda_h R_h - \mu_h S_h, \\ \frac{dI_h}{dt} = \beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_h + \delta_h + \gamma_h) I_h, \\ \frac{dR_h}{dt} = \gamma_h I_h - (\mu_h + \lambda_h) R_h, \\ \frac{dS_v}{dt} = b_2^\alpha - \gamma_v S_v - \beta_3 S_v I_h, \\ \frac{dI_v}{dt} = \beta_3 S_v I_h - (\gamma_v + \delta_v) I_v. \end{cases} \quad (3.1)$$

Here, the overall $N_h(t)$ is by susceptibility, infection, and recovery of three room area, namely, $N_h(t) = S_h(t) + I_h(t) + R_h(t)$. At the same time, medium total $N_v(t)$ is composed of infection and restoring the two districts, that is, $N_v(t) = S_v(t) + I_v(t)$.

In addition, Table 1 is the meaning of the parameters in the model.

Table 1. Model 3.1 parameter description.

Parameter	Description
b_1	Humans recruitment rate
b_2	Animals recruitment rate
λ_h	Recovery rate of infected persons
δ_h	Human disease fatality rate
μ_h	Natural human mortality rate
δ_v	Vector-induced mortality
γ_v	Vector natural mortality
γ_h	The transfer rate from I_h to R_h
β_1	The contact rate between S_h and I_h
β_2	The contact rate between the I_v and the S_h
β_3	The contact rate between S_v and I_h

The initial constraints:

$$S_h(0) = S_{h_0}, I_h(0) = I_{h_0}, R_h(0) = R_{h_0}, S_v(0) = S_{v_0}, I_v(0) = I_{v_0}. \quad (3.2)$$

By definition, the CFD solves the fractional initial value problem in R-L fractional calculus. The definition of the Caputo-type derivative takes the same form as the integer order differential equation, including the restriction of the integer order derivative value to the value of the unknown function at the endpoint $t = a$, and so on. Therefore, the description of the Caputo differential equation is more suitable for nonzero initial value problems. So, we introduce the Caputo fractional differential equation to reconstruct the model. At the same time, to solve the problem of inconsistent orders on both sides of the equal sign, the parameters are modified to fractional order, and the equation obtained is as follows:

$$\begin{cases} {}^C_0 D_t^\alpha S_h = b_1^\alpha - \beta_1^\alpha S_h I_h - \beta_2^\alpha S_h I_v + \lambda_h^\alpha R_h - \mu_h^\alpha S_h, \\ {}^C_0 D_t^\alpha I_h = \beta_1^\alpha S_h I_h + \beta_2^\alpha S_h I_v - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_h, \\ {}^C_0 D_t^\alpha R_h = \gamma_h^\alpha I_h - (\mu_h^\alpha + \lambda_h^\alpha) R_h, \\ {}^C_0 D_t^\alpha S_v = b_2^\alpha - \gamma_v^\alpha S_v - \beta_3^\alpha S_v I_h, \\ {}^C_0 D_t^\alpha I_v = \beta_3^\alpha S_v I_h - (\gamma_v^\alpha + \delta_v^\alpha) I_v. \end{cases} \quad (3.3)$$

Figure 1 shows the transmission process of leptospirosis.

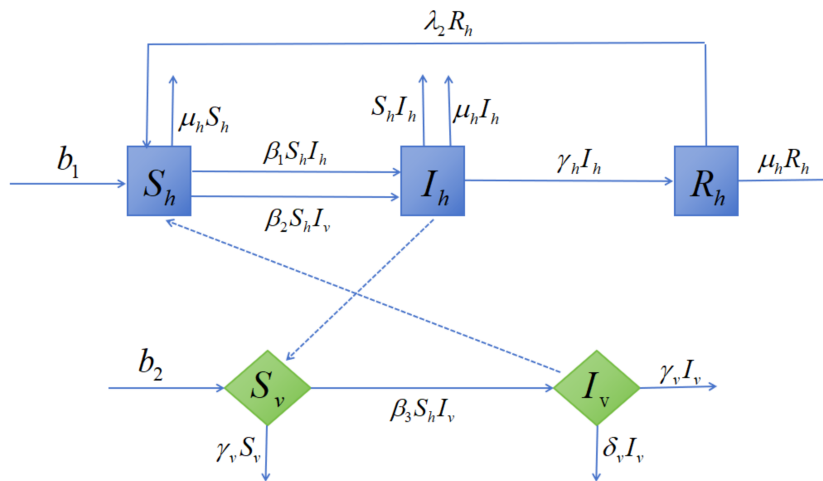


Figure 1. The transmission of leptospirosis.

4. Model analysis

In this section, to ensure that our model (3.3) makes sense, first we prove the nonnegative property of the model and the boundedness. Then, the local stability of DFEP and EEP is proved, respectively. Finally, the global stability of DFEP and EEP is proved by constructing different Lyapunov functions.

4.1. Positivity and boundedness

We need to look at the generalized mean value theorem to prove nonnegativity.

Lemma 4.1. [28] *Let $f(x) \in C[c, d]$ and $D_c^\alpha f(x) \in C[c, d]$, for $0 < \alpha \leq 1$, then we have*

$$f(x) = f(c) + \frac{1}{\Gamma(\alpha)} (D_c^\alpha f)(\xi)(x - c)^\alpha,$$

with $c \leq \xi \leq x, \forall x \in (c, d]$.

Remark 4.1. *Obviously, according to the generalized mean value theorem above Lemma 4.1, when $\forall x \in (c, d], \alpha \in (0, 1], f(x) \in C[c, d], D_c^\alpha f(x) \in C[c, d]$, if $D_c^\alpha f(x) \geq 0$, then $f(x)$ is nondecreasing; If $D_c^\alpha f(x) \leq 0$, then $f(x)$ is nonincreasing.*

Theorem 4.1. *The region*

$$\Omega_+ = \{(S_h, I_h, R_h, S_v, I_v); S_h > 0, I_h > 0, R_h > 0, S_v > 0, I_v > 0\},$$

is a positivity invariant set for the model (3.3).

Proof. In [29], the problem of fractional differential equations and their initial values is investigated in

detail. Then, the nonnegativities of the solution for model (3.3) are

$$\begin{aligned} {}_0^C D_t^\alpha S_h |_{S_h=0} &= b_1^\alpha + \lambda_h^\alpha R_h > 0, \\ {}_0^C D_t^\alpha I_h |_{I_h=0} &= \beta_2^\alpha S_h I_v > 0, \\ {}_0^C D_t^\alpha R_h |_{R_h=0} &= \gamma_h^\alpha I_h > 0, \\ {}_0^C D_t^\alpha S_v |_{S_v=0} &= b_2^\alpha > 0, \\ {}_0^C D_t^\alpha I_v |_{I_v=0} &= \beta_3^\alpha S_v I_h > 0. \end{aligned}$$

According to Remark 4.1, the nonnegative property of model (3.3) is proved.

Theorem 4.2. *Boundedness of region*

$$\Omega = \left\{ (S_h, I_h, R_h, S_v, I_v,) : 0 \leq S_h + I_h + R_h \leq 1, 0 \leq S_v \leq \frac{b_2^\alpha}{\gamma_v^\alpha \beta_3^\alpha}, 0 \leq I_v \leq \frac{\beta_3^\alpha}{\gamma_v^\alpha + \delta_v^\alpha} \right\}.$$

Proof. Let $N = S_h + I_h + R_h$. $N(t)$ transforms \tilde{N} after the Laplace transform and can be obtained from model (3.3)

$${}_0^C D_t^\alpha N(t) = b_1^\alpha - \mu_h^\alpha N(t) - S_h I_h \leq b_1^\alpha - \mu_h^\alpha N(t).$$

That is,

$${}_0^C D_t^\alpha N(t) \leq b_1^\alpha - \mu_h^\alpha N(t).$$

We take the Laplace transform of both sides of this inequality

$$s^\alpha \tilde{N}(s) - s^{\alpha-1} N(0) \leq \frac{b_1^\alpha}{s} - b_1^\alpha \tilde{N}(s).$$

Then,

$$\tilde{N}(s) \leq \frac{s^{-1}}{s^\alpha + b_1^\alpha} b_1^\alpha + \frac{s^{\alpha-1}}{s^\alpha + b_1^\alpha}.$$

From the Eqs (2.1) and (2.2), inverse Laplace transformation can obtain the above inequality.

$$\begin{aligned} N(t) &\leq b_1^\alpha t^\alpha E_{\alpha, \alpha+1}(-b_1^\alpha t^\alpha) + N(0) E_{\alpha, 1}(-b^\alpha t^\alpha) \\ &\leq 1 - E_{\alpha, 1}(-b^\alpha t^\alpha) + N(0) E_{\alpha, 1}(-b^\alpha t^\alpha) \leq 1. \end{aligned}$$

The bounded range of $N(t)$ is $0 \leq N \leq 1$. Besides, for S_v ,

$$\begin{aligned} {}_0^C D_t^\alpha S_v &= b_2^\alpha - \gamma_v^\alpha \delta_v^\alpha - \beta_3^\alpha S_v^\alpha I_h \\ &\leq b_2^\alpha - \gamma_v^\alpha \delta_v^\alpha - \beta_3^\alpha S_v. \end{aligned}$$

Then,

$$0 \leq S_v \leq \frac{b_2^\alpha}{\gamma_v^\alpha \beta_3^\alpha}.$$

Similarly, we can get

$$0 \leq I_v \leq \frac{\beta_3^\alpha}{\gamma_v^\alpha + \delta_v^\alpha}.$$

4.2. Stability

To start, set the right side of the model (3.3) to 0.

$$\begin{cases} b_1^\alpha - \beta_1^\alpha S_h I_h - \beta_2^\alpha S_h I_v + \lambda_h^\alpha R_h - \mu_h^\alpha S_h = 0, \\ \beta_1^\alpha S_h I_h + \beta_2^\alpha S_h I_v - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_h = 0, \\ \gamma_h^\alpha I_h - (\mu_h^\alpha + \lambda_h^\alpha) R_h = 0, \\ b_2^\alpha - \gamma_v^\alpha S_v - \beta_3^\alpha S_v I_h = 0, \\ \beta_3^\alpha S_v I_h - (\gamma_v^\alpha + \delta_v^\alpha) I_v = 0. \end{cases}$$

Solving the above equation gives two EPs, which are the DFEP $X^0 = (\frac{b_1^\alpha}{\mu_h^\alpha}, 0, 0, \frac{b_2^\alpha}{\gamma_v^\alpha}, 0)$ and the EEP $X^* = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$, where

$$\begin{cases} S_h^* = \frac{k_1 k_3 (\gamma_v^\alpha + \beta_3^\alpha I_h^*)}{b_2^\alpha \beta_2^\alpha \beta_3^\alpha + \beta_1^\alpha k_3 (\gamma_v^\alpha + \beta_3^\alpha I_h^*)}, \\ I_h^* = \frac{k_2 R_h^*}{\gamma_h^\alpha}, \\ R_h^* = \frac{\gamma_h^\alpha I_h^*}{k_2}, \\ S_v^* = \frac{b_2^\alpha}{\gamma_v^\alpha + \beta_3^\alpha I_h^*}, \\ I_v^* = \frac{\beta_3^\alpha b_2^\alpha I_h^*}{k_3 (\gamma_v^\alpha + \beta_3^\alpha I_h^*)}. \end{cases}$$

Here, $\kappa_1 = b^\alpha + \rho^\alpha, \kappa_2 = b^\alpha + v^\alpha, \kappa_3 = \eta^\alpha + b^\alpha - \theta^\alpha$.

Next, the basic reproduction number (BRM) is calculated through the next generation matrix (NGM) [30]. Let compartment $\varphi = (I_h, R_h, S_v, I_v, S_h)^\top$, then the model (3.3) is rewritten as

$$\mathcal{F}(\varphi) = \begin{pmatrix} \beta_1^\alpha S_h I_h + \beta_2^\alpha S_h I_v \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \mathcal{V}(\varphi) = \begin{pmatrix} k_1 I_h \\ k_2 R_h - \gamma_h^\alpha I_h \\ \beta_3^\alpha S_v I_h + \gamma_v^\alpha S_v - b_2^\alpha \\ k_3 I_v - \beta_3^\alpha S_v I_h \\ \beta_1^\alpha S_h I_h + \beta_2^\alpha S_h I_v + \mu_h^\alpha S_h - \lambda_h^\alpha R_h - b_1^\alpha \end{pmatrix}.$$

Because the basic regeneration number is $R_0 = \rho(\mathbb{F}\mathbb{V}^{-1})$, the matrices \mathbb{F} and \mathbb{V} are as follows [31]:

$$\mathbb{F} = \frac{\partial \mathcal{F}(\varphi)}{\partial \varphi} = \begin{pmatrix} \beta_1^\alpha S_h & 0 & 0 & \beta_2^\alpha S_h & \beta_1^\alpha I_h \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$\mathbb{V} = \frac{\partial \mathcal{V}(\varphi)}{\partial \varphi} = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 \\ -\gamma_h^\alpha & k_2 & 0 & 0 & 0 \\ \beta_3^\alpha S_v & 0 & \beta_3^\alpha I_h + \gamma_v^\alpha & 0 & 0 \\ -\beta_3^\alpha S_v & 0 & -\beta_3^\alpha I_h & -k_3 & 0 \\ \beta_1^\alpha S_h & -\lambda_h^\alpha & 0 & \beta_2^\alpha S_h & \beta_1^\alpha I_h + \beta_2^\alpha I_v + \mu_h^\alpha \end{pmatrix}.$$

By calculating $R_0 = \mathbb{FV}^{-1}$, we get the BRN R_0 :

$$R_0 = R_1 + R_2 = \frac{\beta_2^\alpha \beta_3^\alpha b_1^\alpha b_2^\alpha}{k_1 k_3 \gamma_v^\alpha \mu_h^\alpha} + \frac{\beta_1^\alpha b_1^\alpha}{\mu_h^\alpha k_1}.$$

The BRN R_0 means that if $R_0 < 1$, the disease will become extinct. On the contrary, if $R_0 > 1$, the disease will persist. Next, we will explain the stability of $X^0 = (\frac{b_1^\alpha}{\mu_h^\alpha}, 0, 0, \frac{b_2^\alpha}{\gamma_v^\alpha}, 0)$ and $X^* = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$.

Theorem 4.3. *If $R_0 < 1$, then the disease-free equilibrium point X^0 of model (3.3) is locally asymptotically stable.*

Proof. To begin, solve the Jacobian matrix

$$\mathcal{M} = \begin{bmatrix} (-\beta_1^\alpha I_h - \beta_2^\alpha I_v - \mu_h^\alpha) & -\beta_1^\alpha S_h & \lambda_h^\alpha & 0 & -\beta_2^\alpha S_h \\ \beta_1^\alpha I_h + \beta_2^\alpha I_v & (\beta_1^\alpha S_h - k_1) & 0 & 0 & \beta_2^\alpha S_h \\ 0 & \gamma_h^\alpha & -k_2 & 0 & 0 \\ 0 & -\beta_3^\alpha S_v & 0 & (-\gamma_v^\alpha - \beta_3^\alpha I_h) & 0 \\ 0 & \beta_3^\alpha S_v & 0 & \beta_3^\alpha I_h & -k_3 \end{bmatrix}.$$

Bring in the DFEP, and get by simplification X^0

$$\mathcal{M}(X^0) = \begin{bmatrix} -\mu_h^\alpha & -k_1 & \lambda_h^\alpha & 0 & 0 \\ 0 & \beta_1^\alpha S_h - k_1 & 0 & 0 & \beta_2^\alpha S_h \\ 0 & \gamma_h^\alpha & -k_2 & 0 & 0 \\ 0 & 0 & 0 & -\gamma_v^\alpha & -k_3 \\ 0 & \beta_3^\alpha S_h & 0 & 0 & -k_3 \end{bmatrix}.$$

We know that to judge model (3.3) to be locally asymptotically stable at X^0 , all eigenvalues of its matrix must satisfy the following equation [32].

$$|\arg(\text{eig}(\mathcal{M}))| = |\arg(\lambda_i)| > \alpha \frac{\pi}{2}. \tag{4.1}$$

The characteristic equation obtained from $|(\mathcal{M}(X^0) - \lambda I)| = 0$ is as follows:

$$(\lambda + \mu_h^\alpha)(\lambda + k_2)(\lambda + \gamma_v^\alpha)(\lambda^2 + (k_1 + k_3 - \beta_1^\alpha S_h)\lambda + k_1 k_3 - \beta_1^\alpha S_h k_3 - \beta_2^\alpha S_h \beta_3^\alpha S_v) = 0.$$

Obviously, $\lambda = -k_2$, $\lambda = -\mu_h^\alpha$, $\lambda = -\gamma_v^\alpha$ all satisfy Eq (4.1). Therefore, we only need to discuss the following formula:

$$\lambda^2 + \rho_1 \lambda + \rho_2 = 0.$$

When $R_0 \leq 1$, it satisfies

$$\begin{aligned}\rho_1 &= k_1 + k_3 - \beta_1^\alpha S_h = k_1 + k_3 - \frac{\beta_1^\alpha b_1^\alpha}{\mu_h^\alpha} \\ &= k_3 + k_1 \left(1 - \frac{\beta_1^\alpha b_1^\alpha}{\mu_h^\alpha k_1}\right) = k_3 + k_1(1 - R_2) > 0, \\ \rho_2 &= k_1 k_3 - \beta_1^\alpha S_h k_3 - \beta_2^\alpha S_h \beta_3^\alpha S_v = k_1 k_3 - \left(\frac{\beta_1^\alpha b_1^\alpha k_3}{\mu_h^\alpha} + \frac{\beta_2^\alpha \beta_3^\alpha b_1^\alpha b_2^\alpha}{\mu_h^\alpha \gamma_h^\alpha}\right) \\ &= k_1 k_3 \left(1 - \left(\frac{\beta_1^\alpha b_1^\alpha}{\mu_h^\alpha k_1} + \frac{\beta_2^\alpha \beta_3^\alpha b_1^\alpha b_2^\alpha}{\mu_h^\alpha \gamma_h^\alpha k_1 k_3}\right)\right) \\ &= k_1 k_3(1 - R) > 0.\end{aligned}$$

Then, $\rho_1 \rho_2 \geq \rho_3$, therefore, according to the Routh-Hurwitz criterion, the DFEP X^0 of model (3.3) is locally asymptotically stable.

Theorem 4.4. *If $R_0 \geq 1$, then the EEP X^* of model (3.3) is locally asymptotically stable.*

Proof. The \mathcal{M} at the EEP X^* is

$$\mathcal{M}(X^*) = \begin{bmatrix} (-\beta_1^\alpha I_h^* - \beta_2^\alpha I_v^* - \mu_h^\alpha) & -\beta_1^\alpha S_h^* & \lambda_h^\alpha & 0 & -\beta_2^\alpha S_h^* \\ \beta_1^\alpha I_h^* + \beta_2^\alpha I_v^* & (\beta_1^\alpha S_h^* - k_1) & 0 & 0 & \beta_2^\alpha S_h^* \\ 0 & \gamma_h^\alpha & -k_2 & 0 & 0 \\ 0 & -\beta_3^\alpha S_v^* & 0 & (-\gamma_v^\alpha - \beta_3^\alpha I_h^*) & 0 \\ 0 & \beta_3^\alpha S_v^* & 0 & \beta_3^\alpha I_h^* & -k_3 \end{bmatrix}.$$

For easy calculation, the matrix is expressed as

$$\mathcal{M}(X^*) = \begin{bmatrix} -B_1 & -B_7 & \lambda_h^\alpha & 0 & -B_6 \\ B_2 & B_3 & 0 & 0 & -B_6 \\ 0 & \gamma_h^\alpha & -k_2 & 0 & 0 \\ 0 & -B_5 & 0 & -B_4 & 0 \\ 0 & B_5 & 0 & B_8 & -k_3 \end{bmatrix},$$

where

$$B_1 = \beta_1^\alpha I_h + \beta_2^\alpha I_v + \mu_h^\alpha, B_2 = \beta_1^\alpha I_h + \beta_2^\alpha I_v, B_3 = \beta_1^\alpha S_h - k_1, B_4 = \gamma_v^\alpha + \beta_3^\alpha I_h,$$

$$B_5 = \beta_3^\alpha S_v, B_6 = \beta_2^\alpha S_h, B_7 = \beta_1^\alpha S_h, B_8 = \beta_3^\alpha I_h.$$

Its characteristic equation is expressed as

$$\lambda^5 + G_1 \lambda^4 + G_2 \lambda^3 + G_3 \lambda^2 + G_4 \lambda + G_5 = 0,$$

where

$$\begin{aligned}
 G_1 &= B_4 + k_2 + k_3 - (B_1 + B_3), \\
 G_2 &= B_4 k_3 + B_5 B_6 - (B_4 + k_3)(k_2 - B_1 - B_3) + (B_2 B_7 - B_1 B_3 - k_2(B_1 + B_3)), \\
 G_3 &= k_2(B_2 B_7 - B_1 B_3) - B_2 \gamma_h^\alpha \lambda_h^\alpha + [(B_2 B_7 - B_1 B_3) - k_2(B_1 + B_3)](B_4 + k_3) \\
 &\quad + [k_2 - (B_1 + B_3)] B_4 k_3 + [\gamma_h^\alpha + (2\mu_h^\alpha + \lambda_h^\alpha)] B_5 B_6, \\
 G_4 &= [k_2(B_2 B_7 - B_1 B_3) - B_2 \gamma_h^\alpha \lambda_h^\alpha](B_4 + k_3) + B_4 k_3 [(B_2 B_7 - B_1 B_3) - k_2(B_1 + B_3)] \\
 &\quad + [k_2 \mu_h^\alpha + \gamma_h^\alpha (2\mu_h^\alpha + \lambda_h^\alpha)] B_5 B_6, \\
 G_5 &= [k_2(B_2 B_7 - B_1 B_3) - B_2 \gamma_h^\alpha \lambda_h^\alpha] B_4 k_3 + B_5 B_6 k_2 \mu_h^\alpha \gamma_v^\alpha.
 \end{aligned}$$

According to the Routh-Hurwitz criterion

$$\begin{aligned}
 D_5 &= \begin{vmatrix} G_1 & G_3 & G_5 & 0 & 0 \\ 1 & G_2 & G_4 & 0 & 0 \\ 0 & G_1 & G_3 & G_5 & 0 \\ 0 & 1 & G_2 & G_4 & 0 \\ 0 & 0 & G_1 & G_3 & G_5 \end{vmatrix} \\
 &= G_1 G_4 G_5 (G_2 G_3 - G_1 G_4) - G_1 G_2^2 G_5^2 + G_1 G_4 G_5^2 - G_4 G_5 (G_3^2 - G_1 G_5) + G_2 G_3 G_5^2 - G_5^2 > 0.
 \end{aligned}$$

According to the Routh-Hurwitz criterion, if $D_5 > 0$ and $R_0 \geq 1$, $G_i > 0$ ($i = 1, 2, 3, 4, 5$), then the eigenvalue of the eigen-equation is negative. The EEP X^* of model (3.3) is locally asymptotically stable.

Next, we will introduce a lemma so that we can better construct Lyapunov to prove the global stability of the model (3.3) [33–35].

Lemma 4.2. *When $t \geq 0$, $\alpha \in (0, 1)$, $f(t) \in R^+$ is continuous and differentiable.*

Satisfy the following relation

$$\frac{1}{2} {}_0^C D_t^\alpha g^2(t) \leq {}_0^C D_t^\alpha g(t), \quad (4.2)$$

and

$${}_0^C D_t^\alpha \left(g(t) - g^* - g^* \ln \frac{g(t)}{g^*} \right) \leq \left(1 - \frac{g^*}{g(t)} \right) {}_0^C D_t^\alpha g(t). \quad (4.3)$$

The inequality takes an equal sign if, and only if, $\alpha = 1$.

Theorem 4.5. *If $R_0 < 1$, the model (3.3) at the DFEP X^0 is global asymptotically stable.*

Proof. Define $F_1(t)$ as a Lyapunov function:

$$F_1(t) = \frac{\beta_3^\alpha b_2^\alpha}{\gamma_v^\alpha} I_h + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_v + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) S_v,$$

$t \geq 0$, $F_1(t)$ is continuous and positive definite, and when $S_h(t) = I_h(t) = R_h(t) = S_v(t) = I_v(t) = 0$, it satisfies $F_1(t) = 0$.

Now, we have

$$\begin{aligned} {}_0^C D_t^\alpha F_1(t) &= \left(\frac{\beta_3^\alpha b_2^\alpha}{\gamma_v^\alpha}\right) {}_0^C D_t^\alpha I_h + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) {}_0^C D_t^\alpha I_v + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) {}_0^C D_t^\alpha S_v \\ &= \frac{\beta_3^\alpha b_2^\alpha}{\gamma_v^\alpha} (\beta_1^\alpha S_h I_h + \beta_2^\alpha S_h I_v - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_h) \\ &\quad + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (\beta_3^\alpha S_v I_h - (\gamma_v^\alpha + \delta_v^\alpha) I_v) \\ &\quad + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (b_2^\alpha - \gamma_v^\alpha S_v - \beta_3^\alpha S_v I_h). \end{aligned}$$

Bring into $X^0 = (\frac{b_1^\alpha}{\mu_h^\alpha}, 0, 0, \frac{b_2^\alpha}{\gamma_v^\alpha}, 0)$, then

$$\begin{aligned} {}_0^C D_t^\alpha F_1(t) &= \frac{\beta_1^\alpha \beta_3^\alpha b_1^\alpha b_2^\alpha}{\gamma_v^\alpha \mu_h^\alpha} I_h + \frac{\beta_3^\alpha \beta_2^\alpha b_1^\alpha b_2^\alpha}{\gamma_v^\alpha \mu_h^\alpha} I_v - \frac{\beta_3^\alpha b_2^\alpha (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha)}{\gamma_v^\alpha} I_h + \frac{\beta_3^\alpha b_2^\alpha (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha)}{\gamma_v^\alpha} I_h \\ &\quad - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (\gamma_v^\alpha + \delta_v^\alpha) I_v + (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) b_2^\alpha - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) \gamma_v^\alpha \frac{b_2^\alpha}{\gamma_v^\alpha} \\ &\quad - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) \beta_3^\alpha \frac{b_2^\alpha}{\gamma_v^\alpha} I_h \\ &= \frac{\beta_1^\alpha \beta_3^\alpha b_1^\alpha b_2^\alpha}{\gamma_v^\alpha \mu_h^\alpha} I_h - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) \beta_3^\alpha \frac{b_2^\alpha}{\gamma_v^\alpha} I_h + \frac{\beta_3^\alpha \beta_2^\alpha b_1^\alpha b_2^\alpha}{\gamma_v^\alpha \mu_h^\alpha} I_v \\ &\quad - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (\gamma_v^\alpha + \delta_v^\alpha) I_v \\ &= \frac{k_1^\alpha \beta_3^\alpha b_2^\alpha I_h}{\gamma_v^\alpha} \left(1 - \frac{\beta_1^\alpha b_1^\alpha}{\mu_h^\alpha k_1}\right) - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (\gamma_v^\alpha + \delta_v^\alpha) I_v \left(1 - \frac{\beta_2^\alpha \beta_3^\alpha b_1^\alpha b_2^\alpha}{k_1 k_3 \gamma_v^\alpha \mu_h^\alpha}\right) \\ &= - \frac{k_1^\alpha \beta_3^\alpha b_2^\alpha I_h}{\gamma_v^\alpha} (1 - R_2) - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (\gamma_v^\alpha + \delta_v^\alpha) I_v (1 - R_1). \end{aligned}$$

Thus,

$${}_0^C D_t^\alpha F_1(t) = - \frac{k_1^\alpha \beta_3^\alpha b_2^\alpha I_h}{\gamma_v^\alpha} (1 - R_2) - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) (\gamma_v^\alpha + \delta_v^\alpha) I_v (1 - R_1).$$

So if $R_0 < 1$, then $R_1 < 1$ and $R_2 < 1$, thus ${}_0^C D_t^\alpha F_1(t) \leq 0$. Besides, ${}_0^C D_t^\alpha F_1(t) = 0$ if, and only if, $I_h(t) = I_v(t) = 0$. Therefore, based on Lassalle's invariance theorem, all solutions tend to the maximum invariant set [36, 37]. When $R_0 < 1$, the DFEP X^0 of model (3.3) is globally asymptotically stable.

Theorem 4.6. *The model (3.3) at the EEP $X^* = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$ is global asymptotically stable if $R_0 > 1$.*

Proof. Define $F_2(t)$ as a Lyapunov function.

$$\begin{aligned} F_2(t) &= \frac{1}{\mu_h^\alpha} (S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*}) + \frac{1}{\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha} (I_h - I_h^* - I_h^* \ln \frac{I_h}{I_h^*}) \\ &\quad + \frac{1}{\mu_h^\alpha + \lambda_h^\alpha} (R_h - R_h^* - R_h^* \ln \frac{R_h}{R_h^*}) + \frac{Q}{\gamma_v^\alpha + \delta_v^\alpha} (I_v - I_v^* - I_v^* \ln \frac{I_v}{I_v^*}), \end{aligned}$$

where $Q = R_0 - 1$, and if $R_0 > 1, Q > 0$. When $t \geq 0, F_2(t)$ is continuous and positive definite. $F_2(t) = 0$ if, and only if, $S_h(t) = S_h^*(t), I_h(t) = I_h^*(t), R_h(t) = R_h^*(t), S_v(t) = S_v^*(t), I_v(t) = I_v^*(t)$. Now, we have

$$\begin{aligned} {}_0^C D_t^\alpha F_2(t) &= \frac{1}{\mu_h^\alpha} (S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*}) + \frac{1}{\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha} (I_h - I_h^* - I_h^* \ln \frac{I_h}{I_h^*}) \\ &\quad + \frac{1}{\mu_h^\alpha + \lambda_h^\alpha} (R_h - R_h^* - R_h^* \ln \frac{R_h}{R_h^*}) + \frac{Q}{\gamma_v^\alpha + \delta_v^\alpha} (I_v - I_v^* - I_v^* \ln \frac{I_v}{I_v^*}). \end{aligned}$$

According to Lemma 4.2, we get

$$\begin{aligned} {}_0^C D_t^\alpha F_2(t) &\leq \frac{1}{\mu_h^\alpha} \left(1 - \frac{S_h^*}{S_h(t)}\right) {}_0^C D_t^\alpha S_h(t) + \frac{1}{\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha} \left(1 - \frac{I_h^*}{I_h(t)}\right) {}_0^C D_t^\alpha I_h(t) \\ &\quad + \frac{1}{\mu_h^\alpha + \lambda_h^\alpha} \left(1 - \frac{R_h^*}{R_h(t)}\right) {}_0^C D_t^\alpha R_h(t) + \frac{Q}{\gamma_v^\alpha + \delta_v^\alpha} \left(1 - \frac{I_v^*}{I_v(t)}\right) {}_0^C D_t^\alpha I_v(t) \\ &= \frac{1}{\mu_h^\alpha} \left(1 - \frac{S_h^*}{S_h(t)}\right) [b_1^\alpha - \beta_1^\alpha S_h I_h - \beta_2^\alpha S_h I_v + \lambda_h^\alpha R_h - \mu_h^\alpha S_h] \\ &\quad + \frac{1}{\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha} \left(1 - \frac{I_h^*}{I_h(t)}\right) [\beta_1^\alpha S_h I_h + \beta_2^\alpha S_h I_v - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_h] \\ &\quad + \frac{1}{\mu_h^\alpha + \lambda_h^\alpha} \left(1 - \frac{R_h^*}{R_h(t)}\right) [\gamma_h^\alpha I_h - (\mu_h^\alpha + \lambda_h^\alpha) R_h] \\ &\quad + \frac{Q}{\gamma_v^\alpha + \delta_v^\alpha} \left(1 - \frac{I_v^*}{I_v(t)}\right) [\beta_3^\alpha S_v I_h - (\gamma_v^\alpha + \delta_v^\alpha) I_v]. \end{aligned}$$

Bring into the endemic conditions, and we get

$${}_0^C D_t^\alpha F_2(t) \leq -\frac{(S_h(t) - S_h^*)^2}{S_h(t)} - \frac{(I_h(t) - I_h^*)^2}{I_h(t)} - \frac{(R_h(t) - R_h^*)^2}{R_h(t)} - \frac{(I_v(t) - I_v^*)^2}{I_v(t)} (R_0 - 1).$$

When $R_0 > 1, {}_0^C D_t^\alpha F_2(t) \leq 0$ and ${}_0^C D_t^\alpha F_2(t) = 0$, if, and only if, $S_h(t) = S_h^*(t), I_h(t) = I_h^*(t), R_h(t) = R_h^*(t), I_v(t) = I_v^*(t)$. So, based on Lassalle’s invariance theorem, all solutions tend to the maximum invariant set. Therefore, when $R_0 > 1$, the EEP X^* is globally asymptotically stable.

In this section, we proved nonnegativity and boundedness. The local stability of the equilibrium point is proved according to the Rouse-Hurwitz criterion. Finally, the Lyapunov function is constructed to prove the global stability of the equilibrium point. It can be seen that the model (3.3) has certain theoretical significance for our study of leptospirosis.

5. Optimal control problem

In this section, first, because the pathogenic bacteria exists in animal urine, soil, and contaminated water, three control variables $u_1, u_2,$ and u_3 were developed according to the presence status of the bacteria to prevent people from being infected with the bacteria. Among them, u_1 represents personal protection; for example, people who often walk in sewage, people who work in slaughterhouses, etc., can wear protective clothing at work, regularly disinfect, and pay attention to hygiene. u_2 indicates timely symptomatic treatment and hospitalization for observation. u_3 refers to cleaning the environment,

landfill treatment of infected animals, and timely cleaning and sterilization of the polluted environment. Second, the control function is optimized by the forward-backward scanning algorithm. According to our control variables u_1, u_2 , and u_3 , we get the following equations:

$$\begin{cases} {}^C_0D_t^\alpha S_h(t) = b_1^\alpha - \beta_1^\alpha S_h I_h - (1 - u_1)\beta_2^\alpha S_h I_v + \lambda_h^\alpha R_h - \mu_h^\alpha S_h, \\ {}^C_0D_t^\alpha I_h(t) = \beta_1^\alpha S_h I_h + (1 - u_1)\beta_2^\alpha S_h I_v - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_h, \\ {}^C_0D_t^\alpha R_h(t) = \gamma_h^\alpha I_h - (\mu_h^\alpha + \lambda_h^\alpha) R_h + u_2 I_h, \\ {}^C_0D_t^\alpha S_v(t) = b_2^\alpha - \gamma_v^\alpha S_v - \beta_3^\alpha S_v I_h - u_3 S_v, \\ {}^C_0D_t^\alpha I_v(t) = \beta_3^\alpha S_v I_h - (\gamma_v^\alpha + \delta_v^\alpha) I_v - u_3 I_v, \end{cases} \quad (5.1)$$

subject to the initial conditions

$$S_h(0) = S_{h_0}, I_h(0) = I_{h_0}, R_h(0) = R_{h_0}, S_v(0) = S_{v_0}, I_v(0) = I_{v_0}. \quad (5.2)$$

Our control goal is to reduce the number of I_h and I_v with leptospirosis and at a low cost, that is, the minimum objective function is

$$\text{Min } \mathcal{J}(u) = {}_0I_T^\alpha \left(m_1 I_h + m_2 I_v + \frac{1}{2} (n_1 u_1^2 + n_2 u_2^2 + n_3 u_3^2) \right). \quad (5.3)$$

m_1, m_2 are weights of infected people and infected animals. The weights of the costs of the three measures are n_1, n_2 , and n_3 . The implementation cost of measures A, B, and C are $\frac{1}{2}n_1 u_1^2, \frac{1}{2}n_2 u_2^2$, and $\frac{1}{2}n_3 u_3^2$, respectively.

For the above problem, the Lagrange function \mathcal{L} and the Hamiltonian function \mathcal{H} are as follows:

$$\mathcal{L}(I_h, I_v, u_1, u_2, u_3, t) = \left(m_1 I_h + m_2 I_v + \frac{1}{2} (n_1 u_1^2 + n_2 u_2^2 + n_3 u_3^2) \right),$$

and

$$\begin{aligned} \mathcal{H}(S_h, I_h, R_h, S_v, I_v, v_i, u_j, t) &= \mathcal{L}(I_h, I_v, u_1, u_2, u_3, t) + v_1 {}^C_0D_t^\alpha S_h(t) + v_2 {}^C_0D_t^\alpha I_h(t) \\ &\quad + v_3 {}^C_0D_t^\alpha R_h(t) + v_4 {}^C_0D_t^\alpha S_v(t) + v_5 {}^C_0D_t^\alpha I_v(t) \\ &= m_1 I_h + m_2 I_v + \frac{1}{2} (n_1 u_1^2 + n_2 u_2^2 + n_3 u_3^2) \\ &\quad + v_1 (b_1^\alpha - \beta_1^\alpha S_h I_h - (1 - u_1)\beta_2^\alpha S_h I_v + \lambda_h^\alpha R_h - \mu_h^\alpha S_h) \\ &\quad + v_2 (\beta_1^\alpha S_h I_h + (1 - u_1)\beta_2^\alpha S_h I_v - (\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha) I_h) \\ &\quad + v_3 (\gamma_h^\alpha I_h - (\mu_h^\alpha + \lambda_h^\alpha) R_h + u_2 I_h) \\ &\quad + v_4 (b_2^\alpha - \gamma_v^\alpha S_v - \beta_3^\alpha S_v I_h - u_3 S_v) \\ &\quad + v_5 (\beta_3^\alpha S_v I_h - (\gamma_v^\alpha + \delta_v^\alpha) I_v - u_3 I_v). \end{aligned}$$

The adjoint variable of S_h, I_h, R_h, S_v, I_v are v_1, v_2, v_3, v_4, v_5 , respectively. Next, we will use Agrawal's method to solve fractional optimal control problems (FOCP) [38].

Theorem 5.1. *Set u_1^*, u_2^* , and u_3^* for the control system of control variables. Then, the control variable*

can be expressed as

$$\begin{cases} u_1^* = \max \left\{ 0, \min \left(1, \frac{(v_1 - v_2)\beta_2^\alpha S_h I_v}{n_1} \right) \right\}, \\ u_2^* = \max \left\{ 0, \min \left(1, \frac{(v_2 - v_3)I_h(t)}{n_2} \right) \right\}, \\ u_3^* = \max \left\{ 0, \min \left(1, \frac{v_4 S_v + v_5 I_v}{n_3} \right) \right\}, \end{cases} \quad (5.4)$$

where the adjoint variables v_1, v_2, v_3, v_4 , and v_5 satisfying

$$\begin{cases} v_1' = (v_1 - v_2)\beta_1^\alpha I_h + (1 - u_1)(v_1 - v_2)\beta_2^\alpha I_v + v_1 u_h^\alpha, \\ v_2' = -m_1 + (v_1 - v_2)\beta_1^\alpha S_h + v_2(\mu_h^\alpha + \delta_h^\alpha + \gamma_h^\alpha + u_2) - v_3(\gamma_h^\alpha + u_2) + (v_4 - v_5)\beta_3^\alpha S_v, \\ v_3' = v_3(\mu_h^\alpha + \lambda_h^\alpha) - v_1 \lambda_h^\alpha, \\ v_4' = (\gamma_v^\alpha + u_3)v_4 + (v_4 - v_5)\beta_3^\alpha I_h, \\ v_5' = -m_2 + (v_1 - v_2)(1 - u_1)\beta_2^\alpha S_h + v_5(\delta_v^\alpha + \gamma_v^\alpha + u_3), \end{cases} \quad (5.5)$$

have boundary conditions or transverse conditions

$$v_1(t_f) = 0, \quad v_2(t_f) = 0, \quad v_3(t_f) = 0, \quad v_4(t_f) = 0 \quad \text{and} \quad v_5(t_f) = 0.$$

Proof. The adjoint variable Eq (5.5) can be obtained from the \mathcal{H}

$$\frac{dv_1}{dt} = -\frac{\partial \mathcal{H}}{\partial S_h}, \quad \frac{dv_2}{dt} = -\frac{\partial \mathcal{H}}{\partial I_h}, \quad \frac{dv_3}{dt} = -\frac{\partial \mathcal{H}}{\partial R_h}, \quad \frac{dv_4}{dt} = -\frac{\partial \mathcal{H}}{\partial S_v} \quad \text{and} \quad \frac{dv_5}{dt} = -\frac{\partial \mathcal{H}}{\partial I_v},$$

and have boundary conditions or transverse conditions.

Eq (5.4) and the optimal control of the variable u_1^* , u_2^* , and u_3^* can be obtained by the following

$$\frac{\partial \mathcal{H}}{\partial u_1} = 0, \quad \frac{\partial \mathcal{H}}{\partial u_2} = 0, \quad \text{and} \quad \frac{\partial \mathcal{H}}{\partial u_3} = 0.$$

Completion of theorem proving.

In general, one way to calculate v_1 is to use Eq (5.4) as the iterative formula [39]. However, Eq (5.4) is not strictly an iterative formula. Therefore, we use forward and reverse scanning to optimize the control variables.

6. Numerical simulation result

In this section, because fde12 has good accuracy and stability in CFD numerical simulation [40, 41], we use the fde12 solver to simulate models of different fractional orders. At the same time, we propose three disease transmission control strategies and optimize the control function by using the forward-reverse scanning method. Detailed steps of the forward-backward scan algorithm and its convergence are in references [42, 43]. Finally, the characteristics of these three strategies are analyzed. MATLAB (R2018b) was used to simulate the model.

6.1. Disease control strategies

Control the spread of leptospirosis and take into account its cost. We developed the following three control strategies:

Strategy A: Combination of strengthening personal protection, timely hospitalization of infected patients, and cleaning up the environment after slaughtering infected animals (*i.e.*, $u_1, u_2, u_3 \neq 0$).

Strategy B: The combination of timely hospitalization of infected patients and cleaning up the environment after slaughtering infected animals (*i.e.*, $u_2, u_3 \neq 0$, and $u_1 = 0$).

Strategy C: Combination of enhanced personal protection and timely hospitalization of infected patients (*i.e.*, $u_1, u_2 \neq 0$, and $u_3 = 0$).

Parameter values and references are listed in Table 2.

Table 2. Estimated values and references.

Parameters	Estimated value	Reference
b_1	1.6	[44]
b_2	1.2	[44]
λ_h	0.00057	[44]
δ_h	0.0004	[14]
μ_h	0.0094	assumed
δ_v	0.0094	assumed
γ_v	0.17	[44]
γ_h	0.067	assumed
β_1	0.00098	assumed
β_2	0.0098	[45]
β_3	0.0078	[44]

6.2. Simulation result analysis

Figure 2 shows how $S_h, I_h, R_h, S_v,$ and I_v change over time when α takes different values. We found that over time, susceptible people move into infected people. Therefore, Figure 2a gradually becomes stable after decreasing, while Figure 2b first increases slightly, then decreases, and then becomes stable. Second, looking at Figure 2d,e, we find that the same is true of animals, with the number of susceptible animals transferring over time to infected animals. Finally, the study found that, without any control, leptospirosis stabilized after 86 days ($I_h \leq 17, I_v \leq 6$).

Figure 3 shows the numerical analysis results when α takes different values under control Strategy A. We can see that the amount of I_h in Figure 3b decreases, suddenly approaches a stable position on day 10, and stabilizes after 20 days. The validity of our control strategy is verified. It is worth mentioning that when $\alpha = 1$, the amount of I_h goes up and then down. When α takes fractional order, the amount of I_h is directly reduced. It has been proved that fractional order is more beneficial for us in studying the spread control of leptospirosis. In addition, in control Strategy A, our control variable u_3 is to disinfect and clean the environment after killing some infected animals, so the susceptible animals in Figure 3d and the infected animals in Figure 3e directly decline and then stabilize, reflecting our control variable u_3 .

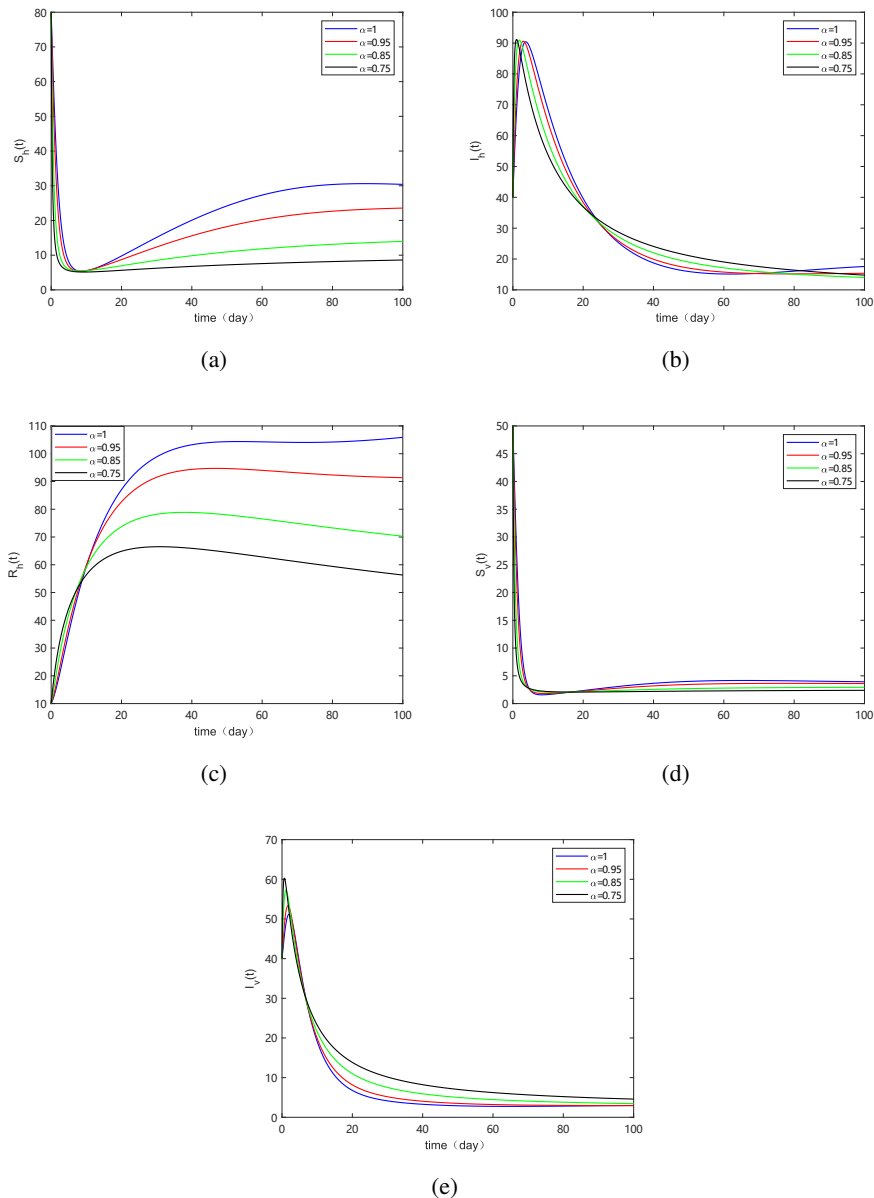


Figure 2. On the premise of no control, when α respectively take different values, $S_h(t)$, $I_h(t)$, $R_h(t)$, $S_v(t)$, $I_v(t)$, of the results of numerical simulation.

Figure 4 compares the control effects of Strategies A, B, and C on leptospirosis. First of all, it can be seen from Figure 4b that when we adopt control strategy A (personal protection, timely treatment of I_h , killing animals to clean the environment), the number of I_h directly declines and then becomes stable. In the case of Strategy B, there is no personal protection. In Strategy C, no animals were killed to clean the environment. The number of infected people rose to a certain extent before falling, reflecting the importance of personal protection and killing animals to clean the environment and control leptospirosis. Then, according to Figure 4b,e, in terms of the number of I_h , the disease was better controlled after the implementation of Strategies A, B, and C on the 6th day, the 8th day, and the 10th day, respectively,

($I_h \leq 5$). Compared with the three strategies, the first is the best for leptospirosis.

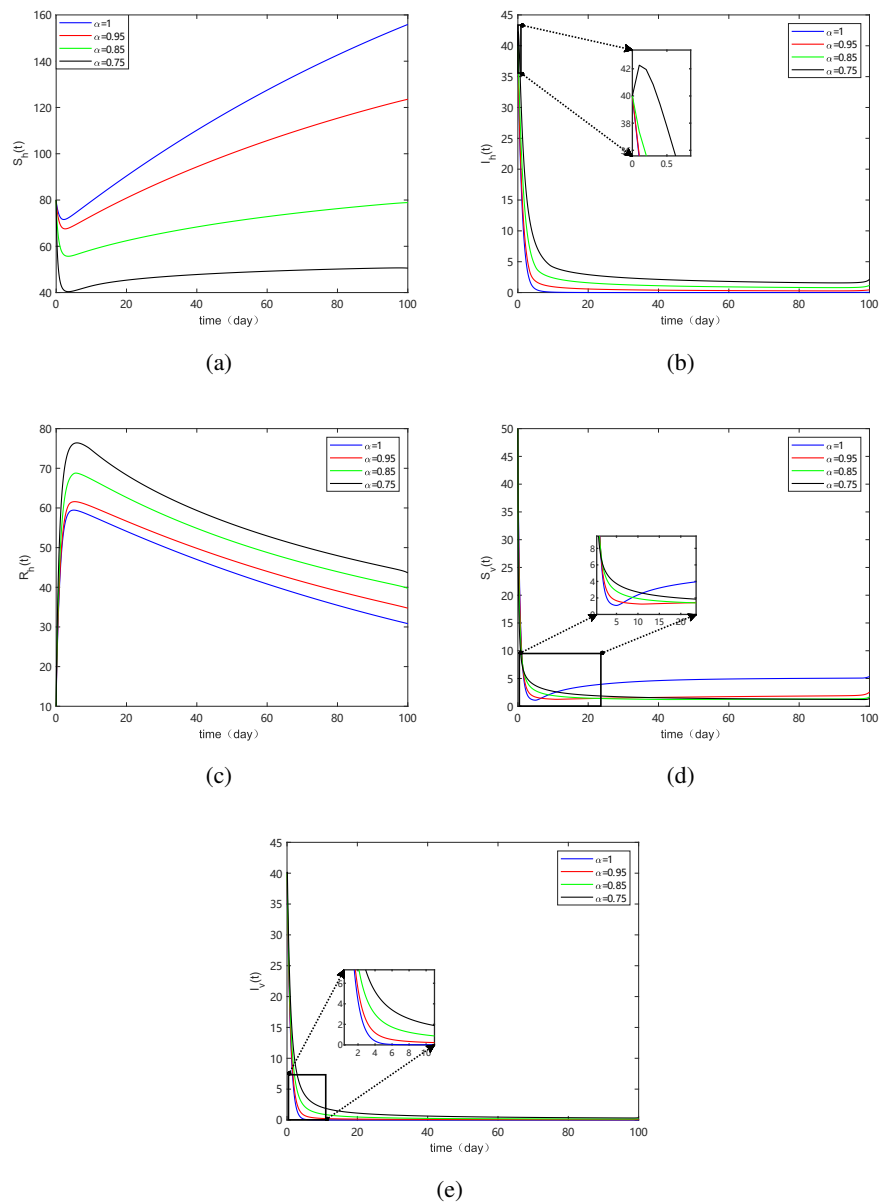


Figure 3. Under control Strategy A, when α takes different values, $S_h(t)$, $I_h(t)$, $R_h(t)$, $S_v(t)$, $I_v(t)$, to analyze the results numerically.

Figure 5 shows the change of the control function over time under three different strategies. Figure 5a shows that we need to ensure that infected people are hospitalized in a timely manner within the first 5 days. After 5 days, depending on the severity of the condition, home treatment or hospitalization can be chosen. After slaughtering infected animals, environmental cleaning must continue until day 42. The control measures can then be reduced according to the actual situation. In contrast, u_1 (personal protection) has always been close to zero because leptospirosis is rarely transmitted from patients to humans. Figure 5b shows that in the absence of personal protection, the measures we need to take are 93

days of hospitalization of the infected person, 98 days of killing animals to clean up the environment, and then reduce the intensity according to the actual situation. Figure 5c shows that an infected person requires immediate hospitalization when the disease is first detected. At the same time, vulnerable people should immediately take high-intensity personal protection until the pathogenic leptospirosis is eliminated. Due to the increased level of personal protection, the amount of I_h has decreased. So, u_2 rapidly drops to close to zero.

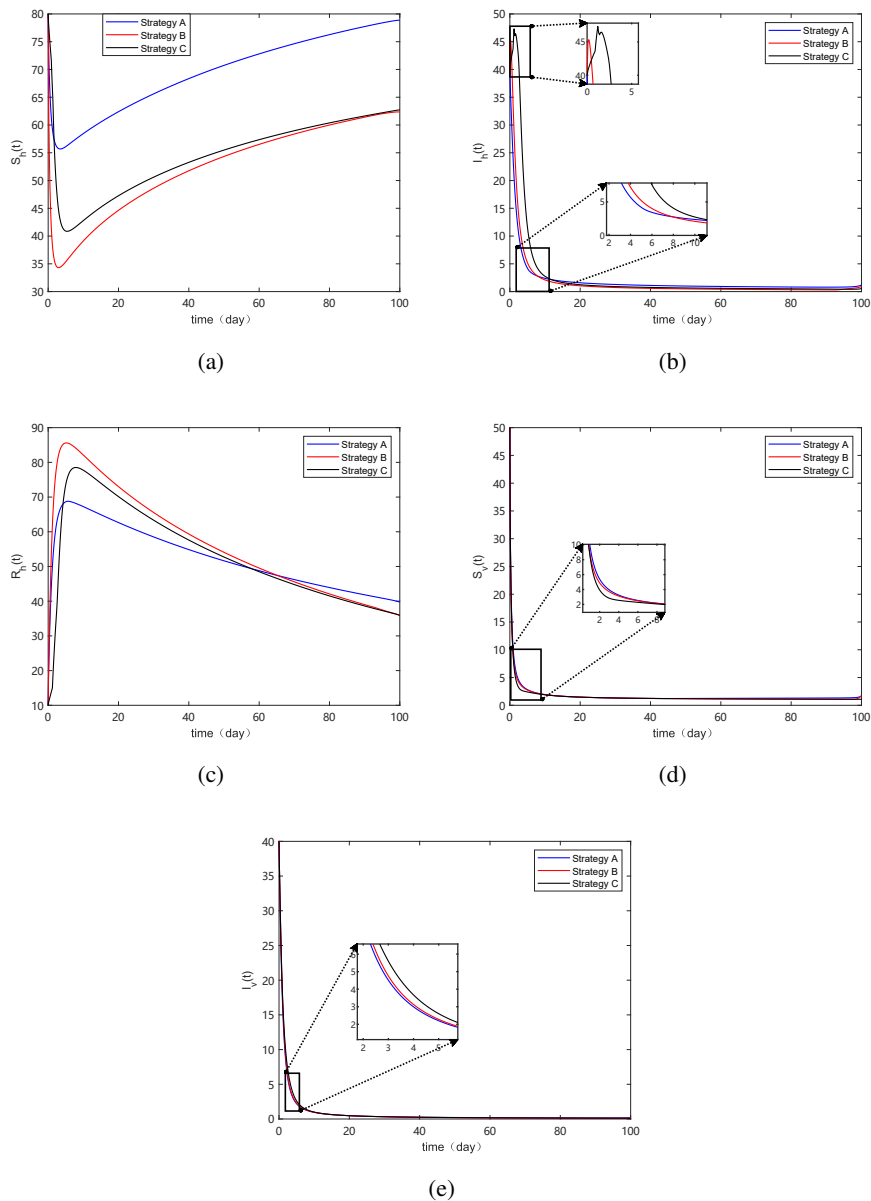


Figure 4. The control effect of Strategies A, B, and C. The fractional order $\alpha = 0.85$ for the above numerical analysis.

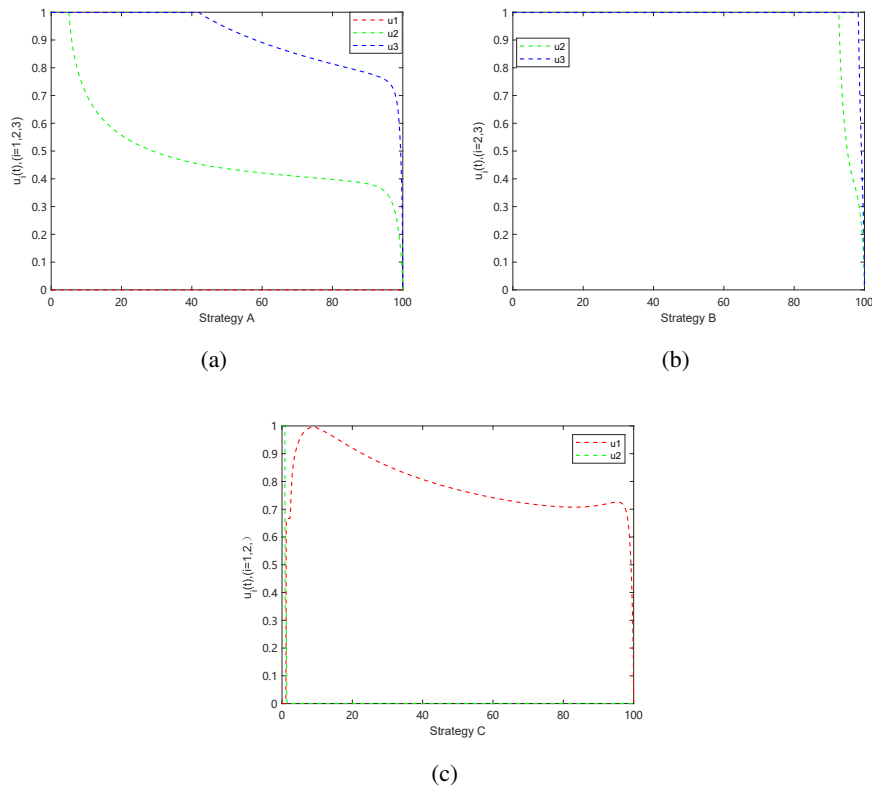


Figure 5. Numerical analysis results of optimal control functions u_1 , u_2 , and u_3 under three strategies, $\alpha = 0.85$.

7. Conclusions

This paper introduces the Caputo fractional differential equation to study the transmission dynamics model of leptospirosis. First, its dynamic characteristics are analyzed and proved, giving the model practical significance. It includes nonnegative, boundedness, and stability. The fde12 solver performs numerical simulations of different fractional order models. The simulation effect of different fractional orders on the transmission of leptospirosis is given. Second, from the perspective of fractional optimal control, combined with the existence mode and transmission characteristics of pathogenic bacteria of leptospirosis, three control variables u_1 (personal protection), u_2 (timely hospitalization of infected persons), and u_3 (disinfection of killed animals and environmental cleaning) were constructed to control the spread of leptospirosis. The forward-back scan algorithm is introduced to optimize the three control functions. Finally, according to the three control variables, three different control strategies were constructed to analyze the spread of leptospirosis. The results showed that strategy A could effectively control the spread of leptospirosis quickly. Without any control measures, the disease was controlled after 86 days. The disease can be effectively controlled after 6 days when the A strategy control measures are taken. It is worth noting that the first strategy is best for leptospirosis. The introduction of forward and backward scanning algorithms has certain optimization effects on the control function.

The above research has achieved certain results. However, there are still some shortcomings. Combined with the actual transmission mode and characteristics of leptospirosis, we found a close

relationship between the transmission of leptospirosis and the environment, so the next step can be to establish a model among humans, the environment, and animals for analysis. Also, the experiment lacks some actual data, but we can collect some actual data later to improve the model further.

Author contributions

Ling Zhang: Methodology, Software, Formal Analysis, and Writing - Original Draft. Xuwen Tan: Validation and Writing - Review & Editing. Jia Li: Project administration. Fan Yang: Conceptualization, Visualization and Writing - Review & Editing.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Acknowledgments

We are grateful to the three anonymous reviewers for their valuable comments and suggestions, which significantly enhanced the presentation of this work. This work was supported by the National Natural Science Foundation of China (Nos. 11361104, 12261104), the Youth Talent Program of Xingdian Talent Support Plan (XDYC-QNRC 2022-0514), the Yunnan Provincial Basic Research Program Project (No. 202301AT070016, No. 202401AT070036), the Yunnan Province International Joint Laboratory for Intelligent Integration and Application of Ethnic Multilingualism (202403AP140014).

Conflict of interest

The authors declare there is no conflict of interest.

References

1. Report of the second meeting of the leptospirosis burden epidemiology reference group, World Health Organization, 2011.
2. F. Costa, J. E. Hagan, J. Calcagno, M. Kane, P. Torgerson, M. S. Martinez-Silveira, Global morbidity and mortality of leptospirosis: A systematic review, *PLoS Negl. Trop. Dis.*, **9** (2015), e0003898. <https://doi.org/10.1371/journal.pntd.0003898>
3. K. V. Evangelista, J. Coburn, *Leptospira* as an emerging pathogen: A review of its biology, pathogenesis and host immune responses, *Future microbiol.*, **5** (2010), 1413–1425. <https://doi.org/10.2217/fmb.10.102>
4. K. B. Karpagam, B. Ganesh, Leptospirosis: A neglected tropical zoonotic infection of public health importance—an updated review, *Eur. J. Clin. Microbiol. Infect. Dis.*, **39** (2020), 835–846. <https://doi.org/10.1007/s10096-019-03797-4>
5. D. A. Haake, P. N. Levett, Leptospirosis in humans, *Leptospira and Leptospirosis*, Springer-Verlag GmbH, (2015), 65–97.

6. L. Biscornet, J. de Comarmond, J. Bibi, P. Mavingui, K. Dellagi, P. Tortosa, et al., An observational study of human leptospirosis in Seychelles, *Am. J. Trop. Med. Hyg.*, **103** (2020), 999–1008. <https://doi.org/10.4269/ajtmh.19-0228>
7. E. Bierque, R. Thibeaux, D. Girault, M. E. Soupé-Gilbert, C. Goarant, A systematic review of *Leptospira* in water and soil environments, *PLoS one*, **15** (2020), e0227055. <https://doi.org/10.1371/journal.pone.0227055>
8. G. López-Robles, F. N. Córdova-Robles, E. Sandoval-Petris, M. Montalvo-Corral, Leptospirosis at human-animal-environment interfaces in Latin-America: Drivers, prevention, and control measures, *Biotechnia*, **23** (2021), 89–100. <https://doi.org/10.18633/biotechnia.v23i3.1442>
9. M. H. A. A. Rahman, S. M. Hairon, R. A. Hamat, T. Z. M. T. Jamaluddin, M. N. Shafei, N. Idris, et al., Leptospirosis health intervention module effect on knowledge, attitude, belief, and practice among wet market workers in northeastern Malaysia: An intervention study, *Int. J. Environ. Res. Public Health*, **15** (2018), 1396. <https://doi.org/10.3390/ijerph15071396>
10. J. E. Sykes, D. A. Haake, C. D. Gamage, W. Z. Mills, J. E. Nally, A global one health perspective on leptospirosis in humans and animals, *J. Am. Vet. Med. Assoc.*, **260** (2022), 1589–1596. <https://doi.org/10.2460/javma.22.06.0258>
11. M. A. Mwachui, L. Crump, R. Hartskeerl, J. Zinsstag, J. Hattendorf, Environmental and behavioural determinants of leptospirosis transmission: A systematic review, *PLoS Negl. Trop. Dis.*, **9** (2015), e0003843. <https://doi.org/10.1371/journal.pntd.0003843>
12. Y. Guo, T. Li, Modeling the competitive transmission of the Omicron strain and Delta strain of COVID-19, *J. Math. Anal. Appl.*, **526** (2023), 127283. <https://doi.org/10.1016/j.jmaa.2023.127283>
13. Y. Guo, T. Li, Modeling and dynamic analysis of novel coronavirus pneumonia (COVID-19) in China, *J. Math. Anal. Appl.*, **68** (2022), 2641–2666. <https://doi.org/10.1007/s12190-021-01611-z>
14. Z. Zhang, Y. Wang, J. Zhang, Z. Ai, F. Liu, Novel stability results of multivariable fractional-order system with time delay, *Chaos, Solitons Fractals*, **157** (2022), 111943. <https://doi.org/10.1016/j.chaos.2022.111943>
15. N. H. Tuan, H. Mohammadi, S. Rezapour, A mathematical model for COVID-19 transmission by using the Caputo fractional derivative, *Chaos, Solitons Fractals*, **140** (2020), 110107. <https://doi.org/10.1016/j.chaos.2020.110107>
16. R. Almeida, A. M. C. B. da Cruz, N. Martins, M. T. T. Monteiro, An epidemiological MSEIR model described by the Caputo fractional derivative, *nt. J. Dyn. Control*, **7** (2019), 776–784. <https://doi.org/10.1007/s40435-018-0492-1>
17. K. Mukdasai, Z. Sabir, M. A. Z. Raja, R. Sadat, M. R. Ali, P. Singkibud, A numerical simulation of the fractional order Leptospirosis model using the supervise neural network, *Alexandria Eng. J.*, **61** (2022), 12431–12441. <https://doi.org/10.1016/j.aej.2022.06.013>
18. S. Ullah, M. A. Khan, Modeling and analysis of fractional leptospirosis model using Atangana–Baleanu derivative, *Fractional Derivatives with Mittag-Leffler Kernel: Trends and Applications in Science and Engineering*, Springer Cham, (2019), 49–67. https://doi.org/10.1007/978-3-030-11662-0_4

19. K. O. Okosun, M. Mukamuri, D. O. Makinde, Global stability analysis and control of leptospirosis, *Open Math.*, **14** (2016), 567–585. <https://doi.org/10.1515/math-2016-0053>
20. D. Baca-Carrasco, D. Olmos, I. Barradas, A mathematical model for human and animal leptospirosis, *J. Biol. Syst.*, **23** (2015), S55–S65. <https://doi.org/10.1142/S0218339015400057>
21. M. A. Khan, S. F. Saddiq, S. Islam, I. Khan, S. Shafie, Dynamic behavior of leptospirosis disease with saturated incidence rate, *Int. J. Appl. Comput. Math.*, **2** (2016), 435–452. <https://doi.org/10.1007/s40819-015-0102-2>
22. F. K. Alalhareth, U. Atta, A. H. Ali, A. Ahmad, M. H. Alharbi, Analysis of leptospirosis transmission dynamics with environmental effects and bifurcation using fractional-order derivative, *Alexandria Eng. J.*, **80** (2023), 372–382. <https://doi.org/10.1016/j.aej.2023.08.063>
23. H. D. Ngoma, P. R. Kiogora, I. Chepkwony, A fractional order model of leptospirosis transmission dynamics with environmental compartment, *Global J. Pure Appl. Math.*, **18** (2022), 81–110.
24. H. Qu, S. Saifullah, J. Khan, A. Khan, M. Ur Rahman, G. Zheng, Dynamics of leptospirosis disease in context of piecewise classical-global and classical-fractional operators, *Fractals*, **30** (2022), 2240216. <https://doi.org/10.1142/S0218348X22402162>
25. F. Scarselli, M. Gori, A. C. Tsoi, M. Hagenbuchner, G. Monfardini, The graph neural network model, *IEEE Trans. Neural Networks*, **20** (2008), 61–80. <https://doi.org/10.1109/TNN.2008.2005605>
26. T. Abdeljawad, On Riemann and Caputo fractional differences, *Comput. Math. Appl.*, **62** (2011), 1602–1611. <https://doi.org/10.1016/j.camwa.2011.03.036>
27. C. Gallicchio, A. Micheli, Graph echo state networks, in *The 2010 International Joint Conference on Neural Networks (IJCNN)*, IEEE, Barcelona, Spain, (2010), 1–8. <https://doi.org/10.1109/IJCNN.2010.5596796>
28. Z. M. Odibat, N. T. Shawagfeh, Generalized Taylor’s formula, *Appl. Math. Comput.*, **186** (2007), 286–293. <https://doi.org/10.1016/j.amc.2006.07.102>
29. W. Lin, Global existence theory and chaos control of fractional differential equations, *J. Math. Anal. Appl.*, **332** (2007), 709–726. <https://doi.org/10.1016/j.jmaa.2006.10.040>
30. L. K. Hansen, P. Salamon, Neural network ensembles, *IEEE Trans. Pattern Anal. Mach. Intell.*, **12** (1990), 993–1001. <https://doi.org/10.1109/34.58871>
31. R. E. Schapire, The strength of weak learnability, *Mach. Learn.*, **5** (1990), 197–227. <https://doi.org/10.1007/BF00116037>
32. Y. Freund, R. E. Schapire, A decision-theoretic generalization of on-Line learning and an application to boosting, *J. Comput. Syst. Sci.*, **55** (1997), 119–139. <https://doi.org/10.1006/jcss.1997.1504>
33. J. H. Friedman, Greedy function approximation: A gradient boosting machine, *Ann. Stat.*, **29** (2001), 1189–1232. <https://doi.org/10.1214/aos/1013203451>
34. L. Breiman, Bagging predictors, *Mach. Learn.*, **24** (1996), 123–140. <https://doi.org/10.1023/A:1018054314350>
35. A. Boukhouima, K. Hattaf, E. M. Lotfi, M. Mahrouf, D. F. M. Torres, N. Yousfi, Lyapunov functions for fractional-order systems in biology: Methods and applications, *Chaos, Solitons Fractals*, **140** (2020), 110224. <https://doi.org/10.1016/j.chaos.2020.110224>

36. L. Breiman, Random forests, *Mach. Learn.*, **45** (2001), 5–32. <https://doi.org/10.1023/A:1010933404324>
37. P. Geurts, D. Ernst, L. Wehenkel, Extremely randomized trees, *Mach. Learn.*, **63** (2006), 3–42. <https://doi.org/10.1007/s10994-006-6226-1>
38. C. Cortes, V. Vapnik, Support-vector networks, *Mach. learn.*, **20** (1995), 273–297. <https://doi.org/10.1023/A:1022627411411>
39. L. Wang, T. Wang, X. Hu, Logistic regression region weighting for weakly supervised object localization, *IEEE Access*, **7** (2019), 118411–118421. <https://doi.org/10.1109/ACCESS.2019.2935011>
40. K. Diethelm, N. J. Ford, A. D. Freed, Detailed error analysis for a fractional Adams method, *Numerical Algorithms*, **36** (2004), 31–52. <https://doi.org/10.1023/B:NUMA.0000027736.85078.be>
41. R. Garrappa, On linear stability of predictor-corrector algorithms for fractional differential equations, *Int. J. Comput. Math.*, **87** (2010), 2281–2290. <https://doi.org/10.1080/00207160802624331>
42. M. McAsey, L. Mou, W. Han, Convergence of the forward-backward sweep method in optimal control, *Comput. Optim. Appl.*, **53** (2012), 207–226. <https://doi.org/10.1007/s10589-011-9454-7>
43. Y. Guo, T. Li, Dynamics and optimal control of an online game addiction model with considering family education, *AIMS Math.*, **7** (2022), 3745–3770. <https://doi.org/10.3934/math.2022208>
44. H. D. Ngoma, P. R. Kiogora, I. Chepkwony, A fractional order model of leptospirosis transmission dynamics with environmental compartment, *Global J. Pure Appl. Math.*, **18** (2022), 81–110.
45. M. El-Shahed, Fractional order model for the spread of leptospirosis, *Int. J. Math. Anal.*, **8** (2014), 2651–2667. <https://doi.org/10.12988/ijma.2014.410312>



AIMS Press

©2024 the Author(s), licensee AIMS Press. This is an open access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by/4.0>)