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Research article

Predicting the trend of leptospirosis in China via a stochastic model with vector and environmental transmission

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Abstract: A stochastic model of leptospirosis with vector and environmental transmission is established in this paper. By mathematical analysis of the model, the threshold for eliminating the disease is obtained. The partial rank correlation coefficient was used to analyze the parameters that have a greater impact on disease elimination, and a sensitivity analysis was conducted on the parameters through numerical simulation. Further, combined with the data of leptospirosis case reports in China from 2003 to 2021, two parameter estimation methods, Least Squares method (LSM) and Markov Chain Monte Carlo-Metropolis Hastings method (MCMC-MH), are applied to estimate the important parameters of the model and the future trend of leptospirosis in China are predicted.

Keywords: stochastic epidemic model; leptospirosis; disease elimination; parameter estimation; disease prediction

1. Introduction

Leptospirosis is an acute systemic infectious disease caused by various pathogenic leptospira, which belongs to natural foci disease. It is epidemic almost all over the world, especially severe in Southeast Asia. Most provinces, cities, and autonomous regions in China have the existence and epidemic of this disease. Rodents and pigs are the two major sources of infection, while other livestock such as cattle, pigs, and pets like cats, dogs, and mice may also transmit leptospirosis. Typically, pathogenic leptospira can survive longer in a warm and humid environment. People may contract the disease through ingestion of contaminated food or water, or when the bacteria enter the body through scratches on the skin or mucous membranes [1].

The application of mathematical models in leptospirosis research has also become increasingly widespread. Through numerical simulations and data analysis, we can delve deeper into the transmission mechanisms and dynamic characteristics of the disease, providing more precise and effective means for disease prevention and control. Regarding the research on mathematical models of lep-

tospirosis, please refer to the literature [2–6]. These models analyze the factors that influence the transmission dynamics of leptospirosis, pointing out that disease transmission is not only related to the interaction between rodents and humans [4], but also to their contact with free bacteria in the environment [6]. They also demonstrate that adopting appropriate intervention mechanisms, such as reducing the transmission rate, increasing the recovery rate, reducing rodent populations, and reducing bacterial contamination in water sources, can greatly assist in reducing the spread of the disease in the population.

In the real world, infectious disease models are inevitably affected by environmental noise, and deterministic models alone cannot accurately reflect the dynamic behavior of the system when describing disease transmission processes. In recent years, most scholars have explored stochastic infectious disease models that consider environmental perturbations [7–12]. The research results indicate that random perturbations have a certain impact on the spread of diseases.

Therefore, it is highly necessary to further establish and study leptospirosis models that consider vector-environment interactions and random disturbances.

To establish the model, we make the following assumptions.

(i) Susceptible individuals who come into contact with infected vectors or free bacteria in the environment can become infected individuals, and susceptible vectors that come into contact with infected individuals or free bacteria in the environment can also become infected vectors.

(ii) Infected individuals and vectors both release free bacteria into the environment.

(iii) The host population $S_h(t), I_h(t), S_h(t)$, vector population $S_v(t), I_v(t)$, and the concentration of bacteria in the environment are all influenced by Gaussian white noise.

(iv) The recruitment rate Λ and the birth rate Π of the vectors are constants. Every parameter within the system is a nonnegative real number.

Base on the above assumptions, we establish and study a stochastic model of leptospirosis with host-vector-environment interactions:

$$\begin{cases} \mathrm{d}S_h(t) = & [\Lambda - \mu_h S_h - \dfrac{\beta_1 S_h I_v}{N_h} - \dfrac{\beta_3 S_h B}{K+B} + \lambda_h R_h]\mathrm{d}t + \sigma_1 S_h \mathrm{d}B_1(t), \\ \mathrm{d}I_h(t) = & [\dfrac{\beta_1 S_h I_v}{N_h} + \dfrac{\beta_3 S_h B}{K+B} - \mu_h I_h - \delta_h I_h - \gamma_h I_h]\mathrm{d}t + \sigma_2 I_h \mathrm{d}B_2(t), \\ \mathrm{d}R_h(t) = & [\gamma_h I_h - \lambda_h R_h - \mu_h R_h]\mathrm{d}t + \sigma_3 R_h \mathrm{d}B_3(t), \\ \mathrm{d}S_v(t) = & [\Pi - \dfrac{\beta_2 I_h S_v}{N_h} - \dfrac{\beta_4 S_v B}{K+B} - \mu_v S_v]\mathrm{d}t + \sigma_4 S_v \mathrm{d}B_4(t), \\ \mathrm{d}I_v(t) = & [\dfrac{\beta_2 I_h S_v}{N_h} + \dfrac{\beta_4 S_v B}{K+B} - \mu_v I_v]\mathrm{d}t + \sigma_5 I_v \mathrm{d}B_5(t), \\ \mathrm{d}B(t) = & [\alpha_1 I_h + \alpha_2 I_v - kB]\mathrm{d}t + \sigma_6 B \mathrm{d}B_6(t), \end{cases} \tag{1.1}$$

where the host population, which represents the human population, is divided into three categories at time t: susceptible individuals $S_h(t)$, infected individuals $I_h(t)$, and recovered individuals $R_h(t)$. The vector population is divided into susceptible vectors $S_v(t)$ and infected vectors $I_v(t)$ at time t. Additionally, $B(t)$ represents the free-floating bacterial population in the environment. The meanings of the parameters are as follows. β_1 and β_2 represent the infection rates of diseased vectors transmitting the disease to humans and of infected humans transmitting the disease to vectors, respectively. β_3 and

β_4 represent the rates at which susceptible humans and susceptible vectors become infected through contact with bacteria in the environment. μ_h and μ_v are natural mortality rate for the human population and the vector population, and γ_h represents the disease-induced mortality rate among humans. δ_h represents the recovery rate for infected humans, while λ_h represents the rate at which recovered humans revert back to the susceptible state. α_1 and α_2 represent the rates at which infected humans and infected vectors release bacteria into the environment, respectively. K serves as a half-saturation infection parameter, and k is the decay rate of bacteria in the environment. $B_i(t)$ $(i = 1, 2, 3, 4, 5, 6)$ are standard Brownian motions. Parameters σ_i $(i = 1, 2, \cdots, 6)$ are the intensities of noise, representing variability and stochastic effects: σ_1 represents the variability in the susceptible individuals $S_h(t)$, which arise from fluctuating contact rates or changes in population behavior that affect exposure to the virus environment and infected vectors; σ_2 reflects the random fluctuations in the number of the infected population $I_h(t)$ due to variations in the disease's infectiousness, or response to treatment; σ_3 represents stochastic factors affecting the recovered population $R_h(t)$, such as loss of immunity or the impact of interventions; σ_4 represents the variability in the susceptible vectors $S_v(t)$, which arise from fluctuating contact rates or changes in population behavior that affect exposure to the Leptospira virus environment and infected individuals; σ_5 reflects the random fluctuations in the number of the infected vectors $I_v(t)$ due to variations in the disease¡¯s infectiousness; σ_6 represents the random variation intensity of Leptospira virus $B(t)$ released into the environment by infected humans or disease vectors.

We assume the initial conditions are

$$S_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0, B(0) \geq 0. \tag{1.2}$$

The aim of this paper is to build a stochastic model of leptospirosis that incorporates both vector-borne and environmental transmission to more comprehensively describe the disease's transmission characteristics. Furthermore, by combining this model with actual reported data on leptospirosis in China in recent years, we aim to estimate important parameters of the model using statistical methods and predict the future trends of leptospirosis in China.

2. The long-term behavior of the solution

To demonstrate that our proposed model is meaningful, we prove that there exists a unique global positive solution of the system (1.1).

Theorem 2.1. *For any initial value* $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0)) \in \mathbb{R}^6_+$*, the system (1.1) has a unique positive solution* $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), B(t))$*, and the solution will remain in* $\mathbb{R}^6_+$ *with probability 1, i.e.,* $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), B(t)) \in \mathbb{R}^6_+$ *for all* $t > 0$ *almost surly (a.s.).*

Proof. Obviously, the system (1.1) has locally Lipschitz continuous coefficients, for any initial value $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0)) \in \mathbb{R}^6_+$, and the system (1.1) exists a unique maximal local solution $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), B(t))$, $t \in [0, \tau_e)$, where τ_e is the explosion time. To verify that this solution of the system (1.1) is global, we just have to prove that $\tau_e = \infty$ a.s. For this, assume $k_0 \geq 1$ is large enough such that $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0))$ all fall within the interval $[1/k_0, k_0]$. For

each integer $k \geq k_0$, define the stopping time as:

$$\tau_k = \inf\Big\{t \in [0, \tau_e) : S_h(t) \notin (\frac{1}{k}, k) \text{ or } I_h(t) \notin (\frac{1}{k}, k) \text{ or } R_h(t) \notin (\frac{1}{k}, k)$$

$$\text{or } S_h(t) \notin (\frac{1}{k}, k) \text{ or } I_v(t) \notin (\frac{1}{k}, k) \text{ or} B(t) \notin (\frac{1}{k}, k)\Big\},$$

where $\inf \varnothing = \infty$ ($\varnothing$ denotes the empty set). Clearly, when $k \to \infty$, τ_k are increasing. Let $\tau_\infty = \lim\limits_{k\to\infty} \tau_k$, then $\tau_\infty \leq \tau_e$ a.s. If $\tau_\infty = \infty$ a.s. holds, then $\tau_e = \infty$ a.s., which means that $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), B(t)) \in \mathbb{R}^6_+$ a.s. for $t \geq 0$. Therefore, it suffices to prove that $\tau_\infty = \infty$ a.s.

Next, we assume that there exist constants $T > 0$ and $\varepsilon \in (0, 1)$, such that $P\{\tau_\infty \leq T\} > \varepsilon$, then, there exists an integer $k_1 \geq k_0$, such that for any $k \geq k_1$,

$$P\{\tau_k \leq T\} \geq \varepsilon. \tag{2.1}$$

Define the function $Q : \mathbb{R}^6_+ \to \mathbb{R}_+$ as follows:

$$Q(S_h, I_h, R_h, S_v, I_v, B) = (S_h - a_1 - a_1 \ln \frac{S_h}{a_1}) + (I_h - 1 - \ln I_h) + (R_h - 1 - \ln R_h)$$

$$+ (S_v - b_1 - b_1 \ln \frac{S_v}{b_1}) + (I_v - 1 - \ln I_v) + \ln(1 + \frac{1}{B}),$$

where a_1, b_1 are positive constants to be determined later. Obviously, the function $u - 1 - \ln u$ is non-negative for all $u > 0$.

Applying Itô's formula, we obtain

$$\mathrm{d}Q = LQ\mathrm{d}t + \sigma_1(S_h - a_1)\mathrm{d}B_1(t) + \sigma_2(I_h - 1)\mathrm{d}B_2(t) + \sigma_3(R_h - 1)\mathrm{d}B_3(t)$$

$$+ \sigma_4(S_v - b_1)\mathrm{d}B_4(t) + \sigma_5(I_v - 1)\mathrm{d}B_5(t) - \frac{\sigma_6}{1 + B}\mathrm{d}B_6(t),$$

where

$$LQ = \Lambda - \mu_h(S_h + I_h + R_h) - \delta_h I_h + \Pi - \mu_v(S_v + I_v) - \frac{\alpha_1 I_h}{B(1 + B)} - \frac{\alpha_2 I_v}{B(1 + B)}$$

$$+ \frac{k}{1 + B} - \frac{a_1\Lambda}{S_h} + a_1\mu_h + \frac{a_1\beta_1 I_v}{N_h} + \frac{a_1\beta_3 B}{K + B} - \frac{a_1\lambda_h R_h}{S_h} - \frac{\beta_1 S_h I_v}{N_h I_h} - \frac{\beta_3 S_h B}{(K + B)I_h}$$

$$+ \mu_h + \delta_h + \gamma_h - \frac{\gamma_h I_h}{R_h} + \lambda_h + \mu_h - \frac{b_1\Pi}{S_v} + \frac{b_1\beta_2 I_h}{N_h} + \frac{b_1\beta_4 B}{K + B} + b_1\mu_v - \frac{\beta_2 S_v I_h}{N_h I_v}$$

$$- \frac{\beta_4 S_v B}{(K + B)I_v} + \mu_v + \frac{1}{2}a_1\sigma_1^2 + \frac{1}{2}\sigma_2^2 + \frac{1}{2}\sigma_3^2 + \frac{1}{2}b_1\sigma_4^2 + \frac{1}{2}\sigma_5^2 + \frac{1}{2}\frac{1 + 2B}{(1 + B)^2}\sigma_6^2$$

$$\leq \Lambda + \Pi + k + a_1\mu_h + (\frac{a_1\beta_1}{M_1} - \mu_v)I_v + a_1\beta_3 + \mu_h + \delta_h + \gamma_h + \lambda_h + \mu_h + (\frac{b_1\beta_2}{M_1}$$

$$- \mu_h)I_h + b_1\beta_4 + b_1\mu_v + \mu_v + \frac{1}{2}a_1\sigma_1^2 + \frac{1}{2}\sigma_2^2 + \frac{1}{2}\sigma_3^2 + \frac{1}{2}b_1\sigma_4^2 + \frac{1}{2}\sigma_5^2 + \frac{1}{2}\sigma_6^2.$$

Choose $a_1 = \dfrac{\mu_v M_1}{\beta_1}, b_1 = \dfrac{\mu_h M_1}{\beta_2}$, such that $\dfrac{a_1\beta_1}{M_1} - \mu_v = 0, \dfrac{b_1\beta_2}{M_1} - \mu_h = 0$, and

$$\begin{aligned} LQ \le{}& \Lambda + \Pi + k + a_1\mu_h + a_1\beta_3 + \mu_h + \delta_h + \gamma_h + \lambda_h + \mu_h + b_1\beta_4 + b_1\mu_v + \mu_v \\ &+ \frac{1}{2}a_1\sigma_1^2 + \frac{1}{2}\sigma_2^2 + \frac{1}{2}\sigma_3^2 + \frac{1}{2}b_1\sigma_4^2 + \frac{1}{2}\sigma_5^2 + \frac{1}{2}\sigma_6^2 := K, \end{aligned}$$

where $K > 0$ is a constant. The remainder of the proof follows the similar approach given in [13].

Now, the sufficient conditions for the elimination of I_h, I_v are presented. Denote $\langle f \rangle = \dfrac{1}{t}\displaystyle\int_0^t f(s)ds$, and the parameter as follows:

$$\mathcal{R}_m = \frac{(\beta_1 + \beta_2 + \beta_4)\mu_h\Pi + \beta_3\mu_v\Lambda}{\mu_h\mu_v(\Lambda + \Pi) + (\delta_h + \gamma_h)\mu_v\Lambda}.$$

To facilitate the proof of the theorem, we first give a related lemma.

Lemma 2.1. *[14–16] For any initial value $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0)) \in \mathbb{R}^6_+$, the solution $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), B(t)) \in \mathbb{R}^6_+$ of model (1.1) possesses the following properties:*

$$\lim_{t\to\infty} \frac{\int_0^t S_h(s)\mathrm{d}B_1(s)}{t} = 0, \ \lim_{t\to\infty} \frac{\int_0^t I_h(s)\mathrm{d}B_2(s)}{t} = 0, \ \lim_{t\to\infty} \frac{\int_0^t R_h(s)\mathrm{d}B_3(s)}{t} = 0,$$

$$\lim_{t\to\infty} \frac{\int_0^t S_v(s)\mathrm{d}B_4(s)}{t} = 0, \ \lim_{t\to\infty} \frac{\int_0^t I_v(s)\mathrm{d}B_5(s)}{t} = 0, \ \lim_{t\to\infty} \frac{\int_0^t B(s)\mathrm{d}B_6(s)}{t} = 0 \ a.s.$$

Proof of Lemma 2.1 can be similarly obtained by following the proof of Lemma 2.2 in reference [14]. The details are omitted here.

Theorem 2.2. *Assume $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), B(t)) \in \mathbb{R}^6_+$ is the solution of model (1.1) that satisfies the initial condition $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0)) \in \mathbb{R}^6_+$. If $\mathcal{R}_m < 1$, then $(I_h(t), I_v(t), B(t))$ converges to $(0, 0, 0)$ exponentially with probability one (a.s.), indicating the elimination of the disease, and furthermore,*

$$\lim_{t\to\infty} S_h(t) = \frac{\Lambda}{\mu_h}, \ \lim_{t\to\infty} S_v(t) = \frac{\Pi}{\mu_v}, \ \lim_{t\to\infty} R_h(t) = 0 \ a.s.$$

Proof. Let $P(t) = I_h(t) + I_v(t)$. Applying Itô's formula, we have

$$\begin{aligned} \mathrm{d}P(t) =& \Big[\frac{\beta_1 S_h}{N_h} I_v + \frac{\beta_3 S_h B}{K + B} - \mu_h I_h - \delta_h I_h - \gamma_h I_h + \frac{\beta_2 I_h}{N_h} S_v + \frac{\beta_4 S_v B}{K + B} \\ &- \mu_v I_v\Big]\mathrm{d}t + \sigma_2 I_h \mathrm{d}B_2(t) + +\sigma_5 I_v \mathrm{d}B_5(t). \end{aligned} \tag{2.2}$$

Integrating both sides of (2.2) from 0 to t and dividing by t, we obtain

$$\begin{aligned}\frac{P(t)}{t} =&\frac{P(0)}{t} + \beta_1\langle\frac{S_h}{N_h}I_v\rangle + \beta_3\langle\frac{S_hB}{K+B}\rangle - (\mu_h+\delta_h+\gamma_h)\langle I_h\rangle + \beta_2\langle\frac{I_h}{N_h}S_v\rangle\\ &+\beta_4\langle\frac{S_vB}{K+B}\rangle - \mu_v\langle I_v\rangle + \frac{1}{t}\int_0^t \sigma_2 I_h(s)\mathrm{d}B_2(s) + \frac{1}{t}\int_0^t \sigma_5 I_v(s)\mathrm{d}B_5(s)\\ \leq&\frac{P(0)}{t} + \beta_1\langle I_v\rangle + \beta_3\langle S_h\rangle - (\mu_h+\delta_h+\gamma_h)\langle I_h\rangle + \beta_2\langle S_v\rangle + \beta_4\langle S_v\rangle -\\ &\mu_v\langle I_v\rangle + \frac{1}{t}\int_0^t \sigma_2 I_h(s)\mathrm{d}B_2(s) + \frac{1}{t}\int_0^t \sigma_5 I_v(s)\mathrm{d}B_5(s).\end{aligned} \tag{2.3}$$

Notice

$$\begin{aligned}&\mathrm{d}(S_h(t)+I_h(t)+R_h(t))\\ \leq&[\Lambda-\mu(S_h+I_h+R_h)]\mathrm{d}t + \sigma_1 S_h(t)\mathrm{d}B_1(t) + \sigma_2 I_h(t)\mathrm{d}B_2(t) + \sigma_3 R_h(t)\mathrm{d}B_3(t)\end{aligned} \tag{2.4}$$

and

$$\mathrm{d}(S_v(t)+I_v(t)) = [\Pi-\mu_v(S_v+I_v)]\mathrm{d}t + \sigma_4 S_v(t)\mathrm{d}B_4(t) + \sigma_5 I_v(t)\mathrm{d}B_5(t). \tag{2.5}$$

Integrating both sides of (2.4) and (2.5) from 0 to t and dividing by t, then, taking the upper limit, we obtain

$$\limsup_{t\to\infty}\langle S_h(t)+I_h(t)+R_h(t)\rangle \leq \frac{\Lambda}{\mu_h}\ a.s.$$

$$\limsup_{t\to\infty}\langle S_v(t)+I_v(t)\rangle = \frac{\Pi}{\mu_v}\ a.s.$$

Thus

$$\limsup_{t\to\infty}\langle S_h(t)\rangle \leq \frac{\Lambda}{\mu_h},\ \limsup_{t\to\infty}\langle I_h(t)\rangle \leq \frac{\Lambda}{\mu_h},\ \limsup_{t\to\infty}\langle R_h(t)\rangle \leq \frac{\Lambda}{\mu_h}\ a.s.$$

$$\limsup_{t\to\infty}\langle S_v(t)\rangle \leq \frac{\Pi}{\mu_v},\ \limsup_{t\to\infty}\langle I_v(t)\rangle \leq \frac{\Pi}{\mu_v}\ a.s.$$

Taking the upper limit of both sides of (2.3), and according to Lemma 2.1, we can obtain the desired result

$$\begin{aligned}\limsup_{t\to\infty}\frac{P(t)}{t} &\leq \beta_1\cdot\frac{\Pi}{\mu_v} + \beta_3\cdot\frac{\Lambda}{\mu_h} - (\mu_h+\delta_h+\gamma_h)\cdot\frac{\Lambda}{\mu_h} + \beta_2\cdot\frac{\Pi}{\mu_v} + \beta_4\cdot\frac{\Pi}{\mu_v} - \mu_v\cdot\frac{\Pi}{\mu_v}\\ &= \frac{\mu_h(\Lambda+\Pi)+(\delta_h+\gamma_h)\Lambda}{\mu_h}(\mathcal{R}_m-1) < 0.\end{aligned}$$

Then

$$\lim_{t\to\infty} P(t) = 0.$$

Hence

$$\lim_{t\to\infty} I_h(t) = 0,\ \lim_{t\to\infty} I_v(t) = 0.$$

For the sixth equation in (1.1), by integrating both sides from 0 to t, dividing by t, and then taking the upper limit, we can derive that $\lim_{t\to\infty} B(t) = 0$.

Similarly, applying the same method to the third equation in (1.1), we can obtain $\lim_{t\to\infty} R_h(t) = 0$. Since

$$\begin{aligned} \mathrm{d}(S_h(t) + I_h(t)) =&[\Lambda - \mu_h S_h - \mu_h I_h - \delta_h I_h - \gamma_h I_h + \lambda_h R_h]\mathrm{d}t \\ &+ \sigma_1 S_h(t)\mathrm{d}B_1(t) + \sigma_2 I_h(t)\mathrm{d}B_2(t), \end{aligned}$$

based on the conclusions obtained above, we can derive that $\lim_{t\to\infty} S_h(t) = \frac{\Lambda}{\mu_h}$. Similarly, we can obtain that $\lim_{t\to\infty} S_v(t) = \frac{\Pi}{\mu_v}$.

To better analyze the impact of different parameters on the spread of infectious diseases on the surface, we will proceed with a further parameter sensitivity analysis. We conduct 1000 samplings of the parameters using the Latin Hypercube Sampling (LHS) method [17]. By calculating the Partial Rank Correlation Coefficient (PRCC), we will be able to screen out the parameters that have a significant impact on the population size. This will help us identify more accurate measures to control the epidemic.

Observing Figure 1, it is evident that the parameters with significant impacts on disease transmission are $\beta_3, \delta_h, \gamma_h, \mu_v$. Here, β_3 is positively correlated with $\mathcal{R}_m$, while $\delta_h, \gamma_h, \mu_v$ are negatively correlated with $\mathcal{R}_m$. In other words, the smaller the contact rate of humans with free bacteria in the environment, the higher the human mortality rate due to the disease and the natural mortality rate of the vector population; and the faster the recovery rate from the disease, the smaller the basic reproduction number will be, making it easier to eliminate the disease. In fact, as the contact rate of humans with free bacteria in the environment declines, so does the likelihood of contracting the virus. Similarly, when the mortality rate stemming from the illness is high, infected individuals may perish during the infection period, thereby diminishing their capacity to spread the disease to others, resulting in a lower average transmission rate per infected individual. Furthermore, a high natural mortality rate among vectors lessens their chances of transmitting the disease to humans and curtails the release of virus particles into the environment. Lastly, an increase in the recovery rate of infected individuals reduces their chances of transmitting the disease to vectors. All these scenarios contribute significantly to a decrease in the R_m value.

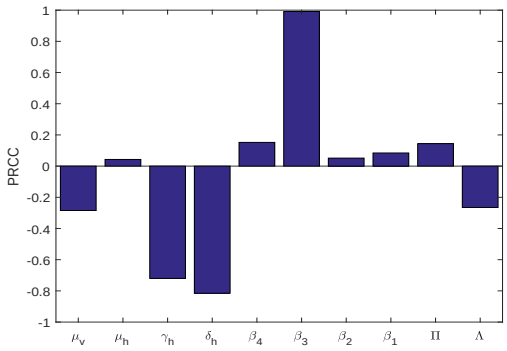
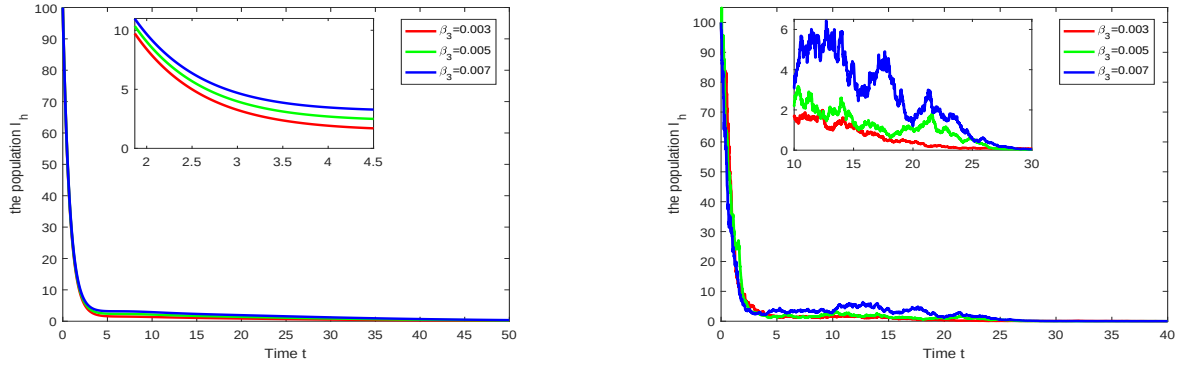


Figure 1. The correlation PRCC index of each parameter on $\mathcal{R}_m$.

Next, we perform numerical simulations on the system (1.1) by using the high-order *Milstein* method mentioned in [18, 19], which is based on the concept of Itô's formula and stochastic Taylor

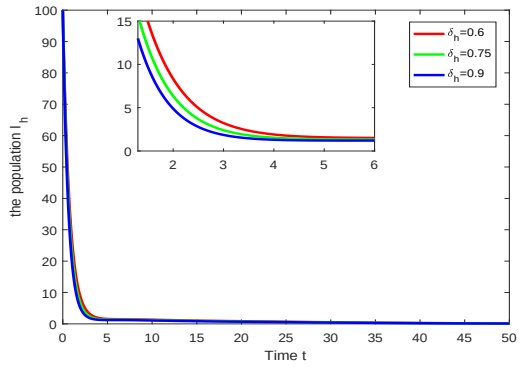
expansion. The *Milstein* method improves the accuracy of the estimates by introducing higher-order infinitesimals. Compared to the *Euler-Maruyama* method, the Milstein method is more precise. However, the *Milstein* method requires the stochastic process to be twice differentiable, which can make its implementation more complex. It is primarily suitable for stochastic differential equations with continuous sample paths. For stochastic differential equations with discontinuous sample paths or jump processes, other types of numerical methods may be required.

Assuming an initial condition of $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0)) = (400, 100, 150, 500, 120, 1000)$, the specific parameter values are as follows: $\Lambda = 35\ day^{-1}$, $\Pi = 30\ day^{-1}$, $\beta_1 = 0.004\ day^{-1}$, $\beta_2 = 0.001\ day^{-1}$, $\beta_3 = 0.003\ day^{-1}$, $\beta_4 = 0.002\ day^{-1}$, $\lambda_h = 0.1\ day^{-1}$, $\delta_h = 0.6\ day^{-1}$, $\mu_h = 0.01\ day^{-1}$, $\mu_v = 0.1\ day^{-1}$, $K = 10\ cells \cdot ml^{-1}$, $k = 0.5\ day^{-1}$, $\alpha_1 = 0.08\ cells \cdot ml^{-1} \cdot day^{-1}$, $\alpha_2 = 0.09\ cells \cdot ml^{-1} \cdot day^{-1}$ and $\gamma_h = 0.7\ day^{-1}$.

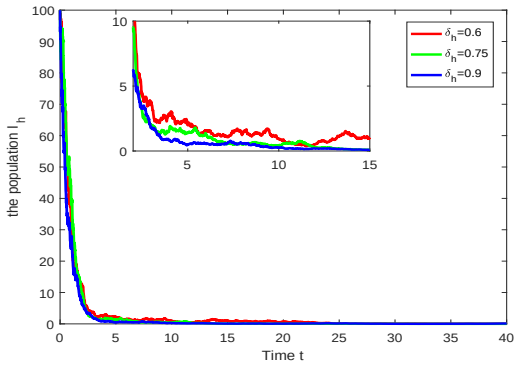


(*a*) $\beta_3 = 0.003, 0.005, 0.007$

(*b*) $\beta_3 = 0.003, 0.005, 0.007$



(*c*) $\delta_h = 0.6, 0.75, 0.9$

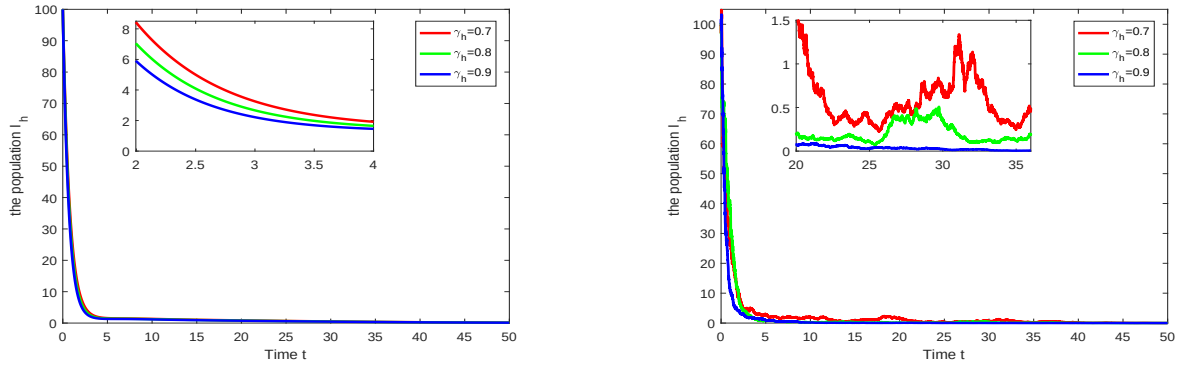


(*d*) $\delta_h = 0.6, 0.75, 0.9$

Figure 2. Numerical simulations of deterministic and stochastic systems I_h under different β_3 and δ_h. (*a*) and (*c*) represent the corresponding deterministic model of (1.1), while (*b*) and (*d*) represent the stochastic model (1.1).

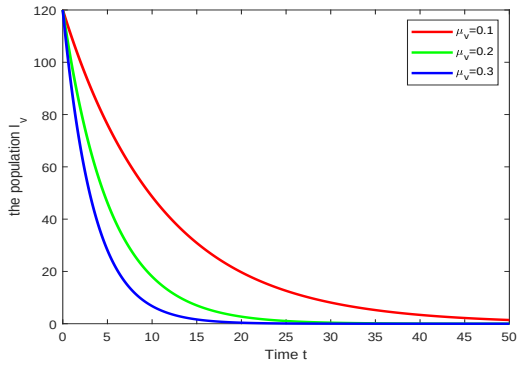
Figures 2 and 3 demonstrate the specific time-varying situation of the number of infected individuals or infected vectors when these four parameters $\beta_3, \delta_h, \gamma_h, \mu_v$ change, while other parameters remain unchanged, respectively. From these two figures, it can be observed that despite changes in the parameters, both the infected population and the infected vectors ultimately go extinct, but the time of extinction differs. Specifically, as β_3 decreases, the extinction time of I_h shortens. Similarly, when δ_h

and γ_h increase, the extinction time of I_h decreases. Additionally, as μ_v increases, the extinction time of I_v also shortens.

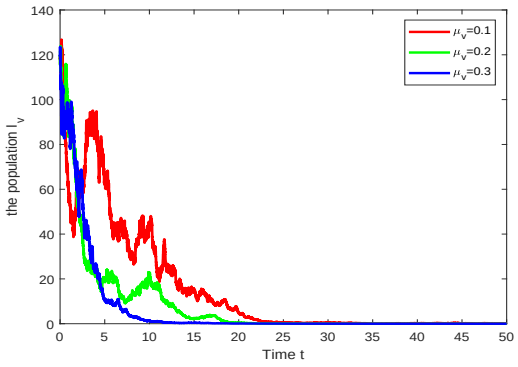


(a) $\gamma_h = 0.7, 0.8, 0.9$

(b) $\gamma_h = 0.7, 0.8, 0.9$



(c) $\mu_v = 0.1, 0.2, 0.3$



(d) $\mu_v = 0.1, 0.2, 0.3$

Figure 3. Numerical simulations of deterministic and stochastic systems I_h and I_v under different γ_h and μ_v. (a) and (c) represent the corresponding deterministic model of (1.1), while (b) and (d) represent the stochastic model (1.1).

3. Predicting the trend of leptospirosis in China

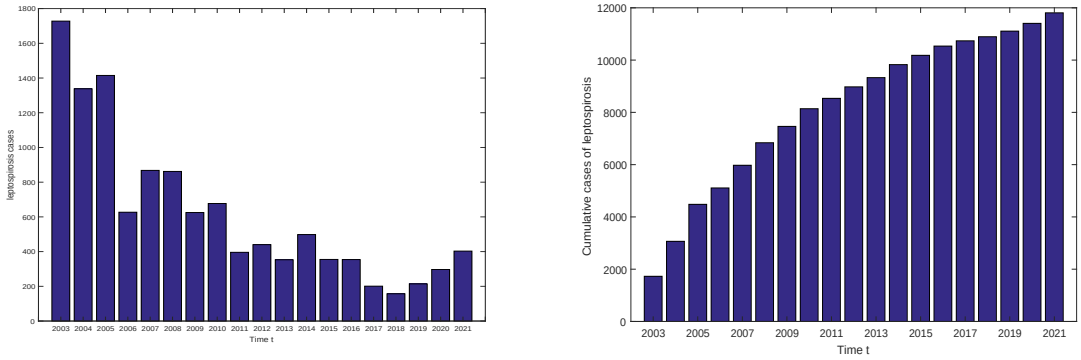
In this section, we utilize the reported leptospirosis case data in China from 2003 to 2021 to predict the future epidemic situation of the disease. The data comes from China's statistical Yearbook [20], as shown in Figure 4. The population recruitment rate of $\Lambda = 7.74 \times 10^6$ is estimated based on China's population statistics from 2003 to 2021, the natural death rate of humans is $\mu_h = 0.0064$, and the number of newly reported leptospirosis cases in 2003 was 1728 [20]. Assuming that the recruitment rate of vectors carrying leptospira is $\Pi = 1.0812 \times 10^5$, these vectors are susceptible to external factors that can lead to death, with a natural death rate of $\mu_v = 0.8125$ [21]. The specific values of the parameters are listed in Table 1.

Let the cumulative number of leptospirosis cases in the human population be defined as $D_h(t)$, and

$$\frac{\mathrm{d}D_h(t)}{\mathrm{d}t} = \frac{\beta_1 S_h I_v}{N_h} + \frac{\beta_3 S_h B}{K + B}. \tag{3.1}$$

To predict the disease, it is necessary to first estimate the two important parameters that affect the

spread of the disease, namely, β_1, β_3. We utilize the numerical solution $D_h(t)$ from model (3.1) to fit the data. Let $\Theta(\beta_1, \beta_3)$ represent the vector of parameters to be estimated, and $D_h(t, \Theta)$ represent the numerical solution of model (3.1) corresponding to the parameters Θ. The vector $Y(Y_k, k = 1, 2, 3, ..., 19)$ represents the 19 statistical data points, and t_k is the corresponding time for each data point. Take the initial value of the variable as $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), B(0), D_h(0)) = (7.74\times10^6, 1728, 307, 1.0812\times10^5, 1.867\times10^3, 1.42\times10^2, 1728)$, and the initial value of the parameter $(\beta_1, \beta_3) = (3.2326 \times 10^{-3}, 1.2 \times 10^{-4})$. Random disturbance intensities are taken as $\sigma_1 = \sigma_2 = \sigma_3 = \sigma_4 = \sigma_5 = \sigma_6 = 0.1$. We estimate the parameters using two methods below: one is the least squares method, and the other is the Markov Chain Monte Carlo (MCMC) method.



(a) Annual case report data

(b) Cumulative case report data

Figure 4. The Report on Leptospirosis Cases in China from 2003 to 2021.

Table 1. Parameter values and sources.

Parameter	Parameter value	Source	Parameter	Parameter value	Source
Λ	7.74×10^6 year^{-1}	[20]	Π	1.0812×10^5 year^{-1}	[22]
β_2	1×10^{-5} year^{-1}	Fitted	β_4	1×10^{-5} year^{-1}	Fitted
K	4.65×10^8 cells·ml^{-1}	Fitted	k	0.162 year^{-1}	[20]
μ_h	0.0064 year^{-1}	[20]	μ_v	0.8125 year^{-1}	[21]
α_1	3 cells·ml^{-1}·year^{-1}	[20]	α_2	100 cells·ml^{-1}·year^{-1}	Fitted
λ_h	0.08082 year^{-1}	[21]	δ_h	0.03328 year^{-1}	[23]
γ_h	0.08889 year^{-1}	[23]			

1) *The least squares method (LSM).* The goal is to find the optimal values of $\Theta(\beta_1, \beta_3)$ that minimize the least squares criterion:

$$LS = \sum_{k=1}^{19} |D_h(t_k, \Theta) - Y_k|^2. \tag{3.2}$$

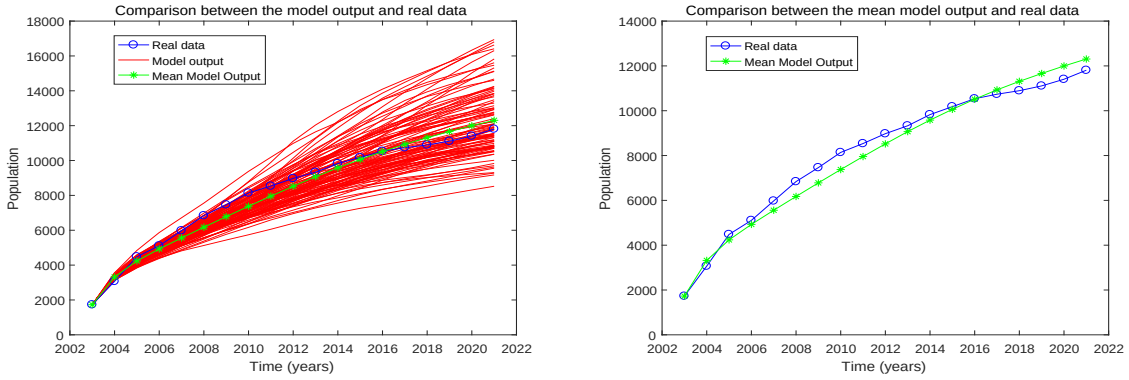
To achieve this, we utilize the *fmincon* command in the mathematical software MATLAB for numerical optimization. Based on the biological background, we set the ranges of Θ to be $((0, 0), [0.5, 0.5])$,

which serve as the constraint conditions. Using the optimization algorithm, we obtain the estimated values of the parameters. Then, we run the program 100 times and calculate the average of the output parameters $\beta_1 = 0.0032308, \beta_3 = 0.00011993$, which serve as the required parameter estimates. Figure 5(a),(b) present numerical simulations of the cumulative number of leptospirosis cases in 100 sample paths and their mean output path, respectively.

2) *Markov Chain Monte Carlo-Metropolis Hastings method (MCMC-MH).* Now, we estimate the parameters using the MCMC parameter estimation method combined with MH sampling. Let $\Theta(\beta_1, \beta_3)$ be the proposed parameter and $\Theta'(\beta_1, \beta_3)$ be the current parameter. The proposed parameter follows $\Theta = \Theta' + \varepsilon$, where ε is the step size of random walk that follows a uniform distribution. According to Bayesian statistical inference, the posterior distribution is given by:

$$P(\Theta|Y) = L(Y|\Theta)P(\Theta), \tag{3.3}$$

where the likelihood function is $L(Y|\Theta) = -\sum_{k=1}^{19} |D_h(t_k, \Theta) - Y_k|^2$, and $P(\Theta)$ is the non-informative prior distribution, assumed to be a constant C. The acceptance probability is defined as: $\alpha(\Theta, \Theta') = \min\{1, \exp(L(Y|\Theta) - L(Y|\Theta'))\}$. The ranges of Θ are also $((0, 0), [0.5, 0.5])$. After performing 5000 iterations of MCMC calculations, with a burn-in period of 1000 iterations, we computed the average of the last 4000 iterations to obtain the estimated values of the parameters as $\beta_1 = 0.0050193, \beta_3 = 0.000096193$. The 95 percent confidence interval for β_1 and β_3 is $(1.432 \times 10^{-3} - 9.941 \times 10^{-3})$, $(1.5036 \times 10^{-5} - 2.2604 \times 10^{-4})$, respectively. By substituting the estimated parameters into the model (3.1), we can obtain any 100 paths of $D_h(t)$. Figure 6(a),(b) present numerical simulations of the cumulative number of leptospirosis cases in 100 sample paths and their mean output path, respectively. Figure 6(c),(d) show the posterior distribution plots and trace plots for β_1, β_3, respectively.



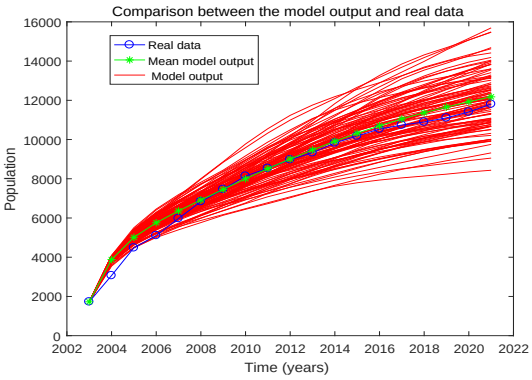
(a) 100 Sample Paths output by the model

(b) Comparing the average of 100 sample paths output by the model with the real data

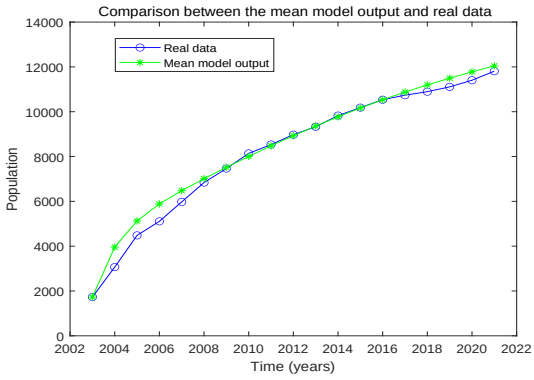
Figure 5. LSM analysis chart for parameters β_1 and β_3.

It can be seen from Figures 5 and 6 that both simulation results of the model (3.1) by two methods match the cumulative data of leptospirosis cases in China from 2003 to 2021. Next, we calculate the error value between the average curve and the real data, and compare the results from both two methods. It can be seen from Table 2 that the parameter values estimated by the two methods are very close, but the estimation error by the MCMC-MH method is smaller than LSM. Finally, using the

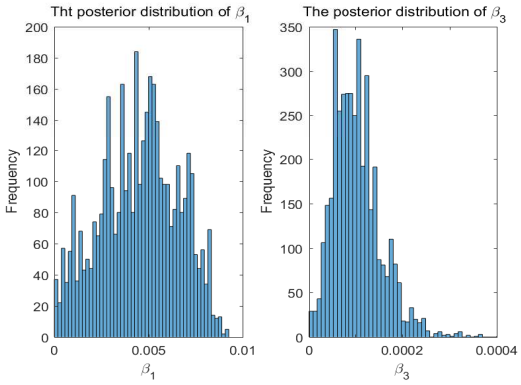
parameters estimated by the MCMC-MH method, we calculate the basic reproduction number for the transmission of leptospirosis in China, $R_m \approx 0.00075197 < 1$, and predict that leptospirosis will be eliminated in China in 26 years (see Figure 7).



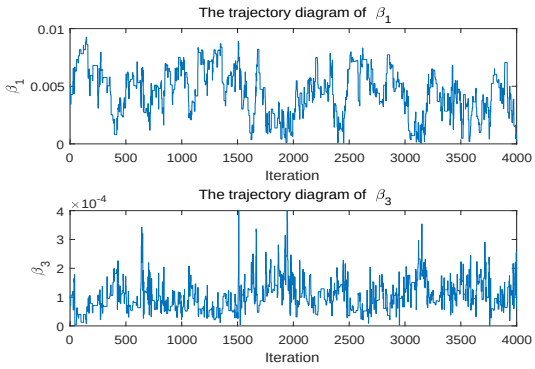
(a) 100 Sample Paths output by the model



(b) Comparing the average of 100 sample paths output by the model with the real data

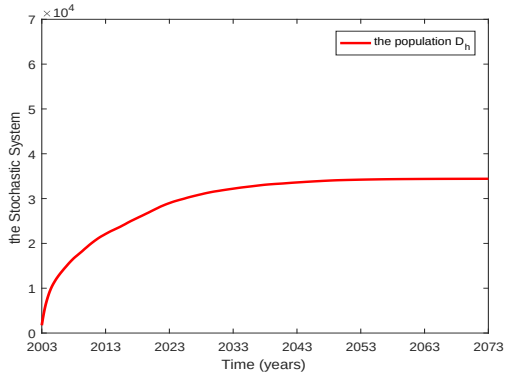


(c) β_1, β_3 Posterior Distribution

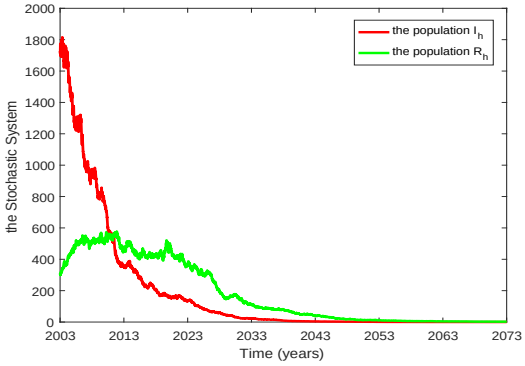


(d) β_1, β_3 trajectory Chart

Figure 6. MCMC analysis chart for parameters β_1 and β_3.



(*a*) D_h forecast chart



(*b*) I_h, R_h forecast chart

Figure 7. Prediction chart of the future trend of leptospirosis.

Table 2. Error comparison.

Method	The estimated value of β_1	The estimated value of β_3	MAPE	RSME
LSM	0.0032308	0.00011993	0.6236	4190.7348
MCMC	0.0050193	0.000096193	0.61821	3968.3587

4. Conclusions

This article establishes a stochastic leptospirosis model with both vector and environmental transmission. Through mathematical analysis of the model, a threshold for disease elimination is derived. Then, using the partial rank correlation coefficient, an impact analysis was conducted on the model parameters to identify the key parameters that have a significant influence on disease elimination. Furthermore, a sensitivity analysis of these parameters was carried out through numerical simulations, which further revealed the mechanisms of their role in the disease transmission process. This analytical approach provides a powerful tool for gaining a deeper understanding of how model parameters affect disease transmission. In the end, using data from China's leptospirosis case reports from 2003 to 2021, two parameter estimation methods, LSM and MCMC-MH, are applied to estimate the crucial parameters of the model. The simulation results of the number of infections in model (1.1) using parameters obtained from two parameter estimation methods align well with the cumulative data of leptospirosis cases in China from 2003 to 2021. It is predicted that under the current control measures, leptospirosis in China will be completely eliminated after 26 years.

Common leptospirosis models [3,5,22] tend to only consider the interaction between hosts and vectors, overlooking the influence of environmental factors. In this paper, by incorporating environmental transmission factors into the model design and considering environmental disturbance, we construct a more comprehensive and realistic stochastic infectious disease model, providing a new perspective for a more accurate understanding of the transmission mechanisms of leptospirosis. Specifically, the parameter estimation method used in this article, which combines MH sampling with MCMC, has served as a good demonstration for parameter estimation in stochastic differential systems with numerous parameters. This approach of combining actual data with parameter estimation not only enhances the accuracy and reliability of the model, but also provides strong support for predicting the future trends of leptospirosis in China. The stochastic model of leptospirosis and its related analysis methods established in this article have important theoretical and practical significance for understanding the transmission patterns of other similar vector-borne diseases and predicting future epidemic trends.

However, it must be said that when we make predictions, we only estimate two important parameters, and some parameters are based on subjective assumptions fitted to the data, which may reduce the accuracy of the prediction. In addition, the model does not fully consider the impact of human behavior, socioeconomic factors, and climate change on disease transmission. The neglect of these factors may limit the accuracy and applicability of the model. In the future, we will incorporate human behavior, socioeconomic factors, and climate change into our model, and strive to utilize actual data to estimate more parameters in order to improve the accuracy and applicability of the model. This will help us gain a deeper understanding of the dynamics of disease transmission and design effective interventions to protect public health.

Use of AI tools declaration

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

Acknowledgments

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Conflict of interest

The authors declare there is no conflicts of interest.

References

1. L. Liu, H. Zhu, G. Yang, Current situation of endemic status, prevention and control of neglected zoonotic diseases in China (in Chinese), *Chin. J. Schistosomiasis Control*, **25** (2013), 307–311.
2. H. T. Alemneh, A co-infection model of dengue and leptospirosis diseases, *Adv. Differ. Equations*, **2020** (2020), 664–687. https://doi.org/10.1186/s13662-020-03126-6
3. A. Bhalraj, A. Azmi, M. H. Mohd, Analytical and numerical solutions of leptospirosis model, *Comput. Sci.*, **16** (2021), 949–961.
4. M. A. Gallego, M. V. Simoy, Mathematical modeling of leptospirosis: a dynamic regulated by environmental carrying capacity, *Chaos, Solitons Fractals*, **152** (2021), 111425. https://doi.org/10.1016/j.chaos.2021.111425
5. H. A. Engida, D. M. Theuri, D. Gathungu, J. Gachohi, H. T. Alemneh, A mathematical model analysis for the transmission dynamics of leptospirosis disease in human and rodent populations, *Comput. Math. Methods Med.*, **2022** (2022), 1806585. https://doi.org/10.1155/2022/1806585
6. 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
7. D. Zhou, X. Y. Shi, X. Y. Zhou, Dynamic analysis of a stochastic delayed SEIRS epidemic model with Lévy jumps and the impact of public health education, *Axioms*, **12** (2023), 560. https://doi.org/10.3390/axioms12060560
8. J. Djordjevic, J. C. Silva, F. D. Torres, A stochastic SICA epidemic model for HIV transmission, *Appl. Math. Lett.*, **84** (2018), 168–175. https://doi.org/10.1016/j.aml.2018.05.005
9. Y. Zhao, D. Jiang, The threshold of a stochastic SIS epidemic model with vaccination, *Appl. Math. Comput.*, **243** (2014), 718–727. https://doi.org/10.1016/j.amc.2014.05.124
10. X. Meng, S. Zhao, T. Feng, T. Zhang, Dynamics of a novel nonlinear stochastic SIS epidemic model with double epidemic hypothesis, *J. Math. Anal. Appl.*, **433** (2016), 227–242. https://doi.org/10.1016/j.jmaa.2015.07.056
11. A. Din, Bifurcation analysis of a delayed stochastic HBV epidemic model: Cell-to-cell transmission, *Chaos, Solitons Fractals*, **181** (2024), 114714. https://doi.org/10.1016/j.chaos.2024.114714

12. A. Din, Y. Li, A. Yusuf, Delayed hepatitis B epidemic model with stochastic analysis, *Chaos, Solitons Fractals*, **146** (2021), 110839. https://doi.org/10.1016/j.chaos.2021.110839
13. X. Mao, G. Marion, E. Renshaw, Environmental Brownian noise suppresses explosions in population dynamics, *Stochastic Processes Appl.*, **97** (2002), 95–110. https://doi.org/10.1016/S0304-4149(01)00126-0
14. Y. Zhao, D. Jiang, The threshold of a stochastic SIS epidemic model with vaccination, *Appl. Math. Comput.*, **243** (2014), 718–727. https://doi.org/10.1016/j.amc.2014.05.124
15. Q. T. Ain, Nonlinear stochastic cholera epidemic model under the influence of noise, *J. Math. Tech. Model.*, **1** (2024), 52–74. https://doi.org/10.56868/jmtm.v1i1.30
16. R. Khasminskii, *Stochastic Stability of Differential Equations*, Springer Berlin, Heidelberg, 2011.
17. S. Marino, I. B. Hogue, C. Ray, et al., A methodology for performing global uncertainty and sensitivity analysis in systems biology, *J. Theor. Biol.*, **254** (2009), 178–196. https://doi.org/10.1016/j.jtbi.2008.04.011
18. D. J. Higham, An algorithmic introduction to numerical simulation of stochastic differential equations, *SIAM Rev.*, **43** (2001), 525–546. https://doi.org/10.1137/S0036144500378302
19. Q. T. Ain, J. Shen, P. Xu, X. Qiang, Z. Kou, A stochastic approach for co-evolution process of virus and human immune system, *Sci. Rep.*, **14** (2024), 10337. https://doi.org/10.1038/s41598-024-60911-z
20. *China's Statistical Yearbook*, the National Bureau of Statistics of China, 2003–2021. Available from: https://www.stats.gov.cn/sj/ndsj/.
21. W. Triampo, D. Baowan, I. M. Tang, N. Nuttavut, J. Wong-Ekkabut, G. Doungchawee, A simple deterministic model for the spread of leptospirosis in Thailand, *Int. J. Bio. Med. Sci.*, **2** (2007), 22–26.
22. M. A. Khan, S. Islam, S. A. Khan, G. Zaman, Global stability of vector-host disease with variable population size, *Biomed Res. Int.*, **2013** (2013), 710917. http://dx.doi.org/10.1155/2013/710917
23. W. Tangkanakul, H. L. Smits, S. Jatanasen, D. A. Ashford, Leptospirosis: an emerging health problem in Thailand, *Southeast Asian J. Trop. Med. Public Health*, **36** (2005), 281–288.