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Research article

Modeling and analysis of the transmission dynamics of cystic echinococcosis: Effects of increasing the number of sheep

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Abstract: A transmission dynamics model with the logistic growth of cystic echinococcus in sheep was formulated and analyzed. The basic reproduction number was derived and the results showed that the global dynamical behaviors were determined by its value. The disease-free equilibrium is globally asymptotically stable when the value of the basic reproduction number is less than one; otherwise, there exists a unique endemic equilibrium and it is globally asymptotically stable. Sensitivity analysis and uncertainty analysis of the basic reproduction number were also performed to screen the important factors that influence the spread of cystic echinococcosis. Contour plots of the basic reproduction number versus these important factors are presented, too. The results showed that the higher the deworming rate of dogs, the lower the prevalence of echinococcosis in sheep and dogs. Similarly, the higher the slaughter rate of sheep, the lower the prevalence of echinococcosis in sheep and dogs. It also showed that the spread of echinococcosis has a close relationship with the maximum environmental capacity of sheep, and that they have a remarkable negative correlation. This reminds us that the risk of cystic echinococcosis may be underestimated if we ignore the increasing number of sheep in reality.

Keywords: epidemic model; cystic echinococcosis; basic reproduction number; global asymptotic stability; sensitivity analysis

1. Introduction

Cystic echinococcosis (CE) is a worldwide parasitic disease produced by echinococcus granulosus. In the life cycle of echinococcosis granulosus (Figure 1), dogs are the primary definitive host, and livestock (such as sheep, goats and swine) constitute the major intermediate host. In usual, CE mainly transmits between dogs and livestock. Humans as accidental hosts of CE, infected by eggs in the environment but not participating in the spread of the disease. This disease mainly occurs in animal

husbandry countries and causes about USD 2 billion dollars of economic losses to the livestock industry every year [1, 2]. The World Health Organization recognizes it as an ignored tropical disease and lists it as a priority disease [3, 4].

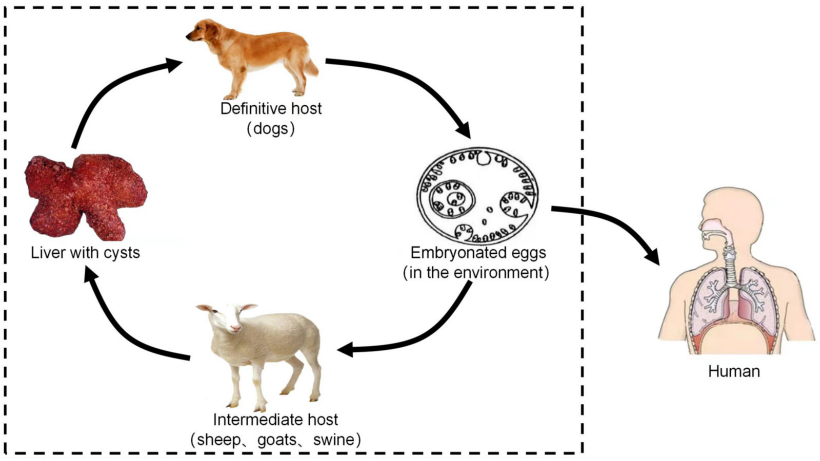


Figure 1. The life cycle of Echinococcus granulosus.

It is a crucial global public health goal to prevent and control the spread of CE [5]. Mathematical modeling is a powerful tool to solve such problems. A great quantity of mathematical models on CE have been developed and analyzed [6–8]. Roberts el al. [9, 10] and Gemmell et al. [11–13] explored a mathematical model of the life cycle of CE in dogs and sheep to discuss previously published experimental and survey data. Considering that CE cannot spread without eggs in the environment, Wang et al. [14] constructed a new deterministic model incorporating dogs, sheep, humans and eggs in the environment, and they used it to examine the transmission dynamics of echinococcosis in Xinjiang. Recently, Rong et al. [15, 16] improved the compartmental model presented in [14] and used the new model to investigate the effect of stray dogs and searched the optimal control measures. Zhao and Yang [17] considered an echinococcosis model that contains four control measures and discussed the optimal control problem to decrease and eliminate the spread of echinococcosis between dogs and livestock with comprehensive interventions. Cui [18] analyzed the prevention and control effects of CE in Pengyang County, predicted the epidemic trend of CE in Pengyang County and evaluated the impact of prevention and control measures on the spread of the disease. For more details, the reader can refer to [19–21].

However, these studies disregard the effect of the number of sheep on the prevention and control of echinococcosis. In fact, the demand for sheep is increasing as the population increases. For example, the production of mutton in China increased from 4.45 million tons in 2012 to 5.14 million tons in 2021, and more than 146.2 million sheep were slaughtered in 2017, which is almost 20 million more than six years ago (http://ncpscxx.moa.gov.cn/). Moreover, sheep, as the main intermediate host, represent the key link in the spread of echinococcosis, and they are also the main income of many farmers and herders. Specifically, the importance of considering the effects of the prevalence of echinococcus in sheep was once mentioned by Yang et al. [22]. They studied the prevalence of CE in slaughtered

sheep in Emin County, Xinjiang, China. In addition, Xiao et al. [23] investigated the epidemiology of sheep echinococcosis in Kashi, Xinjiang during 2014–2017. Recently, Gao et al. [24] investigated the epidemic situation of sheep echinococcus in China from 1983 to 2020 by using meta-analysis. These studies focused on the epidemiological survey of sheep echinococcosis, and there is no clear qualitative analysis of the effect of sheep on the spread of CE.

Thus, in this paper, we attempt to elucidate the impact of increasing sheep on the spread of CE by constructing and analyzing a mathematical model. The remainder of this paper is organized as follows. Section 2 presents the development of a CE model to highlight the logistic growth of sheep in reality. The basic reproduction number and the dynamical behaviors of the model are presented in Section 3. In the next section, the effects of sheep and other control measures are investigated through numerical simulations. A brief conclusion and discussion are presented in the last section.

2. Model formulation

In this section, a dynamical model based on the transmission of CE among animals is formulated. Based on the idea of compartmental modeling, the definitive host dogs are divided into susceptible dogs and infected dogs, and the corresponding numbers at time t are respectively denoted by $S_D(t)$ and $I_D(t)$; the intermediate host sheep is divided into susceptible, immune and infected individuals, and the corresponding numbers at time t are recorded as $S_L(t)$, $V_L(t)$ and $I_L(t)$, respectively; and the number of parasite eggs in the environment is denoted by $E(t)$ at time t. Let $N_L(t) = S_L(t)+V_L(t)+I_L(t)$ be the total number of intermediate host sheep that satisfies

$$\frac{dN_L}{dt} = (b_2 - d_2)\left(1 - \frac{N_L}{K}\right)N_L - \theta N_L, \tag{2.1}$$

where b_2 represents the natural birth rate of sheep, d_2 denotes the natural death rate of sheep, K represents the maximum environmental capacity of sheep and θ is the slaughter rate of sheep. To model $N_L(t)$ as increasing in reality, we assume that $b_2 \geq d_2 + \theta$. Based on this compartmental scheme (Figure 2), a dog-sheep-egg life cycle transmission dynamics model can be given as

$$\begin{cases} \dfrac{dS_D}{dt} = A_1 - \beta_1 S_D I_L - d_1 S_D + \sigma I_D, \\ \dfrac{dI_D}{dt} = \beta_1 S_D I_L - (d_1 + \sigma) I_D, \\ \dfrac{dS_L}{dt} = b_2 N_L - \beta_2 S_L E - (\theta + d_2) S_L - (b_2 - d_2)\dfrac{N_L}{K} S_L - \omega S_L + \delta V_L, \\ \dfrac{dV_L}{dt} = \omega S_L - \delta V_L - (\theta + d_2) V_L - (b_2 - d_2)\dfrac{N_L}{K} V_L, \\ \dfrac{dI_L}{dt} = \beta_2 S_L E - (\theta + d_2) I_L - (b_2 - d_2)\dfrac{N_L}{K} I_L, \\ \dfrac{dE}{dt} = \alpha I_D - d_e E. \end{cases} \tag{2.2}$$

For the definitive host dogs, parameter A_1 represents the number of dogs born per year, d_1 is the natural mortality rate of dogs, σ is the deworming recovery rate for infectious dogs, β_1 is the dogs'

transmission rate and $\beta_1 S_D I_L$ represents the transmission of echinococcosis to dogs by ingesting cyst-containing organs of infectious sheep. For the intermediate host sheep, ω represents the vaccination rate of sheep, δ is the immune failure rate of sheep, β_2 is the transmission rate of sheep and $\beta_2 S_L E$ denotes the transmission of echinococcosis to sheep by ingesting parasite eggs in the environment. Moreover, α represents the release rate of parasite eggs in the environment, and d_e denotes the natural extinction rate of parasite eggs.

It should be noted that the release rate of parasite eggs in the environment, α, is determined by the average annual amount of parasites in each dog, q, the number of eggs laid per adult per unit time, b_e, the average life span of a dog, t_d, and adult mortality in dogs, d. It follows from literature [6] that $\alpha = b_e q\left(1 - e^{-dt_d}\right)/d$.

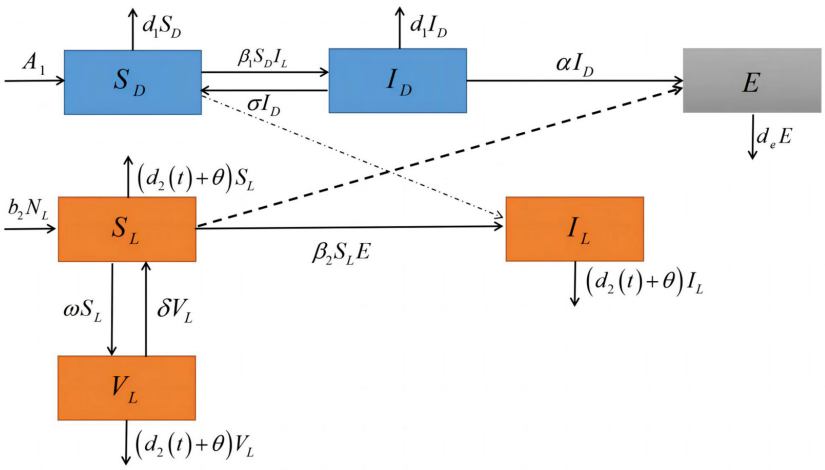


Figure 2. Compartmental model of the transmission dynamics of CE.

Based on the biological meaning of the model, all solutions of the system (2.2) are assumed to satisfy the following initial conditions:

$$\begin{aligned} S_D(0) = S_{D0} > 0, I_D(0) = I_{D0} \geq 0, S_L(0) = S_{L0} > 0, \\ V_L(0) = V_{L0} > 0, I_L(0) = I_{L0} \geq 0, E(0) = E_0 > 0. \end{aligned} \tag{2.3}$$

Define

$$\Gamma = \left\{(S_D, I_D, S_L, V_L, I_L, E) \in \mathbb{R}_+^6, S_D + I_D \leq \frac{A_1}{d_1}, S_L + V_L + I_L \leq \frac{(b_2 - d_2 - \theta) K}{b_2 - d_2}, E \leq \frac{\alpha A_1}{d_e d_1}\right\}.$$

We have the following result.

Theorem 1. *All of the solutions satisfying the initial conditions of the system* (2.2) *are positive;* Γ *is the positively invariant set of system* (2.2).

Proof. Let $x(t) = (S_D(t), I_D(t), S_L(t), V_L(t), I_L(t), E(t))$ be the solution of the system (2.2) satisfying the initial conditions given by (2.3). We first prove that $x(t)$ is the positive solution.

Assume that the conclusion is not true; then, there exists a constant $t_1 > 0$ such that

$$x^0(t_1) = \min\{S_D(t_1), I_D(t_1), S_L(t_1), V_L(t_1), I_L(t_1), E(t_1)\} = 0, \text{ for any } t \in [0, t_1),$$

and

$$x^m(t) = \min\{S_D(t), I_D(t), S_L(t), V_L(t), I_L(t), E(t)\} > 0.$$

If $x^0(t_1) = I_D(t_1)$, by the second equation of system (2.2), we have

$$\frac{dI_D(t)}{dt} \geq -(d_1 + \sigma) I_D, \forall t \in [0, t_1).$$

One can easily calculate that

$$I_D(t_1) \geq I_D(0) \exp(-(d_1 + \sigma) t_1) > 0.$$

This is inconsistent with $x^0(t_1) = I_D(t_1)$.

If $x^0(t_1) = E(t_1)$, it can be known from the last equation of system (2.2) that

$$\frac{dE(t)}{dt} \geq -d_e E, \forall t \in [0, t_1).$$

Then, we have

$$E(t_1) \geq E(0) \exp(-d_e t_1) > 0.$$

This is inconsistent with $x^0(t_1) = E(t_1)$.

Similarly results can be obtained when $S_D^0(t_1) = S_D(t_1), S_L^0(t_1) = S_L(t_1), I_L^0(t_1) = I_L(t_1)$ and $V_L^0(t_1) = V_L(t_1)$. Therefore, all of the solutions satisfying the initial conditions given by (2.3) are positive solutions for system (2.2).

Next, let us prove that Γ is the positively invariant set of the system (2.2). From the first two equations of system (2.2), we have

$$\frac{d(S_D(t) + I_D(t))}{dt} \leq A_1 - (S_D + I_D) d_1.$$

Simple calculation implies that

$$\lim_{t\to\infty} (S_D(t) + I_D(t)) \leq \frac{A_1}{d_1}.$$

Similarly, we have

$$\lim_{t\to\infty} (S_L(t) + V_L(t) + I_L(t)) \leq \frac{(b_2 - d_2 - \theta) K}{b_2 - d_2} \text{ and } \lim_{t\to\infty} E(t) \leq \frac{\alpha A_1}{d_e d_1}.$$

Therefore, Γ is the positively invariant set of system (2.2).

3. Mathematical analysis

In this section, we first investigate the existence of the disease-free equilibrium and endemic equilibrium and then discuss the global dynamics of system (2.2).

3.1. Equilibrium and basic reproduction number

Set the right-hand side of system (2.2) as zero; then, we have the following algebraic equation

$$\begin{cases} A_1 - \beta_1 S_D I_L - d_1 S_D + \sigma I_D = 0, \\ \beta_1 S_D I_L - (d_1 + \sigma) I_D = 0, \\ b_2 N_L - \beta_2 S_L E - (\theta + d_2) S_L - (b_2 - d_2) \frac{N_L}{K} S_L - \omega S_L + \delta V_L = 0, \\ \omega S_L - \delta V_L - (\theta + d_2) V_L - (b_2 - d_2) \frac{N_L}{K} V_L = 0, \\ \beta_2 S_L E - (\theta + d_2) I_L - (b_2 - d_2) \frac{N_L}{K} I_L = 0, \\ \alpha I_D - d_e E = 0. \end{cases} \tag{3.1}$$

In the absence of disease, it follows from Eq (3.1) that system (2.2) always has a disease-free equilibrium $E_{dfe} = \left(S_D^0, 0, S_L^0, V_L^0, 0, 0\right)$, where

$$S_D^0 = \frac{A_1}{d_1}, S_L^0 = h\frac{(b_2 - d_2 - \theta) K}{b_2 - d_2}, V_L^0 = (1 - h) \frac{(b_2 - d_2 - \theta) K}{b_2 - d_2}, h = \frac{\delta + b_2}{\omega + \delta + b_2}.$$

According to the next-generation operator approach proposed by Diekmann et al. [25] and van den Driessche and Watmough [26], we define

$$\mathcal{F} = \begin{bmatrix} \beta_1 S_D I_L \\ \beta_2 S_L E \\ \alpha I_D \end{bmatrix}, \mathcal{V} = \begin{bmatrix} (d_1 + \sigma) I_D \\ (\theta + d_2) I_L + (b_2 - d_2) \frac{N_L}{K} I_L \\ d_e E \end{bmatrix}.$$

Note that the disease-free equilibrium of system (2.2) is E_{dfe}; then,

$$F = \begin{bmatrix} 0 & \frac{\beta_1 A_1}{d_1} & 0 \\ 0 & 0 & \frac{\beta_2 h(b_2 - d_2 - \theta) K}{b_2 - d_2} \\ \alpha & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} d_1 + \sigma & 0 & 0 \\ 0 & b_2 & 0 \\ 0 & 0 & d_e \end{bmatrix}.$$

Hence, the next generation matrix reads as

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_1 A_1}{b_2 d_1} & 0 \\ 0 & 0 & \frac{\beta_2 h(b_2 - d_2 - \theta) K}{(b_2 - d_2) d_e} \\ \frac{\alpha}{d_1 + \sigma} & 0 & 0 \end{bmatrix}.$$

Then the basic reproduction number, which is calculated from $\rho\left(FV^{-1}\right)$, is as follows:

$$R_0 = \sqrt[3]{\underbrace{\frac{\alpha}{d_e}}_{\text{eggs by per dog}} \cdot \underbrace{\frac{\beta_2 h (b_2 - d_2 - \theta) K}{(d_1 + \sigma)(b_2 - d_2)}}_{\text{infected sheep by eggs}} \cdot \underbrace{\frac{\beta_1 A_1}{b_2 d_1}}_{\text{infected dogs}}}. \tag{3.2}$$

Here, R_0 represents the average number of new infections produced by infectious dogs during their respective infection period [26, 27]. In the non-negative neighborhood of the disease-free equilibrium, the density of echinococcus eggs released by each infectious dog is α/d_e. In the dogs' expected infectious period $1/(d_1 + \sigma)$, the susceptible sheep $[h(b_2 - d_2 - \theta) K]/(b_2 - d_2)$ are infected by contacting parasites eggs with the probability β_2. Meanwhile, in the sheep expected infectious period

$1/b_2$, the total dogs (number: A_1/d_1) are infected by ingesting infectious cyst-containing organs of sheep with the probability β_1.

If there exist infected dogs or sheep, it follows from Eq (3.1) that system (2.2) has an endemic equilibrium $E_e^* = \left(S_D^*, I_D^*, S_L^*, V_L^*, I_L^*, E^*\right)$ when $R_0 > 1$, where

$$S_D^* = \frac{A_1 - d_1 I_D^*}{d_1},\ I_D^* = \frac{d_e b_2 (b_2 - d_2)(d_1 + \sigma)\left(R_0^3 - 1\right)}{\alpha\beta_2 h\left[(b_2 - d_2 - \theta) K\beta_1 + (b_2 - d_2)(d_1 + \sigma)\right]},$$

$$S_L^* = \frac{(b_2 - d_2 - \theta) hK - (b_2 - d_2) hI_L^*}{b_2 - d_2},\ V_L^* = \frac{\omega}{\delta + b_2} S_L^*,$$

$$I_L^* = \frac{d_1 (d_1 + \sigma) I_D^*}{\beta_1\left(A_1 - d_1 I_D^*\right)},\quad E^* = \frac{\alpha I_D^*}{d_e}.$$

3.2. Global dynamical behaviors

Based on Theorem 2 presented by van den Driessche and Watmough [26], when $R_0 < 1$, the disease-free equilibrium E_{dfe} of system (2.2) is locally asymptotically stable, and unstable when $R_0 > 1$. The global asymptotic stability of the disease-free equilibrium E_{dfe} can be given by performing a direct Lyapunov approach.

Theorem 2. *If $R_0 \leq 1$, the disease-free equilibrium E_{dfe} of system* (2.2) *is globally asymptotically stable.*

Proof. Let $(S_D(t), I_D(t), S_L(t), V_L(t), I_L(t), E(t))$ be any solution of the system (2.2) that satisfies

$$S_D(t) \leq \frac{A_1}{d_1},\ S_L(t) \leq \frac{h(b_2 - d_2 - \theta) K}{b_2 - d_2},\ N_L(t) \leq \frac{(b_2 - d_2 - \theta) K}{b_2 - d_2},$$

for $\forall t \geq t_0,$ where $\exists t_0 > N,\ N \in \mathbb{R}^+$.

Define

$$V(I_D, I_L, E) = \frac{\alpha\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)(d_1 + \sigma)} I_D + \frac{1}{b_2} I_L + \frac{\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)} E.$$

By applying its derivative along the solutions of system (2.2) and using the expression of R_0, we calculate that

$$\begin{aligned}
\frac{dV}{dt} &= \frac{\alpha\beta_1\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)(d_1 + \sigma)} S_D I_L - \frac{\alpha\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)} I_D + \frac{\beta_2 S_L E}{b_2} \\
&\quad - \frac{\theta + d_2}{b_2} I_L - (b_2 - d_2)\frac{N}{b_2 K} I_L + \frac{\alpha\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)} I_D - \frac{\beta_2 h(b_2 - d_2 - \theta) K}{b_2 (b_2 - d_2)} E \\
&\leq \frac{\alpha\beta_1\beta_2 A_1 h(b_2 - d_2 - \theta) K}{d_1 b_2 d_e (b_2 - d_2)(d_1 + \sigma)} I_L - \frac{\alpha\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)} I_D + \frac{\beta_2 h(b_2 - d_2 - \theta) K}{b_2 (b_2 - d_2)} E \\
&\quad - \frac{\theta + d_2}{b_2} I_L - \frac{(b_2 - d_2 - \theta)}{b_2} I_L + \frac{\alpha\beta_2 h(b_2 - d_2 - \theta) K}{b_2 d_e (b_2 - d_2)} I_D - \frac{\beta_2 h(b_2 - d_2 - \theta) K}{b_2 (b_2 - d_2)} E \\
&= \frac{\alpha\beta_1\beta_2 A_1 h(b_2 - d_2 - \theta) K}{d_1 b_2 d_e (b_2 - d_2)(d_1 + \sigma)} I_L - I_L
\end{aligned}$$

$$= \left(\frac{\alpha\beta_1\beta_2 A_1 h\,(b_2 - d_2 - \theta)\,K}{d_1 b_2 d_e\,(b_2 - d_2)\,(d_1 + \sigma)} - 1 \right) I_L$$
$$= \left(R_0^3 - 1 \right) I_L.$$

Therefore, $dV/dt \leq 0$ if $R_0 \leq 1$ and $dV/dt = 0$ if and only if $I_L = 0$. It is not hard to verify that $\left(S_D^0, 0, S_L^0, V_L^0, 0, 0\right)$ is the unique invariant set of system (2.2). According to Lasalle's invariant principle [28], the disease-free equilibrium E_{dfe} is globally asymptotically stable.

The proof of Theorem 1 implies that system (2.1) has a unique positive equilibrium N_L^0 and one can easily verify that N_L^0 is globally asymptotically stable in Γ, that is,

$$\lim_{t\to\infty} N_L(t) = \frac{(b_2 - d_2 - \theta)\,K}{b_2 - d_2} \triangleq N_L^0.$$

Using the results from Castillo-Chavez and Thieme [29] and Mischaikow et al. [30], we can obtain the analytical results by considering the following limit system of system (2.2) in which the sheep population is assumed to be constant N_L^0:

$$\begin{cases} \dfrac{dS_D}{dt} = A_1 - \beta_1 S_D I_L - d_1 S_D + \sigma I_D, \\ \dfrac{dI_D}{dt} = \beta_1 S_D I_L - (d_1 + \sigma) I_D, \\ \dfrac{dS_L}{dt} = b_2 N_L^0 - \beta_2 S_L E - (\theta + d_2) S_L - (b_2 - d_2) \dfrac{N_L^0}{K} S_L - \omega S_L + \delta V_L, \\ \dfrac{dV_L}{dt} = \omega S_L - \delta V_L - (\theta + d_2) V_L - (b_2 - d_2) \dfrac{N_L^0}{K} V_L, \\ \dfrac{dI_L}{dt} = \beta_2 S_L E - (\theta + d_2) I_L - (b_2 - d_2) \dfrac{N_L^0}{K} I_L, \\ \dfrac{dE}{dt} = \alpha I_D - d_e E. \end{cases} \tag{3.3}$$

Theorem 3. *If $R_0 > 1$, the endemic equilibrium E_e^* is globally asymptotically stable .*

Proof. Let $g(x) = x - 1 - \ln x, x > 0$. Obviously, $g(x) \geq 0$, if and only if $x = 1$, $g(x) = 0$. Note that $(x-1)(1-y) = g(x) + g(y) - g(xy)$ for all $x, y \in \mathbb{R}_+$. Let $V_\# = \#^* g\left(\frac{\#}{\#^*}\right)$, where # represents S_D, I_D, S_L, V_L, I_L and E.

Then, using the equilibrium equation $A_1 - \beta_1 S_D^* I_L^* - d_1 S_D^* + \sigma I_D^* = 0$ and differentiating V_{S_D} along system (2.2), one has

$$\begin{aligned} \frac{dV_{S_D}}{dt}\Big|_{(2.2)} &= \left(1 - \frac{S_D^*}{S_D}\right) S_D' \\ &= \left(1 - \frac{S_D^*}{S_D}\right) \left[-\beta_1 (S_D I_L - S_D^* I_L^*) - d_1 (S_D - S_D^*) + \sigma (I_D - I_D^*)\right] \\ &= -\beta_1 S_D^* I_L^* \left(\frac{S_D I_L}{S_D^* I_L^*} - 1\right)\left(1 - \frac{S_D^*}{S_D}\right) - d_1 S_D^* \left(\frac{S_D}{S_D^*} - 1\right)\left(1 - \frac{S_D^*}{S_D}\right) \end{aligned}$$

$$
\begin{aligned}
&+\sigma I_D^*\left(\frac{I_D}{I_D^*}-1\right)\left(1-\frac{S_D^*}{S_D}\right)\\
&=-\beta_1 S_D^* I_L^* g\left(\frac{S_D I_L}{S_D^* I_L^*}\right)-\beta_1 S_D^* I_L^* g\left(\frac{S_D^*}{S_D}\right)+\beta_1 S_D^* I_L^* g\left(\frac{I_L}{I_L^*}\right)-d_1 S_D^* g\left(\frac{S_D}{S_D^*}\right)\\
&\quad-d_1 S_D^* g\left(\frac{S_D^*}{S_D}\right)+\sigma I_D^* g\left(\frac{I_D}{I_D^*}\right)+\sigma I_D^* g\left(\frac{S_D^*}{S_D}\right)-\sigma I_D^* g\left(\frac{I_D S_D^*}{I_D^* S_D}\right)\\
&=-\beta_1 S_D^* I_L^* g\left(\frac{S_D I_L}{S_D^* I_L^*}\right)+\beta_1 S_D^* I_L^* g\left(\frac{I_L}{I_L^*}\right)-d_1 S_D^* g\left(\frac{S_D}{S_D^*}\right)+\sigma I_D^* g\left(\frac{I_D}{I_D^*}\right)\\
&\quad-\sigma I_D^* g\left(\frac{I_D S_D^*}{I_D^* S_D}\right)-A_1 g\left(\frac{S_D^*}{S_D}\right).
\end{aligned}
$$

Using the equilibrium equation $\beta_1 S_D^* I_L^* - (d_1+\sigma) I_D^* = 0$ and differentiating V_{I_D} along system (2.2), one has

$$
\begin{aligned}
\frac{dV_{I_D}}{dt}\Big|_{(2.2)} &= \left(1-\frac{I_D^*}{I_D}\right) I_D'\\
&= \left(1-\frac{I_D^*}{I_D}\right)\left[\beta_1\left(S_D I_L - S_D^* I_L^*\right)-(d_1+\sigma)\left(I_D-I_D^*\right)\right]\\
&= \beta_1 S_D^* I_L^*\left(\frac{S_D I_L}{S_D^* I_L^*}-1\right)\left(1-\frac{I_D^*}{I_D}\right)-(d_1+\sigma) I_D^*\left(\frac{I_D}{I_D^*}-1\right)\left(1-\frac{I_D^*}{I_D}\right)\\
&= \beta_1 S_D^* I_L^* g\left(\frac{S_D I_L}{S_D^* I_L^*}\right)+\beta_1 S_D^* I_L^* g\left(\frac{I_D^*}{I_D}\right)-\beta_1 S_D^* I_L^* g\left(\frac{S_D I_L I_D^*}{S_D^* I_L^* I_D}\right)\\
&\quad-(d_1+\sigma) I_D^* g\left(\frac{I_D}{I_D^*}\right)-(d_1+\sigma) I_D^* g\left(\frac{I_D^*}{I_D}\right)\\
&= \beta_1 S_D^* I_L^* g\left(\frac{S_D I_L}{S_D^* I_L^*}\right)-\beta_1 S_D^* I_L^* g\left(\frac{S_D I_L I_D^*}{S_D^* I_L^* I_D}\right)-d_1 I_D^* g\left(\frac{I_D}{I_D^*}\right)-\sigma I_D^* g\left(\frac{I_D}{I_D^*}\right).
\end{aligned}
$$

Similarly, we have

$$
\begin{aligned}
\frac{dV_{S_L}}{dt}\Big|_{(2.2)} &= -\beta_2 S_L^* E^* g\left(\frac{S_L E}{S_L^* E^*}\right)+\beta_2 S_L^* E^* g\left(\frac{E}{E^*}\right)-(\theta+d_2+\omega) S_L^* g\left(\frac{S_L}{S_L^*}\right)\\
&\quad-\frac{b_2-d_2}{K} N_L^0 S_L^* g\left(\frac{S_L}{S_L^*}\right)+\delta V_L^* g\left(\frac{V_L}{V_L^*}\right)-\delta V_L^* g\left(\frac{V_L S_L^*}{V_L^* S_L}\right)-b_2 N_L^0 g\left(\frac{S_L^*}{S_L}\right).
\end{aligned}
$$

$$
\begin{aligned}
\frac{dV_{V_L}}{dt}\Big|_{(2.2)} &= -\omega S_L^* g\left(\frac{S_L V_L^*}{S_L^* V_L}\right)-(\delta+\theta+d_2) V_L^* g\left(\frac{V_L}{V_L^*}\right)-\frac{b_2-d_2}{K} N_L^0 V_L^* g\left(\frac{V_L}{V_L^*}\right)\\
&\quad+\omega S_L^* g\left(\frac{S_L}{S_L^*}\right).\\
\frac{dV_{I_L}}{dt}\Big|_{(2.2)} &= \beta_2 S_L^* E^* g\left(\frac{S_L E}{S_L^* E^*}\right)-\beta_2 S_L^* E^* g\left(\frac{S_L E I_L^*}{S_L^* E^* I_L}\right)-b_2 I_L^* g\left(\frac{I_L}{I_L^*}\right).
\end{aligned}
$$

$$\frac{dV_E}{dt}\Big|_{(2.2)} = \alpha I_D^* g\left(\frac{I_D}{I_D^*}\right) - \alpha I_D^* g\left(\frac{I_D E^*}{I_D^* E}\right) - d_e E^* g\left(\frac{E}{E^*}\right).$$

Then, consider the following Lyapunov candidate function:

$$\bar{V} = \frac{\alpha\beta_2 S_L^*}{d_1 d_e}\left(V_{S_D} + V_{I_D}\right) + V_{S_L} + V_{V_L} + V_{I_L} + \frac{\beta_2 S_L^*}{d_e} V_E.$$

Add the derivatives of $V_{S_D}, V_{I_D}, \ldots, V_E$ along system (2.2); then, combining with $\sigma = 0$, we have

$$\begin{aligned}\frac{d\bar{V}}{dt}\Big|_{(2.2)} = &-\frac{\alpha\beta_2 S_L^* S_D^*}{d_e} g\left(\frac{S_D}{S_D^*}\right) - \frac{\alpha\beta_2 S_L^* \sigma I_D^*}{d_1 d_e} g\left(\frac{I_D S_D^*}{I_D^* S_D}\right) - \frac{\alpha A_1 \beta_2 S_L^*}{d_1 d_e} g\left(\frac{S_D^*}{S_D}\right)\\ &- \frac{\alpha\beta_2 S_L^* \beta_1 S_D^* I_L^*}{d_1 d_e} g\left(\frac{S_D I_L I_D^*}{S_D^* I_L^* I_D}\right) - (\theta + d_2) S_L^* g\left(\frac{S_L}{S_L^*}\right) - \frac{b_2 - d_2}{K} N_L^0 S_L^* g\left(\frac{S_L}{S_L^*}\right)\\ &- \delta V_L^* g\left(\frac{V_L S_L^*}{V_L^* S_L}\right) - b_2 N_L^0 g\left(\frac{S_L^*}{S_L}\right) - \omega S_L^* g\left(\frac{S_L V_L^*}{S_L^* V_L}\right) - (\theta + d_2) V_L^* g\left(\frac{V_L}{V_L^*}\right)\\ &- \frac{b_2 - d_2}{K} N_L^0 V_L^* g\left(\frac{V_L}{V_L^*}\right) - \beta_2 S_L^* E^* g\left(\frac{S_L E I_L^*}{S_L^* E^* I_L}\right) - \frac{\alpha\beta_2 S_L^* I_D^*}{d_e} g\left(\frac{I_D E^*}{I_D^* E}\right).\end{aligned}$$

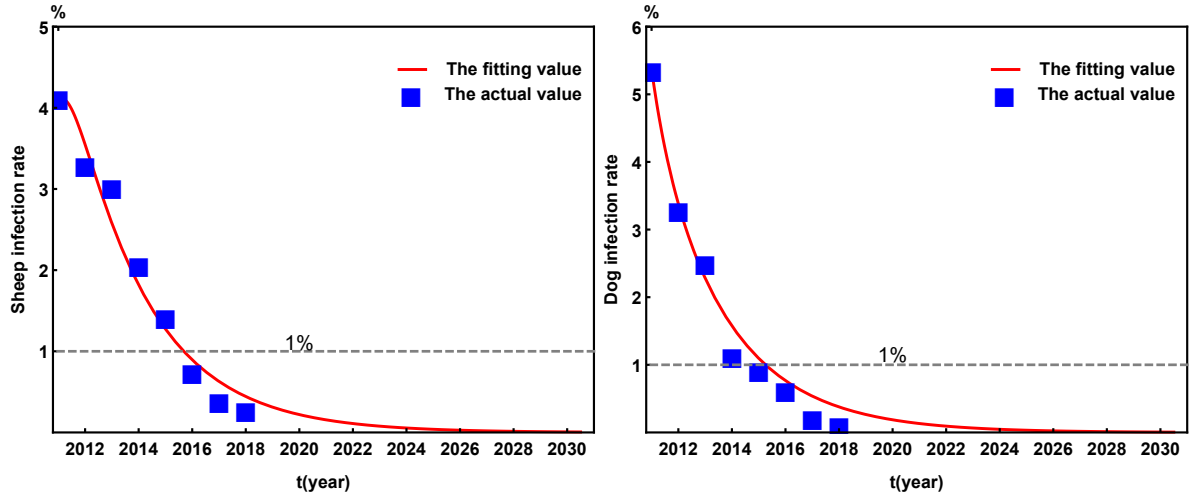
Therefore, $d\bar{V}/dt \le 0$ and $d\bar{V}/dt = 0$ if and only if $S_D = S_D^*, I_D = I_D^*, S_L = S_L^*, V_L = V_L^*, I_L = I_L^*, E = E^*$. Then, the maximal invariant set of system (2.2) in the set $\left\{(S_D, I_D, S_L, V_L, I_L, E)\,\middle|\, d\bar{V}/dt = 0\right\}$ is $\left(S_D^*, I_D^*, S_L^*, V_L^*, I_L^*, E^*\right)$. Additionally, because the Lyapunov function $\bar{V}$ has a lower bound of 0 on $\mathbb{R}^6$, when $\|(S_D, I_D, S_L, V_L, I_L, E)\| \to +\infty$, we have that $\bar{V} \to +\infty$. It follows from LaSalle's invariant principle [28] that $E_e^* = \left(S_D^*, I_D^*, S_L^*, V_L^*, I_L^*, E^*\right)$ is globally asymptotically stable.

4. Numerical simulations

In this section, numerical simulation is used to further illustrate the effects of sheep on the spread of CE.

4.1. Sensitivity analysis and uncertainty analysis of R_0

First, we fit the parameters of the model based on the statistical yearbook of Ningxia Hui Autonomous Region [31] and surveillance indicators of echinococcosis in Pengyang County, Ningxia Hui Autonomous Region, from 2011 to 2018 [32]. The monitoring indicators included the infection rate of sheep (the ratio of infected sheep to the total number of tested sheep) and the infection rate of dogs (the ratio of canine antigen positive feces to tested feces). Some parameters in the model were assumed based on actual conditions, and other parameters were selected from the literature. The biological significance and values of parameters in the model are shown in Table 1. The software used in this study was Mathematica.



(a) (b)

Figure 3. Time plots of sheep and dog infection rates in Pengyang County. The initial values of system (2.2) are $S_D(0) = 0.9463$, $I_D(0) = 0.0537$, $S_L(0) = 0.8587$, $V_L(0) = 0.1$, $I_L(0) = 0.0413$, $E(0) = 40$.

Table 1. Parameters, biological significance and values for system (2.2).

Parameter	Biological significance	Value	Basis
A_1	Annual crop of newborn puppies	0.08	Calculation
β_1	Transmission rate from sheep to dog	0.4 (0, 0.6)	Fitting
d_1	Dog natural mortality rate	0.08	[14]
σ	Recovery rate of infected dogs	0.74 (0, 2)	Fitting
b_2	Annual crop of newborn sheep	0.8	Assumption
β_2	Transmission rate from parasite eggs to sheep	0.00085 (0, 0.003)	Fitting
d_2	Natural mortality rate of sheep	0.152	[16]
θ	The slaughter rate of sheep	0.49 (0, 0.648)	[31]
d_e	Parasite egg mortality rate	10.42	[14]
ω	Vaccination rate of sheep	0.65 (0, 1)	Fitting
δ	Vaccination failure rate of sheep	0.153 (0, 1)	[18]
K	The maximum environmental capacity of sheep	4.5	Assumption
b_e	Number of eggs laid by each adult per unit time	560	[8]
q	Average annual amount of parasites in each dog	42	[33]
d	Adult mortality in dogs	12/5	[33]
t_d	Average life span of a dog	5	[33]

Note: The release rate of eggs in the environment $\alpha = b_e q\left(1 - e^{-dt_d}\right)/d = 9799$.

Based on the parameter values in Table 1, the infection rates of sheep and dogs in Pengyang County

from 2011 to 2018 are shown in Figure 3. Figure 3 shows that the fitting value is basically consistent with the actual value. The correlation coefficient R^2 in the statistical index is often used to measure the accuracy of fitting. The closer the value is to 1, the better the fitting effect. In this paper, the R^2 values obtained by using the parameters in Table 1 are 0.9699 and 0.9905, respectively, which proves that the fitting effect meets the requirements. DISO values (distance between indices of simulation and observation) can easily and quantitatively obtain the accuracy between models. Models with small DISO values indicate high overall performances, and vice versa [34]. In this study, $DISO_{sheep} = 0.0438$ and $DISO_{dog} = 0.05488$; the DISO values are close to 0, which also indicates a good fitting effect. According to the "Health Industry Standards of the People's Republic of China" issued by the National Health Commission, the prevalence of echinococcosis in livestock should be less than 1% and the infection rate of dogs should be less than 1%. According to this standard, it can be seen from Figure 3 that Pengyang County reached the national control standard in 2016. In addition, by 2030, the infection rate of sheep will be controlled to 0.0068%, and the infection rate of dogs will be controlled to 0.0058%, which is far lower than the national standard, indicating that the prevention and control measures of echinococcosis in Pengyang County are very effective.

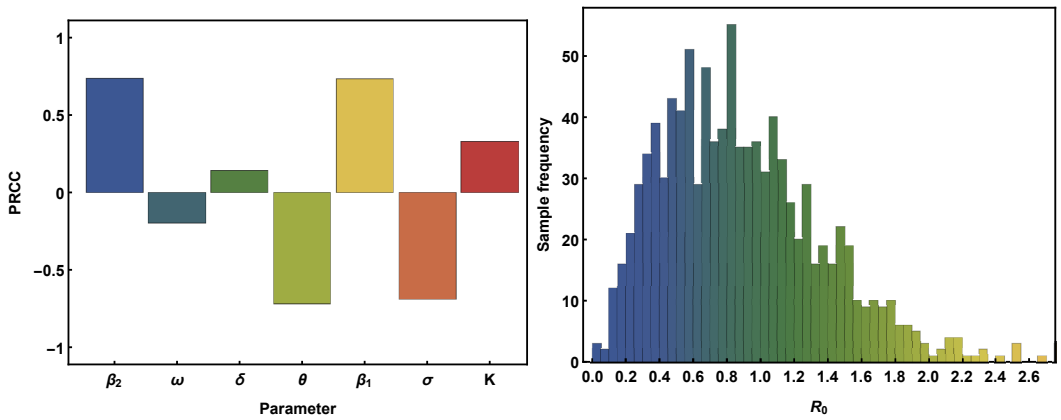


Figure 4. Sensitivity analysis and uncertainty analysis of R_0. Parameter values are shown in Table 1; all parameters are assumed to be uniformly distributed within the range.

As the theoretical results have shown, the basic reproduction number R_0 is a significant threshold for the dynamics of CE transmission. Therefore, we first present the sensitivity analysis and uncertainty analysis of R_0. We used the partial rank correlation coefficient (PRCC) method to study the sensitivity analysis of R_0, which illustrates the degree of influence of different parameters on R_0 (Figure 4). Figure 4(a) shows that R_0 has strong negative correlation with the dogs deworming recovery rate (σ) and the fraction of annual slaughtered sheep (θ). Moreover, it also illustrates that R_0 has strong positive correlation with the dog infection rate β_1 and the sheep infection rate β_2. Birhan et al. [20] also pointed out that the most sensitive parameter of the spread of CE is the transmission rate β_2. That is, control measures must be taken for dogs and sheep, and infection routes must be cut off to reduce the risk of CE. It should be noted that R_0 also has strong positive correlation with the maximum environmental capacity of sheep (K). This implies that the maximum environmental capacity of sheep is a crucial

factor affecting the spread of CE. However, this important fact has been neglected for a long time. Uncertainty analysis of R_0 was conducted by using the Latin hypercube method, and it was used to quantify the influence of parameter uncertainty on R_0 (Figure 4(b)). We assumed that the parameters follow a uniform distribution in their range as given in Table 1; then, the distribution histogram of R_0 can be given by using Mathematica software (sample number: 1000). The results show that the range of R_0 is $[0.05, 2.8]$, with a mean of 0.899 and a variance of 0.507 (Figure 4(b)). Combined with the theoretical analysis, it is possible to control the epidemic of echinococcosis and even eliminate it completely.

4.2. Contour plot of R_0

The sensitivity analysis in this paper indicates that the vaccination rate of sheep (ω), the dogs' deworming recovery rate (σ) and the sheep slaughter rate (θ) have strong negative correlations with R_0. Therefore, we constructed the contour plots of R_0 with these three parameters to better understand their respective influences on R_0. The results show that, if we want to make the basic reproduction number less than 1 ($R_0 \leq 1$), we can increase the dogs' deworming recovery rate to equal or be more than 0.185 ($\sigma \geq 0.185$) (Figure 5(a)) or increase the slaughter rate of sheep to equal or be more than 0.15 ($\theta \geq 0.15$) (Figure 5(b)) when the vaccination rate of sheep is equal to 0.65 ($\omega = 0.65$). It also shows that we should increase the dogs' deworming recovery rate to decrease the value of the basic reproduction number to less than 1 if we fix the slaughter rate of sheep, and vice versa (Figure 5(c)). Figure 5 also shows the effect of control patterns, with an emphasis on canine deworming and a secondary focus on sheep immunization, on preventing and controlling the spread of CE. It also shows the important role of the slaughter rate of sheep on the spread of CE, which is always ignored in the theoretical studies.

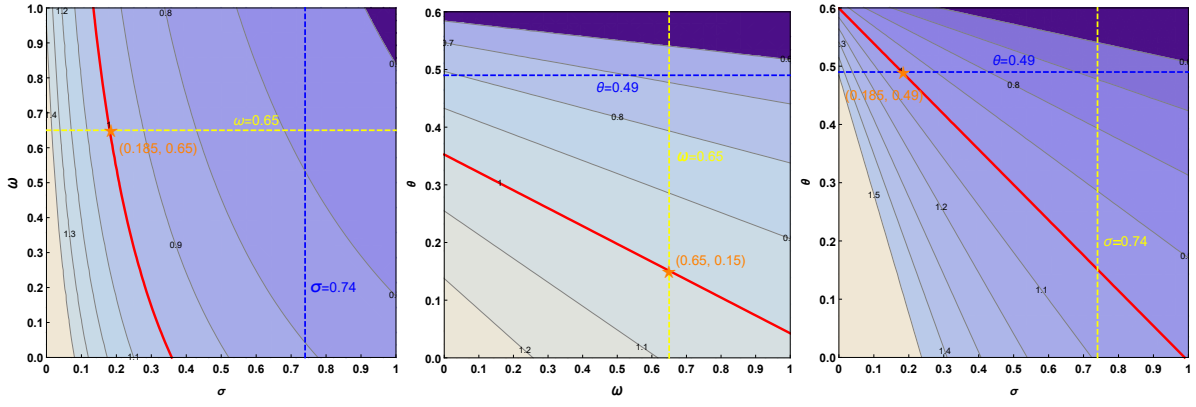
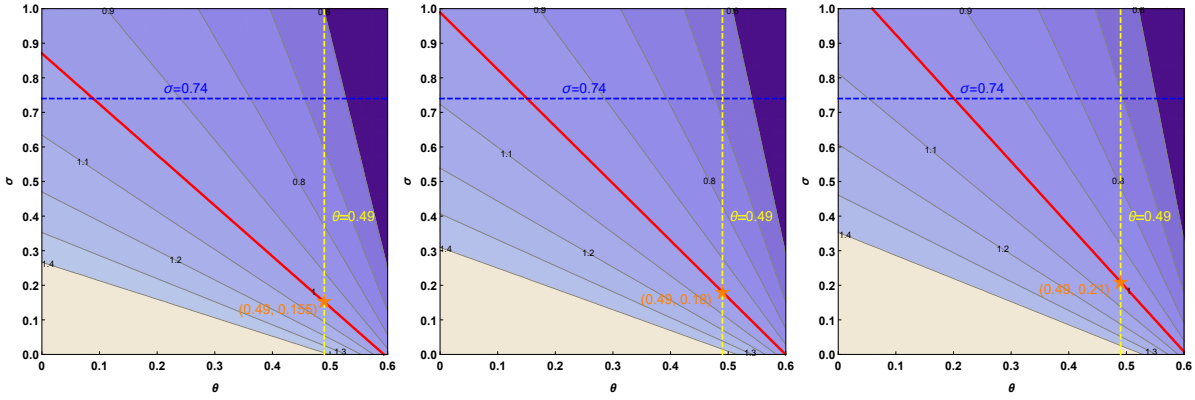


Figure 5. Contour plots of R_0: (a) the (σ, ω) plane, (b) the (ω, θ) plane and (c) the (σ, θ) plane. Other parameters are shown in Table 1.



(a) $K = 4$ (b) $K = 4.5$ (c) $K = 5$

Figure 6. Contour plots of R_0 at (θ, σ) plane with different maximum environmental capacities of sheep, K. Other parameters are the same as in Table 1.

Except the transmission rates β_1, β_2 that have strong positive correlations with R_0, the maximum environmental capacity of sheep, K, also has strong correlation with R_0. In former studies, researchers usually assumed the total number of sheep to be a constant [14–16,20,21]. There is a lack of studies that investigate how K affects the spread of CE. Combined the slaughter rate of sheep (θ) and the deworming recovery rate of infectious dogs (σ) are the main factors that influence the value of R_0 (Figures 4 and 5). We also show the contour plots of R_0 at the (θ, σ) plane when the maximum environmental capacity of sheep, K, is different (Figure 6). Figure 6 shows that both the sheep slaughter rate and the dogs deworming recovery rate increase as the the maximum environmental capacity of sheep increases if we want to keep $R_0 = 1$. For example, if we fix $\theta = 0.49$, the value of σ increases from 0.155 to 0.18 and then to 0.21 when K respectively increases to 4.5 and 5 from 4. Similar results can be obtained if we fix σ. That is, the premise of expanding the number of sheep is to increase the deworming strength for dogs and the slaughter rate of sheep.

4.3. Time plots of infected hosts

Figure 7 shows that the number of sheep tends to be stable over time, and that, when the maximum environmental capacity of sheep increases, the growth rate of the sheep population becomes faster. Time plots of infected sheep I_l and infected dogs I_d (Figure 8(a),(b)) indicate that, if we only expand the number of sheep without any additional control measures, reducing the sheep and dog infection rates below national standards takes longer and is more difficult to control. Due to the sheep slaughter rate having strong correlation with R_0, here, we evaluate the effect of the sheep slaughter rate on CE. As shown in Figure 8(c),(d), when the slaughter rate of sheep increases, it becomes less difficult to reduce the sheep and dog infection rates below the national standard. When $K = 9$, that is, when the number of sheep increases, the control difficulty increases. At this time, the slaughter rate of sheep increases to 1.25 times and 1.5 times the original rate ($\theta = 0.49$); then, the time to control the sheep infection rate is significantly shortened. The results show that the premise of increasing sheep size is to increase the slaughter rate of sheep at the same time, which could effectively control the transmission

of CE and reduce the difficulty of controlling CE transmission.

Finally, in order to evaluate the priorities of CE prevention and control in a region with different breeding scales, we analyzed the effects of different intensities of dog deworming and sheep immunization measures on CE prevention and control. When $K = 4.5$, the original dog deworming rate ($\sigma = 0.74$) and sheep immunity rate ($\omega = 0.65$) are the baselines, the results are as shown by the red solid lines in Figure 9. When K increases to 9, if we do not change the strength of dog deworming and increase the vaccination rate of sheep by 1.25 and 1.5 times, and then for sheep populations, the greater the strength of sheep immunization, the shorter the time it takes to reduce sheep infection rate to below the national standard; for dog populations, there is little change in the dog infection rate (see yellow and blue dashed lines in the Figure 9). If the immunity intensity of sheep is not changed and the deworming rate of dogs is increased to 1.25 times and 1.5 times of the original, and then for sheep populations, the greater the deworming rate of dogs, the less difficult it is to reduce the infection rate of sheep to below the national standard; for dog populations, the increase of the deworming intensity of dogs leads to a rapid decline in the infection rate of dogs; it significantly reduces the time required to control the infection rate in dogs (see pink and purple dashed lines in Figure 9). This indicates that when the number of sheep increases, improving dogs deworming rate allows for better control of the transmission of CE. If the dog deworming strength and sheep immunity strength are increased at the same time, the infection rates of sheep and dogs and the time required to control CE are reduced more quickly (see gray and cyan dashed lines in Figure 9). The results indicate that the dog deworming rate is more effective in controlling echinococcosis than the sheep vaccination rate, which theoretically verified the rationality of the control strategy of placing an emphasis on canine deworming and a secondary focus on sheep immunization to prevent and control the spread of CE.

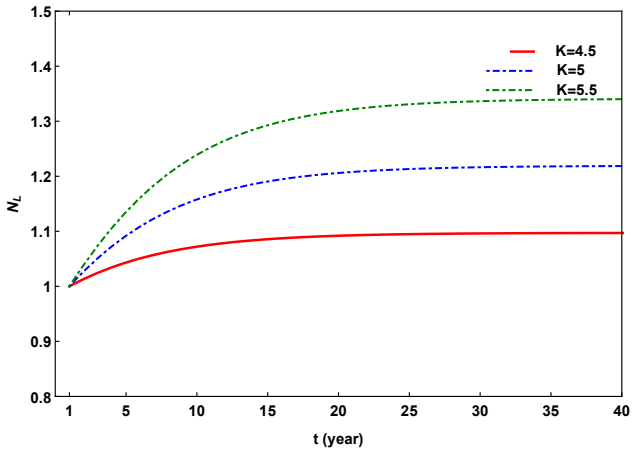


Figure 7. Time plot of N_L with different maximum environmental capacities K. Other parameters are the same as in Table 1.

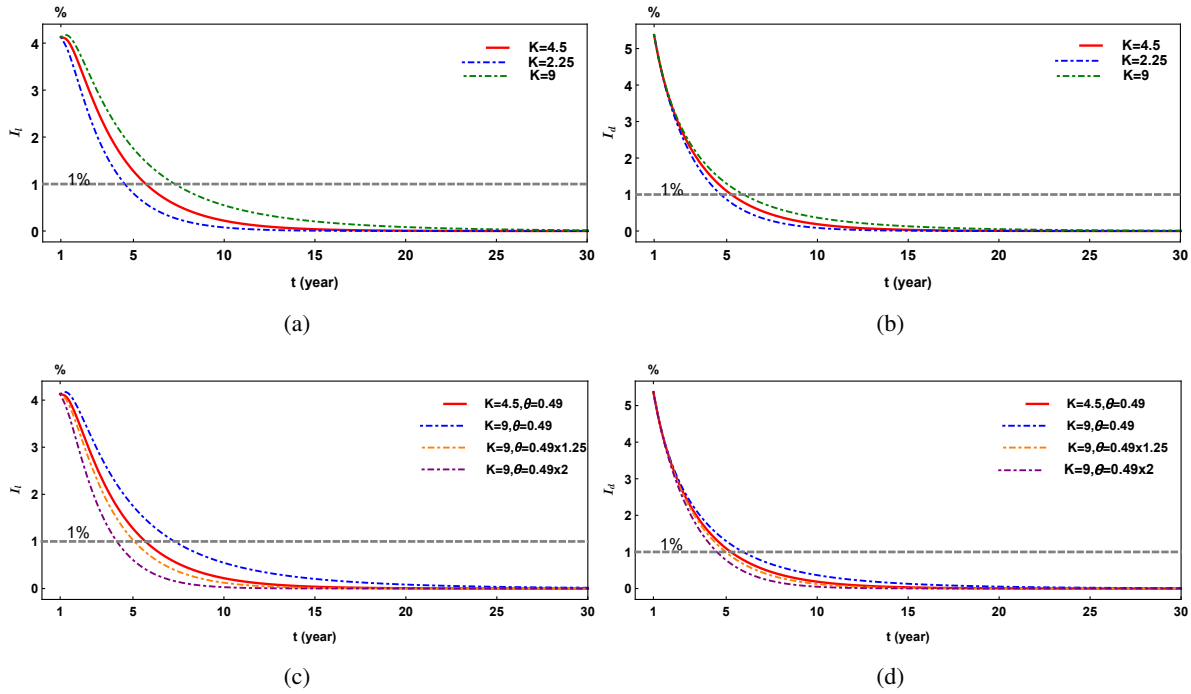


Figure 8. Time plots of I_l and I_d. Other parameters are the same as in Table 1.

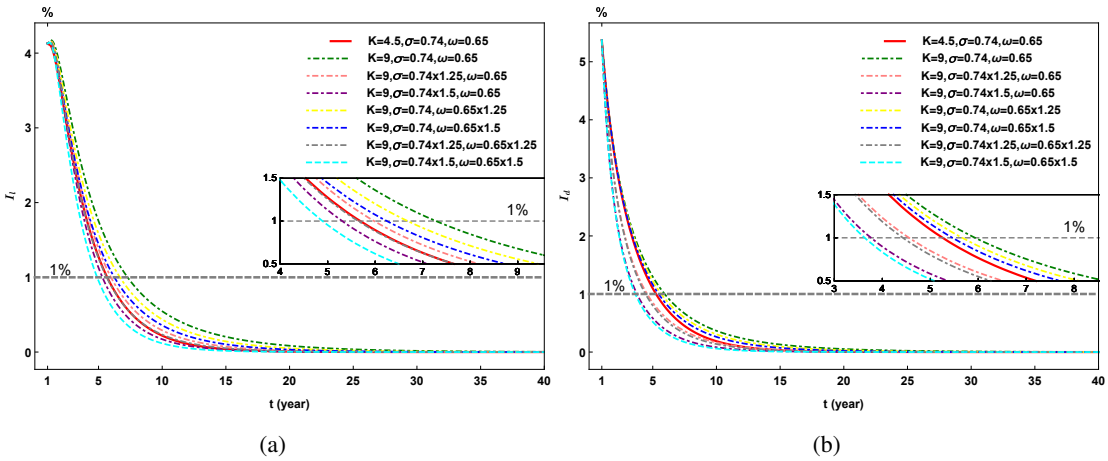


Figure 9. Time plots of I_l and I_d under different efforts of dog deworming and sheep immunity control.

5. Conclusions

In this study, we attempted to reveal the influence of sheep in the prevention and control of CE. Based on the mechanisms for transmission of CE among dogs, sheep and eggs in the environment, we

established a mathematical model and then analyzed its global dynamical behaviors. We computed the basic reproduction number of the model and obtained that the transmission of CE is mainly determined by R_0. The results showed that, when $R_0 < 1$, the disease-free equilibrium E_{dfe} is globally asymptotically stable, and that, when $R_0 > 1$, the endemic equilibrium E_e^* is globally asymptotically stable. That is, when $R_0 < 1$, the disease gradually dies out, and the disease continuous to persist if $R_0 > 1$. Sensitivity analysis and uncertainty analysis of R_0 were conducted to reveal the important influential factors of R_0. The results theoretically validate control patterns with an emphasis on dog deworming and a secondary focus on sheep immunization aimed at preventing and controlling CE (Figure 4). Figure 4 also indicated that we cannot ignore the effect of the slaughter rate of sheep and the maximum environmental capacity of sheep in the control of CE. Contour plots of R_0 further illustrated that the important roles of the slaughter rate of sheep and dog deworming on the spread of CE (Figure 5). In addition, with an increasing number of sheep, Figure 6 showed that we should increase the sheep slaughter rate and the deworming rate of dogs to control CE. That is, the risk of CE will be underestimated if we do not consider the increasing flock of sheep.

Use of AI tools declaration

The authors declare that they have not used artificial intelligence tools in the creation of this article.

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Conflict of interest

The authors declare that they have no competing interests.

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