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Research article

Modeling and analysis of Cystic Echinococcosis epidemic model with health education

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Abstract: The prevention and control of the spread of Cystic Echinococcosis is an important public health issue. Health education has been supported by many governments because it can increase public awareness of echinococcosis, promote the development of personal hygiene habits, and subsequently reduce the transmission of echinococcosis. In this paper, a dynamic model of echinococcosis is used to integrate all aspects of health education. Theoretical analysis and numerical model fitting were used to quantitatively analysed by the impact of health education on the spread of echinococcosis. Theoretical findings indicate that the basic reproduction number is crucial in determining the prevalence of echinococcosis within a given geographical area. The parameters of the model were estimated and fitted by using data from the Ningxia Hui Autonomous Region in China, and the sensitivity of the basic reproduction number was analysed by using the partial rank correlation coefficient method. These findings illustrate that all aspects of health education demonstrate a negative correlation with the basic reproduction number, suggesting the effectiveness of health education in reducing the basic reproduction number and mitigating the transmission of echinococcosis, which is consistent with reality. Particularly, the basic reproduction number showed a strong negative correlation with the burial rate of infected livestock (b) and the incidence of infected livestock viscera that is not fed to dogs (q). This paper further analyzes the implementation plan for canine deworming rates and sheep immunity rates, as well as the transmission of infected hosts over time under different parameters b and q. According to the findings, emphasizing the management of infected livestock in health education has the potential to significantly reduce the risk of echinococcosis transmission. This study will provide scientific support for the creation of higher quality health education initiatives.

Keywords: Cystic Echinococcosis; mathematical model; basic reproduction number; health education; prevention and control

Mathematics Subject Classification: 93D05, 90C31

1. Introduction

Cystic Echinococcosis (CE) is one of the most widespread parasitic diseases in China, especially in western China [1,2]. It is estimated that approximately 380,000 people are affected by Echinococcoses, and 50 million are at risk of infection nationwide [3]. The transmission of CE seriously threatens the health of farmers and herdsmen and hampers the development of local animal husbandry. Moreover, it is also considered as a disease that can drive farmers and herdsmen into poverty or return them to poverty [4].

CE always in remote rural areas and urban slums across the world. These places most often have no safe drinking water, poor sanitation and limited access to basic health care and it is impossible to control CE just relying on treatment [5]. The most important means of prevention is to change people's attitude towards CE from the way of thinking and living habits, and then control its root cause. Actually, health education can popularize knowledge, modify attitudes, and change behaviors among the populations [6]. Regarding history, Iceland's success in eradicating CE relied heavily on health education [7, 8]. Recently, diverse health education products have been designed and applied in the control of CE in China, and some of them have garnered first-class awards [2, 9]. For example, the health education film *Galsang Flowers in Blossom* depicts the journey of a big family of Tibetan compatriots who did not know about Echinococcosis as they learned with the help of the government. It showed that this film does play a positive role in improving the knowledge, attitude, and behavior intention of CE prevention among the high-risk groups [10, 11].

This paper highlights the importance of mathematical modeling and operations research in the understanding of the dynamics of the transmission of CE, and, in particular, of the impact of health education. The proposed study was designed to contribute to existing knowledge by comprehensively analysing the mathematical model and providing insights that can inform effective CE control and prevention strategies. Compartmental modelling is a powerful tool for the qualitative and quantitative analysis of the dynamics of the transmission of infectious diseases. Based on the mechanism of disease transmission, these models typically divide the population into different compartments [12–16]. Several mathematical models have been developed to study the transmission dynamics and control of CE, such as [17–26]. Wang et al. [27] proposed a deterministic model centered around dogs, livestock, humans and eggs in the environment to study the dynamics of the transmission of echinococcosis in Xinjiang. He et al. [28] formulated a mathematical model analyzing livestock and dogs to reveal the effect of increasing the sheep number. Sun et al. [29] studied the dynamics of stochastic echinococcosis infection with environmental noise. They presented the sufficient condition for ergodic stochasticity. However, there are very few models that have been developed to examine the impact of health education on the spread of CE. Rong et al. [30, 31] studied the potential role of free-roaming dogs in transmitting echinococcosis and provided effective measures to control free-roaming dogs. Zhang and Xiao [32] proposed an impulsive intervention mathematical model with periodic transmission to describe multi-host echinococcosis transmission dynamics and explore the effectiveness of control and prevention measures. Their findings suggest that key strategies for controlling the spread of echinococcosis in

humans include culling wild dog populations and implementing environmental sanitation.

Based on the works of Wang et al. [27], Rong et al. [30] and Zhao et al. Zhao and Yang [24] in the field of mathematical modeling, our study formulated a compartmental model of echinococcosis with health education to find effective health education products that can control the spread of echinococcosis. We have extended the existing model to include a death compartment and incorporated the most important aspects of health education. This allows us to qualitatively assess the impact of health education on CE transmission dynamics. The research directly addresses a critical issue in zoonoses: CE control. CE remains a global burden for several countries, and understanding its dynamics through analysis is in line with the interests of prevention and control of parasitic diseases.

To the best of our knowledge, this is the first study to consider the combination of the death compartment and the main aspects of health education on CE. Our main objectives are to assess the impact of health education, and to investigate how health education influences the transmission dynamics of CE. This research represents a significant advance the understanding of the dynamics of CE and may contribute to the development of more effective strategies to control the spread of the disease. The rest of the paper is organized as follows: A CE model with health education and control measures will be formulated in the next section. The existence and stability of equilibria will be analyzed in Section 3, and numerical simulations are given in Section 4. The discussion of our theoretical and numerical results will be presented in the final section.

2. Model formulation

CE is a multi-host parasitic disease, which is produced by Echinococcus granulosus. In Echinococcus granulosus's life cycle (Figure 1 (left)), most adult worms inhabit the small intestine of dogs; the *E. granulosus* eggs produced by an adult worm are released through the feces of dogs, ultimately polluting the water source, grassland, and so on; after viable *E. granulosus* eggs are ingested by a suitable host (e.g., under natural conditions: sheep, goat, and other livestock; under contingent condition: human), then the oncosphere emerges from the egg, penetrates the intestinal mucosa, and reaches different organs via the bloodstream, mainly, the liver and lungs. In the organ tissues, the oncosphere develops into a cystic larva. The cycle is completed when the dog eats the infected organs of dead livestock. It is obvious that *E. granulosus* eggs in the environment are an integral part of the life cycle of *Echinococcus granulosus*. Moreover, in the life cycle of *Echinococcus granulosus*, dogs are considered as the definitive host, and livestock and humans are considered as the intermediate hosts. It is worth noting that the human being, as an intermediate host of the accident, does not take part in the spread of the CE.

By referencing the modeling ideas of existing works [24, 27, 30], in this paper, we divide definitive host dogs into two classes (susceptible dogs S_d and infected dogs I_d), intermediate host livestock into four classes (susceptible livestock S_l, vaccinated livestock V_l, infected livestock I_l, and dead livestock D_l), and humans into three classes (susceptible human S_h, exposed human E_h, and infected human I_h); we also denote the *E. granulosus* eggs in the environment as x.

In the model, the probability that one person is infected by ingesting a viable *E. granulosus* egg is assumed to be β and the rate of ingestion of viable *E. granulosus* eggs by a person is assumed to be r. Then, the transmission rate for a person who ingests an *E. granulosus* egg should be $\beta \cdot r$, which is simply denoted as β_h. Similarly, β_l is the transmission rate of an *E. granulosus* egg via ingestion by

livestock.

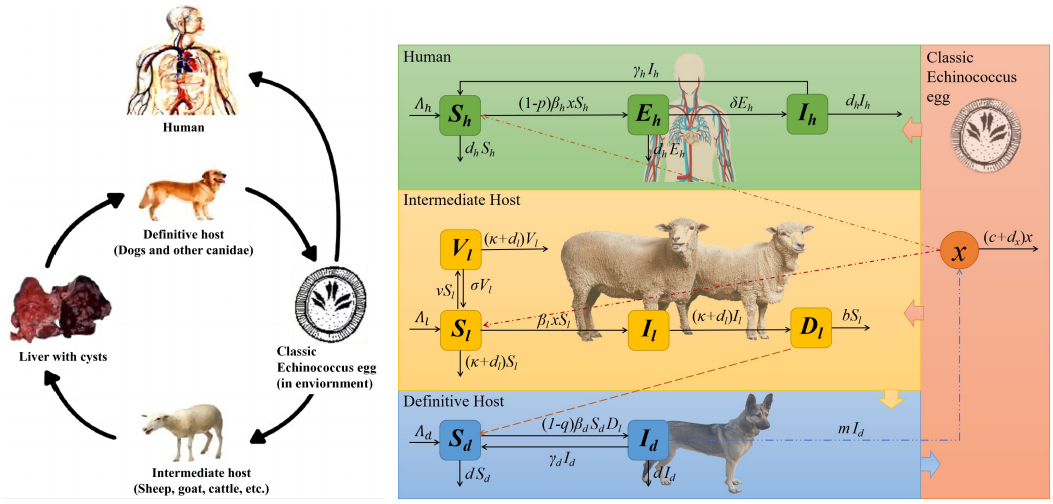

Figure 1. Life cycle of *Echinococcosis granulosus* (left) and transmission diagram of CE among dogs, livestock, and humans (right).

In reality, health education primarily involves assisting individuals in the practice of good hygiene (e.g., washing hands before meals, consuming boiled water, eating thoroughly cooked meat) and proper disposal of livestock carcasses (e.g., through burial or incineration, or by not feeding them to dogs) [10, 33]. To better incorporate these parts of health education in our model, we make the following assumptions for each host:

First, health education can help people to form good health habits such as, washing hands before meals or after playing with dogs or cats, drinking boiled water, and so on. Good health habits cut off the contact between humans and *E. granulosus* eggs; that is, good health habits can decrease the infection rate for humans. If we denote coefficient of influence of health education on people as p, then the transmission rate from *E. granulosus* eggs to humans is $(1 - p)\beta_h$.

Second, health education helps people to develop the habit of deeply burying the livestock carcasses, especially the internal organs of dead livestock. To better describe this situation, we chose to add a dead compartment class (D_l) in the model, which is considered in a CE model for the first time, and it is reasonable and necessary, as it could better understand the effect of livestock carcass disposal. If we denote b as the buried rate of livestock carcasses, then the dead livestock D_l will reduce by bD_l per unit time. And, it is also convenient to describe the habit of not feeding dead livestock organs to dogs. As health education raises the awareness of humans who do not feed livestock carcasses to dogs. This decreases the transmission rate from dead livestock to susceptible dogs. If we denote the coefficient of influence of health education on dogs as q, then the effective transmission rate from dead livestock (D_l) to susceptible dogs (S_d) is $(1 - q)\beta_d$.

Finally, dog deworming and sheep immunization, as two primary control measures, are also considered in the model. The recovery rate γ_d of dogs can reflect the degree of dog deworming. It was observed that the canine was prone to reinfection after deworming [34]; this can be modeled

by returning from compartment I_d to S_d. The vaccination rate μ and the rate of loss of immunity σ of livestock can reflect the immune sheep. As *E. granulosus* eggs can remain viable for several weeks or months in a water source and grassland, we also consider environmental disinfection in the model. Environmental disinfection can reduce the density of *E. granulosus* eggs. We denote c as the environmental disinfection rate; then, the density of eggs will reduce cx per unit time.

Based on the above assumptions and the flowchart shown in Figure 1 (right), the CE model with health education can be given by the following system of differential equations:

$$\begin{cases} \frac{dS_h}{dt} = \Lambda_h - (1-p)\beta_h x S_h + \gamma_h I_h - d_h S_h, \\ \frac{dE_h}{dt} = (1-p)\beta_h x S_h - (\delta + d_h)E_h, \\ \frac{dI_h}{dt} = \delta E_h - (\gamma_h + d_h)I_h, \\ \frac{dS_l}{dt} = \Lambda_l - \beta_l x S_l + \sigma V_l - (\nu + \kappa + d_l)S_l, \\ \frac{dV_l}{dt} = \nu S_l - (\sigma + \kappa + d_l)V_l, \\ \frac{dI_l}{dt} = \beta_l x S_l - (\kappa + d_l)I_l, \\ \frac{dD_l}{dt} = (\kappa + d_l)I_l - bD_l, \\ \frac{dS_d}{dt} = \Lambda_d - (1-q)\beta_d S_d D_l + \gamma_d I_d - dS_d, \\ \frac{dI_d}{dt} = (1-q)\beta_d S_d D_l - (\gamma_d + d)I_d, \\ \frac{dx}{dt} = mI_d - (c + d_x)x, \end{cases} \tag{2.1}$$

where Λ_h is the annual recruitment rate for human population, β_h is the transmission rate from the environment to humans, p is the coefficient of influence of health education on humans, δ is the rate of transition rate from exposed humans to infectious humans, γ_h is the treatment rate for infectious humans, d_h is the natural death rate of humans, Λ_l is the annual recruitment rate for livestock, β_l is the rate of transmission from hte environment to livestock, q is incidence of infected livestock viscera that is not fed to dogs, ν is the vaccination rate of livestock, σ is the invalid livestock vaccination rate, κ is the fraction of annual slaughtered livestock, b is the burial rate for infected livestock carcasses, d_l is the natural death rate of livestock, Λ_d is the annual recruitment rate for dogs, β_d is the rate of transmission from livestock carcasses with CE to dogs, γ_d is the deworming recovery rate for infectious dogs, d is the natural death rate of dogs, m is the release rate from infectious dogs, c is the cleaning/disinfection rate of *E. granulosus* eggs in the environment, and d_x is the natural death rate of *E. granulosus* eggs in the environment. All parameters in this paper are assumed to be non-negative.

Based on the biological meaning, we assume that all solutions of model (2.1) satisfy the following positive initial conditions:

$$\begin{array}{llll} S_h(0) = S_{h0} > 0, & E_h(0) = E_{h0} > 0, & I_h(0) = I_{h0} > 0, & \\ S_l(0) = S_{l0} > 0, & V_l(0) = V_{l0} > 0, & I_l(0) = I_{l0} > 0, & D_l(0) = D_{l0} > 0, \\ S_d(0) = S_{d0} > 0, & I_d(0) = I_{d0} > 0, & x(0) = x_0 > 0. & \end{array} \tag{2.2}$$

Let $(S_h(t), E_h(t), I_h(t), S_l(t), V_l(t), I_l(t), D_l(t), S_d(t), I_d(t), x(t))$ be any solution of model (2.1) with the initial conditions given by (2.2); using a similar argument as in [27], one can prove that $S_h(t) > 0, E_h(t) > 0, I_h(t) > 0, S_l(t) > 0, V_l(t) > 0, I_l(t) > 0, D_l(t) > 0, S_d(t) > 0, I_d(t) > 0$, and $x(t) > 0$ for all $t > 0$.

Define

$$\begin{aligned}\Gamma = \Big\{(S_h, E_h, I_h, S_l, V_l, I_l, D_l, S_d, I_d, x) \in \mathbb{R}_+^{10} : S_h + E_h + I_h \le \frac{\Lambda_h}{d_h},\\ S_l + V_l + I_l \le \frac{\Lambda_l}{\kappa + d_l}, D_l \le \frac{(\kappa + d_l)\Lambda_l}{b(\kappa + d_l)}, S_d + I_d \le \frac{\Lambda_d}{d}, x \le \frac{m\Lambda_d}{(c + d_x)d}\Big\}.\end{aligned} \tag{2.3}$$

It is not difficult to show that Γ is positively invariant with respect to model (2.1), such that any solution with positive initial values will ultimately enter Γ as $t \to +\infty$.

3. Dynamical behaviors of model (2.1)

Let all of the right-hand sides of model (2.1) equal to zero; one can calculate that model (2.1) always has a unique disease-free equilibrium $E_0 = (S_h^0, 0, 0, S_l^0, V_l^0, 0, 0, S_d^0, 0, 0)$, where

$$S_h^0 = \frac{\Lambda_h}{d_h},\ S_l^0 = \frac{\epsilon\Lambda_l}{\kappa + d_l},\ V_l^0 = \frac{(1-\epsilon)\Lambda_l}{\kappa + d_l},\ S_d^0 = \frac{\Lambda_d}{d},\ \text{and } \epsilon = \frac{\sigma + \kappa + d_l}{\nu + \sigma + \kappa + d_l}.$$

It follows from the concepts of the next-generation matrix provided by Diekmann et al. [35] and van den Driessche and Watmough [36] that the basic reproduction number can be given as

$$R_0 = \sqrt[3]{\underbrace{\frac{m}{\gamma_d + d}}_{\text{eggs by dog}} \cdot \underbrace{\beta_l \frac{1}{c + d_x}\frac{\epsilon\Lambda_l}{\kappa + d_l}}_{\substack{\text{infected sheep}\\ \text{by eggs}}} \cdot \underbrace{(1-q)\beta_d \frac{1}{b}\frac{\Lambda_d}{d}}_{\substack{\text{infected dogs}\\ \text{by sheep}}}}. \tag{3.1}$$

The basic reproduction number (R_0) denotes the expected number of secondary cases from an infected individual in the susceptible population [35]. That is, R_0 represents the number of susceptible people that an infected person can infect during the infectious period. Here, R_0 is the number of new infected human/sheep that are produced by infected dogs during the infectious period. Each infected dog releases *E. granulosus* eggs at the density $m/(\gamma_d + d)$. All susceptible livestock, i.e., $\epsilon\Lambda_l/(\kappa + d_l)$, are infected via contact with *E. granulosus* eggs at the rate β_l during the dogs' expected infectious period $1/(c + d_x)$. Moreover, the total number of dogs Λ_d/d that are infected at the rate $(1 - q)\beta_d$ via ingestion of infectious cyst-containing organs of dead livestock during the livestock's burial period $1/b$. The cube root depicts a complete transmission cycle of echinococcosis, that is, the transmission from infectious dogs to *E. granulosus* eggs, to livestock, and then to infectious dogs again.

Besides the disease-free equilibrium E_0, there also exists a unique endemic equilibrium $E^* = (S_h^*, E_h^*, I_h^*, S_l^*, V_l^*, I_l^*, D_l^*, S_d^*, I_d^*, x^*)$ when $R_0 > 1$, where

$$\begin{aligned}S_h^* &= \frac{\Lambda_h}{d_h} - (d_h + d_h\frac{\gamma_h + d_h}{\delta})I_h^*,\ E_h^* = \frac{\gamma_h + d_h}{\delta}I_h^*,\\ I_h^* &= \frac{(1-p)\beta_h\Lambda_h m\delta I_d^*}{(1-p)m\beta_h(d_h(\delta + d_h) + \gamma_h d_h)I_d^* + (c + d_x)(\delta + d_h)(\gamma_h + d_h)d_h},\end{aligned}$$

$$S_l^* = \frac{\epsilon\Lambda_l}{\kappa + d_l} - \frac{\epsilon(\kappa + d_l)}{\kappa + d_l} I_l^*,\ V_l^* = \frac{\nu}{\sigma + \kappa + d_l} S_l^*,\ I_l^* = \frac{bd(c + d_x)(\gamma_d + d)(\kappa + d_l)(R_0^3 - 1)}{(1 - q)\beta_d(\kappa + d_l)(d(c + d_x)(\kappa + dl) + m\epsilon\beta_l\Lambda_d},$$
$$D_l^* = \frac{\kappa + d_l}{b} I_l^*,\ S_d^* = \frac{\Lambda_d}{d} - I_d^*,\ I_d^* = \frac{b(c + d_x)(\gamma_d + d)(\kappa + d_l)(R_0^3 - 1)}{m\epsilon\beta_l((1 - q)\beta_d\Lambda_l + b(\gamma_d + d))},\ x^* = \frac{m}{c + d_x} I_d^*.$$

For the stability of disease-free equilibrium E_0 and endemic equilibrium E^*, we have the following results.

Theorem 1. *If $R_0 < 1$, then the disease-free equilibrium E_0 of model* (2.1) *is globally asymptotically stable in* Γ.

Proof. The Jacobian matrix at E_0 is

$$J(E_0) = \begin{bmatrix} J_1 & J_2 \\ 0 & J_3 \end{bmatrix},$$

where

$$J_1 = \begin{bmatrix} -d_h & 0 & \gamma_h \\ 0 & -(\delta + d_h) & 0 \\ 0 & \delta & -(\gamma_h + d_h) \end{bmatrix},\quad J_2 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & -(1 - p)\beta_h\frac{\Lambda_h}{d_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & (1 - p)\beta_h\frac{\Lambda_h}{d_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$J_3 = \begin{bmatrix} -(\nu + \kappa + d_l) & \sigma & 0 & 0 & 0 & 0 & -\beta_l\frac{\epsilon\Lambda_l}{\kappa + d_l} \\ \nu & -(\sigma + \kappa + d_l) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\kappa + d_l) & 0 & 0 & 0 & \beta_l\frac{\epsilon\Lambda_l}{\kappa + d_l} \\ 0 & 0 & \kappa + d_l & -b & 0 & 0 & 0 \\ 0 & 0 & 0 & -(1 - q)\beta_d\frac{\Lambda_d}{d} & -d & \gamma_d & 0 \\ 0 & 0 & 0 & (1 - q)\beta_d\frac{\Lambda_d}{d} & 0 & -(\gamma_d + d) & 0 \\ 0 & 0 & 0 & 0 & 0 & m & -(c + d_x) \end{bmatrix}.$$

□

The corresponding characteristic equation is

$$\Phi(\lambda) := (\lambda + d_h)(\lambda + \gamma_h + d_h)(\lambda + \delta + d_h)(\lambda + d)(\lambda + \kappa + d_l) \times(\lambda + \nu + \sigma + \kappa + d_l)(\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4) = 0,$$

where

$$a_1 = \kappa + d_l + b + \gamma_d + d + c + d_x > 0,$$
$$a_2 = (\kappa + d_l)(b + \gamma_d + d + c + d_x) + b(\gamma_d + d + c + d_x) + (\gamma_d + d)(c + d_x) > 0,$$
$$a_3 = (\kappa + d_l)b(\gamma_d + d + c + d_x) + (b + \kappa + d_l)(\gamma_d + d)(c + d_x) > 0,$$
$$a_4 = (\kappa + d_l)(\gamma_d + d)(c + d_x)b(1 - R_0^3).$$

When $R_0 < 1$, direct calculation yields

$$H_2 := a_1a_2 - a_3 = (b + \kappa + d_l)(b + \gamma_d + d + c + d_x)(\gamma_d + d + c + d_x + \kappa + d_l) + (\gamma_d + d + c + d_x)(\gamma_d + d)(c + d_x) > 0,$$
$$H_3 := a_3(a_1a_2 - a_3) - a_1^2a_4 > (\kappa + d_l + b)(\gamma_d + d + c + d_x)(\gamma_d + d)^2(c + d_x)^2 > 0.$$

Meanwhile, $H_1 := a_1 > 0$ and $H_4 := a_4H_3 > 0$. Therefore, by Routh-Hurwitz criteria, all roots of $\Phi(\lambda)$ have negative real parts; hence E_0 is locally stable.

Notice that the first three equations of model (2.1) are independent of the remaining equations; we then split it up into two subsystems:

$$\begin{cases} \dfrac{dS_h}{dt} = \Lambda_h - (1-p)\beta_h x S_h + \gamma_h I_h - d_h S_h, \\ \dfrac{dE_h}{dt} = (1-p)\beta_h x S_h - (\delta + d_h)E_h, \\ \dfrac{dI_h}{dt} = \delta E_h - (\gamma_h + d_h)I_h, \end{cases} \tag{3.2}$$

and

$$\begin{cases} \dfrac{dS_l}{dt} = \Lambda_l - \beta_l x S_l + \sigma V_l - (\nu + \kappa + d_l)S_l, \\ \dfrac{dV_l}{dt} = \nu S_l - (\sigma + \kappa + d_l)V_l, \\ \dfrac{dI_l}{dt} = \beta_l x S_l - (\kappa + d_l)I_l, \\ \dfrac{dD_l}{dt} = (\kappa + d_l)I_l - bD_l, \\ \dfrac{dS_d}{dt} = \Lambda_d - (1-q)\beta_d S_d D_l + \gamma_d I_d - dS_d, \\ \dfrac{dI_d}{dt} = (1-q)\beta_d S_d D_l - (\gamma_d + d)I_d, \\ \dfrac{dx}{dt} = mI_d - (c + d_x)x. \end{cases} \tag{3.3}$$

To proof the global attractivity of E_0, we first consider subsystem (3.3). Let $g(x) = x - 1 - \ln x$, $x > 0$. One can easily verify that $g(x) \geq 0$ with $g(x) = 0$ if and only if $x = 1$ and $(x-1)(y-1) = g(x) + g(y) - g(xy)$ for any $x, y \in \mathbb{R}_+$. Define

$$V_1(t) = S_l^0 g\Big(\frac{S_l}{S_l^0}\Big) + V_l^0 g\Big(\frac{V_l}{V_l^0}\Big).$$

Differentiating V_1 along subsystem (3.3) and using S_l^0 and V_l^0 is the solution of subsystem (3.3); simple calculation implies that

$$\begin{aligned} \frac{dV_1(t)}{dt}\Big|_{(3.3)} &= \Big(1 - \frac{S_l^0}{S_l}\Big)S_l' + \Big(1 - \frac{V_l^0}{V_l}\Big)V_l' \\ &= \Big(1 - \frac{S_l^0}{S_l}\Big)\Big[- \beta_l x S_l + \sigma V_l^0\Big(\frac{V_l}{V_l^0} - 1\Big) - (\nu + \kappa + d_l)S_l^0\Big(\frac{S_l}{S_l^0} - 1\Big)\Big] \\ &\quad + \Big(1 - \frac{V_l^0}{V_l}\Big)\Big[\nu S_l^0\Big(\frac{S_l}{S_l^0} - 1\Big) - (\sigma + \kappa + d_l)V_l^0\Big(\frac{V_l}{V_l^0} - 1\Big)\Big] \\ &= -\beta_l x S_l + \beta_l x S_l^0 - (\kappa + d_l)g\Big(\frac{S_l}{S_l^0}\Big) - (\kappa + d_l)g\Big(\frac{V_l}{V_l^0}\Big) \\ &\quad - \Lambda_l g\Big(\frac{S_l^0}{S_l}\Big) - \sigma V_l^0 g\Big(\frac{S_l^0 V_l}{S_l V_l^0}\Big) - \nu S_l^0 g\Big(\frac{S_l V_l^0}{S_l^0 V_l}\Big). \end{aligned} \tag{3.4}$$

Let $(S_l(t), V_l(t), I_l(t), D_l(t), S_d(t), I_d(t), x(t))$ be any solution of system (3.3) in Γ; then, for any $t \geq 0$, we have that $S_d(t) \leq \Lambda_d/d = S_d^0$. Define

$$V_2(t) = I_l + \frac{m\beta_l S_l^0(1-q)\beta_d S_d^0}{b(\gamma_d + d)(c + d_x)} D_l + \frac{m\beta_l S_l^0}{(\gamma_d + d)(c + d_x)} I_d + \frac{\beta_l S_l^0}{c + d_x} x.$$

Differentiating $V_2(t)$ along subsystem (3.3), one can derive

$$\begin{aligned}
\frac{dV_2(t)}{dt}\Big|_{(3.3)} &= \beta_l x S_l - (\kappa + d_l) I_l + \frac{m\beta_l S_l^0(1-q)\beta_d S_d^0}{b(\gamma_d + d)(c + d_x)}[(\kappa + d_l) I_l - bD_l] \\
&\quad + \frac{m\beta_l S_l^0}{(\gamma_d + d)(c + d_x)}[(1-q)\beta_d S_d D_l - (\gamma_d + d) I_d] + \frac{\beta_l S_l^0}{c + d_x}[mI_d - (c + d_x)x] \\
&\leq \beta_l x S_l - (\kappa + d_l) I_l + \frac{m\beta_l S_l^0(1-q)\beta_d S_d^0}{b(\gamma_d + d)(c + d_x)}[(\kappa + d_l) I_l - bD_l] \\
&\quad + \frac{m\beta_l S_l^0}{(\gamma_d + d)(c + d_x)}[(1-q)\beta_d S_d^0 D_l - (\gamma_d + d) I_d] + \frac{\beta_l S_l^0}{c + d_x}[mI_d - (c + d_x)x] \\
&= \beta_l x S_l - (\kappa + d_l) I_l + \frac{m\beta_l S_l^0(1-q)\beta_d S_d^0}{b(\gamma_d + d)(c + d_x)}(\kappa + d_l) I_l - \beta_l S_l^0 x.
\end{aligned} \tag{3.5}$$

Consider the following Lyapunov candidate function:

$$V(t) = V_1(t) + V_2(t).$$

Then, combining (3.4) and (3.5) and using the expression of R_0, we have

$$\begin{aligned}
\frac{dV}{dt}\Big|_{(3.3)} &= -\Lambda_l g\Big(\frac{S_l^0}{S_l}\Big) - (\kappa + d_l)\Big[g\Big(\frac{S_l}{S_l^0}\Big) + g\Big(\frac{V_l}{V_l^0}\Big)\Big] - \sigma V_l^0 g\Big(\frac{S_l^0 V_l}{S_l V_l^0}\Big) \\
&\quad - \nu S_l^0 g\Big(\frac{S_l V_l^0}{S_l^0 V_l}\Big) - \Big(1 - R_0\Big)(\kappa + d_l) I_l.
\end{aligned} \tag{3.6}$$

Therefore, if $R_0 \leq 1$, then $dV/dt \leq 0$ and $dV/dt = 0$ if and only if $S_l = S_l^0$, $V_l = V_l^0$, and $I_l = 0$. It is not difficult to verify that $(S_l^0, V_l^0, 0, 0, S_d^0, 0, 0)$ is the only invariant set of subsystem (3.3). By LaSalle's invariant principle, $(S_l^0, V_l^0, 0, 0, S_d^0, 0, 0)$ is globally asymptotically stable.

Now, we consider the subsystem (3.2). Since $x \to 0$ as $t \to +\infty$, it is not difficult to show that $S_h(t) \to \Lambda_h/d_h, E_h(t) \to 0$, and $I_h(t) \to 0$ as $t \to +\infty$. Hence, $(\Lambda_h/d, 0, 0)$ is attractive with respect to subsystem (3.2). Thus, according to the theory of asymptotic autonomous systems [37], the disease-free equilibrium E_0 of system (2.1) is globally asymptotically stable when $R_0 \leq 1$.

This theorem suggests that the propagation of CE can be eliminated when the basic reproduction number R_0 is less than one. In other words, it is necessary to keep the value of the basic reproduction number as less than unity to ensure the extinction of echinococcosis. The lower the value, the better the prevention and control of the CE.

Theorem 2. *If $R_0 > 1$, then the endemic equilibrium E^* of model* (2.1) *is globally asymptotically stable in* Γ.

Proof. First, we investigate the global asymptotic stability of the endemic equilibrium of subsystem (3.3). Let $g(x) = x - 1 - \ln x$ and

$$V_\sharp = \sharp^* g\Big(\frac{\sharp}{\sharp^*}\Big),$$

where $\sharp$ represents $S_l, V_l, I_l, D_l, S_d, I_d$, and x. Note that $(x-1)(1-y) = g(x)+g(y)-g(xy)$ for all $x, y \in \mathbb{R}_+$. Then, using the equilibrium equation $\Lambda_l - \beta_l x^* S_l^* + \sigma V_l^* - (\nu + \kappa + d_l)S_l^* = 0$ and differentiating V_{S_l} along subsystem (3.3), one has

$$\begin{aligned}
\frac{dV_{S_l}}{dt}\Big|_{(3.3)} &= \Big(1 - \frac{S_l^*}{S_l}\Big)S_l' \\
&= \Big(1 - \frac{S_l^*}{S_l}\Big)[-\beta_l x S_l + \beta_l x^* S_l^* + \sigma(V_l - V_l^*) - (\nu + \kappa + d_l)(S_l - S_l^*)] \\
&= \beta_l x^* S_l^*\Big(1 - \frac{S_l^*}{S_l}\Big)\Big(1 - \frac{xS_l}{x^* S_l^*}\Big) + \sigma V_l^*\Big(1 - \frac{S_l^*}{S_l}\Big)\Big(\frac{V_l}{V_l^*} - 1\Big) \\
&\quad -(\nu + \kappa + d_l)S_l^*\Big(1 - \frac{S_l^*}{S_l}\Big)\Big(\frac{S_l}{S_l^*} - 1\Big) \\
&= \beta_l x^* S_l^* g\Big(\frac{x}{x^*}\Big) - \beta_l x^* S_l^* g\Big(\frac{S_l^*}{S_l}\Big) - \beta_l x^* S_l^* g\Big(\frac{xS_l}{x^* S_l^*}\Big) + \sigma V_l^* g\Big(\frac{V_l}{V_l^*}\Big) \\
&\quad +\sigma V_l^* g\Big(\frac{S_l^*}{S_l}\Big) - \sigma V_l^* g\Big(\frac{S_l^* V_l}{S_l V_l^*}\Big) - (\nu + \kappa + d_l)S_l^*\Big[g\Big(\frac{S_l^*}{S_l}\Big) + g\Big(\frac{S_l}{S_l^*}\Big)\Big] \\
&= \beta_l x^* S_l^* g\Big(\frac{x}{x^*}\Big) - \Lambda_l g\Big(\frac{S_l^*}{S_l}\Big) - \beta_l x^* S_l^* g\Big(\frac{xS_l}{x^* S_l^*}\Big) + \sigma V_l^* g\Big(\frac{V_l}{V_l^*}\Big) \\
&\quad -\sigma V_l^* g\Big(\frac{S_l^* V_l}{S_l V_l^*}\Big) - (\nu + \kappa + d_l)S_l^* g\Big(\frac{S_l}{S_l^*}\Big).
\end{aligned} \tag{3.7}$$

Using the equilibrium equation $\nu S_l^* - (\sigma + \kappa + d_l)V_l^* = 0$ and differentiating V_{V_l} along subsystem (3.3), one can derive

$$\begin{aligned}
\frac{dV_{V_l}}{dt}\Big|_{(3.3)} &= \Big(1 - \frac{V_l^*}{V_l}\Big)V_l' \\
&= \Big(1 - \frac{V_l^*}{V_l}\Big)[\nu(S_l - S_l^*) - (\sigma + \kappa + d_l)(V_l - V_l^*)] \\
&= \nu S_l^*\Big(1 - \frac{V_l^*}{V_l}\Big)\Big(\frac{S_l}{S_l^*} - 1\Big) - (\sigma + \kappa + d_l)V_l^*\Big(1 - \frac{V_l^*}{V_l}\Big)\Big(\frac{V_l}{V_l^*} - 1\Big) \\
&= \nu S_l^* g\Big(\frac{S_l}{S_l^*}\Big) - \nu S_l^* g\Big(\frac{S_l V_l^*}{S_l^* V_l}\Big) + \nu S_l^* g\Big(\frac{V_l^*}{V_l}\Big) \\
&\quad -(\sigma + \kappa + d_l)V_l^* g\Big(\frac{V_l^*}{V_l}\Big) - (\sigma + \kappa + d_l)V_l^* g\Big(\frac{V_l}{V_l^*}\Big) \\
&= \nu S_l^* g\Big(\frac{S_l}{S_l^*}\Big) - \nu S_l^* g\Big(\frac{S_l V_l^*}{S_l^* V_l}\Big) - (\sigma + \kappa + d_l)V_l^* g\Big(\frac{V_l}{V_l^*}\Big).
\end{aligned} \tag{3.8}$$

Similarly, we have

$$\begin{aligned}
\frac{dV_{I_l}}{dt}\Big|_{(3.3)} &= \beta_l x^* S_l^* g\Big(\frac{xS_l}{x^*S_l^*}\Big) - \beta_l x^* S_l^* g\Big(\frac{xS_l I_l^*}{x^*S_l^* I_l}\Big) - (\kappa + d_l) I_l^* g\Big(\frac{I_l}{I_l^*}\Big),\\
\frac{dV_{D_l}}{dt}\Big|_{(3.3)} &= (\alpha + \kappa + d_l) I_l^* g\Big(\frac{I_l}{I_l^*}\Big) - (\alpha + \kappa + d_l) I_l^* g\Big(\frac{I_l D_l^*}{I_l^* D_l}\Big) - b D_l^* g\Big(\frac{D_l}{D_l^*}\Big),\\
\frac{dV_{S_d}}{dt}\Big|_{(3.3)} &= -\Lambda_d g\Big(\frac{S_d^*}{S_d}\Big) - (1-q)\beta_d S_d^* D_l^* g\Big(\frac{S_d D_l}{S_d^* D_l^*}\Big) + (1-q)\beta_d S_d^* D_l^* g\Big(\frac{D_l}{D_l^*}\Big)\\
&\quad + \gamma_d I_d^* g\Big(\frac{I_d}{I_d^*}\Big) - \gamma_d I_d^* g\Big(\frac{S_d^* I_d}{S_d I_d^*}\Big) - d S_d^* g\Big(\frac{S_d}{S_d^*}\Big),\\
\frac{dV_{I_d}}{dt}\Big|_{(3.3)} &= (1-q)\beta_d S_d^* D_l^* g\Big(\frac{S_d D_l}{S_d^* D_l^*}\Big) - (1-q)\beta_d S_d^* D_l^* g\Big(\frac{S_d D_l I_d^*}{S_d^* D_l^* I_d}\Big) - (\gamma_d + d) I_d^* g\Big(\frac{I_d}{I_d^*}\Big),\\
\frac{dV_x}{dt}\Big|_{(3.3)} &= m I_d^* g\Big(\frac{I_d}{I_d^*}\Big) - m I_d^* g\Big(\frac{x^* I_d}{x I_d^*}\Big) - (c + d_x) x^* g\Big(\frac{x}{x^*}\Big).
\end{aligned} \tag{3.9}$$

Then, consider the following Lyapunov candidate function:

$$\begin{aligned}
\bar{V} = \; & V_{S_l} + V_{V_l} + V_{I_l} + \frac{m\beta_l S_l^*(1-q)\beta_d S_d^*}{b(\gamma_d + d)(c + d_x)} V_{D_l}\\
& + \frac{m\beta_l S_l^*}{b(\gamma_d + d)(c + d_x)}(V_{S_d} + V_{I_d}) + \frac{\beta_l S_l^*}{c + d_x} V_x.
\end{aligned} \tag{3.10}$$

It follows from the equations $\beta_l x^* S_l^* - (\kappa + d_l) I_l^* = 0$ and $(1-q)\beta_d S_d^* D_l^* - (\gamma_d + d) I_d^* = 0$ that

$$\frac{m\beta_l S_l^*(1-q)\beta_d S_d^*}{b(\gamma_d + d)(c + d_x)} = \frac{m\frac{(\kappa + d_l) I_l^*}{x^*} \cdot \frac{(\gamma_d + d) I_d^*}{D_l^*}}{b(\gamma_d + d)(c + d_x)}.$$

And, $D_l^* = \frac{\kappa + d_l}{b} I_l^*$ and $x^* = \frac{m}{c + d_x} I_d^*$ imply that

$$\frac{m\beta_l S_l^*(1-q)\beta_d S_d^*}{b(\gamma_d + d)(c + d_x)} = 1. \tag{3.11}$$

Combining (3.7) (3.8), and (3.9) and using (3.11), we have

$$\begin{aligned}
\frac{d\bar{V}}{dt}\Big|_{(3.3)} = \; & -\Lambda_l g\Big(\frac{S_l^*}{S_l}\Big) - \sigma V_l^* g\Big(\frac{S_l^* V_l}{S_l V_l^*}\Big) - (\kappa + d_l) S_l^* g\Big(\frac{S_l}{S_l^*}\Big) - \nu S_l^* g\Big(\frac{S_l V_l^*}{S_l^* V_l}\Big)\\
& - (\kappa + d_l) V_l^* g\Big(\frac{V_l}{V_l^*}\Big) - \beta_l x^* S_l^* g\Big(\frac{xS_l I_l^*}{x^* S_l^* I_l}\Big) - \frac{m\beta_l S_l^* \Lambda_d}{(\gamma_d + d)(c + d_x)} g\Big(\frac{S_d^*}{S_d}\Big)\\
& - \frac{m\beta_l S_l^*(1-q)\beta_d S_d^*(\alpha + \kappa + d_l) I_l^*}{b(\gamma_d + d)(c + d_x)} g\Big(\frac{I_l D_l^*}{I_l^* D_l}\Big) - \frac{m\beta_l S_l^*(1-q)\beta_d S_d^* D_l^*}{(\gamma_d + d)(c + d_x)} g\Big(\frac{S_d D_l I_d^*}{S_d^* D_l^* I_d}\Big)\\
& - \frac{m\beta_l S_l^* \gamma_d I_d^*}{(\gamma_d + d)(c + d_x)} g\Big(\frac{S_d^* I_d}{S_d I_d^*}\Big) - \frac{md\beta_l S_l^* S_d^*}{(\gamma_d + d)(c + d_x)} g\Big(\frac{S_d}{S_d^*}\Big) - \frac{m\beta_l S_l^* I_d^*}{c + d_x} g\Big(\frac{x^* I_d}{x I_d^*}\Big).
\end{aligned} \tag{3.12}$$

Therefore, $d\bar{V}/dt \le 0$ and $d\bar{V}/dt = 0$ if and only if $S_l = S_l^*, V_l = V_l^*, I_l = I_l^*, D_l = D_l^*, S_d = S_d^*, I_d = I_d^*$, and $x = x^*$, that is, $(S_l^*, V_l^*, I_l^*, D_l^*, S_d^*, I_d^*, x^*)$ is the only invariant set of subsystem (3.3). It follows from LaSalle's invariant principle that $(S_l^*, V_l^*, I_l^*, D_l^*, S_d^*, I_d^*, x^*)$ is globally asymptotically stable.

The pervious proof implies that $x \to x^*$ as $t \to +\infty$. Therefore, the limiting system of subsystem (3.2) can be given as

$$\begin{cases} \dfrac{dS_h}{dt} = \Lambda_h - (1-p)\beta_h x^* S_h + \gamma_h I_h - d_h S_h, \\ \dfrac{dE_h}{dt} = (1-p)\beta_h x^* S_h - (\delta + d_h)E_h, \\ \dfrac{dI_h}{dt} = \delta E_h - (\gamma_h + d_h)I_h, \end{cases} \tag{3.13}$$

Using the Lyapunov candidate function

$$\tilde{V} = S_h^* g\Big(\frac{S_h}{S_h^*}\Big) + E_h^* g\Big(\frac{E_h}{E_h^*}\Big) + I_h^* g\Big(\frac{I_h}{I_h^*}\Big),$$

one can easily derive that $d\tilde{V}/dt|_{(3.13)} \leq 0$ and $d\tilde{V}/dt|_{(3.13)} = 0$ if and only if $S_h = S_h^*, E_h = E_h^*$, and $I_h = I_h^*$. Therefore, (S_h^*, E_h^*, I_h^*) is the only invariant set of (3.13), and it is globally asymptotically stable based on LaSalle's invariant principle. Hence, according to the theory of asymptotic autonomous systems [37], if $R_0 > 1$, the unique endemic equilibrium E^* of system (2.1) is globally asymptotically stable. □

A simple interpretation and epidemiological implication of Theorem 2 is that CE will persist among humans, livestock, and dogs when the basic reproduction number is larger than one. In other words, CE will never become extinct when $R_0 > 1$.

4. Numerical simulation

In this paper, the data were mainly taken from the Data Center of China Public Health Science (https://www.phsciencedata.cn/) and Ningxia Statistical Yearbook (NSY) (http://nxdata.com.cn/). Nevertheless, part of the data could not be acquired easily. We had to rely on published papers and our estimation, such as the number of *E. granulosus* eggs in the environment and the number of infected dogs in Ningxia Hui Autonomous Region (NHAR) in China. Wolfram Mathematica 10.2 was employed for the calculation, curve fitting, and sensitivity analysis. The values of the model parameters were estimated based on the following facts and assumptions:

(a) The number of dogs in NHAR was estimated to be 515000 in 2004 (NSY). The average life span of dogs was set as 12.5 years [27]. Thus, the natural death rate of dogs depends on the life span of dogs, with $d = 1/12.5 = 0.08$. The annual recruitment rate for dogs, Λ_d, equals the total number of dogs times the natural death rate with $\Lambda_d = 515000 \times 0.08 = 41200$. The deworming recovery rate for infectious dogs γ_d was estimated as 0.91, and the coefficient of influence q on dogs has been assumed to be 0.1.

(b) The natural death rate of sheep d_l was set as 0.152 [31]. Based on the number of livestock (only considering the sheep in this study, as sheep constitute the main livestock infected with CE) slaughtered and handled in the NSY, the fraction of the annual slaughtered rate for livestock κ was calculated as 0.49, and the annual recruitment rate for livestock Λ_l was estimated to be 7.48×10^6. According to the assumption of immune effect on livestock, the vaccination rate of livestock ν and the invalid vaccination rate among livestock σ were assumed to be 0.92 and 0.14, respectively. In addition, the burial rate b of infected livestock carcasses was set as 0.09.

(c) The life span of *E. granulosus* eggs was assumed to be 35 days [38]; then, $d_x = 365/35 = 10.42$. We supposed that the farmers perform a major disinfection once a year, which means that $c = 1/1 = 1$. Based on the assumptions in [39], the release rate for infectious dogs can be expressed as $m = \phi(h/\eta)(1 - \exp(-\eta t_d)$, where ϕ denotes the number of *E. granulosus* eggs released by adult worms per year, h is the average number of *E. granulosus* eggs in the small intestines of dogs during a one-year period, t_d represents the average life span of dogs, and η means the natural mortality rate of adult worms. From Budke et al. [40] and Torgerson and Heath [41], the *E. granulosus* egg release rate for one worm per unit time was 42, the worm production rate per dog per year was set as 560, the *E. granulosus* egg mortality rate in the dogs' body is 12/5, and the average life span of dogs was set as 5; thus, $m = 9799$.

(d) The human annual birth population in NHAR was applied as 1.57×10^5 (NSY). Then, we have that $\Lambda_h = 1.57 \times 10^5$. The average life span of people in NHAR is 76.5 years; thus, $d_h = 1/76.5$. The recovery rate γ_h was estimated to be 35%, and the coefficient of influence of health education on humans was assumed to be 0.2. The incubation period of CE can be months to years, even decades; we chose 14 years [27]; thus, $\delta = 1/14$. And, the parameters β_h, β_l, and β_d were fitted by using the data in NHAR.

For clarity, we summarize the description, default values, and references of the parameters of model (2.1) in Table 1.

Table 1. Description of model parameters, default values (ranges), and references.

Parameters	Biological descriptions	Value(Range)	Source
Λ_h	Annual recruitment rate for human population	1.57×10^5	NSY
β_h	Transmission rate from environment to humans	3.1×10^{-8}	Fitting
p	Coefficient of influence of health education on humans	0.2 (0,1)	Assumption
δ	Transition rate from exposed humans to infectious humans	1/14	[27]
γ_h	Treatment rate for infectious humans	0.35	Fitting
d_h	Natural death rate of humans	1/76.5	NSY
Λ_l	Annual recruitment rate for livestock	7.48×10^6	NSY
β_l	Transmission rate from environment to livestock	3.2×10^{-9}	Fitting
q	Incidence of infected livestock viscera that is not fed to dogs	0.1	Assumption
ν	Vaccination rate of livestock	0.92	Assumption
σ	Invalid livestock vaccination rate	0.14	Assumption
κ	Fraction of annual slaughtered livestock	0.49	NSY
b	Burial rate for infected livestock carcass	0.09	Assumption
d_l	Natural death rate of livestock	0.152	[31]
Λ_d	Annual recruitment rate for dogs	4.12×10^4	NSY
β_d	Transmission rate from livestock carcass with CE to dogs	1.33×10^{-8}	Fitting
γ_d	Deworming recovery rate for infectious dogs	0.91	Assumption
d	Natural death rate of dogs	0.08	[27]
m	Release rate for infectious dogs	9799	Calculation
c	Cleaning/disinfection rate of *E. granulosus* eggs in the environment	1	Assumption
d_x	Natural death rate of *E. granulosus* eggs in the environment	10.42	[38]

NSY: Ningxia Statistical Yearbook.

Using the parameter values in Table 1, the time plot for infected human cases in NHAR was constructed as presented in Figure 2. One can see that our model provides a relatively good match

with the reported data in NHAR. That means that our studies based on this model are rational. In what follows, we will use model (2.1) and the parameter values listed in Table 1 to investigate the effect of health education on the spread of CE.

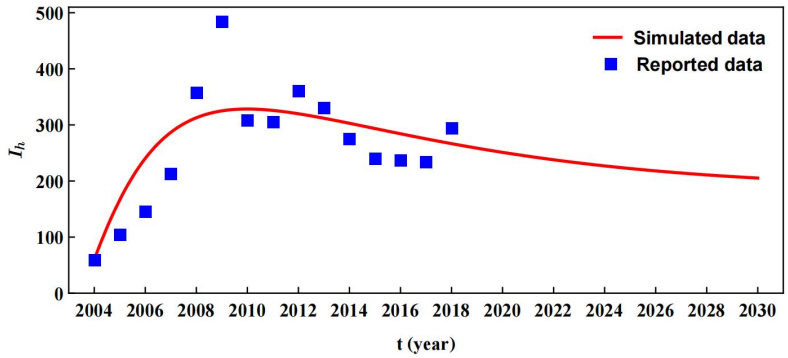

Figure 2. Simulation and prediction of human CE in NHAR. The initial conditions are $S_h(0) = 5.837\times10^6, E_h(0) = 2000, I_h(0) = 60, S_l(0) = 8.528\times10^6, V_l(0) = 2.38\times10^5, I_l(0) = 4.68\times10^5, D_l(0) = 1600, S_d(0) = 4.077\times10^5, I_d(0) = 3.121\times10^4$, and $x(0) = 3.57\times10^7$.

Notice the important role of the basic reproduction number R_0. We first use the partial rank correlation coefficient (PRCC) to identify critical parameters that have a significant impact on the value of R_0. From Figure 3, one can see that the biggest values of the PRCC for R_0 were observed for parameters q (the incidence of infected livestock viscera that is not fed to dogs) and b (burial rate for infected livestock carcasses). That means that q and b have the most significant impact on R_0. These two parameters were first considered in the CE model, and their values completely depend on health education. This implies that health education is strongly related to R_0. Figure 3 also shows that R_0 is more sensitive to γ_d (deworming recovery rate for infectious dogs) and ν (vaccination rate of livestock). This is consistent with the Chinese policy of canine deworming as primary and sheep immunization as auxiliary. Regarding the livestock vaccination, the PRCC values for ν and σ inform us that vaccination coverage plays a more significant role in preventing the prevalence of CE. Moreover, κ and c are also negatively correlated with R_0, that is, the higher the livestock slaughter rate and environmental disinfection rate, the lower the value of R_0. In all, Figure 3 shows that health education plays a positive role in reducing the values of R_0, especially education on the infected livestock carcass disposal (b and q).

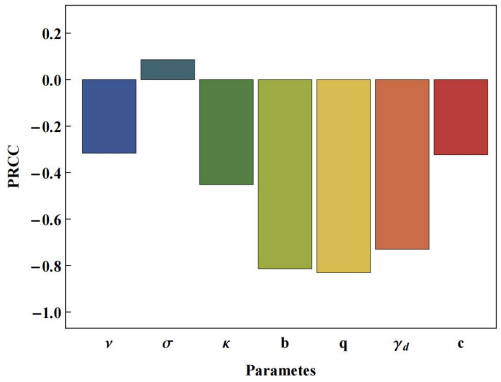

Figure 3. PRCC for the basic reproduction number R_0.

To further clarify the effects of b, q, ν, and γ_d on R_0, we further present the contour plots for R_0 from the perspectives of γ_d and ν with different values of q and b. From Figure 4(a) and (b) or Figure 4(c) and (d), we have that, once we increase the value of b by 20%, the value of γ_d sharply decreases from 0.9267 to 07589 when we fix $\nu = 0.92$ to keep $R_0 = 1$. Similar results can be obtained for ν if we fix γ_d. If we want to increase the value q from 0.1 to 0.12, Figure 4(a) and (c) tell us that the dependence on γ_d will slightly decrease if we fix ν to keep $R_0 = 1$. Similarly results can be derived from Figure 4(a) and (c) or Figure 4(b) and (d). These four plots illustrate that increases in b and q are both can independently the dependence on γ_d and ν. However, from the perspective of effects, the influence of b is more obvious. That means that deeply burying infected livestock carcasses is the most important behavior to reduce the value of R_0. Meanwhile, the time plots of Figure 5 tell us that the trends of infected humans, livestock, and dogs all indicate high correlation with b and q. The higher the values of b and q, the lower the risk for humans, livestock, and dogs. These results indicate that infected livestock carcass disposal awareness should be stressed during health education.

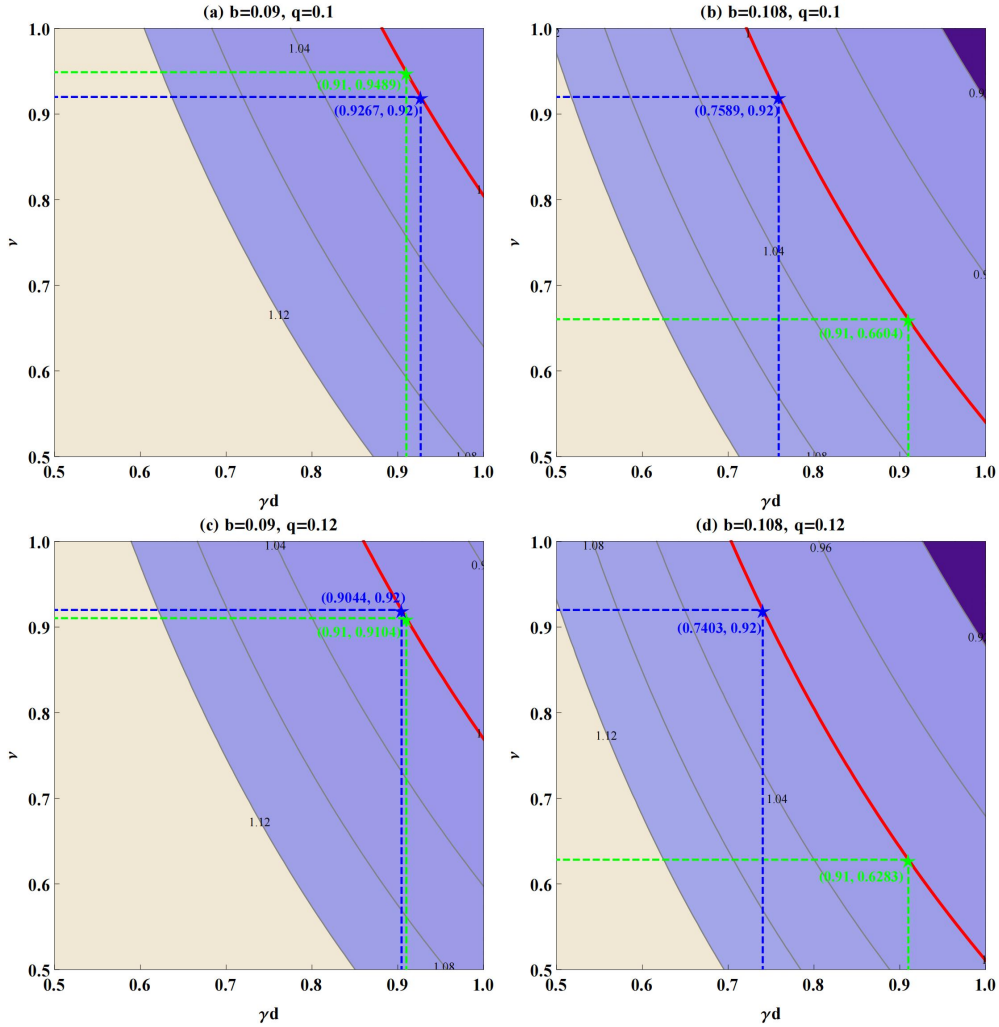

Figure 4. Contour plots for γ_d and ν with different values of b and q. The red line indicates that $R_0 = 1$, the dashed green line is the scenario of a fixed $\gamma_d = 0.91$, and the dashed blue line represents the scenario when $\nu = 0.92$. The other parameters are the same as in Table 1.

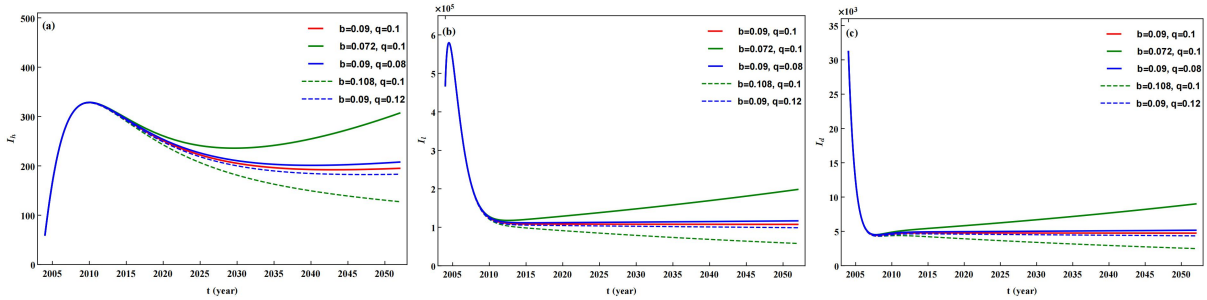

Figure 5. Time plots for I_h, I_l, and I_d with different values of b and q. The red line represents the baseline with $b = 0.09$ and $q = 0.1$. The green (dashed green) lines indicate a decrease (increase) in b by 20%, and the blue (dashed blue) lines denote a (increase) decrease in q by 20%. The other parameters are the same as in Table 1.

Finally, time plots for infected humans, livestock, and dogs with different values of p are given in Figure 6. It shows that I_h proportionally decreases with the increase of p, while the time series of I_l and I_d are steady without much change. That is because humans do not participate in the spread of CE. In the model, p reflects the actions of washing hands before meals, drinking boiled water, and eating thoroughly cooked meal. That is, if people pay attention to personal hygiene, the spread of CE in humans will be mitigated.

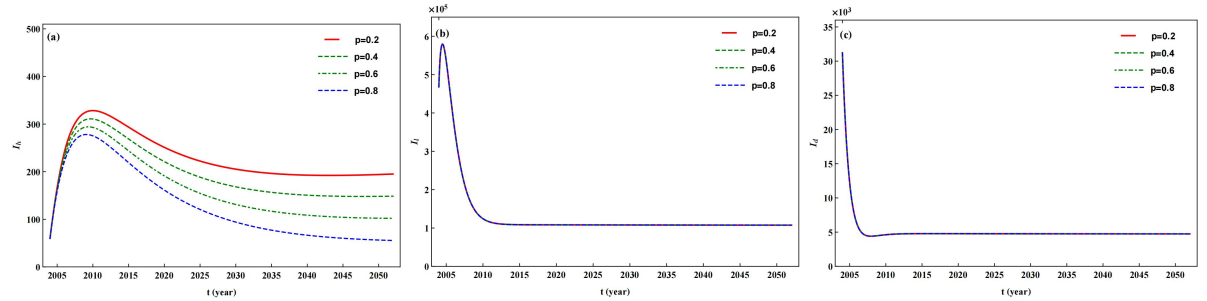

Figure 6. Time plots for I_h, I_l and I_d with different values of p. The red line represents the baseline with $p = 0.2$. The other parameters are the same as in Table 1.

5. Conclusions

CE, as one of the most important diseases in the world, is mainly distributed in the western part of China, including Ningxia, Tibet, and other western provincial capitals [42]. Health education plays a vital role in controlling and preventing CE, which can mitigate and reduce the transmission of CE. This paper presents a deterministic mathematical model that explores the dynamics of a CE model, encompassing the main aspects of health education while also incorporating dog deworming and sheep immunity into the model. The study include a rigorous analysis of the model, with a number of key findings. The dynamical behaviors indicate that the transmission of CE is mainly determined by the basic reproduction number R_0. That is, CE will persist when $R_0 > 1$, while it will die out when $R_0 < 1$. With the parameter values in Table 1, model (2.1) can mimic the reported human echinococcosis data from 2004 to 2018 in NHAR (Figure 2). It should be noted that the basic reproduction number in

NHAR is 1.0056 > 1, which means that CE is an endemic disease in NHAR.

For the sensitivity analysis of R_0, we analyzed the correlation of various parameters of R_0. The strong negative correlation between R_0 and the deworming recovery rate for dogs γ_d is consistent with the results in [30, 31]. It is interesting that R_0 has strong negative correlations with the burial rate for infected livestock carcasses b and the incidence of infected livestock viscera that is not fed to dogs q (Figures 3 and 4). This indicates that suitable disposal of infected livestock carcasses is very important for the prevention and control of the transmission of CE.

The study presented here is just a primary investigation into the effect of health education on the prevention and control of the spread of CE. The major conclusions are provided as follows.

1) The spread of CE is determined by the basic reproduction number, and the lower the value the better the ability to prevent and control the spread.

2) Health education is an effective means for the prevention of CE.

3) Infected livestock carcass disposal awareness should be stressed in the process of health education.

Various directions can be improved and generalized in the future. As in [43], we can further explore cost-effective approaches for CE. We could further explore the most effective health education products and treatment measures for CE, investigate the impact of climate change on the spread of CE, or analyze the geographical distribution of CE in China for our next study.

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

We declare that we have no financial or personal relationships with other people or organizations that could have inappropriately influenced our work.

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