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Research Article

# Mathematical modeling of Echinococcosis in humans, dogs and livestock with optimal control strategies

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**Abstract**

In this paper, we present a deterministic model of Cystic Echinococcosis disease of eleven differential equations, describing complex interactions between three types of hosts, specifically humans, livestock and dogs. The model is analyzed; disease-free and endemic equilibrium exist and are globally asymptotically stable if the basic reproduction number is less than or greater than one, respectively. The optimal control represents the efficiency of health education and EG95 sheep vaccination. The existence of optimal controls is proven, the characterization is formulated using Pontriyagin's maximum principle, and the optimal system is derived. For the numerical simulation, the data used are from some studies done in Morocco.

**AMS subject classifications (2020):** Primary 45D05; Secondary 42C10, 65G99.

**Keywords:** Cystic Echinococcosis; deterministic modeling; basic reproduction number; global stability; optimal control.

## 1 Introduction

Mathematical modeling is a branch of science that seeks to illustrate real-life problems using mathematical techniques to better understand them, predict them, and act accordingly. It presents great interest in several fields, especially epidemiology [5, 12, 16, 17].

Cystic Echinococcosis (CE), also known as hydatid disease, is a globally known parasitic disease for its serious medical and economic impacts, it is caused by a parasite called the tapeworm CE. This disease affects humans, domestic and wild animals such as dogs, cats, rodents, livestock, horses, foxes, and wolves. The transmission of the disorder goes through a cycle [27].

Indeed eggs of CE are passed in the environment in the feces of definitive hosts such as dogs. Those eggs are then ingested by intermediate hosts like sheep. Inside those, the eggs hatch and form cysts in the liver, lungs, and sometimes brain, those cysts act like tumors that can disturb the function of the organ where they are found, they can cause poor growth, reduced production of milk and meat, which has severe economic impacts. When the

infected organs of intermediate hosts are eaten by a definitive carnivore host, the cycle restarts again [25].

Humans contract the infection by ingesting parasite eggs by touching contaminated soil, water, or an infected domestic dog for example [26]. There is no direct transmission between humans and those are called to be accidental hosts of the Echinococcosis disease. For them the malady can be dangerous, threatening, and occasionally fatal. Moreover, the treatment is heavy, lengthy, and expensive. In fact, the 2015 WHO Foodborne Disease Burden Epidemiology Reference Group (FERG) predicted Echinococcosis to be the cause for 19 300 deaths and approximately 871,000 disability-adjusted life-years (DALYs) globally each year [25].

Echinococcosis commonly affects rural areas where farming is the main activity, and dogs are kept in great numbers to protect livestock and provide companionship. Their presence is important for keeping safety and managing the pastoral community. So, contact between the three populations (humans, livestock, dogs) speeds the spread of the disease, especially because of the lack of knowledge of pastoralists about how CE is transmitted, and the open access of dogs to uncooked meat or carcasses that can be infectious [20].

In Morocco, a country where many regions depend on sheep farming, Echinococcosis can be dangerous and can threaten food security, animal and human health, and even life. However, studies and states on this disease are poor in Morocco, but the last ones assumed that the disease is critically endemic in regions where livestock are highly produced (Middle Atlas, El Hajeb, Azrou, Timahdit, Ouarzazate, Tiznit-Sidi Ifni, etc.) [9, 19, 2]. Different levels of prevalence of CE are recorded in [9]: 3.6 to 71.4% for cattle, 0.1 to 81.1% in sheep, and 0 to 25.7 % in goats. Concurrently, high degrees of CE infection (up to 70%) were spotted in dogs in various regions of the country. For humans, it has been reported from a study in the Middle Atlas region that CE prevalence is 1.9% out of 5367 people examined in 2017 [6].Other studies show high infection degrees for humans and animals in Morocco [8, 2, 3, 19].

According to [20, 2], the mortality rate for CE in Morocco ranges between 2% and 3%, and the surgery and treatment of each infected patient costs approximately 17,000 and 32,000 Moroccan dirham MAD (US$ 1700 and US$ 3200), for simple and recurrence instances, respectively. Also, according

to a socio-economic study in [18] on the country's CE burden, the DALY ( Disability-adjusted life years ) is 160 years (0.5 years per 100,000 people) per year, with total annual losses of US$ 73 million (US$ 54.92 million) due to organ seizures, healthcare bills, and missed income for infected individuals, and all these stats are likely under-notified.

Many deterministic and statistical models are developed to describe and study the transmission of CE disease [4, 5, 14, 23, 24, 11], and so on. Based on these studies, in this article, we propose a developed Echinococcosis model that contains compartments representing humans, dogs, livestock, CE eggs, and infectious meat and that considers relapse after the surgery for humans and dogs. For humans, this rate ranges from 4.65% to 36% of cases in different regions of the world [22]. This recurrence causes a real problem in the management of the disease as stated by Velasco-Tirado et al. [22]. On the other hand, the optimal control theory is applied by integrating two control measures to study their effect on the spread of the disease. Therefore, in this work, based on some results from [13], we investigate the effectiveness of two prevention methods, the first is health education due to the current lack of knowledge of CE among citizens, and the second is the vaccination of livestock with an E. granulosus recombinant antigen (EG95) in Morocco [13], an approach that has been previously done in China and Argentina [25], because livestock prevention is probably the best approach to disrupt the CE life cycle.

A comparison between the current article and one of the articles cited above will be made next to show the key differences and innovation in our article.

The rest of this paper is organized as follows: In Section 2, a deterministic model is presented. Then in Section 3, the positivity and boundedness of the solutions are studied, and the basic reproduction number and the two equilibria of the model are computed. Following that we study the stability of the disease-free equilibrium (DFE) and the endemic equilibrium. Thus in Section 4, we apply the optimal control theory to the deterministic model studied. After that, in Section 5, numerical simulations are presented and discussed based on some studies carried out in Morocco. Finally, conclusion is made in Section 6.

# 2 Model formulation

## 2.1 Description of the model

To mathematically describe the spread of CE, we use a deterministic model that takes into account three types of population, humans being accidental hosts, dogs being definitive hosts, and livestock being intermediate hosts according to the diagram in Figure 1.

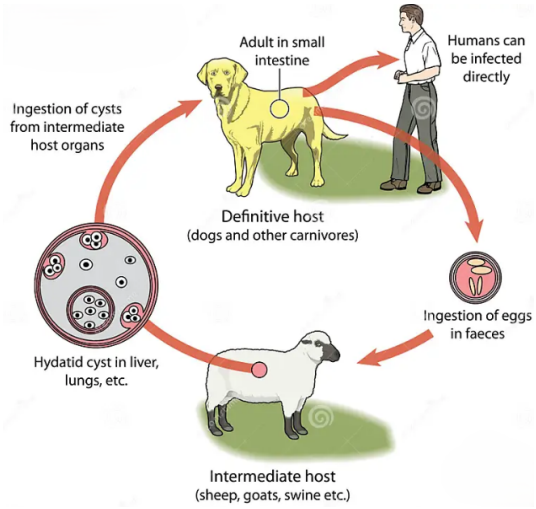


Figure 1: Life cycle of Echinococcus granulosus

**Hypothesis**

1. The human population is divided into four classes: The susceptible $H_S$, the exposed $H_E$, the infected $H_I$, and the recovered $H_R$.

   Humans act like accidental hosts, they are infected by touching the eggs of CE, due to lack of personal hygiene. Moreover, there is no direct transmission of CE disease from one human to another, and humans do not get infected from infectious meat.

   For the humans submodel, we suppose a constant recruitment rate $B_H$ due to birth or immigration into the susceptible class $H_S$, those decrease owing to exposure to CE parasite's eggs at a rate $\beta_H$ or to natural death

$\mu_{HS}$. As relapse of the disease exists even after recovery, we added a rate $\alpha_S$ that denotes the rate at which recovered individuals become susceptible again.

The exposed class $H_E$ increases at a rate $\beta_H$ and decreases when moving to the infected class $H_I$ at a rate $\sigma_H$ or due to natural death $\mu_{HE}$. Infected humans are regarded to suffer death caused by the CE disease at a rate $d_{kh}$, and natural death at a rate $\mu_{HI}$, they decrease when moving to the recovered class $H_R$ at a pace $r_H$.

Recovered class $H_R$ diminishes due to the relapse of the healed individuals into the susceptible class at a pace $\alpha_S$, or due to natural death $\mu_{HR}$.

2. The dog population is separated into three classes: The susceptible $D_H$, the infected $D_I$, and the recovered $D_R$.

   Dogs represent definitive hosts infected by consuming contaminated meat at a rate $\beta_D$, and they spread CE eggs present in their feces in the environment at a pace $B_E$. Moreover, we suppose that cured dogs can become susceptible again at a rate $\alpha_D$. The rest of the parameters of the dog submodel follow the same trend of the human population and are explained in Table 1 and Figure 2.

3. We divide livestock population into two classes: The susceptible $O_S$ and the infected $O_I$.
   Livestock population are infected by ingesting CE eggs at a rate $\beta_O$, which causes the formation of cysts in some of their organs, those cysts grow with time and stay in the infected organs. Moreover, they are slaughtered at a pace $\eta_1$ when susceptible, and $\eta_2$ when infected. The rest of the parameters are described in Table 1 and Figure 2.

4. We also integrate the infected meat compartment denoted by $V$, and it grows at a rate $\eta_2$ when contaminated sheep or cattle are slaughtered and diminishes when it is eaten by humans at a rate $\gamma$, and by dogs at a rate $\beta_D$.

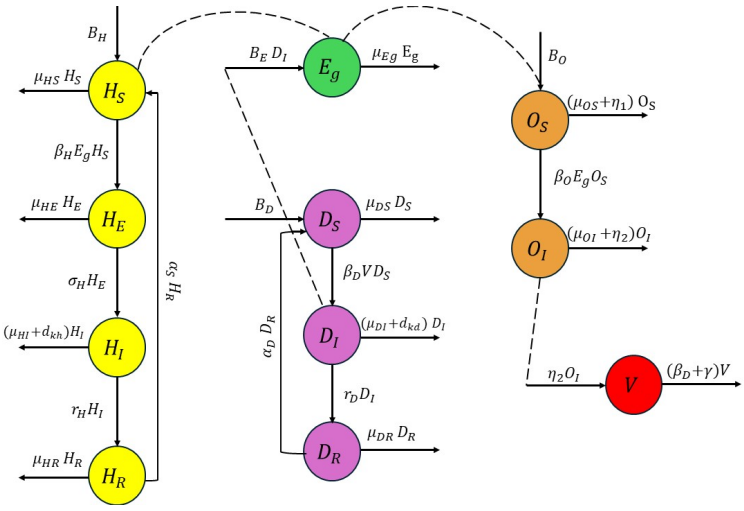


Figure 2: Diagram representation of the CE disease

5. The model also includes the density of Echinoccocosis eggs present in the environment denoted by $E_g$, they are spread by infected dogs at a rate $B_E$, and they face natural death at a rate $\mu_{Eg}$.

Table 1: Parameters of the model

| Parameters | Details |
|---|---|
| $B_D$ | Dogs birth or immigration rate |
| $B_O$ | Livestock birth or immigration rate |
| $B_E$ | Rate of shedding CE eggs by infected dogs |
| $\mu_{DS}$ | Natural mortality rate of susceptible dogs |
| $\mu_{DI}$ | Natural mortality rate of infected dogs |
| $\mu_{DR}$ | Natural mortality rate of recovered dogs |
| $\mu_{OS}$ | Natural mortality rate of susceptible livestock |
| $\mu_{OI}$ | Natural mortality rate of infected livestock |
| $\mu_{Eg}$ | Natural mortality rate of CE eggs |
| $\alpha_S$ | Relapse of humans after surgery |
| $\alpha_D$ | Relapse of dogs after recovery |
| $\sigma_H$ | Transfer rate to the infected humans class |
| $d_{kh}$ | Humans mortality rate due to the CE disease |
| $d_{kd}$ | Dogs mortality rate due to the CE disease |
| $\eta_1$ | Slaughtering rate of susceptible livestock |
| $\eta_2$ | Slaughtering rate of infected livestock |
| $r_D$ | Recovery rate of dogs |

The deterministic model is then given by this system of eleven equations bellow:

$$\begin{cases} \frac{dH_S}{dt} = B_H - \beta_H E_g H_S - \mu_{HS} H_S + \alpha_S H_R, \\ \frac{dH_E}{dt} = \beta_H E_g H_S - (\sigma_H + \mu_{HE})\, H_E, \\ \frac{dH_I}{dt} = \sigma_H H_E - (\mu_{HI} + d_{kh} + r_H)\, H_I, \\ \frac{dH_R}{dt} = r_H H_I - (\mu_{HR} + \alpha_S)\, H_R, \\ \frac{dD_S}{dt} = B_D - \beta_D V D_S - \mu_{DS} D_S + \alpha_D D_R, \\ \frac{dD_I}{dt} = \beta_D V D_S - (r_D + d_{kd} + \mu_{DI})\, D_I, \\ \frac{dD_R}{dt} = r_D D_I - (\mu_{DR} + \alpha_D) D_R, \\ \frac{dO_S}{dt} = B_O - \beta_O E_g O_S - (\mu_{OS} + \eta_1) O_S \\ \frac{dO_I}{dt} = \beta_O E_g O_S - (\mu_{OI} + \eta_2)\, O_I, \\ \frac{dV}{dt} = \eta_2 O_I - (\beta_D + \gamma)\, V, \\ \frac{dE_G}{dt} = B_E D_I - \mu_{Eg} E_g. \end{cases} \tag{1}$$

In system (1), we assume that all the parameters (described in Table 1) are positive.

## 2.2 Comparison with a previous study

In this section, a comparison is made with the article [5] by Chacha et al.

**- Overview of the article**

- In their work, Chacha et al. [5] developed a deterministic then a stochastic CTMC model to describe the dynamic of CE in humans, dogs, and cattle. The results emphasized that disease prevention requires intervention strategies for the populations of dogs and cattle.

**- Comparison of our model with the Chacha et al. model**

- Building on the work of Chacha et al. [5], our model also describes CE transmission between three types of population; humans, dogs and livestock and

studies the density of infected meat and CE eggs. Indeed unlike the model in [5], we adopted different types of submodels to address reality more:

- For human submodel, unlike [5], we included the compartment of recovered persons $H_R$. This inclusion allows us to give importance to the healing phase and the recovery processes which was neglected in [5]. In fact it has supposed that once the individual is infected, he either stay infected or dies,which is not true in real life, where several people take their treatment or/and surgery to recover. Another reason for adding this compartment is that patients that have experienced the disease and were recovered due to treatment will certainly change and develop some health and hygiene practices to prevent themselves from a new contamination, they will be aware of the danger of the disease and will help raise awareness among their family and neighbors circle conducting to a decrease of being susceptible or exposed to the disease, as improving hygiene is the most effective way to prevent from infection.

- For dog submodel, contrary to [5], we excluded the Exposed dogs compartment, because dogs become infectious almost few time after consuming infected meat of intermediate host, and there is a negligible latent period where dogs doesn't spread CE eggs. Moreover, in real life it is so difficult to detect if a dog is exposed but not yet infectious. Instead, we added a compartment for recovered dogs as there is a category of dogs that get cured by anthelmintics or by removing adult tapeworm in surgery, which was ignored in [5], where infectious dogs either stay infected or die.

- In the same context we added the parameters $\alpha_S$ and $\alpha_D$ that stand for the rate of recovered returning susceptible again among humans and dogs, respectively. This parameter aligns the biological behavior of CE, so if a recovered person stops his good hygiene practices, he is at direct risk of being infected again, especially in an endemic area. Similarly, if the immunity of a recovered dog wanes, then it becomes susceptible again to infection at a certain rate.

- For livestock submodel, we disregarded the exposed compartment introduced for cattle in [5], and used only susceptible and infected compartments for livestock. The cause behind this choice is that livestock are intermediate

hosts; they affect the life cycle of the disease only with the cysts present in their infected organs when they are slaughtered or naturally dead, so exposed and infected compartments are considered as one in this case. Adding exposed livestock will not affect the transmission cycle or the dynamic of CE.

- The model in [5] is developed as a stochastic model and our model will be developed as an optimal control model.

## 3 Model analysis

Grounded in the biological foundation of the model (1), we assume that all the initial conditions of solutions' system are positive as

$$\begin{aligned} &H_S(0) > 0, \quad H_E(0) > 0, \quad H_I(0) > 0, \quad H_R(0) > 0, \\ &D_S(0) > 0, \quad D_I(0) > 0, \quad D_R(0) > 0, \\ &O_S(0) > 0, \quad O_I(0) > 0, \\ &V(0) > 0, \quad E_g(0) > 0. \end{aligned} \tag{2}$$

Moreover, we suppose $H_P$ the total human population given by

$$H_P = H_S + H_E + H_I + H_R, \tag{3}$$

and $D_P$ the total dog population given by

$$D_P = D_S + D_I + D_R. \tag{4}$$

As the same, the total livestock population is modeled by $O_P$ and given as

$$O_P = O_S + O_I. \tag{5}$$

### 3.1 Positivity and boundedness of system solutions

**Theorem 1.** 1. The solutions of model system (1) with initial conditions (2) are nonnegative for all $t \geq 0$.

2. The solutions of model system (1) with initial conditions (2) are bounded in $\Gamma_\epsilon$ when $t \to \infty$.

Here for any $\varepsilon > 0$, $\Gamma_\varepsilon$ is defined as

$$\Gamma_\varepsilon = \left\{ (H, D, O, V, E_g) \in \mathbb{R}_+^{11} \quad | \quad \begin{array}{l} H_N \leq \dfrac{B_H}{\mu_1} + \varepsilon, \\ D_N \leq \dfrac{B_D}{\mu_2} + \varepsilon, \\ O_N \leq \dfrac{B_O}{\mu_3} + \varepsilon, \\ V \leq W, \\ E_g \leq F, \end{array} \right\} \tag{6}$$

with

$$\begin{aligned} &H = (H_S, H_E, H_I, H_R), \quad D = (D_S, D_I, D_R), \quad O = (O_S, O_I), \\ &W = \frac{\eta_2 B_O}{\mu_3(\beta_D + \gamma)} + (\frac{\eta_2}{\beta_D + \gamma} + 1)\varepsilon, \quad F = \frac{B_E B_D}{\mu_2 \mu_{Eg}} + \varepsilon(B_E + 1), \\ &\mu_1 = \min\{\mu_{HS}, \mu_{HE}, \mu_{HI}, \mu_{HR}\}, \quad \mu_2 = \min\{\mu_{DS}, \mu_{DI}, \mu_{DR}\}, \\ &\mu_3 = \min\{\mu_{OS}, \mu_{OI}\}. \end{aligned}$$

*Proof.* **Positivity of solutions**

1. First, for convenience, we write a solution of the model (1) as $(H(t), D(t), O(t), V(t), E_g(t))$, with $H(t) = (H_S(t), H_E(t), H_I(t), H_R(t))$, $D(t) = (D_S(t), D_I(t), D_R(t))$, and $O(t) = (O_S(t), O_I(t))$.

We consider $(H(t), D(t), O(t), V(t), E_g(t))$, a solution of the model (1) with the initial positive conditions (2).

We proceed by contradiction, and we suppose that there is $t^* > 0$ such that

$$\min(H(t^*), D(t^*), O(t^*), V(t^*), E_g(t^*)) = 0.$$

So that $\min(H(t), D(t), O(t), V(t), E_g(t)) > 0$ for all $t \in [0, t^*[$.

- **Case 1**: $\min(H(t^*), D(t^*), O(t^*), V(t^*), E_g(t^*)) = H_S(t^*)$.

We have in this case from the first equation of the system (1) that

$$\frac{dH_S}{dt} \geq -(\beta_H E_g + \mu_{HS})H_S \quad \text{for all} \quad t \in [0, t^*],$$

and by applying separation method, it yields

$$\frac{dH_S}{H_S} \geq -(\beta_H E_g + \mu_{HS})dt \quad \text{for all} \quad t \in [0, t^*].$$

Then we integrate in initial conditions,

$$0 = H_S(t^*) \geq H_S(0)e^{-\int_0^{t^*}(\beta_H E_g(s)+\mu_{HS})ds} > 0.$$

That results in a contradiction.

- **Case 2:** $\min(H(t^*), D(t^*), O(t^*), V(t^*), E_g(t^*)) = H_E(t^*)$.

We obtain from the second equation of the model (1) and by adopting the same method as in case 1, that

$$0 = H_E(t^*) \geq H_E(0)e^{-(\sigma_H+\mu_{HE})t^*} > 0.$$

This leads to a contradiction in this case too.

The same approach is applied for the rest of cases when $\min(H(t^*), D(t^*), O(t^*), V(t^*), E_g(t^*))$ is equal to $H_I(t^*), H_R(t^*), D_S(t^*)$, $D_I(t^*), D_R(t^*), O_S(t^*), O_I(t^*), V(t^*)$, and $E_g(t^*)$, respectively, where we have from the corresponding equations of the system (1), respectively:

$$0 = H_E(t^*) \geq H_E(0)e^{-(\sigma_H+\mu_{HE})t^*} > 0,$$
$$0 = H_I(t^*) \geq H_I(0)e^{-(\mu_{HI}+d_{kh}+r_H)t^*} > 0,$$
$$0 = H_R(t^*) \geq H_R(0)e^{-(\mu_{HR}+\alpha_S)t^*} > 0,$$
$$0 = D_S(t^*) \geq D_S(0)e^{-\int_0^{t^*}(\beta_D V(s)+\mu_{DS})ds} > 0,$$
$$0 = D_I(t^*) \geq D_I(0)e^{-(r_D+d_{kd}+\mu_{DI})t^*} > 0,$$
$$0 = D_R(t^*) \geq D_R(0)e^{-(\mu_{DR}+\alpha_D)t^*} > 0,$$
$$0 = O_S(t^*) \geq O_S(0)e^{-\int_0^{t^*}(\beta_O E_g(s)+\mu_{OS}+\eta_1)ds} > 0,$$
$$0 = O_I(t^*) \geq O_I(0)e^{-(\mu_{OI}+\eta_2)t^*} > 0,$$

$0 = V(t^*) \geq V(0)e^{-(\beta_D+\gamma)t^*} > 0,$

$0 = E_g(t^*) \geq E_g(0)e^{-\mu_{Eg}t^*} > 0.$

It yields to a contradiction in each case.

Finally, we conclude that all the solutions of system (1) are nonnegative for all $t \geq 0$.

**Boundedness of the solutions**

2. In this part of the demonstration, we use the total populations of humans (3), dogs (4), and livestock (5), respectively, to show that the solutions are bounded in $\Gamma_\varepsilon$ (6).

From (3), and the first four equations of system (1), we got

$$\frac{dH_P}{dt} = B_H - \mu_{HS}H_S - \mu_{HE}H_E - \mu_{HI}H_I - d_{kh}H_I - \mu_{HR}H_R.$$

So

$$\frac{dH_P}{dt} \leq B_H - \mu_{HS}H_S - \mu_{HE}H_E - \mu_{HI}H_I - \mu_{HR}H_R.$$

Let $\mu_1 = \min\{\mu_{HS}, \mu_{HE}, \mu_{HI}, \mu_{HR}\}$.

Then

$$\frac{dH_P}{dt} \leq B_H - \mu_1(H_S + H_E + H_I + H_R),$$
$$\frac{dH_P}{dt} \leq B_H - \mu_1 H_P.$$

Thus,

$$\lim_{t\to\infty} H_P(t) \leq \frac{B_H}{\mu_1}.$$

Hence for any $\varepsilon > 0$, there exists $t_1 > 0$ such that

$$H_P(t) \leq \frac{B_H}{\mu_1} + \varepsilon \qquad \text{for all } t \geq t_1.$$

Adopting the same method for the rest of the equation of system (1), we obtain the following results: From (4) and the fifth, sixth, and seventh equations from (1) we obtain that

there exists $t_2 > 0$ such as

$$D_P(t) \leq \frac{B_D}{\mu_2} + \varepsilon \quad \text{for all } t \geq t_2, \tag{7}$$

with $\mu_2 = \min\{\mu_{DS}, \mu_{DI}, \mu_{DR}\}$.

Similarly, we have from (5)

There exists $t_3 > 0$ such that

$$O_P(t) \leq \frac{B_O}{\mu_3} + \varepsilon \quad \text{for all } t \geq t_3, \tag{8}$$

with $\mu_3 = \min\{\mu_{OS}, \mu_{OI}\}$.
Likewise for the tenth equation of system (1) and from (8), we obtain

$$\begin{aligned} &\frac{dV(t)}{dt} = \eta_2 O_I(t) - (\beta_D + \gamma)V(t), \\ &\frac{dV(t)}{dt} \leq \eta_2 \frac{B_O}{\mu_3} + \eta_2\varepsilon - (\beta_D + \gamma)V(t) \quad \text{for all } t \geq t_3, \end{aligned}$$

so there is $t_4 > t_3$ such that

$$\begin{aligned} &V(t) \leq \frac{\eta_2(\frac{B_O}{\mu_3} + \varepsilon)}{\beta_D + \gamma} + \varepsilon, \\ &V(t) \leq \frac{\eta_2 B_O}{\mu_3(\beta_D + \gamma)} + (\frac{\eta_2}{\beta_D + \gamma} + 1)\varepsilon; \quad \text{for all } t \geq t_4. \end{aligned}$$

Finally, from the last equation of system (1) and inequality (7), we obtain the following result:
There exists $t_5 > t_2$ such that

$$E_g \leq \frac{B_E B_D}{\mu_2 \mu_{Eg}} + \varepsilon(B_E + 1) \quad \text{for all } t \geq t_5.$$

Take $t^{\#} = \max\{t_1, t_4, t_5\}$; then for all $t > t^{\#}$ we have

$$(H(t), D(t), O(t), V(t), E_g(t)) \in \Gamma_\varepsilon.$$

Recall that

$$H = (H_S, H_E, H_I, H_R), \quad D = (D_S, D_I, D_R), \quad O = (O_S, O_I).$$

Therefore the region $\Gamma_\varepsilon$ is positive and invariant. Moreover, all the solutions of system (1) with initial conditions (2) are bounded.

Finally, the proof of Theorem 1 is concluded. □

### 3.2 Disease free equilibrium (DFE) and basic reproduction number

Basic computations assume that the system (1) has a DFE given as

$$N_0 = (H_S^0, 0, 0, 0, D_S^0, 0, 0, O_S^0, 0, 0, 0),$$

where

$$\begin{aligned} H_S^0 &= \frac{B_H}{\mu_{HS}}, \\ D_S^0 &= \frac{B_D}{\mu_{DS}}, \\ O_S^0 &= \frac{B_O}{\mu_{OS} + \eta_1}. \end{aligned} \tag{9}$$

The DFE denotes the case when the disease does not occur in the three types of populations.

The basic reproduction number $R_0$ is the number of secondary infection caused by an infected person during the period of infection [7]. To compute it for the system (1) we use the next generation matrix method [21] and we define then

$$\mathcal{F} = \begin{pmatrix} \beta_H E_g H_S \\ 0 \\ \beta_D V D_S \\ \beta_O E_g O_S \\ 0 \\ 0 \end{pmatrix} \mathcal{V} = \begin{pmatrix} (\sigma_H + \mu_{HE}) H_E \\ -\sigma_H H_E + (\mu_{HI} + d_{kh} + r_H) H_I \\ (r_D + d_{kd} + \mu_{DI}) D_I \\ (\mu_{OI} + \eta_2) O_I \\ -\eta_2 O_I + (\beta_D + \gamma) V \\ -B_E D_I + \mu_{E_g} E_g \end{pmatrix}.$$

Then for the DFE (9), we obtain

$$\mathbb{F} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{\beta_H B_H}{\mu_{HS}} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_D B_D}{\mu_{DS}} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{\beta_O B_O}{\mu_{OS} + \eta_1} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

Moreover,

$$\mathbb{V} = \begin{pmatrix} a & 0 & 0 & 0 & 0 & 0 \\ -\sigma_H & b & 0 & 0 & 0 & 0 \\ 0 & 0 & c & 0 & 0 & 0 \\ 0 & 0 & 0 & d & 0 & 0 \\ 0 & 0 & 0 & -\eta_2 & e & 0 \\ 0 & 0 & -B_E & 0 & 0 & \mu_{Eg} \end{pmatrix},$$

where

$$a = (\sigma_H + \mu_{HE}), \quad b = (\mu_{HI} + d_{kh} + r_H), \quad c = (r_D + d_{kd} + \mu_{DI}),$$

$$d = (\mu_{OI} + \eta_2), \quad e = (\beta_D + \gamma).$$

Accordingly, as $R_0 = \rho\left(\mathbb{F}\mathbb{V}^{-1}\right)$, $R_0$ is given by

$$R_0 = \sqrt{\frac{\beta_D \beta_O B_D B_E \eta_2 B_O}{\mu_{DS}\mu_{Eg}(\eta_2 + \mu_{OI})(\mu_{OS} + \eta_1)(r_D + d_{kd} + \mu_{DI})(\beta_D + \gamma)}}. \tag{10}$$

For the seek of simplicity, we can write $R_0$ as

$$R_0 = \sqrt{\beta_D \beta_O \frac{B_E}{\mu_{Eg}} \frac{B_D}{\mu_{DS}} \frac{\eta_2}{(\eta_2 + \mu_{OI})} \frac{B_O}{(\mu_{OS} + \eta_1)} \frac{1}{(r_D + d_{kd} + \mu_{DI})} \frac{1}{(\beta_D + \gamma)}}. \tag{11}$$

In the expression of $R_0$ (11), we interpret $\dfrac{B_E}{\mu_{Eg}}$ to be the density of CE eggs released in the environment by contaminated dogs, $\dfrac{B_D}{\mu_{DS}}$ and $\dfrac{B_O}{(\mu_{OS} + \eta_1)}$ to be the size of dog and livestock populations in the beginning respectively. The fraction $\dfrac{\eta_2}{(\eta_2 + \mu_{OI})}$ stands for the contaminated population of livestock slaughtered for consuming. The expression $\dfrac{1}{(r_D + d_{kd} + \mu_{DI})}$ signifies the outflow rate of dogs from the infected compartment $D_I$. Finally, the standard rate describing when infectious meat is disease-spreading, is given by $\dfrac{1}{(\beta_D + \gamma)}$.

It is assumed that when $R_0 < 1$, the disease will stop on its own, and when $R_0 > 1$ it will persist.

### 3.3 Endemic equilibrium

The endemic equilibrium of system (1) denotes the case when the CE disease is endemic. It is represented by

$$N^* = (H_S^*, H_E^*, H_I^*, H_R^*, D_S^*, D_I^*, D_R^*, O_S^*, O_I^*, V^*, E_g^*), \tag{12}$$

where

$$H_S^* = \frac{B_H + \alpha_S H_R^*}{\beta_H E g^* + \mu_{HS}},$$
$$H_E^* = \frac{\beta_H E_g^*(B_H + \alpha_S H_R^*)}{(\sigma_H + \mu_{HE})(\beta_H E_g^* + \mu_{HS})},$$
$$H_I^* = \frac{\sigma_H \beta_H E_g^*(B_H + \alpha_S H_R^*)}{(\sigma_H + \mu_{HE})(\beta_H E_g^* + \mu_{HS})(\mu_{HI} + d_{kh} + r_H)},$$
$$H_R^* = \frac{B_H r_H \sigma_H \beta_H E_g^*}{(\mu_{HR} + \alpha_S)(\sigma_H + \mu_{HE})(\beta_H E_g^* + \mu_{HS})(\mu_{HI} + d_{kh} + r_H) - \alpha_S r_H \sigma_H \beta_H E_g^*},$$
$$D_S^* = \frac{B_D + \alpha_D D_R^*}{\beta_D V^* + \mu_{DS}},$$
$$D_I^* = \frac{\beta_D V^*(B_D + \alpha_D D_R^*)}{(\beta_D V^* + \mu_{DS})(r_D + d_{kd} + \mu_{DI})},$$
$$D_R^* = \frac{B_D r_D \beta_D V^*}{(\mu_{DR} + \alpha_D)(\beta_D V^* + \mu_{DS})(r_D + d_{kd} + \mu_{DI}) - \alpha_D r_D \beta_D V^*},$$
$$O_S^* = \frac{B_O}{\beta_O E_g^* + \mu_{OS} + \eta_1},$$
$$O_I^* = \frac{\beta_O E_g^* B_O}{(\mu_{OI} + \eta_2)(\beta_O E_g^* + \mu_{OS} + \eta_1)},$$
$$V^* = \frac{\eta_2 \beta_O E_g^* B_O}{(\beta_D + \lambda)(\mu_{OI} + \eta_2)(\beta_O E_g^* + \mu_{OS} + \eta_1)},$$
$$E_g^* = \frac{A}{B}(R_0 - 1)(R_0 + 1),$$

with $A = \mu_{DS}\mu_{Eg}(\mu_{DR}+\alpha_D)^2(r_D+d_{kd}+\mu_{DI})(\beta_D+\gamma)(\mu_{OI}+\eta_1)(\mu_{OS}+\eta_1)$, and $B = \mu_{Eg}\beta_D\eta_2\beta_O B_O[(\mu_{DR}+\alpha_D)(r_D+d_{kd}+\mu_{DI})-\alpha_D r_D]+\mu_{DS}\mu_{Eg}\beta_O(\mu_{DR}+\alpha_D)(r_D+d_{kd}+\mu_{DI})(\beta_D+\lambda)(\mu_{OI}+\eta_2)$.

According to the expression of the endemic equilibrium of system (1), we can say that the CE disease persist in humans, dogs and livestock if $R_0 > 1$, which is confirmed by this theorem.

**Theorem 2.** The system (1) modeling the dynamic of transmission of the Echinoccocosis disease has an endemic equilibrium when $R_0 > 1$.

## 3.4 Stability analysis of the equilibria

### 3.4.1 Global stability of the DFE

**Theorem 3.** The DFE $N_0$ is globally asymptotically stable when $R_0 < 1$.

*Proof.* To prove the global stability of DFE (9) when $R_0 < 1$, we use the Lyapunov function given by

$$L = \frac{1}{(r_D + d_{kd} + \mu_{DI})} D_I + \frac{B_E}{\mu_{DI}\mu_{Eg}} E_g. \tag{13}$$

So from the equation six and eleven of model system (1), we have

$$\begin{aligned}
\frac{dL}{dt} &= \frac{1}{r_D + d_{kd} + \mu_{DI}} \frac{dD_I}{dt} + \frac{B_E}{\mu_{DI}\mu_{Eg}} \frac{dE_g}{dt}, \\
\frac{dL}{dt} &= \frac{1}{r_D + d_{kd} + \mu_{DI}} [\beta_D V D_S - (r_D + d_{kd} + \mu_{DI}) D_I] \\
&\quad + \frac{B_E}{\mu_{DI}\mu_{Eg}} (B_E D_I - \mu_{Eg} E_g), \\
\frac{dL}{dt} &= \frac{\beta_D V D_S}{r_D + d_{kd} + \mu_{DI}} - D_I + \frac{B_E^2 D_I}{\mu_{DI}\mu_{Eg}} - \frac{B_E}{\mu_{DI}} E_g, \\
\frac{dL}{dt} &= \frac{\beta_D V D_S}{r_D + d_{kd} + \mu_{DI}} - D_I + \frac{B_E^2 D_I}{\mu_{DI}\mu_{Eg}} - \frac{B_E^2 D_I}{\mu_{DI}\mu_{Eg}}, \\
\frac{dL}{dt} &= \frac{\beta_D V D_S}{r_D + d_{kd} + \mu_{DI}} - D_I, \\
\frac{dL}{dt} &= \frac{\beta_D \eta_2 O_I B_D}{\mu_{DS}(r_D + d_{kd} + \mu_{DI})(\beta_D + \gamma)} - D_I, \\
\frac{dL}{dt} &= \frac{\beta_D \eta_2 B_D \beta_O E_g O_S}{\mu_{DS}(r_D + d_{kd} + \mu_{DI})(\beta_D + \gamma)(\mu_{OI+\eta_2})} - D_I, \\
\frac{dL}{dt} &= \left(\frac{\beta_D \eta_2 B_D \beta_O B_O B_E}{\mu_{DS}\mu_{Eg}(r_D + d_{kd} + \mu_{DI})(\beta_D + \gamma)(\mu_{OI} + \eta_2)(\mu_{OS} + \eta_1)} - 1\right) D_I, \\
\frac{dL}{dt} &= (R_0^2 - 1) D_I.
\end{aligned}$$

Then

$$\frac{dL}{dt} = (R_0 - 1)(R_0 + 1) D_I. \tag{14}$$

From Theorem 1, we have $D_I$ is nonnegative, so in (14), if $R_0 = 1$, then $\frac{dL}{dt} = 0$. If $R_0 < 1$, then $\frac{dL}{dt} < 0$

Based on what precedes we can conclude then that the DFE (9) is globally asymptotically stable in $\Gamma_\varepsilon$(6) when $R_0 < 1$.

Therefore the proof of this theorem comes to an end. □

### 3.4.2 Global stability of endemic equilibrium

**Theorem 4.** The system of differential equation (1), is said to have a unique, globally asymptotically stable (GAS), endemic equilibrium (12) if $R_0 > 1$.

*Proof.* We know that endemic equilibrium exists if and only if $R_0 > 1$, so in this proof we will suppose that we are in the case when $R_0 > 1$.

We consider the function $h(x) = x-1-\ln(x), x > 0$, we have clearly $h(x) \geq 0$ and that $h(1) = 0$.
Moreover, we can easily prove that

$$(x-1)(y-1) = h(x) + h(y) - h(xy) \quad \text{for all} \quad x > 0 \quad \text{and} \quad y > 0. \tag{15}$$

We consider $\mathcal{L}_{\#} = \#^* h(\frac{\#}{\#^*})$ where we will replace $\#$ by $H_S, H_E, H_I, H_R, D_S, D_I, D_R, O_S, O_I, V$ and $E_g$, respectively.

First, we have

$$\frac{d\mathcal{L}_{H_S}}{dt} = \left(1 - \frac{H_S^*}{H_S}\right), \frac{dH_S}{dt} \tag{16}$$

and from the first equation of system (1) and the equilibrium equation, we have
$B_H - \beta_H E_g^* H_S^* - \mu_{HS} H_S^* + \alpha_S H_R^* = 0$, and we get

$$\begin{aligned}
\frac{d\mathcal{L}_{H_S}}{dt} &= \left(1 - \frac{H_S^*}{H_S}\right)\left(\beta_H E_g^* H_S^* + \mu_{HS} H_S^* - \alpha_S H_R^* - \beta_H E_g H_S - \mu_{HS} H_S + \alpha_S H_R\right) \\
&= \left(1 - \frac{H_S^*}{H_S}\right)\left[-\beta_H (E_g H_S - E_g^* H_S^*) - \mu_{HS}(H_S - H_S^*) + \alpha_S (H_R - H_R^*)\right] \\
&= -\beta_H E_g^* H_S^* \left(1 - \frac{E_g H_S}{E_g^* H_S^*}\right)\left(1 - \frac{H_S^*}{H_S}\right) - \mu_{HS} H_S^* \left(\frac{H_S}{H_S^*} - 1\right)\left(1 - \frac{H_S^*}{H_S}\right) \\
&\quad + \alpha_S H_R^* \left(\frac{H_R}{H_R^*} - 1\right)\left(1 - \frac{H_S^*}{H_S}\right).
\end{aligned} \tag{17}$$

Using the expression (15) and the equilibrium equation yields

$$\frac{d\mathcal{L}_{H_S}}{dt} = -\beta_H E_g^* H_S^* h(\frac{E_g H_S}{E_g^* H_S^*}) - \beta_H E_g^* H_S^* h(\frac{H_S^*}{H_S}) + \beta_H E_g^* H_S^* h(\frac{E_g}{E_g^*})$$

$$
\begin{aligned}
&- \mu_{HS} H_S^* h(\frac{H_S}{H_S^*}) - \mu_{HS} H_S^* h(\frac{H_S^*}{H_S}) \\
&+ \alpha_S H_R^* h(\frac{H_R}{H_R^*}) + \alpha_S H_R^* h(\frac{H_S^*}{H_S}) - \alpha_S H_R^* h(\frac{H_R H_S^*}{H_S H_R^*}) \\
=& - \beta_H E_g^* H_S^* h(\frac{E_g H_S}{E_g^* H_S^*}) + \beta_H E_g^* H_S^* h(\frac{E_g}{E_g^*}) - \mu_{HS} H_S^* h(\frac{H_S}{H_S^*}) \\
&- B_H h(\frac{H_S^*}{H_S}) + \alpha_S H_R^* h(\frac{H_R}{H_R^*}) - \alpha_S H_R^* h(\frac{H_R H_S^*}{H_S H_R^*}). \qquad (18)
\end{aligned}
$$

Similarly, from the second equation of the system (1), and the endemic equilibrium equation, $\beta_H E_g^* H_S^* - (\sigma_H + \mu_{HE}) H_E^* = 0$, we obtain

$$
\begin{aligned}
\frac{d\mathcal{L}_{HE}}{dt} &= \left(1 - \frac{H_E^*}{H_E}\right) \frac{dH_E}{dt} \\
&= \left(1 - \frac{H_E^*}{H_E}\right) \left[\beta_H (E_g H_S - E_g^* H_S^*) - (\sigma_H + \mu_{HE}) (H_E - H_E^*)\right] \\
&= \beta_H E_g^* H_S^* (1 - \frac{H_E^*}{H_E})(\frac{E_g H_S}{E_g^* H_S^*} - 1) \\
&\quad - (\sigma_H + \mu_{HE}) H_E^* (1 - \frac{H_E^*}{H_E})(\frac{H_E}{H_E^*} - 1). \qquad (19)
\end{aligned}
$$

Using the expression (15) and the equilibrium equation yields

$$
\frac{d\mathcal{L}_{H_E}}{dt} = \beta_H E_g^* H_S^* h(\frac{E_g H_S}{E_g^* H_S^*}) - \beta_H E_g^* H_S^* h(\frac{H_E^* E_g H_S}{H_E E_g^* H_S^*}) - (\sigma_H + \mu_{HE}) H_E^* h(\frac{H_E}{H_E^*}).
$$

Similarly, we get

$$
\begin{aligned}
\frac{d\mathcal{L}_{H_I}}{dt} &= \sigma_H H_E^* h(\frac{H_E}{H_E^*}) - \sigma_H H_E^* h(\tfrac{H_I^* H_E}{H_I H_E^*}) - (\mu_{HI} + d_{kh} + r_H) H_I^* h(\frac{H_I}{H_I^*}), \\
\frac{d\mathcal{L}_{H_R}}{dt} &= r_H H_I^* h(\frac{H_I}{H_I^*}) - r_H H_I^* h(\frac{H_R^* H_I}{H_R H_I^*}) - (\mu_{HR} + \alpha_S) H_R^* h(\frac{H_R}{H_R^*}), \\
\frac{d\mathcal{L}_{D_S}}{dt} &= -\beta_D V^* D_S^* h(\tfrac{V D_S}{V^* D_S^*}) + \beta_D V^* D_S^* h(\frac{V}{V^*}) - \mu_{DS} D_S^* h(\frac{D_S}{D_S^*}) - B_D h(\frac{D_S^*}{D_S}) \\
&\quad + \alpha_D D_R^* h(\frac{D_R}{D_R^*}) - \alpha_D D_R^* h(\frac{D_R D_S^*}{D_S D_R^*}), \\
\frac{d\mathcal{L}_{D_I}}{dt} &= \beta_D V^* D_S^* h(\frac{V D_S}{V^* D_S^*}) - \beta_D V^* D_S^* h(\frac{V D_S D_I^*}{V^* D_S^* D_I}) - (r_D + d_{kd} + \mu_{DI}) D_I^* h(\frac{D_I}{D_I^*}), \\
\frac{d\mathcal{L}_{D_R}}{dt} &= r_D D_I^* h(\frac{D_I}{D_I^*}) - r_D D_I^* h(\frac{D_R^* D_I}{D_R D_I^*}) - (\mu_{DR} + \alpha_D) D_R^* h(\frac{D_R}{D_R^*}), \\
\frac{d\mathcal{L}_{O_S}}{dt} &= -\beta_O E_g^* O_S^* h(\frac{E_g O_S}{E_g^* O_S^*}) + \beta_O E_g^* O_S^* h(\frac{E_g}{E_g^*}) - (\mu_{OS} + \eta_1) O_S^* h(\frac{O_S}{O_S^*})
\end{aligned}
$$

$$- B_O h(\frac{O_S^*}{O_S}),$$

$$\frac{d\mathcal{L}_{O_I}}{dt} = \beta_O E_g^* O_S^* h(\frac{E_g O_S}{E_g^* O_S^*}) - \beta_O E_g^* O_S^* h(\frac{E_g O_S O_I^*}{E_g^* O_S^* O_I}) - (\mu_{OI} + \eta_2) O_I^* h(\frac{O_I}{O_I^*}),$$

$$\frac{d\mathcal{L}_V}{dt} = \eta_2 O_I^* h(\frac{O_I}{O_I^*}) - \eta_2 O_I^* h(\frac{V^* O_I}{V O_I^*}) - \beta_D V^* h(\frac{V}{V^*}) - \gamma V^* h(\frac{V}{V^*}),$$

$$\frac{d\mathcal{L}_{E_g}}{dt} = B_E D_I^* h(\frac{D_I}{D_I^*}) - B_E D_I^* h(\frac{D_I E_g^*}{D_I^* E_g}) - \mu_{Eg} E_g^* h(\frac{E_g}{E_g^*}).$$

We define the Lyapunov function given by

$$\begin{aligned}\mathcal{L} =& \frac{\mu_{Eg}}{B_H H_S^*}(\mathcal{L}_{H_S} + \mathcal{L}_{H_E} + \mathcal{L}_{H_I} + \mathcal{L}_{H_R}) + \frac{2B_E}{d_{kd}}(\mathcal{L}_{D_S} + \mathcal{L}_{D_E} + \mathcal{L}_{D_R}) \\ &+ \frac{\mu_{Eg}}{\beta_O O_S^*}(\mathcal{L}_{O_S} + \mathcal{L}_{O_I}) + \frac{2B_E D_S^*}{d_{kd}}\mathcal{L}_V + 2\mathcal{L}_{E_g}.\end{aligned} \tag{20}$$

The derivative of the Lyapunov function regarding time is then given as

$$\begin{aligned}\frac{d\mathcal{L}}{dt} =& \frac{\mu_{Eg}}{\beta_H H_S^*}\Big[-\mu_{HS} H_S^* h(\frac{H_S}{H_S^*}) - B_H h(\frac{H_S^*}{H_S}) - \alpha_S H_R^* h(\frac{H_R H_S^*}{H_S H_R^*}) \\ &- \beta_H E_g^* H_S^* h(\frac{H_E^* E_g H_S}{H_E E_g^* H_S^*}) - \mu_{HE} H_E^* h(\frac{H_E}{H_E^*}) - \sigma_H H_E^* h(\frac{H_I^* H_E}{H_I H_E^*}) \\ &- (\mu_{HI} + d_{kh}) H_I^* h(\frac{H_I}{H_I^*}) - r_H H_I^* h(\frac{H_R^* H_I}{H_R H_I^*}) - (\mu_{HR} + \alpha_S) H_R^* h(\frac{H_R}{H_R^*})\Big] \\ &+ \frac{2B_E}{d_{kd}}\Big[-\mu_{DS} D_S^* h(\frac{D_S}{D_S^*}) - B_D h(\frac{D_S^*}{D_S}) - \alpha_D D_R^* h(\frac{D_R D_S^*}{D_S D_R^*}) \\ &- \beta_D V^* D_S^* h(\frac{V D_S D_I^*}{V^* D_S^* D_I}) - (d_{kd} + \mu_{DI}) D_I^* h(\frac{D_I}{D_I^*}) \\ &- r_D D_I^* h(\frac{D_R^* D_I}{D_R D_I^*}) - \mu_{DR} D_R^* h\,(\frac{D_R}{D_R^*})\Big] \\ &+ \frac{\mu_E g}{\beta_O O_S^*}\Big[-(\mu_{OS} + \eta_1) O_S^* h(\frac{O_S}{O_S^*}) - B_O h(\frac{O_S^*}{O_S}) \\ &-\beta_O E_g^* O_S^* h(\frac{E_g O_S O_I^*}{E_g^* O_S^* O_I}) - \mu_{OI} O_I^* h(\frac{O_I}{O_I^*})\Big] \\ &+ \frac{2B_E D_S^*}{d_{kd}}\Big[-\eta_2 O_I^* h(\frac{V^* O_I}{V O_I^*}) - \beta_D V^* h(\frac{V}{V^*}) - \gamma V^* h(\frac{V}{V^*})\Big] \\ &- 2\Big[B_E D_I^* h(\frac{D_I E_g^*}{D_I^* E_g})\Big].\end{aligned}$$

We have that all the solutions and parameters of the system (1) are positive. Moreover the function $h$ is positive, so the derivative of our Lyapunov function

is clearly less than zero.

So $\frac{d\mathcal{L}}{dt} \leq 0$, and $\frac{d\mathcal{L}}{dt} = 0$ if and only if $H_S = H_S^*, H_E = H_E^*, H_I = H_I^*, H_R = H_R^*, D_S = D_S^*, D_I = D_I^*, D_R = D_R^*, O_S = O_S^*, O_I = O_I^*, V = V^*, E_g = E_g^*$.

Then the largest invariant set where $\frac{d\mathcal{L}}{dt} = 0$, is the singleton $N^*$, which is the endemic equilibrium (12).

Hence using Lassale's invariant principle, when $t \to +\infty$, all solutions of (1) approach the endemic equilibrium if $R_0 > 1$.

Finally the endemic equilibrium is GAS when $R_0 > 1$. □

## 4 Optimal control

### 4.1 Optimal control problem

In this section, we propose a formulation of an optimal control problem of the CE disease, based on some prevention techniques proposed in some medical studies in Morocco such as [9].

Indeed we introduce to model (1) two controls $u_1$ and $u_2$ described as follows:

- The first control $u_1$ describes health education in the time interval $[0, T]$. Indeed, due to the poor knowledge about CE mechanism in rural areas [20], it is a potent approach to help people understand the behavior of CE disease and how it is transmitted among domestic animals and humans to promote good hygiene practices, secure management of livestock, and show the importance of stopping dogs from consuming infected meat. This will help raise awareness among citizens and improve community engagement to fight the disease more effectively.

- The second control $u_2$ models the vaccination of livestock with an E. granulosus recombinant antigen (EG95) [25] in the interval of time $[0, T]$. As livestock are the intermediate hosts in the transmission cycle of CE (see Figure1), their vaccination will help effectively break the life cycle of the parasite. The vaccine has shown its worth in China where it is highly utilized, and in

Argentina [25]. In Morocco the EG95 vaccine produced in the country was found in a study elaborated in 2019 [25] to be securing from CE in sheep, during an experimental period of 18 months for 4 groups of 20 animals. However, this approach is not yet applied to the majority of livestock in Morocco.

After applying controls to system (1), we obtain the following controlled system of eleven equations:

$$\begin{aligned}
\frac{dH_S}{dt} &= B_H - (1-u_1(t))\,\beta_H E_g(t) H_S(t) - \mu_H H_S(t) + \alpha_S H_R(t),\\
\frac{dH_E}{dt} &= (1-u_1(t))\,\beta_H E_g(t) H_S(t) - (\sigma_H + \mu_H)\, H_E(t),\\
\frac{dH_I}{dt} &= \sigma_H H_E(t) - (\mu_H + d_{kh} + r_H)\, H_I(t),\\
\frac{dH_R}{dt} &= r_H H_I(t) - (\mu_H + \alpha_S)\, H_R(t),\\
\frac{dD_S}{dt} &= B_D - \beta_D V(t) D_S(t) + \alpha_D D_R(t) - \mu_D D_S(t),\\
\frac{dD_I}{dt} &= \beta_D V(t) D_S(t) - (r_D + \mu_D + d_{kd})\, D_I(t),\\
\frac{dD_R}{dt} &= r_D D_I(t) - (\mu_D + \alpha_D)\, D_R(t),\\
\frac{dO_S}{dt} &= B_O - (1-u_2(t))\,\beta_O E_g(t) O_S(t) - (\mu_O + \eta_1)\, O_S(t),\\
\frac{dO_I}{dt} &= (1-u_2(t))\,\beta_O E_g(t) O_S(t) - (\mu_O + \eta_2)\, O_I(t),\\
\frac{dV}{dt} &= \eta_2 O_I(t) - (\beta_D + \gamma)\, V(t),\\
\frac{dE_g}{dt} &= \sigma_E D_I(t) - \mu_{E_g} E_g(t).
\end{aligned} \tag{21}$$

In this model, we suppose that all the compartments of humans, dogs, and livestock have a unique death rate, $\mu_H, \mu_D$, and $\mu_O$, respectively.
Moreover, the same initial conditions of model (1), given by (2), are also applied to this controlled model.

We associate to system model (21) the objective functional $J$ given as

$$J(u_1,u_2)=\int_0^T [C_1H_E(t)+C_2H_I(t)+C_3D_I(t)+C_4O_I(t)+\frac{1}{2}\sum_{i=1}^{i=2}\theta_i u_i^2(t)]dt, \tag{22}$$

where $C_i \geq 0$, for $i=1,\ldots,4$ are the gains of the populations $H_E(t), H_I(t)$, $D_I(t)$, and $O_I(t)$, respectively. The constants $\theta_i$ for $i=1,2$ represent the weights that balance the associated controls.

Furthermore, the integrand of the objective functional is given by

$$\begin{aligned}&\mathcal{L}(H_S,H_E,H_I,H_R,D_S,D_I,D_R,O_S,O_I,V,E,u)\\&\quad=C_1H_E(t)+C_2H_I(t)+C_3D_I(t)+C_4O_I(t)+\frac{1}{2}\sum_{i=1}^{i=2}\theta_i u_i^2(t).\end{aligned} \tag{23}$$

More precisely, the optimal control problem can be defined as follows:

$$J(u_1^*,u_2^*)=\min_{\Omega} \quad J(u_1,u_2), \tag{24}$$

where

$$\Omega=\left\{(u_1,u_2) \quad | \quad \begin{array}{l} u_i(t) \text{ is Lebesgue measurable} \\ 0\leq u_i(t)\leq 1, \quad t\in[0,T], \text{for} \quad i=1,2, \end{array}\right\} \tag{25}$$

is the set of admissible controls.

Therefore, the optimal control problem is solved when $(u_1^*,u_2^*)\in\Omega$ that minimize the function (24), are founded.

## 4.2 Existence of optimal control

In order to solve the optimal control problem, it is first necessary to show the existence of the solution of system (21).

Consider the state variables $H_S,H_E,H_I,H_R,D_S,D_I,D_R,O_S,O_I,V,E_g$ and the control variables $u_1$, $u_2$ with nonnegative initial conditions as given in (2), the system (21) can be written as

$$\mathbb{X}_t=\mathbb{A}\mathbb{X}+\mathbb{B}(\mathbb{X}), \tag{26}$$

where $\mathbb{X}$ and $\mathbb{B}$ are given bellow and $\mathbb{A}$ is given by (27):

$$\mathbb{X} = \begin{bmatrix} H_S \\ H_E \\ H_I \\ H_R \\ D_S \\ D_I \\ D_R \\ O_S \\ O_I \\ V \\ E_g \end{bmatrix}, \qquad \mathbb{B}(\mathbb{X}) = \begin{bmatrix} B_H - (1-u_1(t))\,\beta_H E_g H_S \\ (1-u_1(t))\,\beta_H E_g H_S \\ 0 \\ 0 \\ B_D - \beta_D V D_S \\ \beta_D V D_S \\ 0 \\ B_O - (1-u_2(t))\,\beta_O E_g O_S \\ (1-u_2(t))\,\beta_O E_g O_S \\ 0 \\ 0 \end{bmatrix},$$

$\mathbb{X}_t$ is the derivative of $\mathbb{X}$ with respect to time $t$. It is clear that the system (26) is a nonlinear system:

$$\mathbb{A} = \begin{bmatrix} \mu_H & 0 & 0 & \alpha_S & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\sigma_H - \mu_H & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_H & -(\mu_H + d_{KH} + r_H) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & r_H & -(\mu_H + \alpha_S) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_D & 0 & \alpha_D & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(r_D + \mu_D + d_{kd}) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_D + \alpha_D) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_O + \eta_1) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_O + \eta_2) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \eta_2 & -(\beta_D + \gamma) & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_E & 0 & 0 & 0 & 0 & -\mu_{E_g} \end{bmatrix}. \tag{27}$$

We pose

$$\mathbb{H}(\mathbb{X}) = \mathbb{A}\mathbb{X} + \mathbb{B}(\mathbb{X}). \tag{28}$$

Let $\mathbb{X}_1$ and $\mathbb{X}_2$ be two solutions of system (21). Then,

$$\mathbb{B}(\mathbb{X}_1) - \mathbb{B}(\mathbb{X}_2) = \begin{bmatrix} (1-u_1(t))\,\beta_H\,[-E_{g_1}H_{S_1} + E_{g_2}H_{S_2}] \\ (1-u_1(t))\,\beta_H\,[E_{g_1}H_{S_1} - E_{g_2}H_{S_2}] \\ 0 \\ 0 \\ \beta_D\,(-V_1 D S_1 + V_2 D S_2) \\ \beta_D\,(V_1 D_{S_1} - V_2 D_{S_2}) \\ 0 \\ (1-u_2(t))\,\beta_O\,(-E_{g_1}O_{S_1} + E_{g_2}O_{S_2}) \\ (1-u_2(t))\,\beta_O\,(E_{g_1}O_{S_1} - E_{g_2}O_{S_2}) \\ 0 \\ 0 \end{bmatrix}.$$

This term satisfies the following:

$$
\begin{aligned}
|\mathbb{B}(\mathbb{X}_1) - \mathbb{B}(\mathbb{X}_2)| =& 2\beta_H \left(1 - u_1(t)\right) |E_{g_1}(t) H_{S_1}(t) - E_{g_2}(t) H_{S_2}(t)| \\
&+ 2\beta_D |V_1 D_{S_1} - V_2 D_{S_2}| \\
&+ 2\beta_O \left(1 - u_2(t)\right) |E_{g_1} O_{S_1} - E_{g_2} O_{S_2}| \\
\leq& 2\beta_H \left(1 - u_1(t)\right) [|E_{g_2}||H_{S_1}(t) - H_{S_2}(t)| \\
&+ |H_{S_1}||E_{g_1}(t) - E_{g_2}(t)|] \\
&+ 2\beta_D [|D_{S_1}||V_1(t) - V_2(t)| + |V_2||D_{S_1} - D_{S_2}|] \\
&+ 2\beta_O \left(1 - u_2(t)\right) [|E_{g_1}||O_{S_1} - O_{S_2}| + |O_{S_2}||E_{g_1} - E_{g_2}|] \\
\leq& 2\beta_H \left(1 - u_1(t)\right) [F|H_{S_1}(t) - H_{S_2}(t)| \\
&+ \frac{B_H}{\mu_H} |E_{g_1}(t) - E_{g_2}(t)|\Big] \\
&+ 2\beta_D \left[\frac{B_D}{\mu_D} |V_1(t) - V_2(t)| + W|D_{S_1} - D_{S_2}|\right] \\
&+ 2\beta_O \left(1 - u_2(t)\right) \left[F|O_{S_1} - O_{S_2}| + \frac{B_O}{\mu_O} |E_{g_1} - E_{g_2}|\right] \\
\leq& 2\beta_H F \left(1 - u_1(t)\right) |H_{S_1}(t) - H_{S_2}(t)| \\
&+ 2\beta_H \frac{B_H}{\mu_H} \left(1 - u_1(t)\right) |E_{g_1}(t) - E_{g_2}(t)| \\
&+ 2\beta_D \frac{B_D}{\mu_D} |V_1(t) - V_2(t)| + 2\beta_D W |D_{S_1} - D_{S_2}| \\
&+ 2\beta_O F \left(1 - u_2(t)\right) |O_{S_1} - O_{S_2}| \\
&+ 2\beta_O \frac{B_O}{\mu_O} \left(1 - u_2(t)\right) |E_{g_1} - E_{g_2}| \\
\leq& M[|H_{S_1}(t) - H_{S_2}(t)| + |E_{g_1}(t) - E_{g_2}(t)| + |V_1(t) - V_2(t)| \\
&+ |D_{S_1} - D_{S_2}| + |O_{S_1} - O_{S_2}|],
\end{aligned}
$$

where $M > 0$ is independent of the variables $H_S$, $E_g$, $V$, $D_S$, and $O_S$. Therefore;

$$|\mathbb{H}(\mathbb{X}_1) - \mathbb{H}(\mathbb{X}_2)| \leq K|\mathbb{X}_1 - \mathbb{X}_2|, \text{ with } K = ||\mathbb{A}|| + M.$$

Thus, it follows that the function $\mathbb{H}$ satisfies the Lipschitz condition, uniformly with respect to non-negative state variables.

Therefore, there exists a solution of the system (21).

Now, we present a result that will show the existence of an optimal control

that minimizes the objective functional $J$ in a finite interval $[0, T]$, subjected to the system (21).

**Theorem 5.** There exists an optimal control pair $u^* = \{u_1^*, u_2^*\}$ in $\Omega$ and a corresponding solution $\mathbb{X}^* = \left\{H_S^*, H_E^*, H_I^*, H_R^*, D_S^*, D_I^*, D_R^*, O_S^*, O_I^*, V^*, E_g^*\right\}$ such that

$$J(u_1^*, u_2^*) = \min_{\Omega} \quad J(u_1, u_2).$$

*Proof.* The existence of an optimal control is proved using Fleming's results (Theorem (III.4.1) and its corresponding corollary in [10]). Indeed we must verify those five conditions:

**P1.** The set solutions to the system (21)–(2) and its corresponding controls are nonempty (proved lastly).

**P2.** The set of controls is convex and closed.

We consider

$$\Omega = \left\{u \in \mathbb{R}^2 : ||u|| \leq 1\right\}.$$

Let $u$, $v \in \Omega$ such that $0 \leq ||u|| \leq 1$ and $0 \leq ||v|| \leq 1$.

Then, for any $\varepsilon \in [0, 1]$, we have

$$0 \leq ||\varepsilon u + (1-\varepsilon)v|| \leq \varepsilon||u|| + (1-\varepsilon)||v|| \leq 1.$$

**P3.** The state system can be written as linear function of control variables with coefficients depending on time and state variable.

**P4.** The integrand (23) of objective function is convex with respect to controls.

Let $\mathbb{X} = (H_S, H_E, H_I, H_R, D_S, D_I, D_R, O_S, O_I, V, E)$, $u = (u_1, u_2) \in \Omega$ and $v = (\{)v_1, v_2\} \in \Omega$.

Then, for $\varepsilon \in [0, 1]$, we have

$\mathcal{L}(\mathcal{X}, \varepsilon u + (1-\varepsilon)v) = C_1 H_E(t) + C_2 H_I(t) + C_3 D_I(t) + C_4 O_I(t) + \frac{1}{2}\sum_{i=1}^{i=2} \theta_i (\varepsilon u_i + (1-\varepsilon)v_i)^2$,

and

$$\varepsilon\mathcal{L}(\mathcal{X}, u) + (1-\varepsilon)\mathcal{L}(\mathcal{X}, v) = C_1 H_E(t) + C_2 H_I(t) + C_3 D_I(t) + C_4 O_I(t)$$

$$+\frac{1}{2}\varepsilon\sum_{i=1}^{i=2}\theta_i u_i^2+\frac{1}{2}(1-\varepsilon)\sum_{i=1}^{i=2}\theta_i v_i^2.$$

Therefore,

$$\mathcal{L}(\mathcal{X},\varepsilon u+(1-\varepsilon)v)-(\varepsilon\mathcal{L}(\mathcal{X},u)+(1-\varepsilon)\mathcal{L}(\mathcal{X},v))=\frac{1}{2}(\varepsilon^2-\varepsilon)\sum_{i=1}^{i=2}\theta_i(u_i-v_i)^2,$$

and

$$\mathcal{L}(\mathcal{X},\varepsilon u+(1-\varepsilon)v)-(\varepsilon\mathcal{L}(\mathcal{X},u)+(1-\varepsilon)\mathcal{L}(\mathcal{X},v))\leq 0,\quad \text{since}\quad \varepsilon\in[0,1].$$

**P5.** There exist constants $\vartheta_1,\vartheta_2>0$ and $\vartheta_3>1$ as the integrand (23) is bounded by
$\vartheta_1(|u_1|^2+|u_2|^2)^{\vartheta_3/2}-\vartheta_2$.
In fact it is easy to verify that

$$\begin{aligned}\mathcal{L}(\mathcal{X},u)&\geq\frac{1}{2}\textstyle\sum_{i=1}^{i=2}\theta_i u_i^2\\&\geq\vartheta_1(|u_1|^2+|u_2|^2)^{\vartheta_3/2}-\vartheta_2,\end{aligned}$$

where $\vartheta_1=\frac{1}{2}\min\{\theta_1,\theta_2\}$, $\vartheta_2>0$ and $\vartheta_3=2$. □

## 4.3 Characterization of optimal control

Theorem 5 assures the existence of an optimal control $(u_1^*,u_2^*)$ that minimizes the objective functional (22).

The Pontryagin's maximum principle transform system (21) with (22) and (24) to a minimization problem of Hamiltonian, that we define as

$$\begin{aligned}&\mathrm{H}(\mathbb{X},u,\Lambda,t)\\&=C_1H_E(t)+C_2H_I(t)+C_3D_I(t)+C_4O_I(t)+\frac{1}{2}\sum_{i=1}^{i=2}\theta_i u_i^2(t)\\&\quad+\lambda_1(t)\frac{dH_S(t)}{dt}+\lambda_2(t)\frac{dH_E(t)}{dt}+\lambda_3(t)\frac{dH_I(t)}{dt}+\lambda_4(t)\frac{dH_R(t)}{dt}\\&\quad+\lambda_5(t)\frac{dD_S(t)}{dt}+\lambda_6(t)\frac{dD_I(t)}{dt}+\lambda_7(t)\frac{dD_R(t)}{dt}+\lambda_8(t)\frac{dO_S(t)}{dt}\\&\quad+\lambda_9(t)\frac{dO_I(t)}{dt}+\lambda_{10}(t)\frac{dV(t)}{dt}+\lambda_{11}(t)\frac{dE(t)}{dt},\end{aligned}\tag{29}$$

with $\mathbb{X} = \{H_S, H_E, H_I, H_R, D_S, D_I, D_R, O_S, O_I, V, E_g\}$, $u = \{u_1, u_2\} \in \Omega$, and $\Lambda = \{\lambda_i, i = 1, \ldots, 11\}$ the adjoint variables.

Necessary condition that $\{\mathbb{X}^*(t), u^*(t)\}$ can be an optimal solution for the optimal control problem is the existence of a nontrivial vector function $\Lambda(t) = \{\lambda_i(t), i = 1, \ldots, 11\}$ such that

$$\begin{aligned} \frac{d\mathbb{X}}{dt} &= \frac{\partial \mathrm{H}(\mathbb{X}^*(t), u^*(t), \Lambda(t), t)}{d\Lambda}, \\ 0 &= \frac{\partial \mathrm{H}(\mathbb{X}^*(t), u^*(t), \Lambda(t), t)}{du}, \\ \Lambda'(t) &= -\frac{\partial \mathrm{H}(\mathbb{X}^*(t), u^*(t), \Lambda(t), t)}{d\mathbb{X}}. \end{aligned} \tag{30}$$

The next result presents the adjoint system and the characterization of optimal control.

**Theorem 6.** Given an optimal control $u^* = \{u_1^*, u_2^*\}$ and corresponding solutions $\mathbb{X}^* = \{H_S^*, H_E^*, H_I^*, H_R^*, D_S^*, D_I^*, D_R^*, O_S^*, O_I^*, V^*, E_g^*\}$ that minimize $J(u)$ over $\Omega$, there exist adjoint variables $\lambda_i$, $i = 1, 2, \ldots, 11$ satisfying

$$\frac{d\lambda_1(t)}{dt} = (1 - u_1(t))\,\beta_H E_g(t)\,[\lambda_1(t) - \lambda_2(t)] + \mu_H \lambda_1(t),$$

$$\frac{d\lambda_2(t)}{dt} = -C_1 + \sigma_H\,[\lambda_2(t) - \lambda_3(t)] + \mu_H \lambda_2(t),$$

$$\frac{d\lambda_3(t)}{dt} = -C_2 + (\mu_H + d_{KH} + \ r_H)\,\lambda_3(t) - r_H \lambda_4(t),$$

$$\frac{d\lambda_4(t)}{dt} = -\alpha_S \lambda_1(t) + (\mu_H + \alpha_S)\,\lambda_4(t),$$

$$\frac{d\lambda_5(t)}{dt} = \beta_D V[\lambda_5(t) - \lambda_6(t)] + \mu_D \lambda_5(t),$$

$$\frac{d\lambda_6(t)}{dt} = -C_3 + (r_D + \mu_D + d_{KD})\,\lambda_6(t) - r_D \lambda_7(t) - \sigma_E \lambda_{11}(t),$$

$$\frac{d\lambda_7(t)}{dt} = -\alpha_D \lambda_5(t) + (\mu_D + \alpha_D)\,\lambda_7(t),$$

$$\frac{d\lambda_8(t)}{dt} = (1 - u_2(t))\,\beta_O E_g(t)[\lambda_8(t) - \lambda_9(t)] + (\eta_1 + \mu_O)\,\lambda_8(t),$$

$$\frac{d\lambda_9(t)}{dt} = -C_4 + (\eta_2 + \mu_O)\,\lambda_9(t) - \eta_2 \lambda_{10}(t),$$

$$\frac{d\lambda_{10}(t)}{dt} = (\beta_D + \gamma)\,\lambda_{10}(t),$$

$$\begin{aligned}\frac{d\lambda_{11}(t)}{dt} &= (1-u_1(t))\,\beta_H H_S(t)\,[\lambda_1(t)-\lambda_2(t)]\\ &\quad + (1-u_2(t))\,\beta_O O_S(t)[\lambda_8(t)-\lambda_9(t)] + \mu_{E_g}\lambda_{11}(t),\end{aligned}$$

where final time conditions are

$$\lambda_i(T) = 0, \qquad i = 1, 2, \ldots, 11 \tag{31}$$

Moreover, the following characterization holds:

$$\begin{cases} u_1^*(t) = \max\{\min\{1, \dfrac{\beta_H[-\lambda_1(t)+\lambda_2(t)]E_g^*(t)H_S^*(t)}{\theta_1}\}, 0\}, \\ u_2^*(t) = \max\{\min\{1, \dfrac{\beta_O[-\lambda_8(t)+\lambda_9(t)]E_g^*(t)O_S^*(t)}{\theta_2}\}, 0\}. \end{cases} \tag{32}$$

*Proof.* The form of the adjoint system endowed with terminal conditions (31) results from Pontryagin's maximum principle by differentiating the Hamiltonian function (29) at the respective solutions of the state system (21).
Also, to get the characterizations of the optimal control given by (32), we use the optimality conditions.
After solving the equation

$$\frac{\partial \mathrm{H}(\mathcal{X}^*(t), u^*(t), \Lambda(t), t)}{du_i} = 0, \text{ for } i = 1, 2,$$

we obtain directly (32) by taking to account the boundedness condition given in (25). □

## 5 Numerical simulations

In this section, we present numerical simulations done using MATLAB to understand more the comportment of the model of transmission of Cystic Echinococcosis, and the influence of the two controls $u_1$ and $u_2$ representing health education and Vaccination of sheep with the EG95 vaccine respectively on the dynamic of the studied populations. Note that in this work, we use the data found in some studies elaborated in some regions of Morocco (Middle Atlas region, Ifrane and El Hajeb ) [2, 1, 18, 19].

The system of stats (1) is resolved in the sens $t: \ 0 \to T$ using the ode45 solver in MATLAB, with the initial conditions $HS(0) = 5367$, $H_E(0) = 0$, $H_I(0) = 0$, $H_R(0) = 0$, $D_S(0) = 700$, $D_I(0) = 37$, $D_R(0) = 3$, $O_S(0) = 797$ $O_I(0) = 159$, $V(0) = 34$, $E_g(0) = 240$, where the values of $H_S(0), D_S(0)$, and $O_S(0)$ are derived from [6] and the other initial conditions of (1) are assumed. However the optimality system consisting of control system (21) and the adjoint equations (31), is resolved in the opposite sens $t: \ T \to 0$, with the transversality conditions $\lambda_i(T) = 0, 1 \leq i \leq 11$, where $T = 60$ months.

We set in (22) also $C_1 = 50, C_2 = 90, C_3 = 70, C_4 = 60, \theta_1 = 10, \theta_2 = 10$.

**Parameter selection**

- Some parameters of the system (21) have been considered relying on online news, data fitting, or assumption because Echinococcosis is an overlooked disease and only few studies have been done on it in Morocco, and we found it difficult at the time of the study to find all parameters values in the studied areas. The values of the parameters of the system (21) are given in Table 2.
- Some of the table's parameters were based on a study in China because such parameters were unavailable in Morocco, and the choice of those specific values is explained below:
- Parameters $\mu_{Eg}$ and $B_E$ are considered universal because they depend on biological and environmental factors, such as humidity and temperature, that are comparable in regions where CE is endemic.
- For the parameter $r_H$: the recovery rate is also a universal parameter that depends on the physical context and is common among populations.
- Parameters $\mu_D$ and $\mu_O$: Specific parameters related to the regions studied in Morocco were not available at the time of the study, so we tried to derive those rates from other studies conducted in areas where farming practices and socioeconomic conditions do not vary much.
- So, we assumed that those parameters would be close to their real values in Morocco.

We calibrated the parameters derived from references in Table 2 in months.
-The parameters $B_H$, $B_D$, and $B_O$ are calculated using the same method as in [24].

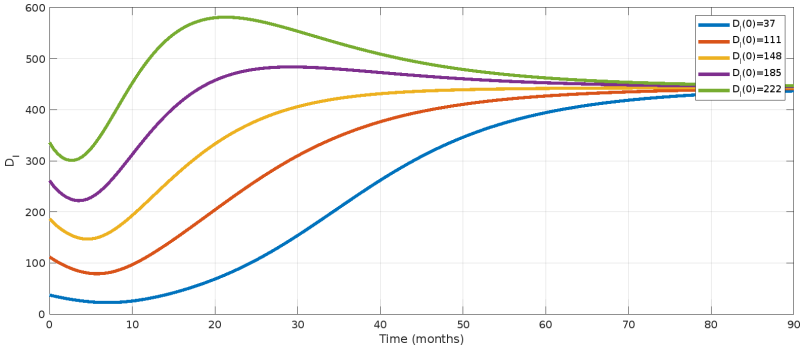


Figure 3: The influence of initial conditions on the number of infected dogs

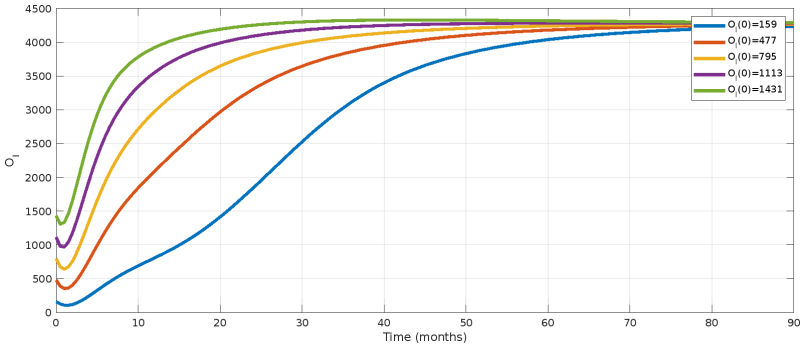


Figure 4: The influence of initial conditions on the number of infected livestock

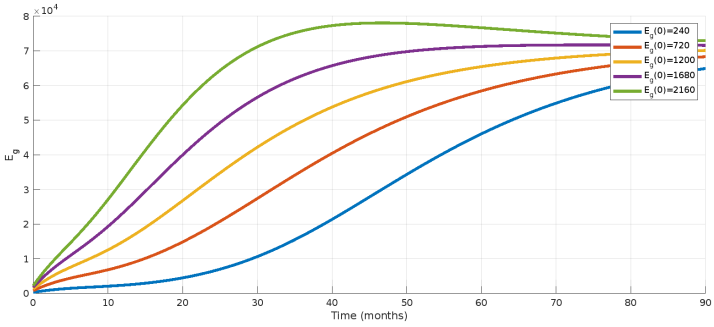


Figure 5: The influence of initial conditions on the number of CE eggs

Table 2: Parameters of system (1)

| Parameters | Values | Source |
|---|---|---|
| $B_H$ | 277.3 | Calculation |
| $\beta_H$ | 1.6% - 2.4% | [6] |
| $\mu_H$ | 5.167 | [15] |
| $\sigma_H$ | 0.0714 | Estimated |
| $d_{kh}$ | 2% - 3% | [20] |
| $r_H$ | 0.075 | [11] |
| $B_D$ | 56 | Calculation |
| $\beta_D$ | 2% - 41% | [1] |
| $\mu_D$ | 0.08 | [24] |
| $d_{kd}$ | 0.010 | Estimated |
| $r_D$ | 22.3% | [1] |
| $B_O$ | 121 | Calculation |
| $\beta_O$ | 0.007 | [9] |
| $\mu_O$ | 0.152 | [12] |
| $\eta_1$ | 0.37 | Estimated |
| $\eta_2$ | 0.37 | Estimated |
| $\gamma$ | 0.315 | Estimated |
| $B_E$ | 0.808 | [11] |
| $\mu_{Eg}$ | 10.42 | [11] |
| $\alpha_S$ | 8.5% | [22] |
| $\alpha_D$ | 4% | [1] |

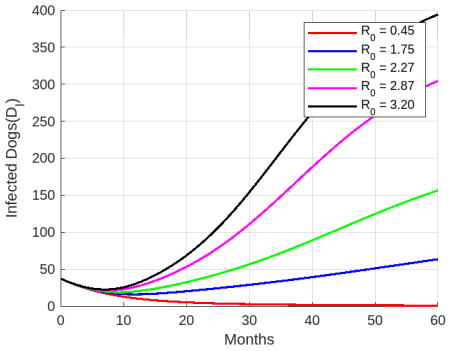


Figure 6: Sensitivity of the infected dogs to $R_0$

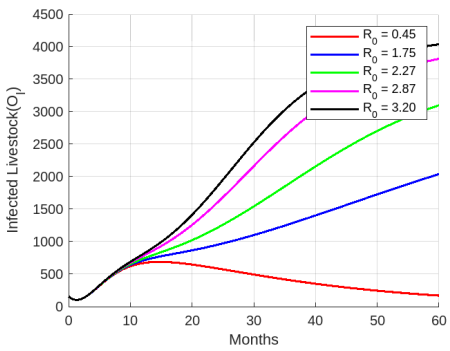


Figure 7: Sensitivity of the infected livestock to $R_0$

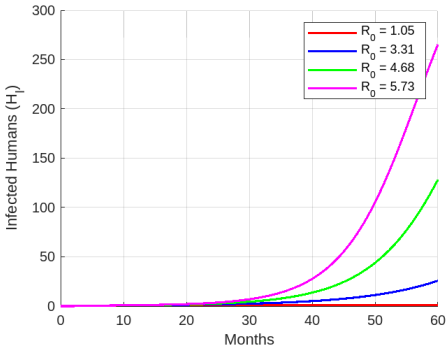


Figure 8: Sensitivity of the infected Humans to $R_0$

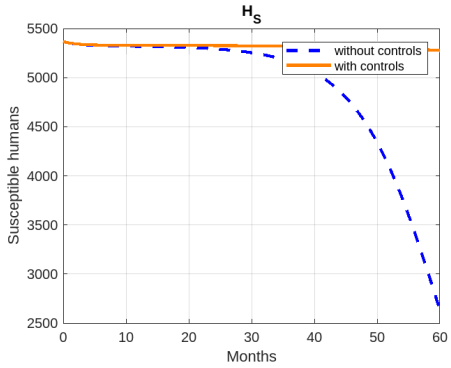


Figure 9: Evolution of CE in susceptible humans with and without control

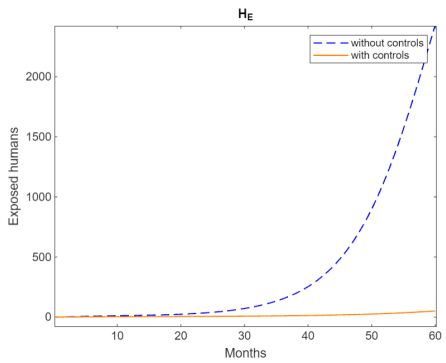


Figure 10: The evolution of CE in exposed humans with and without control

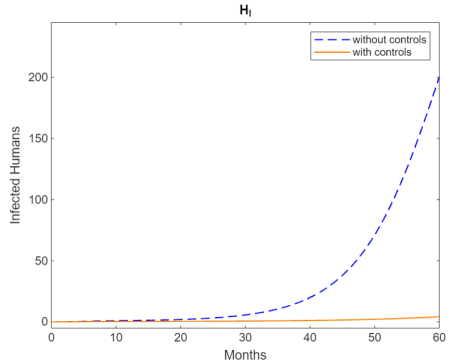


Figure 11: The evolution of CE in infected humans with and without control

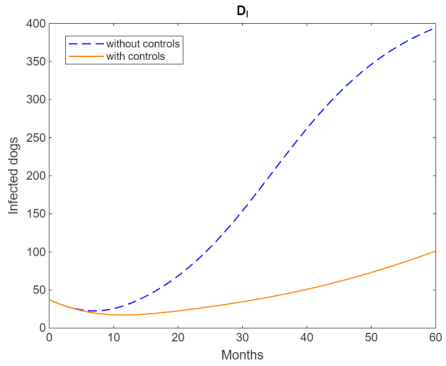


Figure 12: The evolution of CE in infected dogs with and without control

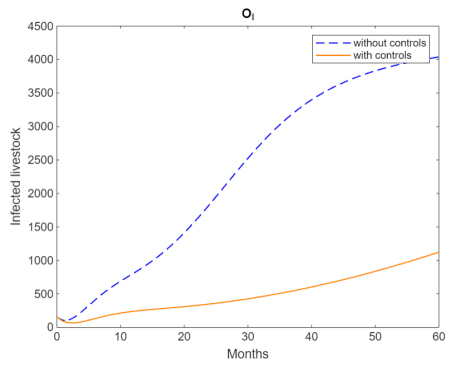


Figure 13: The evolution of CE in infected livestock with and without control

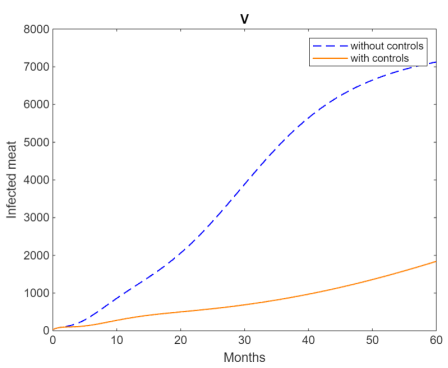


Figure 14: The evolution of infected meat with and without control

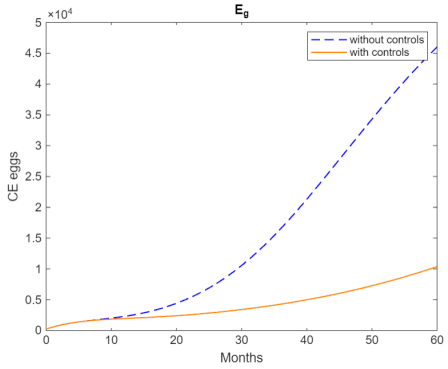


Figure 15: The evolution of CE eggs with and without control

## 5.1 Discussion

Figures 3, 4, and 5 show that despite the different values of initial conditions, the system reaches the same steady state, that is, in our case, the endemic equilibrium. The results of the simulations in the figures match the property describing that the endemic equilibrium is globally asymptotically stable. That confirms our theoretical results.

In Figures 8, 6, and 7, we changed the values of the basic reproduction number, and we studied the impact of those variations on the number of infected humans, dogs, and livestock, respectively. The values of $R_0$ were computed by modifying the rates $\beta_D$ and $\beta_O$ at each time.
We remarked that those two rates directly affect the $R_0$, and the bigger they get, the greater $R_0$ becomes. As a result, the infection becomes more important in the three populations with each rise of $\beta_D$ and $\beta_O$.
Based on the results of simulations in Figures 8, 6, and 7, we have chosen two control strategies that directly affect the rates of transmission for humans, dogs and livestock $\beta_H$, $\beta_D$, and $\beta_O$, by implementing health education and vaccination of livestock, respectively, as described in the previous section.

The purpose of numerical simulations given in Figures 10, 9, 11, 12, 13, 14, 15 is to evaluate the effectiveness and the impact of health education and

livestock vaccination on the spread of CE, those simulations are done while implementing the two controls $u_1$ and $u_2$ in the same time.

From the figures above, we remark that in the uncontrolled scenario (dashed blue lines ), there is a clear increase in the infected compartments including exposed and infected humans $H_E, H_I$ (after 20 months) in Figures 10 and 11, infected dogs $D_I$ (after nearly 7 months) in Figure 12, infected livestock $O_I$ (after almost 1 month) in Figure 13, infected meat $V$ (from the start) in Figure 14, and Echinococcosis eggs $E_g$ (from the start) in Figure 15. That means that without control there is a growing prevalence of the disease in the concerned regions of Morocco starting after month 20 of the study or even from the very beginning for some compartments and reaching the extreme after 60 months. In fact ,in this case, the parasite continues its life cycle normally, causing smooth transmission between the intermediate and definitive hosts. In this situation, we spot that the density of the concerned populations begins to increase $(H_E, H_I, D_I, O_I, V, E_g)$ until they stabilize at values representing the endemic equilibrium (12) after the 60 months.

However, while implementing the two controls $u_1$ and $u_2$ (orange lines), we observe an overall notable decrease in the prevalence of CE. In fact, the curves show that exposed and infected humans remain at a level low to zero (Figures 10 and 11). This means that the control $u_1$ consisting of health education was very beneficial in stopping so many people from becoming exposed or infected.
From Figure 12, we spot that infected dogs in the controlled case also grow, but at a significantly lower level ($\sim$100) compared to the uncontrolled case ($\sim$400). The other compartments $H_E, H_I, D_I, O_I, V, E_g$ follow the same trend. This means that the second control $u_2$ of sheep vaccination clearly decreases infected populations over time because it reduces the chance of carrying the parasite inside the intermediate hosts.
Less contamination in sheep leads to lower infection in dogs and also humans due to the break of the life-cycle of the parasite.

Even with the success of the optimal control strategy, the curves representing the Echinococcosis eggs in Figure 15 show that the density of these eggs remains important (approximately 12,000) and is expected to grow more af-

ter the study period, this can be explained by the very high resistance of the eggs to environmental conditions, and it is the source of the remaining infected cases among the populations of dogs and sheep (Figures 12 and 13). This suggests that more long-term control alternatives are needed to stop the disease more effectively by stopping or reducing the continued presence of CE eggs.

The results of the simulations show the effectiveness of health education and sheep vaccination controls in reducing disease in the regions studied in Morocco. However, if no control measures are taken in the country, CE disease will be sustained in human, livestock, and dog populations, which can be very dangerous to the health and food security of Moroccans in the next few years.

## 6 Conclusion

To understand the dynamic of the transmission of CE among humans, dogs, and livestock, we proposed a mathematical model based on the transmission cycle of the disease and taking into consideration the relapse after recovery for humans and dogs. The mathematical analysis of the model allowed us to define a basic reproduction number $R_0$ that determines the transmission of CE. The results suggested that, when $R_0 < 1$, the DFE $N_0$ is globally asymptotically stable and in this case, the disease dies. When $R_0 > 1$, the endemic equilibrium $N^*$ is globally asymptotically stable, the disease persistently remains. Those theoretical results are confirmed by some numerical simulations. Based on those results, we introduced two control strategies that are health education and sheep vaccination, and we performed the analysis using the optimal control theory. We proved the existence of optimal controls and characterized them using Pontryagin's maximum principle. The plots of the controlled system applied to some regions of Morocco facilitated the comparison between controlled and uncontrolled scenarios. The results revealed that health education combined with the vaccination of sheep with the EG95 vaccine is a must to reduce the prevalence of the disease in Morocco for the next decades. Therefore to sustain the prevention of Cystic

Echinococcosis in Morocco, it is crucial to adopt a continued investment in livestock vaccination, and raise awareness among citizens, especially in rural areas of the kingdom. Due to the relatively high founded level of CE eggs in the environment even with applying the current control strategies, in our future studies, we will integrate into the model other control measures such as inspection of infection in dog populations at least 4 times/year and slaughterhouse monitoring to reduce the environmental contamination. Moreover, we will try to address the randomness in the transmission process of CE disease by adding some stochastic parameters to the deterministic system, and analyze the new stochastic model.

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