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Mathematical Modeling and Optimal Control Strategies for Toxoplasmosis with Multiple Hosts

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Toxoplasmosis is one of the most prevalent infectious diseases in the world due to its harmful effects on both humans and animals. In this study, we present the dynamics of Toxoplasmosis in cat and mouse populations. We implement continuous vaccination for cats, horizontal transmission in both populations, and include vertical (congenital) transmission only in the cat population. Additionally, we consider the impact of oocysts of the parasite Toxoplasma gondii, which is responsible for causing the Toxoplasmosis infection. The paper offers a comprehensive analysis of positivity, boundedness, and stability of equilibrium points. Furthermore, we propose a controlled system accompanied by two suggested control strategies that aim to minimize the infected population while optimizing costs. To support the analytical findings, a numerical example is provided.

Keywords: *Toxoplasma gondii; vaccination; stability analysis; vertical transmissions, horizontal transmissions; optimal control.*

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1. Introduction

The protozoan parasite Toxoplasma gondii is the cause of toxoplasmosis, which affects a significant proportion of the global population. About 11% of people in the United States aged six years or older are thought to have been infected, and in certain parts of the world, the infection rate is more than 60%. Because such environmental conditions enhance the survival of the parasite's oocysts, the prevalence is noticeably higher in hot, humid climates and lower elevations. There are three major routes of transmission: foodborne, zoonotic (animal-to-human), and congenital (mother-to-child). Pregnant women and people with compromised immunity may be at risk, even though it is frequently asymptomatic in those with robust immune systems. There are several ways that toxoplasmosis can spread, such as eating raw meat or shellfish, drinking tainted water or food, coming into contact with cat excrement, pregnant women can transfer the disease to their unborn child, and in rare cases, organ donation or blood transfusion can also transmit the disease. Many people with toxoplasmosis have no symptoms at all, and symptoms might vary greatly. Severe instances of toxoplasmosis can cause damage to the brain or eyes, while mild ones may just cause muscle aches and lymph node swelling. Ocular toxoplasmosis may require treatment by an ophthalmologist, and infected neonates may show signs later in life, including potentially fatal brain or eye damage (see [1]).

Worldwide, Toxoplasma gondii is a protozoan parasite that is often found in domestic and wild animals, especially in cats [2,3]. Most cats that have Toxoplasma gondii do not display any symptoms, however, cats with compromised immune systems may display symptoms that affect multiple organs [4].

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According to Hartmann et al. (2013), cats only shed these environmentally resistant oocysts once in their lifetime, but infected cats may still pose a risk to people after that. According to [5,6], Toxoplasma gondii may infect a wide variety of warm-blooded animals, with significant seroprevalence rates seen in South American cats.

The diagram 1 given in [7] shows the life cycle of the Toxoplasma gondii in the environment.

The Felidae family, especially domestic cats, represents the primary group of definitive hosts for Toxoplasma gondii. Cats excrete unsporulated oocysts in their feces, a process that can produce a significant amount of oocysts and usually takes 1–3 weeks. The sporulation of these oocysts in the environment takes one to five days. After consuming soil, water, or plant material contaminated with these sporulated oocysts, intermediate hosts like birds and rats become infected. After ingestion, oocysts release sporozoites that differentiate into tachyzoites, which settle in muscle and neural tissue and mature into tissue cyst bradyzoites. Both consuming intermediate hosts that contain tissue cysts or swallowing sporulated oocysts directly can cause infection in cats [1]. Furthermore, wild game and animals raised for human consumption can become infected with tissue cysts by ingesting sporulated oocysts in the environment. Toxoplasma gondii can infect humans through various means, including eating raw meat from animals with tissue cysts, consuming food or drink contaminated with cat excrement or environmental samples (such as soil contaminated with feces), cleaning a pet cat's litter box, organ transplantation or blood transfusion, and transmission from mother to fetus transplacentally (congenital transmission) [1, 7, 8].

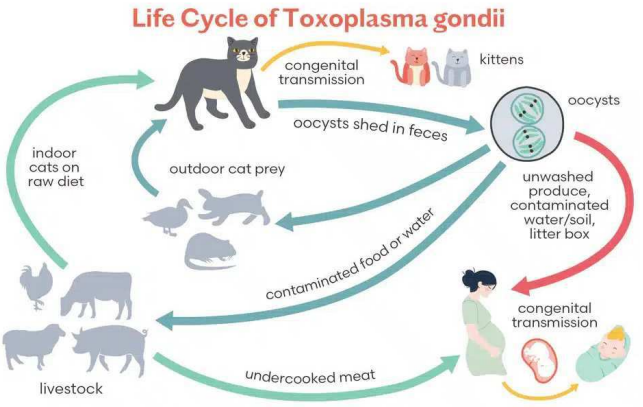

Figure 1. Life Cycle of Toxoplasma gondii.

Once inside the human host, the parasites form tissue cysts, which are commonly found in the brain, eyes, heart, and skeletal muscle. These cysts can persist throughout the host's life. Although stained biopsy specimens may reveal tissue cysts, serology is typically used for diagnosis. In cases of congenital infections, molecular methods like polymerase chain reaction (PCR) can be employed to detect Toxoplasma gondii DNA in amniotic fluid for diagnostic purposes [1].

A mathematical framework called the compartmental model, initiated by Kermack et al. [9], provides insights into the dynamics of infectious diseases such as mouse typhoid [10]. In compartmental epidemiological models, the population is divided into three separate groups: the susceptible (S) class, the infected (I) class, and the removed (R) class. When susceptible individuals come into contact with an infected individual, they become infected. Individuals in the infected class may eventually recover from the illness and transition to the removed class, where they are assumed to remain immune to it. This particular model is referred to as the "SIR" model. Mathematical epidemiological models have contributed significantly to understanding diseases caused by viruses and parasites [11–14]. Several studies [15, 16] have all used these models to study the dynamics of toxoplasmosis in specific populations, such as humans, cats, and mice. To reduce the spread of Toxoplasma gondii to people and other animals, several studies have particularly examined the effects of cat vaccination [16,17]. A number of studies [16, 18] have explicitly examined the effect of immunizing cats as a strategy to reduce Toxoplasma gondii transmission to humans and other animals. However, developing a vaccine against toxoplasmosis is still a top priority [19]. This work presents a mathematical model of Toxoplasma gondii transmission involving mice and oocysts, in which cats become infected by coming into contact with oocysts.

One key characteristic of the life cycle of oocysts is that they can be transmitted within populations via two routes: horizontal transmission between cats and mice, and vertical transmission from cats to their neonatal offspring [8, 20]. If horizontal transmission incurred no costs, it would evolve to

always increase rates. While the mouse population is divided into susceptible and infectious classes, with the recovered class excluded due to the parasite's systemic spread in infected mice, the model separates the cat population into susceptible, infectious, and vaccinated/recovered sub-populations. Toxoplasma gondii oocysts are the primary environmental source of infection, and the mathematical model explores the dynamics of disease transmission. Animals can get the disease in a number of ways, such as by consuming contaminated meat, ingesting feces from recently infected cats, or through vertical transmission from mother to fetus. According to several studies [17, 18], cats act as important reservoirs for the disease.

2. Toxoplasmosis model

In the following sections, we develop a mathematical model to describe the spreading dynamics of toxoplasmosis within cat population and mouse population. This model incorporates a constant vaccination program for cats [16–18] and includes the oocyst population, as these are primarily responsible for maintaining Toxoplasma gondii in the environment [21]. This inclusion is critical since cats are the only known excretors of Toxoplasma gondii oocysts [17, 21]. The model accounts for direct contact between cats and environmental oocysts. Environmental contamination by oocysts is well-documented [22], and the likelihood of acquiring Toxoplasma gondii infection is directly related to the quantity of oocysts present in the environment [23].

Infections generally originate from two sources: tissue cysts in prey and environmental oocysts [22]. However, prey infection ultimately traces back to oocyst shedding by cats. Our model is based on the premise that infection risk is proportional to the environmental oocyst load, which is influenced by the number of infected cats in preceding weeks [17]. This mathematical model employs a system of ordinary differential equations and includes parameters related to vaccination rates and oocyst survival times. We assume lifelong immunity post-recovery, as cats, while they can be reinfected and shed oocysts again, tend to shed significantly fewer oocysts in subsequent episodes [1, 2]. Given that the vaccine is assumed to provide complete immunity, we consolidate vaccinated and recovered cats into a single compartment.

The model also considers vertical transmission within the cat population, supported by several studies [24, 25] and evidence of lactational transmission of Toxoplasma gondii [25]. A natural exponential decay is applied to the oocysts. While we do not include a subpopulation of exposed oocysts in this model, future work may incorporate this aspect, as oocysts become infective only after 24 to 48 hours post-shedding. Sporulated oocysts can survive for extended periods under typical environmental conditions [26].

The model proposed is presented by the following ordinary differential equations system

$$\begin{aligned}
\frac{dS(t)}{dt} &= \mu\, V_R(t) - \beta\, S(t)\, O(t) - \gamma S(t) + \mu\, t_{v_2} I(t),\\
\frac{dI(t)}{dt} &= \beta\, S(t)\, O(t) - \alpha\, I(t) + \mu\, t_{v_1} I(t),\\
\frac{dV_R(t)}{dt} &= \alpha\, I(t) + \gamma\, S(t) - \mu\, V_R(t),\\
\frac{dO(t)}{dt} &= k\, I(t) - \mu_0 O(t),\\
\frac{dS_m(t)}{dt} &= (b - \mu_m)\, S_m - \beta_m S_m(t)\, O(t),\\
\frac{dI_m(t)}{dt} &= (b - \mu_m)\, I_m + \beta_m S_m(t)\, O(t).
\end{aligned} \tag{1}$$

The total cat population is denoted $N(t)$ and it is classified into three disjoint sub-populations: susceptible cats $S(t)$, infected cats $I(t)$, and vaccinated/recovered cats $V_R(t)$. The mice population $N_m(t)$ is classified in two different sub-populations: susceptible mice $S_m(t)$ and infectious mice $I_m(t)$. Assuming the rate of births is the same as the natural death rate (μ), then the population of cats stays constant

($\dot{N}(t) = 0$). However, susceptible cats or mice become infected after direct connection with oocysts at the two rates (β) and (β_m), respectively. It is assumed that oocysts become infective immediately after being shed by cats. Additionally, there is an oocyst population $O(t)$ in the environment. The parameter $k > 0$ is the rate of appearance of new oocysts in the environment per infected cat. Susceptible cats move to the vaccinated sub-population $V_R(t)$ at rate (γ), while infectious cats move to $V_R(t)$ at rate (α). The fluctuation in the oocysts number $O(t)$ at time t is in relation with the amount of infectious cats $I(t)$. Note also that (μ_0) is oocysts death rate. In the system model (1) the two parameters (t_{v_1}) and (t_{v_2}) denote vertical transmission rates, which are crucial for understanding the persistence of toxoplasmosis in cat populations. Vertical transmission refers to the transfer of infection from mother to offspring, either during pregnancy or through breastfeeding. The parameter (t_{v_1}) represents the rate at which infected mother cats transmit the infection to their kittens. This mode of transmission helps sustain the prevalence of Toxoplasma gondii within cat populations, even in the absence of environmental oocysts [27, 28]. The parameter t_{v_2} accounts for the offspring of infected mothers being born susceptible but not immediately infected, highlighting the potential for rapid re-infection cycles due to close contact with the mother or contaminated environments [29]. Incorporating (t_{v_1}) and (t_{v_2}) into the model allows for a more accurate simulation of disease dynamics and aids in evaluating effective control strategies.

Furthermore, we assume that the total populations of cats and mice are scaled: $N(t) = S(t) + I(t) + V_R(t) = 1$, and $N_m(t) = S_m(t) + I_m(t)$ respectively, without loss of generality. Note that the variables related to births (b) and deaths (μ_m) in the mouse population disappear due to the scaling of the population. Since $I_m(t) = 1 - S_m(t)$ and $V_R(t) = 1 - S(t) - I(t)$ taking into account the scaled populations we can reduce the model (1) to a simpler one as follows

$$\begin{aligned}
\frac{dS(t)}{dt} &= \mu - \beta\, S(t)\, O(t) - (\mu + \gamma)\, S(t) - \mu\, I(t) + \mu\, t_{v_2} I(t),\\
\frac{dI(t)}{dt} &= \beta\, S(t)\, O(t) - \alpha\, I(t) + \mu\, t_{v_1} I(t),\\
\frac{dO(t)}{dt} &= k\, I(t) - \mu_0 O(t),\\
\frac{dS_m(t)}{dt} &= -\beta_m S_m(t)\, O(t),
\end{aligned} \tag{2}$$

with initial conditions

$$S(0) \geqslant 0, \quad I(0) \geqslant 0, \quad O(0) \geqslant 0, \quad S_m(0) \geqslant 0. \tag{3}$$

This paper aims to analyze the dynamics of toxoplasmosis. We take in count two types of transmissions (horizontal and vertical). By deriving the basic reproduction number, that is expressed by the system parameters, we prove sufficient conditions for the stability of two equilibrium points of the system. Furthermore, we present a description of optimal control to minimize the numbers of infected cats and oocysts in the environment.

The rest of this paper is organized in the following form. In Section 3, we determine the equilibrium values of the model (2), and we show the existence, positivity and boundlessness of system solution to guarantee that our system is mathematically and biologically well-posed. The characterization of optimal controls is investigated in Section 4. In Section 5, we illustrate our analytical results with numerical simulations. In the end, we finish the paper with conclusion and future directions,

$$\begin{aligned}
\frac{dS}{dt} &= \mu\,(1 - S - I) - \beta\, S\, O - \gamma\, S + \mu\, t_{v_2} I,\\
\frac{dI}{dt} &= \beta\, S\, O - \alpha I + \mu\, t_{v_1}\, I,\\
\frac{dV_R}{dt} &= \alpha\, I + \gamma\, S - \mu\,(1 - S - I).
\end{aligned}$$

3. Basic mathematical analysis

The proposed model below describes the dynamics of a biological population, therefore, the solutions of the system must be nonnegative and bounded.

We can also verify the existence and uniqueness of the solutions of the system. Thus, we can rewrite the system (2) by

$$\psi(t) = A(X(t)) + B(X(t)),$$

where

$$X(t) = \begin{bmatrix} S(t) \\ I(t) \\ O(t) \\ S_m(t) \end{bmatrix}, \quad \Psi(t) = \begin{bmatrix} \frac{dS(t)}{dt} \\ \frac{dI(t)}{dt} \\ \frac{dO(t)}{dt} \\ \frac{dS_m(t)}{dt} \end{bmatrix},$$

$$A = \begin{bmatrix} -\mu+\gamma & \mu+\mu t_{v_2} & 0 & 0 \\ 0 & -\alpha+\mu t_{v_1} & 0 & 0 \\ 0 & k & -\mu & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad B(X(t)) = \begin{bmatrix} \mu - \beta\, S(t)\, O(t) \\ \beta\, S(t)\, O(t) \\ 0 \\ \beta_m S_m(t)\, O(t) \end{bmatrix}.$$

There exists a Lipschtiz constant L such that $\|B(X_1(t)) - B(X_2(t))\| \leqslant L\|X_1(t) - X_2(t)\|$, then $\|\psi(X_1) - \psi(X_2)\| \leqslant W\|X_1(t) - X_2(t)\|$, where $W = \max(L, \|A\|) < \infty$. This shows that the function ψ is uniformly Lipschitz continuous, and with the restrictions on $S(t) \geqslant 0$, $I(t) \geqslant 0$, $O(t) \geqslant 0$ and $S_m(t) \geqslant 0$, we prove the existence of a solution for the system (2) [30].

3.1. Positivity

Since the populations must remain nonnegative at all times, the following theorem shows that every solution of (2), with nonnegative initial values (3) will remain in $\mathbb{R}^4_+$.

Theorem 1. *For any initial conditions (3), the solutions $S(t)$, $I(t)$, $O(t)$, $S_m(t)$ of system (2) are positive for all $t \geqslant 0$.*

Proof. We assume that the initial values $(S(0), I(0), O(0), S_m(0))$ of system (2) are in $\mathbb{R}^4_+$. We prove that system (2) is a non-negative dynamical system using Proposition 2.1 in [31]. Then, we have

$$\left.\frac{dS}{dt}\right|_{S=0} = \mu\,(1-I(t)) + \mu\, t_{v_2} I(t) \geqslant 0, \quad \left.\frac{dI}{dt}\right|_{I=0} = \beta\, S(t)\, O(t) \geqslant 0, \quad \left.\frac{dO}{dt}\right|_{O=0} = k\, I(t) \geqslant 0, \quad \left.\frac{dS_m}{dt}\right|_{S_m=0} = 0.$$

Then, $\mathbb{R}^4_+$ is an invariant set. Therefore, if the initial values belong to the nonnegative cone, every solution will remain in $\mathbb{R}^4_+$. That is complete the proof. ∎

3.2. Boundedness of solutions

The following theorem presents the boundedness of solution. In order to show this, we consider the following set:

$$\Omega = \left\{ (S, I, O, S_m) \in \mathbb{R}^4_+ : 0 \leqslant S \leqslant \frac{\mu(1+t_{v_2})}{\mu+\gamma},\ 0 \leqslant I \leqslant \frac{k\,\beta\,\mu\,(1+t_{v_1}) + \mu\, t_{v_2} \mu_0 (\mu+\gamma)}{\alpha\,\mu_0(\mu+\gamma)}, \right.$$
$$\left. 0 \leqslant O \leqslant \frac{k}{\mu_0},\ I + S \leqslant 1,\ I_m + S_m \leqslant 1 \right\}. \quad (4)$$

Theorem 2. *The set Ω is positively invariant with respect to system (2), with the initial conditions (3).*

Proof. Taking the third equation of system (2)

$$\frac{dO(t)}{dt} = k\, I(t) - \mu_0 O(t),$$

and since $I(t) \leqslant 1$, then

$$\frac{dO(t)}{dt} \leqslant k - \mu_0 O(t).$$

Therefore, according to Birkhoff and Rota's [30] differential inequality, we get

$$O(t) \leqslant e^{-\mu_0 t}\left(\frac{k}{\mu_0} + O(0)(e^{\mu_0 t} - 1)\right), \quad \forall t \geqslant 0.$$

Then, since $O(0) \leqslant \frac{k}{\mu_0}$ we have $O(t) \leqslant \frac{k}{\mu_0}$ for all $t \geqslant 0$. The first equation gives

$$\begin{aligned}\frac{dS(t)}{dt} &= -\beta\, S(t)\, O(t) - (\mu + \gamma)\, S(t) + \mu\,(1 - (1 - t_{v_2})\, I(t)) \\ &\leqslant \mu - (\mu + \gamma)\, S(t) + \mu\, t_{v_2} I(t),\end{aligned}$$

and since $I(t) \leqslant 1$, then

$$\frac{dS(t)}{dt} \leqslant \mu\,(1 + t_{v_2}) - (\mu + \gamma)\, S(t),$$

which implies

$$S(t) \leqslant e^{-(\mu+\gamma)t}\, S(0) + \frac{\mu\,(1 + t_{v_2})}{\mu + \gamma}\big(1 - e^{-(\mu+\gamma)\,t}\big), \quad \forall t \geqslant 0,$$

with $S(0)$ the initial condition. Letting $t \to \infty$, we get

$$\lim_{t\to\infty} S(t) \leqslant \frac{\mu\,(1 + t_{v_2})}{\mu + \gamma}.$$

Then, from the second equation

$$\begin{aligned}\frac{dI(t)}{dt} &= \beta\, S(t)\, O(t) - \alpha\, I(t) + \mu\, t_{v_1}\, I(t) \\ &\leqslant \frac{k\,\beta\,\mu\,(1 + t_{v_2})}{\mu_0(\mu + \gamma)} + \mu\, t_{v_1}\, I(t),\end{aligned}$$

and since $I(t) \leqslant 1$, then

$$\frac{dI(t)}{dt} \leqslant \frac{k\beta\mu(1 + t_{v_1})}{\mu_0(\mu + \gamma)} + \mu\, t_{v_2} - \alpha\, I(t),$$

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$$I(t) \leqslant e^{-\alpha t} I(0) + \frac{k\,\beta\,\mu\,(1 + t_{v_1}) + \mu\, t_{v_2}\mu_0(\mu + \gamma)}{\alpha\,\mu_0(\mu + \gamma)}\big(1 - e^{-\alpha\, t}\big), \quad \forall t \geqslant 0$$

with $I(0)$ the initial condition. Letting $t \to \infty$, we get

$$\lim_{t\to\infty} I(t) \leqslant \frac{k\,\beta\,\mu\,(1 + t_{v_1}) + \mu\, t_{v_2}\mu_0(\mu + \gamma)}{\alpha\,\mu_0(\mu + \gamma)}.$$

Therefore, we can focus on the system (2) in the region Ω defined by (4). ∎

Remark 1. According to Theorems 1 and 2, we confirm that the problem is mathematically and biologically well defined.

3.3. Basic reproduction number

Using the next-generation matrix method developed by Van den Driessche and Watmough [32], we can express the basic reproduction number of the system. Here, the infected compartment is I. The outflow term $\mathfrak{V}$ and the new-infection terms $\mathfrak{F}$ are as follows

$$\mathfrak{F} = \begin{bmatrix}\beta S(t) O(t) \\ 0\end{bmatrix}, \quad \mathfrak{V} = \begin{bmatrix}\alpha\, I(t) - \mu\, t_{v_1} I(t) \\ -k\, I(t) + \mu_0 O(t)\end{bmatrix}.$$

The Jacobian matrix of $\mathcal{F}$ and $\mathcal{V}$ are expressed as follows:

$$\mathcal{F} = \begin{bmatrix}0 & \beta S(t) \\ 0 & 0\end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix}\alpha - \mu t_{v_1} & 0 \\ -k & \mu_0\end{bmatrix}.$$

Then the matrix $\mathcal{F} \times \mathcal{V}^{-1}$ is given as follow:

$$\mathcal{F} \times \mathcal{V}^{-1} = \begin{bmatrix}\frac{\beta\, k\, S(t)}{\mu_0(\alpha - \mu\, t_{v_1})} & \frac{\beta\, S(t)}{\mu_0} \\ 0 & 0\end{bmatrix}.$$

The linearized form of the matrix $\mathcal{F} \times \mathcal{V}^{-1}$ at the equilibrium E_0 is expressed as follows,

$$(\mathcal{F} \times \mathcal{V}^{-1})(E_0) = \begin{bmatrix} \frac{\beta\, k\, \mu}{\mu_0(\mu+\gamma)(\alpha-\mu\, t_{v_1})} & \frac{\beta\, \mu\, S(t)}{\mu_0(\mu+\gamma)} \\ 0 & 0 \end{bmatrix}.$$

The dominant eigenvalue of $\mathcal{F} \times \mathcal{V}^{-1}(E_0)$ represents the basic reproduction numbers, and it is given by $R_0 = \rho(\mathcal{F} \times \mathcal{V}^{-1})$ (with ρ the spectral radius of $\mathcal{F} \times \mathcal{V}^{-1}(E_0)$). Then,

$$R_0 = \frac{\beta\, k\, \mu}{\mu_0(\mu+\gamma)(\alpha-\mu\, t_{v_1})}.$$

Remark 2. For a deterministic system, the reproduction number has a critical signification. It is the expected amount of secondary cases generated by one infective individual introduced into a population constituted totally by susceptible individuals in an average period of time. It determines the eradication or continuance of an epidemic.

3.4. Equilibrium points

In this part, we calculate the equilibrium points of the system (2) to study their stability. For this, we solve the following system

$$\begin{aligned} \mu - \beta\, S(t)\, O(t) - (\mu+\gamma)S(t) - \mu\, I(t) + \mu\, t_{v_2} I(t) &= 0, \\ \beta\, S(t)\, O(t) - \alpha\, I(t) + \mu\, t_{v_1} I(t) &= 0, \\ k\, I(t) - \mu_0 O(t) &= 0, \\ -\beta_m S_m(t)\, O(t) &= 0. \end{aligned}$$

The equilibrium points are given as follows:

• **Disease-free equilibrium state.** The free equilibrium state stands for the absence of infection. Thus, the infected and oocysts groups will be empty, and the whole population will incorporate only susceptible. Hence, the free equilibrium of the model is given by $E_0\big(\frac{\mu}{\mu+\gamma}; 0; 0; S_m^*\big)$.

• **Endemic equilibrium state.** The endemic equilibrium state is when both the infection and oocysts are present in the system. Thus, we have $E_e(S^*; I^*; O^*; S_m^*)$, such that

$$\begin{aligned} S^* &= \frac{\mu_0(\alpha - \mu t_{v_1})}{\beta k}, \\ I^* &= \frac{\mu\beta k - \mu_0(\mu+\gamma)(\alpha-\mu t_{v_1})}{\beta k \alpha} = \frac{\mu_0(\mu+\gamma)(\alpha-\mu t_{v_1})}{\beta k \alpha}(R_0 - 1), \\ O^* &= \frac{\mu\beta k - \mu_0(\mu+\gamma)(\alpha-\mu t_{v_1})}{\beta \mu_0 \alpha} = \frac{\mu_0(\mu+\gamma)(\alpha-\mu t_{v_1})}{\beta \mu_0 \alpha}(R_0 - 1), \\ S_m^* &= 0. \end{aligned}$$

This endemic equilibrium exists and feasible biologically if $\alpha - \mu\, t_{v_1} > 0$ and $R_0 > 1$.

3.5. Stability

Theorem 3. *Given $R_0 < 1$, the equilibrium point E_0 is locally asymptotically stable.*

Proof. The Jacobian matrix of system (2) at the equilibrium point E_0 is given by

$$J_{E_0} = \begin{bmatrix} -\gamma-\mu & \mu\, t_{v_2} - \mu & -\frac{\beta\,\mu}{\mu+\gamma} \\ 0 & \mu\, t_{v_1} - \alpha & \frac{\beta\,\mu}{\mu+\gamma} \\ 0 & k & -\mu_0 \end{bmatrix}. \tag{5}$$

Its characteristic polynomial equation is given by,

$$P_0(X) = [X + \mu + \gamma]\left[X^2 + q_{01}X + q_{02}\right].$$

It is clear that, the first eigenvalue associated with the matrix (5) is $X_1 = -\mu - \gamma < 0$. The rest eigenvalues of the matrix (5) can be obtained by solving the following equation:

$$X^2 + q_{01}X + q_{02} = 0, \tag{6}$$

where

$$q_{01} = -\mu\, t_{v_1} + \alpha + \mu_0,$$
$$q_{02} = -\mu_0(\mu\, t_{v_1} - \alpha) - \frac{k\,\beta\,\mu}{\mu+\gamma} = -\mu_0(-\mu\, t_{v_1} + \alpha)\,[R_0 - 1]\,.$$

Since $\mu\, t_{v_1} < \alpha$ and $R_0 < 1$ we obtain $q_{01} > 0$ and $q_{02} > 0$. Then, according to Rooth–Hurwitz criteria [33], we conclude that the real parts of the eigenvalues of (6) are strictly negative. Therefore, the free equilibrium point E_0 is locally asymptotically stable. ∎

Theorem 4. *Assuming that $R_0 > 1$, the equilibrium point E_e is locally asymptotically stable.*

Proof. At the equilibrium point E_e, the Jacobian matrix of system (2) is defined as follows

$$J_{E_e} = \begin{bmatrix} -\frac{\beta\,\mu\,k+\mu_0(\mu+\gamma)(\mu\, t_{v_1}-\alpha)}{\mu_0\alpha} - \gamma - \mu & \mu\, t_{v_2} - \mu & -\frac{\mu_0(-\mu\, t_{v_1}+\alpha)}{k} \\ \frac{\beta\,\mu\,k+\mu_0(\mu+\gamma)(\mu\, t_{v_1}-\alpha)}{\mu_0\alpha} & \mu\, t_{v_1} - \alpha & \frac{\mu_0(-\mu\, t_{v_1}+\alpha)}{k} \\ 0 & k & -\mu_0 \end{bmatrix}.$$

Its characteristic polynomial equation $P_e(X)$ is as follows:

$$P_e(X) = X^3 + q_{11}X^2 + q_{12}X + q_{13}, \tag{7}$$

where

$$q_{11} = \frac{\alpha\,\mu_0(\alpha - \mu t_{v_1}) + \gamma\,\mu\, t_{v_1}\mu_0 + \mu^2 t_{v_1}\mu_0 + k\,\beta\,\mu + \alpha\,\mu_0^2}{\mu_0\alpha} > 0,$$
$$q_{12} = \frac{\mu\left(\gamma\, t_{v_1}\mu_0^2 + \mu\, t_{v_1}\mu_0 + k\,\alpha\,\beta + k\,\beta\,\mu_0\right)}{\mu_0\,\alpha} > 0,$$
$$q_{13} = \mu_0(\gamma+\mu)(\alpha - \mu\, t_{v_1})\,(R_0 - 1) > 0,$$

$$\begin{aligned} q_{11}q_{12} - q_{13} = {} & \big[-\alpha\gamma\mu^2 t_{v1}^2\mu_0^3 + \gamma^2\mu^2 t_{v1}^2\mu_0^3 + \gamma\mu^3 t_{v1}^2\mu_0^3 - \zeta\alpha^2\beta\mu^2 t_{v1}\mu_0 + \zeta\alpha\beta\gamma\mu^2 t_{v1}\mu_0 + \zeta\alpha\beta\mu^3 t_{v1}\mu_0 \\ & - \zeta\alpha\beta\mu^2 t_{v1}\mu_0^2 + 2\zeta\beta\gamma\mu^2 t_{v1}\mu_0^2 + \eta\beta\mu^3 t_{v1}\mu_0^2 + \alpha^2\gamma\mu t_{v1}\mu_0^3 + \alpha\gamma\mu t_{v1}\mu_0^4 + \gamma\mu^3 t_{v1}^2\mu_0^2 \\ & - \alpha\mu^3 t_{v1}^2\mu_0^2 + \mu^4 t_{v1}^2\mu_0^2 + \zeta^2\alpha\beta^2\mu^2 + \zeta^2\beta^2\mu^2\mu_0 + \zeta\alpha^3\beta\mu\mu_0 + 2\zeta\alpha^2\beta\mu\mu_0^2 + \zeta\beta\mu^3 t_{v1}\mu_0 \\ & + \zeta\alpha\beta\mu\mu_0^3 + \alpha^2\mu^2 t_{v1}\mu_0^2 + \alpha\mu^2 t_{v1}\mu_0^3\big] \\ & - \big[\alpha^2\gamma\mu t_{v1}\mu_0^3 + \alpha^2\mu^2 t_{v1}\mu_0^3 + \zeta\alpha^2\beta\mu\mu_0^2 - \alpha^3\gamma\mu_0^3 - \alpha^3\mu\mu_0^3\big] \\ = {} & \gamma^2\mu^2 t_{v1}^2\mu_0^3 + \gamma\mu^3 t_{v1}^2\mu_0^3 + \zeta\alpha\beta\gamma\mu^2 t_{v1}\mu_0 + \zeta\alpha\beta\mu^3 t_{v1}\mu_0 + 2\zeta\beta\gamma\mu^2 t_{v1}\mu_0^2 + \eta\beta\mu^3 t_{v1}\mu_0^2 \\ & + \alpha\gamma\mu t_{v1}\mu_0^4 + \gamma\mu^3 t_{v1}^2\mu_0^2 + \mu^4 t_{v1}^2\mu_0^2 + \zeta^2\alpha\beta^2\mu^2 + \zeta^2\beta^2\mu^2\mu_0 + \zeta\alpha\beta\mu\mu_0^3 + \zeta\beta\mu^3 t_{v1}\mu_0 \\ & + \alpha\mu^2 t_{v1}\mu_0^3 + \big[-\alpha\gamma\mu^2 t_{v1}^2\mu_0^3 + \alpha^2\gamma\mu t_{v1}\mu_0^3\big] + \big[2\zeta\alpha^2\beta\mu\mu_0^2 - \zeta\alpha\beta\mu^2 t1\mu_0^2 - \zeta\alpha^2\beta\mu\mu_0^2\big] \\ & + \big[-\alpha^2\gamma\mu t_{v1}\mu_0^3 + \alpha^3\gamma\mu_0^3\big] + \big[-\alpha^2\mu^2 t_{v1}\mu_0^3 + \alpha^3\mu\mu_0^3\big] \\ & + \big[-\zeta\alpha^2\beta\mu^2 t_{v1}\mu_0 + \zeta\alpha^3\beta\mu\mu_0\big] + \big[-\alpha\mu^3 t_{v1}^2\mu_0^2 + \alpha^2\mu^2 t_{v1}\mu_0^2\big] \\ = {} & \gamma^2\mu^2 t_{v1}^2\mu_0^3 + \gamma\mu^3 t_{v1}^2\mu_0^3 + \zeta\alpha\beta\gamma\mu^2 t_{v1}\mu_0 + \zeta\alpha\beta\mu^3 t_{v1}\mu_0 + 2\zeta\beta\gamma\mu^2 t_{v1}\mu_0^2 + \eta\beta\mu^3 t_{v1}\mu_0^2 \\ & + \alpha\gamma\mu t_{v1}\mu_0^4 + \gamma\mu^3 t_{v1}^2\mu_0^2 + \mu^4 t_{v1}^2\mu_0^2 + \zeta^2\alpha\beta^2\mu^2 + \zeta^2\beta^2\mu^2\mu_0 + \zeta\alpha\beta\mu\mu_0^3 + \zeta\beta\mu^3 t_{v1}\mu_0 \\ & + \alpha\mu^2 t_{v1}\mu_0^3 + \big[\alpha\gamma\mu t_{v1}\mu_0^3 + \zeta\alpha\beta\mu\mu_0^2 + \alpha^2\gamma\mu_0^3 + \alpha^2\mu\mu_0^3 + \zeta\alpha^2\beta\mu\mu_0 + \alpha\mu^2 t_{v1}\mu_0^2\big][\alpha - \mu t_{v1}] \\ > {} & 0. \end{aligned}$$

Since $\alpha - \mu\, t_{v1} > 0$ and according to Rooth–Hurwitz criteria [33], we conclude that the real parts of the eigenvalues of (7) are strictly negative. Therefore, the endemic equilibrium point E_e is locally asymptotically stable. ∎

4. Optimal control problem

Health problems have always been of a great issue for governments in all countries. Several governments have proposed health intervention strategies to manage and eradicate the spread of infectious diseases. The problem is that the cost of transforming these strategies into the field is very high. Therefore, it is

necessary to develop an efficient method that reduces the number of infections and the costs associated with it.

Controlling toxoplasmosis transmission involves a two-pronged approach focusing on treatments for infected cats and control of oocysts in the environment. It is primarily imperative to treat cats that have contracted the infection since they are the main hosts that excrete Toxoplasma gondii oocysts. Frequent veterinary examinations and timely administration of antiprotozoal drugs, such as azithromycin or clindamycin, might decrease oocyst shedding. To reduce the amount of oocysts in the environment, it is necessary to manage and reduce them. Developing strict sanitation protocols, including routinely cleaning and disinfecting cat litter boxes, disposing of cat waste properly, and ensuring that cat feces are properly disposed of to prevent contamination of water and soil, is necessary for this. Additionally, educating cat owners about the importance of these practices can further reduce environmental contamination and the number of infected cats, ultimately controlling the spread of toxoplasmosis [1]. In this section, we investigate two optimal control strategies:

1. The control by treatment strategy for infected cats, which is the time-dependent control variable denoted by $w_1(t)$.
2. The control via managing and treating oocysts in the environment. This control targets the oocyst population, and is represented by the time-dependent variable $w_2(t)$.

After considering the proposed controls $w_1(t)$ and $w_2(t)$, we get the following controlled system

$$\begin{aligned}
\frac{dS(t)}{dt} &= \mu - \beta\, S(t)\, O(t) - (\mu+\gamma)\, S(t) + w_1 I(t) - \mu\, I(t) + \mu\, t_{v_2} I(t),\\
\frac{dI(t)}{dt} &= \beta\, S(t)\, O(t) - \alpha\, I(t) - w_1 I(t) + \mu\, t_{v_1} I(t),\\
\frac{dO(t)}{dt} &= k\, I(t) - \mu_0 O(t) - w_2 O(t),\\
\frac{dS_m(t)}{dt} &= -\beta_m S_m(t)\, O(t).
\end{aligned} \tag{8}$$

The aim is to minimize the following objective functional

$$J(w_1, w_2) = \int_0^T \left(I(t) + O(t) + \frac{C_1 w_1^2(t)}{2} + \frac{C_2 w_2^2(t)}{2} \right) dt, \tag{9}$$

where the parameters $C_1 \geqslant 0$ and $C_2 \geqslant 0$ balance the size of the terms and represent the weight factor's characterization based on the costs and benefits of the treatment. The purpose is to minimize the objective function presented in the equation (9) by decreasing the numbers of the infected cats and oocysts in the environment. To put it differently, we are looking for an optimal control $w^* = (w_1^*, w_2^*)$ which satisfies

$$J(w_1^*, w_2^*) = \min\{J(w_1, w_2) \colon (w_1, w_2) \in W\},$$

where W is the control's set defined by:

$$W = \big\{ w = (w_1, w_2) \colon w_{i=1,2} \text{ measurable},\ 0 \leqslant w_i(t) \leqslant 1,\ t \in [0, t_f] \big\}.$$

4.1. Optimal control existence

According to Fleming and Rishel [34], we can obtain the existence of the optimal control w^*. Thus, the five following steps must be checked:

1. W is a nonempty set.
2. W is convex and closed.
3. solution of (8) is bounded by a linearity in the state and control variables.
4. The objective function integrand is convex.
5. There exist $C_1 > 0$, $C_2 > 0$ and $\psi > 1$ such that the integrand $L(S, I, O, S_m)$ of the objective functional satisfies $L(S, I_N, I_R, R) \geqslant C_1 + C_2(\|w_1\|^2 + \|w_2\|^2)^{\frac{\psi}{2}}$.

The following theorem shows the existence of the optimal control.

Theorem 5. *For the controlled system (8), there exist optimal controls* $(w_1^*, w_2^*) \in W$ *such that* $J(w_1^*, w_2^*) = \min\{J(w_1, w_2) \colon (w_1, w_2) \in W\}$.

Proof. The existence of the optimal control can be obtained using a result by Fleming and Rishel [34], checking the above steps. According to Lukes [35], an existence result was used to give the existence of solution of system (8) with bounded coefficients, which gives condition 1. By definition the control set W is convex and closed. Using the boundedness of the solution and it is linearity in W, the right side of (8) verify the third condition. The integrand in the objective functional (9) is convex on W. In addition, we can easily see that there exist a constant $\psi > 1$ and positive numbers C_1 and $C_2 > 0$ satisfying $L(S, I, O, S_m) \geqslant C_1 + C_2(\|w_1\|^2 + \|w_2\|^2)^{\frac{\psi}{2}}$. This complete the proof. ∎

4.2. Optimal control characterization

This subsection provides the necessary conditions for the optimal control problem using the Pontryagin's Maximum Principle [36]. In order to characterize the optimal control $w^* = (w_1^*, w_2^*)$, the Hamiltonian H is defined from the formulation of objective functional (9) as follows:

$$H = I(t) + O(t) + \frac{C_1}{2} w_1^2(t) + \frac{C_2}{2} w_2^2(t) + \sum_{i=1}^{4} \lambda_i g_i, \tag{10}$$

where g_i is the right hand side of the differential equation of each state variable of the system (8). Using Pontryagin's maximum principle [36], we can determine the optimal control $w^* = (w_1^*, w_2^*)$ for the problem (8) and its associated trajectory $X^* = (S^*, I^*, O^*, S_m^*)^T$. Then the following theorem is stated.

Theorem 6. *Given the optimal control* $w^* = (w_1^*, w_2^*)$ *and the corresponding solution* $X^* = (S^*, I^*, O^*, S_m^*)^T$ *of the system (8), there exists adjoint functions* λ_1, λ_2, λ_3 *and* λ_4 *satisfying the following equations:*

$$\begin{aligned}
\lambda_1' &= (\lambda_1 - \lambda_2)\beta\, O(t) + \lambda_1(\mu + \gamma),\\
\lambda_2' &= -1 + (\lambda_1 - \lambda_2)w_1 + (\lambda_1 - \lambda_1 t_{v_1} - \lambda_2 t_{v_2})\mu + \alpha\, \lambda_2 - k\, \lambda_3,\\
\lambda_3' &= -1 + (\lambda_1 - \lambda_2)\beta\, S(t) + \lambda_3(\mu_0 + w_2) + \lambda_4 \beta_m S_m(t),\\
\lambda_4' &= \lambda_4 \beta_m O(t),
\end{aligned}$$

with the transversality conditions at time t_f*:*

$$\lambda_i(t_f) = 0, \quad \forall i = 1, \ldots, 4.$$

Moreover, for $t \in [0, t_f]$*, the optimal controls* w_1^* *and* w_2^* *are given by*

$$w_1^* = \min\left(1, \max\left(0, \tfrac{\lambda_2 - \lambda_1}{C_1} I(t)\right)\right), \tag{11}$$

$$w_2^* = \min\left(1, \max\left(0, \tfrac{\lambda_3}{C_2} O(t)\right)\right). \tag{12}$$

Proof. Let the Hamiltonian H defined by (10). The adjoint equations and transversality conditions can be obtained by using Pontryagin's Maximum Principle [36] such that

$$\begin{aligned}
\lambda_1' &= -\frac{\partial H}{\partial S} = (\lambda_1 - \lambda_2)\,\beta\, O(t) + \lambda_1(\mu + \gamma),\\
\lambda_2' &= -\frac{\partial H}{\partial I} = -1 + (\lambda_1 - \lambda_2)\, w_1 + (\lambda_1 - \lambda_1 t_{v_1} - \lambda_2 t_{v_2})\,\mu + \alpha\, \lambda_2 - k\, \lambda_3,\\
\lambda_3' &= -\frac{\partial H}{\partial O} = -1 + (\lambda_1 - \lambda_2)\,\beta\, S(t) + \lambda_3\,(\mu_0 + w_2) + \lambda_4\,\beta_m\, S_m(t),\\
\lambda_4' &= -\frac{\partial H}{\partial S_m} = \lambda_4\,\beta_m\, O(t),
\end{aligned}$$

with the transversality conditions at t_f given by $\lambda_1(t_f) = 0$, $\lambda_2(t_f) = 0$, $\lambda_3(t_f) = 0$ and $\lambda_4(t_f) = 0$. The optimal controls w_1^* and w_2^* can be solved from the following optimality conditions,

$$\frac{\partial H}{\partial w_1} = 0, \quad \frac{\partial H}{\partial w_2} = 0,$$

this gives

$$\frac{\partial H}{\partial w_1} = C_1 w_1 + (\lambda_1 - \lambda_2)\, I(t) = 0, \quad \text{and} \quad \frac{\partial H}{\partial w_2} = C_2 w_2 - \lambda_3 O(t) = 0.$$

Finally using the bounds λ_1, λ_2, λ_3 and λ_4 on the control W, we can easily obtain w_1^* and w_2^* in the form (11) and (12), respectively. ∎

5. Numerical simulation

This section focuses on numerical simulations of the mathematical model (2) to explore various toxoplasmosis scenarios and the impact of controls w_1 and w_2. We investigate both $R_0 < 1$ and $R_0 > 1$ scenarios to validate theoretical predictions. Key strategies for reducing toxoplasmosis prevalence include vaccination and environmental oocyst removal. Therefore, our simulations will vary vaccination rates and the rate of oocyst removal. Additionally, we examine different transmission rates between oocysts and populations of cats and mice. Varying the vaccination rate γ and the oocysts mortality rate μ_0 allows us to simulate diverse outcomes.

Each simulation computes steady states to confirm the theoretical stability results discussed earlier. A critical parameter is the transmissibility of toxoplasmosis via oocysts, which determines the basic reproduction number R_0 [37]. Initial estimates of environmental oocyst loads are derived from an adapted equation based on [23].

Most simulations employ parameter values detailed in Table 1, reflecting real-world conditions with varying degrees of certainty. The values used here are drawn from different studies [4, 8, 20, 38].

Table 1. Parameter values in the simulation.

Parameter	Figure 2	Figure 3	Figure 4	Figure 5	Figure 6	Figure 7	Figure 8	Figure 9
α	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$
k	$2 \cdot 10^7$	$2 \cdot 10^7$	$2 \cdot 10^7$	$2 \cdot 10^7$	$2 \cdot 10^7$	$2 \cdot 10^7$	$2 \cdot 10^7$	$2 \cdot 10^7$
μ	$\frac{1}{260}$	$\frac{1}{260}$	$\frac{1}{260}$	$\frac{1}{260}$	$\frac{1}{260}$	$\frac{1}{260}$	$\frac{1}{260}$	$\frac{1}{260}$
β_m	10^{-9}	10^{-9}	10^{-9}	10^{-9}	10^{-9}	10^{-9}	10^{-9}	10^{-9}
t_{v_1}	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02
t_{v_2}	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05
β	$0.18 \cdot 10^{-9}$	$0.18 \cdot 10^{-8}$	$0.18 \cdot 10^{-7}$	$0.18 \cdot 10^{-7}$	$0.18 \cdot 10^{-8}$	$0.18 \cdot 10^{-8}$	$0.18 \cdot 10^{-8}$	$0.18 \cdot 10^{-8}$
γ	0.001	0.001	0.001	0.08	0	0	0.001	0.001
μ_0	$\frac{1}{26}$	$\frac{1}{26}$	$\frac{1}{26}$	$\frac{1}{26}$	$\frac{1}{168}$	$\frac{1}{13}$	$\frac{1}{26}$	$\frac{1}{26}$
u	—	—	—	—	—	—	—	varied
v	—	—	—	—	—	—	—	varied
R_0	0.1486	1.4859	14.8594	0.8588	12.0979	0.9361	1.4859	—

We first consider a case where $R_0 = 0.1486 < 1$. In this case, we have taken a low vaccination rate of $\gamma = 0.001$ in the vaccination program with the transmission rate $\beta = 1.8 \cdot 10^{-10}$. The dynamics of the subpopulation are depicted in Figure 2, and the parameter values used are given in Table 1. As a result, the infection dies out as the solution approaches the steady state E_0. The disease eventually disappears despite the low immunization rate. The theoretical stability analysis and these numerical results are in agreement.

For the second case, when $R_0 = 1.4859 > 1$, the illness becomes endemic when the number of sick cats reaches a steady state that differs from zero, as seen in Figure 3. Therefore, the endemic equilibrium point E_e is reached when the transmission rate β is increased (equal $18 \cdot 10^{-10}$) such that $R_0 > 1$.

Next, we examine the effects of immunization programs on the dynamics of mouse and cat populations. We examine a scenario in which cats are extremely vulnerable to oocyst infection. It should be noted that cats can become infected with Toxoplasma gondii by consuming food contaminated by the excrement of another cat that is shedding the parasite's tiny feces, as well as through vertical transmission. Initially, we set the value of the transmission rate $\beta = 0.18 \cdot 10^{-7}$ so that $R_0 = 14.8594 > 1$ as we

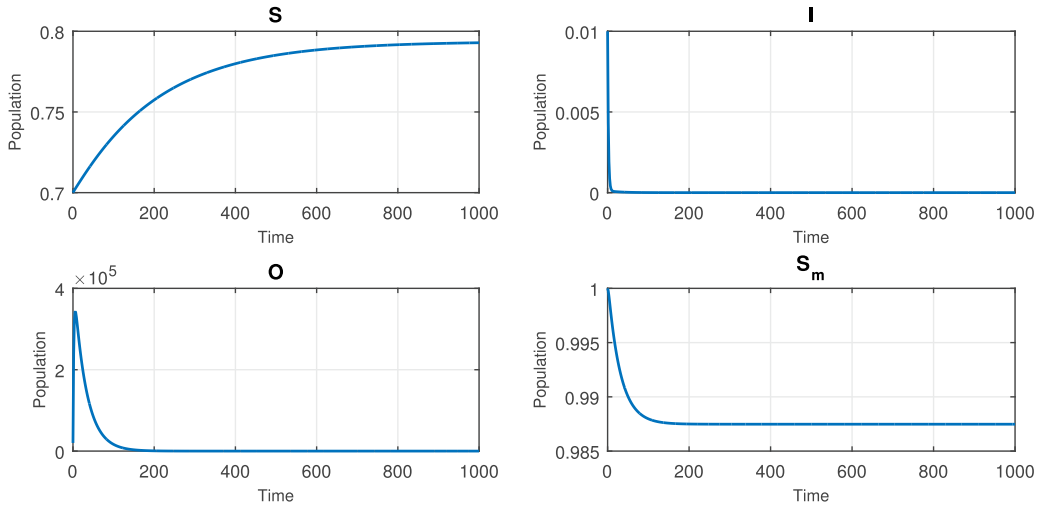

Figure 2. The dynamics $(S(t), I(t), O(t), S_m(t))$ across the free equilibrium point E_0.

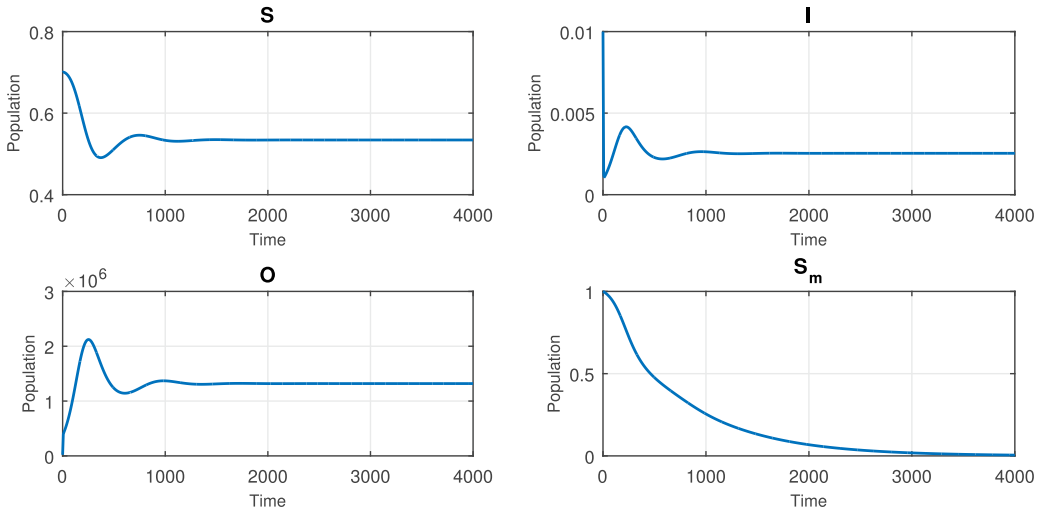

Figure 3. The dynamics $(S(t), I(t), O(t), S_m(t))$ across the endemic equilibrium point E_e.

can see in Figure 4, the infection is still present. Furthermore, we place the initial conditions far from the disease-free equilibrium E_0. Then, we have increased the vaccination rate to $\gamma = 0.08$, resulting in $R_0 < 1$ ($R_0 = 0.8588$). Figure 5 illustrates that even though oocysts have a high infectivity, both the proportion of infected cats and the total quantity of oocysts go extinct. This specific outcome, where the basic reproduction number $R_0 < 1$, demonstrates the efficacy of a cat immunization program in eradicating the illness.

In the following case we investigate the effects of a public health initiative that aims to lower oocyst levels in the absence of immunization ($\gamma = 0$). In order to ensure that $R_0 < 1$, we vary the oocyst mortality rate ($\mu_0 = 1/168$). Assuming a high oocyst infection to cats ($R_0 = 12.0979$) which increases the number of infected individuals and the number of oocysts in the environment, as shown in Figure 6. To highlight the local stability of the endemic state E_e, we intentionally set initial conditions significantly different from the disease-free equilibrium E_0. Figure 7 shows how the system can approach a steady state free of illness by increasing the rate at which oocysts are cleared ($\mu_0 = 1/13$ in this case we get $R_0 = 0.9361$). This implies that reducing the prevalence of toxoplasmosis without a vaccination campaign is achievable, as long as an effective method for cleaning the oocyst habitat is used.

The above figures show the usefulness of using vaccination (high value of $\gamma \neq 0$, Figure 5) and cleaning oocysts from the environment (high value of m_0, Figure 7). Therefore, in the following, we examine the effectiveness of the control optimal, as we can see in Figure 9 and compared to Figure 8, the numbers of infected cats and oocysts are decreased and then disappeared from the environment.

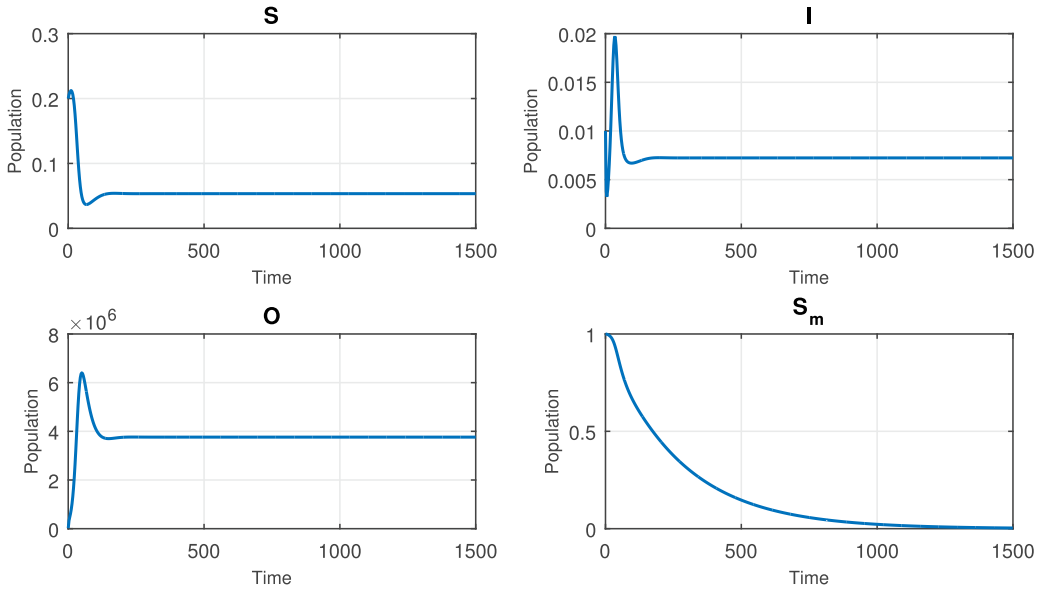

Figure 4. The dynamics $(S(t),\ I(t),\ O(t),\ S_m(t))$ when $\gamma = 0.001$ and $R_0 = 14.8594$.

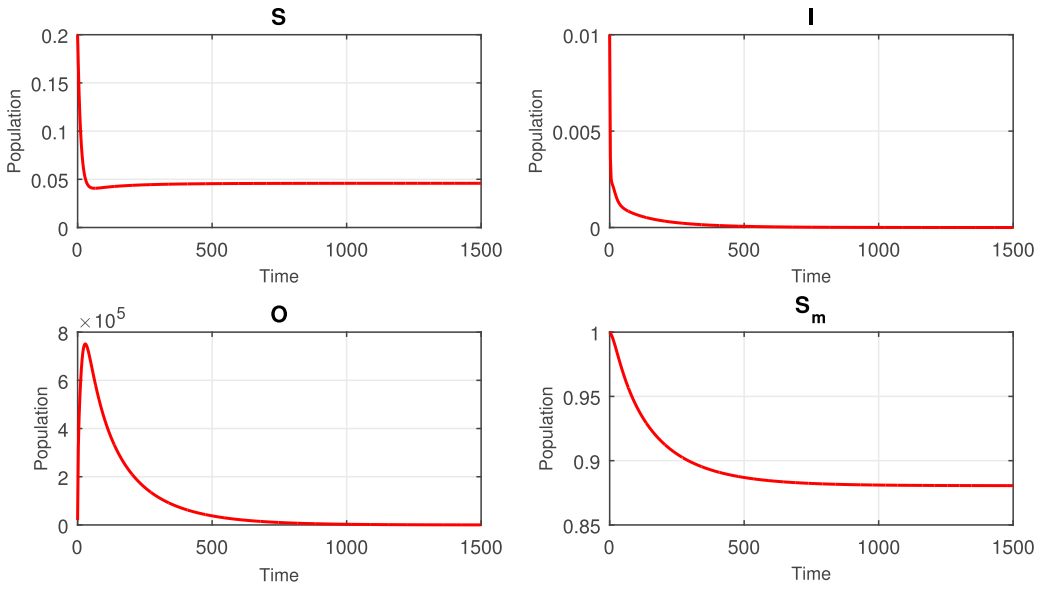

Figure 5. The dynamics $(S(t),\ I(t),\ O(t),\ S_m(t))$ when $\gamma = 0.08$ then $R_0 = 0.8588$.

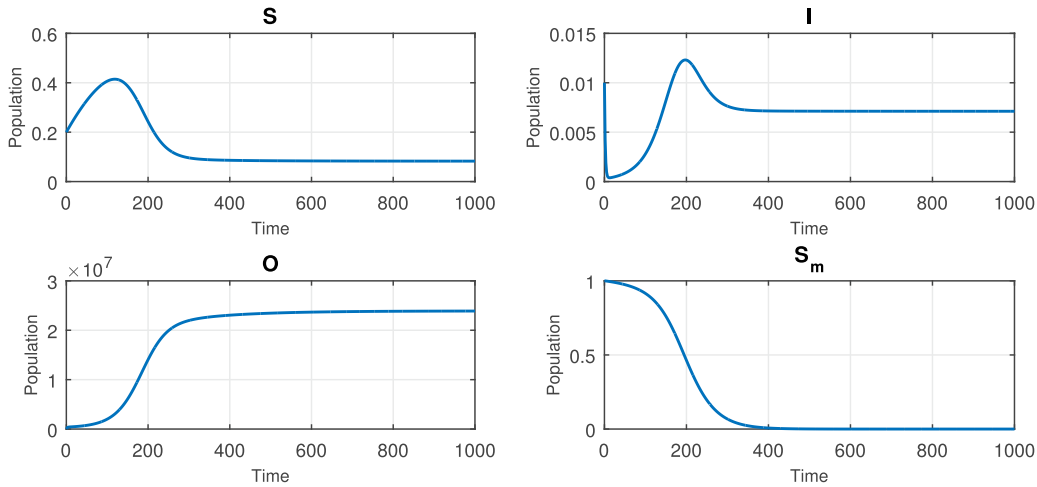

Figure 6. The dynamics $(S(t),\ I(t),\ O(t),\ S_m(t))$ when $\mu_0 = 1/168$ and $\gamma = 0$ then $R_0 = 12.0979$.

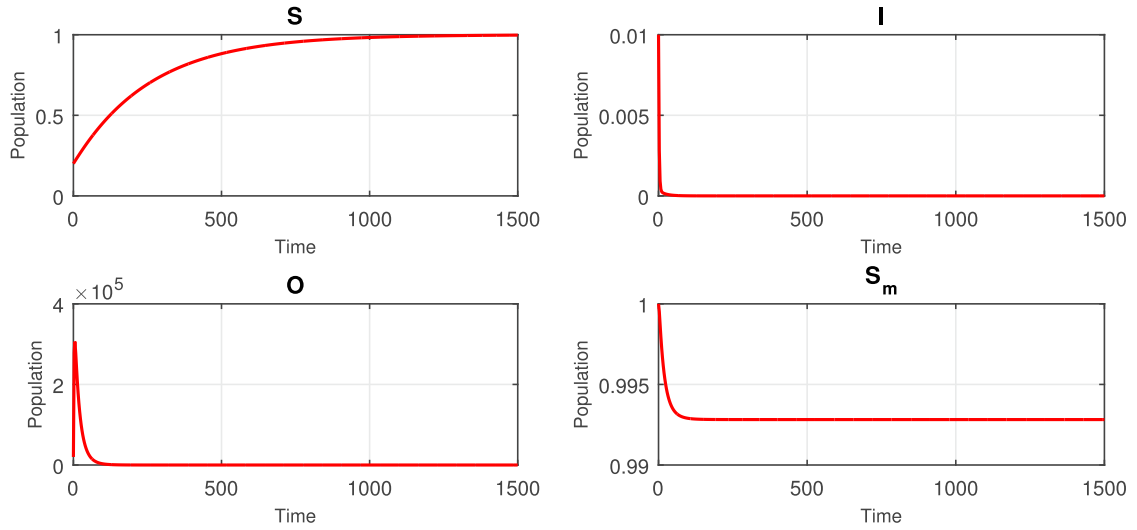

Figure 7. The dynamics $(S(t),\ I(t),\ O(t),\ S_m(t))$ when $\mu_0 = 1/13$ and $\gamma = 0$ then $R_0 = 0.9361$.

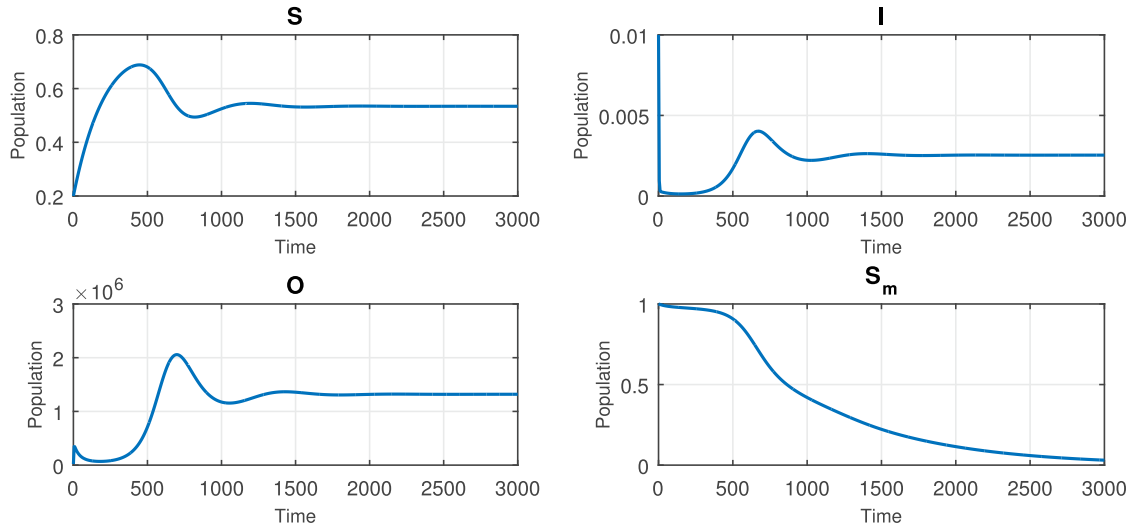

Figure 8. The dynamics $(S(t),\ I(t),\ O(t),\ S_m(t))$ without the controls w_1 and w_2.

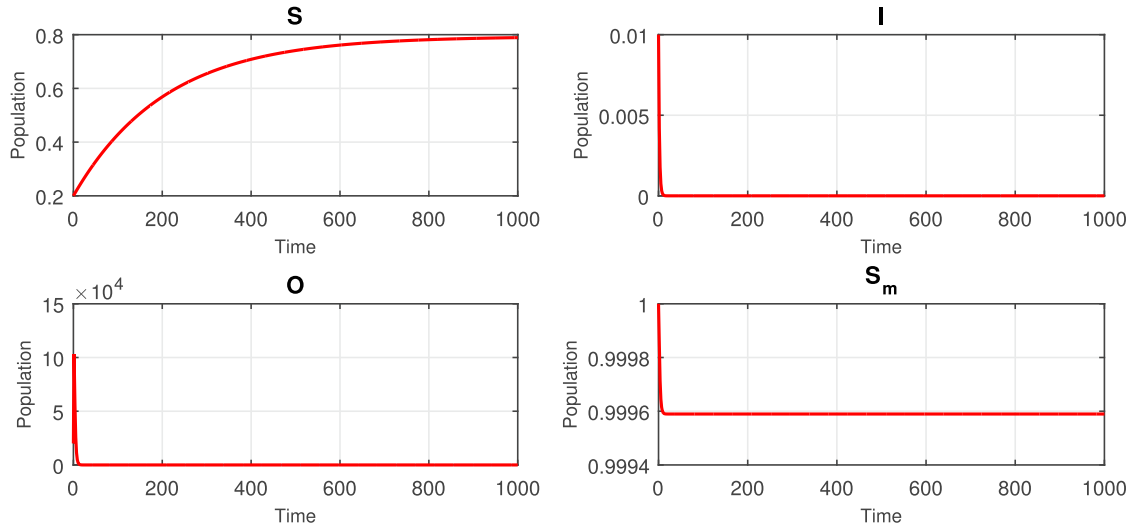

Figure 9. The dynamics $(S(t),\ I(t),\ O(t),\ S_m(t))$ with the controls w_1 and w_2.

6. Conclusion

This study presents a mathematical model for the transmission dynamics of *Toxoplasma gondii* in cat and mouse populations, incorporating vaccination and environmental contamination by oocysts. The model is shown to be mathematically and biologically well posed through proofs of existence, uniqueness, boundedness, and positivity of solutions.

Using the basic reproduction number R_0, we establish that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, while the endemic equilibrium is locally asymptotically stable when $R_0 > 1$. An optimal control framework is further developed to reduce the number of infected cats and the environmental oocyst load. Treatment of infected cats and management of environmental oocysts are shown, both analytically and numerically, to be effective in mitigating disease transmission.

Numerical simulations support the theoretical analysis and highlight the impact of vaccination and environmental control strategies. Future work will extend the model to include human populations and explicitly account for both vertical and horizontal transmission pathways of *Toxoplasma gondii*.

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Математичне моделювання та стратегії оптимального керування токсоплазмозом із декількома хазяями

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Токсоплазмоз є одним із найпоширеніших інфекційних захворювань у світі через його шкідливий вплив як на людей, так і на тварин. У цьому дослідженні представлено динаміку токсоплазмозу в популяціях котів і мишей. Розглянуто безперервну вакцинацію котів, горизонтальну передачу в обох популяціях та враховано вертикальну (вроджену) передачу лише для популяції котів. Крім того, враховано вплив ооцист паразита Toxoplasma gondii, який є збудником токсоплазмозної інфекції. У роботі наведено комплексний аналіз додатності, обмеженості та стійкості точок рівноваги. Крім того, запропоновано керовану систему з двома стратегіями керування, які спрямовані на мінімізацію інфікованої популяції за одночасної оптимізації витрат. Для підтвердження аналітичних результатів наведено чисельний приклад.

Ключові слова: *Toxoplasma gondii; вакцинація; аналіз стійкості; вертикальна передача; горизонтальна передача; оптимальне керування.*