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Mathematical modeling of the influence of vertical transmission and mild infection on the dynamics of toxoplasmosis

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Abstract

In this article, a novel model for the dynamics of toxoplasmosis in human and cat populations with vertical transmission and contribution of oocysts to the environment from the mildly infected cats is constructed. The non-negative properties of the model's solutions are proved. We demonstrate that a secondary quantity that affects the overall dynamics of *T. gondii* in human and cat populations is the reproductive ratio $\mathcal{R}_\circ$. The impact of the contribution of oocysts from the mildly infected cats as well as the impact of vertical transmission and the impact of effective contact between cat and cat and cat and humans on the reproductive ratio are shown. The model's endemic and disease-free equilibria are derived, and their local and global stabilities are proved. The bifurcation and sensitivity of the model's parameters to *T. gondii* dynamics are studied. Finally, simulations are performed with the aid of the computer-in-built Runge-Kutta package implemented in the software Maple to illustrate the behavior of the model graphically. The results indicate that vertical transmission, contact with the infected cats and contribution of *T. gondii* from the mildly infected cats have a significant impact on the dynamics of toxoplasmosis.

Keywords: Oocysts, reproductive ratio, simulation, toxoplasmosis, vertical transmission
2020 MSC: 92B05, 92D30, 34D20

1 Introduction

Toxoplasma gondii (or *T. gondii*), one of the most pervasive zoonotic parasites in the world, may infect almost all warm-blooded species. Toxoplasmosis, a potentially deadly infection in humans that places a significant burden on the public health system and causes significant financial losses in the livestock industry owing to abortion and stillbirth, is caused by *T. gondii* [48].

The life cycle of *T. gondii* is complex and complicated. It involves both sexual and asexual development, many hosts, and three infectious stages (invasive tachyzoites rapidly dividing and spreading in nucleated cells, slowly dividing bradyzoites in tissue cysts, and sporozoites in oocysts) [20]. Members of the Felidae family have the most significant role in *T. gondii* transmission because they are the parasite's sole known host [60].

Any of the three *T. gondii* infection phases can cause an infection in cats [20]. After sexual reproduction in their intestines, cat can release millions of oocysts through their faces [43]. This could go on for up to 20 days [11]. In the

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environment, oocysts sporulate in a matter of days and can stay contagious in soil or water for over a year [2]. By consuming sporulated oocysts from contaminated habitats, a variety of warm-blooded animals can become infected and act as the parasite's intermediate hosts [32]. Additionally, *T. gondii* can spread across intermediate hosts through carnivory, such as when individuals eat undercooked meat that contains tissue cysts [14]. Also, the tachyzoites from *T. gondii* can pass through the placenta and infect the fetus, causing congenital toxoplasmosis, if the original infection happens during pregnancy [22].

Models are useful for studying the dynamics of infectious disease transmission in many contexts because they employ mathematical language to explain the behavior of a system. They can be used to validate, compare, and optimize the preventive measures in addition to helping us better understand how a virus spreads. Compartmental models, for instance, are frequently used in epidemiological research to examine the transmission of contagious diseases at the population level. In these models, the entire population is usually divided into various compartments [15].

Many mathematical models have been developed to evaluate the dynamics of *T. gondii* in the population of cats [5, 7, 10, 31, 32, 37, 39, 53, 56], population of humans [4] and, in the populations of both cat and human [24, 30, 43, 50, 58, 59]. In [50], the dangers of *T. gondii* oocyst exposure to farm animals and people were investigated. The authors x-rayed the impacts of kittens in maintaining environmental contamination by *T. gondii* oocysts on farms. The authors however utilized probabilistic elements to allow various ambiguities surrounding the dynamics of *T. gondii*. In [17], an exceptional x-ray of previous toxoplasmosis mathematical models is presented. In [5, 10, 41, 44, 45, 54, 56], vaccinations were incorporated into toxoplasmosis studies to assess the effect of vaccines as control strategies and it was discovered that vaccines were strong tools to alter the dynamics of *T. gondii*. Vaccines can confer life long immunity to cats thereby reducing the rate of spread of *T. gondii* [26, 27].

The majority of the sickness that has been linked to human *T. gondii* is caused by congenital toxoplasmosis [17]. The burden of congenital toxoplasmosis was assessed to be 1.2 million DALYS (95% CI: 0.76-1.90) [55]. Despite dangers of toxoplasmosis to humans and public health, few mathematical models have considered the dynamics of the disease in human population as well as in human and cat populations [4, 24, 30, 43, 58, 59]. Aranda et al. [4] were credited for developing the first model of toxoplasmosis transmission in human population though the model does not include the major transmitter of the disease, the cats. Gonzalez-Parra et al. [30] modified [4] the following year and the work caught the attention of Mathematical Biologists. The model is made up of five equations but does not include the tendency of toxoplasmosis transmission from human-to-human as well as the tendency of contribution of oocysts to the environment by the mildly infected cats. Like [30], other models of *T. gondii* spread in human and cat populations lack human-to-human transmission of the disease and the contribution of oocysts to the environment by the mildly infected cats.

In the present analysis, we develop a system of seven nonlinear ODEs to capture the dynamics of *T. gondii* in human and cat populations. The seven system of equations allows the analysis of how various epidemiological parameters affect the overall behavior of toxoplasmosis in the populations of humans and cats. Further, the proposed model involves modeling the contacts between susceptible and infectious populations of both species. The horizontal spread of the infection to humans is assumed to occur via interactions with infectious cats and humans while the existence of vertical transmission is assumed in both human and cat populations. The model is studied analytically by deriving its equilibria and reproductive ratio, examining the stability of its equilibria and performing bifurcation and sensitivity analyses. To verify the theoretical results, we solve the model numerically and the conclusion of the findings of the study is offered.

The paper is structured as follows: we design the model and outline the key assumptions in section 2. We analyze the steady states for local and global stability and also perform bifurcation and sensitivity analyses in section 3. We offer a range of simulations in section 4. Finally, we give the conclusion in section 5.

2 Materials and Methods

In this part, a mathematical model for the spread of toxoplasmosis disease in populations of humans and cats is formulated. The model incorporates vertical transmission in both human and cat population though the probability of vertical transmission in cats is very low [37]. The possibility of *T. gondii* spreading from human-to-human is also included though it is omitted in most existing toxoplasmosis models [4, 7, 30, 31, 46, 53]. Studies have shown that toxoplasmosis can spread from human-to-human through unprotected sexual intercourse, blood transfusion or organ transplantation [3, 25, 35, 40, 42, 47, 52]. The constructed model also incorporates the possibility of shedding of oocysts by the mildly infected cats. A cat becomes mildly infected when the oocysts shedding has been terminated and the parasites have become dormant in the cat. This stage of infection is excluded in most known toxoplasmosis models

because it is considered that cats do not contribute oocysts to the environment at this stage [4, 7, 10, 30, 31, 32, 46, 53]. However, the contribution of oocysts into the environment by the mildly infected cats has been mentioned in [42, 52] and considered recently in [37]. The *T. gondii* parasites primarily infect cats, who then shed the parasites into the environment [40]. In the model constructed, there is a tendency for cats and oocysts to interact directly while the environment is frequently contaminated by oocysts [21]. The prevalence of *T. gondii* in the environment has an expected effect on the chance of infection. As a result, the number of oocysts in the environment, which is dependent on the number of infected cats, is used to model the rate of infection [33].

The model assumes that when there are no infected cats, the oocysts deteriorate naturally [33]. This is a typical premise used in biological process modeling. Oocysts that have been sporulated can persist for a very long time under a range of environmental circumstances. Oocysts can indeed last in damp soil for a long time [33]. The following presumptions form the basis of the model:

1. The entire population $N_h(t)$ for humans is categorized into three subpopulations:

- Susceptible $S_h(t)$: people who could contract the disease among the human population.
- Infected $I_h(t)$: members of human population who are *T. gondii*- infected.
- Recovered $R_h(t)$: people who have had toxoplasmosis treatment.

2. The population of cats $N_c(t)$ is categorized into three subpopulation

- Susceptible $S_c(t)$: individuals of the cat population who could contract the disease.
- Acutely Infected $I_c(t)$: members of the *T. gondii* parasite infected cat population.
- Mildly infected $M_c(t)$: members of the infected cats who have passed the stage of acute infection.

3. Through effective contact with an infected cat or human, a susceptible human can contract the disease at different rates β_2 and β_3 and moves to the infected class $I_h(t)$. An infected individual then moves to the recovered class $R_h(t)$ at a rate of γ_1.

4. A susceptible cat can contract the disease through direct contact with an infected cat or contaminated environment at different rates β_2 and β_1 and moves to infected compartment $I_c(t)$. Infected cats never make a full recovery but move to the mildly infected compartment $M_c(t)$ at rate γ_2 when the parasites become dormant in them [37]. Susceptible humans and cats contract the disease from the infected cats at the same rate β_2 [30].

5. Vertical transmission is assumed in both human and cat populations though the probability of it in cats is very low [37]. The human and cat populations are recruited at rates b_1 and b_2 respectively such that a fraction ϵ_1 and ϵ_2 from humans and cats respectively are born infected from their infected mothers with $\epsilon_1 > \epsilon_2$.

6. Both acutely and mildly infected cats, $I_c(t)$ and $M_c(t)$, contribute oocyst to the environment compartment $E(t)$ at different rates η_1 and η_2 respectively ($\eta_2 < \eta_1$) [37]. Oocyst are lost in the environment at rates, δ and ϕ, due to natural occurrences and human intervention (sanitation).

7. Death due to *T. gondii* is not considered but natural mortality occurs in human and cat populations at rate u_1 and u_2 respectively.

We denote the entire human population by

$$N_h(t) = S_h(t) + I_h(t) + R_h(t),$$

and the entire cat population by

$$N_c(t) = S_c(t) + I_c(t) + M_c(t).$$

Given the assumptions above, the dynamic toxoplasmosis disease model for the population of humans and cats is graphically shown in Figure 1 and is analytically represented by the ODEs shown below

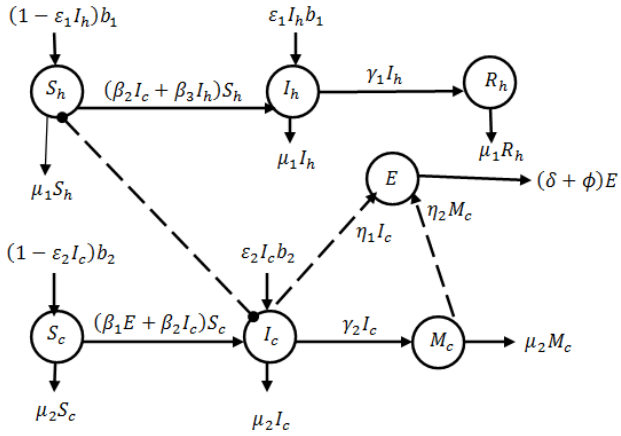


Figure 1: Transmission diagram of the model defined in system (2.1)

$$
\begin{aligned}
\frac{dS_h}{dt} &= (1-\epsilon_1 I_h)b_1 - (\beta_2 I_c+\beta_3 I_h)S_h - \mu_1 S_h,\\
\frac{dI_h}{dt} &= \epsilon_1 I_h b_1 + (\beta_2 I_c+\beta_3 I_h)S_h - (\mu_1+\gamma_1)I_h,\\
\frac{dR_h}{dt} &= \gamma_1 I_h - \mu_1 R_h,\\
\frac{dS_c}{dt} &= (1-\epsilon_2 I_c)b_2 - (\beta_1 E+\beta_2 I_c)S_c - \mu_2 S_c,\\
\frac{dI_c}{dt} &= \epsilon_2 b_2 I_c + (\beta_1 E+\beta_2 I_c)S_c - (\mu_2+\gamma_2+\eta_1)I_c,\\
\frac{dM_c}{dt} &= \gamma_2 I_c - (\mu_2+\eta_2)M_c,\\
\frac{dE}{dt} &= \eta_1 I_c + \eta_2 M_c - (\delta+\phi)E,
\end{aligned}
\tag{2.1}
$$

subject to the initial conditions $S_{0h}>0, I_{0h}\geq 0, R_{0h}\geq 0, S_{0c}>0, I_{0c}\geq 0, M_{0c}\geq 0, E_0\geq 0$.

Table 1: Nomenclatures for the model parameters

model parameters	nomenclatures	values	reference
b_1	recruitment rate for humans	100	Assumed
b_2	recruitment rate for cats	5	Assumed
ϵ_1	fraction of humans born from infected mothers	0.005	Assumed
ϵ_2	fraction of cats born from infected mothers	0.001	Assumed
ϵ_2	fraction of cats born from infected mothers	0.001	Assumed
β_1	contact rate between cats and the environment	0.01-0.15	[37]
β_2	contact rate between human and cats	0.008	Assumed
β_3	contact rate between humans	0.001	Assumed
μ_1	natural mortality rate for humans	0.000039	Estimamed
μ_2	natural mortality rate for cats	0.00021	Estimated
γ_1	recovery rate for humans	0.5	Assumed
γ_2	rate of termination of oocyst shedding	0.47-1	[37]
η_1	oocysts shedding rate for acutely infected cats	0.027-0.3	[10]
η_2	oocysts shedding rate for mildly infected cats	0.00027-0.003	Assumed
δ	removal rate of oocysts due to nature	0.058-0.096	[37]
ϕ	removal rate of oocysts due to human intervention	0.085-0.5	[10]

As in [57], we exclude the equation for the recovered human in (2.1) and base the analysis on the reduced system

$$\begin{aligned}
\frac{dS_h}{dt} &= (1-\epsilon_1 I_h)b_1 - (\beta_2 I_c + \beta_3 I_h)S_h - \mu_1 S_h,\\
\frac{dI_h}{dt} &= \epsilon_1 I_h b_1 + (\beta_2 I_c + \beta_3 I_h)S_h - (\mu_1+\gamma_1)I_h,\\
\frac{dS_c}{dt} &= (1-\epsilon_2 I_c)b_2 - (\beta_1 E + \beta_2 I_c)S_c - \mu_2 S_c,\\
\frac{dI_c}{dt} &= \epsilon_2 b_2 I_c + (\beta_1 E + \beta_2 I_c)S_c - (\mu_2+\gamma_2+\eta_1)I_c,\\
\frac{dM_c}{dt} &= \gamma_2 I_c - (\mu_2+\eta_2)M_c,\\
\frac{dE}{dt} &= \eta_1 I_c + \eta_2 M_c - (\delta+\phi)E.
\end{aligned} \tag{2.2}$$

Now bringing the change in the total populations $N_h(t)$ and $N_c(t)$ for the two species together in an equation then

$$\begin{aligned}
\frac{d}{dt}N_h(t) &= b_1 - \mu_1 N_h - \gamma_1 I_h,\\
\frac{d}{dt}N_c(t) &= b_2 - \mu_2 N_c - \eta_1 I_c - \eta_2 M_c.
\end{aligned} \tag{2.3}$$

Also, the change in oocyst population represented in the last equation in (2.2) can be expressed as

$$\frac{dE}{dt} = (I_c + M_c)\lambda - (\delta+\phi)E, \tag{2.4}$$

where $\lambda = \min(n\beta_1, n\beta_2)$ [6]. λ is the total contribution of the infectious cats (both acutely and mildly infected) to the spread of oocyst. Before being considered valid, the model has to meet the boundedness and positivity features. Further, it needs to be well-posed biologically and mathematically. To confirm the model's validity, each of the features must be verified one at a time.

2.1 Positivity of solutions

Since the system tracks the populations of human and animal, its solutions must be favorably positive. We will demonstrate that the system's solutions are nonnegative for any $t > 0$.

Theorem 2.1. The system's solutions $(S_h, I_h, S_c, I_c, M_c, E)$ are nonnegative for every $t > 0$ given the nonnegative starting variables $S_{0h} > 0, I_{0h} > 0, R_{0h} > 0, S_{0c} > 0, I_{0c} > 0, M_{0c} > 0, E_0 > 0$.

Proof . From (2.3), the following holds

$$\begin{aligned}
\lim_{t\to\infty} \sup N_h(t) &\le \frac{b_1}{\mu_1},\\
\lim_{t\to\infty} \sup N_c(t) &\le \frac{b_2}{\mu_2}.
\end{aligned} \tag{2.5}$$

Supposing $\nabla_L = \sup \ \ t > 0 : S_h(t) > 0, I_h(t) > 0, S_c(t) > 0, I_c(t) > 0, M_c(t) > 0, E(t) > 0$ then $\nabla_L > 0$. Assuming also that $\nabla_L > \infty$, then $S_h, I_h, S_c, I_c, M_c, E$ become zero at ∇_L. Hence, from the first equation in (2.2), it follows that

$$\frac{d}{dt}S_h(t)\exp[(\beta_2 I_c + \beta_3 I_h + \mu_1)t] = \int_0^{\nabla_L} (1-\epsilon_1 I_h)b_1 \exp[(\beta_2 I_c + \beta_3 I_h + \mu_1)p]dp. \tag{2.6}$$

Then

$$S_h(\nabla_L) = S_h(0)\exp[-(\beta_2 I_c+\beta_3 I_h+\mu_1)\nabla_L] + \exp[-(\beta_2 I_c+\beta_3 I_h+\mu_1)\nabla_L] \times \int_0^{\nabla_L} (1-\epsilon_1 I_h)b_1 \exp[(\beta_2 I_c+\beta_3 I_h+\mu_1)p]dp > 0. \tag{2.7}$$

Following the same technique, we can show that $I_h > 0, S_c(t) > 0, I_c(t) > 0, M_c(t) > 0, E(t) > 0$, for all $t > 0$. □

2.2 Boundedness of solutions

The solutions $(S_h, I_h, S_c, I_c, M_c, E)$ of the system are bounded.

Proof . When there is no infection in the two populations then (2.3) reduces to

$$\begin{aligned} \frac{d}{dt}N_h(t) &\leq b_1 - \mu_1 N_h, \\ \frac{d}{dt}N_c(t) &\leq b_2 - \mu_2 N_c. \end{aligned} \tag{2.8}$$

Integrating the first equation in (2.8),

$$e^{\mu_1 t}N_h(t) = \int b_1 e^{\mu_1 t}dt + c_1 \leq \frac{b_1}{\mu_1}e^{\mu_1 t} + c_1,$$

this implies that

$$N_h(t) \leq \frac{b_1}{\mu_1} + c_1 e^{-\mu_1 t}.$$

When $t = 0$, we have, $c_1 \geq N_h(0) - \dfrac{b_1}{\mu_1}$, therefore

$$N_h(t) \leq \frac{b_1}{\mu_1} + \left(N_h(0) - \frac{b_1}{\mu_1}\right)e^{-\mu_1 t}.$$

Hence,

$$N_h(t) \leq N_h(0)e^{-\mu_1 t} + \frac{b_1}{\mu_1}(1 - e^{-\mu_1 t}). \tag{2.9}$$

Also, integrating the second equation in (2.8), $e^{\mu_2 t}N_c(t) = \int b_2 e^{\mu_2 t}dt + c_2$, then

$$e^{\mu_2 t}N_c(t) \leq \frac{b_2}{\mu_2} + c_2,$$

and so,

$$N_c(t) \leq \frac{b_2}{\mu_2} + c_2 e^{-\mu_2 t}.$$

When $t = 0$, we have, $c_2 \geq N_c(0) - \dfrac{b_2}{\mu_2}$, therefore

$$N_c(t) \leq \frac{b_2}{\mu_2} + \left(N_c(0) - \frac{b_2}{\mu_2}\right)e^{-\mu_2 t}.$$

Hence,

$$N_c(t) \leq N_c(0)e^{-\mu_2 t} + \frac{b_2}{\mu_2}(1 - e^{-\mu_2 t}). \tag{2.10}$$

Following [13], (2.9) and (2.10) become

$$0 \leq N_h(t) \leq \frac{b_1}{\mu_1} \quad \text{and} \quad 0 \leq N_c(t) \leq \frac{b_2}{\mu_2} \quad \text{as} \quad t \to \infty.$$

Particularly, $N_h(t) \leq \dfrac{b_1}{\mu_1}$ and $N_c(t) \leq \dfrac{b_2}{\mu_2}$ if $N_h(0) \leq \dfrac{b_1}{\mu_1}$ and $N_c(0) \leq \dfrac{b_2}{\mu_2}$ respectively. Hence, the solutions of the two species populations enter

$$\Omega_1 = \left\{\Omega_h \cup \Omega_c \in \mathbb{R}_+^2 \times \mathbb{R}_+^3\right\},$$

where

$$\Omega_1 = \left\{(S_h, I_h) \in \mathbb{R}_+^2; N_h(t) \leq \frac{b_1}{\mu_1}, (S_c, I_c, M_c) \in \mathbb{R}_+^3; N_c(t) \leq \frac{b_2}{\mu_2}\right\}. \tag{2.11}$$

As for the oocyst concentration in the environment in (2.4), i.e.,

$$\frac{dE}{dt} = (I_c + M_c)\lambda - (\delta + \phi)E,$$

I_c and M_c are of cats then $I_c + M_c \leq \frac{b_2}{\mu_2}$ [6]. Therefore, $\frac{dE}{dt} \leq \frac{b_2\lambda}{\mu_2} - (\delta + \phi)E$, then

$$E(t) \leq \frac{b_2\lambda}{\mu_2(\delta + \phi)}\left(1 - c_3 e^{-(\delta+\phi)t}\right).$$

As $t \to \infty$ then,

$$E(t) \leq \frac{b_2\lambda}{\mu_2(\delta + \phi)}.$$

Hence, the solution for the oocyst concentration exists in the feasible region

$$\Omega_2 = \left\{E \in \mathbb{R}_+; E(t) \leq \frac{b_2\lambda}{\mu_2(\delta + \phi)}\right\}.$$

Consequently, the entire set of solutions for the system enters

$$\Omega = \left\{(\Omega_1 \cup \Omega_2) \in \mathbb{R}^2_+ \times \mathbb{R}^3_+ \times \mathbb{R}_+\right\},$$

where

$$\Omega = \left\{(S_h, I_h) \in \mathbb{R}^2_+; N_h(t) \leq \frac{b_1}{\mu_1}, (S_c, I_c, M_c) \in \mathbb{R}^3_+; N_c(t) \leq \frac{b_2}{\mu_2}, E \in \mathbb{R}_+; E(t) \leq \frac{b_2\lambda}{\mu_2(\delta + \phi)}\right\}. \tag{2.12}$$

Given (2.12), all the model's solutions remain in Ω and Ω is positively invariant. In Ω where the model is epidemiologically and mathematically properly posed, the dynamics of the disease regulated by (2.2) can therefore be taken into consideration. □

3 Model Analysis

3.1 Equilibria

The model equilibrium points are obtained to analyze the long-term dynamics of toxoplasmosis. The system of equations (2.2) is set to zero to obtain two equilibria. i.e.

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dS_c}{dt} = \frac{dI_c}{dt} = \frac{dM_c}{dt} = \frac{dE}{dt} = 0. \tag{3.1}$$

The solutions $S_h^\circ, I_h^\circ, S_c^\circ, I_c^\circ, M_c^\circ$ and E° satisfy equation (3.1) and clearly indicate that the equilibrium is not trivial and the populations do not go into extinction as long as human recruitment rate b_1 and the cats recruitment rate b_2 are not zero. Hence, $I_h = I_c = M_c = E = 0$ when *T. gondii* is totally absent from the community and the system admits a steady state, $W_\circ$, that represents the infection-free equilibrium (DFE). Therefore, the system has the DFE denoted by

$$W_\circ = (S_h^\circ, I_h^\circ, S_c^\circ, I_c^\circ, M_c^\circ, E^\circ) = \left(\frac{b_1}{\mu_1}, 0, \frac{b_2}{\mu_2}, 0, 0, 0\right).$$

However, if the community is invaded with *T. gondii*, each of the variables become nonzero. Assuming W^* defines the system's steady state when the community is invaded with the parasite with points $S_h^*, I_h^*, S_c^*, I_c^*, M_c^*, E^*$. We solve the system (2.2) in terms of I_h^* and I_c^* and obtain

$$\begin{aligned}
S_h^* &= \frac{(1 - \epsilon_1 I_h^*)b_1}{\beta_2 I_c^* + \beta_3 I_h^* + \mu_1},\\
S_c^* &= \frac{(1 - \epsilon_2 I_c^*)b_2(\mu_2 + \eta_2)(\delta + \phi)}{\beta_1\eta_1(\mu_2 + \eta_2) + \beta_1\eta_2\gamma_2(\beta_2 I_c^* + \mu_2)(\mu_2 + \eta_2)(\delta + \phi)},\\
M_c^* &= \frac{\gamma_2}{(\mu_2 + \eta_2)} I_c^*,\\
E^* &= \frac{\eta_1(\eta_2 + \mu_2) + \eta_2\gamma_2}{(\mu_2 + \eta_2)(\delta + \phi)} I_c^*.
\end{aligned} \tag{3.2}$$

Since all the solutions of the model must be positive at W^*, the endemic equilibrium W^* exists if and only if I_h^* and I_c^* are positive.

3.2 Reproduction Number

The epidemiological quantity of the main interest is the reproductive ratio, ($\mathcal{R}_\circ$), i.e., the average number of secondary cases produced by an infectious agent introduced into a community that is fully susceptible [18]. Its values typically dictate whether or not a contagious disease can propagate through a community. If $\mathcal{R}_\circ$ is below unity, the population finally finds an equilibrium where there is no longer any sickness. The infection will, nevertheless, be able to transmit and persist in the community if the reproductive ratio is more than unity. The quantity ($\mathcal{R}_\circ$) can be determined using the approach described in [19]. For toxoplasmosis, in addition to being spread between definitive hosts, the parasite also has a reservoir in the environment and is capable of spreading through intermediate hosts. Therefore, the toxoplasmosis model in (2.2) consists of four infected states I_h, I_c, M_c and E and uninfected states S_h and S_c. The rate of new infections and state transition, which are represented by $\mathcal{F}$ and $\mathcal{V}$, respectively, are given by

$$\mathcal{F} = \begin{pmatrix} \epsilon_1 b_1 I_h + \beta_2 I_c S_h + \beta_3 I_h S_h \\ \eta_2 b_2 I_c + \beta_1 E S_c + \beta_2 I_c S_c \\ 0 \\ 0 \end{pmatrix}; \quad \mathcal{V} = \begin{pmatrix} (\mu_1 + \gamma_1) I_h \\ (\mu_2 + \gamma_2 + \eta_1) I_c \\ -\gamma_2 I_c + (\mu_2 + \eta_2) M_c \\ -\eta_1 I_c - \eta_2 M_c + (\delta + \phi) E \end{pmatrix}, \tag{3.3}$$

At the DFE, $S_h = S_h^\circ = p_1 = \dfrac{b_1}{\mu_1}, I_h = 0, S_c = S_c^\circ = p_2 = \dfrac{b_2}{\mu_2}$ and $I_c = M_c = E = 0$. The Jacobian at the DFE of $\mathcal{F}$ and $\mathcal{V}$ are F and V, respectively with

$$\begin{aligned} F &= \begin{pmatrix} (\epsilon_1 b_1 + \beta_3 S_h) & \beta_2 S_h & (\beta_2 I_c + \beta_2 I_h) & 0 \\ 0 & (\epsilon_2 b_2 + \beta_2 S_c) & 0 & \beta_1 S_c \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \\ V &= \begin{pmatrix} (\mu_1 + \gamma_1) & 0 & 0 & 0 \\ 0 & (\mu_2 + \gamma_1 + \eta_1) & 0 & 0 \\ 0 & -\gamma_2 & (\mu_2 + \gamma_2) & 0 \\ 0 & -\eta_1 & -\eta_2 & (\delta + \phi). \end{pmatrix}, \end{aligned} \tag{3.4}$$

The rates for fresh infections and changes that take place close to equilibrium are F and V. The duration spent in each of the states and the overall number of new infections produced throughout each illness which are generally denoted by V^{-1} and FV^{-1} are determined using Maple 18. The dominant eigenvalue of (FV^{-1}) is $\mathcal{R}_\circ$ and is given by

$$\mathcal{R}_\circ = \max(\mathcal{R}_{\circ hc}, \mathcal{R}_{\circ ec}) = \frac{p_1(\beta_2 + \beta_3) + \epsilon_1 b_1}{(\mu_1 + \gamma_1)}, \frac{p_2\beta_2 + b_2\epsilon_2}{(\mu_2 + \gamma_2 + \eta_1)} + \frac{p_2\beta_1(\eta_2\gamma_2 + \eta_1(\mu_2 + \eta_2))}{(\mu_2 + \eta_2)(\mu_2 + \gamma_2 + \eta_1)(\delta + \phi)}. \tag{3.5}$$

In (3.5), while $\mathcal{R}_{\circ hc}$ measures the rate of spread of *T. gondii* into human population through contact rate between infected cats and susceptible humans (β_2) and contact rate between infected and susceptible humans (β_3), $\mathcal{R}_{\circ ec}$ measures the rate of spread of *T. gondii* into cat population through contact rate between infected and susceptible cats (β_2) and contact rate between susceptible cats and contaminated environment (β_1). It is assumed that *T. gondii* spreads from cats to cats and to humans at the same rate β_2 [30]. The spread of *T. gondii* is significantly influenced by the quantity ($\mathcal{R}_\circ$). Consider the model's second and fourth equations in (2.2) with $I_c = I_h = 1$ in the second equation, the following equations are derived

$$\begin{aligned} \frac{dI_h}{dt} &= k_1(\mathcal{R}_{\circ hc} - 1)I_h, \\ \frac{dI_c}{dt} &= k_2(\mathcal{R}_{\circ ec} - 1)I_c, \end{aligned} \tag{3.6}$$

where

$$\begin{aligned} k_1 &= (\mu_1 + \gamma_1), \\ k_2 &= (\mu_2 + \gamma_2 + \eta_1). \end{aligned} \tag{3.7}$$

Some information about the transmission and control of toxoplasmosis is revealed in (3.7). $\mathcal{R}_{\circ hc} < 0$ and $\mathcal{R}_{\circ ec} < 0$ are necessary for the change in the populations of infected humans and cats to be negative so that the disease does

not spread into both human and cat populations. However, since the main transmitter of *T. gondii* is cat, we can discuss the transmission of the parasite in both human and cat populations in terms of $\mathcal{R}_{\circ ec}$. it is revealed in (3.7) that when $\mathcal{R}_{\circ ec} < 1$, the infected cats spread the parasite to less than one susceptible populations (both human and cat). This is so because the change in the population of infected cats is negative when $\mathcal{R}_{\circ ec} < 1$ and the infection just dies out. On the contrary, the change in the population of infected cats is positive when $\mathcal{R}_{\circ ec} > 1$ which signifies the possibility of disease inversion because each infected cat can contribute to the growth of toxoplasmosis by more than one infection. Therefore, $\mathcal{R}_{\circ ec} > 1$ is necessary for the existence of endemic equilibrium. Furthermore, If $\mathcal{R}_{\circ ec} = 1$, it implies that each infected cat contributes to the growth of the disease by just one as a whole. Hence, the population of the infected cats does not change when $\mathcal{R}_{\circ ec} = 1$ and the infection subsequently persists in the community.

3.3 Local and global stability of zero equilibrium, $\mathcal{W}_\circ$

The stability of $\mathcal{W}_\circ$, both local and global, depends on $\mathcal{R}_\circ$. $\mathcal{W}_\circ$ is stable locally and globally if $\mathcal{R}_\circ < 1$ but it is unstable if $\mathcal{R}_\circ > 1$. To verify the existence of local stability for $\mathcal{W}_\circ$, we compute the Jacobian matrix of the system (2.2) as follows

$$J = \begin{pmatrix} -q_1 & -(\epsilon_1 b_1 + \beta_3 S_h) & 0 & -\beta_2 S_h & 0 & 0 \\ q_2 & (\epsilon_1 b_1 + \beta_3 S_h) & 0 & \beta_2 S_h & 0 & 0 \\ 0 & 0 & -q_3 & -(\epsilon_2 b_2 + \beta_2 S_c) & 0 & -\beta_1 S_c \\ 0 & 0 & q_4 & \epsilon_2 b_2 + \beta_2 S_c - (\mu_2 + \gamma_2 + \eta_1) & 0 & \beta_1 S_c \\ 0 & 0 & 0 & \gamma_2 & -(\mu_2 + \eta_2) & 0 \\ 0 & 0 & 0 & \eta_1 & \eta_2 & -(\delta + \phi) \end{pmatrix}, \tag{3.8}$$

where $q_1 = (\mu_1 + \beta_3 I_h + \beta_2 I_c), q_2 = (\beta_3 I_h + \beta_2 I_c), q_3 = (\mu_2 + \beta_1 E + \beta_2 I_c), q_4 = (\beta_1 E + \beta_2 I_c)$. Evaluating J at $\mathcal{W}_\circ$ then (3.8) becomes

$$J(\mathcal{W}_\circ) = \begin{pmatrix} -\mu_1 & -(\epsilon_1 b_1 + \beta_3 p_1) & 0 & -\beta_2 p_1 & 0 & 0 \\ 0 & \epsilon_1 b_1 + \beta_3 p_1 - (\mu_1 + \gamma_1) & 0 & \beta_2 p_1 & 0 & 0 \\ 0 & 0 & -\mu_2 & -(\epsilon_2 b_2 + \beta_2 p_2) & 0 & -\beta_1 p_2 \\ 0 & 0 & 0 & \epsilon_2 b_2 + \beta_2 p_2 - (\mu_2 + \gamma_2 + \eta_1) & 0 & \beta_1 p_2 \\ 0 & 0 & 0 & \gamma_2 & -(\mu_2 + \eta_2) & 0 \\ 0 & 0 & 0 & \eta_1 & \eta_2 & -(\delta + \phi) \end{pmatrix}. \tag{3.9}$$

In (3.9), $\lambda_1 = -\mu_1, \lambda_2 = -\mu_2$ and the remaining solutions of $J(\mathcal{W}_\circ)$ are contained in B given as

$$B = \begin{pmatrix} \epsilon_1 b_1 + \beta_3 p_1 - (\mu_1 + \gamma_1) & \beta_2 p_1 & 0 & 0 \\ 0 & \epsilon_2 b_2 + \beta_2 p_2 - (\mu_2 + \gamma_2 + \eta_1) & 0 & \beta_1 p_2 \\ 0 & -\gamma_2 & -(\mu_2 + \eta_2) & 0 \\ 0 & \eta_1 & \eta_2 & -(\delta + \phi) \end{pmatrix}. \tag{3.10}$$

Following Gershgorin's circle theorem [29, 12], the following hold from matrix B

$$\begin{aligned} R_1 : 1 &> \frac{\epsilon_1 b_1 + \beta_3 p_1}{(\mu_1 + \gamma_1)} + \frac{\beta_2 p_1}{(\mu_1 + \gamma_1)}, \\ R_1 : 1 &> \frac{\epsilon_2 b_2 + \beta_2 p_2}{(\mu_2 + \gamma_2 + \eta_1)} + \frac{\beta_1 p_2}{(\mu_2 + \gamma_2 + \eta_1)}, \\ R_3 : 1 &> \frac{\gamma_2}{(\mu_2 + \eta_2)} \\ R_4 : 1 &> \frac{\eta_1}{(\delta + \phi)} + \frac{\eta_2}{(\delta + \phi)}. \end{aligned} \tag{3.11}$$

From R_1 in (3.11),

$$1 > \frac{p_1(\beta_2 + \beta_3) + \epsilon_1 b_1}{(\mu_1 + \gamma_1)}.$$

Then $\mathcal{R}_{\circ hc} < 1$. Also, multiplying R_3 and R_4 and use the result to multiply the second term of R_2 gives

$$1 > \frac{p_2\beta_2 + b_2\epsilon_2}{(\mu_2 + \gamma_2 + \eta_1)} + \frac{p_2\beta_1(\eta_2\gamma_2 + \eta_1(\mu_2 + \eta_2))}{(\mu_2 + \eta_2)(\mu_2 + \gamma_2 + \eta_1)(\delta + \phi)},$$

which indicates that $\mathcal{R}_{\circ ec} < 1$, hence the DFE is locally asymptotically stable if $\mathcal{R}_\circ < 1$. To establish the global stability of DFE that guarantees total elimination of *T. gondii* regardless of the initial populations of humans and cats, we construct a Lyapunov function $\mathcal{U}(t)$ as in [9, 49] as follows

$$\mathcal{U}(t) = A_1 I_h + A_2 I_c + A_3 M_c + A_4 E,$$

with time derivative

$$\dot{\mathcal{U}}(t) = A_1 \dot{I}_h + A_2 \dot{I}_c + A_3 \dot{M}_c + A_4 \dot{E},$$

where $A_1, \cdots, A_4$ are nonnegative constants whose values do not alter the positivity or negativity of $\dot{\mathcal{U}}(t)$. Therefore, we neglect them and assume that $I_c = I_h = 1$ in the second equation in (2.2) and write

$$\begin{aligned}\dot{\mathcal{U}}(t) =&[(\epsilon_1 I_h b_1 + (\beta_2 I_h + \beta_3 I_h)S_h - (\mu_1 + \gamma_1)I_h)] + (\epsilon_2 I_c b_2 + (\beta_1 E + \beta_2 I_c)S_c - (\mu_2 + \gamma_2 + \eta_1)I_c)\\ &+ \gamma_2 I_c - (\mu_2 + \eta_2)M_c + \eta_1 I_c + \eta_2 M_c - (\delta + \phi)E\\ =&(\mu_1 + \gamma_1)\left\{\frac{(\beta_2 + \beta_3)S_h + \epsilon_1 b_1}{(\mu_1 + \gamma_1)} - 1\right\} I_h\\ &+ (\mu_2 + \gamma_2 + \eta_1)\left\{\frac{S_c\beta_2 + b_2\epsilon_2}{(\mu_2 + \gamma_2 + \eta_1)} + \frac{S_c\beta_1(\eta_2\gamma_2 + \eta_1(\mu_2 + \eta_2))}{(\mu_2 + \eta_2)(\mu_2 + \gamma_2 + \eta_1)(\delta + \phi)} - 1\right\} I_c.\end{aligned}$$

Since $S_h \le p_1$ and $S_c \le p_2$, then

$$\begin{aligned}\dot{\mathcal{U}}(t) \le&(\mu_1 + \gamma_1)\left\{\frac{(\beta_2 + \beta_3)p_1 + \epsilon_1 b_1}{(\mu_1 + \gamma_1)} - 1\right\} I_h\\ &+ (\mu_2 + \gamma_2 + \eta_1)\left\{\frac{p_2\beta_2 + b_2\epsilon_2}{(\mu_2 + \gamma_2 + \eta_1)} + \frac{p_2\beta_1(\eta_2\gamma_2 + \eta_1(\mu_2 + \eta_2))}{(\mu_2 + \eta_2)(\mu_2 + \gamma_2 + \eta_1)(\delta + \phi)} - 1\right\} I_c,\end{aligned}$$

Then

$$\dot{\mathcal{U}}(t) = K(\mathcal{R}_\circ - 1)I, \tag{3.12}$$

where K is a positive constant and I denotes the existence of *T. gondii* parasite in the community. Equation (3.12) indicates that $\dot{\mathcal{U}}(t) < 0$ if $\mathcal{R}_\circ < 1$. Again, $\dot{\mathcal{U}}(t) = 0$ at $\mathcal{W}_\circ$. Putting $I_h = I_c = M_c = E = 0$ in the equations of $S_h(t)$ and $S_c(t)$ in (2.2) then $S_h(t) \to \dfrac{b_1}{\mu_1}$ and $S_c(t) \to \dfrac{b_2}{\mu_2}$ as $t \to \infty$. Also, putting $I_h = I_c = M_c = E = 0$ in the equations for $I_h(t), I_c(t), M_c(t)$ and $E(t)$ in (2.2) indicates that $(I_h(t), I_c(t), M_c(t), E(t)) \to (0, 0, 0, 0)$ as $t \to \infty$. Hence, $\mathcal{U}(t)$ remains the Lyapunov function. Following Theorem 2.3.1 in [51] as employed in [23] then there exists DFE $\mathcal{W}_\circ$ that is globally asymptotically stable in Ω if $\mathcal{R}_\circ < 1$.

3.4 Local and global stability of nonzero equilibrium, $\mathcal{W}^*$

As in DFE, $\mathcal{W}_\circ$, the stability of $\mathcal{W}^*$ depends on $\mathcal{R}_\circ$. However, unlike in $\mathcal{W}_\circ$, $\mathcal{W}^*$ is stable locally and globally if $\mathcal{R}_\circ > 1$ while it is unstable if $\mathcal{R}_\circ < 1$. To examine the local stability of $\mathcal{W}^*$, we linearize (2.2) about $\mathcal{W}^*$ and the result obtained is in the form of (3.8) but with the asterisk in the variables to indicate endemic points. We perform a simple row operation for the result obtained as in [49] to get the matrix in (3.13)

$$J^* = \begin{pmatrix} -q_1 & -\Gamma_1 & 0 & -\beta_2 S_h^* & 0 & 0\\ 0 & \dfrac{q_1}{q_2}\Gamma_1 - \Gamma_1 & 0 & \dfrac{q_1}{q_2}\beta_2 S_h^* - \beta_2 S_h^* & 0 & 0\\ 0 & 0 & -q_3 & -\Gamma_2 & 0 & -\beta_1 S_c^*\\ 0 & 0 & q_4 & \Gamma_2 - (\mu_2 + \gamma_2 + \eta_1) & 0 & \beta_1 S_c^*\\ 0 & 0 & 0 & \gamma_2 & -(\mu_2 + \eta_2) & 0\\ 0 & 0 & 0 & \eta_1 & \eta_2 & -(\delta + \phi), \end{pmatrix} \tag{3.13}$$

where $q_1 = (\mu_1 + \beta_3 I_h^* + \beta_2 I_c^*), q_2 = (\beta_3 I_h^* + \beta_2 I_c^*), q_3 = (\mu_2 + \beta_1 E^* + \beta_2 I_c^*), q_4 = (\beta_1 E^* + \beta_2 I_c^*), \Gamma_1 = (\epsilon_1 b_1 + \beta_3 S_h^*), \Gamma_2 = (\epsilon_2 b_2 + \beta_2 S_c^*)$. Elements of J^* are represented in terms of f and we have

$$J^* = \begin{pmatrix} -f_1 & -f_2 & 0 & -f_3 & 0 & 0\\ 0 & f_4 - f_5 & 0 & f_7 - f_8 & 0 & 0\\ 0 & 0 & -f_9 & -f_{10} & 0 & -f_{11}\\ 0 & 0 & f_{12} & f_{13} - f_{14} & 0 & f_{15}\\ 0 & 0 & 0 & f_{16} & -f_{17} & 0\\ 0 & 0 & 0 & f_{18} & f_{19} & -f_{20}, \end{pmatrix} \tag{3.14}$$

The eigenvalues of J^* can be obtained from (3.15) given by

$$(f_1+\lambda)(d_0\lambda^5+d_1\lambda^4+d_2\lambda^3+d_3\lambda^2+d_4\lambda+d_5)=0. \tag{3.15}$$

One root in (3.15) is already negative (i.e., $\lambda_1=-f_1$) then the endemic equilibrium $\mathcal{W}^*$ is locally asymptotically stable if all the roots of

$$(d_0\lambda^5+d_1\lambda^4+d_2\lambda^3+d_3\lambda^2+d_4\lambda+d_5)=0$$

are also negative. Following Routh-Hurwitz criteria [8], the roots of the equation are all negative and $\mathcal{W}^*$ is locally asymptotically stable if the following inequalities are true

$$d_1d_2d_3>d_3^2+d_1^2d_4,\quad (d_1d_4-d_5)(d_1d_2d_3-d_3^2-d_1^2d_4)>d_5(d_1d_2-d_3)^2+d_1d_5^2,$$

where
$d_0=1$,
$d_1=f_{13}+f_{14}+f_{17}+f_{20}$,
$d_2=-f_{12}f_{13}-(f_{13}-f_{14})(f_{17}-f_{20})-f_{18}f_{15}+f_{17}f_{20}$,
$d_3=f_{12}f_{13}+(f_{13}-f_{14})(f_{17}+f_{20})+f_{15}f_{18}-f_{17}f_{20}+f_{12}f_{13}(f_{17}+f_{20})+f_{12}f_{15}+f_{15}f_{17}f_{18}+f_{15}f_{16}f_{19}+(f_{13}-f_{14})f_{17}f_{20}$,
$d_4=-f_{12}f_{13}(f_{13}-f_{14})+f_{15}f_{12}f_{18}+f_{15}f_{17}f_{18}+f_{15}f_{16}f_{19}+(f_{13}-f_{14})f_{17}f_{20}+f_{12}f_{13}f_{17}f_{20}-f_{12}f_{15}f_{16}f_{19}-f_{12}f_{15}f_{17}f_{18}$,
$d_5=f_{12}f_{15}f_{17}f_{18}+f_{12}f_{15}f_{16}f_{19}-f_{12}f_{13}f_{17}f_{20}$.

For the global attractiveness of $\mathcal{W}^*$, we formulate a Lyapunov function as in [1, 28] as follows

$$\begin{aligned}\mathcal{V}(t)&=(S_h-S_h^*\ln(S_h))+A\,(I_h-I_h^*\ln(I_h))+(S_c-S_c^*\ln(S_c))\\&\quad+B\,(I_c-I_c^*\ln(I_c))+C\,(M_c-M_c^*\ln(M_c))+D\,(E-E^*\ln(E))\,,\end{aligned}$$

with time derivative

$$\begin{aligned}\dot{\mathcal{V}}(t)&=\left(\dot S_h-\frac{S_h^*}{S_h}\dot S_h\right)+A\left(\dot I_h-\frac{I_h^*}{I_h}\dot I_h\right)+\left(\dot S_c-\frac{S_c^*}{S_c}\dot S_c\right)+B\left(\dot I_c-\frac{I_c^*}{I_c}\dot I_c\right)+C\left(\dot M_c-\frac{M_c^*}{M_c}\dot M_c\right)+D\left(\dot E-\frac{E^*}{E}\dot E\right)\\&=\left(1-\frac{S_h^*}{S_h}\right)\dot S_h+A\left(1-\frac{I_h^*}{I_h}\right)\dot I_h+\left(1-\frac{S_c^*}{S_c}\right)\dot S_c+B\left(1-\frac{I_c^*}{I_c}\right)\dot I_c+C\left(1-\frac{M_c^*}{M_c}\right)\dot M_c+D\left(1-\frac{E^*}{E}\right)\dot E.\end{aligned} \tag{3.16}$$

At $\mathcal{W}^*$, the following hold from (2.2)

$$\begin{aligned}b_1&=\epsilon_1b_1I_h^*+\beta_2I_c^*S_h^*+\beta_3I_h^*S_h^*+\mu_1S_h^*\\\mu_1+\gamma_1&=\frac{\epsilon_1b_1I_h^*+\beta_2I_c^*S_h^*+\beta_3I_h^*S_h^*}{I_h^*}\\b_2&=\epsilon_2b_2I_c^*+\beta_1E^*S_c^*+\beta_2I_c^*S_c^*+\mu_2S_c^*\\\mu_1+\gamma_2+\eta_1&=\frac{\epsilon_2b_2I_c^*+\beta_1E^*S_c^*+\beta_2I_c^*S_c^*}{I_c^*}\\\mu_2+\eta_2&=\frac{\gamma_2I_c^*}{M_c^*}\\\delta+\phi&=\frac{\eta_1I_c^*+\eta_2M_c^*}{E^*}.\end{aligned} \tag{3.17}$$

Appropriate substitution of (2.2) and (3.17) into (3.16) gives

$$\begin{aligned}\dot{\mathcal{V}}(t)&=\left(1-\frac{S_h^*}{S_h}\right)[\epsilon_1b_1I_h^*+\beta_2I_c^*S_h^*+\beta_3I_h^*S_h^*+\mu_1S_h^*-\epsilon_1b_1I_h-\beta_2I_cS_h-\beta_3I_hS_h-\mu_1S_h]\\&\quad+A\left(1-\frac{I_h^*}{I_h}\right)\left[\epsilon_1b_1I_h+\beta_2I_cS_h+\beta_3I_hS_h-\left(\frac{\epsilon_1b_1I_h^*+\beta_2I_c^*S_h^*+\beta_3I_h^*S_h^*}{I_h^*}\right)I_h\right]\\&\quad+\left(1-\frac{S_c^*}{S_c}\right)[\epsilon_2b_2I_c^*+\beta_1E^*S_c^*+\beta_2I_c^*S_c^*+\mu_2S_c^*-\epsilon_2b_2I_c-\beta_1ES_c-\beta_2I_cS_c-\mu_2S_c]\\&\quad+B\left(1-\frac{I_c^*}{I_c}\right)\left[\epsilon_2b_2I_c+\beta_1ES_c+\beta_2I_cS_c-\left(\frac{\epsilon_2b_2I_c^*+\beta_1E^*S_c^*+\beta_2I_c^*S_c^*}{I_c^*}\right)I_c\right]\\&\quad+C\left(1-\frac{M_c^*}{M_c}\right)\left[\gamma_2I_c-\left(\frac{\gamma_2I_c^*}{M_c^*}\right)M_c\right]+D\left(1-\frac{E^*}{E}\right)\left[\eta_1I_c+\eta_2M_c-\left(\frac{\eta_1I_c^*+\eta_2M_c^*}{E^*}\right)E\right].\end{aligned} \tag{3.18}$$

Simplifying (3.18), then

$$\begin{aligned}\dot{\mathcal{V}}(t) =&\mu_1 S_h^*\left(2-\frac{S_h}{S_h^*}-\frac{S_h^*}{S_h}\right)+\mu_2 S_c^*\left(2-\frac{S_c}{S_c^*}-\frac{S_c^*}{S_c}\right)\\&+\epsilon_1 b_1 I_h^*\left(1-\frac{S_h^*}{S_h}-\frac{I_h}{I_h^*}+\frac{I_h S_h^*}{I_h^* S_h}\right)+\epsilon_2 b_2 I_c^*\left(1-\frac{S_c^*}{S_c}-\frac{I_c}{I_c^*}+\frac{I_c S_c^*}{I_c^* S_c}\right)\\&+\beta_3 I_h^* S_h^*\left(1-\frac{I_h S_h}{I_h^* S_h^*}-\frac{S_h^*}{S_h}+\frac{I_h}{I_h^*}+A\frac{I_h S_h}{I_h^* S_h^*}-A\frac{I_h}{I_h^*}-\frac{S_h}{S_h^*}+A\right)\\&+\beta_2 I_c^* S_h^*\left(1-\frac{I_c S_h}{I_c^* S_h^*}-\frac{S_h^*}{S_h}+\frac{I_h}{I_h^*}+A\frac{I_c S_h}{I_c^* S_h^*}-A\frac{I_h}{I_h^*}-\frac{I_c S_h I_h^*}{I_c^* S_h^* I_h^*}+A\right)\\&+\beta_2 I_c^* S_c^*\left(1-\frac{I_c S_h}{I_c^* S_c^*}+\frac{S_c^*}{S_c}+\frac{I_c}{I_c^*}+B\frac{I_c S_h}{I_c^* S_c^*}-B\frac{I_c}{I_c^*}-B\frac{S_c}{S_c^*}+B\right)\\&+\beta_1 E^* S_c^*\left(1-\frac{E S_c}{E^* S_c^*}-\frac{S_c^*}{S_c}+\frac{E}{E^*}+B\frac{E S_c}{E^* S_c^*}-B\frac{I_c}{I_c^*}-B\frac{E S_c I_c^*}{E^* S_c^* I_c^*}+B\right)\\&+C\gamma_2 I_c^*\left(1+\frac{I_c}{I_c^*}-\frac{M_c}{M_c^*}-\frac{I_c M_c^*}{I_c^* M_c^*}\right)+D\eta_1 I_c^*\left(1+\frac{I_c}{I_c^*}-\frac{E}{E^*}-\frac{I_c}{I_c^*}\frac{E^*}{E}\right)+D\eta_2 M_c^*\left(1+\frac{M_c}{M_c^*}-\frac{E}{E^*}-\frac{M_c}{M_c^*}\frac{E^*}{E}\right).\end{aligned} \tag{3.19}$$

As in [1], $A=B=C=D=1$ and $I_h(t)\le I_h^*, I_c(t)\le I_c^*, M_c(t)\le M_c^*, E(t)\le E^*$ which implies that $\frac{I_h}{I_h^*}\le 1$, $\frac{I_c}{I_c^*}\le 1$, $\frac{M_c}{M_c^*}\le 1$, $\frac{E}{E^*}\le 1$. Hence, $\dot{\mathcal{V}}(t)$ reduces to

$$\begin{aligned}\dot{\mathcal{V}}(t) \le&\mu_1 S_h^*\left(2-\frac{S_h}{S_h^*}-\frac{S_h^*}{S_h}\right)+\mu_2 S_c^*\left(2-\frac{S_c}{S_c^*}-\frac{S_c^*}{S_c}\right)+\beta_3 I_h^* S_h^*\left(2-\frac{S_h^*}{S_h}-\frac{S_h}{S_h^*}\right)+\beta_2 I_c^* S_h^*\left(2-\frac{S_h^*}{S_h}-\frac{S_h}{S_h^*}\right)\\&+\beta_2 I_c^* S_c^*\left(2-\frac{S_c^*}{S_c}-\frac{S_c}{S_c^*}\right)+\beta_1 E^* S_c^*\left(2-\frac{S_c^*}{S_c}-\frac{S_c}{S_c^*}\right).\end{aligned} \tag{3.20}$$

Since $GM\le AM$ [36], $\dot{\mathcal{V}}(t)\le 0$, and by La Salle's Invariance Principle [38], the invariant set in $\{(S_h,I_h,S_c,I_c,M_c,E)\in\mathbb{R}_+^6 : (S_h,I_h,S_c,I_c,M_c,E)\to\mathcal{W}^*\}$ is the singleton $\mathcal{W}^*$. Therefore, any solution to (2.2) that intersect the interior of $\mathbb{R}_+^6$ limits to $\mathcal{W}^*$. Hence, $\mathcal{W}^*$ is globally asymptotically stable in Ω whenever $\mathcal{R}_\circ>1$.

3.5 Bifurcation analysis

Toxoplasmosis can be eradicated from the community if $\mathcal{R}_\circ<1$ but some factors which depend on the model parameters can make the eradication difficult even when $\mathcal{R}_\circ<1$, a phenomenon known as backward bifurcation [7, 34]. We investigate the existence of backward bifurcation for our model using the center manifold theory [16] as in [7, 34]. The Jacobian of the system (2.2) at DFE $\mathcal{W}_0$ with $\beta_1=\beta_1^*$ is computed as

$$J(\mathcal{W}_0)|_{\beta_1=\beta_1^*}=\begin{pmatrix}-\mu_1 & -(\epsilon_1 b_1+\beta_3 p_1) & 0 & -\beta_2 p_1 & 0 & 0\\ 0 & \epsilon_1 b_1+\beta_3 p_1-(\mu_1+\gamma_1) & 0 & \beta_2 p_1 & 0 & 0\\ 0 & 0 & -\mu_1 & -(\epsilon_2 b_2+\beta_2 p_2) & 0 & -\beta_1^* p_2\\ 0 & 0 & 0 & \epsilon_2 b_2+\beta_2 p_2-(\mu_2+\gamma_2+\eta_1) & 0 & \beta_1^* p_2\\ 0 & 0 & 0 & \gamma_2 & -(\mu_2+\eta_2) & 0\\ 0 & 0 & 0 & \eta_1 & \eta_2 & -(\delta+\phi)\end{pmatrix}. \tag{3.21}$$

The right eigen vector corresponding to $J^* = J(\mathcal{W}_0)|_{\beta_1=\beta_2^*}$ is computed as

$$\begin{aligned}
w_1 &= \frac{1}{\mu_1}\left\{\frac{\beta_2 p_1(\mu_1+\gamma_1)}{(\epsilon_1 b_1+\beta_3 p_1-(\mu_1+\gamma_1))}\right\}w_4,\\
w_2 &= -\frac{\beta_2 p_1}{(\epsilon_1 b_1+\beta_3 p_1-(\mu_1+\gamma_1))}w_4,\\
w_3 &= -\frac{\mu_2+\gamma_2+\eta_1}{\mu_2}w_4<0,\\
w_4 &= w_4>0\\
w_5 &= \frac{\gamma_2}{(\mu_2+\eta_2)}w_4>0,\\
w_6 &= -\frac{(\epsilon_2 b_2+\beta_2 p_2-(\mu_2+\gamma_2+\eta_1))}{\beta_1^* p_2}w_4.
\end{aligned} \tag{3.22}$$

The left eigne vector for $J^* = J(\mathcal{W}_0)|_{\beta_1=\beta_1^*}$ which met $\mathbf{v}.\mathbf{w} = \mathbf{1}$ is $v_1 = v_2 = v_3 = v_5 = v_6 = 0$ but $v_4 = v_4 > 0$. The bifurcation coefficients of a and b are then computed following [16] as

$$\begin{aligned}
a &= 2v_4w_3\{\beta_1^*+\beta_2\}(w_4+w_6) = -\frac{2v_2}{\mu_2\beta_1^* p_2}(\beta_1^*+\beta_2)(\mu_2+\gamma_2+\eta_1)\{1-(\epsilon_2 b_2+\beta_2 p_2-(\mu_2+\gamma_2+\eta_1))\}v_4w_4^2,\\
b &= p_2v_4w_4>0.
\end{aligned} \tag{3.23}$$

Considering (3.23), backward bifurcation is possible for the *T.gondii* model as the bifurcation coefficient *"a"* can be positive especially with the existence of parameters ϵ_2 and β_2, fraction of cats born from infected mothers and contact rate between humans and cats respectively. This shows that vertical transmissions in both humans and cats as well as contact with the infected cats can make *T.gondii* eradication difficult.

3.6 Sensitivity analysis

There is need to investigate the relative contributions of the model parameters to *T. gondii* spread and control to determine the parameters to be focused while designing interventions against *T. gondii* . Following the approach in [6], the sensitivity of some parameters to *T. gondii* dynamics is computed as follows

$$\begin{aligned}
\Gamma_{\beta_2}^{\mathcal{R}_{\circ hc}} &= \frac{b_1}{\mu_1(\mu_1+\gamma_1)}\times\frac{\beta_2}{\mathcal{R}_{\circ hc}},\\
\Gamma_{\beta_3}^{\mathcal{R}_{\circ hc}} &= \frac{b_1}{\mu_1(\mu_1+\gamma_1)}\times\frac{\beta_3}{\mathcal{R}_{\circ hc}},\\
\Gamma_{\epsilon_1}^{\mathcal{R}_{\circ hc}} &= \frac{b_1}{\mu_1(\mu_1+\gamma_1)}\times\frac{\epsilon_1}{\mathcal{R}_{\circ hc}},\\
\Gamma_{b_2}^{\mathcal{R}_{\circ ec}} &= \frac{\beta_2+\epsilon_2}{\mu_2(\mu_2+\gamma_2+\eta_1)}+\frac{\beta_1(\eta_2\gamma_2+\eta_1(\mu_2+\eta_2))}{(\mu_2+\eta_2)(\mu_2+\gamma_2+\eta_1)(\delta+\phi)}\times\frac{b_2}{\mathcal{R}_{\circ ec}},\\
\Gamma_{\beta_1}^{\mathcal{R}_{\circ ec}} &= \frac{b_2(\eta_2\gamma_2+\eta_1(\mu_2+\eta_2))}{(\mu_2+\eta_2)(\mu_2+\gamma_2+\eta_1)(\delta+\phi)}\times\frac{\beta_1}{\mathcal{R}_{\circ ec}},\\
\Gamma_{\epsilon_2}^{\mathcal{R}_{\circ ec}} &= \frac{b_2}{\mu_2(\mu_2+\gamma_2+\eta_1)}\times\frac{\epsilon_2}{\mathcal{R}_{\circ ec}}.
\end{aligned} \tag{3.24}$$

4 Numerical Simulations and graphs

We gather data from several sources and solve the system using fourth order Runge-Kutta technique. Some parameters' values are real; for instance, the natural mortality rate of human is $\mu_1 = 0.000039$ per day, which corresponds to a 70-year old person's life expectancy, and the mortality rate of cats is $\mu_2 = 0.00021$ per day, which corresponds to a cat's average lifespan of 13 years. A number of values for the parameters is selected from [37, 10]. The daily recruitment rates for humans and cats are $b_1 = 100$ and $b_2 = 5$, respectively. The rates of contribution of the acutely infected cats to environmental contamination are $\eta_1 = 0.027 - 0.3$ and the rate of contribution of the mildly infected cats to environmental contamination are $\eta_2 = 0.00027 - 0.003$, respectively. $\beta_1 = 0.01 - 0.15$, $\beta_2 = 0.008$,

are the spreading rates of *T. gondii* from environment to cat and human to cat population, respectively, $\beta_3 = 0.001$ is spreading rate of *T. gondii* from human to human. The human recovery rate is $\gamma_1 = 0.5$ while oocyst shedding is terminated at rate $\gamma_2 = 0.47 - 1$. We also make assumptions about the initial population sizes, $\epsilon_1 = 0.005$ and $\epsilon_2 = 0.001$. In rare instances the new offspring of infected cats are infected with *T. gondii* so take $\epsilon_2 = 0.001$ and consider that humans transmit *T. gondii* vertically at $\epsilon_1 = 0.005$. The rate of loss of oocysts due to nature and due to human intervention are assumed to be $\delta = 0.058 - 0.096$ and $\phi = 0.085 - 0.5$ respectively. The definitions and values for the parameters can be accessed at a glance from Table 1. With these values, we obtain the indices of sensitivity for the model parameters in Table 2.

Table 2: Nomenclatures for the model parameters

parameters	signs	sensitivity indices
β_2	+	0.57
β_3	+	0.07
ϵ_1	+	0.36
b_2	+	1
β_1	+	0.0037
ϵ_2	+	0.2

In Table 2, the contributions of some parameters to *T. gondii* spread are revealed. It is deduced that recruitment rate of cats b_2 has the highest contribution to *T. gondii* spread. This shows that toxoplasmosis is likely to be a challenge in a community where there is no restriction in the population of cats. Another parameter with high rate of contribution to *T. gondii* spread is effective contact between susceptible humans and infectious cats and susceptible cats and infectious cats β_2. The index for β_2 in Table 2 means that failure to identify infected cats on time for prompt treatment can escalate *T. gondii* transmission. Also, vertical transmission in humans ϵ_1 is sensitive to toxoplasmosis transmission. The implication of index for ϵ_1 is that pregnant women have to be monitored for toxoplasmosis to prevent *T. gondii* spread in human population.

Let $S_h(0) = 800, I_h(0) = 75, S_c(0) = 150, I_c(0) = 35, M_c(0) = 20$, and $E(0) = 300$ be the initial values for the model's variables. We graphically depict the outcomes after solving to demonstrate the effects of recruitment rate for cats, vertical transmission, sanitation and contact with the infected cats. Figure 2 depicts how recruitment rate for cats affects the population of infectious humans. Figure 3 depicts how vertical transmission affects the population of susceptible and infectious humans. Figure 4 depicts how oocysts contribution from the mildly infected cats affects the population *T. gondii* parasites. Figure 5 depicts how improved sanitation affects the population of infectious humans and pathogens. Figure 6 depicts how poor sanitation affects the population of infectious humans and pathogens. Lastly, Figure 7 depicts how contact with the infected cats affects population susceptible and infectious humans.

We can observe from Figure 2 that increasing the recruitment rate for cats from 5 to 15 increases the number of individuals who are infected with *T. gondii* and the increase reach the peak when the highest number of cats is recruited. This is an indication that prevention and control measures have to be intensified should the population of cats increase because *T. gondii* infection might spread if the population of cats is not controlled.

Figure 3 shows that as more and more children are born from infected women, individuals who are susceptible to *T. gondii* declines (Figure 3a) while those who are infected with the parasite rise (Figure 3b). It is therefore shown that infection with *T. gondii* during pregnancy can exarcerbate *T. gondii* transmission in human population.

In Figure 4, although the population of oocysts declines continuously over a period of a decade which might be as a result of mortality from natural and human factors, the possibility of oocysts contribution from the mildly infected cats still pushes the curves outward, an indication of increase in the oocysts population in some regards. The implication of mildly infection in cats for *T. gondii* transmission in human population as shown in Figure 4 is that every cat should be regarded as a potential carrier of *T. gondii* and should be put under close monitoring to forstall cat-to-human spread of toxoplasmosis.

In Figure 5, as environmental sanitation improved and increased from more the 30% through to 50%, the population of individuals who are infected with *T. gondii* decreased and the decrease is highest when the improvement in sanitation attained 50% (Figure 5a). Also, the population of pathogen in the environment reduced with the improved sanitation (Figure 5b). This shows that improved sanitation is key to the elimination of the parasite as well as the eradication of *T. gondii* infection in human population.

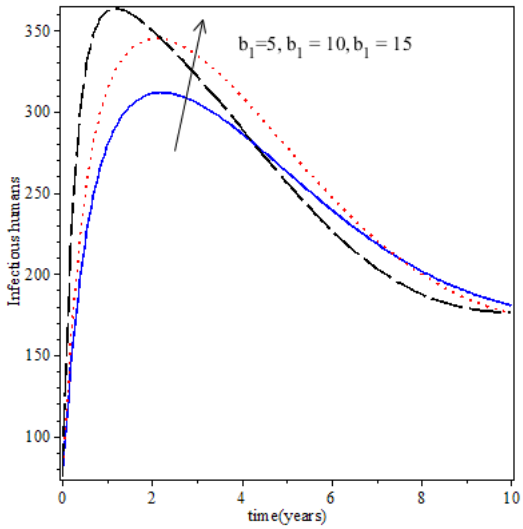
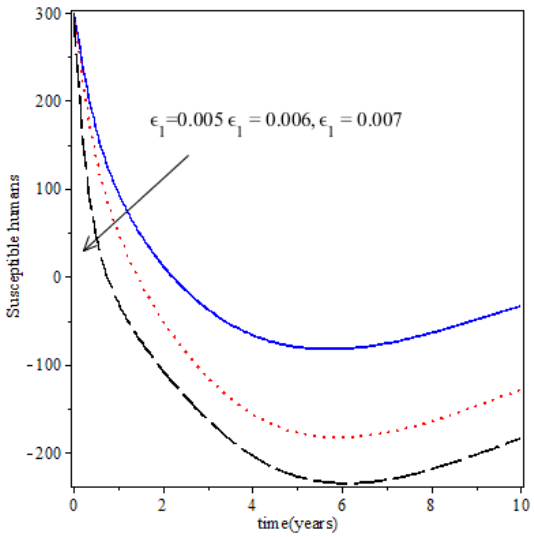
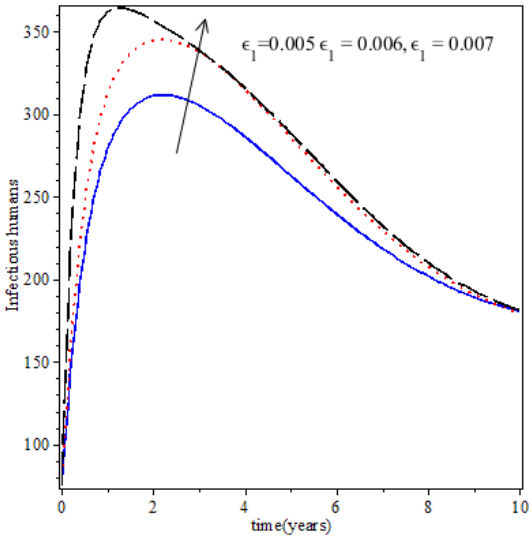


Figure 2: Effect of increasing recruitment rate for cats.



(a) Effect of increasing vertical transmission on susceptible humans



(b) Effect of increasing vertical transmission on infectious humans

Figure 3: Effect of *T. gondii* infection during pregnancy

Unlike in Figure 5, Figure 6 indicates the effect of poor sanitation on the population of infectious human and pathogen. As environmental sanitation fell from more that 30% through to 20%, the number of infected people increased (Figure 6a) and the population of the pathogen in the environment also increased (Figure 6b). Therefore, *T. gondii* parasites multiply and the infection escalates in a human population where sanitation is poor.

In Figure 7, increase in contact with infected cats reduces the population of humans who are susceptible to the disease (Figure 7a) but increases the population of those who are infected (Figure 7b). Since avoidance of cats is not easy for individuals who keep them and the fact that cats are asymptomatic to *T. gondii* infection, frequent examination of *T. gondii* infection is necessary for cats to prevent *T. gondii* transmission from cats to humans.

5 Conclusion

Toxoplasmosis transmission has serious health and economic implications. It is transmitted to humans and other animals through parasites that can spread from the infected agents to the susceptible populations via various modes. This study developed a model for the dynamics of toxoplasmosis that include horizontal and vertical transmissions as

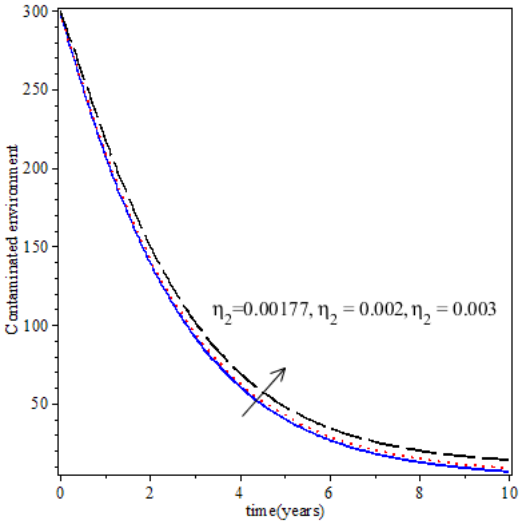
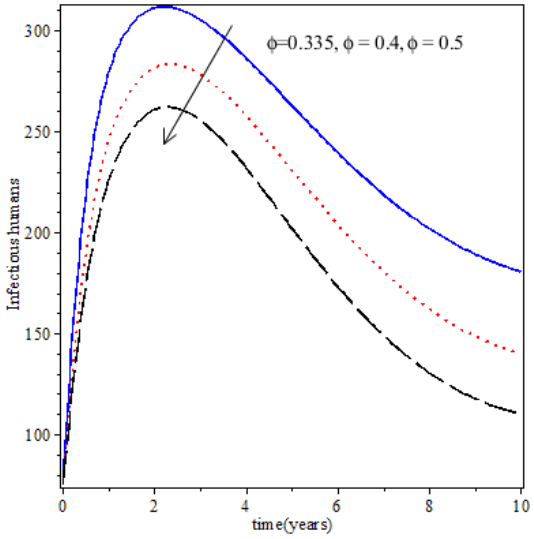
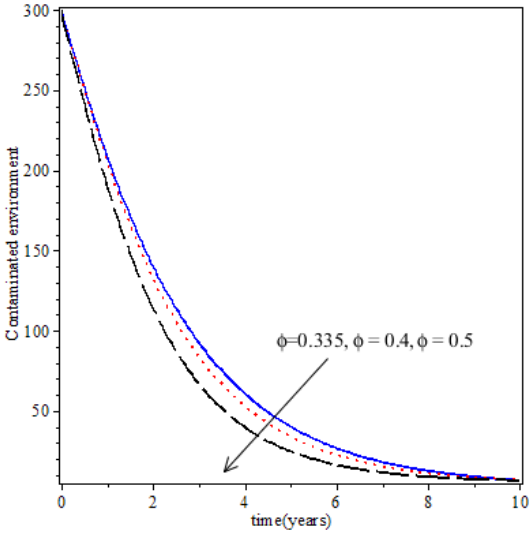


Figure 4: Effect of increasing oocysts contribution from the mildly infected cats



(a) Effect of improved sanitation on the population of infectious humans



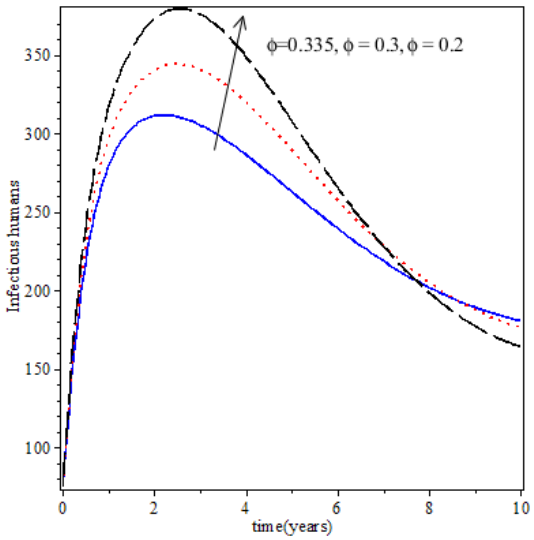
(b) Effect of improved sanitation on the population of parasites

Figure 5: Effect of sanitation on the spread of *T. gondii* infection in humans

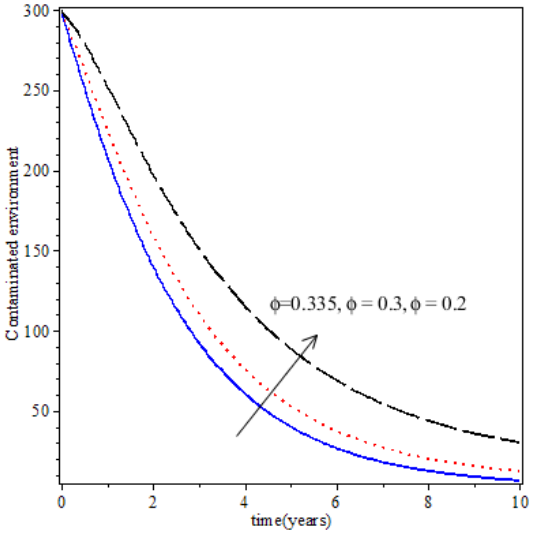
well as the contribution of the parasites from the mildly infected cats. In the work, we identified the factors that can cause *T. gondii* to be eradicated and those that can cause it to persist. We examined *T. gondii* dynamics with a focus on the equilibria stability. To investigate the equilibria stability locally, we computed and applied the characteristic equations. We show that the DFE is locally stable if $\mathcal{R}_0 < 1$. In contrast, the endemic equilibrium is locally stable if $\mathcal{R}_0 > 1$. The Lyapunov theorem was used to determine the global stability of the DFE. In particular, the DFE is globally stable if $\mathcal{R}_0 < 1$, and *T. gondii* eventually goes extinct.

Overall, our findings give a better understanding of *T. gondii* dynamics. Some parameters were selected from toxoplasmosis literature. Though, some of the parameters have some degree of uncertainties as are general in biological processes. We can observe from the expression for $\mathcal{R}_0$ that the endemic equilibrium and the value for $\mathcal{R}_0$ are influenced by the recruitment rate for cats, vertical transmission rates, contact rate with infected cats and oocysts contributions to the environment. For example, when all the stated parameters rise, $\mathcal{R}_0$ increases and this has negative control effects on *T. gondii*.

Graphical simulations are used to display the behavior of the disease. The effect of increase in the population of cats on the spread of toxoplasmosis is indicated in Figure 2 where an increase in the population of cats is accompanied with a

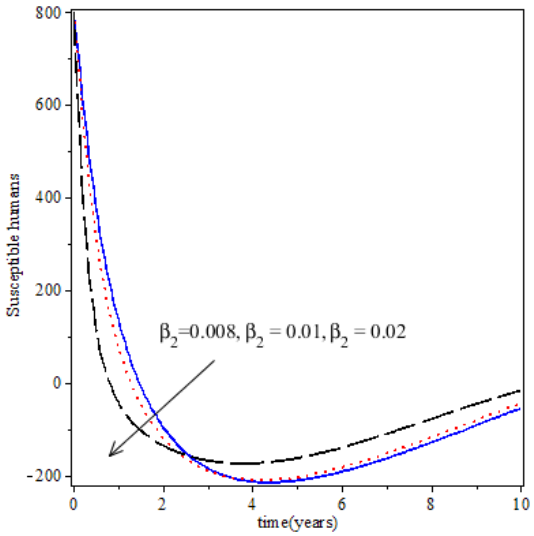


(a) Effect of poor sanitation on the population of infectious humans

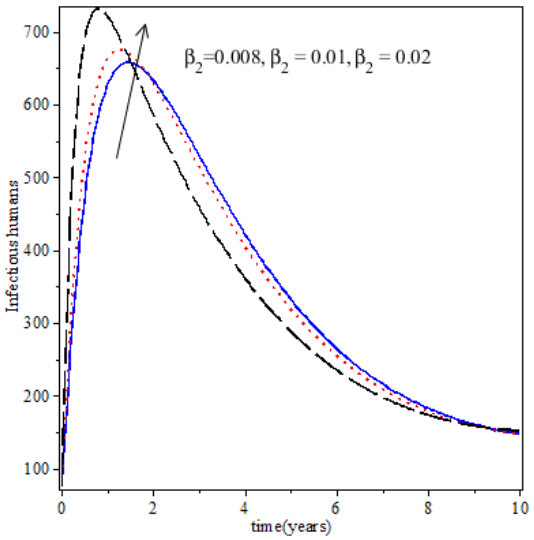


(b) Effect of poor sanitation on the population of parasites

Figure 6: Effect of sanitation on the spread of *T. gondii* infection in humans



(a) Effect of increased contacts with infectious cats on the population of susceptible humans



(b) Effect of increased contacts with infectious cats on the population of infectious humans

Figure 7: Effect of contact with infectious cats on humans.

corresponding increase in the population of infectious humans. The impact of vertical transmission on the transmission of the disease is depicted in Figure 3. As the vertical transmission rises, the susceptible human population falls while the infected human population rises. Figure 4 indicates the effect of contribution of the parasites from the mildly infected cats. As the contribution of the parasites from the mildly infected cats increases, the environment becomes more contaminated with *T. gondii*.

Figures 5 and 6 show the effect of sanitation on the dynamics of *T. gondii*. While improved sanitation reduces the population of infectious humans and pathogen, poor sanitation increases their populations. Figure 7 indicates the effects of contact with the infected cats on the human populations. As contact with the infected cat rises, the susceptible human population falls but the infected human population rises. The analysis shows that recruitment rate for cats, vertical transmission, contact with the infected cats, sanitation and contribution of *T. gondii* from the mildly infected cats can shape the dynamics of toxoplasmosis. Therefore, effective monitoring of pregnant women and cats

for *T. gondii* infection and every other intervention that can make the environment free from *T. gondii* are necessary to prevent or control the spread of toxoplasmosis.

This study is not without limitations and some of the limitations would be mentioned to give direction for fututure work. The model developed in this study is based on the assumption of homogeneous mixing which might not be true for toxoplasmosis in all regions. Therefore, further studies are needed in this regard. The values used for basic transmission rates are not clear and vary from one place to another. The effective contact rate between infected and susceptible cats and infected cats and susceptible humans is assumed equal. This might not be true but calls for extensive more biological field research.

Finally, we want to emphasize that studies of this kind shed more light on toxoplasmosis condition and can aid public health officials in lowering the incidence of toxoplasmosis.

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