Journal of Fractional Calculus and Applications
Vol. 14(1) Jan. 2023, pp. 75-87
ISSN: 2090-5858.
http://math-frac.org/Journals/JFCA/

MATHEMATICAL MODELING OF *TOXOPLASMA GONDII* BETWEEN THE ENVIRONMENT AND CAT POPULATION UNDER VACCINATION AND SANITATION

A. A. AYOADE, T. OYEDEPO, S. AGUNBIADE

Abstract. We examined the transmission dynamics of *Toxoplasma gondii* between the environment and cat population taking vaccination and sanitation as control strategies. Because of adverse effects of toxoplasmosis on fetuses, adequate knowledge about its dynamics in cat population is crucial to planning interventions against the parasite. Considering five compartments - four for cats and one for the environment, a mathematical model of toxoplasmosis spread in cat population was developed. The model and its results were presented and the conditions for disease eradication and persistence were established. Particularly, it was discovered from sensitivity analysis that poor vaccination and sanitation could instigate disease persistence due to higher shedding rates of oocysts and larger rates of infection. Based on the results obtained, discussions were offered on how vaccination and sanitation could be applied to reduce the spread of *Toxoplasma gondii* to other animals that are at risk of toxoplasmosis.

1. Introduction

Toxoplasmosis is a parasitic infection caused by *Toxoplasma gondii* (*T. gondii*). The disease was discovered in 1908 and affects about two-third of the global population, the majority of infections occurs in the tropics [1]. The primary host for *T. gondii* is cats [2], although animals like sheep, dog, birds, rodents, goats, cattle, swine, etc. are potential reservoirs of the parasite [3]. *T. gondii* infections may spread from unhygienic contact with infected excreta of the hosts especially cat litters, consumption of infected food, or in rare occasion, from infected mothers to their fetuses (vertical transmission) [4].

Like cats, many hosts for *T. gondii* remain asymptomatic. However, *T. gondii* infection is severe in high risks individuals with impaired immunity. Individuals in "high risk" group include babies (fetuses), pregnant women, very elderly individuals, young children and immunosuppressed individuals e.g. individuals living with AIDS, individuals on anticancertherapy and organ transplant recipients on immunosuppressive treatment [5]. In "high risks" individuals, toxoplasmosis may

2010 *Mathematics Subject Classification.* 92B05, 92D30, 34D20.
Key words and phrases. Toxoplasma gondii, toxoplasmosis, cat, parasite, vaccination, sanitation.
Submitted May 17, 2022. Revised July 1, 2022.

instigate severe illnesses such as inflammation of the brain (encephalitis), stillbirths, miscarriages, birth defect and other illnesses that are capable of distorting eyes and the nervous system [6]. The disease has grave effects on pregnant women. In a study of toxoplasmosis conducted on some infected children, it was discovered that out of one hundred and sixty-two children that were infected with *T. gondii*, sixty-six were put to bed "seriously sick", fifty were born with optical disorder, and thirty-eight out of the fifty children that were born with optical disorder were also suffering neurological impairments [7].

Adequate knowledge of *T. gondii* transmission in cats is important because cats remain the only hosts in which *T. gondii* attains maturity and reproduces [8, 9]. For this reason, cats are important component of the life cycle of the parasite. It is therefore important to study the transmission dynamics of *T. gondii* in cat population to understand how the parasite propagates in cat population and possibly spreads into other animals' populations.

Several mathematical models have been proposed to analyze the transmission dynamics of *T. gondii* in cat population only [10, 11, 12, 13, 14, 15] and also, in both human and cat populations [5, 16, 17, 18]. In the epidemiology of toxoplasmosis, the environment is contaminated with *T. gondii* through the shedding of oocysts from the infectious cat excreta. Besides, a cat remains infected throughout its life once it is infected with *T. gondii*, although infectious cats only shed oocysts in their excreta for 1-3 three weeks after which the shedding terminates and the parasite becomes dormant in the cats [13, 19, 20, 21, 22].

Some existing toxoplasmosis models do not account for permanence of *T. gondii* in the infected cats by adding recovered compartment in the *T. gondii* models [14]. Also, with the exemption of the model in [13], existing toxoplasmosis models lack chronic stage of *T. gondii* infection in cats when the shedding of oocysts terminates and the parasite becomes dormant in the infected cats for life. This is an important aspect of toxoplasmosis dynamics, the omission of which leaves a serious gap in toxoplasmosis study. Although the model in [13] appears elegant and robust, it does not include any prevention strategies. Susceptibility to *T. gondii* infection can be reduced by vaccination while oocysts revoval or elimination from the environment can be increased by improved sanitation. Therefore, vaccination and sanitation can play key roles in limiting infections with *T. gondii* and shape dynamics of the disease.

In this study, we extend a toxoplasmosis model in [13] by incorporating a compartment for vaccination and adding a sanitation parameter to the compartment for the environment. We investigate how vaccination and sanitation can limit susceptibility and the population of oocysts in the environment and subsequently influence the propagation of *T. gondii* in the population of cat. Both the computational and analytical methods from differential equations (numerical solutions together with steady-state, stability and sensitivity analyzes) are employed to gain insight into the transmission dynamics of *T. gondii* between cats taking the impacts of vaccination and sanitation into consideration.

2. Model Formulation

To develop the model, cat population which is sub-categorized into susceptible (S(t)); vaccinated (V(t)); seriously infected (I(t)); mildly infected (M(t)) together

with the environment (E(t)) is considered. The population of cats that are successfully vaccinated (V(t)) is increased via recruitment either by birth or migration with a fraction ϕ ($0 \leq \phi \leq 1$) at a rate k (ϕ quantifies the number of cats that are temporarily immune to the parasite at the point of entry). The vaccinated group however decreases by σ and μ, the rates of loss of acquired immunity and natural death respectively. Hence, the rate of change of vaccinated cats with time is given as

$$\frac{dV}{dt} = \phi k - (\sigma + \mu)V.$$

The population of cats that are susceptible to *T. gondii* (S(t)) increases by $(1-\phi)k$ and σ, the recruitment rate and the rate of loss of acquired immunity respectively. The population, however, decreases through infection, at a rate ω and through natural death, at a rate μ, so that the rate of change in the population of susceptible cats with respect to time is given as

$$\frac{dS}{dt} = (1 - \phi)k + \sigma V - \omega ES - \mu S.$$

The population of seriously infectious cats (I(t)) is increased through effective contact between susceptible cats and contaminated environment at a rate ω but reduces owing to termination of shedding of oocysts and natural mortality at rates γ and μ respectively. Therefore, the rate of change of the population of seriously infectious cat with time is given as

$$\frac{dI}{dt} = \omega ES - (\gamma + \mu)I.$$

The population of mildly infected cats is increased at the expiration of shedding of oocysts at a rate γ but reduces through natural death at a rate μ so that the rate of change of the population of mildly infectious cats with time is given as

$$\frac{dM}{dt} = \gamma I - \mu M.$$

Lastly, the population of oocysts increases in the environment when they are shed by the seriously infectious cats at a rate ϵ but reduces through natural death and sanitation at rates δ and η respectively. Hence, the rate of change of oocyst population in the environment with respect to time is given as

$$\frac{dE}{dt} = \epsilon I - (\delta + \eta)E.$$

The transfer between the compartments of the model is illustrated in Figure 1.

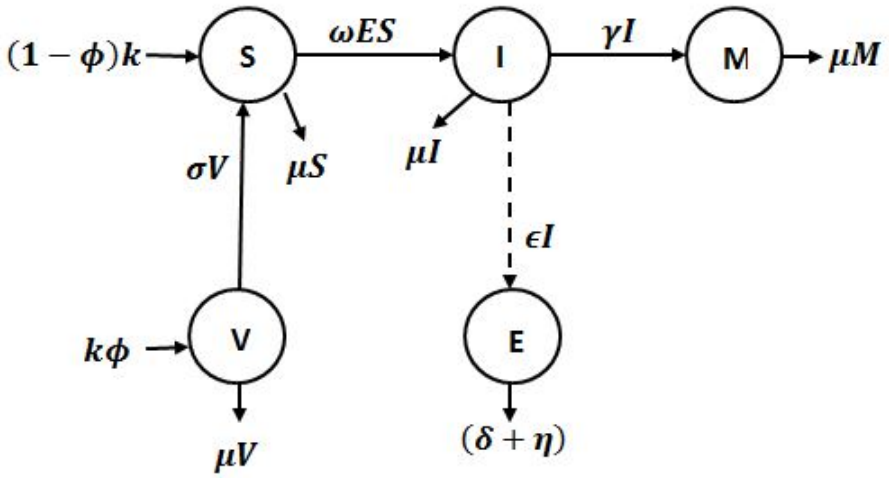

Figure 1: Transfer diagram of toxoplasmosis model

Based on the aforesaid assumptions, formulations and flow diagram, the mathematical model for the transmission of toxoplasmosis in cat population is made up of the below system of ODEs.

$$\frac{dV}{dt} = \phi k - (\sigma + \mu)V, \tag{1}$$

$$\frac{dS}{dt} = (1-\phi)k + \sigma V - \omega ES - \mu S, \tag{2}$$

$$\frac{dI}{dt} = \omega ES - (\gamma + \mu)I, \tag{3}$$

$$\frac{dM}{dt} = \gamma I - \mu M, \tag{4}$$

$$\frac{dE}{dt} = \epsilon I - (\delta + \eta)E, \tag{5}$$

subject to the initial conditions

$$V(0) = V_\circ, S(0) = S_\circ, I(0) = I_\circ, M(0) = M_\circ, E(0) = E_\circ.$$

The model is built around the following main assumptions:

(1) mildly infected cats do not contribute to the growth of oocysts in the environment;
(2) cats in the vaccinated group do not interact with the contaminated environment and do not pick up infections unless their immunity is lost or they become stray.

The associated parameters of the model together with their range of values measured per week, which are from [13] as well as assumptions, are restated in Table 1. The parameter values from [13] are estimated by the author in the appendix.

Table 1. Description of parameters for the model

Parameters' Descriptions	Symbols	Rang of Values	Reference
Natural death rate	μ	0.0019-0.0058	[13]
Recruitment rate by birth and immigration	k	0.58-1.73	[13]
Effective contact rate	ω	0.01-0.15	[13]
Fraction of cats that are successfully vaccinated at birth	ϕ	0.001-0.1	Assumed
Termination rate of shedding of oocysts	γ	0.47-1	[13]
Waning rate of immunity	σ	0.008-0.08	Assumed
Shedding rate of oocysts	ϵ	0.027-0.3	Assumed
Rate of removal of oocysts unrelated to sanitation	δ	0.058-0.096	[13]
Rate of removal of oocysts due to sanitation	η	0.085-0.5	Assumed

In Figure 1, compartment M does not flow into any of the other compartments, hence the system dynamics is not affected by equation (4). Compartment M is therefore excluded from the model and the analysis is restricted to the reduced system [23, 24].

2.1. **Basic properties of the model.** For the toxoplasmosis model to be mathematically and epidemiologically valid, it is necessary to establish that it is well-posed and has positive solution at all time.

2.1.1. *Boundedness and solution positivity.* Since the system considers cat and pathogen populations, all the parameters and state variables of the system must be positive. Consider the feasible region

$$\Omega = \left\{ (V, S, I, E) \in \Re_+^4 : V, S, I, E \geq 0; N(t) \leq \frac{k}{\mu}; E(t) \leq \frac{\epsilon k}{\mu(\delta + \eta)} \right\}.$$

We can show that Ω is positively invariant and at the same time, a global attractor. That is, whatever phase trajectory emanated at anywhere within the positive region $\Re_+^4$ finally enters Ω and remains in Ω.
Let $V_\circ, S_\circ, I_\circ, E_\circ \geq 0$ such that $V_\circ, S_\circ, I_\circ = N_\circ$ and $E_\circ = E_\circ$. Hence, there exists the solutions $V(t), S(t), I(t), E(t)$ for the model, with initial conditions $V_\circ, S_\circ, I_\circ, E_\circ$ at initial time $t = 0$. Each solution exists for all $t \geq 0$. In fact, $V(t), S(t), I(t), E(t)$ are positive such that $V(t) + S(t) + I(t) = N(t)$ and $E(t) = E(t)$ for all t. Since $N = V + S + I$, it suffices that the change in the total population of cat with time is given as

$$\frac{dN}{dt} \leq k - \mu N,$$

so that $N(t) \to \dfrac{k}{\mu}$ as $t \to \infty$. Also, the change in the pathogen population with time is given as

$$\frac{dE}{dt} = \epsilon I - (\delta + \eta)E.$$

Notice that I is part of $N(t)$ and $N(t) \leq \dfrac{k}{\mu}$. Therefore, substituting $\dfrac{k}{\mu}$ for I and integrating then, $E(t) \to \dfrac{\epsilon k}{\mu(\delta + \eta)}$ as $t \to \infty$. Hence, the total population for cat and pathogen are asymptotically constant.

Theorem 1. *The model solution $F(t) = (V, S, I, E)$ with non-negative initial condition $F \geq 0$ is positive for $t \geq 0$.*

Proof. Consider equation (1),

$$\frac{dV}{dt} = \phi k - (\sigma + \mu)V.$$

Integrating within $[0, T]$,

$$\frac{d}{dt}\left[V(t)exp\left\{\int_0^T (\sigma+\mu)T\right\}\right] = \phi k\ exp[(\sigma+\mu)T]dT,$$

such that,

$$\begin{aligned} V(T) =& V(0)exp\left[-\int_0^T (\sigma+\mu)T\right] + exp\left[-\int_0^T (\sigma+\mu)T\right] \\ &\times \int_0^T \phi k\ exp\left[\int_0^y (\sigma+\mu)y\right] dy > 0. \end{aligned}$$

Following the same idea, $S(t), I(t), E(t)$ can be proved positive for all $T > 0$. Hence, $F > 0$ for all $t > 0$. □

3. Dynamical behavior of the model

The theoretical results for the toxoplasmosis model are investigated in this section as follows.

3.1. Infection-free equilibrium.

The disease-free equilibrium for the model is obtained as

$$\mathcal{W}_0 = (V^\circ, S^\circ, I^\circ, E^\circ) = \left(\frac{\phi k}{\mu+\sigma}, \frac{(\mu+\sigma)(1-\phi)k + \sigma\phi k}{\mu(\mu+\sigma)}, 0, 0\right).$$

3.2. Effective reproduction number.

In mathematical epidemiology, the reproduction number, whose definition is in [25], is a nondimensional quantity that measures the average number of infections generated by an infectious individual throughout the period of his infectiousness. Following the notations and method in [26], the effective reproduction number $\mathcal{R}_z$ for the model is computed as

$$\mathcal{R}_z = \frac{\omega\epsilon[(\mu+\sigma)(1-\phi)k + \sigma\phi k]}{\mu(\mu+\sigma)(\mu+\gamma)(\delta+\eta)}. \tag{6}$$

3.3. Global stability analysis of infection-free equilibrium.

Theorem 2. *The infection-free equilibrium of the model is globally asymptotically stable if $\mathcal{R}_z < 1$ and the conditions $(H1)$ and $(H1)$ in [27] are satisfied. The conditions $(H1)$ and $(H2)$ are satisfied if the derivative of uninfectious compartments is zero $(H1)$ whenever the derivative of infectious compartments is nonzero $(H2)$.*

Proof. Based on the procedure in [27],

$$\hat{G}(X, Y) = AY - G(X, Y),$$

where X and Y are uninfectious and infectious compartments of the model respectively and A is a Metzler-matrix. A and G are determined after some algebraic processes so that

$$\hat{G}(X,Y) = \begin{pmatrix} -(\mu+\gamma) & \omega S^{\circ} \\ \epsilon & -(\delta+\eta) \end{pmatrix} \begin{pmatrix} I \\ E \end{pmatrix} - \begin{pmatrix} \omega ES & -(\mu+\gamma)I \\ \epsilon I & -(\delta+\eta)E \end{pmatrix},$$
$$\Rightarrow \hat{G}(X,Y) = \begin{pmatrix} \omega E(S^{\circ}-S) \\ 0 \end{pmatrix} = \begin{pmatrix} G_1(X,Y) \\ G_2(X,Y) \end{pmatrix}.$$

It follows that $G_2(X,Y) = 0$ and $G_1(X,Y) \geq 0$ if $S^{\circ} \geq S$. Hence, $\hat{G}(X,Y) \geq 0$ since the inequality $S^{\circ} < S$ cannot hold. Therefore, conditions $(H1)$ and $(H2)$ in [27] hold and the DFE $\mathcal{W}_0$ of the model is globally asymptotically stable if $\mathcal{R}_z < 1$. The implication of Theorem 2 is that whenever $\mathcal{R}_z$ is below unity (i.e., $\mathcal{R}_z < 1$), toxoplasmosis can be eradicated from cat population if the initial size of infectious cats and pathogen sub-populations are in the basin of attraction of DFE ($\mathcal{W}_0$). The result in Theorem 2 also guarantees $\mathcal{R}_z < 1$ as the necessary and sufficient condition for the eradication of *T. gondii* in cat population and rules out the possibility of existence of backward bifurcation for the model. □

3.4. **Existence and global stability of disease persistence equilibrium.** The endemic equilibrium for the toxoplasmosis model is denoted by $\mathcal{W}_*$ with coordinates

$$\mathcal{W}_* = (V^*, S^*, I^*, E^*) = \left(\frac{\phi k}{\mu+\sigma}, \frac{S^{\circ}}{\mathcal{R}_z}, \frac{\delta+\eta}{\epsilon}(\mathcal{R}_z - 1), \frac{\mu}{\omega}(\mathcal{R}_z - 1) \right).$$

Since the model variables are non-negative, the endemic equilibrium exists only if $\mathcal{R}_z > 1$.

Theorem 3. *The toxoplasmosis model contains a unique infection persistence equilibrium $\mathcal{W}_*$ that is globally asymptotically stable if $\mathcal{R}_z > 1$.*

Proof. If $\mathcal{J}$ is a Lyapunov function, the infection-persistence equilibrium $\mathcal{W}_*$ is globally asymptotically stable if $\dfrac{d\mathcal{J}}{dt} \leq 0$.
Define $\mathcal{J}$ as

$$\begin{aligned} \mathcal{J} = &\left(V - V^* - V^* \log \frac{V}{V^*} \right) + \left(S - S^* - S^* \log \frac{S}{S^*} \right) \\ &+ \left(I - I^* - I^* \log \frac{I}{I^*} \right) + \left(E - E^* - E^* \log \frac{E}{E^*} \right). \end{aligned} \tag{7}$$

Finding the derivative of $\mathcal{J}$ and after a few algebraic simplifications,

$$\begin{aligned} \frac{d\mathcal{J}}{dt} = &- \left(1 - \frac{V^*}{V} \right) [(\mu+\sigma)(V - V^*)] - \left(1 - \frac{S^*}{S} \right) [(\omega E - \mu)(S - S^*)] \\ &- \left(1 - \frac{I^*}{I} \right) [(\mu+\gamma)(I - I^*)] - \left(1 - \frac{E^*}{E} \right) [(\delta+\eta)(E - E^*)]. \end{aligned} \tag{8}$$

Since all the variables are non-negative and $\mathcal{W}_{\circ} \geq \mathcal{W}_*$ then $\dfrac{d\mathcal{J}}{dt} \leq 0$. Again, $\dfrac{d\mathcal{J}}{dt} = 0$ if $\mathcal{W}_{\circ} = \mathcal{W}_*$. That is, if all the points in $\mathcal{W}_{\circ}$ are equal to all the points in $\mathcal{W}_*$. Therefore, $\mathcal{W}_*$ is the largest invariant set in $\left\{ (V,S,I,E) \in \Omega : \dfrac{d\mathcal{J}}{dt} = 0 \right\}$. Hence, $\mathcal{W}_*$ is GAS in the interior Ω by LaSalle's invariance principle [28].

The implication of the result of Theorem 3 is that toxoplasmosis will remain in cat population irrespective of the initial size of the seriously infectious cats if $\mathcal{R}_z > 1$. □

4. Sensitivity analysis and simulations

The transmission potential of *T. gondii* is a function of a number of parameters that have been theoretically derived in equation (6). The existence of disease eradication $\mathcal{R}_z < 1$ as well as the disease persistence $\mathcal{R}_z > 1$ are governed by these parameters. Following the approach in [29] and using parameter values in Table 1, the relative contributions of some of the key parameters to toxoplasmosis spread and management are computed and the results are displayed in Table 2.

Table 2. Sensitivity indices of major parameters

Parameters	Indices
ω	+1.000000
ϵ	+1.000000
k	+1.000000
η	-0.810811
ϕ	-0.000032

The result in Table 2 reveals important information about toxoplasmosis dynamics. Effective contact rate (ω), shedding rate of oocyst (ϵ) and the recruitment rate of cats (k) contribute immensely to the spread of *T. gondii* in cat population to the extent that toxoplasmosis spread in cat population approximately increases by 100% if any of the parameters increases by 1. On the other hand, in Table 2, the spread of toxoplasmosis is effectively inhibited by removal rate of oocyst due to sanitation (η) and the fraction of cats that are successfully vaccinated at birth (ϕ). It can be deduced from Table 2 that a 81% improvement in sanitation (η) could prevent toxoplasmosis transmission in cat population by 81%. Since environment is contaminated through shedding of oocyst (ϵ) and infections spread into cat population through contaminated environment (ω), it is therefore evident that any strategy that can aid removal of oocyst from the environment is vital to the elimination of *T. gondii* in cat population. The remaining factor that instigates toxoplasmosis transmission, which is recruitment rate of cats, can be checked by the vaccination of cats at birth. The higher the population of cats that are immune to *T. gondii*, the lower the *T. gondii* susceptibility and infectivity. Therefore, effective vaccination can play a crucial role in limiting toxoplasmosis spread in cat population.

To buttress sensitivity impact of the parameters in Table 2 on toxoplasmosis spread and management and to demonstrate the point where the disease is eradicated ($\mathcal{R}_z < 1$) and the point where it persists ($\mathcal{R}_z > 1$), two illustrations are provided. The exact values of the parameters used to obtained Table 2 are used in illustration 1 while the values of some of these parameters are varied in illustration 2.

Example 1: For $\omega = 0.011, \epsilon = 0.045, \mu = 0.003, \sigma = 0.02, \phi = 0.08, k = 1.5, \gamma = 0.8, \delta = 0.07, \eta = 0.3$, then ($\mathcal{R}_z = 0.8 < 1$), hence the *T. gondii*-free equilibrium ($\mathcal{W}_\circ$) is globally asymptotically stable.

Example 2: For $\omega = 0.019, \epsilon = 0.055, \mu = 0.003, \sigma = 0.02, \phi = 0.079, k = 1.5, \gamma = 0.8, \delta = 0.07, \eta = 0.29$, then ($\mathcal{R}_z = 1.8 > 1$), hence the *T. gondii*-endemic equilibrium ($\mathcal{W}_*$) is globally asymptotically stable.
We see that a small perturbation in the values of the vaccination parameter (ϕ) and the sanitation parameter (η) in example 2 compared to example 1 instigate disease persistence ($\mathcal{R}_z > 1$). To visualize the influence of vaccination and sanitation on the dynamics of *T. gondii*, the parameter space in Example 1 together with the variables initial conditions $V(0) = 200, S(0) = 500, I(0) = 55$ and $E(0) = 250$ are used to generate plots for the model. The impacts of increased vaccination and sanitation on the populations of susceptible and infectious cats as well as the pathagens are therefore displayed in Figures 2-4.

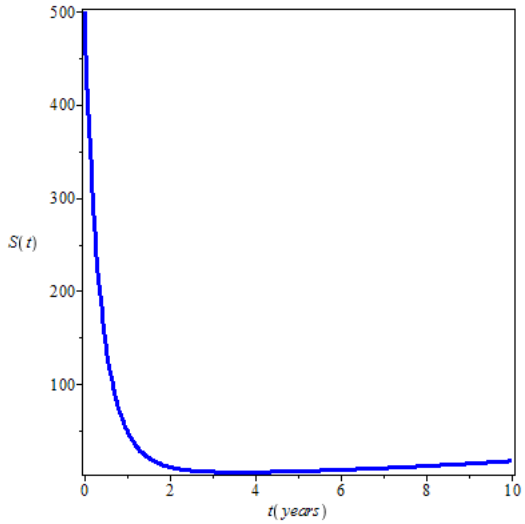

Figure 2: Influence of vaccination on susceptible cats

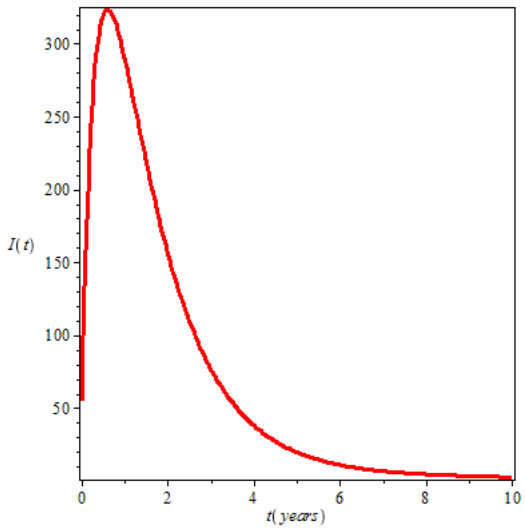

Figure 3: Influence of vaccination on infected cats

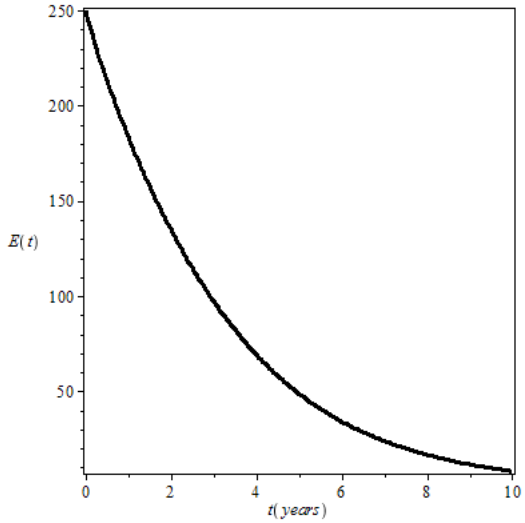

Figure 4: Influence of sanitation on *T. gondii*

It is observed in Figure 2 that the population of susceptible susceptible cats reduces continuously. Also, in Figure 4, the population of *T. gondii* falls continuously and tends to zero after 10 years. However, in Figure 3, the population of infectious cats firstly rises but eventually falls after 2 years and tends to zero after 8 years. Therefore, vaccination and sanitation are indispensable in the eradication of *T. gondii* in cat population. As regards the global stability of both the disease-free and endemic equilibria, Figure 5 and Figure 6 are generated to visualize the behavior of the model variables for the two cases when $\mathcal{R}_z < 1$ and $\mathcal{R}_z > 1$ respectively.

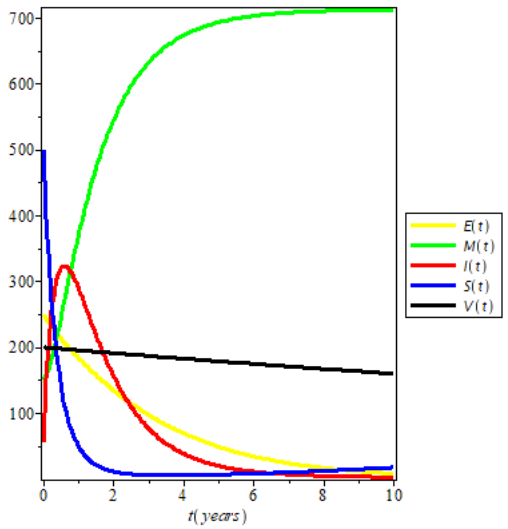

Figure 5: Behavior of model variables when $\mathcal{R}_z < 1$

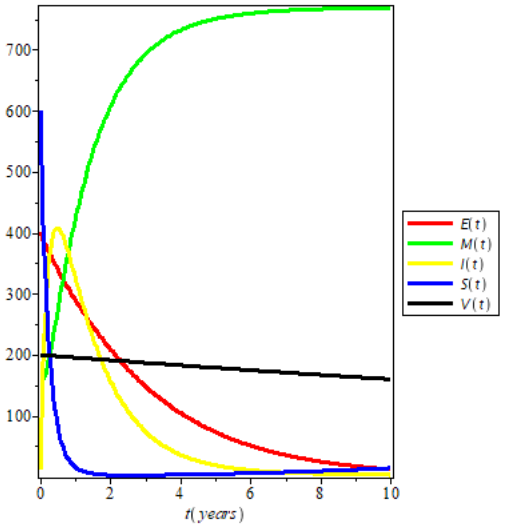

Figure 6: Behavior of model variables when $\mathcal{R}_z > 1$

Figure 5 and Figure 6 show the behaviors of the model when the disease-free equilibrium is stable (i.e., $\mathcal{R}_z < 1$) and when the endemic equilibrium is stable (i.e., $\mathcal{R}_z > 1$)

5. Conclusion

In this paper, we have extended a model of *T. gondii* by incorporating a separate class for vaccination and including a sanitation parameter to the environmental compartment to examine how vaccination and sanitation can influence the propagation of *T. gondii* in cat population. We have carried out a robust analysis to establish the validity of the model and derived the quantity ($\mathcal{R}_z$), the effective reproduction number, that dictates toxoplasmosis spread and eradication. We have also derived condition for the existence of global stability of the infection-free equilibrium and infection-persistence equilibrium in terms of the threshold quantity ($\mathcal{R}_z$) and performed sensitivity analysis for the quantity ($\mathcal{R}_z$) to investigate the impact of each parameter on the transmission and control of toxoplasmosis. The result indicated the effective contact rate (ω), shedding rate of oocyst (ϵ), recruitment rate of cats (k), removal rate of oocyst due to sanitation (η) and vaccination rate (ϕ) as the most sensitive parameters to the threshold parameter ($\mathcal{R}_z$). Based on the theoretical results, simulations were performed to demonstrate the effect of the sensitive parameters on the spread and management of *T. gondii*. In general, the results of this study show that any strategies that ensure removal of oocyst from the environment and at the same time, reduce *T. gondii* susceptibility will drive *T. gondii* eradication in cat population and subsequently prevent the spread of toxoplasmosis into the population of other animals that are at the risk of *T. gondii*. A good example of such strategies is to enforce environmental sanitation practices and to mandate periodic cat vaccination.

References

[1] E. M. Lilly, and C. D. Wortham, High prevalence of *Toxoplasma gondii* oocyst shedding in stray and pet cats *(Felis catus)* in Virginia, United States, Parasites & Vectors, 6, 1, 266-285, 2003.

[2] G. Ramakrishnan, S. Maier, R. A. Walker, H. Rehrauer et al., An experimental genetically attenuated live vaccine to prevent transmission of *Toxoplasma gondii* by cats, Scientic Reports, Vol. 9, 1479-1488, 2019. https://doi.org/10.1038/s41598-018-37671-8.

[3] D. L. Zulpo, A. S. Sammi, J. R. dos Santos et al., *Toxoplasma gondii*: A study of oocyst re-shedding in domestic cats, J. Vet. Parasitol., Vol. 249, 17-33, 2018.

[4] J. A. León Marín and I. D. Gandica, A mathematical model for the reproduction dynamics of toxoplasma gondii, J. Biol. Syst., Vol. 23, S91, 2015. doi: 10.1142/S0218339015400082.

[5] J. D. Ferreira, L. M. Echeverry and C. A. Peña Rincon, Stability and bifurcation in epidemic models describing the transmission of toxoplasmosis in human and cat populations, Math. Meth. Appl. Sci., Vol. 40, 55-75, 2017. doi: 10.1002/mma.4410.

[6] L. L. Manuel, G. G. Santos-Gomes and E. V. Noormahomed, Human toxoplasmosis in Mozambique: gaps in knowledge and research opportunities, Parasites & Vectors, Vol. 13, 571, 2020. https://doi.org/10.1186/s13071-020-04441-3

[7] M. M. Avelino, W. N. Amaral, I. M. X. Rodrigues, A. R. Rassi, M. B. F. Gomes, T. L. Costa, and A. M. Castro, Congenital toxoplasmosis and prenatal care state programs, BMC Infectious Diseases, Vol. 14, 1, 33-4, 2014.

[8] M. T. Balcha, B. I. Aga, D. D. Disasa and G. Berhanu, Public health and economic significance of toxoplasmosis, American-Eurasian Journal of Scientific Research, Vol. 15, 3, 112-121, 2020.

[9] S. Almeria, F. H. A. Murata, C. K. Cerqueira-Cézar, O. C. H. Kwok, A. Shipley, J. P. Dubey, Epidemiological and public health signicance of *Toxoplasma gondii* infection in wild rabbits and hares: 2010-2020, Microorganisms, Vol. 9, 597, 2021. https://doi.org/10.3390/microorganisms9030597

[10] A. J. Arenas, G. González-Parra, and R. J. Villanueva Micó, Modeling toxoplasmosis spread in cat populations under vaccination, Theor. Popul. Biol., Vol. 77, 227-239, 2010. https://doi.org/10.1016/j.tpb.2010.03.005.

[11] M. Hari and Y. Zulfahmi, Bifurcation analysis of toxoplasmosis epidemic control on increased controlled rate of suppressing the rate of infected births, Int. J. Comp. Sci. Appl. Math, Vol. 6, 1, 1-5, 2020 .

[12] M. Langlais, M. Lélu, C. Avenet, and E. Gilot-Fromont, A simplied model system for *Toxoplasma gondii* spread within a heterogeneous environment, Nonlinear Dyn., Vol. 68, 381-340, 2012. doi:10.1007/s11071-011-0255-4.

[13] E. Kelting, (2018). *Toxoplasma gondii*: a mathematical model of its transfer between cats and the environment, https://doi.org/10.1137/175016580

[14] I. B. Muhammada and S. Usaini, Dynamics of toxoplasmosis disease in cats population with vaccination, J. Nig. Soc. Phys. Sci., Vol. 3, 17-25, 2021.

[15] D. F. Aranda, R. J. Villanueva, A. J. Arenas, and G. C. GonzalezParra, Mathematical modeling of toxoplasmosis disease in varying size populations, Comp. Math. Appl., Vol. 56, 690, 2008. https://doi.org/10.1016/j.camwa.2008.01.008.

[16] C. Peña, and K. G. HermesMartinez, Hybrid model of the spread of toxoplasmosis between two towns of Colombia, Tecciencia, Vol. 7, 18, 1-6, 2015. DOI: http:/dx.doi.org/10.18180/tecciencia.2015.18.1

[17] G. C. González-Parra, A. J. Arenas, D. F. Aranda, R. J. Villanueva and L. Jódar, Dynamics of a model of toxoplasmosis disease in human and cat population, Comp.Math. Appl., Vol. 57, 16921700, 2009. https://doi.org/10.1016/j.camwa.2008.09.012.

[18] Z. Zafar, A. Ali and D. Baleanu, Dynamics and numerical investigations of a fractional-order model of toxoplasmosis in the population of human and cat, Chaos, Soliton & Fractals, 2021. https://doi.org/10.1016/j.chaos.2021.111261

[19] R. Blaizot, C. Nabet, L. Laghoe, B. Faivre, S. Escotte-Binet, F. Djossou, E. Mosnier, F. Henaff, D. Blanchet, A. Mercier, M-L. Dardé, I. Villena and M. Demar, Outbreak of Amazonian toxoplasmosis: a one health investigation in a remote Amerindian community, Front. Cell. Infect. Microbiol., 2020. 10:401. doi: 10.3389/fcimb.2020.00401

[20] K. Hatam-Nahavandi, R. Calero-Bernal, M. T. Rahimi, A. S. Pagheh, M. Zarean, A. Dezhkam and E. Ahmadpou, *Toxoplasma gondii* infection in domestic and wild felids as public health

concerns: a systematic review and meta-analysis, *Scientific Reports*, Vol. 11, 9509, 2021. https://doi.org/10.1038/s41598-021-89031-8

[21] G. Milne, J. P. Webster and M. Walker, *Toxoplasma gondii*: an underestimated threat, Trends in Parasitology, Vol. 36, 12, 2020. https://doi.org/10.1016/j.pt.2020.08.005

[22] H. Deng, R. Cummins, G. Schares, C. Trevisan, H. Enemark, H. Waap et al., Mathematical modeling of *Toxoplasma gondii* transmission: a systematic review, Food and Water Parasitology, Vol.22, e00102, 2021. https://doi.org/10.1016/j.fawpar.2020.e00102

[23] J. Wang and C. Modnak, Modeling cholera dynamics with controls, Canadian Applied Mathematics Quarterly, Vol. 19, 3, 255-273, 2011.

[24] A. Ayoade, S. Agboola and M. Ibrahim, Mathematical analysis of the effect of maternal immunity on the global eradication of measles, Annals. Computer Science Series, 17th Tome 1st Fasc. 2019. 2019.

[25] O. Diekmann, J. A. J. Heesterbeek and J. A. J. Metz, On the definition and the computation of the basic reproduction ratio $R_\circ$ in models for infectious diseases in heterogeneous populations, Journal of Mathematical Biology, Vol. 28, 4, 365-373, 1990.

[26] O. Diekmann, J. A. J. Heesterbeek and M. J. Roberts, The construction of next generation matrices for compartmental epidemic models, Journal of The Royal Society Interface, Vol. 7, 47, 873-885, 2010.

[27] C. Castillo-Chavez, Z. Feng and W. Huang, On the Computation of $R_\circ$ and its Role on Global Stability, 2001.

[28] J. P. LaSalle, Stability theory for ordinary dierential equations, Journal of differential equations, Vol. 4, 57-65, 1968.

[29] A. A. Ayoade and O. Aliu, COVID-19 outbreak and mitigation by movement restrictions: a mathematical assessment of economic impact on Nigerian households, Journal of Quality Measurement and Analysis, Vol. 18, 2, 29-44, 2022.

Abayomi A. Ayoade

Department of Mathematics, Faculty of Science, University of Lagos, Lagos, Nigeria

E-mail address: ayoadeabbayomi@gmail.com, ayoayoade@unilag.edu.ng

T. Oyedepo

Department of Applied Sciences, Federal College of Dental Technology and Therapy, Enugu, Nigeria

E-mail address: oyedepotaiye@yahoo.com

S. Agunbiade

Department of Basic Sciences, Babcock University, Ilishan-Remo, Nigeria

E-mail address: agunbiade1971@gmail.com, agunbiades@babcock.edu.ng