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Dynamics of a model of Toxoplasmosis disease in human and cat populations

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ABSTRACT

A mathematical model for the transmission of Toxoplasmosis disease in human and cat populations is proposed and analyzed. We explore the dynamics of the Toxoplasmosis disease at the population level using an epidemiological type model. Discussion of the basic concepts of the Toxoplasmosis transmission dynamics on human and cat populations are presented. The cats in this model plays a role of infectious agents and host of the protozoan Toxoplasma Gondii parasite. Qualitative dynamics of the model is determined by the basic reproduction number, R_0. If the threshold parameter $R_0 < 1$, then the solution converges to the disease free equilibrium point. On the other hand if $R_0 > 1$ the convergence is to the endemic equilibrium point. Numerical simulations of the model illustrates several different dynamics depending on the threshold parameter R_0 and show the importance of this parameter.

1. Introduction

The protozoan Toxoplasma Gondii is a prevalent parasite in wild and domestic animals worldwide. The life cycle of Toxoplasma is unusual in that the organism is capable of indefinite replication using either sexual or asexual subcycles. The asexual cycle can occur in virtually any warm-blooded animal especially in cats [1], and humans. The cats are considered immune to toxoplasma and it can cast more than 20 million oocysts between 4 and 13 days after the infection and these can infect humans [2]. The T. Gondii can be transmitted vertically by tachyzoites that are passed to the fetus via the placenta [3]. The asexual portion of its life cycle consists of just two stages, the rapidly dividing tachyzoites and the more slowly dividing bradyzoites, which can encyst in the brain, heart and other tissues. Transmission occurs when an animal ingests bradyzoite-infected tissue through carnivores or scavenging [4–6]. It can also occur accidentally through feed that is contaminated with animal parts. Theoretically, this asexual portion of the life cycle could continue indefinitely, cycling around the food chain [6].

Throughout history, humans have domesticated different animals, mainly the dogs and cats domesticated 12,000 years and 4000 years ago, respectively. However, the cat is now on the verge of becoming the western world's most popular pet; current predictions are that cats will soon overtake dogs as the most commonly kept pet. According to the Pet Food Institute in Washington, DC, cats already outnumber dogs in the United States, with 70.2 million, in Spain with 5.5 million cats [7] and 10% of households in Colombia have a cat as pet [8].

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The cat is considered a transmission vector of the toxoplasmosis disease, as is considered the A. aegypti to dengue disease [9] and in some small islands where there are different kind of animals but not cats, the prevalence of toxoplasmosis is null [4]. The oocysts released in the feces of infected cats contaminate the environment, including vegetables and other kind of food. Therefore, the Toxoplasma Gondii can be acquired through oral via. This type of contagious has been considered the main route of infection in tropical countries [10]. The prevalence of the Toxoplasma Gondii antibody in some animals ranges from 12% to 60% and in pigs is from 26% to 78%. This ranges show the variability in different Latin American countries, such as Argentina, Brazil and Colombia [11,12]. Toxoplasma infection can be transmitted to humans either by ingestion of tissue cysts in meat or by ingestion of oocysts in cat feces. Some researches have been reported a increasing prevalence of infection with Toxoplasma Gondii in human population [13]. Therefore, based on all the aforementioned facts is important to construct models to study and prevent toxoplasmosis disease.

Mathematical models, simpler than the reality, allow us to understand the global dynamical behavior of the toxoplasmosis disease in the human and cat populations. An important issue that is addressed here is the impact of the epidemics on the human population with the cat population as a vector transmission. In order to explore the dynamics of the toxoplasmosis disease at both populations an epidemiological type model is used. A system of nonlinear differential equations to study the dynamics of the human and cat infected populations is developed. This modeling approach is a standard way to investigate the dynamics of diseases in populations from a epidemiological point of view [14–16]. The proposed system consists on modeling the interactions among susceptible and infective individuals of the two species assuming that the horizontal transmission of the disease to humans is only through the contact with infected cats and vertical transmission in both cat and human populations. In the model, human population is divided in three classes or subpopulations, susceptibles $S_h(t)$, infectious $I_h(t)$ and controlled $C_h(t)$, and the cat population into two classes, susceptibles $S_c(t)$ and infectious $I_c(t)$.

The model is described by a system of nonlinear ordinary differential equations with five equations, which allows to discuss how the different epidemiological parameters influence the global behavior in the evolution of the toxoplasmosis disease in the human and the cat populations. The model include the interactions among susceptible and infective individuals of the two species assuming that the horizontal transmission of the disease to humans is through the contact with infected cats. This transmission is modeled with a classical incidence rate, however may be modeled using another incidence rate such a polynomial one [15,17]. Additionally, vertical transmission in both cat and human populations is considered. In human population vertical transmission is assumed with probability 1. In the cat population it is assumed that a cat borne from a infected one has a probability p_c of not being infected. Furthermore, we assume that both populations of cats and human are constant which is reasonable in some environments where births are approximately balanced by the natural deaths and for short time horizons. In [18] a computer simulation of the transmission of Toxoplasma Gondii was developed considering equilibrium in the cat population size.

In this paper we study the stability of the steady states of the system and we find that the basic reproductive number $\mathcal{R}_0$ controls completely the dynamics of the infection. This basic reproductive number should be regarded as a measure of the capacity of the cats to transmit toxoplasmosis. We proved that the basic reproduction number $\mathcal{R}_0$ is a threshold value that completely determines the global dynamics and the outcome of the disease. If the threshold parameter $R_0 < 1$, then the solution converges to the disease free equilibrium point. On the other hand if $R_0 > 1$ the convergence is to the endemic equilibrium point. Numerical simulations of the model illustrates several different dynamics depending on the threshold parameter R_0. Additionally, the importance of the vector vertical transmission to the dynamics of the infection is studied through numerical simulations.

It is important to remark that some prevention and control strategies against the Toxoplasmosis can be modeled using numerical simulations. In [19] a first approach for modeling the evolution of Toxoplasmosis disease in human population has been proposed, but considering that cat population is constant and has a uniform behavior with respect to the disease. However, in this work modeling of the evolution of toxoplasmosis in a human population take into account the cat as a vector of transmission is proposed. This model is more complex since two populations with interactions are included.

The organization of this paper is as follows. In the next section we formulate the mathematical model. Section 3 is devoted to analyze the steady states and find the threshold value $\mathcal{R}_0$ which determines the global dynamics and the outcome of the disease. Section 4 contains numerical results and finally in Section 5 we present the conclusions.

2. Mathematical model

In this section, we present a continuous mathematical model for the transmission and evolution Toxoplasmosis disease in human and cat populations. Following the basic ideas and structure of mathematical modeling in epidemiology, the Toxoplasmosis disease model will be developed under the next basic hypotheses [14,16].

(1) The total population of human $N_h(t)$ is divided in three subpopulations:
- Susceptible $S_h(t)$: members of the human population who may become infected.
- Infected $I_h(t)$: members of human population infected by the Toxoplasma Gondii parasite.
- Controlled $C_h(t)$: members of the population who have been treated for the Toxoplasmosis.

(2) The total population of cats $N_c(t)$ is divided in two subpopulations:
- Susceptible $S_c(t)$: members of the cats population who may become infected.
- Infected $I_c(t)$: members of cats population infected by the Toxoplasma Gondii parasite.

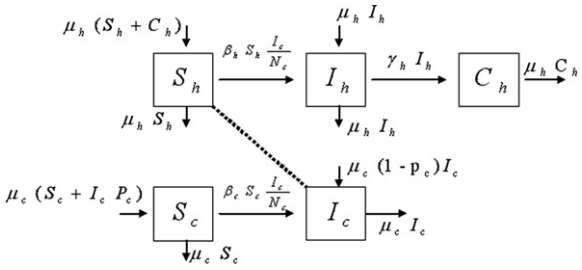

Fig. 1. Flow diagram of the Toxoplasmosis disease model for human and cat populations as defined in system (1).

(3) A susceptible human can be infected through a effective direct or indirect contact with an infected cat and transit to the infected subpopulation $I_h(t)$. An infected human transits to the controlled subpopulation $C(t)$ at a rate γ.
(4) A susceptible cat can be infected through a effective contact with an infected cat and transit to the infected subpopulation $I_c(t)$. A cat never recover from infection.
(5) Both human and cat birth rate are assumed equal to the their natural death rates, therefore constant population size is assumed.
(6) All members of the susceptible subpopulations $S_h(t)$ and $S_c(t)$ have the same probability to be infected.
(7) Vertical transmission is assumed in human population, but in cat population it is assumed that occurs with probability $[1 - p_c]$, where p_c is the probability that a susceptible cat born from a infected one.

The total population of humans is denoted by

$$N_h(t) = S_h(t) + I_h(t) + C_h(t)$$

and the total population of cats is denoted by

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

Under the above assumptions the dynamic Toxoplasmosis disease model for human and cat population is depicted graphically in Fig. 1 and is given analytically by the first order following nonlinear system of ordinary differential equation,

$$\begin{aligned}
\dot{S}_h(t) &= \mu_h C_h - \beta_h S_h \frac{I_c}{N_c}, \\
\dot{I}_h(t) &= \beta_h S_h \frac{I_c}{N_c} - \gamma I_h, \\
\dot{C}_h(t) &= \gamma I_h - \mu_h C_h, \\
\dot{S}_c(t) &= \mu_c I_c p_c - \beta_c S_c \frac{I_c}{N_c}, \\
\dot{I}_c(t) &= \beta_c S_c \frac{I_c}{N_c} - \mu_c I_c p_c.
\end{aligned} \tag{1}$$

3. Stability analysis of the model

In this section, the model (1) will be dynamically analyzed to investigate the existence and stability of its associated equilibria. This analysis allows us to study different scenarios about the spread of the toxoplasmosis disease in the human population caused by direct or indirect contact with infected cats.

3.1. Scaling model

Following the ideas developed in [20,21] in regard to scaling population models, it is defined the next ratios (depending of time),

$$X(t) = \frac{S_h(t)}{N_h(t)}, \qquad Y(t) = \frac{I_h(t)}{N_h(t)}, \qquad Z(t) = \frac{C_h(t)}{N_h(t)}, \qquad A(t) = \frac{S_c(t)}{N_c(t)}, \qquad B(t) = \frac{I_c(t)}{N_c(t)}. \tag{2}$$

Thus, using (1) and (2) one gets,

$$\begin{aligned}
\dot{X}(t) &= \mu_h Z(t) - \beta_h X(t) B(t),\\
\dot{Y}(t) &= \beta_h X(t) B(t) - \gamma Y(t),\\
\dot{Z}(t) &= \gamma Y(t) - \mu_h Z(t),\\
\dot{A}(t) &= \mu_c p_c B(t) - \beta_c A(t) B(t),\\
\dot{B}(t) &= \beta_c A(t) B(t) - \mu_c p_c B(t).
\end{aligned} \tag{3}$$

Since human and cat total populations have been normalized to unity, the following equations are obtained:

$$Z(t) = 1 - X(t) - Y(t) \tag{4}$$

and

$$B(t) = 1 - A(t). \tag{5}$$

Thus, from (3), and using (4) and (5) one gets the following simplified equivalent system

$$\begin{aligned}
\dot{X}(t) &= \mu_h (1 - X(t) - Y(t)) - \beta_h X(t)(1 - A(t)),\\
\dot{Y}(t) &= \beta_h X(t)(1 - A(t)) - \gamma Y(t),\\
\dot{A}(t) &= \mu_c p_c (1 - A(t)) - \beta_c A(t)(1 - A(t)).
\end{aligned} \tag{6}$$

For the sake of clarity and without loss of generality, analysis of equilibrium points are performed using the reduced system (6). Moreover, the dynamics of system (6) is restricted in the positive invariant subset $\Omega \subset \mathbb{R}^3_+$ defined by

$$\Omega = \left\{ (X, Y, A)^{\mathrm{T}} \in \mathbb{R}^3_+ / 0 \le X \le 1, 0 \le Y \le 1, 0 \le A \le 1 \right\}.$$

3.2. Equilibrium points of the model

We denote by (X^*, Y^*, A^*) the equilibrium points of system (6), i.e., the steady state where $\dot{X}(t) = 0, \dot{Y}(t) = 0, \dot{A}(t) = 0$, for all $t > t_0$. Therefore, from the last equation of system (6) one gets that

$$(1 - A^*)[\mu_c p_c - \beta_c A^*] = 0. \tag{7}$$

Therefore,

$$A_1^* = 1 \tag{8}$$

or

$$A_2^* = \frac{\mu_c p_c}{\beta_c}. \tag{9}$$

Now, if (8) holds, then from second equation of system (6) one can obtain that $Y_1^* = 0$, and from first equation of system (6) one gets that $X_1^* = 1$. Hence, it obtains the disease free point as $(X_1^*, Y_1^*, A_1^*) = (1, 0, 1)$.

On the other hand, if (9) holds, then from second equation of system (6) it follows that

$$Y_2^* = \frac{\beta_h X^* (1 - A_2^*)}{\gamma}, \tag{10}$$

and replacing in the first equation of system (6) it follows that

$$X_2^* = \frac{\mu_h \gamma}{\gamma \mu_h + \beta_h (1 - A_2^*)(\mu_h + \gamma)} \tag{11}$$

and that

$$Y_2^* = \frac{\mu_h \beta_h (1 - A_2^*)}{\gamma \mu_h + \beta_h \mu_h (1 - A_2^*) + \beta_h \gamma (1 - A_2^*)}. \tag{12}$$

Indeed, it has the endemic point of coordinates (X_2^*, Y_2^*, A_2^*).

3.3. Stability analysis

Computing the Jacobian of system (6) evaluated at (X^*, Y^*, A^*), one gets the following matrix

$$J(X^*, I^*, A^*) = \begin{pmatrix} -\mu_h - \beta_h(1-A^*) & -\mu_h & \beta_h X^* \\ \beta_h(1-A^*) & -\gamma & -\beta_h X^* \\ 0 & 0 & -\mu_c p_c - \beta_c + 2\beta_c A^* \end{pmatrix}.$$

Disease free point

In the absence of infection ($I_h = 0$) the model (6) has a disease free point $F_1^* = (X_1^*, Y_1^*, A_1^*)$ and evaluating the Jacobian $J(F_1^*)$ it follows that

$$J(F_1^*) = \begin{pmatrix} -\mu_h & -\mu_h & \beta_h \\ 0 & -\gamma & -\beta_h \\ 0 & 0 & \beta_c - \mu_c p_c \end{pmatrix}.$$

The stability of the equilibrium point F_1^* is determined using the eigenvalues of $J(F_1^*)$. The disease free equilibrium point F_1^* is locally asymptotically stable if the real part of eigenvalues are all negative. Thus, computing the eigenvalues of $J(F_1^*)$ one gets that all are negative if

$$\beta_c < \mu_c p_c. \tag{13}$$

Therefore, if we define

$$\mathcal{R}_0 = \frac{\beta_c}{\mu_c p_c} < 1, \tag{14}$$

the disease free point F_1^* is locally asymptotically stable for $\mathcal{R}_0 < 1$. Thus, we have established the following Lemma:

Lemma 1. *The disease free point F_1^* is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

In addition, it can be shown that the disease free point F_1^* is globally asymptotically stable for $\mathcal{R}_0 \leqslant 1$. The following theorem can be established,

Theorem 1. *If $\mathcal{R}_0 < 1$, then the disease free point F_1^* is globally asymptotically stable.*

Proof. In order to prove this, we consider the following *Lyapunov* function $V : \Omega \longrightarrow \mathbb{R}_+$, defined by

$$V(t) = V(X(t), Y(t), A(t)) = \frac{1 - A(t)}{\mu_c p_c}. \tag{15}$$

It is clear that $V \in C^1(\Omega)$ and $V(X(t), Y(t), A(t)) \geq 0$ for all $(X(t), Y(t), A(t))^{\mathrm{T}} \in \Omega$. Next, taking the derivative of (15) with respect to time along of the solutions of system (6) and using the third equation of (6), one gets that

$$\dot{V}(t) = -(1 - A(t)) - \mathcal{R}_0 A(t)(1 - A(t)) = -(1 - \mathcal{R}_0 A(t))(1 - A(t)). \tag{16}$$

Since that $1 - A(t) \geq 0$ and $1 - \mathcal{R}_0 A(t) > 0$, from (16) it follows that $\dot{V}(t) \leq 0$. Therefore, the Liapunov–Lasalle theorem guarantees the global stability of the disease free point (X_1^*, Y_1^*, A_1^*), if $\mathcal{R}_0 < 1$. □

Endemic point

In presence of infection ($I_h \neq 0$), from (9), (11) and (12) the model (6) has an endemic point $E_2^* = (X_2^*, Y_2^*, A_2^*)$. Evaluating the Jacobian $J(E_2^*)$ one gets that

$$J(E_2^*) = \begin{pmatrix} L & -\mu_h & \dfrac{\beta_h \gamma}{H} \\ -\mu h - L & -\gamma & -\dfrac{\beta_h \gamma}{H} \\ 0 & 0 & M, \end{pmatrix},$$

where

$$\begin{aligned} H &= \gamma\mu_h + (\mu_h + \gamma)\beta_h \left(1 - \frac{1}{\mathcal{R}_0}\right), \\ K &= \mu_h \beta_h \left(1 - \frac{1}{\mathcal{R}_0}\right), \\ L &= -\mu_h - \beta_h \left(1 - \frac{1}{\mathcal{R}_0}\right), \\ M &= -\mu_c p_c - \beta_c + \frac{2\beta_c}{\mathcal{R}_0}. \end{aligned} \tag{17}$$

Table 1
Parameters of the model

Parameter	Values
μ_h	0.233
β_h	0.0206
μ_c	0.066
p_c	0.01
γ	0.000232

The eigenvalues of $J(E_2^*)$ are calculated using $\text{Det}(J(E_2^*) - \lambda I_3) = 0$, i.e.,

$$\text{Det}\begin{pmatrix} L-\lambda & -\mu_h & \dfrac{\beta_h\gamma}{H} \\ \mu h - L & -\gamma-\lambda & -\dfrac{\beta_h\gamma}{H} \\ 0 & 0 & M-\lambda \end{pmatrix} = 0.$$

It follows that an eigenvalue is $\lambda_1 = M$ and the other two eigenvalues are λ_2, λ_3 the roots of the polynomial

$$\lambda^2 - \lambda(L-\gamma) + \mu_h(\mu_h - L) - L\gamma = 0. \tag{18}$$

Now, the Eq. (18) has negative solutions if $L - \gamma < 0$ and $\mu_h(\mu_h - L) - L\gamma > 0$. Indeed

$$-\gamma + L = -\gamma - \mu_h - \beta_h\left(1 - \frac{1}{\mathcal{R}_0}\right) < 0,$$

since $\mathcal{R}_0 > 1$, and

$$\mu_h\left[\mu_h + \mu_h + \beta_h\left(1 - \frac{1}{\mathcal{R}_0}\right)\right] + \left[\mu + \beta_h\left(1 - \frac{1}{\mathcal{R}_0}\right)\right]\gamma > 0.$$

Therefore, if $\mathcal{R}_0 = \frac{\beta_c}{\mu_c p_c} > 1$, then the endemic point is locally asymptotically stable. The following *Lemma* has been established.

Lemma 2. *The endemic point E_2^* is locally asymptotically stable if $\mathcal{R}_0 > 1$ and unstable if $\mathcal{R}_0 < 1$.*

Based on the previous analysis we can resume and say that there are two realistic equilibrium points: One is the disease free point and the other is the unique endemic equilibrium. $\mathcal{R}_0$ is a unique threshold parameter which determines the behavior of the Toxoplasmosis spread model. Assuming that the stability result for the endemic equilibrium is also global and assuming that initially there is at least one infectious cat, then if $\mathcal{R}_0 < 1$, we expect the disease to die out, whereas if $\mathcal{R}_0 > 1$, then we expect the Toxoplasmosis disease to tend to the unique endemic equilibrium, thereby establishing itself in the region.

4. Numerical simulations

In this section, we simulate different possible scenarios in order to observe the effect that some relevant parameters has over the dynamics of the toxoplasmosis disease in human and cat populations. This is important from an epidemiological point of view, since it is possible to obtain the best strategies to tackle the disease. The first two scenarios are computed to check dynamic consistency between the theoretical results obtained in the previous section and the numerical simulations of the model. One scenario is the disease free with $\mathcal{R}_0 < 1$ and other is the endemic with $\mathcal{R}_0 > 1$.

Another scenario is simulated varying the cats vertical transmission parameter p_c. This numerical simulation allows to observe the effect of this parameter on the transmission dynamics of the toxoplasmosis disease in human and cat populations.

In order to perform the numerical simulations we take into account that in Chile approximately 55% of the cat population have antibodies against the Toxoplasma Gondii and that in Colombia approximately 47, 74% of the population have antibodies against the Toxoplasma and only the 1% is controlled [19,22]. Parameter values of human birth rate μ_h, β_h and transition to controlled population rate γ were taken from [19]. The cats birth rate μ_c was obtained by sources specialists in zootechnic [23,24]. Thus, for numerical simulations it is assumed as initial condition in the simulated scenarios the following values,

$$X(0) = 0.5253 \qquad Y(0) = 0.47 \qquad \text{and} \qquad A_c(0) = 0.45. \tag{19}$$

The time invariant parameters of the model (6) are showed in Table 1.

Disease free point

Here, it is assumed a value for the parameter β_c such that $R_0 < 1$. As it can be observed in Fig. 2 and as expected from the theoretical results of previous section, the system approach to the disease free equilibrium point $(X_1^*, Y_1^*, A_1^*) = (1, 0, 1)$.

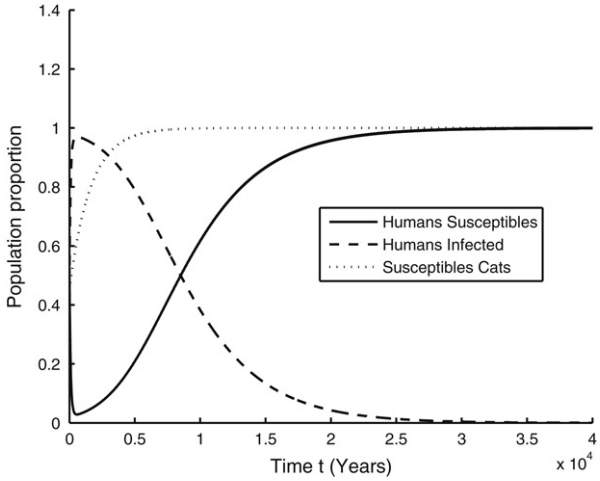

Fig. 2. Dynamics of the different subpopulations when $\beta_c = 0.00066$, $\gamma = 0.000232$ and $\mathcal{R}_0 = 0.909$. ($X_1^* = 1$, $Y_1^* = 0$ and $A_1^* = 1$).

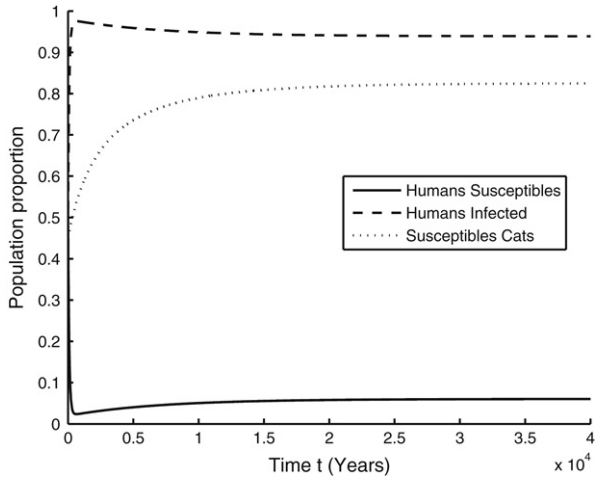

Fig. 3. Dynamics of the different subpopulations when $\beta_c = 0.0008$, $\gamma = 0.000232$ and $\mathcal{R}_0 = 1.2121$. ($X^* = 0.0603$, $Y^* = 0.9386$ and $A^* = 0.825$).

Endemic point

Here, it is assumed a value for the parameter β_c such that $R_0 > 1$. As it can be observed in Fig. 3 and as expected from the theoretical results of previous section, the system approach to the endemic equilibrium point ($X^* = 0.0602$, $Y^* = 0.9388$ and $A^* = 0.8246$).

High vertical transmission in cat population

Here, it is assumed a value for the parameter $\beta_c = 0.0008$ and $p_c = 0.0001$. As it can be observed in Fig. 4, the system approach to the endemic equilibrium point ($X^* = 0.0112$, $Y^* = 0.9878$ and $A^* = 0.00825$). Notice that this equilibrium point has more infected cats and infected humans at the steady state than the previous case. This fact is expected from a logical point of view, since high vertical transmission implies easily transmission from one generation of cats to other. Furthermore, since there are more infected cats, transmission to humans is also enhanced.

5. Discussion and conclusions

In this paper one proposes a mathematical model to study the dynamics of Toxoplasmosis disease in the human and cat populations. The model consists on modeling the interactions among susceptible and infective individuals of the two species assuming that the horizontal transmission of the disease to humans is only throughout the contact with infected cats. Human population is divided in three classes or subpopulations, susceptibles $S_h(t)$, infectious $I_h(t)$ and controlled $C_h(t)$, and the cat population into two classes or subpopulations, susceptibles $S_c(t)$ and infectious $I_c(t)$. We assume that both populations of cats and human are constant. Vertical and horizontal transmission in the cat population was considered. Transmission is assumed in the human population through an effective direct or indirect contact with an infected cat and

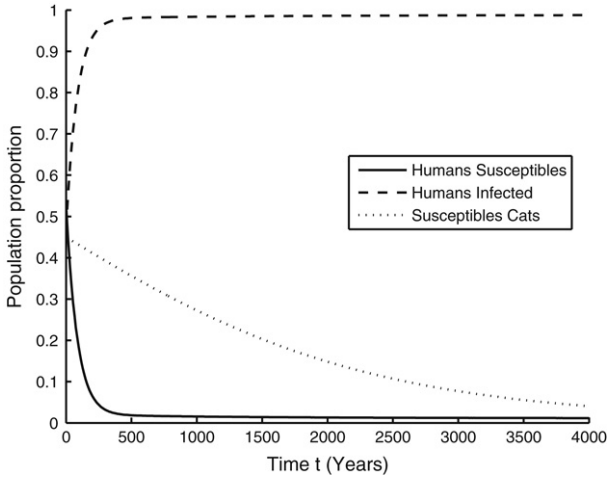

Fig. 4. Dynamics of the different subpopulations when $\beta_c = 0.0008$, $\gamma = 0.000232$, $p_c = 0.0001$ and $\mathcal{R}_0 = 121.21$. ($X^* = 0.0112$, $Y^* = 0.9878$ and $A^* = 0.00825$).

vertical transmission is assumed with probability 1. However, vertical transmission in cat population it is assumed that occurs with probability $[1 - p_c]$.

We proved that the basic reproduction number $\mathcal{R}_0$ is a threshold value that completely determines the global dynamics and the outcome of the disease. If the threshold parameter $R_0 < 1$, then the solution converges to the disease free equilibrium point. On the other hand if $R_0 > 1$ the convergence is to the endemic equilibrium point. Additionally, the importance of cats vertical transmission to the dynamics of the infection is studied through numerical simulations. When a high vertical transmission is assumed in cat population, the endemic equilibrium point has a higher proportion of infected cats and infected humans at the steady state. This fact is expected from an intuitive point of view, since high vertical transmission implies easily transmission from one generation of cats to other. Furthermore, since there are more infected cats, transmission to humans is also enhanced. Therefore, the dynamics of toxoplasmosis disease is strongly influenced by vertical transmission in cat population. The numerical simulations show that this transmission favors the establishment of a constant endemic level in both populations. Also, they show an important increase in the endemic level of the cat and human population. This last result reinforces the idea that vertical transmission can be an important mechanism that favors the maintenance of the virus areas with low human densities.

It is important to remark that the threshold number $\mathcal{R}_0$ is directly proportional to the probability of effective infectious contact among cats and does not depends on direct or indirect effective infectious contacts between humans and cats. Therefore, a control strategy to reduce toxoplasmosis prevalence, should focus on reducing this probability. One way of control is a vaccine program for cats population. However, prevalence in human population may be reduced, but not eradicated with hygienic actions and educations programs. In addition, probability of vertical transmission in cat population is an intrinsic value that seems invariable to control strategies. Therefore, this mechanism of transmission may be responsible for the permanence of the toxoplasmosis infection as has been suggested in [25] for dengue virus.

Finally, it is important to mention that our mathematical model considers the evolution of Toxoplasmosis in the human and cat populations with interaction among them, but it is necessary to have further knowledge of the parameters values involved in order to have more accurate estimates about future health scenarios in the human and cat populations. Moreover, the parameters values of the model vary depending on the environment, therefore these parameters should be adjusted for different cities or regions for different real world applications. Future work should include a model with vaccination and variable population size, and more variables such seasonal birth rate or oocyst survival time.

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