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Research article

A stochastic model incorporating an implicit delay effect for toxoplasmosis: Evaluation of intervention policies for public health

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Abstract: According to the World Health Organization (WHO), toxoplasmosis affects more than 60% of the global population. The prevalence of this infection is particularly high in hot, humid, and low-altitude regions, as such environments favor the survival of oocysts in the ecosystem. In this study, we investigated the transmission dynamics of toxoplasmosis using a stochastic model with an implicit delay effect approach. The host populations were divided into compartments representing susceptible cats $S(t)$, infected cats $I_c(t)$, recovered cats $V_R(t)$, susceptible mice $S_m(t)$, infected mice $I_m(t)$, and the number of oocysts in the environment $O(t)$. In the delayed deterministic model, fundamental mathematical properties such as positivity, boundedness, existence, and uniqueness of solutions were established. Furthermore, the local and global stability of the steady states were analyzed using second-order stability conditions. In the stochastic delayed formulation, we investigated the positivity, boundedness, extinction, and persistence of the infection under random environmental fluctuations. To address the nonlinear complexity of the proposed system, several computational methods were employed, including the Euler–Maruyama, stochastic Euler, stochastic Runge–Kutta, and the stochastic non-standard finite difference (SNSFD) schemes. A comparative numerical analysis demonstrated that the SNSFD scheme preserves the qualitative features of the continuous model and remains stable under large time steps, confirming its suitability for modeling biologically realistic epidemic dynamics.

Keywords: toxoplasmosis; stochastic delayed model; existence and uniqueness; basic reproduction number; vaccination strategy; numerical simulation; stochastic non-standard finite difference; computational methods
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1. Introduction

Toxoplasmosis is a biting infection caused by *Toxoplasma gondii*, named after the rounded figure of its infectious phase. People often get infected from eating undercooked meat, ingesting unfiltered water and raw seafood, and coming into contact with cat feces; additionally, these parasites can pass to a baby during pregnancy. Often, individuals with toxoplasmosis do not have any sort of symptom of the disease; others have flu-like symptoms, including fever, muscle aches, headaches, body aches, fatigue, and tender lymph nodes. People with a weakened immune system are much more prone to disease.

T. gondii infection occurs all over the world, but the infection rate is country-specific. In 1908, Nicolle and Manceaux in Tunisia and Splendore in Brazil described *Toxoplasma gondii* for the first time. In 1923, the first case of toxoplasmosis was recorded. In 1937, Sabin and Olitsky examined *T. gondii* in research laboratory mice and monkeys and established it as a pathogen transmissible between animals [1]. Toxoplasmosis is more common in women than in men. At the international level, around six billion individuals are infected with *T. gondii*. Its seropositivity rate is much lower in developed than in developing countries. The seroprevalence of toxoplasmosis varies with age groups within an area and also in diverse topographical sections within a country. Limited studies from Pakistan have stated that toxoplasmosis prevalence was 11.33%–29.45% [2]. As cats are the transmission vector of toxoplasmosis, in areas where they are not present, the prevalence of toxoplasmosis is zero [3]. More than 200,000 humans are infected with toxoplasmosis. Infection during pregnancy mostly results in miscarriage, stillbirth, or abnormal birth.

In 2008, Aranda proposed a mathematical model to study the dynamics of toxoplasmosis infection in Colombia. By using a linear system of ODEs, the initial system was formulated and converted to obtain comparative values and to characterize the qualitative behavior of the system. The modified system is a nonlinear system of ordinary differential equations [3]. In 2012, Sullivan introduced a mathematical model to examine the intra-host dynamics of *T. gondii*, including incursion, reproduction, and stage conversion [4]. In 2018, Peng et al. studied a dynamics of a model of toxoplasmosis disease in cats and humans with varying population sizes [5]. In 2017, Ferreira et al. studied a stability and bifurcation in epidemic models describing the transmission of toxoplasmosis in human and cat populations [6]. In 2024, Raza et al. studied well-established techniques to investigate disease modeling with delay strategies and demonstrated how such delay mechanisms can effectively control disease dynamics within a population [7]. In 2021, Zafar et al. proposed an epidemiological model to examine the dynamics of random-order toxoplasmosis infection in the hominid and catlike populations with the support of non-integer Multistep Generalized Differential Transform method (MSGDTM) [8]. In 2021, a non-integer and nonlinear mathematical model was described by Zafar for toxoplasmosis infection in human and cat populations. The special effects of toxoplasmosis infection were evaluated on humans by taking cats as a diffusion trajectory [9]. In 2019, Raza et al. proposed a mathematical framework to examine vector-borne disease transmission in a population and implemented several numerical methods to solve the resulting complex

stochastic system, evaluating the efficiency and reliability of the proposed approaches [10]. In 2018, Kelting et al. proposed a mathematical model for the effects of *T. gondii* on the cat population in order to understand its dynamics and develop preventative measures against this parasite [11]. Effects of leucocytes, antibiotics, and immunologic adjuvants against *T. gondii* were studied by Liu et al. in 2012, by proposing three models. In leucocytes, toxoplasmosis depends on the immune strength of the host. Antibiotics seem to have a significant impact on toxoplasmosis infection, and the immune strength of the host is improved by ingestion of an immunological adjuvant that ends up killing *T. gondii* [12].

Toxoplasmosis spread in the Netherlands and in the rest of Europe directly by infected cats and raw meat. In 2020, Marinović et al. suggested a system of cat immunization for dipping oocytes that originated from *T. gondii* human contagions; it is still unclear whether this system is effective [13]. In 2022, Parra et al. projected an epidemiological system to study toxoplasmosis infection with numerous congregations. They included mouse populations as an intermediary host and showed that the basic reproduction number R_0 governs the outcome of the infection [14]. In 2016, Li et al. developed a mathematical system to stimulate toxoplasmosis spread between cats and oocyte populations. They studied asymptotic behaviors around the equilibrium by using stochastic Lyapunov functions [15].

Here, a stochastic model with an implicit delay effect model is established for the study of toxoplasmosis transmission between cats, mice, and environmental oocyst populations. Mathematical results on positivity, boundedness, threshold behavior, and stability of steady states are obtained for the deterministic and stochastic models. In addition, a stochastic non-standard finite difference method is designed and computationally tested, outperforming the classical stochastic schemes in terms of stability and qualitative behavior. Furthermore, stochastic modeling employs efficient computational methods to identify essential epidemiological risk factors and guide public health innovations. Stochastic techniques offer a unique perspective on the complex dynamics of toxoplasmosis, resulting in enhanced recovery strategies and informed decision-making. The stochastic terms capture environmental randomness and uncertainties in transmission events associated with the variability in survival times of oocysts and host–environment interactions. This could not be represented by the OSE model.

This paper is organized as follows: Section II presents the formulation of the stochastic model and the basic deterministic model attributes. Sections II and IV present the model's stability analysis at both local and global levels. Section V describes the sensitivity of the model equilibria. Sections VI and VII describe the stochastic methods developed for the stochastic model. Section VIII presents asymptotic behavior of the model. Section IX presents an investigation of the stochastic NSFD scheme. Finally, a conclusion is provided.

1.1. Basic notations

Definition 1. A deterministic system is one in which the evolution of the state variables is completely determined by a set of differential equations without random perturbations.
Formally, a deterministic system is expressed as

$$\frac{dX(t)}{dt} = F(X(t), t),$$

where $X(t) \in \mathbb{R}^n$ is the state vector and $F: \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n$ is a continuously differentiable vector function.

Given an initial condition $X(0) = X_0$, the system's trajectory is uniquely defined for all $t > 0$.

Definition 2. A stochastic differential equation (SDE) introduces randomness into the system dynamics through a stochastic term, usually modeled by Brownian motion.

It is generally represented as

$$dX(t) = f(X(t), t)\, dt + g(X(t), t)\, dB(t),$$

where:

- $f(X(t), t)$ is the drift term, representing the deterministic trend of the process.
- $g(X(t), t)$ is the diffusion term, representing the stochastic fluctuations.
- $B(t)$ denotes standard Brownian motion, satisfying $E[dB(t)] = 0$ and $E[dB(t)^2] = dt$.

Definition 3. A stochastic model with an implicit delay effect is a stochastic differential equation in which memory or latency effects are incorporated through survival or weighting functions, rather than explicit delayed state variables.

In particular, the delay effect is modeled via a survival factor of the form

$$e^{-\mu\tau},$$

which represents the probability that individuals or pathogens survive a latent or maturation period of length τ under a constant mortality or decay rate μ. Such formulations capture biologically realistic incubation or environmental survival processes without introducing explicit delay terms.

Definition 4. An equilibrium point (or steady state) of a dynamical system is a constant solution X^* such that

$$F(X^*, t) = 0 \text{ or equivalently, } \frac{dX}{dt} = 0.$$

For epidemic models, the equilibria often correspond to:

- Disease-free equilibrium (DFE): No infection persists, $I^* = 0$.
- Endemic equilibrium (EE): Infection persists at a constant positive level, $I^* > 0$.

Definition 5. A solution $X(t)$ of a biological model is said to be positive if

$$X_i(t) \geq 0, \forall i = 1,2, \dots, n, \forall t \geq 0.$$

Positivity ensures that population variables (e.g., susceptible or infected classes) remain biologically meaningful.

Definition 6. A solution $X(t)$ is said to be bounded if there exists a constant $M > 0$ such that

$$\| X(t) \| \leq M, \forall t \geq 0.$$

Boundedness implies that the populations cannot grow without limit, preserving biological realism.

Definition 7. The basic reproduction number, R_0, is defined as the expected number of secondary infections produced by a single infectious individual in a fully susceptible population. For a model linearized around the disease-free equilibrium, it can be expressed using the next-generation matrix approach,

$$R_0 = \rho(FG^{-1}),$$

where F is the transmission matrix, G is the transition matrix, and $\rho(\cdot)$ denotes the spectral radius (dominant eigenvalue).

- If $R_0 < 1$: The disease dies out.
- If $R_0 > 1$: The disease persists.

Definition 8. An equilibrium point X^* of the system $\frac{dX}{dt} = F(X)$ is said to be locally asymptotically stable (L.A.S.) if for all initial conditions $X(0)$ sufficiently close to X^*, we have

$$\lim_{t\to\infty} X(t) = X^*.$$

This property indicates that small perturbations around X^* decay over time.

Definition 9. An equilibrium X^* is said to be globally asymptotically stable (G.A.S.) if it is both:

1. Stable in the Lyapunov sense: Solutions remain near X^* when initial perturbations are small, and
2. Attractive: $\lim_{t\to\infty} X(t) = X^*$ for all initial conditions $X(0) \in \mathbb{R}^n_+$.

Definition 10. A Lyapunov function $V(X)$ is a continuously differentiable, positive-definite function that satisfies

$$V(X) > 0 \text{ for } X \neq X^*, \text{ and } \frac{dV}{dt} \leq 0.$$

If such a function exists, the equilibrium X^* is stable. In stochastic systems, Itô's lemma is used to extend this concept via stochastic Lyapunov functions.

Definition 11. For a stochastic process $X(t)$ satisfying $dX(t) = f(X,t)\,dt + g(X,t)\,dB(t)$, and a twice-differentiable function $V(X,t)$, Itô's lemma gives

$$dV(X,t) = V_t\,dt + V_X\,dX + \frac{1}{2}V_{XX}\,(dX)^2.$$

This formula is fundamental in deriving the stochastic differential of Lyapunov functions and in stability analysis of stochastic systems.

Definition 12. The stochastic system $dX(t) = f(X,t)\,dt + g(X,t)\,dB(t)$ is mean-square stable if

$$\lim_{t\to\infty} E[\| X(t) - X^* \|^2] = 0.$$

This ensures that, on average, the system tends to equilibrium despite random perturbations.

2. Model formulation

This section provides a survival-delay model to study toxoplasmosis transmission in cat and mouse populations. This concept is built around a consistent cat vaccination strategy. Although the system does not explicitly include delayed state variables (e.g., $S(t-\tau)$ or $O(t-\tau)$), a delay effect is incorporated implicitly through the survival factor $e^{-\mu\tau}$, which represents the probability of surviving the latent/incubation interval of length τ under an exponential mortality rate. The model contains oocytes, which are the cause of *T. gondii* in the environment. The cat population, N(t), is split into three distinct subpopulations: Susceptible S(t), infected I(t), and vaccine-recovered V_R(t). The mouse population, N_m(t), is also split into two different subpopulations: susceptible S_m(t) and infected $I_m(t)$. O(t) is the number of oocytes in the environment. Affective contact with oocytes at rate β and β_m causes a susceptible cat or mouse, respectively, to join the infected subpopulation. The parameter γ represents the rate of transmission from a susceptible cat into the vaccinated subpopulation V_R(t). Similarly, the parameter α represents the transmission of an infected cat into the vaccinated/recovered subpopulation V_R(t). Oocytes and infected cats are directly proportional to each other. The increase of oocytes at any time t is proportional to the number of infected cats I_t. μ_o is the death rate of oocytes. μ is the death/birth rate of cats (See Table 1).

Table 1. Description of model parameters and variables used in the toxoplasmosis model with a saturated incidence rate.

Symbol	Description
$\boldsymbol{\mu}$	Natural birth/death rate of cats (day^{-1})
$\boldsymbol{\alpha}$	Removal/recovery rate of infected cats (day^{-1}) (so 1/α is the mean infectious period)
$\boldsymbol{\mu_o}$	Decay/removal rate of oocysts in the environment (day^{-1})
$\boldsymbol{k}$	Oocyst production rate per infected cat (oocysts·cat^{-1}·day^{-1})
$\boldsymbol{\beta}$	Transmission coefficient from environmental oocysts to cats (units consistent with incidence term)
$\boldsymbol{\beta_m}$	Transmission coefficient from environmental oocysts to mice (units consistent with incidence term)
$\boldsymbol{\gamma}$	Vaccination/acquired-immunity rate removing susceptible cats from S-class (day^{-1})
$\boldsymbol{b}$	Birth rate of mice (day^{-1})
$\boldsymbol{\mu_m}$	Natural death rate of mice (day^{-1})

Here, τ (days) denotes the oocyst maturation period before oocysts become infective. The factor $e^{-\mu_0\tau}$ represents the probability that oocysts survive this period under the environmental decay rate μ_0. Empirically, oocysts typically sporulate and become infective within 0.1–0.5 days, hence we consider $\tau \in [0.1,0.5]$ days in the numerical investigations.

The delayed differential equations of the toxoplasmosis epidemic model, as nonlinear, delayed first-order, and coupled, are as follows:

$$S'(t) = \mu\big(1 - I(t)\big) - \beta S(t)O(t)e^{-\mu_o\tau} - (\mu + \gamma)S(t), \qquad t \geq 0 \;\; \tau \leq t, \tag{1}$$

$$I'(t) = \beta S(t)O(t)e^{-\mu_o\tau} - \alpha I(t), \qquad t \geq 0 \;\; \tau \leq t, \tag{2}$$

$$O'(t) = kI(t) - \mu_o O(t), \qquad t \geq 0, \tag{3}$$

$$S'_m(t) = bS_m(t) - \beta_m S_m(t)O(t) - \mu_m S_m(t), \qquad t \geq 0, \tag{4}$$

Here, $S(0) \geq 0,\ I(0) \geq 0,\ O(0) \geq 0,\ S_m(t) \geq 0$. The exponential factor $e^{-\mu_o\tau}$ represents the probability that oocysts survive the maturation period τ required to become infective. Thus, the incidence term $\beta S(t)O(t)e^{-\mu_o\tau}$ does not imply instantaneous infection but rather accounts for the effective transmission arising from oocysts that remain viable after environmental decay during the latent period.

2.1. Dynamical properties

To investigate the dynamics of toxoplasmosis transmission in cat and mouse populations, all system parameters in Eqs (1)–(4) are assumed to be nonnegative, i.e., greater than or equal to zero. For the epidemiological model to be meaningful, the state variables must also remain nonnegative for all $t \geq 0$ and $\tau \leq t$. Consequently, the model's feasible region is defined as positive and bounded within a biologically relevant domain.

$$\Omega = \{(S, I, O, S_m) \in \mathbb{R}^4_+ : N_c(t) \leq \tfrac{\mu}{\mu+\gamma},\ N_m(t) \leq \tfrac{b}{\mu_m},\ S \geq 0, I \geq 0, O \geq 0, S_m \geq 0\}.$$

Theorem 1. Positivity of solutions: For any initial conditions $(S(0), I(0), O(0), S_m(0)) \in \mathbb{R}^4_+$, the corresponding solutions

$$(S(t), I(t), O(t), S_m(t)) \in \mathbb{R}_+^4$$

of the system (1)–(4) remain positive for all $t \geq 0$.

Proof. Let us define the norm

$$\lambda_\infty = \sup_{t \in D_\lambda} | \lambda(t) |.$$

For the susceptible population $S(t)$, we have

$$\frac{dS}{dt} = \mu(1 - I_\infty) - \beta S O_\infty e^{-\mu_o \tau} - (\mu + \gamma)S.$$

Thus,

$$\frac{dS}{dt} \geq -\beta S O_\infty e^{-\mu_o \tau} - (\mu + \gamma)S.$$

Dividing both sides by S and integrating over time gives

$$\frac{dS}{S} \geq -[\beta O_\infty e^{-\mu_o \tau} + (\mu + \gamma)]dt,$$

which yields

$$S(t) \geq S(0)\, e^{-[\beta O_\infty e^{-\mu_o \tau} + (\mu+\gamma)]t} \geq 0.$$

Hence, $S(t) \geq 0$ for all $t \geq 0$.

Similarly, for the other compartments,

$$I(t) \geq I(0)e^{-\alpha t} \geq 0, O(t) \geq O(0)e^{-\mu_o t} \geq 0, S_m(t) \geq S_m(0)e^{-[\beta_m O_\infty + \mu_m]t} \geq 0.$$

Therefore, all state variables remain nonnegative for all $t \geq 0$**.**

Theorem 2. Boundedness of solutions: The solutions $(S, I, O, S_m) \in \mathbb{R}_+^4$ of the system (1)–(4) are bounded for all $t \geq 0$**.**

Proof. Consider the total population function

$$N(t) = S(t) + I(t) + O(t).$$

Differentiating with respect to t,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dO}{dt}.$$

Using the system equations, we obtain the inequality

$$\frac{dN}{dt} \leq -\mu(S + I + O) + \mu,$$

or equivalently,

$$\frac{dN}{dt} \leq -\mu N + \mu.$$

This can be rewritten as

$$\frac{dN}{dt} + \mu N \leq \mu.$$

Applying Gronwall's inequality, we find

$$N(t) \leq N(0)e^{-\mu t} + 1.$$

Therefore,

$$\lim_{t\to\infty} \sup N(t) \leq 1.$$

Hence, the total population remains bounded as $t \to \infty$.

2.2. Model equilibria

In this section, we determine two distinct equilibrium points of the system:

- The toxoplasmosis-free equilibrium (TFE, T_0), and
- the toxoplasmosis-endemic equilibrium (TEE, T^*).

These states are gained by having the right-hand sides of system (1)–(4) set to zero and solving for the steady-state values.

Toxoplasmosis-free equilibrium (TFE). At the disease-free state $(I = 0, O = 0)$, the system reduces to the steady solution:

$$T_0 = (S_0, I_0, O_0, S_{m0}) = (\frac{\mu}{\mu+\gamma}, 0, 0, \frac{b}{\mu_m}).$$

This represents a healthy population with no infection among cats or mice.

Toxoplasmosis-endemic equilibrium (TEE). At the endemic steady state $(I^*, O^* > 0)$, the system admits the equilibrium

$$T^* = (S^*, I^*, O^*, S_m^*) = (\frac{\alpha\mu_o}{\beta k e^{-\mu\tau}}, \frac{\mu\beta k e^{-\mu_o\tau} - (\gamma+\mu)\alpha\mu_o}{\beta k e^{-\mu_o\tau}(\mu+\alpha)}, \frac{\mu\beta k e^{-\mu_o\tau} - (\gamma+\mu)\alpha\mu_o}{\mu_o k e^{-\mu_o\tau}(\mu+\alpha)}, 0).$$

The endemic equilibrium exists only when $R_0 > 1$, ensuring that infection persists in the population.

Basic reproduction number (R_0). The basic reproduction number R_0 quantifies the expected number of secondary infections generated by a single infectious cat introduced into a completely susceptible population. It is derived using the next-generation matrix (NGM) method.

Let F denote the new infection matrix and G the transition (removal) matrix. Considering the infectious classes I and O (and excluding S' and S'_m from the Jacobian), we have

$$F = \begin{bmatrix} 0 & \beta S e^{-\mu_o\tau} \\ 0 & 0 \end{bmatrix}, G = \begin{bmatrix} \alpha & 0 \\ -k & \mu_o \end{bmatrix}.$$

Then,

$$FG^{-1} = \begin{bmatrix} 0 & \beta S e^{-\mu_o\tau} \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\alpha} & 0 \\ \frac{k}{\alpha\mu_o} & \frac{1}{\mu_o} \end{bmatrix}.$$

The dominant eigenvalue (spectral radius) of FG^{-1} gives the basic reproduction number

$$R_0 = \frac{k\mu\beta e^{-\mu_o\tau}}{\alpha\mu_o(\gamma+\mu)}.$$

If $R_0 < 1$, the infection will eventually disappear, while if $R_0 > 1$, the infection will persist in the

host populations.

3. Local stability analysis

We shall prove the following well-known conclusions for local stability in both balances of the model. Consider the function as follows: The elements of the Jacobian matrix are

$$J = \begin{bmatrix} -\beta O e^{-\mu_o \tau} - (\gamma + \mu) & -\mu & -\beta S e^{-\mu_o \tau} & 0 \\ \beta O e^{-\mu_o \tau} & -\alpha & \beta S e^{-\mu_o \tau} & 0 \\ 0 & k & -\mu_\circ & 0 \\ 0 & 0 & -\beta_m S_m & b - \beta_m O - \mu_m \end{bmatrix}. \tag{5}$$

Theorem 3. The toxoplasmosis-free equilibrium (TFE-T_0), $T_0 = (S_0, I_0, O_0, S_{m_0}) = \left(\frac{\mu}{\mu+\gamma}, 0,0, \frac{b}{\mu_m}\right)$ is locally asymptotical stable (LAS) if $R_0 < 1$. However, if $R_0 > 1$, the system will be unstable at T_0.

Proof. For stability at $T_0 = (S_0, I_0, O_0, S_{m_0}) = \left(\frac{\mu}{\mu+\gamma}, 0,0, \frac{b}{\mu_m}\right)$, the Jacobian matrix (5) becomes:

$$J(T_0) = \begin{bmatrix} -(\gamma + \mu) & -\mu & -\beta S_0 e^{-\mu_o \tau} & 0 \\ 0 & -\alpha & \beta S_0 \ e^{-\mu_o \tau} & 0 \\ 0 & k & -\mu_\circ & 0 \\ 0 & 0 & -\beta_m S_{m_0} & b - S_{m_0} O - \mu_m \end{bmatrix}.$$

$$|J(T_0) - \lambda| = \begin{vmatrix} -(\gamma + \mu) - \lambda & -\mu & -\beta \left(\frac{\mu}{\mu+\gamma}\right) e^{-\mu_o \tau} & 0 \\ 0 & -\alpha - \lambda & \beta \left(\frac{\mu}{\mu+\gamma}\right) e^{-\mu_o \tau} & 0 \\ 0 & k & -\mu_\circ - \lambda & 0 \\ 0 & 0 & -\beta_m \left(\frac{b}{\mu_m}\right) & b - \mu_m - \lambda \end{vmatrix}.$$

Here, the eigenvalues of $J(T_0)$ are as follows: $\lambda_1 = -(\gamma + \mu) < 0$, $\lambda_2 = b - \mu_m$. Then, $\lambda_2 < 0$ if $b < \mu_m$.

$$\lambda^2 + a_1 \lambda + a_0 = 0.$$

Then, $a_1 = \alpha + \mu$, $a_0 = (\alpha \mu_o)(1 - R_0)$. So, $a_1, a_0 > 0$.

Thus, by the Routh–Hurwitz criterion polynomial, the values of a_1 and a_0 are positive if $R_0 < 1$. Therefore, the toxoplasmosis-free equilibria (TFE-T_0) of the system (1)–(4) is locally stable. On the other hand, if $R_0 > 1$, Routh–Hurwitz's condition of stability is violated. Thus, T_0 is unstable locally.

Theorem 4. The toxoplasmosis endemic equilibrium (TEE-T^*), $T^* = (S^*, I^*, O^*, S^*{}_m)$ is locally asymptotical stable (LAS) if $R_0 > 1$.

Proof. Let us consider a Jacobian matrix at T^*, we have

$$J(T^*) = \begin{bmatrix} -\beta O^* e^{-\mu_o \tau} - (\gamma + \mu) & -\mu & -\beta S^* e^{-\mu_o \tau} & 0 \\ \beta O^* e^{-\mu_o \tau} & -\alpha & \beta S^* e^{-\mu_o \tau} & 0 \\ 0 & k & -\mu_\circ & 0 \\ 0 & 0 & -\beta_m S^* m & b - \beta_m O^* - \mu_m \end{bmatrix}.$$

For the eigenvalue, consider $|J(T^*) - \lambda I| = 0$,

$$\lambda_1 = b - \beta_m O^* - \mu_m < 0.$$

$$\lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_o = 0.$$

$$a_2 = \alpha + \mu_\circ + \beta O^* e^{-\mu_o \tau} + (\gamma + \mu) > 0.$$

$$a_1 = \alpha\mu_\circ + (\alpha + \mu_\circ)\left(\beta O^* e^{-\mu_o \tau} + (\gamma + \mu)\right) - \left((k\beta S^* e^{-\mu_o \tau}) + (\mu\beta O^* e^{-\mu_o \tau})\right),$$

where $a_1 > 0$ if $\frac{\alpha\mu_\circ + (\alpha+\mu_\circ)\left(\beta O^* e^{-\mu_o \tau} + (\gamma+\mu)\right)}{\left((k\beta S^* e^{-\mu_o \tau}) + (\mu\beta O^* e^{-\mu_o \tau})\right)} > 1$.

$$a_o = (\alpha\mu_\circ)\left(\beta O^* e^{-\mu_o \tau} + (\gamma + \mu)\right) - \left[\left(\beta O^* e^{-\mu_o \tau} + (\gamma + \mu)\right)\right](k\beta S^* e^{-\mu_o \tau}) + (\beta O^* e^{-\mu_o \tau})\left(\mu_\circ + (k\beta S^* e^{-\mu_o \tau})\right),$$

where $a_0 > 0$ if $(\alpha\mu_\circ)\left(\beta O^* e^{-\mu_o \tau} + (\gamma + \mu)\right) > \left[\left(\beta O^* e^{-\mu_o \tau} + (\gamma + \mu)\right)\right](k\beta S^* e^{-\mu_o \tau}) + (\beta O^* e^{-\mu_o \tau})\left(\mu_\circ + (k\beta S^* e^{-\mu_o \tau})\right)$.

According to the Routh–Hurwitz criterion for a third-degree characteristic polynomial, the local stability of the equilibrium depends on the signs of the polynomial coefficients. For the characteristic equation obtained from system (1)–(4), all coefficients are positive under the condition $R_0 > 1$. Therefore, when $R_0 > 1$, the toxoplasmosis endemic equilibrium of the model is locally asymptotically stable.

Conversely, when $R_0 < 1$, one or more of the Routh–Hurwitz stability conditions are violated. Consequently, the endemic equilibrium becomes locally unstable, indicating that infection cannot persist in the population.

4. Global stability analysis

Well-known results are presented for the stability of the toxoplasmosis delayed epidemic model in the global sense as follows:

Theorem 5. The system at toxoplasmosis-free equilibrium (TFE-T_0) is GAS if $R_0 < 1$.

Proof. Define the Volterra Lyapunov function $U: \Omega \to \mathbb{R}$ as

$$U = \left[S - S_0 - S_0 log \frac{S}{S_0}\right] + I + O + S_m.$$

$$\frac{dU}{dt} = \left[1 - \frac{S_0}{S}\right]\frac{dS}{dt} + \frac{dI}{dt} + \frac{dO}{dt} + \frac{dS_m}{dt}.$$

$$\frac{dU}{dt} = \left[\frac{S - S_0}{S}\right][\mu(1 - I) - \beta SOe^{-\mu_o \tau} - (\mu + \gamma)S] + [\beta SOe^{-\mu_o \tau} - \alpha I] + [kI - \mu_\circ O] + [bS_m - \beta_m S_m O - \mu_m S_m].$$

$$\frac{dU}{dt} \leq -\mu\frac{(S - S_0)^2}{SS_0} - \mu_\circ O[1 - \frac{\beta Se^{-\mu_o \tau}}{\mu_\circ}] - \alpha I - \beta_m S_m O - \mu_m S_m.$$

This implies that $\frac{dU}{dt} \leq 0$ if $R_0 < 1$ and $\frac{dU}{dt} = 0$ if $S = S_0, I = O = S_m = 0$.

Therefore, T_0 is globally asymptotically stable.

Theorem 6. The system at $T^* = (S^*, I^*, O^*, S^*_m)$ is globally asymptotically stable if $R_0 > 1$.

Proof. Letting the Lyapunov function $W: \Omega \to \mathbb{R}$ be defined as

$$W = k_1\left(S - S^* - S^* \ln\left(\frac{S}{S^*}\right)\right) + k_2\left(I - I^* - I^* \ln\left(\frac{I}{I^*}\right)\right) + k_3\left(O - O^* - O^* \ln\left(\frac{O}{O^*}\right)\right) + k_4\left(S_m - S^*_m - S^*_m \ln\left(\frac{S_m}{S^*_m}\right)\right).$$

Given positive constants $k_i(i = 1,2,3,4)$, we can express the following equation:

$$\frac{dW}{dt} = k_1\left[\frac{S-S^*}{S}\right]\frac{dS}{dt} + k_2\left[\frac{I-I^*}{I}\right]\frac{dI}{dt} + k_3\left[\frac{O-O^*}{O}\right]\frac{dO}{dt} + k_4\left[\frac{S_m-S^*m}{S_m}\right]\frac{dS_m}{dt}.$$

$$\frac{dW}{dt} = -k_1\mu\frac{(S-S^*)^2}{SS^*} - k_2\frac{(I-I^*)^2}{II^*}(\alpha O)\left[1-\frac{\beta Se^{-\mu_o\tau}}{\alpha}\right] - k_3(kI)\frac{(O-O^*)^2}{OO} - k_4(bS_m)\frac{(S_m-S^*m)^2}{S_mS^*m}.$$

If we choose k_i where $(i = 1,2,3,4)$,

$$\frac{dW}{dt} = -\mu\frac{(S-S^*)^2}{SS^*} - \frac{(I-I^*)^2}{II^*}(\alpha O)\left[1-\frac{\beta Se^{-\mu_o\tau}}{\alpha}\right] - (kI)\frac{(O-O^*)^2}{OO} - (bS_m)\frac{(S_m-S^*m)^2}{S_mS^*m}.$$

$\frac{dW}{dt} \le 0$ for $R_0 > 1$ and $\frac{dW}{dt} = 0$ if and only if $S = S^*,\ I = I^*,\ O = O^*,\ S_m = S^*{}_m$.
Hence, by Lasalle's invariance principle, T^* is globally asymptotical stable.

Theorem 7. (Second-order global stability) The toxoplasmosis-free equilibrium (TFE-T_0), $T_0 = (S_0, I_0, O_0, S_{m_0}) = \left(\frac{\mu}{\mu+\gamma}, 0,0, \frac{b}{\mu_m}\right)$ is globally asymptotically stable if $\mathcal{R}_0 < 1$.

Proof. Define the function B: $\Omega \to \mathbb{R}$ as

$$\mathrm{B}'(I) = \frac{1}{I}\frac{dI}{dt},$$

$$\mathrm{I}''(I) = \frac{1}{I}\frac{d^2I}{dt^2} - \frac{1}{I^2}\left(\frac{dI}{dt}\right)^2,$$

$$\mathrm{B}''(I) = \frac{1}{I}\left(\frac{k\beta SIe^{-\mu_o\tau}}{\mu_\circ} - \alpha I\right)^2 - \frac{1}{I^2}(\alpha)^2\left(1-\frac{k\beta SIe^{-\mu_o\tau}}{\alpha\mu_\circ}\right)^2,$$

$$\mathrm{B}''(I) = (\alpha)^2(\mathcal{R}_0-1)^2 - (\alpha)^2\left(1-\frac{k\beta Se^{-\mu_o\tau}}{\alpha\mu_\circ}\right)^2,$$

$$\mathrm{B}''(I) \le 0 \ \ if\ \ \mathcal{R}_0 < 1.$$

Thus, the system (1)–(4) is globally asymptotically stable at toxoplasmosis-free equilibrium $T_0 = (S_0, I_0, O_0, S_{m_0})$.

Theorem 8. (Second-order global stability) The toxoplasmosis endemic equilibrium (TEE-T^*), $T^* = (S^*, I^*, O^*, S^*{}_m)$ is globally asymptotical stable (GAS) if $R_0 > 1$.

Proof. Define the Lyapunov function W: $\Omega \to \mathbb{R}$ as

$$W = k_1\left(S - S^* - S^*\ln\left(\frac{S}{S^*}\right)\right) + k_2\left(I - I^* - I^*\ln\left(\frac{I}{I^*}\right)\right) + k_3\left(O - O^* - O^*\ln\left(\frac{O}{O^*}\right)\right)$$
$$+k_4\left(S_m - S^*{}_m - S^*{}_m\ln\left(\frac{S_m}{S^*{}_m}\right)\right).$$

$$\frac{d^2W}{dt^2} = \frac{S^*}{S^2}\left(\frac{dS}{dt}\right)^2 + \left(1-\frac{S^*}{S}\right)\frac{d^2S}{dt^2} + \frac{I^*}{I^2}\left(\frac{dI}{dt}\right)^2 + \left(1-\frac{I^*}{I}\right)\frac{d^2I}{dt^2} + \frac{O^*}{O^2}\left(\frac{dO}{dt}\right)^2$$
$$+\left(1-\frac{O^*}{O}\right)\frac{d^2O}{dt^2} + \frac{S^*{}_m}{S_m{}^2}\left(\frac{dS_m}{dt}\right)^2 + \left(1-\frac{S^*{}_m}{S_m}\right)\frac{d^2S_m}{dt^2}.$$

$$\frac{d^2W}{dt^2} = \left((\mu)^2 + (\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right)\frac{S^*}{S^2} - \left(2\mu(\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right)\frac{S^*}{S^2} +$$
$$\left(\mu\left(\beta Oe^{-\mu_o\tau} + (\mu+\gamma)\right)\right)\frac{S^*}{S} - \left(\left(\beta Oe^{-\mu\tau} + (\mu+\gamma)\right)(\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right)\frac{S^*}{S} +$$
$$\left(\left(\beta Oe^{-\mu_o\tau} + (\mu+\gamma)\right)(\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right) - \left(\mu\left(\beta Oe^{-\mu_o\tau} + (\mu+\gamma)\right)\right) +$$
$$((\beta SOe^{-\mu_o\tau})^2 + (\alpha I)^2)\frac{I^*}{I^2} - \left(2(\beta SOe^{-\mu_o\tau})(\alpha I)\right)\frac{I^*}{I^2} + \left((\alpha\beta SOe^{-\mu_o\tau})\right)\frac{I^*}{I} - (\alpha^2 I)\frac{I^*}{I} + (\alpha^2 I) -$$

$\left((\alpha\beta SOe^{-\mu_o\tau})\right) + ((\mu_\circ O)^2 + (kI)^2)\frac{O^*}{O^2} - \left(2(\mu_\circ O)(kI)\right)\frac{O^*}{O^2} + (\mu_\circ kI)\frac{O^*}{O} - ((\mu_\circ)^2 O)\frac{O^*}{O} + ((\mu_\circ)^2 O) - (\mu_\circ kI) + ((bS_m)^2 + (\beta_m S_m O + \mu_m S_m)^2)\frac{S_m{}^*}{S_m{}^2} - \left(2(bS_m)(\beta_m S_m O + \mu_m S_m)\right)\frac{S_m{}^*}{S_m{}^2} + \left(((b)^2 S_m + \beta_m S_m O + \mu_o S_m)\right)\frac{S_m{}^*}{S_m} - \left(2b(\beta_m S_m O + \mu_o S_m)\right)\frac{S_m{}^*}{S_m} + \left(2b(\beta_m S_m O + \mu_o S_m +)\right) - \left(((b)^2 S_m + \beta_m S_m O + \mu_o S_m)\right).$

For simplification, we choose

$\frac{d^2W}{dt^2} = \chi_1 - \chi_2,$

$\chi_1 = \left((\mu)^2 + (\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right)\frac{S^*}{S^2} + \left(\mu\left(\beta Oe^{-\mu_o\tau} + (\mu+\gamma)\right)\right)\frac{S^*}{S} + \left(\left(\beta Oe^{-\mu_o\tau} + (\mu+\gamma)\right)(\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right) + ((\beta SOe^{-\mu_o\tau})^2 + (\alpha I)^2)\frac{I^*}{I^2} + \left((\alpha\beta SOe^{-\mu_o\tau})\right)\frac{I^*}{I} + (\alpha^2 I) + ((\mu_\circ O)^2 + (kI)^2)\frac{O^*}{O^2} + (\mu_\circ kI)\frac{O^*}{O} + ((\mu_\circ)^2 O) + ((bS_m)^2 + (\beta_m S_m O + \mu_m S_m)^2)\frac{S_m{}^*}{S_m{}^2} + \left(((b)^2 S_m + \beta_m S_m O + \mu_o S_m)\right)\frac{S_m{}^*}{S_m} + \left(2b(\beta_m S_m O + \mu_o S_m)\right),$

$\chi_2 = \left(2\mu(\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right)\frac{S^*}{S^2} + \left(\left(\beta Oe^{-\mu\tau} + (\mu+\gamma)\right)(\mu I + \beta SOe^{-\mu_o\tau} + (\mu+\gamma)S)\right)\frac{S^*}{S} + \left(\mu\left(\beta Oe^{-\mu_o\tau} + (\mu+\gamma)\right)\right) + \left(2(\beta SOe^{-\mu_o\tau})(\alpha I)\right)\frac{I^*}{I^2} + (\alpha^2 I)\frac{I^*}{I} + \left((\alpha\beta SOe^{-\mu_o\tau})\right) + \left(2(\mu_\circ O)(kI)\right)\frac{O^*}{O^2} + ((\mu_\circ)^2 O)\frac{O^*}{O} + (\mu_\circ kI) + \left(2(bS_m)(\beta_m S_m O + \mu_m S_m)\right)\frac{S_m{}^*}{S_m{}^2} + \left(2b(\beta_m S_m O + \mu_o S_m)\right)\frac{S_m{}^*}{S_m} + \left(((b)^2 S_m + \beta_m S_m O + \mu_o S_m)\right).$

We can see that

$$\chi_1 > \chi_2,\ \frac{d^2W}{dt^2} > 0, \quad \chi_1 < \chi_2,\ \frac{d^2W}{dt^2} < 0, \quad \chi_1 = \chi_2,\ \frac{d^2W}{dt^2} = 0.$$

5. Sensitivity analysis

A derivative-based local sensitivity approach was applied to assess how variations in model parameters influence the basic reproduction number R_0. This method evaluates the partial derivatives of R_0 with respect to each parameter, thereby quantifying the relative impact of each parameter on disease transmission dynamics.

The sensitivity indices are obtained as follows:

$$T_k = \frac{k}{R_0}\frac{\partial R_0}{\partial k} = 1 > 0,\ T_\beta = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta} = 1 > 0,\ T_\alpha = \frac{\alpha}{R_0}\frac{\partial R_0}{\partial \alpha} = -1 < 0,$$
$$T_{\mu_o} = \frac{\mu_o}{R_0}\frac{\partial R_0}{\partial \mu_o} = -1 < 0, \qquad T_\gamma = \frac{\gamma}{R_0}\frac{\partial R_0}{\partial \gamma} = -\frac{1}{\mu+\gamma} < 0.$$

The results show that the parameters k and β have positive and larger sensitivity indices, indicating that an increase in either parameter enhances the potential for disease transmission. In contrast, the parameters μ_o, α, and γ exhibit negative sensitivity indices, implying that higher values of these parameters reduce the infection potential. Therefore, k and β are the most influential factors in driving the system from a disease-free to an endemic state.

6. Stochastic formulation phase 1

Let

$$U(t) = (S(t), I(t), O(t), S_m(t))^\top$$

denote the state vector of the toxoplasmosis epidemic model corresponding to system (1)–(4). To incorporate random environmental fluctuations and demographic uncertainty, we derive a stochastic formulation based on a diffusion approximation of the underlying Markov jump process.

6.1. Transition structure

The possible transition events, their state-change vectors $T_i \in \mathbb{R}^4$, and corresponding transition probabilities $P_i(U,t)$ are summarized in Table 2. Over a small time interval Δt, the conditional expectation and covariance of the increment $\Delta U(t) = U(t+\Delta t) - U(t)$ satisfy

$$\mathbb{E}^*[\Delta U] = \sum_{i=1}^{9} P_i\,(U,t)\,T_i,$$

$$\mathbb{E}^*[\Delta U\,(\Delta U)^\top] = \sum_{i=1}^{9} P_i\,(U,t)\,T_i T_i^\top,$$

where $\mathbb{E}^*(\cdot)$ denotes the conditional expectation given the current state $U(t)$.

Table 2. Possible transition events and their probabilities.

Transition	Change vector (T_i)	Transition probability (P_i)	Biological interpretation
$\boldsymbol{T_1}$	$[1,0,0,0]^T$	$P_1 = \mu(1-I)\Delta t$	Birth of susceptible hosts
$\boldsymbol{T_2}$	$[-1,1,0,0]^T$	$P_2 = \beta SOe^{-\mu_o\tau}\Delta t$	Infection of susceptible hosts
$\boldsymbol{T_3}$	$[-1,0,0,0]^T$	$P_3 = (\alpha+\mu)S\Delta t$	Death or removal of susceptible hosts
$\boldsymbol{T_4}$	$[0,-1,0,0]^T$	$P_4 = (\alpha+\mu)I\Delta t$	Death or recovery of infected hosts
$\boldsymbol{T_5}$	$[0,0,1,0]^T$	$P_5 = kI\Delta t$	Shedding of oocysts by infected hosts
$\boldsymbol{T_6}$	$[0,0,-1,0]^T$	$P_6 = \mu_o O\Delta t$	Decay or removal of oocysts
$\boldsymbol{T_7}$	$[0,0,0,1]^T$	$P_7 = bS_m\Delta t$	Birth of susceptible intermediate hosts
$\boldsymbol{T_8}$	$[0,0,0,-1]^T$	$P_8 = \beta_m S_m O\Delta t$	Infection of intermediate hosts
$\boldsymbol{T_9}$	$[0,0,0,-1]^T$	$P_9 = \mu_m S_m\Delta t$	Death of intermediate hosts

Dividing by Δt and letting $\Delta t \to 0$, the drift term is obtained as

$$G(U,t) = \lim_{\Delta t\to 0}\frac{\mathbb{E}^*[\Delta U]}{\Delta t} = \begin{pmatrix} \mu(1-I) - \beta SOe^{-\mu_0\tau} - (\mu+\gamma)S \\ \beta SOe^{-\mu_0\tau} - (\alpha+\mu)I \\ kI - \mu_0 O \\ bS_m - \beta_m S_m O - \mu_m S_m \end{pmatrix}. \tag{6}$$

6.2. Diffusion approximation

To represent the stochastic variability induced by the transition events, we introduce a

vector-valued Brownian motion,

$$W(t) = (W_1(t), W_2(t), \ldots, W_9(t))^\top,$$

where each $W_i(t)$ is an independent standard Brownian motion.
The diffusion matrix $H(U,t) \in \mathbb{R}^{4\times 9}$ is constructed directly from the transition structure as

$$H(U,t) = [T_1\sqrt{P_1(U,t)}, T_2\sqrt{P_2(U,t)}, \ldots, T_9\sqrt{P_9(U,t)}].$$

By construction, this matrix satisfies

$$H(U,t)H(U,t)^\top = \sum_{i=1}^{9} P_i\,(U,t)\,T_i T_i^\top,$$

which coincides with the infinitesimal covariance matrix of the jump process.

6.3. Stochastic differential equation model

The stochastic toxoplasmosis model is therefore given by the system of stochastic differential equations

$$dU(t) = G(U,t)\,dt + H(U,t)\,dW(t), \tag{7}$$

that is,

$$\begin{pmatrix} dS \\ dI \\ dO \\ dS_m \end{pmatrix} = \begin{pmatrix} \mu(1-I) - \beta SOe^{-\mu_0\tau} - (\mu+\gamma)S \\ \beta SOe^{-\mu_0\tau} - (\alpha+\mu)I \\ kI - \mu_0 O \\ bS_m - \beta_m S_m O - \mu_m S_m \end{pmatrix} dt + H(U,t)\,dW(t).$$

This formulation ensures that the stochastic perturbations correctly reflect the correlations induced by the biological transition events, while the covariance structure remains positive semidefinite.

6.4. Numerical approximation

To numerically approximate system (7), we apply the Euler–Maruyama scheme. Let $t_n = n\Delta t$. Then

$$U_{n+1} = U_n + G(U_n, t_n)\,\Delta t + H(U_n, t_n)\,\Delta W_n, \tag{8}$$

where $\Delta W_n = W(t_{n+1}) - W(t_n)$ is a vector of independent normal random variables with mean zero and variance Δt.

7. Stochastic formulation phase 2

To explicitly represent uncertainty in each compartment, the stochastic formulation of the toxoplasmosis model can be rewritten as follows:

$$dS(t) = \Big(\mu\big(1-I(t)\big) - \beta S(t)O(t)e^{-\mu_o\tau} - (\mu+\gamma)S(t)\Big)\,dt + \sigma_1 S(t)dB(t), \quad t \geq 0, \tau \leq t. \tag{9}$$

$$dI(t) = \big(\beta S(t)O(t)e^{-\mu_o\tau} - \alpha I(t)\big)dt + \sigma_2 I(t)dB(t), \qquad t \geq 0, \tau \leq t. \tag{10}$$

$$dO(t) = \big(kI(t) - \mu_o O(t)\big)dt + \sigma_3 O(t)dB(t), \qquad t \geq 0, \tag{11}$$

$$dS_m(t) = \big(bS_m(t) - \beta_m S_m(t)O(t) - \mu_m S_m(t)\big)dt + \sigma_4 S_m(t)dB(t), \qquad t \geq 0. \tag{12}$$

Here, σ_i (for $i = 1,2,3,4$) are the intensity parameters that quantify the level of stochastic perturbation in each compartment, while $B(t)$ denotes the standard Brownian motion process representing environmental randomness.

7.1. Feasible properties

This section examines the positivity and boundedness properties of the stochastic system (9)–(12). Let the state vector be defined as

$$V(t) = (S(t), I(t), O(t), S_m(t)),$$

and introduce the Euclidean norm

$$|V(t)| = \sqrt{S^2(t) + I^2(t) + O^2(t) + {S_m}^2(t)}. \tag{13}$$

Let $D_1^{3,1}(\mathbb{R}^4 \times (0,\infty); \mathbb{R}_+)$ denote the set of all nonnegative functions $U_1(V,t)$ defined on $\mathbb{R}^4 \times (0,\infty)$ that are twice continuously differentiable with respect to V and once differentiable with respect to t.

Consider the stochastic model with an implicit delay effect system

$$dV(t) = D_1(V,t)dt + k_1(V,t)\mathrm{dB(t)} \tag{14}$$

and define the associated differential operator T_1 by

$$T_1 = \frac{\partial}{\partial t} + \sum\nolimits_{i=1}^{4} D_{1i}(V,t)\ \frac{\partial}{\partial V_i} + \frac{1}{2}\sum_{i,j=1}^{4} [k_1^T(V,t)\ k_1(V,t)]_{ij}\ \frac{\partial^2}{\partial V_i\, \partial V_j}].$$

When the operator T_1 acts on a function $V^* \in D_1^{3,1}(\mathbb{R}^4 \times (0,\infty); \mathbb{R}_+)$, we have

$$T_1 V^*(V,t) = V_t^*(V,t) + V_V^*(V,t)\ D_1(V,t) + \frac{1}{2}\mathrm{Tr}[k_1^T(V,t)\ V_{VV}^*(V,t)\ k_1(V,t)].$$

Theorem 9. Positivity and existence of a unique global solution.

For system (9)–(12) and any initial condition $X(\theta) = \phi(\theta) \in \mathbb{R}_+^4$ for $\theta \in [-\tau, 0]$, there exists a unique global solution $X(t)$ on $t \geq 0$ that remains in $\mathbb{R}_+^4$ almost surely.

Proof. Let $X(t) = (S(t), I(t), O(t), S_m(t))^{\mathsf{T}}$. Denote by $b(x)$ and $\Sigma(x)$ the drift and diffusion coefficients of (9)–(12), so the system can be written in vector form

$$dX(t) = b(X(t))\ dt + \Sigma(X(t))\ dW(t).$$

Step 1: Local existence and uniqueness (via truncation).

The coefficients $b(x)$ and $\Sigma(x)$ are locally Lipschitz on $\mathbb{R}^4$ (polynomial-type terms) but not necessarily globally Lipschitz. For each $n \in \mathbb{N}$, define truncated coefficients

$$b_n(x) = b(\pi_n(x)), \Sigma_n(x) = \Sigma(\pi_n(x)),$$

where $\pi_n(x) = \frac{n}{\max\{n, \|x\|\}} x$ is the radial projection onto the closed ball $B(\bar{0}, n)$. Then, b_n, Σ_n are globally Lipschitz and satisfy linear growth. Hence, by the standard existence-uniqueness theorem for SDEs, there exists a unique global strong solution $X_n(t)$ for

$$dX_n(t) = b_n(X_n(t))\,dt + \Sigma_n(X_n(t))\,dW(t), X_n(0) = X(0).$$

Define the stopping time

$$\tau_n := \inf\{t \geq 0 : \| X_n(t) \| \geq n\}.$$

By construction, $X_n(t) = X_{n+1}(t)$ for $t < \tau_n$ almost surely, so we can define a maximal local solution $X(t)$ on $[0, \tau_e)$, where $\tau_e := \lim_{n\to\infty} \tau_n$ is the explosion time. This yields a unique local solution of (9)–(12) on $[0, \tau_e)$.

Step 2: Positivity (invariance of $\mathbb{R}^4_+$).

We now show that, starting from nonnegative initial data, the solution cannot cross the coordinate hyperplanes. Observe that on each boundary component, the drift points inward and the diffusion term vanishes in the corresponding component (multiplicative noise structure). For example, at $S = 0$, the S-equation in (9) has the form

$$dS(t) = (\mu N(\cdot) - (\text{nonnegative}) \cdot S(t))\,dt + \sigma_1 S(t)\,dB_1(t),$$

so when $S(t) = 0$, both the diffusion term $\sigma_1 S(t)\,dB_1(t)$ and the loss terms vanish, while the remaining drift term is nonnegative; hence, $S(t)$ cannot become negative. The same argument applies to $I(t)$, $O(t)$, and $S_m(t)$: each component has diffusion proportional to itself, and the drift at zero is nonnegative (no "negative source" at the boundary). Therefore,

$$X(t) \in \mathbb{R}^4_+ \text{for all } t < \tau_e \text{ a.s.}$$

Step 3: Global existence (non-explosion) via stopping times + stochastic Lyapunov.

It remains to show $\tau_e = \infty$ almost surely. Define stopping times

$$\tau_n := \inf\{t \geq 0 : \| X(t) \| \geq n\}, n \geq n_0,$$

and note that $\tau_n \uparrow \tau_e$ as $n \to \infty$. Consider a nonnegative C^2 Lyapunov function $V : \mathbb{R}^4_+ \to \mathbb{R}_+$ (for instance, $V(x) = \sum_{i=1}^{4}(x_i - 1 - \ln x_i)$, which is finite and nonnegative on $\mathbb{R}^4_+$). Applying Itô's formula to $V(X(t \wedge \tau_n))$ yields

$$\mathbb{E}\,V(X(t \wedge \tau_n)) = V(X(0)) + \mathbb{E}\int_0^{t\wedge\tau_n} \mathcal{L}V(X(s))\,ds,$$

where $\mathcal{L}$ is the generator of (9)–(12). Using the model structure and nonnegativity of state variables, we obtain an estimate of the form

$$\mathcal{L}V(x) \leq C_1 + C_2 V(x) \text{for all } x \in \mathbb{R}^4_+,$$

for some constants $C_1, C_2 > 0$. By Grönwall's inequality,

$$\sup_{0\leq s\leq t} \mathbb{E}\,V(X(s \wedge \tau_n)) \leq (V(X(0)) + C_1 t)e^{C_2 t} < \infty.$$

This prevents $V(X(t \wedge \tau_n))$ (hence $\| X(t \wedge \tau_n) \|$) from blowing up in finite time, implying $\mathbb{P}(\tau_e < \infty) = 0$. Therefore, $\tau_e = \infty$ almost surely and the solution is global and remains in $\mathbb{R}^4_+$. This completes the proof.

Theorem 10. If $R_0^S < 1$ and $\sigma^2 < \alpha$, then the infected individuals of the system (9)–(12) exponentially tend to zero.

Proof. Let us consider the initial condition

$$(S(0), I(0), O(0), S_m(0)) \in \mathbb{R}^4_+,$$

and assume that the stochastic system (9)–(12) admits a solution

$$(S(t), I(t), O(t), S_m(t))$$

that satisfies the corresponding stochastic model with an implicit delay effect equation, where σ denotes the diffusion (randomness) term and c represents the drift coefficient.
For the infected population $I(t)$, the stochastic equation can be written as

$$dI(t) = [\,\beta S(t)O(t)e^{-\mu_o \tau} - \alpha I(t)\,]\,dt + c\,\sigma_2 I(t)\,dB(t).$$

Applying Itô's lemma to the logarithmic transformation $g(I) = \ln I$, we obtain

$$dg(I(t)) = [\frac{1}{I(t)}\,dI(t) - \frac{1}{2I^2(t)}\,(dI(t))^2].$$

Substituting into Itô's formula gives

$$d(\ln I) = \frac{1}{I}\,dI - \frac{1}{2I^2}\,(dI)^2.$$

Because for an Itô process, $(dB(t))^2 = dt$, we obtain

$$(dI)^2 = \sigma_2^2 I^2\,dt.$$

Hence,

$$d(\ln I) = \frac{1}{I}\,dI - \frac{1}{2}\sigma_2^2\,dt.$$

Substituting $dI(t)$ from the stochastic equation,

$$d(\ln I) = \frac{1}{I}[\beta S(t)O(t)e^{-\mu_o \tau} - \alpha I(t)]dt + \sigma_2\,dB(t) - \frac{1}{2}\sigma_2^2\,dt.$$

Simplifying,

$$d(\ln I) = [\frac{\beta S(t)O(t)e^{-\mu_o \tau}}{I(t)} - \alpha - \frac{1}{2}\sigma_2^2]dt + \sigma_2\,dB(t).$$

Integrating from 0 to t gives

$$\ln I(t) - \ln I(0) = \int_0^t [\frac{\beta S(s)O(s)e^{-\mu_o \tau}}{I(s)} - \alpha - \frac{1}{2}\sigma_2^2]\,ds + \int_0^t \sigma_2\,dB(s).$$

Let

$$N(t) = \int_0^t \sigma_2\,dB(s),$$

which denotes a stochastic integral with mean zero. Then,

$$\ln I(t) = \ln I(0) + \int_0^t [\frac{\beta S(s)O(s)e^{-\mu_o \tau}}{I(s)} - \alpha - \frac{1}{2}\sigma_2^2]\,ds + N(t).$$

8. Asymptotic behavior

Rearranging terms:

$$\ln I(t) = \ln I(0) + \int_0^t \frac{\beta S(s) O(s) e^{-\mu_o \tau}}{I(s)} ds - (\alpha + \frac{1}{2}\sigma_2^2)t + N(t).$$

1. If $\sigma_2^2 > 4\alpha$, then

$$-(\alpha + \frac{1}{2}\sigma_2^2)t < \alpha t,$$

hence

$$\ln I(t) > \ln I(0) + N(t) + \alpha t.$$

Dividing by t and taking limits,

$$\lim_{t\to\infty} \frac{\ln I(t)}{t} > \alpha.$$

2. If $\sigma_2^2 < 4\alpha$, then

$$\ln I(t) < \ln I(0) + N(t) + \alpha t,$$

which implies

$$\frac{\ln I(t)}{t} < \frac{\ln I(0)}{t} + \frac{N(t)}{t} + \alpha.$$

Since $N(t)/t \to 0$ almost surely as $t \to \infty$, we obtain

$$\lim_{t\to\infty} \frac{\ln I(t)}{t} < \alpha(R_0 - 1)\frac{\alpha\mu_o(\mu+\gamma)}{\mu\beta k - (\mu+\gamma)\alpha\mu_o}.$$

When $R_0 < 1$, this implies

$$\lim_{t\to\infty} \sup \frac{\ln I(t)}{t} \leq 0 \ \ a.s.$$

showing that the infection dies out in the stochastic environment, as required.

9. Stochastic delayed non-standard finite difference scheme

For the stochastic delayed system defined by Eqs (9)–(12), the corresponding non-standard finite difference (NSFD) discretization is expressed as

$$S^{n+1} = \frac{S^n + h\mu(1-I^n) + h\sigma_1 S^n \Delta B_n}{1 + h\beta O^n e^{-\mu_o \tau} + h(\mu+\gamma)}. \tag{24}$$

$$I^{n+1} = \frac{I^n + h\beta S^n O^n e^{-\mu_o \tau} + h\sigma_2 I^n \Delta B_n}{1 + h\alpha}. \tag{25}$$

$$O^{n+1} = \frac{O^n + hkI^n + h\sigma_3 O^n \Delta B_n}{1 + h\mu_\circ}. \tag{26}$$

$$S_m^{n+1} = \frac{S_m^n + hbS_m^n + h\sigma_4 S_m^n \Delta B_n}{1 + h\beta_m O^n + h\mu_m}. \tag{27}$$

Here, h is the discretization time step and $n = 0,1,2,\ldots$. The stochastic component of the scheme is characterized by

$$\Delta B_n = B(t_{n+1}) - B(t_n),$$

where $\Delta B_n \sim \mathcal{N}(0,1)$ represents a normally distributed random variable.

9.1. Stability analysis

To analyze local stability, we first assume that the stochastic intensities satisfy ($\sigma_i = 0$, i = 1,2,3,4). Under this condition, the system (24)–(27) can be represented by the functions

$$E = \frac{S+h\mu(1-I)}{1+h\beta Oe^{-\mu_o\tau}+h(\mu+\gamma)},\ F = \frac{h\beta SOe^{-\mu_o\tau}+I}{1+\alpha h},\ G = \frac{O+hkI}{1+\mu_\circ h},\ H = \frac{S_m(1+hb)}{1+h\beta_m O+h\mu_m}.$$

Jacobian matrix. The Jacobian matrix J of the discrete system is composed of the partial derivatives:

$$\frac{\partial E}{\partial S} = \frac{1}{1+h\beta Oe^{-\mu_o\tau}+h(\mu+\gamma)}, \frac{\partial E}{\partial I} = \frac{-h\mu}{1+h\beta Oe^{-\mu_o\tau}+h(\mu+\gamma)},$$
$$\frac{\partial E}{\partial O} = \frac{-(h\mu(1-I)+S)(h\beta e^{-\mu\tau})}{[1+h\beta Oe^{-\mu_o\tau}+h(\mu+\gamma)]^2}, \frac{\partial E}{\partial S_m} = 0,$$
$$\frac{\partial F}{\partial S} = \frac{h\beta Oe^{-\mu_o\tau}}{1+\alpha h}, \frac{\partial F}{\partial I} = \frac{1}{1+\alpha h}, \frac{\partial F}{\partial O} = \frac{h\beta Se^{-\mu_o\tau}}{1+\alpha h}, \frac{\partial F}{\partial S_m} = 0,$$
$$\frac{\partial G}{\partial S} = 0, \frac{\partial G}{\partial I} = \frac{hk}{1+\mu_o h}, \frac{\partial G}{\partial O} = \frac{1}{1+\mu_o h}, \frac{\partial G}{\partial S_m} = 0,$$
$$\frac{\partial H}{\partial S} = 0, \frac{\partial H}{\partial I} = 0, \frac{\partial H}{\partial O} = \frac{-(S_m(1+hb)h\beta_m)}{[1+h\beta_m O+h\mu_m]^2}, \frac{\partial H}{\partial S_m} = \frac{1+hb}{1+h\beta_m O+h\mu_m}.$$

Theorem 11. Local stability of the toxoplasmosis-free equilibrium.
For all $n \geq 0$, the eigenvalues of the Jacobian matrix of system (24)–(27) evaluated at the toxoplasmosis-free equilibrium,

$$T_0 = (S_0, I_0, O_0, S_{m0}) = \left(\frac{\mu}{\mu+\gamma}, 0, 0, \frac{b}{\mu_m}\right),$$

lie inside the unit circle if and only if $R_0 < 1$.
Proof. Evaluating the Jacobian matrix $J(T_0)$ at T_0, we obtain:

$$J(T_0) = \begin{bmatrix} 1+h(\mu+\gamma) & \frac{-h\mu}{1+h(\mu+\gamma)} & \frac{-(h\beta e^{-\mu_o\tau})(\frac{\mu}{\mu+\gamma}+h\mu)}{[1+h(\mu+\gamma)]^2} & 0 \\ 0 & \frac{1}{1+\alpha h} & \frac{\frac{h\beta\mu e^{-\mu_o\tau}}{\mu+\gamma}}{1+\alpha h} & 0 \\ 0 & \frac{hk}{1+\mu_\circ h} & \frac{1}{1+\mu_o h} & 0 \\ 0 & 0 & \frac{-(1+hb)(h\beta_m)}{[1+h\mu_m]^2} & \frac{1+hb}{1+h\mu_m} \end{bmatrix}.$$

The characteristic equation $|J(T_0) - \lambda I| = 0$ yields the eigenvalues

$$\lambda_1 = \frac{1}{1+h(\mu+\gamma)} < 1,$$
$$\lambda_2 = \frac{1}{1+\alpha h} < 1,$$
$$\lambda_3 = \frac{1}{1+\mu_o h} < 1,$$
$$\lambda_4 = \frac{1 - hk\frac{(h\beta\mu e^{-\mu_o \tau})}{\mu+\gamma}}{1+h\mu_m}.$$

Using the definition of the basic reproduction number $R_0 = \frac{k\mu\beta e^{-\mu_o \tau}}{\alpha\mu_o(\mu+\gamma)}$:

- If $R_0 < 1$, then $\lambda_4 < 1$, and all eigenvalues lie within the unit circle. Hence, the equilibrium T_0 is locally asymptotically stable (L.A.S.).
- Conversely, if $R_0 > 1$, then $\lambda_4 > 1$, indicating that the equilibrium T_0 is unstable. $R_0 < 1 \Rightarrow T_0$ is L.A.S., $R_0 > 1 \Rightarrow T_0$ is unstable.

9.2. Computational results

We take into consideration the system (24)–(27) with reported instances of toxoplasmosis in [14] in order to get the numerical results. Time is measured in days, and the nonlinear least-square curve approach is used to fit the parameter values shown in Table 3. This section compares the newly developed construction for the specific model as a stochastic NSFD scheme, across different step sizes, with the properties of the graphs representing the number of infected individuals using methods already available in the literature, such as stochastic Euler and stochastic Runge–Kutta schemes. Parameter values used in the stochastic toxoplasmosis model were obtained from previous scientific studies and literature searches.

Table 3. Parameter values.

Symbol	Value (per day)	Range (from literature)	Source [14]
$\boldsymbol{\mu}$	0.50	0.1–0.7	Assumed
$\boldsymbol{\alpha}$	0.50	0.2–0.8	Assumed
$\boldsymbol{\mu_o}$	0.385	0.2–0.5	[14]
$\boldsymbol{k}$	0.02	0.01–0.05	[14]
$\boldsymbol{\beta}$	1.10	0.8–1.5	[14]
$\boldsymbol{\beta_m}$	0.10	0.05–0.2	[14]
$\boldsymbol{\gamma}$	0.0005	0.0001–0.001	[14]
$\boldsymbol{b}$	0.50	0.3–0.7	Assumed
$\boldsymbol{\mu_m}$	0.50	0.2–0.6	Assumed

9.3. Discussion

All the simulations are performed using the same set of parameters used in the analytical section. The basic reproductive number R_0 is calculated for this set of parameters to determine the theoretical behavior: If R_0 <1, the theory suggests a convergent solution to the disease-free equilibrium and extinction of the disease; for R_0 > 1, the theory suggests persistence and convergent

behavior to the endemic equilibrium. Figures 1–3 above demonstrate the theoretical results and the numerical performance of the proposed algorithm. Indeed, it is observed that the SNSFD algorithm is able to retain positivity, boundedness, and stability even for large-step sizes, which may not necessarily happen using classical stochastic algorithms. Figures 1(a),(b) illustrate the comparison between the stochastic Euler method and the SNSFD method for the infected cat's equation. For the smaller step size (h = 0.01), both methods exhibit stable results with similar patterns. For the larger step size (h = 1.0), however, the results from the stochastic Euler method become unstable and exhibit diverging patterns, whereas the SNSFD method remains stable with converging results. A similar observation can be made from Figures 1(c),(d), where the results from the stochastic Runge–Kutta method agree with SNSFD results for a smaller step size (h = 0.01) but become unstable and diverge for a larger step size (h = 2.0), whereas the SNSFD method remains stable with converging results. Figure 2 makes it clear how the parameter τ affects the susceptible and infected cat populations. It is clear how the transmission of the disease will be impaired by the survival factor with the increase in the value of τ. Consequently, the value of the susceptible population increases, along with a reduction in the infected population. This also matches the analysis. Figure 3 also confirms the same results because the value of R_0 decreases with the increase in the value of τ.

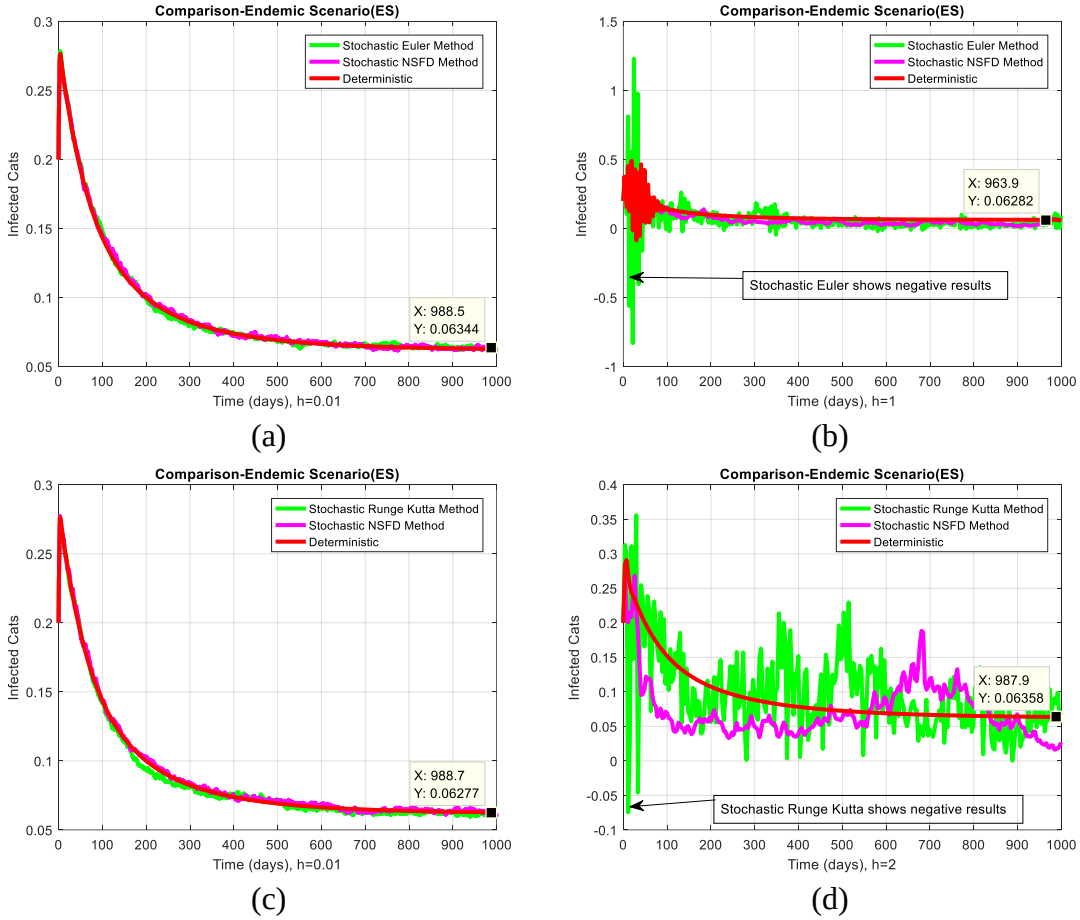
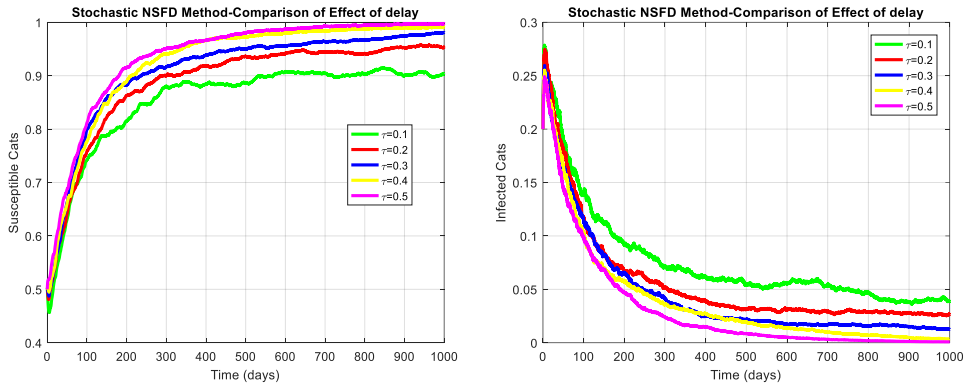


Figure 1. Computational methods used at the toxoplasmosis-endemic equilibrium of the model. (a) Stochastic Euler method's perception of the infected cat population at h = 0.01; (b) stochastic Euler method's perception of the infected cat population at h = 1; (c) stochastic Runge–Kutta method's visualization of the infected cat population at h = 0.01; (d) stochastic Runge–Kutta method's perception of the infected cat population at h = 2.



(e) (f)

Figure 2. Time plot with the time delay on susceptible and infected cat populations. (e) Effect of delay in susceptible cat populations. (f) Effect of delay in infected cat populations.

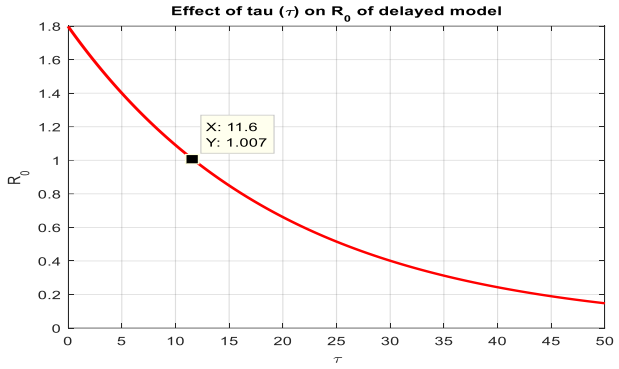


Figure 3. Time plot of the effect of time delay (τ) with reproduction number (R_0).

In general, the results obtained are in agreement with the conclusions drawn and serve to emphasize the major computational contribution of this work: the new SNSFD scheme preserves the essential dynamics characteristics of the continuous model—positive solutions, boundedness, and qualitative stability—while standard stochastic approximation algorithms like Euler-Maruyama, stochastic Euler methods, and stochastic Runge–Kutta fail to do so for particular step sizes because of step-size dependence. This adds stronger credibility to the new stochastic model of toxoplasmosis.

10. Conclusions

In this study, a stochastic non-standard finite difference (SNSFD) scheme was developed to describe the communication dynamics of toxoplasmosis infection. Previous numerical techniques, such as the stochastic Euler and stochastic Runge–Kutta (SRK) methods of order four, were found to be inadequate due to their strong dependence on the time step size. These classical methods exhibit only temporary convergence; as the time step increases, their numerical solutions diverge and deviate significantly from the expected dynamical behavior. Furthermore, such conventional schemes fail to preserve essential structural properties of the continuous model, including positivity, boundedness, and dynamical consistency. To overcome these limitations, the stochastic non-standard finite difference method was proposed. The SNSFD scheme maintains the intrinsic characteristics of the continuous system, ensuring stability, positivity, and boundedness while remaining independent of the

time step size. This approach provides a robust and reliable framework that accurately reproduces the qualitative behavior of the stochastic toxoplasmosis model. A key feature of the proposed framework is the incorporation of an implicit delay effect through a survival probability function, rather than explicit delayed state variables. This approach captures the biological latency associated with oocyst maturation and environmental survival while maintaining analytical tractability. The exponential survival factor modifies the effective transmission rate by accounting for pathogen decay during the latent period, providing a biologically meaningful and mathematically robust representation of delayed infection processes. The methodology presented in this work can be extended to a variety of complex dynamical systems. Future research will focus on applying the SNSFD framework to spatiotemporal, fractional-order, fractal-fractional, and delay-based stochastic models, enabling a deeper understanding of uncertainty and memory effects in real-world biological and epidemiological processes.

Author contributions

Ali Raza: Conceptualization, methodology, software, validation, formal analysis, investigation, data curation, resources, writing–original draft, writing–review & editing, visualization, supervision, project administration; Mansoor Alsulami: Methodology, writing–review & editing, validation, funding acquisition; Eman Ghareeb Rezk: writing–review & editing, validation, funding acquisition. All authors have read and approved the final version of the manuscript for publication.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors affirm they have no conflicts of interest to disclose concerning the current study.

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