Research article

Dynamics of a toxin-mediated aquatic population model with delayed toxic responses

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Abstract: Traditional mathematical models for studying the effects of environmental toxins on population dynamics usually assume that the toxic effects are immediate. However, the effects of toxins on the reproduction and mortality of aquatic populations can be delayed in reality. In this paper, we propose a new mathematical model with delayed toxic responses for aquatic populations in polluted aquatic environments. The delayed model is analyzed in terms of steady states, stability, and bifurcation. The results show that the delayed effect on the reproduction of aquatic populations does not affect the stability of the interior equilibrium, but the delayed effect on the mortality of aquatic populations can destabilize the interior equilibrium. Numerical results corroborate the theoretical ones are presented.

Keywords: aquatic population model; toxin; time delays; stability and bifurcation analysis

1. Introduction

With the development of industry and agriculture, a variety of chemical toxins, such as heavy metals and pesticides etc., have been released into aquatic ecosystems. These toxins have the potential to cause an adverse impact on a diverse range of organisms [1], which has become a primary concern all over the world. Many countries have made a list of priority chemicals of concern and designated policies and measures to mitigate the adverse effects of chemical toxins on aquatic environments [2, 3]. Accurately assessing the risks of toxins requires understandings of the effects of toxins on organisms, as well as on complex ecological interactions.

During the last several decades, mathematical models including individual-based model, matrix population models, differential equation models, have been developed to understand the ecological effects of toxin exposure. Bartell [4] and Pastorok et al. [5] make a complete review on the realism, relevance, and applicability of distinct types of models from the point of view of evaluating risks caused

by environmental toxins. A literature search shows that since a series of work by Hallam et al. [6–9], a number of differential equation models have been proposed and studied to examine the effects of toxins on aquatic populations. A common feature of these differential equation models is that they involve three state variables: the population concentration, the concentration of toxins in the population, and the concentration of toxins in the environment, hence these models describe the interactions between populations and environmental toxins. In practice, because toxins contained in populations are extremely small portion of the whole environment, population birth, mortality, and metabolism do not significantly affect the concentration of toxins in the environment where the population habit. Based on this fact, Huang et al. [10] developed a body burden-dependent population model in which the concentration of toxins in the environment is treated as an important parameter and the effect of the population on environmental toxins is ignored. The model was then used to understand the toxic effects of methylmercury on the long-term behavior of the rainbow trout (*Oncorhynchus mykiss*). In terms of the results of model parameterization, the authors provided a threshold concentration of methylmercury in the external environment to maintain population persistence. Moreover, a complete global and bifurcation analysis of the model can be found in [11].

Time delays are very common in the biological processes, which can cause complex dynamic phenomena including limit cycle oscillations, quasi-periodic oscillations, and even chaos. Time delayed models are widely used to model biological systems, such as population dynamics [12–14], predator prey system [15, 19], tumor immune system [18], gut microbiota system [20], Escherichia coli Tetrahymena system [21], food chain system [22] and so on. Recently, many authors have considered time delays in modeling aquatic population dynamics with the effects of environmental toxins [16, 17, 23]. For example, Chattopadhyay et al. [16] proposed a toxin-producing phytoplankton and zooplankton interaction model and revealed that the toxin substances produced by the phytoplankton play a significant role in the termination of planktonic blooms. Furthermore, Chattopadhyay et al. [17] extended the previous model [16] by considering the time delay of the liberation of toxic substances by the phytoplankton population, and investigated the delayed effect of toxic phytoplankton causing oscillation behaviors of phytoplankton and zooplankton populations. Besides, Jiang et al. [23] developed a delayed phytoplankton-zooplankton model with the coefficients depending on the delay. They showed that the maturity delay of toxic substances might serve as a key factor in the periodic phytoplankton and algae blooms.

The external changes in the environment have some kind of delayed effects on population dynamics, in particular, when a species live in a polluted aquatic environment, toxins have delayed responses on its growth and death [26]. Casarini et al. used Frog Embryo Teratogenesis Assay–Xenopus (FETAX) assay to measure mortality, delayed growth and embryo deformation. The results showed that the growth rate of the embryos was significantly delayed at the concentrations of 0.1 to 10 *nM* of exposure to Okadaic Acid (OA) [27]. And the delayed mortality is observed in several species 4 to 12 d following the exposure to very low toxicant concentrations [28]. A delayed model for studying the interaction among phytoplankton, zooplankton, and fish in the presence of toxins has been developed in [24]. The delayed effect of toxins in a polluted environment on the growth of the population biomass has been considered in [25].

In this work, we extend the above-mentioned body burden-dependent model in [10, 11] to a delay model by incorporating delayed toxic responses. The extension is based on the fact that the effects of toxins on the growth and mortality maybe not immediate, but time-delayed, especially when the

population is subject to sublethal toxin levels [29]. We analyze the model in terms of steady states, stability, and bifurcation. By analyzing the transcendental characteristic equation, we investigate the effects of the time delays on the local stability of equilibria. We find that the Hopf bifurcation can occur in the model as the threshold value increases through certain values of the delays.

The paper consists of five sections. In the next section, we propose the toxin-mediated population model with time-delayed toxic responses. Section 3 contains nondimensionalization, existence and local stability of equilibria, and bifurcation analysis. In section 4, we make numerical simulations to verify the theoretical results. The conclusion and discussion are in the final section.

2. The model

Our model with time delays is based on the following toxin-dependent aquatic population model studied in [10, 11]:

$$\frac{dx(t)}{dt} = \left(\frac{\alpha_1 \max\{0, 1 - \alpha_2 y(t)\}}{1 + \alpha_3 x(t)} - ky(t) - m \right) x(t), \tag{2.1a}$$

$$\frac{dy(t)}{dt} = aT_e - \left(\sigma + \frac{\alpha_1 \max\{0, 1 - \alpha_2 y(t)\}}{1 + \alpha_3 x(t)} \right) y(t), \tag{2.1b}$$

where $x(t)$ represents the concentration of population biomass — the mass of the population per unit volume of the aquatic environment — at time t, $y(t)$ represents the body burden of the population — the mass of toxin per unit population biomass — at time t. The model parameters $\alpha_1, \alpha_2, \alpha_3, k, m, a, T_e$, and σ are all positive constants. See [11] for the detailed model construction.

Equation (2.1a) presents the rate of change of the population biomass under the influence of the toxin. The term, $\alpha_1 \max\{0, 1 - \alpha_2 y(t)\}/(1 + \alpha_3 x(t))$, describes the population gain rate that depends on the population biomass and the body burden, $\alpha_1/(1 + \alpha_3 x(t))$, a decreasing function with respect to population biomass, is a density-dependent biomass gain rate due to birth and growth, where α_1 is the maximum gain rate and α_3 denotes the crowding effect. The expression, $\max\{0, 1 - \alpha_2 y(t)\}$, is a fraction between 0 and 1, which represents linear toxic responses for the gain rate. If there is no toxic effect (i.e., body burden $y(t) = 0$), then $\max\{0, 1 - \alpha_2 y(t)\} = 1$, the gain rate of population biomass is $\alpha_1/(1 + \alpha_3 x(t))$. If the body burden y_i reaches a threshold level $1/\alpha_2$, then $\max\{0, 1 - \alpha_2 y(t)\} = 0$, the gain rate of population biomass is 0. In this case, the population stops reproduction and growth. The term, $ky(t) + m$, means that the population mortality rate linearly depends on the body burden $y(t)$, where k is the effect coefficient of toxin on mortality, and m is the natural mortality rate. If there is no toxic effect, i.e., $y(t) = 0$, then the mortality rate is given by the natural mortality rate m.

Equation (2.1b) is a balance equation for the body burden, where a represents the toxin uptake rate by the population from the environment and T_e is the concentration of toxin in the environment — the mass of toxin per unit volume of the aquatic environment. σ is the toxin elimination rate due to the metabolic processes of the population. The last term of (2.1b), $\alpha_1 \max\{0, 1 - \alpha_2 y(t)\}y(t)/(1 + \alpha_3 x(t))$, means toxin dilution due to birth and growth.

Model (2.1) assumes that the toxic responses (i.e., the effects of toxin on population growth and mortality) are immediate. In this paper, we extend model (2.1) by considering delayed toxic responses. To this end, we introduce two time delays τ_1 and τ_2, which are associated with the effects of toxins on

growth and mortality, respectively. Then a modified model from (2.1) reads

$$\frac{dx(t)}{dt} = \left(\frac{\alpha_1 \max\{0, 1 - \alpha_2 y(t - \tau_1)\}}{1 + \alpha_3 x(t)} - k y(t - \tau_2) - m \right) x(t), \tag{2.2a}$$

$$\frac{dy(t)}{dt} = a T_e - \left(\sigma + \frac{\alpha_1 \max\{0, 1 - \alpha_2 y(t - \tau_1)\}}{1 + \alpha_3 x(t)} \right) y(t), \tag{2.2b}$$

We non-dimensionalize system (2.2) by introducing the following dimensionless quantities:

$$\tilde{x}(\tilde{t}) = \alpha_3 x(t), \ \tilde{y}(\tilde{t}) = \alpha_2 y(t), \ \tilde{t} = \alpha_1 t, \ \tilde{\tau}_1 = \alpha_1 \tau_1, \ \tilde{\tau}_2 = \alpha_1 \tau_2, \ \tilde{k} = \frac{k}{\alpha_1 \alpha_2}, \ \tilde{m} = \frac{m}{\alpha_1}, \ \tilde{T} = \frac{\alpha_2 a T_e}{\alpha_1}, \ \tilde{\sigma} = \frac{\sigma}{\alpha_1}.$$

Dropping the tildes for notational simplicity, we obtain the following scaled model:

$$\frac{dx(t)}{dt} = \left(\frac{\max\{0, 1 - y(t - \tau_1)\}}{1 + x(t)} - k y(t - \tau_2) - m \right) x(t), \tag{2.3a}$$

$$\frac{dy(t)}{dt} = T - \left(\sigma + \frac{\max\{0, 1 - y(t - \tau_1)\}}{1 + x(t)} \right) y(t). \tag{2.3b}$$

We denote by C the Banach space of continuous functions $\phi : [-\tau, 0] \to \mathbf{R}^2$ equipped with the suitable norm, where $\tau = \max\{\tau_1, \tau_2\}$. Furthermore, let

$$C_+ = \phi = (\phi_1, \phi_2) \in C : \phi_i(\theta) \geq 0 \text{ for all } \theta \in [-\tau, 0], i = 1, 2.$$

The initial conditions of system (2.3) are given as

$$x(\theta) = \phi_1(\theta) > 0, \ y(\theta) = \phi_2(\theta) > 0, \ \theta \in [-\tau, 0]. \tag{2.4}$$

3. Local stability and bifurcation analysis

First of all, we show that solutions of system (2.3) behave in a biologically reasonable manner.

Proposition 3.1. The solutions of system (2.3) are always nonnegative and bounded under the initial conditions (2.4).

Proof. Since the right-hand side of the system (2.3) is locally Lipschitz-continuous in $\mathbf{R}_+^2 = \{(x(t), y(t)) | x(t), y(t) \geq 0\}$, we obtain

$$x(t) = x(t_0) \exp \left\{ \int_{t_0}^t \left(\frac{\max\{0, 1 - y(s - \tau_1)\}}{1 + x(s)} - k y(s - \tau_2) - m \right) ds \right\} > 0,$$

$$y(t) = y(t_0) \exp \left\{ \int_{t_0}^t \left(\frac{T}{y(s)} - \sigma - \frac{\max\{0, 1 - y(s - \tau_1)\}}{1 + x(s)} \right) ds \right\} > 0.$$

Using the positive property of solutions, we get

$$\frac{dx(t)}{dt} \leq x \left(\frac{1}{1 + x} - m \right) < 1 - mx,$$

$$\frac{dy(t)}{dt} \leq T - \sigma y.$$

According to the comparison principle, we have

$$\limsup_{t\to\infty} x(t) \leq \frac{1}{m},$$

$$\limsup_{t\to\infty} y(t) \leq \frac{T}{\sigma}.$$

So, any solutions of system (2.3) with the initial conditions (2.4) will stay in the rectangular region:

$$\left\{(x, y) \in \mathbf{R}^2 \Big| \; 0 \leq x < \frac{1}{m}, 0 \leq y < \frac{T}{\sigma}\right\}.$$

$\square$

The existence and stability conditions of equilibria of system (2.3) without time delays were discussed in [11]. Here we summarize the results as follows.

Lemma 3.1. Consider system (2.3) when $\tau_1 = 0$ and $\tau_2 = 0$.

(i) When $0 < \sigma < 1$, system (2.3) has the boundary equilibrium

$$E_1^0 = (0, y_1^0), \quad \text{where } y_1^0 \in (0, (\sigma + 1)/2), \qquad\qquad 0 < T < \sigma,$$

$$E_i^0 = (0, y_i^0), \; i = 1, 2, \quad \text{where } 0 < y_1^0 < \frac{\sigma + 1}{2} < y_2^0 < 1, \quad \sigma < T < \frac{(\sigma + 1)^2}{4},$$

$$E_3^0 = (0, y_3^0), \quad \text{where } y_3^0 > 1, \qquad\qquad\qquad T > \frac{(\sigma + 1)^2}{4}.$$

(ii) When $\sigma \geq 1$, system (2.3) has the boundary equilibrium

$$E_1^0 = (0, y_1^0), \quad \text{where } y_1^0 \in (0, 1), \quad 0 < T < \sigma,$$

$$E_3^0 = (0, y_3^0), \quad \text{where } y_3^0 > 1, \qquad T > \sigma,$$

where

$$y_1^0 = \frac{\sigma + 1 - \sqrt{(\sigma + 1)^2 - 4T}}{2}, \quad y_2^0 = \frac{\sigma + 1 + \sqrt{(\sigma + 1)^2 - 4T}}{2}, \quad y_3^0 = \frac{T}{\sigma}.$$

(iii) When

$$0 < m < 1, \quad T < \frac{(1 - m)\left[\sigma(k + 1) + k + m\right]}{(k + 1)^2} := T_c,$$

system (2.3) has a unique interior equilibrium

$$E^* = \left(\frac{1 - y^*}{ky^* + m} - 1, \; y^*\right),$$

where

$$y^* = \frac{-(m + \sigma) + \sqrt{(m + \sigma)^2 + 4kT}}{2k}.$$

Lemma 3.2. Consider system (2.3) when $\tau_1 = 0$ and $\tau_2 = 0$.

(i) When E_1^0 exists, it is stable if $\sigma > \frac{1 - (2m + k)}{1 + k}$.

(ii) When E_2^0 exists, it is always unstable.

(iii) When E_3^0 exists, it is always stable.

(iv) When E^* exists, it is stable if $T < k\theta^2 + (m + \sigma)\theta := T_\theta$, where

$$\theta = \frac{-(2km + 3m - 2k + \sigma) + \sqrt{(2km + 3m - 2k + \sigma)^2 - 4(k^2 + 3k)(m^2 - 2m - \sigma)}}{2(k^2 + 3k)}.$$

Next, we discuss the effects of τ_1 and τ_2 on the stability of the interior equilibrium E^*. Note that $0 < y^* < 1$, we have $\max\{0, 1 - y^*\} = 1 - y^*$.

Case (I) $\tau_1 > 0$, $\tau_2 = 0$.

Theorem 3.1. For system (2.3), suppose that $\tau_1 > 0$, $\tau_2 = 0$, then when E^* exists, it is locally asymptotically stable if $T < T_\theta$.

Proof. When $\tau_1 > 0$, $\tau_2 = 0$, linearizing system (2.3) at E^*, we have

$$\frac{dx(t)}{dt} = \left(\frac{1 - y^*}{(1 + x^*)^2} - ky^* - m\right) x(t) - kx^* y(t) - \left(\frac{x^*}{1 + x^*}\right) y(t - \tau_1), \tag{3.1a}$$

$$\frac{dy(t)}{dt} = \left(\frac{y^*(1 - y^*)}{(1 + x^*)^2}\right) x(t) - \left(\sigma + \frac{1 - y^*}{1 + x^*}\right) y(t) + \left(\frac{y^*}{1 + x^*}\right) y(t - \tau_1). \tag{3.1b}$$

Let $\vec{Z}(t) = (x(t), y(t))^T$, system (3.1) can be transformed as

$$\frac{d\vec{Z}}{dt} = A\vec{Z}(t) + B\vec{Z}(t - \tau_1), \tag{3.2}$$

where

$$A = \begin{bmatrix} \frac{1 - y^*}{(1 + x^*)^2} - ky^* - m & -kx^* \\ \frac{y^*(1 - y^*)}{(1 + x^*)^2} & -(\sigma + \frac{1 - y^*}{1 + x^*}) \end{bmatrix}, \quad B = \begin{bmatrix} 0 & -\frac{x^*}{1 + x^*} \\ 0 & \frac{y^*}{1 + x^*} \end{bmatrix}.$$

Let $\vec{Z} = e^{\lambda t}\vec{\xi}$, $\vec{\xi} \neq \vec{0}$ in (3.2), then we obtain

$$\lambda e^{\lambda t}\vec{\xi} = Ae^{\lambda t}\vec{\xi} + Be^{\lambda(t - \tau_1)}\vec{\xi},$$

that is,

$$(\lambda I - A - Be^{-\lambda \tau_1})\vec{\xi} = \vec{0},$$

where I is an identity matrix. Therefore, the characteristic equation is

$$\det(\lambda I - A - Be^{-\lambda \tau_1}) = \begin{vmatrix} \lambda - \frac{1 - y^*}{(1 + x^*)^2} + ky^* + m & kx^* + \frac{x^*}{1 + x^*}e^{-\lambda \tau_1} \\ -\frac{y^*(1 - y^*)}{(1 + x^*)^2} & \lambda + \sigma + \frac{1 - y^*}{1 + x^*} - \frac{y^*}{1 + x^*}e^{-\lambda \tau_1} \end{vmatrix} = 0,$$

which can be written as the form

$$P(\lambda) + Q(\lambda)e^{-\lambda \tau_1} = 0, \tag{3.3}$$

where

$$P(\lambda) = \lambda^2 + D_1\lambda + E_1, \quad Q(\lambda) = D_2\lambda,$$

and

$$D_1 = \frac{(k^2 + 2k)(y^*)^2 + (2km + 2m - 2k + \sigma)y^* + m^2 - 2m - \sigma}{y^* - 1},$$

$$E_1 = \frac{(ky^* + m)(ky^* + y^* - 1 + m)(2ky^* + \sigma + m)}{y^* - 1},$$

$$D_2 = \frac{k(y^*)^2 + my^*}{y^* - 1}.$$

Since $E^* = \left(\frac{1 - y^*}{ky^* + m} - 1, y^*\right)$ implies that $0 < y^* < 1$ and $ky^* + y^* + m < 1$, we have that $E_1 > 0$, $D_2 < 0$.

Let $\omega > 0$ and suppose $\lambda = i\omega$ is a pure imaginary root of equation (3.3). Separating the real and imaginary parts, we have

$$\omega D_2 \sin(\omega \tau_1) = \omega^2 - E_1, \tag{3.4a}$$

$$\omega D_2 \cos(\omega \tau_1) = -\omega D_1. \tag{3.4b}$$

Thus

$$h_1(\omega) = \omega^4 + a_1 \omega^2 + a_2 = 0, \tag{3.5}$$

where

$$a_1 = D_1^2 - 2E_1 - D_2^2, \quad a_2 = E_1^2 > 0.$$

Then we have

$$\Delta := (a_1)^2 - 4a_2 = (D_1^2 - 2E_1 - D_2^2)^2 - 4E_1^2 = (D_1 + D_2)(D_1 - D_2)(D_1^2 - 4E_1 - D_2^2). \tag{3.6}$$

Equation (3.5) has positive roots if and only if

$$a_1 < 0, \ \Delta \geq 0.$$

Since $T < T_\theta$, $D_1 + D_2 > 0$, we have $D_1 > 0$, $D_1 - D_2 > 0$. Besides, due to $D_1^2 - 4E_1 - D_2^2 = a_1 - 2E_1 < 0$, we have $\Delta < 0$. It is a contradiction. So equation (3.5) has no positive root.

Therefore, when $\tau_1 > 0$, $\tau_2 = 0$ and E^* exists, it is locally asymptotically stable if $T < T_\theta$. $\qquad \square$

Case (II) $\tau_1 = 0, \tau_2 > 0$.

Theorem 3.2. For system (2.3), suppose that $\tau_1 = 0, \tau_2 > 0$, if (A1) holds, then there is a positive integer m such that
$$0 < \tau_{2,+}^{(0)} < \tau_{2,-}^{(0)} < \tau_{2,+}^{(1)} < \cdots < \tau_{2,-}^{(m-1)} < \tau_{2,+}^{(m)}.$$

E^* is locally asymptotically stable if $\tau_2 \in \left(0, \tau_{2,+}^{(0)}\right) \cup \left(\tau_{2,-}^{(0)}, \tau_{2,+}^{(1)}\right) \cup \cdots \cup \left(\tau_{2,-}^{(m-1)}, \tau_{2,+}^{(m)}\right)$, and E^* is unstable if $\tau_2 \in \left(\tau_{2,+}^{(0)}, \tau_{2,-}^{(0)}\right) \cup \left(\tau_{2,+}^{(1)}, \tau_{2,-}^{(1)}\right) \cup \cdots \cup \left(\tau_{2,+}^{(m)}, \infty\right)$. Furthermore, if (A2) holds, then system (2.3) undergoes Hopf bifurcation at τ_2^*, where $\tau_2^* = \tau_2^{(0)} = \min\{\tau_{2,+}^{(0)}, \tau_{2,-}^{(0)}\}$.

Proof. When $\tau_1 = 0$, $\tau_2 > 0$, linearizing system (2.3) at E^*, we get

$$\frac{dx(t)}{dt} = \left(\frac{1 - y^*}{(1 + x^*)^2} - ky^* - m\right)x(t) - kx^*y(t - \tau_2) - \left(\frac{x^*}{1 + x^*}\right)y(t),$$

$$\frac{dy(t)}{dt} = \left(\frac{y^*(1 - y^*)}{(1 + x^*)^2}\right)x(t) - \left(\sigma + \frac{1 - 2y^*}{1 + x^*}\right)y(t).$$

Similarly, the characteristic equation is given by

$$P(\lambda) + Q(\lambda)e^{-\lambda\tau_2} = 0, \tag{3.7}$$

where

$$P(\lambda) = \lambda^2 + F_1\lambda + G_1, \quad Q(\lambda) = G_2,$$

and

$$F_1 = \frac{(k^2 + 3k)(y^*)^2 + (2km + 3m - 2k + \sigma)y^* + m^2 - 2m - \sigma}{y^* - 1},$$

$$G_1 = \frac{(ky^* + m)(ky^* + y^* - 1 + m)(ky^* + \sigma + m)}{y^* - 1},$$

$$G_2 = \frac{(ky^* + m)(ky^* + y^* - 1 + m)ky^*}{y^* - 1}.$$

Since $0 < y^* < 1$ and $ky^* + y^* + m < 1$, we have $G_1 > 0$, $G_2 > 0$. And since $T < k\theta^2 + (m + \sigma)\theta$, we have $F_1 > 0$.

Let $\omega > 0$ and suppose $\lambda = i\omega$ is a pure imaginary root of equation (3.7). Separating the real and imaginary parts, we have

$$G_2 \cos(\omega\tau_2) = \omega^2 - G_1, \tag{3.8a}$$

$$G_2 \sin(\omega\tau_2) = \omega F_1. \tag{3.8b}$$

Thus

$$h_2(\omega) = \omega^4 + b_1\omega^2 + b_2 = 0, \tag{3.9}$$

where

$$b_1 = F_1^2 - 2G_1, \quad b_2 = G_1^2 - G_2^2 > 0.$$

(A1) : Suppose $b_1 < 0, (b_1)^2 - 4b_2 > 0$.

Equation (3.9) has two positive roots ω_+ and ω_-, where

$$\omega_+ = \left(\frac{-b_1 + \sqrt{(b_1)^2 - 4b_2}}{2}\right)^{1/2}, \tag{3.10a}$$

$$\omega_- = \left(\frac{-b_1 - \sqrt{(b_1)^2 - 4b_2}}{2}\right)^{1/2}. \tag{3.10b}$$

According to (3.8), we obtain

$$\cos(\omega_\pm \tau_2) = \frac{\omega_\pm^2 - G_1}{G_2}, \tag{3.11a}$$

$$\sin(\omega_\pm \tau_2) = \frac{F_1 \omega_\pm}{G_2} > 0. \tag{3.11b}$$

Then solving equation (3.11) for τ_2 yields

$$\tau_2^{(j)} = \frac{1}{\omega_\pm} \arccos\left(\frac{\omega_\pm^2 - G_1}{G_2}\right) + \frac{2j\pi}{\omega_\pm}, \ j = 0, 1, 2, \ldots. \tag{3.12}$$

Define

$$\tau_2^* = \tau_2^{(0)} = \min\{\tau_{2,+}^{(0)}, \tau_{2,-}^{(0)}\}, \ \omega^* = \omega_2^{(0)}. \tag{3.13}$$

Next we obtain the transversality conditions for the Hopf bifurcation at $\tau_2 = \tau_2^*$.

Let $\lambda(\tau_2) = \upsilon(\tau_2) + i\omega(\tau_2)$ be the root of equation (3.7). When $\tau_2 = \tau_2^*$, we have $\upsilon(\tau_2^*) = 0$ and $\omega(\tau_2^*) = \omega^*$.

Differentiating equation (3.7) with respect to τ_2, we have

$$\left(\frac{d\lambda}{d\tau_2}\right)^{-1} = \frac{2\lambda + F_1 - G_2\tau_2 e^{-\lambda\tau_2}}{G_2\lambda e^{-\lambda\tau_2}} = \frac{(2\lambda + F_1)e^{\lambda\tau_2}}{G_2\lambda} - \frac{\tau_2}{\lambda} = \frac{2\omega^2 + F_1^2 - 2G_1}{(\omega^2 - G_1)^2 + F_1^2\omega^2} - \frac{\tau_2}{\lambda}.$$

Thus

$$\begin{aligned}
\text{sign}\left[\frac{d(\text{Re}(\lambda))}{d\tau_2}\right]_{\tau_2=\tau_2^*} &= \text{sign}\left[\text{Re}\left(\frac{d\lambda}{d\tau_2}\right)^{-1}\right]_{\tau_2=\tau_2^*} \\
&= \text{sign}\left[\frac{2\omega^{*2} + F_1^2 - 2G_1}{(\omega^{*2} - G_1)^2 + F_1^2\omega^{*2}}\right] \\
&= \text{sign}[h_2'(\omega^{*2})].
\end{aligned}$$

Hence if the following conditon
(A2) : $h_2'(\omega^{*2}) \neq 0$
holds, then the transversality condition for the Hopf bifurcation is satisfied. □

Case (III) $\tau_1 \in (0, \infty), \ \tau_2 > 0.$

Theorem 3.3. For system (2.3), suppose that for each fixed $\tau_1 \in (0, \infty), \ \tau_2 > 0$, if (A3) holds, then there is a positive integer n such that

$$0 < \tau_2^{(1,0)} < \tau_2^{(2,0)} < \tau_2^{(3,0)} < \tau_2^{(4,0)} < \tau_2^{(1,1)} < \cdots < \tau_2^{(4,n-1)} < \tau_2^{(1,n)} < \tau_2^{(2,n)} < \tau_2^{(3,n)} < \tau_2^{(4,n)}.$$

E^* is locally asymptotically stable if $\tau_2 \in \left(0, \tau_2^{(1,0)}\right) \cup \left(\tau_2^{(2,0)}, \tau_2^{(3,0)}\right) \cup \cdots \cup \left(\tau_2^{(3,n)}, \tau_2^{(4,n)}\right)$, and E^* is unstable if $\tau_2 \in \left(\tau_2^{(1,0)}, \tau_2^{(2,0)}\right) \cup \left(\tau_2^{(3,0)}, \tau_2^{(4,0)}\right) \cup \cdots \cup \left(\tau_2^{(4,n)}, \infty\right)$. Furthermore, if (A4) holds, then system (2.3) undergoes Hopf bifurcation at $\tau_2^\star$, where $\tau_2^\star = \tau_2^{(k_0,0)} = \min_{k\in\{1,\ldots,k_m\}} \{\tau_2^{(k,0)}\}$.

Proof. We assume that τ_1 is arbitrarily fixed within the stable interval $\tau_1 \in (0, \infty)$, while τ_2 is varied as a control parameter. We linearize the system (2.3) at E^* to obtain

$$\frac{dx(t)}{dt} = \left(\frac{1 - y^*}{(1 + x^*)^2} - ky^* - m\right)x(t) - \left(\frac{x^*}{1 + x^*}\right)y(t - \tau_1) - kx^*y(t - \tau_2),$$

$$\frac{dy(t)}{dt} = \left(\frac{y^*(1 - y^*)}{(1 + x^*)^2}\right)x(t) - \left(\sigma + \frac{1 - y^*}{1 + x^*}\right)y(t) + \left(\frac{y^*}{1 + x^*}\right)y(t - \tau_1).$$

Similarly, the characteristic equation is given by

$$P(\lambda) + Q(\lambda)e^{-\lambda\tau_1} + R(\lambda)e^{-\lambda\tau_2} = 0, \tag{3.14}$$

where $P(\lambda) = \lambda^2 + H_1\lambda + I_1$, $Q(\lambda) = H_2\lambda$, $R(\lambda) = I_2$,
and

$$H_1 = \frac{(k^2 + 2k)(y^*)^2 + (2km + 2m - 2k + \sigma)y^* + m^2 - 2m - \sigma}{y^* - 1} > 0,$$

$$I_1 = \frac{(ky^* + m)(ky^* + y^* - 1 + m)(ky^* + \sigma + m)}{y^* - 1} > 0,$$

$$H_2 = \frac{k(y^*)^2 + my^*}{y^* - 1} < 0,$$

$$I_2 = \frac{(ky^* + m)(ky^* + y^* - 1 + m)ky^*}{y^* - 1} > 0.$$

The corresponding real and imaginary parts become

$$I_2 \cos(\omega\tau_2) = \omega^2 - I_1 - H_2\omega\sin(\omega\tau_1), \tag{3.15a}$$

$$-I_2 \sin(\omega\tau_2) = -H_1\omega - H_2\omega\cos(\omega\tau_1). \tag{3.15b}$$

We obtain

$$h_3(\omega) = \omega^4 + c_1\omega^3 + c_2\omega^2 + c_3\omega + c_4 = 0, \tag{3.16}$$

where c_i ($i = 1, 2, 3, 4$) are given by

$$c_1 = -2H_2 \sin(\omega\tau_1), \quad c_2 = H_1^2 + 2H_1H_2\cos(\omega\tau_1) + H_2^2 - 2I_1, \quad c_3 = 2H_2I_1 \sin(\omega\tau_1), \quad c_4 = I_1^2 - I_2^2.$$

(A3) : Suppose that (3.16) has at least one positive root.

Let k_m denote the maximum number of positive real roots of equation (3.16). For $k \in 1, \cdots, k_m$, positive roots of equation (3.16) are denoted by ω_k.

According to (3.15), we obtain

$$\cos(\omega_k\tau_2) = \frac{\omega_k^2 - I_1 - H_2\omega_k \sin(\omega_k\tau_1)}{I_2} := J(\omega_k), \tag{3.17a}$$

$$\sin(\omega_k\tau_2) = \frac{H_1\omega_k + H_2\omega_k \cos(\omega_k\tau_1)}{I_2} := K(\omega_k). \tag{3.17b}$$

Then solving the equation (3.17) for τ_2 yields

$$\tau_2^{(k,j)} = \begin{cases} \dfrac{1}{\omega_k}\left[\arccos\left(\dfrac{\omega_k^2 - I_1 - H_2\omega_k\sin(\omega_k\tau_1)}{I_2}\right) + 2j\pi\right], & K(\omega_k) \geq 0, \ j = 0, 1, 2\ldots \\ \dfrac{1}{\omega_k}\left[2\pi - \arccos\left(\dfrac{\omega_k^2 - I_1 - H_2\omega_k\sin(\omega_k\tau_1)}{I_2}\right) + 2j\pi\right], & K(\omega_k) < 0, \ j = 0, 1, 2\ldots \end{cases}$$

We define

$$\tau_2^\star = \tau_2^{(k_0,0)} = \min_{k\in\{1,\ldots,k_m\}}\{\tau_2^{(k,0)}\}, \quad \omega^\star = \omega_{k_0}. \tag{3.18}$$

By differentiating equation (3.14) with respect to τ_2, we get that

$$P_1(\tau_2)\left[\frac{d(\mathrm{Re}\lambda)}{d\tau_2}\right]_{\tau_2=\tau_2^\star} - Q_1(\tau_2)\left[\frac{d\omega}{d\tau_2}\right]_{\tau_2=\tau_2^\star} = R_1(\tau_2), \tag{3.19}$$

$$Q_1(\tau_2)\left[\frac{d(\mathrm{Re}\lambda)}{d\tau_2}\right]_{\tau_2=\tau_2^\star} + P_1(\tau_2)\left[\frac{d\omega}{d\tau_2}\right]_{\tau_2=\tau_2^\star} = S_1(\tau_2). \tag{3.20}$$

where

$$P_1(\tau_2) = H_1 + H_2\cos(\omega^\star\tau_1) + H_2\tau_1\omega^\star\sin(\omega^\star\tau_1) - I_2\tau_2\cos(\omega^\star\tau_2),$$
$$Q_1(\tau_2) = 2\omega^\star - H_2\sin(\omega^\star\tau_1) - H_2\omega^\star\tau_1\cos(\omega^\star\tau_1) + I_2\tau_2\sin(\omega^\star\tau_2),$$
$$R_1(\tau_2) = -H_2(\omega^\star)^2\cos(\omega^\star\tau_1) + I_2\omega^\star\sin(\omega^\star\tau_2),$$
$$S_1(\tau_2) = H_2(\omega^\star)^2\sin(\omega^\star\tau_1) + I_2\omega^\star\cos(\omega^\star\tau_2).$$

Consequently, we obtain

$$\left[\frac{d(\mathrm{Re}\lambda)}{d\tau_2}\right]_{\tau_2=\tau_2^\star} = \frac{P_1(\tau_2)R_1(\tau_2) + Q_1(\tau_2)S_1(\tau_2)}{P_1(\tau_2)^2 + Q_1(\tau_2)^2}\Bigg|_{\tau_2=\tau_2^\star}.$$

Hence, if the conditon
(A4): $[P_1(\tau_2)R_1(\tau_2) + Q_1(\tau_2)S_1(\tau_2)]|_{\tau_2=\tau_2^\star} \neq 0$
holds, then the transversality condition for Hopf bifurcation is satisfied. $\qquad\square$

4. Numerical simulations

In this section, we numerically study the influence of time delays τ_1 and τ_2 on the stability of the interior equilibrium E^* of system (2.3). We choose a set of parameter values as $k = 0.2$, $m = 0.01$, $\sigma = 0.1$, $T = 0.16$. By some calculations, we have $E^* = (1.38658, 0.660748)$, $T_\theta = 0.170329$, $T_c = 0.226875$. According to Lemma 3.2, E^* is stable when $\tau_1 = \tau_2 = 0$. Stability regions of the interior equilibrium E^* of system (2.3) in (τ_1, τ_2) plane are drawn in Figure 1. We then consider the following three cases and plot some graphs of the numerical solutions of system (2.3).
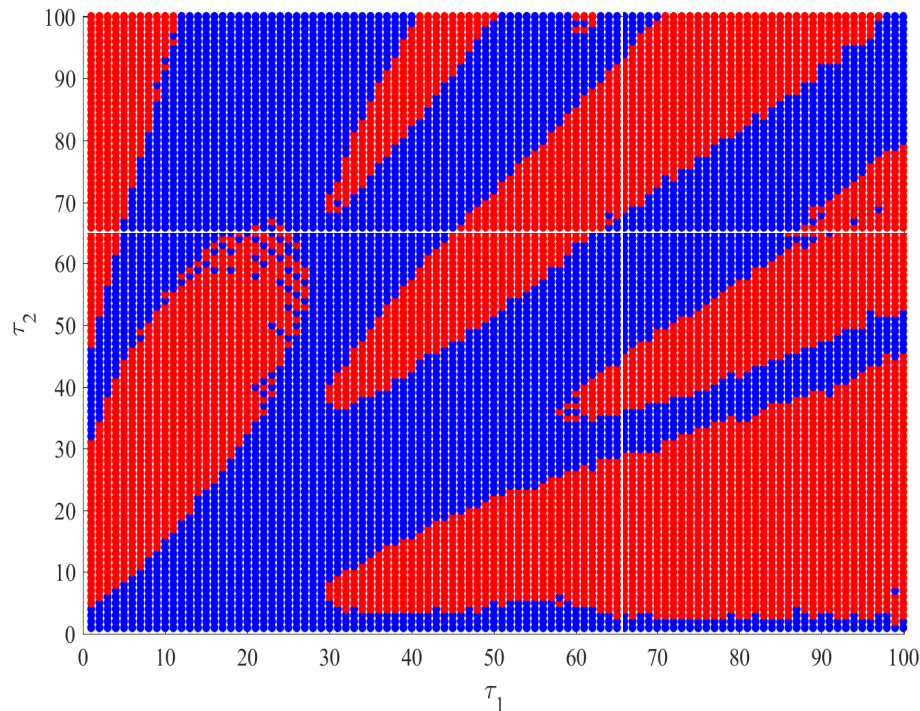
Figure 1. Stability regions of E^* of system (2.3) in (τ_1, τ_2) plane. E^* is locally asymptotically stable in blue regions while it is unstable in red regions.

Case(I) $\tau_1 > 0$, $\tau_2 = 0$.

From (3.5) and (3.6), we have $a_1 = -0.0330221 < 0$ and $\Delta = -0.00273187 < 0$. So equation (3.5) has no positive root. According to Theorem 3.1, E^* is locally asymptotically stable when $\tau_1 > 0$, $\tau_2 = 0$, which means that τ_1 can not change the stability of E^* (see Figure 2).

Case(II) $\tau_1 = 0, \tau_2 > 0$.

From (3.10a) and (3.10b), we have $\omega_+ = 0.165797, \omega_- = 0.101075$. According to Theorem 3.2, there is a positive integer $m = 1$ such that $0 < \tau_{2,+}^{(0)} < \tau_{2,-}^{(0)} < \tau_{2,+}^{(1)}$, where $\tau_{2,+}^{(0)} = 4.9121$, $\tau_{2,-}^{(0)} = 26.5368$, $\tau_{2,+}^{(1)} = 42.8089$. E^* is locally asymptotically stable if $\tau_2 \in \left(0, \tau_{2,+}^{(0)}\right) \cup \left(\tau_{2,-}^{(0)}, \tau_{2,+}^{(1)}\right)$, and E^* is unstable if $\tau_2 \in \left(\tau_{2,+}^{(0)}, \tau_{2,-}^{(0)}\right) \cup \left(\tau_{2,+}^{(1)}, \infty\right)$. When we choose $\tau_2 = 4$, E^* is stable (see Figure 3a). However, when τ_2 increases to $5 > 4.9121$, E^* becomes unstable and tends to the boundary equilibrium. In this case, the subcritical Hopf bifurcation appears resulting in an unstable limit cycle (see Figure 3b). As τ_2 increase to 30, E^* becomes stable again (see Figure 3c). When τ_2 increases to $45 > 42.8089$, E^* becomes unstable again and tends to the boundary equilibrium (see Figure 3d). Furthermore, system (2.3) undergoes Hopf bifurcation at $\tau_2^* = \tau_{2,+}^{(0)} = 4.9121$ (see Figure 1).

Case(III) $\tau_1 \in (0, \infty), \tau_2 > 0$.

Here we fix $\tau_1 = 5$. By calculations, equation (3.16) has two positive roots: $\omega_1 = 0.105931, \omega_2 = 0.0646036$. According to Theorem 3.3, there is a positive integer $n = 1$ such that $0 < \tau_2^{(1,0)} < \tau_2^{(2,0)} < \tau_2^{(1,1)}$, where $\tau_2^{(1,0)} = 9.29205$, $\tau_2^{(2,0)} = 42.7927$, $\tau_2^{(1,1)} = 68.6063$. E^* is locally asymptotically stable if $\tau_2 \in \left(0, \tau_2^{(1,0)}\right) \cup \left(\tau_2^{(2,0)}, \tau_2^{(1,1)}\right)$, and E^* is unstable if $\tau_2 \in \left(\tau_2^{(1,0)}, \tau_2^{(2,0)}\right) \cup \left(\tau_2^{(1,1)}, \infty\right)$. When we choose $\tau_2 = 8$,
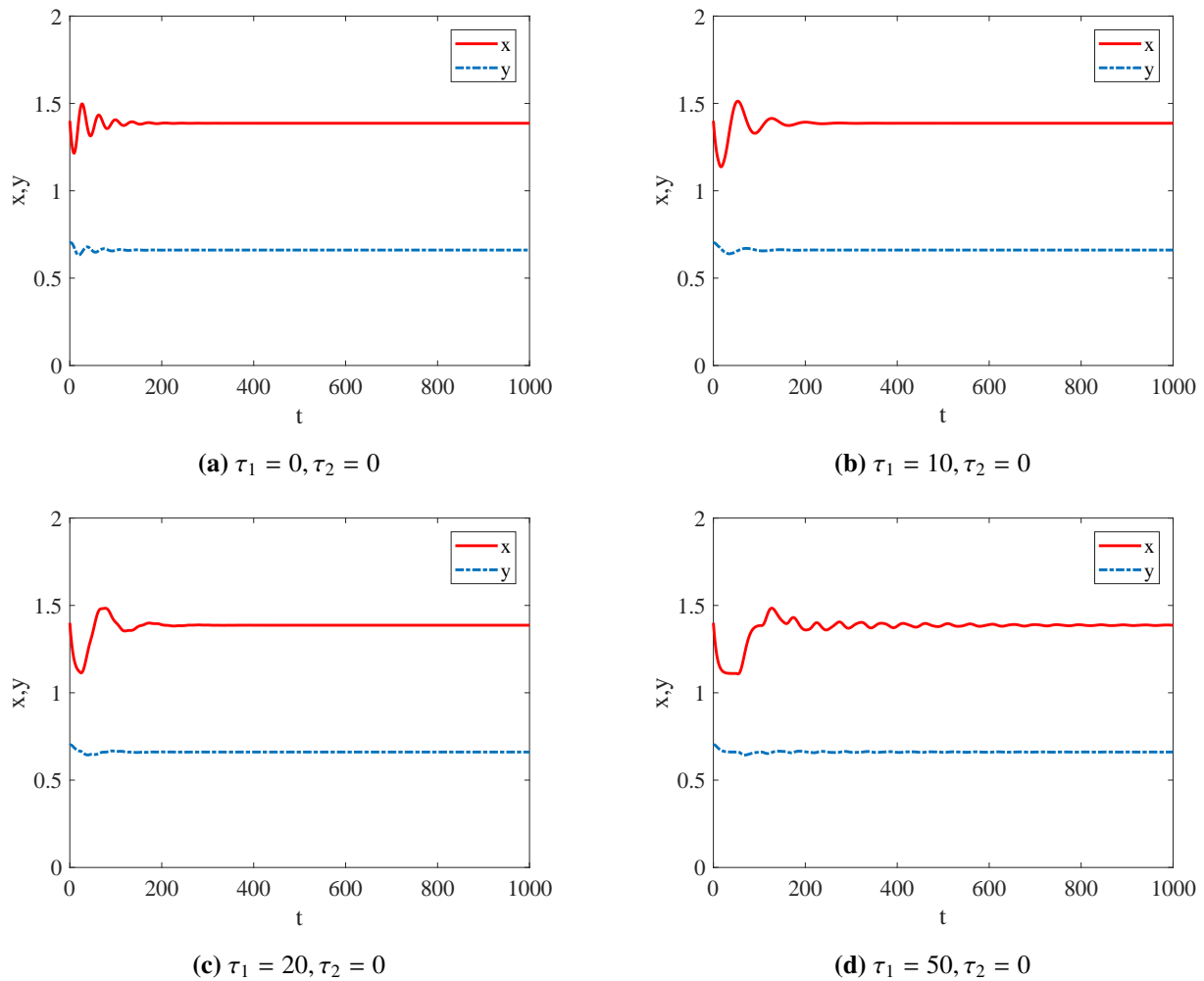
Figure 2. The time evolution curves of system (2.3) with initial conditions $(1.4, 0.7)$. When $\tau_1 = 0, 10, 20, 50$ and $\tau_2 = 0$, E^* is locally asymptotically stable.
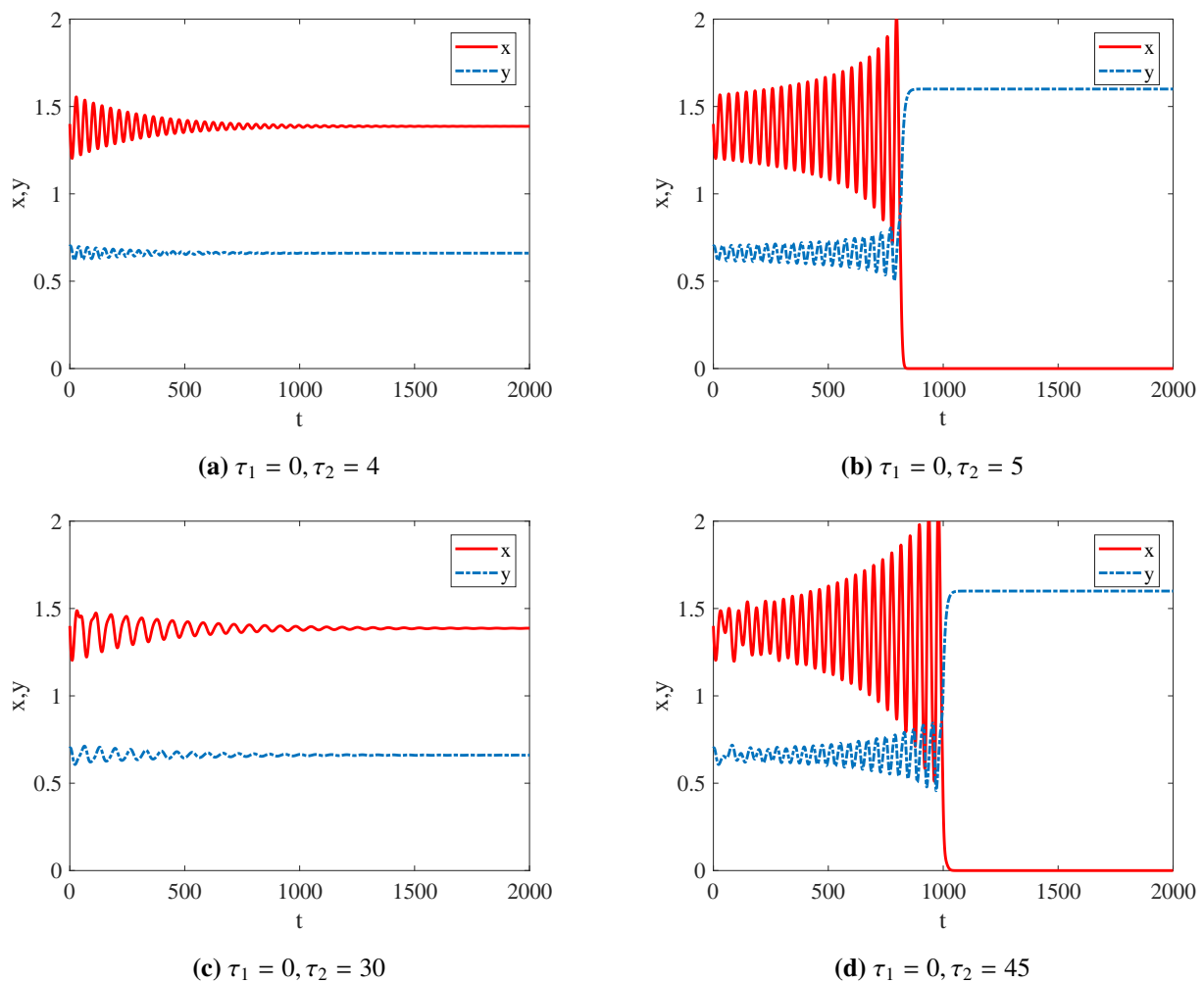
(a) $\tau_1 = 0, \tau_2 = 4$

(b) $\tau_1 = 0, \tau_2 = 5$

(c) $\tau_1 = 0, \tau_2 = 30$

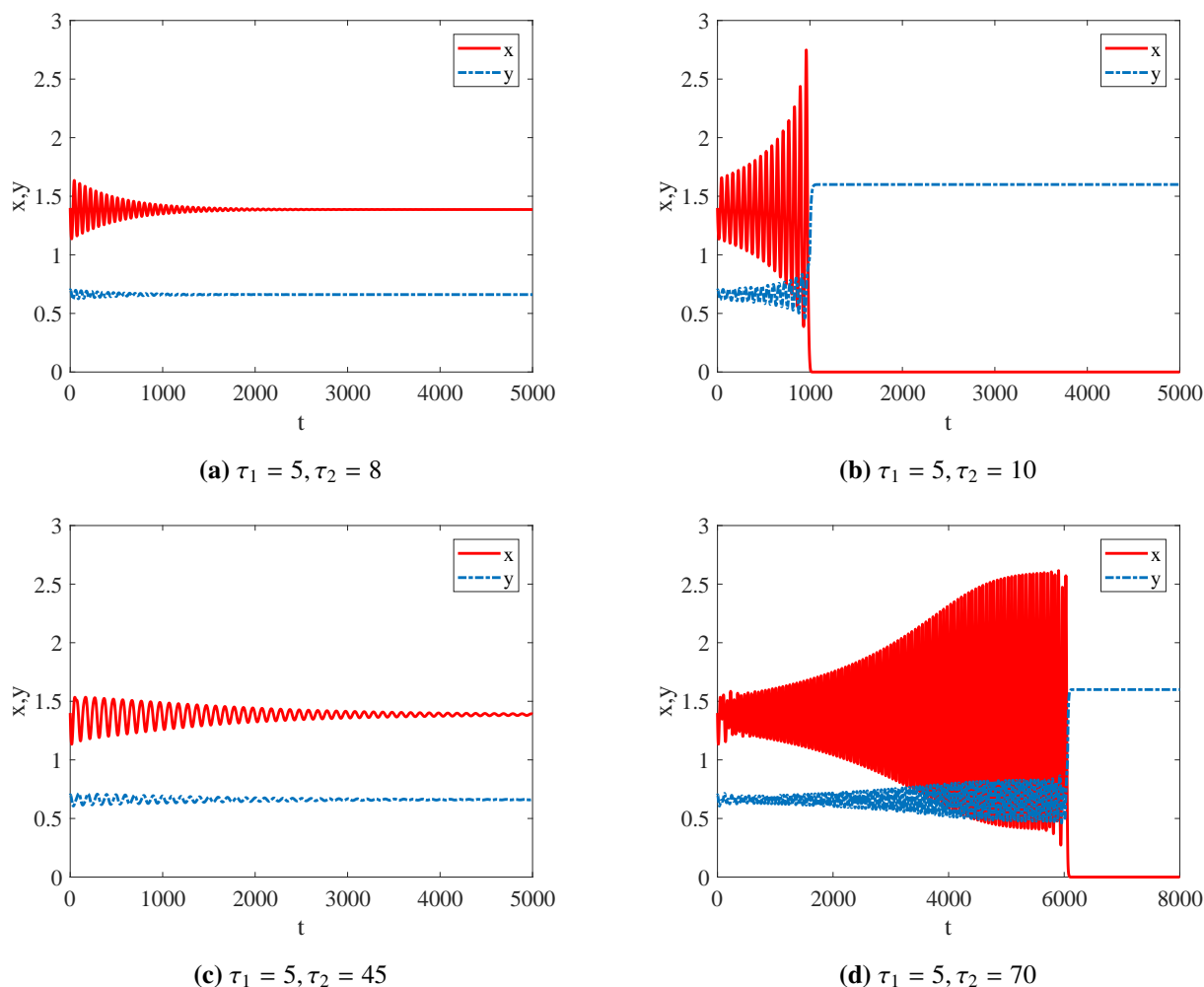**(d)** $\tau_1 = 0, \tau_2 = 45$

Figure 3. The time evolution curves of system (2.3) with initial conditions $(1.4, 0.7)$. (a) When $\tau_1 = 0$ and $\tau_2 = 4$, E^* is locally asymptotically stable. (b) When $\tau_1 = 0$ and $\tau_2 = 5$, E^* is unstable. (c) When $\tau_1 = 0$ and $\tau_2 = 30$, E^* is locally asymptotically stable. (d) When $\tau_1 = 0$ and $\tau_2 = 45$, E^* is unstable.

E^* is stable (see Figure 4a). However, when τ_2 increases to $10 > 9.29205$, E^* becomes unstable and tends to the boundary equilibrium. In this case, the subcritical Hopf bifurcation appears resulting in an unstable limit cycle (see Figure 4b). As τ_2 increase to 45, E^* becomes stable again (see Figure 4c). When τ_2 increases to $70 > 68.6063$, E^* becomes unstable again and tends to the boundary equilibrium (see Figure 4d). Furthermore, system (2.3) undergoes Hopf bifurcation at $\tau_2^\star = \tau_2^{(1,0)} = 9.29205$ (see Figure 1). Although the introduction of τ_1 has no effects on the stability of E^*, it can increase the critical values of τ_2 which can destabilize E^*.

(a) $\tau_1 = 5, \tau_2 = 8$

(b) $\tau_1 = 5, \tau_2 = 10$

(c) $\tau_1 = 5, \tau_2 = 45$

(d) $\tau_1 = 5, \tau_2 = 70$

Figure 4. The time evolution curves of system (2.3) with initial conditions $(1.4, 0.7)$. (a) When $\tau_1 = 5$ and $\tau_2 = 8$, E^* is locally asymptotically stable. (b) When $\tau_1 = 5$ and $\tau_2 = 10$, E^* is unstable. (c) When $\tau_1 = 5$ and $\tau_2 = 45$, E^* is locally asymptotically stable. (d) When $\tau_1 = 5$ and $\tau_2 = 70$, E^* is unstable.

5. Conclusion and discussion

In order to understand the effects of toxins on the dynamics of populations in polluted environments, a large number of mathematical models have been developed [4, 5]. The existing models usually as-

sume that the effects of toxins on population reproduction and mortality are instant. However, there are evidences showing that toxic effects on populations may be delayed. This motivates us to propose a new mathematical model (2.3) with delayed toxic responses in this paper. In the model, two discrete delays τ_1 and τ_2 represent the delayed effects of toxins on the reproduction and mortality of the population, respectively. We mainly focused on the effects of τ_1 and τ_2 on the stability of interior equilibrium E^*, at which the population persists. The results of theoretical analysis showed that the delayed effect on the reproduction of the aquatic population does not change the stability of E^*, but the delayed effect on the mortality of the population significantly affects long-term population dynamics. It turns out that the delayed mortality effects not only produces population oscillations, but also change the fluctuation amplitudes. So, we suggest that experimental biologists should pay more attention to the delayed mortality caused by environmental toxins.

An interesting and challenging future work is the global stability analysis of the equilibria of model (2.3). The delay model (2.3) we studied in this work can be extended in several biologically meaningful ways: (1) The delayed toxic responses in our model are constant. Indeed, the delays can depend on toxin levels or some other environmental factors. We can modify our model such that the delays are functions of the body burden. (2) In a population, different development stages may have different sensitivities to toxins and different delayed toxic responses, so it is worth extending the current model to a stage-structured population model by taking these factors into account. (3) Different species living in the same environment may have different vulnerabilities to toxins, the impact of toxins on the interactions between different species in polluted aquatic environments was studied in [30] and [31]. We plan to investigate how delayed toxic responses affect the long-term dynamics of two interactive species in the future.

Acknowledgments

Conflict of interest

The author declares no conflicts of interest in this paper.

References

1. C. H. Walker, R. Sibly, S. Hopkin, D. B. Peakall, Principles of Ecotoxicology, CRC Press, Boca Raton, 2012.

2. The Canadian Council of Ministers of the Environment, Canadian water quality guidelines for the protection of aquatic life: Guidance on the site-specific application of water quality guidelines in Canada: procedures for deriving numerical water quality objectives, 2003, http://ceqg-rcqe.ccme.ca/download/en/221.

3. US national archives and records administration, code of federal regulations, title 40-protection of environment, Appendix A to part 423–126 priority pollutants.

4. S. M. Bartell, R. A. Pastorok, H. R. Akcakaya, H. Regan, S. Ferson, C. Mackay, Realism and relevance of ecological models used in chemical risk assessment, *Hum. Ecol. Risk Assess.*, **9** (2003), 907–938.

5. R. A. Pastorok, S. M. Bartell, S. Ferson, L. R. Ginzburg, *Ecological Modeling in Risk Assessment: Chemical Effects on Populations, Ecosystems, and Landscapes*, CRC Press, Boca Raton, 2001.

6. T. G. Hallam, C. E. Clark, R. R. Lassiter, Effects of toxicants on populations: Aqualitative approach I. Equilibrium environmental exposure, *Ecol. Model.*, **18** (1983), 291–304.

7. T. G. Hallam, C. E. Clark, G. S. Jordan, Effects of toxicants on populations: A qualitative approach II. First order kinetics, *J. Math. Biol.*, **18** (1983), 25–37.

8. T. G. Hallam, J. T. De Luna, Effects of toxicants on populations: A qualitative approach III. Environmental and food chain pathways, *J. Theor. Biol.*, **109** (1984), 411–429.

9. J. T. De Luna, T. G. Hallam, Effects of toxicants on populations: A qualitative approach IV. Resource-consumer-toxicant models, *Ecol. Model.*, **109** (1987), 249–273.

10. Q. Huang, L. Parshotam, H. Wang, C. Bampfylde, M. A. Lewis, A model for the impact of contaminaants on fish population dynamics, *J. Theor. Biol.*, **334** (2013), 71–79.

11. Q. Huang, G. Seo, C. Shan, Bifurcations and global dynamics in a toxin-dependent aquatic population model, *Math. Biosci.*, **296** (2018), 26–35.

12. N. MacDonald, *Biological Delay Systems: Linear Stability Theory*, Cambridge University Press, Cambridge, 1989.

13. Y. Kuang, *Delay Differential Equations: With Applications in Population Dynamics*, Academic Press, 1993.

14. J. M. Cushing, *Integro-differential Equations and Delay Models in Population Dynamics*, Springer Science & Business Media, 2013.

15. S. Ruan, Absolute stability, conditional stability and bifurcation in Kolmogorov-type predator-prey systems with discrete delays, *Quart. Appl. Math*, **59** (2001), 159–173.

16. J. Chattopadhayay, R. R. Sarkar, S. Mandal, Toxin-producing plankton may act as a biological control for planktonic blooms—field study and mathematical modelling, *J. Theor. Biol.*, **215** (2002), 333–344.

17. J. Chattopadhayay, R. R. Sarkar, A. El Abdllaoui, A delay differential equation model on harmful algal blooms in the presence of toxic substances, *IMA J. Math. Appl. Med. Biol.*, **19** (2002), 137–161.

18. Y. Dong, G. Huang, R. Miyazaki, Y. Takeuchi, Dynamics in a tumor immune system with time delays, *Appl. Math. Comput.*, **252** (2015), 99–113.

19. Z. Jiang, L. Wang, Global Hopf bifurcation for a predator-prey system with three delays, *Int. J. Bifur. Chaos*, **27** (2017), 1750108.

20. Y. Dong, Y. Takeuchi, S. Nakaoka, A mathematical model of multiple delayed feedback control system of the gut microbiota-Antibiotics injection controlled by measured metagenomic data, *Nonlinear Anal. Real World Appl.*, **43** (2018), 1–17.

21. Y. Dong, M. Sen, M. Banerjee, Y. Takeuchi, S. Nakaoka, Delayed feedback induced complex dynamics in an Escherichia coli and Tetrahymena system, *Nonlinear Dyn.*, **94** (2018), 1447–1466.

22. R. K. Upadhyay, S. Mishra, Y. Dong, Y. Takeuchi, Exploring the dynamics of a tritrophic food chain model with multiple gestation periods, *Math. Biosci. Eng.*, **16** (2019), 4660–4691.

23. Z. Jiang, L. Wang, Global Hopf bifurcation of a delayed phytoplankton-zooplankton system considering toxin producing effect and delay dependent coeffcient, *Math. Biosci. Eng.*, **16** (2019), 3807–3829.

24. P. Panja, S. K. Mondal, D. K. Jana, Effects of toxicants on Phytoplankton-Zooplankton-Fish dynamics and harvesting, *Chaos Soliton. Fract.*, **104** (2017), 389–399.

25. S. K. Sahani, How delay can affect the survival of species in polluted environment, in "Soft Computing for Problem Solving" (eds. J. C. Bansal, K. N. Das, A. Nagar, K. Deep, A. K. Ojha), Springer, Singapore, (2019), 913–923.

26. V. Vasconcelos, J. Azevedo, M. Silva, V. Ramos, Effects of marine toxins on the reproduction and early stages development of aquatic organisms, *Mar. Drugs*, **8** (2010), 59–79.

27. L. Casarini, A. Franchini, D. Malagoli, E. Ottaviani, Evaluation of the effects of the marine toxin okadaic acid by using FETAX assay, *Toxicol. Lett.*, **169** (2007), 145–151.

28. M. A. Beketov, M. Liess, Acute and delayed effects of the neonicotinoid insecticide thiacloprid on seven freshwater arthropods, *Environ. Toxicol. Chem.*, **27** (2008), 461–470.

29. A. C. Gutleb, J. Appelman, M. C. Bronkhorst, J. H. J. van den Berg, A. Spenkelink, A. Brouwer et al., Delayed effects of pre- and early-life time exposure to polychlorinated biphenyls on tadpoles of two amphibian species (*Xenopus lae6is and Rana temporaria*), *Environ. Toxicol. Pharmacol.*, **8** (1999), 1–14.

30. Q. Huang, H. Wang, M. A. Lewis, The impact of environmental toxins on predator-prey dynamics, *J. Theor. Biol.*, **378** (2015), 12–30.

31. C. Shan, Q. Huang, Direct and indirect effects of toxins on competition dynamics of species in an aquatic environment, *J. Math. Biol.*, **78** (2019), 739–766.