

Received XXXX

(www.interscience.wiley.com) DOI: 10.1002/sim.0000

MOS subject classification: 34F05; 37A30; 92D25

# Dynamics of a stochastic phytoplankton-toxic phytoplankton-zooplankton system under regime switching

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In this paper, a stochastic phytoplankton-toxic phytoplankton-zooplankton system with Beddington-DeAngelis functional response, where both the white noise and regime switching are taken into account, is studied analytically and numerically. The aim of this research is to study the combined effects of the white noise, regime switching and toxin-producing phytoplankton (TPP) on the dynamics of the system. Firstly, the existence and uniqueness of global positive solution of the system is investigated. Then some sufficient conditions for the extinction, persistence in the mean and the existence of a unique ergodic stationary distribution of the system are derived. Significantly, some numerical simulations are carried out to verify our analytical results, and show that high intensity of white noise is harmful to the survival of plankton populations, but regime switching can balance the different survival states of plankton populations and decrease the risk of extinction. Additionally, it is found that an increase in the toxin liberation rate produced by TPP will increase the survival change of phytoplankton, while it will reduce the biomass of zooplankton. All these results may provide some insightful understanding on the dynamics of phytoplankton-zooplankton system in randomly disturbed aquatic environments. Copyright © 0000 John Wiley & Sons, Ltd.

**Keywords:** stochastic phytoplankton-toxic phytoplankton-zooplankton system, white noise, regime switching, extinction, stationary distribution

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## 1. Introduction

Plankton are the basis of the freshwater and seawater food chains, and their importance for the wealth of aquatic ecosystems and ultimately for the planet itself is nowadays widely recognized [1]. Phytoplankton, particularly, can create energy for the aquatic life through photosynthesis and produce large amounts of oxygen by absorbing carbon dioxide from their surroundings. However, the rapid growth of phytoplankton can cause large-scale blooms in one area and the occurrences of harmful phytoplankton blooms have been reported globally with an increasing frequency in the past decades [2], which are detrimental to the public health, fisheries, tourisms as well as the balance of ecosystems [3]. For example, some freshwater lakes in China, such as Lake Taihu, Lake Poyanghu, Lake Chaohu, etc., have suffered varying degrees of toxic cyanobacterial blooms in recent years. In 2011, Lake Erie experienced the largest harmful algal blooms (HABs) in its recorded history, with a peak intensity over three times greater than any previously observed bloom [4]. Based on the huge effects of planktonic blooms and the mechanisms behind them are not yet clearly understood, therefore, it is necessary and important to understand the dynamic mechanisms of changes in plankton populations.

Many researchers have attempted different approaches to explain the dynamic mechanisms of planktonic blooms in the past decades. The results from experiments suggested the toxic or noxious chemicals produced by blue-green algae may reduce the grazing pressure of zooplankton population and even cause their mortality for a long time [5, 6], which could be one of the key parameters for planktonic blooms [7]. Some experimental evidence demonstrated that the grazing pressure by microzooplankton can represent an important factor for the controlling and regulation of HABs [8, 9]. In addition, there is an experiment revealing that under some suitable conditions, the formation of *Microcystis* blooms is closely related to the presence or absence of zooplankton population and to its selective grazing of the naturally occurring zooplankton [10]. Another approach the researchers are trying to explain the bloom phenomenon is the role of toxicity. The result that toxicity may be as a strong mediator in the zooplankton feeding rate is found in the field observation [11], as well as the laboratory experiment [12]. Moreover, the experimental findings and field study revealed that TPP can suppress the grazing pressure of zooplankton and may act as a biological control way for the termination of planktonic blooms [13, 14]. All these results imply that the toxin production plays a significant role in the interaction between phytoplankton and zooplankton populations, which may greatly stimulate researchers to explore the way how these toxin production affects the coexistence and survival prospect of plankton populations in the presence of non-toxic and toxic phytoplankton.

Due to the complexity and openness of the real aquatic ecosystems, establishing mathematical models is now classical way to study the planktonic blooms [15], which can provide quantitative insights into the dynamic mechanisms of changes in plankton populations. In recent years, many deterministic mathematical models for plankton dynamics, such as the delayed nutrient-phytoplankton models [16, 17], a diffusive nutrient-toxic phytoplankton model [18], the viral infection nutrient-phytoplankton models [19, 20], a phytoplankton-toxin producing phytoplankton-zooplankton model [13], and so on, have been developed and studied extensively, and many interesting results have been shown. However, plankton populations in the real aquatic environments often fluctuate unpredictably because of the unpredictability of environmental stochasticity, and these deterministic models did not capture the random environmental fluctuations which is important feature of aquatic ecosystems. In fact, some experiments shown that the environmental noise has a significantly effect on population systems in ecology [21, 22]. For example, the work of Reichwaldt et al. [23] demonstrated that the wind can be as the most likely driver to control the biomass of cyanobacteria. In addition, the growth rate of toxic *Microcystis* and environmental biomass rely heavily on the temperature and nutrient concentration [24, 25]. Thus, plankton systems with environmental fluctuations are significantly more reasonable, and the issue on how the environmental fluctuations affect plankton systems have attracted increasing attention and great effort has been made towards the study of the dynamics of stochastic plankton systems recently [26, 27, 28, 29, 30, 31, 32]. But the study of stochastic phytoplankton-toxic phytoplankton-zooplankton system still in its infancy, especially the dynamics of the phytoplankton-toxin producing-phytoplankton-zooplankton system with white noise and regime switching is not very clear currently. Thus, we mainly present the effects of white noise, regime switching and toxic substances produced by TPP on the stochastic phytoplankton-toxic phytoplankton-zooplankton system in this paper.

The rest of this paper is organized as follows. The model is presented in section 2. Section 3 introduces some preliminaries firstly, and then we give the main results, including the existence and uniqueness of the global positive solution, extinction and persistence in the mean as well as the stationary distribution and ergodicity of the system. Some numerical simulations are carried out to study the dynamics of the system in section 4. We summarize the results and present our conclusions in section 5.

## 2. Model formation

In this section, we will establish a stochastic two preys-predator model in which the zooplankton feeds on two types of phytoplankton species, including a non-toxic phytoplankton (NTP) and a toxic one. The construction of the stochastic phytoplankton-toxic phytoplankton-zooplankton system is based on the following assumptions:

- It is assumed that  $P_1(t), P_2(t), Z(t)$  are the population densities of NTP, TPP and zooplankton, respectively;  $m$  is the natural death rate of zooplankton.
- It is considered that the growths of NTP and TPP in the absence of the grazer zooplankton are generally considered as logistic type with the intrinsic growth rates  $r_1$  and  $r_2$ , respectively, and their corresponding environmental carrying capacities  $K_1$  and  $K_2$ .
- Assuming that  $a_1$  and  $a_2$  measure the competitive effects of TPP on NTP and NTP on TPP, respectively. In fact, these competitions have been introduced into ecological systems to explore the properties of plankton dynamics, such as stability [29], oscillation and chaos [33], etc.
- Behavior of the entire community is assumed to arise from the coupling of these interacting species. Both groups of phytoplankton exhibit Beddington-DeAngelis functional response to the grazer zooplankton as given by  $\frac{\alpha Z(t)}{1+b_1 P_1(t)+b_2 P_2(t)}$  and  $\frac{\beta Z(t)}{1+b_1 P_1(t)+b_2 P_2(t)}$ , where  $\alpha$  and  $\beta$  are the attack rates of zooplankton on NTP and TPP, respectively;  $b_1$  and  $b_2$  are the product of attack rate and handling time on NTP and TPP, respectively. In addition, the term  $\frac{\gamma P_2 Z}{1+b_1 P_1+b_2 P_2}$ , which describes the resultant reduction for the growth of zooplankton due to the ingestion of TPP, where  $\gamma$  is the inhibition rate of zooplankton growth, while the term  $\frac{\delta P_1 Z}{1+b_1 P_1+b_2 P_2}$  can be regarded as the growth form of zooplankton in the present of NTP, where  $\delta$  is the conversion efficiency. The Beddington-DeAngelis functional response [34, 35] here provides a more appropriate in case of plankton population due to the fact that the predator individuals either search, consume or interfere with each other [29].
- It is assumed that the environmental noise exists in the realistic aquatic ecosystems because of the unpredictability of the environmental stochasticity, such as nutrients supply, water temperature, and some other small environmental fluctuations, which may affect population growths of the system. Actually, the work of May [36] have pointed out that these small environmental fluctuations can affect the ecological parameters of a model more or less, which can be described by white noise. Thus, following the idea of [26, 27, 30, 31], the convenient formulations, which describe the intrinsic growth rates of phytoplankton populations and the death rate of zooplankton population that are influenced by white noise, are taken as  $r_i \rightarrow r_i + \sigma_i dB_i(t)$  ( $i = 1, 2$ ) and  $-d \rightarrow -d + \sigma_3 dB_3(t)$ , respectively. Here the terms  $dB_i(t)$  denote the white noises and  $\sigma_i^2 > 0$  are their intensities of white noises,  $i = 1, 2, 3$ .
- We further consider the regime switching into the model, where the biomass of plankton often suffer switching abruptly to a contrasting alternative stable state in the real world due to some kinds of moderate environmental fluctuations, for example, environmental pollution, rain falls [37, 38] and biotic exploitation [39]. The plankton population models in this case can be characterised by the telegraph noise or colored noise [40], which may cause the population systems switching from one environmental regime to any other regimes [41, 42]. In addition, the switching is generally memoryless and the waiting time between two shifts follows exponential distribution. The convenient formulation here is to take  $\xi(t), t \geq 0$  as regime switching, which is a continuous-time Markov chain with state space  $\aleph = \{1, 2, \dots, n\}, 1 \leq n < \infty$ .
- It is assumed that the Markov chain  $\xi(t)$  and the Brownian motions  $B_i(t), i = 1, 2, 3$  are defined on a completed probability space  $(\Omega, \mathcal{L}_t, \{\mathcal{L}_t\}_{t \geq 0}, \mathcal{P})$  with a filtration  $\{\mathcal{L}_t\}$  satisfying the usual normal conditions, and  $\xi(t)$  is independent of  $B_i(t), i = 1, 2, 3$ .

Based on above assumptions, a stochastic phytoplankton-toxic producing phytoplankton-zooplankton system under regime switching is presented as follows:

$$\begin{cases} dP_1(t) = P_1(t) \left[ r_1(\xi(t)) \left( 1 - \frac{P_1(t)}{K_1(\xi(t))} \right) - a_1(\xi(t)) P_2(t) \right. \\ \quad \left. - \frac{\alpha(\xi(t)) Z(t)}{1 + b_1(\xi(t)) P_1(t) + b_2(\xi(t)) P_2(t)} \right] dt + \sigma_1(\xi(t)) P_1(t) dB_1(t), \\ dP_2(t) = P_2(t) \left[ r_2(\xi(t)) \left( 1 - \frac{P_2(t)}{K_2(\xi(t))} \right) - a_2(\xi(t)) P_1(t) \right. \\ \quad \left. - \frac{\beta(\xi(t)) Z(t)}{1 + b_1(\xi(t)) P_1(t) + b_2(\xi(t)) P_2(t)} \right] dt + \sigma_2(\xi(t)) P_2(t) dB_2(t), \\ dZ(t) = Z(t) \left[ \frac{\delta(\xi(t)) P_1(t) - \gamma(\xi(t)) P_2(t)}{1 + b_1(\xi(t)) P_1(t) + b_2(\xi(t)) P_2(t)} - d(\xi(t)) \right] dt + \sigma_3(\xi(t)) Z(t) dB_3(t). \end{cases} \quad (1)$$

where  $r_1(k), K_1(k), a_1(k), b_1(k), r_2(k), K_2(k), a_2(k), b_2(k), \alpha(k), \beta(k), \gamma(k), \delta(k)$  and  $\sigma_i(k)$  ( $i = 1, 2, 3$ ) are all positive constants for each  $k \in \mathbb{N}$ .

### 3. Main results

In this section, we investigate mainly the existence and uniqueness of global positive solutions, extinction and persistence in the mean of system (1), and the positive recurrence and ergodic property of solution is considered as well.

#### 3.1. Preliminaries

Denote  $\mathbb{R}_+ = [0, +\infty)$  and  $\mathbb{R}_+^n = \{(x_1, \dots, x_n) \in \mathbb{R}^n : x_i > 0, i = 1, 2, \dots, n\}$ , and  $|x| = \sqrt{\sum_{i=1}^n x_i^2}$ . For convenience, if  $\varphi(t)$  is a bounded and integrable function on  $\mathbb{R}_+$ , we define  $\tilde{\varphi} = \limsup_{t \rightarrow +\infty} \langle \varphi \rangle$  and  $\hat{\varphi} = \liminf_{t \rightarrow +\infty} \langle \varphi \rangle$ , here  $\langle \varphi \rangle = \frac{1}{T} \int_0^T \varphi(s) ds, T > 0$ .

Let  $\{\xi(t), t \geq 0\}$  be a right-continuous Markov chain on the probability space  $(\Omega, \mathcal{L}_t, \{\mathcal{L}_t\}_{t \geq 0}, \mathcal{P})$  with initial value  $\xi(0) = \xi_0$ , taking values in a finite-state space  $\mathbb{N} = \{1, 2, \dots, m\}$  with the transition rate matrix  $Q = (q_{ij})_{m \times m}$  of  $\xi(t)$  given by the following form:

$$\mathcal{P}\{\xi(t + \Delta t) = j | \xi(t) = i\} = \begin{cases} q_{ij} \Delta t + o(\Delta t), & i \neq j, \\ 1 + q_{ii} \Delta t + o(\Delta t), & i = j, \end{cases}$$

where  $o(\Delta t)$  is the infinitesimal of higher order,  $\Delta t > 0$  and  $q_{ij} \geq 0$  is the transition rate from  $i$  to  $j$  if  $i \neq j$  and  $q_{ii} = -\sum_{j \neq i} q_{ij}$ . Throughout this paper, we always suppose that Markov chain  $\xi(t), t \geq 0$  is irreducible, which means the system can switch from one regime to any other regimes, indicating that there exist finite numbers  $i_1, i_2, \dots, i_m \in \mathbb{N}$  such that  $q_{i_1, i_1} q_{i_1, i_2} \dots q_{i_m, i_m} > 0$ , for any  $i, j \in \mathbb{N}$ . Under this assumption, the Markov chain  $\xi(t), t \geq 0$  has a unique stationary distribution  $\pi = (\pi_1, \pi_2, \dots, \pi_m) \in \mathbb{R}^{1 \times m}$ , which can be determined by solving the linear equation  $\pi Q = 0$  subject to  $\sum_{k=1}^m \pi_k = 1$  and  $\pi_k > 0, \forall k \in \mathbb{N}$ . For any vector  $\phi = (\phi(1), \dots, \phi(m))^T$ , we define  $\phi^* = \max_{k \in \mathbb{N}} \{\phi(k)\}$  and  $\phi_* = \min_{k \in \mathbb{N}} \{\phi(k)\}$ .

Now, we introduce some fundamental results on the stationary distribution of stochastic differential equations under regime switching. Let  $(X(t), \xi(t))$  be the diffusion process described by the following equation:

$$\begin{cases} dX(t) = f(X(t), \xi(t)) dt + g(X(t), \xi(t)) dB(t), \\ X(0) = X_0 \in \mathbb{R}^n, \xi(0) = \xi_0 \in \mathbb{N}, \end{cases} \quad (2)$$

where  $B(\cdot)$  and  $\xi(\cdot)$  are the  $d$ -dimensional Brownian motion and right continuous Markov chain, respectively.  $f(\cdot, \cdot) : \mathbb{R}^n \times \mathbb{N} \rightarrow \mathbb{R}^n$  and  $g(\cdot, \cdot) : \mathbb{R}^n \times \mathbb{N} \rightarrow \mathbb{R}^{n \times d}$  satisfy  $g(X, k)g(X, k)^T = (d_{ij}(X, k))$ . For each  $k \in \mathbb{N}$  and any twice continuously differentiable function  $V(X, k)$  that are non-negative, we define a generator  $\mathcal{L}$ :

$$\mathcal{L}V(X, k) = \sum_{i=1}^n f_i(X, k) \frac{\partial V(X, k)}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^n d_{ij}(X, k) \frac{\partial^2 V(X, k)}{\partial x_i \partial x_j} + \Gamma(V, \cdot)(k),$$

where

$$\Gamma(X, \cdot)(k) = \sum_{h=1}^m q_{kh} \mathcal{L}(X, h) = \sum_{k \neq h, h \in \mathbb{N}} q_{kh} (\mathcal{L}(X, h) - \mathcal{L}(X, k)), h \in \mathbb{N}.$$

From Theorem 3.13 [43], the following lemma that describes a criterion for the ergodic stationary distribution of system (2) can be presented.

**Lemma 1.** *If the following conditions are satisfied:*

- (i) for  $i \neq j$ ,  $q_{ij} > 0, i, j \in \mathbb{N}$ ;
- (ii) for each  $k \in \mathbb{N}$ ,  $\lambda |\mu|^2 \leq \mu^T d_{ij}(X, k) \mu \leq \lambda^{-1} |\mu|^2$  for all  $\mu \in \mathbb{R}^n$  with some constant  $\lambda \in (0, 1]$  for all  $X \in \mathbb{R}^n$ ;
- (iii) there exists a bounded open subset  $\Xi$  of  $\mathbb{R}^n$  with a regular (i.e. smooth) boundary satisfying that, for each  $k \in \mathbb{N}$ , there exists a non-negative function  $V(\cdot, k) : \Xi^c \rightarrow \mathbb{R}$  such that  $V(\cdot, k)$  is twice continuously differentiable and that for some  $\varsigma > 0$ ,  $\mathcal{L}V(\cdot, k) \leq -\varsigma$  for any  $(X, k) \in \Xi^c \times \mathbb{N}$ .

Then system (2) is ergodic and positive recurrent. That is, there exists a unique stationary distribution  $\rho(\cdot, \cdot) = (\rho(\cdot, i) : i \in \mathbb{N})$ , and for any Borel measurable function  $f(\cdot, \cdot) : \mathbb{R}^n \times \mathbb{N} \rightarrow \mathbb{R}$  such that  $\sum_{k \in \mathbb{N}} \int_{\mathbb{R}^n} |f(X, k)| \rho(X, k) dx < \infty$ , we have

$$\mathcal{P} \left( \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t f(X(s), \xi(s)) ds = \sum_{k \in \mathbb{N}} \int_{\mathbb{R}^n} |f(X, k)| \rho(X, k) dx \right) = 1.$$

**Lemma 2.** [44] *Let  $Q = (q_{ij})$  be irreducible and  $\varpi, \eta \in \mathbb{R}^m$ , then the following linear system:*

$$Q\varpi = \eta$$

has a solution if and only if  $\pi\eta = 0$ .

### 3.2. Existence and uniqueness of global positive solutions

Before investigating the dynamics of system (1), we should first guarantee the existence of global positive solutions according to the biological interpretation. Therefore, in this subsection, we will discuss the existence of global positive solutions of system (1). The following result can be presented.

**Theorem 1.** *For any given initial value  $(P_1(0), P_2(0), Z(0), \xi(0)) \in \mathbb{R}_+^3 \times \mathbb{N}$ , system (1) exists a unique solution  $(P_1(t), P_2(t), Z(t), \xi(t))$  on  $\mathbb{R}_+$  and the positive solution will remain in  $\mathbb{R}_+^3 \times \mathbb{N}$  with probability one, namely  $(P_1(t), P_2(t), Z(t), \xi(t)) \in \mathbb{R}_+^3 \times \mathbb{N}$  for all  $t > 0$  almost surely.*

*Proof.* From the method of the Theorem 3.15 [45], obviously, we can verify that all the coefficients of system (1) are locally Lipschitz continuous and system (1) admits a unique local solution  $(P_1(t), P_2(t), Z(t), \xi(t))$  on  $[0, \tau_e)$  for any given initial value  $(P_1(0), P_2(0), Z(0), \xi(0)) \in \mathbb{R}_+^3 \times \mathbb{N}$ , where  $\tau_e$  represents the explosion time. In order to illustrate the solution is global, we only need to prove  $\tau_e = \infty$  a.s.. Let  $n_0 \geq 1$  enough large satisfying  $(P_1(0), P_2(0), Z(0), \xi(0)) \in [\frac{1}{n_0}, n_0]$ . For each integer  $n \geq n_0$ , we define the following stopping time:

$$\tau_n = \inf \{ t \in [0, \tau_e) : \max \{ (P_1(t), P_2(t), Z(t)) \} \leq \frac{1}{n} \text{ or } \min \{ (P_1(t), P_2(t), Z(t)) \} \geq n \},$$

and the set  $\inf \emptyset = \infty$  ( $\emptyset$  denotes the empty set). Obviously,  $\tau_n$  is increasing as  $n \rightarrow \infty$ . Let  $\tau_\infty = \lim_{n \rightarrow +\infty} \tau_n$ , then we can obtain that  $\tau_\infty \leq \tau_e$  a.s.. Thus, if we can prove that  $\tau_\infty = \infty$  a.s. in the following, then  $\tau_e = \infty$  and  $(P_1(0), P_2(0), Z(0), \xi(0)) \in \mathbb{R}_+^3 \times \mathbb{N}$  a.s. for all  $t \geq 0$ . In other words, to complete the proof, we only need to prove  $\tau_\infty = \infty$  a.s. Otherwise, the statement is false, then there exist two constants  $T > 0$  and  $\varepsilon \in (0, 1)$  such that  $\mathcal{P}\{\tau_\infty \leq T\} > \varepsilon$ . Hence, for all  $n \geq n_1$ , there exists an integer  $n_1 \geq n_0$  such that  $\mathcal{P}\{\tau_\infty \leq T\} \geq \varepsilon$ .

Define a  $C^2$ -function  $\bar{V} : \mathbb{R}_+^3 \rightarrow \mathbb{R}_+$  by

$$\bar{V}(P_1, P_2, Z, \xi) = \delta^*(P_1 - 1 - \log P_1) + (P_2 - 1 - \log P_2) + \alpha_*(Z - 1 - \log Z).$$

Obviously, the function  $\bar{V}(P_1, P_2, Z, \xi)$  is non-negative. Applying the Itô's formula to  $\bar{V}(P_1, P_2, Z, \xi)$ , we have

$$\begin{aligned} d\bar{V}(P_1, P_2, Z, \xi) &= \mathcal{L}\bar{V}(P_1, P_2, Z, \xi)dt + \delta^*\sigma_1(\xi)(P_1 - 1)dB_1(t) \\ &\quad + \sigma_2(\xi)(P_2 - 1)dB_2(t) + \alpha_*\sigma_3(\xi)(Z - 1)dB_3(t) \end{aligned}$$

where  $\mathcal{L}\bar{V} : \mathbb{R}_+^3 \rightarrow \mathbb{R}$  is defined by

$$\begin{aligned} &\mathcal{L}\bar{V}(P_1, P_2, Z, \xi) \\ &= \delta^*(P_1 - 1) \left( r_1(\xi) \left( 1 - \frac{P_1}{K_1(\xi)} \right) - a_1(\xi)P_2 - \frac{\alpha(\xi)Z}{1 + b_1(\xi)P_1 + b_2(\xi)P_2} \right) + \frac{\delta^*\sigma_1^2(\xi)}{2} \\ &\quad + (P_2 - 1) \left( r_2(\xi) \left( 1 - \frac{P_2}{K_2(\xi)} \right) - a_2(\xi)P_1 - \frac{\beta(\xi)Z}{1 + b_1(\xi)P_1 + b_2(\xi)P_2} \right) + \frac{\sigma_2^2(\xi)}{2} \\ &\quad + \alpha_*(Z - 1) \left( \frac{\delta(\xi)P_1 - \gamma(\xi)P_2}{1 + b_1(\xi)P_1 + b_2(\xi)P_2} - d(\xi) \right) + \frac{\alpha_*\sigma_3^2(\xi)}{2} \\ &\leq \left( \alpha_*d^* + \frac{\alpha_*\gamma^*}{(b_2)_*} - \delta^*(r_1)_* - (r_2)_* + \frac{1}{2}(\delta^*\sigma_1^* + \sigma_2^* + \alpha_*\sigma_3^*) \right) \\ &\quad + \left( \frac{\delta^*r_1^*}{(K_1)_*} + \delta^*r_1^* + a_2^* \right) P_1 - \frac{\delta^*(r_1)_*}{K_1^*} P_1^2 + \left( \frac{r_2^*}{(K_2)_*} + r_2^* + \delta^*a_1^* \right) P_2 - \frac{(r_2)_*}{K_2^*} P_2^2 \\ &\quad + |\delta^*\alpha^* + \beta^* - \alpha_*d_*|Z \\ &\leq M + |\delta^*\alpha^* + \beta^* - \alpha_*d_*|Z. \end{aligned}$$

where

$$\begin{aligned} M &= \left( \alpha_*d^* + \frac{\alpha_*\gamma^*}{(b_2)_*} - \delta^*(r_1)_* - (r_2)_* + \frac{1}{2}(\delta^*\sigma_1^* + \sigma_2^* + \alpha_*\sigma_3^*) \right) \\ &\quad + \max_{P_1 \in (0, +\infty)} \left\{ \left( \frac{\delta^*r_1^*}{(K_1)_*} + \delta^*r_1^* + a_2^* \right) P_1 - \frac{\delta^*(r_1)_*}{K_1^*} P_1^2 \right\} \\ &\quad + \max_{P_2 \in (0, +\infty)} \left\{ \left( \frac{r_2^*}{(K_2)_*} + r_2^* + \delta^*a_1^* \right) P_2 - \frac{(r_2)_*}{K_2^*} P_2^2 \right\}. \end{aligned}$$

Notice that

$$Z \leq 2(Z - 1 - \log Z) + 2 \log 2 \leq \frac{2}{\alpha_*} \bar{V}_1(P_1, P_2, Z, \xi) + 2 \log 2$$

for all  $Z > 0$ , then one can obtain that

$$\mathcal{L}\bar{V} \leq M + 2|\delta^*\alpha^* + \beta^* - \alpha_*d_*| \log 2 + \frac{2}{\alpha_*} |\delta^*\alpha^* + \beta^* - \alpha_*d_*| \bar{V} \leq \Upsilon(1 + \bar{V}),$$

where

$$\Upsilon = \max \left\{ M + 2|\delta^*\alpha^* + \beta^* - \alpha_*d_*| \log 2, \frac{2}{\alpha_*} |\delta^*\alpha^* + \beta^* - \alpha_*d_*| \right\}.$$

The remainder of the proof follows that in [46], here, we omit it. □

### 3.3. Extinction and persistence in the mean

Based on the Theorem 1 and the perspective of study of population dynamics, it is necessary and important to consider whether a population can sustain development or become extinct in the long time. Thus, we will discuss the persistence in the mean and extinction of system (1) in this subsection. For convenience of discussion in the following, we define

$$A = \sum_{i=1}^m \pi_i \left( r_1(i) - \frac{1}{2}\sigma_1^2(i) \right), B = \sum_{i=1}^m \pi_i \left( r_2(i) - \frac{1}{2}\sigma_2^2(i) \right), C = \sum_{i=1}^m \pi_i \left( d(i) + \frac{1}{2}\sigma_3^2(i) \right).$$

**Theorem 2.** Suppose that  $(P_1(t), P_2(t), Z(t), \xi(t))$  is the solution of system (1) with initial value  $(P_1(0), P_2(0), Z(0), \xi(0)) \in \mathbb{R}_+^3 \times \mathbb{N}$ , and if the conditions  $A < 0$  and  $B < 0$  hold, then system (1) tends to extinction.

*Proof.* We firstly consider the phytoplankton species  $P_1(t)$ . Applying the Itô's formula to the first equation of system (1), one can obtain that

$$d \ln P_1(t) = \left[ r_1(\xi(t)) \left( 1 - \frac{P_1(t)}{K_1(\xi(t))} \right) - a_1(\xi(t)) P_2(t) - \frac{\alpha(\xi(t)) Z(t)}{1 + b_1(\xi(t)) P_1(t) + b_2(\xi(t)) P_2(t)} - \frac{1}{2} \sigma_1^2(\xi(t)) \right] dt + \sigma_1(\xi(t)) dB_1(t),$$

Integrating the above from 0 to  $t$  and dividing  $t$  on both sides yield

$$\begin{aligned} \frac{1}{t} \ln \frac{P_1(t)}{P_1(0)} &= \left\langle r_1(\xi(t)) - \frac{1}{2} \sigma_1^2(\xi(t)) \right\rangle - \left\langle \frac{r_1(\xi(t))}{K_1(\xi(t))} P_1(t) \right\rangle - \langle a_1(\xi(t)) P_2(t) \rangle \\ &\quad - \left\langle \frac{\alpha(\xi(t))}{1 + b_1(\xi(t)) P_1(t) + b_2(\xi(t)) P_2(t)} Z(t) \right\rangle + \frac{M_1(t)}{t}, \end{aligned} \quad (3)$$

where

$$M_1(t) = \int_0^t \sigma_1(\xi(s)) dB_1(s).$$

By the strong law of large numbers for martingales [47] yields

$$\lim_{t \rightarrow +\infty} \frac{M_1(t)}{t} = 0 \quad a.s. \quad (4)$$

According to the ergodic theorem of Markov chain  $\xi(t)$  and (3), (4), we have

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln \frac{P_1(t)}{P_1(0)} \leq \lim_{t \rightarrow \infty} \langle r_1(\xi(t)) - \frac{1}{2} \sigma_1^2(\xi(t)) \rangle = \sum_{i=1}^m \pi_i \left( r_1(i) - \frac{1}{2} \sigma_1^2(i) \right) = A < 0, \quad a.s.$$

which implies that  $\lim_{t \rightarrow \infty} P_1(t) = 0 \quad a.s.$

For species  $P_2(t)$  and species  $Z(t)$ , similarly, we have

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln \frac{P_2(t)}{P_2(0)} \leq \lim_{t \rightarrow \infty} \langle r_2(\xi(t)) - \frac{1}{2} \sigma_2^2(\xi(t)) \rangle = \sum_{i=1}^m \pi_i \left( r_2(i) - \frac{1}{2} \sigma_2^2(i) \right) = B < 0, \quad a.s.$$

and

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln \frac{Z(t)}{Z(0)} \leq - \lim_{t \rightarrow \infty} \langle d(\xi(t)) + \frac{1}{2} \sigma_3^2(\xi(t)) \rangle = - \sum_{i=1}^m \pi_i \left( d(i) + \frac{1}{2} \sigma_3^2(i) \right) < 0, \quad a.s.$$

which imply that  $\lim_{t \rightarrow \infty} P_2(t) = 0 \quad a.s.$  and  $\lim_{t \rightarrow \infty} Z(t) = 0 \quad a.s.$ , respectively.  $\square$

**Theorem 3.** Suppose that  $(P_1(t), P_2(t), Z(t), \xi(t))$  is the solution of system (1) with initial value  $P_1(0), P_2(0), Z(0), \xi(0) \in \mathbb{R}_+^3 \times \mathbb{N}$ , and the following conditions

$$A - \frac{a_1^*(K_2)^*}{r_2^*} B > 0, B - \frac{a_2^*(K_1)^*}{r_1^*} A > 0, \frac{\delta^*(K_1)^*}{r_1^*} A - C < 0$$

hold, then the species  $P_1(t)$  and  $P_2(t)$  are persistence in the mean, while the species  $Z(t)$  undergoes extinction.

*Proof.* From the Lemma 4 [48, 49], we can obtain that

$$\langle \widetilde{P_1} \rangle \leq \frac{(K_1)^*}{r_1^*} A, \quad \langle \widetilde{P_2} \rangle \leq \frac{(K_2)^*}{r_2^*} B \quad (5)$$

We first consider the species  $Z(t)$ . Applying the Itô's formula to the third equation of system (1) and then integrating from 0 to  $t$ , we have

$$\begin{aligned} \frac{1}{t} \ln \frac{Z(t)}{Z(0)} = & - \left\langle d(\xi(t)) + \frac{1}{2} \sigma_3^2(\xi(t)) \right\rangle + \left\langle \frac{\delta(\xi(t))}{1 + b_1(\xi(t))P_1(t) + b_2(\xi(t))P_2(t)} P_1(t) \right\rangle \\ & - \left\langle \frac{\gamma(\xi(t))}{1 + b_1(\xi(t))P_1(t) + b_2(\xi(t))P_2(t)} P_2(t) \right\rangle + \frac{M_3(t)}{t}, \end{aligned} \quad (6)$$

Taking upper limit on both sides of (6) and using the strong law of large number of local martingale yields

$$\left[ \frac{1}{t} \ln \frac{Z(t)}{Z(0)} \right] \leq \frac{\delta^*(K_1)_*}{r_1^*} A - C < 0, \text{ a.s.}$$

which implies  $\lim_{t \rightarrow \infty} Z(t) = 0$ , namely the species  $Z(t)$  is extinctive.

Now, we consider the species  $P_1(t)$ . By (5) and integrating (3) on the interval  $[0, t]$  and making some estimations, one can obtain that

$$\frac{1}{t} \ln P_1(t) \geq \frac{1}{t} \ln P_1(0) + A - \frac{r_1^*}{(K_1)_*} \langle P_1 \rangle - \frac{a_1^*(K_2)_*}{r_2^*} B - \alpha^* \tilde{Z} + \frac{M_1(t)}{t}. \quad (7)$$

In addition, since the fact that

$$\lim_{t \rightarrow +\infty} \frac{\ln P_1(0)}{t} = \lim_{t \rightarrow +\infty} \frac{M_1(t)}{t} = 0,$$

and from the definition of  $A$  and  $B$ , then we can obtain that for arbitrary  $\epsilon_2 > 0$ , there exists a constant  $T_2 > 0$  such that

$$\langle P_2 \rangle \leq \frac{(K_2)_*}{r_2^*} B + \frac{\epsilon_2}{2\alpha_1^*}, \quad \langle Z \rangle \leq \frac{\epsilon_2}{2\alpha^*}, \quad \langle r_1(\xi(t)) - \frac{1}{2} \sigma_1^2(\xi(t)) \rangle \geq A - \frac{\epsilon_2}{3}, \quad \frac{\ln P_1(0)}{t} \geq -\frac{\epsilon_2}{3}, \quad \frac{M_1(t)}{t} \geq -\frac{\epsilon_2}{3}.$$

Substituting the above inequalities into (7) and for all  $t \geq T_2$ , we have

$$\frac{1}{t} \ln \frac{P_1(t)}{P_1(0)} \geq \left( A - \frac{a_1^*(K_2)_*}{r_2^*} B \right) t - \frac{r_1^*}{(K_1)_*} \langle P_1 \rangle + \frac{M_1(t)}{t},$$

From (5), one can obtain that

$$\widehat{P_1(t)} \geq \frac{(K_1)_*}{r_1^*} \left( A - \frac{a_1^*(K_2)_*}{r_2^*} B \right) > 0.$$

For the species  $P_2(t)$ , the same analysis to the species  $P_1(t)$ , we have

$$\widehat{P_2(t)} \geq \frac{(K_2)_*}{r_2^*} \left( B - \frac{a_2^*(K_1)_*}{r_1^*} A \right) > 0.$$

That is, the species  $P_2(t)$  is persistence in the mean. □

### 3.4. Stationary distribution and ergodic property

In this subsection, by constructing a suitable Lyapunov function and using Khasminskii's method [50], some sufficient conditions for the positive recurrence and the existence of uniqueness of stationary distribution are obtained.

**Theorem 4.** *If the following condition*

$$\Pi = \sum_{\xi=1}^m \pi_{\xi} \Lambda_{\xi} > 0$$



holds, where

$$\Lambda_\xi = \left( r_1(\xi) - \frac{1}{2}\sigma_1^2(\xi) \right) + \left( r_2(\xi) - \frac{1}{2}\sigma_2^2(\xi) \right) - \left( d(\xi) + \frac{1}{2}\sigma_3^2(\xi) \right) - \frac{\gamma(\xi)}{b_2(\xi)} \\ - \frac{\alpha(\xi)d(\xi)K_1(\xi) \left[ \frac{r_1(\xi)\beta(\xi)(\alpha(\xi)+\beta(\xi)+1)}{\alpha(\xi)d(\xi)} + \frac{r_1(\xi)}{K_1(\xi)} + a_2(\xi) \right]^2}{4r_1(\xi)\beta(\xi)(\alpha(\xi)+\beta(\xi)+1)} - \frac{K_2(\xi) \left[ r_2(\xi) + \frac{r_2(\xi)}{K_2(\xi)} + a_1(\xi) \right]^2}{4r_2(\xi)}.$$

Then for any given initial value  $(P_1(0), P_2(0), Z(0), \xi(0)) \in \mathbb{R}_+^3 \times \mathbb{N}$ , system (1) admits a unique ergodic stationary distribution.

*Proof.* In order to prove this theorem, we only need to prove all the three conditions of the Lemma 1 one by one. Obviously, the condition (i) of the Lemma 1 is satisfied by the assumption  $q_{ij} > 0$  for any  $i \neq j$ ,  $i, j \in \mathbb{N}$  in the subsection 3.1. On the other hand, it is easy to verify that the diffusion matrix  $d_{ij}(X, k) = \text{diag}\{\sigma_1^2(\xi), \sigma_2^2(\xi), \sigma_3^2(\xi)\}$  of system (1) is positive definite, which implies that the condition (ii) of the Lemma 1 holds.

In the following, we prove the condition (iii) of the Lemma 1. We construct a  $C^2$ -function  $V : \mathbb{R}_+^3 \times \mathbb{N} \rightarrow \mathbb{R}$  as follows:

$$V(P_1, P_2, Z, \xi) = (c_1P_1 + P_2 + c_2Z) - (\ln P_1 + 1 + \ln c_1 + \ln P_2 + 1 + \ln Z + 1 + \ln c_2) \\ + (\varpi_\xi + |\varpi|) \triangleq V_1(P_1, P_2, Z, \xi) + V_2(P_1, P_2, Z, \xi) + V_3(\xi),$$

where  $c_1$  and  $c_2$  are positive constants,  $\varpi = (\varpi_1, \varpi_2, \dots, \varpi_m)^T$ ,  $|\varpi| = \sqrt{\varpi_1^2 + \dots + \varpi_m^2}$  and  $\varpi_\xi$  ( $\xi \in \mathbb{N}$ ) will be determined later and the reason for  $|\varpi|$  being here is to make  $\varpi_\xi + |\varpi|$  non-negative. Obviously, the function  $V(P_1, P_2, Z, \xi)$  is non-negative. Applying the Itô's formula to  $V_1$  and  $V_2$ , we have

$$\mathcal{L}V_1(P_1, P_2, Z, \xi) = c_1r_1(\xi)P_1 - \frac{c_1r_1(\xi)}{K_1(\xi)}P_1^2 - c_1a_1(\xi)P_1P_2 - \frac{c_1\alpha(\xi)P_1Z}{1+b_1(\xi)P_1+b_2(\xi)P_2} \\ + r_2(\xi)P_2 - \frac{r_2(\xi)}{K_2(\xi)}P_2^2 - a_2(\xi)P_1P_2 - \frac{\beta(\xi)P_2Z}{1+b_1(\xi)P_1+b_2(\xi)P_2} \\ + \frac{c_2\delta(\xi)P_1Z}{1+b_1(\xi)P_1+b_2(\xi)P_2} - \frac{c_2\gamma(\xi)P_2Z}{1+b_1(\xi)P_1+b_2(\xi)P_2} - c_2d(\xi)Z \\ \leq -\frac{c_1r_1(\xi)}{K_1(\xi)}P_1^2 + c_1r_1(\xi)P_1 - \frac{r_2(\xi)}{K_2(\xi)}P_2^2 + r_2(\xi)P_2 - c_2d(\xi)Z \\ - \frac{[c_1\alpha(\xi) - c_2\delta(\xi)]P_1Z}{1+b_1(\xi)P_1+b_2(\xi)P_2},$$

and

$$\mathcal{L}V_2(P_1, P_2, Z, \xi) = -r_1(\xi) + \frac{r_1(\xi)}{K_1(\xi)}P_1 + a_1(\xi)P_2 + \frac{\alpha(\xi)Z}{1+b_1(\xi)P_1+b_2(\xi)P_2} + \frac{\sigma_1^2(\xi)}{2} \\ - r_2(\xi) + \frac{r_2(\xi)}{K_2(\xi)}P_2 + a_2(\xi)P_1 + \frac{\beta(\xi)Z}{1+b_1(\xi)P_1+b_2(\xi)P_2} + \frac{\sigma_2^2(\xi)}{2} \\ - \frac{\delta(\xi)P_1 - \gamma(\xi)P_2}{1+b_1(\xi)P_1+b_2(\xi)P_2} + d(\xi) + \frac{\sigma_3^2(\xi)}{2} \\ \leq \left[ \frac{r_1(\xi)}{K_1(\xi)} + a_2(\xi) \right] P_1 + \left[ \frac{r_2(\xi)}{K_2(\xi)} + a_1(\xi) \right] P_2 + [\alpha(\xi) + \beta(\xi)]Z \\ + \left[ \frac{\gamma(\xi)}{b_2(\xi)} - r_1(\xi) - r_2(\xi) + d(\xi) + \frac{\sigma_1^2(\xi) + \sigma_2^2(\xi) + \sigma_3^2(\xi)}{2} \right].$$

Thus,

$$\begin{aligned} & \mathcal{L}V_1(P_1, P_2, Z, \xi) + \mathcal{L}V_2(P_1, P_2, Z, \xi) \\ & \leq -\frac{c_1 r_1(\xi)}{K_1(\xi)} P_1^2 + \left[ c_1 r_1(\xi) + \frac{r_1(\xi)}{K_1(\xi)} + a_2(\xi) \right] P_1 - \frac{r_2(\xi)}{K_2(\xi)} P_2^2 \\ & \quad + \left[ r_2(\xi) + \frac{r_2(\xi)}{K_2(\xi)} + a_1(\xi) \right] P_2 + [\alpha(\xi) + \beta(\xi) - c_2 d(\xi)] Z - \frac{[c_1 \alpha(\xi) - c_2 \delta(\xi)] P_1 Z}{1 + b_1(\xi) P_1 + b_2(\xi) P_2} \\ & \quad + \left[ \frac{\gamma(\xi)}{b_2(\xi)} - r_1(\xi) - r_2(\xi) + d(\xi) + \frac{\sigma_1^2(\xi) + \sigma_2^2(\xi) + \sigma_3^2(\xi)}{2} \right]. \end{aligned}$$

Choosing  $c_1 = \frac{\beta^*}{\alpha_*} c_2$  and  $c_2 = \frac{\alpha^* + \beta^* + 1}{d_*}$ , one can obtain

$$\begin{aligned} & \mathcal{L}V_1(P_1, P_2, Z, \xi) + \mathcal{L}V_2(P_1, P_2, Z, \xi) \\ & \leq -\frac{\beta(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1)}{\alpha(\xi) d(\xi) K_1(\xi)} \left[ P_1 - \frac{\beta(\xi) K_1(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1)}{2[\beta(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1)]} \right. \\ & \quad \left. + \frac{\alpha(\xi) d(\xi)(r_1(\xi) + K_1(\xi) a_2(\xi))}{2[\beta(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1)]} \right]^2 - \frac{r_2(\xi)}{K_2(\xi)} \left( P_2 - \frac{r_2(\xi) + K_2(\xi)(r_2(\xi) + a_1(\xi))}{2r_2(\xi)} \right)^2 \\ & \quad + \frac{\alpha(\xi) d(\xi) K_1(\xi) \left[ \frac{r_1(\xi) \beta(\xi)(\alpha(\xi) + \beta(\xi) + 1)}{\alpha(\xi) d(\xi)} + \frac{r_1(\xi)}{K_1(\xi)} + a_2(\xi) \right]^2}{4r_1(\xi) \beta(\xi)(\alpha(\xi) + \beta(\xi) + 1)} + \frac{K_2(\xi) \left[ r_2(\xi) + \frac{r_2(\xi)}{K_2(\xi)} + a_1(\xi) \right]^2}{4r_2(\xi)} \\ & \quad + \left[ \frac{\gamma(\xi)}{b_2(\xi)} - \left( r_1(\xi) - \frac{1}{2} \sigma_1^2(\xi) \right) - \left( r_2(\xi) - \frac{1}{2} \sigma_2^2(\xi) \right) + \left( d(\xi) + \frac{1}{2} \sigma_3^2(\xi) \right) \right]. \end{aligned}$$

Moreover,

$$\mathcal{L}V_3(\xi) = \sum_{h=1}^m q_{\xi h} \varpi_h.$$

Note that

$$\sum_{\xi=1}^m \pi_{\xi} = 1, \quad \pi[\Lambda - (\pi\Lambda)I_m] = 0,$$

where  $\Lambda = (\Lambda_1, \Lambda_2, \dots, \Lambda_m)^T$ ,  $I_m = (1, 1, \dots, 1)^T \in \mathbb{R}^m$ . By the Lemma 2, we can obtain that the equation  $Q\varpi = \Lambda - (\pi\Lambda)I_m$  has a solution  $\varpi = (\varpi_1, \varpi_2, \dots, \varpi_m)^T \in \mathbb{R}^m$ , which implies

$$-\Lambda_{\xi} + \sum_{h=1}^m q_{\xi h} \varpi_h = -\sum_{h=1}^m \pi_h \Lambda_h = -\Pi.$$

Then

$$\mathcal{L}V(P_1, P_2, Z, \xi) \leq -\Pi + f(P_1) + g(P_2),$$

where

$$\begin{aligned} \Pi &= \sum_{\xi=1}^m \pi_{\xi} \left\{ \left( r_1(\xi) - \frac{1}{2} \sigma_1^2(\xi) \right) + \left( r_2(\xi) - \frac{1}{2} \sigma_2^2(\xi) \right) + \left( d(\xi) + \frac{1}{2} \sigma_3^2(\xi) \right) - \frac{\gamma(\xi)}{b_2(\xi)} \right. \\ & \quad \left. - \frac{\alpha(\xi) d(\xi) K_1(\xi) \left[ \frac{r_1(\xi) \beta(\xi)(\alpha(\xi) + \beta(\xi) + 1)}{\alpha(\xi) d(\xi)} + \frac{r_1(\xi)}{K_1(\xi)} + a_2(\xi) \right]^2}{4r_1(\xi) \beta(\xi)(\alpha(\xi) + \beta(\xi) + 1)} - \frac{K_2(\xi) \left[ r_2(\xi) + \frac{r_2(\xi)}{K_2(\xi)} + a_1(\xi) \right]^2}{4r_2(\xi)} \right\}, \\ f(P_1) &= -\frac{\beta(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1)}{\alpha(\xi) d(\xi) K_1(\xi)} \left[ P_1 \right. \\ & \quad \left. - \frac{\beta(\xi) K_1(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1) + \alpha(\xi) d(\xi)(r_1(\xi) + K_1(\xi) a_2(\xi))}{2[\beta(\xi) r_1(\xi)(\alpha(\xi) + \beta(\xi) + 1)]} \right]^2 \end{aligned}$$

and

$$g(P_2) = -\frac{r_2(\xi)}{K_2(\xi)} \left( P_2 - \frac{r_2(\xi) + K_2(\xi)(r_2(\xi) + a_1(\xi))}{2r_2(\xi)} \right)^2 + \frac{K_2(\xi) \left[ r_2(\xi) + \frac{r_2(\xi)}{K_2(\xi)} + a_1(\xi) \right]^2}{4r_2(\xi)}.$$

Thus,

$$\Pi + f(P_1) + g(P_2) \leq \begin{cases} -\Pi + f(P_1) + g^*(P_2) \rightarrow -\infty, & \text{as } P_1 \rightarrow +\infty, \\ -\Pi + f(P_1) + g^*(P_2) \leq -\Pi, & \text{as } P_1 \rightarrow 0^+, \\ -\Pi + f^*(P_1) + g(P_2) \rightarrow -\infty, & \text{as } P_2 \rightarrow +\infty, \\ -\Pi + f^*(P_1) + g(P_2) \leq -\Pi, & \text{as } P_2 \rightarrow 0^+, \\ -\Pi + f^*(P_1) + g^*(P_2) \leq -\Pi, & \text{as } Z \rightarrow 0^+ \text{ or } Z \rightarrow +\infty. \end{cases}$$

Therefore, we can take  $\varepsilon > 0$  sufficiently small such that for any  $(P_1, P_2, Z, \xi) \in \mathcal{F}_\varepsilon^c \times \mathbb{N}$ ,

$$\mathcal{LVP}_1, P_2, Z, \xi \leq -1,$$

where  $\mathcal{F}_\varepsilon = \left( \varepsilon, \frac{1}{\varepsilon} \right) \times \left( \varepsilon, \frac{1}{\varepsilon} \right) \times \left( \varepsilon, \frac{1}{\varepsilon} \right)$ . Hence, the condition (iii) of the Lemma 1 is verified. It follows from the Lemma 1 that system (1) admits a unique ergodic stationary distribution.  $\square$

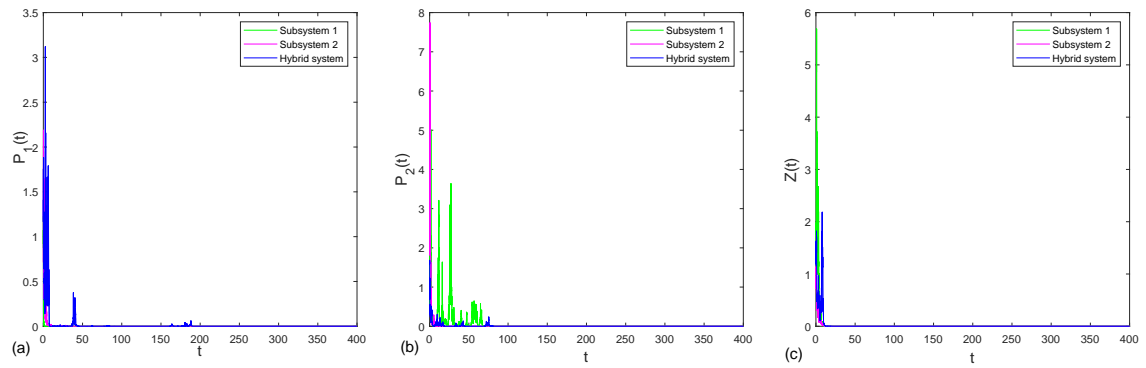
## 4. Numerical simulation

In this section, based on the Milstein's Higher Order Method [51], some numerical simulations are carried out to study the effects of the white noise, regime switching and TPP on the dynamics of system (1). In these numerical simulations, unless otherwise specified, we always assume that the right-continuous Markov chain  $\xi(t)$  takes values on state space  $\mathbb{N} = \{1, 2\}$  and the values of parameters are listed in the table 1. In order to study how the white noise, regime switching and TPP affect the dynamics of system (1), we firstly consider that there is no regime switching in system (1). Fixed  $(\gamma(1), \gamma(2)) = (0.2, 0.3)$  and choose  $(\sigma_1(1), \sigma_1(2)) = (1.5, 1.5)$ ,  $(\sigma_2(1), \sigma_2(2)) = (1.3, 1.4)$ ,  $(\sigma_3(1), \sigma_3(2)) = (0.7, 0.8)$ . By direct computation, the sufficient conditions of the extinction for both Subsystems are easily to verify. Thus, all the species of Subsystems 1 and 2 are extinct (see Figure 1). Furthermore, suppose that the generator  $Q$  of the Markov chain  $\xi(t)$  is  $\begin{pmatrix} -\frac{1}{8} & \frac{1}{8} \\ \frac{1}{12} & -\frac{1}{12} \end{pmatrix}$ , by the irreducible property, we can obtain that the stationary distribution of  $\xi(t)$  is  $\pi = (0.4, 0.6)$ , which satisfies the conditions of Theorem 2, then all the species of system (1) undergo extinction (see Figure 1). This result suggests that the regime switching can not change the extinction behavior of system (1) in this case, that is, system (1) is extinct if both two Subsystems die out simultaneously.

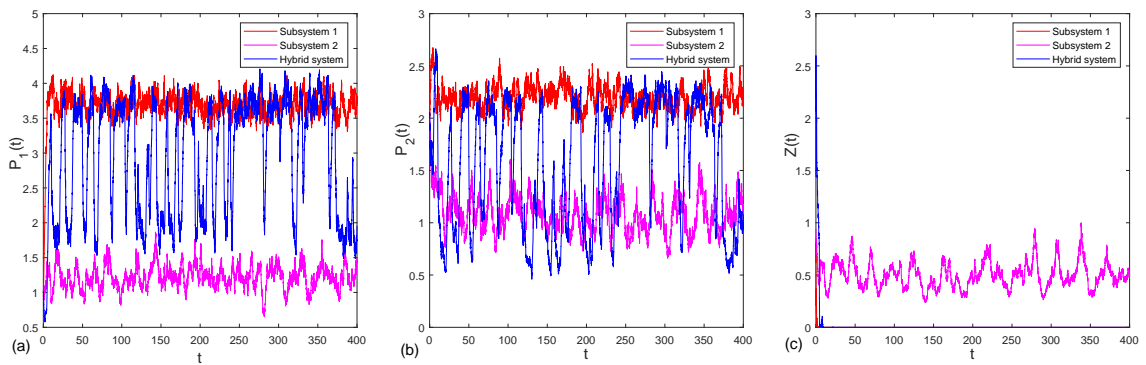
**Table 1** Parameter values

States	Coefficients											
	$r_1$	$K_1$	$r_2$	$K_2$	$a_1$	$a_2$	$\alpha$	$\beta$	$b_1$	$b_2$	$\delta$	$d$
1	0.8	1.8	0.65	2.5	0.01	0.28	0.85	0.08	0.2	0.5	0.6	0.28
2	0.85	3.8	0.8	3.5	0.01	0.08	0.95	0.01	0.1	0.8	0.75	0.2

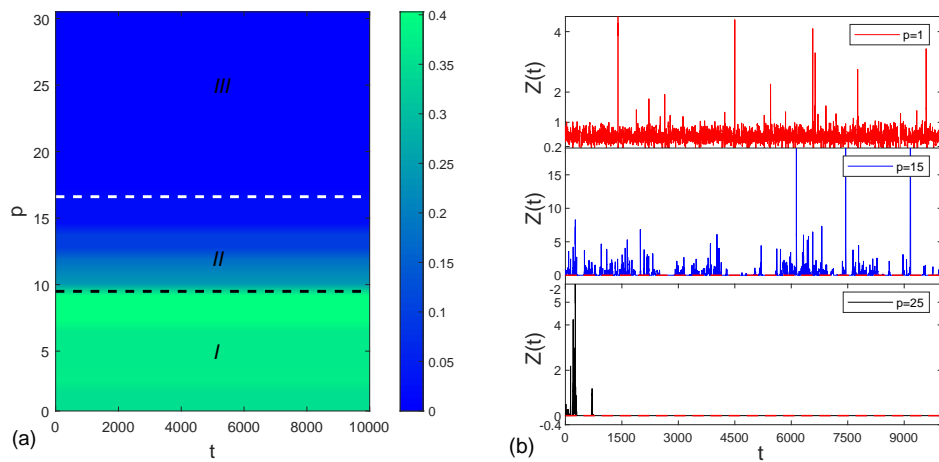
On the other hand, to illustrate the effect of regime switching on the dynamics of system (1), we choose  $(\sigma_1(1), \sigma_1(2)) = (0.1, 0.05)$ ,  $(\sigma_2(1), \sigma_2(2)) = (0.1, 0.05)$  and  $(\sigma_3(1), \sigma_3(2)) = (0.1, 1.8)$  and all other parameters remain unchanged. By a simple computation, we can easily verify the conditions of Theorem 3, which follows that both species  $P_1(t)$  and  $P_2(t)$  of system (1) are persistence in the mean, while species  $Z(t)$  tends to extinction, as shown in Figure 2. From Figure 2, it is clear that Subsystems 1 and 2 have different persistence-extinction behaviors and system (1) can switch from one Subsystem to another Subsystem due to the regime shift, which implies that regime switching can balance the density of the population under different regimes. Significantly, it should be pointed out that the zooplankton of system (1) is extinct due to the extinction of zooplankton in Subsystem 1. This indicates that the regime switching may not change persistence-extinction behaviors in



**Figure 1.** (a), (b) and (c) denote the solution trajectories of  $P_1(t)$ ,  $P_2(t)$  and  $Z(t)$  for system (1) with  $(\gamma(1), \gamma(2)) = (0.2, 0.3)$  and  $(\sigma_1(1), \sigma_1(2)) = (1.5, 1.5)$ ,  $(\sigma_2(1), \sigma_2(2)) = (1.3, 1.4)$ ,  $(\sigma_3(1), \sigma_3(2)) = (0.7, 0.8)$ , respectively. Here the initial value is  $(P_1(0), P_2(0), Z(0)) = (1, 2, 1)$ .

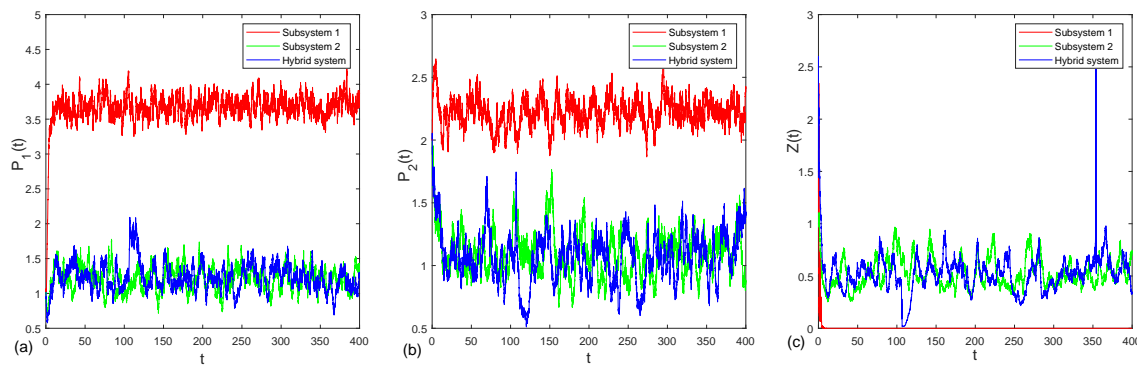


**Figure 2.** (a), (b) and (c) denote the solution trajectories of  $P_1(t)$ ,  $P_2(t)$  and  $Z(t)$  for system (1) with  $(\gamma(1), \gamma(2)) = (0.2, 0.3)$  and  $(\sigma_1(1), \sigma_1(2)) = (0.1, 0.05)$ ,  $(\sigma_2(1), \sigma_2(2)) = (0.1, 0.05)$ ,  $(\sigma_3(1), \sigma_3(2)) = (0.1, 1.8)$ , respectively. Here the initial value is  $(P_1(0), P_2(0), Z(0)) = (1, 2, 1)$ .



**Figure 3.** The effect of regime switching on the stochastic behaviors of zooplankton species  $Z(t)$  for system (1). (a) denotes the stochastic behaviors between extinction and persistence in the mean of zooplankton species  $Z(t)$  for system (1) with different values of  $p$  in different areas of  $I, II, III$  and other parameters as in Figure 2; (b) denotes the solution trajectories of zooplankton species  $Z(t)$  with respect to Figure 3(a) for  $p = 1$ ,  $p = 15$  and  $p = 25$ , respectively.

this case. However, changing the generator  $Q$  to  $Q = \begin{pmatrix} -\frac{p}{100} & \frac{p}{100} \\ \frac{100-p}{100} & -\frac{100-p}{100} \end{pmatrix}$  by controlling the value of  $p$ , it is easy to obtain

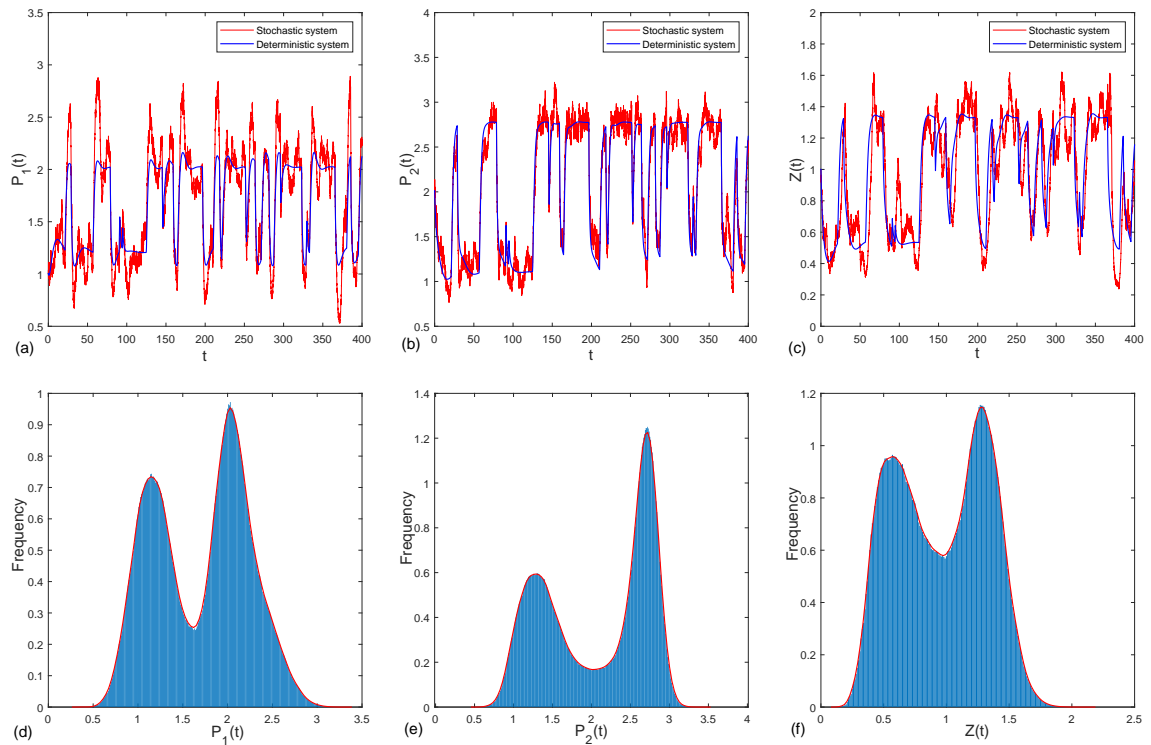


**Figure 4.** (a), (b) and (c) denote the solution trajectories of  $P_1(t)$ ,  $P_2(t)$  and  $Z(t)$  for system (1) with  $(\gamma(1), \gamma(2)) = (0.2, 0.3)$  and  $(\sigma_1(1), \sigma_1(2)) = (0.1, 0.05)$ ,  $(\sigma_2(1), \sigma_2(2)) = (0.1, 0.05)$ ,  $(\sigma_3(1), \sigma_3(2)) = (0.1, 1.8)$ , respectively. Here the initial value is  $(P_1(0), P_2(0), Z(0)) = (1, 2, 1)$ .

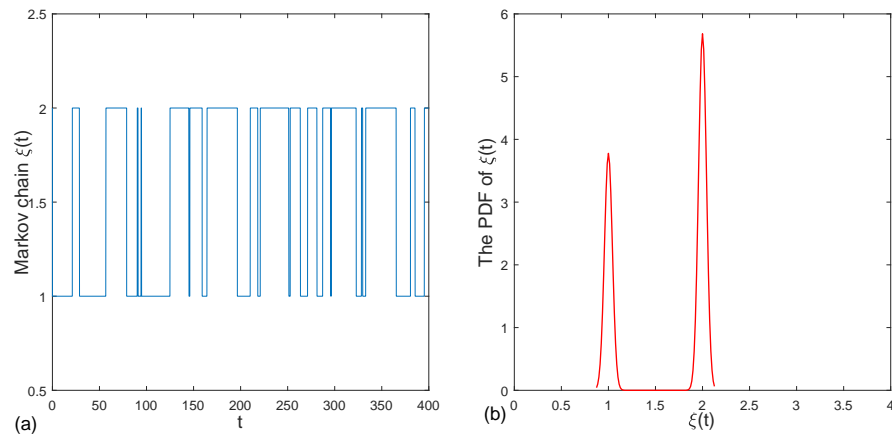
that the stationary distribution of  $\xi(t)$  is  $\pi = (\pi_1, \pi_2) = \left( \frac{100-p}{100}, \frac{p}{100} \right)$  ( $0 \leq p \leq 100$ ). From Figure 3 (a), one can observe that with the increasing value of  $p$ , the dynamical behaviors of species  $Z(t)$  change from persistence in the mean to extinction in different areas of *I*, *II* and *III* and Figure 3 (b) depicts the dynamical behaviors of species  $Z(t)$  with respect to Figure 3 (a) for  $p = 1$ ,  $p = 15$  and  $p = 25$ , respectively. Taking  $p = 1$  for example, we can see from the Figure 4 that system (1) becomes persistence in the mean, whereas other Subsystems remain unchanged and almost all of the sample trajectories of system (1) are in that of Subsystem 2 due to  $\pi_1 > \pi_2$ . This means that plankton species can choose a better living environmental state to survive due to Markov chain. For the case of  $p = 25$ , we can obtain that the zooplankton of system (1) becomes extinction again (Figures are not given due to the similarity to Figure 4). Thus, under the effect of the regime switching, we can obtain the result from Figure 3 and Figure 4 that even if one population undergoes extinction in one state, it will become persistence in the mean in another state because of its staying longer in a better living environmental state. Therefore, it can be asserted that the regime switching can change the persistence-extinction behaviors of system (1) and the distribution of Markov chain  $\xi(t)$  is beneficial to the survival of plankton.

Next, the impact of the white noise on the dynamics of system (1) will be shown. From Figure 5, it can be seen that NTP, TPP and zooplankton populations can coexist at a relatively stable state when the intensities of white noise are comparatively small  $((\sigma_1(1), \sigma_1(2)) = (0.1, 0.05)$ ,  $(\sigma_2(1), \sigma_2(2)) = (0.1, 0.05)$ ,  $(\sigma_3(1), \sigma_3(2)) = (0.1, 0.05))$  and all other parameters as in Figure 1. Actually, according to Theorem 4, system (1) has a unique ergodic stationary distribution (see Figure 5), which are consistent with our numerical analysis. Moreover, it is clear to see from Figure 4 (a),(b),(c) that white noise keeps the stochastic processes  $P_1(t)$ ,  $P_2(t)$  and  $Z(t)$  moving up and down randomly and the solution (the red lines) of system (1) fluctuates in a small neighborhood of that (the blue lines) of its corresponding deterministic system. Thus, we can obtain that white noise can affect the distribution of phytoplankton and zooplankton populations. That is, white noise can significantly affect the dynamic evolution mechanism of plankton populations. Significantly, we can observe from Figure 4 (d),(e),(f) that the probability density functions of NTP, TPP and zooplankton have two wave curves that are corresponding to the two states  $\aleph = \{1, 2\}$  of the Markov switching, respectively. Comparing Figures 1, 2 and 5, it is obvious to find that the high density of white noise can accelerate the extinction of the plankton populations and be advantageous to the rapid disappearance of planktonic blooms, which may help us to control the density of plankton populations in real aquatic ecosystems. Therefore, it can be asserted that the plankton systems incorporating white noise can better simulate planktonic blooms than its corresponding deterministic counterparts. Figure 6 describes that system (1) switches from one state  $\xi = 1$  to another state  $\xi = 2$  by the law of Markov chain  $\xi(t)$  over time.

Finally, the influence of the toxin liberation rate produced by TPP under the effects of the white noise and regime switching is studied as well. By choosing  $(\gamma(1), \gamma(2)) = (0.5, 0.55)$ , and a simple computation shows that  $\Pi > 0$ . According to the Theorem 4, we know that system (1) has a unique stationary distribution and the probability density functions of NTP, TPP and zooplankton populations have two wave curves due to the regime shift (see Figure 7). Comparing Figure 5 and Figure 7, we can observe that the peak values of the probability density functions for  $P_1(t)$ ,  $P_2(t)$  and  $Z(t)$  of system (1) are higher than that in the earlier case  $((\gamma(1), \gamma(2)) = (0.2, 0.3))$ . Moreover, we can also observe that with the increasing value of  $\gamma$ , the mean values of  $P_1(t)$  and  $P_2(t)$  of system (1) are getting larger, while that of  $Z(t)$  is becoming smaller. Therefore, it



**Figure 5.** (a), (b) and (c) denote the solution trajectories of system (1) and its corresponding deterministic counterparts, and (d), (e) and (f) denote the density function diagrams of system (1) with  $(\sigma_1(1), \sigma_1(2)) = (0.1, 0.05)$ ,  $(\sigma_2(1), \sigma_2(2)) = (0.1, 0.05)$ ,  $(\sigma_3(1), \sigma_3(2)) = (0.1, 0.05)$ ,  $(\gamma(1), \gamma(2)) = (0.2, 0.3)$  and initial condition  $(P_1(0) = 1, P_2(0) = 2, Z(0) = 1, \xi(0) = 1)$  in regime  $\xi = 1, \xi = 2$ , respectively and other parameters are the same with Figure 1.

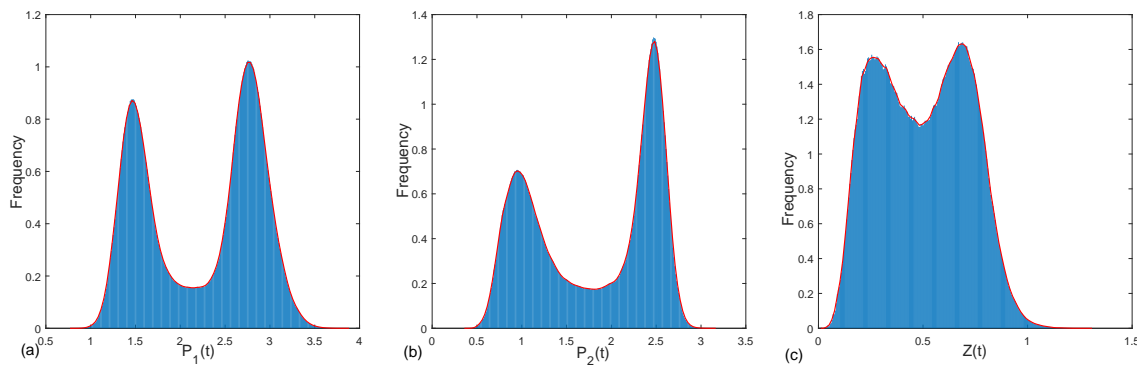


**Figure 6.** (a) denotes the movement of Markov chain in the state space  $\aleph = \{1, 2\}$  over time. (b) denotes the probability density function (PDF) of  $\xi(t)$ .

is obliged to be stressed that the introduce of TPP can be beneficial to the persistence in the mean of three species through the termination of planktonic blooms and may be acted as a controlling agent to control planktoic blooms.

## 5. Conclusions

The occurrences of harmful phytoplankton blooms have been reported globally with an increasing frequency in the past decades [2], and TPP are among the contributors in these blooms [2, 52, 53]. Moreover, plankton populations in the real



**Figure 7.** (a), (b) and (c) denote the histograms of probability density function for  $P_1(t)$ ,  $P_2(t)$  and  $Z(t)$  of system (1) with  $(\gamma(1), \gamma(2)) = (0.5, 0.55)$ , respectively and other parameters are the same with Figure 5.

aquatic ecosystems often fluctuate unpredictably because of the predictability of the environmental stochasticity, which plays an important role in the ecosystems [54]. In order to better understand the effects of environmental fluctuations and TPP on the dynamics of plankton systems, in this paper, therefore, we propose a stochastic phytoplankton-toxic producing phytoplankton-zooplankton system with Beddington-DeAngelis functional response, which incorporates with white noise and regime switching, and study how these factors affect the dynamics of system (1) analytically and numerically. We firstly investigate the existence and uniqueness of global positive solutions, and then derive some sufficient conditions for the extinction and persistence in the mean of system (1). To prove the existence of the stationary distribution, the theory of Khasminskii [50] for periodic Markov process and a method based on constructing a Lyapunov function are employed. Numerical analysis illustrates our theoretical results and further indicates that the white noise, regime switching and TPP play an important role in controlling planktonic blooms as follows:

- (i) Regime switching plays an important role in the balance of the different survival states of plankton populations. On the one hand, Subsystem 1, Subsystem 2 and system (1) have the same persistence-extinction behaviors. Actually, the regime switching can not change the persistence-extinction behaviors of these systems (see Figures 1 and 5), which means that system (1) still becomes persistence in the mean (or extinction) if Subsystems 1 and 2 becomes persistence in the mean (or extinction). On the other hand, the persistence-extinction behaviors of system (1) rely heavily on that of Subsystem 1 and Subsystem 2 due to the role of regime shifts. In the case of Subsystem 1 is persistence in the mean and Subsystem 2 dies out, then system (1) will tend to extinction (see Figure 2). However, the system (1) becomes persistence in the mean although one Subsystem is extinct by controlling the value of  $p$  (see Figure 4). Thus, the presence of regime switching in the stochastic system can change the survival of plankton populations and reduce the risk of extinction. Therefore, it can be asserted that whether the regime switching is conducive to the survival of plankton populations or not strongly depends on its staying longer in a 'good' or 'bad' environmental state.
- (ii) White noise is adverse to the survival of plankton populations. As the Figure 5 points out, if the white noise densities are relatively small satisfying the conditions of Theorem 4, then system (1) has a unique ergodic stationary distribution, which means NTP, TPP and zooplankton can coexist at a stable state for a long time. However, by enhancing the intensity of white noise on zooplankton only or on all three species simultaneously, the zooplankton of Subsystem 2 or all the three species of every system will go to extinction (see Figures 1 and 2). From Figures 1, 2 and 5, by controlling the intensity of white noise, the dynamic behaviours of system (1) can be significantly changed. That is, high intensity of white noise is disadvantageous to the development of plankton and increases the risk of extinction. This is ecologically meaningful as the species deteriorates drastically because of high environmental fluctuations. Thus, it is obliged to be stressed that the controlling of the white noise may be acted as a possible biological way to control planktonic blooms.
- (iii) TPP can increase the survival chance of phytoplankton but reduce the biomass of zooplankton. With the increasing value of the toxin liberation rate  $\gamma$  ensuring the condition of Theorem 5 holds, system (1) has a unique stationary distribution (see Figure 7), which describes the long time asymptotic behaviors of the system (1) from a statistical viewpoint. Additionally, comparing Figures 5 and 7, we can conclude that the toxin liberation rate is conducive to the persistence in the mean of phytoplankton but is adverse to the survival of zooplankton population. Therefore, TPP plays an important role in controlling planktonic blooms.

## Acknowledgements

This work was supported by the National Natural Science Foundation of China (Grant No. 61871293), the National Key Research and Development Program of China (Grant No. 2018YFE0103700) and the National Natural Science Foundation of China (Grant No.61901303).

## Conflict interest

We declare that this work does not have any conflicts of interest.

## References

1. Huppert A, Blasius B, Stone L. A model of phytoplankton blooms. *Am Nat.* 2002;159:156-171.
2. Hallegraeff GM. A review of harmful algal blooms and the apparent global increase. *Phyco.* 1993;32:79-99.
3. Anderson DM, Kaoru Y, White AW. *Estimated Annual Economic Impacts from Harmful Algal Blooms (HABs) in the United States Sea Grant Woods Hole.* NCCOS, Silver Spring, 2000.
4. Michalak AM, Anderson EJ, Beletsky D, Boland S, Bosch NS, Bridgeman TB, ... Zagorski MA. Record-setting algal bloom in Lake Erie caused by agricultural and meteorological trends consistent with expected future conditions. *Proc Natl Acad Sci.* 2013;110(16):6448-6452.
5. Fulton III RS, Paerl HW. Toxic and inhibitory effects of the blue-green algae *Microcystis aeruginosa* on herbivorous zooplankton. *J Plankton Res.* 1987;9(5):837-855.
6. Lampert W. Inhibitory and toxic effects of blue-green algae on *Daphnia*. *Int Revue ges Hydrobiol.* 1981;66(3):285-298.
7. Krik K, Gilbert J. Variations in herbivore response to chemical defences: zooplankton foraging on toxic cyanobacteria. *Ecol.* 1992;73:2208-2217.
8. Calbet A, Vaque D, Felipe J, et al. Relative grazing impact of microzooplankton and mesozooplankton on a bloom of the toxic dinoflagellate *Alexandrium minutum*. *Mari Ecol Prog Ser.* 2003;259:303-309.
9. Johansson M, Coats DW. Ciliate grazing on the parasite *Amoebophrya* sp. decreases infection of the red-tide dinoflagellate *Akashiwo sanguinea*. *Aquat Microb Ecol.* 2002;28(1):69-78.
10. Wang XD, Qin BQ, Gao G, Paerl HW. Nutrient enrichment and selective predation by zooplankton promote *Microcystis* (Cyanobacteria) bloom formation. *J Plankton Res.* 2010;32(4):457-470.
11. Estep KW, Nejstgaard JC, Skjoldal HR, Rey F. Predation by copepods upon natural populations of *Phaeocystis pouchetii* as a function of the physiological state of the prey. *Mar Ecol Prog Ser.* 1990;67:333-344.
12. Huntley ME, Sykes P, Rohan S, Marin V. Chemically mediated rejection of dinoflagellate prey by the copepods *Calanus pacificus* and *Paracalanus parvus*: mechanism, occurrence and significance. *Mar Ecol Prog Ser.* 1986;28:105-120.
13. Chattopadhyay J, Sarkar R, Pal S. Mathematical modeling of harmful algal blooms supported by experimental findings. *Ecol Complex.* 2004;1:225-235.
14. Chattopadhyay J, Sarkar R, Mandal R. Toxin-producing plankton may act as a biological control for the termination of planktonic blooms-field study and mathematical modeling. *J Theor Biol.* 2002;215(3):333-344.
15. Truscott J, Brindley J. Ocean plankton populations as excitable media. *Bull Math Biol.* 1994;56:981-998.
16. Dai CJ, Zhao M, Yu HG. Dynamics induced by delay in a nutrient-phytoplankton model with diffusion. *Ecol Complex.* 2016;26:29-36.
17. Guo Q, Dai CJ, Yu HG, Liu H, Sun XX, Li JB, Zhao M. Stability and bifurcation analysis of a nutrient-phytoplankton model with time delay. *Math Meth Appl Sci.* 2019;1-22.
18. Chakraborty S, Tiwarib PK, Misrab AK, Chattopadhyay J. Spatial dynamics of a nutrient-phytoplankton system with toxic effect on phytoplankton. *Math Biosci.* 2015;264:94-100.
19. Li JJ, Gao WJ. Analysis of a nutrient-phytoplankton model in the presence of viral infection. *Acta Math Appl Sin E.* 2016;1:113-128.
20. Chattopadhyay J, Sarkar RR, Pal S. Dynamics of nutrient-phytoplankton interaction in the presence of viral infection. *Biosys.* 2003;68:5-17.
21. Richardson K, Heilmann JP. Primary production in the Kattegat: past and present. *Ophelia.* 1995;41:317-328.
22. Carpenter SR, Cole JJ, Pace ML, Batt R, Brock WA, Cline T, Coloso J, Hodgson JR, Kitchell JF, Seekell DA, Smith L, Weidel B. Early warnings of regimes shifts: a whole-ecosystem experiment. *Science* 2011;332:1079-1082.



23. Reichwaldt ES, Song H, Ghadouani A. Effects of the distribution of a toxic *Microcystis* bloom on the small scale patchiness of zooplankton. *PLOS ONE* 2013;8(6):1.
24. Davis TW, Berry DL, Boyer GL, Gobler CJ. The effects of temperature and nutrients on the growth and dynamics of toxic and non-toxic strains of *Microcystis* during cyanobacteria blooms. *Harmful Alg.* 2009;8:715-725.
25. Fujimoto N, Sugiura N, Inamori Y. Nutrient-limited growth of *Microcystis aeruginosa* and *Phormidium tenue* and competition under various N:P supply ratios and temperatures. *Limnol Oceanogr.* 1997;42(2):250-256.
26. Yu XW, Yuan SL, Zhang TH. The effects of toxin-producing phytoplankton and environmental fluctuations on the planktonic blooms. *Nonlinear Dyna.* 2017;3:1653-1668.
27. Zhao QY, Liu ST, Niu XL. Stationary distribution and extinction of a stochastic nutrient-phytoplankton-zooplankton model with cell size. *Math Meth Appl Sci.* 2020; 1-17.
28. Sarkar RR, Chattopadhyay J. The role of environmental stochasticity in a toxic phytoplankton-non-toxic phytoplankton-zooplankton system. *Environmetrics* 2003;14:775-792.
29. Pal S, Chatterjee S, Das KP, Chattopadhyay J. Role of competition in phytoplankton population for the occurrence and control of plankton bloom in the presence of environmental fluctuations. *Ecol Model.* 2009;220:96-110.
30. Chen ZW, Tian ZY, Zhang SW, Wei CJ. The stationary distribution and ergodicity of a stochastic phytoplankton-zooplankton model with toxin-producing phytoplankton under regime switching. *Physica A* 2020;537:122728.
31. Chen ZW, Zhang RM, Li J, Zhang SW, Wei CJ. A stochastic nutrient-phytoplankton model with viral infection and Markov switching. *Chaos, Solitons & Fractals* 2020;140:110109.
32. Wang H, Liu M. Stationary distribution of a stochastic hybrid phytoplankton-zooplankton model with toxin-producing phytoplankton. *Appl Math Lett.* 2020;101:106077.
33. Huisman J, Weissing FJ. Biodiversity of plankton by species oscillations and chaos. *Nature* 1999;402:407-410.
34. Beddington JR. Mutual interference between para-sites or predators and its effect on searching efficiency. *J Anim Ecol.* 1975;44:331-340.
35. DeAngelies DL, Goldstein RA, O'Neill RV. A model for trophic interactions. *Ecol.* 1975;56:881-892.
36. May RM. *Stability and Complexity in Model Ecosystems*. Princeton University Press, Princeton, NJ, 1973.
37. Du NH, Kon R, Sato K, Takeuchi Y. Dynamical behavior of Lotka-Volterra competition systems: Non-autonomous bistable case and the effect of telegraph noise. *J Comput Appl Math.* 2004;170:399-422.
38. Slatkin M. The dynamics of a population in a Markovian environment. *Ecol.* 1978;59:249-256.
39. Scheffer M, Carpenter SR, Foley JA, Folke C, Walker B. Catastrophic shifts in ecosystems. *Nature* 2001;413:591-596.
40. Mao XR, Sabanis S, Renshaw E. Asymptotic behavior of stochastic Lotka-Volterra model. *J Math Anal Appl.* 2003;287:141-156.
41. Luo Q, Mao XR. Stochastic population dynamics under regime switching. *J Math Anal Appl.* 2007;334:69-84.
42. Li X, Jiang DQ, Mao XR. Population dynamical behavior of Lotka-Volterra system under regime switching. *J Comput Appl Math.* 2009;232:427-448.
43. Zhu C, Yin G. Asymptotic properties of hybrid diffusion systems. *SIAM J Control Optim.* 2007;46:1155-1179.
44. Yin G, Zhu C. *Hybrid Switching Diffusions: Properties and Applications*, Springer-Verlag, New York, 2009.
45. Mao XR, Yuan C. *Stochastic Differential Equations with Markovian Switching*, Imperial College Press, London, 2006.
46. Yu XW, Yuan SL, Zhang TH. Persistence and ergodicity of a stochastic single species model with Allee effect under regime switching. *Commun Nonlinear Sci Numulat.* 2018;59:359-374.
47. Mao XR. *Stochastic Differential Equations and Applications*, Horwood Chichester, 1997.
48. Liu M, Wang K, Wu Q. Survival analysis of stochastic competition models in a polluted environment and stochastic competition exclusion principle. *Bull Math Biol.* 2011;73:1969-2012.
49. Liu M, Bai CZ. Analysis of a stochastic tri-trophic food-chain model with harvesting. *J Math Biol.* 2016;73:597-625.
50. Khasminskii R. *Stochastic Stability of Differential Equations*, Sijthoff & Noorhoff, The Netherlands, 1980.
51. Higham DJ. An algorithmic introduction on numerical simulation of stochastic differential equations. *SIAM Rev* 2001;43:525-546.
52. Philips E, Badylak S, Youn S, Kelley K. The occurrence of potentially toxic dinoflagellates and diatoms in a subtropical lagoon, the Indian River Lagoon, Florida, USA. *Harmful Alg.* 2004;3:39-49.
53. Hallam TG, Luna JT. Effects of toxicants on populations: a qualitative: approach III. Environmental and food chain pathways. *J Theor Biol.* 1984;109:411-429.
54. Carpenter SR, Cole JJ, Pace ML, et al. Early warnings of regime shifts: a whole-ecosystem experiment. *Science* 2011;332:1079-1082.