Occurrence of planktonic blooms under environmental fluctuations and its possible control mechanism—mathematical models and experimental observations

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Abstract

Planktonic blooms and its control is an intriguing problem in ecology. To investigate the oscillatory successions of blooms, three simple phytoplankton–zooplankton systems are proposed. It is observed that if the uptake function is linear and the process of toxin liberation is instantaneous, the oscillatory nature of blooms is not observed. On the other hand, periodic planktonic blooms are observed when toxin liberation process follows discrete time variation. The bloom phenomena described by this mechanism can be controlled through toxin producing phytoplankton (TPP). Introducing environmental fluctuation in the system, a critical value of time delay in terms of correlation time of the fluctuation is worked out. We observed from our mathematical analysis, numerical simulation and field observation that TPP and control of the rapidity of environmental fluctuation are key factors for the termination of planktonic blooms.

Keywords: Toxin producing plankton (TPP); Zooplankton; Bloom; Time delay; Colored noise; Control

1. Introduction

The dynamics of rapid (or massive) increase or decrease of plankton populations is an important subject in marine plankton ecology. Generally, high nutrient levels and favorable conditions play a key role in rapid or massive growth of algae and low nutrient concentration as well as unfavorable conditions inevitably limit their growth. The water must contain high levels of inorganic nutrients (nitrogen and phosphorus) for the algae to feed on and also water temperature and salinity levels must be within a certain range to be conducive to planktonic growth. A frequent outcome of planktonic bloom formation is massive cell lysis and rapid disintegration of large planktonic populations. This is closely followed by an equally rapid increase in bacterial numbers, and in turn by a fast deoxygenation of water, which could be detrimental to aquatic plants and animals. These blooms also reduce the chance of growth for aquatic vegetation. Hence studies regarding the pattern of blooms are necessary towards this serious ecological problem. In recent years, there has been considerable scientific attention towards harmful algal blooms (HABs) (see, Blaxter and Southward, 1997; Stoermer and Smol, 1999; Anderson, 1989; Smayda, 1990; Hallegraeff, 1993; Chattopadhyay et al., 2002a, etc.). A broad classification of HAB species distinguishes two groups: the toxin producers, which can contaminate seafood or kill fish, and the high-biomass producers, which can cause anoxia and indiscriminate mortalities of marine life after reaching dense concentrations. Some HAB species have characteristics of both groups. The term 'bloom' may be misleading, particularly when used in reference to organisms that produce toxin. A broader definition of the harmful marine organisms includes (i) planktonic or benthic microalgae that produce toxin (e.g. the motile stage of *Alexandrium*, the benthic *Gambierdiscus*); (ii) other harmful dinoflagellates (e.g. *Pfiesteria*); (iii) macroalgae that results in noxious smells (e.g. *Pilayella*), (iv) a few species of Cyanobacteria or blue green algae (e.g. *Microcystis*) and (v) non-toxic microorganisms that result in hypoxic

conditions (e.g. *Chaetoceros, Mesodinium*). The adverse effects of HABs on human health, commercial fisheries, subsistence fisheries, recreational fisheries, tourism and coastal recreation, ecosystem and environment are well established. Nevertheless, despite the attention towards this issue, the mechanism for the occurrence of planktonic blooms and its possible control strategy are not yet well established and required special attention. Hence experimental work as well as mathematical modelling is necessary.

In a broad sense planktonic blooms can be derived into two types, "spring blooms" and "red tides". Spring blooms occur seasonally for the changes in temperature or nutrient availability which are connected with seasonal changes. Red tides are localized outbreaks and occur due to high water temperature (see, Truscott and Brindley, 1994). Nature of blooms, in the sense of the rapid onset and disappearance of oscillations under supposedly favorable environmental condition is one of the main characteristics in plankton ecosystem. It is convenient to define the meaning of blooms. This can be explained by two ways, namely multistability (in which the system tends to one of the coexisting stable equilibria) and sustained oscillation (Hopf bifurcation, in which the system oscillates around an unstable equilibrium). Several researchers have tried to explain the dynamics of the nature of blooms in planktonic systems by different approaches: for example, nutrient upwelling has been investigated by Edwards and Brindley (1996), spatial patchiness by Mathews and Brindley (1996) and species diversity by Pitchford and Brindley (1998). Steele and Henderson (1993); Edwards and Brindley (1996) observed that the choice of functional form and mortality of zooplankton has a major influence in the dynamics of excitable nature of blooms.

Harmful phytoplankton certainly plays an important role in the blooms and succession. Probably, the main reason behind population succession and bloom is due to the toxin produced by harmful phytoplankton. When a bloom of a particular harmful phytoplankton occur, the cumulative effect of all the toxins released may affect the other organisms, causing mass mortality. Such extreme concentrations or blooms are responsible for the massive localized mortality observed in fish and invertebrates in various places (see, Rice, 1984). Reduction of grazing pressure of zooplankton due to release of toxic substances by phytoplankton is one of the key parameters in this context (e.g., see Kirk and Gilbert, 1992). There is also some evidence that herbivore (zooplankton, see Odum, 1971) grazing plays a crucial role in the initial stages of a red tide outbreak (e.g., see Uye, 1986). Buskey and Stockwell (1993) have demonstrated in their field studies that micro and meso zooplankton populations are reduced during the blooms of a chrysophyte *Aureococcus anophagefferens* in the southern Texas coast. Toxicity may be a strong mediator of zooplankton feeding rate, as shown in both field studies (see Nielsen et al., 1990) and laboratory studies (see Ives, 1987; Nejstgaard and Solberg, 1996). These observations indicate that the toxic substance plays an important role on the growth of the zooplankton population and has a great impact on phytoplankton–zooplankton interactions. Researchers are trying to establish that viral infection on plankton plays an important role for the termination of blooms (Sarno and Forlani, 1999; Beltrami and Carroll, 1994). But the identification of viruses is a difficult task and also its control mechanism is still a matter of debate. Chattopadhyay et al. (2002a) established an alternative approach (the effect of toxic chemicals on zooplankton in contrast to viral infection on phytoplankton) to explain the mechanism for the occurrence of planktonic blooms and its possible control.

Another interesting problem is the dynamics of externally forced systems. Natural forcing is of course superposed by a certain environmental noise. Massive phytoplankton blooms were observed in Seto Inland Sea, Japan (Prakash, 1987) and in Hong Kong Harbour (Lam and Ho, 1989) and were due to artificial eutrophication. It has been observed that artificial eutrophication plays an important role for excitable behavior of the system. It has also been suggested that eutrophication (Smayda, 1989, 1990), changes in N:P ratio (Egge and Heimdal, 1994) and certain contaminants in the marine environment (Papathanassiou et al., 1994) affect phytoplankton succession. Richardson and Heilmann (1995) have suggested that eutrophication resulting from human activities has led to an increase in phytoplankton production in Kattegat throughout the annual period in which nutrients are predicted to be limiting for phytoplankton growth. Thus, plankton populations often fluctuate unpredictably due to the fluctuating environment and the study of the dynamics of the system under these circumstances is necessary.

May (1973) analysed a prey–predator system under stochastic fluctuation considering white noise for the population and observed that when the population deviates more from the equilibrium point, the system shows instability i.e. for instability of the system, the deviations from mean population will increase. The ecological effects for terrestrial and marine systems will depend on the character of the physical frequency distributions and the general qualitative response of these systems could be inherently different. For terrestrial system, the environmental variability is large at both short and long time periods and could be expected to develop internal mechanisms to the system which would cope with short-term variability and minimize the effects of long-term variations, hence analysis of the system with white noise gives better results. But for marine systems, particularly in planktonic systems, less

robust internal processes are needed to handle the smaller amplitude variability at short time periods commensurate with the life span of the organisms. Steele and Henderson (1984) observed more complicated models containing specific descriptions of ecological relations particularly with stochastic inputs for different frequency ranges of color and white noise and used specifically for marine plankton. They found that for white noise the system would fluctuate irregularly at much shorter time-scales compare to the color noise though the comparable forcing period is much more for the case of white noise than that of the color noise. Marine ecological systems are more likely to have color noise instead of white noise as compared to terrestrial systems (Steele, 1985; Pimm, 1982). Furthermore, several authors (Cushing, 1989; Kiørboe, 1993) have pointed out that the growth rates of the herbivores capable of consuming large phytoplankton cells are slow relative to the growth rates of the algae and that there will be a relatively long lag time between the onset of an increase in growth rate in large phytoplankton species and a build up in the biomass of their predators. There are also several reports that the zooplankton mortality due to the toxic phytoplankton bloom, occurs after some time lapse (see, www.mote.org, www.mdsg.umd.edu).

The above observations indicate that the nature of planktonic blooms is very much complex. In this paper we propose a simple phytoplankton–zooplankton model with an additional factor that the release of toxic substance by phytoplankton species reduces the growth of zooplankton. We shall try to explain the nature of blooms by changing the following two constituents of toxin liberation one at a time, (1) liberation process is instantaneous and (2) it follows the discrete time variation. Our theoretical result suggests that in the latter case periodic planktonic blooms can be explained. We shall also attempt to search a possible mechanism for controlling the planktonic blooms by introducing environmental stochasticity in the model system. The main objectives of this paper are the following:

- to find a suitable mechanism by which one can explain the nature of periodic planktonic blooms;
- to find a possible control mechanism by which the planktonic blooms can be checked;
- to find a critical value of delay factor in a noise-induced system, which is not yet available in literature.

To establish the theoretical results we shall use our field observations. We have considered the toxin producing plankton (TPP) species as *Noctiluca scintillans* and zooplankton species as *Paracalanus* sp. (the taxonomical and functional distinctions of both the species are given in Chattopadhyay et al., 2002a). Our field study suggest that the zooplankton population fluctuates unpredictably in the collection zone nearer to the river (where artificial eutrophication increased due to discharge of sewages, etc.) and the presence of TPP in this region is much less. Hence rapid appearance and disappearance of the zooplankton population (which may be termed as 'blooms') have been observed. Further in the collection zone, which is far from the riverside, the presence of TPP terminates the bloom of zooplankton population after some time lag and a sustained existence of the population has been observed without oscillation. Also, we observe that the blooms of the zooplankton population near the riverside can be controlled by introducing a random fluctuation with low intensity in the observed data and this phenomena agrees with our analytical study. This establishes the fact that both TPP and control of artificial eutrophication may act as a control for planktonic blooms.

2. Background of mathematical models

From the above discussion, it is now clear that for termination of blooms, TPP and control of artificial eutrophication have major role. To establish this we shall propose some mathematical models and the findings will be supported through our field observations.

Monitoring of plankton population was carried out since 1999 in the NorthWest coast of Bay of Bengal. As we are interested to report the effect of toxin producing phytoplankton on zooplankton, we choose *Noctiluca scintillans* (TPP) and *Paracalanus* sp. (zooplankton) for this study. The materials and method of the study have been discussed elaborately in Chattopadhyay et al. (2002a). In this paper we will consider the data of the field observation from March, 1999 to January, 2001 (30 sample collection dates). To establish our theoretical realization we shall mainly focus on the dynamics of TPP–zooplankton system in different collection regions, the schematic diagram of which is given in Fig. 1. Here stations 1, 2, 6 and 7 of the collection region (zone 1) are nearer to the Talsari river (which are artificially eutrophicated by the discharge of sewage etc. from the

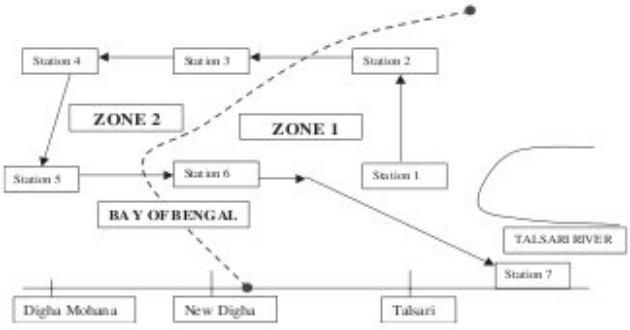

Fig. 1. Collection region of marine plankton samples in the North West Coastal Region of Bay of Bengal.

districts Balasore, Orissa and Midnapur, West Bengal, India) and stations 3, 4 and 5 (zone 2) are far from the river side (thus less affected by artificial eutrophication).

It is interesting to note that in zone 1, the presence of TPP is much less than that of zone 2. The rapid appearance and disappearances of zooplankton population in zone 1 clearly demonstrate the bloom phenomena (see, Fig. 2). We also observe that most of the zooplankton population is above the average level of population (calculated by the average of population density throughout our study time). But in zone 2 most of the zooplankton population lies below the average level. Also the sustained oscillations which we observed in zone 1 settled down (see, Fig. 3). Hence the role of TPP in the termination of zooplankton bloom is clear.

Motivated from the literature and our field observations, a series of dynamic models to describe bloom phenomena have been proposed and the role of toxic phytoplankton as well as artificial eutrophication in the termination of planktonic blooms have been observed. Our first aim is to find a suitable model which explains the oscillatory succession of planktonic bloom and then propose suitable mechanisms by which the blooms thus formed may be terminated or controlled. The following issues have been explored in this work and the details are given in subsections.

First, we propose a mathematical model of TPP and zooplankton interaction. It has been already stated that TPP population do not release toxic chemicals always, release only in the presence of dense zooplankton population around it. This phenomenon has been included in the interaction terms as Holling type I and type II functional forms, respectively. Our analysis shows that this model formulation cannot explain the bloom phenomena and hence a modified model is needed.

To search for a mechanism of oscillatory succession in planktonic blooms, we further develop the first model considering the biological fact that the reduction of grazing pressure due to toxic substance is not instantaneous, but is mediated by some time lag required to release toxic substances by the phytoplankton species. There are also several reports that the zooplankton mortality due to toxic phytoplankton bloom, occurs

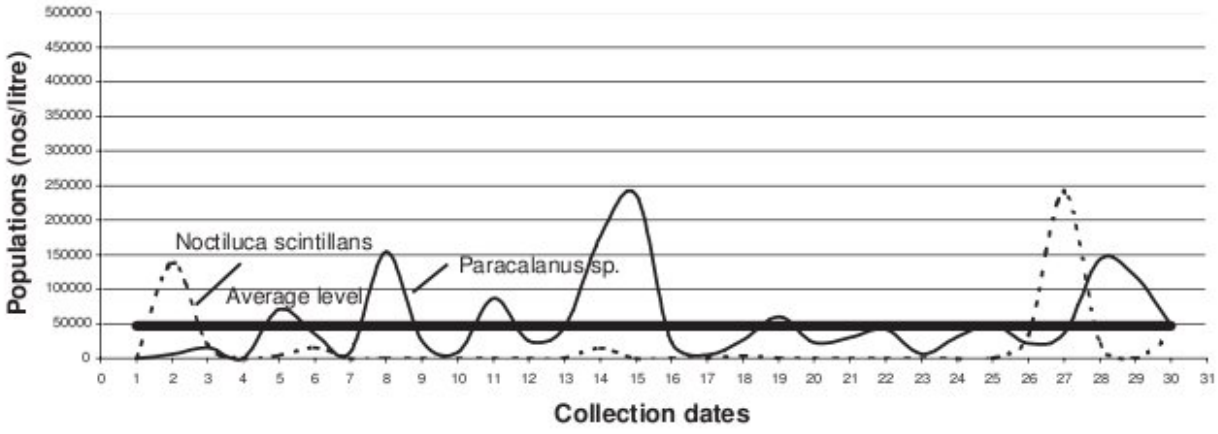

Fig. 2. Blooms of Paracalanus sp. and less presence of Noctiluca scintillans in zone 1.

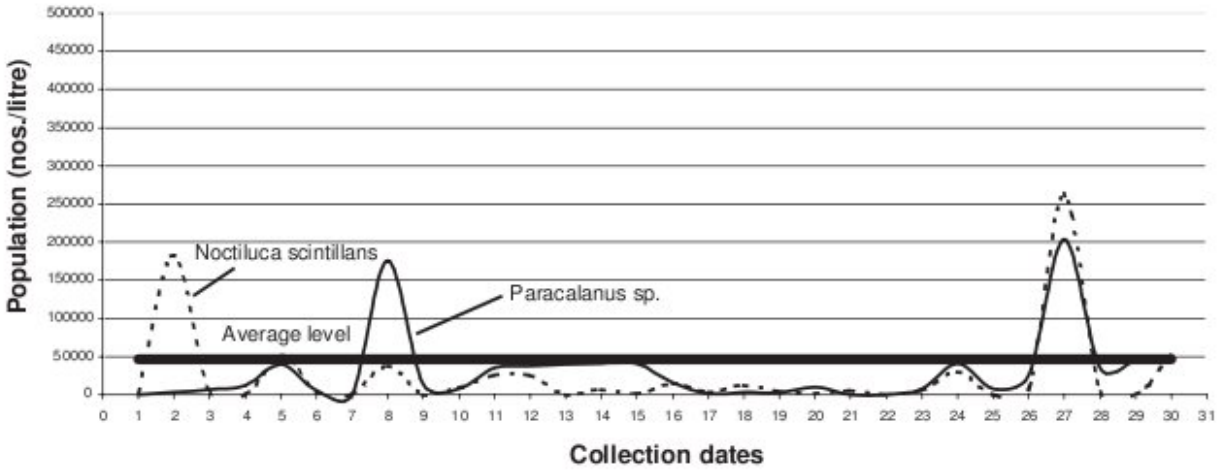

Fig. 3. Termination of blooms of Paracalanus sp. and more presence of Noctiluca scintillans in zone 2.

after some time lapse (see www.mote.org, www.mdsg.umd.edu). Our field observations (see Chattopadhyay et al., 2002a) also suggest that the abundance of *Paracalanus* (zooplankton) population reduces after some time lapse of the bloom of toxin producing phytoplankton *Noctiluca scintillans*. This depicts the fact that, there is a time lag between the occurrence of harmful algal bloom and the reduction of grazing pressure of zooplankton population. Further, Chattopadhyay et al. (2002b) observed that when the release of toxic substances by phytoplankton population follows a gamma distribution then the cyclic nature of blooms cannot be explained by this type of distribution of toxic substances or toxic phytoplankton. The prediction based on the system involving distributed delay illustrates that concentration of toxic substances or toxic phytoplankton eventually approach equilibrium concentration and hence no periodic solutions are possible. It is also worth noting that if the order of the delay kernel, go to infinity while keeping the mean delay fixed, then the distributed delay can be viewed as discrete delay (see Cooke and Grossman, 1982). Thus, we have considerable freedom to modify the first system by introducing discrete time variation in the grazing term.

Thirdly, we want to explain a more realistic feature as it is already mentioned in the introduction that phytoplankton zooplankton interaction would be more visible if one can study the dynamics in the presence of externally forced system. Inter annual variability in phytoplankton production can to a large extent be explained by changing weather conditions and changing land use, as the water shed and rainfall determine the nutrient and sediment input to coastal sea water. Thus in order to understand ecosystem functioning better, we need to understand what determines phytoplankton species composition and succession during blooms, what happens when blooms decay, how contaminants influence bloom dynamics and what is the interplay between nutrient enrichment (through artificial eutrophication) and harmful algal blooms. This forcing appears rather normally due to daily, seasonal or annual cycles, photosynthetically active radiation, temperature, nutrient availability, eutrophication, etc. and phytoplankton populations often fluctuate unpredictably in numbers (Evans and Parslow, 1985; Truscott, 1995; Popova et al., 1997; Ryabchenko et al., 1997). Further, the historically increased nutrient loads to estuaries have been reflected in similar trends of increased nutrient concentrations in at least some coastal seawater (Hickel et al., 1993; Allen et al., 1998), raising concerns about nutrification stimulating both primary production and nuisance blooms of algae, not only within estuaries but also in adjacent coastal seawater. However, trends of nutrient increase in coastal seas have usually been confined to localized areas subjected to severe impact by large local inputs, such as in German Bight by the Rhine and Scheldt plumes (Gieskes and Kraay, 1977; Jickells, 1998). While annual loads give comparative information between estuaries, there may be large seasonal differences in the inputs of nutrient, with consequent ecological effects (Nedwell et al., 1999). The impact on the estuary of nutrient load will amongst other factors, depend on the residence time of the nutrient within the estuary. The Freshwater Flushing Time (FWFT), which expresses the volume of freshwater within the estuary as a function of the freshwater input (Dyer, 1973), will be influenced by tidal mixing and exchange and affect the volume of freshwater remaining within the estuary. It has been observed through the longitudinal profiles of salinity that FWFT varied between as little as 15 days during winter peak flows as much as 765 days in late summer when river flow was least and the freshwater volume in the estuary was replaced only slowly by riverflow (Nedwell et al., 1999). The algal bloom formation can result from two often simultaneously operating processes: (1) bottom-up control of photosynthesis and (2) top-down control of biomass. The interplay of these factors will determine whether there is an increase in biomass. Hence there is always a time lag for the change of phytoplankton biomass which depends on the nutrient concentration, light availability, grazing and is also influenced by physical conditions such as residence time of the nutrient or FWFT (Underwood and Kromkamp, 1999). Hence, the study of the dynamics under a certain environmental noise with the autocorrelation time of the noise process which can be interpreted as the residence time, is an appropriate consideration in this context. Now, we try to explain the development of different phases of model formulation systematically in the following subsections:

2.1. The Mathematical model with instantaneous toxin liberation

In the formulation of the model it has been assumed that the growth of phytoplankton population follows the logistic law (see, Murray, 1989; Odum, 1971) with intrinsic growth rate 'r' and environmental carrying capacity 'K'. It is mentioned in the introduction that some phytoplankton genera release toxic substances and thereby reduce the growth of zooplankton by decreasing the grazing pressure. It is also stated in the previous section that zooplankton grazing plays an important role in the initial stages of outbreaks. Keeping these properties of phytoplankton–zooplankton population in mind, two different types of predational forms have been assumed: simple law of mass action and Holling-type response term (Holling, 1959) which is also known from Monod or Michaelis-Menten saturation models of enzyme kinetics (Michaelis and Menten, 1913; Monod and Jacob, 1961). When phytoplankton populations do not produce toxin, the predation rate will follow the

simple law of mass action and in this case zooplankton eating is proportional to the phytoplankton density and thus limiting the production of the phytoplankton (since an individual zooplankton encounters in direct proportion to prey abundance, thus in the absence of limiting factors feeding rate increases linearly with food, see, Andersen and Nival, 1988). But as liberation of toxin reduces the growth of zooplankton, causes substantial mortality of zooplankton and in this period phytoplankton population is not easily available, hence a more common and intuitively obvious choice is of the Holling type II functional form to describe the grazing phenomena in the presence of toxic substances.

From the above assumptions the following differential equations can be formed:

$$\frac{dP}{dt} = rP\left(1 - \frac{P}{K}\right) - \alpha PZ,$$
$$\frac{dZ}{dt} = \beta PZ - cZ - \frac{\theta P}{\gamma + P}Z. \quad (1)$$

Here P and Z represent the density of phytoplankton and zooplankton population, respectively, $\alpha(>0)$ is the specific predation rate and $\beta(>0)$ represents the ratio of biomass consumed per zooplankton for the production of new zooplankton. $c(>0)$ is the mortality rate of zooplankton. $\theta(>0)$ is the rate of toxin production per phytoplankton species and $\gamma(>0)$ is the half saturation constant.

System (1) has to be analysed with the following initial conditions:

$$P(0) > 0, \quad Z(0) > 0. \quad (2)$$

System (1) has the following nonnegative equilibria, namely, a trivial equilibrium $E_0(0,0)$, an axial equilibrium $E_1(K,0)$ and the interior equilibrium $E^*(P^*,Z^*)$, where

$$P^* = \frac{-(\beta\gamma - c - \theta) + \sqrt{(\beta\gamma - c - \theta)^2 + 4\beta\gamma c}}{2\beta}, \quad (3)$$

$$Z^* = \frac{r}{\alpha}\left(1 - \frac{P^*}{K}\right). \quad (4)$$

A simple algebraic calculation shows that a necessary and sufficient condition for the existence of positive equilibrium E^* is

$$\theta < (\beta K + \beta\gamma - c) - \frac{\gamma c}{K}. \quad (5)$$

It is observed that the right-hand side of system (1) is a smooth function of the variables (P,Z) and the parameters, as long as these quantities are nonnegative, so local existence and uniqueness properties hold in the positive quadrant. From the first equation of system (1), it follows that $P = 0$ is an invariant subset, that is $P = 0$ if and only if $P(t) = 0$ for some t. Thus, $P(t) > 0$ for all t if $P(0) > 0$. Similar argument follows for $Z = 0$ from the second equation of system (1). Now, let us consider the boundedness of solutions of system (1).

Lemma 2.1.1. *All the solutions which initiate in* $\{R_+^2 \backslash 0\}$ *are uniformly bounded.*

The proof of the lemma is given in the appendix (A).

Stability analysis: Local stability analysis (LAS) of system (1) around the equilibria can be studied by computing the variational matrix. It is easy to see that the trivial equilibrium E_0 is an unstable saddle point. Existence of a positive interior equilibrium implies that the axial equilibrium E_1 is also an unstable saddle in character. Non-existence of positive equilibrium ensures that E_1 is locally asymptotically stable. The characteristic equation of system (1) around the positive interior equilibrium E^* is given by

$$\lambda^2 - M\lambda + N = 0, \quad (6)$$

where

$$M = -\frac{rP^*}{K}(<0),$$
$$N = \frac{\alpha P^* Z^*}{(\gamma + P^*)^2}[\beta(\gamma + P^*)^2 - \theta\gamma].$$

It can be easily verified from second equation of system (1) that $\theta \geqslant \beta(\gamma + P^*)^2/\gamma$ can never be a solution of the system, hence instability, even simple bifurcation is also not possible in this case. Now since $M < 0$ and $N > 0$ system (1) around $E^*(P^*,Z^*)$ is locally asymptotically stable. To investigate the global behavior of system (1) let us first prove that system (1) around E^* has no nontrivial periodic solutions.

Lemma 2.1.2. *Existence of positive equilibrium of system* (1) *ensures that there is no limit cycle in the first quadrant.*

The proof of the lemma is given in Appendix A.

Theorem 2.1.1. *Existence of a positive interior equilibrium ensures that system* (1) *around* $E^*(P^*,Z^*)$ *is globally asymptotically stable.*

The proof of the theorem is given in Appendix A.

From the above observations we can now write down the results of system (1) in the following remark.

Remark. If the growth of phytoplankton species follows logistic law, uptake function is of linear type in the absence of toxic substances, reduction of grazing pressure due to toxic substances is of Holling type II and the distribution of toxic substance is instantaneous, oscillatory succession of planktonic blooms is not observed by this model formulation.

2.2. Model with discrete time variation in toxin liberation

In this subsection we shall try establish a suitable mechanism to explain the oscillatory succession in planktonic blooms by introducing discrete time variation in the grazing term in system (1). We state the main results for oscillatory successions of planktonic blooms following the paper of Chattopadhyay et al. (2002b). System (1) now takes the following form:

$$\frac{dP}{dt} = P\left(r - \frac{rP}{K}\right) - \alpha PZ,$$
$$\frac{dZ}{dt} = \beta PZ - cZ - \frac{\theta P(t-\tau)}{\gamma + P(t-\tau)}Z, \quad (7)$$

where τ is the discrete time delay.

System (7) has the same equilibria as in the previous case. The nature of trivial and axial equilibrium are the same as in system (1). We now perturb system (7) around $E^*(P^*, Z^*)$ and obtain the following linearized system of differential equations:

$$\frac{dx}{dt} = Ax + By,$$
$$\frac{dy}{dt} = Cx + Ex(t-\tau), \quad (8)$$

where

$$x = P - P^*, \quad y = Z - Z^*, \quad A = -\frac{rP^*}{K}, \quad B = -\alpha P^*,$$
$$C = \beta Z^*, \quad E = -\frac{\theta\gamma Z^*}{(\gamma + P^*)^2}. \quad (9)$$

We assume a solution of the form $x(t) = e^{\lambda t}$. We have the corresponding characteristic equation as

$$\Delta(\lambda, \tau) = \lambda^2 - A\lambda - BC - BEe^{-\lambda\tau} = 0. \quad (10)$$

Now substituting $\lambda = \alpha_1 + i\omega$ in Eq. (10) and separating the real and imaginary parts we obtain the system of transcendental equations

$$\alpha_1^2 - \omega^2 - A\alpha_1 - BC - BEe^{-\alpha_1\tau}\cos\omega\tau = 0,$$
$$2\alpha_1\omega - A\omega + BEe^{-\alpha_1\tau}\sin\omega\tau = 0. \quad (11)$$

The stability or instability of the system is determined by the sign of those λ satisfying Eq. (10) if λ is real or the sign of α_1 satisfying Eq. (11) if λ is complex.

Theorem 2.2.1. *The following are necessary and sufficient conditions for E^* to be asymptotically stable for every $\tau \geqslant 0$ (see, Saaty, 1981):*

1. *The real part of every root of $\Delta(\lambda, 0) = 0$ is negative.*
2. *For all real ω_0 and $\tau \geqslant 0$, $\Delta(i\omega_0, \tau) \neq 0$, where $i = \sqrt{-1}$.*

Theorem 2.2.2. *As $A < 0$ and $B < 0$, then in the parametric region $-E < C$ the interior equilibrium E^* of system (7) is locally asymptotically stable for $0 < \tau < \pi/\omega_0$.*

Now we have the following condition under which the system goes through a point $\tau(=\tau_0)$ and $\omega(=\omega_0)$ where a Hopf-bifurcation occurs.

Lemma 2.2.1. *If $A^2 + 2BC < 0$ and $0 \leqslant C < -E$ then there exists a unique pair (ω_0, τ_0) with $\omega_0, \tau_0 \geqslant 0$, $\omega_0\tau_0 < 2\pi$ such that $\Delta(i\omega_0, \tau_0) = 0$, where ω_0 and τ_0 are given by the following formulae:*

$$\omega_0^2 = \tfrac{1}{2}\left[-(A^2 + 2BC) + \sqrt{(A^2 + 2BC)^2 - 4(B^2C^2 - B^2E^2)}\right],$$

$$\tau_0 = \frac{1}{\omega_0}\arcsin\left(\frac{-A\omega_0}{BE}\right) + \frac{2n\pi}{\omega_0}, \quad n = 0, 1, 2, \ldots .$$

Lemma 2.2.2. *Let $A^2 + 2BC < 0, 0 \leqslant C < -E$. Then the real parts of the solutions of Eq. (11) are negative for $\tau < \tau_0$, where $\tau_0 > 0$ is the smallest value for which there is a solution to Eq. (10) with real part zero. For $\tau > \tau_0$, E^* is unstable. Further as τ increases through τ_0, E^* bifurcates into small amplitude of periodic solutions. Chattopadhyay et al. (2002b) also derived the conditions for which system (7) around E^* enters into supercritical or subcritical bifurcation.*

Note. From Lemma 2.2.1 we have

$$\tau_0 = \frac{1}{\omega_0}\arcsin\left(\frac{-A\omega_0}{BE}\right) \text{ (for } n = 0).$$

Now as $K \to \infty, \tau_0 \to 0$ (since $\omega_0 \to$ a fixed positive value). This result shows that enhancement of environmental carrying capacity decreases the critical value of the delay factor to zero. As a result the instability region of the system increases. This observation also exhibits the nature of blooms through periodicity.

2.3. Model with discrete delay and color noise

In the previous subsection we observed that oscillatory succession of planktonic blooms can be explained if the production of toxic substance follows the discrete time variation which occurs due to time lapse required for liberation of toxic substances when there is a dense concentration of zooplankton population. From the above point of view we shall again modify and study system (7) taking into account the additive color noise with proper choice of autocorrelation time of the noise process as the residence time for nutrient concentration.

It is well known that the rapid perpetual, highly irregular motions of a small particles (in this case phytoplankton) can be described by Brownian motion. Generally, the Wiener process is used to model Brownian motion i.e. it is chosen as a stochastic process to represent the position of the Brownian particle. But if

the Wiener process is used then the instantaneous velocity is not defined in the model as the sample paths of the Wiener process are nowhere differentiable and the process is infinite. This difficulty can be avoided considering the main random quantity as Uhlenbeck and Ornstein process, which is also the appropriate choice to model a colored noise environment in most of the applications (see Horsthemke and Lefever, 1983). This supports the usefulness of the color noise idealization in applications to the natural aquatic systems. Here we have assumed the Stratonovich interpretation of the stochastic differential equations, which conserves the ordinary rule of calculus and in this case the ensemble average of the solutions of stochastic differential equations can generally be related to the solutions of appropriately constructed ordinary differential equations.

The aim of this section is to find out a relationship between the critical value of delay factor and the correlation time, so that one can have an idea for controlling the planktonic blooms. The dynamics of the system in a random environment will be considered within the frame work of the following model taking into account the additive form of the colored noise process:

$$\frac{dP}{dt} = P\left(r + \eta(t) - \frac{rP}{K}\right) - \alpha PZ,$$
$$\frac{dZ}{dt} = \beta PZ - cZ - \frac{\theta P(t-\tau)Z}{\gamma + P(t-\tau)}, \tag{12}$$

where the perturbed term $\eta(t)$ is a colored noise or follows Ornstein–Uhlenbeck process (Uhlenbeck and Ornstein, 1954). The mathematical expectation and correlation function of the process $\eta(t)$ are given by

$$\langle \eta(t) \rangle = 0, \langle \eta(t_1)\eta(t_2) \rangle = \varepsilon\delta_0 \exp(-\delta_0|t_1 - t_2|), \tag{13}$$

where $\varepsilon, \delta_0 > 0$ are, respectively, the intensity and the correlation time of the noise and $\langle . \rangle$ represents average over the ensemble of the stochastic process. The correlation function $\eta(t)$ is the solution of the stochastic differential equation

$$\frac{d\eta}{dt} = -\delta_0\eta + \delta_0\sqrt{2\varepsilon}\frac{d\omega}{dt}, \tag{14}$$

where $\xi(t) = d\omega/dt$ denotes the standard zero mean Gaussian white noise characterized by

$$\langle \xi(t) \rangle = 0, \langle \xi(t_1)\xi(t_2) \rangle = \delta(t_1 - t_2) \tag{15}$$

with $\delta(t)$ as the Dirac delta function. Substituting $X = \log P$ and $Y = \log Z$ in Eq. (12), we obtain

$$\frac{dX}{dt} = r + \eta(t) - \frac{re^X}{K} - \alpha e^Y,$$
$$\frac{dY}{dt} = \beta e^X - c - \frac{\theta e^{X(t-\tau)}}{\gamma + e^{X(t-\tau)}}.$$

Now using the transformation $u = X - X^*, v = Y - Y^*$, respectively, in the above system of equations we obtain

$$\frac{du}{dt} = r + \eta(t) - \frac{re^{u+X^*}}{K} - \alpha e^{v+Y^*},$$
$$\frac{dv}{dt} = \beta e^{u+X^*} - c - \frac{\theta e^{u(t-\tau)+X^*}}{\gamma + e^{u(t-\tau)+X^*}}.$$

Then using Taylor series expansion up to first order of $u(t), v(t), u(t-\tau)$ and using small delay expansion in the above system of equations we finally obtain the linearized system as:

$$\frac{du}{dt} = \eta(t) - \frac{rP^*}{K}u - \alpha Z^* v,$$
$$\frac{dv}{dt} = \theta\gamma P^*\tau\eta(t) + \left(\beta - \theta\gamma - \frac{r\theta\gamma P^*\tau}{K}\right)P^* u - \theta\gamma\tau\alpha P^* Z^* v, \tag{16}$$

where (P^*, Z^*) is the positive interior equilibrium point of the system in the absence of noise.

By eliminating v and u from (16), respectively, we get

$$\frac{d^2u}{dt^2} + a\frac{du}{dt} + bu = F_1(t),$$
$$\frac{d^2v}{dt^2} + a\frac{dv}{dt} + bv = F_2(t), \tag{17}$$

where $F_1(t) = -\delta_0\eta(t) + \delta_0\sqrt{2\varepsilon}\xi(t)$ and $F_2(t) = (\beta - \gamma\theta\delta_0 - \theta\gamma\tau)P^*\eta(t) + \theta\gamma\tau P^*\delta_0\sqrt{2\varepsilon}\xi(t)$. Here $a = (r/K + \theta\gamma\alpha\tau Z^*)P^*$ and $b = \alpha P^* Z^*(\beta - \gamma\theta)$. We are now in a position to solve the stochastic differential equation given in Eq. (17). To solve this system we shall follow the approach of Hoel et al. (1993). We obtain

$$u(t) = u(0)\phi_1(t) + u'(0)\phi_2(t) + \eta(t),$$
$$v(t) = v(0)\phi_1(t) + v'(0)\phi_2(t) + \left[(\beta - \gamma\theta)P^*\sqrt{2\varepsilon}\xi(t) - \frac{(\beta - \theta\gamma - \theta\gamma\tau\delta_0)P^*}{\delta_0}\eta(t)\right]. \tag{18}$$

Here

$$\phi_1(t) = \frac{r_1' e^{r_1'' t} - r_1'' e^{r_1' t}}{r_1' - r_1''} \quad \text{when, } a^2 - 4b > 0$$
$$= e^{\alpha_1'}(\cos\beta_1' t - \frac{\alpha_1'}{\beta_1'}\sin\beta_1' t) \quad \text{when } a^2 - 4b < 0$$
$$= e^{r_1 t}(1 - r_1 t) \quad \text{when } a^2 - 4b = 0,$$
$$\phi_2(t) = \frac{e^{r_1' t} - e^{r_1'' t}}{r_1' - r_1''} \quad \text{when } a^2 - 4b > 0$$
$$= \frac{e^{\alpha_1'}}{\beta_1'}\sin\beta_1' t \text{ when } a^2 - 4b < 0$$
$$= t e^{r_1 t} \text{ when } a^2 - 4b = 0,$$

where $a=(r/K+\theta\gamma\alpha\tau Z^*)P^*$, $b=\alpha P^*Z^*(\beta-\gamma\theta)$, $r_1'=(-a+\sqrt{a^2-4b})/2, r_1''=(-a-\sqrt{a^2-4b})/2$, $\alpha_1'=-a/2$, $\beta_1'=(\sqrt{4b-a^2})/2$, $r_1=-a/2$ and $u(0)=\log P(0)/P^*, u'(0)=r-rP(0)/K-\alpha Z(0), v(0)=\log Z(0)/Z^*$, $v'(0)=\beta P(0)-c-\theta P(0)/(\gamma+P(0))$.

The solution without noise term takes the following form:

$$u(t)=u(0)\phi_1(t)+u'(0)\phi_2(t),$$
$$v(t)=v(0)\phi_1(t)+v'(0)\phi_2(t). \tag{19}$$

In this case the ensemble average of the population are given by

$$\langle u(t)\rangle = u(0)\langle\phi_1(t)\rangle + u'(0)\langle\phi_2(t)\rangle$$

and

$$\langle v(t)\rangle = v(0)\langle\phi_1(t)\rangle + v'(0)\langle\phi_2(t)\rangle.$$

Now for $t\to\infty$, $\langle\phi_1(t)\rangle\to 0$ and also $\langle\phi_2(t)\rangle\to 0$ (since in this case $a^2-4b>0$ which is satisfied as the condition in Theorem 2.2.2 holds for locally asymptotic stability of the system).

Hence we get $\langle P(t)\rangle = P^*$, $\langle Z(t)\rangle = Z^*$ and also the deviations of both the populations $\sigma_P^2=0$ and $\sigma_Z^2=0$.

Now we are in a position to see the effect of environmental fluctuation in the system. Recently, Sarkar et al. (2001) developed a method to estimate the optimal values of the parameters and the safe region for an eco-epidemiological model of Tilapia and Pelican populations proposed by Chattopadhyay and Bairagi (2001) and Chattopadhyay et al. (2001) successfully used this technique to estimate the inaccessible parameters in a plant–herbivore–parasitoid system. Here we shall use their method to explain the dynamics of TPP–zooplankton system under environmental fluctuation.

From Tchebycheff's inequality we have a pre-assigned small value $\varepsilon_0>0$ for which $\lim_{t\to\infty} Prob(|P-P^*|<\varepsilon_0)=1$ (as $\sigma_P=0$) and $\lim_{t\to\infty} Prob(|Z-Z^*|<\varepsilon_0)=1$ (as $\sigma_Z=0$) i.e. the probability of P-population and Z-population to lie inside the tolerance intervals $(P^*-\varepsilon_0, P^*+\varepsilon_0)$ and $(Z^*-\varepsilon_0, Z^*+\varepsilon_0)$, respectively are maximum.

Now the question is how these tolerance intervals are affected due to influence of stochastic fluctuations?

The solutions of the noise induced system are given by

$$P(t)=P^*\exp\left[\log\frac{P(0)}{P^*}\phi_1(t)+\left(r-\frac{rP(0)}{K}-\alpha Z(0)\right)\phi_2(t)+\eta'(t)\right],$$
$$Z(t)=Z^*\exp\left[\log\frac{Z(0)}{Z^*}\phi_1(t)+\left(\beta P(0)-c-\frac{\theta P(0)}{\gamma+P(0)}\right)\phi_2(t)+\eta''(t)\right], \tag{20}$$

where $\eta'(t)=\eta(t), \eta''(t)=[(\beta-\gamma\theta)P^*\sqrt{2\varepsilon}\xi(t)-[(\beta-\theta\gamma\delta_0-\theta\gamma\tau)P^*/\delta_0]\eta(t)]$. For $t\to\infty$, we have $\langle P(t)\rangle = P^*$, $\langle Z(t)\rangle = Z^*$. But in this case the deviations are different from zero and are given by

$$\sigma_P^2=2\varepsilon\delta_0 P^{*2},$$

$$\sigma_Z^2=2\varepsilon\left[(\beta-\gamma\theta)^2+\frac{2(\beta-\theta\gamma\delta_0-\theta\gamma\tau)^2}{\delta_0}\right]P^{*2}Z^{*2}.$$

Now for different choice of system parameters when σ_P^2 is greater than ε_0 and similarly σ_Z^2 is also greater than ε_0, both populations will deviate from the tolerance level and the system becomes unstable around the positive equilibrium. In terms of system parameters the deviations of two populations P and Z from the mean are, respectively, given by

$$\sigma_P^2=2\varepsilon\delta_0\left[\frac{-(\beta\gamma-c-\theta)+\sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}}{2\beta}\right]^2,$$

$$\sigma_Z^2=\frac{\varepsilon r^2}{8\alpha^2\beta^4K^2}\left[(\beta-\gamma\theta)^2+\frac{2(\beta-\theta\gamma\delta_0-\theta\gamma\tau)^2}{\delta_0}\right]\times\left[-(\beta\gamma-c-\theta)+\sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}\right]^2\times\left[2\beta K+(\beta\gamma-c-\theta)-\sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}\right]^2. \tag{21}$$

Now $\lim_{\delta_0\to\infty}\sigma_P^2=\infty$ and $\lim_{\delta_0\to\infty}\sigma_Z^2=\infty$.

So we can say that as δ_0 increases the deviations also increase and the system becomes unstable. It is well known that the population will remain stable if the variances from the equilibrium level are minimum (May, 1973) i.e. the probability of the population to lie inside the tolerance level which is described previously are maximum. To minimize σ_P^2 and σ_Z^2 and also to find out the critical value of τ we differentiate the second equation of (21) with respect to τ (since there is no τ in the first equation of (21)) and we get

$$\frac{d(\sigma_Z^2)}{d\tau}=\frac{\varepsilon r^2}{8\alpha^2\beta^4K^2}\left[\frac{-4(\beta-\theta\gamma\delta_0-\theta\gamma\tau)\theta\gamma\tau}{\delta_0}-\frac{2(\beta-\theta\gamma\delta_0-\theta\gamma\tau)^2}{\delta_0^2}\right]\left[-(\beta\gamma-c-\theta)+\sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}\right]^2\left[2\beta K+(\beta\gamma-c-\theta)-\sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}\right]^2. \tag{22}$$

Now from $d(\sigma_Z^2)/d\tau=0$ we get

$$\tau=\frac{\beta-\theta\gamma\delta_0}{\theta\gamma}(=\tau_0 \quad say). \tag{23}$$

Further we obtain

$$\frac{d^2(\sigma_Z^2)}{d\tau^2}\Big|_{\tau=\tau_0} = \frac{\varepsilon r^2(\beta-\theta\gamma)^2}{8\alpha^2\beta^4K^2\delta_0^3}[-(\beta\gamma-c-\theta) + \sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}]^2 \times \left[2\beta K+(\beta\gamma-c-\theta) - \sqrt{(\beta\gamma-c-\theta)^2+4\beta\gamma c}\right]^2$$

and using the existence condition of E^* we observe that

$$\frac{d^2(\sigma_Z^2)}{d\tau^2}\Big|_{\tau=\tau_0} > 0.$$

It is interesting to note that

$$\tau_0 = \frac{1}{\omega_0}\arcsin\left(\frac{-A\omega_0}{BE}\right) \text{ (for } n=0)$$

(from Lemma 2.2.1)

$$\simeq -\frac{A}{BE}$$

(when retaining only first order terms of ω_0)

$$= \frac{(r+P^*)^2}{\theta\gamma(K-P^*)}. \quad (24)$$

Now from the conditions of existence of the positive equilibrium E^* we have, $\tau_0 \leqslant P^{*^2}\theta^2/\theta\gamma(K-P^*)$ (from Eq. (24)) and further from the conditions of Lemma 2.2.1 we finally have

$$\tau_0 \leqslant \frac{\beta}{\theta\gamma}\frac{2P^*\theta^2K}{r}. \quad (25)$$

We can further observe that when there is no noise i.e. when the perturbed term $\eta \to 0$ (which suggests that the correlation time $\delta_0 \to 0$) then the stochastic threshold τ reduces to

$$\tau_0 \to \frac{\beta}{\theta\gamma} \quad \text{(from Eq.(23))}.$$

This result is closely resembled to the deterministic threshold τ given in Eq. (25).

Thus we can conclude that when $\tau \leqslant \tau_0$, (τ_0 is given in (23)), the deviations of the population from mean level are minimum. Further it is to be noted here that if we vary only two parameters δ_0, τ_0 and keeping all other parameters fixed, we see that from Eq. (23), $\tau_0 = f(\theta) - \delta_0$ (where $f(\theta) = \beta/\theta\gamma$). This result shows that there is a linear relationship between the delay factor and correlation time with a negative slope. The delay factor (τ) of the species is an inherent property and cannot be controlled in other way. We observe that the oscillatory succession of the planktonic blooms may be controlled either through toxin or correlation time of artificial fluctuations.

3. Results and discussion

It is to be well noted that for the justification of linearized stochastic delay-differential equations (Eq. (16)), the delay expansion τ must be small compared to the autocorrelation time (δ_0^{-1}) of the noise process (see Guillouzic et al., 1999). In our model formulation we have considered the delay factor as time lag required for the maturity of the phytoplankton species to release toxic substances when there is a dense concentration of zooplankton species. Thus to resemble with the short lifespan of phytoplankton species it is quite reasonable to consider the unit of the delay term (τ) in minutes. Also in our field study (see Chattopadhyay et al., 2002a), we observe that the blooms of phytoplankton and zooplankton species are occurring nearly every 7 months. The environmental variability connected with this system is mainly due to artificial eutrophication from the sewages that requires a long time interval to dissolve and react with seawater. As a result there is a change in the nutrient configuration of the water (discussed thoroughly in Section 2.3). Keeping in mind the previous history of the bloom phenomena, it is therefore reasonable to consider the unit of the autocorrelation time (δ_0^{-1}) as per 7 months signifying the physical reasonability of the problem. To establish the above analytical findings numerically, we shall use the other parameters values which are presented and discussed elaborately in the paper of Edwards and Brindley (1999) and also used by several authors. Abbreviations, default values (which have been used here) and ranges of the parameters are given in Table 1. From the above set of parameter values it has been observed that the numerical simulation of the solution represented by Eq. (20), depicts the stable situation of the system for $\tau = 0$ and $\delta_0 = 0.1$ since the maximum portion of the population lies inside the tolerance limit as mentioned in the earlier section (see Fig. 4). But we observe that the system remains stable for $\tau \in (0, 170)$ and above the limit the system becomes unstable (maximum portion of the populations lie outside the tolerance limit) around the positive interior equilibrium point (see Figs. 5 and 6). This critical value of τ_0 is also very close to the numerical value obtained from Eq. (23) for the above set of parameter values. Now if we increase the value of the correlation time (δ_0) from 0.1 to 1, we observe that the system around the positive interior equilibrium point becomes stable for $\tau < 125$ and unstable for $\tau > 125$ (see Figs. 7 and 8). Further if we decrease the value of δ_0 to $\delta_0 = 0.01$ then we observe that the system around the positive interior equilibrium point becomes stable for $\tau < 1250$ and unstable for $\tau > 1250$ (see Figs. 9 and 10). The above numerical results also depict the fact that there exists an opposite relation between the correlation time (δ_0) and the delay factor (τ). This is consistent with our analytical

Table 1
Abbreviations, default values and ranges of the parameters. The ranges cover values used by different authors in their different models as mentioned by Edwards and Brindley (1999)

Parameters	Symbols	Default Values	Reported Ranges
Growth rate of phytoplankton population (P)	r	0.0083 ($hour^{-1}$)	0.00292–0.0117 ($hour^{-1}$)
Environmental carrying capacity	K	1.667 ($g\ C\ m^{-3}$)	—
Grazing efficiency of zooplankton population (Z)	α	0.0375 ($m^3 g^{-1} C^{-1} hr^{-1}$)	0.025–0.0583 ($m^3 g^{-1} C^{-1} hr^{-1}$)
Growth efficiency of zooplankton population (Z)	ß	0.0125 ($m^3 g^{-1} C^{-1} hr^{-1}$)	0.0083–0.0208 ($m^3 g^{-1} C^{-1} hr^{-1}$)
Higher predation on Z or natural death rate	c	0.00083 ($hour^{-1}$)	0.000625–0.00625 ($hour^{-1}$)
Zooplankton grazing half saturation coefficient	γ	0.0025 ($g\ C\ m^{-3}$)	0.00083–0.00417 ($g\ C\ m^{-3}$)
Toxin production rate	θ	0.167 ($hour^{-1}$)	—

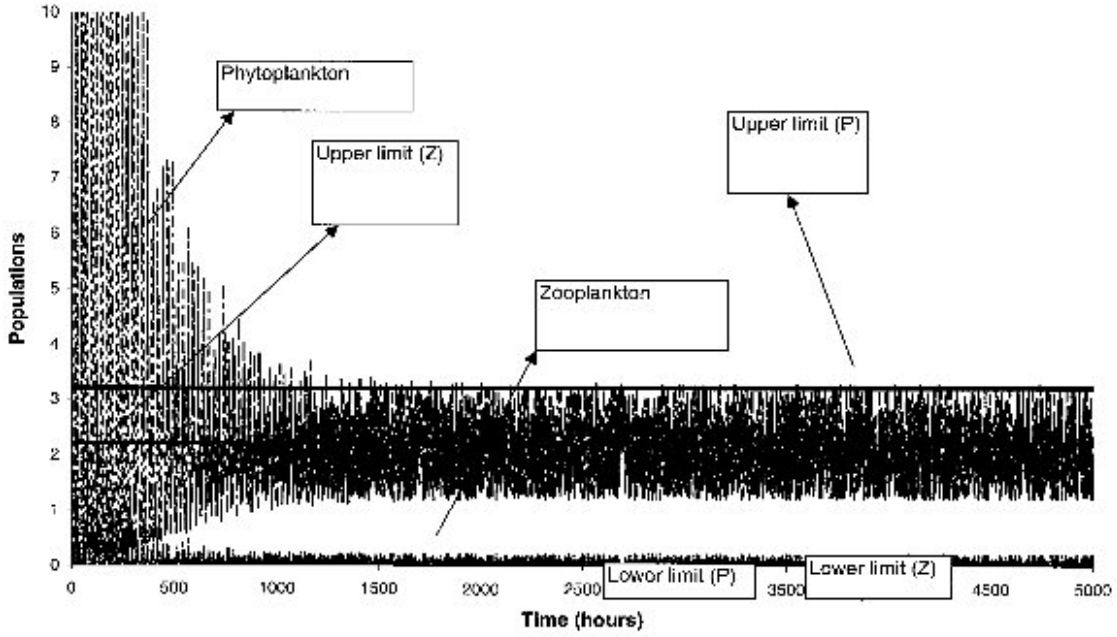

Fig. 4. Numerical solutions of equation (20) depicting stable situation for tau = 0 and delta_0 = 0.1.

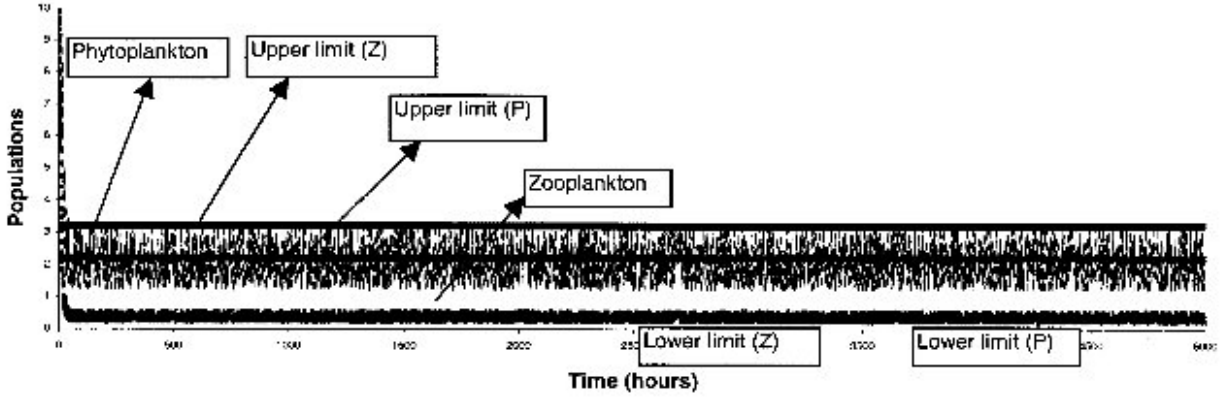

Fig. 5. Numerical solutions of equation (20) depicting stable situation for tau = 170 and delta_0 = 0.1.

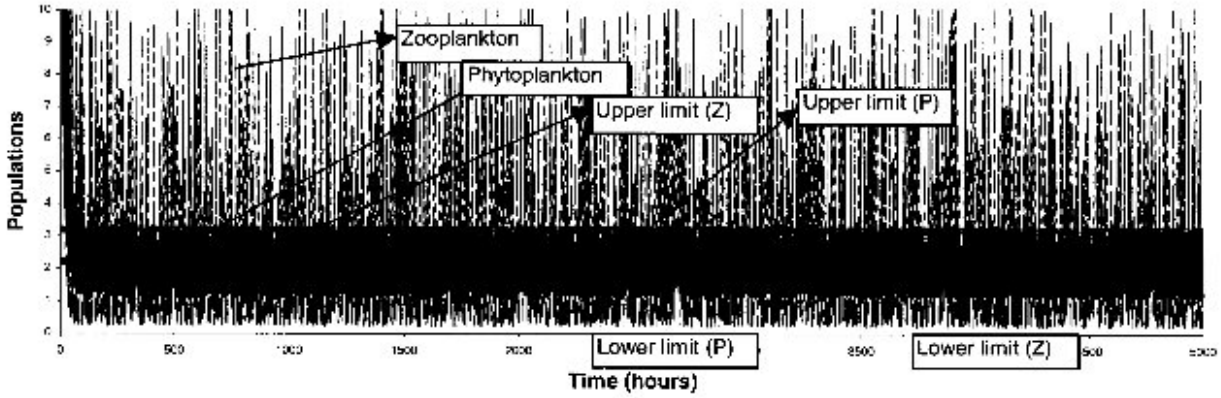

Fig. 6. Numerical solutions of equation (20) depicting unstable situation for tau = 180 and delta_0 = 0.1.

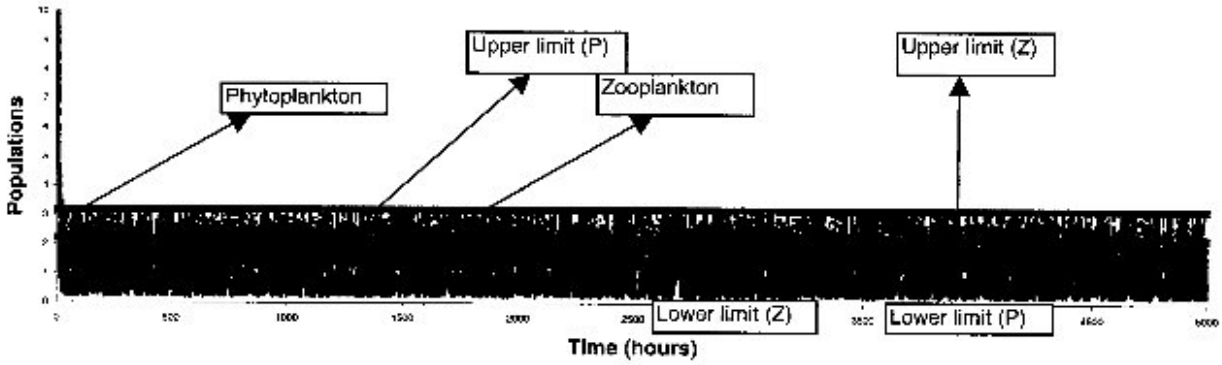

Fig. 7. Numerical solutions of equation (20) depicting stable situation for tau = 120 and delta_0 = 1.

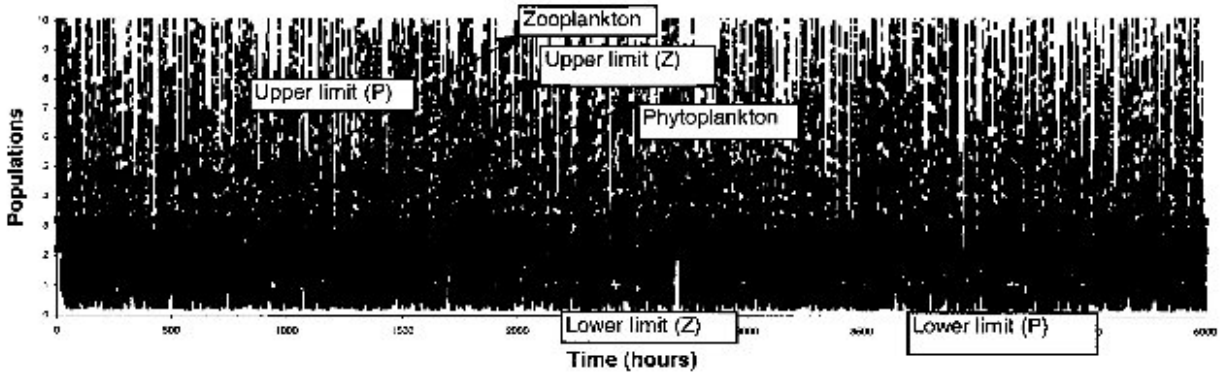

Fig. 8. Numerical solutions of equation (20) depicting unstable situation for tau = 130 and delta_0 = 1.

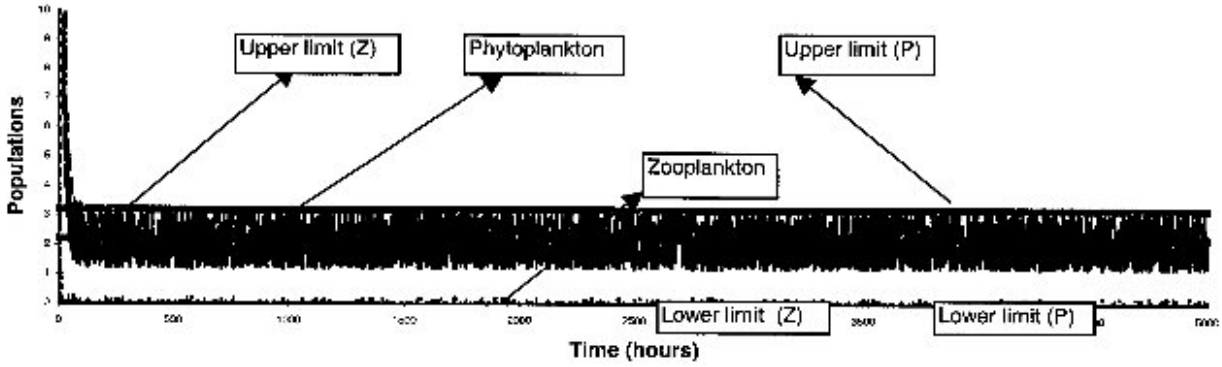

Fig. 9. Numerical solutions of equation (20) depicting stable situation for tau = 1240 and delta_0 = 0.01.

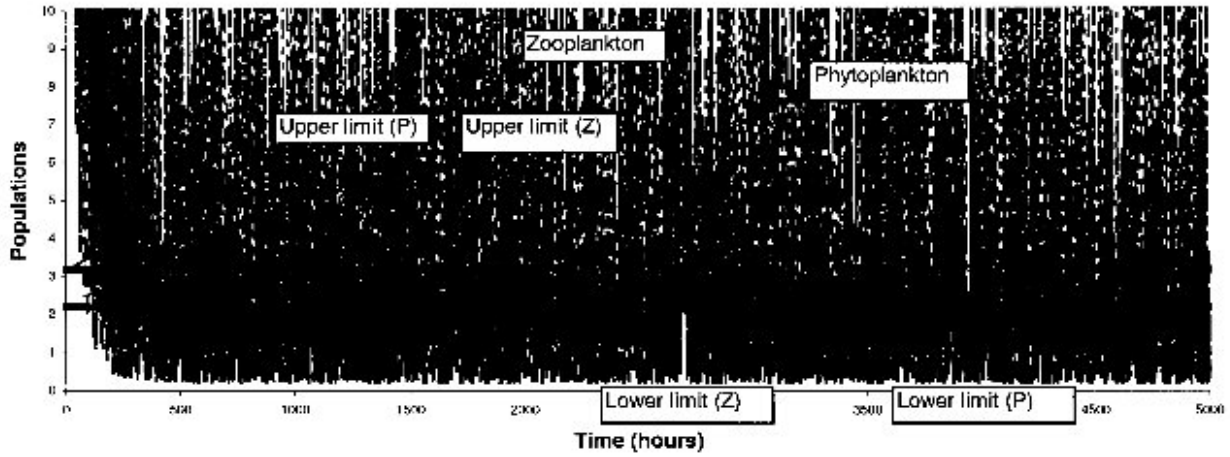

Fig. 10. Numerical solutions of equation (20) depicting unstable situation for tau = 1260 and delta_0 = 0.01.

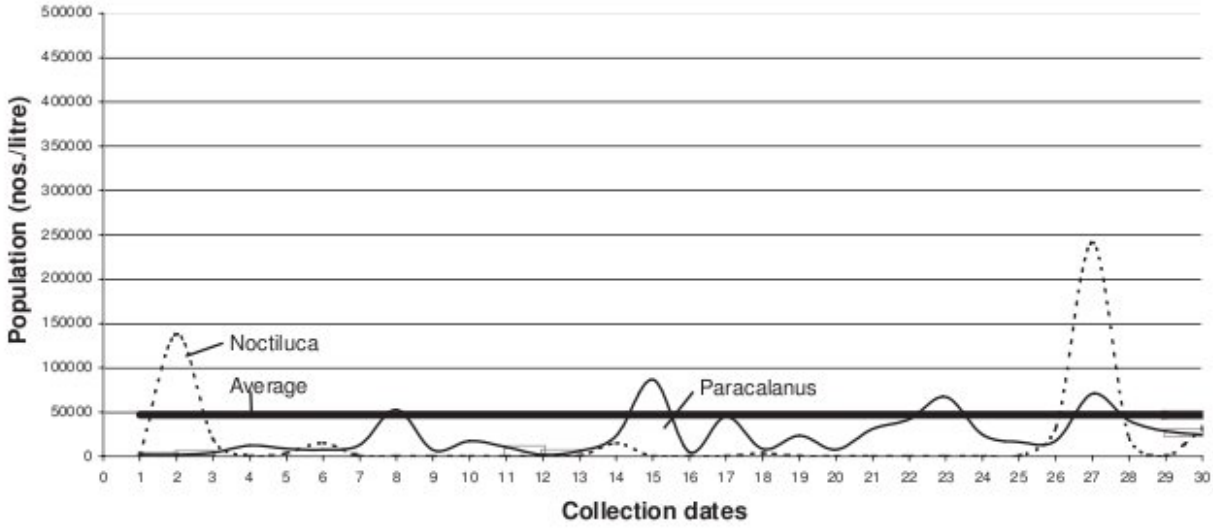

Fig. 11. Termination of blooms of Paracalanus sp. after introduction of artificial fluctuation.

findings. Thus, we conclude that the correlation factor may be used as a key parameter for controlling the planktonic blooms. It is also interesting to note that the region of stability of the system around the positive interior equilibrium point increases due to decrease of correlation time. This shows that, the system becomes unstable around the positive interior equilibrium when the time variation (τ) exceeds the critical value (τ_c) but introduction of less rapid fluctuations stabilizes the above unstable situation.

Hence we may conclude that rapidity of artificial eutrophication or enhancement of nutrients increase the region of instability. On the other hand if the process of artificial eutrophication is slow, the system exhibits less fluctuations. As a result the region of stability increases.

To establish the above findings, we shall use our field collected samples of zone 1 (please see, Section 2). We incorporated a random fluctuation to each data point of zooplankton population with very low intensity and correlation time. This provides a negative feedback to zooplankton population. We observed most of the population lie inside the average level and the rapid onset and disappearance of oscillations are settled down (see, Fig. 11). This experimental observations resembles our analytical results which are presented in Section 2.3.

To explain the idea of tolerance intervals in our study, we have considered the average level of the zooplankton population as the upper limit of the tolerance interval and the zero level as the lower limit of the tolerance interval. From our field observations we observe that in zone 1 the zooplankton population deviates more from the average level than in zone 2. It is also observed in the presence of artificial fluctuation the deviations in zone 1 are less than that of zone 1 without artificial fluctuation. The entire results have been summarized in Table 2. These observations clearly indicate that TPP populations in zone 2 and control of artificial eutrophication in zone 1, may help in the termination of blooms.

Table 2
Measure and comparison of deviations of zooplankton population in different zones

Deviation of Zooplankton population from average level in Zone 1	Deviation of Zooplankton population from average level in Zone 2	Deviation of Zooplankton population from average level in Zone 1 under artificial fluctuation	Percentage of decrease in deviations of Zone 1 and Zone 2	Percentage of decrease in deviations of Zone 1 and Zone 1 under artificial fluctuation
58017.92	46711.87	25516.73	19.487 %	56.019%

4. Conclusion

This paper attempted to search a suitable mechanism by which one can explain the oscillatory succession of planktonic blooms and also tried to find out a possible mechanism for controlling blooms. To explain these we used models made up of three main constituents: phytoplankton–zooplankton (prey-predator) coupling with instantaneous toxin liberation, the discrete time variation in toxin liberation and the environmental stochasticity in the phytoplankton population as an additive color noise. Our theoretical results show that if the uptake function is linear in nature, reduction of grazing pressure due to toxic substances is of type II functional form and the distribution of toxic substances is instantaneous, the oscillatory successions of planktonic blooms have not been observed. But if the toxin liberation process follows the discrete time variation then the periodic planktonic blooms can be explained. It is also observed that bloom phenomena described by this mechanism can be controlled by two ways, (i) through toxin producing phytoplankton (TPP) and (ii) through introduction of less rapid artificial eutrophication. From our analytical study we have observed that there exist a functional relationship between the critical value of time delay (τ) and rate of toxin production (θ) as well as the correlation time (δ_0). Hence, enhancing the rate of toxin production one can increase the region of stability of the system and as a result can control the periodicity of planktonic blooms. We have observed from our field study that when the presence of TPP is much more then the bloom of zooplankton population terminates (observed in zone 2 of Fig. 2). Thus the role of TPP in termination of planktonic bloom is clear from analytical and experimental point of view. Moreover, we have observed that there exists a linear relationship between the critical value of time delay and the correlation time of the fluctuation with a negative slope. Since correlation time measures the rapidity of environmental fluctuations and delay factor is an inherent property of the system, hence delay factor cannot be used as a possible control mechanism. Thus control of correlation time of environmental fluctuations may be used as a possible control mechanism for planktonic blooms. This mechanism has been successfully used in our field study to control the blooms of zooplankton population (observed in zone 1, see Fig. 11). Finally, we may conclude that TPP and control of the rapidity of environmental fluctuation are the key factors in termination of planktonic blooms.

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Appendix A

Proof of Lemma 2.1.1. Let us define a function

$$W = \beta P + \alpha Z. \tag{A.1}$$

The time derivative of (A.1) along the solutions of (1) is

$$\begin{aligned}\frac{dW}{dt} &= r\beta P\left(1-\frac{P}{K}\right) - \alpha c Z - \frac{\beta\theta P}{\gamma+P}Z \\ &\leqslant r\beta P\left(1-\frac{P}{K}\right) - \alpha c Z \\ &= \left[r\beta P\left(1-\frac{P}{K}\right) + c\beta P\right] - cW.\end{aligned}$$

The term $[r\beta P(1-\frac{P}{K}) + c\beta P]$ has a maximum value, so the above expression reduces to

$$\frac{dW}{dt} + cW \leqslant D,$$

where

$$D = \frac{\beta K(c+\gamma)^2}{4r}.$$

Applying a theorem of differential inequality (Birkoff and Rota, 1982), we obtain

$$0 < W(P,Z) \leqslant \frac{D}{c}(1-e^{-ct}) + W(P(0),Z(0))e^{-ct}$$

and for $t \to \infty$, we have

$$0 < W \leqslant \frac{D}{c}.$$

Hence all the solutions of (1) that initiate in $\{R_+^2 \backslash 0\}$ are confined in the region

$$B = \left\{(P,Z) \in R_+^2 : W = \frac{D}{c} + \varepsilon'\right\},$$

for any $\varepsilon' > 0$ and for t large enough. □

Proof of Lemma 2.1.2. The proof is based on an application of a divergence criterion (Hale, 1993). Let $h(P,Z) = 1/PZ$, obviously $h(P,Z) > 0$ if $P > 0, Z > 0$. Let us define

$$g_1(P,Z) = rP\left(1 - \frac{P}{K}\right) - \alpha PZ,$$

$$g_2(P,Z) = \beta PZ - cZ - \frac{\theta PZ}{\gamma + P},$$

$$\Delta(P,Z) = \frac{\partial}{\partial P}(g_1 h) + \frac{\partial}{\partial Z}(g_2 h),$$

we find that

$$\Delta(P,Z) = -\frac{r}{KZ} \tag{A.2}$$

which is less than zero for all $P > 0, Z > 0$. Therefore by the Bendixon–Dulac criterion, there will be no limit cycle in the first quadrant. □

Proof of Theorem 2.1.1. The proof is based on the following arguments:

(a) System (1) is bounded and positively invariant in the first quadrant if $\theta < (\beta K + \beta\gamma - c) - \gamma c/K$.

(b) Trivial equilibrium E_0 is always an unstable saddle point and existence of positive equilibrium confirms that the axial equilibrium E_1 is also an unstable saddle point.

(c) Positive equilibrium E^* is LAS.

(d) System (1) around E^* has no non-trivial periodic solutions.

Hence system (1) around the positive equilibrium is also globally asymptotically stable. □

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