Toxin-producing Plankton May Act as a Biological Control for Planktonic Blooms—Field Study and Mathematical Modelling

J. CHATTOPADHAYAY*, R. R. SARKAR† AND S. MANDAL†

†*Embryology Research Unit, Indian Statistical Institute*, 203, *B. T. Road, Kolkata* 700 035, *India*

(*Received on* 25 *September* 2001, *Accepted in revised form on* 5 *December* 2001)

Termination of planktonic blooms is of great importance to human health, ecosystem, environment, tourism and fisheries. Toxic substances released by plankton play an important role in this context. The effect of toxin-producing plankton (TPP) on zooplankton is observed from the field-collected samples and mathematical modelling. Information from both the studies led us to suggest that TPP may terminate the planktonic blooms by decreasing the grazing pressure of zooplankton and thus acts as a biological control.

Introduction

There has been a global increase in harmful plankton blooms in last two decades (Anderson, 1989; Smayda, 1990; Hallegraeff, 1993) and considerable scientific attention towards harmful algal blooms (HABs) has been paid in recent years (Blaxter & Southward, 1997; Stoermer & Smol, 1999). 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 result in noxious smells (e.g. *Pilayella*), (iv) 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 pattern of planktonic blooms and its possible control mechanism are not yet well established and required special attention. Hence, the experimental as well as mathematical modelling are necessary in this field.

Several researchers attempted to explain bloom phenomena by different approaches. The seasonal succession is most often and clearly seen in temperate seas, which have a marked variation in temperature over the course of a year, temperature has been suggested as a cause. Certainly, this may be one of the factors, but it is

unlikely to be solely responsible, because certain dominant species recur at different temperatures. Furthermore, than the temperature changes in seawater, the replacement of dominant species is often much more rapid. Changes in the nutrient level over the year are also a cause for planktonic blooms. While this factor may also contribute, observations suggest population changes are not closely correlated with the changes in the nutrient concentration (see, Nybakken, 1982). Harmful phytoplankton certainly plays an important role in the blooms and succession. Probably, the main reason behind population succession and bloom is the toxin produced by harmful phytoplankton. When a bloom of a particular harmful phytoplankton occurs, 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). It is now well established that quite a good number of phytoplankton species produce toxin, such as *Pseudo-nitzschia* sp., *Gambierdiscus toxicus*, *Prorocentrum* sp., *Ostrepsis* sp., *Coolia monotis*, *Thecadinium* sp., *Amphidinium carterae*, *Dinophysis* sp., *Gymnodinium breve*, *Alexandrium* sp., *Gymodinium catenatum*, *Pyrodinium bahamense*, *Pfiesteria piscicida*, *Chrysochromulina polylepis*, *Prymnesium patelliferum*, *P. parvum* (e.g., see Steidinger *et al.*, 1996; Nielsen *et al.*, 1990; Hallegraeff, 1993).

Reduction of grazing pressure of zooplankton due to the release of toxic substances by phytoplankton is one of the most important parameters in this context (e.g. see Kirk & Gilbert, 1992). There are also some good evidences 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 & 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 & Solberg, 1996). These observations indicate that the toxic substance plays one of the important role on the growth of the zooplankton population and have 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 & Forlani, 1999; Beltrami & Carroll, 1994). But the identification of viruses is a difficult task and also its control mechanism is still a matter of debate. The present paper attempts to establish an alternative approach (the effect of toxic chemicals on zooplankton on the contrary to viral infection on phytoplankton) to explain the suitable mechanism for the occurrence of planktonic blooms and its possible control. As far as knowledge goes, the role of toxin-producing plankton (TPP) on the termination of planktonic blooms is not observed either experimentally or from modelling point of view.

In the present study we shall put our emphasis to observe the effects of TPP on planktonic blooms and succession. Our tested TPP species is *Noctiluca scintillans* belonging to the group Dinoflagellates of the Division Dinophyta. It is a very common heterotrophic dinoflagellate and is known to feed on bacteria, diatoms, other flagellates and ciliate protozoans. This species has already been reported as harmful when appeared as bloom and exert adverse ecological effects, via its toxins to other planktonic organisms including fish. This species is taxonomically a phytoplankton but functionally it is a phagotroph or heterotroph.† Among zooplankton species we choose *Paracalanus* belonging to the group Copepoda which dominate the zooplankton community in all over the world oceans, and are the major herbivore which determine the form of the phytoplankton curve. Our field study suggests us that TPP may help in the termination of planktonic blooms and succession. These field observations helped us to formulate a mathematical model on TPP–zooplankton system in which the grazing pressure of zooplankton decreases due to the release of toxic substances by TPP species. The mathematical results also

† We are indebted to the learned referee for pointing out the taxonomical and functional distinctions of *Noctiluca scintillans*.

reveal the fact that TPP may act as a biological control for planktonic blooms.

The aim of the paper is to find out suitable mechanism for understanding the excitable nature of planktonic blooms and its possible control. Our presentation has three parts. Firstly, we present our field observation, which shows that TPP has a positive role in the termination of planktonic blooms. Secondly, we give a mathematical explanation for the occurrence of planktonic blooms. Thirdly, we present numerical simulations to substantiate our analytical findings and also suggest a possible way to control the planktonic blooms.

Materials and Methods

The study area extends from Talsari (Orissa, India) to Digha Mohana (West Bengal, India). Geographically, the area is situated between 21°37′ Northern Latitude and 87°25′ Eastern Longitude to 21°42′ Northern Latitude and 87°31′ Eastern Longitude. The geographical location of the study area is given in Fig. 1.

The study was carried out from January 2000 to February 2001. Samplings were done aboard 10 m fishing vessel hired each time from Talsari fish landing centre. Frequency of sampling was once in every fortnight except the months of September and October. During this time because of the roughness of the sea sampling programme had to be suspended.

Plankton samples were collected both from the surface and subsurface water (1–2 m depth) by a horizontal plankton tow with a 20 μm mesh net and 0.3 m in diameter. The collected samples were preserved in 3% formaldehyde in seawater.

Counting of phytoplankton was made under microscope using Sedgewick–Rafter counting cell and are expressed in nos. l^{-1}.

Identification of plankton community was done following the method of Tomas (1997).

Field Observation and Data Analysis

There were altogether 19 sampling days in the year 2000–2001. Number of samples (surface collection) analysed were 133.

The present study reveals the presence of altogether 115 number of phytoplanktonic species of which 65 are from the diatoms followed by 19 of green algae (Chlorophyceae), 9 of blue greens (Cyanophyceae) and 22 of Dinoflagellates from the surface waters.

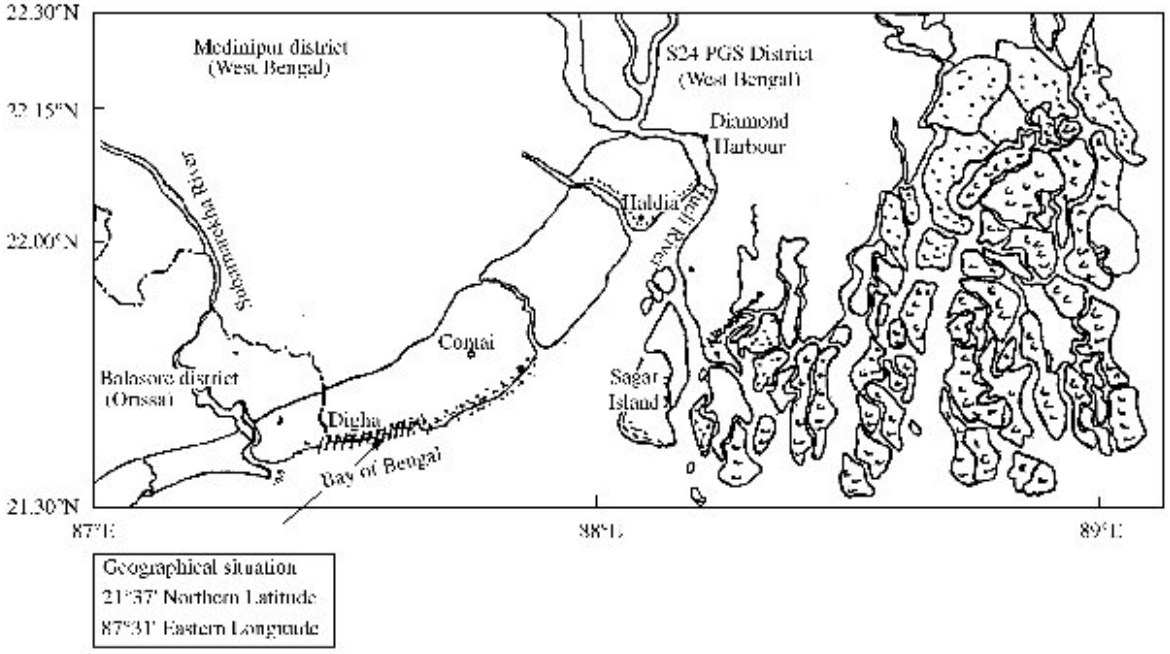

FIG. 1. Map of Coastal Region of West Bengal and part of Orissa, India. (Source: CIFRI, Barrackpore, India.) Region investigated (//////).

Previous study on harmful algal population in an area is important for an early warning system and the knowledge of the threshold point of each species is of utmost need to avoid any toxicity to the standing crop or fishermen who are engaged in harvesting and trade. In this regard an attempt has also been made to identify such harmful algal species which have an adverse effect on the other biota present in the system. Table 1 gives the list of the harmful phytoplankton observed in the years 2000 and 2001.

Harmful effects and allelopathic effects of planktonic algae are the recent topics in aquatic research. It is to be noted that bloom-forming phytoplankton are directly harmful to the other marine biota including humans. Six species of the diatoms, as examined presently in both the surface and subsurface water, viz. *Chaetoceros* spp., *Skeletonema costatum*, *Cerataulina* spp., *Leptocylindricus* spp., *Nitzschia* spp. and *Phaeocystis* spp. are harmful alga (also noticed by Sournia, 1995). However, Sournia (1995), showed that the dinoflagellates contribute quite a good number of harmful bloom-forming toxic species. In the present study out of total 22 species of dinoflagellates identified both from surface and subsurface water, only three species, viz. *Dinophysis acuta*, *Noctiluca scintillans* and *Prorocentrum* sp. were noticed to be harmful (also noticed by Richardson, 1997).

TABLE 1
List of harmful phytoplankton observed in 2000 *and* 2001 *in our study area*

	Name of the species
1	*Phaeocystis* sp.
2	*Nitzschia* sp.
3	*Favella* sp.
4	*Cerataulina* sp.
5	*Chaetoceros* spp.
6	*Skeletonema costatum*
7	*Thallassiosira* sp.
8	*Skujaella thiebautii*
9	Dinoflagellate group
a	*Noctiluca scintillans*
b	*Dinophysis* sp.
c	*Peridinium* sp.
d	*Prorocentrum* sp.

The blooms of *Noctiluca scintillans* and *Paracalanus* occur in the months of January and December. Figure 2 shows that after the bloom of both the species (see the high peak obtained on the sampling date 20.01.2000), *Noctiluca scintillans* decreases and simultaneously the *Paracalanus* also decreases. Further the population of *Noctiluca scintillans* remains very low up to the sampling date 29.11.2000 and during this period the *Paracalanus* does not show any large change in population. Again on the sampling date 13.12.2000 we observe that both the species attain another high peak and then slowly decrease. This observation indicates that *Noctiluca scintillans* attaining the first peak in January (*Paracalanus* also present in high abundance) starts to release toxic substance, as a result it controls the bloom of *Paracalanus* population and also its own bloom. This phenomenon persists for a long time (probably due to the effect of toxin concentration) and again when there is low concentration of toxin, both the population lead to another bloom and the process continues. Our experimental result is also similar to the observation at Vasilev Bay, where *Paracalanus* sp. decreased drastically since 1987 due to the increase in biomass of *Noctiluca scintillans* (see, Kideys *et al.*, 2001). This result

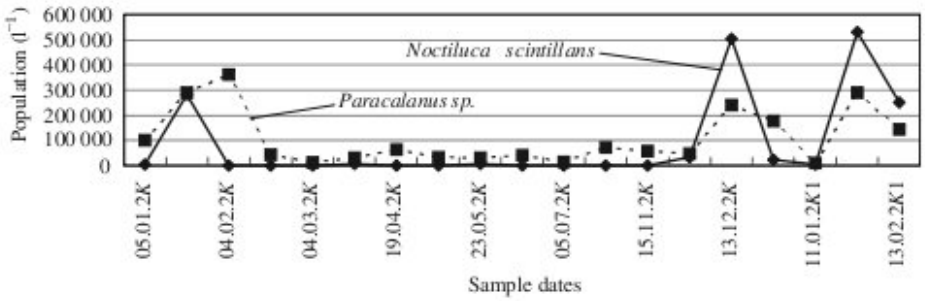

FIG. 2. Field observation during the year 2000–2001.

indicates us that the process of toxin liberation starts only in the presence of dense zooplankton around it and termination of planktonic blooms is the result of toxic chemical(s).

To establish the result statistically we observe the lag correlation or covariation of the two populations. In this case, we have calculated the correlation of *Paracalanus* population with the *Noctiluca scintillans* population at next time points and found that they are negetively correlated. This inverse relationship between *Paracalanus* and *Noctiluca scintillans* is also represented in a scatterplot of the data (see, Fig. 3). Now to test the significance of the observed sample correlation coefficient we performed Student's t-test. If r is the observed correlation coefficient in the sample of n pairs of observations then under the null hypothesis H_0 : $\rho = 0$ (i.e. population correlation coefficient is zero), the statistic

$$t = \frac{r}{\sqrt{1-r^2}}\sqrt{n-2},$$

follows Student's t-distribution with $(n-2)$ degrees of freedom (d.f.). If the value of t comes out to be significant, we refuse H_0 at a certain level of significance adopted, to conclude that

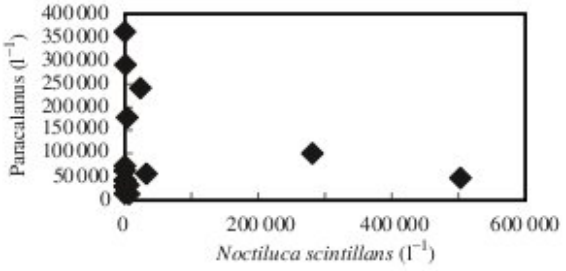

FIG. 3. Scatter diagram for *Noctiluca scintillans* and *Paracalanus* depicting negative correlation.

$\rho \neq 0$ implies that r is significance of correlation in the population. Our statistical result gives that the two species are negetively correlated in the population at 5% level of significance (for details see, Table 2).

General Mathematical Model and Assumptions

In the formulation of the model we assume that the growth of toxin-producing plankton (TPP) population follows the logistic law (see, Murray, 1989; Odum, 1971) with intrinsic growth rate "r" and environmental carrying capacity "K". It is already mentioned that TPP reduces the grazing pressure of its predator, zooplankton. As the fractional changes in the TPP population per unit time effectively illustrates the impact of predation on the population at any particular time, it is interesting to examine the specific predation rate for the system as the outbreak advances (see, Truscott & Brindley, 1994). Keeping the above-mentioned properties in mind two different types of functional forms ($f(P)$ and $g(P)$) have been assumed for the formulation of the model. Here $f(P)$ represents the predational response function and $g(P)$ represents the distribution of toxic substances.

From the above assumptions the following model may be formulated by means of ordinary differential equations:

$$\begin{aligned} \frac{dP}{dt} &= rP\left(1-\tfrac{P}{K}\right) - \alpha f(P)Z, \\ \frac{dZ}{dt} &= \beta f(P)Z - \mu Z - \theta g(P)Z. \end{aligned} \tag{1}$$

Here, P is the density of TPP population and Z is the density of zooplankton population. $\alpha(>0)$ is the rate of predation of zooplankton on TPP population. Further $\beta(>0)$ denotes the ratio of

TABLE 2
Correlation Coefficient and t-test for the observed sample

Species	Pearson's correlation coefficient, r	No. of observation n	Test statistic $\lvert t \rvert$	Degrees of freedom (d.f.)	Tabulated t at 5% level of significance	Remark
Noctiluca & Paracalanus	−0.1134	19	0.4745	17	0.389*	Correlation is significant at 0.05 level (one-tailed)

*For reference see Sheskin (1997).

biomass consumed by zooplankton for its growth and $\mu(>0)$ is the mortality rate of zooplankton due to natural death as well as due to higher predation. θ denotes the rate of toxin liberation by TPP population.

The general basic assumption of the above system and the conditions for the existence of the positive equilibria (or equilibrium) is given in Appendix A.

We have already mentioned that the predational response functions and the toxin liberation processes play an important role in planktonic dynamics. Different possible permutations of these functional forms give us the following nine cases:

Case 1: Both $f(P)$ and $g(P)$ are linear, i.e. $f(P) = g(P) = P$.

Case 2: Both $f(P)$ and $g(P)$ are of Holling type II, i.e. $f(P) = g(P) = P/(\gamma + P)$.

Case 3: Both $f(P)$ and $g(P)$ are of Holling type III, i.e. $f(P) = g(P) = P^2/(\gamma^2 + P^2)$.

Further there may be other cases, which can arise, and these are

Case 4: $f(P)$ is linear and $g(p)$ is of Holling type II, i.e. $f(P) = P$, $g(P) = P/(\gamma + P)$.

Case 5: $f(P)$ is of Holling type II and $g(p)$ is linear, i.e. $f(P) = P/(\gamma + P)$, $g(P) = P$.

Case 6: $f(P)$ is linear and $g(p)$ is of Holling type III, i.e. $f(P) = P$, $g(P) = P^2/(\gamma^2 + P^2)$.

Case 7: $f(P)$ is of Holling type III and $g(p)$ is linear, i.e. $f(P) = P^2/(\gamma^2 + P^2)$, $g(P) = P$.

Case 8: $f(P)$ is of Holling type II and $g(p)$ is of Holling type III, i.e. $f(P) = P/(\gamma + P)$, $g(P) = P^2/(\gamma^2 + P^2)$.

Case 9: $f(P)$ is of Holling type III and $g(p)$ is of Holling type II, i.e. $f(P) = P^2/(\gamma^2 + P^2)$, $g(P) = P/(\gamma + P)$.

In all the above cases γ denotes the half-saturation constant.

As liberation of toxin reduces the growth of zooplankton, causes substantial mortality of zooplankton and in this period TPP population is not easily accessible, hence a more common and intuitively obvious choice is of the Holling type II or type III functional form to describe the grazing phenomena. Moreover, saturation of grazing function allows the TPP population to escape from grazing pressure of zooplankton and forms a tide. This suppression of grazing is usually associated with active hunting behaviour on the part of the predator, as opposed to passively waiting to encounter food. The Holling type II or type III predational form (Ludwig *et al.*, 1978) is an obvious choice to represent the hunting behaviour of predator. In reality, the raptorial behaviour of copepods is highly complex and exhibits a hunting behaviour (Uye, 1986) and hence type II or type III is an appropriate choice.

The main objective for taking into consideration different combinations of functional forms is to understand mechanisms for excitable nature of planktonic blooms and its possible control. If the population exhibits large and rapid response for a suitable perturbation around its equilibrium and in the absence of further perturbation, if it comes back to the original state and remains there, the system may be described as an excitable system (Murray, 1989). Excitability of the system may happen through multistability (in which the system tends to one of the coexisting stable equilibrium) or periodicity (the Hopf-bifurcation, in which the system oscillates around an unstable equilibrium).

Local Stability Analysis

In this section, local stability analysis of the system around the biological feasible equilibria for different cases will be performed. The aim of this part is to find out the suitable mechanism to explain the excitability of planktonic blooms.

We observe that in cases 1 and 4, the trivial equilibrium ($E_0 : (0, 0)$) of the system is always unstable. In case 1, existence of the positive interior equilibrium $E_*(\mu/(\beta - \theta), \frac{r}{\alpha}(1 - P^*/K))$ implies that the axial equilibrium $E_1(K, 0)$ is also an unstable saddle point and non-existence of a positive equilibrium ensures that E_1 is a stable point. Further from the characteristic equation associated with the positive equilibrium, E_* one can easily verify that the system is locally asymptotically stable around the positive equilibrium E_*. The same argument is also valid for case 4. Hence in these cases bloom phenomena of the system cannot be explained.

OCCURRENCE OF BLOOMS THROUGH PERIODICITY

In cases 2, 3 and 5, we observe that the trivial equilibrium is always unstable. It is also

TABLE 3
Local stability analysis of cases 2, 3 and 5

Cases	Functional forms	Conditions for existence of E_*	Parametric relation for which the Hopf-bifurcation occurs
2	$f(P)=\frac{P}{\gamma+P}$	$\theta<\beta-\mu-\frac{\gamma\mu}{K}$	$\beta=\frac{K+\gamma}{K-\gamma}\mu$, when $\theta=0$
	$g(P)=\frac{P}{\gamma+P}$		$\beta=\theta+\frac{K+\gamma}{K-\gamma}\mu$, when $\theta\neq 0$
3	$f(P)=\frac{P^2}{\gamma^2+P^2}$	$\theta<\beta-\mu-\frac{\gamma^2\mu}{K^2}$	$\beta=\mu+\frac{16\mu\gamma^2}{(K+\sqrt{K^2-4\gamma^2})^2}$, when $\theta=0$
	$g(P)=\frac{P^2}{\gamma^2+P^2}$		$\beta=\theta+\mu+\frac{16\mu\gamma^2}{(K+\sqrt{K^2-4\gamma^2})^2}$, when $\theta\neq 0$
5	$f(P)=\frac{P}{\gamma+P}$	$\theta\gamma+\mu<\beta<\min\{(\theta\gamma+\mu+$	$\beta=\mu+\frac{2\mu\gamma}{K-\gamma}$, when $\theta=0$
	$g(P)=P$	$2\sqrt{\theta\gamma\mu}),(\theta\gamma+\mu+2K\theta+\frac{2\mu\gamma}{K})\}$	$\beta=\theta\gamma+\frac{\theta}{2}+\mu+\frac{2\mu\gamma}{K-\gamma}$, when $\theta\neq 0$

Note: It is clear from the above table that the stability region increases due to toxic substance.

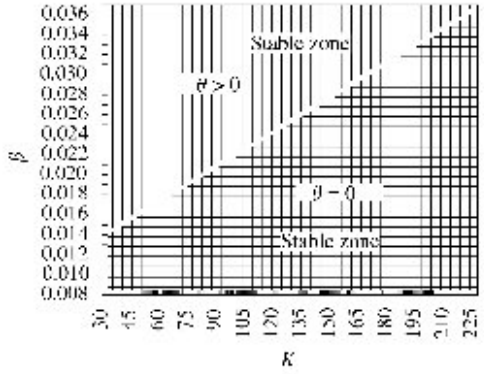

FIG. 4. Region of stability in parametric space.

observed in these cases that the existence of E_* implies that axial equilibrium E_1 is also an unstable saddle point and from the characteristic equation associated with the positive equilibrium E_*, we observe that the Hopf-bifurcation occurs. Existence conditions of E_* and the parametric values at which the Hopf-bifurcation occurs are given in Table 3.

From Table 3, one can easily note that the range of stability increases with the release of toxic chemicals which is also verified with the help of a set of numerical values of the parameters (see Fig. 4).

OCCURRENCE OF BLOOMS THROUGH MULTISTABILITY OF POSITIVE EQUILIBRIUM

We observe that the systems represented in cases 6–9 exhibit planktonic blooms through multistability of the positive equilibrium. Here, we shall present the full analysis of case 6 only and the results of other cases are presented in Table 4.

For case 6, the system (1) reduces to

$$\begin{aligned}\frac{dP}{dt}&=rP\left(1-\frac{P}{K}\right)-\alpha PZ=f(P,Z),\\ \frac{dZ}{dt}&=\beta PZ-\mu Z-\frac{\theta P^2 Z}{\gamma^2+P^2}=g(P,Z).\end{aligned}\tag{2}$$

The system has three equilibria $E_0(0,0)$, $E_1(K,0)$ and $E_*(P^*,Z^*)$.

For the system to exhibit excitable behaviour, it is necessary that the null cline $g(P,Z)=0$ retains its characteristic shape, i.e. that $g(P,Z)=0$ should have two turning points at values of $P>0$. The position of these turning points is the

TABLE 4
Local stability analysis of cases 7–9

Cases	Functional forms	Position of the turning points	Parametric range in which the real roots occur	Behaviour of the system
7	$f(P)=\dfrac{P^2}{\gamma^2+P^2}$	P_{H1}, P_{Z1}	$0<\dfrac{\gamma}{K}<\dfrac{1}{3\sqrt{3}}$	see note 1
	$g(P)=P$			
8	$f(P)=\dfrac{P}{\gamma+P}$	P_{H2}, P_{Z2}	$H_2<0,\ G_2^2+4H_2^3<0$*	see note 2
	$g(P)=\dfrac{P^2}{\gamma^2+P^2}$		$P_{H2}=\dfrac{K-\gamma}{2}$	
9	$f(P)=\dfrac{P^2}{\gamma^2+P^2}$	P_{H3}, P_{Z3}	$0<\dfrac{\gamma}{K}<\dfrac{1}{3\sqrt{3}}$	see note 3
	$g(P)=\dfrac{P}{\gamma+P}$			

* $H_2=\dfrac{\gamma^2[3(\beta-\mu)(\beta-\mu-\theta)-(\mu+\theta)^2]}{9}$, $G_2=\dfrac{\gamma^3(\mu+\theta)(\beta-\mu)(\beta-\mu-\theta)}{3}-\mu\gamma^3(\beta-\mu-\theta)^2-\dfrac{2\gamma^3(\mu+\theta)^3}{27}$.

Note 1: When P_{Z1} is below the lower or above the upper root (P_{H1}), the system behaves excitably. With P_{Z1} falling between the two roots, the system has no stable equilibrium and solutions follow a periodic trajectory. The out breaks are modelled with the system in region of parameter space $P^*<P_{H1}$.

Note 2: When P_{H2} is below the lower or above the upper root (P_{Z2}), the system behaves excitably. With P_{H2} falling between the two roots, the system has no stable equilibrium and solutions follow a periodic trajectory. The out breaks are modelled with the system in region of parameter space $P^*<P_{Z2}$.

Note 3: When P_{Z3} is below the lower or above the upper root (P_{H3}), the system behaves excitably. With P_{Z3} falling between the two roots, the system has no stable equilibrium and solutions follow a periodic trajectory. The out breaks are modelled with the system in region of parameter space $P^*<P_{H3}$.

solutions of $g(P,Z)=0$ which gives

$$\theta P^3-(\mu+\theta)P^2+\beta\gamma^2P-\mu\gamma^2=0. \qquad (3)$$

Clearly the positions of the turning points (say, P_Z) are functions of θ,μ,β and γ. Equation (3) has three real positive roots, which ensures that there are at least two turning points to retain the excitability of the system. By considering the number of real roots of eqn (3) it can be shown that this occurs only within the parameter range

$$H_1<0, G_1^2+4H_1^3<0,$$

where $H_1=(3\beta\gamma^2\theta-(\mu+\theta)^2)/9$ and $G_1=\beta\gamma^2\theta(\mu+\theta)/3-\mu\gamma^2\theta^2-2(\mu+\theta)^3/27$.

The null cline $f(P,Z)=0$ occurs on the line $P=P_H$ (say) and from $\partial f/\partial P=0$ along with $f(P,Z)=0$ we get

$$P_H=K\left(\frac{1-\alpha}{2-\alpha}\right). \qquad (4)$$

The system is capable of exhibiting two different types of behaviour, depending upon the position of P_H with respect to the two roots of eqn (3). When P_H falls below the lower root or above the upper root, the system behaves excitably. When P_H falls between the two roots, the system has no stable equilibrium and solutions follow a periodic trajectory. It can be easily shown that the Hopf-bifurcation separates these regions of parameter space (see Appendix B).

The out breaks are modelled with the system in the region of parameter space defined by

$$P^*<P_Z. \qquad (5)$$

Discussion

Our field observation shows that there is a noticeable effect of TPP on zooplankton and the statistical analysis also shows that both the species are negatively correlated and the effect of toxin in the termination of bloom is expected. To confirm this observation, a mathematical model of TPP–zooplankton system in which the grazing pressure of zooplankton decreases due to the release of toxic substances by TPP species is

proposed and analysed. As the fractional changes in the TPP population have a great impact on predation and also the nature of toxic liberation process is not known, different possible combinations of these functional forms have been considered to search a suitable mechanism for explaining the planktonic blooms. We observe that when $f(P)$, $g(P)$ are linear and also $f(P)$ is linear, $g(P)$ is Holling type II, the bloom phenomena have not been observed. But if the uptake functions are homographic type whatever be the distribution of toxic substances (linear or homographic), the excitable nature of blooms through periodicity has been observed. It is also noted that when the toxin liberation process is of Holling type III and uptake function is linear, the excitable nature of blooms through periodicity has been observed under some parametric restrictions (case 6). The above observations show that predational response functions describe the bloom phenomena, whereas the termination of planktonic blooms is the result of toxic chemicals.

To substantiate analytical findings a set of hypothetical parameter values have been considered for numerical simulation (see, Table 5).

We observe that the region of stability increases in $\beta - K$ parametric space due to toxic substances (see Fig. 4), resembles our analytical results (see Table 3). Numerical simulations of case 3 (in other cases we also observe the similar nature) depicts that there is an outbreak (or bloom) of both TPP and zooplankton in the absence of toxic substances (i.e. when $\theta = 0$, see Fig. 5). But in the presence of toxic substances (for $\theta = 0.5$), both the populations attain a stable steady state through decaying oscillations (see Fig. 6). Hence the role of TPP in the termination of planktonic blooms is clear.

TABLE 5
Parameter values used in numerical simulation

Parameters	Symbols	Values
Growth of phytoplankton population (P)	r	2 (day^{-1})
Environmental carrying capacity	K	108 ($l\,day^{-1}$)
Grazing efficiency of zooplankton population (Z)	α	0.7 ($l\,day^{-1}$)
Growth efficiency of zooplankton population (Z)	β	0.6 ($l\,day^{-1}$)
Higher predation on Z or natural death rate	μ	0.012 (day^{-1})
Zooplankton grazing half-saturation coefficient	γ	5.7 (l^{-1})
Toxin-production rate	θ	0.5 (day^{-1})

As mentioned in the Introduction, the presence and abundance of viruses in seawater has been known for long time (at least since 1950). Since then the researchers are trying to answer the question—what do the viruses do in the ocean? Sarno & Forlani (1999) reported that viruses are considered to be important for the termination of phytoplankton blooms. The role of viruses in the termination of algal blooms (*Emiliania huxleyi*, *Micromonas pusilla* and *Noctiluca scintillans*) was modelled by Beltrami and Carroll (1994). The predicted dynamics of an infected monospecific phytoplankton bloom shown in the model resembles that observed in natural blooms. But what controls viral activity in natural waters is still a matter of debate. Finding out the reason for occurrence of planktonic blooms is necessary but knowledge of the mechanisms for controlling the blooms are much more important and research into this direction needs urgent priority. The release of

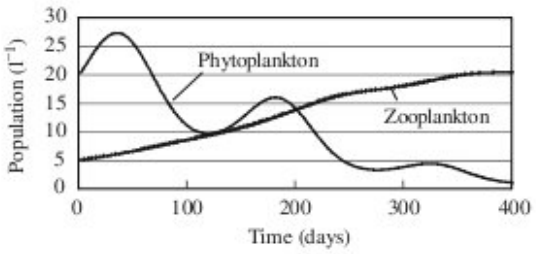

FIG. 5. Numerical solution of case 3 for $\theta = 0$ depicting outbreak (bloom).

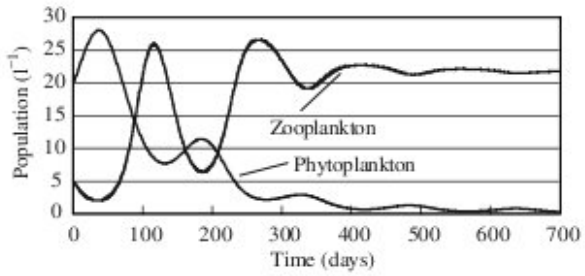

FIG. 6. Numerical solution of case 3 for $\theta = 0.5$ depicting stable state.

toxic substances by TPP may terminate the planktonic blooms, which is not yet well recognized but cannot be ignored. Naturally the interdisciplinary involvement of experimental ecologists and mathematical ecologists is necessary in this issue. The present field study, analytical observation and numerical findings lead us to conclude that toxic substance released by TPP plays an important role in the termination of planktonic blooms. Thus, toxic chemicals not only act as a biocontrol of other plankton species but also have a self-regulatory activity. This sort of control mechanism will be more applicable to closed aquatic systems like ponds, lake, artificial fishery, etc. Before application, the laboratory-based study is necessary to identify the toxic chemical and the amount of doses required for the termination of planktonic blooms in a specified area. This study is presently running being conducted in our laboratory.

The authors are grateful to the Director and Professor-in-charge, Biological Sciences Division of Indian Statistical Institute, Kolkata, India for support. The help rendered by B. Bandyapadhyay (Visiting Scientist), H. Chanda, A. K. Ghosh, T. Modak and N. Banik of Embryology Research Unit is also thankfully acknowledged. The authors are also grateful to their teacher Professor P. K. Tapaswi for his valuable suggestions and comments on this article.

The authors are also very much grateful to the anonymous referee of this paper for his valuable comments and suggestions in improving the standard of the work.

REFERENCES

ANDERSON, D. M. (1989). Toxic algae blooms and red tides: a global perspective. In: *Red Tides: Biology, Environmental Science and Toxicology* (Okaichi, T., Anderson, D. M., & Nemoto, T., eds), pp. 11–21, New York, U.S.A: Elsevier.

BELTRAMI, E. & CARROLL, T. O. (1994). Modelling the role of viral disease in recurrent phytoplankton blooms. *J. Math. Biol.* **32,** 857–863.

BLAXTER, J. H. S. & SOUTHWARD, A. J. (1997). *Advances in Marine Biology*. London, U.K: Academic Press.

BUSKEY, E. J. & STOCKWELL, D. A. (1993). Effects of a persistent "brown tide" on zooplankton population in the Laguna Madre of southern Texas. In: *Toxic Phytoplankton Blooms in the Sea* (Smayda, T. J. & Shimuzu, Y., eds), pp. 659–666, Amsterdam, Netherlands: Elsevier.

HALLEGRAEFF, G. M. (1993). A review of harmful algae blooms and the apparent global increase. *Phycologia* **32,** 79–99.

IVES, J. D. (1987). Possible mechanism underlying copepod grazing responses to levels of toxicity in red tide dinoflagellates. *J. Exp. Mar. Biol. Ecol.* **112,** 131–145.

KIDEYS, A. E., KOVALEV, A. V. & NIERMANN, U. (2001). Results of recent Zooplankton and Ichthyoplankton investigations in the Black Sea. (www.metu.edu.tr)

KIRK, K. & GILBERT, J. (1992). Variations in herbivore response to chemical defences: zooplankton foraging on toxic cyanobacteria. *Ecology* **73,** 2208.

LUDWIG, D., *et al*. (1978). Qualitative analysis of an insect outbreak system: the spruce budworm and forest. *J. Anim. Ecol.* **47,** 315.

MURRAY, J. M. (1990). *Mathematical Biology*. Berlin: Springer Verlag.

NEJSTGAARD, J. C. & SOLBERG, P. T. (1996). Repression of copepod feeding and fecundity by the toxic haptophyte *Prymnesium patelliferum*. *Sarsia* **81,** 339–344.

NIELSEN, T. G., KIØRBOE, T. & BJØRNSEN, P. K. (1990). Effects of a *Chrysochromulina polylepis* sub surface bloom on the plankton community. *Mar. Ecol. Prog. Ser.* **62,** 21–35.

NYBAKKEN, J. W. (1982). *Marine Biology: An Ecological Approach*, pp. 78–81. New York: Harper & Row.

ODUM, E. P. (1971). *Fundamentals of Ecology*. London: W. B. Saunders Company.

RICE, E. L. (1984). *Allelopathy*, 2nd edn, London: Academic Press. pp. 189–205.

RICHARDSON, K. (1997). Harmful or exceptional phytoplankton blooms in the marine ecosystem. In: *Advances in Marine Biology*, (Blaxter, J. H. S. & Southward, A. J., eds) London, U.K: Academic Press. Vol. 31, pp. 301–385.

SARNO, Z. A. D. & FORLANI, G. (1999). Seasonal dynamics in the abundance of *Micromonus pusilla* (Prasinophyceae) and its viruses in the Gulf of Naples (Mediterranean Sea). *J. Plankton. Res.* **21,** 2143–2159.

SHESKIN, D. J. (1997). *Handbook of Parametric and Nonparametric Statistical Procedures*. New York: CRC Press.

SMAYDA, T. (1990). Novel and nuisance phytoplankton blooms in the sea: evidence for a global epidemic. In: *Toxic Marine Phytoplankton* (Graneli, E., Sundstrøm, B., Edler, L., & Anderson, D. M., eds), pp. 29–40, New York, U.S.A: Elsevier.

SOURNIA, A. (1995). Red tide and toxic marine phytoplankton of the world ocean: an enqury into biodiversity. In: *Harmful Marine Algal Blooms* (Lassus, P. *et al*., eds), pp. 103–112. Paris, France: Lavoisier/Intercept.

STEIDINGER, K. A., BUKHOLDER, J. M., GLASGOW, H. B. Jr., TRUBY, E. W., GARETT, J. K., NOGA, E. K. & SMITH, S. A. (1996). *Pfiesteria Piscicida*, a new toxic dinoflagellate genus and species of the order Dinamoebales. *J. Phycol.* **32,** 157–164.

STOERMER, E. F. & SMOL, J. P. (1999). *The Diatoms*. Cambridge U.K: Cambridge University Press.

TOMAS, C. R. (1997). *Identifying Marine Diatoms and Dinoflagellates*, pp. 858. San Diego, CA: AP.

TRUSCOTT, J. E. and BRINDLEY, J. (1994). Ocean plankton populations as excitable media. *Bull. Math. Biol.* **56,** 981–998.

UYE, S. (1986). Impact of copepod grazing on the red tide flagellate *Chattonella antiqua*. *Mar. Biol.* **92,** 35.

APPENDIX A

The general basic assumptions of system (1) are:

(i) f and g are increasing,
(ii) $P(0)>0$, $Z(0)>0$.

Equation (1) has invariant subset: $Z=0$. In this case the equation reduces to the logistic equation for the phytoplankton with two equilibria $P=0$ (unstable) and $P=K$ (stable). Thus, the point (0,0) is unstable with respect to the full system, while the point $(K,0)$ will be stable or unstable according to whether the expression

$$\beta f(K)-\mu-\theta g(K)<0 \quad \text{or} \quad >0. \tag{A.1}$$

The equations for the positive equilibrium (or equilibria, if any) are

$$rP\left(1-\frac{P}{K}\right)-\alpha f(P)Z=0 \quad \text{and}$$
$$\beta f(P)Z-\mu Z-\theta g(P)Z=0$$

Looking for the solutions with $Z\neq 0$ and $P\neq 0$, the first equation yields

$$Z=\frac{rP(1-P/K)}{\alpha f(P)}, \tag{A.2}$$

while the second one may be written as

$$\beta f(P)-\mu-\theta g(P)=0. \tag{A.3}$$

Note that with the general assumption if the equilibrium $(K,0)$ is stable, then eqn (A.3) does not have any solution. Assuming now that $(K,0)$ is unstable, in this case $(A1)$ is positive, then system (A.2)–(A.3) may have solutions. Further from eqn (A.3) we have $\beta f(P)-\theta g(P)=\mu$ and the positivity of μ ensures

$$\beta f(P)>\theta g(P). \tag{A.4}$$

Now looking at eqn (A.1), we can see that the right-hand side is expressed in terms of $P(1-P/K)$, which is not a monotone function. The graph of that function on the interval $[0,K]$ (the only interval which matters here) is a piece of parabola with maximum at $P=K/2$.

We assume that (P^*,Z^*) is the positive equilibrium of system (1). The characteristic equation of system (1) around (P^*,Z^*) is

$$\lambda^2-\left[r-\frac{2rP^*}{K}-\alpha f'(P^*)Z^*\right]\lambda$$
$$+\alpha[\beta f'(P^*)-\theta g'(P^*)]f(P^*)Z^*=0. \tag{A.5}$$

From eqn (A.5) we observe that since $f(P^*)>0$ (always, as f is an increasing function and P^* is a positive equilibrium) and from eqn (A.4) and assumption (ii) we get that

$$\beta f'(P^*)>\theta g'(P^*)$$

so

$$\alpha[\beta f'(P^*)-\theta g'(P^*)]f(P^*)Z^*>0. \tag{A.6}$$

Hence, it is obvious that the stability criteria of the system entirely depend on the term $[r-2rP^*/K-\alpha f'(P^*)Z^*]$.

APPENDIX B

The stability of the various equilibria of the system in case 6 can be ascertained by examining the eigen values of the stability matrix J.

$$J=\begin{pmatrix}\dfrac{\partial f}{\partial P} & \dfrac{\partial f}{\partial Z}\\[2ex] \dfrac{\partial g}{\partial P} & \dfrac{\partial g}{\partial Z}\end{pmatrix},$$

where the elements of the matrix are the partial derivatives of f and g defined above.
The resulting values of J at the three equilibrium points are shown below. For $E_0(0,0)$

$$J=\begin{pmatrix} r & 0\\ 0 & -\mu\end{pmatrix}.$$

In this case the eigenvalues of J are clearly r and $-\mu$, indicating $(0,0)$ is a saddle point and unstable. For $E_1(K,0)$,

$$J=\begin{pmatrix} -r & -\alpha K\\ 0 & \beta K-\mu-\dfrac{\theta K^2}{\gamma^2+K^2}\end{pmatrix}.$$

In this matrix, the eigenvalues are $-r$ and $\beta K-\mu-\theta K^2/(\gamma^2+K^2)$. Existence of $E_*(P^*,Z^*)$

implies that the point $(K,0)$ is also an unstable saddle.

The eigenvalues of J at (P^*,Z^*) cannot be obtained by simple inspection. They can be expressed as the solutions of the characteristic equation,

$$\lambda^2 - Trace\, J\lambda + Det.\, J = 0. \qquad \text{(B.1)}$$

From the expressions for the derivatives above, it can be seen that $Trace\, J = \partial f/\partial P$ and $Det.\, J > 0$ at (P^*,Z^*) [since $2\theta\gamma^2 PZ/(\gamma^2+P^2)^2 \geqslant \beta Z$ can never be a solution of the system, thus $\partial g/\partial P > 0$ always]. The real parts of the eigenvalues of J are therefore both of the same sign. The equilibrium is consequently not a saddle point, but either stable or unstable depending on the sign of $\partial f/\partial P$. Values of (P^*,Z^*) at which $\partial f/\partial P = 0$ are points at which the eigenvalues of J have zero real part, i.e. the Hopf-bifurcation. These points also fulfil the conditions in eqn (3) and are therefore also the turning points of the null cline $g(P,Z) = 0$.