A delay differential equation model on harmful algal blooms in the presence of toxic substances

J. CHATTOPADHYAY* AND R. R. SARKAR

Embryology Research Unit, Indian Statistical Institute, 203, B.T. Road, Kolkata 700108, India

AND

A. EL ABDLLAOUI

Département de Mathématiques et Informatique, Faculté des Sciences, Université M^ed V de Rabat, Maroc

[Received on 15 July 2000; revised on 7 August 2001; accepted on 23 September 2002]

The periodic nature of blooms is the main characteristic in marine plankton ecology. Release of toxic substances by phytoplankton species or toxic phytoplankton reduce the growth of zooplankton by decreasing grazing pressure and have an important role in planktonic blooms. A simple mathematical model of phytoplankton–zooplankton systems with such characteristics is proposed and analysed. As the process of liberation of toxic substances by phytoplankton species is still not clear, we try to describe a suitable mechanism to explain the cyclic nature of bloom dynamics by using different forms of toxin liberation process. To substantiate our analytical findings numerical simulations are performed and these adequately resemble the results obtained in our field study.

Keywords: phytoplankton; zooplankton; toxin; bloom; Hopf bifurcation.

1. Introduction

Plankton are the basis of all aquatic food chains and phytoplankton in particular occupy the first trophic level. Phytoplankton do huge services for our earth: they provide food for marine life, oxygen for human life and also absorb half of the carbon dioxide which may be contributing to global warming (Duinker & Wefer, 1994). The dynamics of rapid (massive) increase or almost equally decrease of phytoplankton populations is a common feature in marine plankton ecology and known as bloom. This phenomenon can occur in a matter of days and can disappear just as rapidly. In recent years there has been considerable scientific attention towards harmful algal blooms (HABs) (e.g. Blaxter & Southward, 1997; Stoermer & Smol, 1999). Several authors have argued that there has been a global increase in harmful phytoplankton blooms in recent decades (e.g. Anderson *et al.*, 1990; Smayda, 1990; Hallegraeff, 1993). In a broad sense planktonic blooms may be categorized into two types, 'spring blooms' and 'red tides'. Spring blooms occur seasonally as a result of changes in temperature or nutrient availability which are connected with seasonal changes in thermocline depth and strength, and consequent mixing. Red tides are the result of

localized outbreaks associated with water temperature (see Truscott & Brindley, 1994). They are also associated with greater stability of the water column and higher growth rates.

Blooms of blue-green algae have been linked to health problems ranging from skin irritation to liver damage depending on time and duration of exposure. The livelihood of many fish and shellfish have also been endangered due to toxin. Blooms of red tide produce chemical toxins, a type of paralytic poison which can be harmful to zooplankton, finfish, shellfish, fish, birds, marine mammals and humans also. Only a few dozen of the many thousands of species of microscopic or macroscopic algae are repeatedly associated with toxic or harmful blooms. Some species, such as the dinoflagellate *Alexandrium tamarense* and the diatom *Pseudo-nitzschia australis* (Work *et al.*, 1993) produce potent toxins which are liberated into the water before they are eaten and they may well affect zooplankton when they are in water. It is now well established that a significant 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* (see Steidinger *et al.*, 1996; Nielsen *et al.*, 1990; Aure & Rey, 1992; Hallegraeff, 1993).

Reduction of grazing pressure of zooplankton due to release of toxic substances by phytoplankton is one of the most vital parameters in this context (see Keating, 1976; Lefevre *et al.*, 1952; Kirk & Gilbert, 1992; Fay, 1983). 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 (Wyatt & Horwood, 1973; Levin & Segel, 1976; Uye, 1986). Areas rich in some phytoplankton organisms, e.g. *Phaeocystis*, *Coscinodiscus*, *Rhizosolenia*, are unaccepted/avoided by zooplankton due to dense concentration of phytoplankton or the production of toxic as well as unpleasant factors by them and this phenomena can be well explained by the 'exclusion' principle (see Odum, 1971; Boney, 1976). 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* off the southern Texas coast. Toxicity may be a strong mediator of zooplankton feeding rate, as shown in both field studies (Estep *et al.*, 1990; Nielsen *et al.*, 1990; Hansen, 1995) and laboratory studies (Huntley *et al.*, 1986; Ives, 1987; Buskey & Hyatt, 1995; Nejstgaard & Solberg, 1996). These observations indicate that the toxic substance as well as toxic phytoplankton plays an important role in the growth of the zooplankton population and has a great impact on phytoplankton–zooplankton interactions.

The process of production of toxic substances by phytoplankton species is still not clear. Modelling on plankton communities in HABs is very rare in the literature. Franks (1997) reviewed different models which describe the phenomenon of red tide outbreak. To our knowledge, in describing bloom phenomena HAB models do not take into account the effect of toxin which causes the grazing pressure of zooplankton to decrease. The release of toxic substances by phytoplankton may terminate the planktonic blooms—something which is not yet well recognized but cannot be ignored; naturally, interdisciplinary involvement of experimental ecologists and mathematical ecologists is necessary. This study is devoted to establishing the role of toxin in the reduction of grazing pressure of zooplankton with the help of both field study and mathematical modelling. We believe that it is the first model in this direction. Monitoring of plankton population was carried

out throughout the year 2000 off the north west coast of the Bay of Bengal. As we are interested to report the effect of toxic phytoplankton on zooplankton, we chose *Noctiluca scintillans* (phytoplankton) and *Paracalanus* (zooplankton) for this study. Motivated by our field observations, a mathematical model of the phytoplankton–zooplankton system in which the grazing pressure of zooplankton decreases due to toxic phytoplankton species is proposed and analysed. As the process of toxin liberation is still not clear, we shall try to explain the bloom dynamics by assuming various forms of toxin liberation process and also by the cyclic nature of the system through periodicity.

2. Formulation of the model

2.1 *Observational background*

In this study we concentrate our observations on the effects of harmful phytoplankton on planktonic blooms, and on what follows. The study area extends from Talsari (Orissa, India) to Digha Mohana (West Bengal, India) on the north west coast of the Bay of Bengal (geographically the area is situated between 21°37′ Northern Latitude, 87°25′ Eastern Longitude and 21°42′ Northern Latitude, 87°31′ Eastern Longitude, see Fig. 1). The study was carried out during the period Jan–Dec 2000. Samplings were done aboard a 10 m fishing vessel hired from the Talsari fish landing centre. Frequency of sampling was every fortnight except for the months of September and October when, because of the roughness of the sea, the 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 0.3 m in diameter. The collected samples were preserved in 3% formaldehyde in seawater. Counting of phytoplankton was done under microscope using a Sedgewick–Rafter counting cell and counts are expressed in no/litre. Identification of the plankton community was done following Davis (1955); Newell & Newell (1979) and Tomas (1997). There were altogether 16 sampling days in the year 2000. Numbers of samples (surface collection) analysed were 112. The study reveals the presence of altogether 115 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. In each group there were some unidentified species. Out of the total 22 species of Dinoflagellates identified both from surface and subsurface water, only three species (*Dinophysis acuta, Noctiluca scintillans* and *Prorocentrum sp.*) were noted as harmful (Richardson, 1997). Six species of the diatoms examined in both the surface and subsurface water (*Chaetoceros spp., Skeletonema costatum, Cerataulina spp. Leptocylindricus spp., Nitzschia spp.* and *Phaeocystis spp.*) are believed to be harmful alga (Sourina, 1995).

Our tested phytoplankton species is *Noctiluca scintillans* belonging to the group Dinoflagellates, which is also capable of producing toxin that are released into the seawater. Among zooplankton species we chose *Paracalanus* belonging to the group Copepoda which dominates the zooplankton in community in all the world oceans, and is the major herbivore which determines the form of the phytoplankton curve. The blooms of *Noctiluca scintillans* occur in January and December. *Paracalanus* bloom also coexists with *Noctiluca scintillans*. 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*

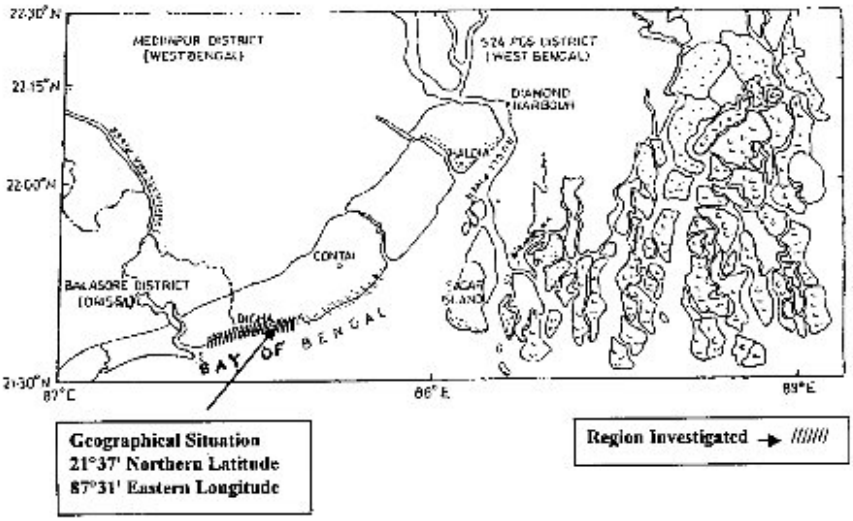

FIG. 1. Map of coastal region of West Bengal and part of Orissa, India. (Source: CIFRI, Barrackpore, India.)

decreases and simultaneously the *Paracalanus* also decreases. The population of *Noctiluca scintillans* then 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. On the sampling date 13/12/2000 we again observe that both species attain another high peak and then slowly decrease. This observation indicates that *Noctiluca scintillans* attaining the first peak in January (with *Paracalanus* also present in high abundance) starts to release toxic substance, and 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) until there is again a low concentration of toxin, both populations again bloom and the process continues. Our experimental result is similar to the observation at Vasilev Bay, where *Paracalanus sp.* decreased drastically after 1987 due to increase in biomass of *Noctiluca scintillans* (see Kideys *et al.*, 2001). Although the chemical toxin released by phytoplankton is not yet tested, the results of our field observations as well as what is already known motivaties us to formulate a mathematical model on the phytoplankton–zooplankton system in which the grazing pressure of zooplankton decreases due to release of toxic substances by the phytoplankton species. It may be noted that the reduction of grazing pressure of zooplankton due to release of toxic substances will have an important role in the termination of planktonic bloom—our analysis bears out this fact very nicely.

2.2 *Mathematical model*

In the formulation of the model we assume 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 already confirmed by our field observation and the literature that toxic substances released by phytoplankton reduce the grazing pressure

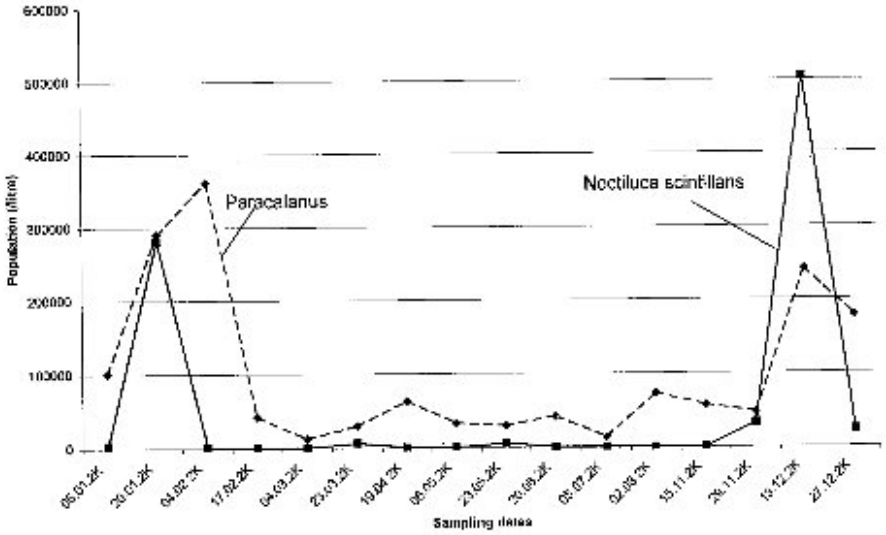

FIG. 2. Field observation during the year 2000.

of its predator, zooplankton. As the fractional changes in the phytoplankton 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). It is well known that at certain times, conditions (adequate temperature, proper light intensity, warmer water and minimal predation pressure) are adequate for planktonic growth. The phytoplankton will continue to bloom until one or more of the key factors prompting phytoplankton growth is no longer available. Keeping the above-mentioned facts of phytoplankton–zooplankton population in mind, we assume two predational forms for describing the dynamics. When phytoplakton populations do not produce toxin, we assume that the predation rate will follow the simple law of mass action. But as liberation of toxin reduces the growth of zooplankton, it causes substantial mortality of zooplankton and in this period phytoplankton population is not easily accessible, hence a more common and intuitively obvious choice is of the Holling type II functional form to describe the grazing phenomenon. Moreover, saturation of grazing function allows the phytoplankton population to escape from the grazing pressure of the zooplankton and form 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, and there is a maximum rate of consumption per individual however large the phytoplankton population becomes. 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.

From the above assumptions we can write down the following differential equations:

$$\begin{cases} \dfrac{dP}{dt} = rP\left(1 - \dfrac{P}{K}\right) - \alpha PZ \\ \dfrac{dZ}{dt} = \beta PZ - \mu Z - \dfrac{\theta P}{\gamma + P}Z. \end{cases} \tag{1}$$

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. $\mu(> 0)$ 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:

$$P(0) > 0, Z(0) > 0. \tag{2}$$

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

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

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

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

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

We first observe 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 non-negative, 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 $P(0) > 0$. A similar argument follows for $Z = 0$ from the second equation of system (1).

Now, we consider the boundedness of solutions of system (1).

LEMMA 2.1 All the solutions which initiate in $\{R_+^2 \backslash 0\}$ are uniformly bounded.

Proof. We define a function

$$W = \beta P + \alpha Z. \tag{6}$$

The time derivative of (6) along the solutions of (1) is

$$\frac{dW}{dt} = r\beta P\left(1 - \frac{P}{K}\right) - \alpha\mu Z - \frac{\beta\theta P}{\gamma + P}Z \tag{7}$$

$$\leqslant r\beta P\left(1 - \frac{P}{K}\right) - \alpha\mu Z \tag{8}$$

$$= [r\beta P\left(1 - \frac{P}{K}\right) + \mu\beta P] - \mu W. \tag{9}$$

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

$$\frac{\mathrm{d}W}{\mathrm{d}t}+\mu W \leqslant C \tag{10}$$

$$\text{where } C=\frac{\beta K(\mu+\gamma)^2}{4r}. \tag{11}$$

Applying the theorem of differential inequality (Birkhoff & Rota, 1982), we obtain

$$0 < W(P,Z) \leqslant \tfrac{C}{\mu}(1-\mathrm{e}^{-\mu t})+W(P(0),Z(0))\mathrm{e}^{-\mu t}$$

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

$$0 < W \leqslant \frac{[\beta K\mu(1+\frac{\gamma}{\mu})^2/4]}{r}.$$

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

$$B=\left\{(P,Z)\in R_+{}^2 : W=\frac{C}{\mu}+\epsilon\right\},$$

for any $\epsilon > 0$ and for t large enough. □

Note. The upper bound of W implies that the linear combination of phytoplankton–zooplankton population is less than a finite quantity which is determined by the ratio of the effective growth rate of zooplankton to the net growth rate of phytoplankton.

Before analysing the model system, we would like to mention the meaning of the periodic nature of blooms. It is well established that the occurrence of more than one bloom in a season suggests that the features influencing a red tide event are cyclic (e.g. see Satora & Laws, 1989). The periodic nature of blooms, in the sense of the rapid onset and disappearance of oscillations under supposedly favourable environmental condition, is one of the main characteristics in plankton ecosystems. This may happen in two ways: namely multistability, in which the system tends to one of the coexisting stable equilibria, and periodicity (Hopf bifurcation), in which the system oscillates around an unstable equilibrium. At this point it may be mentioned that an external forcing agent in a proper measure can also bring out the essential physicalities of the system under study. But we feel that such an addition only suppresses a proper understanding of the system. This is because, given the extent of the regulatory behaviour shown by the system, the external forcing agent remains to a large extent arbitrary and needs very fine tuning for which there may not be any adequate explanation.

Hence we are trying to explore a suitable mechanism for planktonic blooms which is present within the system.

3. Stability analysis

Local stability analysis (LAS) of system (1) around the equilibria can be studied by computing a 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. Violation 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

$$\lambda^2 - M\lambda + N = 0 \tag{12}$$

where

$$M = -\frac{rP^*}{K} (< 0) \tag{13}$$

$$N = \frac{\alpha P^* Z^*}{(\gamma + P^*)^2} [\beta(\gamma + P^*)^2 - \theta\gamma]. \tag{14}$$

It can be easily verified from the second equation of system (1) that $\theta \geqslant \frac{\beta(\gamma+P^*)^2}{\gamma}$ can never be a solution of the system, hence simple bifurcation is also not possible in this case. Now since $M < 0$, the system (1) around $E^*(P^*, Z^*)$ is locally asymptotically stable.

To investigate the global behaviour of system (1) we first prove that system (1) around E^* has no nontrivial periodic solutions. The proof is based on an application of a divergence criterion (Hale, 1993).

Let $h(P, Z) = \frac{1}{PZ}$. Obviously $h(P, Z) > 0$ if $P > 0, Z > 0$.

We define

$$g_1(P, Z) = rP\left(1 - \frac{P}{K}\right) - \alpha PZ \tag{15}$$

$$g_2(P, Z) = \beta PZ - \mu Z - \frac{\theta PZ}{\gamma + P} \tag{16}$$

$$\Delta(P, Z) = \frac{\partial}{\partial P}(g_1 h) + \frac{\partial}{\partial Z}(g_2 h), \tag{17}$$

and find that

$$\Delta(P, Z) = -\frac{r}{KZ} \tag{18}$$

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.

Now, we are in a position to prove the following theorem.

THEOREM 3.1 Existence of a positive interior equilibrium ensures that system (1) around $E^*(P^*, Z^*)$ is globally asymptotically stable.

Proof. The proof is based on the following arguments:

(a) System (1) is bounded and positively invariant in the first quadrant if $\theta < (\beta K + \beta\gamma - \mu) - \frac{\gamma\mu}{K}$.
(b) Trivial equilibrium E_0 is always an unstable saddle point and existence of a 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.

From the above observations we find that there is no chance of exchange of stability. Hence the cyclic nature of the bloom phenomenon which is very common in marine phytoplankton–zooplankton systems cannot be explained by the above mechanism. At this stage we wish to mention that various combinations of predational functional response and toxin liberation process give rise to exchange of stability through Hopf bifurcation or multistability of the positive equilibrium. But in this study we are mainly interested in presenting a mechanism for planktonic blooms in which the liberation of toxic substance or the effect of toxic phytoplankton is not an instantaneous process but is mediated by some time lag.

4. Model with distributed delay

We assume that the liberation of toxic substances by phytoplankton species is not an instantaneous process but is mediated by some time lag required for maturity of the species. There are also several reports that the zooplankton mortality due to the toxic phytoplankton bloom occurs after some time lapse (see `http://www.mote.org`, `http://www.mdsg.umd.edu`). Our field observation also suggests that the abundance of *Paracalanus* (zooplankton) population reduces after some time lapse of the bloom of toxic phytoplankton *Noctiluca scintillans* (see Fig. 2) and this allows us some considerable freedom for considering the delay factor in the model construction.

It is not usually possible to know the past history of the release of toxic substances by phytoplankton or the actual form of the delay kernel. So a particular member of the family of kernels is at best an approximation. To search for excitability (and/or) a cyclic nature of blooms in the system we now assume that the release of toxic substances by the phytoplankton population follows a gamma distribution. This form of distributed delay kernel has been widely used in biological modelling (see Cushing, 1997; MacDonald, 1978, and references therein) and seems to be the most useful family of reducible kernels (i.e. delay kernels that allow a distributed delay model to be converted to an equivalent system of ordinary differential equations). These kernels are not only mathematically convenient, but also linear combinations of them represent a generic class of distributed delay kernels (see Busenberg & Travis, 1982). In this case system (1) can be represented as

$$\begin{cases} \dfrac{\mathrm{d}P}{\mathrm{d}t} = rP\left(1-\dfrac{P}{K}\right) - \alpha PZ \\ \dfrac{\mathrm{d}Z}{\mathrm{d}t} = \beta PZ - \mu Z - \theta\left[\displaystyle\int_{-\infty}^{t} \sigma^{k+1}\frac{(t-s)^k}{k!}\mathrm{e}^{-\sigma(t-s)}\frac{P(s)}{\gamma+P(s)}\,\mathrm{d}s\right]Z. \end{cases} \tag{19}$$

Here k, a non-negative integer, is the order of the delay kernel and σ, is real non-negative. These are linked to the mean time lag by $T = \frac{k+1}{\sigma}$. It is interesting to note that when the value of k increases then the phytoplankton consumed in the past by zooplankton become more important compared to the case when k is small. In particular, when $k = 1$ we have a strong kernel and when $k = 0$ we have a weak kernel in the memory function. This system also possesses the same equilibria as in system (1).

Stability analysis of each equilibrium can be performed by using a variational matrix. The behaviour of this system around E_0 and E_1 is the same as we observed in the previous case.

The characteristic equation of system (19) around $E^*(P^*, Z^*)$ is

$$\lambda\left(\lambda + \frac{rP^*}{K}\right) - \frac{\theta\alpha\gamma P^*Z^*}{(\gamma + P^*)^2}G_k(\lambda) + \alpha\beta P^*Z^* = 0 \tag{20}$$

where

$$G_k(\lambda) = \int_{-\infty}^{t} \sigma^{k+1}\frac{(t-s)^k}{k!}\mathrm{e}^{-(\sigma+\lambda)(t-s)}\,\mathrm{d}s. \tag{21}$$

We shall study system (19) with $k = 1$.

In this case $G_1(\lambda) = \left(\frac{\sigma}{\lambda+\sigma}\right)^2$ and the characteristic equation becomes

$$\lambda\left(\lambda + \frac{rP^*}{K}\right)(\lambda+\sigma)^2 - \frac{\theta\alpha\gamma P^*Z^*}{(\gamma + P^*)^2}\sigma^2 + \alpha\beta P^*Z^*(\lambda+\sigma)^2 = 0. \tag{22}$$

Equation (22) can be written in the form

$$\lambda^4 + f_1(\sigma)\lambda^3 + f_2(\sigma)\lambda^2 + f_3(\sigma)\lambda + f_4(\sigma) = 0$$

where

$$f_1(\sigma) = 2\sigma + \frac{rP^*}{K} \tag{23}$$

$$f_2(\sigma) = \sigma^2 + \frac{2\sigma rP^*}{K} + \alpha\beta P^*Z^* \tag{24}$$

$$f_3(\sigma) = \frac{\sigma^2 rP^*}{K} + 2\alpha\beta\sigma P^*Z^* \tag{25}$$

$$f_4(\sigma) = \alpha\beta\sigma^2 P^*Z^* - \frac{\theta\alpha\gamma\sigma^2 P^*Z^*}{(\gamma + P^*)^2}. \tag{26}$$

By using the Routh–Hurwitz criterion, we find that the real part of all roots are negative. So in this case also there is no possibility for exchange of stability. Hence the cyclic nature of blooms cannot be explained by this type of distribution of toxic substance or toxic phytoplankton. The prediction based on the system involving distributed delay illustrates that concentration of toxic substances or toxic phytoplankton eventually approaches equilibrium concentration and hence no periodic solutions are possible. It is also worth noting that if the order (k) of the delay kernel, goes to infinity while keeping the mean delay, $T = \frac{k+1}{\sigma}$ fixed, then the distributed delay can be viewed as a discrete delay (for details see Wolkowicz *et al.*, 1997). Now, to explain the periodic nature of bloom phenomena we shall assume the process of toxic liberation as a break-even point by discrete delay.

5. Model with discrete delay

We now assume that the process of toxic liberation follows a discrete time variation. System (1) now takes the form

$$\left.\begin{aligned}\frac{dP}{dt} &= rP\left(1-\frac{P}{K}\right)-\alpha PZ\\ \frac{dZ}{dt} &= \beta PZ-\mu Z-\frac{\theta P(t-\tau)}{\gamma+P(t-\tau)}Z\end{aligned}\right\} \tag{27}$$

where τ is the discrete time delay.

As in the previous two cases system (27) has the same equilibria. System (27) around E_0 is an unstable saddle. Existence of E^* implies that E_1 is also an unstable saddle. Non-existence of E^* implies that E_1 is stable in nature.

To investigate local asymptotic stability of system (27) around E^* we perturb the system (27) around $E^*(P^*, Z^*)$ and obtain the following system of differential equations:

$$\begin{aligned}\frac{dx}{dt} &= Ax+By+a_{11}xy+a_{20}x^2\\ \frac{dy}{dt} &= Cx+Ex(t-\tau)+b_{11}xy+b'_{11}\frac{x(t-\tau)y}{\gamma+x(t-\tau)}+b'_{12}\frac{x(t-\tau)}{\gamma+x(t-\tau)}\end{aligned} \tag{28}$$

where

$$\begin{aligned}&x=P-P^*,\ y=Z-Z^*,\ A=-\frac{rP^*}{K},\ B=-\alpha P^*,\\ &C=\beta Z^*,\ E=-\frac{\theta\gamma Z^*}{(\gamma+P^*)^2},\\ &a_{11}=-\alpha,\ a_{20}=\frac{-r}{K},\ b_{11}=\beta,\\ &b'_{11}=\theta,\ b'_{12}=-\theta Z^*.\end{aligned} \tag{29}$$

Retaining only the linear terms in (28), the linearized system becomes

$$\begin{aligned}\frac{dx}{dt} &= Ax+By\\ \frac{dy}{dt} &= Cx+Ex(t-\tau).\end{aligned} \tag{30}$$

System (30) can be written as

$$\frac{d^2x}{dt^2}-A\frac{dx}{dt}-BCx-BEx(t-\tau)=0. \tag{31}$$

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

$$\Delta(\lambda,\tau)=\lambda^2-A\lambda-BC-BEe^{-\lambda\tau}=0. \tag{32}$$

Now substituting $\lambda = \alpha_1 + i\omega$ in (32) and separating the real and imaginary parts we obtain the system of transcendental equations

$$\begin{aligned} &{\alpha_1}^2 - \omega^2 - A\alpha_1 - BC - BEe^{-\alpha_1\tau}\cos\omega\tau = 0 \\ &2\alpha_1\omega - A\omega + BEe^{-\alpha_1\tau}\sin\omega\tau = 0. \end{aligned} \tag{33}$$

The stability or instability of the system is determined by the sign of those λ satisfying (32) if λ is real or the sign of α_1 satisfying (33) if λ is complex.

THEOREM 5.1 The following are necessary and sufficient conditions for E^* to be asymptotically stable for every $\tau \geqslant 0$:

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

THEOREM 5.2 As $A < 0$ and $B < 0$, then in the parametric region $-E < C$ the interior equilibrium E^* of system (27) is locally asymptotically stable for $0 < \tau < \frac{\pi}{\omega_0}$.

Proof. From (32) it is clear that E^* is asymptotically stable for $\tau = 0$ if $-E < C$.

Proving the second condition of Theorem 3.1 requires the Nyquist criterion and its consequences. Consider (30) and the space of all real-valued continuous functions defined on $[-\tau, \infty)$ satisfying the initial condition $x(t) = 0$ for $-\tau \leqslant t < 0$, $x(0^+) = P_1 > 0$ and $\dot{x}(0^+) = P_2 > 0$. After taking the Laplace transform of (31) and simplifying, we have

$$L(x(s)) \equiv L(s) = \frac{P_1 s + P_2 - AP_1}{s^2 - As - BC - BEe^{-\tau s}}. \tag{34}$$

The inverse Laplace transform of $L(s)$ will have terms which increase exponentially with t if $L(s)$ has poles with positive real part. Thus it is clear that a condition for stability of E^* is that all poles of $L(s)$ have negative real parts. We apply the Nyquist criterion (see Thingstad & Langeland, 1974) to conclude whether $L(s)$ has any pole in the right half-plane. This criterion leads us to the conditions

$$\text{Im } \psi(i\omega_0) > 0 \tag{35}$$

$$\text{Re } \psi(i\omega_0) = 0 \tag{36}$$

where

$$\psi(s) = s^2 - As - BC - BEe^{-\tau s}, \tag{37}$$

with ω_0 the smallest positive value of ω for which (36) holds. Now,

$$\psi(i\omega_0) = -{\omega_0}^2 - iA\omega_0 BC - BE(\cos\omega_0\tau - i\sin\omega_0\tau) \cdot \tag{38}$$

$$\text{Im } \psi(i\omega_0) = -A\omega_0 + BE\sin\omega_0\tau \tag{39}$$

and

$$Re\ \psi(i\omega_0) = -{\omega_0}^2 - BC - BE\cos\omega_0\tau. \tag{40}$$

Writing conditions (35) and (36) using the expressions (39) and (40) and taking account of $B < 0$ and $E < 0$ we obtain $\frac{A}{BE\tau} < \frac{\sin\omega_0\tau}{\omega_0\tau}$ and ${\omega_0}^2 = -BC - BE\cos\omega_0\tau$.

Since $A < 0$, $-(BE) < 0$, condition (35) is satisfied for $0 < \tau < \frac{\pi}{\omega_0}$.

Further since $B < 0,\ E < 0$ we have

$$-BC - BE < -BC - BE\cos\omega_0\tau < -BC + BE.$$

Hence $z = {\omega_0}^2$ and $z = -BC - BE\cos\omega_0\tau$ intersect on $0 < \omega_0 < \frac{\pi}{\tau}$. From (40) we also have (in the parametric region $-E < C$)

$$0 < -BC - BE < {\omega_0}^2 < -BC + BE, \text{ for } 0 < \omega_0 < \frac{\pi}{\tau} \tag{41}$$

so we have an upper bound ω_+ of ω_0 given by

$$\omega_+ = \sqrt{BE - BC}. \tag{42}$$

Hence we can conclude that in our case the Nyquist criterion holds and the interior equilibrium E^* of the system (27) is locally asymptotically stable for all values of τ satisfying $0 < \tau < \frac{\pi}{\omega_0}$. □

6. Bifurcation of the solutions

In this section we state a condition under which the system goes through a point where a Hopf bifurcation occurs. We show the existence of such a $\tau\ (= \tau_0)$ and $\omega\ (= \omega_0)$.

LEMMA 6.1 If $A^2 + 2BC < 0$ and $0 \leqslant C < -E$ then there exists a unique pair $(\omega_0,\ \tau_0)$ 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 (46) and (51), respectively.

Proof. From $\Delta(\mathrm{i}\omega_0, \tau_0) = 0$ and from (39) and (40) we have

$$-A\omega_0 + BE\sin\omega_0\tau_0 = 0 \tag{43}$$

and

$$-{\omega_0}^2 - BC - BE\cos\omega_0\tau_0 = 0. \tag{44}$$

Squaring and adding together (43) and (44) we arrive at

$${\omega_0}^4 + (A^2 + 2BC){\omega_0}^2 + (B^2C^2 - B^2E^2) = 0. \tag{45}$$

We see from (45) that λ has a pair of purely imaginary roots of the form $\pm\mathrm{i}\omega_0$ provided $A^2 + 2BC < 0$ and $0 \leqslant C \leqslant -E$.

The corresponding roots of (45) in this case are

$${\omega_0}^2 = \frac{1}{2}[-(A^2 + 2BC) + \sqrt{(A^2 + 2BC)^2 - 4(B^2C^2 - B^2E^2)}\ ] \tag{46}$$

Using (43) in (44), we obtain

$$-[\frac{(BE)^2(1 - \cos^2\omega_0\tau_0)}{A^2}] - BC - BE\cos\omega_0\tau_0 = 0$$

or

$$(BE)^2\cos^2\omega_0\tau_0 - BEA^2\cos\omega_0\tau_0 - BCA^2 - (BE)^2 = 0. \tag{47}$$

Set

$$f(z) = (BE)^2z^2 - BEA^2z - BCA^2 - (BE)^2 = 0. \tag{48}$$

We have

$$f(1) = -BE^2(C + E) < 0 \tag{49}$$

and

$$f(-1) = -BE^2(C - E) > 0. \tag{50}$$

Hence $f(z)$ has a real solution in $(-1, 1)$ of the form $\cos\omega_0\tau_0 = k$, where $|k| < 1$. From (43),

$$\tau_0 = \frac{1}{\omega_0}\arcsin\left(\frac{-A\omega_0}{BE}\right) + \frac{2n\pi}{\omega_0},\ n = 0,\ 1,\ 2, \ldots. \tag{51}$$

In (46) we assume $A^2 + 2BC < 0$, so that there is only one imaginary solution $\lambda = i\omega_0$ ($\omega_0 > 0$) and therefore the only crossing of imaginary axis is from left to right as τ increases and the stability of the trivial solution can only be lost and not regained. Obviously in this case $n = 0$. □

LEMMA 6.2 Let $A^2+2BC < 0, 0 \leqslant C < -E$. Then the real parts of the solutions of (32) are negative for $\tau < \tau_0$, where $\tau_0 > 0$ is the smallest value for which there is a solution to (32) 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.

Proof. For $\tau = 0$, it is obvious that E^* is stable. Hence by Butler's lemma (see Freedman & Rao, 1983), E^* remains stable for $\tau < \tau_0$. We have now to show that $\frac{d\alpha_1}{d\tau}|_{\tau=\tau_0} > 0$ where $\omega = \omega_0$ (for $n = 0, 1, 2, \ldots$). This will signify that there exists at least one eigenvalue with positive real part for $\tau > \tau_0$, and hence E^* is unstable for $\tau > \tau_0$. Moreover, the conditions for Hopf bifurcation (see Hale, 1993) are then satisfied yielding the required periodic solutions. Now differentiating (43) with respect to τ, we get

$$\begin{aligned}\{2\alpha_1 - A + BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}\frac{d\alpha_1}{d\tau} + \{-2\omega + BE\tau e^{-\alpha_1\tau}\sin\omega\tau\}\frac{d\omega}{d\tau}\\ = BEe^{-\alpha_1\tau}\{-\alpha_1\cos\omega\tau - \omega\sin\omega\tau\}\end{aligned} \tag{52}$$

and

$$\begin{aligned}\{2\omega - BE\tau e^{-\alpha_1\tau}\sin\omega\tau\}\frac{d\alpha_1}{d\tau} + \{2\alpha_1 - A + BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}\frac{d\omega}{d\tau}\\ = BEe^{-\alpha_1\tau}\{\alpha_1\sin\omega\tau - \omega\cos\omega\tau\}.\end{aligned} \tag{53}$$

Therefore

$$[\{2\alpha_1 - A + BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}^2 - (2\omega - BE\tau e^{-\alpha_1\tau}\sin\omega\tau)\{-2\omega + BE\tau e^{-\alpha_1\tau}\sin\omega\tau\}]\frac{d\alpha_1}{d\tau}$$
$$= BE\tau e^{-\alpha_1\tau}[(-\alpha_1\cos\omega\tau - \omega\sin\omega\tau)\{2\alpha_1 - A + BE\tau e^{-\alpha_1\tau}\cos\omega\tau\}$$
$$-(\alpha_1\sin\omega\tau - \omega\cos\omega\tau)(-2\omega + BE\tau e^{-\alpha_1\tau}\sin\omega\tau)].$$

Now at $\alpha_1 = 0,\ \tau = \tau_0,\ \omega = \omega_0$, we have

$$[\{-A + BE\tau_0\cos\omega_0\tau_0\}^2 + \{2\omega_0 - BE\tau_0\sin\omega_0\tau_0\}^2]\frac{d\alpha_1}{d\tau}|_{(\alpha_1=0,\ \tau=\tau_0,\ \omega=\omega_0)}$$
$$= BE[-\omega_0\sin\omega_0\tau_0\{-A + BE\tau_0\cos\omega_0\tau_0\} + \omega_0\cos\omega_0\tau_0(-2\omega_0 + BE\tau_0\sin\omega_0\tau_0)]$$
$$= BE\omega_0[A\sin\omega_0\tau_0 - 2\omega_0\cos\omega_0\tau_0]$$
$$= BE\omega_0 p_1\cos(\omega_0\tau_0 - \theta_1) > 0 \tag{54}$$

where ${p_1}^2 = A^2 + 4{\omega_0}^2,\ \tan\theta_1 = (\frac{-A}{2\omega_0})$.

Hence $\frac{d\alpha_1}{d\tau}|_{(\alpha_1=0,\ \tau=\tau_0,\ \omega=\omega_0)} > 0$. Therefore the transversality condition holds and hence a Hopf bifurcation occurs at $\omega = \omega_0,\ \tau = \tau_0$. □

The stability of the bifurcating branches is given in the appendix.

7. Discussion

The dynamics of planktonic bloom is very complex and the role of algal toxin in the complex ecology of HABs is still not clear. Researchers are trying to find a suitable mechanism for this. Apart from some noticeable poisoning by phytoplankton, the ecological consequences of algal toxins are also not well elaborated. This allows us some considerable freedom to formulate a mathematical model.

A simple mathematical model of phytoplankton–zooplankton (prey–predator) system in which the grazing pressure of zooplankton reduce due to release of toxic chemical by phytoplankton or due to toxic phytoplankton being eaten by zooplankton has been proposed and analysed. In our study we have tried to establish the following three major processes:

(i) the cyclic nature of the phytoplankton–zooplankton system around the positive equilibrium,
(ii) that phytoplankton start to release toxic chemical or become toxic very quickly in the presence of dense zooplankton population; as a result the grazing pressure decreases, and
(iii) the toxic effect on zooplankton will help in the termination of blooms.

It was stated clearly in the introduction that toxic phytoplankton or toxic chemicals reduce the growth of zooplankton populations and as the process of toxic liberation is still not clear, we have investigated the model under three types of distribution of toxic substances. We have observed that the cyclic nature of blooms which are a very common feature in the planktonic world cannot be explained by our model formulation if the distribution of toxic substances is of Holling type II or if it follows a gamma distribution,

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 & Brindley (1999)*

Parameters	Symbols	Default values	Reported ranges
Maximum P growth rate	r	0·2 (h^{-1})	0·07–0·28 (h^{-1})
Maximum Z grazing rate	α	0·9 (1 h^{-1})	0·6–1·4 (l h^{-1})
Z growth efficiency	β	0·3 (1 h^{-1})	0·2–0·5 (l h^{-1})
Natural death rate of Z	μ	0·02 (h^{-1})	0·015–0·15 (h^{-1})
Z grazing half-saturation coefficient	ν	0·06 (l^{-1})	0·02–0·1 (l^{-1})

whereas if the distribution of toxic substances is of discrete type, we have observed that the system around the positive equilibrium enters a Hopf bifurcation and exhibits the cyclic nature of blooms for a certain amount of time delay. To ascertain this local behaviour we have performed the stability analysis of bifurcating periodic solutions (see the appendix) and obtained the conditions for supercritical or subcritical bifurcation. In most situations, the oscillation phenomena of ecological systems are generally described by distributed delay models. The point is that reduction of grazing pressure on phytoplankton due to release of toxin is not continuous but follows a discrete fashion. The research by JoAnn Burkholder and others at North Carolina State University also reflects our observation. They suggest that *Pfiesteria piscicida* assumes more than 20 different forms during its lifetime, including a difficult-to-detect cyst stage, an amoeboid stage and a toxic vegetative stage, in which, propelled by its flagella, it can kill its predator (see `http://www.mdsg.umd.edu/MarineNotes/Jul-Aug97`).

To substantiate the analytical findings we have used the parameter values which are presented and discussed elaborately in Edwards & Brindley (1999). Abbreviations, default values (which we have used) and the ranges of the parameter values are given in Table 1. For these sets of values and for $\tau = 18$ h, $K = 400\,l^{-1}$ and $\theta = 0{\cdot}9\ h^{-1}$, we have obtained the values of $\beta_1 = 0{\cdot}0223115 - 6{\cdot}337212i$, $\overline{\nu_1} = -0{\cdot}0007769111 + 0{\cdot}07877564i$, $\overline{\nu_2} = 0{\cdot}4992216 + 0{\cdot}004632169i$, $g_{20} = 0{\cdot}5868627 + 3{\cdot}29125i$, $g_{02} = 2{\cdot}349286 + 3{\cdot}496705i$, $g_{11} = -7{\cdot}952592 - 0{\cdot}08011834i$, $g_{21} = 33{\cdot}582 - 13{\cdot}22366i$, $\mathrm{Re}\ C_1(0) = -13{\cdot}89942$, $\mu_2 = 53499{\cdot}1$ and $\tau_2 = 621{\cdot}9235$.

For these sets of parameter values we have obtained $\mu_2 > 0$, the bifurcation is supercritical and the system exhibits a stable limit cycle. Further, since $\tau_2 > 0$, the period of the oscillations increases with τ. Numerical solutions of (27) were carried out using the modified fourth-order Runge–Kutta method. The results indicate that the equilibrium solution is stable (by decaying oscillations) for $0 \leqslant \tau < 18$ and unstable (by growing oscillations) for $\tau > 18$ (see Figs 3 and 4). The system exhibits a stable limit cycle periodic solution at the bifurcation value $\tau_0 = 18$ h (see Fig. 5, which is quite reasonable for the life span of phytoplankton). This observation indicates that there is a threshold limit τ, below which the system shows no excitability and above which the system enters into excitable range. These findings demonstrate the delayed effect of toxic phytoplankton and the cyclic nature of blooms in this phytoplankton–zooplankton system. We would like to mention here that in our field study we observed that the blooms reappear after 10 months whereas our model simulation shows that the blooms reappear after 6 months. The above findings

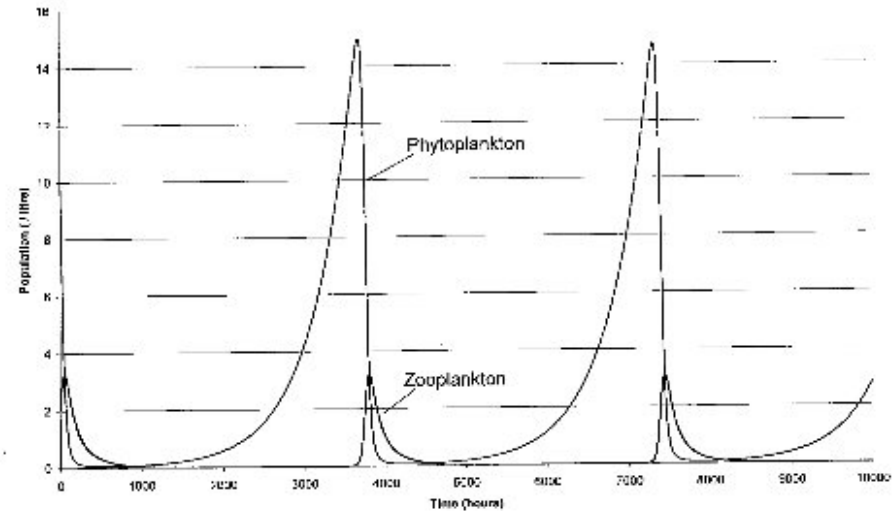

FIG. 3. Numerical solution of equation (9) for $\tau < 18$ h depicting stable situation (decaying oscillation).

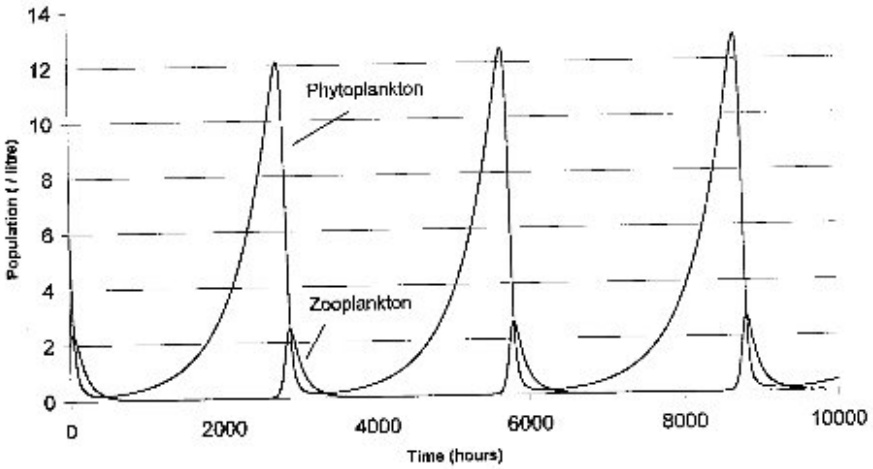

FIG. 4. Numerical solution of equation (9) for $\tau > 18$ h depicting unstable situation (growing oscillation).

show that the toxin producing planktons may act as biological control for the termination of planktonic blooms. Although these results give only qualitative agreement (this may be due to sampling process, environmental factors, etc.) this fact cannot be ignored. We believe that biologists might be interested in this idea and will perform more explicit studies in the laboratory in this direction.

We further observed that when the ratio of initial phytoplankton–zooplankton population was 5 : 1, the system around positive equilibrium exhibits a stable limit cycle

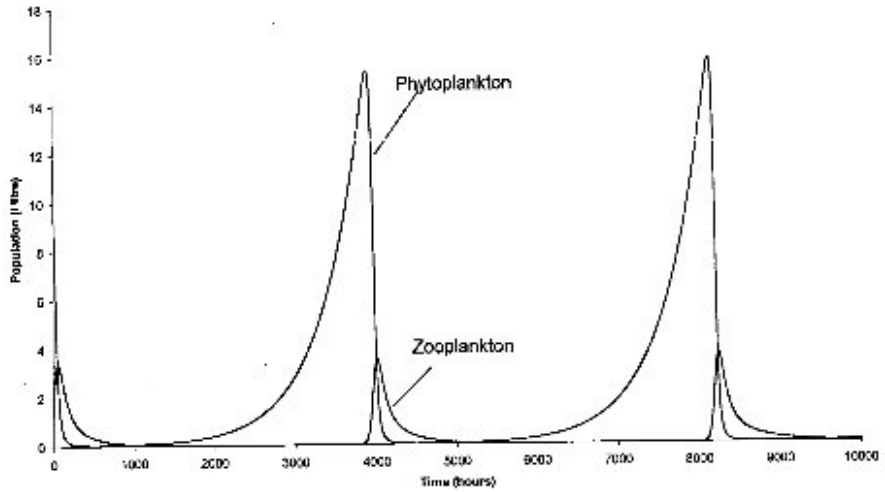

FIG. 5. Numerical solution of equation (9) for $\tau = 18$ h depicting periodic situation (stable limit cycle).

for $\tau = 18$ h, but when there is a dense concentration of zooplankton (we chose initial phytoplankton–zooplankton population ratio as 3 : 1) the time lag decreases and the periodicity (through the stable limit cycle) occurs at $\tau = 6$ h (see Fig. 6). This result shows the sensitivity of toxic phytoplankton in the presence of dense zooplankton populations. Diminution of time lag also implies that toxin acts as a controlling agent in the presence of dense zooplankton. The results obtained by our field observation also suggest that toxic substances or toxic phytoplankton may serve as a key factor in the termination of planktonic blooms. Thus, we may conclude that the above observations establish the role of toxin in the reduction of grazing pressure of zooplankton. It may also be noted that the experimental and mathematical observations of Chattopadhyay *et al.* (2002) and the experimental research of Buskey & Stockwell (1993) support our conclusion.

Finally, we would like to mention that the dynamics of the planktonic community, specifically the understanding of the role of HABs in the planktonic world, is still in a state of infancy and hence interdisciplinary involvement is necessary. For example, the life stage of an individual (larva, juvenile or adult) will also greatly affect the response to a toxic substance. In general, larvae and juveniles are more vulnerable to injury or death from exposure to these substances. Studies of the effects of toxic substances must consider both the age and species of specimens to fully access the chemical toxicity. Also, to study the dynamics under the presence of external force may be another interesting problem in this context, as massive phytoplankton blooms were observed in Seto Inland Sea, Japan (Prakash, 1987) and in Hong Kong Harbour (Lam & Ho, 1989) which were due to artificial eutrophication, although we feel such an approach may be viewed as very artificial and hence at present we have avoided it. So, all the possible mechanisms existing in the planktonic world may not be captured in a single mathematical model. However, the present simple model with its outcome may give some insight to researchers of this very complex and important issue.

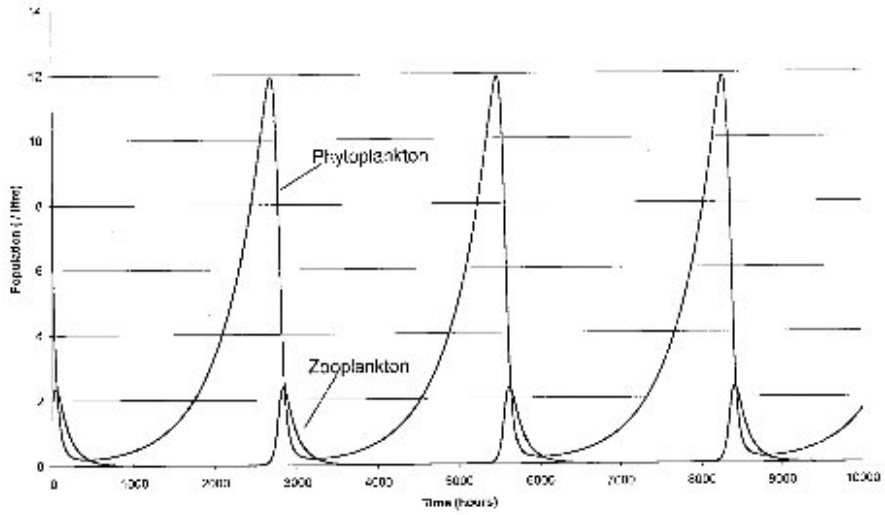

FIG. 6. Numerical solution of equation (9) for $\tau = 6$ h depicting periodic oscillations (stable limit cycle).

Acknowledgements

The authors are grateful to Ovide Arino for discussions and useful suggestions. Part of this work was completed when the authors J.Chattopadhyay and A.El Abdllaoui were at Institut de Recherche pour le Developpement (IRD), U. R. Geodes, Paris, France, and were supported by an IRD grant. The authors are grateful to the anonymous referees of this paper for their valuable comments and suggestions in improving the standard of the work.

REFERENCES

ANDERSON, D. M. *et al.* (1990) Dynamics and physiology of saxitoxin production by the dinoflagllates. *Alexandrium spp. Marine Biology*, **104**, 511–524.

AURE, J. & REY, F. (1992) Oceanographic conditions in the Sandsfjord System, western Norway, after a bloom of the prymnesiophyte *Prymnesium parvum* Carter in August 1990. *Sarsia*, **76**, 247–254.

BIRKHOFF, G. & ROTA, G. C. (1982) *Ordinary Differential Equations*. Ginn Boston.

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

BONEY, A. D. (1976) *Phytoplankton*. London: Arnold.

BUSENBERG, S. N. & TRAVIS, C. C. (1982) On the use of reducible-functional differential equations in biological models. *J. Math. Anal. Appl.*, **89**, 46–66.

BUSKEY, E. J. & HYATT, C. J. (1995) Effects of the Texas (USA) brown tide alga on planktonic grazers. *Mar. Ecol. Prog. Ser.*, **126**, 285–292.

BUSKEY, E. J. & STOCKWELL, D. A. (1993) Effects of a persistent 'brown tide' on zooplankton population in the Laguna Madre of southern Texas. *Toxic Phytoplankton Blooms in the Sea*. (T. J. Smayda & Y. Shimuzu, eds). Amsterdam: Elsevier, pp. 659–666.

CHATTOPADHYAY, J., SARKAR, R. & MANDAL, S. (2002) Toxin producing plankton may act as a biological control for planktonic blooms—field study and mathematical modelling. *J. Theor. Biol.*, **215**, 333–344.

CUSHING, J. M. (1977) *Integrodifferential Equations and Delay Models in Population Dynamics*, Lecture Notes in Biomathematics, 20. Heldelberg: Springer.

DAVIS, C. C. (1955) *The marine and freshwater plankton*. Michigan State Univ. Press, p. 562.

DUINKER, J. & WEFER, G. (1994) Das CO_2-Problem und die Rolle des Ozeans. *Naturwissenschahten*, **81**, 237–242.

EDWARDS, A. M. & BRINDLEY, J. (1999) Zooplankton mortality and the dynamical behaviour of Plankton population model. *Bull. Math. Biol.*, **61**, 303.

ESTEP, K. W., NEJSTGAARD, J. C., SKJOLDAL, H. R. & REY, F. (1990) Predation by copepods upon natural populations of *Phaeocystis pouchetii* as a function of the physiological state of the prey. *Mar. Ecol. Prog. Ser.*, **67**, 235–249.

FAY, P. (1983) *The Blue-Greens*. London: Arnold.

FRANKS, P. J. S. (1997) Models of harmful algal blooms. *Limaol. Oceangr*, **42**, 1273–1282.

FREEDMAN, H. I. & RAO, V. S. H. (1983) The trade-off between mutual interference and time lags in predator-prey systems. *Bull. Math. Biol.*, **45**, 991.

HALE, J. (1993) *Theory of Functional Differential Equation with Applications in Population Dynamics*. New York: Academic.

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

HANSEN, F. C. (1995) Trophic interaction between zooplankton and *Phaeocystis cf. Globosa*. *Helgol Meeresunters*, **49**, 283–293.

HASSARD, B. D. *et al.* (1981) *Theory and Application of Hopf Bifurcation*. Cambridge: Cambridge University.

HUNTLEY, M. E., SYKES, P., ROHAN, S. & MARIN, V. (1986) Chemically mediated rejection of dinoflagellate prey by the copepods *Calanus pacificus* and *Paracalanus parvus*: mechanism, occurence and significance. *Mar. Ecol. Prog. Ser.*, **28**, 105–120.

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.

KEATING, K. I. (1976) Algal metabolite influence on bloom sequence in eutrophic freshwater ponds. *E.P.A. Ecological monograph series* (EPA. 600/3-76-081. Washington, DC: Govt. Print. OH., p. 148.

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

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

LAM, C. W. Y. & HO, K. C. (1989) Red Tides in Tolo Harbor, Hong Kong. *Red Tides: Biology, Environmental Science and Toxicology*. (T. Okaichi *et al.*, eds). Amsterdam: Elsevier, pp. 49–52.

LEFEVRE, M., JAKOB, H. & NISBET, M. (1952) Auto et heteroantagonisme chezles algnes d'eau dome invitro et daus les collections d'eau naturrells. *Ann. Sta. Contr. Hydro. App.*, **4**, 1.

LEVIN, S. A. & SEGEL, L. (1976) Hypothesis for the origin of plankton patchiness. *Nature*, **259**, 659.

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

MACDONALD, N. (1978) *Time Lags in Biological Models*, Lecture Notes in Biomathematics, 27. Heldelberg: Springer.

MURRAY, J. M. (1989) *Mathematical Biology*. Berlin: Springer.

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

NEWELL, G. E. & NEWELL, R. C. (1979) *Marine plankton*, A practical guide. 5th Edn.. Hutchinson.

NIELSEN, T. G. *et al.* (1990) Effects of a *Chrysochromulina polylepis* subsurface bloom on the plankton community. *Marine Ecology Progress Series*, **62**, 21–35.

ODUM, E. P. (1971) *Fundamentals of Ecology*. Philadelphia, PA: Saunders.

PRAKASH, A. (1987) Coastal organic pollution as a contributing factor to red tide development. *Rapports et Proces—verbaux des Reunions du Conseil Permanent International pour l'Exploration de la Mer*, **187**, 61–65.

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

SATORA, T. & LAWS, A. (1989) Periodic blooms of the silicoflagellate *Dictyocha perlaevis* in the subtropical inlet, Kaneohe Bay, Hawaii. *Red Tides: Biology, Environmental Science and Toxicology*, Vol. 69. (T. Okaichi *et al.*, eds). Amsterdam: Elsevier.

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

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

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

STEIDINGER, K. A. *et al.* (1996) Pfiesteria Piscicida a new toxic dinoflagellate genus and species of the order Dinamoebales. *J. Phycology*, **32**, 157–164.

THINGSTAD, T. F. & LANGELAND, T. I. (1974) Dynamics of chemostat culture: The effect of a delay in cell response. *J. Theo. Biol.*, **48**, 149.

TOMAS, C. R. (1997) *Identifying marine diatoms and dinoflagellates*. San Diego, CA: Academic Press, p. 858.

TRUSCOTT, J. E. & 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.

WOLKOWICZ, G. S. K., XIA, H. & RUAN, S. (1997) Competition in the chemostat: A distributed delay model and its global asymptotic behaviour. *SIAM. J. Appl. Math.*, **57**, 1281–1310.

WORK, T. M. *et al.* (1993) Domoic acid in toxication of brown pelicans and cormorants in Santa Cruz, California. *Toxic Phytoplankton Blooms in the Sea*, Vol. 3. (T. J. Smayda & Y. Shimuza, eds). Amsterdam: Elsevier, pp. 643–649.

WYATT, T. & HORWOOD, J. (1973) Model which generates red tides. *Nature*, **244**, 238–240.

Appendix. Stability of the bifurcation

Here we determine a formula that establishes the stability of bifurcating periodic orbits. The calculation is based on Hassard *et al.* (1981). We assume the case where Hopf bifurcation occurs (at $\tau = \tau_0$ and $\omega = \omega_0$) and using the standard notation as in Hassard

et al. (1981) we rewrite (28) in the form

$$\dot{x}_t = A_{\alpha'} x_t + R x_t \tag{A1}$$

where $x_t \in C([-\tau, 0], \Re)$ is given by $x_t(\theta') = x(t + \theta')$; α' represents the parameter values at $\tau = \tau_0$, $\omega = \omega_0$.

$$A_{\alpha'}\phi(\theta') = \begin{cases} \dfrac{\mathrm{d}\phi}{\mathrm{d}\theta'} & -\tau \leqslant \theta' < 0 \\ \displaystyle\int_{-\tau}^{0} \mathrm{d}\eta(\rho, \alpha')\phi(\rho) & \theta' = 0. \end{cases} \tag{A2}$$

$$R\phi(\theta') = \begin{cases} \begin{pmatrix} 0 \\ 0 \end{pmatrix} & -\tau \leqslant \theta' < 0 \\ \begin{pmatrix} a_{11}\phi_1(\theta')\phi_2(\theta') + a_{20}\phi_1^2(\theta') \\ \{b_{11}\phi_1(\theta')\phi_2(\theta') + b_{11}' \dfrac{\phi_1(\theta' - \tau)}{\gamma + \phi_1(\theta' - \tau)}\phi_2(\theta') \\ + b_{12}' \frac{\phi_1(\theta' - \tau)}{\gamma + \phi_1(\theta' - \tau)}\} \end{pmatrix} & \theta' = 0 \end{cases} \tag{A3}$$

$$\mathrm{d}\eta(\theta'; \alpha') = \begin{pmatrix} A\,\delta(\theta') & B\,\delta(\theta') \\ C\,\delta(\theta') + \alpha'\delta(\theta' + \tau) & D\,\delta(\theta') \end{pmatrix} \mathrm{d}\theta'. \tag{A4}$$

An eigenfunction of the problem corresponding to the eigenvalue $\mathrm{i}\omega_0$

$$q(\theta') = \begin{pmatrix} \beta_1 \\ 1 \end{pmatrix} \mathrm{e}^{\mathrm{i}\omega_0\theta'} \tag{A5}$$

where

$$\beta_1 = \frac{B}{\lambda - A}.$$

At $\lambda = \mathrm{i}\omega_0$

$$\beta_1 = \frac{AB + \mathrm{i}B\omega_0}{A^2 + \omega_0^2}. \tag{A6}$$

Now we define the following bilinear form:

$$\langle \psi, \phi \rangle = \overline{\psi(0)}\phi(0) - \int_{\theta'=-\tau}^{0} \int_{\xi=0}^{\theta'} \overline{\psi(\xi - \theta')}[\mathrm{d}\eta(\theta')]\phi(\xi)\mathrm{d}\xi. \tag{A7}$$

To obtain the corresponding adjoint eigenfunction $\hat{q}(\theta')$, we use the standard result

$\langle \acute{q}, q\rangle = 1$ and $\langle \acute{q}, \bar{q}\rangle = 0$, letting $\acute{q} = e^{i\omega_0\theta'}(\nu_1, \nu_2)$, then we have

$$\begin{aligned}\langle \acute{q}, q\rangle &= \overline{\acute{q}(0)}q(0) - \int_{-\tau}^{0}\int_{0}^{\theta'} \overline{\acute{q}(\xi-\theta')}\,d\eta(\theta')q(\xi)\,d\xi \\ &= \beta\bar{\nu}_1 + \bar{\nu}_2 - \int_{-\tau}^{0}\int_{0}^{\theta'} e^{-i\omega_0(\xi-\theta')}(\bar{\nu}_1 \;\; \bar{\nu}_2)\begin{pmatrix} 0 \\ \alpha'\delta(\theta'+\tau)\end{pmatrix}\begin{pmatrix}\beta_1 \\ 1\end{pmatrix} e^{i\omega_0\xi}\,d\theta'\,d\xi \\ &= \beta\bar{\nu}_1 + \bar{\nu}_2 - \int_{-\tau}^{0}\int_{0}^{\theta'} e^{i\omega_0\theta'}\alpha'\bar{\nu}_2\delta(\theta'+\tau)\,d\theta'\,d\xi \\ &= \beta\bar{\nu}_1 + \bar{\nu}_2 - \alpha'\bar{\nu}_2(\tau\cos\omega_0\tau - i\tau\sin\omega_0\tau). \end{aligned} \tag{A8}$$

Therefore,

$$\beta_1\bar{\nu}_1 + \bar{\nu}_2(1 - \alpha'\tau\;\cos\omega_0\tau + i\alpha'\tau\;\sin\omega_0\tau) = 1 \tag{A9}$$

$$\begin{aligned}\langle \acute{q}, \bar{q}\rangle &= \bar{\beta}_1\bar{\nu}_1 + \bar{\nu}_2 \\ &\quad - \int_{-\tau}^{0}\int_{0}^{\theta'} e^{-i\omega_0(\xi-\theta')}(\bar{\nu}_1 \;\; \bar{\nu}_2)\begin{pmatrix} 0 \\ \alpha'\delta(\theta'+\tau)\end{pmatrix}\begin{pmatrix}\bar{\beta}_1 \\ 1\end{pmatrix} e^{-i\omega_0\xi}\,d\theta'\,d\xi \\ &= \bar{\beta}_1\bar{\nu}_1 + \bar{\nu}_2 - \int_{-\tau}^{0}\int_{0}^{\theta'} e^{-i\omega_0(\xi-\theta')}\alpha'\bar{\nu}_2\delta(\theta'+\tau)e^{-i\omega_0\xi}\,d\theta'\,d\xi \end{aligned} \tag{A10}$$

Therefore,

$$\bar{\beta}_1\bar{\nu}_1 + \bar{\nu}_2 + i\alpha'\frac{\bar{\nu}_2}{\omega_0}\sin\omega_0\tau = 0. \tag{A11}$$

So, the required equations for $\bar{\nu}_1$ and $\bar{\nu}_2$ are

$$\left.\begin{aligned}\beta_1\bar{\nu}_1 + e_1\bar{\nu}_2 &= 1 \\ \bar{\beta}_1\bar{\nu}_1 + e_2\bar{\nu}_2 &= 0\end{aligned}\right\} \tag{A12}$$

where

$$\left.\begin{aligned}e_1 &= 1 - \alpha'\tau\cos\omega_0\tau + i\alpha'\tau\;\sin\omega_0\tau \\ e_2 &= 1 + i\frac{\alpha'}{\omega_0}\sin\omega_0\tau\end{aligned}\right\} \tag{A13}$$

$$\left.\begin{aligned}\bar{\nu}_1 &= \frac{e_2}{e_2\beta_1 - e_1\bar{\beta}_1} \\ \bar{\nu}_2 &= \frac{-\bar{\beta}_1}{e_2\beta_1 - e_1\bar{\beta}_1}.\end{aligned}\right\} \tag{A14}$$

Finally, we have the values of ν_1 and ν_2 by taking the complex conjugate of (A14). Using the notation as in Hassard *et al.* (1981), we write

$$\begin{pmatrix} x \\ y\end{pmatrix} = zq + \bar{z}\bar{q} + W \tag{A15}$$

$$z = \left\langle \acute{q}\begin{pmatrix} x \\ y\end{pmatrix}\right\rangle \tag{A16}$$

$$\begin{aligned}\dot{z}(t) &= \mathrm{i}\omega_0 z(t) + \bar{q}(0)\cdot f(w(z,\bar{z},\theta') + \mathrm{Re}\{z(t)q(\theta')\}) \\ &= \mathrm{i}\omega_0 z(t) + \bar{q}(0)\cdot f_0(z,\bar{z})\end{aligned} \tag{A17}$$

where

$$f_0 = \begin{pmatrix} f_0^1 \\ f_0^2 \end{pmatrix} \tag{A18}$$

$$\left.\begin{aligned} f_0^1 &= \{W^1(0) + 2\mathrm{Re}(z(t)\beta_1)\}[a_{11}(W^2(0) + 2\mathrm{Re}\, z(t)) \\ &\quad + a_{20}(W^1(0) + 2\mathrm{Re}\,(z(t)\beta_1))], \\ f_0^2 &= \{W^2(0) + 2\mathrm{Re}\, z(t)\}[b_{11}(W^1(0) + 2\mathrm{Re}\,(z(t)\beta_1)) \\ &\quad + b_{11}'(W^1(-\tau) + 2\mathrm{Re}\,(z(t)\mathrm{e}^{-\mathrm{i}\omega_0\tau}\beta_1))] \\ &\quad + b_{12}'(W^1(-\tau) + 2\mathrm{Re}\,(z(t)\mathrm{e}^{-\mathrm{i}\omega_0\tau}\beta_1))^2 \end{aligned}\right\} \tag{A19}$$

Using the result $\Delta(\mathrm{i}\omega,\tau) = 0$, for $\omega = \omega_0$, $\tau = \tau_0$, and letting $\Omega_0 = \mathrm{e}^{-\mathrm{i}\omega_0\tau_0}$ also we have assumed that $W = O(|z|^2)$. We have retained only the terms necessary to compute $C_1(0)$. Therefore,

$$\left.\begin{aligned} f_0^1 &= a_{11}(W^1(0) + \beta_1 z + \bar{\beta}_1\bar{z})(W^2(0) + z + \bar{z}) \\ &\quad + a_{20}(W^1(0) + \beta_1 z + \bar{\beta}_1\bar{z})^2 \\ &= (a_{11}\beta_1 + a_{20}{\beta_1}^2)z^2 + (a_{11}\bar{\beta}_1 + a_{20}{\bar{\beta}_1}^2)\bar{z}^2 \\ &\quad + (a_{11}(\beta_1 + \bar{\beta}_1) + 2a_{20}\beta_1\bar{\beta}_1)z\bar{z} + O(|z|^4) \end{aligned}\right\} \tag{A20}$$

$$\left.\begin{aligned} f_0^2 &= b_{11}(W^1(0) + \beta_1 z + \bar{\beta}_1\bar{z})(W^2(0) + z + \bar{z}) \\ &\quad + (W^1(0) + (\beta_1 z + \bar{\beta}_1\bar{z})\Omega_0)b_{11}'(W^2(0) + z + \bar{z}) \\ &\quad + b_{12}'(W^1(0) + (\beta_1 z + \bar{\beta}_1\bar{z})\Omega)^2 \\ &= (b_{11}\beta_1 + b_{12}'\beta_1{\Omega_0}^2)z^2 + (b_{11}\bar{\beta}_1 + \bar{\beta}_1 b_{12}'{\Omega_0}^2)\bar{z}^2 \\ &\quad + (b_{11}(\beta_1 + \bar{\beta}_1) + 2b_{12}'\beta_1\bar{\beta}_1)z\bar{z} \\ &\quad b_{11}'{\beta_1}^2{\Omega_0}^2 z^3 + b_{11}'{\bar{\beta}_1}^2{\Omega_0}^2\bar{z}^3 + b_{11}'(\beta_1^2 + 2\beta_1\bar{\beta}_1){\Omega_0}^2 z^2\bar{z} \\ &\quad + b_{11}'(2\beta_1\bar{\beta}_1 + {\bar{\beta}_1}^2)z\bar{z}^2 + O(|z|^4). \end{aligned}\right\} \tag{A21}$$

So, after taking the dot product of f_0 and $\bar{q}(0)$ and after expanding, we have

$$\begin{aligned}\dot{z} &= \mathrm{i}\omega_0 z + \bar{v}_1 f_0^1 + \bar{v}_2 f_0^2 \\ &= \mathrm{i}\omega_0 z + \frac{1}{2}g_{20}z^2 + \frac{1}{2}g_{02}\bar{z}^2 + g_{11}z\bar{z} + \frac{1}{6}g_{30}z^3 \\ &\quad + \frac{1}{6}g_{03}\bar{z}^3 + \frac{1}{2}g_{21}z^2\bar{z} + \frac{1}{2}g_{12}z\bar{z}^2 + O(|z|^4)\end{aligned} \tag{A22}$$

where

$$\left.\begin{aligned}
g_{20} &= 2[\bar{\nu}_1(a_{11}\beta_1 + a_{20}\beta_1^2) + \bar{\nu}_2(b_{11}\beta_1 + b_{12}'\beta_1\Omega_0)^2] \\
g_{02} &= 2[\bar{\nu}_1(a_{11}\bar{\beta}_1 + a_{20}\bar{\beta}_1^2) + \bar{\nu}_2(b_{11}\bar{\beta}_1 + b_{12}'\bar{\beta}_1\Omega_0)^2] \\
g_{11} &= \bar{\nu}_1(a_{11}(\beta_1 + \bar{\beta}_1) + 2a_{20}\beta_1\bar{\beta}_1) + \bar{\nu}_2(b_{11}(\beta_1 + \bar{\beta}_1) \\
&\quad + 2b_{12}'\beta_1\bar{\beta}_1) \\
g_{12} &= 2\bar{\nu}_2 b_{11}'(2\beta_1\bar{\beta}_1 + \bar{\beta}_1^2)\Omega_0^2 \\
g_{21} &= 2\bar{\nu}_2 b_{11}'(2\beta_1\bar{\beta}_1 + \beta_1^2)\Omega_0^2 \\
g_{30} &= 6\bar{\nu}_1 b_{11}'\beta_1^2\Omega_0^2 \\
g_{03} &= 6\bar{\nu}_1 b_{11}'\bar{\beta}_1^2\Omega_0^2.
\end{aligned}\right\} \tag{A23}$$

Finally we use the expression of Hassard *et al.* (1981):

$$\left.\begin{aligned}
C_1(0) &= \frac{i}{2\omega_0}(g_{20}g_{11} - 2|g_{11}|^2 + \frac{1}{3}|g_{02}|^2) + \frac{1}{2}g_{21} \\
\mu_2 &= -\frac{\mathrm{Re}\ C_1(0)}{\alpha'(0)} \\
\tau_2 &= -\frac{Im\ C_1(0) + \mu_2\omega'(0)}{\omega_0}.
\end{aligned}\right\} \tag{A24}$$

So, the bifurcation is supercritical if $\mu_2 > 0$ and subcritical if $\mu_2 < 0$. Further if $\tau_2 > 0$, the period of the solution increases with τ.