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TEMPORAL AND SPATIO-TEMPORAL DYNAMICS OF A MATHEMATICAL MODEL OF HARMFUL ALGAL INTERACTION

B. MUKHOPADHYAY* AND R. BHATTACHARYYA

ABSTRACT. The adverse effect of harmful plankton on the marine ecosystem is a topic of deep concern. To investigate the role of such phytoplankton, a mathematical model containing distinct dynamical equations for toxic and non-toxic phytoplankton is analyzed. Stability analysis of the resulting three equation model is carried out. A continuous time variation in toxin liberation process is incorporated into the model and a stability analysis of the resulting delay model is performed. The distributed delay model is then extended to include the spatial distribution of plankton and the delay-diffusion model is analyzed with spatial and spatiotemporal kernels. Conditions for diffusion-driven instability in both the cases are derived and compared to explore the significance of these kernels. Numerical studies are performed to justify analytical findings.

AMS Mathematics Subject Classification : Primary 35K55, 35K57, 92D25
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1. Introduction

Plankton are the basis of all marine aquatic food chains. There are two types of plankton. The plant species is known as *phytoplankton* and the herbivore animals feeding on these plants are termed *zooplankton*. Apart from occupying the first trophic level, phytoplankton do a huge service by generating oxygen needed for maintaining life and by absorbing carbon-di-oxide and thereby reducing global warming. Besides oxygen and carbon-di-oxide, other substances including phosphorus, nitrogen and sulphur are also recycled by phytoplankton [1,2]. Hence, phytoplankton are one of the fundamental components controlling further development of the climate and there is a vast literature on this subject [3,4].

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In spite of the important service rendered by these tiny organisms, some phytoplankton species are known to have adverse effects on their surrounding environment. There has been a considerable scientific attention towards the study of these harmful phytoplankton in recent times [5-11]. These phytoplankton can be classified into two categories - (i) the toxin producers that contaminate sea food and destroy fish; (ii) high biomass producers that cause mortality of surrounding marine creatures after attaining dense concentrations. Species like *Alexandrium tamarense* and *Pseudo-nitzschia australis* liberate toxins into the environment before they are eaten up [12]. It has been well established through field studies and experimental observations that a significant number of phytoplankton species produce toxins [8, 13-15] and the harmful impact of these toxic substances on ecosystem and environment as a whole are recognized.

Time delays in the dynamics of several interacting species can arise from a great variety of causes and are always present to some extent [16]. In the context of plankton ecology, different researchers have incorporated time delays in nutrient recycling, growth response to nutrient uptake etc. [17-19]. It is well known that liberation of toxic elements by harmful phytoplankton species is not an instantaneous process, but is mediated by some time delay. This phenomenon is amply supported by the observation that zooplankton mortality due to toxic phytoplankton bloom occurs after some time lapse. The field study conducted by Chattopadhyay and his associates [10-11] suggests that the abundance of *Paracalanus* (zooplankton) population reduces after some time lapse of the bloom of the toxic phytoplankton *Noctiluea Scintillans.* This time delay can be interpreted ecologically as the time required for the phytoplankton to mature before they can produce toxic materials. Several authors [20-21] have suggested that the growth rate of zooplankton which consumes large phytoplankton cells are slow relative to the growth rate of phytoplankton and there will be a relatively long time gap between the onset of an increase in growth rate of phytoplankton species and a build up in the biomass of zooplankton. Based on their field study, Chattopadhyay et.al.[10] performed a mathematical study of two-species plankton models under the influence of toxication delay both in discrete and distributed form and observed that discrete time delay is capable of generating periodic oscillation of different system components resembling phytoplankton bloom. Sarkar et.al. [9] studied the same model with discrete time delay and environmental fluctuations to predict a control mechanism for blooms.

An interesting aspect of modern biological research is the formation of spatial and spatiotemporal patterns. It characterizes the dynamics of both aquatic and terrestrial populations in their natural setting. In the natural marine environment, the spatial horizontal distribution of plankton is highly inhomogeneous [22-23]. This inhomogeneity is evident in all scales and are related to various factors such as the underlying hydrophysical fields like temperature and nutrient [24-25], physical features of the fluid motion for example eddies, fronts and turbulence [26-30]. In other cases where there is no such clear cut forcing mechanism, it is conjectured that the biological process inherent to the population dynamics are important in producing such patchy distribution [31-35]. Using

nonlinear reaction-diffusion equations as a mathematical tool [36-38], different researchers have studied the problem of patchy plankton distribution and attributed the formation of spatial patterns to well known general mechanisms such as diffusion-driven Turing instability [39-40] or differential-flow-induced instability [41]. Ruan [42] studied a two-species plankton model which considered distributed time delays in nutrient recycling and in plankton growth response in a spatially heterogeneous environment. By using a spatial kernel he has demonstrated the existence of Turing instability and subsequent travelling waves. Bousaba et.al. [43] extended the work of Ruan for spatiotemporal kernels. Using the method developed by Gourley and Britton [44] for a general reaction-diffusion system, Mukhopadhyay and Bhattacharyya [45] investigated a delay-diffusion phytoplankton-zooplankton model with spatiotemporal kernel and established the role of toxin on phytoplankton bloom. They have also studied a nutrient-plankton model in a purely spatial context and modelled the impact of allelopathic phytoplankton on nutrient-plankton dynamics using a type-IV zooplankton functional response as well as toxin-induced cross-diffusion [46].

In the present analysis, we consider a three species plankton model consisting of (i) Nontoxic phytoplankton (ii) toxic phytoplankton and (iii) zooplankton. The time needed to release toxic substances is included as a distributed time delay and the stability criteria of the delay model is investigated. Spatial non-homogeneity in plankton distribution is modelled by incorporating physical diffusion of the plankton communities. The resulting delay-diffusion model is studied both with spatial and spatiotemporal kernels to explore the dynamical complexity of the system under different realistic assumptions. Numerical simulations are carried out to support analytical results.

2. The model

Let $N(x,t)$, $T(x,t)$ and $Z(x,t)$ denote the densities of non-toxic phytoplankton, toxic phytoplankton and zooplankton respectively at time t and location x, where $0 \leq t < \infty$, $-\infty < t < \infty$.

Suppose, d_1, d_2 and d_3 be the constant diffusion coefficients of the three plankton populations. Then we have the following reaction-diffusion model with delay in toxin production

$$\begin{aligned}
\frac{\partial N}{\partial t} &= d_1 \frac{\partial^2 N}{\partial x^2} + r_1 N\left(1 - \frac{N}{k_1}\right) - \alpha_1 \frac{NZ}{a+N} - \mu NT \\
\frac{\partial T}{\partial t} &= d_2 \frac{\partial^2 T}{\partial x^2} + r_2 T\left(1 - \frac{T}{k_2}\right) - \alpha_2 \frac{TZ}{b+T} \\
\frac{\partial Z}{\partial t} &= d_3 \frac{\partial^2 Z}{\partial x^2} + \beta_1 \frac{NZ}{a+N} + \beta_2 \frac{TZ}{b+T} - dZ - \theta Z \int_{-\infty}^{t} \alpha e^{-\alpha(t-\tau)} T(\tau) d\tau
\end{aligned} \tag{1}$$

with initial conditions $N(x,0) \geq 0$, $T(x,\lambda) = \eta_1(x,\lambda)$ and $Z(x,0) \geq 0$ where $\lambda \in (-\infty, 0)$ and η_1 is a nonnegative continuous function and $\alpha > 0$.

Table 1
Values of model Parameters

Parameters	Values/day
r_1	0.5
r_2	0.4
k_1	10
k_2	11
α_1	0.4
α_2	0.5
μ	0.02
β_1	0.24
β_2	0.25
a	0.6
b	0.5
d	0.09

The growth process of both the phytoplankton species are taken to be logistic with maximum growth rate r_i, $(i = 1, 2)$ and environmental carrying capacity k_i, $(i = 1, 2)$. Loss of both phytoplankton species due to grazing by zooplankton follows a simple saturating functional response formulated as a *Michaelis-Menten function* of phytoplankton density with half saturation constants a (for non-toxic) and b (for toxic) and maximum per capita grazing rates of α_1 and α_2. The non-toxic phytoplankton suffer additional losses due to the inhibitory effect of toxic phytoplankton at a rate μ. As is already mentioned, the harmful phytoplankton is capable of reaching dense concentration through rapid growth and hence can inhibit the growth and development of the non-toxic ones. The inhibitory effect of non-toxic phytoplankton on toxic ones is neglected since it is insignificantly small compared to the effect of toxic phytoplankton on nontoxic ones. Food intake by zooplankton is converted into growth with efficiencies β_1 and β_2. In addition to a fixed mortality d, zooplankton population is also destroyed due to the toxin produced by harmful phytoplankton at a rate θ. As is already mentioned, production of toxin by harmful phytoplankton is not an instantaneous process and the delay is due to the time taken by phytoplankton to mature before they can release toxins. However, different harmful plankton species is expected to have distinct maturation time and consequently we have used a *distributed time delay with a weak kernel.*

We denote the interior equilibrium point of system (1) by $E^* \equiv (N^*, T^*, Z^*)$ where

$$Z^* = \frac{(b+T^*)r_2}{\alpha_2}\left(1 - \frac{T^*}{k_2}\right) \tag{2}$$

(N^*,T^*) is the positive root of the system

$$r_1\left(1 - \frac{x}{k_1}\right) - \left(\frac{\alpha_1}{a+x}\right)\frac{r_2}{\alpha_2}(b+y)\left(1 - \frac{y}{k}\right) - \mu xy = 0$$

$$\beta_1\frac{x}{a+x} + \beta_2\frac{y}{b+y} - d - \theta y = 0 \tag{3}$$

3. The delayed homogeneous system

First we consider the model (1) in a spatially homogeneous environment

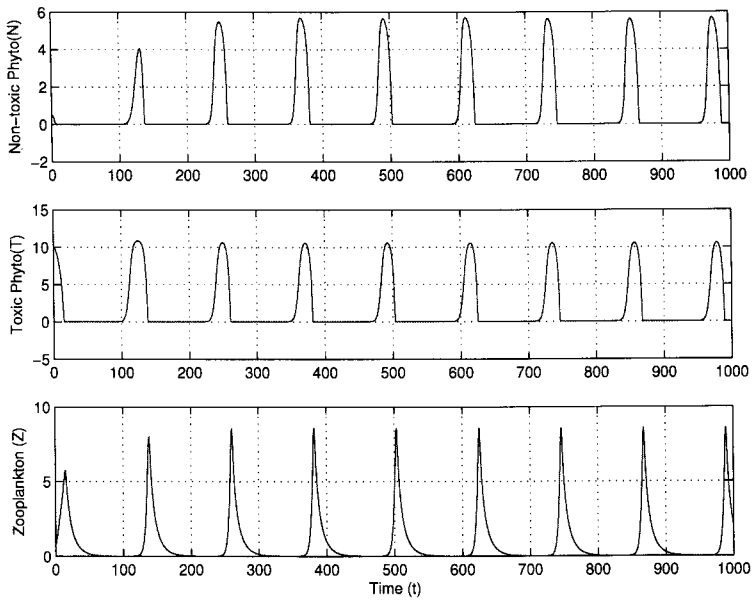

FIGURE 1. Numerical simulation of the distributed delay model. Parameter values are taken from Table 1 and $\theta = 0.01$. The figure exhibits oscillatory coexistence of all populations.

namely,

$$\begin{aligned}
\frac{dN}{dt} &= r_1 N\left(1-\frac{N}{k_1}\right) - \alpha_1 \frac{NZ}{a+N} - \mu NT \\
\frac{dT}{dt} &= r_2 T\left(1-\frac{T}{k_2}\right) - \alpha_2 \frac{TZ}{b+T} \\
\frac{dZ}{dt} &= \beta_1 \frac{NZ}{a+N} + \beta_2 \frac{TZ}{b+T} - dZ - \theta Z \int_{-\infty}^{t} \alpha e^{-\alpha(t-\tau)} T(\tau) d\tau
\end{aligned} \tag{4}$$

with $\alpha > 0$. The characteristic equation of the linearized system corresponding to (4) about E^* (obtained by using *linear chain trick*) will be

$$\lambda^4 + P_1(\theta)\lambda^3 + P_2(\theta)\lambda^2 + P_3(\theta)\lambda + P_4(\theta) = 0 \tag{5}$$

where

$$\begin{aligned}
P_1(\theta) &= A(\theta) + \alpha \\
P_2(\theta) &= A\alpha + B - a_{34}\alpha \\
P_3(\theta) &= B\alpha + C + \alpha a_{34}(a_{22} + a_{11}) \\
P_4(\theta) &= C\alpha - a_{11}a_{22}a_{34}\alpha \\
a_{34} &= -\theta Z^*
\end{aligned} \tag{6}$$

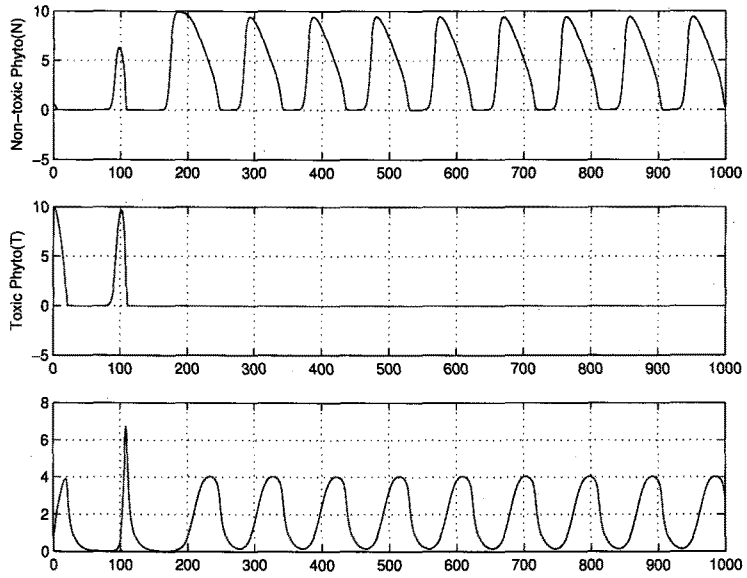

FIGURE 2. Numerical simulation of the DDE model with $\theta = 0.03$. The figure exhibits oscillatory behavior of nontoxic phytoplankton and zooplankton and extinction of toxic phytoplankton.

Now the delayed system will be asymptotically stable about E^* if

$$\begin{aligned} &P_1(\theta) > 0 \\ &P_1(\theta)P_2(\theta) - P_3(\theta) > 0 \\ &P_1(\theta)[P_2(\theta)P_3(\theta) - P_1(\theta)P_4(\theta)] - [P_3(\theta)]^2 > 0 \\ &P_4(\theta) > 0 \end{aligned} \tag{7}$$

Obviously, $P_1(\theta) > 0$ if $A > 0$. Also for small θ, the second and the last inequalities will automatically hold. Thus, the system will exhibit stable or unstable behavior according as

$$\Psi(\theta) = P_1(\theta)[P_2(\theta)P_3(\theta) - P_1(\theta)P_4(\theta)] - [P_3(\theta)^2] \tag{8}$$

is positive or negative. From the above analysis it is seen that the toxication rate θ is an important parameter in controlling the system dynamics for the delayed homogeneous system. A numerical study of model (4) is performed and the results are shown in Figures $1-5$. Figure 1 exhibits the oscillatory coexistence of all the three species for $\theta = 0.01$. As θ increases ($\theta = 0.03$), the nontoxic phytoplankton and the zooplankton survive in an oscillatory fashion whereas the toxic ones undergo ultimate extinction (Figure-2). A further increase of θ shows that for $\theta \in [0.04, 0.05]$, the toxic phytoplankton and the zooplankton exhibit oscillatory coexistence and the nontoxic ones undergo extinction (Figure-3,4). When θ is increased beyond $\theta = 0.06$, the toxic ones and the zooplankton coexist in a stable manner and the nontoxic ones undergo total extinction (Fig 5).

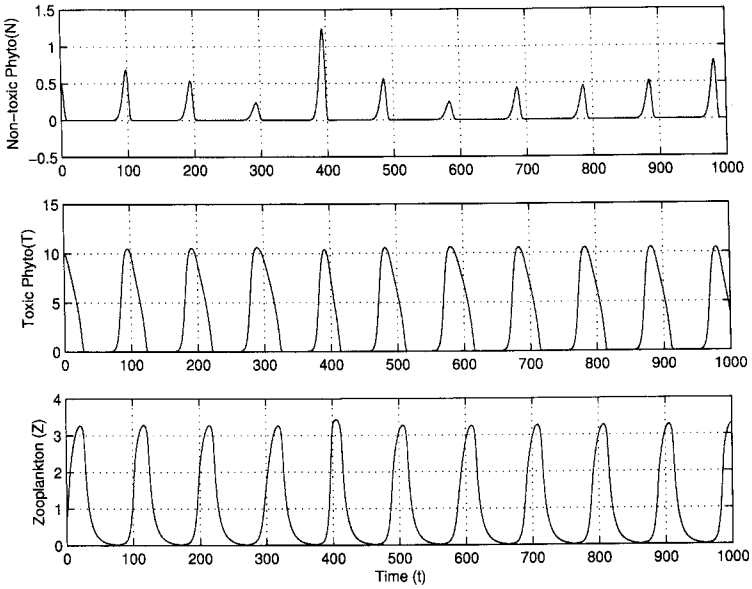

FIGURE 3. Numerical simulation of the DDE model with $\theta = 0.04$. The figure exhibits oscillatory coexistence of toxic phytoplankton and zooplankton and almost extinction of nontoxic ones.

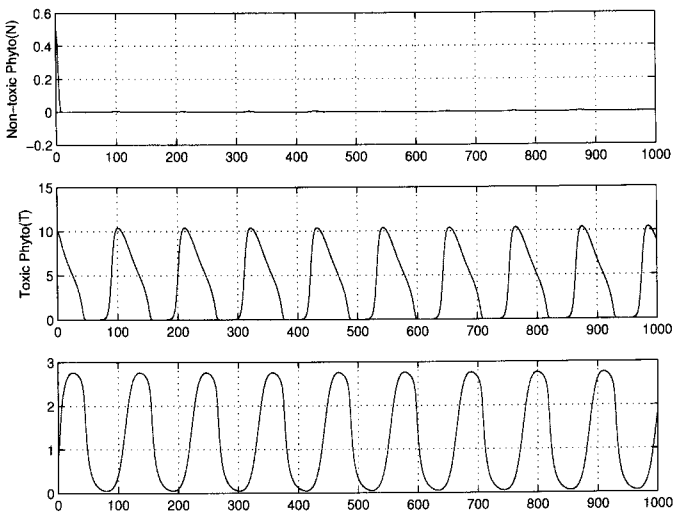

FIGURE 4. Numerical simulation of the DDE model with $\theta = 0.05$. The figure exhibits oscillatory coexistence of toxic phytoplankton and zooplankton and extinction of nontoxic ones.

Interestingly, in all the above cases, the delay parameter (α) has no significant impact on population evolution.

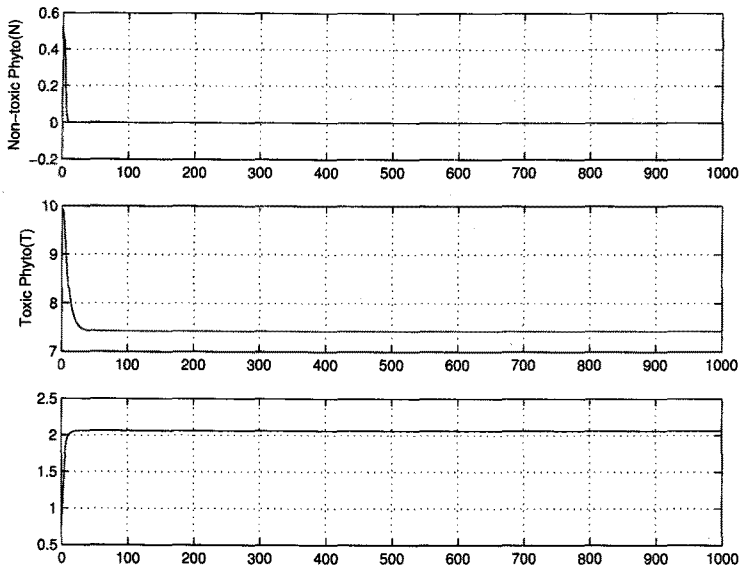

FIGURE 5. Numerical simulation of the DDE model with $\theta = 0.07$. The figure exhibits stable coexistence of toxic phytoplankton and zooplankton and extinction of nontoxic ones.

4. Effect of spatial kernel

In this section, we study the model with spatial kernel, namely model (1). We start the analysis by letting

$$R = \int_{-\infty}^{t} \alpha e^{-\alpha(t-\tau)} T(\tau) d\tau$$

system (1) reduces to

$$\begin{aligned}
\frac{\partial N}{\partial t} &= d_1 \frac{\partial^2 N}{\partial x^2} + r_1 N \left(1 - \frac{N}{k_1}\right) - \alpha_1 \frac{NZ}{a+N} - \mu NT \\
\frac{\partial T}{\partial t} &= d_2 \frac{\partial^2 T}{\partial x^2} + r_2 T \left(1 - \frac{T}{k_2}\right) - \alpha_2 \frac{TZ}{b+T} \\
\frac{\partial Z}{\partial t} &= d_3 \frac{\partial^2 Z}{\partial x^2} + \beta_1 \frac{NZ}{a+N} + \beta_2 \frac{TZ}{b+T} - dZ - \theta ZR \\
\frac{\partial R}{\partial t} &= \alpha(Z - R)
\end{aligned} \tag{9}$$

The positive interior equilibrium point of (9) is $E_1^* = (N^*, T^*, Z^*, R^*)$ where $R^* = Z^*$. Let,

$$u_1 = N - N^*; \quad u_2 = T - T^*; \quad u_3 = Z - Z^*; \quad u_4 = R - R^*. \tag{10}$$

Then the linearized system corresponding to (9) at E_1^* is

$$\begin{aligned}
\frac{\partial u_1}{\partial t} &= d_1\frac{\partial^2 u_1}{\partial x^2} + a_{11}u_1 + a_{12}u_2 + a_{13}u_3 \\
\frac{\partial u_2}{\partial t} &= d_2\frac{\partial^2 u_2}{\partial x^2} + a_{22}u_2 + a_{23}u_3 \\
\frac{\partial u_3}{\partial t} &= d_3\frac{\partial^2 u_3}{\partial x^2} + a_{31}u_1 + a_{32}u_2 + a_{34}u_4 \\
\frac{\partial u_4}{\partial t} &= \alpha(u_3 - u_4)
\end{aligned} \tag{11}$$

We assume a solution of (11) in the form

$$\begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} = \begin{pmatrix} \alpha_1 \\ \alpha_2 \\ \alpha_3 \\ \alpha_4 \end{pmatrix} \cos(qx)e^{\lambda t} \tag{12}$$

where q is the wave number of perturbation in the direction of x and λ is the

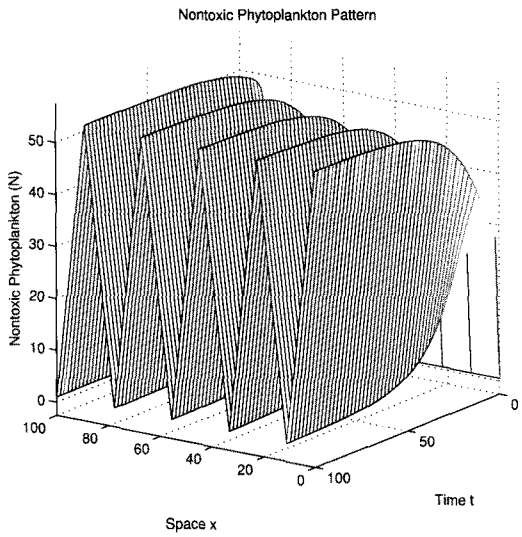

FIGURE 6. Numerical simulation of the delay-diffusion model showing the nontoxic phytoplankton pattern. The simulation is done for $0 < x < 100$ with zero flux boundary. Parameter values are taken from Table 1 together with $\alpha = 1.5$, $D_1 = 0.0065$; $D_2 = 0.005$; $D_3 = 0.007$.

frequency. The characteristic equation corresponding to (11) will be

$$\lambda^4 + m_1(\theta, q^2)\lambda^3 + m_2(\theta, q^2)\lambda^2 + m_3(\theta, q^2)\lambda + m_4(\theta, q^2) = 0 \tag{13}$$

where

$$\begin{aligned}
m_1(\theta, q^2) &= A + \alpha + (d_1 + d_2 + d_3)q^2 \\
m_2(\theta, q^2) &= \alpha[A + (d_1 + d_2 + d_3)q^2] + B + Ad_3q^2 + (d_1d_2 + d_1d_3 + d_2d_3)q^4 \\
&\quad -\alpha a_{34}
\end{aligned}$$

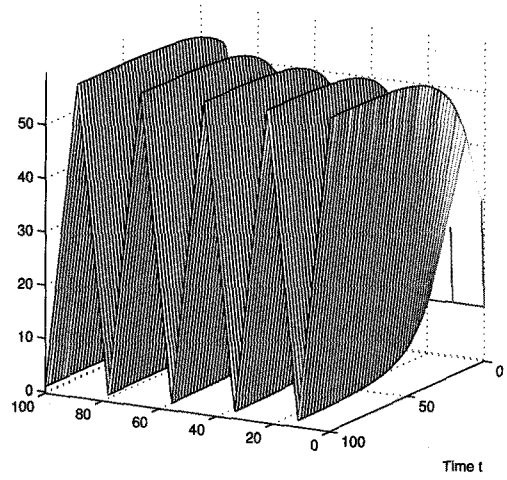

FIGURE 7. Numerical simulation of the delay-diffusion model showing the toxic phytoplankton pattern. Parameter values are same as that in Figure 6.

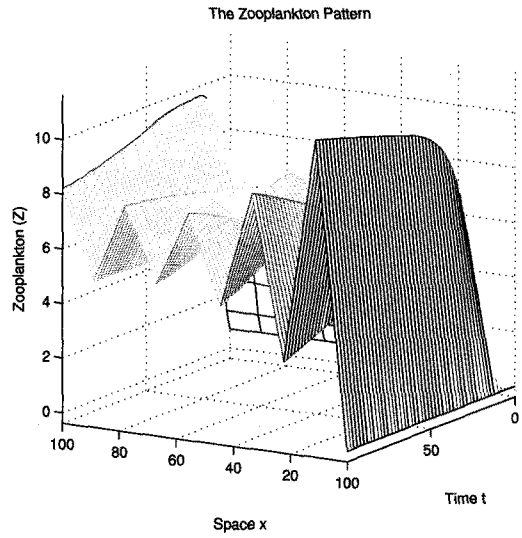

FIGURE 8. Numerical simulation of the delay-diffusion model showing the zooplankton pattern. Parameter values are same as that in Figure 6.

$$
\begin{aligned}
m_3(\theta, q^2) \;=\; & B\alpha + C + (A\alpha d_3 - a_{11}d_2\alpha - a_{22}d_1\alpha - a_{23}a_{32}d_1 - a_{31}a_{23}d_2)q^2 \\
& -a_{11}a_{22}d_3 + d_3q^4(a_{11}d_2 + a_{22}d_1) + (d_1d_3 + d_2d_3 + d_1d_2)\alpha q^4 \\
& +d_1d_2d_3q^6 - \alpha a_{34}\{A + (d_1 + d_2)q^2\}
\end{aligned}
$$

$$\begin{aligned} m_4(\theta, q^2) &= \alpha[C - a_{23}a_{32}d_1q^2 - \alpha a_{13}a_{31}d_2q^2 + a_{11}a_{22}d_3q^2 \\ &+ d_3q^4(-a_{11}d_2 - a_{22}d_1) + d_1d_2d_3q^6] - \alpha a_{34}(a_{11} - d_1q^2)(a_{22} - d_2q^2) \end{aligned} \tag{14}$$

Now for diffusive instability, one of the following conditions must be violated.

$$\begin{aligned} &m_1(\theta, q^2) > 0 \\ &m_1(\theta, q^2)m_2(\theta, q^2) - m_3(\theta, q^2) > 0 \\ &m_1(\theta, q^2)\{m_2(\theta, q^2)m_3(\theta, q^2) - m_1(\theta, q^2)m_4(\theta, q^2)\} - \{m_3(\theta, q^2)\}^2 > 0 \\ &m_4(\theta, q^2) > 0 \end{aligned} \tag{15}$$

The first condition holds for $P_1 > 0$. Also, for $q^2 > (a_{22}/d_2) \equiv q_c$ the fourth condition will be satisfied. The second condition will hold provided $P_1(\alpha)P_2(\alpha) - P_3(\alpha) > 0$ which is one of the criteria for stability of the non-diffusive system. Thus system (1) will undergo diffusion-driven instability when

$$m_1(\theta, q^2)[m_2(\theta, q^2)m_3(\theta, q^2) - m_1(\theta, q^2)m_4(\theta, q^2)] - m_3(\theta, q^2)^2 < 0 \tag{16}$$

Hence the toxication rate θ together with the wave number of perturbation q play an important role in the occurrence of diffusion-driven instability for the system. A numerical simulation of the model system (9) is performed using MATLAB and the resulting spatial patterns of toxic and non-toxic phytoplankton and the zooplankton are shown in Figure 6 – 8. The figures indicate that the spatial models exhibit unstable behaviour for the same parameter values which impart stability to the non-spatial system; this numerically confirms existence of diffusion-driven instability.

5. Role of spatiotemporal kernel

The delay-diffusion model of the previous section using a spatial kernel ignores the fact that harmful phytoplankton, that are undergoing continuous drift due to lateral diffusion, are not at the same location as at previous times. In this section, we take into account this *nonlocal effect* [44] within a finite interval I by incorporating a second convolution in the integral term of equation (1) which converts the spatial kernel of (1) into a spatiotemporal kernel. The effect of toxins on zooplankton now depends not only on phytoplankton density at one point in space and time, but on the weighted average involving values at all previous times and all points in space within I. With these assumptions, we have the following modified model

$$\begin{aligned} \frac{\partial N}{\partial t} &= d_1\frac{\partial^2 N}{\partial x^2} + r_1N\left(1 - \frac{N}{k_1}\right) - \alpha_1\frac{NZ}{a+N} - \mu NT \\ \frac{\partial T}{\partial t} &= d_2\frac{\partial^2 T}{\partial x^2} + r_2T\left(1 - \frac{T}{k_2}\right) - \alpha_2\frac{TZ}{b+T} \\ \frac{\partial Z}{\partial t} &= d_3\frac{\partial^2 Z}{\partial x^2} + \beta_1\frac{NZ}{a+N} + \beta_2\frac{TZ}{b+T} - dZ \\ &\quad -\theta Z\int_{-\infty}^{t}\int_0^L \frac{e^{-\beta|x-y|}\alpha e^{-\alpha(t-s)}T(s,y)}{\int_0^L e^{-\beta|x-z|}dz}dsdy \end{aligned} \tag{17}$$

with the boundary condition $\frac{\partial N}{\partial x} = \frac{\partial T}{\partial x} = \frac{\partial Z}{\partial x} = 0$ at $x = 0, L$ where $I = (0, L)$ and same initial conditions as in (1).
System (17) is equivalent to

$$\begin{aligned}
\frac{\partial N}{\partial t} &= d_1 \frac{\partial^2 N}{\partial x^2} + r_1 N\left(1 - \frac{N}{k_1}\right) - \alpha_1 \frac{NZ}{a+N} - \mu NT \\
\frac{\partial T}{\partial t} &= d_2 \frac{\partial^2 N}{\partial x^2} + r_2 T\left(1 - \frac{T}{k_2}\right) - \alpha_2 \frac{TZ}{b+T} \\
\frac{\partial Z}{\partial t} &= d_3 \frac{\partial^2 N}{\partial x^2} + \beta_1 \frac{NZ}{a+N} + \beta_2 \frac{TZ}{b+T} - dZ - \theta Z \int_0^L \frac{e^{-\beta|x-y|} R(t,y)}{\int_0^L e^{-\beta|x-z|} dz} dy
\end{aligned} \tag{18}$$

Using the transformation as in (10) and linearizing we find

$$\begin{aligned}
\frac{\partial u_1}{\partial t} &= d_1 \frac{\partial^2 u_1}{\partial x^2} + a_{11}u_1 + a_{12}u_2 + a_{13}u_3 \\
\frac{\partial u_2}{\partial t} &= d_2 \frac{\partial^2 u_2}{\partial x^2} + a_{22}u_2 + a_{23}u_3 \\
\frac{\partial u_3}{\partial t} &= d_3 \frac{\partial^2 u_3}{\partial x^2} + a_{31}u_1 + a_{32}u_2 + a_{34} \int_0^L \frac{e^{-\beta|x-y|} R(t,y)}{\int_0^L e^{-\beta|x-z|} dz} dy \\
\frac{\partial u_4}{\partial t} &= \alpha(u_3 - u_4)
\end{aligned} \tag{19}$$

We assume solution of (19) in the form

$$\begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \end{pmatrix} = \begin{pmatrix} \alpha_1 \\ \alpha_2 \\ \alpha_3 \\ \alpha_4 \end{pmatrix} \cos(qx) e^{\lambda t} \tag{20}$$

where q is the wave number and λ is the frequency. The boundary conditions imply that $q = \frac{n\pi}{L}$ where n is the half-wavelength and

$$\gamma = \frac{1}{\int_0^L \Gamma^2(x) dx} \int_0^L \left[\int_0^L \frac{e^{-\beta|x-y|} \Gamma(y)}{\int_0^L e^{-\beta|x-z|} dz} dy \right] \Gamma(x) dx \tag{21}$$

where

$$\Gamma(x) = \cos(kx) \tag{22}$$

The characteristic equation of (19) will be

$$\lambda^4 + S_1\lambda^3 + S_2\lambda^2 + S_3\lambda + S_4 = 0 \tag{23}$$

where

$$\begin{aligned}
S_1 &= m_1 \\
S_2 &= S_{21} - \gamma\alpha a_{34} \\
S_3 &= S_{31} - \gamma\alpha a_{34}(A + d_1q^2 + d_2q^2) \\
S_4 &= S_{41} - \gamma\alpha a_{34}(a_{11} - d_1q^2)(a_{22} - d_2q^2) \\
S_{21} &= m_2 + \alpha a_{34}; \quad S_{31} = m_3 + \alpha a_{34}(A + d_1q^2 + d_2q^2) \\
S_{41} &= m_4 + \alpha a_{34}(a_{11} - d_1q^2)(a_{22} - d_2q^2)
\end{aligned} \tag{24}$$

To study the stability of the system we have to look at the following inequalities.

$$\begin{aligned} S_1 &> 0 \\ S_1S_2 - S_3 &> 0 \\ S_1(S_2S_3 - S_1S_4) - S_3^2 &> 0 \\ S_4 &> 0 \end{aligned} \tag{25}$$

Now, $m_1 > 0$ implies $S_1 > 0$. Also, positivity of S_4 is assured from $P_4 > 0$ and $S_1S_2 - S_3 > 0$ follows from $m_1m_2 - m_3 > 0$. So, the stability criteria of (19) will be determined by the third inequality above. Let us denote

$$S_1(S_2S_3 - S_1S_4) - S_3^2 \equiv I_1 + \gamma I_2 \tag{26}$$

where I_1 represent terms devoid of γ and

$$\begin{aligned} I_2 &= \alpha a_{34}[\alpha a_{34}S_1\{A + (d_1 + d_2)q^2\} - (S_1S_{21} - 2S_{31})\{A + (d_1 + d_2)q^2\} \\ &- S_1S_{31} + S_1^2(a_{11} - d_1q^2)(a_{22} - d_2q^2)] \end{aligned} \tag{27}$$

From (21) it is clear that $|\gamma| \leq 1$. Simple algebra reveals that

$$m_1(m_2m_3 - m_1m_4) - m_3^2 = I_1 + I_2 \tag{28}$$

Rewriting (27) in the form

$$\begin{aligned} I_2 &= \alpha a_{34}[(d_1d_2S_1^2)q^4 + \{\alpha a_{34}S_1(d_1 + d_2) - (S_1S_{21} - 2S_{31})(d_1 + d_2) \\ &- S_1^2(a_{11}d_2 - a_{22}d_1)\}q^2 + \{A\alpha a_{34}S_1 - A(S_1S_{21} - 2S_{31}) + S_1^2a_{11}a_{22}\}] \end{aligned} \tag{29}$$

it is seen that I_2 is a quadratic in q^2. Therefore, if $I_2 = 0$ has two real roots say, q_-^2 and q_+^2 with $q_+^2 > 0$, then I_2 will be positive provided $q_-^2 < q^2 < q_+^2$ or $0 < q^2 < q_+^2$ according as $q_-^2 > 0$ or $q_-^2 < 0$.

Now we consider the following cases

1 If I_1 and I_2 are both positive then the population will have uniform distribution and no pattern can be generated with or without nonlocal effect.

2 If $I_1 > 0$, $I_2 < 0$ but $I_1 + I_2 > 0$ then $I_1 + \gamma I_2$ will also be positive as $|\gamma| \leq 1$. In this case also, no diffusive instability will occur.

3 If $I_1 < 0$ and $I_2 < 0$ (and consequently $I_1 + I_2 < 0$) then the system with spatial as well as spatiotemporal kernel will exhibit diffusion induced instability.

4 Finally, when $I_1 < 0$, $I_2 > 0$ but $I_1 + I_2 > 0$, then $I_1 + \gamma I_2$ can have negative values. Thus in this situation the model with spatiotemporal kernel can exhibit diffusion-driven instability under the same parameter restriction which ensures stability for the model with spatial kernel. Moreover, diffusive instability in this case is controlled by the toxication rate θ together with q.

6. Discussion

In the present paper we have investigated the effect of spatial and spatiotemporal variation on a mathematical model of harmful algae where the toxic and nontoxic phytoplankton have distinct dynamical evolution.

In section 3, we have studied the effect of toxication delay on the system dynamics, where we have taken the delay in the distributed form. Our analysis showed that instead of the delay parameter α, the dynamics is controlled by the toxication rate θ. From our numerical study, we have obtained *two threshold*

values of θ, the first one of which facilitates extinction of toxic phytoplankton and the second one, that of nontoxic phytoplankton while the other two populations coexist in an oscillatory way. Thus when delay in toxin production is taken into account, extinction of zooplankton is not possible and the plankton bloom is contributed either by the toxic or the nontoxic phytoplankton together with the zooplankton. We have also obtained stable coexistence of toxic phytoplankton and zooplankton for further higher values of θ. Thus, the toxication rate θ can be a very significant parameter in the context of occurrence and/or termination of algal blooms.

Next we have analyzed a spatial extension of the delay model. Spatial patchiness is a very prominent aspect of marine plankton ecology and hence spatial models are of paramount interest in this context. We have divided our spatial study into two parts. In section 4, we have utilized a spatial kernel which measures toxin production *point wise in space.* In section 5, we have explored a spatiotemporal kernel that incorporates the so called *nonlocal effect* [44] and hence takes into account the fact that the plankton may not be at the same point in space as at earlier times due to continuous drift. Our analysis in both these cases revealed the occurrence of diffusive instability which is controlled by the toxication parameter θ together with the wave number q. Thus, the toxication rate θ is seen to have a very significant impact on the system dynamics in the presence and absence of spatial effect and with spatial as well as spatiotemporal kernel.

Another noticeable outcome of the above analysis is that the spatiotemporal kernel is capable of generating diffusion-driven instability under the same parametric conditions for which the spatial model remains stable. As diffusion process is known to have a destabilizing effect on plankton dynamics, the above finding exemplifies the realism of spatiotemporal kernels over the spatial ones in the context of marine plankton ecology.

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Dr. Banibrata Mukhopadhyay has obtained the Ph.D. degree in Applied Mathematics from the University of Calcutta in 2007. He has worked under the guidance of Prof. C.G. Chakrabarti, S.N. Bose Professor of Theoretical Physics, Department of Applied Mathematics, University of Calcutta. His research interests are Dynamical modelling using diffusion and delay differential equations with application to complex biological systems.

Department of Science, Central Calcutta Polytechnic, 21, Convent Road, Kolkata -700 014, India.

E-mail : banibrat001@yahoo.co.in

Dr. Rakhi Bhattacharyya has completed her Ph.D. from the University of Calcutta. She has obtained her M.Sc. and M.Phil. degrees from the University of Calcutta. Her research interest focus on stability and bifurcation analysis of dynamical models with application to biological systems.

Department of Science, The Calcutta Technical School, 110, S.N. Banerjee Road, Kolkata-700 013, India

E-mail : rakhi_bhattach@yahoo.co.in