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Spatial dynamics of a nutrient–phytoplankton system with toxic effect on phytoplankton

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ABSTRACT

The production of toxins by some species of phytoplankton is known to have several economic, ecological, and human health impacts. However, the role of toxins on the spatial distribution of phytoplankton is not well understood. In the present study, the spatial dynamics of a nutrient–phytoplankton system with toxic effect on phytoplankton is investigated. We analyze the linear stability of the system and obtain the condition for Turing instability. In the presence of toxic effect, we find that the distribution of nutrient and phytoplankton becomes inhomogeneous in space and results in different patterns, like stripes, spots, and the mixture of them depending on the toxicity level. We also observe that the distribution of nutrient and phytoplankton shows spatiotemporal oscillation for certain toxicity level.

1. Introduction

Phytoplankton are the microscopic organisms and form the base of all aquatic food chains. Their presence is important for the large-scale global processes such as the global carbon cycle, ocean-atmosphere dynamics and climate change. In natural systems, phytoplankton are known to occur in aggregates or patches which results due to the inhomogeneous distribution of phytoplankton in space [1]. The formation of patches alters the optical and acoustical properties of the water column and can have significant impact on the ecological dynamics of marine communities. Several mechanisms have been proposed to describe the patchiness of phytoplankton in the ocean. Some of these mechanisms can be found in Malchow et al. [2].

On a spatio-temporal scale, the complexity of species interaction is considered as one of the main factors behind the regulation of phytoplankton dynamics [3]. In this respect, toxic chemicals released by some species of phytoplankton (known as toxin producing phytoplankton (TPP)) which are common in most aquatic ecosystems, has the ability to affect species interaction by suppressing the growth and establishment of other species of phytoplankton [4]. However, the release of toxic chemicals has other ecological roles, such as precursors for subcellular organelles, cell-wall degradation products, nucleic acid synthesis, nitrogen storage, and defense against predation [5,6]. Previous studies revealed the importance of toxins affecting other species of phytoplankton in maintaining biodiversity of phytoplankton [7,8] and also on the bloom dynamics [9]. In spite of such huge importance, there has been little research focused on the role of toxins on the spatial patch formation of phytoplankton.

To the best of our knowledge, there are only few studies showing the occurrence of inhomogeneous biomass distribution of phytoplankton in the presence of TPP due to the spatial movements of plankton. Rao [10] considered a simple TPP–zooplankton system with toxic effect on zooplankton and showed different complex pattern formations depending on the environmental carrying capacity. Chaudhury et al. [11] investigated the effect of toxins released by TPP on grazer zooplankton in a system consisting of two competing phytoplankton species among which one is toxic and one grazer zooplankton. However, they did not consider the effects of toxins on the competing phytoplankton species. Another paper by Roy [12] dealt with two different models; one is a two phytoplankton-one zooplankton system with toxic effect on zooplankton, qualitatively similar in structure with the model of Chaudhury et al. [11], and another one consists of three competing species of phytoplankton among which one is able to release toxic chemicals which negatively affect the growth of the other two species. In both of these studies, abundance of TPP was considered as the representative of the toxic effects. However, in real plankton systems, there is no strict correlation between the toxicity and the abundance of toxic species [13,14]. Some species, like *Phaeocystis*, become toxic only when they are abundant. On the

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other hand, *Dinophysis* and *Alexandrium* can be potentially toxic at very low concentrations. Moreover, several other environmental and physical factors also affect the release of toxic chemicals by TPP and accordingly, the rate of the release of toxic chemicals and its toxic effects on other species can vary drastically [15–17]. Therefore, the abundance of TPP cannot always be a perfect representative of the toxic effects on other species of phytoplankton. Here, we attempt to overcome this shortcoming by considering a simple bottom up model without the presence of TPP but with toxic effects on other phytoplankton species. Moreover, algebraically simple models are more suitable for examining the generic behavior of a system [18].

In the present study, we consider the model originally developed by Chakraborty et al. [9] to describe the interaction of a nutrient and a species of phytoplankton with toxic effects on them, applicable to situations where the species is supposed to experience spatial homogeneity. We modify the system by incorporating the effect of spatial movement in the form of diffusion. Our aim is to investigate how the influence of toxic effect affects the spatial distribution of phytoplankton. To do this, we vary the toxic effect on phytoplankton in the presence of spatial movement of nutrient and phytoplankton and explore the distribution of nutrient and phytoplankton in space.

Rest of the paper is organized as follows. In Section 2, we describe the nutrient–phytoplankton (NP) model with toxic effect on phytoplankton in the presence of diffusion. In Section 3, first we analyze the model and state the stability conditions in the absence of diffusion, then analyze the system in the presence of diffusion and find the stability conditions. Specifically, we search for the condition under which Turing instability occurs, where the system without diffusion remains stable, but becomes unstable in the presence of diffusion. In Section 4, we provide numerical evidences of the effects of toxicity on the variation of spatial distribution of nutrient and phytoplankton. Finally, the paper ends with a discussion in Section 5.

2. Basic model structure

The spatial extension of the model developed by Chakraborty et al. [9] describing the interaction of a nutrient and a species of phytoplankton can be written as:

$$\begin{aligned}\frac{\partial N}{\partial t} &= a - bNP - eN + D_N\left(\frac{\partial^2 N}{\partial x^2} + \frac{\partial^2 N}{\partial y^2}\right),\\ \frac{\partial P}{\partial t} &= cNP - dP - \theta\frac{P^2}{\mu^2 + P^2} + D_P\left(\frac{\partial^2 P}{\partial x^2} + \frac{\partial^2 P}{\partial y^2}\right),\end{aligned} \tag{1}$$

where $N(x, y, t)$ (g C m^{-3}) and $P(x, y, t)$ (g C m^{-3}), respectively, represent the nutrient concentration and phytoplankton abundance at any time t and location (x, y). Here, a is the rate of constant external nutrient input into the system, b is the maximal nutrient uptake rate of phytoplankton, c is the maximal growth rate of phytoplankton, d is the per capita-mortality rate of phytoplankton, e is the per capita-loss rate of nutrient due to sinking from the epilimnion down to the hypolimnion and therefore making these nutrients unavailable for phytoplankton uptake, θ represents the rate of release of toxic chemicals by the TPP population which measures the strength of toxic effect on phytoplankton population, and μ denotes the half-saturation constant. Here, the rate of toxin released by the TPP is proportional to the crowding of the phytoplankton and the toxic effect becomes significant when the phytoplankton population reaches high concentration [19]. D_N and D_P represent the rate coefficients of self-diffusion of N and P, respectively. Let Ω be the two-dimensional bounded connected square domain with $\partial\Omega$ as boundary, and $\frac{\partial}{\partial\eta}$ be the outward drawn normal derivative on the boundary. In Ω, we take the following initial conditions for system (1):

$$N(x, y, 0) = N_0(x, y) > 0, \quad P(x, y, 0) = P_0(x, y) > 0, \quad \forall \quad (x, y) \in \Omega$$

and the zero-flux boundary conditions

$$\left.\frac{\partial N}{\partial \eta}\right|_{(x,y)} = \left.\frac{\partial P}{\partial \eta}\right|_{(x,y)} = 0, \quad \text{where } (x, y) \in \partial\Omega.$$

It is to be noted here that, the similar system without toxic effect for a homogeneous environment was considered and analyzed by Huppert et al. [20]. We also want to mention here that, although our aim is to model the effect of toxins produced by TPP on the spatial distribution of phytoplankton, and according to that we choose our model system, but the term corresponding to the toxic effect can also represent predation on phytoplankton [21]. Therefore, the model can be also used to study the role of predation on the spatial distribution of phytoplankton. However, in the present study, we prefer to stick to the notation of toxic effects, though the model itself is much more general.

3. Stability analysis

3.1. Temporal model and its stability analysis

First, we perform the stability analysis of the model system (1) in the absence of diffusion. In this case, the system has two non-negative equilibria, namely; trivial equilibrium $E_1 = (\frac{a}{e}, 0)$ and interior equilibrium $E^*(N^*, P^*)$ with $N^* = \frac{a}{e+bP^*}$, and P^* is a positive root of the equation

$$bdP^3 + (b\theta - (ac - de))P^2 + (\theta e + bd\mu^2)P - (ac - de)\mu^2 = 0.$$

It is clear that the equilibrium E_1 always exists. Moreover, a unique positive interior equilibrium exists if $0 < ac - de \le b\theta$.

From the biological point of view, we are mainly interested in studying the stability of the interior equilibrium point E^*. The Jacobian matrix corresponding to E^* can be written as:

$$J = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix},$$

where

$$\begin{aligned} a_{11} &= -(bP^* + e), \quad a_{12} = -bN^*,\\ a_{21} &= cP^*, \quad a_{22} = \theta\frac{P^{*2} - \mu^2}{(\mu^2 + P^{*2})^2}P^*. \end{aligned}$$

The corresponding characteristic equation of J is

$$\lambda^2 + A\lambda + B = 0,$$

where

$$\begin{aligned} A &= -(a_{11} + a_{22}),\\ B &= a_{11}a_{22} - a_{12}a_{21}. \end{aligned}$$

Using the Routh–Hurwitz criterion together with the above equation, the following theorem follows immediately:

Theorem 1. *The equilibrium point E^* of system (1) in the absence of diffusion is locally asymptotically stable iff $A > 0$ and $B > 0$.*

3.2. Spatiotemporal model and Turing instability

Now, we study the effect of diffusion on the stability of the system about the interior equilibrium point E^*. Specifically, we are interested in investigating the Turing instability of the system where the uniform steady state of the system without diffusion is stable, but it is unstable in the partial differential equations with diffusion terms. To study this, first we consider the linearized form of system (1) about $E^*(N^*, P^*)$ as