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Avoiding toxic prey may promote harmful algal blooms

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ABSTRACT

Blooms of freshwater cyanobacteria are a worldwide spread environmental issue. Despite toxin producing planktonic species are generally expected to be poor competitors for resources, dense blooms of toxic cyanobacteria, such as *Microcystis*, do often occur in nature. Experimental results suggest that the formation of such blooms is promoted by the predatory activity of zooplankton. In fact, such predator grazes on both the nontoxic and toxic species despite the toxicity of the latter actually inhibits its growth. We model this phenomenon through a Lotka–Volterra reaction–diffusion system. Our goal is to investigate the coupled role of toxicity and zooplankton's predation in the persistence of the toxic prey and to study the mechanisms behind the formation of spatially local toxic blooms. It is known that the classical Lotka-Volterra system consisting of one prey and one predator never exhibits pattern formation. In this paper, we show that the introduction of a toxic prey may destabilize the spatially homogeneous coexistence and trigger spatial pattern formation. We also show that local blooms more likely occur when predators avoid the toxic prey and when the strength of the toxicity is of an intermediate level.

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1. Introduction

The term harmful algal bloom (in short, HAB) indicates an algal bloom that has negative impacts on other organisms via the production of toxins, mechanical damage, or by other means. HAB include different types of taxa such as dinoflagellates, diatoms, and cyanobacteria (commonly known as blue-green algae). The latter are of special importance because of their potential impact on drinking or recreational waters. In fact, they can produce a variety of potent toxins called *cyanotoxins* (e.g., Falconer and Humpage, 1996). These compounds have been found to be hepatotoxic or neurotoxic for a wide range of organisms, including humans, and several intoxication cases have been reported worldwide (Jochimsen et al., 1998). Therefore, in the recent years, the formation of toxic blooms of cyanobacteria in lakes and rivers has been causing more and more concern.

Among the great variety of cyanobacteria that can produce toxins, a common bloom forming one is *Microcystis*. This species is usually composed of both toxic and nontoxic strains (Kardinaal et al., 2007). Toxin producing planktonic species are generally expected to be poor competitor for resources (Porter and Orcutt, 1980; Mitra and Flynn, 2006), even though experimental results suggest that increments in temperature and changes in the ratios of nutrients might reverse the situation (Davis et al., 2009; Fujimoto et al., 1997). These evidence suggest that toxic and nontoxic species of freshwater phytoplankton hardly coexist in absence of other species. In particular, competition experiments have shown that the toxic strain of *Microcystis* is a very poor competitor for light (Kardinaal et al., 2007). In these experiments the toxic strain always lost the competition against the nontoxic one, even when given a strong initial advantage. Then how can these species survive and actually bloom?

Toxin-producing *Microcystis* has overall an inhibitory effect on the growth of most herbivore taxa. Nevertheless, zooplankton usually grazes on both toxic and nontoxic species (Fulton and Paerl, 1988). This is interesting, since the toxic or noxious chemicals produced by blue-green algae may inhibit feeding and, over long term, cause mortality of zooplankton (Porter and Orcutt, 1980; Lampert, 1981, 1982; Fulton and Paerl, 1987). In particular, while a few species like the rotifer *Brachionus calyciflorus* and the cladoceran *Bosmina longirostris* apparently make no great distinction between toxic and non toxic prey, the feeding rates of other small-bodied cladocerans, rotifers, and copepods seem to be strongly related to the toxicity of *Microcystis* (Fulton and Paerl, 1987). In view of these observations, we consider predator and toxic prey to have an inhibitory effect on each other. Can the existence of such interaction promote the spatial pattern formation and local algal blooms?

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Lampert (1981) suggests that toxicity to planktonic grazers has a very protective value: it reduces the grazing pressure directly (a fast mechanism), and it lowers the population density of predators (a long lasting effect). Wang et al. (2010) propose that the formation of surface blooms of *Microcystis* is strongly connected to the presence or absence of zooplankton, and to its selective predatory activity. In fact, the authors observed that if the zooplankton was removed from the water at the start of the experiment, no surface bloom of *Microcystis* appeared regardless of the addition of nutrients.

In this paper, through the analysis of a mathematical model, we investigate the mechanisms behind the formation of spatial blooms of toxic cyanobacteria. Previous studies have shown that the classical one-prey and one-predator Lotka-Volterra system never exhibits spatial pattern formation, while higher nonlinearities such as Allee effect can induce such a phenomenon (Murray, 1975; Mimura and Murray, 1978; Mimura, 1978). Thus, a complicated functional response is a candidate for the mechanism of spatial blooms. Biologically, other factors such as the spatial variation in light, nutrients and water temperature might play an important role (for example see the above mentioned Fujimoto et al., 1997; Davis et al., 2009). Here we study another possibility. Can spatially local blooms be explained only by the presence of toxin producing species that have a negative effect on the growth of zooplankton? To answer this question, we propose a two-prey-one-predator Lotka-Volterra system in which one of the prey has a toxic effect that inhibits the growth of the predators. We assume that the toxic prey is a weaker competitor for resources and the competition between the two prey in absence of the predator is monostable in favor of the nontoxic prey. We will further introduce a function that, depending on the toxicity and another parameter, describes whether the zooplankton is more or less inclined to avoid the toxic prey. After a preliminary analysis of the ordinary differential equations, we focus on the reaction–diffusion system in order to study the spatiotemporal dynamics of the model. Our goal is to show the effect of the existence of toxin-producing phytoplankton species on the spatial pattern formation and to investigate how the formation of toxic blooms is related to the selective predatory activity of zooplankton. Toward this goal, we perform the bifurcation analysis and numerical calculations of the reaction–diffusion system.

2. The mathematical model

In general, a three-species Lotka–Volterra system is written as follows:

$$\frac{dN_i}{dT} = N_i\left(r_i - \sum_{j=i}^{3}\beta_{ij}N_j\right), \quad i = 1, 2, 3; \quad T > 0. \tag{2.1}$$

When N_1 and N_2 represent the abundance of two prey species and N_3 represents the abundance of a predator at time T, the coefficients can be set as:

$$\begin{aligned}&\beta_{ij} > 0 \quad \text{for } i = 1,2; \quad j = 1,2,3;\\ &\beta_{ij} < 0 \quad \text{for } i = 3; \quad j = 1,2;\\ &\beta_{33} = 0.\end{aligned}$$

This model was proposed and studied by Parrish and Saila (1969) who showed that the introduction of a predator can delay the extinction of one prey species. Cramer and May (1972) gave conditions for the coexistence of three species. During the 70s, this model received a lot of attention and since then it has been studied under many points of view by several authors. Fujii (1977) refined the results of Cramer and May (1972) and suggested the existence of a stable limit cycle in the three species state space. His results were then improved by Takeuchi and Adachi (1983). This type of system also showed interesting chaotic dynamics, which have been studied by Gilpin (1979) and Schaffer and Kot (1985). Klebanoff and Hastings (1994) used bifurcation analysis to show that chaotic dynamics can be expected in such type of systems.

2.1. Assumptions

To construct a system aiming to model our biological situation, we make the following assumptions:

- **A1**: In absence of toxic prey, predator and nontoxic prey coexist;
- **A2**: In absence of nontoxic prey, predators go extinct;
- **A3**: In absence of predators, the nontoxic prey is a superior competitor for resources (i.e., monostable).

Assumption **A1** corresponds to the co-occurrence of different species of (nontoxic) phytoplankton and zooplankton (e.g., Hutchinson, 1961). Assumption **A2** is based on the empirical results showing that the toxicity of some species of phytoplankton inhibits or have a non-nutritious effect on zooplankton (Lampert, 1981; Fulton and Paerl, 1987, 1988). Assumption **A3** is based on the idea that toxic strains are eventually outcompeted by nontoxic ones (e.g., Kardinaal et al., 2007; Lampert, 1981). We will further assume that the two prey species share the same growth rate and carrying capacity. This assumption may seem not realistic. However, our main goal is to investigate the effects that the parameter controlling the toxicity has on the dynamics. Therefore, we keep the dependency of the system on other factors at minimum. Note that some experimental results are consistent with our simplification (Briand et al., 2008; Imai et al., 2009; Schatz et al., 2005; for details see the final discussion).

2.2. The model

Let N_1, N_2, and N_3 denote the nontoxic, toxic, and predator species respectively in (2.1). Then we notice that β_{32} is positive. We now rewrite (2.1) as the following system of ordinary differential equations:

$$\begin{aligned}\frac{dN_1}{dT} &= N_1(\rho - \alpha N_1 - \alpha_{12}N_2 - \alpha_{13}N_3)\\ \frac{dN_2}{dT} &= N_2(\rho - \alpha N_2 - \alpha_{21}N_1 - \alpha_{23}N_3); \quad T > 0\\ \frac{dN_3}{dT} &= N_3(\alpha_{31}N_1 - \alpha_{32}N_2 - \rho_3)\end{aligned} \tag{2.2}$$

This system can be schematized as shown in Fig. 1 and Table 1 lays out the parameters and their units. In order to simplify the system, we rescale some parameters to one by using the following transformations,

$t = \rho_3 T, \quad P = \frac{N_1\alpha_{31}}{\rho_3}, \quad X = \frac{N_2\alpha_{31}}{\rho_3}, \quad Z = \frac{N_3\alpha_{13}}{\rho}.$

We obtain the following non-dimensional system:

$$\begin{aligned}\frac{dP}{dt} &= rP\left(1 - \frac{P + aX}{K} - Z\right)\\ \frac{dX}{dt} &= rX\left(1 - \frac{X + bP}{K} - dZ\right); \quad t > 0\\ \frac{dZ}{dt} &= Z(P - \mu X - 1)\end{aligned} \tag{2.3}$$

where:

$$a = \frac{\alpha_{12}}{\alpha}, \quad b = \frac{\alpha_{21}}{\alpha}, \quad d = \frac{\alpha_{23}}{\alpha_{13}}, \quad K = \frac{\rho\alpha_{31}}{\rho_3\alpha}, \quad r = \frac{\rho}{\rho_3}, \quad \mu = \frac{\alpha_{32}}{\alpha_{31}}.$$

The parameters r, a, b, d, K and μ are all positive.

The most interesting parameter to our purposes is μ, to which from now on we will refer as "toxicity". Toxicity then is defined

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