MIMICKING THE SPATIAL SELF DEFENSE EFFECT IN A TOXIC PHYTOPLANKTON - ZOOPLANKTON SYSTEM

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Abstract

Plankton dynamics is a fascinating and interesting subject of research. There are lots of aspects influencing plankton dynamics. Approximately 7% of the phytoplankton species are known to form large-scale blooms, dramatically affecting marine communities [1]. These blooms are formed because of the formation of different patches/ colonies by the phytoplankton population. Various studies have demonstrated that the formation of colonies/ patches by green alga offers considerable protection against grazing by zooplankton [2]. The potent neurotoxin production by many microalgal species may have some direct or indirect effect in forming a patch and might be perceived by its grazer as group defense. The defense strategy and patch formation of toxin producing phytoplankton (TPP) may give a possible answer to the evergreen crucial ecological question of why do many microalgal species produce neurotoxins. In the present paper we propose a simple model of TPP-zooplankton interactions in which the former is assumed to be able to detect the presence of zooplankton and counteract it by forming colonies or patches and releasing some toxic chemicals in the surrounding water. We observe that the fraction of TPP population that aggregates to form colonies or patches, and the number of colonies or patches they form, plays an important role in the recurrent bloom phenomenon. We also observe that the formation of patch by the TPP decreases the grazing pressure of zooplankton resulting in stronger coupling between the interacting species determined by the fraction of the phytoplankton population that aggregates to form colonies or patches and also on the number of patches.

Keywords: Phytoplankton-Zooplankton, toxic chemicals, patch, recurrent bloom, coexistence.

Presenting Author's Biography

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1 Introduction

Toxic or otherwise Harmful algal blooms (HAB) are increasing in frequency worldwide [3, 4] and have negative impact on aquaculture, coastal tourism and human health [5]. The appearance of a bloom can have devastating implications. But, Jansen and coworkers [6] remarked that bloom forming phytoplankton species are not harmful and serve as energy resource at the base of the food web. The complex and inconsistent interactions between toxin producing phytoplankton (TPP) and their grazers may be due to the level and solubility of toxicity. However, knowledge about interactions between TPP and their potential grazers are only rudimentary [7, 8]. Also, we know a little about how phytoplankton bloom occurs. There are lot of theories available to explain the bloom phenomenon. Some of them use 'top-down' mechanism [9, 10, 11, 12] to explain the bloom, i.e., according to them the occurrence of phytoplankton bloom depends on their grazing pressure, while some use 'bottom-up' mechanism [13, 14, 15, 16], i.e., according to them the occurrence of bloom depends on the availability of the nutrient. Some researchers use simultaneous effect of both top down and bottom up mechanisms to explain the bloom phenomenon [17]. Quite a good number of studies [18, 19, 20] with the above mentioned mechanisms have considered TPP as an important factor to explain the recurring bloom formation which can be used to control bloom in aquatic systems [21, 22]. But, none of the above studies have taken into account the formation of patch by TPP which then demands more in depth studies.

The toxin liberated by the phytoplankton may be regarded as an anti-grazing strategy [23]. Among the various other anti-grazing strategies observed so far for phytoplankton, cell morphology [24], presence of gelatinous substances, or the formation of colonies [25] and filamentous structures [26] are widely recognized. Since phytoplankton in the ocean are small relative to their predatory enemies, they will not survive an encounter with a grazer. But, phytoplanktons are not defenseless food-particles that are easily harvested by the consumers. Several algal species are able to adjust their phenotype (colony formation, spines, size) in such a way that it results in a reduced grazing pressure [27]. The anti-grazing strategy is not only important for the existence of the phytoplankton species but also for many zooplankton species. It is largely determined by the ways in which the species of phytoplankton can resist mutual extinction due to competition or persistence despite grazing pressure from zooplankton [28]. This coupled defense mechanism through patching and poison release then results in the coexistence of the interacting species. Huisman and coworkers [29, 30] have used the coexistence of the species for explaining the plankton paradox and biodiversity.

It is now known that increased spine length and cells in a colony of members of a phytoplankton species (like genus *Scenedesmus*), when zooplankton grazing is intense, helps in reducing zooplankton filtering rates, the effect of these defense mechanisms at the population level has been observed in few studies [28]. The study of the defense mechanism through the formation of colonies or patches becomes more important if such colonies or patches have the ability to release toxin chemicals, like in case of dinoflagellate [32]. Toxic chemicals released through chemical signals by aquatic organisms may have indirect and cascading effects on the ecology of entire community and ecosystems. These signals between microbial predators and prey may contribute to food selection or avoidance and to defense, factors that probably affect trophic structure and algal blooms [23]. For example zooplankters, like Copepods being highly selective often can avoid eating the toxic phytoplankton and thus escape its adverse effects [33]. In such cases do the level of toxicity and the fraction of the phytoplankton population that aggregates to form patches enhance the strength of coupling between interacting species? We are also interested to find out the role of colonies/ patches and the toxic chemicals in the recurrent bloom phenomenon. As such, this unknown mechanism offers considerable intellectual challenges to the theoretical and experimental ecologists.

The present paper is devoted to understand such dynamics by proposing a toxic phytoplankton–zooplankton system modeling where the phytoplankton populations are assumed to aggregate into patches as a defense mechanism. With these preliminaries in mind it is our interest to assume that the predator response function is not necessarily a monotone increasing function of prey density, but rather it is only monotonically increasing up to a certain threshold density and then becoming monotone decreasing. We will also assume that the release of phycotoxins have a negative impact on the growth of zooplankton.

2 The mathematical model

We consider a two population predator-prey system in which the prey is able to detect the presence of the predator and to act in self defense by grouping together and releasing toxin chemicals. The latter is assumed to diffuse in the surrounding water bed through the surface of the patch.

Let $P(t)$ and $Z(t)$ denote the toxin producing phytoplankton (TPP) and zooplankton population sizes respectively. Assume that the TPP population follows the law of logistic growth and the zooplankton consume phytoplankton for their growth. The dynamics of the latter shows positive growth due to predation, then we must account for natural mortality μ, and finally we include the poisoning effect.

The sketch of the model is then

$$\begin{aligned} \dot{P} &= \text{growth - predation} \\ \dot{Z} &= \text{predation - natural mortality - poisoning} \end{aligned} \tag{1}$$

We assume also that only a fraction k of the phytoplankton aggregates to form N patches. For the predation term the Holling type-II functional form is taken over

the fraction $1-k$ of the "free" phytoplankton. We propose a more complicated mechanism for the release of poison. Notice that the population in each patch will be $\frac{1}{N}kP$. Let us introduce a new parameter $\rho \equiv \left(\frac{k}{N}\right)^{\frac{2}{3}}$. If the 3D patch in the ocean can be assumed roughly to be spherical, its radius will be proportional to $\left[\frac{1}{N}kP\right]^{\frac{1}{3}}$, so that its surface is proportional to $\left[\frac{1}{N}kP\right]^{\frac{2}{3}} = \rho P^{\frac{2}{3}}$. We assume that the phytoplankton can detect the presence of zooplankton and release the poison in self defense through the surface of the patch. We are then led to the following equations

$$\begin{aligned} \dot{P} &= rP - bP^2 - \frac{c(1-k)ZP}{a+\gamma P} \equiv F_1(P,Z) \quad (2) \\ \dot{Z} &= \frac{e(1-k)ZP}{a+\gamma P} - \mu Z - e\rho P^{\frac{2}{3}} Z \equiv F_2(P,Z) \end{aligned}$$

where all parameters are nonnegative and $0 \le k \le 1$. We also assume $c \ge e$. ρ may be defined as the measure of the toxicity.

Positive invariance

By setting $X = (P,Z)^T \in R^2$ and $F(X) = [F_1(X), F_2(X)]^T$, with $F : C_+ \to R^2$ and $F \in C^\infty(R^2)$, equation (2) becomes

$$\dot{X} = F(X), \quad (3)$$

together with $X(0) = X_0 \in {R_+}^2$. It is easy to check that whenever choosing $X(0) \in {R_+}^2$ with $X_i = 0$, for i=1, 2, then $F_i(x) \mid_{X_i=0} \ge 0$. Due to the lemma of Nagumo [34] any solution of equation (3) with $X_0 \in {R_+}^2$, say $X(t) = X(t; X_0)$, is such that $X(t) \in {R_+}^2$ for all $t > 0$.

3 Equilibria and stability analysis

The system (2) has only three equilibria $E_i = (P_i, Z_i)$, $i = 0, 1, 2$: the origin E_0, the boundary equilibrium point $E_1 = \left(\frac{r}{b}, 0\right)$ and another feasible non boundary equilibrium E_2. Its positive coordinates are found in the $P-Z$ phase plane by solving the nonlinear system $\frac{e(1-k)P}{a+\gamma P} - \mu - e\rho P^{\frac{2}{3}} = 0$ and $r - bP - \frac{c(1-k)Z}{a+\gamma P} = 0$. Solving these two equations we find $Z_2 = \frac{(r-bP_2)(a+\gamma P_2)}{c(1-k)}$, where P_2 is the positive real root of the following equation,

$$\begin{aligned} \phi(P) \equiv\ & \gamma^3 e^3 \rho^3 P^5 + 3a\gamma^2 e^3 \rho^3 P^4 \\ & - \left\{(e(1-k)-\mu\gamma)^3 - 3a^2\gamma e^3\rho^3\right\} P^3 \\ & + \left\{3\mu a(e(1-k)-\mu\gamma)^2 + a^3 e^3 \rho^3\right\} P^2 \\ & - 3\mu^2 a^2 \left\{e(1-k)-\mu\gamma\right\} P + \mu^3 a^3 = 0. \end{aligned} \quad (4)$$

From Descartes' rule of sign, we observe that either there exist no positive root or more than one positive real roots for the above equation (2) depending on certain parametric conditions and if those roots are less than $\frac{r}{b}$, then there exist one or more positive equilibrium points E_2.

Tab. 1 A hypothetical set of parameter values. Unit of P and Z is $g\ m^{-3}$ and time t is days.

Parameters	values	Units
r	0.27	day^{-1}
b	0.1	$m^3\ g^{-1}\text{day}^{-1}$
c	0.3	day^{-1}
e	0.09	$m^3\ g^{-1}\text{day}^{-1}$
μ	0.1	day^{-1}
k	0.75	–
γ	0.1	–
a	0.1	$g\ m^{-3}$
ρ^3	0.0225	$g\ m^{-3}$

For example, let us consider the hypothetical set of parameter values given in Table 1. The parameter values are so chosen that the number of patches $N = 5$. With this parameter set the equation (4) becomes,

$$\begin{aligned} \phi(P) \equiv\ & 0.164\times10^{-7}P^5 + 0.492\times10^{-7}P^4 \\ & -0.1904\times10^{-5}P^3 + 0.4704\times10^{-5}P^2 \\ & -0.375\times10^{-5}P + 10^{-6} = 0. \end{aligned} \quad (5)$$

Equation (5) has two positive root 1.423 and 7.71. For $P_2 = 1.423$, we have $Z_2 = 0.412$ and for $P_2 = 7.17$, we have $Z_2 = -5.822$. Thus the value of parameters given in Table 1, gives a unique interior equilibrium point $E_2 \equiv (1.423,\ 0.412)$.

Boundedness of the solutions

Let us first recall the following lemma [35].

Lemma 1. *Let g be a real valued differential function defined on some half line $[a, +\infty)$, $a \in (-\infty, +\infty)$. If (i) $\lim_{t\to+\infty} g(t) = \alpha$; $\mid \alpha \mid < +\infty$, (ii) $g'(t)$ is uniformly continuous for $t > a$, then $\lim_{t\to+\infty} g'(t) = 0$.*

We shall prove the following key lemma.

Lemma 2. *Assume at first that the initial condition of equation (2) satisfies $P(t_0) \ge \frac{r}{b}$ then either (i): $P(t) \ge \frac{r}{b}$ for all $t \ge 0$ and therefore as $t \to +\infty$, $(P(t), Z(t)) \to E_1 = (\frac{r}{b}, 0)$ or (ii): there exists a $t_1 > 0$ such that $P(t) < \frac{r}{b}$ for all $t > t_1$. If instead $P(t_0) < \frac{r}{b}$, then $P(t) < \frac{r}{b}$ for all $t \ge 0$.*

Proof. We consider first the case $P(t) \ge \frac{r}{b}$ for all $t \ge 0$. From the first equation of (2) we get

$$\frac{dP}{dt} = rP - bP^2 - \frac{c(1-k)ZP}{a+\gamma P}. \quad (6)$$

Hence, for all $t \ge 0$, we have that $\frac{dP(t)}{dt} \le 0$. Let

$$\lim_{t\to\infty} P(t) = \eta. \quad (7)$$

If $\eta > \frac{r}{b}$, then by the Barbalat lemma [35], we have

$$\begin{aligned} 0 &= \lim_{t\to\infty} \frac{dP(t)}{dt} \\ &= \lim_{t\to\infty} \left[P(t)(r - bP(t)) - \frac{c(1-k)Z(t)P(t)}{a+\gamma P(t)} \right] \\ &\leq \lim_{t\to\infty} [P(t)(r - bP(t))] \\ &= [\eta(r - b\eta)] \ < \ 0. \end{aligned}$$

This contradiction shows that $\eta = \frac{r}{b}$ i.e.,

$$\lim_{t\to\infty} \ P(t) = \frac{r}{b}. \tag{8}$$

Of course, P(t) is differentiable and $P'(t)$ is uniformly continuous for $t \in (0, \ +\infty)$. Thus, with equation (8) all the assumptions of the Barbalat lemma are verified, so that

$$\lim_{t\to\infty} \ \frac{dP}{dt} = 0. \tag{9}$$

Combining then (8) with (2) we have

$$\lim_{t\to\infty} \frac{dP(t)}{dt} = -\lim_{t\to\infty} \frac{c(1-k)Z(t)P(t)}{a+\gamma P(t)}. \tag{10}$$

Hence, equation (8), (9) and (10) are in agreement if and only if $\lim_{t\to\infty} Z(t) = 0$. This completes the case (i).

Suppose that assumption (i) is violated. Then there exists $t_1 \ > 0$ at which for the first time $P(t_1) = \frac{r}{b}$. From equation (2) we have

$$\frac{dP(t)}{dt} \ |_{t=t_1} = \frac{-c(1-k)Z(t_1)P(t_1)}{a+\gamma P(t_1)} < 0.$$

This implies that once a solution with P has entered into the interval $(0, \frac{r}{b})$ then it remains bounded there for all $t > t_1$, i.e., $P(t) \ < \frac{r}{b}$ for all $t > \ t_1$.

Finally, if $P(t_0) \ < \ \frac{r}{b}$, then applying the previous argument it follows that $P(t) < \frac{r}{b}$ for all $t > 0$, i.e. (iii) holds true. This completes the proof.

Lemma 3. *Letting $l = \frac{(r+\eta)^2}{4b}$ there is $\eta \in (0, \mu]$ such that for any positive solution $(P(t), \ Z(t))^T$ of the system (2) for all large t we have $Z(t) < M$, with $M = \frac{l}{\eta}$.*

Proof. Lemma 2 implies that for any $(P(t_0), Z(t_0))$ such that $P(t_0) \geq \frac{r}{b}$, then either a time $t_1 > 0$ exists for which $P(t) < \frac{r}{b}$ for all $t > t_1$, or $\lim_{t\to\infty} P(t) = \frac{r}{b}$. Furthermore: if $P(t_0) < \frac{r}{b}$ then $P(t) \leq \frac{r}{b}$ for all $t > 0$. Hence in any case a non-negative time, say t^*, exists such that $P(t) \ < \ \frac{r}{b} + \epsilon$, for some $\epsilon \ > \ 0$ and for all $t > t^*$.

Set $W = P(t) + Z(t)$. Calculating the derivative of W along the solution of system (2), we find for $t > t^*$

$$\begin{aligned} \frac{dW}{dt} &= rP - bP^2 - \frac{c(1-k)ZP}{a+\gamma P} \\ &\quad + \frac{e(1-k)ZP}{a+\gamma P} - \mu Z - e\rho P^{\frac{2}{3}} Z \\ &\leq rP - bP^2 - \mu Z, \quad (\text{since}, c \geq e). \end{aligned}$$

Taking $\eta > 0$ we get,

$$\frac{dW}{dt} + \eta W \leq (r - bP + \eta)P + (\eta - \mu)Z$$

Now if we choose $\eta \leq \mu$, then

$$\begin{aligned} \frac{dW}{dt} + \eta W &\leq (r - bP + \eta)P \\ &\leq \frac{(r+\eta)^2}{4b} \equiv l \end{aligned}$$

It is clear that the right-hand side of the above expression is bounded. Thus, there exist a positive constant M, such that $W(t) < M$ for all large t. The assertion of lemma 2 now follows from the ultimate boundedness of P.

Let Ω be the following subset of $R^2_{0,+}$:

$$\Omega = \left\{ (P, Z)\varepsilon R^2_{0,+} : P \leq \frac{r}{b}, Z \leq M \right\}. \tag{11}$$

Theorem 1. *The set Ω is a global attractor in $R^2_{0,+}$ and, of course, it is positively invariant.*

Proof. Due to lemmas 2 and 3 for all initial conditions in $R^2_{+,0}$ such that $(P(t_0), \ Z(t_0))$ does not belong to Ω, either there exists a positive time, say T, $T = \max\{t_1, t^*\}$, such that the corresponding solution $(P(t), Z(t)) \in$ int Ω for all $t > T$, or the corresponding solution is such that $(P(t), \ Z(t)) \to E_1(\frac{r}{b}, 0)$ as $t \to +\infty$. But, $E_1 \in \partial\Omega$. Hence the global attractivity of Ω in $R^2_{0,+}$ has been proved.

Assume now that $(P(t_0), \ Z(t_0)) \in$ int Ω. Then Lemma 2 implies that $P(t) < \frac{r}{b}$ for all $t > 0$ and also by lemma 3 we know that $Z(t) < M$ for all large t. Finally note that if $(P(t_0), \ Z(t_0)) \in \partial\Omega$, because $P(t_0) = \frac{r}{b}$ or $Z(t_0) = M$ or both, then still the corresponding solutions $(P(t), \ Z(t))$ must immediately enter intΩ or coincide with E_1.

We have proved that the trajectories of (2) are bounded. Next we shall study the stability property of different equilibrium points.

The Jacobian matrix of the system (2) has the form

$$J_i \equiv \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix} \tag{12}$$

where,

$$\begin{aligned} a_{11} &= r - 2bP_i - \frac{c(1-k)Z_i}{(a+\gamma P_i)^2}, \ a_{12} = \frac{-c(1-k)P_i}{a+\gamma P_i}, \\ a_{21} &= e\left[\frac{(1-k)a}{(a+\gamma P_i)^2} - \frac{2}{3}\rho P_i^{-\frac{1}{3}} \right] Z_i, \\ a_{22} &= \frac{e(1-k)P_i}{a+\gamma P_i} - \mu - e\rho P_i^{\frac{2}{3}} \end{aligned}$$

At the origin, the eigenvalues are $r, \ -\mu$ are found showing its instability. At E_1, we have the eigenvalues $-r, \ e\left[\frac{(1-k)r}{ab+r\gamma} - \frac{\mu}{e} - \rho\left(\frac{r}{b}\right)^{\frac{2}{3}}\right]$. Thus, E_1 is conditionally stable if $\frac{(1-k)r}{ab+r\gamma} \leq \frac{\mu}{e} + \rho\left(\frac{r}{b}\right)^{\frac{2}{3}}$. Finally, at the

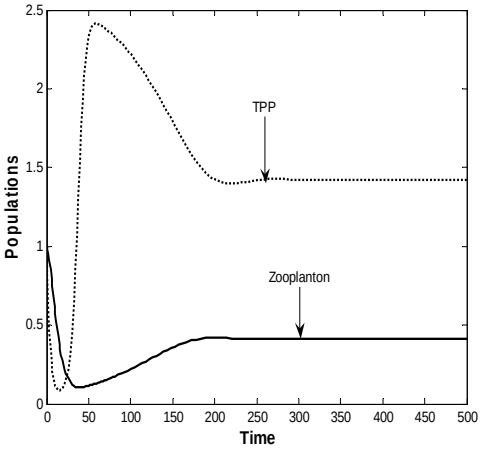

Fig. 1 The figure depicts the local stability of the system (2) around the interior equilibrium point E_2.

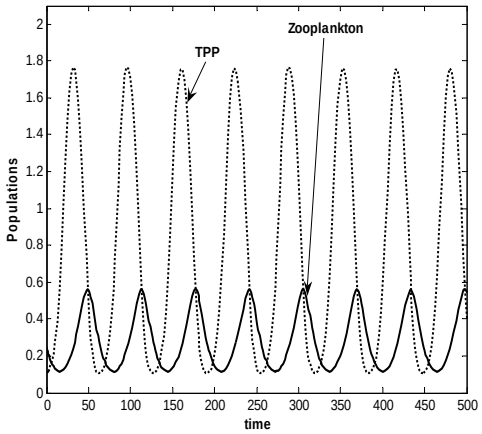

Fig. 2 The figure depicts coexistence of all the species through periodic oscillation.

interior equilibrium E_2

$$J_2 \equiv \begin{pmatrix} \frac{r(a+\gamma P_2-1)+bP_i(1-2(a+\gamma P_2)}{(a+\gamma P_2)} & \frac{-c(1-k)P_2}{a+\gamma P_2} \\ e\left[\frac{(1-k)a}{(a+\gamma P_i)^2} - \frac{2}{3}\rho P_i^{-\frac{1}{3}}\right] Z_i & 0 \end{pmatrix} \tag{13}$$

Thus the eigenvalues in this case are obtained as roots of the quadratic $\lambda^2 - \mathrm{tr}(J_2)\lambda + \det(J_2) = 0$, where $\mathrm{tr}(J_2) = r - 2bP_2 - \frac{r-bP_2}{a+\gamma P_2}$, and $\det(J_2) = \frac{ec(1-k)P_2}{a+\gamma P_2}\left\{\frac{(1-k)}{(a+\gamma P_2)^2} - \frac{2}{3}\rho P_2^{-\frac{1}{3}}\right\}$. Now, $\mathrm{tr}(J_2) < 0$, iff $r < 2bP_2 + \frac{r-bP_2}{a+\gamma P_2}$ with $P_2 < \frac{r}{b}$ and we find that the Routh-Hurwitz criterion for stability is satisfied if $\det(J_2) > 0$, i.e. if $\frac{(1-k)}{a+\gamma P_2} > \frac{2}{3}\rho P_2^{-\frac{1}{3}}$, a condition which is equivalent to

$$P_2 \geq \frac{8}{27}\left(\frac{\rho(a+\gamma P_2)^2}{1-k}\right)^3. \tag{14}$$

Now we are in the position to state the following theorem.

Theorem 2. *The positive equilibrium E_2 of the system (2) is locally asymptotically stable if the following conditions hold:*

$$1)\ r < 2bP_2 + \frac{r-bP_2}{a+\gamma P_2} \text{ with } P_2 < \frac{r}{b},$$

$$2)\ P_2 \geq \frac{8}{27}\left(\frac{\rho(a+\gamma P_2)^2}{1-k}\right)^3.$$

4 Numerical simulation and discussion

Theorem 2 ascertains that the system (2) is locally asymptotically stable around the interior equilibrium point under certain parametric conditions. With the set of parameter values given in Table 1, we observe that the system (2) is locally asymptotically stable around the interior equilibrium point $E_2 \equiv (1.423,\ 0.412)$, see Figure 1. If we take $k = 0.65$, retaining the other parameter values fixed, we observe periodic solution depicting the recurrent bloom phenomenon, see Figure 2. Thus, our model shows the recurrent bloom phenomenon too.

Next, we shall study the role of toxin chemicals and the patches in the formation and termination of the planktonic blooms. We begin with the parameter k. To study the role of the parameter k which is the fraction of the TPP population that aggregates to form patches, we do the bifurcation diagram of both the species with k as the bifurcation parameter, see Figure 3. To obtain the bifurcation diagram we run the system (2) for 10000 time steps and examined the last 3000 time step to eliminate transient behaviour. Then we have plotted the successive maxima and minima of all the species with k as a function of the control parameter and other parameters are kept fixed at the level given in the Table 1. We vary ρ along with k such that the number of patches formed remains same, i.e., $N = 5$. We observe that for the lower value of k the solution shows periodic oscillation, see Figure 3. With an increase in the value of k, the system becomes stable around the positive equilibrium point. Finally, for higher values of k, there is a huge increase

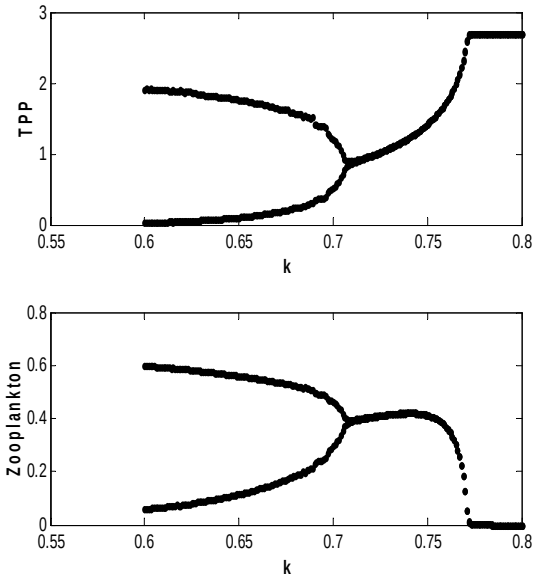

Fig. 3 The figure depicting the bifurcation diagram with k as the bifurcation parameter.

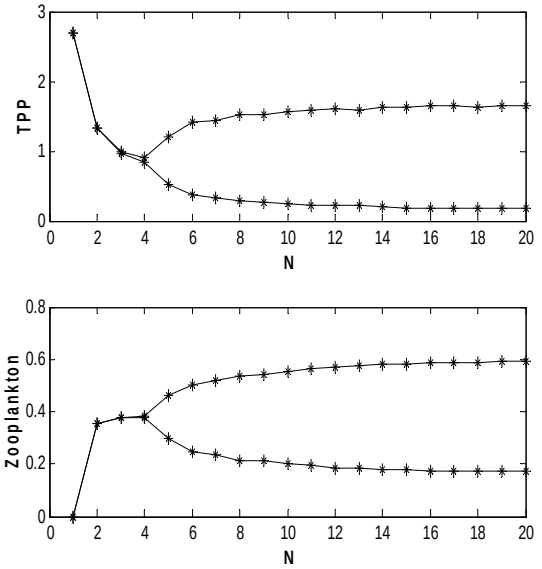

Fig. 4 The figure depicting the bifurcation diagram with N as the bifurcation parameter.

in the TPP population and the zooplankton population is washed away from the system. Thus, we may conclude that the fraction of phytoplankton that aggregate to form patches plays an important role in the recurrent bloom phenomenon and also in the coexistence of all the species. For stability of the system around the interior equilibrium point, the fraction of TPP population that aggregates to form patches must be between certain lower and upper threshold values. If the fraction is less than that of the lower threshold value, then it may cause recurrent bloom and if it is higher than the upper threshold value then it may cause the extinction of the zooplankton population.

In the above simulation we have considered a fixed number of colonies (N=5). It is interesting to see what happens to the to the dynamical nature of the system, when the number of colonies/ patches N changes. To observe the role of N, we consider $k = 0.75$ and vary ρ so that N always remains an integer, retaining other parameter values the same as in Table 1. We observe that if the TPP population forms a single patch it is very difficult for the zooplankton population to survive. We also observe that if the number of patches is higher, then it results in the reccurent bloom. So, we may again conclude that for the stability of the system around the interior equilibrium point, the number of patches N has to be between certain critical values. For example, here N has to be between $2 \leq N \leq 4$ for the stability of the system around the interior equilibrium point, see Figure 4.

Our results indicate that the fraction of TPP population that form patches and the number of patches it forms determine occurrence and termination of the bloom. Before ending our article we would like to mention that our results shows that the formation of colonies or patches specially by TPP population plays an important role in the aquatic system and so need special attention from the experimental biologists.

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