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Aggregation of toxin producing phytoplankton acts as a defense mechanism– a model based study*

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Abstract

We propose a simple model of toxin producing phytoplankton-zooplankton interactions in which the former is assumed to be able to detect the presence of zooplankton and to counteract it by forming patches and by releasing some toxic chemicals in the surrounding water. We observe that the formation of patch by the toxin producing phytoplankton 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 patches. **Finally the results were validated by comparing them with an alternative spatial model.**

Keywords: Phytoplankton-Zooplankton; patch; Phase plane analysis; Stability criteria; level of toxicity.

AMS Classification 92D25, 92D40, 37C10.

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1 Introduction

Toxic or otherwise Harmful algal blooms (HAB) are increasing in frequency worldwide [20, 53] and have negative impact on aquaculture, coastal tourism and human health [2]. Many theories are available explaining the bloom phenomenon. Some of them use ‘top-down’ mechanism [11, 15, 42, 60] 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 [10, 25, 45, 50], i.e., 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 [41]. Quite a good number of studies [6, 7, 17, 40] with the above mentioned mechanisms have considered toxin producing phytoplankton (TPP) as an important factor to explain the recurring bloom formation.

The toxin liberated by the phytoplankton may be regarded as an anti-grazing strategy [64]. Many researchers observe that the production of endo-extracellular toxins is also a common property of several strains of pelagic primary producers, serving as an efficient grazer defense [29, 12]. **The evolution of toxins as a defence against the appropriate herbivores is often unclear and remains vigorously debated [62].** Most of these toxins are always present in the phytoplankton cells, but their concentration may go up because of the following chemical cues- (i) chemicals may released from lysed cells (mechanical damage) of algal species that have not been in contact with a herbivore’s digestive system [19], (ii) herbivore-released chemicals that are produced by the herbivore and these chemicals are not related to feeding. For example, chemicals like sex pheromones or aggregation pheromones (intra-specific competition) [62], and (iii) specific chemical related directly to feeding, i.e. when cells and/or their contents of the phytoplankton come into contact with the feeding apparatus and digestive system of the grazer [55]. However, still it is not clear whether the algal cell use them uniquely or in combination. It is also possible there may exists some more possibilities

for the toxin release.

The anti-grazing strategy is not only important for the existence of the phytoplankton species but also for many zooplankton species 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 [35]. Among the various other anti-grazing strategies observed so far for phytoplankton, cell morphology [21], presence of gelatinous substances, or aggregate to form patches [28] and filamentous structures [32] are widely recognized. Since phytoplankton in the pelagic are small relative to their predatory enemies, they will not survive an encounter with a grazer without any anti-grazing strategy. Various studies have demonstrated that the formation of patches by green alga offers considerable protection against grazing by zooplankton [13]. Short-term grazing studies of zooplankton revealed that the rate of phytoplankton consumption decreases with increasing patch size, if the nutritional quality of algae is optimal [21]. 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. Phytoplanktons may also form patches as an immediate response to some chemical stimulus released by the grazer [62].

The relation between defense strategies like patch formation and toxin release may give a possible answer to the evergreen crucial ecological question of why do many toxin releasing microalgal species aggregate together to form patch leading to bloom. Toxic chemicals released through chemical signals by the phytoplankton patches 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 [64]. So the question is, in the above 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? 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 simple toxic phytoplankton–zooplankton system where the phytoplankton populations are assumed to aggregate into patches as a defense mechanism. In spite of the simple model structure and analysis, we obtained some interesting result which we later validate through an alternative diffusion-reaction equation, following the concepts**

of [33, 47].

The organization of the paper is as follows: Section 2 deals with the mathematical model. The analysis is performed in Section 3. In section 4 we proposed an alternative spatial model and finally, the article ends with a conclusion in the Section 5.

2 The basic mathematical model

The study of the defense mechanism through the formation of patches becomes more important if such patches have the ability to release toxin chemicals, like in case of dinoflagellate [54]. The phytoplankton defense mechanism through the formation of patches were studied mainly using reaction diffusion system. **In population dynamics, this type of equations were first used by Segel and Jackson [48]. Later, Levin and Segel [30] suggested this scenario of spatial pattern formation for a possible origin of planktonic patchiness. After this there were many articles that proposed reaction-diffusion model and study them under different conditions [22, 34, 63].** For example, Serizawa et al. [51] studied a two-component diffusion model that can exhibit various types of spatial patterns including patchiness. Medvinsky et al. [36] also studied spatiotemporal complexity of plankton using reaction-diffusion equation and demonstrated that the diffusive instability can lead the system to spatiotemporal chaos even though starting from simple initial conditions. There are many more articles, such as [1, 39, 61], which tried to capture the effect of phytoplankton patchiness using diffusion equation.

Though the simulation images created by the diffusion systems are closer to real patchiness patterns, solving those partial differential equations are not easy. Sometimes the analytical solutions lead to complex relations. So, here we proposed a simple mathematical model of TPP–zooplankton interactions in the presence of plankton patchiness using a system of ordinary differential equations (ODEs). In our predator-prey system, the prey as an act of defense group together and release toxin chemicals. The latter is assumed to diffuse in the surrounding water bed through the surface of the patch. **This total defense strategy may involve some cost. For example, one distinct ecological cost of algal aggregation is limitation in photosynthesis and uptake of nutrient by reducing the available surface-per-volume [62]. But the literature on the costs**

of (inducible) defences is mixed. Some studies failed to detect cost in terms of nutrient or energy investment [31], while some found high costs [57, 9] although most of them are found in terrestrial systems [62]. If there is any cost for the defense mechanism then it is beneficial for the prey to activate its defense mechanism only in the presence of the predator. If there is no cost associated with the defence mechanism then the benefit of the protection could be permanently enjoyed [59]. In the present study we assume no cost associated with the defense mechanism and hence we model it as a continuous function.

In the present study, the space dimension is introduced in the model without using diffusion parameters. To capture the effect of phytoplankton patches on the zooplankton community, we propose a functional response which is not a monotonically increasing function of the prey density, but rather it is only monotonically increasing up to a certain threshold density and then becomes monotone decreasing. We also assumed that these patches have a negative impact on the growth of zooplankton.

Mathematically, let $P(t)$ and $Z(t)$ denote the phytoplankton (TPP) and zooplankton population sizes respectively. The phytoplankton population is assumed to follow 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 and finally we include the poisoning effect. The sketch of the model is then

$$\begin{aligned} \text{rate of change of } P &= \text{growth} - \text{predation} & (1) \\ \text{rate of change of } Z &= \text{predation} - \text{natural mortality} - \text{poisoning} \end{aligned}$$

In the above equation the poisoning effect needs to be coupled with the formation of the patches. we assume that the patch size is proportional to the phytoplankton density. This assumption is quite reasonable because many habitat fragmentation experiments show that the patch size is proportional to the population density [5]. For example, Bender et al. [4] show that patch size depends on population density. Root's [46] resource concentration hypothesis also shows that there is a positive relationship between patch size and population density. Suppose a fraction k ($0 \leq k \leq 1$) of the phytoplankton population aggregates to form N patches. For the predation term the standard mass action incidence can easily be taken, over the fraction $1 - k$ of the "free" phytoplankton. We propose here a more complicated mechanism

for the release of poison. Note 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 to be roughly 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 and this will leak into the surrounding water through the surface of the patch which is proportional to $\rho P^{\frac{2}{3}}$.

We are then led to the following equations

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

where all parameters are nonnegative. r is the growth rate, b is the phytoplankton's death rate, c is the predation rate and e is the conversion rate (assuming $c \geq e$) and μ is the natural mortality rate of the zooplankton. The most important parameter is ρ which may be defined as the measure of the toxicity, which is directly proportional to the fraction of phytoplankton forming patches and inversely proportional to the number of patches formed by the phytoplankton.

2.1 Model validation

To analyze our proposed model close to real world scenario, we take parameter values from different literature sources (Table 1) and simulate our system (2) to compare it with the available literature on plankton dynamics [23]. For the above simulation we considered hypothetical values for the parameters associated with the patch, varying them to see their effect on the system.

In [23] time series data are produced from of *Peridinium gatunense* phytoplankton blooms in Lake Kinneret, Israel, from 1970 to 1999. The phase-plane analysis supports mechanistic grounds for such phytoplankton's behaviour. Such phase-plane is symbolically sufficient to represent the plankton dynamics observed in real world. Here we reproduce their results with our system and experimentally estimate the parameter values, validating our proposed system. For the simulation we used our own software written in Matlab

Table 1: The set of parameter values taken from different literature sources for our model. Here the units of P and Z are in g m^{-3} and time is measured in days.

Parameters	definition	values considered	Range/ value from literature
r	the growth rate of phytoplankton	1.5	1– 2.9 [18, 16]
b	death coefficient of phytoplankton	0.09	0.09–0.1 [58, 52]
c	predation rate	0.14	0.04– 0.17 [14]
e	conversion efficiency of zooplankton	$0.6 \times c$	$(0.5– 0.9) \times c$ [14]
μ	death rate of zooplankton	0.035	0.021– 0.051 [18, 16, 14]
k	fraction of phytoplankton aggregates	0.55	–
ρ	degree of toxicity	0.0121	–

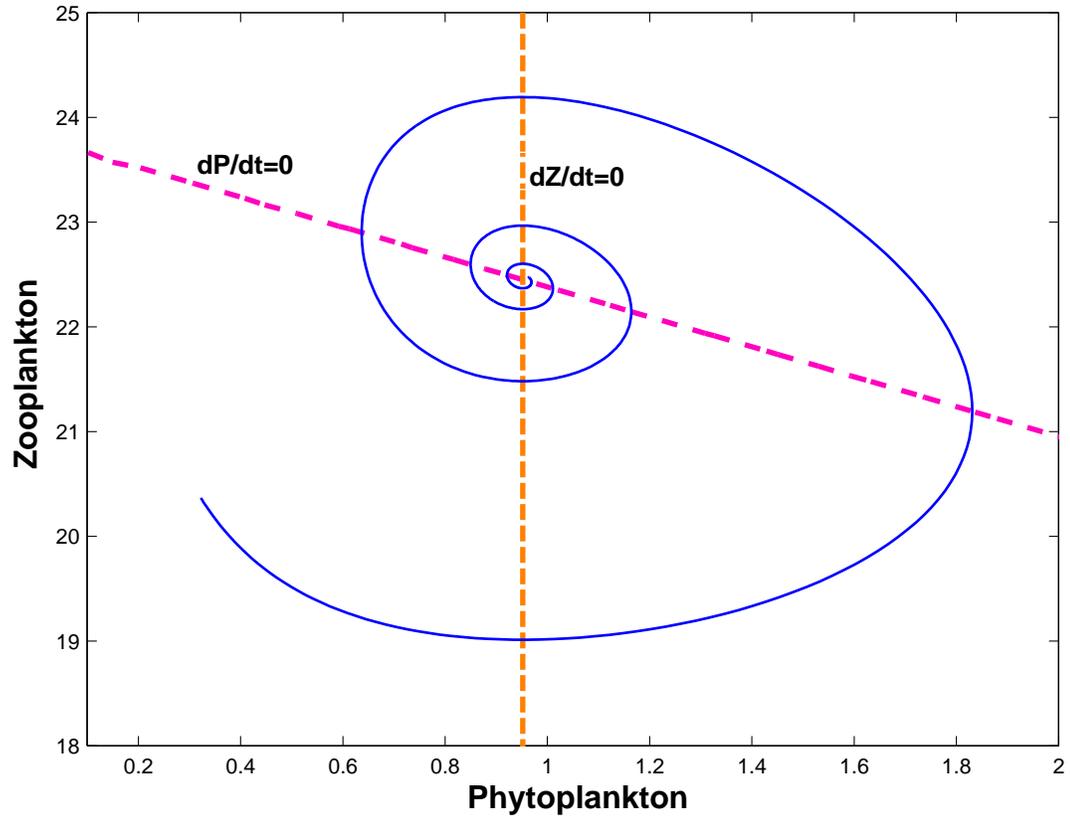


Figure 1: Phase plane diagram for the model (2). The phase plane may be used as a guide to trace out how the trajectory of the model changes with time as it is attracted towards equilibrium. A careful examination of \dot{P} and \dot{Z} in the four regions shows that the trajectory must move counterclockwise through the phase plane in its approach to equilibrium.

7.0. The phase plane is created using the free Matlab macro `pplane6.m` written by John Polking, Rice University, 1995, an interactive tool for studying planar autonomous systems of differential equations.

The time evolution of the simulation (Figure 1) shows the curves for Z against P with the $Z - P$ nullclines and the obtained flow in the phase plane is divided in different stages as done by Huppert et al. [23], each with its own ecological meaning. The first stage is the lower left region ($\dot{Z} < 0$ and $\dot{P} > 0$) where the zooplankton population declines, and there is a slow constant increase in the TPP population P . P continues to grow until they crosses the threshold level ($\dot{Z} = 0$) and the zooplankton population starts to grow (lower-right region, the second stage). Next, the trajectory crosses the P nullcline ($\dot{P} = 0$) where the TPP attains its maximum level P_{max} and moves into the upper-right region ($\dot{Z} > 0$ and $\dot{P} < 0$) of the phase plane. In this region, zooplankton dramatically increases while TPP starts to decrease. But this is the point at which TPP feels the lump of zooplankton population around itself and as a defense strategy starts releasing toxin chemicals [64]. Due to the action of these toxic chemicals, after some time the zooplankton population starts to decline. This is observed in the figure, when the trajectory passes from the upper-right into the upper-left region and crosses the z nullcline ($\dot{Z} = 0$) where the zooplankton population attains its maximum level Z_{max} .

3 Analysis of the model

3.1 Equilibrium points

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 $E_1 = (\frac{r}{b}, 0)$ and another feasible non boundary equilibrium E_2 . Its positive coordinates are found in the $P - Z$ phase plane by solving the nonlinear system $e(1 - k)P - \mu - e\rho P^{\frac{2}{3}} = 0$ and $r - bP - c(1 - k)Z = 0$. Solving these two equations we find $Z_2 = \frac{r - bP_2}{c(1 - k)}$, where P_2 is the positive real root of the following cubic equation,

$$\phi(P) \equiv e^3(1 - k)^3 P^3 - \{3e^2(1 - k)^2 \mu + e^3 \rho^3\} P^2 + 3e(1 - k)\mu^2 P - \mu^3 = 0. \quad (3)$$

From Descartes' rule of sign, we observe that there exists at least one positive real root of the above equation (2) and if that root is less than $\frac{r}{b}$, then there exists a unique positive equilibrium point E_2 .

For example, with the parameter set considered in Table 1, the equation (3) becomes,

$$\phi(P) \equiv 0.000054P^3 - 0.00015P^2 + 0.0001389P - 0.00004287 = 0. \quad (4)$$

Equation (4) has exactly one positive root, $P_2 = 0.9488$ and we obtain a unique interior equilibrium point $E_2 \equiv (0.9488, 22.4493)$.

3.2 Boundedness

Lemma 1. Assume at first that the initial condition of equation (2) satisfies $P(t_0) \geq \frac{r}{b}$ then either (i): $P(t) \geq \frac{r}{b}$ for all $t \geq 0$ and therefore as $t \rightarrow +\infty$, $(P(t), Z(t)) \rightarrow E_1 = (\frac{r}{b}, 0)$ 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 $t \geq 0$.

Proof. See Appendix A.

Lemma 2. 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. See Appendix A.

Theorem 1. The set Ω , where

$$\Omega = \left\{ (P, Z) \in R_{0,+}^2 : P \leq \frac{r}{b}, Z \leq M \right\}. \quad (5)$$

is a global attractor in $R_{0,+}^2$ and, of course, it is positively invariant.

Proof. See Appendix A.

3.3 Local stability analysis

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

$$J_i \equiv \begin{pmatrix} r - 2bP_i - c(1-k)Z_i & -c(1-k)P_i \\ e \left[(1-k) - \frac{2}{3}\rho P_i^{-\frac{1}{3}} \right] Z_i & e(1-k)P - \mu - e\rho P_i^{\frac{2}{3}} \end{pmatrix} \quad (6)$$

At the origin, the eigenvalues are $r, -\mu$ showing its instability. At E_1 , we have the eigenvalues $-r, e \left[(1-k)\frac{r}{b} - \frac{\mu}{e} - e\rho \left(\frac{r}{b}\right)^{\frac{2}{3}} \right]$. Thus, E_1 is conditionally stable if and only if $(1-k)\frac{r}{b} < \frac{\mu}{e} + e\rho \left(\frac{r}{b}\right)^{\frac{2}{3}}$. Finally, at the interior equilibrium E_2 the eigenvalues are obtained as roots of the quadratic

$\lambda^2 - \text{tr}(J_2)\lambda + \det(J_2) = 0$. Since $\text{tr}(J_2) = -bP_2 < 0$ and $\det(J_2) = ceP_2Z_2 \left[(1-k) - \frac{2}{3}\rho P_2^{-\frac{1}{3}} \right]$, we find that the Routh Hurwitz criterion for stability is satisfied if $\det(J_2) > 0$, i.e. if $(1-k) > \frac{2}{3}\rho P_2^{-\frac{1}{3}}$, which is equivalent to

$$27\mu(1-k)^2 > 8e\rho^3 \quad (7)$$

Before proceeding further let us recall for the benefit of the reader Dulac's criterion, [27].

Dulac criterion. Let us consider a system

$$\dot{x} = g(x)$$

where $g = (X, Y)^t$ and $x = (x, y)^t$. Further, $g \in C^1(E)$ where E is a simply connected region of the plane. If there exists a function $p(x, y) \in C^1(E)$ such that the divergence of the vector field $p(x, y)g$, i.e.,

$$\nabla \cdot p(x, y)g = \frac{\partial (p(x, y)X)}{\partial x} + \frac{\partial (p(x, y)Y)}{\partial y},$$

is always of the same sign but not identically zero then there are no periodic solution in the region E of the planar system.

Theorem 2. The coexistence equilibrium E_2 of the system (2) is globally asymptotically stable if the following conditions hold:

$$(1-k)\frac{r}{b} > \frac{\mu}{e} + e\rho \left(\frac{r}{b}\right)^{2/3}, \quad (8)$$

and

$$27\mu(1-k)^2 > 8e\rho^3. \quad (9)$$

Proof. The trajectories of (2) are bounded and the equilibrium point E_0 is a saddle, the equilibrium point E_1 is a repeller if $(1-k)\frac{r}{b} > \frac{\mu}{e} + e\rho \left(\frac{r}{b}\right)^{2/3}$, and the interior equilibrium point E_2 is locally asymptotically stable if $27\mu(1-k)^2 > 8e\rho^3$.

Now, we will test for the existence or non-existence of periodic solution around the positive equilibrium by using the Dulac criterion. Let $h(P, Z) = \frac{1}{PZ}$ and

$$\frac{\partial(hf)}{\partial P} + \frac{\partial(hg)}{\partial Z} = -\frac{bP}{Z} < 0.$$

Hence, there is no non-trivial positive periodic solution around the interior equilibrium point, proving the theorem.

Remark 1. One can see from relation (9) that all the species coexist if the level of toxicity (ρ) falls below a certain threshold value depending on the fraction of phytoplankton that aggregate to form patches.

3.4 Numerical analysis

Our analytical result (see Theorem 2) ascertains that the system (2) is globally asymptotically stable around the positive interior equilibrium point, and cannot explain the recurrent bloom phenomenon. We have seen from the experimentally obtained parameters that our system replicates plankton dynamics starting from bloom formation to termination. To understand the role of defense mechanism like toxin chemicals and the patches in governing the plankton dynamics, we plotted the ratio between the zooplankton population at the interior equilibrium point E_2 with the phytoplankton population at the same point, (*i.e.*, $\frac{Z_2}{P_2}$) against k , while all the other parameter values were kept fixed as in Table 1 except ρ . We vary both ρ and k in such a way that the number of patches N remains the same, here $N = 3$. The system (2) is numerically integrated using the built-in MATLAB function `ode45` and after integration we collected the final steady state values for different combinations of ρ and k and plotted those values in the $\rho - k$ space.

We observe the following values: for $k = 0.01$ the ratio is $\frac{Z_2}{P_2} = 24.36$ and for $k = 0.79$ it is $\frac{Z_2}{P_2} = 1$, Figure 2. Finally, when k crosses some critical value k_c , here $k_c = 0.82$, the ratio $\frac{Z_2}{P_2}$ tends to zero, which means there is a huge increase in the phytoplankton population with respect to the zooplankton population. Thus if the fraction of TPP population aggregates to form patches is small, it is easy for the zooplankton population to survive. But if the fraction of the phytoplankton population aggregating to form patches is larger, then there is a huge increase in the size of the phytoplankton population with respect to the zooplankton population. Thus, we may conclude that the formation of patches acts as a defense mechanism for the phytoplankton population, but the fraction of phytoplankton aggregating to form patches must not exceed a certain critical point.

In the above simulation we have considered a fixed number of patches ($N=3$). It is interesting to see what happens to the value of the equilibrium

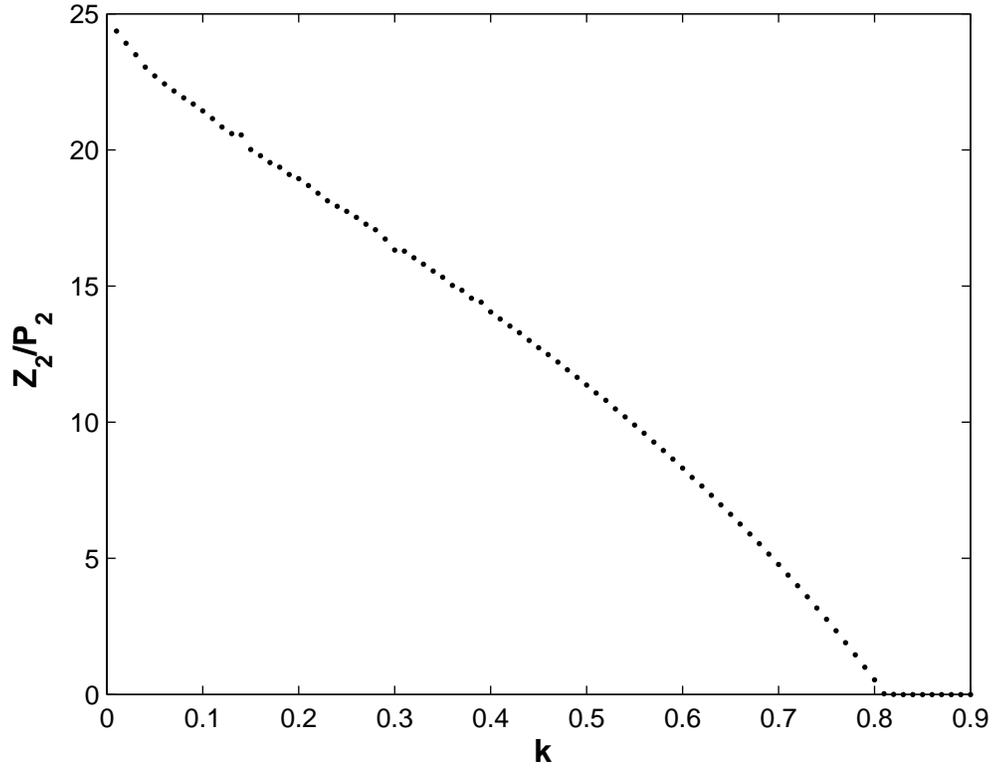


Figure 2: We vary the ratio between Z_2 (the value of Z at the equilibrium point E_2) and P_2 (the value of P at the equilibrium point E_2), (*i.e.*, $\frac{Z_2}{P_2}$) with k .

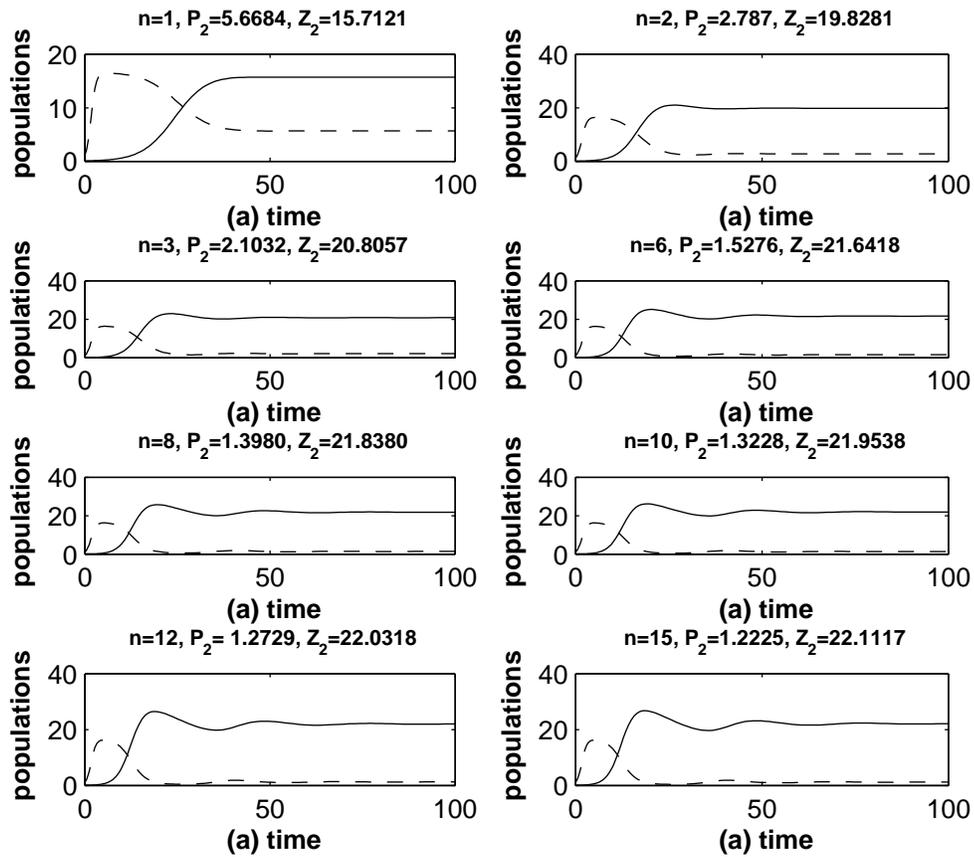


Figure 3: The figure depicts the dynamical behaviour of both populations for different values of N . ‘—’ denotes Zooplankton population and ‘- - -’ denotes TPP population.

point E_2 , when the number of patches N changes. To observe the role of N , we vary N and also ρ so that N always remains an integer, retaining the same other parameter values as in Table 1. For the numerical integration, we used the matlab built-in function `ode45` and plotted the time series for different values of N . We observe that Z_2/P_2 attains the smallest value for $N = 1$, see Figure 3. Thus when the TPP forms only one single patch, it is difficult for the zooplankton species to survive. But with an increasing number of patches there is an increase in the size of the zooplankton population. Note that clearly the patch size reduces when their number increases, because we assumed a phytoplankton population of fixed size. Thus if the number of patches is larger, it helps the zooplankton population to survive. Finally, when the value of N is increased beyond a certain threshold value, here $N = 8$, we observe only small changes in the values of E_2 , i.e the values of P_2 and Z_2 almost remain the same.

4 A space-dependent model

From our previous model we obtained a relation between the percentage and size of phytoplankton aggregation and survival of the plankton population. Now to see the robustness of our results from the simple model, we propose and study a spatial version of our system (2) in which the patch formation is absent, in order to compare the previous results. This constitutes an important validation of our results, in view of the fact that we formulate a simple model to study a very complicated dynamics. If our result does not change much in the diffusion model then we can claim that our proposed lumped parameters model can adequately capture the complex dynamics associated with the phytoplankton aggregation mechanism.

We explicitly incorporate diffusion in our model, without phytoplankton aggregation. We introduce diffusion in our model following [33, 47]. Mathematically, in our new system we replace the term $\rho P^{\frac{2}{3}}$ by θP , where θ represent the degree of toxicity and we also of course drop the term k associated with the aggregation. Moreover, in the original model we assumed that phytoplankton aggregates, but no aggregation was assumed for zooplankton. Here we will assume spatial changes in both the species. Thus a spatial

analogue of the model (2) is presented here below.

$$\begin{aligned}\frac{\partial P}{\partial t} &= rP - bP^2 - cZP + D\frac{\partial^2 P}{\partial x^2} \\ \frac{\partial Z}{\partial t} &= (e - \theta)ZP - \mu Z + D\frac{\partial^2 Z}{\partial x^2}\end{aligned}\tag{10}$$

where D_1 and D_2 represent the diffusion coefficients of P and Z respectively. It is written in one dimension, but the extension to two dimensions is straightforward.

Let Υ represent a suitable spatial domain in one or two dimension as the case considered suggests. For the system (10) in the one-dimensional domain Υ we take the following initial conditions

$$P(0, x) > 0, \quad Z(0, x) > 0,$$

and the zero-flux boundary condition

$$\frac{\partial P}{\partial x} \Big|_{\partial\Upsilon} = \frac{\partial Z}{\partial x} \Big|_{\partial\Upsilon} = 0.$$

This means that no external input occurs across the boundary of these populations.

4.1 Model analysis

There are three equilibrium points for the model (10) in the absence of diffusion, out of which two are same as obtained from equation (2), i.e., the origin $E_0=(0,0)$, the axial equilibrium point $E_1 = (\frac{r}{b}, 0)$. The third equilibrium point is the coexistence point denoted by $E^* = \left(\frac{\mu}{e-\theta}, \frac{r(e-\theta)-b\mu}{c(e-\theta)}\right)$.

Denoting the general uniform steady state (USS) of the model (10) by $(\widehat{P}, \widehat{Z})$, we investigate perturbations of the following form,

$$\begin{pmatrix} P(t, x) \\ Z(t, x) \end{pmatrix} = \begin{pmatrix} \widehat{P} \\ \widehat{Z} \end{pmatrix} + \begin{pmatrix} P_d(t) \\ Z_d(t) \end{pmatrix} \cos(lx) \exp(\lambda t),$$

where $l > 0$ is the wave number of the spatial perturbation and $\lambda > 0$ is the time evolution rate.

By substituting these expressions in the equation (10) and differentiating (using the fact that $(\widehat{P}, \widehat{Z})$ represents a steady state), we obtain the following differential equations

$$\begin{aligned}\frac{dP_d}{dt} &= -\lambda P_d - \left(\widehat{P} + P_d \cos(lx) \exp(\lambda t) \right) (bP_d + cZ_d) - D_1 l^2 P_d \\ \frac{dZ_d}{dt} &= -\lambda Z_d + (e - \theta) \left(\widehat{Z} + Z_d \cos(lx) \exp(\lambda t) \right) P_d - D_2 l^2 Z_d\end{aligned}$$

After linearization we seek the system's equilibria. The problem is then reduced to an eigenvalue problem in the parameter λ for the following matrix at the generic USS $(\widehat{P}, \widehat{Z})$,

$$J_D \equiv \begin{pmatrix} r - 2b\widehat{P} - c\widehat{Z} - l^2 D_1 & -c\widehat{P} \\ (e - \theta)\widehat{Z} & (e - \theta)\widehat{P} - \mu - l^2 D_2 \end{pmatrix} \quad (11)$$

Its eigenvalues will thus provide the needed time dependency λ .

The eigenvalues for the plankton-free E_0 , are $r - l^2 D_1$ and $-\mu - l^2 D_2$. Hence, our diffusion model (10) showed that in presence of diffusion E_0 is uniformly stable if and only if $r < l^2 D_1$, i.e., when the prey diffusion coefficients exceed a threshold, the system can collapse, wiping out both the populations. At E_1 , the eigenvalues are $-r - l^2 D_1$ and $(e - \theta)\frac{r}{b} - \mu - l^2 D_2$. So, the system is stable around E_1 if and only if $(e - \theta)\frac{r}{b} < \mu + l^2 D_2$. Thus the condition for the stability of $(\frac{r}{b}, 0)$ in the presence of diffusion (obtained from the diffusion model (10)) is similar to what we observed from the original model. Thus the diffusion model does not change the result related to the preservation of phytoplankton and the washing out the zooplankton from the system.

For the interior equilibrium point E^* the eigenvalues are obtained as roots of the quadratic

$$x^2 - \text{tr}(J_D^*)x + \det(J_D^*) = 0, \quad (12)$$

where $\text{tr}(J_D^*) = -(bP^* + (D_1 + D_2)l^2) < 0$, and $\det(J_D^*) = l^2 D_2 (bP^* + l^2 D_1) + c(e - \theta)P^*Z^* > 0$ if $\theta < e + \frac{l^2 D_2 (bP^* + l^2 D_1)}{cP^*Z^*}$. Thus from the Routh-Hurwitz criterion the system shows stability in the presence of diffusion if $\theta < e + \frac{l^2 D_2 (bP^* + l^2 D_1)}{cP^*Z^*}$. Hence the system (10) does not show diffusive instability around E^* , see Fig. 4 and the conditions

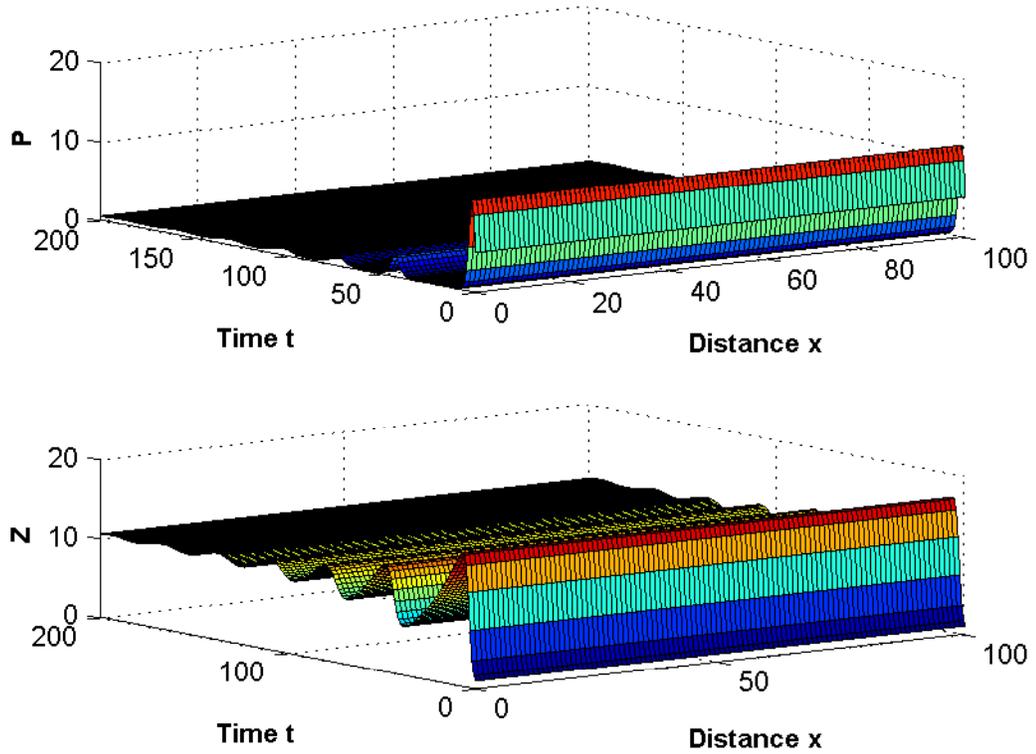


Figure 4: Biomass distribution of phytoplankton and zooplankton over time and space for the model (10) for $D_1 = 10$ and $D_2 = 2$ with other parameter values from Table 1. Here the value of θ is same as ρ . For this parameter set the non-diffusive system shows stable coexistence. Here $(P^*, Z^*) = (0.45, 10.45)$.

are almost the same as obtained from equation (2). In both the models we observed that for the stability of coexistence equilibrium the degree of toxicity has to be less than some threshold values.

To observe the turbulence effect in our system we consider the same diffusion rate for both the species as done by [33]. Thus if we consider the mixing due to turbulence, the condition for the stable coexistence of the species becomes $\theta < e + \frac{l^2 D(bP^* + l^2 D)}{cP^*Z^*}$, where D is the diffusion coefficient. Here we have not considered the effect of turbulence explicitly because our model describes the general path size, while turbulence mainly controls the small scale plankton patches, which are of size less than one hundred meters [33, 43, 44].

We can now summarize the whole discussion into the following remark.

Remark 2. The stability properties of all the steady states of the system (10), except the trivial equilibrium point, remain almost the same as observed for the original model (2). The trivial steady state which is unstable in the original model is instead conditionally stable in the diffusion model.

5 Conclusion

Our proposed model assumes that the TPP population aggregates to defend itself from the zooplankton predation. This is well accordance with results showing that the phytoplankton population forms patches to protect itself from grazers [54]. The coupled defense mechanism through patching and poison release results in the coexistence of the interacting species. Huisman and coworkers [24, 26, 56] have used the species coexistence to explain biodiversity and the plankton paradox. Our observations also indicate that there is a threshold in the number of patches enhancing the coupling strength of interacting species. Patch formation is important for the existence of the phytoplankton, but for zooplankton's survival the patch size should not be large. So, to maintain biodiversity, phytoplankton needs to gather in small patches.

As discussed earlier, the common practice of mathematically capturing the effect of phytoplankton patchiness is through diffusion equations [1, 39, 61]. Researchers have used spatial models based on diffusion equa-

tions to find the critical-length scales of phytoplankton patchiness in terms of phytoplankton growth and herbivore grazing [65]. **Our simple model also provides a good explanation for the relation between the percentage and the size of the phytoplankton aggregation and the survival of the plankton population.** Moreover, we observed that incorporation of reaction-diffusion in the proposed model does not change the basic nature of the system's behaviour except that there is a chance of total extinction if the prey diffusion is sufficiently high, larger than a certain threshold directly proportional to the phytoplankton reproduction rate.

Although we have proved the robustness of our results under diffusion mechanisms by using a simple reaction-diffusion system, the proposed model can be improved in a number of ways. For example, we could consider a more complex functional response to capture parameters like the saturation effect. We already mentioned that the activation of defense strategy is not clear and here we assumed a continuous function, but one could also take a step function to capture the discontinuity and compare the result with the present study. In the present study we have ignored the explicit effect of turbulence. Turbulence has its own importance in these studies, since it quickly disrupts patches in nature, certainly preventing them from behaving like perfect spheres. More importantly, turbulence actively prevents group defense from being possible. It is observed that under active turbulence not only the size and the shape of the patches change but also the spacing between patches get modified [38]. Finally, one can also consider separating the phytoplankton present outside the patch from the one inside the patch. Recent studies showed that the growth of a phytoplankton, grazing pressure and even nutrient availability is different for the inner and outer layers in a patch [37]. In the present model there is no term for zooplankton feeding inside and outside of patches. To obtain a more realistic model, separate equations for phytoplankton inside and outside of patches and for zooplankton inside and outside of patches could be formulated.

Although the proposed model can be extended and improved by the above modifications, **we believe that increasing the complexity of the model will not change the basic results.** We already observed this fact using the simple reaction-diffusion scheme in comparison to the lumped parameters model. We also believe that our study will open new

door for the plankton ecologists to relate plankton survival with patch size and the fraction of population that aggregates to form patches.

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Appendix A

Let us first recall (without proof) the Barbălat [3] lemma:

Let g be a real valued differentiable function defined on the half line $[a, \infty)$, $a \in (-\infty, \infty)$. If (i) $\lim_{t \rightarrow \infty} g(t) = \alpha$; $|\alpha| < +\infty$, (ii) $g'(t)$ is uniformly continuous for $t > a$, then $\lim_{t \rightarrow +\infty} g'(t) = 0$.

Proof of Lemma 1. With the help of a Barbălat's lemma, one can easily prove the Lemma and hence the proof is omitted.

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

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

Taking $\mu \geq \eta > 0$ we get,

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

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 1 now follows from the ultimate boundedness of P .

Proof of Theorem 1. Due to lemmas 1 and 2 for all initial conditions in $R_{+,0}^2$ 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 $(P(t), Z(t)) \in \text{int } \Omega$ for all $t > T$, or the corresponding solution is such that $(P(t), Z(t)) \rightarrow E_1(\frac{r}{b}, 0)$ as $t \rightarrow +\infty$. But, E_1 is at the boundary of Ω , i.e., $E_1 \in \partial\Omega$, where $\partial\Omega$ represents the boundary of Ω . Hence the global attraction of Ω in $R_{0,+}^2$ has been proved.

Assume now that $(P(t_0), Z(t_0)) \in \text{int } \Omega$. Then Lemma 1 implies that $P(t) < \frac{r}{b}$ for all $t > 0$ and also by lemma 2 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 $\text{int}\Omega$ or coincide with E_1 .

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