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Hopf bifurcation in a periodic toxin producing phytoplankton model.

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Abstract

Harmful algal blooms (HABs) caused by toxin-producing phytoplankton (TPP) have become increasingly common worldwide. Understanding the complex interactions between TPP and other organisms in the ecosystem is crucial. This study focuses on the Hopf bifurcation analysis of plankton interactions between TPP and zooplankton, with uptake function and a periodic toxin production. The maximum toxin liberation rate is considered as a bifurcation parameter. The aim is to determine how the toxin liberation rate affects the system. One of the proposed models assumes constant toxin production by TPP, resulting in an autonomous system of ordinary differential equations. To incorporate natural day and night, tidal, or seasonal cycles, the model is extended to a periodic system. The study examines the existence of steady states and trivial periodic solutions and analyses the stability of both models. Moreover, using the concept of uniform persistence, we derive sufficient conditions for the coexistence of the periodic system based on the model parameters. Due to instability of equilibria, we observe Hopf bifurcations in the constant toxin-producing model, providing insights into the system's dynamic behaviour. Numerical simulations are performed to validate the analytical findings of the proposed models and their implications.

Keywords

Harmful algal blooms, Hopf bifurcation, Periodic systems, Toxin-Producing Phytoplankton, Zooplankton.

Introduction

Aquatic ecosystems heavily rely on plankton, specifically phytoplankton and zooplankton, as fundamental components of the food web. Phytoplankton, through photosynthesis, play a vital role in producing oxygen and regulating carbon dioxide levels. Zooplankton, in turn, act as grazers and energy transfer agents, facilitating the flow of energy through the ecosystem. However, the growing prevalence of Harmful Algal Blooms (HABs) caused by TPP poses a significant threat to both aquatic life and human health (Hubbart, 2012), (Phlips, 2004), (Roelke, 2011), (Sopanen, 2011). Mathematical models consist of either autonomous systems of ordinary differential equations or delay differential equations have been developed to explore the mechanisms and impacts of toxin-producing phytoplankton populations (Chattopadhyay J. S., 2002), (Khare, 2010), (Mukhopadhyay, 2006), (Saha, 2009). However, it is plausible that toxin release by phytoplankton is not constant (DeAngelis, 1992), (McGillicuddy Jr, 2003), (Phlips, 2004). The main objective of this study is to discuss the bifurcation analysis of the proposed periodic system that models the interactions between TPP and zooplankton by considering natural cycles. It also aims to analyse the effect of toxin liberation on plankton survival, persistence, and its accumulation in the food chain.

Methodology/materials and methods

Spatial homogenous models

$$\frac{dP}{dt} = r \left(P \left(1 - \frac{P}{K} \right) - af(P)Z \right) = rP \left(1 - \frac{P}{K} - \frac{aZ}{m+P} \right) \quad (1)$$

$$\frac{dZ}{dt} = bZ(f(P) - d - c\gamma(t)g(P)) = bZ \left(\frac{(1 - c\gamma(t))P}{m+P} - d \right)$$

where $P(t)$, $Z(t)$ are TPP, zooplankton populations at time t respectively. K is carrying capacity, r is intrinsic growth rate of phytoplankton, b is intrinsic growth rate of zooplankton, a is maximum uptake rate of zooplankton, $f(P)$ is zooplankton's uptake function, $g(P)$ is phytoplankton's toxin-producing function, d is natural mortality rate of zooplankton ($0 < d < 1$), c is rate of toxic substances produced by per unit biomass of phytoplankton. m is the half saturation constant for a Holling type II functional response. $\gamma(t) = \gamma(1 + Aq(t))$ is the periodic function. Here $q(t)$ is τ -periodic, γ is toxin liberation rate, and A is magnitude of periodicity.

Proposition 1: Solutions of (1) remain non negative and are bounded for $t > 0$.

The Model with constant toxin production

$$\frac{dP}{dt} = r \left(P \left(1 - \frac{P}{K} \right) - af(P)Z \right) \quad (2)$$

$$\frac{dZ}{dt} = bZ(f(P) - d - c\gamma g(P))$$

$A = 0$ yields a constant toxin production model (2), where the toxin production rate (γ) remains constant.

The Model with periodic toxin production

$$\frac{dP}{dt} = r \left(P \left(1 - \frac{P}{K} \right) - af(P)Z \right) \quad (3)$$

$$\frac{dZ}{dt} = bZ(f(P) - d - c\gamma(1 + A \sin(Tt))g(P))$$

The positive magnitude of periodicity results in a periodic system, where the toxin production rate varies periodically.

Stability Analysis of the equilibria of constant toxin production

Proposition 2: System (2) has two boundary steady states $E_0 = (0, 0)$ and $E_1 = (k, 0)$, where E_0 is a saddle point, and E_1 is locally asymptotically stable if $f(K) - d - c\gamma g(K) < 0$ and is unstable if $f(K) - d - c\gamma g(K) > 0$. Moreover, (2) has at least one interior steady state if $f(K) - d - c\gamma g(K) > 0$.

When both f and g are of Holling type II with different half saturation constants, we analyze the stability of the unique interior steady state $E^* = (P^*, Z^*)$ by ensuring that the assumptions $f(K) - d - cg(K)\gamma > 0$ and $1 - d - c\gamma > 0$ are satisfied. The functions $f(K)$ and $g(K)$ represent values evaluated at the carrying capacity K . Then bifurcation occurs when the *trace* of the Jacobian matrix at $E^* = 0$.

Hopf Bifurcation

By considering the maximal toxin liberation rate (γ) as the bifurcation parameter, we can demonstrate a Hopf bifurcation. For that, we employ the following theorem.

Theorem 1: Consider constant toxin production system (2), (i.e., $A = 0$) and assume $f(K) - d - c\gamma g(K) > 0$ and $1 - d - c\gamma > 0$ hold. Then system constant toxin production has a unique interior steady state $E^* = (P^*, Z^*)$. If $g(P) = \frac{P}{k+P}$ and $f(P) = \frac{P}{m+P}$, then there exists a unique $\gamma_0 > 0$ such that a Hopf bifurcation occurs at $\gamma = \gamma_0$ provided $K > m$.

Proof: Hopf bifurcation occurs at $\gamma = \gamma_0$, we verify that the eigenvalues cross the imaginary axis transversally, i.e.,

$$\text{tr}' \left(J(E^*(\gamma_0)) \right) = \left(\frac{dP^*}{d\gamma} \Big|_{\gamma=\gamma_0} \right) \left(\frac{-2r}{K} - arf''(P^*(\gamma_0))Z^*(\gamma_0) \right) \neq 0.$$

Stability analysis of periodic toxin production

Proposition 3: Periodic system (3) always has two trivial τ -periodic solutions $(0, 0)$ which is unstable and $(K, 0)$ which is asymptotically stable if $f(K) - d - c\hat{\gamma}g(K) < 0$ and unstable otherwise.

Theorem 2: If $f(K) - d - c\hat{\gamma}g(K) > 0$, then the periodic system (3) is uniformly persistent where $\hat{\gamma} = 1/\tau \int_0^\tau \gamma(t)dt$.

Proof: Notice that the boundary is invariant under (3) and the flow F is dissipative. Let $M_1 = \{E_0\}$ and $M_2 = \{E_1\}$. Then $M = \{M_1, M_2\}$ is composed of disjoint, compact, and isolated invariant sets for ∂F . We prove that the set

$$N_1 = \{(P, Z) \in R_+^2 : d((P, Z), M_1) < \epsilon\}$$

is an isolated neighbourhood, where d is the Euclidean metric. Similarly, by the assumption $f(K) - d - c\hat{\gamma}g(K) > 0$, we can choose $\delta > 0$ sufficiently small. We prove that the set N_2 is an isolated neighborhood of M_2 .

$$N_2 = \{(P, Z) \in R_+^2 : d((P, Z), M_2) < \delta\}$$

Therefore M is an isolated covering for ∂F . Moreover, we prove that M is an acyclic covering for ∂F .

Results and Discussion

The model (3) exhibits positive periodic solutions and periodic outbreaks of planktonic blooms. The graphical representations presented in Figure 1 and Figure 2 are derived from the data presented in Table 1 and Table 2, respectively.

Table 1. Parameter values for model (2).

Parameter	Value	Unit	Source
r	0.7	day^{-1}	Assumed
b	0.6	day^{-1}	Assumed
K	100	μgNI^{-1}	Assumed
m	$(5.7)^2$	$(\mu gNI^{-1})^2$	(Jang, 2014)
a	0.7	day^{-1}	(Jang, 2014)
c	0.8	day^{-1}	Assumed
d	0.4	day^{-1}	Assumed
k	20	day^{-1}	(Jang, 2014)

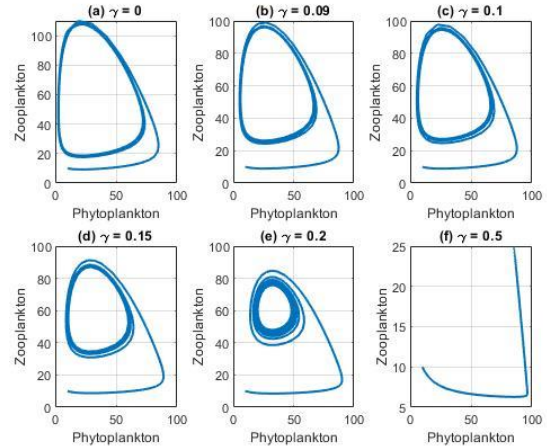


Figure 1. Effect of Toxin Liberation Rate γ of (2) with $A = 0$.

Table 2. Parameter values for model (3).

Parameter	Value	Unit	Source
r	0.7	day^{-1}	Assumed
b	0.6	day^{-1}	Assumed
K	80	μgNI^{-1}	Assumed
m	$(5.7)^2$	$(\mu gNI^{-1})^2$	(Jang, 2014)
a	0.7	day^{-1}	(Jang, 2014)
c	0.5	day^{-1}	(Jang, 2014)
d	0.4	day^{-1}	Assumed

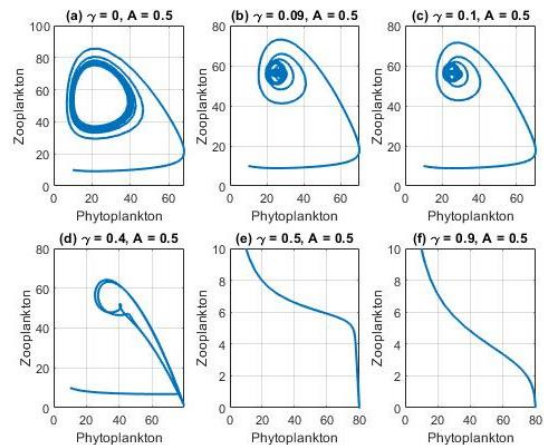


Figure 2. Effect of Toxin Liberation Rate γ of (3) with Periodicity $A = 0.5$.

Figure 2 (a) - (d) exhibit unstability, while Figure 2 (e) - (f) display stability.

Conclusion

In this work, we propose a periodic model to investigate the effects of toxin liberation by phytoplankton on planktonic interactions. We incorporate the concept of the periodic

function $\gamma(t)$ and outline the conditions it follows. Since solutions remain nonnegative and are bounded, the proposed model is biologically sound. Additionally, we introduce two variations: constant toxin production and periodic toxin production. We obtain the stability conditions for the model with constant toxin production. Using the concept of uniform persistence and based on the boundary dynamics of the model, we derive sufficient conditions for coexistence under certain parameter conditions. Using toxin liberation rate as a bifurcation parameter, we prove that system can undergo a Hopf bifurcation when interior steady state loses its stability. Specifically, this occurs when the carrying capacity (K) of the phytoplankton is small relative to the half-saturation constant (m) of the zooplankton grazing rate. This finding aligns with biological reasoning, as phytoplankton cannot sustain high population densities when the carrying capacity (K) is small. Consequently, the toxic effects on zooplankton are minimal, allowing both populations to coexist in a unique interior steady state. However, when the carrying capacity (K) is large, the autonomous system exhibits positive periodic solutions. This leads to periodic outbreaks of planktonic blooms, indicating fluctuating population dynamics. Finally, we present the numerical simulations which validate our analytical findings. We demonstrate that altering the toxin liberation rate γ affects the stability of both the constant toxin production system and the periodic toxin production system.

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