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STUDY ON THE IMPACT OF ALLELOCHEMICALS ON ALGAL-FISH DYNAMICS WITH MONOD- HALDANE TYPE FUNCTIONAL RESPONSE IN A CORAL REEF ECOSYSTEM

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Abstract Coral reefs face a myriad of threats from both natural phenomena and human activities worldwide. Among these threats, the proliferation of algae within coral reef ecosystems stands out as a prominent and damaging factor contributing to the decline of healthy coral populations. Certain species of algae produce allelochemicals that pose harm to corals, especially when they are already stressed. The presence of these toxic allelochemicals released by algae not only damages coral reefs but also reduces the populations of reef fishes, which are vital for regulating algae levels. In reef habitats, algae-feeding fishes offer a crucial role in preserving ecological equilibrium by managing algae proliferation. However, the presence of toxic algae can impede the growth of herbivorous fish populations and disturb the delicate ecological balance. To better understand the dynamics of this interaction, we have developed a mathematical model that simulates the relationships between corals, toxic algae, and herbivorous fish biomass in reef ecosystem. The stability of coral reef ecosystems hinges on factors such as the toxicity of macroalgae within the coral population. The model system displays instability behavior under conditions of high inhibitory effects from algae, consequently adversely impacting both coral and fish populations. Additionally, bifurcation scenarios have been explored via numerical simulations, concerning the inhibitory effect of algae on coral and fish populations. The numerical analysis further establishes that under the release of allelochemicals by the algal population, both the coral reefs as well the residing fish populations face survival challenges.

1 Introduction

Reef ecosystems serve as a major source of world's ocean biodiversity, providing habitat for approximately one-third of all oceanic species [1]. Reefs offer vital habitats and refuges for numerous oceanic species, playing a crucial role in sustaining marine life. Additionally, coral reefs provide essential services such as coastal protection against wave action and storms [2], as well as serving as a source of nutrients for marine food chains. Furthermore, coral reefs contribute significantly to economies through fisheries and tourism, providing livelihoods and income for many people. Despite being highly vulnerable, the reef system belongs to one of the richest and most diversified aquatic ecosystems. These ecosystems are subjected to various natural and human-induced stresses, and their responses are not always gradual. Instead, they can undergo rapid transitions to new states once a critical threshold is surpassed—a phenomenon known as phase shift [3]. Reef ecosystem resilience refers to the ecosystem's ability to withstand and recuperate after repeated stressors before shifting into a different steady phase. Macroalgae, regardless of being crucial to coral reef ecology, have been increasingly linked to a decline in reef resilience [4, 5]. Research by Done [6] and Bellwood et al.[7], has demonstrated that as coral reefs lose their regaining ability, often due to decreased adaptive capacity, they can shift from coral-dominated states to alternative states predominated by algae or different marine creatures [8]. Such phase shifts primarily occur because algae outcompete corals through shading and the release of allelopathic chemicals [9]. Additionally, fast-growing macroalgae further exacerbate

this competition by occupying space that would otherwise be available for coral settlement [10]. This underscores the dynamic nature of coral reef ecosystems and the possibility of recovery despite significant challenges. Both natural and human-induced stresses can gradually erode corals, often unnoticed, until a perturbation occurs, leading to a catastrophic devastating change into a new condition.

Studies show that fast herbivore extinction and increased macroalgal poisoning are causing reductions in corals that form reefs in a number of different places [11, 12]. When chemicals released by macroalgae affect their target species, it is referred to as algal allelopathy [13]. These effects can encompass various aspects such as growth, health, origin, or population biology of both the donor and recipient organisms [13, 14]. The complex processes such as the synthesis of active toxic chemicals and their effective transfer to nearby species are responsible for the allelopathic behaviour of algae [15]. Macroalgae, predominantly benthic organisms firmly anchored to corals, engage in competition for nutrients, sunlight, and stratum with other marine creatures. This competitive interaction underscores the complex dynamics within coral reef ecosystems.

The levels of nutrients in an ecosystem appear to have a significant impact on release of allelopathic compounds produced by macroalgae. Harmful algal blooms have been occurring more frequently in recent times. [16, 17].The macroalgae are opportunists and frequently flourish on reefs that are exposed to disturbance, such as those where elevated nutrient concentrations and terrestrial runoff occur. Nutrient enrichment indirectly reduces coral cover because high nutrient concentrations promote the growth of macroalgae and phytoplankton, which compete with coral by producing toxic substances for space and light, respectively [18, 19]. The end outcome of this disturbance is almost always a decline in reef covering and a rise in fleshy algae. Algal toxins exert a direct impact on the ecosystem and are implicated in suspected instances of mass fish kills feeding on them[20]. Furthermore, there are reports indicating that toxins originating from macroalgae undergo biomagnification up the food chain, leading to significant mortality among herbivorous fishes [21]. Algal-feeding fishes have a prominent part in shaping the distribution and abundance of reef macroalgae, and their intensive grazing may be necessary for the growth of reef-building corals. Some coral reef ecosystems exhibit at least two alternative states- one dominated by algae and the other by corals. The concept of the coral-algal-herbivore fish triangle serves as a useful generalization within the intricate network of biotic interactions and abiotic conditions present in coral reef ecosystems.

Mathematical models based on different assumptions have been used to effectively explain the changing patterns of relationships among organisms. However, the choice of functional response has been particularly intriguing in the study of prey-predator interactions. Trophic interactions have been shown using various functional responses and mathematical expressions that go along with them, emphasising their important significance in modelling interacting organisms. Traditionally, monotonic functional responses like Holling types I, II, and III have been widely utilized in modeling population dynamics. These functional responses offer a robust description of ecological scenarios where predators capture innocuous prey. However, they may not be suitable for situations where prey exhibit defensive mechanisms. In 1930, Haldane included a non-monotonic type-IV functional response, incorporating the intensity of toxin, making it better suited for modeling situations involving prey defence [22]. Subsequently, Sokol and Howell (1981) proposed an improved Monod-Haldane (MH) functional response, formulated as $\phi(x) = \frac{x}{bx^2+e}$, aimed at providing a better description of group defense phenomena [23]. Here, b represents the measure of the inhibitory effect, while e denotes the half-saturation constant. This MH functional response offers a more nuanced understanding of trophic interactions, particularly in scenarios involving prey defense mechanisms. Pal et al. [24] utilized a simplified MH (Michaelis-Menten and Holling) functional response to investigate the fluctuations in toxin-producing aquatic microorganisms. Their study revealed that the initiation and cessation of blooms are influenced by the toxication process of phytoplankton. Lui and Tan [25] examined a two-dimensional prey-predator population model incorporating impulsive harvesting and stocking, employing the MH functional response to model group defense theory. Numerous researchers have explored the concept of defense mechanisms [26, 27], and Mishra et al. [28] investigated a tri-trophic prey and two-predator framework, incorporating prey defense mechanisms into their analysis.

To examine the dynamics of coral reefs, P.J. Mumby et al. [29] developed a straightforward analytical framework with a fully parameterized simulation model to demonstrate how

significantly the system's dynamical behaviour changes among both extremes of grazing in a coral-algal structure [29]. This model was further extended by Blackwood et al. [30] by incorporating fishing pressure management, taking into account feeding as an evolving process, and two distinct instances of coral recovery along with finding key fishing intensity to enable coral regeneration. Other models, such as those by Singh et al. [31] and Wang et al. [32], looked into the structure of fish biomass and how a coral reef can serve as a refuge for fish. An extended ODE model from Blackwood et al. [30] was taken into consideration by Fattahpour et al. [33] in order to explicitly assess the effect of herbivores fish abundance on macroalgal feeding and management practices on the reef ecosystem. Fleshy macroalgae and turf algae, which are more prevalent on reefs along the coast, are two examples of the wide range of food sources that herbivorous fish can consume. When there were nutrients available, herbivores were able to manage macroalgae, and the more nutrients present, the more powerful the herbivores' control over macroalgae was. Therefore, an effective way to safeguard reefs from nutrient pollution and its impact is to keep herbivore fish population from declining [34, 35]. The competition between seaweeds and corals for turf algae and its behavior in a coral reef benthic environment with herbivorous Parrotfish is also explored [36]. An eco-epidemiological model was developed in [37] to analyse the patterns of coral reefs under the effect of algal toxicity and illness caused by free-living pathogens. Existing models concentrated on the variations of coral-algal dynamics and a few factors affecting them, particularly the impact of overfishing, harmful fishing techniques, and disease-causing pathogens. Recent studies have found that nutrient pollution in reef ecosystems alters the coral-algal-herbivory structure, resulting in the mass killing of the fish population and coral degradation.

The central focus of this study is to explore how inhibitory effects, particularly allelochemicals, impact the interplay between algae and fish within coral reef ecosystems. This investigation encompasses a range of biological interactions, including predator-prey relationships, commensalism, and competition. While the coral-algal-herbivore fish triangle offers a useful conceptual framework, it is crucial to examine how algae-produced toxins influence the dynamics of these ecosystems. The paper is organized to guide the reader through a comprehensive analysis of the mathematical model developed. In Section 2, the Mathematical Model Formulation outlines the creation of the model that underpins the study. Section 3, Positivity and Boundedness Analysis examines conditions ensuring that the model's solutions remain both positive and bounded, which is crucial for realistic interpretations. Section 4 provides a detailed Equilibrium Analysis, divided into three subsections: Section 4.1 focuses on the Computation of Equilibrium Points, identifying various potential equilibrium states within the model; Section 4.2 covers Local Stability Analysis, assessing the stability conditions of each feasible equilibrium point; and Section 4.3 discusses Global Stability Analysis, exploring the conditions for global stability of the interior equilibrium. In Section 5, Bifurcation Analysis delves into the occurrence of Hopf bifurcations and their implications for the system's behavior. The Numerical Simulation Results in Section 6 present the findings from numerical simulations, offering insights into the dynamic behavior of the model under study. Section 7 discusses all the findings, highlighting the key results and implications of the research. Finally, Section 8 presents the conclusion of the research work.

2 Model formulation

In this work, we introduce a model to study a reef ecosystem comprising three interacting components: corals, toxic algae, and herbivorous fish. Our model incorporates the interaction between these components, with a specific focus on the influence of toxic algae on fish predation rates. We hypothesize that algae produce allelochemicals that reduce the predation pressure exerted by fish on phytoplankton density. We model algal-fish interaction using a modified Type-IV function, specifically the modified Holling (MH) type function. This function captures the adverse effect of algae on fish dynamics, mimicking a group defense mechanism exhibited by algae under stressful conditions, both against other algal species and their predators.

Let $C(t)$ denote the coral population, $A(t)$ denote the toxic algal population, and $F(t)$ denote the fish population. The model formulation is achieved using the following assumptions:

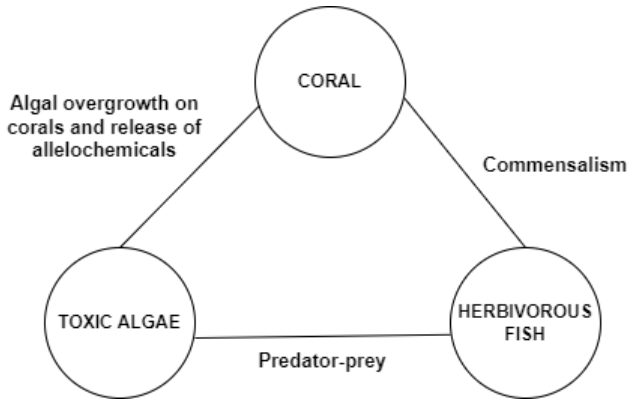

Figure 1. Population Diagram Flow

(i) Three different populations namely: algae $A(t)$, corals $C(t)$, and herbivorous fish $F(t)$ and their interactions are considered for the dynamical system.

(ii) Corals and algae exhibit logistic growth, characterized by rates r_1 and r_2, respectively, and with carrying capacities K_1 and K_2, respectively.

(iii) Corals experience overgrowth by algae at a rate α. Increased algal cover reduces coral settlement space, leading to more frequent and intense coral-algal interactions, diminishing coral recruitment, reducing coral growth rates, and inducing mortality.

(iv) The predation rate of algae by fish m_1 follows a Monod-Haldane functional form, where e and b represent the half-saturation constant and a direct measure of the inhibitory effect resulting from allelochemical secretion by the algal population, respectively. The maximum conversion rate of fish biomass m_2 represents the conversion of food into fish biomass.

(v) The predator (fish) grows logistically at a rate r_3 with a carrying capacity K_3. The commensalism relation among the predator and coral is observed in the predator's potential to sustain within the maximum capacity of coral population, represented by hC, where h quantifies the benefit derived by the predator from coral.

The model developed after implementing the assumption is as follows :

$$\begin{aligned}
\frac{dC}{dt} &= C[r_1(1-\frac{C}{K_1}) - \alpha A] \\
\frac{dA}{dt} &= A[r_2(1-\frac{A}{K_2}) + \alpha C - \frac{m_1 F}{bA^2+e}] \\
\frac{dF}{dt} &= F[r_3(1-\frac{F}{K_3+hC}) + \frac{m_2 A}{bA^2+e}]
\end{aligned} \tag{2.1}$$

with initial conditions $C(0) > 0$, $A(0) > 0$ and $F(0) > 0$.

3 Boundedness and positivity of solution

Theorem 3.1. *The solutions of system (2.1) exhibit non-negativity.*

Proof. The first equation of system (2.1) yields, $\frac{dC}{C} = \phi(C, A, F)dt$
here $\phi(C, A, F) = r_1\left(1 - \frac{C}{K_1}\right) - \alpha A$
By integrating the above equation through $(0, t)$, we have

$$C(t) = C(0)esp(\int_0^t \phi(C, A, F)dt) > 0, \forall\, t$$

Likewise, considering the next equation in (2.1), we get $\frac{dA}{A} = \psi(C, A, F)dt$
here $\psi(C, A, F) = r_2\left(1 - \frac{A}{K_2}\right) + \alpha C - \frac{m_1 F}{bA^2+e}$
By integrating the above equation through $(0, t)$, we get

$$A(t) = A(0)exp(\int_0^t \psi(C, A, F)dt) > 0, \forall t$$

Finally, derived from the last equation in (2.1), we get $\frac{dF}{F} = \chi(C, A, F)dt$
here $\chi(C, A, F) = r_3\left(1 - \frac{F}{K_3+hC}\right) + \frac{m_2 A}{bA^2+e}$
By integrating the above equation through $(0, t)$, then

$$F(t) = F(0)exp(\int_0^t \chi(C, A, F)dt) > 0, \forall t$$

Therefore, every system solution of (2.1) is non-negative. □

Theorem 3.2. *All solutions of system (2.1) are bounded.*

Proof. Consider the solution $(C(t), A(t), F(t))$ for system (2.1).
Since $\frac{dC}{dt} \leq Cr_1(1 - \frac{C}{K_1}) \leq \frac{r_1}{4} = N(say)$.
Again since $\frac{dA}{dt} \leq Ar_2(1 - \frac{A}{K_2}) + \alpha N \leq \frac{r_2}{4} + \alpha N = N_1(say)$.
Finally, derived from the third equation in system (2.1),
$\frac{dF}{dt} \leq F[r_3(1 - \frac{F}{K_3+hN}) + \frac{m_1 N_1}{bN_1^2+e}] \leq N_2[r_3(1 - \frac{N_2}{K_3+hN}) + \frac{m_1 N_1}{bN_1^2+e}] = N_3(say)$,
where $N_2 = \frac{K_3+hN}{2r_1}[r_2 + \frac{m_1 N_1}{bN_1^2+e}]$.
Thus every solution to the system (2.1) are bounded. □

4 Eqilibrium points and stability analysis

The identification of various possible equilibria within the model and the analysis of the stability conditions of all feasible equilibrium points are included in this section.

4.1 Equilibrium points

The five equilibria will be indicated as follows:

(i) Extinction equilibrium $E_0(0, 0, 0)$

(ii) Coral only equilibrium $E_1(C_1, 0, 0)$ and algae only equilibrium $E_2(0, A_1, 0)$ where $C_1 = K_1$ and $A_1 = K_2$

(iii) Algae free equilibrium $E_3(C_1, 0, F_1)$ where $F_1 = K_3 + hK_1$

(iv) Coexistence equilibrium $E^*(C^*, A^*, F^*)$ where

$$C^* = \frac{1}{h}\left[\frac{r_3 F^{*2}(bA^{*2} + e)}{r_3(bA^{*2} + e) + m_2 A^*} - K_3\right], A^* = \frac{r_1}{\alpha}\left(1 - \frac{C^*}{K_1}\right),$$

$$F^* = \frac{bA^{*2} + e}{m_1}\left[r_2(1 - \frac{A^*}{K_2}) + \alpha C^*\right] \tag{4.1}$$

4.2 Local stability analysis

This section focuses on analyzing the local behavior of system (2.1) near each equilibrium point. We calculate the variational matrix associated with each equilibrium in order to evaluate the stability conditions of the equilibria.

$$V(C,A,F)=\begin{bmatrix} r_1(1-\frac{2C}{K_1})-\alpha A & -\alpha C & 0 \\ \alpha A & r_2(1-\frac{2A}{K_2})+\alpha C-\frac{m_1F(-bA^2+e)}{(bA^2+e)^2} & -\frac{m_1A}{bA^2+e} \\ \frac{r_3F^2}{(K_3+hC)^2} & \frac{m_2F(-bA^2+e)}{(bA^2+e)^2} & r_3(1-\frac{2F}{K_3+hC})+\frac{m_2A}{bA^2+e} \end{bmatrix}$$

Theorem 4.1. *The extinction equilibrium $E_0(0,0,0)$ is always unstable.*

Proof. The variational matrix for system (2.1) at extinction equilibrium E_0 is given by

$$V(E_0)=\begin{bmatrix} r_1 & 0 & 0 \\ 0 & r_2 & 0 \\ 0 & 0 & r_3 \end{bmatrix}$$

The determinantal equation of the variational matrix $V(E_0)$ is given by

$$(r_1-\lambda)(r_2-\lambda)(r_3-\lambda)=0$$

The eigenvalues of the above determinantal equation are r_1, r_2, r_3. Thus, the equilibrium point E_0 is unstable since the eigenvalues are always positive. □

Theorem 4.2. *The coral-only equilibrium $E_1(C_1,0,0)$ is unstable.*

Proof. The variational matrix for system of equations (2.1) at coral-only equilibrium point E_1 is given by

$$V(E_1)=\begin{bmatrix} -r_1 & -\alpha K_1 & 0 \\ 0 & r_2+\alpha K_1 & 0 \\ 0 & 0 & r_3 \end{bmatrix}$$

The determinantal equation of the variational matrix $V(E_1)$ is given by

$$(-r_1-\lambda)(r_2+\alpha K_1-\lambda)(r_3-\lambda)=0$$

The eigenvalues of the above determinantal equation are $-r_1, r_2+\alpha K_1$ and r_3. E_1 is unstable since one eigenvalue is always positive. □

Theorem 4.3. *The algal-only equilibrium $E_2(0,A_1,0)$ is locally asymptotically stable if $r_1<\alpha K_2$ and $r_3<\frac{-m_2K_2}{bK_2^2+e}$.*

Proof. The variational matrix for system of equations (2.1) at algal-only equilibrium point E_2 is given by

$$V(E_2)=\begin{bmatrix} r_1-\alpha K_2 & 0 & 0 \\ \alpha K_2 & -r_2 & -\frac{m_1K_2}{bK_2^2+e} \\ 0 & 0 & r_3+\frac{m_2K_2}{bK_2^2+e} \end{bmatrix}$$

The determinantal equation of the variational matrix $V(E_2)$ is given by

$$(r_1-\alpha K_2-\lambda)(-r_2-\lambda)(r_3+\frac{m_2K_2}{bK_2^2+e}-\lambda)=0$$

So, all the roots of the above equation are $r_1-\alpha K_2$, $-r_2$ and $r_3+\frac{m_2K_2}{bK_2^2+e}$. The algal only equilibria E_2 is locally asymptotically stable if $r_1<\alpha K_2$ and $r_3<\frac{m_2K_2}{bK_2^2+e}$.

□

Theorem 4.4. *The algal free equilibrium $E_3(C_1, 0, F_1)$ is locally asymptotically stable if* $r_2 + \alpha K_1 < \frac{m_1}{e}(K_3 + hK_1)$

Proof. The determinantal equation for system of equations (2.1) at algal-free equilibrium point E_3 is given by

$$V(E_3) = \begin{bmatrix} -r_1 & -\alpha K_1 & 0 \\ 0 & r_2 - \alpha K_1 - \frac{m_1}{e}(K_3 + hK_1) & 0 \\ r_3 & \frac{m_2}{e}(K_3 + hK_1) & -r_3 \end{bmatrix}$$

The determinantal equation of the variational matrix $V(E_3)$ is given by

$$(-r_1 - \lambda)(r_2 + \alpha K_1 - \frac{m_1}{e}(K_3 + hK_1) - \lambda)(-r_3 - \lambda) = 0$$

Roots of the above equation are $-r_1$, $r_2 + \alpha K_1 - \frac{m_1}{e}(K_3 + hK_1)$ and $-r_3$. Therefore, in the preceding equation, every solution are negative if $r_2 + \alpha K_1 < \frac{m_1}{e}(K_3 + hK_1)$.

□

Theorem 4.5. *The coexistence equilibrium $E^*(C^*, A^*, F^*)$ is locally asymptotically stable if* $\sigma_1 > 0, \sigma_3 > 0$ *and* $\sigma_1\sigma_2 - \sigma_3 > 0$.

Proof. The variational matrix for system of equations (2.1) at coexistence equilibrium equilibrium point E^* is given by

$$V(E^*) = \begin{bmatrix} a_{11} & a_{12} & 0 \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix}$$

where $\sigma_1 = -(a_{11} + a_{22} + a_{33}), \sigma_2 = a_{11}a_{22} + a_{11}a_{33} + a_{22}a_{33} + a_{12}a_{21} - a_{23}a_{33}, \sigma_3 = a_{11}a_{23}a_{32} - a_{11}a_{22}a_{33} + a_{12}a_{23}a_{31} - a_{12}a_{21}a_{33}, a_{11} = r_1(1 - \frac{2C^*}{K_1}) - \alpha A^*, a_{12} = -\alpha C^*, a_{21} = \alpha A^*, a_{22} = r_2(1 - \frac{2A}{K_2}) + \alpha C - \frac{m_1 F(-bA^2+e)}{(bA^2+e)^2}, a_{23} = -\frac{m_1 A}{bA^2+e}, a_{31} = \frac{r_3 F^2}{(K_3+hC)^2}, a_{32} = \frac{m_2 F(-bA^2+e)}{(bA^2+e)^2}$ and $a_{33} = r_3(1 - \frac{2F}{K_3+hC}) + \frac{m_2 A}{bA^2+e}$.
The determinantal equation of the variational matrix $V(E^*)$ is given by

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0 \tag{4.2}$$

As per Routh-Hurwitz criteria, $E^*(C^*, A^*, F^*)$ is locally asymptotically stable if
$\sigma_1 > 0, \sigma_3 > 0$ and $\sigma_1\sigma_2 > \sigma_3$

□

4.3 Global stability analysis

Explores the global stability of the interior equilibrium point E^* in this section.

Theorem 4.6. *The interior equilibrium $E^*(C^*, A^*, F^*)$ is globally asymptotically stable if* $\alpha^2 < \frac{1}{4K_1K_2}$ *and* $\frac{(m_1e+m_2e)^2}{(bA^2+e)^2(bA^{*2}+e)^2} < \frac{K_3}{K_2(K_3+hC)(K_3+hC^*)}$.

Proof. To demonstrate the global stability of the equilibrium state E^*, we assume the utilization of the following positive definite function:

$$Y = (C - C^*) - C^* ln\frac{C}{C^*} + (A - A^*) - A^* ln\frac{A}{A^*} + (F - F^*) - F^* ln\frac{F}{F^*}$$

Upon differentiation of the aforementioned equation with respect to time, we obtain

$$\frac{\mathrm{d}Y}{\mathrm{d}t} = \left(\frac{C - C^*}{C}\right)\frac{\mathrm{d}C}{\mathrm{d}t} + \left(\frac{A - A^*}{A}\right)\frac{\mathrm{d}A}{\mathrm{d}t} + \left(\frac{F - F^*}{F}\right)\frac{\mathrm{d}F}{\mathrm{d}t}$$

$$\frac{\mathrm{d}Y}{\mathrm{d}t} = (C-C^*)\left[r_1\left(1-\frac{C}{K_1}\right)-\alpha A - r_1\left(1-\frac{C^*}{K_1}\right)+\alpha A^*\right]$$
$$+(A-A^*)\left[r_2\left(1-\frac{A}{K_2}\right)+\alpha C-\frac{m_1F}{bA^2+e}-r_2\left(1-\frac{A^*}{K_2}\right)-\alpha C^*+\frac{m_1F^*}{bA^{*2}+e}\right]$$
$$+(F-F^*)\left[r_3\left(1-\frac{F}{K_3+hC}\right)+\frac{m_2A}{bA^2+e}-r_3\left(1-\frac{F^*}{K_3+hC^*}\right)-\frac{m_2A^*}{bA^{*2}+e}\right]$$

$$\frac{dY}{dt} = -\frac{(C-C^*)^2}{K_1}-\frac{(A-A^*)^2}{K_2}-\frac{K_3(F-F^*)^2}{(K_3+hC)(K_3+hC^*)}-2\alpha(C-C^*)(A-A^*)$$
$$-(m_1e+m_2e)\frac{(A-A^*)(F-F^*)}{(bA^2+e)(bA^{*2}+e)}$$
$$-\frac{m_1b(A-A^*)(FA^{*2}-F^*A^2)+m_2b(F-F^*)(AA^{*2}-A^*A^2)}{(bA^2+e)(bA^{*2}+e)}$$
$$\leq -\frac{(C-C^*)^2}{K_1}-\frac{(A-A^*)^2}{K_2}-\frac{K_3(F-F^*)^2}{(K_3+hC)(K_3+hC^*)}-2\alpha(C-C^*)(A-A^*)$$
$$-(m_1e+m_2e)\frac{(A-A^*)(F-F^*)}{(bA^2+e)(bA^{*2}+e)} \tag{4.3}$$

if $\frac{C}{C^*}>1$, $\frac{A}{A^*}>1$ and $\frac{F}{F^*}>1$
Then we obtain

$$\frac{dY}{dt} \leq -\frac{(C-C^*)^2}{K_1}-\frac{(A-A^*)^2}{K_2}-\frac{K_3(F-F^*)^2}{(K_3+hC)(K_3+hC^*)}-2\alpha(C-C^*)(A-A^*)$$
$$-(m_1e+m_2e)\frac{(A-A^*)(F-F^*)}{(bA^2+e)(bA^{*2}+e)}$$

Sufficient conditions for $\frac{dY}{dt}$ to be negative definite obtained by Sylvester's criteria are:

$$\alpha^2 < \frac{1}{4K_1K_2} \tag{4.4}$$

$$\frac{(m_1e+m_2e)^2}{(bA^2+e)^2(bA^{*2}+e)^2} < \frac{K_3}{K_2(K_3+hC)(K_3+hC^*)} \tag{4.5}$$

Hence the proof. □

5 Bifurcation analysis

In relation to b (inhibitory effect), the Hopf bifurcation of our suggested system (2.1) has been examined in this section.

Theorem 5.1. *If the inhibitory effect of algae, b crosses the critical value b^* then the system enters into Hopf−bifurcation around the equilibrium E^*.*

Proof. The necessary and sufficient conditions for Hopf-bifurcation to appear from the interior equilibrium E^*, there must be a critical point for a given value for the parameter b, let's say $b=b^*$, such that such that

(i) $\sigma_1(b^*)>0$

(ii) $Q(b^*) = \sigma_1(b^*)\sigma_2(b^*) - \sigma_3(b^*)$=0

(iii) $\left[\frac{\mathrm{d}}{\mathrm{d}b}(\sigma_1\sigma_2 - \sigma_3)\right]_{b=b^*} \neq 0$

The determinantal equation is given by

$$\left[\lambda^2 + \sigma_2(b^*)\right]\left[\lambda + \sigma_1(b^*)\right] = 0$$

The roots of the above determinantal equation are $\lambda_{1,2}(b^*) = \pm i\sqrt{\sigma_2(b^*)}$, and $\lambda_3 = -\sigma_1(b^*)$. We can write the root as follows

$$\lambda_1(b) = \alpha_1(b) + i\alpha_2(b), \lambda_2 = \alpha_1(b) - i\alpha_2(b), \lambda_3(b) = -\sigma_1(b)$$

The transversally condition is given as

$$\left[\frac{\mathrm{d}Re(\lambda_j(b))}{\mathrm{d}b}\right]_{b=b^*} \neq 0, j = 1, 2$$

Upon replacing $\lambda_j(b) = \alpha_1(b) \pm i\alpha_2(b)$ in equation (4.2) and splitting both the real and imaginary components, we obtain

$$\alpha_1^3 - 3\alpha_1\alpha_2^2 + \sigma_1(\alpha_1^2 - \alpha_2^2) + \sigma_2\alpha_1 + \sigma_3 = 0 \tag{5.1}$$

$$3\alpha_1^2\alpha_2 - \alpha_2^3 + 2\sigma_1\alpha_1\alpha_2 + \sigma_2\alpha_2 = 0 \tag{5.2}$$

Here $\alpha(b) \neq 0$
Therefore from (5.2), we have

$$\alpha_2^2 = 3\alpha_1^2 + 2\sigma_1\alpha_1 + \sigma_2$$

Substituting this in (5.1), we get

$$8\alpha_1^3 + 8\sigma_1\alpha_1^2 + 2\alpha_1(\sigma_1^2 + \sigma_2) + \sigma_1\sigma_2 - \sigma_3 = 0$$

Since $\alpha_1(b^*) = 0$, we get

$$\begin{aligned}\alpha_1{}'(b^*) &= \frac{d}{db}\left[\{Re\lambda(b)\}\right]_{b=b^*} \\ &= -\left[\frac{1}{2(\sigma_1^2 + \sigma_2)}\frac{d}{db}(\sigma_1\sigma_2 - \sigma_3)\right]_{b=b^*}\end{aligned}$$

which is non-zero if

$$\left[\frac{d}{db}(\sigma_1\sigma_2 - \sigma_3)\right]_{b=b^*} \neq 0$$

Therefore the transversally condition holds. This implies that a Hopf−bifurcation occurs at $b = b^*$. Hence the proof. □

6 Numerical Simulation

We have employed numerical methods, specifically utilizing MATLAB, to solve equation (2.1) and gain deeper insights into our proposed model. Setting the parametric values from the set allows us to perform numerical simulations

$$r_1 = 1, r_2 = 0.2, r_3 = 0.9, K_1 = 20, K_2 = 20, K_3 = 15, \alpha = 0.32,$$

$$m_1 = 0.65, b = 0.25, e = 20, h = 0.6, m_2 = 0.6. \tag{6.1}$$

This combination of parametric parameters has been utilised to create fig.2. The proposed system (2.1) using the aforementioned collection of parameters has a single positive equilibrium value (1.0282, 2.9672, 17.0093) which is in accordance with the theory. It is shown from the system dynamics analysis that the coexistence equilibrium shows local asymptotic stability. This suggests that under appropriate conditions, the prey, predator, and coral species can coexist within the ecosystem.

The bifurcation diagram of the system (2.1) concerning the parameter b (inhibitory effect) is depicted in fig.3 for the specified collection of parameters (6.1). From this illustration, it is evident that when the value of b (inhibitory effect) is augmented, specifically for $b > 0.252$, the stable equilibrium point E^* of the system transitions into instability. This clearly indicates that the inhibitory effect negatively affects the stability of the system. Again when decreasing the value of h, i.e. the measure of conversion factor quantifying the benefit that fish derive from coral, the interior equilibrium point E^* becomes unstable which is depicted in fig.4.

7 Discussion

In this study, we explore the dynamic interactions among prey (algae), predator (fish), and coral populations, incorporating a MH type functional response. We investigated the boundedness and positivity of solutions for our suggested framework (2.1). For all possible equilibria of the mathematical model (2.1), we have analytically found the local stability constraints. As shown in fig.2, we have observed that the coexistence equilibrium $E^*(C^*, A^*, F^*)$ exhibits local asymptotic stability under certain circumstances that support the combined existence of three populations. Furthermore, as shown by equations (4.4-4.5), the coexistence equilibrium has been the subject of research on the global stability of our suggested model. Hopf bifurcation happens when the entire system becomes unstable due to an algal toxicity that exceeds its critical value of 0.252. Figure3 makes it evident that our suggested model exhibits oscillatory behaviour as the inhibitory impact of prey (b) connected to the Monod–Haldane functional response fluctuates which is demonstrated by numerical simulations. Our study shows there is a decrease in coral density for the increased values of $(b > 0.252)$ (inhibitory effect) which is shown in fig.5. From the analysis of proposed framework (2.1), we can conclude that the density of coral and fish population are directly linked which is understood from eq.(4.1). Hence any change in coral population due to the allelochemical produced by algae will automatically affect the fish population confirming the commensalism relation that fish share with corals. Further investigation of the factor quantifying the benefit that fish derive from coral (h) shows that by decreasing the value h, the system becomes unstable which is shown in fig.4. Additionally, increased toxicity in algae raises the probability of coral species extinction. Due to the complex dynamical system arising as a result of the toxic production of algae, the commensalism relation between fish and corals as well as the benefits fish derive from corals are affected.

More in-depth assessments of dynamical models are becoming greater importance to ecologists, particularly when it comes to comprehending the consequences of shifting ecological systems or historical reliance on biological structures. A region of attraction for various asymptotic conditions may need to be identified in order to ascertain the future trajectory of ecosystems, particularly as a consequence of external disturbances. When too many nutrients, such as nitrogen and phosphorus, are released into the oceans through industrial emissions, sewerage and pollutants, runoff from agriculture, and atmospheric release, it can lead to nutrient pollution in coral reef ecosystems and toxic algal blooms. Methods like field sampling, lab analysis, and remote sensing are used in the analysis of nutrient contamination in the ocean. A thorough grasp of the chemical relationships that exist between hazardous algae and their surroundings is necessary to analyse nutrient contamination using the levels of allelochemicals generated by these algae. While investigating nutrient pollution in aquatic ecosystems, one can make use of the outcomes of this work to validate their results by examining the level of toxicity of algae in the surrounding environment.

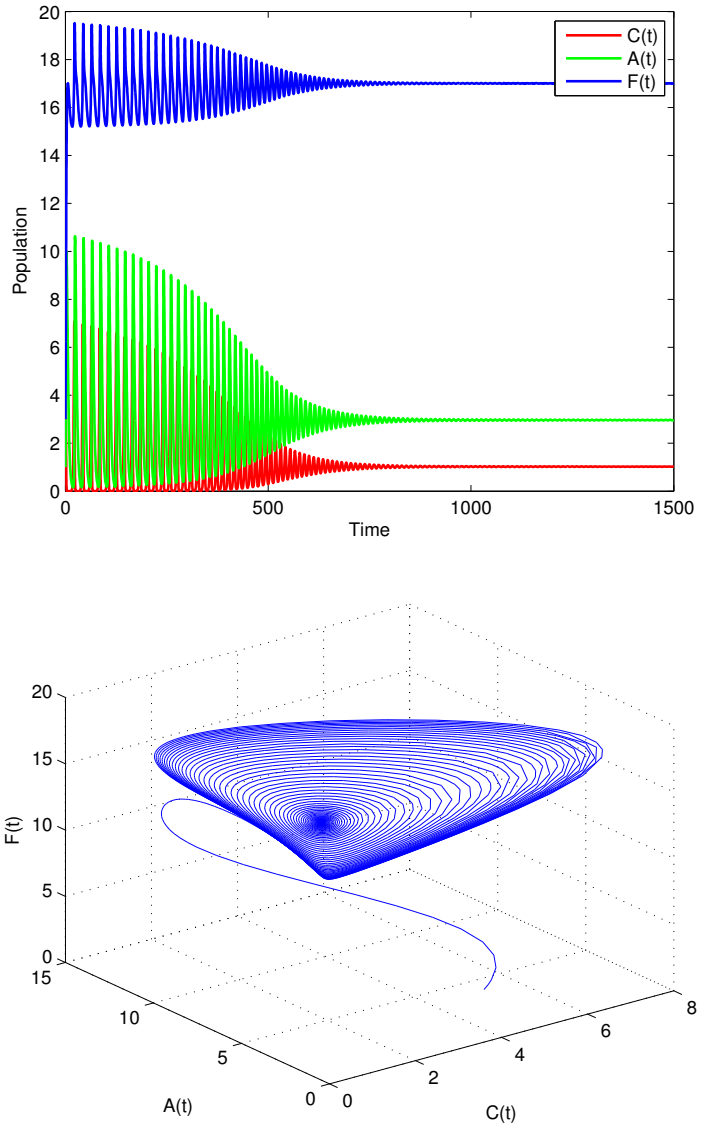

Figure 2. Model dynamics of (2.1) illustrating the coexistence equilibrium E^*'s stability behaviour over time for the parametric values given in (6.1)

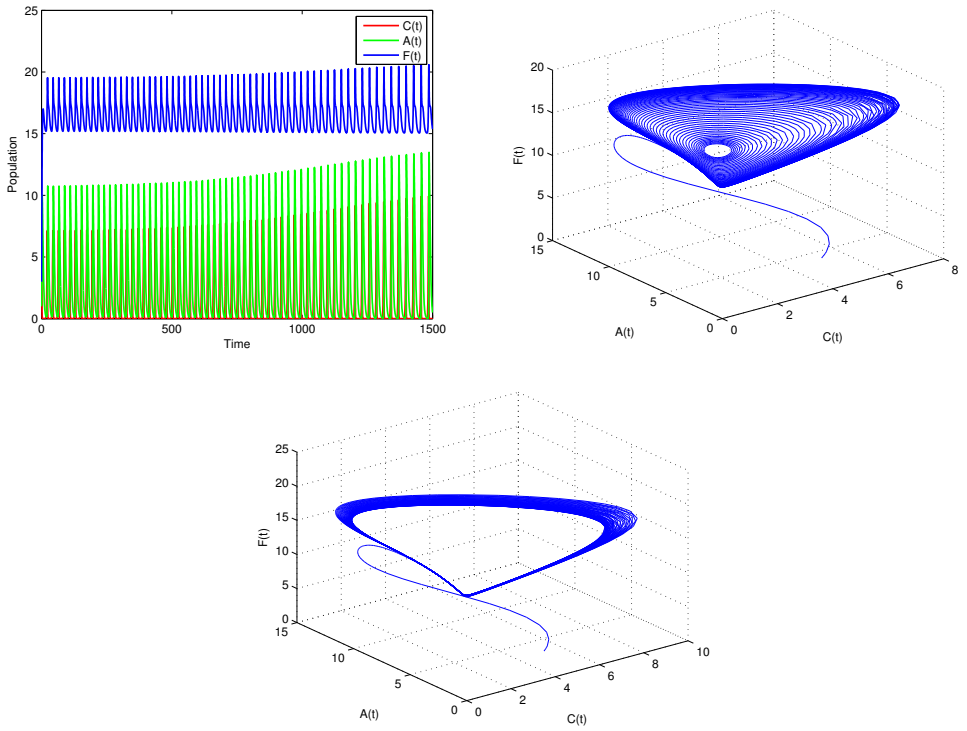

Figure 3. Bifurcation diagram for model system (2.1) w.r.t the value of 'b'

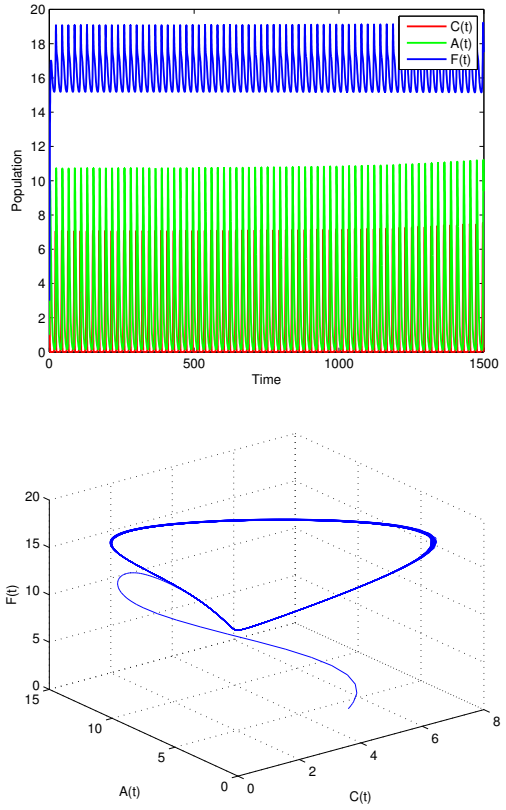

Figure 4. Bifurcation diagram for model system (2.1) w.r.t the value of 'h'

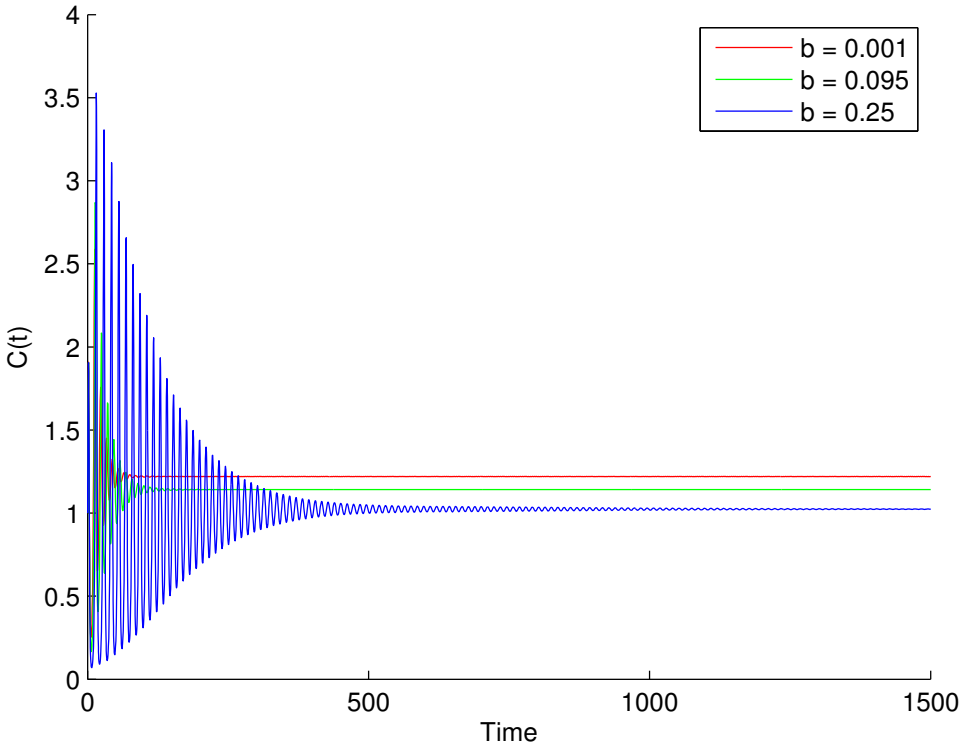

Figure 5. Graph between population (C) and time t with increasing values of b

8 Conclusion

From the research work, it is concluded that when there is nutrient enrichment in the reef ecosystem, fleshy algae producing allelochemicals overgrow corals as a result of competition for space, light, and food. As a result, the coral cover diminishes and will negatively affect the growth of algae feeding herbivorous fishes that rely on coral. This algal growth can be controlled only by enhancing the water quality by keeping the nutrient level under control. This work provides insight for future researchers, policymakers, and conservation management over the world to understand the impacts on coral reef ecosystems that have increased rapidly caused by nutrient pollution, land runoff, and toxic algal blooms. From our findings, we suggest that the complex dynamical system changing due to the release of allelochemicals by algae should not be allowed to exceed the critical value of toxicity as mentioned in this paper. Researchers can utilise the results to guide the development of management plans that lessen the effects of nutrient contamination and reduce it. We suggest that in order to avoid mass fish killing due to the chemicals released by the proliferation of toxic algae, nutrient levels in the ecosystems should be put under control. These effects could have significant management possibilities for coral reefs along with other biological systems with dynamic behaviour related to them.

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