MACROALGAL ALLELOPATHY IN THE EMERGENCE OF CORAL DISEASES

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Abstract. Microbial disease in corals associated with the proliferation of benthic macroalgae are the major contributors to the decline of coral reefs over the past few decades. Several benthic macroalgae species produce allelopathic chemical compounds that negatively affect corals. The emergence of microbial diseases in corals occurs simultaneously with the elevated abundance of benthic macroalgae. The release of allelochemicals by toxic-macroalgae enhances microbial activity on coral surfaces via the release of dissolved compounds. Proliferation of benthic macroalgae in coral reefs results in increased physical contacts between corals and macroalgae, triggering the susceptibility of coral disease. The abundance of macroalgae changes the community structure towards macroalgae dominated reef ecosystem. We investigate coral-macroalgal phase shift in presence of macroalgal allelopathy and microbial infection on corals by means of an eco-epidemiological model under the assumption that the transmission of infection is mediated by the pathogens shed by infectious corals and under the influence of macroalgae in the environment. We perform equilibrium and stability analysis on our non-linear ODE model and found that the system is capable of exhibiting the existence of two stable configurations of the community under the same environmental conditions by allowing saddle-node bifurcations that involves in creation and destruction of fixed points and associated hysteresis effect. It is shown that the system undergoes a sudden change of transition when the transmission rate of the infection crosses some certain critical thresholds. Computer simulations have been carried out to illustrate different analytical results.

1. Introduction. Macroalgal proliferation and coral diseases have been identified as among the most important contributors to the global degradation of coral reef ecosystems. Some of the microbial diseases in corals have been found to be positively correlated with increasing macroalgal abundance [27, 35]. White Plague Disease (WPD) is one of the most common and prevalent diseases that has been observed affecting coral reefs in the Caribbean basin [34]. Throughout the Caribbean and Western Atlantic, WPD is recognized as a destructive coral infectious bacterial disease that affects a number of coral species [30]. Depending on the type of WPD...
(I, II or III), progression rates vary and different coral species are affected. Experimental observations show that direct contact of the green algae *Halimeda opuntia* with healthy corals could trigger WPD-II associated with the bacterial pathogen, *Aurantimonas coralicaida* present on *H. opuntia* [26]. This microbial community has been identified as the main causative agent of WPD-II in the Caribbean corals by creating a chemical environment that digests off and dissolves the coral tissue away from the skeleton [33] which may result in coral bleaching. Several macroalgae species contain multiple hydrophobic compounds that directly damage coral tissues by transferring hydrophobic allelochemicals present on algal surfaces [7, 9]. *Halimeda opuntia* exhibits allelopathic activity against corals, and are known to be rich in secondary metabolites which are damaging to most corals. Also, the contact with the *Halimeda opuntia* can trigger WPD-II in corals. Field observations by [1, 29] demonstrate that corals, when in contact with toxic-macroalgae species experience reduced fecundity and even higher mortality. The release of allelochemicals by toxic-macroalgae influences the microbes associated with corals by transmitting pathogens. Macroalgae release large amounts of organic carbon into the surrounding environment, providing nutrients for coral pathogens and triggering chemotactic behaviour of pathogens [31]. Proliferation of benthic macroalgae in coral reefs results in increased physical contacts between corals and macroalgae leading to an increase in exposure to pathogens. The abundance of macroalgae changes the community structure towards macroalgae dominated reef ecosystem [11, 24]. The combinations of outbreak of WPD-II and proliferation of macroalgae is detrimental to coral reef ecosystems.

In coral reef ecosystems, macroalgae and corals compete intensively for the available space in seabed and the grazing by reef herbivores prevents algae to proliferate. In the absence of herbivory by reef fishes, the faster growing macroalgae often overgrow corals by causing their decline [18, 20, 25]. Phase shifts in an ecosystem occur when a smooth change in an active parameter value triggers a complete different behaviour of the system. As observed by Done [13] and Bellwood et al. [3], degradation of coral reefs often exhibits phase shifts in community structure for which corals decline with an increase in abundance of macroalgae [6]. Researchers observe that coral disease together with the reduction in herbivory can lead to a permanent shift in regime in which macroalgae, once dominant, inhibit coral settlement [7, 10]. The grazing of macroalgae by herbivores contributes to the resilience of the coral-dominated reef [19, 21, 28]. The response of coral reef ecosystems to changing external perturbations vary from smooth to discontinuous [12]. In case of smooth phase transitions, the state of reef ecosystems may respond in a gradual and continuous way to external perturbations. In discontinuous phase shifts, reef ecosystems exhibit hysteresis over a range of conditions, typically corresponding to a saddle-node bifurcation. A catastrophic shift of regime with hysteresis effect occurs when the grazing rate of herbivores and the transmission rate of WPD-II cross some critical thresholds. As different sets of feedbacks stabilize each alternative regime, returning the system from macroalgae-dominated regime to coral-dominated regime becomes difficult [14].

Our model originates from the models used in [8, 23] and is a direct extension of the models studied in [3, 5] by assuming that the disease spreads among corals according to a Susceptible → Infected (SI) type compartmental eco-epidemiological model [2]. For free-moving terrestrial organisms, transmission rates are mainly dependent on the contact among susceptible and infectious hosts, whereas in the
marine environment disease agents move more widely and rapidly within the water column \[15\]. Since corals are sessile in nature, in our proposed model, considering an indirect mode of transmission of WPD-II from susceptible to infected corals seems more appropriate rather than choosing a contagious mode of transmission. We have incorporated a growth equation of pathogens in our model to study the dynamics of this indirect mode of disease transmission under the assumption that infectious corals contaminate the environment by shedding pathogens and the susceptible corals become infectious by the contaminated environment. It is also assumed that macroalgal species promote the transmission of the disease by acting as a reservoir of the pathogens. The main emphasis of our study will be put in analyzing the dynamic behaviour of the system and to find out the long-term consequences of WPD-II and reduction in herbivory on coral resilience and persistence.

2. The basic model. To study the dynamics of three benthic groups - corals, turf algae and toxic-macroalgae, competing for space on the seabed in presence of microbial infection, we consider an SI-eco-epidemiological model by assuming that macroalgae are always present in the system irrespective of the abundance of corals in sea bed and by ignoring the possibility of any empty space in seabed. Let \( M(t) \) be the proportion of seabed covered by macroalgae, \( C_S(t) \) be the proportion of seabed covered by susceptible coral, \( C_I(t) \) be the proportion of seabed covered by infected coral and \( T(t) \) represent the fraction of seabed occupied by turf-algae so that \( M(t) + C_S(t) + C_I(t) + T(t) = c_0 \) (constant) at any instant \( t \). Infectious corals shed pathogens in the environment and the susceptible corals become infectious through the contaminated environment. The higher pathogen density increases the chance that susceptible corals become infected. We assume that the infection passes in coral population at a rate proportional to the product of susceptible coral cover and pathogen concentration in the environment. Also, we assume that corals are born free of infection and the infected coral population does not contribute to the reproduction. Let \( W(t) \) be the population density (cells L\(^{-1}\)) of free-living pathogen (FLP) in the environment at time \( t \). As observed by Hibbing et al. \[17\], in a well-mixed bacteria culture, individuals of the same bacteria population compete for acquisition of nutrients as they become depleted by the growing population. Since the mechanisms by which WPD-II act upon the coral polyp are not well known, the crowding effect of the bacterial populations for WPD-II cannot be ruled out. In our model we choose \( \delta \) as a crowding parameter in the growth equation of pathogens. Grazing rate of herbivores on macroalgae is \( \frac{g}{M+T} \) per unit area of algal cover, where \( g \) is the maximal grazing rate of herbivorous fish. For simplicity, we have ignored the possibility of any empty space in the sea bed. A schematic diagram of the system is given in Fig.1.

Assuming that the grazing of herbivores as a dynamic process, the reef dynamics are thus described as a system of nonlinear differential equations:

\[
\begin{align*}
\frac{dM}{dt} &= M \left\{ \alpha(C_S + C_I) - \frac{g}{M+T} - d_1 \right\} + (aM + b)T \\
\frac{dC_S}{dt} &= C_S \left\{ rT - (\alpha + \gamma)M - \lambda W - d_2 \right\} 
\end{align*}
\]
Figure 1. Schematic representation of the model

\[
\begin{align*}
\frac{dC_I}{dt} &= \lambda C_S W - C_I \left\{ (\alpha + \gamma) M + d_2 + \eta \right\} \\
\frac{dT}{dt} &= \frac{gM}{M + T} + d_1 M + (d_2 + \gamma M)(C_S + C_I) + \eta C_I - T(aM + b + rC_S) \\
\frac{dW}{dt} &= W(\nu_1 M + \nu_2 C_I - d_3 - \delta W)
\end{align*}
\]

where \(M(0) > 0, C_S(0) \geq 0, C_I(0) \geq 0, T(0) > 0\) and \(W(0) \geq 0\).

All the parameters used in the model (1) are positive and are presented in the Table 1. Most of the parameter values in Table 1 are from field observations with an yearly estimated value. Box et al. [10] observed that the growth rate \(r\) of corals in cage control is 1 cm yr\(^{-1}\) and in the presence of macroalgae corals have a reduced growth of 0.55 cm yr\(^{-1}\). No-cage controls represent an yearly coral mortality rate \(d_2\) of 0.42 ± 1.62. The upper and lower 95% confidence intervals for the yearly mortality rate of 0.42 were calculated as 0.108 and 0.9. In Table 1 we have taken the coral mortality rate as 0.24 yr\(^{-1}\) which is well within lower 95% confidence intervals for the yearly mortality rate. The death rate of corals due to macroalgal toxicity \(\gamma\) and the disease-induced death of corals \(\eta\) are taken as 0.1 yr\(^{-1}\) and 0.01 yr\(^{-1}\) respectively. Thus, the total death rate of corals is 0.35 yr\(^{-1}\) which is still within lower 95% confidence interval for the yearly mortality rate of 0.42. The macroalgal limited overgrowth on corals \(\alpha\) is given in Lirman [19]. As observed by Mumby et al. [22], in absence of hurricane disturbances, the average macroalgal vegetative growth rate \(a\) on turf algae is 1.2 yr\(^{-1}\), whereas
during hurricane period the average growth rate becomes 0.35 yr\(^{-1}\). Therefore, the average macroalgal vegetative growth rate can be taken as 0.77 yr\(^{-1}\). In [22] it is also observed that macroalgal cover suffers severe losses during hurricane impact and the average yearly loss of macroalgal cover due to hurricane impact is 0.083. In Table 1 we choose macroalgal mortality rate \((d_1)\) as a combination of natural mortality, impact by fishing boats and hurricane impact as 0.1 yr\(^{-1}\).

Since pathogens in the marine environment survive for longer periods and can remain viable for several hundreds of years without its primary host [16], we assume that pathogen decay rate due to decontamination or natural mortality to be very small \((d_3)=0.01 yr^{-1}\).

The rate of macroalgal immigration on algal turf \((b)\) and the maximal grazing rate of herbivores \((g)\) are given in [15]. Without any loss of generality, we assume that \(c_0=1\). Then from (1) we obtain

\[
\begin{align*}
\frac{dM}{dt} &= M\left\{\alpha(C_S + C_I) - \frac{g}{1 - C_S - C_I} - d_1\right\} + (aM + b)(1 - M - C_S - C_I) \equiv f^1 \\
\frac{dC_S}{dt} &= C_S \{r(1 - M - C_S - C_I) - (\alpha + \gamma)M - \lambda W - d_2\} \equiv f^2 \\
\frac{dC_I}{dt} &= \lambda C_SW - C_I \{(\alpha + \gamma)M + d_2 + \eta\} \equiv f^3 \\
\frac{dW}{dt} &= W(\nu_1M + \nu_2C_I - d_3 - \delta W) \equiv f^4
\end{align*}
\]

where \(M(0) > 0, C_S(0) \geq 0, C_I(0) \geq 0\) and \(W(0) \geq 0\).

**Table 1.** Parameters used in the model (1).

| Parameter | Description                                      | Value          | Reference |
|-----------|--------------------------------------------------|----------------|-----------|
| \(\alpha\) | Rate of macroalgal direct overgrowth over coral  | 0.1 yr\(^{-1}\) | [8, 15, 19]|
| \(r\)     | Recruitment rate of corals on turf algae         | 0.55 yr\(^{-1}\) | [10, 15]  |
| \(a\)     | Macroalgal vegetative growth rate on turf algae  | 0.77 yr\(^{-1}\) | [15, 22]  |
| \(b\)     | Immigration rate of macroalgae on algal turf     | 0.005 yr\(^{-1}\) | [16]      |
| \(d_1\)   | Mortality rate of macroalgae                    | 0.1 yr\(^{-1}\) | [4, 22]   |
| \(d_2\)   | Natural mortality rate of corals                 | 0.24 yr\(^{-1}\) | [4, 10]   |
| \(\gamma\) | Toxin-induced death rate of corals              | 0.1 yr\(^{-1}\) | -         |
| \(\nu_1\) | Rate of release of pathogens by macroalgae       | 0.1 yr\(^{-1}\) | -         |
| \(\nu_2\) | Pathogen-shedding rate by infectious corals      | 0.3 yr\(^{-1}\) | -         |
| \(\frac{1}{\delta}\) | Average time pathogens exist in environment     | 100 yrs        | [16]      |
| \(\lambda\) | Rate of infection                               | 0.2 L cell\(^{-1}\)yr\(^{-1}\) | -         |
| \(\eta\)  | Disease induced death rate of infected corals    | 0.01 yr\(^{-1}\) | -         |
| \(g\)     | The maximal grazing rate of herbivorous fish     | 0.6 yr\(^{-1}\) | [15]      |
| \(\delta\) | Crowding parameter                              | 0.01 L cell\(^{-1}\)yr\(^{-1}\) | -         |

We observe that right-hand sides of the equations in the system (2) are smooth functions of the variables \(M, C_S, C_I, W\) and the parameters. As long as these quantities are non-negative, local existence and uniqueness properties hold in the positive octant.

**Lemma 2.1.** For all \(\epsilon > 0\), there exists \(t_\epsilon > 0\) such that all the solutions of (2) enter into the set

\[
\{ M(0), C_S(0), C_I(0), W(0) \} \subset \{ M(t), C_S(t), C_I(t), W(t) \} : t \in [0, t_\epsilon]
\]
Lemma 3.2. The system \((M, C_S, C_I, W) \in \mathbb{R}^4 : M(t) + C_S(t) + C_I(t) + W(t) < \frac{\nu_1 + \nu_2 + \delta - d_3}{3} + \epsilon\) whenever \(t \geq t_*\).

Proof. Since \(M(t), C_I(t) \leq 1\) for all \(t\), we have \(\frac{dW}{dt} \leq d_4 W \left( \frac{\nu_1 + \nu_2 - d_3}{3} - W \right)\).

Let \(U(t)\) be the solution of \(\frac{dU}{dt} = d_4 U \left( \frac{\nu_1 + \nu_2 - d_3}{3} - U \right)\), satisfying \(U(0) = W(0)\).

Then \(U(t) = k \frac{\nu_1 + \nu_2 - d_3}{3} e^{\frac{\delta}{1 + ke^{\frac{\nu_1 + \nu_2 - d_3}{3}}}}\), where \(k = \frac{\delta w(0)}{\nu_1 + \nu_2 - d_3 - \delta w(0)}\).

If \(\nu_1 + \nu_2 \leq d_3\), then \(U(t) \rightarrow 0\) as \(t \rightarrow \infty\).

If \(\nu_1 + \nu_2 > d_3\), then \(U(t) = \frac{\nu_1 + \nu_2 - d_3}{3} e^{\frac{\delta}{k + e^{-\frac{\nu_1 + \nu_2 - d_3}{3}}}}\) as \(t \rightarrow \infty\).

Applying the standard theorem of differential inequality we obtain \(\lim_{t \rightarrow \infty} \sup W(t) \leq \frac{\nu_1 + \nu_2 - d_3}{3}\).

Therefore, \(\lim_{t \rightarrow \infty} \sup [M(t) + C_S(t) + C_I(t) + W(t)] \leq \frac{\nu_1 + \nu_2 + \delta - d_3}{3}\). \(\square\)

3. Equilibria and their stability. In this section we determine biologically feasible equilibrium solutions of the model and investigate the dependence of their stability on several key parameters.

The system (2) possesses the following equilibria:

(i) Coral and pathogen-free equilibrium \(E_0 = (M_0, 0, 0, 0)\) exists always, where \(M_0 = \frac{a - b - d_1 - g + \sqrt{(a - b - d_1 - g)^2 + 4ab}}{2}\).

(ii) Coral-free equilibrium in presence of pathogens \(E_1 = (M_0, 0, 0, W_1)\), where \(W_1 = \frac{\nu_1 M_0 - d_3}{\delta}\). \(E_1\) exists if \(\nu_1 > \frac{d_3}{M_0}\).

(iii) Infection-free equilibrium \(E_2 = (M_1, C_S, 0, 0)\), where \(M_1\) is a positive root of the equation

\[M \left\{ \alpha(p_1 + q_1 M) - \frac{g}{1 - p_1 - q_1 M} - d_1 \right\} + \frac{(aM + b)(\lambda M + d_2)}{r} = 0, \quad C_S = p_1 + q_1 M, \quad p_1 = 1 - \frac{d_2}{r}, \quad q_1 = -\frac{\lambda d_2}{r^2}\.

(iv) Infected coral-free equilibrium in presence of pathogens \(E_3 = (M_1, C_S, 0, W_2)\), where \(W_2 = \frac{\nu_1 M_1 - d_3}{\delta}, C_{S_2} = p_2 + q_2 M_1, p_2 = 1 - \frac{d_2}{r} - \frac{\lambda d_2}{r^2}, q_2 = -\frac{(r + \alpha + \gamma)}{r}\).

\(E_3\) exists if \(\nu_1 > \frac{d_3}{M_0}\).

(v) Interior equilibrium \(E^* = (M^*, C_S^*, C_I^*, W*)\), where \(M^*\) is a positive root of the equation \(r (1 - M - f_2(M^*) - f_3(M^*)) - \alpha M + \gamma M - \lambda f_1(M^*) - d_2 = 0, \quad C_S^* = f_2(M^*), \quad C_I^* = f_3(M^*)\) and \(W^* = f_1(M^*)\).

We analyze the stability of system (2) by using eigenvalue analysis of the Jacobian matrix evaluated at the appropriate equilibrium.

At \(E_0\) the eigenvalues of the Jacobian matrix of the system (2) are \(\nu_1 M_0 - d_3, -\{\alpha + \gamma\} M_0 + d_3 + \eta\}, -\sqrt{(a - b - d_1 - g)^2 + 4ab}\) and \(r - d_2 - (r + \alpha + \gamma) M_0\).

Therefore, all the eigenvalues of the Jacobian matrix are negative if \(\nu_1 < \frac{d_3}{M_0}\) and \(\gamma > \frac{r - d_2}{M_0} - (r + \alpha)\). This gives the following lemma:

Lemma 3.1. The system (2) is locally asymptotically stable at \(E_0\) if \(\nu_1 < \frac{d_3}{M_0}\) and \(\gamma > \gamma^*\), where \(\gamma^* = \frac{r - d_2}{M_0} - (r + \alpha)\).

The existence of \(E_1\) implies the instability at \(E_0\). At \(E_1\) the eigenvalues of the Jacobian matrix of the system (2) are \(-\sqrt{(a - b - d_1 - g)^2 + 4ab}, r - d_2 - (r + \alpha + \gamma) M_0 - AW_1, -\{\alpha + \gamma\} M_0 + d_3 + \eta\) and \(\nu_1 M_0 - d_3 - 2d_4 W_1\). Therefore, all the eigenvalues of the Jacobian matrix are negative if \(\nu_1 > \frac{d_3}{M_0}\) and \(\gamma > \gamma^* = \frac{\lambda d_2}{r^2} \left( \nu_1 - \frac{d_3}{M_0} \right)\).

This gives the following lemma:

Lemma 3.2. The system (2) is locally asymptotically stable at \(E_1\) if \(\nu_1 > \frac{d_3}{M_0}\) and \(\gamma > \gamma_*\), where \(\gamma_* = \gamma^* - \frac{\lambda}{4} \left( \nu_1 - \frac{d_3}{M_0} \right)\).
From lemma (3.1) and (3.2) it follows that, high macroalgal toxicity leads to the elimination of corals. Further, $\gamma^* > \gamma_*$ implies that with higher rate of macroalgae-mediated release of the pathogens in the system, corals become less tolerant to macroalgal toxicity.

**Lemma 3.3.** If $\alpha > a + \frac{b}{M_0}, \nu_1 < \frac{d_3}{M_0}$ and $g < \frac{(a-a)M_0-b}{M_0}$, the system (2) undergoes a transcritical bifurcation at $E_0$ when $\gamma$ crosses $\gamma^*$.

**Proof.** At $\gamma = \gamma^*$ the Jacobian matrix of the system (2) at $E_0$ is

$$J_0 = \begin{pmatrix}
-\sqrt{(a-b-d_1-g)^2+4ab} & (a-g-a)M_0-b & (a-g-a)M_0-b & 0 \\
0 & 0 & -(a+\gamma^*)M_0-d_2-\eta & \nu_1 M_0-d_1 \\
0 & 0 & 0 & 0 \\
\nu_1 (\nu_1 M_0-d_3) & 0 & 0 & d_3-\nu_1 M_0 \\
\end{pmatrix}$$

Therefore, the zero eigenvalue of the Jacobian matrix is simple.

Let $U_0$ and $V_0$ be the eigenvectors corresponding to the zero eigenvalue for $J_0$ and $J_1^T$ respectively.

Then we obtain $U_0 = (u_0 \ 1 \ 0 \ 0)^T$ and $V_0 = (0 \ 1 \ 0 \ 0)^T$, where

$$u_0 = \frac{(a-a)M_0-b}{\sqrt{(a-b-g-d_1)^2+4ab}}$$

Let us express the system (2) in the form $\dot{X} = f(X; \gamma)$, where

$$X = \begin{pmatrix} M \ C_S \ C_I \ W \end{pmatrix}^T$$

and

$$f(X; \gamma) = \begin{pmatrix} f_1 \ f_2 \ f_3 \ f_4 \end{pmatrix}^T$$

Then $V_0^T f_\gamma(E_0; \gamma^*) = 0$ and so no saddle-node bifurcation occurs at $E_0$ when $\gamma$ crosses $\gamma^*$.

Since $Df_\gamma(E_0; \gamma^*) U_0 = (0 -M_0 \ 0 \ 0)^T$, it follows that $V_0^T [Df_\gamma(E_0; \gamma^*) U_0]$ is the Jacobian matrix of the system (2) at $E_0$ when $\gamma$ crosses $\gamma^*$. Therefore, the zero eigenvalue of the Jacobian matrix is simple.

**Lemma 3.4.** If $\alpha > a + \frac{b}{M_0}, \nu_1 < \frac{d_3}{M_0}$ and $g < \frac{(a-a)M_0-b}{M_0}$, the system (2) undergoes a transcritical bifurcation at $E_1$ when $\gamma$ crosses $\gamma_*$.

**Proof.** At $\gamma = \gamma_*$ the Jacobian matrix of the system (2) at $E_1$ is

$$J_1 = \begin{pmatrix}
-\sqrt{(a-b-d_1-g)^2+4ab} & (a-g-a)M_0-b & (a-g-a)M_0-b & 0 \\
0 & 0 & -(a+\gamma_*)M_0-d_2-\eta & \nu_1 M_0-d_1 \\
0 & 0 & 0 & 0 \\
\nu_1 (\nu_1 M_0-d_3) & 0 & 0 & d_3-\nu_1 M_0 \\
\end{pmatrix}$$

Therefore, the zero eigenvalue of the Jacobian matrix is simple.

Let $U_1$ and $V_1$ be the eigenvectors corresponding to the zero eigenvalue for $J_1$ and $J_1^T$ respectively.
If $V_u \gamma$ then there is a change in the stability of the system through transcritical bifurcation. According to Lemma 3.5, it follows that the system (2) undergoes a transcritical bifurcation at $u = \frac{\lambda(\nu_1 M_0 - d_3)}{d_4((\alpha + \gamma) M_0 + d_2 + \eta)}$ and $u_2 = \frac{\lambda(\nu_1 M_0 - d_3)}{d_4((\alpha + \gamma) M_0 + d_2 + \eta)}$.

We obtain $V_1^T f_2(M_0, 0, W_1; \gamma^*) = 0$ and so no saddle-node bifurcation occurs at $E_1$ when $\gamma$ crosses $\gamma^*$.

We have $Df_\gamma(E_1; \gamma^*)U_1 = (0 - M_0 - M_0 u_2 0)^T$, and so $V_1^T[Df_\gamma(E_1; \gamma^*)U_1] = -M_0 < 0$.

Now, we have

$$D^2f(E_1; \gamma^*)(U_1, U_1) = \begin{pmatrix} -2au_1^2 + 2u_1(1 + 2u_2)(\alpha - g - a) - 2gM_0(1 + u_2)^2 \\ -2 \{(r + \alpha + \gamma)u_1 + r(1 + u_2) + \lambda u_3 \} \\ -2 \{(a + \gamma^*)u_1 u_2 - \lambda u_3 \} \\ 2u_3(\nu u_2 - d_4 u_3) \end{pmatrix}$$

This gives $V_1^T[D^2f(E_1; \gamma^*)(U_1, U_1)] = \xi$, where

$$\xi = -2 \left(1 + \frac{\lambda(\nu_1 M_0 - d_3)}{d_4((\alpha + \gamma) M_0 + d_2 + \eta)} \right) \left[ \frac{(r + \alpha + \gamma)(\alpha - a - g)M_0 - b}{d_4((\alpha - g - d_1)^2 + 4ab)} + r \right]$$

$$- \frac{2\lambda}{d_4} \left[ \frac{\nu_1(\alpha - a - g)M_0 - b}{d_4((\alpha - g - d_1)^2 + 4ab)} + \nu_2 \frac{\lambda(\nu_1 M_0 - d_3)}{d_4((\alpha + \gamma) M_0 + d_2 + \eta)} \right]$$

If $\alpha > a + b \frac{M_0}{M_6}$ and $g < \frac{(a - a) M_0 - b}{M_6}$ hold, then we have $V_1^T[D^2f(E_1; \gamma^*)(U_1, U_1)] < 0$.

Therefore, if $\alpha > a + b \frac{M_0}{M_6}$, $\nu_1 > \frac{d_3}{M_6}$ and $g < \frac{(a - a) M_0 - b}{M_6}$ is satisfied, by Sotomayor theorem [28] it follows that the system (2) undergoes a transcritical bifurcation at $E_1$ when $\gamma$ crosses $\gamma^*$. □

Thus, with low macroalgae-mediated release of the pathogens in the system and high macroalgal growth on corals, if the grazing intensity of herbivores becomes low, then there is a change in the stability of the system through transcritical bifurcation at $\gamma = \gamma^*$. The Jacobian $J_2 = J(E_2)$ of the system (2) evaluated at $E_2$ is

$$J_2 = \begin{pmatrix} f_{M|E_1}^1 & f_{C|E_1}^1 & f_{C|E_1}^1 & 0 \\ -r + \alpha + \gamma & C_{S_1} & -rC_{S_1} & -rC_{S_1} \\ 0 & 0 & -\{(\alpha + \gamma)M_1 + d_2 + \eta \} & -C_{S_1} \\ 0 & 0 & 0 & \nu_1 M_1 - d_3 \end{pmatrix}$$

The two roots of the characteristic equation of the Jacobian $J_2$ of the system (2) are $\nu_1 M_1 - d_3$ and $-\{(\alpha + \gamma)M_1 + d_2 + \eta \}$, the remaining two roots are given by the equation

$$\mu^2 + \mu \left( r C_{S_1} - f_{M|E_1}^1 \right) + C_{S_1} \left( r + \alpha + \gamma \right) f_{C|E_1}^1 - r C_{S_1} f_{M|E_1}^1 = 0.$$ 

Therefore, all the eigenvalues of the Jacobian matrix $J_2$ are negative if $\nu_1 < \frac{d_3}{M_1}$, $r C_{S_1} > f_{M|E_1}^1$ and $(r + \alpha + \gamma) f_{C|E_1}^1 > r f_{M|E_1}^1$. This gives the following lemma:

**Lemma 3.5.** The system (2) is locally asymptotically stable at $E_2$ if $\nu_1 < \frac{d_3}{M_1}$, $r C_{S_1} > f_{M|E_1}^1$ and $(r + \alpha + \gamma) f_{C|E_1}^1 > r f_{M|E_1}^1$. 

The Jacobian $J_3 \equiv J(E_3)$ of the system (2) evaluated at $E_3$ is

$$J_3 = \begin{pmatrix}
-(r + \alpha + \gamma)CS_2 & f_{CS_{|E_3}} & f_{C_{I_{|E_3}}} & 0 \\
0 & -rCS_2 & -rCS_2 & -\lambda CS_3 \\
\nu_1 W_2 & \lambda W_2 & -\{(\alpha + \gamma)M_1 + d_2 + \eta\} & \lambda CS_2 \\
0 & \nu_2 W_2 & -d_4 W_2 & -d_4 W_2
\end{pmatrix}$$

The characteristic equation of the Jacobian $J_3$ of the system (2) is

$$\mu^4 + \mu^3 Q_1 + \mu^2 Q_2 + \mu Q_3 + Q_4 = 0,$$

where

$$Q_1 = rCS_2 + dW_2 + \{(\alpha + \gamma)M_1 + d_2 + \eta\} - f_{M_{|E_3}}^1,$$

$$Q_2 = CS_2\{W_2(rd_4 - \lambda \nu_2) + r + \alpha + \gamma \} + \{(\alpha + \gamma)M_1 + d_2 + \eta\}(rCS_2 + dW_2 - f_{M_{|E_3}}^1) - f_{M_{|E_3}}^1(rCS_2 + dW_2),$$

$$Q_3 = \{(\alpha + \gamma)M_1 + d_2 + \eta\}dW_2 + rCS_2(1 - f_{M_{|E_3}}^1) + (r + \alpha + \gamma)CS_2 f_{C_{I_{|E_3}}}^1 - \lambda CS_2 W_2\{\nu_2 (rCS_2 + W_2) + r\nu + dW_2 \},$$

$$Q_4 = CS_2 W_2\{(r + \gamma)M_1 + d_2 + \eta\}\{\nu_2 (rCS_2 + W_2) + r\nu + dW_2 \} + \lambda f_{C_{I_{|E_3}}}^1(r\nu + dW_2 - \nu_2 W_2) + \lambda (\nu_1 (rCS_2 + W_2 - rCS_2)) + (r + \alpha + \gamma)\}W_2(dW_2 - \nu_2 W_2).$$

If $Q_i > 0$ ($i = 1, \ldots, 4$) and $Q_1 Q_2 Q_3 > Q_3^2 + Q_1^2 Q_4$ hold, all the eigenvalues of the Jacobian matrix $J_3$ become negative and consequently, the system (2) will be locally asymptotically stable at $E_3$. Due to the complexity in the algebraic expressions involved, it is difficult to interpret the results in ecological terms, however, numerical simulations are used to illustrate the dynamical behaviour of the system about $E_3$.

The characteristic equation of the Jacobian of the system (2) evaluates at $E^*$ is

$$F(\mu) = \mu^4 + \mu^3 Q_1 + \mu^2 Q_2 + \mu Q_3 + Q_4 = 0,$$

where

$$Q_1^* = rCS_2^* + dW^* + \{(\alpha + \gamma)M^* + d_2 + \eta\} - f_{M_{|E^*}}^1,$$

$$Q_2^* = CS_2^*\{W^*(rd_4 - \lambda \nu_2) + r + \alpha + \gamma \} + \{(\alpha + \gamma)M^* + d_2 + \eta\}(rCS_2 + dW^* - f_{M_{|E^*}}^1) - f_{M_{|E^*}}^1(rCS_2 + dW^*) + \lambda f_{C_{I_{|E^*}}}^1(r\nu + dW^*) - \lambda CS_2 W^*\{\nu_2 (rCS_2 + W^*) + r\nu + dW^* \},$$

$$Q_3^* = \{(\alpha + \gamma)M^* + d_2 + \eta\}dW^* + rCS_2^*(1 - f_{M_{|E^*}}^1) + (r + \alpha + \gamma)CS_2 f_{C_{I_{|E^*}}}^1 - \lambda CS_2 W^*\{\nu_2 (rCS_2 + W^*) + r\nu + dW^* \} + \lambda f_{C_{I_{|E^*}}}^1(r\nu + dW^* - \nu_2 W^*) + \lambda (\nu_1 (rCS_2 + W^*) - \lambda CS_2 W^*) + (r + \alpha + \gamma)\}W^*(dW^* - \nu_2 W^*).$$

Therefore, for local asymptotic stability of the system at $E^*$ we must have $Q_i^* > 0$ ($i = 1, \ldots, 4$) and $Q_1^* Q_2^* Q_3^* > Q_3^{*2} + Q_1^2 Q_4^*$. We will study the saddle-node bifurcation of the system (2) taking $\lambda$ as a bifurcation parameter. Solving $Q_1^*(\lambda) = 0$, the critical value of $\lambda$, say, $\lambda = \lambda^*$ can be obtained. Also, if $Q_2^*(\lambda^*) \neq 0$, then the characteristic equation $F(\mu) = 0$ has a simple zero eigenvalue.

Let $V^*$ and $W^*$ are the eigenvectors corresponding to the zero eigenvalue for $J^*$ and $J^{*T}$ respectively.

Then we obtain $V^* = \begin{pmatrix} 1 & v_1^* & v_2^* & v_3^* \end{pmatrix}^T$ and $W^* = \begin{pmatrix} w_1 & w_2 & w_3 \end{pmatrix}^T$, where

$$v_1^* = \frac{-\lambda W_2 - rCS_2 - dW_2}{f_{C_{I_{|E^*}}}}, \quad v_2^* = \frac{rdW_2 - rCS_2}{(r\nu + dW_2)}\frac{f_{C_{I_{|E^*}}}(\nu_2 r + dW_2 \nu_2 + dW_2 \nu_2)}{f_{C_{I_{|E^*}}}(\nu_2 r + dW_2 \nu_2 + dW_2 \nu_2)} - \frac{rdW_2 - rCS_2}{d_4}, \quad v_3^* = \nu_1 + \nu_2 W_2.$$
implies that a saddle-node bifurcation occurs at $E^w$. Therefore, when the disease transmission rate is increased through $\lambda^*$, the interior equilibria undergo mutual annihilation, leading to a catastrophic shift of regime to a macroalgae-dominated and coral-depleted steady state.

Now, we will study the Hopf-bifurcation of the system (2) at $E^*$, taking $g$ as the bifurcation parameter.

Solving $Q_1^2Q_2Q_3^2 - Q_3^2 - Q_1^2Q_3^4 = 0$, the critical value of $g$ can be obtained, say, $g = g^*$.

At $g = g^*$, the characteristic equation is $\left(\mu^2 + \frac{Q_1}{Q_3}\right)\left(\mu^2 + \mu Q_1^3 + \frac{Q_1^2}{Q_3}\right) = 0$.

For $g = g^*$, let $\mu_i (i = 1, \ldots, 4)$ be the roots of the characteristic equation with the pair of purely imaginary roots as $\mu_1 = i\omega_0$ and $\mu_2 = \bar{\mu}_1$, where $\omega_0 = \frac{\sqrt{Q_3(q^*)}}{Q_1(q^*)}$.

Now, if $\mu_3$ and $\mu_4$ are not real, then $Re\mu_3 = \frac{1}{2} (\mu_3 + \mu_4) = -\frac{1}{2}Q_1(q^*) < 0$.

If $\mu_3$ and $\mu_4$ are real roots, then $\mu_3 + \mu_4 < 0$ and $\mu_3\mu_4 = \frac{Q_3(g^*)}{\omega_0^2} > 0$ implies $\mu_3, \mu_4 < 0$.

Therefore, only a pair of eigenvalues of the characteristic equation $F(\mu) = 0$ at $g = g^*$ are purely imaginary.

Let $\phi(g) = f_1(g) - f_2(g)$, where $f_1(g) = Q_1^4(g)Q_2^3(g)Q_3(g)$, and $f_2(g) = Q_1^2(g) + Q_1^3(g)Q_3(g)$.

Since $\phi$ is continuously differentiable function of $g$, there exists an open interval $(g^* - \epsilon, g^* + \epsilon)$ such that $\mu_i(g) = p_i(g) + iq_i(g), \forall g \in (g^* - \epsilon, g^* + \epsilon)$.

To verify the conditions for Hopf-bifurcation at $g = g^*$, we put $p + iq$ in $F(\mu) = 0$ and then by separating the real and imaginary parts, we obtain
\[
p^4 + p^3Q_1^* + p^2(Q_2^* - 6q^2) + p(Q_3^2 - 3q^2) + (q^4 + Q_3^*) = 0,
q^2(Q_1^* + 4p) = 4p^3 + 3p^2Q_1^* + 2pQ_2^* + Q_4^*.
\]
Substituting the value of $q^2$ we get
\[
p^4 + p^3Q_1^* + p^2(Q_2^* - 6h(p)) + p(Q_3^2 - 3h(p)) + (h(p))^2 + Q_4^* = 0,
\]
where $h(p) = 4p^2 + 3p^2Q_1^* + 2pQ_2^* + Q_4^* + \frac{Q_3^2}{Q_1^*} + 4Q_4^*.$

Differentiating $F(\mu) = 0$ with respect to $g$ and then putting $g = g^*$, we get
\[
\left(\frac{dg}{dg}\right)_{g=g^*} = \frac{Q_1^3(g^*)Q_2^3(g^*) - 3Q_1(g^*)Q_2^2(g^*) + Q_4^2(g^*)}{2(3Q_1^2(g^*)Q_2^3(g^*) - Q_1^3(g^*)Q_2^2(g^*) - 6Q_2^2(g^*))} = 0.
\]
Therefore, the system (1) undergoes a Hopf-bifurcation at $E^*$ when $g$ crosses $g^*$ if
(i) $Q_1^i(g^*) > 0, i = 1, \ldots, 4$
(ii) $Q_1^i(g^*)Q_2^i(g^*) > Q_3^i(g^*)$
(iii) $Q_1^i(g^*)Q_2^i(g^*)Q_3^i(g^*) > Q_3^2(g^*) + Q_1^2(g^*)Q_3^i(g^*)$
(iv) $Q_1^2(g^*)Q_3^2(g^*) \neq 3Q_1(g^*)Q_5^2(g^*) - Q_1^2(g^*) \left(\frac{dQ_1}{dg} + \frac{dQ_1}{dg}\right)_{g=g^*}$ and $Q_1^i(g^*) \neq 0$.

Again, the condition (iv) is equivalent to $\frac{d\phi(g)}{dg} \mid_{g=g^*} \neq 0$. Thus, by using numerical methods, condition (iii) can be verified by showing that the curves $y = f_1(g)$ and $y = f_2(g)$ intersect at $g = g^*$, whereas the condition (iv) can be verified by...
showing that the tangent to the curve \( y = \phi(g) \) at \( g = g^* \) is not parallel to \( g \) axis.

The grazing rate \( g \) depends on the abundance of herbivores and is thus subject to variation with changes in available refuge and food abundance. To identify the impact of changes in grazing intensity on coral cover, in Fig. 2, we plot the solutions of the nullcline equations in the \( C - g \) plane \( (C = C_S + C_I) \), yielding a bifurcation diagram. Coordinates of stable interior equilibria are shown in blue, stable boundary equilibria \( E_0 \) and \( E_2 \) are shown in black and cyan respectively and unstable equilibria are shown in red. The region \( I \) represents bistability at \( E_0 \) and \( E_1 \) for \( 0 \leq g < 0.1739 \), depending upon the initial conditions. In this region, the system will ultimately arrive at a macroalgae-dominated and coral-depleted steady state corresponding to low levels of grazing. Increase of grazing intensity determines a tristable region \( II \) with the three possible stable states \( E_0, E_1 \) and \( E_2 \) for \( 0.1739 < g < 0.3588 \). The bistable region \( III \), for \( 0.3588 < g < 0.5367 \), determines two possible steady states \( E_1 \) and \( E_2 \), depending upon the initial conditions. Once the grazing intensity surpasses the threshold \( g = 0.5367 \), apart from the two possible steady states \( E_1 \) and \( E_2 \), the system can become oscillatory around the interior equilibrium \( E^* \) in the region represented by \( IV \), for \( 0.5367 < g < 0.5442 \). Further increase of the grazing intensity \( (0.5442 < g < 0.637) \) generates the tristable region \( V \) where the system have the possible steady states \( E_1, E_2 \) and \( E^* \). For \( g > 0.637 \), macroalgae and coral coexists in either one of the two stable states \( E_2 \) and \( E^* \) in the region \( VI \).

From Fig. 2(b) we see that for \( g > g^* = 0.5442 \), the real part of all the eigenvalues of the Jacobian of the system at \( E^* \) are negative and so \( E^* \) is a stable focus. The stability of the system changes when \( g \) is lowered through \( g^* \). For \( 0.5367 < g < g^* \), the real part two eigenvalues of the Jacobian of the system at \( E^* \) becomes positive, representing the instability of the system at \( E^* \). Also, \( E^* \) ceased to exist for \( g < 0.5367 \). From Fig. 2(c) we observe that \( f_1(g) \) and \( f_2(g) \) intersect at \( g^* \), indicating that the system changes its stability when the parameter crosses the threshold \( g^* \). More specifically, for \( g > g^* \) we see that \( f_1(g) > f_2(g) \), satisfying Routh Hurwitz condition and therefore the system is locally asymptotically stable at \( E^* \). Moreover, we observe that the tangent to \( \phi(g) \) at \( g = g^* \) is not parallel to \( g \) axis, satisfying the condition \( \frac{dg}{dg}(g=g^*) \neq 0 \). Therefore, the system undergoes a Hopf bifurcation when \( g \) crosses \( g^* \).

From Fig. 2(d) it follows that with low rate of microbial infection (viz. \( \lambda = 0.05 \)), coexistence of macroalgae and corals can be possible even with low grazing intensity of herbivores. From figure 3(a) we see that at \( \lambda = 0.05 \) and \( g = 0.342 \), the Jacobian of the system at \( E^* \) has a pair of purely imaginary eigenvalues. Fig. 3(b) verifies that for \( \lambda = 0.05 \), the system undergoes a Hopf bifurcation at \( E^* \) when \( g \) crosses \( g = 0.342 \). Also, for \( g < 0.3415, E^* \) does not exist. Fig. 3(c) represents a two-parameter bifurcation diagram with \( g \) and \( \lambda \) as active parameters, representing a cusp point at \( (g,\lambda) = (0.2565,0.3536) \) and a Bogdanov-Takens (BT) point at \( (g,\lambda) = (0.2905,0.0396) \) on the saddle-node curve. The Hopf curve intersects the saddle-node curve at BT which is a neutral saddle point. Further, from Figs. 4(a) and 4(b) it follows that \( E^* \) does not exist in absence of microbial infection \( (\lambda = 0) \) on corals or with high disease-induced death rate (\( \eta = 0.5 \)) of corals.

To identify the impact of macroalgal toxicity on coral cover, in Fig. 5(a), we plot the solutions of the nullcline equations in the \( C - \gamma \) plane, yielding a bifurcation diagram. The region \( VII \) represents tristability at \( E_1, E_2 \) and \( E^* \) for \( 0 \leq \gamma < 1.001 \).
for all non-negative initial conditions. For $1.001 < \gamma < 1.678$, the system becomes bi-stable at $E_1$ and $E_2$ in the region $VIII$ where $E^*$ loses its stability. From Fig. 5(b) we see that at $\gamma = 1.001$, two eigenvalues of the Jacobian of the system at $E^*$ becomes purely imaginary with a change in the stability of $E^*$. Also, Fig. 5(c) it follows that $E^*$ undergoes a Hopf bifurcation when $\gamma$ crosses $\gamma = 1.001$. For $1.678 < \gamma < 17.5$, the system becomes tristable at $E_0$, $E_1$ and $E_2$ in the regions $IX, X$ and $XI$. It is seen that a change of stability st $E_0$ occurs when $\gamma$ crosses $\gamma^* = 1.678$. To determine the nature of bifurcation at $\gamma = \gamma^*$ and to verify the analytical results, we use numerical simulations.

At $\gamma = \gamma^* = \frac{rM_0}{E_0} - (r + \alpha) = 1.678$, we have $E_0 = (0.1332, 0, 0, 0)$ and

$$J_0 = \begin{pmatrix} -0.1401 & -0.1741 & -0.1741 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -0.4868 & 0 \\ 0 & 0 & 0 & 0.0033 \end{pmatrix}$$
Coral cover (C) + (C)

Real part of 1st eigenvalue

φ (g) = f_1(g) - f_2(g), f_1(g) < f_2(g)

This implies, the system undergoes a transcritical bifurcation at E_0 when γ crosses γ* = 1.678.

Figure 3. (a) Eigenvalues for the interior equilibrium E* as functions of g for λ = 0.05. (b) The relative positions of f_1(g), f_2(g) and φ(g) showing that a Hopf bifurcation occurs when the two curves intersect at g = 0.342. (c) Two parameter bifurcation diagram with g and λ as active parameters. (The saddle-node curve is in blue, Hopf curve is in red and codimension one bifurcation curve with λ = 0.05 is in green)

has a simple zero eigenvalue. The eigenvectors corresponding to the zero eigenvalue for J_0 and J_0^T are U = ( -1.243 1 0 0 )^T and V = ( 0 1 0 0 )^T respectively. This gives V^T f_1(E_0; γ*) = 0, V^T [Df_1(E_0; γ*)] U = -0.1332 < 0 and V^T [D^2 f(E_0; γ*)](U, U) = 4.6869 > 0.
Further, for high macroalgal toxicity (viz. $\gamma > 17.5$), both $E_2$ and $E^*$ become nonexistent and the system is bistable at $E_0$ and $E_1$ in the region $X_{II}$.

From Figs. 6(a) and 6(b) it follows that the increase in grazing intensity of herbivores increases the tolerance of macroalgal toxicity on corals, however, from Figs. 6(c) and 6(d) it is observed that the change in the transmission rate of microbial infection on corals exhibit no apparent changes in the tolerance level of macroalgal toxicity on corals. Also, from Fig. 7(a) we can conclude that the system is coral-dominated if the rate of macroalgal toxicity is low together with high grazing intensity of herbivores. Fig. 7(b) shows that with low rate of transmission of microbial disease, the system is coral-dominated even with high macroalgal toxicity.

To identify the effect of WPD-II transmission rate on coral cover, in Fig. 8(a), we plot the solutions of the nullcline equations in the $C - \lambda$ plane, yielding a bifurcation diagram. The region $X_{III}$ represents bistability at $E_1$ and $E_2$ for $0 \leq \lambda < 0.0364$ for all non-negative initial conditions. From Fig. 8(b) we see that at $\lambda = 0.0364$, the Jacobian of the system at $E^*$ has a simple zero eigenvalue. The system (2) has a stable focus at $E^*$ for $0.0364 < \lambda < 0.0513$ and has a stable node at $E^*$ for $\lambda > 0.0513$. Also, $E^*$ ceased to exist for $\lambda < 0.0364$. It is seen that a change of stability at $E^*$ occurs when $\lambda$ crosses $\lambda = 0.0364$.

At $\lambda = 0.0364$, we have $E^* = (0.0036, 0.3729, 0.0836, 1.5442)$ and

$$J^* = \begin{pmatrix}
-0.7504 & -0.0148 & -0.0148 & 0 \\
-0.2797 & -0.2051 & -0.2051 & -0.0136 \\
-0.0167 & 0.0562 & -0.2507 & 0.0136 \\
0.1544 & 0 & 0.4633 & -0.0154
\end{pmatrix}$$

has a simple zero eigenvalue. We obtain $U = \begin{pmatrix} 1 & -26.1515 & 24.4822 & 744.4646 \end{pmatrix}^T$, $V = \begin{pmatrix} 1.0594 & 1 & -3.9601 & 0.8789 \end{pmatrix}^T$, $V^Tf_\lambda(E^*; \lambda = 0.0364) = -2.8561 \neq 0$, $V^T[Df_\lambda(E^*; \lambda = 0.0364)U] = -0.00117$ and $V^T[D^2f(E^*; \lambda = 0.0364)(U,U)] = -9.7426 \times 10^9 < 0$.

This implies, the system undergoes a saddle-node bifurcation at $E^*$ when $\lambda$ crosses $\lambda = 0.0364$.

The system is tristable at $E_1$, $E_2$ and $E^*$ in the region $X_{IV}$ for $0.0364 < \lambda < 0.4333$. From Fig. 9(b) we see that at $\lambda = 0.4333$ and $\lambda = 0.6216$, the Jacobians of
Figure 5. (a) Bifurcation diagram of $\gamma$ versus the equilibrium value of coral cover. (b) Eigenvalues for the interior equilibrium $E^*$ as functions of $\gamma$. (c) The relative positions of $f_1(\gamma)$, $f_2(\gamma)$ and $\phi(\gamma)$ showing that a Hopf bifurcation occurs when the two curves intersect at $\gamma = 1.001$.

the system at $E^*$ has a pair of imaginary eigenvalues. For $0.4333 < \lambda < 0.6216$, the real part of two eigenvalues of the Jacobian of the system at $E^*$ becomes positive, representing the instability of the system at $E^*$ in the region XV of Fig. 8(a). Also, $E^*$ ceased to exist for $\lambda > 0.63$. In Fig. 9(c) we use numerical simulations to very that the system undergoes Hopf bifurcations at $\lambda = 0.4333$ and $\lambda = 0.6216$. For $\lambda > 0.6216$, the system becomes bistable at $E_1$ and $E_2$.

To identify the effect of WPD-II induced coral death, in Fig. 10(a), we plot the solutions of the nullcline equations in the $C - \eta$ plane, yielding a bifurcation diagram. The system is tristable at $E_1$, $E_2$ and $E^*$ for $0 < \eta < 0.4377$. From Fig. 10(b) we see that at $\eta = 0.4377$, the Jacobian of the system at $E^*$ has a simple
Toxicity rate ($\gamma$)  

| Value | 0 | 1.383 | 5.025 | 7.905 | 10 |
|-------|---|--------|--------|--------|----|

Coral cover ($C + C_I$)  

| Value | 0 | 0.1 | 0.2 | 0.3 | 0.4 | 0.5 | 0.6 |
|-------|---|-----|-----|-----|-----|-----|-----|

Figure 6. Bifurcation diagrams of $\gamma$ versus the equilibrium value of coral cover for (a) $g = 0.4$, (b) $g = 1$, (c) $\lambda = 0$ and (d) $\lambda = 1$.

Figure 7. Coexistence regions in (a) $\gamma - g$ parameter space, (b) $\gamma - \lambda$ parameter space and (c) $\eta - g$ parameter space (Blue indicates coral-dominated $E^*$, red indicates macroalgae-dominated $E^*$ and green indicates coral-dominated $E_2$).

zero eigenvalue. Also, $E^*$ ceased to exist for $\eta > 0.4377$. It is seen that a change of stability at $E^*$ occurs when $\eta$ crosses $\eta = 0.4377$. 
Figure 8. (a) Bifurcation diagram of $\lambda$ versus the equilibrium value of coral cover. (b) Eigenvalues for the interior equilibrium $E^*$ as functions of $\lambda$.

At $\eta = 0.4377$, we have $E^* = (0.0061, 0.3138, 0.0490, 1.5296)$ and

\[
J^* = \begin{pmatrix}
-0.7504 & -0.0148 & -0.0148 & 0 \\
-0.2797 & -0.2051 & -0.2051 & -0.0136 \\
0.0167 & 0.0562 & -0.2507 & 0.0136 \\
0.1544 & 0 & 0.4633 & -0.0154
\end{pmatrix}
\]

has a simple zero eigenvalue. We obtain $U = \begin{pmatrix}1 & -27.0799 & 2.2282 & 76.8473 \end{pmatrix}^T$, $V = \begin{pmatrix}1.564 & 1 & -2.3818 & 11.8521 \end{pmatrix}^T$, $V^T f_\eta(E^*; \eta = 0.4377) = 2.3818 \neq 0$, $V^T[D_f(E^*; \eta = 0.4377)U] = -33.0598$ and $V^T[D^2 f(E^*; \lambda = 0.0364)(U, U)] = -1.3999 \times 10^9$.

This implies, the system undergoes a saddle-node bifurcation at $E^*$ when $\eta$ crosses $\eta = 0.4377$.

Figs. 11(a – c) show the effect of WPD-II induced death with different grazing intensity of herbivores. It is observed that the system exhibits discontinuous (hysteretic) threshold response with the increase of disease-induced death for $g > 0.4$ (cf. Figs. 11(a)&(b)), while at $g = 0.4$, the response is abrupt with continuous threshold, $\eta$ being an active parameter (cf. Fig. 11(c)).

To identify the effect of macroalgae-mediated growth of FLP on coral cover, in Fig. 12(a), we plot the solutions of the nullcline equations in the $C - \nu_1$ plane, yielding a bifurcation diagram. It is observed that for $0 < \nu_1 < 0.751$, the system is stable at either disease-free coral-macroalgae coexistence state $E_2$, or at the interior equilibrium $E^*$. Increase in the rate of macroalgae-mediated pathogen-growth increases the persistence of FLP in the system. It is seen that the system is tristable at $E_1, E_2$ and $E^*$ for $0.751 < \nu_1 < 0.1862$. For $\nu_1 > 0.1862$, the interior equilibrium ceased to exist followed by a sudden change in transition when $\nu_1$ crosses $\nu_1 = 0.1862$. Since the growth of macroalgae is controlled by the grazers, in Fig. 12(b) we plot a bifurcation diagram of $\nu_1$ versus equilibrium value of coral cover for different grazing rates of herbivores. It is observed that the increase of grazing rate of herbivores increases the tolerance of the coral-dominated steady state to the proliferation of FLP and therefore, increases the resilience of coral-dominated
regime even with high rate of macroalgae-induced pathogen growth measured by the threshold differences of saddle-node bifurcating point (LP) and a transcritical bifurcating point (BP).

4. **Discussion.** An eco-epidemiological model is considered to study the dynamics of coral reef benthic system in presence of a microbial infection, WPD-II on corals. We have modified our previous models [4, 5] by introducing macroalgal toxicity on corals and by considering the dynamics of pathogens. Our model assumes the transmission of WPD-II through free living pathogens shed by infected corals at a rate proportional to the product of susceptible coral cover and pathogen concentration in the environment. It is also assumed that macroalgae influence in the proliferation of pathogens in the system. We first perform equilibrium and stability analysis of the system and found that the system is capable of exhibiting the existence of multiple
stable configurations of the community under the same environmental conditions with saddle-node, transcritical and Hopf bifurcations. The system with stability at the interior equilibrium exhibits a sudden change of transition associated with saddle-node bifurcation when the WPD-II transmission rate becomes low. This supports our previous claim \[5\] that the system will be coral-dominated and stable with low rate of infection. However, in our previous study \[5\], with low contagious infection rate we found no noticeable change in the dynamics of the system even with high rate of non-contagious infection. The current paper demonstrates a Hopf bifurcation followed by coral-depleted steady state with high rate of infection. In this case, the extinction of corals is due to natural fluctuations which become very likely when the oscillation drives the coral population to small size. Increase in macroalgal toxicity and macroalgae-mediated pathogen growth also induces catastrophic
changes in the community structure. In each of the cases, increase of grazing intensity of herbivores increases the resilience of the coral-dominated regime. Further, analytical and numerical simulations demonstrate the following conclusions:

(i) The system is macroalgae-dominated with low macroalgal grazing rate by herbivores. Increase of grazing intensity by herbivores increases the resilience of the coral-dominated regime, justifying the observations of Blackwood et al. [8] and Mumby et al. [23].

(ii) The system becomes macroalgae-dominated with the increase of WPD-II transmission on corals. In this case, the increase of herbivory increases the resilience of the coral-dominated regime. With low rate of infection, the system is coral-dominated and stable even with low grazing intensity.

(iii) With low macroalgal toxicity, the coexistence equilibrium becomes stable. Increase of grazing intensity increases the tolerance of macroalgal toxicity of the coexistence stable state.

(iv) With high rate of transmission of WPD-II, corals become depleted from the system even with low macroalgal toxicity. Decrease in the WPD-II transmission rate on corals can generate a coral-dominated regime even with moderately high rate of macroalgal toxicity. Also, it is observed that with high grazing rate of herbivores and high rate of virulence, corals dominate the disease free stable coral-macroalgae-coexistence equilibrium.

Throughout the article an attempt is made to search for a suitable way to control the growth of macroalgae and corals and maintain stable coexistence. From analytical and numerical observations, it is seen that the reduction in herbivory reduces the tolerance of macroalgal toxicity and WPD-II infection rate on corals. Higher grazing rate of herbivores reduces macroalgal cover and increases the resilience of the coral-dominated regime.

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