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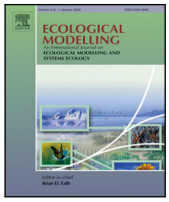

Is toxicity a curse or blessing, or both?—Searching answer from a disease-induced consumer-resource system

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ABSTRACT

Chemical toxins exposed in environments and disease outbreaks are global threats to ecosystems in the present era of the anthropocene. Toxin favors disease progression trivially. However, it is still unclear whether the toxin impacts disease elimination too. Toxin also has a significant role in amplifying the risk of disease-induced consumer extinction. Identification of the extinction vortex and its associated precursors are the two most important pillars for understanding the effect of the toxin on the sustainability of ecosystems. On the other hand, the contribution of toxin as a potential agent for stabilizing a disease-induced consumer-resource system is still unclear. Although disease stabilizes the system in absence of toxicity. In order to address this, we consider a mathematical model of disease transmission in the consumer population where both ecological and epidemiological traits are affected by environmental toxins. The proposed model integrates two compartments (susceptible and infected) for consumers and the resource, where the toxin is incorporated in the form of species body burdens. Apart from the formal stability analysis, we extensively use codim-1 and codim-2 bifurcation through MATCONT software for understanding the different dynamical regimes of disease progression and elimination. These derived regimes will be helpful to raise the alarm and take intervention policies.

1. Introduction

Environmental pollution and disease outbreaks are global threats to ecosystems in the present era of the Anthropocene (Lafferty et al., 2004; Van Bressem et al., 2009; Reckendorf et al., 2023; Trevisan et al., 2022). Toxins released in the environment due to anthropogenic factors have long-drawn consequences on the ecosystem health (Huang et al., 2013, 2015; Garay-Narváez et al., 2013; Banerjee et al., 2021). The emergence of disease outbreaks in many ecosystems is also of utmost concern for ecologists (Lafferty and Kuris, 1999; Lafferty and Holt, 2003; Lafferty et al., 2004; Khan, 1990; Van Bressem et al., 2009). It is well known that environmental toxins can influence the impact of disease spread on the ecosystem, but the manner of it needs to be correctly understood. The toxin may restrict movement and increase the infected host's mortality, thus negatively affecting disease spread. Conversely, the toxin can reduce the host immunity, thus making them more susceptible, which increases disease prevalence (Khan, 1990; Beck and Levander, 2000). For instance, harbor seals readily accumulate toxicants such as polyhalogenated aromatic hydrocarbons (PHAHs) and polychlorinated biphenyls (PCBs) if they feed on herring fish from the more polluted Baltic Sea compared to the less polluted Atlantic Sea. These substances decrease their immunity and make them more prone to infections such as from morbilliviruses, which may lead to mass mortality (Ross et al., 1996; De Swart et al., 1994).

While toxins can affect disease transmission rate, it can also impact host-resource interaction, which in turn can significantly impact disease dynamics (Lafferty and Holt, 2003; De Luna and Hallam, 1987; Banerjee et al., 2019). In fact, the host-resource dynamics have received much attention in recent years regarding disease progression in ecosystems (Hurtado et al., 2014; Hilker and Schmitz, 2008). In this context, it is essential to note that the environmental toxin can influence even life-history traits like growth rate and mortality of both resource and the host. Further, the host (consumers) can accumulate toxins by ingesting resources. This accumulation of toxins higher up the food chain, known as bioaccumulation, may affect the abundance of the host population and, thus, disease prevalence. Mathematical models to understand the effect of toxicity on disease progression based on the above factors, such as transmission, life history traits, etc., are limited (Sinha et al., 2010; Liu et al., 2012; Chauhan et al., 2015; Wang and Ma, 2004).

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Toxicity has been introduced as just additional mortality in these studies. Banerjee et al. (2019) made a seminal attempt to address the gap. However, the scope of the study is limited to a narrow and very specialized *Daphnia*-algae-fungus system.

Moreover, identifying the threshold toxin level above which the consumer population is extinct must be a byproduct of the research output of the above-mentioned dynamical systems. So, this must be an exciting research question that needs to be answered. Further, such threshold toxin level for consumer extinction must depend on the disease's degree of intensity (disease transmission and virulence). There may be multiple precursors of toxin-induced consumer extinction. Disease eradication and the changes in dynamic states are a few examples of such precursors. Determination of extinction vortex and its associated precursors are two most important yardsticks for comprehending the effect of the toxin on disease dynamics. Disease invasion can promote two contrary effects in pursuing its host population to extinction or stabilizing prey-predator cycles (Hilker and Schmitz, 2008). Is this phenomenon also valid for toxin-mediated consumer-resource systems? The plausible answer to this question is a clue in understanding the tolerance level of toxicity in consumer-resource systems.

Although few earlier studies (Freedman and Shukla, 1991; Thomas et al., 1996) incorporated a carrying capacity dependent on exogenous input of toxin, it is challenging to analyze such effect quantitatively. We explore this gap by considering toxin-induced increases in disease transmission in a host-resource system. The resource abundance depends on carrying capacity, which must be affected by an environmental toxin but largely ignored; instead, only toxin-dependent growth rate is assumed (Hallam et al., 1983a,b; Prosnier et al., 2015; Banerjee et al., 2019, 2021). Beverton–Holt growth rate instead of the conventional logistic formulation is the right choice to explain the scenario of toxin-dependent carrying capacity (Thieme, 2003; Huang et al., 2013) and used more recently in predator–prey systems by Huang et al. (2015). We use a similar approach to model the impact of the toxin on the host-resource dynamics.

Based on the above discussions, we set the following interrelated objectives in a questionary format: (1) Does toxicity always trivially favor disease progression, or is it also a game-maker in disease eradication? (2) Can we identify a threshold toxin level above which a sudden collapse of the consumer (host) population occurs? (3) How do the transmission rate and virulence influence the precursors of consumer extinction? (4) Is Hilker's (Hilker and Schmitz, 2008) fundamental implications of disease-induced stabilization under increasing transmission rate still valid in a toxic environment?

First, we describe our model and toxin-mediated response functions in Section 2. Also, we make our model parameters dimensionless and use a quasi-steady state approximation in this section to reduce model complexity. The analytical and simulation techniques to meet the above-mentioned questions are addressed in Section 3. The positivity and boundedness of the solutions and the existence and the stability criterion of the equilibriums are discussed in Appendix B. In Section 4, we first investigate the possible asymptotic states of our system with the help of bifurcation diagrams (Section 4.1). Then, the answers to our proposed question are addressed with plausible explanations in Section 4.2. Finally, our paper is summarized with the conclusions in Section 5.

2. Mathematical model

To find the answers to our objectives, we need to consider a disease-induced consumer-resource model as a testbed. So naturally, the study variables are resource, susceptible and infected consumers and the body burdens of both resource and consumers due to the toxin.

2.1. Model description

We consider a consumer-resource model, where the consumer population is affected by an infectious disease (Hilker and Schmitz, 2008). Let $x(t)$ and $y(t)$ be the concentration of the resource and the consumer biomass, respectively. In the spirit of Hilker and Schmitz (2008), we introduce the disease in the consumer through the compartmental models. So the consumer (host) population can be further segregated into susceptible, $S(t)$, and infected, $I(t)$, classes such that $S(t) + I(t) = y(t)$ (see Fig. 1). The studies (Hilker and Schmitz, 2008; Huang et al., 2013, 2015) assumed several ecological and epidemiological traits in terms of model parameters. For example, ecological traits are resource growth rate, consumers' conversion efficiency, and both species' natural death rate, and the epidemiological trait is the disease transmission rate. These traits must be affected when toxins are present in the system. The best way to capture these effects is through the concept of body burdens of the resource and the consumers (Huang et al., 2013, 2015). Let $u(t)$ and $v(t)$ be the toxin body burden of the resource and consumer species, respectively, which is defined as the ratio of the total toxin in a population to the total biomass concentration (Huang et al., 2015). Note that when we combined disease and toxins in a consumer-resource system, the model parameters indicating traits are the functions of body burdens of respective species. Combining the idea of Hilker and Huang, the disease dynamics under the influence of environmental toxins can be described below:

$$\frac{dx}{dt} = (\beta(x,u) - \mu_1(u))x - \frac{ax}{H+x}y \tag{1a}$$

$$\frac{dS}{dt} = e(v)\frac{ax}{H+x}y - \lambda(v)\frac{SI}{y} - \mu_2(v)S \tag{1b}$$

$$\frac{dI}{dt} = \lambda(v)\frac{SI}{y} - \mu_2(v)I - \eta I \tag{1c}$$

$$\frac{dy}{dt} = \frac{d(S+I)}{dt} = e(v)\frac{ax}{H+x}y - \mu_2(v)y - \eta I \tag{1d}$$

From the above Eq. (1), the rate of change of disease prevalence, $i(t) = \frac{I(t)}{y(t)}$, can be expressed as follows (see Appendix A for the derivation):

$$\frac{di}{dt} = \lambda(v)i(1-i) - e(v)\frac{ax}{H+x}i - \eta i(1-i) \tag{2}$$

Finally, following De Castro and Bolker (2005) and Hilker and Schmitz (2008), the model Eqs. (1a)–(1d), (2) can be summarized only by the three study variables, x, y and i. This is particularly advantageous not only because it helps to remove the singularity in the disease transmission term when the host population is zero but also allows us to establish the cause of the extinction of the host population. We can clearly distinguish the case of whether the consumers become extinct due to (i) ecological factors or (ii) the disease, based on the zero and non-zero i values, respectively. The plausible ecological factors for consumer extinction when the disease prevalence is absent, are a high mortality rate and less conversion efficiency. On the other hand, the epidemiological factor is the high disease-induced mortality of consumers.

Now, let us describe the specific forms of the functional responses due to the toxin into different traits in the subsequent section.

2.2. Toxicity incorporation

The first term on the right-hand side of Eq. (1a) represents the net growth of the resource in the absence of consumers, which is taken to be Beverton–Holt type (see Thieme (2003) for the derivation of this term). The justification for this choice is that increasing toxicity can reduce the carrying capacity in this case. Here, the reproduction and growth rate is represented by $\beta(x,u) = \frac{\alpha_1}{1+\alpha_3 x}b(u)$, where $b(u)$ is the effect of the toxin on the resource's growth rate. The death rate is denoted by $\mu_1(u)$, which depends on the toxin body burden of the resource, u. The second term is biomass loss due to the consumer's predation. The functional response is taken to be Holling type-II, where a is the maximum feeding rate, and H is the half-saturation constant. In Eqs. (1b)–(1c), $e(v)$ is the food conversion efficiency, $\lambda(v)$ denotes

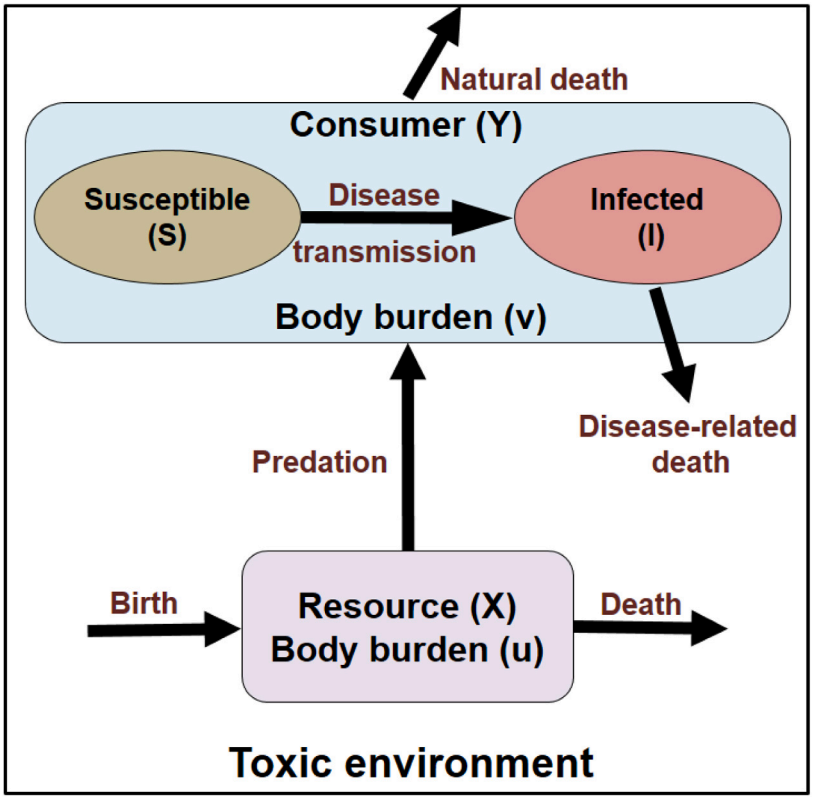

Fig. 1. Schematic representation of our model. Resource growth is regulated by birth, death, and predation by the consumer. The consumer is divided into susceptible and infected classes. The system is under the influence of environmental toxins and the body burden of the resource and consumer are u and v, respectively.

the rate of disease transmission, and $\mu_2(v)$ is the natural mortality rate of the consumer. All these parameters are assumed to be dependent on the toxin body burden of the consumer, v. Infected consumers have an additional disease-induced death term (virulence), η. In our model, disease transmission is assumed to be frequency-dependent, which implies that the per-capita force of infection increases with disease prevalence, $\frac{I}{y}$ (De Koeijer et al., 1998; Hilker and Schmitz, 2008).

For simplicity, we assume that the transmission rate is the only epidemiological trait that is toxin-dependent. Also, we do not consider any recovery from the disease. For readers' convenience, we used largely the same notations as in Hilker and Schmitz (2008) and Huang et al. (2015) for parameters and state variables in this paper.

Modeling toxin accumulation

In order to incorporate the effect of the toxin on the population dynamics of the interacting species, we must track the time evolution of the amount of the accumulated toxin concentration of the resource and consumer species ($U(t)$ and $V(t)$ respectively). Following Huang et al. (2015), their dynamical equations can be written as:

$$\frac{dU}{dt} = a_1 T x - \sigma_1 U - \mu_1(u)U - \frac{axy}{H+x}u \tag{3a}$$

$$\frac{dV}{dt} = a_2 T y - \sigma_2 V + \frac{axy}{H+x}u - \mu_2(v)V - \eta i V \tag{3b}$$

Here, T is the environmental toxicant concentration, a_i, and σ_i ($i = 1, 2$) are the uptake and depuration coefficients of the toxin for resource and consumers, respectively. The concentration of toxins accumulated in both the resource and consumer population is regulated by uptake from the environment and depuration due to metabolism. Additionally, the toxin is lost due to the natural death of both populations and the disease-induced death of the consumer. Predation by consumers also leads to the transfer of toxins from the resource to itself, resulting in biomagnification.

The body burden of the resource and consumer population, already defined above, can thus be expressed as $u(t) = \frac{U(t)}{x(t)}$ and $v(t) = \frac{V(t)}{y(t)}$ respectively, the rate of change of which is given below (see Appendix A):

$$\frac{du}{dt} = a_1 T - \sigma_1 u - \beta(x,u)u \tag{4a}$$

$$\frac{dv}{dt} = a_2 T - \sigma_2 v + \frac{ax}{H+x}(u - ve(v)) \tag{4b}$$

The system can now be fully described using the state variables x, y, and i together with the toxin body burdens u and v. So the Eqs. (1a), (1d), (2), and (4) are the equations of our interest for the remaining part of the paper.

2.2.1. Choice of the functional responses

For analyzing our model, we describe specific forms of toxin body burden dependence for each of the concerned parameters mentioned in the earlier paragraphs.

Environmental toxicity is responsible for reducing the growth and reproduction of species in several ways. It can cause habitat degradation via changing chemical properties like salinity and acidity of marine surface and hamper the growth of the primary producers like phytoplankton by changing the nutrient cycle (Cheevaporn and Menasveta, 2003; Roberts et al., 2013; Zeng et al., 2015). Thus, we consider the effect of the toxin on the resource's growth rate, $b(u)$, to be a monotonically decreasing function of the toxin body burden, u, i.e., $max(0, 1 - \alpha_2 u)$ (Huang et al., 2013, 2015; Thieme, 2003). So the new maximum reproduction rate is given by $\alpha_1 b(u) = \alpha_1 max(0, 1 - \alpha_2 u)$ (see Fig. 2A), which decreases linearly with u up to the threshold value of $\frac{1}{\alpha_2}$, after which it becomes zero and so the resource stops growing. α_2 is the effect coefficient of the toxin on the growth rate of the resource.

Furthermore, toxicants lead to a decrease in the food conversion efficiency of the consumer (Huang et al., 2015; Garay-Narváez et al.,

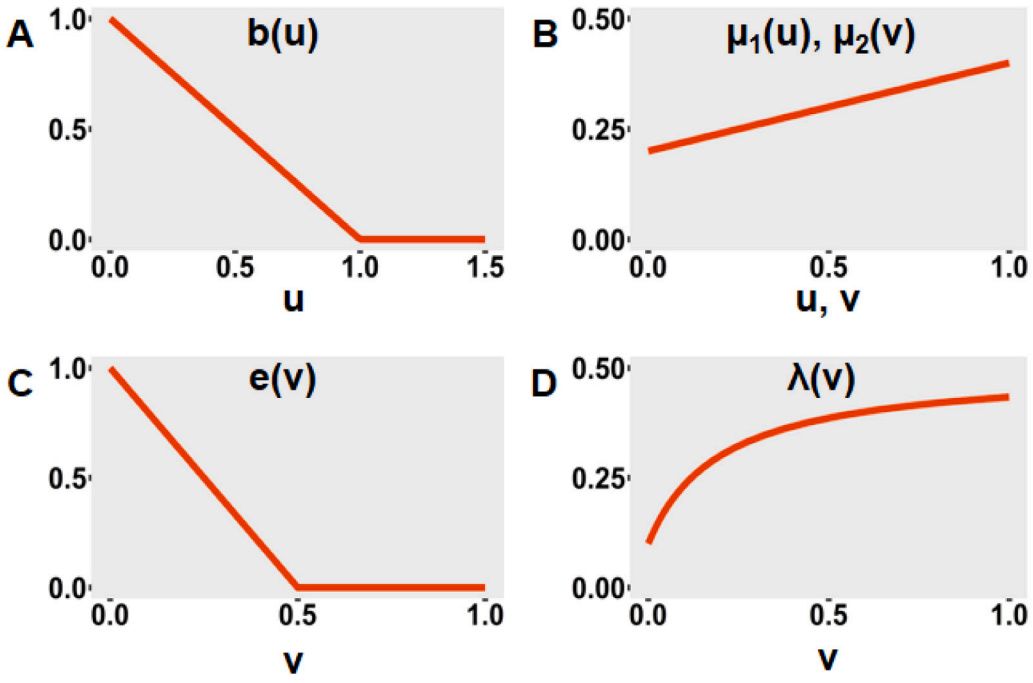

Fig. 2. The responses of toxin body burden on parameters: (a) maximum reproduction rate, (b) mortality of the resource and consumer, (c) conversion efficiency of the consumer, (d) disease transmission rate.

Table 1
Variables and parameters description.

Symbols	Unit	Description
Variables		
x	g/L	Resource density
y	g/L	Consumer density
S	g/L	Susceptible consumer density
I	g/L	Infected consumer density
U	μg/L	Concentration of the toxin in the resource
V	μg/L	Concentration of the toxin in the consumer
u	μg/g	Body burden of the resource
v	μg/g	Body burden of the consumer
Parameters		
α_1	day^{-1}	Maximum reproduction rate of the resource
α_2	g/μg	Effect of toxin on the growth of resource
α_3	L/g	Crowding effect of resource
k_1	g/μg/day	Effect coefficient of the toxin on the resource mortality
p	day^{-1}	Natural mortality of resource
a	day^{-1}	Per-capita feeding rate
H	g/L	Half saturation constant
β_1	–	Reproduction efficiency of consumer
β_2	g/μg	Effect of toxin on the reproduction of consumer
m	–	Effect coefficient of the toxin on the transmission of the disease
b	μg/g	Crowding effect of the consumer
λ	day^{-1}	Disease transmission coefficient
k_2	g/μg/day	Effect coefficient of the toxin on the consumer mortality
μ	day^{-1}	Natural mortality of consumer
η	day^{-1}	Disease related mortality of consumer
a_1	L/g/day	Uptake coefficient of resource
σ_1	day^{-1}	Depuration coefficient of resource
a_2	L/g/day	Uptake coefficient of consumer
σ_2	day^{-1}	Depuration coefficient of consumer
T	μg/L	Toxin concentration in the environment

2013). We assume the consumer's reproduction efficiency to be a linearly decreasing function of the consumer body burden, v, given by $e(v) = \beta_1 max(0, 1 - \beta_2 v)$ (see Fig. 2C), which becomes zero after the threshold value $\frac{1}{\beta_2}$. β_1 is the maximum conversion efficiency of the consumer, and β_2 is the effect coefficient of the toxin on the consumer reproduction (Huang et al., 2015).

Environmental toxins decrease the immunity of species against diseases (De Swart et al., 1994, 1996; Ross et al., 1996), which increases the transmission rate. Keeping this in mind, we incorporate the effect of the toxin on disease transmission. This is assumed to be a monotonically increasing function of the consumer body burden, v, Wang et al. (2018) and is given by $\lambda(v) = (1 + \frac{mv}{b+v})\lambda$ which eventually saturates to a limiting value $(1 + m)\lambda$ (see Fig. 2D). Here, the parameter m is the effect coefficient of the toxin on the transmission, and b is the crowding effect of the consumer. The parameter m corresponds to the saturating limit of the effective disease transmission rate, the increase in which

increase $\lambda(v)$ (see Fig. A.10. (A) in Appendix A.1). On the other hand, the reciprocal of b represents how fast $\lambda(v)$ increases with v (Fig. A.10. (B) in Appendix A.1).

All of the resources, susceptible and infected consumers, are assumed to have toxin-dependent morality terms in addition to their natural mortality, which are linear functions of their toxin body burdens (Fig. 2B). The form of the mortality terms is $\mu_1(u) = p + k_1 u$ and $\mu_2(v) = \mu + k_2 v$ respectively, $k_i, (i = 1, 2)$ being the effect coefficients of the toxin-related mortality for resource and consumers respectively. See Table 1 for the description and units for all parameters and state variables mentioned so far.

2.3. Non-dimensionalization and quasi-steady state approximation

We introduce the standard non-dimensionalization and quasi-steady state approximation techniques for reducing the model complexity. We use the dimensionless model for stability and bifurcation analysis. The first step of the abovementioned method is to introduce the following dimensionless variables and parameters:

$$\bar{x} = \alpha_3 x, \quad \bar{y} = \frac{a\alpha_3}{\alpha_1} y, \quad \bar{t} = \alpha_1 t, \quad \bar{u} = \alpha_2 u, \quad \bar{v} = \beta_2 v,$$
$$\bar{T} = \frac{\alpha_2 a_1}{\sigma_1} T, \quad \bar{k_1} = \frac{k_1}{\alpha_1 \alpha_2}, \quad \bar{k_2} = \frac{k_2}{\alpha_1 \beta_2}, \quad \bar{p} = \frac{p}{\alpha_1}, \quad \bar{h} = H\alpha_3,$$
$$\bar{\beta_1} = \frac{a\beta_1}{\alpha_1}, \quad \bar{\beta_2} = \frac{a\beta_2}{\alpha_2 \sigma_1}, \quad \bar{\sigma_2} = \frac{\sigma_2}{\sigma_1}, \quad \bar{\mu} = \frac{\mu}{\alpha_1}, \quad \bar{\eta} = \frac{\eta}{\alpha_1},$$
$$\bar{\lambda} = \frac{\lambda}{\alpha_1}, \quad \bar{b} = b\beta_2, \quad c = \frac{a_2 \beta_2}{a_1 \alpha_2}, \quad \epsilon = \frac{\alpha_1}{\sigma_1}$$

Substituting the above dimensionless variables and parameters into the Eqs. (1a), (1d), (2) and (4) and omitting the bars, we rewrite the system of equations as follows:

$$\frac{dx}{dt} = max(0, 1-u)\frac{x}{1+x} - (k_1 u + p)x - \frac{xy}{h+x} \tag{5a}$$
$$\frac{dy}{dt} = \beta_1 max(0, 1-v)\frac{xy}{h+x} - (k_2 v + \mu)y - \eta i y \tag{5b}$$
$$\frac{di}{dt} = ((1 + \frac{mv}{b+v})\lambda - \eta)i(1-i) - \beta_1 max(0, 1-v)\frac{xi}{h+x} \tag{5c}$$
$$\epsilon \frac{du}{dt} = (T-u) - \epsilon max(0, 1-u)\frac{u}{1+x} \tag{5d}$$
$$\epsilon \frac{dv}{dt} = cT - \sigma_2 v + \frac{x}{h+x}(\beta_2 u - \epsilon \beta_1 v max(0, 1-v)) \tag{5e}$$

We employ the methodology presented in Huang et al. (2015) to simplify our model by applying the steady-state approximation to the consumer and resource body burdens. The toxin body burden dynamics operate on a considerably faster timescale than the growth of species biomass. For instance, prior investigations by Luoma and Rainbow (2005), Lebrun et al. (2012) and Prosnier et al. (2015) have highlighted the rapid copper assimilation in aquatic species. The toxin depuration rate exceeds the resource reproduction rate. Our parameter ϵ represents the ratio of the resource reproduction rate to the toxin depuration rate and must, therefore, be exceedingly small. Given the small value of ϵ, the positive terms in both the rate equations for u and v dominate, causing them to approach steady-state much more rapidly in comparison to the population time scale. So, letting ϵ tend to zero, Eqs. (5d), (5e) approaches a quasi-steady state, which is given below:

$$u = T \tag{6a}$$
$$v = \frac{cT}{\sigma_2} + \frac{\beta_2 T}{\sigma_2}\frac{x}{h+x} \tag{6b}$$

Substituting the quasi-steady states of the body burden equations, our simplified model becomes:

$$\frac{dx}{dt} = (\frac{max(0, 1-u)}{1+x})x - (k_1 u + p)x - \frac{xy}{h+x} \tag{7a}$$
$$\frac{dy}{dt} = \beta_1 max(0, 1-v)\frac{xy}{h+x} - (k_2 v + \mu)y - \eta i y \tag{7b}$$
$$\frac{di}{dt} = [(1 + \frac{mv}{b+v})\lambda - \eta]i(1-i) - \beta_1 max(0, 1-v)\frac{x}{h+x} i \tag{7c}$$

This model (Eq. (7)) is further analyzed in the remaining part of the paper to study the role of environmental toxins in infectious disease dynamics in ecosystems.

3. Targeting objectives: model-based analysis

If we carefully look at objective 1, there are two questions. It is a trivial fact that toxicity enhances disease progression. Transmission rate and virulence are the two key metaphors for disease progressions. Basic intuition promotes that incorporating toxicity in the consumer-resource system must accelerate the disease progression in consumers. Now, a fundamental question arises whether the toxicity has any pragmatic effect on disease eradication. We first use the simple local stability analysis to find the condition of disease-free equilibrium based on the parameters Toxin level (T), transmission rate (λ), and virulence (η). Bifurcation software is an essential tool to determine the value of critical parameters and the parameters of the relevance of the consumer-resource dynamical system with disease in consumers. We can extensively use MATCONT software in MATLAB for this purpose. The plot of the codim-1 bifurcation diagram concerning toxins for high and low disease transmission rates may be useful and supportive graphics through MATCONT software. Moreover, codim-2 bifurcation diagrams for two pairs of parameters (T, λ and T, η) are helpful tools for understanding the different dynamical regimes of disease progression and elimination.

Intuitively, toxin tolerance levels should decrease with the disease transmission rate. But does this relationship uniformly hold? Beyond a critical value of disease transmission rate, consumers may become extinct at a fixed low toxin level. But can consumers be back in the system after the collapse for a higher transmission rate? Toxin level directly impacts the loss of the consumers' immunity level. As a consequence, susceptible consumers are more exposed to disease when the toxin level increases. Thus, the way of the toxin-induced collapse of the consumers should depend on the disease-related parameters. More specifically, the precursors of such toxin-mediated collapse may vary depending on different epidemic conditions. As stated above, we use the same bifurcation analysis to meet our second and third objectives.

We have already mentioned that the transmission rate and virulence are the two significant components of disease dynamics. The equilibrium points associated with disease-free and disease-induced consumer extinction as an output of the formal stability analysis are the primary tools to meet objective 4. Hilker's model (Hilker and Schmitz, 2008), where the toxicity issue is missing in the system, is a special case of our proposed model. It is obvious that the codim-2 bifurcations with respect to λ and η must be a powerful tool for identifying the different regimes of oscillations and stabilizations. The codim-2 bifurcations with four(4) panels indicating four toxicity levels must be potential visual display measures of understanding the influence of the toxicity in disease-induced stabilization.

First, the mathematical proof of positive invariance and boundedness of the solutions of our model was carried out (see Appendix B.1). Equilibria and their local stability analysis are done in Appendix B.2. The bifurcation analyses are incorporated in the result section for better understanding. For bifurcation analysis, all of the parameters are chosen from published literature (Huang et al., 2015; Hilker and Schmitz, 2008), which includes calibrated as well as hypothetical sets of values. In the absence of disease, our model reduces to that of Huang et al. (2015), and in a toxin-free environment, it is equivalent to Hilker and Schmitz (2008). So, the parameters related to toxins are chosen from the former, while the disease-related ones are chosen from the latter. We use the MATCONT 6p11 (Dhooge et al., 2008) in MATLAB software for the bifurcation analysis of our system.

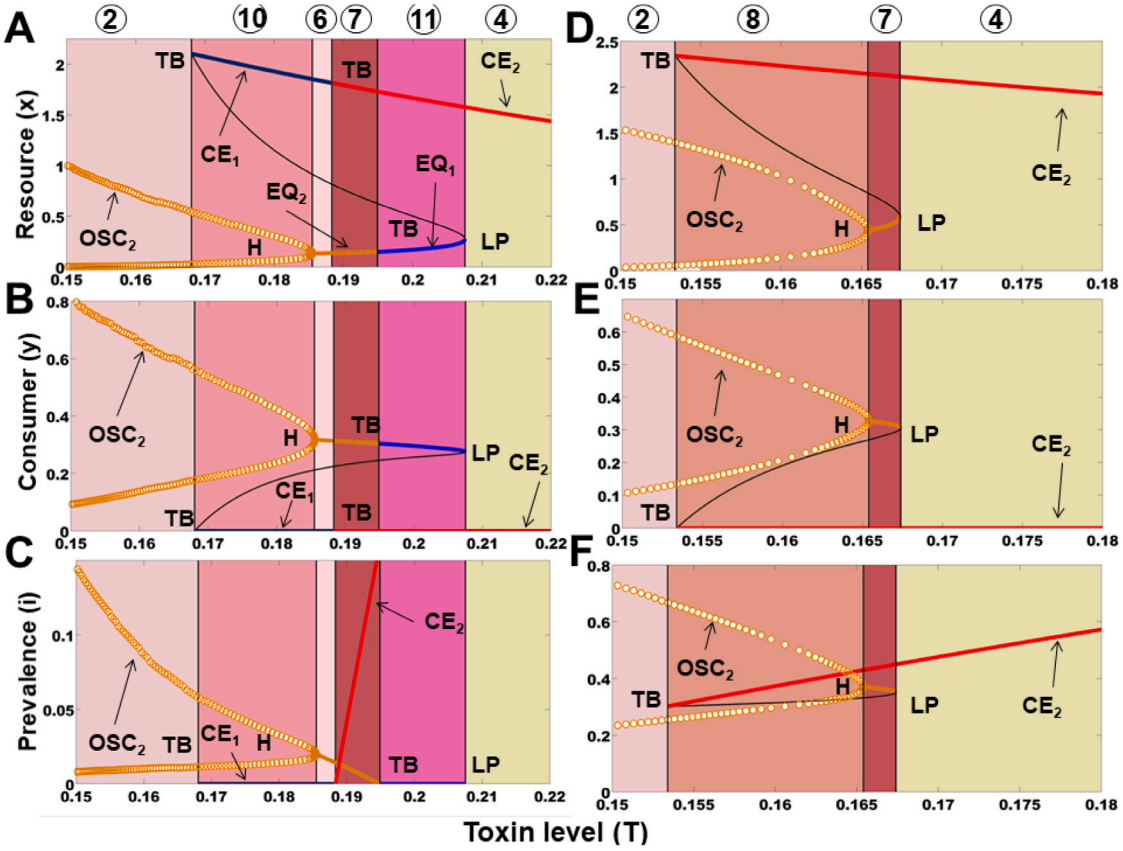

Fig. 3. Effect of changing toxin concentration on the resource (x), the consumer (y), and prevalence (i) for different levels of disease transmission, (A–C) $\lambda = 0.3$ and (D–F) $\lambda = 0.5$. The other parameter values are $b_1 = 1$, $b_2 = 4$, $h = 0.6$, $m = 0.1$, $b = 1$, $c = 1.5$, $\sigma_2 = 1$, $k_1 = 1$, $p = 0.1$, $k_2 = 0.2$, $\mu = 0.02$. Orange and light blue curves indicate the endemic (EQ_2) and disease-free coexistence equilibrium (EQ_1), respectively. Orange circles represent the maximum and minimum population density of the endemic cycle (OSC_2). Deep blue and red curves indicate the ecological and epidemiological extinction of the consumers (CE_1 and CE_2, respectively). Black curves represent the unstable equilibrium branch. The description of the bifurcation points is as in Fig. 6.

4. Results and discussions

Our system exhibits positive invariance and boundedness, ensuring that trajectories originating in the positive quadrant remain within the positive quadrant and do not blow up (see Appendix B.1 for the proof). We have identified six equilibria, and their stability is discussed in Appendix B.2.

We perform extensive bifurcation analysis to meet the objective explained in Section 3. We also attempt to provide intuitive explanations of the different dynamical phenomena observed in the system. In the rest of the paper, EQ_1 denotes the disease-free coexistence equilibrium, and OSC_1 denotes disease-free oscillations. The endemic equilibrium and population cycles are indicated as EQ_2 and OSC_2, respectively. Finally, the ecological extinction of the host is denoted by CE_1 and epidemiological extinction by CE_2.

4.1. Bifurcation analysis

We first plot one-parameter bifurcation diagrams with respect to toxicity (T) for low and high levels of transmission rates (e.g., for $\lambda = 0.3$ and 0.5) to understand the role of toxin for disease dynamics (see Fig. 3).

Effect of toxin for low disease transmission rate

When $\lambda = 0.3$ (Fig. 3(A–C)), the system is in endemic oscillations (OSC_2) state for low level of toxin. Increasing toxicity introduces an alternative stable ecological extinction state of the host, CE_1. With further increase in toxicity, the endemic oscillations become stable, resulting in bistability between EQ_2 and CE_1. In this scenario, when the initial resource density is high, the host population cannot persist (refer to Fig. 4). The steady-state consumer body burden is directly proportional to the resource density (see Eq. (6b)), meaning that a high resource level effectively raises the consumer body burden. This increased body burden results in diminished food conversion efficiency, elevated disease transmission rates, and increased consumer mortality. Consequently, the consumer population goes extinct, yielding a counter-intuitive outcome. Such toxicity-induced bistability has also been demonstrated in previous studies (Huang et al., 2015; Banerjee et al., 2019). On increasing T, EQ_2 undergoes a transcritical bifurcation leading to disease eradication (EQ_1). Ecologically, this happens due to a decrease in host density, which reduces the effective encounter with the host and hence the disease prevalence.

Alternatively, the other equilibrium state, CE_1, also undergoes transcritical bifurcation, leading to disease-induced consumer extinction, CE_2. Such dynamics lead to two more different types of bistability with CE_2 as one of the states, as demonstrated in Fig. 3(A–C). Here, high initial disease prevalence increases the host's disease-induced death, leading to its extinction (Fig. 5. A, C). The bistability between EQ_1 and CE_2 is especially noteworthy. Depending on the disease prevalence, the system exists in disease-free coexistence, or if the disease persists, it leads to consumer extinction (see Figs. 5.C, B.11.C).

Analysis reveals that on increasing toxin concentration, there may be an abrupt transition due to saddle–node bifurcation (LP), which leads to the vanishing of the coexistence equilibrium, thus rendering the host's epidemiological extinction (CE_2) as the only stable state of the system. This transition is irreversible, i.e., once the system has passed the critical threshold (LP), decreasing toxin concentration can no longer return the system to the coexistence state.

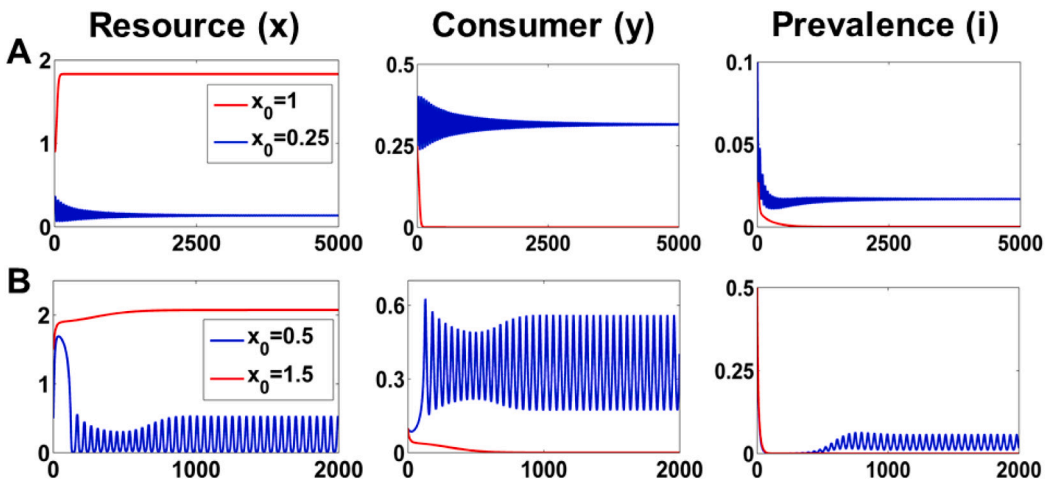

Fig. 4. Time series solution demonstrating bistability between disease-free consumer extinction (CE_1) and (A) endemic coexistence (EQ_2) ($y_0 = 0.25, i_0 = 0.1$; $T = 0.187, \lambda = 0.3$), (B) endemic oscillation (OSC_2) ($y_0 = 0.1, i_0 = 0.5$; $T = 0.17, \lambda = 0.3$). Consumer extinction occurs when resource density is initially high (red line), and it survives for low initial resource density (blue line). Other parameters are the same as in Fig. 8.

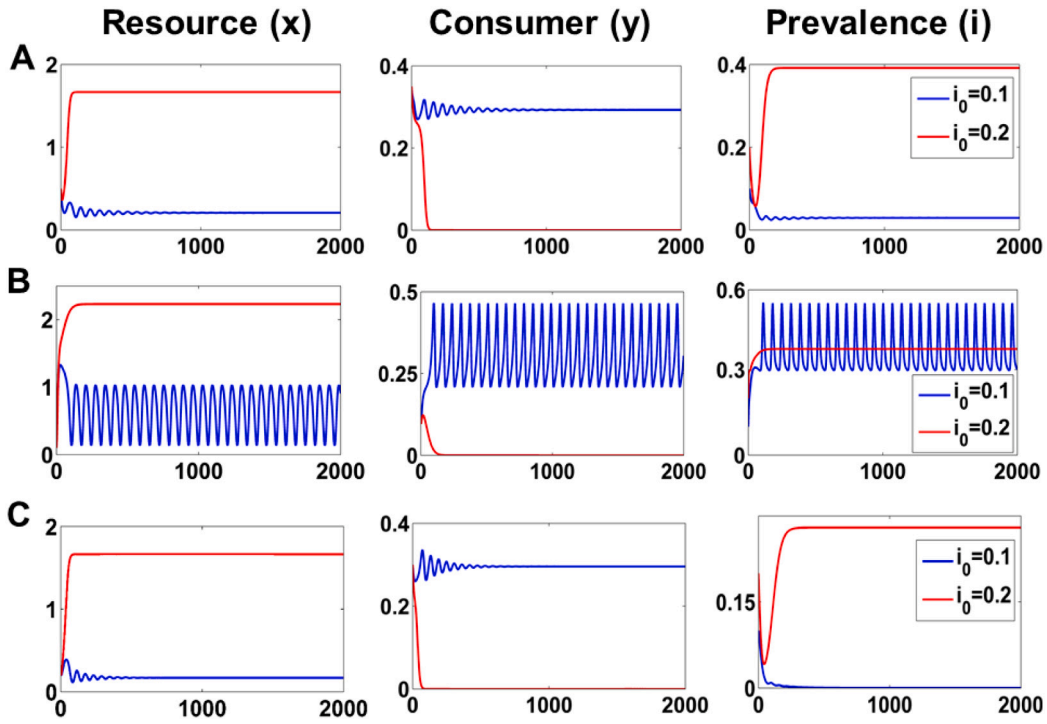

Fig. 5. Time series solution demonstrating bistability between disease-induced consumer extinction (CE_2) and (A) endemic coexistence (EQ_2) ($x_0 = 0.5, y_0 = 0.35$; $T = 0.16, \lambda = 0.5$). (B) endemic oscillation (OSC_2) ($x_0 = 0.1, y_0 = 0.1$; $T = 0.2, \lambda = 0.32$). (C) disease-free coexistence (EQ_1) ($x_0 = 0.2, y_0 = 0.3$; $T = 0.2, \lambda = 0.3$). Disease-induced consumer extinction occurs when initial disease prevalence is high (red line), and endemic coexistence occurs for low initial disease prevalence (blue line). Other parameters are the same as in Fig. 8.

Effect of toxin for high disease transmission rate

For the high transmission rate ($\lambda = 0.5$, Fig. 3D–F), although increased toxin still leads to disease-induced consumer extinction, the route to such extinction differs. Endemic oscillation (OSC_2) is the only state of the system for low toxin levels. Bistability with disease-induced consumer extinction (CE_2) emerges with increasing toxicity. Then oscillation stabilizes, and the system undergoes a saddle–node bifurcation, after which OSC_2 is the only stable state of the system. Unlike the previous scenario, there is no disease-free coexistence in this case, and instead, the endemic oscillations can be bistable with CE_2 (Fig. 5. B).

Two-parameter bifurcation for toxin and disease transmission

The results so far point out the intricate relationship between toxin and disease dynamics. In order to achieve a holistic insight into how disease and toxicity jointly shape the community structure of our system, we carry out a two-parameter bifurcation analysis in the $T - \lambda$ plane (see Fig. 6). An illustration of the equilibrium dynamics in different regions along the two black horizontal lines in Fig. 6 has been provided in the earlier Fig. 3. For a low level of disease transmission rate, the consumer is disease-free (region ①, ⑨, ⑤, see Fig. B.11). The system is in an endemic oscillation state for moderate or high transmission rate (region ②). Increasing toxicity introduces the alternative state of consumer extinction (CE_2), and eventually, this state becomes the only stable state of the system (region ④). For low toxin levels ($T = 0.16$), the disease is established in the system in the form of oscillations with increasing λ. Further, an increase in λ leads to bistability between the endemic oscillation and disease-induced consumer extinction. Oscillations eventually stabilize with λ, and disease-induced consumer

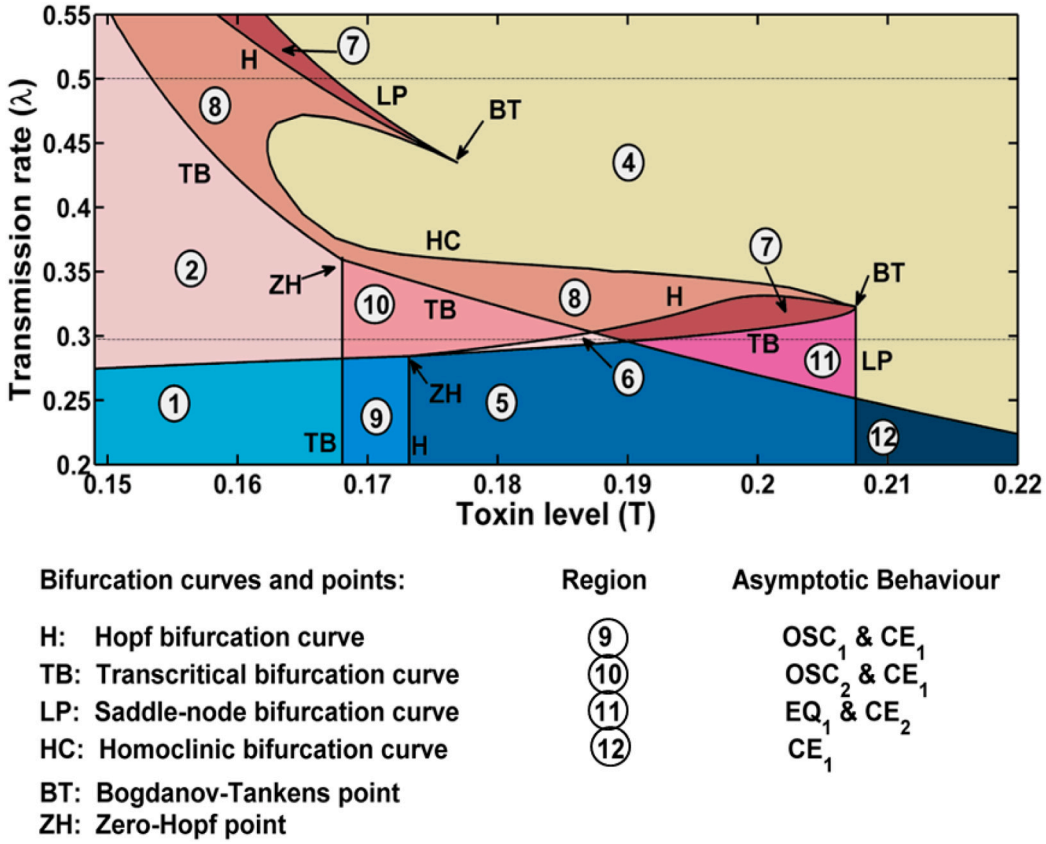

Fig. 6. Two-parameter bifurcation diagram for environmental toxin level (T) and disease transmission rate (λ), demonstrating eleven different dynamical regimes. Asymptotic behaviors: ① OSC_1 (disease-free cycle), ② OSC_2 (endemic cycle), ④ CE_2 (disease-induced consumer extinction), ⑤ EQ_1 and CE_1 (disease-free coexistence and disease-free consumer extinction), ⑥ EQ_2 and CE_1, ⑦ EQ_2 and CE_2, ⑧ OSC_2 and CE_2. Two black horizontal lines indicate the parameter values for which bifurcation diagrams in Fig. 3 are drawn. Here $\eta = 0.2$ and other parameters are same as in Fig. 3.

extinction becomes the only stable state of the system. For a moderate or high level of toxin, bistability between endemic coexistence and consumer extinction is seen with increasing λ. Eventually, endemic oscillation starts, and disease-induced consumer extinction becomes the only stable state of the system after the homoclinic bifurcation for relatively lower values of λ. We also examined the interaction between toxin and transmission at a higher level of virulence (refer to Fig. B.12 in Appendix B.4). In this scenario, the overall system dynamics remain consistent, underscoring the robustness of our findings. However, the bifurcation curves and regions have shifted upward. This shift indicates that a higher λ value is required for the disease to become established. This outcome is attributed to the fact that high virulence effectively eliminates infected hosts from the system, resulting in an inability for the disease to persist at low transmission rates.

Overall, it is observed that for lower values of transmission rate, λ, the system is in the disease-free state represented by different shades of blue in Fig. 6. If we increase the toxin level, the system moves through the regions of different asymptotic behavior to eventually host extinction. It is also noteworthy that, for any level of disease transmission, the host population undergoes catastrophic collapse on increasing toxin concentration. When transmission rate (λ) is high, such a collapse (region ④) occurs for a much lower toxin level, T.

Two-parameter bifurcation for toxin and virulence:

Since the virulence (η) also plays a significant role in disease dynamics (as shown in Hilker and Schmitz (2008)), we plot a two-parameter bifurcation diagram for T and η (see Fig. 7). The regions of dynamical behavior remain the same as in the previous case (Fig. 6). However, the dynamics observed for high η corresponds to that of low λ and vice versa. This is because high virulence increases host mortality and eradicates the disease from the system, whereas disease persists in the system for a low level of η. Also, for any level of η, the host extinction state (CE_1 and CE_2) emerges as an alternative stable state of the system. Finally, these states become the only stable states when the system undergoes a catastrophic shift with increasing T. Two noteworthy behaviors, toxin-induced disease eradication and abrupt extinction from the disease-free coexistence state (EQ_1) to the disease-induced consumer extinction (CE_2), are also observed at a high virulence level.

4.2. An walk towards objectives

4.2.1. Objective 1: Role of the toxin on disease progression and eradication

Toxin promotes consumer extinction: a trivial case

As toxin level increases, bistability with consumer extinction state (CE_1 or CE_2) emerges through a transcritical bifurcation. For low level of λ, or high level of η, bistability with disease-free consumer extinction (CE_1) emerges with increasing toxin (T) (see Figs. 6 and 7, respectively). In this toxin range, the consumer can exist in the system or may go extinct, depending on the initial resource level (Fig. 4). High initial resource level increases the toxin burden of the consumer and hence causes consumer extinction. Further increase in toxin leads to bistability with disease-induced consumer extinction (CE_2). However, for high λ or low η level, toxin directly introduces CE_2. In these cases, the consumer will exist in the system depending on the initial disease level (Fig. 5).

Disease eradication due to toxin: a non-trivial case

Increasing toxicity reduces the consumers' immunity, making them more susceptible to the disease. Toxin increases the effective disease

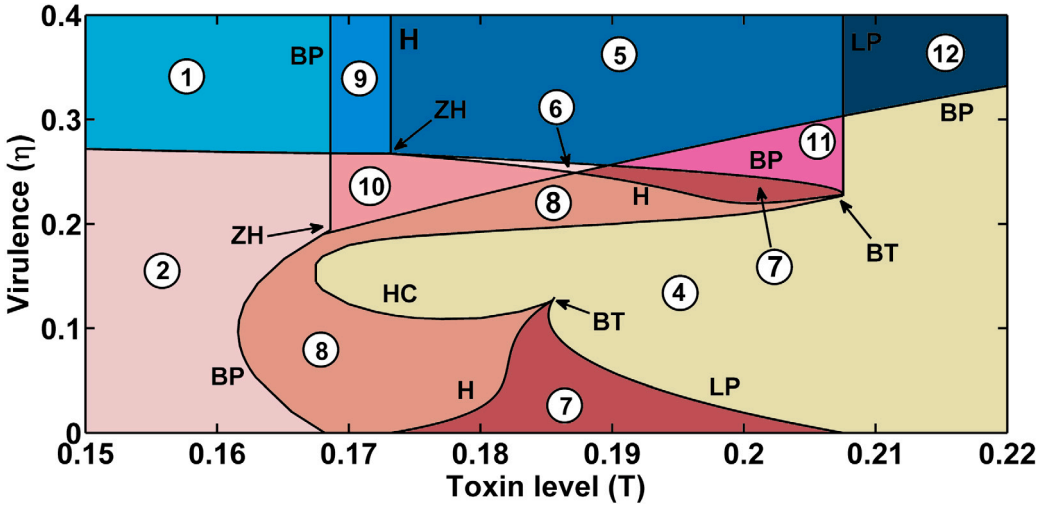

Fig. 7. Two-parameter bifurcation diagram with respect to toxin level (T) and virulence (η), for $\lambda = 0.35$. The description of the regions and bifurcation curves are the same as indicated in Fig. 6. Other parameters are the same as in Fig. 3.

transmission in this way and hence increases disease progression. However, increasing toxins can decrease the disease prevalence and even eradicate the disease from the system for some specific circumstances. Toxicity decreases the disease prevalence from the endemic coexistence state (Fig. 6A-C), where the transmission rate is low. From our results, we see that when the disease transmission rate is low or virulence is high, then increasing toxin moves the system dynamics from region ⑥, ⑦ to ⑤ and ⑪, respectively. In region ⑥, ⑦, endemic equilibrium is one of the stable states. But in region ⑤ and ⑪, disease is not present in the consumers (see Figs. 6, 7, also 3). Increasing toxin levels in the environment increases the body burden of resources and consumers. So, the resource abundance decreases due to the decrease in its growth rate and carrying capacity. Further, the mortality of consumers increases with body burden. So, high host mortality is seen in these circumstances, eliminating the disease due to the lack of hosts. In this way, the toxin can lead to disease eradication when the disease transmission rate is low and virulence is high in the system. So, an increase in toxin levels in the environment may benefit the consumers in such cases. However, toxin-mediated disease eradication is not seen in the case of a high disease transmission rate or low virulence level.

4.2.2. Objective 2: Toxin-induced abrupt consumer extinction

Catastrophic consumer extinction is seen for an increase in the toxin level for any level of fixed disease transmission rate or virulence (see the shift from regions ⑪, ⑧, ⑦ to ④, Figs. 6, 7). For a low or high level of λ and η, increasing toxin leads to a saddle–node bifurcation, and consumers become extinct (region ⑪, ⑦ - ④). For intermediate level of λ or η, consumers extinct through a homoclinic bifurcation (region ⑧ - ④). Increasing disease transmission rate can also lead to abrupt consumer extinction due to disease. Disease transmission rate increases disease prevalence and decreases consumer density due to disease-induced death of the hosts. Eventually, consumers become extinct, and disease-induced consumer extinction becomes the only stable state of the system. However, consumers gradually become extinct because of no toxin in the environment, as seen in Hilker and Schmitz (2008). However, an abrupt consumer extinction event due to increasing disease transmission is seen when the toxin is incorporated into the system. The consumers go to extinction through either a saddle–node bifurcation (LP) or a homoclinic bifurcation (HC) with increasing disease transmission rate for the moderate or high toxin level. A similar role of virulence in the sudden collapse of the consumer is also seen after a certain toxin level.

4.2.3. Objective 3: Precursors of disease-induced consumer extinction

Disease eradication can be considered as a precursor of the abrupt consumer extinction (disease induced) due to toxin, for the low (high) level of λ (η) (see ⑪ - ④, Figs. 6, 7). Disease prevalence decreases with increasing toxicity and the system modes to region ⑪ from region ⑦ through a transcritical bifurcation. In this region ⑪, the system possesses bistability between EQ_1 and CE_2, and the initial disease prevalence level determines the persistence of the consumer in the system. However, the consumers are disease-free if it exists. Further, an increase in toxin level introduces a saddle–node bifurcation, where CE_1 collides with its unstable branch and vanishes. As a result, CE_2 becomes the only stable state of the system. Thus, disease eradication due to the toxin can be a precursor of abrupt and irreversible consumer extinction in this case. For intermediate level of λ or η, the system is in bistability with OSC_2 and CE_2 (region ⑧). Increasing toxin increases the amplitude of the oscillation, and finally, CE_2 becomes the only stable state followed by a homoclinic bifurcation. Thus, the increasing amplitude of the endemic oscillation can be considered a consumer extinction precursor. Finally, for high λ or low η level, endemic oscillation stabilizes with toxin through a hopf bifurcation (region ⑧ - ⑦, Figs. 6, 7). In region ⑦, EQ_2 and CE_2 are the two asymptotic states of the system. A saddle–node bifurcation arises with an increasing toxin, and the system moves to the region ④. So, stabilization of the endemic oscillation with increasing T may be the precursor of the consumer extinction for high (low) λ (η) level.

4.2.4. Objective 4: Effect of toxin on disease-induced stabilization

We compare the dynamical behaviors of our system in the $\lambda - \eta$ plane for low and high contamination levels (see Fig. 8) to understand the effect of the toxin on the stabilizing role of disease, as mentioned in our objective 2.

Disease-induced stabilization for low toxin level

When the toxin is low ($T = 0$, $T = 0.1$, Fig. 8. A, B), the disease is introduced into the disease-free oscillations (region ①) with an increase in λ leading to endemic cycles in region ②. This is followed by a hopf bifurcation (H), resulting in the cycle stabilizing in region ③. Further, an increase in λ leads to epidemiological extinction of the consumers in region ④ by a transcritical bifurcation (TB). This behavior is qualitatively similar to the results demonstrated by Hilker and Schmitz (2008), who analyzed the same model in the absence of toxicity. Interestingly, at higher virulence, η, disease establishes in the

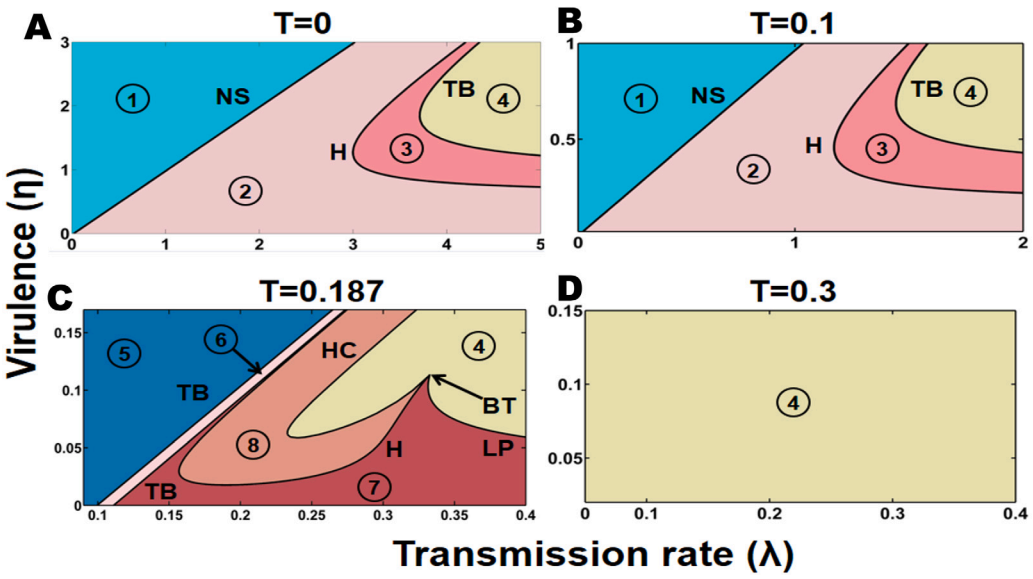

Fig. 8. Two-parameter bifurcation diagram with respect to transmission rate (λ) and virulence (η) showcasing eight different dynamical regimes for environmental toxin levels (a) T = 0, (b) T = 0.1, (c) T = 0.187, (d) T = 0.3. Asymptotic behaviors: ① OSC_1 (disease-free cycle), ② OSC_2 (endemic cycle), ③ EQ_2 (endemic coexistence), ④ CE_2 (disease-induced consumer extinction), ⑤ EQ_1 and CE_1 (disease-free coexistence and disease-free consumer extinction), ⑥ EQ_2 and CE_1, ⑦ EQ_2 and CE_2, ⑧ OSC_2 and CE_2. Description of the bifurcation curves and points are as in Fig. 6.

system only for reasonably high λ. This is because high virulence eliminates the infected host from the system, resulting in the eradication of the disease.

Disease destabilizes the system for high toxin level

The dynamical behavior of our system changed significantly with an increase in the toxin level (for $T = 0.187$, Fig. 8. C). Now, when λ is very low, the system exhibited bistability between two alternative stable states, EQ_1 and CE_1 (region ⑤) (Appendix B.3, Fig. B.11. A). Further, moving along the λ axis, the equilibrium EQ_1 alters its stability with EQ_2, resulting in the persistence of disease in region ⑥. For low virulence (η), further increase in λ will eventually shift the system dynamics to region ⑦ via a transcritical bifurcation (TB) where the system can switch between two alternative stable states, EQ_2 and CE_2. Depending on initial disease prevalence, the consumer survives with a partially infected population or will go extinct (see Fig. 5). On the other hand, for higher virulence (η), on moving along the λ axis, EQ_2 becomes unstable and endemic oscillation starts through a Hopf bifurcation. So bistability between the states OSC_2 and CE_2 is observed in region ⑧. In this context, the toxin induces a destabilization driven by disease, which is in contrast to the findings of Hilker and Schmitz (2008). Elevated toxin levels effectively reduce resource density by diminishing resource growth rates, lowering carrying capacity, and increasing mortality. This reduced resource density, in turn, results in a high consumer-to-resource biomass ratio, causing an imbalance at the top of the food chain Rip and McCann (2011). Consequently, this leads to population cycles with λ in the presence of toxicity. These oscillations can either then become stable, leading to the 'bubbling effect' as demonstrated in Fig. 9A-C, or their amplitude increases with increasing λ until it collapses suddenly to region ④ through a homoclinic bifurcation (see Fig. 9D-F). For the sake of clarity, we do not plot the consumer extinction equilibria (CE_1 and CE_2) in this figure.

5. Conclusions

Previous works, based on the disease-induced consumer resource model, conclude that toxin favors disease progression. However, our proposed model raises an obscure and nontrivial result that toxicity also impacts disease eradication. This result has fundamental implications for understanding the disease-induced consumer-resource dynamics. A robust system prefers a moderate level of toxicity in natural systems. Sudden collapse is inevitable in disease-induced consumer resource systems in the presence of toxicity. Nevertheless, in the absence of toxins, the same system exhibits gradual consumer extinction (Hilker and Schmitz, 2008). This result is an important finding for management experts. The ecologists should emphasize the determination of critical toxin levels for better sustainability of the system. The results obtained from our study indicate that several dynamical regimes can be recognized as the precursors before the abrupt collapse of consumers due to toxins. Disease eradication, endemic oscillations, and stabilization are the precursors associated with the degree of disease intensity. There is scope for management experts to raise the alarm and take intervention policies using the knowledge of those precursors. Finally, Hilker and Schmitz (2008) idea of disease-induced stabilization does not hold when the system is exposed to a certain level of toxins. So, in a nutshell, the toxin has both incremental and detrimental effects on disease-induced consumer-resource systems.

Our study has a limitation. Environmental toxins can impact disease progression in various ways. For instance, they might limit host movement, thereby slowing effective disease transmission, which could ultimately benefit the population in the face of infectious diseases, as suggested by Lafferty and Holt (2003). However, our study focused solely on the immuno-suppressive effect of toxins, which increases disease transmission. Considering both effects could lead to a complex dynamic interplay between toxins and disease. Furthermore, environmental pollution, through biomagnification, has been demonstrated to reduce the persistence of a food web (Garay-Narváez et al., 2013). Conversely, the disease has been shown to enhance community stability, as it can prevent predators from significantly reducing prey populations, thereby increasing stability (Mougi, 2022). Investigating the effects of environmental toxins on an infected food web represents an intriguing research avenue for scientists. Further exploration in this field has the potential to open up new avenues for future research endeavors.

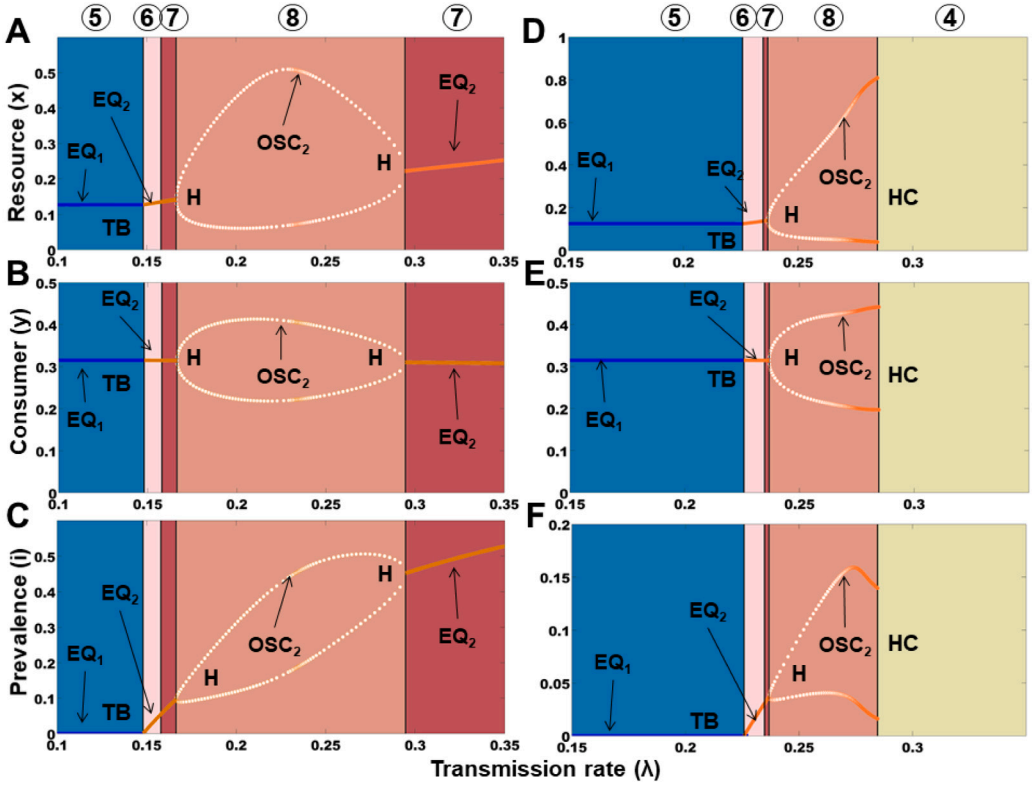

Fig. 9. Effect of the transmission rate (λ) for virulence levels (A–C) $\eta = 0.05$ and (D–F) $\eta = 0.13$ under the high environmental toxin level ($T = 0.187$). Light blue and orange curves represent the disease-free (EQ_1) and endemic equilibrium (EQ_2) respectively. Orange circles represent the maximum and minimum population density of the endemic cycle (OSC_2). Other parameter values are the same as in Fig. 3, and bifurcation points have their usual meaning as mentioned in Fig. 8.

CRediT authorship contribution statement

Arnab Chattopadhyay: Conceived the idea, Refined it, Led the study and designed the simulations, Programmed and ran the simulations, Writing – original draft. **Swarnendu Banerjee:** Conceived the idea, Refined it, Led the study and designed the simulations, Writing – review & editing. **Amit Samadder:** Programmed and ran the simulations. **Sabyasachi Bhattacharya:** Refined the idea, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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Appendix A. Non-dimensionalization and steady-state approximation

We first derive the equations of the disease prevalence and toxin body burdens of both the resource and consumers, respectively as follows:

$$\begin{aligned}
\frac{di}{dt} &= \frac{y\frac{dI}{dt} - I\frac{dy}{dt}}{y^2} \\
&= \frac{1}{y}\frac{dI}{dt} - \frac{I}{y}\left(\frac{y'}{y}\right) \\
&= \frac{1}{y}\left[\left(1+\frac{mv}{b+v}\right)\frac{\lambda SI}{y} - (\mu+k_2v)I - \eta I\right] \\
&\quad - i\left[e(v)\frac{ax}{H+x} - (\mu+k_2v) - \eta\frac{I}{y}\right] \\
&= \left[1+\frac{mv}{b+v}\right]\frac{\lambda Si}{y} - (\mu+k_2v)i - \eta i - i\left[e(v)\frac{ax}{H+x} - (\mu+k_2v) - \eta i\right] \\
&= \left[1+\frac{mv}{b+v}\right]\frac{\lambda i(1-i)y}{y} - e(v)\frac{ax}{H+x}i - \eta i(1-i) \\
&= \left[1+\frac{mv}{b+v}\right]\lambda i(1-i) - e(v)\frac{ax}{H+x}i - \eta i(1-i)
\end{aligned}$$

$$\begin{aligned}
\frac{du}{dt} &= \frac{d}{dt}\left(\frac{U}{x}\right) \\
&= \frac{1}{x}\frac{dU}{dt} - \frac{U}{x}\left(\frac{1}{x}\frac{dx}{dt}\right) \\
&= \frac{1}{x}\left(a_1Tx - \sigma_1U - \mu(u)U - \frac{axy}{H+x}u\right) - u\left(\beta(x,u) - \mu(u) - \frac{ay}{H+x}\right) \\
&= a_1T - \sigma_1u - \beta(x,u)u
\end{aligned}$$

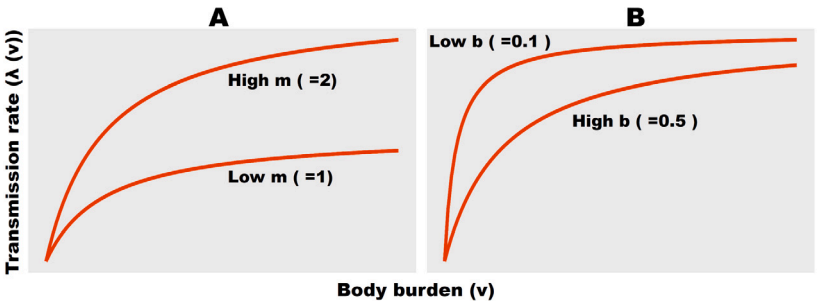

Fig. A.10. Consumer's toxin body burden (v) and effective disease transmission rate ($\lambda(v)$). The saturating value of ($\lambda(v)$) is proportional to m, whereas, how fast ($\lambda(v)$) approaches to its saturating value with v is inversely related to b.

$$\begin{aligned}\frac{dv}{dt} &= \frac{d}{dt}(\frac{V}{y})\\ &= \frac{1}{y}\frac{dV}{dt} - \frac{V}{y}(\frac{1}{y}\frac{dy}{dt})\\ &= \frac{1}{y}(a_2Ty - \sigma_2V + \frac{axy}{H+x}u - (\mu+k_2v)V - \eta\frac{I}{y}V)\\ &\quad - v(e(v)\frac{ax}{H+x} - (\mu+k_2v) - \eta\frac{I}{y})\\ &= a_2T - \sigma_2v + \frac{ax}{H+x}(u - ve(v))\end{aligned}$$

A.1. Functional response of disease due to toxin

Appendix B. Stability analysis

B.1. Proof of positivity and boundedness of solutions

The right-hand side of the system (7a)–(7c) is continuously differentiable and locally Lipschitz in the first quadrant, which implies the existence and uniqueness of solutions for the system in R^3_+. For positive invariance, we rewrite the system as:

$$\frac{dX}{d\tau} = F(X) \tag{B.1}$$

where $X = [x, y, z]^T \in R^3_+$ and $F(X) = [F_1(X), F_2(X), F_3(X)]^T$. The solutions of the system remain in the first quadrant for any non-negative initial condition for all $\tau \geq 0$, since $F_i(X)\ |_{X_i=0} \geq 0$, for all $X_i = 0$ where $i = 1, 2, 3$.

To prove positivity of the solutions of the system (7a)–(7c), we assume,

$$Z(t) = x + y + i$$

Differentiating Z with respect to t we get,

$$\begin{aligned}\frac{dZ}{dt} &= \frac{dx}{dt} + \frac{dy}{dt} + \frac{di}{dt}\\ &= (\frac{[1-u]_+x}{1+x}) - (k_1u+p)x - \frac{xy}{h+x} + \beta_1\frac{[1-v]_+xy}{h+x} - (k_2v+\mu)y\\ &\quad - \eta iy + [(1+\frac{mv}{b+v})\lambda - \eta]i(1-i) - \beta_1[1-v]_+\frac{x}{h+x}i\end{aligned}$$

Since $(\frac{[1-u]_+x}{1+x}) < 1$, $\beta_1\frac{[1-v]_+xy}{h+x} - \frac{xy}{h+x} \leq 0$ we can write,

$$\begin{aligned}\frac{dZ}{dt} &< 1 - (k_1u+p)x - (k_2v+\mu)y - \eta iy + (1+\frac{mv}{b+v})\lambda i(1-i) - \eta i(1-i)\\ &\quad - \beta_1[1-v]_+\frac{x}{h+x}i\end{aligned}$$

As $\frac{v}{b+v} < 1$, $k_1ux \geq 0$, $k_2vy \geq 0$, $\eta i(1-i) \geq 0$ and $\beta_1[1-v]_+\frac{x}{h+x}i \geq 0$,

$$\frac{dZ}{dt} \leq 1 - px - \mu y + (1+m)\lambda i(1-i)$$

For an arbitrary positive real number N we get,

$$\frac{dZ}{dt} + NZ < 1 - x(p-N) - y(\mu-N) + i((\lambda+m\lambda+N) - i(1+m)\lambda) \tag{B.2}$$

Let $N \leq min(p, \mu)$ and the maximum value of $i((\lambda+m\lambda+N) - i(1+m)\lambda)$ is $\frac{(\lambda+m\lambda+N)^2}{4(1+m)}$,

$$\frac{dZ}{dt} + NZ < 1 + \frac{(\lambda+m\lambda+N)^2}{4(1+m)} \tag{B.3}$$

Substituting $A = 1 + \frac{(\lambda+m\lambda+N)^2}{4(1+m)}$ we get,

$$\frac{dZ}{dt} + NZ < A \tag{B.4}$$

By differential inequality

$$0 < Z(x, y, z) < \frac{A(1-exp(-Nt))}{N} + Z(x(0), y(0), z(0))exp(-Nt) \tag{B.5}$$

So for large values of t, we have $0 \leq Z \leq \frac{A}{N}$. Hence, the solution of the system is bounded in the positive quadrant.

B.2. Equilibriums and their stability

The Eqs. (7) can be written in the following form:

$$\frac{dx}{dt} = f(x) - \phi(x)y \tag{B.6a}$$

$$\frac{dy}{dt} = g(x)y - \eta iy \tag{B.6b}$$

$$\frac{di}{dt} = \lambda(x)i(1-i) - e(x)\phi(x)i \tag{B.6c}$$

where,

$$f(x) = \frac{[1-u]_+x}{1+x} - (K_1u+p)x \tag{B.7a}$$

$$\phi(x) = \frac{x}{h+x} \tag{B.7b}$$

$$e(x) = \beta_1[1-v]_+ \tag{B.7c}$$

$$g(x) = e(x)\phi(x) - (\mu+k_2v) \tag{B.7d}$$

$$l(x) = (1+\frac{mv}{b+v})\lambda - \eta \tag{B.7e}$$

The above Eq. (B.1) has the following six equilibrium, $E_j, j = 0, 1, \ldots, 5$.

- The trivial solution $E_0 = (0, 0, 0)$.
- $E_1 = (x_0, 0, 0)$, where $x_0 = \frac{[1-u]_+}{k_1u+p} - 1$.
- $E_2 = (0, 0, 1)$, the trivial extinction due to disease.
- Disease-induced extinction of consumer, $E_3 = (x_0, 0, i_0)$, where $x_0 = \frac{[1-u]_+}{k_1u+p} - 1$ and $i_0 = (1 - \frac{e(x_0)\phi(x_0)}{l(x_0)})$.
- Disease-free coexistence equilibrium, $E_4 = (x_1, \frac{f(x_1)}{\phi(x_1)}, 0)$, where x_1 is the solution of the equation $g(x) = 0$.

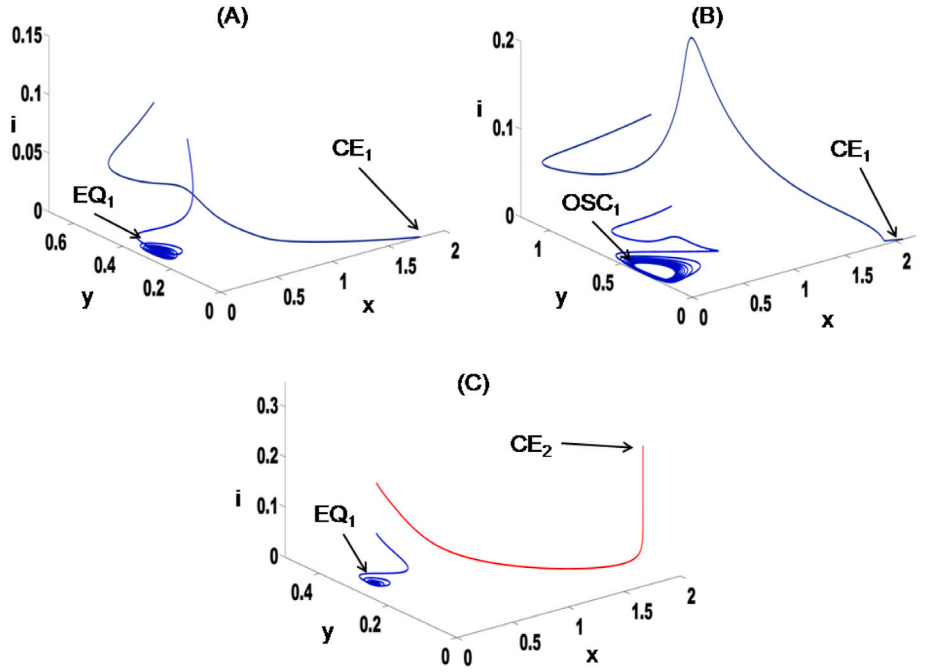

Fig. B.11. The solution trajectories for different bistability regions. (A) region ⑤; (parameters: $T = 0.19$, $\lambda = 0.25$), (B) region ⑨; (parameters: $T = 0.17$, $\lambda = 0.25$), (C) region ⑪; (parameters: $T = 0.2$, $\lambda = 0.3$). The bistability between disease-free coexistence and consumer extinction (disease-free or disease-induced) is seen.

- Endemic equilibrium, $E_5 = (x_2, \frac{f(x_2)}{g(x_2)}, \frac{g(x_2)}{\eta})$, where x_2 is the solution of the equation $1 - \frac{e(x)\phi(x)}{l(x)} = \frac{g(x)}{\eta}$.

The Jacobian matrix at any equilibrium point (x, y, z) is:

$$J = \begin{bmatrix} f'(x) - \phi'(x)y & -\phi'(x) & 0 \\ g'(x)y & g(x) - \eta i & -\eta y \\ l'(x)i(1-i) - (e(x)\phi'(x) & & \\ +e'(x)\phi(x))i & 0 & l(x)(1-2i) - e(x)\phi(x) \end{bmatrix}$$

where,

$$f'(x) = \frac{[1-u]_+}{(1+x)^2} - (K_1 u + p) \tag{B.8a}$$

$$\phi'(x) = \frac{h}{(h+x)^2} \tag{B.8b}$$

$$e'(x) = -\frac{\beta_1\beta_2 T}{\sigma_2}\phi'(x) \tag{B.8c}$$

$$l'(x) = \frac{mb\lambda\beta_2 T}{(b+v)^2\sigma_2}\phi'(x) \tag{B.8d}$$

- Jacobian at trivial equilibrium E_0 is:

$$J(E_0) = \begin{bmatrix} [1-u]_+ - (k_1 u + p) & -\frac{1}{h} & 0 \\ 0 & -(\mu + k_2 v) & 0 \\ 0 & 0 & (1 + m\frac{\frac{cT}{\sigma_2}}{b+\frac{cT}{\sigma_2}})\lambda - \eta \end{bmatrix}$$

Thus E_0 is stable when $T > \frac{1-p}{k_1+1}$ and $\eta > (1 + m\frac{\frac{cT}{\sigma_2}}{b+\frac{cT}{\sigma_2}})\lambda$.

- Jacobian at $E_1 = (x_0, 0, 0)$ is:

$$J(E_1) = \begin{bmatrix} f'(x_0) & -\phi'(x_0) & 0 \\ 0 & g(x_0) & 0 \\ 0 & 0 & l(x_0) - e(x_0)\phi(x_0) \end{bmatrix}$$

Here $f'(x_0) = -(k_1 u + p)\frac{x_0}{x_0+1} < 0$. Thus E_1 is stable if $g(x_0) < 0$ and $l(x_0) - e(x_0)\phi(x_0) < 0$; which together implies : $l(x_0) < e(x_0)\phi(x_0) < (\mu + k_2 v)$.

- Jacobian at the trivial extinction $E_2 = (0, 0, 1)$ is

$$J(E_2) = \begin{bmatrix} [1-u]_+ - (k_1 u + p) & -\frac{1}{h} & 0 \\ 0 & -(\mu + k_2 v) - \eta & 0 \\ -\beta_1[1 - \frac{cT}{\sigma_2}]\frac{1}{h} & 0 & \eta - (1 + m\frac{\frac{cT}{\sigma_2}}{b+\frac{cT}{\sigma_2}})\lambda \end{bmatrix}$$

The eigenvalues of the matrix are the diagonal elements of the matrix and hence E_2 is stable when $T > \frac{1-p}{k_1+1}$ and $\eta < (1+m\frac{\frac{cT}{\sigma_2}}{b+\frac{cT}{\sigma_2}})\lambda$.

- Jacobian at the disease-induced consumer extinction equilibrium $E_3 = (x_0, 0, i_0)$ is:
$J(E_3) =$

$$\begin{bmatrix} f'(x_0) & -\phi'(x_0) & 0 \\ 0 & g(x_0) - \eta i_0 & 0 \\ l'(x_0)i_0(1-i_0) - (e(x_0)\phi'(x_0) & & \\ +e'(x_0)\phi(x_0))i_0 & 0 & l(x_0)(1-2i_0) - e(x_0)\phi(x_0) \end{bmatrix}$$

The diagonal elements are the eigenvalues. Here $f'(x_0) < 0$ and $l(x_0)(1-2i_0) - e(x_0)\phi(x_0) = l(x_0)i_0 < 0$. Thus E_3 is stable if $g(x_0) - \eta i_0 < 0$, i.e., $\eta > \frac{g(x_0)}{i_0}$.

- Jacobian at the disease-free coexisting equilibrium $E_4 = (x_1, \frac{f(x_1)}{\phi(x_1)}, 0)$ is:

$$J(E_4) = \begin{bmatrix} f'(x_1) - \phi'(x_1)\frac{f(x_1)}{\phi(x_1)} & -\phi'(x_1) & 0 \\ g'(x_1)\frac{f(x_1)}{\phi(x_1)} & g(x_1) & -\eta\frac{f(x_1)}{\phi(x_1)} \\ 0 & 0 & l(x_1) - e(x_1)\phi(x_1) \end{bmatrix}$$

Thus E_4 is stable when $l(x_1) - e(x_1)\phi(x_1) < 0$, $f'(x_1) - \phi'(x_1)\frac{f(x_1)}{\phi(x_1)} + g(x_1) < 0$, and $f'(x_1)g(x_1) - \frac{\phi'(x_1)}{\phi(x_1)}f(x_1)(g(x_1) - g'(x_1)) > 0$

B.3. Bistablity in the consumer-resource system

The system exhibits bistable dynamics for various parameter regimes (region ⑤-⑪). Fig. B.11 demonstrates the bi-stable regimes for the low level of disease transmission rate.

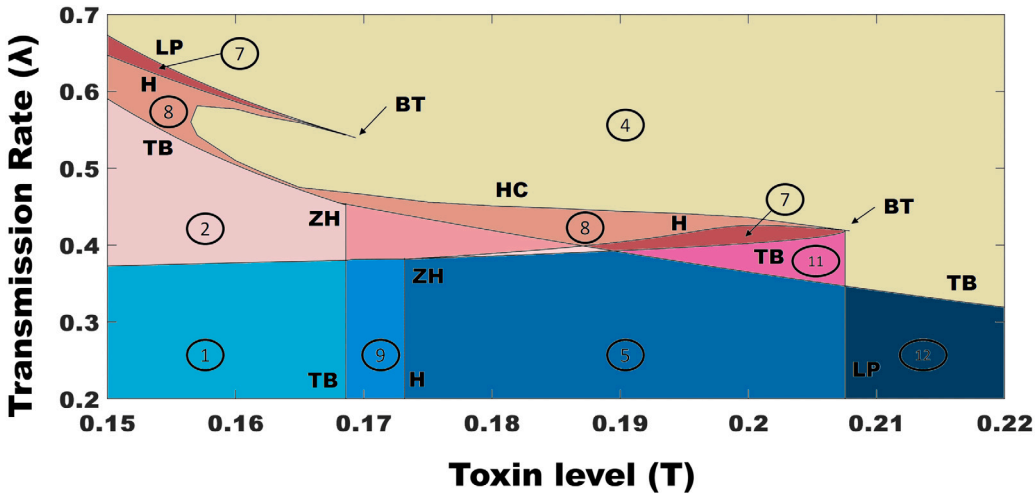

Fig. B.12. Toxin (T)- transmission rate (λ) interplay for higher level of virulence ($\eta = 0.3$). The qualitative behavior of the interplay does not change, but high λ is needed for the establishment of the disease. Other parameters are the same as in Fig. 6.

B.4. Toxin-transmission interplay for high virulence

See Fig. B.12.

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