Enrichment and ecosystem stability: Effect of toxic food

Shovonlal Roy, J. Chattopadhyay*

Agricultural and Ecological Research Unit, Indian Statistical Institute, 203, B.T. Road, Kolkata 700108, India

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Abstract

Enrichment in resource availability theoretically destabilizes predator–prey dynamics (the paradox of enrichment). However, a minor change in the resource stoichiometry may make a prey toxic for the predator, and the presence of toxic prey affects the dynamics significantly. Here, theoretically we explore how, at increased carrying capacity, a toxic prey affects the oscillation or destabilization of predator–prey dynamics, and how its presence influences the growth of the predator as well as that of a palatable prey. Mathematical analysis determines the bounds on the food toxicity that allow the coexistence of a predator along with a palatable and a toxic prey. The overall results demonstrate that toxic food counteracts oscillation (destabilization) arising from enrichment of resource availability. Moreover, our results show that, at increased resource availability, toxic food that acts as a source of extra mortality may increase the abundance of the predator as well as that of the palatable prey.

Keywords: Paradox of enrichment; Food toxicity; Stability; Hopf bifurcation

1. Introduction

Theoretical studies predicted that enrichment of an ecosystem (increasing the available resource) may cause dynamic instability leading to the extinction of species in a finite time (Rosenzweig, 1971; Gilpin, 1972; May, 1972; Abrams and Roth, 1994; Myerscough et al., 1996). Although a few laboratory results (e.g., Luckinbill, 1973; Fussmann et al., 2000) reinforced this 'paradox of enrichment' showing that enrichment leads to oscillation of dynamics that grows with increased carrying capacity, in real ecosystems, destabilization due to enrichment is rarely observed (Vos et al., 2004). Also, a number of experimental results, including McCauley and Murdoch (1990), Kirk (1998), Persson et al. (2001), could not support the paradox of enrichment. Several attempts have been made to explain the discrepancy between theoretical expectations and experimental illustrations (e.g., Brauer and Soudack, 1978; Jansen, 1995, 2001; Scheffer and Deboer, 1995; Abrams and Walter, 1996; Bohannan and Lenski, 1997; Genkai-Kato and Yamamura, 1999; Holyoak, 2000; Persson et al., 2001; Fussmann and Blasius, 2004; Petrovskii et al., 2004; Abrams and Quince, 2005). For instance, in the presence of the prey species that become invulnerable due to either a spatial segregation or some potential survival activity, enrichment cannot destabilize the predator–prey system (Abrams and Walter, 1996).

Spatial interaction between predator and prey species overturns fluctuations and regulates the abundance of the populations (Jansen, 1995). Transition to spatio-temporal chaos can also prevent species extinction in an enriched ecosystem (Petrovskii et al., 2004). Bi- and tri-trophic food chain models that incorporate consumer-induced polymorphism predict that intra-specific heterogeneity in defence levels can overturn instability following enrichment (Vos et al., 2004). A laboratory

experiment with microcosms containing the planktonic rotifer *Synchaeta pectinata* as predator and phytoplankton *Cryptomonas erosa* as prey shows that enrichment can stabilize the population oscillations through autotoxins produced by the predator (Kirk, 1998). However, dynamic instability in an enriched ecosystem is generally an outcome of those simple predator–prey models that incorporate usual 'prey-dependent functional response'.

As an alternative to those functional responses, Arditi and Ginzburg (1989) proposed 'ratio-dependent uptake functions' that do not show instability on enrichment. Although there are debates on the acceptance of ratio-dependent functional forms (Oksanen et al., 1992; Diehl et al., 1993; Abrams, 1994; Gleeson, 1994), the applicability of those functional forms in simple predator–prey models thus overturns the possibility of occurrence of Rosenzweig's paradox.

In the context of ecosystem stability, the energy value or food value of the resource (i.e., prey) is very important. Food value of prey has a significant effect on predator–prey dynamics (e.g., Baalen et al., 2001). A nutrient bound of prey species regulates the dynamics of one-predator–two-prey interaction (e.g., Roy et al., 2005). The experimental work by McCauley and Murdoch (1990) shows that the presence of inedible prey, which acts as a nutrient sponge (Kretzschmar et al., 1993; Murdoch et al., 1998), may provide a plausible mechanism to resolve the paradoxical outcome of ecosystem enrichment. Further, it has been shown theoretically that, in an enriched ecosystem the presence of an unpalatable (less profitable but edible) prey species reduces the amplitude of population oscillations and prevents the minimum abundances of species from falling below certain values (Genkai-Kato and Yamamura, 1999). The stoichiometry or chemical composition of the resource (i.e., prey) generally determines its food value or dietary value for the predator (Jones and Flynn, 2005). A minor change in the stoichiometry of prey may in some cases cause a significant change in its quality as a food (Sterner and Elser, 2002; Mitra and Flynn, 2005). One of the reasons for stoichiometric modulations might be the production of toxin (e.g., Flynn et al., 1996; Calbet et al., 2002).

In marine ecosystems toxin-producing phytoplankton have a significant role in determining the zooplankton level (Chattopadhyay et al., 2002) and regulating the phytoplankton–zooplankton dynamics (Roy et al., 2006). In general, the presence of (common) secondary metabolites in diet is a major cause for food toxicity (Bartosz, 2005). Because of the presence of toxic food in the ecosystem, mixed diets of a predator contain a measurable amount of toxic chemicals (Bartosz, 2005) that act as an inhibitory agent for growth. For example, the dietary composition of human food contains some 1.5 g of plant-originated toxic xenobiotics (Dietrich et al., 2003). However, little attention has been paid to investigate extensively whether the presence of toxin in the composition of food may have any impact on the dynamic instability, predicted theoretically, on enriching the resource availability of an ecosystem.

In this article we theoretically explore the impact of the presence of toxic food in the ecosystem on the dynamic instability caused by increased carrying capacity. For specificity, we consider a simple interaction among two-prey and a predator. One of the two prey is considered as toxic, and we assume that the toxic prey is similar in functional response to the palatable prey, i.e., the toxic prey also exhibits Holling type-II functional response. The predator is assumed to be non-selective; an example could be the filter-feeder (*Daphnia*), however, there are many other examples of filter-feeded organisms. Some typical examples of filter-feeders in the Baltic Sea are mussels and various species of polychates (http://jolly.fimr.fi/boing/encyclopaedia.nsf), and in particular the benthic filter-feeders in Lake Erie are zebra mussels *Dreissena polymorpha* (Edwards et al., 2005). Moreover, there are examples that some big sharks such as *Cetorhinus maximus*, and the whale *Eubalaena* may also behave as filter-feeders (http://www.elasmo-research.org/). We incorporate a toxic prey in the well-known classical Rosenzweig–MacArthur (R–M) model (Rosenzweig and MacArthur, 1963), and analyze the three-component one-predator–two-prey model. In the following sections, first we extend the two-component R–M model to a three-component model for exploring the interaction among a palatable prey, a toxic prey and a common predator. The model is then analyzed to find suitable mathematical bounds on the parameter representing the strength of food toxicity that allows the coexistence of the species and determines the stability of the dynamics. The role of toxic food for preserving the dynamic stability at increased carrying capacity is investigated. The overall results of our study in the context of ecosystem enrichment is discussed with appropriate relevances to the previous studies on this issue.

2. The mathematical model

Assume that one of the two types of prey in a mixed-resource environment is toxic; and the toxicity of those prey, when consumed by the predator, causes an inhibition in the predator growth. We call these toxic prey, and distinguish them from the palatable prey in the mixed-resource. To start with a simplified extension of the two-dimensional R–M model in the presence of these

toxic prey, we assume that the predator is either non-selective or cannot differentiate in the mixed diet the palatable prey from the toxic prey—a situation similar to the interaction between the generalist filter-feeder *Daphnia* and its algal prey in lakes. However, filter feeding is a common feature in many aquatic animals, such as varieties of herbivore fish populations, and also big marine animals such as sharks. The presence of toxic species is common in most naturals waters (Calbet et al., 2002; Chattopadhyay et al., 2002). To the best of our knowledge, a mechanism that accurately describes how a toxic prey may affect the predator is still unknown. There may be several possible ways in which toxic prey might exert a negative effect on predator growth. A common possibility is that toxic prey result in a completely independent mortality term (a negative Holling II functional response term with a coefficient ξ_2) in the equation of predator growth (Sarkar and Chattopadhyay, 2003; Roy et al., 2006). There may be possibilities that toxicity may affect a number of parameters other than mortality rate (e.g., filtering rate) and affect the functional response (Abrams, 1989). However, we concentrate here only on the extra mortality that affects the predator growth on consumption of the toxic species. The original Rosenzweig–MacArthur model for a single prey (with density x) includes a Holling type-II function $(wx)/(D+x)$, where w and D are the rate of predation and half-saturation constant. In the presence of more than one prey, the response function incorporates prey interference, and a two-prey (say densities x_1 and x_2) version of the functional response is expressed by $(w_i x_i)/(D_1 + x_1 + ex_2)$ $(i = 1, 2)$, and $e = D_1/D_2$ (the ratio of the two half-saturation constants) (Fryxell and Lundbegr, 1998). Based on the above assumptions, a plausible model for two-prey–one-predator system, where both the prey exhibit Holling type-II functional response to the predator, but one has a positive and the other has a negative effect on predator growth can be written as follows:

$$\frac{dx_1}{dt} = x_1\left\{r_1\left(1-\frac{x_1}{K_1}\right) - \frac{w_1 y}{D_1 + x_1 + ex_2}\right\}, \quad (1)$$

$$\frac{dx_2}{dt} = x_2\left\{r_2\left(1-\frac{x_2}{K_2}\right) - \frac{w_2 y}{D_1 + x_1 + ex_2}\right\}, \quad (2)$$

$$\frac{dy}{dt} = y\left\{\xi_1\frac{w_1 x_1}{D_1 + x_1 + ex_2} - \xi_2\frac{w_2 x_2}{D_1 + x_1 + ex_2} - c\right\}, \quad (3)$$

with the initial conditions: $x_1(0) > 0$, $x_2(0) > 0$, $y(0) > 0$.

Here, $x_1(t)$, $x_2(t)$ and $y(t)$ are the numbers of individuals (i.e., biomass) of palatable prey, toxic prey and the common predator, respectively, at time t. Let r_i, K_i, w_i, respectively, be the constant intrinsic-growth rate, environmental carrying capacity and the maximum rate of predation for prey $i(i = 1, 2)$; ξ_1 be the assimilation efficiency associated with the predation of palatable prey (species 1) and c be the mortality rate of predator. Let ξ_2 be the efficiency of inhibition in predator growth by toxic material ingested in feeding on toxic prey. The inhibition efficiency ξ_2 is dependent on (proportional to) the strength of toxicity the food, and henceforth we consider this parameter as a measure of food toxicity.

3. Analysis and results

3.1. Local stability

All the solutions of system (1)–(3) which initiate in R^3_+ are uniformly bounded if $q \le c$, where q is a positive constant which should be chosen appropriately (*the proof is obvious.*) The model system (1)–(3) possesses the following equilibria: (i) the species-free equilibrium $E_0 = (0, 0, 0)$; (ii) toxic prey- and predator-free equilibrium $E_1 = (K_1, 0, 0)$; (iii) palatable prey- and predator-free equilibrium, $E_2 = (0, K_2, 0)$; (iv) the predator-free equilibrium, $E_3 = (K_1, K_2, 0)$; (v) toxic prey-free equilibrium $E_4 = (\bar{x}_1, 0, \bar{y})$; (vi) the coexisting (interior) equilibrium $E^* = (x_1^*, x_2^*, y^*)$ where,

$$\bar{x}_1 = \frac{cD_1}{\xi_1 w_1 - c}, \quad (4)$$

$$\bar{y} = \frac{\xi_1 r_1 D_1\{K_1\xi_1 w_1 - c(K_1 + D_1)\}}{K_1(\xi_1 w_1 - c)^2}, \quad (5)$$

and the interior equilibrium point (x_1^*, x_2^*, y^*) is given by the following equations:

$$x_1^* = \frac{K_1(K_2(\theta_1 - 1)\theta_2 - cD_1)}{-\theta_3 K_1 + \theta_1\theta_2 K_2}, \quad (6)$$

$$x_2^* = \frac{K_2(K_1\theta_3(\theta_1 - 1) - cD_1\theta_1)}{-\theta_3 K_1 + \theta_1\theta_2 K_2}, \quad (7)$$

$$y^* = \frac{r_1 r_2(\theta_2 K_2 - K_1\theta_3 + cD_1)}{(-\theta_3 K_1 + \theta_1\theta_2 K_2)^2} \times [D_1(\xi_2\theta_1 K_2 w_2 - \xi_1 K_1 w_1) + (w_1 e\xi_1 + \xi_2 w_2)K_1 K_2(\theta_1 - 1)]. \quad (8)$$

Here $\theta_1 = (w_2 r_1)/(w_1 r_2)$, $\theta_2 = \xi_2 w_2 + ce$ and $\theta_3 = \xi_1 w_1 - c$. After some algebraic manipulations, we find the following conditions sufficient for the existence of

the coexisting equilibrium point of the model system (1)–(3):

$$\theta_1 > 1, \tag{9}$$

$$\theta_2 > \frac{cD_1}{K_2(\theta_1 - 1)}, \tag{10}$$

$$\xi_1 > c\left(\frac{1}{w_1} + \frac{D_1\theta_1}{K_1(\theta_1 - 1)}\right), \tag{11}$$

$$\frac{\xi_2}{\xi_1} > \frac{w_1 K_1}{w_2 K_2}. \tag{12}$$

It can be proved using the Ruth–Hurwitz criterion that all the boundary equilibria (except the predator-free one which is stable if exists) are unstable saddles and the dynamical behavior of the system (1)–(3) around the positive interior equilibrium, where all of the three species coexist, depends on the system parameters. The interior equilibrium of the system, when it exists is locally asymptotically stable in some region of parameter space. Sufficient conditions for local stability of the interior equilibrium, when it exists, are the following:

$$\frac{x_2^*}{x_1^*} > \theta_1 \frac{K_2}{K_1}, \tag{13}$$

$$\theta_2 > D_1 \frac{\theta_1 w_2 K_2 \xi_2 - w_1 K_1 \xi_1}{x_2^* K_1 - \theta_1 x_1^* K_2}. \tag{14}$$

3.2. Dynamics and stability at increased carrying capacity

In the absence of the toxic prey, the model system (1)–(3) reduces to the original R–M model, which is known to exhibit growing oscillations in prey and predator abundances at increased carrying capacity (K_1) (Fig. 1(a)), which is termed the paradox of enrichment. Now if the toxic prey, having an inhibition effect on the predator, is incorporated (i.e., model (1)–(3)), the oscillatory dynamics is affected significantly (Fig. 1). For a fixed level of carrying capacity of the toxic prey, the dynamics moves towards stability for a gradual increase in the strength of food toxicity (Fig. 1(b) and (c)). The stabilizing effect depends on the inhibition efficiency ξ_2 of the toxic food. The predator–prey oscillatory-cycles (Fig. 1(d)) that arise from increased carrying capacity when the toxic food is absent (indicating instability), disappears gradually (Fig. 1(d)–(f)), and the coexisting equilibrium becomes a stable focus (Fig. 1(f)). If the parameter of toxin inhibition is kept fixed and the carrying capacity of the toxic prey is gradually increased, a similar dynamics is obtained (not shown in the figure). The variability of the dynamics is measured on calculating the coefficient of variation (Fig. 2). At increased level of carrying capacity of the palatable prey (K_1), and for a given carrying capacity of the toxic prey (K_2), the coefficient of variation of the biomass of both prey and the predator decreases gradually with a gradual increase in the inhibition efficiency (ξ_2) and settles finally to zero (Fig. 2(a)–(c)). If the toxin inhibition parameter is kept fixed and the carrying capacity of the toxic prey is increased, the coefficient of variation is again decreased gradually to zero (Fig. 2(a)–(c) shows illustrations for three values of $K_1 = 1.5, 1.8, 2.0$). These results suggest that the presence of toxic prey can control the amplitude of unstable oscillation arising from the enrichment.

3.3. Hopf bifurcation at increased carrying capacity

The stability (Fig. 1(f)) of the predator–prey dynamics at increased level of carrying capacity of the palatable prey is possible, because the parameters associated with the toxic prey (carrying capacity K_2 or inhibition efficiency ξ_2) act as bifurcation parameters. It is well known that an increment of the carrying capacity of the palatable prey in the R–M model produces growing oscillation, that occurs due to a change in the dynamic behaviour of the coexisting equilibrium from stability to oscillation through a Hopf bifurcation. However, when a toxic prey is introduced, at increased carrying capacity of the palatable prey and a given carrying capacity of the toxic prey, a Hopf bifurcation occurs for a certain value of ξ_2 (say, ξ_2^c) (Fig. 3). For any value of ξ_2 less than this critical value (the vertical dotted line in Fig. 3(a)–(c)), the dynamics of the coexisting equilibrium of the predator–prey system is unstable (Fig. 3). On the other hand, for an inhibition efficiency greater in magnitude than the critical value ξ_2^c, the equilibrium is stable (Fig. 3). A similar result is obtained (not shown) if ξ_2 is fixed and K_2 is varied. Thus, for an enriched ecosystem, suitable parameter values of K_2 and ξ_2 are obtainable that drive the system back from growing predator–prey cycles to stability. These results demonstrate that at increased resource availability, the presence of toxic food is always stabilizing, and thus favorable for the community in this model.

3.4. Bifurcation in different parameter spaces and stability

Enrichment of an ecosystem is very likely to influence the carrying capacity of both the prey present in the system. To demonstrate the overall effects of enrichment, we examine the dynamical behaviour of the

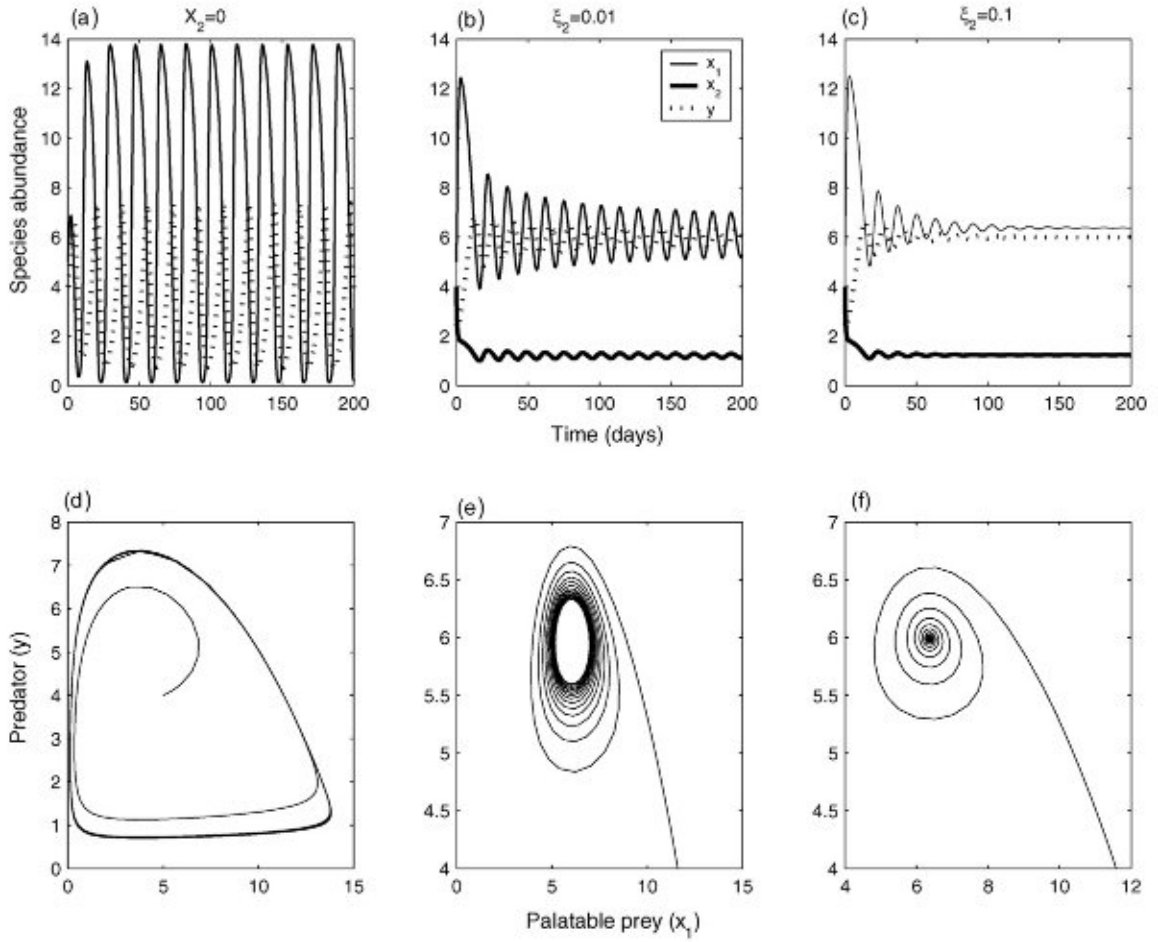

Fig. 1. Different dynamics of predator–prey interaction described by model system (1)–(3). The model is parameterized by the set $r_1 = 2$ day^{-1}, $r_2 = 1.5$ day^{-1}, $w_1 = 2$ day^{-1}, $w_2 = 1.0$ day^{-1}, $c = 0.6$ day^{-1}, $K_1 = 15.0$ nos., $K_2 = 2.0$ nos., $D_1 = 2.5$ nos., $e = 1.25$, $\xi_1 = 0.5$. Dynamical behavior is observed at various strength of inhibition efficiency (ξ_2) of toxic yield. In the panels (a)–(c), thin lines represent palatable prey x_1, bold lines—toxic prey x_2 and dotted lines—predator y. (a) and (d) In the absence of toxic prey (x_2), large amplitude oscillation of the original R–M model at high carrying capacity $K_1 = 15.0$. (b) and (c) Oscillation to stability due to high values of ξ_2. (e) and (f) Gradual disappearance of predator–prey cycle between palatable prey (x_1) and the predator (y) due to high values of ξ_2. (c) Depicts the stable dynamics of the coexisting equilibrium for $\xi_2 = 0.1$, and (f) depicts a stable focus of the coexisting equilibrium in x_1–y plane.

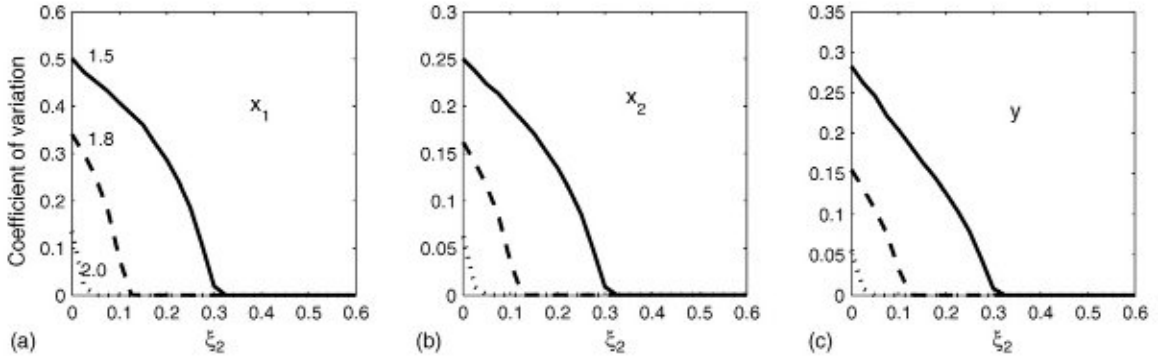

Fig. 2. Coefficient of variation (CV) with gradual increase in ξ_2 at increased carrying capacity $K_1 = 15.0$. (a), (b) and (c), respectively, depicts that CV of the abundance of palatable prey (x_1), toxic prey (x_2) and the predator (y) gradually decrease to zero with increase in the inhibition efficiency. For a fixed ξ_2, CV of all the populations also decrease to zero with a gradual increase in K_2. In all the panels the solid lines correspond to the values of CV for $K_2 = 1.5$, dashed lines for $K_2 = 1.8$ and dotted lines for $K_2 = 2.0$

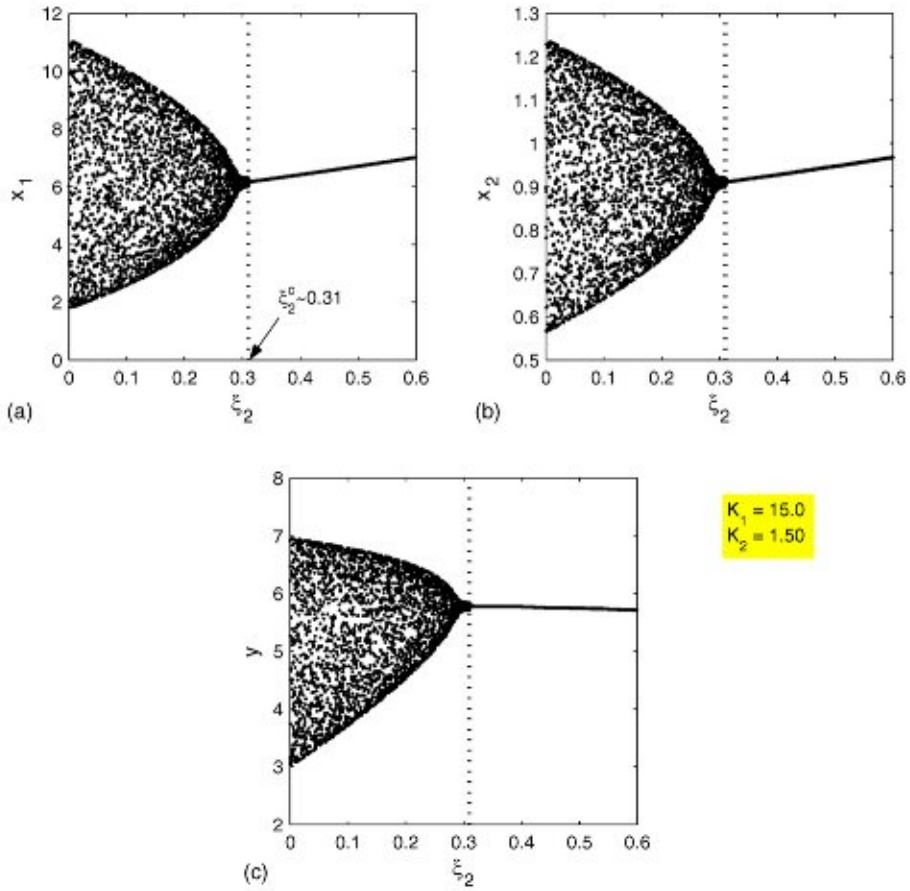

Fig. 3. Crossing the Hopf bifurcation towards dynamic stability; dotted-vertical line in each of figure (a), (b) and (c) represents the critical level of inhibition efficiency ξ_c at which Hopf bifurcation occurs. For values of ξ_2 below this critical level, i.e., for the values of ξ_2 (in horizontal axis) in the left hand side of the vertical-dotted line in (a), (b) and (c), oscillatory dynamics persists at increased carrying capacity ($K_1 = 15.0$, $K_2 = 1.50$). On the other hand, for ξ_2 larger than the critical level, i.e., for the values of ξ_2 (in horizontal axis) in right hand side of the dotted vertical line, the coexisting equilibrium in asymptotically stable (stable focus).

coexisting equilibrium under reasonable ranges of the parameters representing the carrying capacities of palatable prey (K_1) and toxic prey (K_2), and the inhibition efficiency (ξ_2). For a fixed value of ξ_2, a suitable region in the K_1–K_2 plane is obtained where the model system is stable around the positive interior equilibrium (the shaded region of Fig. 4). For an increment in the carrying capacity of the palatable prey (K_1) beyond the critical level (say K_c) at which the original R–M model loses stability, a range of values of K_2 is obtainable (for a fixed value of ξ_2), so that the stability of the system is sustained around the coexisting equilibrium (Fig. 4). In one side of this stability region in the K_1–K_2 parameter plane, a coexisting equilibrium does not exist, however, on the other side, which is separated by the boundary of Hopf bifurcation points, Rosenzweig's paradox occurs (Fig. 4). Hence, for a level of enrichment in the carrying capacity of the palatable prey, a suitable upper bound of the carrying capacity of toxic prey is always obtainable that avoids Rosenweig's paradox of enrichment. In a similar way, in the K_1–ξ_2 plane a stability region exists, such that a suitable range of toxin inhibition parameter is always obtainable for which the coexisting equilibrium in stable (Fig. 5). Here also Rosenzweig's paradox occurs only in a part of the parameter space, separated by the boundary of Hopf bifurcation points, that can be avoided for a suitable choice of ξ_2 (Fig. 5). Furthermore, bifurcation boundaries in the K_2–ξ_2 plane suggests that for an elevated level of carrying capacity of the palatable prey, reasonably large ranges of the parameters K_2 and ξ_2 exist such that, for any combination of those two parameters in the respective ranges, the coexisting equilibria is stable (Fig. 6). These bifurcation diagrams show that the possibility of destabilization of the ecosystem can be avoided efficiently if a toxic prey is present in the system.

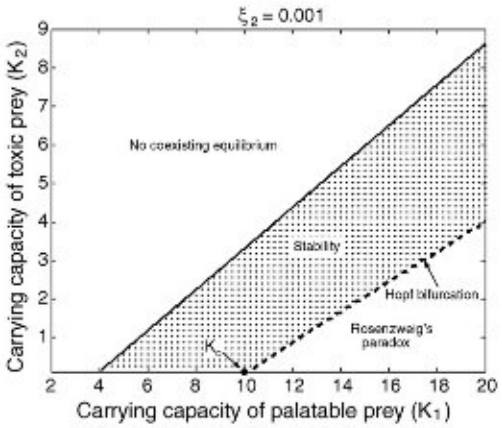

Fig. 4. Stability and bifurcation in K_1–K_2 plane. The value of toxin inhibition is kept fixed at $\xi_2 = 0.001$, other parameters are kept fixed at the values used in Fig. 1. Solid line indicates the boundary, left of which coexisting equilibrium does not exist. The dashed line indicates the boundary where Hopf bifurcations occur. In the shaded region, bounded by the solid line and the dashed line, the coexisting equilibrium is stable. At the right side of the bifurcation boundary, Rosenzweig's paradox occurs. The black-filled dot at K_c indicates the point where the model loses stability if a toxic prey is not present in the system. At any value of K_1 the range of values of K_2, lying in the vertical line passing through that point in the K_1 axis and confined in the shaded region, overturns the growing oscillation and ensures the stability of the coexisting equilibrium.

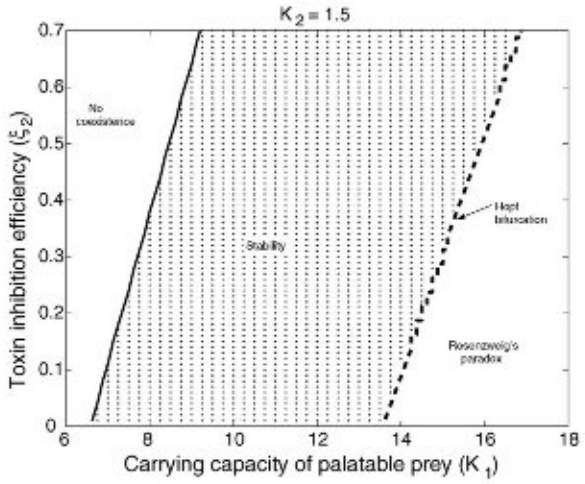

Fig. 5. Stability and bifurcation in K_1–ξ_2 plane. The value of the carrying capacity of the toxic prey is kept fixed at $K_2 = 1.5$, other parameters are kept fixed at the values used in Fig. 1. Solid line indicates the boundary, left of which coexisting equilibrium does not exist. The dashed line indicates the boundary where Hopf bifurcations occur. In the shaded region, bounded by the solid line and the dashed line, the coexisting equilibrium is stable. At the right side of the bifurcation boundary, Rosenzweig's paradox occurs. At any value of K_1 the range of values of K_2, lying in the vertical line passing through that point in the K_1 axis and confined in the shaded region, overturns the growing oscillation and ensures the stability of the coexisting equilibrium.

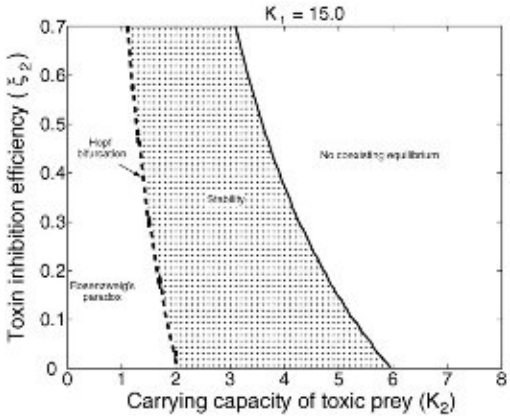

Fig. 6. Stability and bifurcation in K_2–ξ_2 plane. The value of the carrying capacity of the palatable prey is kept fixed at an increased level $K_1 = 15.0$, other parameters are kept fixed at the values used in Fig. 1. Solid line indicates the boundary, right of which coexisting equilibrium does not exist. The dashed line indicates the boundary where Hopf bifurcations occur. In the shaded region, bounded by the solid line and the dashed line, the coexisting equilibrium is stable. At the left side of the bifurcation boundary, Rosenzweig's paradox occurs. For a level of carrying capacity of the palatable prey (here $K_1 = 15.0$), at a fixed point in the axis of K_2 (ξ_2) the range of values of ξ_2 (K_2), lying in the vertical line (the horizontal line) passing through that point in the ξ_2 axis (K_2 axis) and confined in the shaded region, overturns the growing oscillation and ensures the stability of the coexisting equilibrium.

3.5. Effect of toxic inhibition on the abundance of the predator and prey at increased level of carrying capacity

Using Eqs. (1)–(3), we explore the modulations of the equilibrium abundance of the palatable prey and the predator when a toxic prey is present. Calculating $\partial x_1^*/\partial K_2$ and $\partial x_1^*/\partial \xi_2$, we find that if the following condition is satisfied, the equilibrium abundance of the palatable prey increases with the increment in the carrying capacity as well as toxin inhibition efficiency:

$$K_2 > \frac{2}{D_1} + \frac{\theta_3 K_1}{\theta_1 \theta_2}. \tag{15}$$

Similarly, calculating $\partial y^*/\partial K_2$, and $\partial y^*/\partial \xi_2$ the following sufficient condition is derived for which the equilibrium abundance of predator population also increases if the carrying capacity of the toxic prey and/or the toxin inhibition efficiency increases:

$$\frac{\theta_1\theta_2 K_2 - \theta_3 K_1}{K_2(\theta_2 K_2 - K_1\theta_3 + cD_1)} > 2. \tag{16}$$

Simulation results (Fig. 7) depict that, at increased carrying capacity, the effect of inhibition caused by toxic prey is not only to stabilize the community dynamics, but also to determine and regulate the abundance of the palatable

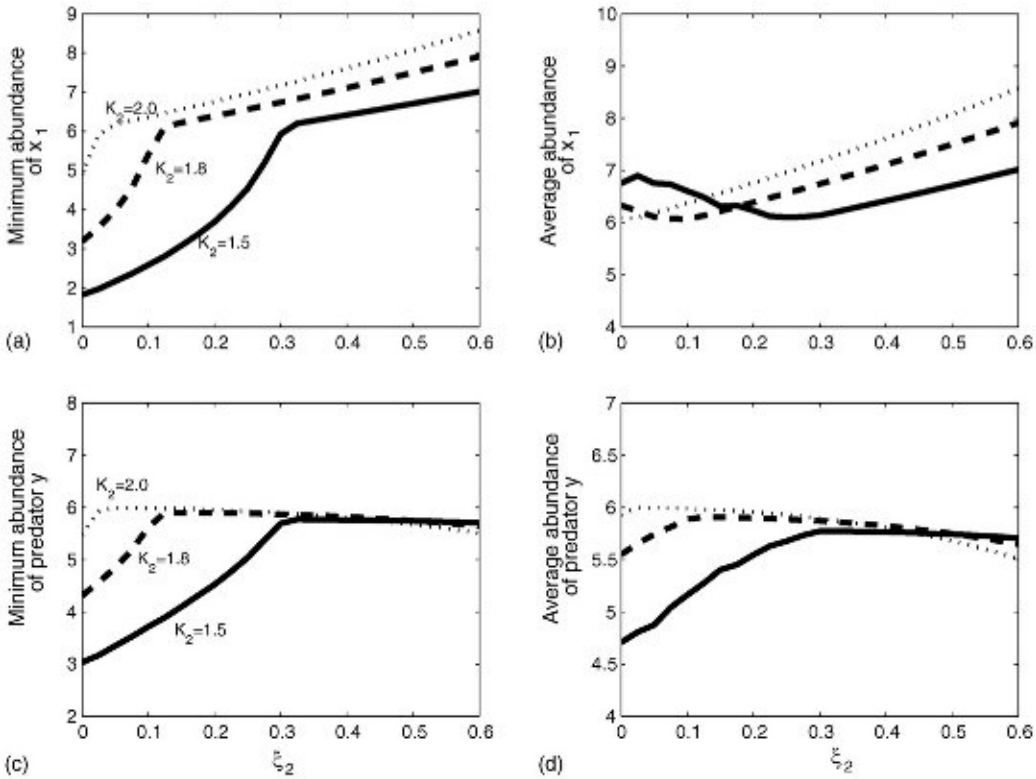

Fig. 7. Degree of toxicity and the growth of palatable prey as well as the predator; for increased carrying capacity K_1 of palatable prey, bold lines are drawn for fixed $K_2 = 1.5$, dashed lines for $K_2 = 1.8$ and dotted lines for $K_2 = 2.0$. The model system was integrated with small step size (< 0.1) using MATLAB software, integration was carried out for a large time period (up to $t = 3000$). To discard the transients, the first half (up to $t = 1500$) of the time-series data for x_1, x_2 and y were rejected, and the calculations of minimum and average abundances were made using only the last half of the time series. The presence of toxic prey is always favorable for the growth of the palatable prey. (a) Minimum abundance of the palatable prey increases and finally saturates for larger inhibition efficiency (ξ_2). (b) For stronger inhibition efficiency, average level of abundance of the palatable prey also increases to a saturation level. (c) Minimum abundance and (d) the average level of abundance of the predator (y) also increases (provided the existence of interior equilibrium is assured) with toxin inhibition efficiency. Food toxicity, at increased level of carrying capacity favours the growth of the predator.

prey as well as the predator. For higher values of inhibition efficiency, minimum abundance as well as the average level of abundance of the palatable prey increase to a saturation level (Fig. 7(a) and (b)). These results indicate that, at increased carrying capacity, (whether or not the system is stable) the threat of extinction of the palatable prey-species due to growing oscillations (as predicted by paradox of enrichment) is overturned if there is a toxic prey in the system. On the other hand, provided that the parameter combination of (K_2, ξ_2) falls in the region where the the interior equilibrium exist (see, Fig. 7), both the minimum abundance and the average level of abundance of the predator increase significantly (Fig. 7(c) and (d)). These results indicate that at increased level of carrying capacity, food toxicity is not harmful, but instead beneficial for the growth of the predator (Fig. 7(c) and (d)), and that the presence of toxic food may promote a realistic predator–prey interaction.

4. Discussions

Generally, enrichment of an ecosystem is studied by increasing the carrying capacity of prey (Rosenzweig, 1971; Gilpin, 1972; May, 1972; Abrams and Roth, 1994; Myerscough et al., 1996). Following this usual practice, we have explored the effect of enrichment on the dynamics of a simple predator–prey interaction. The system that we have considered (1)–(3) consists of two kinds of prey, one is palatable but the other is toxic to the predator. The existence of toxic prey in ecosystem is well known (e.g., Flynn et al., 1996; Chattopadhyay et al., 2002; Calbet et al., 2002). The study demonstrates that, at increased carrying capacity, inhibition due to toxic prey (i.e., toxic food) is an important agent for the stability of the dynamics (Fig. 1). And the toxic food may ensure realistic predator–prey dynamics. The presence of toxic food not only overturns the possibility of dynamic oscillation

(Fig. 1), but also reduces the threat of extinction of the prey species at high carrying capacity (Fig. 7)—a novel result that contributes to a possible resolution to the paradoxical outcome (Rosenzweig, 1971) of theoretical analysis of simple predator–prey models with prey-dependent functional response. A detailed bifurcation analysis shows that Rosenzweig's paradox is always a part of the parameter space that can be avoided on consideration of suitable combination of the carrying capacity of toxic prey and the toxin inhibition efficiency (Figs. 4–6). Moreover, the study suggests that, in an ecosystem enriched by resource availability, the presence of toxic food might be beneficial for the growth of the predator (Fig. 7(c) and (d)). These results of our study implies that, while addressing the food quality (or rather food value) of prey and the enrichment of an ecosystem, sufficient importance might be given to the presence of toxicity.

Although the entire results of our study are based on a simple predator–prey model and the assumptions behind the model formulation are many, which makes the situation considered specific, for a number of reasons the implications of the outcome might be applicable to the general ecosystems. In a broader sense the overall results of our study are consistent with some of the previous studies on this issue. It has been shown by authors that the presence of prey that are either inedible (e.g., Leibold, 1989; Kretzschmar et al., 1993) or invulnerable (Abrams and Walter, 1996) overturns the possibility of ecosystem destabilization following enrichment. Furthermore, Genkai-Kato and Yamamura (1999) has shown that even if prey that is edible but non-profitable, i.e., unpalatable, is present in the system, it can efficiently reduce the predator–prey oscillation and stabilize the dynamics. In the same vein a very likely situation is that in an ecosystem prey that are toxic to the predator are present, and that those prey are accessible to some generalist filter-feeder (such as *Daphnia*). We have analyzed here a simple model that considers such a situation. We find that those toxic prey effectively reduce the high amplitude oscillations and nullify the possibility of an ecosystem to be destabilized following an enrichment. It is well known that in most aquatic ecosystems that some toxic species are present (e.g., Calbet et al., 2002; Chattopadhyay et al., 2002). Thus, for those ecosystems the presence of toxicity might be a very likely explanation to resolve the paradox of enrichment. It has been shown (Abrams, 2002; Matsuda and Abrams, 2004; Abrams and Matsuda, 2005) that, under a range of different predator–prey systems higher mortality of the predator increases its the average population density. Because toxic prey acts as a source of mortality, the model considered here shows that toxicity also stabilizes the predator–prey dynamics and increases the average and minimum abundance of the predator. We suggest that while addressing the issue of ecosystem enrichment, a possible role of food toxicity, which is generally considered as an inhibiting agent for the predator, might be considered to have a positive effect on predator–prey interaction. A careful restriction of toxicity of the toxic food may be treated as beneficial, rather treating as harmful. Some of the recent studies (e.g., Bartosz, 2005) have claimed that food toxicity might be beneficial for calorie restriction, a well-known strategy to prolong the lifespan for not only experimental mammals but also for various non-vertebrate and yeast (Heilbronn and Ravussin, 2003; Merry, 2002). Our results also argue in favour of those studies. However, to confirm that the implications of this study may not be only a model-specific, a number of complimentary theoretical investigations would be necessary. For example, a similar kind of study on consideration of adaptive foraging of zooplankton in presence of toxic prey (Abrams, 1989) and several other possibilities on how toxicity may affect the predator, would be of interest. Finally, some experimental work would be necessary to confirm the results drawn from this study.

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