

# Emergent Allee effects in top predators feeding on structured prey populations

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Top predators that forage in a purely exploitative manner on smaller stages of a size-structured prey population have been shown to exhibit an Allee effect. This Allee effect emerges from the changes that predators induce in the prey-population size distribution and represents a feedback of predator density on its own performance, in which the feedback operates through and is modified by the life history of the prey. We demonstrate that these emergent Allee effects will occur only if the prey, in the absence of predators, is regulated by density dependence in development through one of its juvenile stages, as opposed to regulation through adult fecundity. In particular, for an emergent Allee effect to occur, overcompensation is required in the maturation rate out of the regulating juvenile stage, such that a decrease in juvenile density will increase the total maturation rate to larger/older stages. If this condition is satisfied, predators with negative size selection, which forage on small prey, exhibit an emergent Allee effect, as do predators with positive size selection, which forage on large adult prey. By contrast, predators that forage on juveniles in the regulating stage never exhibit emergent Allee effects. We conclude that the basic life-history characteristics of many species make them prone to exhibiting emergent Allee effects, resulting in an increased likelihood that communities possess alternative stable states or exhibit catastrophic shifts in structure and dynamics.

**Keywords:** structured food chain; Allee effect; size-selective predation; subcritical bifurcation; bistability; catastrophic collapse

## 1. INTRODUCTION

Classical models of food chains (Oksanen *et al.* 1981) predict a stepwise lengthening of the chain with increasing productivity of the environment, in which a new top trophic level is added to the chain at certain threshold values of productivity (Leibold *et al.* 1997). For values of productivity above the threshold the particular trophic level will always be present. Below the threshold productivity it can never occur, as the abundance of its food resource is simply too low. This type of transition at the threshold productivity is technically referred to as a supercritical bifurcation (Kuznetsov 1998). The supercritical nature of the transition or bifurcation point is also observed when we consider the response of the system to changes in the mortality rate of the highest trophic level: there exists a particular threshold value of mortality above which the highest trophic level can never occur in the ecosystem, while below this threshold value it can always persist (see figure 1, curve (ii)). Because of this supercritical bifurcation pattern, classical food-chain models also predict the absence of alternative equilibrium states. Productivity-based models including a higher degree of complexity in the form of omnivory and stage structure have been shown to generate bistability at intermediate levels of productivity (Holt & Polis 1997; Chase 1999; Diehl & Feissel 2000; Mylius *et*

*al.* 2001). However, in these cases, the bifurcation patterns are also supercritical and the alternative states consist of either the predator and the resource or the predator, the consumer and the resource.

The above models of consumers that forage in a purely exploitative manner thus generically predict that an equilibrium with top predators, and one without them, cannot both be stable for the same levels of system productivity or predator mortality. Such a bistability is possible only if the assumption of exploitative foraging is relaxed, and it is explicitly assumed that an Allee effect occurs in the top predator species. Mechanisms giving rise to an Allee effect generally involve social or cooperative processes between multiple individuals that lead to a positive density dependence at low population densities, such that collectively the individuals do better than when alone. Examples of such mechanisms are mate searching, social facilitation of reproduction, predator swamping and anti-predator aggression (Stephens & Sutherland 1999). In a recent paper, De Roos & Persson (2002) showed that in a food chain of consumers that forage purely exploitatively and do not exhibit cooperative behaviour, an Allee effect can none the less occur for top predators, if they forage on their prey population in a size-dependent manner. In this case the transition at the bifurcation point, where top predators can invade an equilibrium population of their prey, is such that for a range of system productivities or predator mortality rates the equilibrium states both with and without predators are stable (see figure 1, curve (i)).

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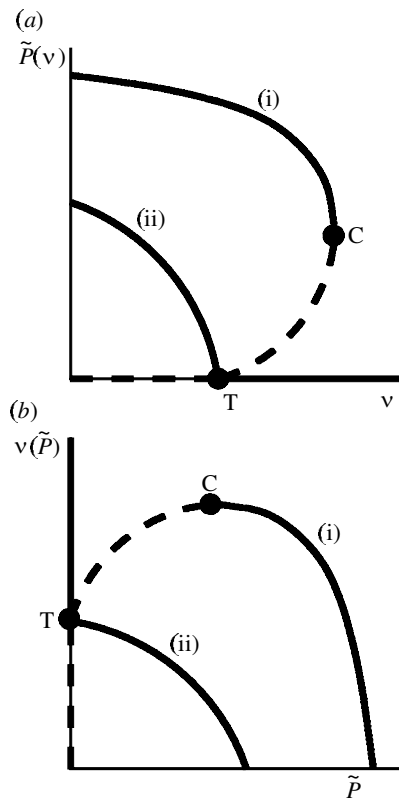


Figure 1. (a) Schematic representation of the relationship between the predator density at equilibrium and its mortality rate with (i) and without (ii) an Allee effect. The point marked T is the transition or branching point at the critical value  $v = v_c$ , where the equilibrium branch representing the prey-only equilibrium (the  $P = 0$  axis). In the case of an Allee effect, the bifurcation at this critical  $v$ -value is referred to as a subcritical, as opposed to a supercritical, bifurcation. With an Allee effect the predator population may collapse and go extinct if the equilibrium is close to the folding point marked C and the predator mortality rate increases slightly. Dashed lines indicate unstable equilibria, solid lines indicate stable equilibria. (b) Illustrates the same relationships but with the axes interchanged. Hence, the predator mortality rate  $v(\tilde{P})$  is shown as a function of the equilibrium predator density  $\tilde{P}$ . This relationship is used for the analysis in this paper.

This type of transition is technically referred to as a subcritical bifurcation (Kuznetsov 1998). The Allee effect that emerges for the top predator makes it possible that a small increase in predator mortality causes a catastrophic collapse of the food chain, in which the predator species is lost and the system reaches a stable equilibrium without it (figure 1, curve (i)). We describe this phenomenon as an ‘emergent Allee effect’, because it emerges through the food-web structure and the life history of another population in the community rather than from the life history of the predator itself. It does, however, refer to a process in which predators can establish themselves in a prey equilibrium only if invading in sufficient numbers, but predators do not cooperate directly. Instead, predators compete with each other through their exploitative feeding on resources.

Mechanistically, the emergent Allee effect follows from the changes that predators induce in the size distribution of their prey population. De Roos & Persson (2002)

showed that if predators are present and forage on the younger prey stages, they reduce the recruitment rate of these small prey to subsequent life stages. As a consequence, predators also reduce the density of individuals in these subsequent life stages and thus relax the intraspecific competition, primarily among older juvenile prey individuals. This relaxation of competition induced by the predators increases the resource levels for the prey and hence increases their fecundity and their growth and maturation rates. Owing to the larger number of adults and their increased fecundity, the total reproduction rate of the prey population is increased, which leads to a higher production of small prey individuals that are vulnerable to predation. Altogether, the predation imposed on the younger prey stages leads to an increase in density of these small prey and hence to an increase in the food available for predators. The emergent Allee effect therefore results as a consequence of the feedback of predator feeding on its own performance, where this feedback operates through and is modified by the life history of their prey individuals.

In De Roos & Persson (2002), the top predator was assumed to exhibit a negative size selection, i.e. to feed only on the smaller size classes of the consumer. Negative size selection is prevalent among many predator taxa (Paine 1976; Paine *et al.* 1985; Tonn *et al.* 1992; Hambright 1994; Wahlstrom *et al.* 2000). However, the opposite pattern, i.e. that the top predator feeds on the largest size classes of the consumer, is also quite common (Brooks & Dodson 1965; Hall *et al.* 1976; Wahlstrom *et al.* 2000). We investigate, more generally, under which conditions an emergent Allee effect can occur in a food chain in which predators forage on a structured prey population. In particular, we investigate whether the assumption of a negative size selection in the top predator is necessary, or whether emergent Allee effects may also be present when the top predator exhibits a positive size selection. We focus primarily on the qualitative aspect, whether the Allee effect occurs or not, and do not discuss the more quantitative aspects, such as the extent of the parameter range for which equilibria with and without predators are both stable.

## 2. MODEL FORMULATION AND ANALYSIS

Identifying the conditions under which an emergent Allee effect occurs is equivalent to distinguishing between the case in which the relationship between equilibrium predator density and predator mortality has a folded shape and exhibits a subcritical bifurcation (as illustrated by curve (i) in figure 1) and the case in which the bifurcation is supercritical (curve (ii) in figure 1). Consider a predator population,  $P$ , foraging on a particular stage of the structured prey population. Let the density of the predator-sensitive prey stage be denoted by  $Y$ . The dynamics of the predator, if it forages on the prey following purely exploitative feeding, can then be described by

$$\frac{dP}{dt} = (\phi f(Y) - \nu)P. \quad (2.1)$$

Here,  $\phi$  indicates the conversion efficiency of prey biomass into newborn predators,  $f(Y)$  represents the predator functional response and  $\nu$  is the background mortality of the predator. We assume that the functional response  $f(Y)$  is a monotonically increasing function of prey density  $Y$ , for

example, a Holling type I, type II or type III functional response. At equilibrium, the predator imposes a density for the predator-sensitive prey stage given by

$$\tilde{Y} = f^{-1}\left(\frac{\nu}{\phi}\right). \quad (2.2)$$

(Here and below we will use a tilde to denote the equilibrium value of a particular variable.) Owing to the monotonicity of the functional response  $f(Y)$ , the equilibrium prey-stage density  $\tilde{Y}$  is an increasing function of predator mortality rate  $\nu$  ( $\partial \tilde{Y} / \partial \nu > 0$ ). Biologically speaking, the higher the predator mortality rate, the higher the prey density that the predator needs for persistence. Equation (2.2) shows that the exact choice of the functional response may have quantitative consequences, but will not affect the monotonicity of the relation between predator mortality and prey equilibrium density.

Let the density of predator-sensitive prey in the absence of predators be denoted by  $Y^*$ . Predator invasion into this equilibrium will be possible when this predator-sensitive prey density  $Y^*$  is larger than required by the predator for its persistence:  $Y^* > \tilde{Y}$ . Owing to the monotonic relation between the equilibrium prey density  $\tilde{Y}$  and the predator mortality  $\nu$  (equation (2.2)) this implies that there exists a critical value  $\nu_c$ , below which the prey equilibrium is unstable against predator invasion. We are interested in the direction of the equilibrium curve relating predator density to its mortality rate at the value  $\nu = \nu_c$  (see figure 1). In the case of an emergent Allee effect the equilibrium predator density,  $\tilde{P}$ , increases with increasing  $\nu$  along this branch at  $\nu = \nu_c$ . If this occurs, the bifurcation is subcritical and an equilibrium with predators is feasible, even for predator mortality rates for which the predator at low density cannot invade the prey-only equilibrium. Considering  $\nu$  as a function of  $\tilde{P}$  (see figure 1b), the condition of an increasing  $\tilde{P}$  with an increase in  $\nu$  can be expressed as

$$\left. \frac{\partial \nu}{\partial \tilde{P}} \right|_{\tilde{P}=0} > 0, \quad (2.3)$$

where the derivative is considered along the branch of equilibria with predators present. Since the predator mortality rate  $\nu$  is monotonically related to the equilibrium prey density  $\tilde{Y}$  this is equivalent to

$$\left. \frac{\partial \tilde{Y}}{\partial \tilde{P}} \right|_{\tilde{P}=0} > 0. \quad (2.4)$$

In other words, an emergent Allee effect occurs owing to a subcritical bifurcation at  $\nu = \nu_c$  where the prey-only equilibrium becomes unstable against predator invasion if, and only if, the equilibrium density of vulnerable prey increases with an increase in equilibrium predator density. In effect, equation (2.4) is a mathematical representation of the essence of the emergent Allee effect: at low predator densities an increase in the number of predators leads to an increase in their food availability and hence to an increase in population growth.

Now, consider that predators impose a mortality rate  $\sigma(P)$  on the vulnerable prey individuals. In general, this predation rate  $\sigma(P)$  will increase with an increase in predator density  $P$ . As a consequence, equation (2.4) can also be expressed as

$$\left. \frac{\partial \tilde{Y}}{\partial \sigma} \right|_{\sigma=0} > 0, \quad (2.5)$$

in which  $\sigma$  denotes the predator-induced mortality rate.

Whether or not size-selective predators will exhibit an emergent Allee effect can therefore be determined by considering only the population dynamics of the prey in the absence of predators. If a small increase in mortality of the predator-sensitive prey individuals leads to an increase in density of these vulnerable prey, predators will have a positive effect on their own food availability through the predation mortality they impose. In the following sections we show that such a counter-intuitive increase in prey density due to increased mortality may indeed occur if the prey population is regulated through density dependence in juvenile development. The resulting Allee effect will give rise to bistability between equilibria with and without predators and the possibility that catastrophic collapses of the predator population will occur (see figure 1, curve (i)). Under these conditions, the predator-invasion bifurcation, where predators can invade a prey-only equilibrium, is subcritical.

### 3. A PREY POPULATION WITH THREE STAGES

We focus on a prey population that is subdivided into three stages: a small larval stage, a large larval stage and an adult stage. For convenience, we refer to these stages as juveniles, subadults and adults, respectively. We consider two different modes by which these populations can be regulated: either by density dependence in the adult fecundity or by density dependence in the maturation and/or mortality rate from the subadult to the adult stage. We consider only intrastage competition, assuming that the density dependence operates purely within a stage, such that the juvenile and subadult densities do not influence adult performance or vice versa, nor do the juvenile and subadult stages influence each other's performance.

#### (a) Prey populations with fecundity regulation

Let the densities of juveniles, subadult and adult individuals be denoted by  $J$ ,  $S$  and  $A$ , respectively. The dynamics of the three stages can now be described by the following system of ordinary differential equations (ODEs):

$$\frac{dJ}{dt} = \beta(A)A - \rho J - (\mu_j + \varepsilon)J, \quad (3.1)$$

$$\frac{dS}{dt} = \rho J - \pi S - (\mu_s + \lambda)S, \quad (3.2)$$

$$\frac{dA}{dt} = \pi S - (\mu_a + \alpha)A. \quad (3.3)$$

The model is an extension of a two-stage system, which is analysed in detail by Thieme (2003, ch. 11). In these equations the function  $\beta(A)$  represents the density-dependent adult fecundity. We assume that fecundity  $\beta(A)$  is a monotonously decreasing function of  $A$ , representing a negative feedback of adult density on population growth. This assumption is necessary for the prey population to be regulated in the absence of predators and can be considered to arise owing to competition for resources. The precise shape of the function  $\beta(A)$  is not important for the purpose of

the analysis presented here. The parameter  $\rho$  represents the maturation rate from the juvenile into the subadult stage ( $\rho^{-1}$  equals the mean duration of the juvenile period) and  $\mu_j$  denotes the background mortality of juveniles. The parameter  $\pi$  is the maturation rate of subadults into adults, while  $\mu_s$  and  $\mu_a$  represent the background mortalities of subadult and adult prey, respectively. The parameters  $\varepsilon$ ,  $\lambda$  and  $\alpha$  represent the predator-induced mortalities of juvenile, subadult and adult prey, respectively. We will consider only predators that forage exclusively on a single stage, on juveniles ( $\varepsilon > 0$ ,  $\lambda = 0$  and  $\alpha = 0$ ), subadults ( $\varepsilon = 0$ ,  $\lambda > 0$  and  $\alpha = 0$ ) or adults ( $\varepsilon = 0$ ,  $\lambda = 0$  and  $\alpha > 0$ ). We will not consider predators that forage on a combination of prey stages.

From equations (3.1)–(3.3) we can deduce that, at equilibrium, juvenile and subadult densities are always proportional to adult density:

$$\tilde{J} = \frac{\mu_s + \lambda + \pi}{\rho} \tilde{S} = \frac{(\mu_s + \lambda + \pi)(\mu_a + \alpha)}{\rho\pi} \tilde{A}, \tag{3.4}$$

$$\tilde{S} = \frac{\mu_a + \alpha}{\pi} \tilde{A}. \tag{3.5}$$

Using the equilibrium relations (3.4) and (3.5) and setting the right-hand side of ODE (3.1) equal to 0 we can derive an expression for the adult fecundity  $\beta(A)$  at equilibrium (ignoring the trivial equilibrium  $\tilde{J} = \tilde{S} = \tilde{A} = 0$ ):

$$\beta(\tilde{A}) = \frac{(\mu_j + \varepsilon + \rho)(\mu_s + \lambda + \pi)(\mu_a + \alpha)}{\rho\pi}. \tag{3.6}$$

From this expression it is clear that the value of  $\beta(\tilde{A})$  at equilibrium always increases with increases in any of the parameters  $\varepsilon$ ,  $\lambda$  or  $\alpha$ . Given that the fecundity  $\beta(A)$  is a decreasing function of  $A$ , this implies that the equilibrium adult density  $\tilde{A}$  always decreases with increases in  $\varepsilon$ ,  $\lambda$  or  $\alpha$ . Since, at equilibrium, both juvenile and subadult densities are proportional to adult density (equations (3.4) and (3.5)), it also implies that all derivatives  $\partial\tilde{J}/\partial\varepsilon$ ,  $\partial\tilde{S}/\partial\lambda$  and  $\partial\tilde{A}/\partial\alpha$  are always negative. In other words, introducing a predator-imposed mortality on juveniles (i.e. increasing  $\varepsilon$  from 0), subadults (i.e. increasing  $\lambda$  from 0) or adults (i.e. increasing  $\alpha$  from 0) will always decrease juvenile, subadult and adult density at equilibrium, respectively. Hence, with fecundity regulation of the prey population in the absence of predators an increase in predator density will always lead to a decrease in their food availability. Predators will therefore never exhibit an emergent Allee effect, regardless of the prey stage they feed on (i.e. the predator-invasion bifurcation is always supercritical; see figure 1; curve (ii)).

**(b) Prey populations with regulation through maturation and/or mortality**

We first consider the case where regulation takes place within the subadult prey stage before addressing the case where it takes place in the juvenile stage. Given a density-dependent maturation and mortality rate, the dynamics of the three-stage prey population can be described by

$$\frac{dJ}{dt} = \beta A - \rho J - (\mu_j + \varepsilon) J, \tag{3.7}$$

$$\frac{dS}{dt} = \rho J - \pi(S)S - (\mu_s(S) + \lambda)S, \tag{3.8}$$

$$\frac{dA}{dt} = \pi(S)S - (\mu_a + \alpha)A, \tag{3.9}$$

where the parameter  $\beta$  represents the (now constant) adult fecundity, the function  $\pi(S)$  represents the (possibly) density-dependent maturation rate from the subadult to the adult stage and the function  $\mu_s(S)$  represents the (possibly) density-dependent mortality rate of the subadults. Regulation through maturation and/or mortality can occur if either the maturation rate  $\pi(S)$  of the subadults decreases or the mortality rate  $\mu_s(S)$  increases with an increase in subadult density, or through a combination of both of these density-dependent effects. These assumptions represent the negative feedback that subadult density might exert on subadult development and mortality, respectively. More formally, we assume that the derivative  $d\pi/dS$  is either 0 or strictly negative, while the derivative  $d\mu_s/dS$  is either 0 or strictly positive for all  $S$ . We exclude the possibility that both derivatives are 0 for all  $S$ , since the prey population would then not be regulated at all.

Setting the right-hand sides of equations (3.7) and (3.9) equal to 0 allows us to express the juvenile and adult densities at equilibrium in terms of the equilibrium subadult density:

$$\tilde{J} = \frac{\beta}{\mu_j + \varepsilon + \rho} \tilde{A} = \frac{\beta}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} \pi(\tilde{S}) \tilde{S}, \tag{3.10}$$

$$\tilde{A} = \frac{1}{\mu_a + \alpha} \pi(\tilde{S}) \tilde{S}. \tag{3.11}$$

Substituting these expressions into the right-hand side of ODE (3.8) and equating it to 0, results in the following equilibrium equation, determining the value of  $\tilde{S}$ :

$$\left( \frac{\beta\rho}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} - 1 \right) \pi(\tilde{S}) = \mu_s(\tilde{S}) + \lambda. \tag{3.12}$$

Here, we have ignored the trivial equilibrium  $\tilde{S} = 0$ . Note that a positive prey equilibrium is feasible only if the left-hand side of this equation, evaluated at  $\tilde{S} = 0$ , is larger than the right-hand side at  $\tilde{S} = 0$ . If this condition is fulfilled, our assumptions about  $\pi(S)$  and  $\mu_s(S)$  ensure that there exists a unique equilibrium subadult density  $\tilde{S}$ .

To determine whether or not the equilibrium juvenile, subadult and adult densities increase or decrease with an increase in predator-induced juvenile, subadult and adult mortality, respectively, we have to investigate the signs of the derivatives  $\partial\tilde{J}/\partial\varepsilon$ ,  $\partial\tilde{S}/\partial\lambda$  and  $\partial\tilde{A}/\partial\alpha$ . In Appendix A we show how expressions for these derivatives can be obtained; here we present only the final results.

First, consider a predator that forages exclusively on subadult prey ( $\varepsilon = 0$ ,  $\lambda > 0$  and  $\alpha = 0$ ). The change in subadult density with an increase in subadult predation mortality is given by (see Appendix A)

$$\frac{\partial\tilde{S}}{\partial\lambda} = \frac{\pi(\tilde{S})}{(\mu_s(\tilde{S}) + \lambda)\pi'(\tilde{S}) - \mu'_s(\tilde{S})\pi(\tilde{S})}. \tag{3.13}$$

Note that  $\pi'(\tilde{S})$  and  $\mu'_s(\tilde{S})$  in these expressions refer to the derivatives  $d\pi/dS$  and  $d\mu_s/dS$ , evaluated at the equilibrium subadult density  $\tilde{S}$ . According to our assumptions,  $\pi'(S)$  is

either 0 or negative and  $\mu'_s(S)$  either 0 or positive, while both are not simultaneously equal to 0. Together these assumptions imply that the partial derivative  $\partial\tilde{S}/\partial\lambda$  is always negative. Increasing predator-imposed mortality on the regulating subadult stage can therefore never increase subadult density. The predator-invasion bifurcation of a predator foraging on subadult prey will always be supercritical and predators of subadults will not show an emergent Allee effect that is induced by the life history of the prey.

Next, consider predators that forage exclusively on either juvenile ( $\varepsilon > 0, \lambda = 0$  and  $\alpha = 0$ ) or adult ( $\varepsilon = 0, \lambda = 0$  and  $\alpha > 0$ ) prey. The changes in juvenile and adult density with an increase in juvenile and adult predation mortality are given by (see Appendix A)

$$\frac{\partial\tilde{J}}{\partial\varepsilon} = \frac{\beta}{\mu_a(\mu_j + \varepsilon + \rho)^2} \frac{(\pi(\tilde{S}))^2}{\mu_s(\tilde{S})\pi'(\tilde{S}) - \mu'_s(\tilde{S})\pi(\tilde{S})} (\pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S})' \quad (3.14)$$

and

$$\frac{\partial\tilde{A}}{\partial\alpha} = \frac{1}{(\mu_a + \alpha)^2} \frac{(\pi(\tilde{S}))^2}{\mu_s(\tilde{S})\pi'(\tilde{S}) - \mu'_s(\tilde{S})\pi(\tilde{S})} (\pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S})', \quad (3.15)$$

respectively. In these equations  $(\pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S})'$  denotes the derivative of the total outflow (maturation plus mortality) rate with respect to the subadult density  $S$  evaluated at the equilibrium subadult density  $\tilde{S}$  (see Appendix A). Our assumptions about  $\pi(S)$  and  $\mu_s(S)$  ensure that the term  $\mu_s(\tilde{S})\pi'(\tilde{S}) - \mu'_s(\tilde{S})\pi(\tilde{S})$  is negative, which leads us to our main result that *both* derivatives  $\partial\tilde{J}/\partial\varepsilon$  and  $\partial\tilde{A}/\partial\alpha$  are positive if, and only if,

$$\left. \frac{d}{dS}(\pi(S)S + \mu_s(S)S) \right|_{S=\tilde{S}} < 0. \quad (3.16)$$

If inequality (3.16) holds, an *increase* in predator-induced mortality of either juvenile or adult prey will *increase* juvenile and adult density, respectively. Hence, a predator that forages selectively on either small or large prey individuals will have a beneficial effect on its own food density through its feeding. Such predator populations will exhibit an emergent Allee effect.

A graphical interpretation of our main result is presented in figure 2: inequality (3.16) holds if the subadult prey equilibrium, in the absence of any predator, is located in a declining part of the curve, which relates the total outflow (maturation plus mortality) rate from the subadult stage to its density. Because we have assumed that  $\mu_s(S)$  is a non-decreasing function, the total mortality rate  $\mu_s(S)S$  is an increasing, possibly accelerating, function of  $S$ . Inequality (3.16) can therefore hold only if the total outflow (maturation plus mortality) rate from the subadult stage first increases from 0 to a maximum with increasing subadult density, subsequently decreases to a minimum and eventually increases to infinity for very high values of subadult density (see figure 2). Such a form of the relationship requires that overcompensation occurs in the total maturation rate  $\pi(S)S$ , i.e. that for certain values of  $S$ , an increase in subadult density actually *decreases* the total maturation rate from the stage, and that this overcompensation is sufficiently strong. Without overcom-

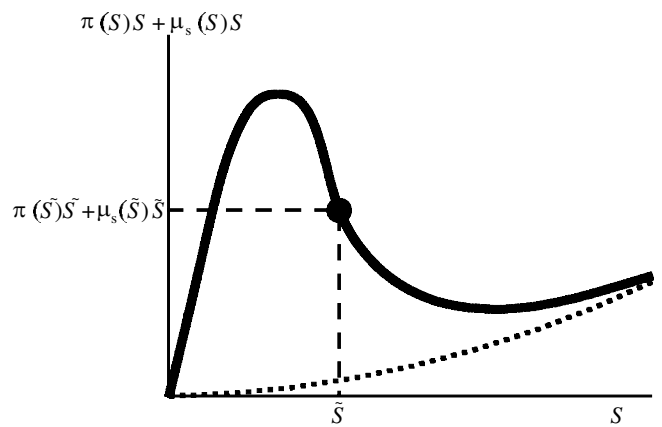


Figure 2. Schematic representation of the conditions required for an emergent Allee effect to occur, when predators exclusively forage on either juvenile or adult prey. The thin dotted line indicates the total mortality rate  $\mu_s(S)S$ , while the thick solid line indicates the total outflow (maturation plus mortality) rate  $\pi(S)S + \mu_s(S)S$  as a function of subadult density  $S$ . The point  $(\tilde{S}, \pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S})$  indicates the prey equilibrium in the absence of predators, which should be located in the declining part of the curve for an emergent Allee effect to occur.

pensation in the maturation rate an emergent Allee effect cannot occur. Also, with a constant density-independent mortality rate  $\mu_s$  inequality (3.16) is more easily satisfied than with a mortality rate that is an increasing function of  $S$ , especially when  $\mu_s$  is small. Emergent Allee effects might, therefore, be expected to occur particularly in predators feeding on long-lived prey species with low background mortality.

We have carried out a completely analogous analysis in which regulation occurs in the juvenile prey stage through a density-dependent maturation rate  $\rho(\mathcal{J})$  and a density-dependent mortality rate  $\mu_j(\mathcal{J})$ . In this case,  $\rho(\mathcal{J})$  and  $\mu_j(\mathcal{J})$  are assumed to be decreasing and increasing functions of juvenile prey density, respectively, while the subadult maturation and mortality rates are assumed to be density-independent constants  $\pi$  and  $\mu_s$ , respectively. The results of this analysis (details not presented) show that under these conditions  $\partial\tilde{J}/\partial\varepsilon$  is always negative, while both  $\partial\tilde{S}/\partial\lambda$  and  $\partial\tilde{A}/\partial\alpha$  are positive if, and only if,

$$\left. \frac{d}{d\mathcal{J}}(\rho(\mathcal{J})\mathcal{J} + \mu_j(\mathcal{J})\mathcal{J}) \right|_{\mathcal{J}=\tilde{\mathcal{J}}} < 0. \quad (3.17)$$

As in the analysis presented earlier, a predator foraging on the regulating stage will always have a decreasing effect on its own food density, but predators foraging on any of the other stages will positively affect their own food density and thus exhibit an emergent Allee effect if the above inequality holds. Also in a two-stage model involving only juveniles and adults (no subadult prey), we have found that if the prey population is regulated through adult fecundity, predators foraging on either juvenile or adult prey cannot exhibit an emergent Allee effect (results not shown). However, if the prey population is regulated through density dependence in the maturation rate from juvenile to adult and the juvenile mortality rate, predators that selectively forage on adult prey will exhibit an emergent Allee effect if inequality (3.17) holds, while predators

foraging only on juvenile prey cannot exhibit such an emergent Allee effect. In general, our main result thus states that in a prey population that is regulated through density dependence in the overall outflow rate (maturation plus mortality) from a particular stage, predators that selectively forage on any of the non-regulating prey stages will exhibit an emergent Allee effect, if there is overcompensation in the regulation, such that the curve relating the outflow rate from the regulating stage to its density is hump shaped, and the prey equilibrium in the absence of any predators is located in the declining part of this curve.

#### 4. DISCUSSION

The results presented extend the study by De Roos & Persson (2002) and reveal more clearly the conditions under which an emergent Allee effect can occur for predators that forage on a structured prey population in an otherwise purely exploitative food chain. First, we predict that predators foraging on prey populations that, in the absence of predators, are regulated through density dependence in adult fecundity will not show an emergent Allee effect. The same holds for prey populations that, in the absence of predators, are regulated only through a (positive) density dependence in the death rate of one of its stages. The transition point at a particular productivity or predator-mortality threshold will always be a supercritical bifurcation point, such that bistability of equilibria with and without predators is not possible. This finding corresponds with the predictions of classical models of food chains, since in these models the top trophic level is always regulated through (indirect) density dependence in adult fecundity.

Second, we show that an emergent Allee effect may commonly occur for predators foraging on prey populations that, in the absence of predators, are regulated by density dependence in development through one of its juvenile stage(s). This hypothesis is outside the scope of the classical unstructured food-chain models as they do not account for the life histories of individual species. The condition for an emergent Allee effect to occur is that there must be overcompensation in the total outflow (i.e. maturation plus mortality) rate from the regulating stage, such that a decrease in the density dependence will increase the total outflow rate. Thus, the curve relating the total maturation and death rate from the regulating stage should be hump shaped and the prey equilibrium in the absence of predators should be located in the declining part of the curve, as illustrated in figure 2. If prey mortality rate is density independent, and prey individuals in the regulating stage experience very low background mortality, this implies that overcompensation in the maturation rate from the regulating stage is sufficient to ensure the occurrence of an emergent Allee effect. However, only predators that forage on stages other than the regulating stage will show the effect. Predators that prefer the regulating stage will always exhibit a supercritical bifurcation without bistability between equilibria with and without predators. Briefly, with overcompensation in development and low background mortality, predators of non-regulating prey stages are likely to exhibit an emergent Allee effect.

Third, our results predict that if the emergent Allee effect is shown to occur for predators foraging on small juvenile stages, it will also occur for predators of large

adult stages of the same prey population. In the model analysed by De Roos & Persson (2002) the regulating prey stage occurs late in the juvenile phase for individuals that have sizes just below the maturation threshold. Growth of these larger juveniles is especially slow and limits their maturation to adulthood. De Roos & Persson (2002) extensively discuss the scenario of predators foraging on small juveniles. Such a negative size selection is present among predators of many taxa where larger stages of the consumer are in a size refuge (Paine 1976; Paine *et al.* 1985; Tonn *et al.* 1992; Hambright 1994; Juanes 1994; Boulton & Polis 1999; Chase 1999). Our present analysis also extends the domain of emergent Allee effects to the case of predators that select adult individuals. Thus, we predict that the classical prey-size selection scenario, in which planktivorous fishes drive the zooplankton community towards smaller sizes through selective feeding on larger individuals (Brooks & Dodson 1965; Hall *et al.* 1976; Zaret 1980; Leibold 1989; Elser *et al.* 1995), may also exhibit an emergent Allee effect.

The model presented was based on a number of simplifying assumptions, to allow a detailed mathematical analysis. We considered regulation through only either adult prey fecundity or juvenile prey development and mortality, while competition was assumed to take place only within stages. The emergent Allee effect, however, was first reported in a study of a three-species food chain, in which the prey life history (growth, metabolism, feeding, reproduction and mortality) was explicitly modelled as a function of resource availability for the prey, and competition for resources occurred among all prey individuals (De Roos & Persson 2002). In addition, in the work of De Roos & Persson (2002) it is argued that the effect also robustly occurs in more complex models, provided that predation is size selective and prey growth is food or density dependent. Our simplifying assumptions with respect to the mode of prey regulation therefore do not seem to be overly restrictive. By contrast, the assumption that predators select only specific size classes of prey is crucial: the more predators that forage on prey from the regulating life stage, the less likely is the occurrence of an emergent Allee effect.

As discussed previously, an important prerequisite for the presence of an emergent Allee effect is the development or maturation regulation. Many populations of amphibians, fishes and zooplankton show clear indications of development or maturation regulation. For example, in fishes, which have a high adult fecundity, producing millions of eggs when spawning, the occurrence of stunted populations is well known. In such populations, individual growth and development is severely reduced by intraspecific competition. It is also known that this stuntedness can be countered by relaxing the competition through increased mortality (Amundsen *et al.* 1993; Klemetsen *et al.* 2002). Similar observations are common in zooplankton species, such as *Daphnia*. McCauley & Murdoch (1987) have shown that maturation regulation plays an important part in both laboratory and field populations of *D. pulex*, as juvenile growth is significantly retarded by density-dependent suppression of food availability. Such food-density-dependent growth has also been shown in many amphibian species (Wilbur 1988; Werner 1994).

In general, our study shows that Allee effects may occur

for many more populations than currently expected, and that the presence of Allee effects does not have to depend on the presence of the mechanisms commonly advanced in the ecological literature, such as cooperative behaviour or mate searching. Emergent Allee effects, as analysed in our study, are solely the result of the changes that predators can induce in their food environment: these changes can be induced only to a limited extent by single foragers and will be much more pronounced when more foragers are present. If the changes in the food environment induced by the species actually promote its own population performance, an emergent Allee effect will be the result. Even though one would expect that foraging on a particular prey species would decrease its abundance, we show that a feedback through the life history of the prey may actually lead to the opposite result: by inducing mortality and thus relaxing intraspecific competition the abundance of vulnerable prey increases. In essence, this phenomenon is comparable to the well-known concept of maximum sustainable yield (MSY) in harvesting. The concept of MSY relies on the assumption that the harvested species follows a logistic growth process, attaining its carrying capacity in the absence of any harvesting. By imposing harvest mortality the yield can be increased up to a maximum level, where the (constant) yield equals the maximum production rate of the species at half its carrying-capacity abundance. The harvesting process itself thus induces changes that only increase the yield. Imposing too high a yield leads to a catastrophic collapse in the harvested species, much like the collapses that may occur with the emergent Allee effects studied here.

Finally, stage-structured interactions, and particularly the presence of size refuges in prey, have in both the theoretical and the empirical literature been suggested to have the potential to produce alternative equilibrium states (Paine 1976; Paine *et al.* 1985; Bazely & Jefferies 1986; Chase 1999). Correspondingly, our analysis predicts that basic life-history characteristics of individuals in these populations may make them prone to exhibit alternative states. More importantly, these alternative states may involve the presence of catastrophic behaviour. Emergent Allee effects have therefore been argued to play a part in the recent collapse of many exploited fish populations, such as cod in the Northwest Atlantic (De Roos & Persson 2002).

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## APPENDIX A: DERIVATION OF PARTIAL DERIVATIVES

The derivatives  $\partial\tilde{J}/\partial\varepsilon$  and  $\partial\tilde{A}/\partial\alpha$  can be found by differentiating equations (3.10) and (3.11), respectively, which results in

$$\frac{\partial\tilde{J}}{\partial\varepsilon} = \frac{\beta}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} \times \left( -\frac{1}{\mu_j + \varepsilon + \rho} \pi(\tilde{S})\tilde{S} + (\pi(\tilde{S})\tilde{S})' \frac{\partial\tilde{S}}{\partial\varepsilon} \right), \quad (\text{A } 1)$$

$$\frac{\partial\tilde{A}}{\partial\alpha} = \frac{1}{\mu_a + \alpha} \left( -\frac{1}{\mu_a + \alpha} \pi(\tilde{S})\tilde{S} + (\pi(\tilde{S})\tilde{S})' \frac{\partial\tilde{S}}{\partial\alpha} \right). \quad (\text{A } 2)$$

In these expressions, we use the shorthand notation  $(\pi(\tilde{S})\tilde{S})'$  to denote the derivative of the total maturation rate  $\pi(S)S$  with respect to the subadult density  $S$  evaluated at the equilibrium subadult density  $\tilde{S}$ .

Equations (A 1) and (A 2) show that the derivatives  $\partial\tilde{J}/\partial\varepsilon$  and  $\partial\tilde{A}/\partial\alpha$  depend on the derivatives of  $\tilde{S}$  with respect to  $\varepsilon$  and  $\alpha$ , respectively. We thus have to derive expressions for all three derivatives  $\partial\tilde{S}/\partial\varepsilon$ ,  $\partial\tilde{S}/\partial\lambda$  and  $\partial\tilde{S}/\partial\alpha$  from the implicit equation (3.12). We therefore rewrite the implicit equation (3.12) in the following form:

$$\left( \frac{\beta\rho}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} - 1 \right) \pi(\tilde{S}) - \mu_s(\tilde{S}) = \lambda. \quad (\text{A } 3)$$

Differentiating with respect to  $\lambda$  yields:

$$\left( \left( \frac{\beta\rho}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} - 1 \right) \pi'(\tilde{S}) - \mu_s'(\tilde{S}) \right) \frac{\partial\tilde{S}}{\partial\lambda} = 1, \quad (\text{A } 4)$$

in which we use  $\pi'(\tilde{S})$  and  $\mu_s'(\tilde{S})$  to denote the derivatives  $d\pi/dS$  and  $d\mu_s/dS$ , respectively, evaluated at the equilibrium subadult density  $\tilde{S}$ . Using the identity

$$\frac{\beta\rho}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} - 1 = \frac{\mu_s(\tilde{S}) + \lambda}{\pi(\tilde{S})} \quad (\text{A } 5)$$

derived from equation (A 3) we can rewrite equation (A 4) as:

$$\frac{(\mu_s(\tilde{S}) + \lambda)\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})}{\pi(\tilde{S})} \frac{\partial\tilde{S}}{\partial\lambda} = 1. \quad (\text{A } 6)$$

Our assumption about the functions  $\pi(S)$  and  $\mu_s(S)$  ensures that the first term on the left-hand side of equation (A 6) will always be strictly negative. We can thus apply the implicit function theorem, which leads to expression (3.13) for  $\partial\tilde{S}/\partial\lambda$  presented in the main text. Differentiating equation (A 3) with respect to  $\varepsilon$  yields

$$\frac{\beta\rho}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} \frac{-1}{\mu_j + \varepsilon + \rho} \pi(\tilde{S}) + \left( \left( \frac{\beta\rho}{(\mu_a + \alpha)(\mu_j + \varepsilon + \rho)} - 1 \right) \pi'(\tilde{S}) - \mu_s'(\tilde{S}) \right) \frac{\partial\tilde{S}}{\partial\varepsilon} = 0. \quad (\text{A } 7)$$

Using identity (A 5) this equation can be rewritten as

$$\frac{(\mu_s(\tilde{S}) + \lambda)\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})}{\pi(\tilde{S})} \frac{\partial\tilde{S}}{\partial\varepsilon} = \frac{1}{\mu_j + \varepsilon + \rho} (\mu_s(\tilde{S}) + \lambda + \pi(\tilde{S})). \quad (\text{A } 8)$$

Application of the implicit function theorem leads to the following expression for the partial derivative  $\partial\tilde{S}/\partial\varepsilon$ :

$$\frac{\partial\tilde{S}}{\partial\varepsilon} = \frac{1}{\mu_j + \varepsilon + \rho} \frac{(\mu_s(\tilde{S}) + \lambda + \pi(\tilde{S}))\pi(\tilde{S})}{(\mu_s(\tilde{S}) + \lambda)\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})}. \quad (\text{A } 9)$$

Substitution of this expression into equation (A 1) and recognizing that for a predator of juvenile prey  $\lambda = 0$  and  $\alpha = 0$ , yields

$$\frac{\partial \tilde{f}}{\partial \varepsilon} = \frac{\beta}{\mu_a(\mu_i + \varepsilon + \rho)^2} \times \left( -\pi(\tilde{S})\tilde{S} + (\pi(\tilde{S})\tilde{S})' \frac{(\mu_s(\tilde{S}) + \pi(\tilde{S}))\pi(\tilde{S})}{\mu_s(\tilde{S})\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})} \right). \quad (\text{A } 10)$$

The parenthetical term of this expression can be rewritten as

$$\frac{(\pi(\tilde{S}))^2}{\mu_s(\tilde{S})\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})} (\pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S}'), \quad (\text{A } 11)$$

in which  $(\pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S})'$  is used as a shorthand notation for the derivative of the total outflow (maturation plus mortality) rate with respect to the subadult density  $\tilde{S}$  evaluated at the equilibrium subadult density  $\tilde{S}$ , i.e.

$$\left. \frac{d}{d\tilde{S}} (\pi(\tilde{S})\tilde{S} + \mu_s(\tilde{S})\tilde{S}) \right|_{\tilde{S}=\tilde{S}}$$

Substitution of expression (A 11) into equation (A 10) leads to the equation for  $\partial \tilde{f} / \partial \varepsilon$  (equation (3.14)).

Following an analogous derivation, differentiation of equation (A 3) with respect to  $\alpha$  leads to

$$\frac{\partial \tilde{S}}{\partial \alpha} = \frac{1}{\mu_a + \alpha} \frac{(\mu_s(\tilde{S}) + \lambda + \pi(\tilde{S}))\pi(\tilde{S})}{(\mu_s(\tilde{S}) + \lambda)\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})}. \quad (\text{A } 12)$$

For predators foraging on adult prey  $\varepsilon = 0$  and  $\lambda = 0$ . Substitution of expression (A 12) for  $\partial \tilde{S} / \partial \alpha$  in equation (A 2) thus leads to

$$\frac{\partial \tilde{A}}{\partial \alpha} = \frac{1}{(\mu_a + \alpha)^2} \times \left( -\pi(\tilde{S})\tilde{S} + (\pi(\tilde{S})\tilde{S})' \frac{(\mu_s(\tilde{S}) + \pi(\tilde{S}))\pi(\tilde{S})}{\mu_s(\tilde{S})\pi'(\tilde{S}) - \mu_s'(\tilde{S})\pi(\tilde{S})} \right). \quad (\text{A } 13)$$

Similar manipulations as discussed above for  $\partial \tilde{f} / \partial \varepsilon$  result in equation (3.15).

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