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E-Article

Food-Dependent Growth Leads to Overcompensation in Stage-Specific Biomass When Mortality Increases: The Influence of Maturation versus Reproduction Regulation

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abstract: We analyze a stage-structured biomass model for size-structured consumer-resource interactions. Maturation of juvenile consumers is modeled with a food-dependent function that consistently translates individual-level assumptions about growth in body size to the population level. Furthermore, the model accounts for stage-specific differences in resource use and mortality between juvenile and adult consumers. Without such differences, the model reduces to the Yodzis and Innes (1992) bioenergetics model, for which we show that model equilibria are characterized by a symmetry property that reproduction and maturation are equally limited by food density. As a consequence, biomass production rate exactly equals loss rate through maintenance and mortality in each consumer stage. Stage-specific differences break up this symmetry and turn specific stages into net producers and others into net losers of biomass. As a consequence, the population in equilibrium can be regulated in two distinct ways: either through total population reproduction or through total population maturation as limiting process. In the case of reproduction regulation, increases in mortality may lead to an increase of juvenile biomass. In the case of maturation regulation, increases in mortality may increase adult biomass. This overcompensation in biomass occurs with increases in both stage-independent and stage-specific mortality, even when the latter targets the stage exhibiting overcompensation.

Keywords: stage-structure, biomass overcompensation, population regulation, food-dependent growth, mortality increases.

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Mortality is the main dynamic process through which population abundances decline. Therefore, one generally expects increased levels of mortality to result in a decrease in density or biomass. Classical experiments suggest, however, that such an inverse relationship between mortality and abundance may be reversed if the mortality is not affecting all individuals of a population. For example, Nicholson (1957) showed that increased adult mortality increased adult density if blowfly populations were regulated by larval competition. In contrast, it increased the abundance of eggs, larvae, and pupae if the blowfly populations were regulated through adult competition. Similar experiments with *Daphnia pulicaria* show that an increase in mortality imposed on small *Daphnia* may result in larger densities of this particular size class (Slobodkin and Richman 1956). Compensating and possibly overcompensating responses—that is, that biomass densities do not decrease (compensation) or even increase (overcompensation) with increases in mortality—were also observed in experiments with *Tribolium* (Watt 1955), populations of blowflies under toxic stress with cadmium (Moe et al. 2002), and soil mites (Cameron and Benton 2004).

As pointed out by DeAngelis and Huston (1993), compensation and overcompensation are basic to many ecological processes. At an individual level, overcompensation in individual growth has been observed in fish and poultry after they were starved of food (Gurney et al. 2003). Similarly, plants exhibit overcompensation in seed production after herbivory (Agrawal 2000). At a within-population scale, overcompensation in maturation rate out of a particular age or size class with increasing mortality may occur (DeAngelis et al. 1993) when individuals are engaged in "scramble competition" (Nicholson 1954; Hassell 1975). At the population level, overcompensation in net primary production of plants may increase the total yield of plants in response to grazing (Dyer 1975; McNaughton 1979), while harvesting may increase the yield in exploited (fish) populations up to a maximum (the "maximum sustainable

yield"). More generally, overcompensation in production rate will occur in populations that are limited by intraspecific competition at high densities.

There are, however, essential differences between overcompensation in ecological process rates and overcompensation in (stage-specific) biomass densities precisely because one involves a rate while the other involves a density. Overcompensation in stage-specific maturation rate or population-level production depends on a release of intraspecific competition and therefore requires a decrease in stage-specific or total biomass density, respectively. Overcompensation in maturation rate may result in higher biomass densities in subsequent stages but will not necessarily do so, and it certainly precludes a positive relationship between mortality and biomass density of the same stage. In contrast, overcompensation in biomass density in response to stage-specific mortality has direct community consequences because it benefits predator populations that prey on the different life stages. For example, predators of newborn *D. pulicaria* may be expected to increase the density of their own prey individuals despite imposing predation mortality, given that newborn density has been found to increase with increasing mortality (Slobodkin and Richman 1956). De Roos and Persson (2002) have shown that this occurs in a model of a tritrophic food chain in which predators forage size-selectively on only the small individuals of a size-structured prey population. Size-selective predation on small prey individuals changes the prey size distribution: it leads to significant increases in biomass of small, newborn as well as adult prey, while it drastically reduces the biomass of larger juveniles. Higher densities of predators can bring about large changes in prey size distribution and therefore increase prey availability. In combination with size-selective predation, biomass overcompensation may thus lead to a positive feedback between predator density and predator persistence, which at the population level manifests itself as an Allee effect for the predator. Because not a single element of predator life history is positively dependent on predator density, this Allee effect is not a direct consequence of an individual-level mechanism and has therefore been dubbed an "emergent Allee effect" (De Roos and Persson 2002; De Roos et al. 2003).

Community consequences may also result when stage-specific biomass overcompensation occurs in response to increased mortality imposed on individuals in another stage. For example, if increased juvenile mortality leads to an increase in adult biomass, stage-specific predators that specialize on juveniles may increase the food availability for predators specializing on adults (De Roos and Persson 2005). Such juvenile-specialized predator species thereby facilitate the persistence of adult-specialized predator species. The tritrophic food-chain model with size-selective predation studied by De Roos and Persson (2002) illustrates this facilitation as the predation on small, newborn prey leads to a significant increase in adult-prey biomass. This type of facilitation is referred to as "emergent facilitation" (De Roos and Persson 2005) because it manifests itself only at the population level and is not a direct consequence of an individual-level mechanism.

The potential community consequences beg the question, under which conditions can overcompensation in stage-specific biomass occur in response to increases in mortality of individuals in either the same or another life-history stage. By analyzing a stage-structured consumer-resource model, which accounts in a mechanistic way for both food-dependent individual growth in body size and reproduction, we show that biomass overcompensation is likely to occur as soon as more than a single life-history process depends on food density and different life stages are food limited to an unequal extent. In Lotka-Volterra-type models, consumer reproduction is the only food-dependent process and represents the only possible target for food limitation in consumers. When, however, other life-history processes, such as growth and development, also depend on food density, it is rare that all of these processes will be simultaneously food limited to an equal extent. Generally, one of them will be limited more than the others. Such an asymmetry between different food-dependent life-history processes entails that in certain life stages, the competition will be stronger than in others. Overcompensation in juvenile biomass will occur when, at equilibrium, the population is mostly regulated by competition among adult individuals and food density limits reproduction more than maturation. In contrast, overcompensation in adult biomass occurs when competition among juveniles mostly regulates the population at equilibrium and food density limits maturation to a larger extent than reproduction. Overcompensation occurs independent of which life stage is subjected to increased mortality. It even occurs when the increased mortality affects all stages equally or targets only the stage exhibiting overcompensation. Stage-specific differences in competition for resources may therefore give rise to hump-shaped relationships between the equilibrium biomass in a particular stage and mortality.

Model Formulation and Parameterization

We analyze the response of a stage-structured consumer-resource model to increases in juvenile or adult mortality. The model is derived as an extension of the bioenergetic approach of Yodzis and Innes (1992) and is described by the following set of ordinary differential equations (ODEs; De Roos et al., forthcoming):

$$\frac{dR}{dt} = \delta(R_{\max} - R) - \frac{R}{H + R}(I_{\max}J + qI_{\max}A), \quad (1)$$

$$\frac{dJ}{dt} = \nu_a(R)A + \nu_j(R)J - \gamma(\nu_j(R))J - \mu_j J, \quad (2)$$

$$\frac{dA}{dt} = \gamma(\nu_j(R))J - \mu_a A. \quad (3)$$

The model describes the change in biomass of resource R, juvenile consumers J, and adult consumers A. In the absence of consumers, the resource R follows semichemostat dynamics, with turnover rate δ and maximum resource density $R_{\max}$. Semichemostat dynamics was assumed for reasons described in Persson et al. (1998). With semichemostat dynamics, resource productivity is constant and independent of resource density, as it equals $\delta R_{\max}$. Therefore, biomass overcompensation cannot occur as a consequence of changes in productivity. The resource is consumed by juveniles and adults following a Holling Type II functional response, with H representing the half-saturation constant of the consumers. The maximum juvenile and adult ingestion rates per unit biomass equal $I_{\max}$ and $qI_{\max}$, respectively. Ingested biomass is assimilated with an efficiency σ. The net biomass production per unit biomass for juveniles and adults, denoted by $\nu_j(R)$ and $\nu_a(R)$, respectively, equals the balance between ingestion and maintenance rate T:

$$\nu_j(R) = \sigma I_{\max}\frac{R}{H + R} - T, \quad (4)$$

$$\nu_a(R) = q\sigma I_{\max}\frac{R}{H + R} - T. \quad (5)$$

As in the Yodzis and Innes model, both ingestion and maintenance are body-mass-specific rates. Larger individuals, therefore, have higher ingestion and maintenance rates than smaller ones, and the differences are proportional to the difference in their body size. Furthermore, juvenile and adult ingestion rates differ by a factor q, the adult : juvenile intake ratio. De Roos et al. (forthcoming) introduce this factor to phenomenologically represent differences in resource exploitation that might occur between juveniles and adults for reasons other than a difference in body size. Notice that q is a body-mass-specific factor. Therefore, in case $q < 1$, the juveniles have a higher intake rate per unit body mass, but not necessarily a higher intake rate per individual, because of the larger body weight of adults.

Equation (2) shows that juvenile biomass increases through recruitment, $\nu_a(R)A$, and through the growth in body mass of individual juveniles, $\nu_j(R)J$. Juveniles are assumed to use their net-biomass production entirely for growth in body size. Maturation, $\gamma(\nu_j(R))J$, decreases juvenile biomass, as does juvenile stage-specific mortality, $\mu_j J$. Adult consumers invest their net-biomass production, $\nu_a(R)$, entirely into reproduction and therefore are assumed not to grow. Adult biomass therefore only increases through maturation of juveniles, $\gamma(\nu_j(R))J$, and decreases through adult stage-specific mortality, $\mu_a A$ (eq. [3]). The maturation function $\gamma(\nu_j(R))$ is food dependent through its dependence on juvenile net-biomass production. It constitutes the unique feature of the model and is given by

$$\gamma(\nu_j(R)) = \frac{\nu_j(R) - \mu_j}{1 - z^{1-\mu_j/\nu_j(R)}}. \quad (6)$$

The parameter z represents the ratio of the consumer body size at birth to its size at maturation. Expression (6) shows that maturation depends on biomass production $\nu_j(R)$, juvenile mortality μ_j, and the size range z over which an individual grows as a juvenile. Both the numerator and denominator vanish in expression (6) when $\nu_j(R) = \mu_j$, but straightforward analysis shows that the function $\gamma(\nu_j(R))$ has a regular limit $-1/\ln(z)$ for $\nu_j(R) = \mu_j$. When z is close to 1, consumers are born at a size close to the maturation size, which effectively removes the stage structure from the model. When the ratio is small, a consumer needs to grow considerably in body size before it matures, resulting in a long juvenile period.

The stage-structured biomass model (eqq. [1]–[3]) constitutes a low-dimensional reformulation in terms of ordinary differential equations of a physiologically structured population model (Metz and Diekmann 1986; De Roos 1997), which accounts for a continuous size distribution of consumers between their birth and maturation size. This continuous size-structured model is based on a core model of a size-dependent individual life history, including size-dependent foraging and food-dependent individual growth as well as size-dependent maturation and reproduction. De Roos et al. (forthcoming) show that the stage-structured model is an exact reformulation of the continuous size-structured model under equilibrium conditions and approximates its dynamics otherwise. Therefore, even though the stage-structured model is formulated in terms of only two differential equations for the biomass dynamics in two stages, it does in fact describe the dynamics of a continuous consumer-size distribution. Juvenile consumers range in size between the size at birth and the size at maturation. The latter equals the body size of all adult consumers, as they are assumed to have stopped growing after maturation. The identity between the two models under equilibrium conditions dictates the form of the mat-

uration function $\gamma(\nu_j(R))$ as given in equation (6). This function consistently translates the individual-level assumption that juveniles use all their net-biomass production for growth in body size to the population level. It therefore takes into account how juvenile mortality (μ_j) and juvenile growth ($\nu_j(R)$) shape the size distribution of juvenile consumers between the size at birth and maturation and thus determine the duration of the juvenile period. For the derivation of the maturation term from its individual-level assumptions on growth and development, we refer to De Roos et al. (forthcoming). These authors present the stage-structured model in a slightly extended form to also account for dynamics under starvation conditions (i.e., when either $\nu_j(R) < 0$ or $\nu_a(R) < 0$). Since such starvation conditions are irrelevant for the results presented here, we only consider the basic version of the model. Notice that in the absence of any differences in mortality and resource use between the stages—that is, when $q = 1$, $\mu_j = \mu_a$, and $\nu_j(R) = \nu_a(R)$—the ODEs (2) and (3) for the dynamics of juvenile and adult biomass can be summed into in a single ODE for the total consumer biomass. Without stage-specific differences, the stage-structured biomass model therefore simplifies to its unstructured analogue, the consumer-resource model developed by Yodzis and Innes (1992).

Parameterization

The parameters H and T can be set equal to 1 without loss of generality. These choices merely represent a scaling of the volume in which we consider the consumer-resource system to exist and a scaling of the time variable, respectively (De Roos et al., forthcoming). After scaling, the model dynamics depend on the four rate parameters δ/T, I_{max}/T, μ_j/T, and μ_a/T, which are expressed as ratios over the mass-specific maintenance rate T. Since the dimension of all five rate parameters—δ, I_{max}, T, μ_j, and μ_a—in the model is per unit time, these four ratios are dimensionless (in fact, because they are mass specific, maximum ingestion and metabolic rate are specified in unit biomass per unit biomass per unit time). Furthermore, dynamics depend on the maximum resource density R_{max}, the conversion efficiency σ, and the dimensionless parameters z and q. We adopt the default values $R_{max} = 5$ and $\delta/T = 1$ for the maximum resource density and the (scaled) resource turnover rate, respectively. The latter value implies that resource turnover rate is approximately equal to the turnover rate of consumer biomass through maintenance. We verified that different choices for R_{max} and δ do not qualitatively change model predictions and only result in proportional scaling of the juvenile and adult biomass at equilibrium. Across differently sized consumer species, mass-specific metabolic rate, mass-specific maximum ingestion rate, and mortality rate (here and below expressed per day) tend to be proportional to the quarter power of adult body size of the species (Peters 1983; Yodzis and Innes 1992; Brown et al. 2004). For the mass-specific maximum ingestion rate, an estimate of 0.13 for the proportionality constant in this quarter-power scaling relation can be derived from data on zooplankton grazing rates presented by Hansen et al. (1997). Peters (1983) estimates the proportionality constant for the mass-specific metabolic rate scaling to equal 0.01, which is in line with estimates provided by Yodzis and Innes (1992) and Brown et al. (2004). Together, these two estimates lead to a default value $I_{max}/T = 13$ for the ratio between the mass-specific maximum ingestion rate and the mass-specific metabolic rate. Gillooly et al. (2001) provide an estimate for the proportionality constant in the quarter-power scaling law of mortality rate equal to 0.001. We therefore assume as default values for juvenile and adult mortality $\mu_j/T = \mu_a/T = 0.1$. The ratio between newborn and maturation size we estimated from data on egg and adult weight for a large number of marine copepod species (Huntley and Lopez 1992). These data show the ratio to range between 0.001 and 0.02 with a median value of 0.01. As default value we therefore adopt $z = 0.01$. For assimilation efficiency we assume as default value $\sigma = 0.7$ (Peters 1983; Yodzis and Innes 1992). Finally, we will investigate model dynamics while varying the value of q representing the ratio between juvenile and adult maximum ingestion rate. An estimate for this parameter can hardly be derived from experimental data as it is only a phenomenological representation of stage-specific differences in resource availability and resource use of juveniles and adults, respectively.

Model Analysis

Explicit expressions for the equilibrium juvenile, adult, and resource density cannot be obtained. It can be proven that for a particular set of parameters, the equilibrium densities of resource, juvenile, and adult biomass are unique, if the maximum resource density R_{max} is sufficiently high for consumers to persist. To compute these equilibrium densities as a function of model parameters, we used Content (Kuznetsov et al. 1996), an interactive software package for numerical bifurcation analysis of dynamic systems (see Kuznetsov 1995 for an introduction and overview). This software package was also used to assess the stability of the consumer-resource equilibrium. For all parameter combinations discussed in this article, the model never predicted any limit cycles to occur (De Roos et al., forthcoming). We consider the effect of varying the adult-juvenile intake ratio q on the consumer-resource equilibrium. Furthermore, we investigate the consequences of increasing juvenile mortality (μ_j/T), adult mortality ($\mu_a/$

T), or stage-independent mortality. The last is represented by a simultaneous and identical increase in both μ_j/T and μ_a/T from their default values.

Results

Figures 1 and 2 summarize the main results of our model analysis: if the population at equilibrium is mostly regulated through strong competition for resources among adult consumers, increases in mortality increase juvenile biomass (fig. 1). The overcompensation in juvenile biomass occurs irrespective of the type of mortality that is increased; even increased mortality of juveniles themselves results in an increase in juvenile biomass. In contrast, if in equilibrium the population is mostly regulated through resource competition among juveniles, increases in mortality result in an increase in adult biomass (fig. 2). Overcompensation in adult biomass also occurs irrespective of the type of mortality that is increased, including increases in adult mortality. Any increase in mortality, be it stage-specific or not, always increases the resource biomass and decreases overall consumer biomass in equilibrium (results not shown). However, mortality also influences the juvenile : adult ratio in the consumer population. The changes in the juvenile : adult ratio with increasing mortality result in increases in either juvenile or adult biomass, as shown in figures 1 and 2, despite the fact that total consumer biomass decreases with mortality.

Strong competition for resources among adult consumers implies that food density limits reproduction more than maturation. Populations that in equilibrium are regulated by adult competition we will therefore refer to as "reproduction regulated." By analogy, populations that in equilibrium are regulated by juvenile competition we will refer to as maturation regulated, since competition among juveniles entails that maturation is more food limited than reproduction. These two distinct modes of population regulation not only determine which type of biomass overcompensation will occur, they also have implications for the life stage that dominates the population at equilibrium. In addition, the mode of population regulation determines which life stage is a net-biomass-production stage and which is a net-biomass-loss stage. Table 1 summarizes the characteristics of the two modes of population regulation, which we discuss in more detail below. Furthermore, we analyze which combinations of mortality and ingestion rates result in which type of regulation and how the different modes of regulation give rise to overcompensation in either juvenile or adult biomass.

Reproduction versus Maturation Regulation

In equilibrium, the right-hand sides of the ODEs (2) and (3) both equal 0. Summing these expressions leads to the following equilibrium condition, which is crucial for distinguishing reproduction from maturation regulation:

$$[\nu_j(R^*) - \mu_j]J^* = -[\nu_a(R^*) - \mu_a]A^*. \tag{7}$$

This equality expresses that if in equilibrium a net gain

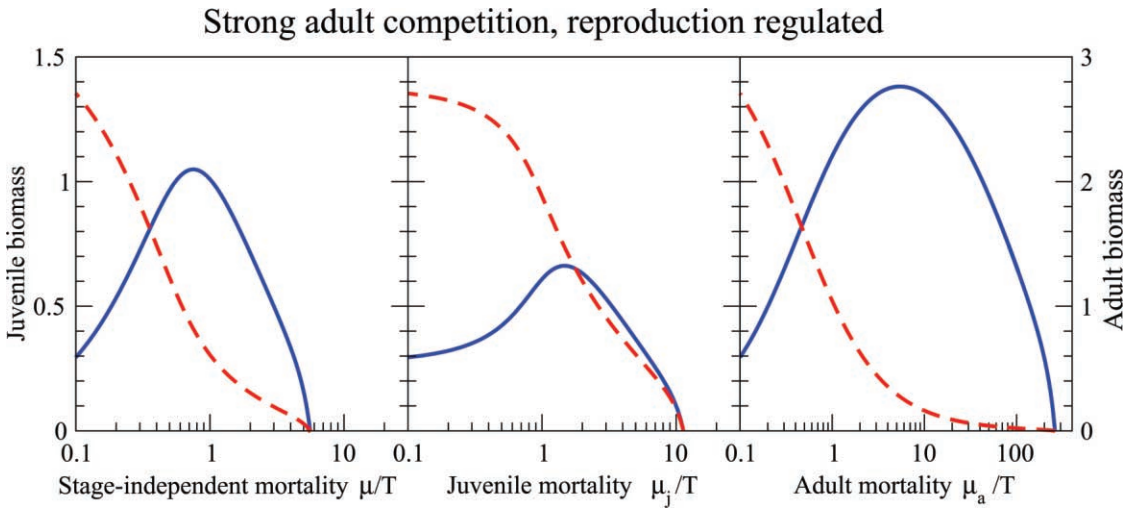

Figure 1: Changes in equilibrium juvenile (*blue*, *solid*) and adult (*red*, *dashed*) biomass with increases in mortality in case the population is reproduction regulated ($q = 0.5$). *Left*, increases in scaled, stage-independent mortality $\mu_j/T = \mu_a/T$. *Middle*, increases in scaled juvenile mortality rate μ_j/T. *Right*, increases in scaled adult mortality rate μ_a/T. All start from $\mu_j/T = \mu_a/T = 0.1$. Parameters: $z = 0.01$; all other parameters have default values.

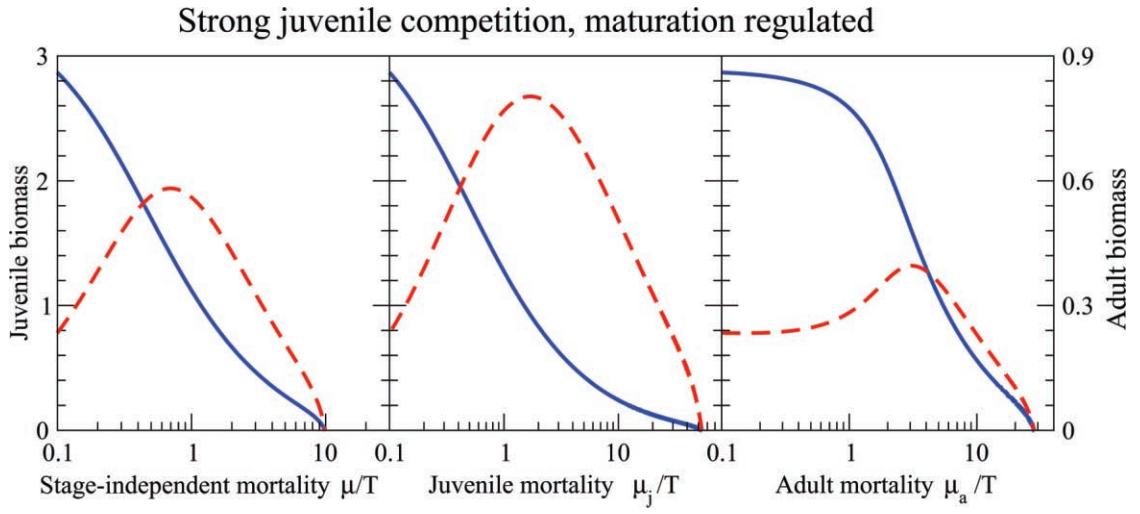

Figure 2: Changes in equilibrium juvenile (*blue*, *solid*) and adult (*red*, *dashed*) biomass with increases in mortality in case the population is maturation regulated ($q = 2.0$). *Left*, increases in scaled, stage-independent mortality $\mu_j/T = \mu_a/T$. *Middle*, increases in scaled juvenile mortality rate μ_j/T. *Right*, increases in scaled adult mortality rate μ_a/T. All start from $\mu_j/T = \mu_a/T = 0.1$. Parameters: $z = 0.5$; all other parameters have default values.

in biomass occurs in one stage, a net loss of equal magnitude occurs in the other. If juveniles and adults do not differ in their mortality and their competition for resources (i.e., $q = 1$, $\mu_j = \mu_a$ and $\nu_j(R^*) = \nu_a(R^*)$), equation (7) can only be satisfied for nonzero equilibrium densities of juveniles and adults if both terms in brackets vanish, which implies that $\nu_j(R^*) = \mu_j$ and $\nu_a(R^*) = \mu_a$. Therefore, in the absence of any stage-specific differences, the equilibrium net-biomass production in each stage exactly equals the biomass loss in equilibrium through mortality in that stage. Both stages are therefore zero net producers of biomass. Any difference between μ_j and μ_a and any value of q unequal to 1 will break up this symmetry between stages. To satisfy equation (7) for nonzero equilibrium densities in that case, the terms in brackets must have opposite signs. Thus, stage-specific differences in resource competition and/or mortality generically give rise to two distinct types of equilibrium situations: either the juvenile stage is a net-biomass-production stage while the adult stage is a net-loss stage, or, vice versa, the adult stage is a net-biomass-production stage while the juvenile stage is a net-loss stage.

The juvenile stage is a net-biomass-production stage in equilibrium if $\nu_j(R^*) > \mu_j$, which occurs when juveniles have either a higher feeding rate ($q < 1$) or a lower mortality rate ($\mu_j < \mu_a$) than adults. Equation (7) dictates that to compensate for the net gain of biomass in the juvenile stage, the adult stage must be a net-loss stage, and therefore $\nu_a(R^*) < \mu_a$. From equation (3) we can infer that in equilibrium, the population-level maturation rate equals the total adult mortality rate: $\gamma(\nu_j(R^*))J^* = \mu_a A^*$. Using this identity, the inequality $\nu_a(R^*) < \mu_a$ can be reformulated as

$$\nu_a(R^*)A^* < \gamma(\nu_j(R^*))J^*. \tag{8}$$

Equation (8) expresses that in equilibrium, biomass leaves the juvenile stage through maturation at a faster rate than it enters through reproduction. The deficit in juvenile biomass turnover is compensated for by somatic growth of juveniles, which exceeds biomass losses through mortality. We will refer to consumer populations, for which in equilibrium inequality (8) holds, as regulated by reproduction, because the population reproduction rate, $\nu_a(R^*)A^*$, limits biomass turnover in the population more than the population maturation rate, $\gamma(\nu_j(R^*))J^*$. As explained above, reproduction regulation is intimately tied to the fact that the adult stage is a net-biomass-loss stage, while the juvenile stage is a net-biomass-production stage (table 1).

In contrast, the adult stage is a net-biomass-production stage in equilibrium when $\nu_a(R^*) > \mu_a$, which occurs when adults have either a higher feeding rate ($q > 1$) or a lower mortality rate than juveniles ($\mu_a < \mu_j$). In this case, the juvenile stage is a net-loss stage, $\nu_j(R^*) < \mu_j$ (eq. [7]), and the population reproduction and maturation rates obey the inequality

$$\nu_a(R^*)A^* > \gamma(\nu_j(R^*))J^*. \tag{9}$$

The rate at which biomass enters the juvenile stage through reproduction is therefore larger than the rate at

Table 1: Characteristics and conditions of the two modes of population regulation, including changes in biomass with increasing mortality

Type of regulation	Relationship population reproduction/ maturation rate	Net-biomass-production stage	Net-biomass-loss stage	Most abundant, strongly competing life stage	Changes with increasing mortality
Reproduction regulation	Reproduction < maturation ($\nu_a(R^*)A^* < \gamma(\nu_j(R^*))J^*$)	Juveniles ($\nu_j(R^*) > \mu_j$)	Adults ($\nu_a(R^*) < \mu_a$)	Adults	Juvenile biomass increases, adult biomass decreases (fig. 1)
Maturation regulation	Reproduction > maturation ($\nu_a(R^*)A^* > \gamma(\nu_j(R^*))J^*$)	Adults ($\nu_a(R^*) > \mu_a$)	Juveniles ($\nu_j(R^*) < \mu_j$)	Juveniles	Adult biomass increases, juvenile biomass decreases (fig. 2)

which biomass leaves the juvenile stage through maturation. There is now an overproduction of biomass in the adult stage through reproduction, compensated for by the more limiting juvenile growth and maturation process. We will refer to populations for which in equilibrium inequality (9) holds as regulated by maturation. Maturation regulation implies that the adult stage is a net-biomass-production stage, while the juvenile stage is a net-biomass -loss stage (table 1).

Figure 3 illustrates the change from reproduction to maturation regulation in the stage-structured biomass model by presenting the population maturation and reproduction rates at equilibrium as a function of q for equal juvenile and adult mortality, $\mu_j = \mu_a$. The two rates equal each other—identifying the switch from reproduction to maturation regulation—for $q = 1$ when, in addition to equal mortality, there are no differences in resource competition between juveniles and adults. For $q < 1$, juveniles are better resource competitors than adults. Adults therefore suffer more from competition than juveniles such that the population is reproduction regulated. In contrast, for $q > 1$, adults are better competitors for resources and juveniles suffer more from competition. As a consequence, the population is maturation regulated. The type of population regulation also determines which life stage dominates the population at equilibrium. When the population reproduction rate is smaller than the maturation rate, juveniles mature quickly, and the population is dominated by adults. This occurs for small values of q when the adult stage is a net-biomass-loss stage. Juveniles dominate the population for large values of q, when the population maturation rate is small compared with the reproduction rate and the juvenile stage is the net-biomass-loss stage. The changes in equilibrium juvenile and adult biomass with q thus parallel the changes in population reproduction and maturation rate (fig. 3), although equal biomass densities of juveniles and adults occur at a value of around $q = 0.85$ (for default parameters). The ratio between juvenile and adult biomass shows particularly rapid changes around this point.

Summarizing, the mode of population regulation in equilibrium is determined by the relationship between the population reproduction rate and the population maturation rate (inequalities [8] and [9]), which takes into account differences between juveniles and adults in competition for resources as well as mortality. Whether a population is regulated through reproduction or maturation also determines whether the juvenile or adult stage is a net-biomass-production stage, whether competition among adults or juveniles is more intense, and whether adults or juveniles dominate the population in equilibrium (table 1).

Increases in Mortality under Reproduction Regulation

If the population in equilibrium is regulated through reproduction and therefore dominated by adults, an increase in mortality translates into an increase in juvenile biomass (fig. 1). For $q = 0.5$, the overcompensating response in juvenile biomass reaches a maximum when mortality is more than an order of magnitude larger than the background mortality levels of $\mu_j/T = \mu_a/T = 0.1$ (fig. 1). We define the strength of the overcompensation as the ratio between the maximum in the hump-shaped biomass-mortality relationship and the biomass when juvenile and adult mortality are equal to background levels. For otherwise identical parameters, the overcompensation is strongest with increases in adult mortality and weakest with increases in juvenile mortality.

From equation (7), we can derive the following expression for the fraction of juvenile biomass in the consumer population at equilibrium:

$$\frac{J^*}{J^* + A^*} = \frac{\mu_a - \nu_a(R^*)}{[\mu_a - \nu_a(R^*)] + [\nu_j(R^*) - \mu_j]}. \qquad (10)$$

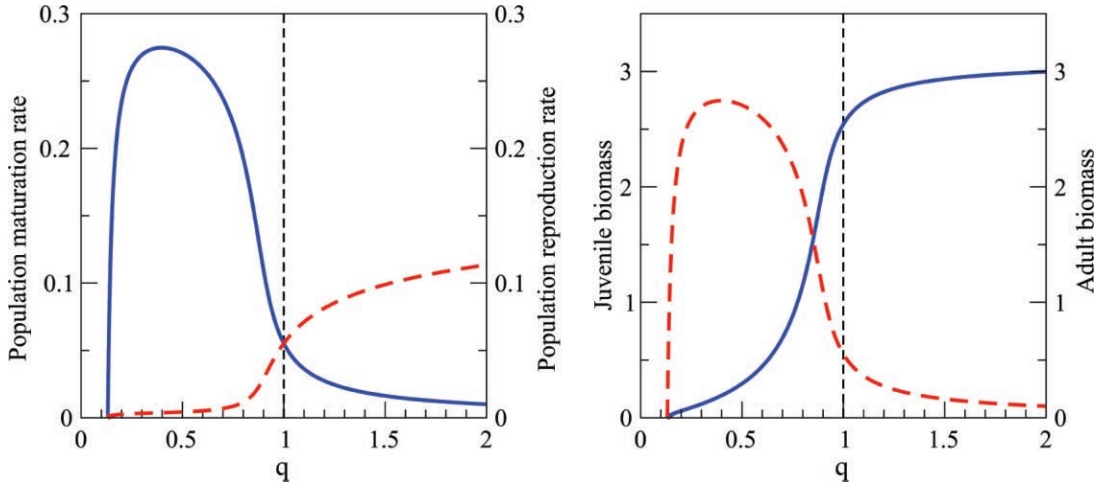

Figure 3: *Left*, population maturation rate, $\gamma(\nu_j(R))J$ (*blue*, *solid*), and population reproduction rate, $\nu_a(R)A$ (*red*, *dashed*), as a function of the adult-juvenile intake ratio q. *Right*, equilibrium juvenile (*blue*, *solid*) and adult (*red*, *dashed*) biomass as a function of the adult-juvenile intake ratio q. Juvenile and adult mortality equal $\mu_j/T = 0.1$ and $\mu_a/T = 0.1$, respectively. Parameters: $z = 0.01$; all other parameters have default values.

Because reproduction regulation implies that the juvenile and adult stages are a net-biomass-production and net-biomass-loss stage, respectively (i.e., $\nu_j(R^*) > \mu_j$ and $\nu_a(R^*) < \mu_a$; table 1), all terms in brackets in the above expression are positive. We can therefore infer that increases in μ_j as well as μ_a increase the right-hand side of the above expression and therefore lead to an increase in the fraction of juvenile biomass. These changes in the fraction of juvenile biomass with increasing mortality translate into overcompensation in juvenile biomass as long as the increased mortality does not lead to a strong decline in total consumer biomass (fig. 1).

An increase in juvenile mortality reduces the juvenile net-biomass production, $\nu_j(R^*) - \mu_j$, and eventually turns the juvenile stage into a net-biomass-loss stage. Therefore, it reduces the population maturation rate and increases the population reproduction rate (fig. 4, *left panel*) and may ultimately induce a shift from reproduction to maturation regulation of the equilibrium altogether. Increases in juvenile mortality for $q < 1$ therefore have a comparable effect on the equilibrium as increases in q, with equal juvenile and adult mortality (fig. 3). Under reproduction regulation, increases in μ_a increase the net-biomass loss, $\mu_a - \nu_a(R^*)$, in the adult stage and therefore do not change the type of population regulation in equilibrium. The fraction of juvenile biomass increases in this case because adults die off more rapidly. When both juvenile and adult mortality are increased simultaneously, the juvenile fraction in the population increases through a combination of these effects. At low background mortality rates of $\mu_j/T = \mu_a/T = 0.1$, the juvenile : adult ratio is primarily determined by the difference in juvenile and adult intake rate. In contrast, when both μ_j and μ_a are increased simultaneously, the difference in net-biomass production of juveniles and adults, $\nu_j(R^*) - \mu_j$ and $\nu_a(R^*) - \mu_a$, respectively, depends more and more on these mortality rates. Increasing stage-independent mortality therefore tends to make the net-biomass-production rates of juveniles and adults more similar and equalizes the juvenile : adult biomass ratio in the population. An increase in stage-independent mortality can, however, never change the fact that the equilibrium is regulated by reproduction.

Increases in Mortality under Maturation Regulation

If the population in equilibrium is regulated through maturation and therefore dominated by juveniles, an increase in mortality translates into an increase in adult biomass (fig. 2). The overcompensation in adult biomass is strongest with increases in juvenile mortality and weakest with increases in adult mortality (fig. 2). With increases in adult mortality, adult biomass may first decrease slightly but thereafter increases to levels above the adult biomass at equal background mortality of $\mu_j/T = \mu_a/T = 0.1$ (fig. 2, *right panel*).

In case of maturation regulation, the fraction of adult biomass in the population can be expressed as

$$\frac{A^*}{J^* + A^*} = \frac{\mu_j - \nu_j(R^*)}{[\mu_j - \nu_j(R^*)] + [\nu_a(R^*) - \mu_a]}. \qquad (11)$$

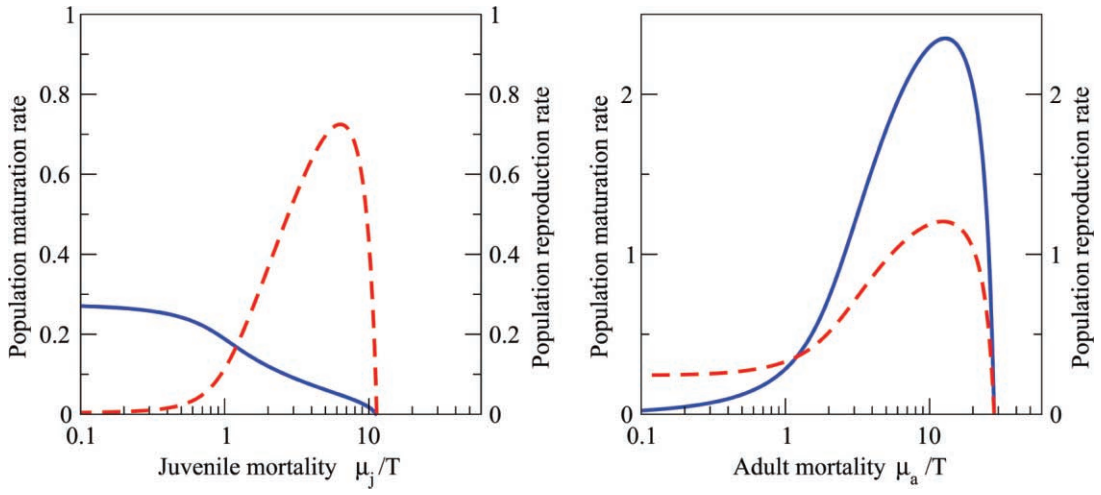

Figure 4: Population reproduction rate (*red, dashed*) increases with increasing juvenile or adult mortality, except when mortality rates are close to extinction levels. Population maturation rate (*blue, solid*), however, may increase (*right*) or decrease (*left*). Parameter values: $q = 0.5$, $z = 0.01$, and $\mu_a/T = 0.1$ (*left*; cf. fig. 1, *middle panel*); and $q = 2.0$, $z = 0.5$, and $\mu_j/T = 0.1$ (*right*; cf. fig. 2, *right panel*). All other parameters have default values.

All terms in brackets in this expression are positive given that maturation regulation implies that the juvenile and adult stages are a net-loss and a net-production stage ($\nu_j(R^*) < \mu_j$ and $\nu_a(R^*) > \mu_a$; table 1), respectively. The expression makes clear that increases in both μ_j and μ_a increase the adult fraction in the population. Adult biomass increases with increasing mortality for similar reasons as juvenile biomass increases in case of reproduction regulation. An increase in adult mortality reduces the adult net-biomass production, $\nu_a(R^*) - \mu_a$, and eventually turns the adult stage into a net-biomass-loss stage. Therefore, it induces a shift from maturation to reproduction regulation of the equilibrium, as also occurs when lowering q from $q > 1$ to $q < 1$ (fig. 3). Accordingly, the ratio between population reproduction and maturation rates increases with increasing μ_a/T (fig. 4, *right panel*), even though in absolute terms both rates increase. The shift from maturation to reproduction regulation leads to the increase in equilibrium adult biomass with increased adult mortality. Increases in μ_j do not change the type of population regulation in equilibrium, as they only increase the net-biomass loss, $\mu_j - \nu_j(R^*)$, during the juvenile stage. The changes in the juvenile : adult ratio now come about because juveniles die off more rapidly. Simultaneous increases in both juvenile and adult mortality combine these two effects without changing the type of population regulation in equilibrium. Such increases therefore also increase the adult fraction in the population and in addition equalize the net-biomass production of juveniles and adults.

Length of Juvenile Stage and Background Mortality

The occurrence of hump-shaped biomass relationships with increasing mortality depends on the ratio between newborn and adult weight z and the background mortality experienced by both juveniles and adults. The results with stage-independent mortality (figs. 1, 2, *left panels*) already show that for background mortalities above $\mu/T = 1$, increasing mortality further only decreases juvenile and adult biomass. We have found this effect to occur independently of the type of mortality that is increased: with higher values of background mortality, the range of mortality rates over which biomass of either juveniles or adults increases is reduced.

For $z = 0.01$ and $q = 0.5$, the maximum in the humped-shaped relationship between juvenile biomass and juvenile mortality is 2 to 2.5 times larger than juvenile biomass at a background mortality of $\mu_j/T = \mu_a/T = 0.1$ (fig. 1, *middle panel*). Halving the background mortality rate increases this factor to 3.5, while increasing the background mortality by a factor of 5 makes the overcompensation disappear altogether. Overcompensation is therefore both more common and more pronounced at lower background mortality. The neonate : adult weight ratio z affects overcompensation in a similar way: smaller values of z increase the ratio between the maximum in the humped-shaped relationship between juvenile biomass and mortality compared with the juvenile biomass at background mortality levels. The overcompensation in juvenile biomass due to increased mortality is therefore stronger

when the juvenile stage covers a larger fraction of the consumer life cycle.

The effect of increased background mortality on overcompensation in adult biomass is similar to that for juveniles: With $z = 0.5$ and $q = 2.0$, the maximum in the humped-shaped relationship between adult biomass and adult mortality is 1.5 to 2 times larger than adult biomass at a background mortality rate of $\mu_j/T = \mu_a/T = 0.1$ (fig. 2, *right panel*). For these parameters, the overcompensation disappears if background mortality is increased with a factor of 2.5. The effect of z on overcompensation in adult biomass is opposite to its effect on juvenile biomass overcompensation: adult biomass overcompensation in response to increased adult mortality is more pronounced when z is close to 1, that is, when the adult stage covers a larger fraction of the life cycle.

We numerically determined the values of the neonate : adult weight ratio z and background mortality at which the maximum in the equilibrium juvenile or adult biomass curve as a function of mortality exactly equals the value at background mortality levels. This combination of z and background mortality represents the threshold below which overcompensation occurs with an increase in mortality (see app. A for computational details). Figure 5 shows these thresholds as a function of z and background mortality, whereby overcompensation occurs for all parameter combinations to the left of a particular boundary. The thresholds differ for increases in juvenile, adult, and stage-independent mortality, and therefore figure 5 shows three boundaries for both cases of reproduction regulation ($q = 0.5$) and maturation regulation ($q = 2.0$). First, we find that with reproduction regulation ($q = 0.5$), the parameter regions for which overcompensation occurs are largest for increases in adult mortality and smallest for increases in juvenile mortality. With maturation regulation ($q = 2.0$), exactly the opposite is the case. Second, with reproduction regulation ($q = 0.5$), a larger value of the neonate : adult weight ratio z requires lower background mortality for the occurrence of overcompensation, as was already argued above. For maturation regulation ($q = 2.0$), it is exactly the other way around, except for increases in juvenile mortality, for which the boundary is virtually independent of the neonate : adult weight ratio z.

Discussion

In Lotka-Volterra-type consumer-resource models, consumer reproduction is the only food-dependent process and represents the only possible target for food limitation in consumers. When other life-history processes, such as growth and development, also depend on food density, only in rare cases, all of these processes will be simultaneously food-limited to an equal extent. Generally, one of them will be limited more than the others. The direct consequence of such an asymmetry between different food-dependent life-history processes is that in equilibrium, certain life stages will be net-biomass-production stages while others will be net-biomass-loss stages. This

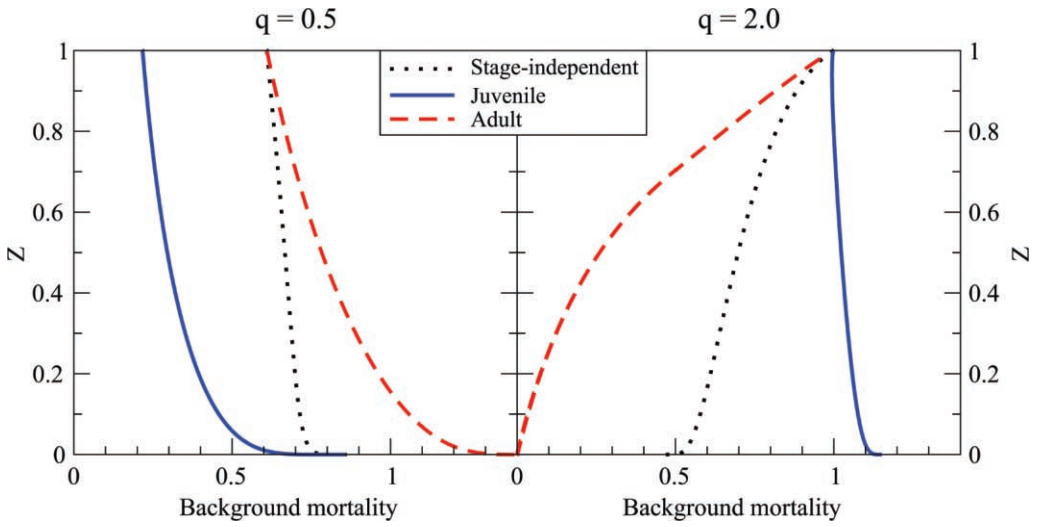

Figure 5: Boundaries delineating the parameter regions of the neonate : adult weight ratio z and background mortality, for which overcompensation in biomass occurs. Boundaries differ for increases in juvenile (*blue*, *solid lines*), adult (*red*, *dashed lines*), and stage-independent mortality (*black*, *dotted lines*). Overcompensation occurs for parameters to the left of a particular boundary. *Left*, reproduction regulation ($q = 0.5$) leading to increases in juvenile biomass. *Right*, maturation regulation ($q = 2.0$) leading to increases in adult biomass. All other parameters have default values.

asymmetry constitutes the fundamental difference between size-structured population models with food-dependent growth and unstructured, Lotka-Volterra-type models or even age-structured population models. Predictions of our stage-structured biomass model therefore differ from predictions of its unstructured analogue, the Yodzis and Innes (1992) model, precisely because of this fact: in equilibrium, stages may differ in their net-biomass-production rate and are not necessarily zero-net-biomass producers. We have shown that if reproduction is more limited than maturation, increases in mortality may increase juvenile biomass, even when juvenile mortality is increased. In our model, these conditions occur for $q < 1$. Similarly, if food density limits maturation more than reproduction, increasing mortality—be it juvenile, adult, or stage-independent mortality—will increase adult biomass. This occurs in our model for $q > 1$. Stage-specific differences in resource use therefore give rise to hump-shaped relationships between the equilibrium biomass in a particular stage and mortality, even if the latter is stage independent.

Our choice to model stage-specific differences in resource availability and resource use with a single parameter q, representing a ratio between adult and juvenile maximum ingestion rate per unit biomass, was merely a strategic one. A model including separate resources for juveniles and adults would account more explicitly and mechanistically for such stage-specific differences in resource use, but for ease of analysis, we chose to avoid this model complexity. We verified, however, that our simple representation of the competitive differences between juveniles and adults does not qualitatively change the results. In fact, in appendix B, we show that qualitatively, the same results as shown in figure 1—a hump-shaped relation between juvenile biomass and mortality—are obtained in the case in which we assume that juveniles have access to an unlimited food supply and therefore grow and develop at the maximum rate, while the population is regulated entirely through exploitative competition among adults for a shared dynamic resource. Obviously, the population is reproduction regulated in this case. Qualitatively, the same results as shown in figure 2—a hump-shaped relation between adult biomass and mortality—can be obtained analogously by assuming that adults have access to an unlimited food supply and therefore reproduce at the maximum rate, while juveniles compete for a shared dynamic resource, leading to a population that is maturation regulated (app. B). Biomass overcompensation may thus occur because of different mechanisms. First of all, it may result because of intraspecific interactions, when juveniles and adults forage on a shared resource but differ in their foraging capacity and are therefore engaged in asymmetric interstage competition. Alternatively, juveniles and adults may not interact or compete with each other but may be food limited to an unequal extent because they occupy separate niches that differ in profitability. Both mechanisms can give rise to the asymmetry in net-biomass production between juveniles and adults that induces the biomass overcompensation in response to increasing mortality.

Even though the stage-structured biomass model contains dynamic equations for only two stages, juveniles and adults, it does account for a continuous size distribution of consumers ranging from the size at birth to the size at maturation (De Roos et al., forthcoming). This low-dimensional representation of a continuous size distribution is only possible if we follow Yodzis and Innes in their assumption that intraspecifically, consumer ingestion and maintenance are proportional to consumer body size (Yodzis and Innes 1992, p. 1154). Note that this assumption is distinct from but easily confused with their assumption that among different species, the mass-specific rates of ingestion and maintenance scale with a power -0.25 of the species body mass (Yodzis and Innes 1992, pp. 1155–1156). This necessary assumption is obviously an idealization, as for both rates, intraspecific size-scaling exponents smaller than 1 have been reported (Peters 1983; Brown et al. 2004). Whether or not a population in equilibrium is maturation or reproduction regulated may depend on these size-scaling exponents of ingestion and maintenance. A decrease in mass-specific maintenance requirements with increasing body size would imply that adults are more competitive than juveniles, which makes it more likely that a population is regulated through maturation. In contrast, a decrease in mass-specific ingestion with increasing body size would make it more likely that a population is regulated through reproduction, as it would imply a competitive advantage of juveniles over adults. As a further complication, we have shown that the mode of population regulation may also be determined by differences in the food supply to juveniles and adults, in case they occupy different niches (app. B). A priori, it therefore seems impossible to draw general conclusions about which mode of population regulation is more likely to occur. Gurney et al. (1996) investigated the equilibrium demography of consumer populations as predicted by four different models of individual energetics. At low background mortality, the juvenile delay tended to vary rapidly with changes in mortality, whereas adult reproduction remained roughly constant. These model analyses therefore suggest that consumer populations are likely to be regulated through maturation. Maturation regulation also seems the rule in populations that combine a high reproductive potential with small offspring size, such as many fish populations. Typically, these populations develop a stunted size distribution dominated by large densities of slow-growing

juveniles, if they are not exposed to any (predation) mortality (Tonn et al. 1992; Ridgway and Chapleau 1994). Maturation regulation may thus be more common than reproduction regulation, despite the fact that most population dynamic models ignore this type of regulation altogether.

Increasing equilibrium densities of predators with increasing predator mortality were already reported by Rosenzweig and MacArthur (1963) for a predator-prey model with logistic prey growth and a saturating predator functional response. This phenomenon was later labeled the "hydra" effect by Abrams and Matsuda (2005; see also Matsuda and Abrams 2004; Abrams and Quince 2005). Although the hydra effect and the biomass overcompensation studied here seem superficially similar, the underlying mechanisms behind these two phenomena are different. The hydra effect arises because prey productivity at equilibrium increases with increasing predator mortality. In contrast, biomass overcompensation in the stage-structured biomass model is not based on such an increase in resource productivity but on a redistribution of consumer biomass over the different stages. A prerequisite for this redistribution of biomass to occur is that different life-history processes (maturation, reproduction) are food limited to unequal extents and therefore respond differentially to increases in mortality and the ensuing increases in food density (fig. 4). Such increases in population-level rates due to population thinning are especially to be expected when individuals are engaged in scramble competition (Nicholson 1954; Hassell 1975).

Model Predictions and Empirical Support

A number of Nicholson's (1957) blowfly experiments show some of the predicted compensatory effects of stage-specific mortality on abundances of different life-history stages. Nicholson first of all showed that switching a blowfly population from being regulated by larval competition (maturation regulation) to being regulated by adult competition (reproduction regulation) has the effect of approximately quadrupling the adult density. Relaxing adult competition by a change from a limited to an unlimited sugar supply was already sufficient to decrease average adult density. These observations correspond with the model prediction that a switch from maturation regulation to reproduction regulation will significantly increase adult biomass and decrease juvenile biomass (fig. 3). In blowfly populations regulated by larval competition, Nicholson showed that a decrease in the water supply shortened adult life span but simultaneously increased adult density. This experimental outcome corresponds to our predictions, illustrated in figure 2, that in maturation-regulated populations, an increase in adult mortality (and therefore decreased life span) may increase adult density. In addition, Nicholson showed that in blowfly populations that were regulated through adult competition, the abundance of eggs, larvae, and pupae doubled when he continuously destroyed 90% of all emerging adults. These observations correspond to our results that in a reproduction-regulated population, juvenile biomass will increase when adult mortality is increased (fig. 1). Finally, without providing any data, Nicholson (1957) also reports that in one of his experiments with blowflies, in which he destroyed 50% of the young larvae each day, the mean adult abundance more than doubled, an observation that is in line with our finding that increases in juvenile mortality will increase adult biomass (fig. 2) when the population is maturation regulated.

Slobodkin and Richman (1956) carried out similar size-selective harvesting experiments with *Daphnia pulicaria* in which they removed varying fractions of newborn individuals. Interpretation of their data is complicated by the fact that the number of individuals they harvested was based on an estimate of the number of *Daphnia* born over a 2- or 4-day time period. As a consequence, the number of individuals to be removed sometimes exceeded the number of small *Daphnia* present at the time of harvesting. Nonetheless, their results show that an increase in harvesting of small *Daphnia* resulted in larger densities in this size class. These findings correspond to the results shown in figure 1, where an increase in juvenile mortality increases juvenile biomass (see also De Roos and Persson 2002). Compensating and possibly overcompensating responses to harvesting were also reported by Watt (1955) in experiments with *Tribolium*, but unfortunately this author does not provide more detailed stage-specific information on abundances.

Since these early experiments in the 1950s, the phenomenon of overcompensation or positive effects of harvesting has been studied surprisingly infrequently. Moe et al. (2002) report positive effects in populations of blowflies under toxic stress with cadmium. These positive effects are attributed to the relaxation of density dependence that is induced by the cadmium stress. However, cadmium affected not only mortality but also adult fecundity, and the results, therefore, do not directly relate to our model analysis. Cameron and Benton (2004) show positive effects on adult density of harvesting eggs in populations of soil mites. These authors argue that adult soil mites are better competitors than juvenile soil mites because of their larger size. Under these conditions, we would predict that the population is regulated by maturation and that an increase in juvenile mortality would indeed increase adult density (fig. 2). Cameron and Benton (2004) also point out the paucity of data on positive effects in response to harvest-

ing, which is especially striking because such effects raise intriguing possibilities for population management.

Overcompensation in Vertebrates and Invertebrates

Vertebrate and invertebrate ectotherms mainly differ in their ecological scope, that is, the ratio between maximum ingestion and maintenance rate (Yodzis and Innes 1992). We found that the ratio I_{max}/T does influence persistence—that is, a species will go extinct at lower mortality rates with a smaller value of this ratio—but overall, the ecological scope has little effect on the extent of the biomass overcompensation (results not shown). We did not find significant differences in the hump-shaped curves of juvenile or adult biomass with increasing mortality when using parameter values for either vertebrate or invertebrate ectotherms. The extent to which we expect to find biomass overcompensation in particular species will therefore mainly be determined by background mortality and the ratio between neonate and adult body mass z. Species in which losses through background mortality are small as compared with the losses through maintenance will be especially prone to exhibit biomass overcompensation (fig. 5). Overcompensation might therefore be more common among the longer-lived vertebrate ectotherms than among invertebrate species. Vertebrate ectotherms might in many cases also have lower values of z, which promotes the occurrence of biomass overcompensation in case of reproduction regulation. An inspection of the estimated weight of hatchlings and adult individuals among 25 freshwater fish species reveals that the neonate-to-adult body mass ranges between 0.00001% and 0.1% and is in general much less than 0.1% (L. Persson, unpublished data). In contrast, data presented by Huntley and Lopez (1992, table A1) on marine copepods suggest that neonate body mass ranges between 0.1% and 2% of adult body mass. Taken together, these considerations imply that a hump-shaped relationship is unlikely to be found for adult biomass with increasing adult mortality, as this requires values of z close to 1 (fig. 5, *right panel*). Otherwise, we expect the other types of biomass overcompensation with increasing mortality to be a rather common phenomenon, especially among vertebrate ectotherms, such as fish.

Effects of Biomass Overcompensation on Communities

Biomass overcompensation due to increased mortality has consequences for the persistence of predator species that forage on the consumer. Using a tritrophic food-chain model that accounts for the entire consumer size distribution, De Roos and Persson (2002) showed that stage-specific predators exhibited an emergent Allee effect when an increase in stage-specific consumer mortality leads to within-stage overcompensation in biomass. The Allee effect occurs when predators specialized on small consumers as well as when they specialized on larger juvenile and adult consumers (De Roos and Persson 2005). Qualitatively, our stage-structured model shows the same results. However, De Roos and Persson (2002) showed that stage-specific predators on small consumers simultaneously increased their own food availability (small consumer biomass) as well as the biomass of large juvenile and adult consumers as a result of increased somatic growth of the latter. These predators therefore simultaneously facilitated their own persistence and the persistence of stage-specific predators on larger consumers. Our stage-structured model cannot show this simultaneous facilitative effect because we assumed that adult consumers do not grow in body size.

The results with stage-independent mortality lead us to postulate that generalist predators, which forage on the entire size range of consumers, may increase food availability for and therefore promote the persistence of stage-specific predators that forage on the stage with the most limited resource use and the lowest abundance. Stage-specific predators on the most abundant consumer stage may similarly facilitate persistence of stage-specific predators on other stages. This so-called emergent facilitation between stage-specific predators has indeed been shown to occur and to promote community persistence in models of a resource, a stage-structured consumer, and two unstructured predator populations that specialize on juvenile and adult consumers, respectively (Van Kooten 2004; De Roos and Persson 2005). Facilitation between multiple predator species foraging on the same prey has been documented in a variety of empirical studies (see Relyea 2003 for a review). In these studies, prey suffer a higher mortality in the presence of two predator species than expected on the basis of single-predator experiments, while predators may show increased growth when both species are present (e.g., Eklöv and VanKooten 2001). The facilitation results from an increased encounter rate between individual predators and prey, for example, due to a behavioral response of the prey to one of the two predator species. In contrast, the emergent facilitation resulting from biomass overcompensation is not an individual-level process but a population-level process: it operates through a release of competition for resources. The change in population feedback on resources and the subsequent differential response in individual growth and reproduction are critical elements of the phenomenon.

Biomass overcompensation with increasing mortality therefore allows predators to alter the resource environment to such an extent that it promotes their own performance (through an emergent Allee effect) or the per-

formance of predators that forage on other ranges of the consumer size distribution (through emergent facilitation). Predators may thus act as "ecological engineers" of their biotic (resource) environment. This suggests that food-dependent growth and size-specific predation may play an important role in sustaining diversity, but little is currrently known about their influence on ecological communities (De Roos and Persson 2005). Even though body size has been recognized to play an important role in ecological communities (Woodward et al. 2005), current community models only take it into account to a limited extent. The Lotka-Volterra cascade model (Cohen et al. 1990) is based on Lotka-Volterra-type interactions between species and not on energetic considerations. The niche model (Williams and Martinez 2000) has been combined with population dynamic equations based on the Yodzis and Innes (1992) model (Williams and Martinez 2004; Brose et al. 2005), but the food web is constructed by ordering species on the basis of their niche value, which is not related to body size. As a consequence, in this model, changes in body size are synonymous with changes in the interaction strength between species. As of yet, Loeuille and Loreau (2005) present the only community model that is consistently based on body size considerations. In this model, the body size of a particular species determines on which other species it can forage, with which species it competes through interference competition, and its loss rate through maintenance and mortality. The model is, however, an unstructured model and ignores within-species size variation. The stage-structured model provides a useful approach with which to investigate the influence of within-species size variation on community dynamics. In particular, it can be used to investigate the generality of the finding that food-dependent growth and size-specific predation promote diversity in ecological communities. Overcompensation in biomass, resulting from stage-specific differences in either resource use or mortality, may thus have important implications for biodiversity as well as for management of exploited populations. In this context, the lack of empirical information on the subject is surprising and represents an obvious gap in our understanding of population responses to stress.

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APPENDIX A

Parameter Bounds to Overcompensation

In this appendix, we focus on the overcompensation in juvenile biomass J due to an increase in juvenile mortality μ_j. We discuss the method to compute the boundary of the set of parameters for which this biomass overcompensation occurs. The procedures to compute the boundaries for the overcompensation in juvenile biomass due to increases in adult or stage-independent mortality, as well as for the overcompensation in adult biomass, are similar and will not be discussed further.

The equilibrium of the stage-structured consumer-resource model is determined by a set of nonlinear equations that we can express in vector notation as

$$\begin{bmatrix} F_1(R^*, J^*, A^*, \mu_j) \\ F_2(R^*, J^*, A^*, \mu_j) \\ F_3(R^*, J^*, A^*, \mu_j) \end{bmatrix} = 0. \tag{A1}$$

Here, the functions $F_1(R^*, J^*, A^*, \mu_j)$, $F_2(R^*, J^*, A^*, \mu_j)$, and $F_3(R^*, J^*, A^*, \mu_j)$ are used to denote the right-hand sides of the ODEs (1)–(3) in the main text, which specify the dynamics of resource biomass R, juvenile biomass J, and adult biomass A. The equilibrium values R^*, J^*, and A^* are functions of the juvenile mortality μ_j, and we can therefore formally write $R^*(\mu_j)$, $J^*(\mu_j)$, and $A^*(\mu_j)$. How these quantities change with a change in μ_j can be computed by differentiating both sides of equation (A1) with respect to μ_j:

$$\begin{bmatrix} \frac{\partial F_1}{\partial R} & \frac{\partial F_1}{\partial J} & \frac{\partial F_1}{\partial A} \\ \frac{\partial F_2}{\partial R} & \frac{\partial F_2}{\partial J} & \frac{\partial F_2}{\partial A} \\ \frac{\partial F_3}{\partial R} & \frac{\partial F_3}{\partial J} & \frac{\partial F_3}{\partial A} \end{bmatrix} \begin{bmatrix} \frac{\partial R^*}{\partial \mu_j} \\ \frac{\partial J^*}{\partial \mu_j} \\ \frac{\partial A^*}{\partial \mu_j} \end{bmatrix} + \begin{bmatrix} \frac{\partial F_1}{\partial \mu_j} \\ \frac{\partial F_2}{\partial \mu_j} \\ \frac{\partial F_3}{\partial \mu_j} \end{bmatrix} = 0. \tag{A2}$$

Note that all differentials occurring in this and all following equations are evaluated at the equilibrium values R^*, J^*, and A^*, respectively.

Applying the implicit function theorem to this last equation yields the following expression for the changes in R^*, J^*, and A^* with changes in μ_j:

$$\begin{bmatrix} \frac{\partial R^*}{\partial \mu_j} \\ \frac{\partial J^*}{\partial \mu_j} \\ \frac{\partial A^*}{\partial \mu_j} \end{bmatrix} = -\begin{bmatrix} \frac{\partial F_1}{\partial R} & \frac{\partial F_1}{\partial J} & \frac{\partial F_1}{\partial A} \\ \frac{\partial F_2}{\partial R} & \frac{\partial F_2}{\partial J} & \frac{\partial F_2}{\partial A} \\ \frac{\partial F_3}{\partial R} & \frac{\partial F_3}{\partial J} & \frac{\partial F_3}{\partial A} \end{bmatrix}^{-1} \begin{bmatrix} \frac{\partial F_1}{\partial \mu_j} \\ \frac{\partial F_2}{\partial \mu_j} \\ \frac{\partial F_3}{\partial \mu_j} \end{bmatrix}. \tag{A3}$$

Geometrically, this equation specifies the direction of the equilibrium curve in the phase space spanned by R^*, J^*, and A^* as a function of μ_j. In practice, we computed the values of $\partial R^*/\partial \mu_j$, $\partial J^*/\partial \mu_j$, and $\partial A^*/\partial \mu_j$ numerically by solving the linear system of equations given in (A2) with a standard numerical solver. The differentials of the functions $F_1(R^*, J^*, A^*, \mu_j)$, $F_2(R^*, J^*, A^*, \mu_j)$, and $F_3(R^*, J^*, A^*, \mu_j)$ that make up equation (A2), however, we computed analytically.

The equilibrium resource, juvenile biomass, and adult biomass at a background mortality level of $\mu_j = \mu_b$ we now denote with R_b^*, J_b^*, and A_b^*, respectively, while the corresponding values at a maximum in the curve relating juvenile biomass to juvenile mortality we denote with R_m^*, J_m^*, and A_m^*, respectively. The juvenile mortality at which this maximum occurs we denote with μ_m. The values of R_b^*, J_b^*, and A_b^* for a given value of μ_b are determined by the following system of three equations:

$$\begin{cases} F_1(R_b^*, J_b^*, A_b^*, \mu_b) = 0 \\ F_2(R_b^*, J_b^*, A_b^*, \mu_b) = 0 \\ F_3(R_b^*, J_b^*, A_b^*, \mu_b) = 0. \end{cases} \tag{A4}$$

The values of R_m^*, J_m^*, and A_m^*, as well as μ_m, are determined by the following system of four equations:

$$\begin{cases} F_1(R_m^*, J_m^*, A_m^*, \mu_m) = 0 \\ F_2(R_m^*, J_m^*, A_m^*, \mu_m) = 0 \\ F_3(R_m^*, J_m^*, A_m^*, \mu_m) = 0 \\ \left.\dfrac{\partial J^*}{\partial \mu_j}\right|_{\mu_j = \mu_m} = 0. \end{cases} \tag{A5}$$

The left-hand side of the last equation in this system is extracted from the direction of the equilibrium curve as a function of μ_j, specified by equation (A3). If we now require $J_m^* = J_b^*$, the seven equations in (A4) and (A5) determine the values of the seven unknown quantities R_b^*, J_b^*, A_b^*, μ_b, R_m^*, A_m^*, and μ_m, for which the maximum in the curve relating juvenile biomass to juvenile mortality μ_j exactly equals the value J_b^* at a background mortality of $\mu_j = \mu_b$. By computing these quantities for a range of values of the neonate : adult body-mass ratio z, we constructed the boundaries shown in figure 5 of the main text.

APPENDIX B

Biomass Overcompensation with Unlimited Juvenile or Adult Food Supply

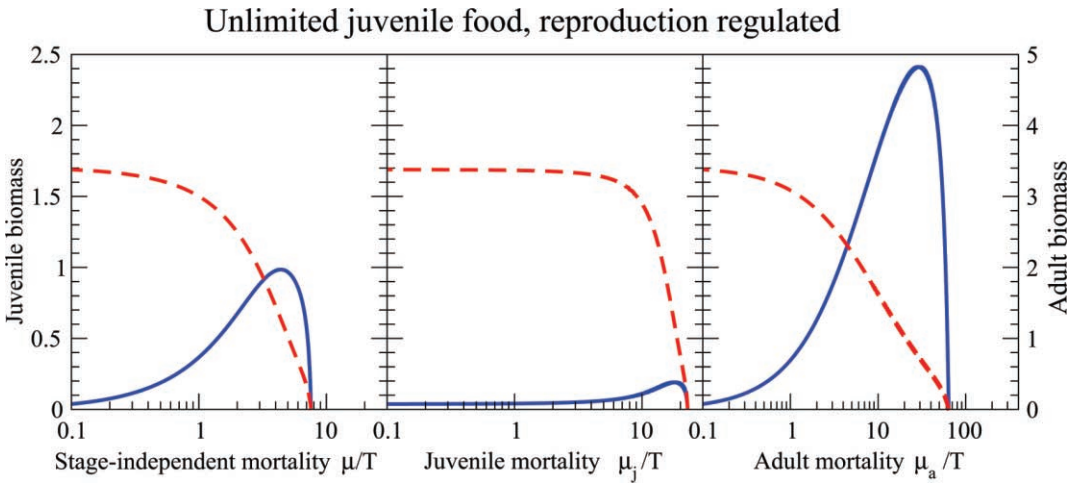

Figure B1: Changes in equilibrium juvenile (*blue*, *solid*) and adult (*red*, *dashed*) biomass with increases in mortality in case juveniles are assumed to have an unlimited food supply. As a consequence, the population is reproduction regulated. *Left*, increases in scaled stage-independent mortality $\mu_j/T = \mu_a/T$. *Middle*, increases in scaled juvenile mortality rate μ_j/T. *Right*, increases in scaled adult mortality rate μ_a/T. All start from $\mu_j/T = \mu_a/T = 0.1$. Parameters: $z = 0.1$; all other parameters have default values. Given the unlimited food supply for juveniles, the stage-structured biomass model is described by the following set of ODEs: $dR/dt = \delta(R_{max} - R) - I_{max}A[R/(H + R)]$, $dJ/dt = \nu_a(R)A + \nu_j J - \gamma(\nu_j)J - \mu_j J$, $dA/dt = \gamma(\nu_j)J - \mu_a A$. Only adults are assumed to exploit and compete for the resource. Juvenile net-biomass production is independent of resource density, $\nu_j = \sigma I_{max} - T$, while net-biomass production of adults is food dependent, $\nu_a(R) = \sigma I_{max} R/(H + R) - T$. We have taken $q = 1$, as we assume differences in resource use between juveniles and adults only result from differences in food supply as opposed to differences in foraging capacity. The maturation function $\gamma(\nu_j)$ is given by equation (6) in the main text.

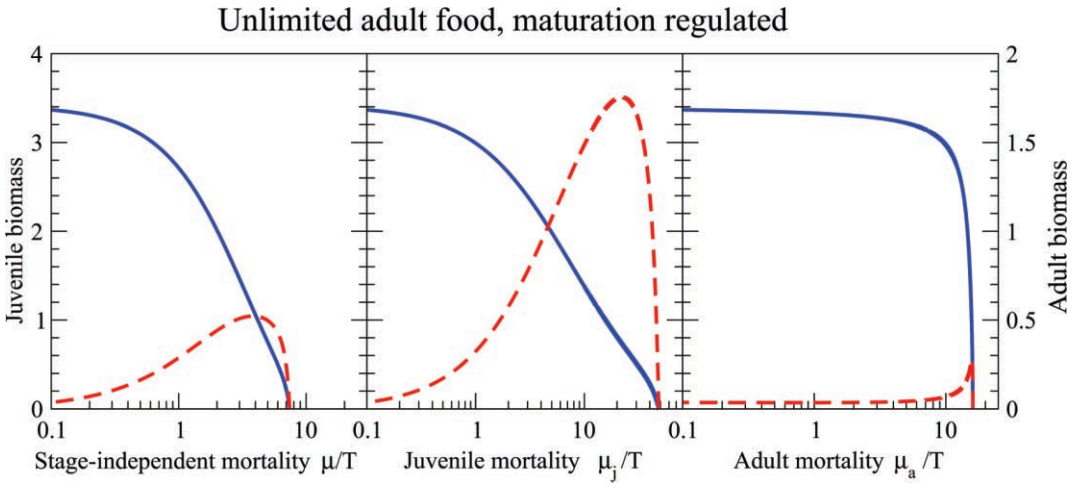

Figure B2: Changes in equilibrium juvenile (*blue*, *solid*) and adult (*red*, *dashed*) biomass with increases in mortality in case adults are assumed to have an unlimited food supply. As a consequence, the population is maturation regulated. *Left*, increases in scaled stage-independent mortality $\mu_j/T = \mu_a/T$. *Middle*, increases in scaled juvenile mortality rate μ_j/T. *Right*, increases in scaled adult mortality rate μ_a/T. All start from $\mu_j/T = \mu_a/T = 0.1$. Parameters: $z = 0.5$; all other parameters have default values. Given the unlimited food supply for adults, the stage-structured biomass model is described by the following set of ODEs: $dR/dt = \delta(R_{max} - R) - I_{max}J[R/(H + R)]$, $dJ/dt = \nu_a A + \nu_j(R)J - \gamma(\nu_j(R))J - \mu_j J$, $dA/dt = \gamma(\nu_j(R))J - \mu_a A$. Only juveniles are assumed to exploit and compete for the resource. Adult net-biomass production is independent of resource density, $\nu_a = \sigma I_{max} - T$, while net-biomass production of juveniles is food dependent, $\nu_j(R) = \sigma I_{max} R/(H + R) - T$. Again, we have taken $q = 1$, as we assume differences in resource use between juveniles and adults only result from differences in food supply as opposed to differences in foraging capacity. The maturation function $\gamma(\nu_j(R))$ is given by equation (6) in the main text.

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