Review

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When less is more: positive population-level effects of mortality

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Experimental and theoretical studies show that mortality imposed on a population can counter-intuitively increase the density of a specific life-history stage or total population density. Understanding positive population-level effects of mortality is advancing, illuminating implications for population, community, and applied ecology. Reconciling theory and data, we found that the mathematical models used to study mortality effects vary in the effects predicted and mechanisms proposed. Experiments predominantly demonstrate stage-specific density increases in response to mortality. We argue that the empirical evidence supports theory based on stage-structured population models but not on unstructured models. We conclude that stage-specific positive mortality effects are likely to be common in nature and that accounting for within-population individual variation is essential for developing ecological theory.

Can less really be more?

Natural populations experience externally imposed mortality arising from predation, parasitism, and disease, or through exploitation, pest control, and eradication programmes. The intuitive expectation is that increasing mortality should result in decreasing population densities, a principle on which population and ecosystem management is generally based. Nevertheless, positive effects of mortality at the population level are increasingly found in many different species. This is true for experiments in the laboratory (e.g., [1,2]) and in the field (e.g., [3,4]). The mechanisms behind positive population-level effects of mortality (see Glossary) and the circumstances under which these can be observed have recently been studied in several mathematical models (e.g., [5–7]). From these studies it has become clear that positive mortality effects have not only wide ranging implications for communities [6,8,9] and ecosystems [10] but also for the sustainable exploitation of natural resources, such as fish stocks [11], and for the successful implementation of pest control and eradication programmes [12,13]. Hence, the occurrence of positive mortality effects at the population level (simply denoted positive mortality effects hereafter) might indeed be a common phenomenon in natural populations, and understanding these effects is imperative for basic and applied ecology.

To facilitate this understanding we reconcile findings from theoretical approaches with evidence from empirical studies. We first review the different mathematical models predicting positive mortality effects and the proposed mechanisms for these effects. Second, we compile results from published mortality experiments and show what types of positive mortality effects occur in natural populations. Finally, theoretical predictions are compared to empirical patterns, leading to an evaluation of the level of support for different hypotheses and mechanisms invoked to explain the occurrence of positive mortality effects.

Positive effects of mortality: theoretical background

Positive mortality effects were first proposed for a discrete-time single-population model that does not explicitly account for resource dynamics [14]. Single-population models can demonstrate positive mortality effects given one or several *a priori* assumed processes, such as overcompensating density dependence, mortality preceding density dependence, scramble competition, or the relative timing of mortality and population census [6,15–18]. In single-population models, however, population growth, resource use, and density dependence are defined in a phenomenological way, meaning that biological processes are modelled as predetermined functions of consumer density.

The objective of this paper is to review the processes and mechanisms resulting in positive mortality effects in theoretical studies and to summarise the empirical support for the different mechanisms by comparing the predicted positive mortality effects to patterns found in empirical settings. In light of this objective, the explicit link between resource availability and consumer population dynamics is essential, because an increase in consumer population density must be based on the transformation of energy from the resource to the consumer. In order to draw such an explicit link, two-trophic consumer-resource models explicitly account for dynamics of the resource density alongside the dynamics of the consumer population. Density dependence in consumer population growth then arises without *a priori* assuming a population-level functional form for that process. The consumer population response to mortality

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Glossary

Bioenergetics: the energy balance of an individual reflecting energy acquisition through consumption, energy losses through excretion, respiration and maintenance, and energy allocation to biomass production in the form of either somatic growth or reproduction; this balance is summarised as the energy budget of an individual.

Bottleneck: the density-dependent process of biomass production that most limits and thus regulates population growth. A bottleneck stage is the life-history stage or size class in which the bottleneck process occurs, which is in contrast to a non-regulatory stage.

Density dependence: the (usually negative) feedback between population growth based on per-capita vital rates (such as somatic growth, maturation, or reproduction) and population density. In consumer-resource models, density dependence can emerge as a result from the intraspecific competition for resources. In single-population models, density dependence is incorporated as a pre-determined relationship between processes reflecting vital rates and population density. Density dependence is overcompensatory when population growth leads to an over- or under-shooting of the carrying capacity or equilibrium, often associated with time lags or delays in the operation of density dependence. Overcompensatory density dependence is a process and must not to be confused with the pattern of stage-specific overcompensation.

Hydra effect: the increase in the equilibrium or time-averaged density of a consumer population with increasing mortality. Hydra effects occur when unstructured consumer populations exhibit cyclic dynamics. The basis for the hydra effect is an exponential increase in resource productivity when resources are depleted and when the consumer population is at the maximum of the consumer-resource cycle. The combination of logistic resource growth and saturating resource ingestion rate of the consumer are hence essential ingredients for hydra effects.

Life-history: the sequence of trait changes over the course of an individual organism's progression through its different life stages from birth to death based on the processes of growth, maturation, reproduction, and survival.

Logistic resource dynamics: a classical formulation of resource dynamics, whereby, in absence of consumption, the resource approaches its carrying capacity in a sigmoidal fashion (if the initial density is below the carrying capacity). In this formulation, resource productivity is dependent on the current resource density and the resource has exponential growth at low resource density. Examples of logistically growing systems are bacterial batch cultures and predators feeding on living, reproducing prey. Mathematically, the logistic growth equation can be given by $\frac{dR}{dt} = rR(1 - \frac{R}{R_{max}})$, where r is the instantaneous growth rate at very low resource densities and R_{max} is the maximum density the resource attains in the absence of consumers.

Ontogenetic asymmetry: ecologically relevant differences between individuals of different body sizes or life-history stages; for example, in resource ingestion rate, biomass maintenance rate, or resource and habitat use. Ontogenetic asymmetry reflects differences in individual competitiveness and therefore determines which life stage is the bottleneck stage.

Positive population-level effects of mortality: the increases in the numbers or biomass of a specific size class or life-history stage of a consumer population or in total population numbers or biomass with increasing mortality. This effect should be distinguished from the often demonstrated positive mortality effects at the individual level, such as higher food ingestion, somatic growth, body conditions, or fecundity of survivors. However, note that positive effects of mortality at the population level ultimately result from positive effects at the individual level of survivors.

Semi-chemostat resource dynamics: a classical formulation of resource dynamics, whereby, in the absence of consumption, the resource approaches its carrying capacity in a smooth (non-sigmoidal) fashion. In this formulation, resource density changes through a constant inflow and outflow. Resource productivity is constant and independent of resource density. Semi-chemostat dynamics would reflect a constant supply of non-reproducing resources in experimental settings or resource populations in nature that have a spatial or size refuge. Mathematically, the equation for semi-chemostat dynamics is given by $\frac{dR}{dt} = b(R_{max} - R)$, where b is the turnover rate of the resource and R_{max} is the maximum or equilibrium density, which the resource attains in the absence of consumers.

Stage-specific overcompensation: an increase in the biomass of the non-regulatory size class or life-history stage of a consumer population with increasing mortality. This effect occurs through relaxation of density dependence in the bottleneck stage. Stage-specific overcompensation occurs irrespective of whether mortality is imposed on the entire population or on a particular life stage.

Stage-specific hydra effects: an increase in stage-specific density of a bottleneck stage with increasing mortality imposed on that stage. Stage-specific hydra effects occur through the temporal separation of density dependence and mortality. Stage-specific hydra effects and their mechanism are a specific case of stage-specific overcompensation when measured in density.

Size- and stage-structure: the division of individuals in a population on the basis of differences in body size or life-history stages, such as juvenile vs adult stage.

emerges from changes in the resource availability for individual consumers, which in turn is determined by the feedback from consumption itself. We will therefore mainly focus on consumer-resource models and only briefly discuss single-species models.

Positive mortality effects in unstructured consumer-resource models

One prominent class of mathematical models predicting positive mortality effects consists of unstructured models of continuous-time consumer-resource systems that are based on the well-known Lotka–Volterra equations [19–21]. These models are unstructured with respect to any within-population variation among individuals; for example, in body size, life-history stage, and ecology. The processes contributing to changes in population density in these unstructured models are therefore identical for all individuals in the population, reflecting the assumption that individuals are represented by the same average body size, life-history stage, and are ecologically equivalent.

Unstructured consumer-resource models predict higher total consumer density with increasing consumer mortality [6,7,22]. This phenomenon has been called the 'hydra effect' [23] and it occurs when consumer-resource models exhibit cyclic dynamics. These consumer-resource cycles are caused by the combination of logistic resource growth and saturating consumption rate of individual consumers [19,20] (Box 1). Under these conditions, hydra effects can appear as an increase in the time-averaged density of consumers if mortality leads to a shorter period and lower amplitude of the consumer-resource cycles. The occurrence and strength of hydra effects caused by this mechanism of 'altered variation in population abundance' [6] (Table 1) then strongly depends on the way the cycle characteristics are affected by mortality [6,7]. In any case, at high mortality rates consumer density declines (and cycles disappear) when the direct negative effect of mortality outweighs the indirect positive effect [6,7].

When these hydra effects occur, the consumer equilibrium density increases with increasing mortality over the same range of mortality rates for which the system displays consumer-resource cycles [6,7,22] (Box 1). Because hydra effects based on higher consumer equilibrium densities also arise via modification of individual traits that alter the trophic interaction between consumer and resource and stabilise dynamics [22–25] (Box 2) this second mechanism underlying positive mortality effects in unstructured models has been termed 'prudent resource exploitation' [6] (Table 1).

When resource productivity in an unstructured consumer-resource model is constant and independent of the resource density (which can be modelled by assuming semi-chemostat resource growth), the consumer does not exhibit hydra effects [22]. The coupling between resource growth and consumer density that is the basis for the two mechanisms leading to hydra effects in unstructured consumer-resource models is absent in systems with semi-chemostat resource dynamics. Consumer-resource cycles do not occur and consumer equilibrium density only decreases with mortality when resource growth follows

Box 1. Resource dynamics and positive mortality effects

One way of illustrating the effects of different resource dynamics on a modelled consumer populations' response to increased mortality is by means of a phase space: the zero growth isoclines of consumer and resource densities (i.e., the solutions of the individual differential equations are zero and resource and consumer densities do not change) are plotted as function of one another (Figure I). The intersections of these isoclines are the steady states (equilibria) of the consumer-resource system described by the two ordinary differential equations for resource R (**Equation I**) and consumer C (**Equation II**):

$$\frac{dR}{dt} = g(R)R - f(R)RC \quad [I]$$

$$\frac{dC}{dt} = e\,f(R)RC - mC \quad [II]$$

where $g(R)$ is the per-capita resource growth rate without consumers and $f(R) = \frac{a}{1+ahR}$ when we define the functional response with a saturating resource consumption rate for individual consumers. The parameter e is the efficiency with which consumers turn ingested resource into new consumers and m is the consumer mortality rate. Straightforward analyses and classical studies show that the consumer isocline $\frac{dc}{dt} = 0$ in this model is defined by a single resource value (e.g., [19,20]) given by $R^* = \frac{m}{a(e-mah)}$ and hence shows up as a straight line in the phase space. The resource isocline $\frac{dR}{dt} = 0$ is determined by both the resource and consumer density, and so is the consumer equilibrium density $C^* = \frac{g(R^*)}{f(R^*)}$ as defined by this isocline. As the saturating consumption rate allows the resource to escape control by consumers it can realise its growth potential.

With logistic resource dynamics $g(R)R = rR(1 - \frac{R}{R_{max}})$, where resource productivity is positively related to resource density, at least at low resource densities, higher consumer mortality can result in higher resource productivity, benefiting consumer reproduction. The consumer equilibrium density increases with increasing mortality in this case (Figure I). With semi-chemostat resource dynamics $g(R)R = b(R_{max} - R)$, resource productivity is independent of resource density and the resource isocline is a monotonously decreasing curve in the phase-space. While higher consumer mortality leads to higher resource density, resource productivity does not increase and the consumer density only declines with increasing consumer mortality (Figure I).

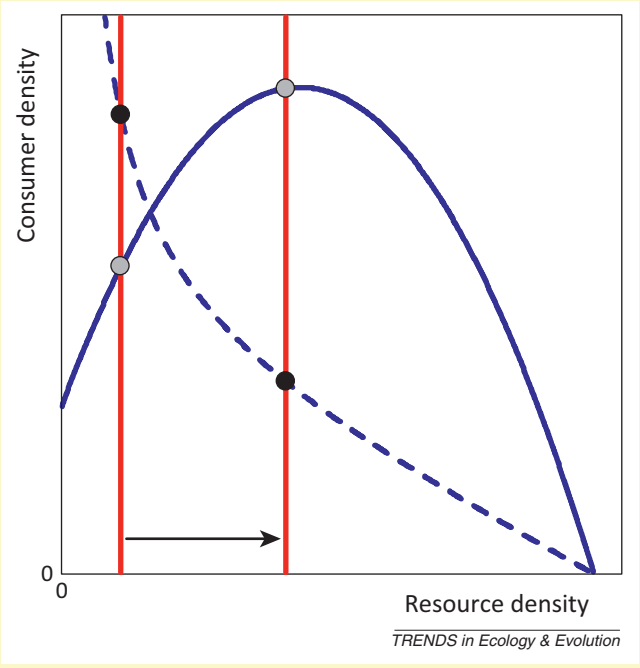

Figure I. The consumer (red) and resource (blue) isoclines in the phase space of an unstructured consumer-resource model when resource dynamics follow logistic (unbroken line) or semi-chemostat dynamics (broken line). The dots represent the internal equilibria for logistic (grey) and semi-chemostat (black) resource dynamics when mortality is increased (arrow). Note that with logistic growth, the equilibria to the left of the maximum are unstable and the system exhibits limit cycles, which are not shown.

semi-chemostat dynamics [22] (Box 1). This finding of the influence of resource dynamics type on the occurrence of positive mortality effects in unstructured consumer populations emphasises the explanatory power of explicitly incorporating resource dynamics into models to capture the interaction that links resource and consumer population growth.

Hydra effects caused by 'altered variation in population density' can also occur in unstructured single-population models formulated in discrete time (Table 1). These models have the tendency to overshoot equilibrium values and to display cycles [6,14–18]. To observe increasing densities with increasing mortality in these models, density dependence must be explicitly defined as overcompensatory [6]. The underlying process leading to an increasing total population density is then implicitly assumed by the phenomenological inclusion of the forms of density dependence and mortality at the population level. Yet another mechanism proposed to result in hydra effects in unstructured single-population models is the 'temporal separation of density dependence and mortality' [6] (Table 1). If mortality is imposed prior to the operation of overcompensatory density dependence, this can lead to higher equilibrium density [6,15]. In unstructured models, this third mechanism is restricted to discrete-time single-population models with their sequential ordering of biological processes.

Positive mortality effects in stage-structured consumer-resource models

Another prominent class of models predicting positive mortality effects consists of stage-structured consumer-resource models, in which individuals within a population are characterised on the basis of their life-history stage (Table 1). This differentiation can reflect developmental life-history stages and also size ranges (e.g., [5,26]). We will use the concept of stage-structure in a broad sense. Stage-structured consumer-resource models describe stage-dependent, biological processes and life-history characteristics at the individual level [9,27,28]. When characterised on the basis of individual body size, different life-history stages can be recognised to exhibit stage-specific process rates. Such differences may influence and determine stage-specific resource consumption rates, the efficiency with which the ingested energy is converted into biomass, and to what extent energy is allocated to growth, biomass maintenance, or reproduction [29]. The emerging ontogenetic asymmetry in ecological performance and bioenergetics determines the size-dependent competitive abilities of individuals. In a stage-structured population that accounts for such individual differences, ontogenetic development (or somatic growth and maturation) and reproduction affect population growth asymmetrically [5,9,28]. The population regulation through development or reproduction depends on whether juveniles or adults are

Table 1. Mechanisms and predictions regarding positive mortality effects in different mathematical models

	Mechanism		Model characteristics	Increase in	Decline in	Type of effect[a]	Refs
1	Altered variation in population size	1.1	Unstructured, discrete time, single-population	Total time-averaged density	-	Hydra effect	[6]
		1.2	Unstructured, continuous time, consumer-resource	Total time-averaged density	-	Hydra effect	[6]
2	Prudent resource exploitation	2.1	Unstructured, continuous time, consumer-resource	Total equilibrium density	-	Hydra effect	[6]
3	Temporal separation of density dependence and mortality	3.1	Unstructured, discrete time, single-population	Total equilibrium density	-	Hydra effect	[15]
		3.2	Stage-structured, discrete time, single-population	Density in the targeted stage (if non-regulatory) and total numbers driven by higher juvenile numbers	Density in bottleneck stage	Stage-specific hydra effects	[12]
		3.3	Stage-structured, continuous time, single-population	Density in the targeted stage (if non-regulatory) and total numbers driven by higher juvenile numbers	Density in bottleneck stage	Stage-specific hydra effects	[6]
4	Relaxation of density-dependent bottleneck	4.1	Stage-structured, discrete time, single-population	Density in the non-regulatory stage and total numbers	Density in bottleneck stage	Stage-specific overcompensation	[12]
		4.2	Stage-structured, continuous time, consumer-resource	Density (number and biomass) in the non-regulatory stage	Density (numbers and biomass) in bottleneck stage and total density	Stage-specific overcompensation	[5]

[a]see Glossary.

competitively superior for a shared resource (see explanation below) [5,9,28].

Stage-structured consumer-resource models with a single resource shared between stages predict density increases in response to increasing mortality in the non-regulatory life-history stage of the consumer only [5,9,28]. Total density as well as the density of the bottleneck stage decline in response to increasing mortality (see Box 2 for more complex scenarios, such as ontogenetic habitat and resource niche shifts). The mechanism behind this 'stage-specific biomass overcompensation' [5] is the relaxation of density dependence within the bottleneck stage with increasing mortality and, hence, increased biomass production in that stage, leading to an altered flow of biomass between stages. Consider, for example, conditions whereby adults are competitively superior to juveniles and the juvenile stage of a consumer species has limited biomass production. In this scenario, the consumer population is dominated by biomass in the juvenile stage and juveniles grow slowly as a result of high intra-stage competition. Maturation into the adult stage is low under these conditions and regulates population density and biomass. Increasing mortality in this consumer population is predicted to relax density dependence among juveniles and to increase biomass production through somatic growth, which results in biomass spilling over to adults. Consequently, adult biomass increases with increasing mortality as a result of the decreased biomass in the juvenile stage and subsequent increased biomass production rate. The pattern is reversed when the population is limited by density-dependent reproduction in the adult stage [5,9,28] (Box 3).

It should be noted that stage-specific biomass overcompensation is caused by a mechanism that is different from the 'temporal separation of density dependence and mortality' as was proposed for positive stage-specific effects of morality in stage-structured single-population models [6,15] (Table 1). In these latter models, mortality must target the non-regulatory stage, or at most act early on in the bottleneck stage, to result in 'stage-specific hydra effects' [6,18] (Table 1). This condition ensures that relaxed density dependence has time to counteract the loss of individuals. Moreover, stage-specific hydra effects have been defined as occurring only in the stage affected by mortality [6,18]. For example, juvenile mortality raises juvenile numbers when the population is regulated by reproduction so that mortality and density dependence do not affect the same stage [12]. However, the temporal separation mechanism does not account for, nor can explain, stage-specific overcompensation in the non-regulatory stage that occurs independent of what stage is affected by mortality [5]. Stage-specific overcompensation and its underlying mechanism therefore pose a more general theory that encompasses the temporal separation mechanism and stage-specific hydra effects.

Experimental results and comparisons with model predictions

This review of mathematical models clarifies that positive effects of mortality at the population level can occur in distinctive forms and arise from various mechanisms. Unstructured models predict hydra effects: mortality-induced increases in total consumer density (at equilibrium or time-averaged in a cyclic population) through changes in resource productivity [6,7,22] or through temporal separation of density dependence and mortality [6,15]. By contrast, stage-structured models predict stage-specific overcompensation: higher density in non-regulatory life-history stages of a consumer through the relaxation of stage-specific density dependence in the bottleneck stage [5,9,28]. This clear-cut distinction allows for a

Box 2. Positive mortality effects with complex interactions or life histories

The mathematical models reviewed here represent the simplest one consumer–one resource systems with interactions between consumer and resource that are independent of consumer or resource traits (in unstructured models) or with life histories distinguishing a juvenile and an adult stage (in stage-structured models). Positive mortality effects have, however, also been reported in systems with larger ecological complexity.

(i) *Unstructured models:* The mortality of conspecifics can have strong non-lethal effects on survivors' plastic behavioural or morphological traits. Individuals may switch to different resources or habitats, reduce their activity, or develop anti-predator morphs when predation or exploitation pressure increases [57,58]. When such mortality-induced trait modifications alter consumer–resource interactions, hydra effects are no longer restricted to unstable systems displaying consumer–resource cycles. Trait-mediated decreased consumption rates raise resource density and higher consumer equilibrium density can be attained [6,7,22–25].

(ii) *Stage-structured models*: Life histories often include more than two stages and hence more possibilities for density-dependent regulatory processes. Nonetheless, increasing mortality will always relax the bottleneck and increase the biomass in the non-regulatory stages not limiting population growth, while total biomass and biomass in the bottleneck stage decline [9]. Overcompensation in total biomass is possible when different life-history stages feed on different resources or exploit different habitats varying in productivity (consumers exhibit an ontogenetic niche shift). Total biomass overcompensation with increased mortality then occurs as a result of a more efficient use of the resource in the bottleneck stage. For example, when juveniles specialise on a resource that provides an unlimited supply, growth and development is rapid and maturation rate is high. The inflow of biomass to the adult stage is then also high, leading to strong competition among adults and regulation through reproduction. Increasing mortality reduces the competition among adults, leading to a higher density of juveniles that still grow and mature rapidly. As a result, population maturation rate increases and hence also adult biomass. In this case, the total population biomass can increase because the biomass produced in the juvenile stage is not lost in the adult stage, but rather transformed more efficiently into new juvenile biomass [9,48]. Note that this increase in total biomass and the underlying mechanism are not equivalent to hydra effects in unstructured models.

None of these predictions have been explicitly tested yet. Behavioural and morphological trait modifications are common in natural systems [57,58], but they have not been quantified in the studies summarised in Table 2. Many of the species studied in Table 2 have multiple juvenile stages (e.g., blow flies, soil mites) or display ontogenetic niche shifts (e.g., Eurasian perch). However, juvenile stages have usually been pooled and ontogenetic niche shifts were unaccounted for when only one resource was provided.

straightforward interpretation of the experimental evidence for positive mortality effects as support for different theoretical concepts.

We review experimental studies that maintained at least one mortality manipulation while comparing the population response to a control without mortality over multiple generations. Also before vs after treatment comparisons were included in our analysis, which are often more practical in field situations. Mortality experiments fulfilling these criteria are currently rare but increasing. The summary of experimental evidence for positive mortality effects (Table 2) demonstrates several important points:

(i) All experiments in Table 2 have been performed using animal species with conspicuous body size differences between juveniles and adults. For many experimental populations, one single regulatory process could be identified (i.e., either ontogenetic development or reproduction). This finding is in line with the idea that relevant ecological differences between individuals existed in the experimental species due to ontogenetic asymmetry.

(ii) Positive mortality effects in experimental populations were always stage-specific. Moreover, the stage in which the positive effect occurred was always the non-regulatory stage while density in the bottleneck stage declined. This stage-specific pattern appeared irrespective of what stage was subjected to mortality. In the majority of cases total population density also declined with mortality (but see points (iv) and (v) below).

Especially conclusive with respect to point (ii) are Nicholson's classic blow fly experiments [30,31] and Schröder *et al.*'s fish experiments [2]. Nicholson experimentally manipulated which life-history stage was regulating population growth the most by modifying stage-specific resource availability. When juveniles had more resources and therefore had an energetic advantage, adult reproduction and survival was limited and mortality led to higher juvenile densities. When adults had an energetic advantage, regulation occurred through ontogenetic development of juveniles and adult numbers increased with mortality [30,31]. In Schröder *et al.* [2], in populations regulated by reproduction, biomass increased with mortality only in the non-regulatory juvenile stage independent of whether juveniles or adults were removed. Moreover, this experiment also showed details of the density response predicted by stage-structured consumer-resource models. Stage-specific overcompensation was weaker and occurred at lower mortality rates when the non-regulatory stage was targeted compared with when the regulatory stage was targeted [2,5].

We interpret these outcomes as support for stage-specific overcompensation associated with the relaxation of density-dependent bottleneck processes as the more general pattern and mechanism for stage-specific positive mortality effects in comparison to stage-specific hydra effects associated with temporal separation of density dependence and mortality.

(iii) Experimental resource availability was, at least in laboratory experiments, controlled by the experimenters such that fixed amounts of non-reproducing food were supplied per time. Usually, experimental populations were fed with non-living food, such as dried food pellets (e.g,. [1,2,32]), or via inflow of living food from a separate growth chamber (e.g., [33]). These procedures represent a constant inflow of resources from a reservoir as can be described by semi-chemostat resource dynamics. Such a reservoir could be a spatial or body-size refuge from which resources are constantly replenished through movement or growth. Resource growth in field situations is less readily attributed to a specific form, but it has been argued that semi-chemostat-like dynamics prevail in nature [34]. The coupling between resource productivity, resource density, and

Table 2. Summary of empirical examples of positive mortality effects

Mortality type	Regulatory process (bottleneck stage)[a]	Resource dynamics[b]	Increase in	Decline in	Density measured in[c]	Type of effect[d]	Laboratory or field study	Species	Refs
Adults (restricted water availability)	Development (Juveniles)	Constant food supply = Semi chemostat	Adults and pupae	Juveniles (eggs, larvae) and total density	Numbers	*Stage-specific overcompensation/stage-specific hydra effect	Lab.	Blow fly (*Lucilia cuprina*)	[30]
Adults 90% daily	Reproduction (Adults)	Constant food supply = Semi-chemostat	Juveniles (eggs, larvae, pupae)	Adults and total density	Numbers	*Overcompensation	Lab.	Blow fly (*Lucilia cuprina*)	[30]
Newborn juveniles 0–90% every 4th day	-	Constant food supply = Semi-chemostat	Juveniles (at 25% mortality)	Adults and total density	Numbers	Stage-specific overcompensation/stage-specific hydra effect	Lab.	Water flea (*Daphnia pulicaria*)	[59]
Juveniles 50% daily	Development (Juveniles)	Constant food supply = Semi-chemostat	Adults	Juveniles and total density	Numbers	*Overcompensation	Lab.	Blow fly (*Lucilia cuprina*)	[31]
Adults (exposure to toxicant)	Development (Juveniles)	Constant food supply = Semi-chemostat	Pupae	Larvae, adults and total density	Biomass	*Stage-specific overcompensation/stage-specific hydra effect	Lab.	Blow fly (*Lucilia sericata*)	[51]
Eggs 15% daily	Development (Juveniles)	Constant food supply = Semi-chemostat	Adults and total density	Juveniles	†Numbers	*Overcompensation	Lab.	Soil mite (*Sancassania berlesei*)	[1]
10%, 30%, 50%, and 70% of total daily	-	Regular transfer to new medium = Logistic	Total density (at 30% mortality)	-	Numbers	Hydra effect	Lab.	Ciliate (*Tetrahymena thermophila*)	[36]
80% of large juveniles and adults over 5 summers	Development (Juveniles)	-	Small juveniles and large adults	Intermediate size classes and total density	Biomass	Overcompensation	Field (whole lake)	Arctic charr (*Salvelinus alpinus*)	[60]
Adults (>20 cm)	-	-	Juveniles and total density	Adults	Numbers	*Overcompensation	Field (whole lake)	Smallmouth bass (*Micropterus dolomieu*)	[35]
Small juveniles 0–0.05% daily	Reproduction (Adults)	Constant food supply = Semi-chemostat	Small juveniles (at 0.008% mortality)	Large juveniles, no decrease in adults, or total density	†Biomass	Stage-specific overcompensation/stage-specific hydra effect	Lab.	Least killifish (*Heterandria formosa*)	[2]
Adults 0–0.05% daily	Reproduction (Adults)	Constant food supply = Semi-chemostat	Small and large juveniles (at 0.015% mortality)	Adults and total density	†Biomass	Overcompensation	Lab.	Least killifish (*Heterandria formosa*)	[2]
20% of total density daily	Development (Juveniles)	Constant food supply = Semi-chemostat	Constant juvenile density	Adults and total density	Biomass	(Over)-compensation	Lab.	Water flea (*Daphnia pulex*)	[33]
Large juveniles and adults	-	-	Juveniles	Adults and total density	Biomass	Overcompensation	Field (enclosure)	Water flea (*Holopedium gibberum*)	[3]
Adults	-	Constant food supply = Semi-chemostat	Adults	No data given for juveniles or total density	Numbers	*Stage-specific overcompensation/stage-specific hydra effect	Lab.	Bruchid beetle (*Callosobruchs maculatus*)	[32]
Adults (through pathogen)	Development (Juveniles)	-	Juveniles	Adults and total density	Biomass	Overcompensation	Field (whole lake)	Eurasian perch (*Perca fluviatilis*)	[4]

[a]Given only when directly manipulated or independent studies (data or simulations) are available, but not when regulatory process was only deduced from the response.

[b]Approximated based on the experimental feeding or transfer procedure, see [30] for prevalence of semi-chemostat dynamics in field systems.

[c]For studies marked with a dagger the density responses to mortality were also measured in biomass ([1], T. Cameron, unpublished) or abundance ([2], A. Schröder, unpublished), which gave the same qualitative results regarding density increases and decreases in total and stage-specific densities.

[d]Effects were classified following the definitions given in the text (see also Glossary). Results marked with an asterisk were interpreted as a hydra effect by [6] or by the author(s) of the reference study, or by both.

Box 3. A consumer-resource model with two consumer life-history stages

We used the stage-structured biomass model from de Roos and Persson [9] to illustrate the impact of increasing consumer mortality in the context of a size-structured consumer population. The model of one unstructured resource and one stage-structured consumer is defined using three ordinary differential equations: one for the resource dynamics (**Equation I**), one for juvenile consumer biomass (**Equation II**), and one for adult consumer biomass (**Equation III**):

$$\frac{dR}{dt} = G(R) - \omega_J(R)J - \omega_A(R)A \quad [I]$$

$$\frac{dJ}{dt} = \nu_A^+(R)A + \nu_j(R)J - \gamma(\nu_J^+, \mu_J)J - \mu_J J \quad [II]$$

$$\frac{dA}{dt} = \gamma(\nu_J^+, \mu_J)J + (\nu_A(R) - \nu_A^+(R))A - \mu_A A \quad [III]$$

With: $G(R)$ – resource growth, following semi-chemostat dynamics; $\omega_i(R)$ – resource intake rate stage i; $\nu_A^+(R)$ – consumer reproduction rate; $\nu_i(R)$ – net biomass production rate stage i; $\gamma(\nu_J^+, \mu_J)$ – juvenile maturation rate; μ_i – mortality rate stage i.

Although the consumer size distribution is discretised into two life-history stages, in equilibrium the model dynamics are completely consistent with an analogous physiologically structured population model accounting for the continuous consumer population size distribution [26]. Regarding the population stage structure, juveniles are assumed to invest their total net energy into somatic growth, while adults are assumed to allocate their total net energy into reproduction.

The net biomass production rates, $\nu_j(R)$ and $\nu_A(R)$, are the only terms we will define in detail here because these terms are particularly relevant for the dynamics shown. The general model definition is given and described in detail elsewhere [5,9,26,28]. The mass-specific biomass production rates of juvenile and adult consumers are defined as $\nu_j(R) = \sigma\frac{MR}{H+R} - T$ and $\nu_A(R) = q\sigma\frac{MR}{H+R} - T$, respectively. Where T represents maintenance rate, M represents maximum ingestion rate, and σ represents conversion efficiency. The factor q in these formulations represents the only mass-specific differentiation between the two life-history stages and represents the relative foraging efficiency of adult consumers with respect to the foraging efficiency of juvenile consumers. We present the consumer equilibrium response to increasing mortality in this model system for three different scenarios: i) juveniles are superior foragers ($q = 0.5$; Figure IA); ii) juveniles and adults do not differ in their foraging capacity ($q = 1.0$; Figure IB, representing the 'symmetric situation'); or iii) adults are superior foragers ($q = 2.0$; Figure IC). Note that the use of the term 'equivalent' here refers to mass-specific intake rates (since larger individuals still have a higher intake in absolute terms). The case with energetic symmetry in juveniles and adults represents a model setting that conforms to the unstructured model formulation by Yodzis and Innes [27], which allows for a direct comparison of the model dynamics in a structured vs unstructured model setting.

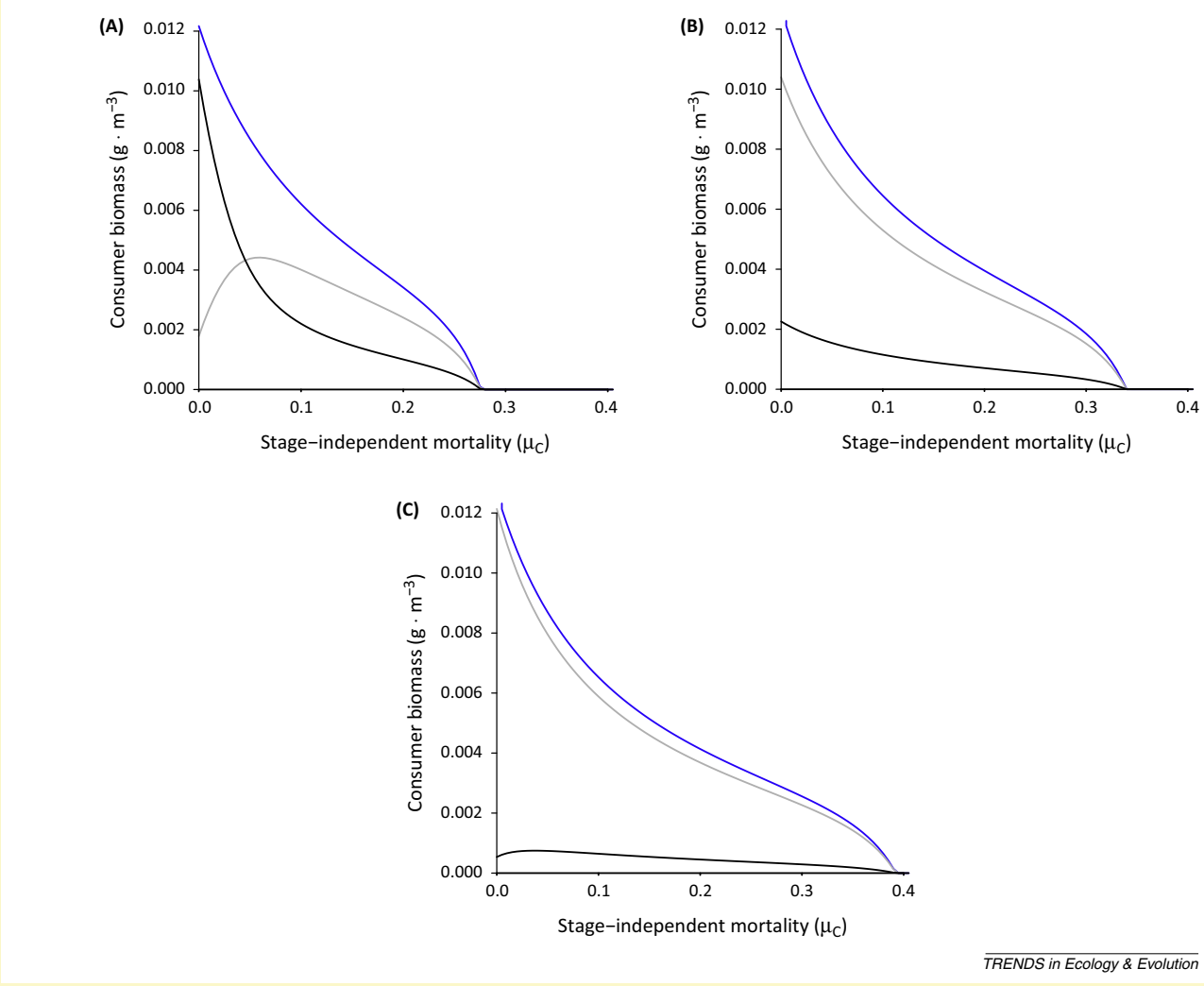

Figure I. Positive mortality effects in a stage-structured biomass model. Consumer equilibrium biomass **(A–C)** as a function of increasing stage-independent mortality, showing the response in total (blue), adult (black), and juvenile (grey) consumer biomass in the stage-structured consumer-resource model of Box 3. We assume that in terms of biomass-specific energetic rates juveniles and adults do not differ, meaning that there are differences on an individual level, since body size differs between stages. Panels show the following cases: juveniles are competitively superior by having an intake rate that is double the adult ingestion rate ($q = 0.5$, A); juveniles and adults have identical intake rates ($q = 1.0$, B); adults are competitively superior ($q = 2.0$, C). Model specification and parameter values are as explained in Box 3 and defined in [9].

Box 4. Population numbers vs biomass

The theoretical background section highlights the distinction between total population density increases with mortality and positive mortality effects in certain life-history stages. Besides the definition of the variable of interest itself, the unit of this variable (i.e., population density) is also highly relevant, especially when dynamics are considered in unstructured vs stage-structured model populations.

The classical approach of measuring population density by a single variable in unstructured models entails the implicit assumption that all individuals of the population are equivalent in their characteristics. Because there is no individual variation in body size, total population density can be readily converted to total biomass, assuming a fixed amount of biomass per individual. Moreover, in an unstructured setting, increasing population biomass necessarily reflects an increase in the numbers of individuals, implicitly reflecting a result of reproduction. As such, the individual-level processes of mortality and reproduction are accounted for, but development (or somatic growth) is ignored in unstructured models [9,28].

By contrast, stage-structured biomass models (Box 3) explicitly account for intraspecific variation in body size and ontogenetic development, which results in a population size distribution. The conversion of total number of individuals to total biomass in the population depends on the population size structure, since individuals with different body sizes do not contribute equally to the population biomass. At the individual level, biomass production in a size-structured population can reflect either somatic growth (i.e., development) or reproductive output, both typically size-dependent processes. Since biomass production through growth and reproduction requires energy, these processes are directly linked to resource availability and can therefore be density dependent and limiting to population growth. The direct link between resource availability and population growth leads to the dual feedback between resources, consumer population density, and size structure that is present in stage-structured models. As a result of that feedback, the size–age relationship of an individual and the population size structure itself are dynamic. The one-to-one relationship between numbers and biomass present in unstructured populations thus disappears in stage-structured models and it is not possible to directly convert population numbers into total biomass.

The relevance of measuring population dynamics in terms of numbers or biomass depends on the research context and question: from a food web perspective, biomass is probably more useful because biomass accounts for the energy flows between species, based on trophic interactions. The same can be true in the context of exploitation and management of a yield-based profit. By contrast, in conservation biology, population numbers are often of higher concern, since the number of individuals determines extinction risk and genetic diversity. In evolutionary ecology, individuals as carriers of alleles between generations are at the heart of theory, and numbers are the relevant metric in that context.

consumer density that is at the basis of hydra effects in unstructured models (Box 1) was thus absent in experiments. We therefore conclude that hydra effects were rarely demonstrated in the experiments even when we saw mortality-induced higher population density (e.g., [1,35], but see [36]). The lack of hydra effects in empirical evidence may therefore reflect a lack of appropriate experimental approach rather than a lack of theoretical support, since the models used to generate hydra effects have only rarely been empirically tested under conditions of logistic resource dynamics. Note also that total density can increase with mortality in ecologically more complex scenarios in stage-structured models with semi-chemostat resource dynamics, especially with ontogenetic niche shifts in resource use (Box 2) (see also points (iv) and (v) below).

(iv) The different positive mortality effects are not necessarily mutually exclusive. For example, juvenile numbers increased with adult mortality in smallmouth bass (*Micropterus dolomieu*) [35], a case of stage-specific overcompensation. At the same time, total population density increased, driven by the higher juvenile numbers, thus representing a hydra effect. This case exemplifies two points: first, the relevance of measuring density in numbers or biomass (Box 4), since it is unclear in this case whether the total density effect in the smallmouth bass population also reflects an increase in total population biomass. Second, the consideration of where the energy sustaining the consumer population is coming from: a clearer picture of whether resource growth follows semi-chemostat dynamics or logistic resource growth can help in distinguishing different types of positive effects (Box 1).

In soil mites, both adult and total numbers increased with egg mortality under maturation regulation [1]. The response in the adult stage is consistent with stage-specific overcompensation while the semi-chemostat resource dynamics theoretically precluded a hydra effect [point (iii)]. The higher total density has been related to a more efficient resource use at the population level of a harvested population where reduced competition through egg mortality prevents the loss of energy through respiration and food consumption of small juveniles that will ultimately starve [1].

(v) The one exception to the points made above is the ciliate experiment by Fryxell *et al.* [36], whereby total numbers increased with mortality. Here, resources likely followed logistic dynamics as ciliates fed on living bacteria and transfer between batches was infrequent relative to generation time. Also, the size differences between protist mother and daughter cells are low compared to birth-maturation size ratios in most multicellular organisms, so that population size structure is less pronounced. Hence, this one experiment might demonstrate a hydra effect, but note that increases in total density can, under some circumstances, also occur in stage-structured models (Boxes 2–4).

Several experiments also report declines in density with mortality (e.g., [37–39]). Both model predictions [5,6] and experiments [2,36] show that stage-specific and total population density ultimately decline when the direct negative effect of mortality reaches an unsustainably high level. Studies in which mortality rates are high or only one mortality treatment is applied might for this reason fail to observe a potential positive mortality effect. Furthermore, not every population fulfils the criteria for the potential of showing positive mortality effects. In unstructured populations, hydra effects are not seen with semi-chemostat resource dynamics [22] or can be masked by the relative timing of discrete census and mortality events [17]. In stage-structured populations no or only weak biomass overcompensation occurs when birth size is close to maturation size or when different life-history stages are ecologically very similar without distinctive ontogenetic asymmetries among stages (symmetry) [5,9,28]

Synthesis and conclusions

The compilation of empirical evidence (Table 2) shows that experiments provide stronger support for stage-specific overcompensation than for hydra effects across both field and laboratory systems. We argue that this is because stage-structured models capture some of the relevant features and processes that determine population growth in natural populations (Box 3), which are ignored by unstructured models. Where unstructured models account just for the processes of mortality and reproduction, stage-structured models in addition account for the process of development (i.e., growth and maturation). Moreover, stage-structured models predict and explain the full diversity of positive mortality effects seen in natural populations. These effects include stage-specific hydra effects as special cases of stage-specific overcompensation and higher total densities in scenarios of ontogenetic niche shifts (Box 2). Theory based on stage-structured models, especially when explicitly accounting for a consistent link between resource availability and the individual energy budget of consumers, hence explains a broader variety of population dynamical effects than does theory based on unstructured models.

Unstructured models do not have the potential for the detailed level of mechanistic explanation present in stage-structured models and do not display the same variety of potential positive mortality effects because they discount size-based differences among individuals in ecological performance and energy budgets. Unstructured models implicitly assume that all individuals have the same body size, or can at least all be represented by an average body size that stays constant under varying environmental (i.e., resource) conditions. Moreover, individuals are ecologically identical and do not grow or mature, but start reproducing directly after birth, while reproduction is the only regulatory process limiting population growth. We argue that these assumptions pose a level of simplification that reduces the dimensionality of the model to an extent at which essential ecological meaning is frequently lost. This point is most clearly substantiated with the observation that there are virtually no organisms that do not undergo some form of ontogenetic development before reproduction can take place; be it growth, metamorphosis, or structural changes in a different form [9,29,40–43].

In this context, it is noteworthy that in unicellular organisms with their allegedly low degree of size structure we still see types of population oscillations, so called single-generation cycles, which are tightly connected to ontogenetic asymmetry and size- and stage-structure [44]. These are precisely the conditions under which stage-specific overcompensation occurs [9,28]. Moreover, single-generation cycles are much more common in nature than consumer-resource cycles [45]; the latter being linked to unstructured models and hydra effects. Together, the widespread occurrence and predominance of single-generation cycles again suggest, as do the patterns of positive mortality effects found in natural populations (Table 2), that explicitly accounting for within-population variation in body size and size-dependent vital rates leads to an intuitive, predictive, and more biologically mechanistic ecological theory than what is currently presented by unstructured modelling approaches.

Based on the experimental support and the prevalence in nature of population size- and stage-structure and ontogenetic asymmetry, we argue that stage-specific overcompensation can be expected to be a common phenomenon in natural populations with all its implications for community and ecosystem dynamics [6,8–10,43], and population management [11–13]. This summary fits well with the current shift in ecological science towards a focus on trait variation among individuals, with body size as a dominant and unifying individual trait [8,42,46], and the limited value of simple, unstructured models for understanding ecological dynamics [47]. By contrast, hydra effects are likely to be scarce and limited to particular life-histories that lack individual differences (ontogenetic symmetry) and to certain environmental circumstances (logistic resource dynamics).

Future research directions

Most theoretical and experimental studies on positive mortality effects assume simple trophic configurations and highly simplified environmental or applied scenarios that rarely occur in nature or management situations. For example, experimental tests of model predictions for positive mortality effects in populations with more complex life histories, including ontogenetic habitat or resource niche shifts, have so far not been conducted (Box 2). Stage-specific overcompensation occurs when resource supply is either constant or variable [1], but how temporally variable mortality, common in pest control or exploitation scenarios, would affect this pattern is not yet clear (but see [17]). Very few studies have investigated commonly practised exploitation strategies beyond proportional harvesting while also varying harvest rates, with inconsistent results [36,39]. The same is true for the effects of mortality in spatially structured populations in which size- and stage-dependent movement can alter local densities through emigration and immigration [32,38]. Stage-specific overcompensation [48,49] and hydra effects [50] can affect the source–sink dynamics of connected habitats differing in mortality rates, with yet unexplored consequences for reserve design in conservation ecology or spatial fish stock management using Marine Protected Areas. Pesticides and pollutants often have latent effects on life-history traits, behaviour, and vital rates [51–53]. These latent effects can indirectly change the recovery time of population size structure [54] or interact with other agents of mortality, relaxing density dependence [55], which together suggest more complex mortality effects of toxicants than is often assumed in studies on positive mortality effects. Stage-specific overcompensation can have wide-ranging effects on communities with few species [9,11]. However, models of more complex food webs usually ignore population size or stage-structure, and ontogenetic asymmetry (e.g., [56]), which nonetheless can affect food web structure and stability [43], an observation again reiterating the potential role that overcompensation may play in the dynamics of food webs.

This review summarises positive effects from increasing mortality in consumer populations both in theoretical and in empirical systems. We have made a distinction among positive mortality effects on the basis of differences in the

underlying mechanisms leading to these effects. Focus of future studies should be on the developmental, ecological, and evolutionary processes that result in positive mortality effects. In particular, more complex environmental and ecological (trophic) scenarios should be addressed in this context. Empirical testing of the critically different hypotheses underlying hydra effects and biomass overcompensation would yield great ecological insight in the responses of consumer populations to changing environmental conditions.

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