They note that, for approximately half of their studies, the original study’s authors, [3], or both claimed that the responses were hydra effects; this is because most used the correct definition. Schröder’s own empirical study in Table 2 exhibits a clear hydra effect in response to juvenile mortality. SVC largely ignore natural enemies as mortality factors. However, decreased foraging in response to increased predation risk readily generates hydra effects. It has been shown to be common and to often increase the prey’s food intake rate because of greater resource abundance (see references in [3]).

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References

Empirical support for different types of positive mortality effects. A reply to Abrams

Arne Schröder¹, Anieke van Leeuwen², and Tom C. Cameron³

¹Department 4: Biology and Ecology of Fishes, Leibniz-Institute of Freshwater Ecology and Inland Fisheries, Müggelseedamm 310, 12587 Berlin, Germany
²Department of Ecology and Evolutionary Biology, Princeton University, 106A Guyot Hall, Princeton, NJ 08544, USA
³School of Biological Sciences, University of Essex, Colchester, CO4 3SQ, UK

We thank Peter Abrams for his comments and thoughts [1] on our recent review of the empirical evidence for positive population-level effects of mortality, defined as an increase with increasing mortality in the numbers or biomass of a total population or of a specific life-history stage or size class within the population [2]. This evidence was compared with predictions of mathematical models. Empirically demonstrated positive mortality effects were predominately stage-specific, a pattern (stage-specific overcompensation [3]) most congruent with predictions by models that account for consumer stage-structure and ontogenetic development of individuals [3–5]. Increases in total density as predicted by unstructured models (hydra effects [6,7]) were rare in empirical studies (occurring in 1 of 15). We concluded that the type of positive mortality effects found in experiments corroborates the perspective of stage-structured population theory [3–5,8,9].

Abrams [1] claims that our ‘conclusion is based on an erroneous definition of hydra effect and an inadequate review of relevant evidence’. His arguments to support this claim are: (i) hydra effects occur under a far wider range of ecological scenarios than the ones we looked at, and (ii) stage-specific positive mortality effects often are hydra effects. By contrast, we argue that Abrams’ claim is based on a misunderstanding of the focus in our review.

First, Abrams [1] argues that hydra effects in general do not require logistic resource growth dynamics and saturating functional responses, and are not tied to consumer-resource cycles, in contrast to how we used the term for our purpose of comparing model predictions and experimental evidence. We agree and explicitly acknowledged that the connection between instability and hydra effects found in unstructured models breaks down in more complex ecological scenarios (Box 2 and p. 615 in [2]). Allee effects in resource populations, multiple interacting species, consumer interference, and reduced foraging activity based on altered morphological traits or behavior of individuals can all lead to hydra effects in stable unstructured consumer-resource systems [1,7]. Nevertheless, we maintain that hydra effects in unstructured one-consumer one-resource models with constant individual behavior or morphology are tightly linked to logistic resource growth and a type II functional response, in other words to systems displaying consumer-resource cycles.

We restricted our review of models predicting positive mortality effects to those of only one consumer and one resource which do not incorporate such ecological scenarios. For example, in the models we considered, per capita foraging activity of individuals does not change with mortality pressure and consumer interference does not occur.
This is also true for the stage-structured models we considered in which individual body size and life-history stage change in response to resource density via food-dependent somatic growth, and are not trait changes directly induced by top predators or other mortality agents.

This restriction was necessary to allow comparison of model predictions with multigenerational experiments explicitly addressing the population responses to mortality. Most experiments accounted for only one consumer and one resource (10 cases; 4 field studies looked mainly at pairwise interactions), and the resource was often dead matter (9 cases). Stage-structure was present in or at least only reported for consumer populations. Because mortality was induced by experimenters (14 cases) and not by manipulating predator density, trait responses and behavioral adjustment to natural enemies were precluded by the experimental designs. In the one case where mortality was due to a pathogen [10], details on the number of interacting populations or individual trait changes were not given. Allee effects and consumer interference were not present, or at least were not quantified, in any of the experiments. Importantly, it was this restriction that allowed us to link empirical findings to processes associated with positive mortality effects, rather than reporting theoretical, phenomenological patterns based on a mixture of mechanisms.

Our conclusions are therefore not based on an inadequate review but rather on a limited set of experimental conditions. Of course we welcome any additional references to manipulation experiments testing model predictions. We do not, however, consider negative responses to immigration in simulated food webs as empirical evidence for positive mortality effects, to say nothing of the mechanistic processes underlying those further unspecified effects. In the same vein, the occurrence of reduced foraging activity under threat of predation is, in our view, not a direct empirical support for any type of positive mortality effect.

Second, Abrams [1] argues that hydra effects occur in stage-structured populations when the stage in which density increases with mortality is subject to this mortality [1,7]. By this definition, which was followed in [2] (see Glossary and Table 2), only 4 of the 14 experimentally demonstrated stage-specific positive mortality effects constitute stage-specific hydra effects. More generally, stage-specific positive mortality effects occurred in the size class or life-history stage least regulating and limiting population growth, independently of what stage was targeted by mortality, and even when mortality was not stage-specific. For example, juvenile biomass in [11] increased through mortality-triggered relaxation of density-dependent adult reproduction rate not only under elevated juvenile mortality but also when adult mortality was raised [11], and even when mortality was stage-independent [12]. While the first result would fall within the definition of a stage-specific hydra effect, the complete pattern of positive mortality effects found in experiments is best captured by the concept of stage-specific overcompensation as predicted by stage-structured one-consumer one-resource models [3–5], including the underlying mechanisms. This distinction illustrates clearly that ‘hydra effect’ is used rather as an umbrella term, covering a variety of predicted patterns caused by diverse ecological mechanisms, many of which currently lack empirical support. By contrast, stage-specific overcompensation refers to a concept that is consistently based on a well-supported biological process (i.e., food-dependent growth) and a distinct mechanism (relaxation of density-dependence), and explains the full range of experimentally demonstrated population-level responses to mortality from an individual-level understanding.

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10 Ohlberger, J. et al. (2011) Stage-specific biomass overcompensation by juveniles in response to increased adult mortality in a wild fish population. Ecology 92, 2175–2182

In Table 2 in [2] we have in several instances denoted stage-specific overcompensation with ‘overcompensation’ for brevity. The columns indicating in what stage density rises or declines with increasing mortality clarifies this meaning.