Life-History Multistability Caused by Size-Dependent Mortality

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Submitted June 11, 2017; Accepted January 22, 2018; Electronically published April 27, 2018

Dryad data: http://dx.doi.org/10.5061/dryad.tb23m.

ABSTRACT: Body size is a key determinant of mortality risk. In natural populations, a broad range of relationships are observed between body size and mortality, including positive and negative correlations. Previous evolutionary modeling has shown that negatively size-dependent mortality can result in life-history bistability, with early maturation at small size and late maturation at large size representing alternative fitness optima. Here we present a general analysis of conditions under which such life-history bistabilities can occur, reporting the following findings. First, alternative fitness optima can be found for any arbitrarily chosen forms of mortality functions, including functions according to which mortality smoothly declines with size. Second, while bistabilities occur more readily under negatively size-dependent mortality, our analysis reveals that they can also emerge under positively size-dependent mortality, a feature missed in earlier work. Third, any sharp drop of mortality with size facilitates bistability. Fourth, if the mortality regime involves more than one such sharp drop, multistable life histories can occur, with alternative fitness optima straddling each of the drops. Paradoxically, our findings imply that, fifth, a species-poor predator community capable of creating a rugged mortality regime is conducive to evolutionary multistability, which could act as a stepping stone toward prey life-history diversification, whereas a species-rich predator community that results in a smoothly varying mortality regime may prevent diversification through this pathway.

Keywords: body size evolution, life history, size-dependent mortality, size refuge, predation, prey diversity, model.

Introduction

Body size is a key life-history trait, which strongly impacts the Darwinian fitness of organisms by affecting the survival and reproduction of individuals (Roff 1992). The functional relationship between fecundity and body mass is often straightforward because the former is typically roughly proportional to the latter. In contrast, the relationships found in nature between body size and mortality are much more diverse and essentially can take any form. Most often, survival prospects have been found to increase with body size (e.g., Kingsolver and Huey 2008), particularly in aquatic environments (Perez and Munch 2010). Reasons for a pervasive survival advantage of large body size include gape limitation among predators and other restrictions on their handling of large prey (Wellborn 1994; Sogard 1997), better predator evasion abilities among prey (Schürch and Taborsky 2005; Husak 2006), and improved starvation resistance in risky environments (Krause et al. 1998; Segers and Taborsky 2011). In contrast, a positive size dependence of mortality can arise when predators preferentially consume larger prey (Hanson et al. 1989; Wellborn 1994; Johnson and Belk 2001) or when larger prey are exploited more heavily because they are easier to detect. When predators prefer intermediate-sized prey (Hayward and Kerley 2005), even hump-shaped relationships between mortality and body size may emerge.

Previous theoretical work has shown that negatively size-dependent mortality readily gives rise to life-history bistability and alternative fitness optima in body size (Ratner and Lande 2001; Day et al. 2002; Taborsky et al. 2003; Gårdmark and Dieckmann 2006), which can even stably coexist under the concurrent action of frequency-dependent selection (Taborsky et al. 2012). This bistability results from two antagonistic selection pressures. First, negatively size-dependent mortality will select for life histories with delayed reproduction, if this allows individuals to outgrow the most intense mortality risk faced at small sizes, that is, if they can reach a size refuge (e.g., Urban 2008). For example, some bivalve species start to reproduce only after reaching a refuge size threshold beyond which they are safe from their major predator (Nakaoka 1998; Boulding et al. 2017). Second, life-history strategies with short juvenile growth periods (and consequently small adult body sizes) can be advantageous because they maximize the chance of reaching

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Am. Nat. 2018. Vol. 192, pp. 62–71.
DOI: 10.1086/697412

adulthood and accruing at least some reproductive output during their lifetime (Taborsky et al. 2003).

In contrast to negative size dependence, the possibility that bistability may arise when mortality increases with body size has been suggested in only one previous study (Gårdmark and Dieckmann 2006). The rationale is that under certain conditions the fecundity advantage of growing to large body sizes may outweigh the disadvantage of higher mortality risk at those body sizes.

Evolutionary bistabilities are important for at least three different reasons. First, they can lead to evolutionary hystereses, through which environmental changes could suddenly push a population from the basin of attraction of one local optimum to that of another. Such changes might not be readily reversed. Second, a bistability can confound environment-trait relationships, obscuring our ability to detect important drivers of trait change. Third, and perhaps most importantly, they can facilitate evolution of within-species polymorphisms and, eventually, speciation. As such, multiple fitness peaks represent alternative evolutionary outcomes, only one of which can persist in the long term. However, if there is an additional mechanism that causes individuals with phenotypes at one fitness peak to compete more intensively among themselves than with those at the other fitness peak, coexistence can result. Such competition is not included in the analysis presented here, but Taborsky et al. (2012) have shown that size-dependent competition can allow phenotypes at different fitness peaks to coexist. Over time, genetic drift or further adaptation could cause such intraspecific morphs to evolve into separate species. Thus, evolutionary bistability may well represent the first step on a path toward speciation.

Here we provide the first comprehensive analysis of general properties that mortality functions with either negative or positive slope must possess in order to give rise to life-history bistability. Moreover, we identify which ecological conditions limit the evolutionary emergence of alternative fitness optima. Finally, we investigate the intriguing possibility that monotonically decreasing mortality functions may even give rise to multiple alternative fitness optima in body size. A worked example shows how multistability may arise in natural communities, potentially affecting their biodiversity.

Model

We consider the mortality rate m as a monotonic function of body size s,

$$m(s) = m_i + m_d M(s), \tag{1}$$

where $m_i \geq 0$ scales the size-independent mortality component, $m_d \geq 0$ scales the size-dependent mortality component, and $M(s) \geq 0$ is a monotonic function describing the shape of size dependence. We assume that the function $M(s)$ respects the following three constraints but can otherwise take any arbitrary form: for negative size dependence, $\lim_{s\to\infty} M(s) = 0$, $M(0) = 1$, and $M'(s) \leq 0$ for all s, whereas for positive size dependence, $\lim_{s\to\infty} M(s) = 1$, $M(0) = 0$, and $M'(s) \geq 0$ for all s. Thus, for negatively size-dependent mortality functions, $\lim_{s\to\infty} m(s) = m_i$, $m(0) - m_i = m_d$, and $m'(s) \leq 0$ for all s, whereas for positively size-dependent mortality functions, $\lim_{s\to\infty} m(s) = m_i + m_d$, $m(0) = m_i$, and $m'(s) \geq 0$ for all s.

Life History

To analyze the evolutionarily optimal body sizes resulting from different shapes of $M(s)$, we model a simple iteroparous life history with determinate growth (Taborsky et al. 2012). This life history consists of a juvenile period with linear growth followed by an adult period with continuous reproduction and no growth. The adult size of an organism thus equals its maturation size $s_M \geq 0$. We obtain fitness as lifetime reproductive success,

$$R_0(s_M) = P(s_M) m^{-1}(s_M) b(s_M). \tag{2a}$$

This fitness function compromises three factors. First,

$$P(s_M) = p \exp\left(-\int_0^{(s_M - s_B)/g} m(s_B + gt)\mathrm{d}t\right) \tag{2b}$$

is the survival probability until maturation, with $s_B > 0$ denoting the size at birth, $0 < p < 1$ denoting the probability of newborn survival, and $g > 0$ denoting the growth rate until maturation. Second, $m^{-1}(s_M)$ is the expected lifetime after maturation. Third,

$$b(s_M) = g\alpha\gamma s_M^{\gamma-1} w_0^{-1} \tag{2c}$$

is the birth rate after maturation, with $w_0 > 0$ denoting the weight of a newborn. This birth rate follows from considering that an individual's weight w is allometrically related to its body size according to $w = \alpha s^\gamma$, with $\alpha > 0$ denoting the allometric scaling coefficient and $\gamma > 1$ the allometric exponent. Because there is no growth after maturation, the birth rate is given by the rate of weight acquisition at maturation,

$$\frac{\mathrm{d}}{\mathrm{d}t} w = \alpha\gamma s^{\gamma-1} \frac{\mathrm{d}}{\mathrm{d}t} s = g\alpha\gamma s^{\gamma-1}$$

for $s = s_M$, divided by the weight of a newborn.

Size-Dependent and Size-Independent Mortality

To exemplify the results of our general analysis of properties of size-dependent mortality, we choose $M(s)$ as a logistic function of the form

$$M(s) = \frac{1}{1 + e^{-(s-s_T)/\tau}}, \tag{3}$$

where s_T denotes the size at which the logistic function has its steepest slope (at its inflection point) and τ defines the steepness and direction of the size dependence. Positive and negative values of τ result in positive and negative size dependence, respectively. Low absolute values of τ result in an abrupt, almost step-like transition, whereas larger values of τ result in a more gradual transition between low and high mortality. We assume that $|\tau|$ is small enough relative to s_T such that the condition $M(0) = 0$ or $M(0) = 1$ is approximately fulfilled.

As an alternative, more mechanistic way of defining size-dependent mortality, we also consider a scenario in which a prey's mortality results from the joint action of multiple predator species. We generate such mortality landscapes by assuming that there are n predator species, each imposing a size-dependent mortality rate following a Gaussian distribution. These distributions have means forming a geometric series and standard deviations determined by a coefficient of variation that is the same for all predator species. The total mortality rate imposed by this predator community is the sum of species-specific contributions and approximately follows a power law with negative exponent, as is often assumed for marine communities (e.g., Peterson and Wroblewski 1984; Andersen et al. 2009; Jørgensen and Holt 2013). For our illustrations, we use $n = 20$ predator species, with the smallest species imposing a mortality rate peaking at a mean body size of 0.1 and with the means for the subsequent pairs of species having a constant ratio of 1.3. We use a coefficient of variation of 40% for all predator species and assume a size-independent mortality rate of $m_i = 0$. Removing a single predator species creates a mortality landscape with a dip when plotting mortality rate against body size on a double-logarithmic scale, which implies a deviation from the aforementioned power law.

Model Reduction

The life-history model based on the logistic mortality function $m(s)$ (eq. [3]; fig. 1*a*), which we examine to illustrate our general analysis, features 10 parameters: p, α, w_0, γ, s_B, m_i, m_d, g, s_T, and τ. The three parameters p, α, and w_0 affect fitness only multiplicatively (eqq. [2]), and the ratio $p\alpha/w_0 = 1$ can thus be set to any value without influencing the position of fitness optima. As shown by (Taborsky et al. 2003), values of γ, as long as $\gamma > 1$, do not affect results qualitatively. Therefore, similar to previous studies of size-dependent mortality (Taborsky et al. 2003, 2012), we perform all analyses for $\gamma = 3$, corresponding to an isometric length-weight relationship. Moreover, in our model, s_B affects only survival until maturation, $P(s_M)$, and we have shown previously that when choosing s_B small relative to s_M, it does not essentially affect $P(s_M)$ (Taborsky et al. 2003), so we let s_B approach 0 in our analysis. Four parameters specify growth and mortality and have the dimensions of time^{-1} (m_i and m_d), size (s_T), or size × time^{-1} (g). We can conveniently choose the units by which we measure size and time as s_T and s_T/g, respectively. Consequently, we can exhaustively analyze our model with logistic mortality by three dimensionless parameters, $r_i = m_i s_T/g$, $r_d = m_d s_T/g$, and τ/s_T (Gårdmark and Dieckmann 2006).

Results

Because successful reproduction requires acquiring energy and surviving until maturation, all fitness functions $R_0(s_M)$ of our model share two properties, namely that reproductive success is 0 if maturation takes place immediately after birth, $R_0(0) = 0$, or if maturation is delayed indefinitely, $\lim_{s_M \to \infty} R_0(s_M) = 0$. This implies that $R_0(s_M)$ in general is hump shaped but can possess one or more local maxima.

An evolutionarily optimal body size at maturation, s_M, maximizes lifetime reproductive success, so

$$R_0'(s_M) = -[s_M m^2(s_M) + g(1-\gamma)m(s_M) + s_M g m'(s_M)] \alpha w_0^{-1} \gamma s_M^{\gamma-2} P(s_M) m^{-2}(s_M) = 0. \tag{4a}$$

This implies that the term in square brackets is 0,

$$F(s_M) = \underbrace{s_M (r_i + r_d M(s_M))^2}_{(1)} + \underbrace{g(1-\gamma)(r_i + r_d M(s_M))}_{(2)} + \underbrace{s_M g r_d M'(s_M)}_{(3)} = 0. \tag{4b}$$

The function $F(s_M)$, thus defined, comprises three additive terms (fig. 1*b*, 1*c*), of which term (1) is always positive, term (2) is always negative, and term (3) is negative (positive) when the size dependence of mortality is negative (positive). Importantly, the magnitude of term (3) is largest at those body sizes at which mortality changes most.

Alternative Evolutionarily Optimal Adult Body Sizes

If two alternative size optima exist, the function $F(s_M)$ must possess three zeros: two corresponding to the fitness maxima and one to the fitness minimum in between. For the two zeros representing the fitness maxima, the second derivative of R_0 is negative, so the first derivative of F is positive. Conversely, for the zero representing the fitness minimum, the first derivative of F is negative. To assess how many zeros the function F possesses, it helps to understand how terms (1)–(3) vary with s_M. Term (1) is nonnegative and sets off at 0 at $s_M = 0$; for negatively size-dependent mortality, it has an initial slope of $(r_i + r_d)^2$ and increases

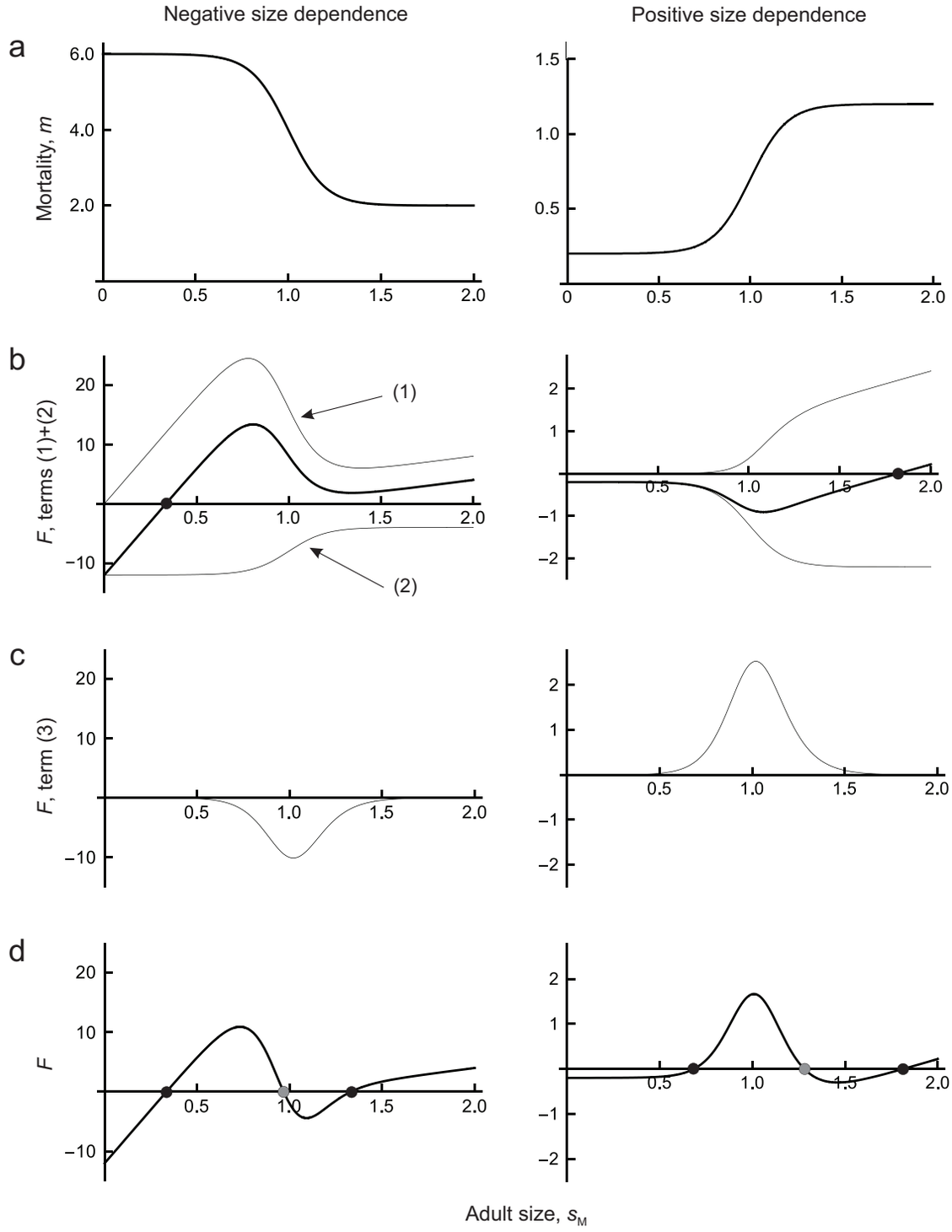

Figure 1: Graphical representation of the three terms of the function $F(s_M)$. All panels are based on the logistic mortality function in equation (3) with negative slope (*left*) or positive slope (*right*). *a*, Negative and positive size dependence of mortality. *b*, Terms (1) and (2) of $F(s_M)$, respectively (thin lines), and their sum (1) + (2) (thick line). *c*, Term (3) of $F(s_M)$. *d*, Sum of all three terms of $F(s_M)$. Black circles indicate maxima and gray circles indicate minima of the fitness $R_0(s_M)$. Parameters: $r_d = 4$, $r_i = 2$, $s_T = 1$, $\tau = -0.1$ (*left*); $r_d = 1.0$, $r_i = 0.1$, $s_T = 1$, $\tau = 0.1$ (*right*).

nonmonotonically to an infinite value with a shallower asymptotic slope of r_i^2 (fig. 1*b*, *left*), whereas for positively size-dependent mortality, it has an initial slope of r_i^2 and increases nonmonotonically to an infinite value with a steeper asymptotic slope of $(r_i + r_d)^2$ (fig. 1*b*, *right*). Term (2) is negative; for negatively size-dependent mortality, it monotonically increases from an initial value of $-g(\gamma - 1)(r_i + r_d)$ at $s_M = 0$ to an asymptotic value of $-g(\gamma - 1)r_i$ (fig. 1*b*, *left*), whereas for positively size-dependent mortality, it monotonically decreases from an initial value of $-g(\gamma - 1)r_i$ at $s_M = 0$ to an asymptotic value of $-g(\gamma - 1)(r_i + r_d)$ (fig. 1*b*, *right*). For negatively size-dependent mortality, term (3) is nonpositive and decreases from 0 at $s_M = 0$ to a minimum, denoted by s_3, before increasing again to an asymptotic value of 0 (fig. 1*c*, *left*). This pattern is mirrored for positive size dependence, for which term (3) is nonnegative (fig. 1*c*, *right*).

Summing the positive term (1) and the negative term (2), we see that this sum is negative at $s_M = 0$ and positive for $s_M \to \infty$. Therefore, this sum has at least one zero, which we denote by $s_{(1)+(2)}$ (fig. 1*b*). If F has more than one zero (fig. 1*d*), this can be due to the nonmonotonic term (3) (fig. 1*c*).

Ecological Constraints on the Existence of Alternative Fitness Optima

The qualitative analysis presented above suggests that for any pair (r_i, r_d), we can choose a function m that will yield three zeros of F and thus two maxima of R_0. We now discuss this conjecture, considering negatively and positively size-dependent mortalities in turn.

For negatively size-dependent mortalities, three zeros of F are obtained by choosing M as a function sharply dropping from 1 to 0 at a suitable size $s_{(3)} > s_{(1)+(2)}$. For less sharp drops of M, the choice of (r_i, r_d) pairs yielding three zeros of F becomes more difficult (1) when r_d is small, since this diminishes the magnitude of term (3) and causes the sum $(1) + (2)$ to increase monotonically; (2) when r_i is large, since this steepens the initial slope of the sum $(1) + (2)$; and (3) when r_i is small, since this flattens the asymptotic slope of the sum $(1) + (2)$.

We illustrate these results by analyzing logistic mortality functions with negative slopes that vary in the parameter $\tau < 0$ determining their steepness (eq. [3]; fig. 2, *top*). This shows that no alternative fitness optima exist (1) when both r_d and r_i are small and (2) when r_i is large. These findings can be understood by considering under which ecological conditions a size refuge at large body size confers fitness benefits. (1) For small r_d and r_i, the mortality risk is so small that reaching maturity quickly and reproducing early is the only optimal life-history strategy. With increasing r_d, mortality at small sizes increases sufficiently to open up an alternative life-history pathway that relies on a size refuge by delaying reproduction and growing large. (2) With increasing r_i, the expected life span decreases, and thereby the benefits of delaying reproduction in order to reach the size refuge diminish: thus, the only optimal life-history strategy is to start reproducing as early as possible.

For positively size-dependent mortalities, three zeros of F are obtained by choosing M as a function sharply increasing from 0 to 1 at a suitable size $s_{(3)} < s_{(1)+(2)}$. Choices of M achieving this are more difficult (1) when r_d is small, since this diminishes the magnitude of term (3); (2) when r_d is large, since this steepens the asymptotic slope of the sum $(1) + (2)$ and thereby lifts its minimum above 0; and (3) when r_i is large, since this steepens the initial and final slopes of the sum $(1) + (2)$.

Again, we illustrate these limits to bistability by varying the parameter $\tau > 0$ in logistic mortality functions with positive slope (eq. [3]; fig. 2, *bottom*). In comparison to the case of negatively size-dependent mortality, we see that for positive size dependence the parameter range where bistability can occur is significantly smaller. In contrast to the case of negative size dependence, for mortalities increasing with size, we were not able to choose a function M yielding three zeros of F for every (r_i, r_d) pair when letting the parameter τ approach 0. When mortality increases with body size, no size refuge can be sought by growing large. For this scenario, growing to a large size yields fitness benefits only through increased fecundity. Therefore, as soon as the overall mortality exceeds a certain level (be it through an increase of the size-dependent or the size-independent component), growing to a large size does not pay, and reproducing early and at small size is the only optimal life-history strategy. Conversely, for very small r_d, where mortality changes only very little with increasing size, the only optimal life-history strategy is to delay reproduction and grow large, thereby enjoying the resultant fecundity advantage.

Shapes of Mortality Functions Causing Bistability

Importantly, the occurrence of bistability is not limited to logistic mortality functions. Instead, mortality functions causing bistability can have arbitrary shapes. In particular, the function F can possess three zeros even when the mortality function is monotonically decreasing. Thus, bistability exists also for an extensive range of (r_i, r_d) pairs when the mortality rate follows a negative exponential function or a negative power function (fig. 3).

Conditions for Multistability

Just as a single sharp drop of mortality can create a threshold size that is straddled by a life-history bistability, multiple sharp drops can lead to evolutionary multistability. More precisely, we predict that choosing $M(s_M)$ with n

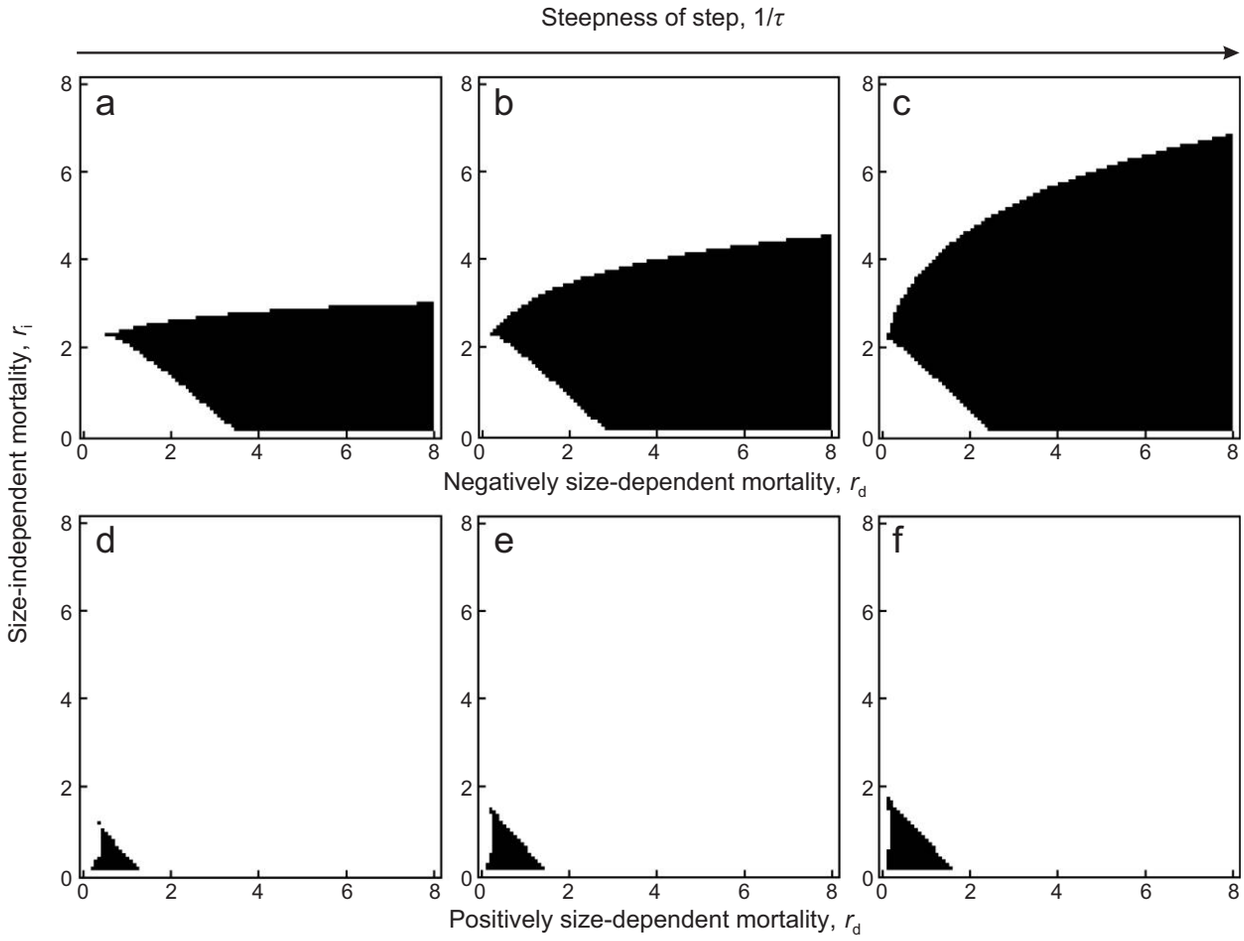

Figure 2: Combinations of size-dependent and size-independent mortalities causing two alternative fitness optima in body size (shaded areas). The mortality functions for negative size dependence (*a–c*) and positive size dependence (*d–f*) are as in figure 1. Panels from left to right are based on logistic mortality functions with an increasing steepness of steps. Parameters: $\tau = -0.1$ (*a*), $\tau = -0.05$ (*b*), $\tau = -0.025$ (*c*), $\tau = 0.1$ (*d*), $\tau = 0.05$ (*e*), $\tau = 0.025$ (*f*). Other parameters: $s_\Gamma = 1$. The code used to generate figures 2 and 3 has been deposited in the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.tb23m (Taborsky et al. 2018).

sharp drops will yield $n + 1$ maxima of R_0. In accordance with this prediction, for negative size dependence of mortality, we find three fitness maxima when the mortality function possesses two sharp drops (fig. 4). This scenario could occur, for example, in a community with two main predators that are differently limited in their prey size spectrum because of their different gape sizes, which opens up two size refuges for the prey.

Role of Predator Diversity

Above we have shown that rugged size-dependent mortality functions may lead to multistability. We suggest that species-poor predator communities represent one ecological scenario for the occurrence of such rugged mortality landscapes: when there are relatively few size-selective predators, it becomes likely that there are prey sizes that are outside the optimal size range of any single predator species. In contrast, a species-rich predator community is likely to result in a mortality landscape that is smoother. Thus, we predict that a loss of diversity in a predator community may trigger the emergence of evolutionary multistability and therefore, somewhat paradoxically, create conditions that favor diversification among the affected prey.

To test this hypothesis, we investigate the fitness consequences of size-selective mortality when the mortality pattern results from a hypothetical predator community. We assume that there are a large number of predator and prey species, such that from the perspective of a single prey species, the mortality imposed by the predator community is

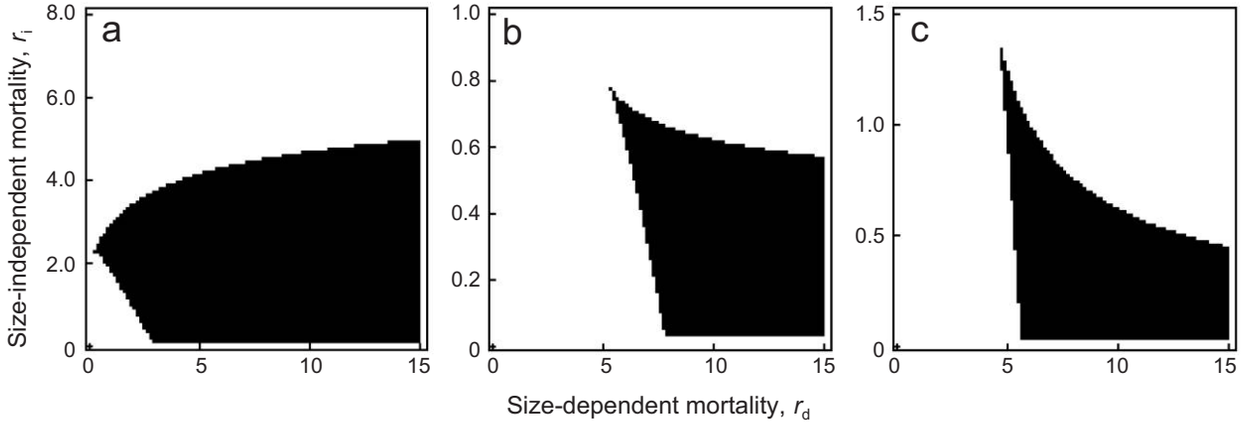

Figure 3: Combinations of size-dependent and size-independent mortalities causing two alternative fitness optima in body size for three mortality functions with monotonically decreasing slopes. *a*, Logistic function (eq. [1]) with $\tau = -0.05$. *b*, Exponential function $m(s) = r_i + r_d \exp(-s/s_0)$ with $s_0 = 1$. *c*, Power function $m(s) = r_i + r_d(s + s_0)^b$ with $s_0 = 0.5$ and $b = -2.5$.

constant, without a feedback from the prey density to the predator densities. Each predator is generating a predation pressure that is described by a Gaussian distribution centered on a species-specific optimal prey body size. When these mean prey body sizes are spaced such that the ratio of adjacent means takes a fixed value, the resulting total mortality is very smooth (fig. 5*a*) and closely follows a power law (for the parameter setting outlined in "Model," it has a slope of −0.98). This smooth mortality function results, as expected, in a single evolutionarily optimal maturation size for the prey (fig. 5*c*). If we remove predator species from this community, the overall mortality is reduced, but more importantly, slight irregularities are created in the mortality landscape, corresponding to the mean optimal prey body sizes of the removed predators (fig. 5*b*). These irregularities translate into additional evolutionarily optimal maturation sizes for the prey (fig. 5*d*). Additional optimal prey maturation sizes arise from removing a predator species only if the removed predator's mean prey size is sufficiently distinct from that of other predators already removed before. In the community of 20 predator species shown in figure 5, the seventh and fourteenth smallest predator species are removed, creating a total of three alternative fitness optima for the prey (fig. 5*b*, 5*d*).

Discussion

Our general analysis of how properties of mortality functions affect fitness shows that (1) life-history bistabilities can arise for any combination of size-dependent and size-independent mortality rates, if mortality decreases with body size by a sharp drop, with the alternative fitness optima straddling the drop; (2) the conditions for bistability become more demanding for mortality functions with smoother drops or arbitrary shape; (3) life-history bistabilities can emerge also under positively size-dependent mortality, a feature missed in earlier work; and (4) multistable life histories can occur if mortality regimes involve more than one abrupt drop. Para-

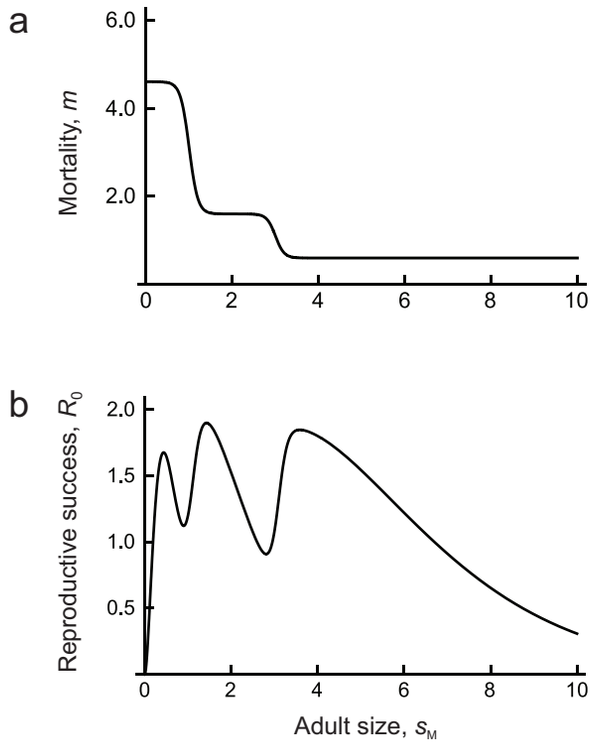

Figure 4: Logistic mortality function with two steps (*a*) gives rise to multistability with three alternative fitness optima in body size (*b*). The two-step mortality function is given by $m(s) = r_i + r_{d1}/[1 + e^{-(s-s_{T1})/\tau}] + r_{d2}/[1 + e^{-(s-s_{T2})/\tau}]$, with $s_{T1} = 1$, $s_{T2} = 3$, $r_{d1} = 3$, $r_{d2} = 1$, $r_i = 0.6$, and $\tau = -0.1$. The ratio $p\alpha/w_0$ is set to 100.

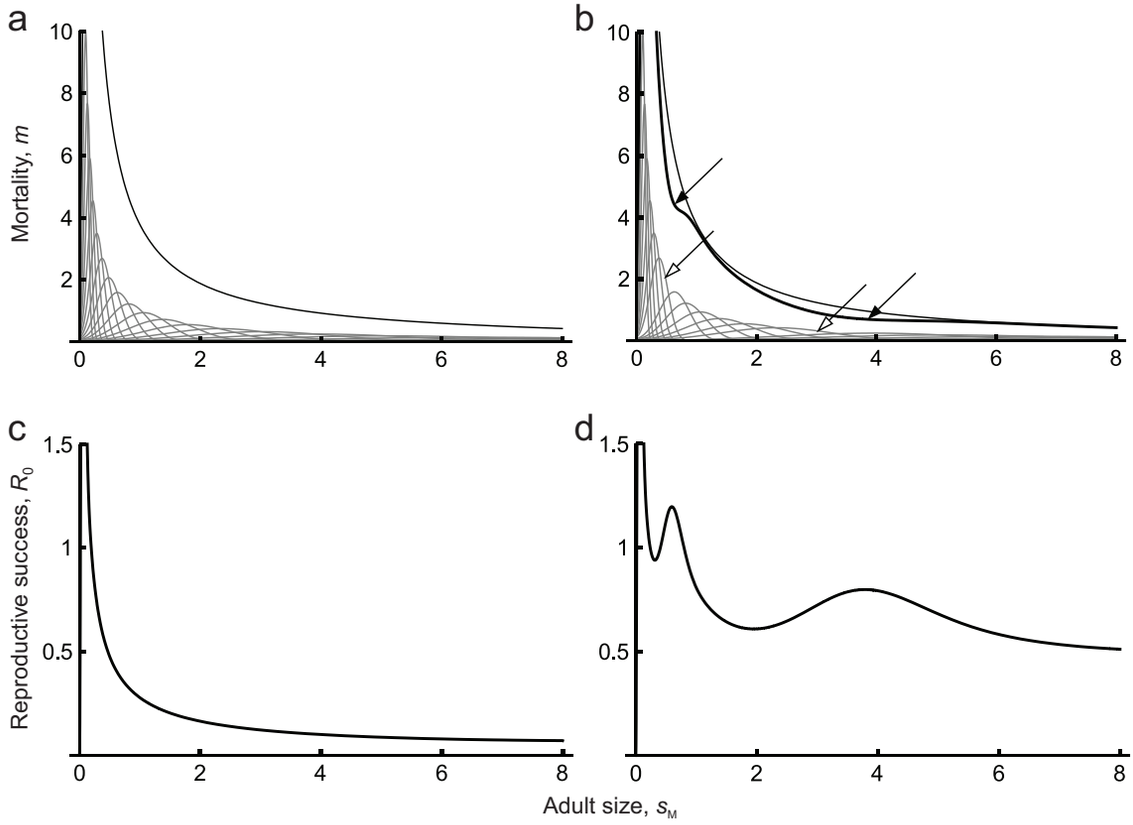

Figure 5: Effect of predator communities on prey diversification in body size. *a*, *c*, A species-rich predator community imposes a smooth size-dependent mortality landscape following a power law with negative exponent; this allows for only a single optimal prey size. *b*, *d*, Removal of the seventh and fourteenth smallest predators from this community causes irregularities in the mortality landscape and opens up two size refuges, allowing for alternative optimal prey sizes. *a*, *b*, Size-frequency distributions of predator species (gray lines) and the mortality rates they impose (black lines). *b* shows the mortality rate resulting from species removal (thick line) and the mortality rate of the original community for reference (thin line), with open arrows pointing toward where the two predator species have been removed and filled arrows pointing toward the induced irregularities in the mortality landscape where their fitness effects are largest. *c*, *d*, Resultant prey fitness $R_0(s_M)$ in dependence of adult body size. The ratio $p\alpha/w_0$ is set to 5,000.

doxically, our findings imply that a species-poor predator community capable of creating a rugged mortality landscape is conducive to multistability that could act as the first step in the life-history diversification of the affected prey species, whereas a species-rich predator community that results in a smoothly varying mortality landscape may prevent diversification through this pathway.

The finding that bistability can be attained for both negative and positive size dependence is particularly noteworthy, as so far theoretical research (Chase 1999; Day et al. 2002; Taborsky et al. 2003, 2012; Gårdmark and Dieckmann 2006) and empirical research (Nakaoka 1998; Chase 1999; Urban 2007, 2008; Engqvist and Taborsky 2016; Boulding et al. 2017) on life-history bistability have focused on negative size dependence. Positively size-dependent mortality can arise by preferences of predators for larger prey (e.g., Hanson et al. 1989; Wellborn 1994; Johnson and Belk 2001). This includes human-induced size-selective mortality, such as resulting from hunting and commercial or sport fishing, which often exert unnatural selection (sensu Allendorf and Hard 2009) against large body size. For instance, when comparing different types of fishing methods in nine populations of sockeye salmon (*Oncorhynchus nerka*), larger than average fish were caught preferentially (positively size-selective fishing). Remarkably, however, in all studied populations of sockeye salmon, fishing produced strong disruptive selection on body size (Kendall and Quinn 2012).

Our model results clearly indicate that the conditions causing life-history bistability are considerably more stringent for positive than for negative size dependence of mortality. While for negatively size-dependent mortality, phenotypes delaying reproduction and growing large enjoy a twofold benefit later in life through a lower mortality risk and higher fecundity, organisms delaying reproduction

when mortality risk increases with size can enjoy only a single benefit through higher fecundity.

Mortality functions with a single drop may result from prey being exposed to a single gape size–limited predator. Life-history bistabilities are then expected to straddle a threshold prey size above which the predator cannot effectively consume prey anymore. Ecological communities with only a single major predator are rarely found in nature; instead, there typically are several major predators, each occupying a different niche and capable of using a different range of prey sizes. With each additional sharp drop or rise of a size-dependent mortality function, an additional alternative fitness optimum can emerge. In the presence of frequency-dependent selection, each such fitness optimum may then correspond to one coexisting body size morph (Taborsky et al. 2012). Our findings thus highlight a simple and generic evolutionary mechanism for how life-history diversity may arise from a set of distinct mortality sources, such as differently sized predators.

Previous models have shown that within populations, each independent selective force (such as a major predator or an important food source) will create an additional ecological niche, allowing for the ecological coexistence of alternative life-history strategies (Heino et al. 1997). Empirical examples show that the presence of different independent ecological dimensions in an environment indeed leads to the emergence of different body size morphs (Wellborn 1994; Claessen et al. 2000), a process which under certain conditions can even lead to sympatric speciation (Parker et al. 2001). Thus, one may expect that the number of coexisting alternative life-history strategies increases with the number of independent ecological dimensions (e.g., different predators). Here we have shown that a densely packed predator community, in which the total resultant prey mortality follows a power law with negative exponent, a mortality regime characteristic of many marine systems (Peterson and Wroblewski 1984; Andersen et al. 2009; Jørgensen and Holt 2013) does not favor the emergence of alternative fitness optima in body size. Counterintuitively, only by removing predator species from such a community—thereby creating irregularities in the resultant size-dependent prey mortality—can the diversity of alternative optimal prey body sizes increase. Thus, for predators with fixed prey preference functions, the removal or extinction of single predators from a rich predator community can open up size refuges for prey, which eventually might contribute to prey diversity through ecological speciation. More generally, our results highlight that multistable life histories can evolve whenever there are small irregularities in mortality landscapes. In addition to predator diversity, such irregularities might readily arise from stochasticity in community composition, differences in relative predator gape size, or other environmental effects impacting mortality regimes.

Here we have shown that life-history multistability caused by size-dependent mortality is a much more general phenomenon than previous work has suggested. Our results enable us to offer three important generalizations beyond previous modeling results, which had demonstrated bistability under negatively size-dependent mortality: (1) alternative fitness optima in body size can occur when mortality decreases or increases with size, (2) mortality functions with arbitrary shapes can lead to life-history bistability, and (3) the conditions for such bistability qualitatively extend to more than two fitness optima, thus allowing for higher degrees of multistability. Arguably, predators are the most important source of size-dependent mortality, and multiple size refuges can result from the presence of multiple predators in a community. Paradoxically, when there are very many predators, reducing the number of predator species can generate additional size refuges: prey diversification through size-dependent mortality is expected to occur only in rather species-poor predator communities. We suggest that the diversity-generating properties of size-dependent mortality, with their partly unexpected consequences for community composition, should be included in future models aiming to explain the evolution and functioning of size-structured communities.

Acknowledgments

B.T. acknowledges financial support by the Austrian Science Fund (project 18647-B16) and by the Swiss National Science Foundation (project 31003A_156881). M.H. acknowledges financial support by the Norwegian Research Council (214189). U.D. acknowledges financial support by the European Commission, the European Science Foundation, the Austrian Science Fund, the Austrian Ministry of Science and Research, and the Vienna Science and Technology Fund.

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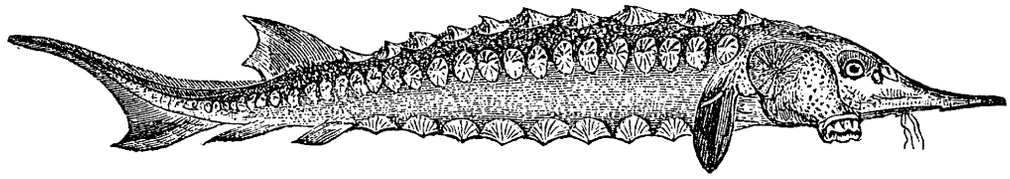
Associate Editor: Franz J. Weissing
Editor: Judith L. Bronstein



"The sturgeons [figured] are genuine scavengers. With their long noses they turn up the bottoms of the streams and feed upon such organic materials as they chance to find, using perhaps the flexible feelers beneath the snout to search out the exact location and nature of the food." From "A Few Words About Scavengers" by Sanborn Tenney (*The American Naturalist*, 1877, 11:129–135).