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Chaparro-Pedraza, P.C.; de Roos, A.M.

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Density-dependent effects of mortality on the optimal body size to shift habitat: Why smaller is better despite increased mortality risk

P. Catalina Chaparro-Pedraza[1,2,3] and **André M. de Roos**[1,4]

[1] *Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, Amsterdam 1090 GE, The Netherlands*

[2] *Eawag—Swiss Federal Institute of Aquatic Science and Technology, Dübendorf, Switzerland*

[3] *E-mail: Catalina.Chaparro@eawag.ch*

[4] *The Santa Fe Institute, Santa Fe, NM, USA*

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Many animal species across different taxa change their habitat during their development. An ontogenetic habitat shift enables the development of early vulnerable-to-predation stages in a safe "nursery" habitat with reduced predation mortality, whereas less vulnerable stages can exploit a more risky, rich feeding habitat. Therefore, the timing of the habitat shift is crucial for individual fitness. We investigate the effect that size selectivity in mortality in the rich feeding habitat has on the optimal body size at which to shift between habitats using a population model that incorporates density dependence. We show that when mortality risk is more size dependent, it is optimal to switch to the risky habitat at a smaller rather than larger body size, despite that individuals can avoid mortality by staying longer in the nursery habitat and growing to safety in size. When size selectivity in mortality is high, large reproducing individuals are abundant and produce numerous offspring that strongly compete in the nursery habitat. A smaller body size at habitat shift is therefore favored because strong competition reduces growth potential. Our results reveal the interdependence among population structure, density dependence, and life history traits, and highlight the need for integrating ecological feedbacks in the study of life history evolution.

KEY WORDS: **Habitat shift, optimal life history trait, size-dependent mortality, size-selective mortality.**

Interactions between organisms do not remain constant throughout their lives. Instead, the outcome of encounters between competitors, between prey and predator, or between parasite and host depends on the developmental stage of the interacting organisms. An increase in body size is the most important ecological aspect of ontogenetic development as it determines to a large extent those interactions as well as individual feeding, growth, and reproduction (de Roos and Persson 2013). Ecological interactions, therefore, change with the increase in size during ontogeny. In particular, smaller or younger individuals of diverse fish (Sogard 1997; Krause et al. 1998; Hampton 2000), amphibian (Semlitsch 1990; Rudolf 2008; Arendt 2009), reptile (Ferguson and Fox 1984; Keren-Rotem et al. 2006), and invertebrate species (Keller and Ribi 1993; Boulton and Polis 1999; Rudolf and Armstrong 2008) experience higher predation or cannibalistic risk than larger ones. To reduce the risk of injury or lethal interactions, small individuals often avoid areas with predators or larger conspecifics (Ohgushi et al. 2012) by using the same habitat differentially (Diehl and Eklov 1995) or using two different habitats for small and large individuals (Dodson et al. 2009).

Among vertebrate animal species, ontogenetic habitat shifts, understood as the use of different habitats in different stages of the life history, have been documented in a wide range of fish and amphibian species, as well as in some reptiles (Werner and Gilliam 1984; Keren-Rotem et al. 2006). By switching habitat, small individuals can develop in a safe "nursery" habitat with reduced predation mortality, whereas large individuals exploit a riskier, but richer feeding habitat. For instance,

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salmon and other anadromous species use freshwater streams as breeding habitats that offer a reduced predation mortality for embryos and larval stages compared to their marine counterparts, whereas larger, less vulnerable-to-predation individuals exploit the productive marine grounds at high latitudes (Dodson et al. 2009). Likewise, in several fish species associated with coral reefs, only large individuals are actually present in this highly productive habitat, and small vulnerable-to-predation stages occur in habitats with lower predation mortality such as mangroves and seagrass beds (Cocheret De La Morinière et al. 2002; Kimirei et al. 2013).

The physical separation between different ontogenetic habitats implies multiple changes in ecological conditions experienced by individuals during the habitat shift: the individuals do not only experience a change in predation vulnerability during the habitat shift but also a diet shift (Hobson 1999), as well as changes in intraspecific competition and food abundance (Diehl and Eklov 1995; Keren-Rotem et al. 2006). Generally, the "nursery" habitat is of relatively small size and low productivity compared to the habitat occupied by older individuals. As a consequence, individuals in the former experience increased density, whereas density dependence is very low to negligible in the latter (Diehl and Eklov 1995; Jonsson et al. 1998; Cocheret De La Morinière et al. 2002). This relaxation of the intraspecific competition in the habitat occupied by older individuals leads to an increase in food abundance after the habitat shift that, in turn, increases the available energy to allocate to both somatic growth and reproduction. An early habitat shift thus enables an early onset of rapid growth and reproduction. However, small individuals are more vulnerable to predation on arrival in the second habitat. A late habitat shift would allow them to reach a larger body size before entering the riskier habitat and therefore lowers predation mortality at the expense of an extended period of slow growth in the first habitat. Under such a trade-off, the timing to shift habitat is a crucial determinant of individual fitness. Werner and Gilliam (1984) concluded that when the two habitats differ in size-specific growth and mortality rates (indicated with g and μ, respectively), fitness is maximized when the switching size minimizes the ratio of mortality to growth rate (also referred to as the "μ/g rule"). However, this conclusion is based on an individual optimization in an invariant environment. It therefore ignores density-dependent processes at the population level caused by the interactions among individuals, such as the difference in intraspecific competition in the two habitats mentioned above. Furthermore, the optimal size to shift habitats determines the outflow and inflow of individuals in the two habitats through growth and reproduction and thus the densities of individuals in each habitat. These changes in population density affect intraspecific competition that, in turn, affects individual growth rate and therefore the optimal strategy to shift habitats. A few studies have investigated the optimal timing of a habitat or niche shift incorporating intraspecific competition (Claessen and Dieckmann 2002), but the role of mortality in the rich feeding habitat and its link with body size has not been explored yet.

Although size-dependent mortality due to predation is usually the main mortality source in the rich feeding habitat, mortality factors that cause random mortality across all size classes (i.e., uniform mortality), such as oxygen depletion and temperature extremes, can sometimes override size-dependent mortality (Sogard 1997). In this study, we investigate how size selectivity in mortality in the rich feeding habitat affects the optimal timing of a habitat shift. To do so, we use a size-structured population model for a consumer-resource interaction that incorporates food-dependent individual growth for the consumers. We analyze the ecological dynamics predicted by the model and use an optimization approach to determine the evolutionary endpoints corresponding to the optimal body size of the habitat shift.

Methods

THE MODEL

We formulate a model that accounts for a population in two habitats. We assume that in each habitat, individuals exploit a different resource. The population is structured by individual body size (body length l). Individual resource consumption, somatic growth, survival, and reproduction follow continuous-time dynamics. We study the population in the ecological equilibrium state.

Individuals are born in the "nursery" habitat (hereafter habitat 1) with size l_0 where they remain until they reach a body size l_s when they shift to the rich feeding habitat (occupied by older individuals, hereafter habitat 2). Juvenile individuals mature and start to reproduce at a body size l_m. Density dependence due to competition for food is considered to be strong in habitat 1, directly influencing growth in body size such as in salmonids (Walters et al. 2013), so we assume the food or resource density in this habitat to be depleted by the foraging of consumer individuals. In the absence of consumers, the resource is assumed to follow semichemostat growth dynamics with maximum density $X_{1\,\max}$ and renewal rate ρ (for an explanation and justification of this type of growth dynamics, see Persson et al. [1998]). Dynamics of the resource density X_1 in habitat 1 in the absence of consumers is hence given by

$$\frac{dX_1}{dt} = \rho\,(X_{1\,\max} - X_1)\,. \tag{1}$$

In contrast, in habitat 2, density dependence is considered negligible, therefore we assume a constant resource density.

The core part of the model describes individual feeding, growth, reproduction, and mortality as a function of the

individual state (i.e., body length l) and the state of the environment (food availability) using the dynamic energy budget approach introduced by Kooijman and colleagues (Kooijman and Metz 1984; Nisbet et al. 2000; Kooijman 2010). More specifically, we adopt the model described in detail by Jager et al. (2013). A derivation of this dynamic energy budget model can be found in the Supporting Information; below we only present the resulting equations for individual feeding, growth, mortality, and reproduction.

In habitat 1, individuals are assumed to feed on the resource following a Holling type II functional response. So, their feeding level f_1 (or scaled functional response), which is the amount of food that is ingested by an individual as a fraction of what it can maximally eat, is described by the following expression:

$$f_1 = \frac{X_1}{K + X_1}\,, \tag{2}$$

where K is the half-saturation resource density. In habitat 2, individuals feed at a constant feeding level f_2. Following Jager et al. (2013), we assume that the individuals in habitat 1 deplete the resource at a rate proportional to their squared body size l and the feeding level f_1. Food intake by individuals in habitat 1 is therefore described by

$$\alpha\,(f_1,\ l) = \begin{cases} I_{\max}\ f_1\ l^2 & \text{if } l < l_s \\ 0 & \text{otherwise} \end{cases}. \tag{3}$$

Here, $I_{\max}$ is a proportionality constant relating maximum ingestion rate to squared body size.

The growth rate in body size $\gamma(f_1, f_2, l)$ is described by the following equation (derivation is presented in the Supporting Information, section Dynamic energy budget model).

$$\gamma\,(f_1, f_2, l) = \begin{cases} \xi\,(l_{\inf}\ f_1\ -\ l) & \text{if } l < l_s \\ \xi\,(l_{\inf}\ f_2 - l) & \text{otherwise} \end{cases}. \tag{4}$$

This equation implies that under constant food conditions, an individual grows in size following a von Bertalanffy growth curve (Kooijman and Metz 1984; Jager et al. 2013) and can reach a maximum size $l_{\inf} f_1$ and $l_{\inf} f_2$ in habitat 1 and 2, respectively, whereas ξ characterizes its growth rate. Because we study the research question only under ecological equilibrium conditions, the inequalities $l_{\inf} f_1 > l$ and $l_{\inf} f_2 > l$ are always fulfilled and hence individuals do not starve and do not shrink in size.

Reproduction is assumed continuous. Adult fecundity is described by

$$\beta(f_1, f_2, l) = \begin{cases} B_{\max}\ f_1\ l^2 & \text{if } l > l_{\mathrm{m}} \text{ and } l < l_{\mathrm{s}} \\ B_{\max}\ f_2\ l^2 & \text{if } l > l_{\mathrm{m}} \text{ and } l \geq l_{\mathrm{s}}, \\ 0 & \text{otherwise} \end{cases} \tag{5}$$

where $B_{\max}$ is a proportionality constant relating maximum fecundity to squared body size.

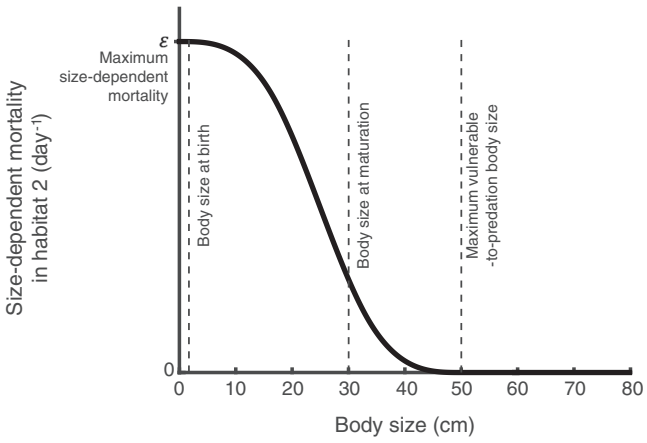

Figure 1. Size-dependent mortality in habitat 2.

Individuals in habitat 1 may die from background mortality μ_1 and in habitat 2 from either background $\mu_{2\,\mathrm{b}}$ or predation mortality $\mu_{2\,\mathrm{p}}$. Background mortality is assumed to be size independent and predation mortality is assumed size dependent. To describe the size-dependent mortality experienced by individuals in habitat 2, we adopt a continuous piecewise-differentiable sigmoid function of body size (Fig. 1):

$$\mu_{2\mathrm{p}}(l) = \begin{cases} \varepsilon\left(-\frac{1}{6}L(l)^3 + 1\right) & \text{if } L\,(l) \leq 1 \\ \varepsilon\left(\frac{1}{3}L(l)^3 - \frac{3}{2}L(l)^2 + \frac{3}{2}L\,(l) + \frac{1}{2}\right) & \text{if } 1 < L(l) \leq 2 \\ \varepsilon\left(-\frac{1}{6}L(l)^3 + \frac{3}{2}L(l)^2 - \frac{9}{2}L\,(l) + \frac{9}{2}\right) & \text{if } 2 < L\,(l) \leq 3 \\ 0 & \text{otherwise} \end{cases}, \tag{6}$$

where $L(l)$ is a scaled body size value, defined as $L\,(l) = 3\,l/l_{\mathrm{v}}$. The sigmoid function is bounded by the maximum size-dependent mortality ε, which occurs at $l = 0$, and the maximum vulnerable-to-predation body size l_{v} at which size-dependent mortality vanishes. This function has been chosen because the parameters ε and l_{v} facilitate biological interpretation. However, size-dependent mortality has been commonly described as an exponential function of body size (Fig. S1; Gislason et al. 2010; Jørgensen and Holt 2013):

$$\mu_{2\,\mathrm{p}}\ (l) = \ c\ l^{-d} \tag{7}$$

therefore, we test the robustness of our results under this assumption (see Supporting Information). The total per capita death rate in habitat 2 is the sum of the background and size-dependent mortality. Thus, the instantaneous mortality rate experienced by an individual is given by

$$\mu(l) = \begin{cases} \mu_1 & \text{if } l < l_{\mathrm{s}} \\ \mu_{2\,\mathrm{b}} + \mu_{2\,\mathrm{p}}\,(l) & \text{otherwise} \end{cases}. \tag{8}$$

Table 1. Model parameters with default values.

Description	Symbol	Value	Unit	References
Resource in the "nursery" habitat				
Resource growth rate	ρ	0.01	day^{-1}	
Maximum resource density	$X_{1\,max}$	4	$g\ m^{-3}$	
Population with habitat shift				
Half saturation resource density	K	1	$g\ m^{-3}$	
Maximum ingestion proportionality constant	I_{max}	0.0025	$g\ cm^{-2}\ day^{-1}$	See Supporting Information
Feeding level in the habitat 2	f_2	0.6	–	
Maximum fecundity proportionality constant	B_{max}	0.0618	$cm^{-2}\ day^{-1}$	See Supporting Information
Body size of a newborn	l_0	2	cm	Gilbey et al. 2009
Body size at the habitat shift	l_s	Varied (evolving)	cm	
Body size at maturation	l_m	30	cm	
Maximum body size at maximum feeding rate	l_{inf}	115	cm	See Supporting Information
Von Bertalanffy growth rate parameter	ξ	0.00051	day^{-1}	See Supporting Information
Maximum vulnerable-to-predation body size*	l_v	50	cm	
Maximum size-dependent mortality*	ε	Varied	day^{-1}	
Scaling coefficient of size-dependent mortality**	c	Varied	day^{-1}	
Exponent of size-dependent mortality**	d	0.75	–	Jørgensen and Holt 2013
Mortality rate in the habitat 1	μ_1	0.002	day^{-1}	Bley and Moring 1988
Size-independent mortality rate in the habitat 2	$\mu_{2\,b}$	Varied	day^{-1}	

*Parameters used only when size-dependent mortality is a sigmoid function of the body size (eq. 7).

**Parameters used only when size-dependent mortality is an exponential function of the body size (eq. 8).

EVOLUTIONARY DYNAMICS

In this study, we are interested in understanding how size selectivity in mortality in the rich feeding habitat 2 affects the optimal timing of a habitat shift. We study the evolution of the body size at the habitat shift l_s using an optimization approach. Because the only impact of the population on its environment is on food resource density in habitat 1 (X_1), the optimal body size at the habitat shift l_s^* is that one that minimizes the resource density evaluated at the equilibrium $\tilde{X}_1$ (Mylius and Diekmann 1995). In the Supporting Information, formal expressions are provided for the individual lifetime reproductive output R_0 as a function of the food resource density in habitat 1 (X_1). This food density is depleted by the food intake of all individuals in habitat 1, which ultimately determines the birth rate of the size-structured population in an ecological equilibrium (see the Supporting Information for the formal expressions). These expressions make clear that for the size-structured population model, the ecological equilibrium cannot be solved analytically. The lifetime reproductive output R_0, the ecological equilibrium values, and the minimum of $\tilde{X}_1$ can therefore only be calculated numerically. We used standard MATLAB functions to perform these calculations (the code is available in the Supporting Information).

MODEL PARAMETERIZATION

Parameters of the population with a habitat shift are shown in Table 1. Default parameter values are loosely based on the biology of Atlantic salmon (*Salmo salar*). Values for the parameters in the functions describing individual feeding, growth, and reproduction are derived from the underlying parameters of the dynamic energy budget model (see Supporting Information and Table S1). Values for the parameters representing life history traits, such as body size at birth, were derived from reported data in the literature (Table 1). Atlantic salmon are considered mature when they return to the streams to spawn (around 50 cm; Hutchings and Jones 1998); however, at this point, individuals had already accumulated large amounts of energy for reproduction. It is unknown, however, when they start to allocate this energy to reproduction. Because we assume reproduction to be a continuous process in the model (i.e., energy allocated to reproduction is immediately converted into offspring), we chose a threshold for maturation

lower than the body size at which Atlantic salmon has been documented to return (30 cm).

MODEL ANALYSIS

We are interested in understanding how size selectivity in mortality in the rich feeding habitat (habitat 2) affects the optimal timing of a habitat shift. However, a simple increase in size-dependent mortality does not only increase the size-selective nature of mortality but also the total mortality experienced in this habitat. Hence, the effect of the size selectivity of mortality per se can only be unraveled while maintaining total mortality constant in habitat 2. To evaluate evolutionary responses, we therefore follow a specific approach, in which the contribution from size-dependent mortality sources is increased but overall mortality in habitat 2 is kept constant through a simultaneous decrease in size-independent mortality. More specifically, we find the optimal body size to shift habitat when there is only size-independent mortality $\mu_{2\,b}$ equal to 0.006/day in habitat 2 and adopt this as our starting, reference population (body size at habitat shift = 19.5 cm). A size-independent mortality $\mu_{2\,b}$ equal to 0.006/day implies that an individual has an expected lifetime of 167 days in habitat 2 from the moment it shifts habitats. Adopting this body size at habitat shift in case it only experiences size-independent mortality $\mu_{2\,b}$of 0.006/day, we infer the combinations of size-independent mortality $\mu_{2\,b}$ and maximum size-dependent mortality ε that also result in a life expectancy of 167 days in habitat 2 (Fig. 2). To determine these combinations, we numerically compute an individual's life expectancy after entering habitat 2 by integrating the following differential equation for its survival $S(\tau)$ as a function of the time τ it has spent in habitat 2:

$$\frac{dS(\tau)}{d\tau} = -\left(\mu_{2\,b} + \mu_{2\,p}(l(\tau))\right) S(\tau), \tag{9}$$

while simultaneously integrating the differential equation $dl/d\tau = \gamma(f_1, f_2, l)$ for the growth in body size l (see eq. 4).

We subsequently determine the optimal body size at shift habitat for each combination of mortality values inferred in the previous step by calculating the body size at the habitat shift that minimizes the resource density evaluated at the equilibrium (Fig. 3). The standard techniques available in Matlab that we use for minimization yield slightly irregular results because of the flatness of the curve around the minimum (Fig. S2). As an alternative method to calculate the evolutionary endpoint for this life history trait and to verify our results, we also use the R package *PSPManalysis* (version 0.2.2; de Roos 2019). This package implements optimized and tailor-made techniques for the analysis of structured population models, including methods to calculate evolutionary endpoints on the basis of the adaptive dynamics approach, which yields the same qualitative results. We use the

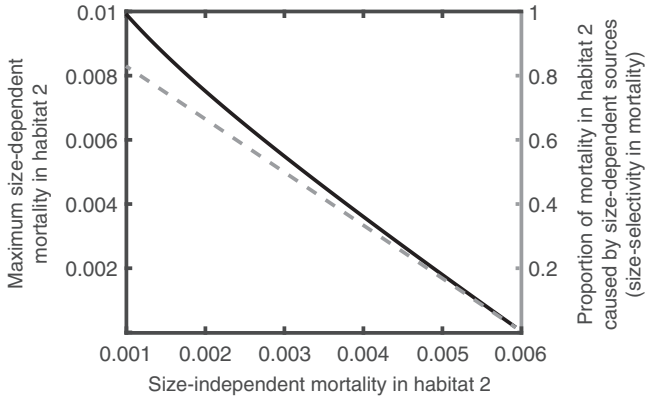

Figure 2. Combinations of maximum size-dependent and size-independent mortality in habitat 2 (black solid line, left axis) that result in the same overall mortality (i.e., equal life expectancy) in this habitat for an individual shifting habitat at 19.5 cm (life expectancy in this habitat is 166.67 days for any combination). This body size at habitat shift is the evolutionary end point when maximum size-dependent mortality and size-independent mortality in habitat 2 equal 0 and 0.006 day-1, respectively (right bottom corner). Each combination of maximum size-dependent and size-independent mortality in habitat 2 corresponds to a certain level of size selectivity (i.e., proportion of mortality caused by size-dependent sources; gray dashed line, right axis).

PSPManalysis package to detect and continue Continuously Stable Strategies (CSSs; according to classification by Eshel 1983, revisited in Geritz et al. 1998) as a function of ε, the scaling factor in the size-dependent mortality function $\mu_{2\,p}$, and $\mu_{2\,b}$, the size-independent mortality parameter in habitat 2. Because the package implements dedicated numerical methods for continuation of CSSs, the resulting curves are smoother as shown in Figure S3.

We additionally test the robustness of the results under the assumption that size-dependent mortality is an exponential function of the body size. A detailed description of these robustness tests can be found in the Supporting Information.

We furthermore evaluate the individual fitness components before and after changes in size selectivity in mortality in habitat 2. This analysis enables us to determine how various fitness components are maximized by selection under the imposed change in size selectivity in mortality (Fig. 4). Likewise, we assess the effect of the variation in size selectivity in mortality on the population size distribution (Fig. 5). According to Diekmann et al. (2003), the shape of the population size distribution is determined by the curves describing the individual survival as a function of age and the body size as a function of age, and its absolute magnitude is determined by the population birth rate (the derivation of this quantity can be found in the Supporting Information, eq. S3.16).

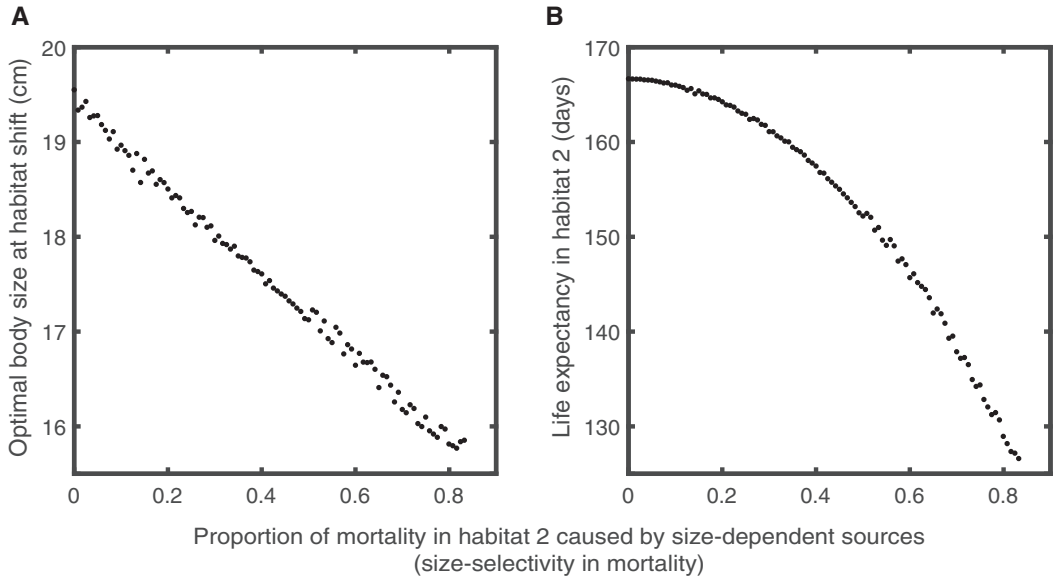

Figure 3. **(A) Evolutionary end points and (B) resulting life expectancy in habitat 2 as a function of the proportion of mortality in habitat 2 caused by size-dependent sources. Size selectivity in mortality is varied (higher toward the right of the horizontal axes) following the combination of size-independent and maximum size-dependent mortalities shown in Figure 2. Other parameter values as in Table 1.**

Results

In the first part of this section, we show the evolutionary effects of size -selectivity in mortality in the rich feeding habitat (habitat 2) on the optimal timing of the habitat shift. Subsequently, we present the cause of these evolutionary responses. For the parameter values explored, there exist a unique local minimum of $\tilde{X}_1$ as function of the body size at habitat shift l_s, therefore there exists only one optimal body size at habitat shift for each size-selectivity in mortality in the habitat 2.

INCREASED SIZE SELECTIVITY IN MORTALITY IN HABITAT 2 DECREASES THE OPTIMAL BODY SIZE AT HABITAT SHIFT

When size selectivity in mortality increases in habitat 2 (i.e., the contribution of size-dependent mortality sources to total mortality increases, given a constant life expectancy in habitat 2), the optimal body size at habitat shift decreases, so individuals evolve to shift habitat at smaller sizes than individuals exposed only to size-independent mortality (Fig. 3A; the irregularities in the results are due to the flatness of the curve relating resource density at the equilibrium $\tilde{X}_1$ to l_s near the minimum, see Figs. S2 and S3). As a consequence of the evolution toward a smaller size at habitat shift, life expectancy in habitat 2 decreases as well (Fig. 3B).

The juveniles switch to the risky habitat 2 at a smaller body size when the risk is more size dependent, despite that mortality would be avoided by staying longer and growing in the nursery habitat to a safer body size. This result is robust to assumptions regarding evolutionary processes (e.g., assumptions of adaptive dynamics framework; Fig. S3) and the size-dependent mortality function (Fig. S4). In the following subsections, we explain the cause of this apparent evolutionary paradox.

INCREASED SIZE SELECTIVITY IN MORTALITY IN HABITAT 2 DECREASES GROWTH POTENTIAL IN HABITAT 1

Considering an individual that initially has an optimal body size at habitat shift, the analysis of its fitness components before and after an increase in size selectivity in mortality in habitat 2 reveals that this increase results in a slower growth rate and thus longer stay in habitat 1, later maturation, and consequently higher survival (compare black solid and dashed lines in Fig. 4A). Following the increase in size selectivity in mortality in habitat 2, a phenotype with a smaller body size at habitat shift is selected for as it maximizes growth rate, leading to earlier maturation and increased fecundity at the expense of lower survival (compare black and gray dashed lines in Fig. 4A).

In contrast, the analysis of the fitness components before and after a decrease in size selectivity in mortality in habitat 2 shows that the decrease leads to shorter stay in habitat 1 as a consequence of the increased growth rate in this habitat, earlier maturation, and thus lower survival (compare black solid and dashed lines in Fig. 4B). After the decrease in size selectivity in mortality, a larger body size at the habitat shift is selected for as it maximizes survival at the expense of slower growth and thus, later maturation (compared black and gray dashed lines in Fig. 4B).

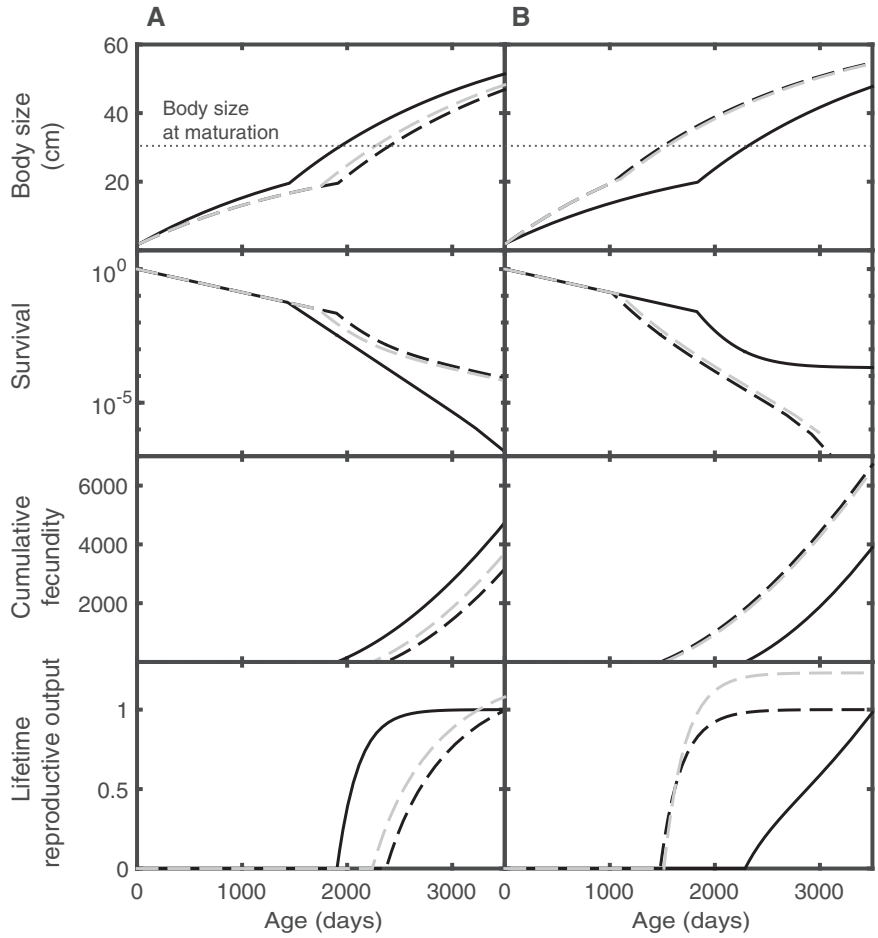

Figure 4. **Individual growth, survival, cumulative fecundity, and expected lifetime reproductive output before (solid lines) and after (black dashed lines) an increase (left column) and a decrease (right column) of size selectivity in mortality in habitat 2. (A) When size selectivity in mortality increases (maximum size-dependent mortality and size-independent mortality in habitat 2 change from 0 and 0.006 to 0.0075 and 0.002 day-1, respectively), an individual that shifts habitat at 19.5 cm does not experience a change in the life expectancy in habitat 2, but its growth rate in habitat 1 decreases (compare solid and dashed black lines). As a consequence, an individual shifting habitat at a smaller body size (gray dashed line, body size at habitat shift is 5% smaller) has a higher fitness than an individual shifting at 19.5 cm. (B) In contrast, when size selectivity in mortality decreases (maximum size-dependent mortality and size-independent mortality in habitat 2 change from 0.015 and 0.0001 to 0.005 and 0.0054 day-1, respectively), an individual that shifts habitat at 19.8 cm does not experience a change in the life expectancy in habitat 2, but it experiences a higher individual growth rate in habitat 1 (compare solid and dashed black lines). Hence, an individual shifting habitat at a larger body size (gray line, body size at habitat shift is 5% larger) has a higher fitness than an individual shifting at 19.8 cm. Other parameter values as in Table 1.**

Figure 4 shows that an increase or decrease in size selectivity in mortality in habitat 2 produces changes in the individual fitness components that are subsequently countered by selection. Although we expected a direct effect of size selectivity in mortality on survival, its effect on growth rate in the nursery habitat needs further explanation.

BY SHAPING POPULATION STRUCTURE, SIZE SELECTIVITY IN MORTALITY INFLUENCES GROWTH POTENTIAL IN HABITAT 1

Because small individuals experience higher mortality rates than large individuals in habitat 2, adult density increases and juvenile density decreases in this habitat (Fig. 5 left panel) when the size selectivity in mortality is increased. This larger density of adults produces more offspring, which raises the density of newborns in habitat 1. As a consequence of the increased density of newborns in habitat 1, competition for food resources is stronger and thus growth rate is slower in this habitat. Given the adverse effects of density on growth potential, by advancing their shift to habitat 2 individuals can escape at an earlier age the reduced body growth they experience in habitat 1.

In contrast, a decreased size selectivity in mortality in habitat 2 causes an increase in juvenile density and a decrease in adult density in this habitat (Fig. 4 right panel). With a

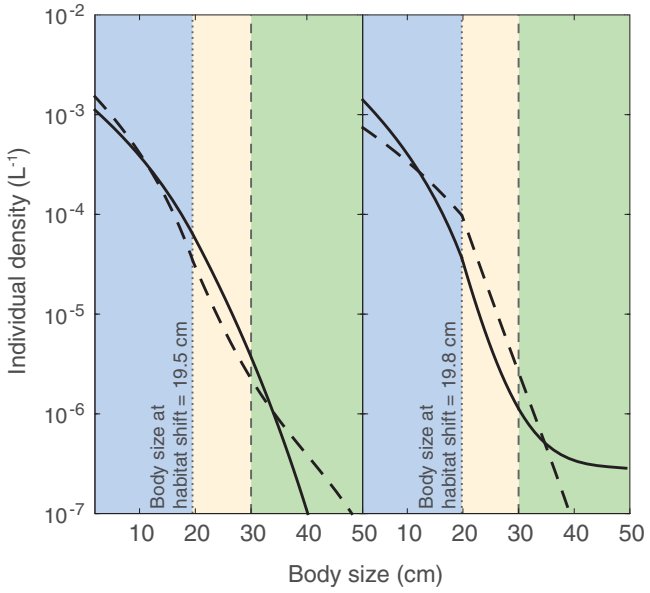

Figure 5. Size distribution of the population just before (solid lines) and immediately after (dashed lines) size selectivity in mortality increases (left panel) and decreases (right panel) in habitat 2. When size selectivity in mortality increases, maximum size-dependent mortality and size-independent mortality in habitat 2 change from 0 and 0.006 to 0.0075 and 0.002 day-1, respectively, whereas when size selectivity in mortality decreases, maximum size-dependent mortality and size-independent mortality in habitat 2 change from 0.015 and 0.0001 to 0.005 and 0.0054 day-1, respectively. Juveniles in habitat 1 (blue region) and in habitat 2 (yellow region), and adults (green region) are delimited by body size at habitat shift (gray vertical dotted line) and at maturation (gray vertical dashed line). Other parameter values as in Table 1.

reduction in adult density, the population birth rate decreases and thus the density of newborns in habitat 1. Therefore, competition is relaxed and growth rate in this habitat increases. With a high growth potential in habitat 1, a later habitat shift enables individuals to increase their survival by postponing the shift to the riskier habitat 2.

In summary, we expected juveniles to switch to the risky habitat 2 at a larger body size when the risk is more size dependent, because the mortality risk would be avoided by growing in the nursery habitat to safety in size. However, the opposite—a habitat shift at a smaller body size–was observed because the potential for growth in body size in the nursery habitat is low when the risk is more size dependent in the risky habitat. Interestingly, this evolutionary response of reducing the body size at habitat shift when size selectivity in the risky habitat increases is strong when total mortality in this habitat is low (or life expectancy is high) and becomes less strong as mortality increases (life expectancy decreases) (Fig. S5). This is the consequence of stronger density dependence effects under low mortality than under high mortality conditions.

Discussion

We have found an unexpected evolutionary response of the timing of a habitat shift to changes in size selectivity in mortality when accounting for feedbacks between the population and its environment, which is represented in this study by the food density in the "nursery" habitat. Our naïve expectation, neglecting the feedback of the population on its environment, was that when size selectivity in mortality in the rich feeding habitat increases, the body size at habitat shift would increase because delaying the habitat shift would cause individuals to benefit from increased survival in a larger part of their life cycle than when mortality is random across all size classes. This expectation holds true if density dependence is neglected (Fig. 6). In contrast, when accounting for density dependence due to competition for food in the "nursery" habitat, the structured population model shows that the body size at habitat shift decreases with an increasing size selectivity in mortality in the rich feeding habitat. This is the consequence of the effect that size-dependent and size-independent mortality in the risky habitat have on the population structure. Specifically, by changing the population structure, higher size selectivity in mortality increases the density of newborns in the "nursery" habitat resulting in increased competition and, thus, triggering an earlier habitat shift.

We have shown that mortality in the rich feeding habitat affects the optimal timing of the habitat shift not only because of its direct effect on survival but also through indirect effects on other fitness components such as growth mediated by density dependence. Werner and Gilliam (1984) have hypothesized that the optimal timing of a habitat shift is determined by both the mortality and growth rate in the two habitats, and that the mortality rate is largely dependent on growth. Yet, the opposite effect that mortality influences growth by regulating the strength of density dependence is a recent concept that emerged from structured population theory: by relaxing competition, mortality affects and in particular promotes food-dependent processes such as growth and reproduction (de Roos et al. 2007). In line with those findings, we show that in populations with a habitat shift, the nature of mortality has effects on body growth.

Multiple studies have reported density, food availability, and growth rate to influence habitat shifts in both experimentally manipulated as well as wild populations. For instance, experimental manipulations have shown that Brown trout is more likely to migrate (shift habitat) when growing slowly at high density but less likely to do so when density is low and growth rate is high (Olsson et al. 2006). This effect was proven to be mediated by food availability (Wysujack et al. 2009). Similarly, Arctic char

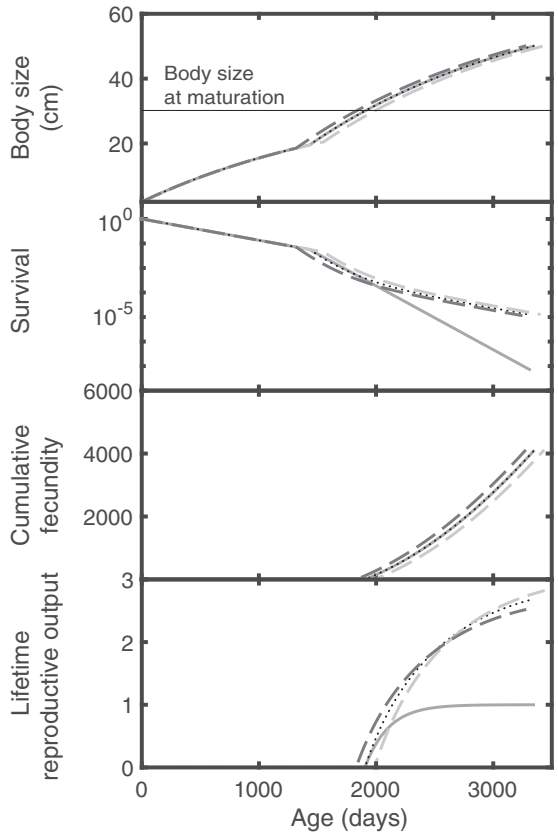

Figure 6. Individual growth, survival, cumulative fecundity, and expected lifetime cumulative fecundity before (solid line) and after (dashed and dotted lines) size selectivity in mortality increases in habitat 2 when density dependence in habitat 1 is neglected (in all cases individuals grow at the same rate: X1 is constant and equal to 0.46 g/L). During the increase in size selectivity in mortality, size-dependent and size-independent mortality in habitat 2 change from 0 and 0.006 to 0.0075 and 0.002 day-1, respectively. Initially, an individual shifts habitat at 19.5 cm (solid and dotted lines), and novel phenotypes shift habitat at a body size 5% larger (light gray dashed lines) and at a body size 5% smaller than the initial phenotype (dark gray dashed lines). A larger fitness is achieved by a novel phenotype that shifts habitat at a larger body size after the increase in size selectivity in mortality. Other parameter values as in Table 1.

(Nordeng 1983) and Atlantic salmon (Lans et al. 2011) are more likely to migrate at low food availability causing slow growth rate. Low food availability causing slow growth rate also results in smaller sizes at metamorphosis than that of fast growers in amphibians (Alford and Harris 1988; Beachy et al. 1999). A long-term study of wild Atlantic salmon populations in the Simojoki river showed that the mean body size at smolting (habitat shift) was negatively correlated with density in the previous autumn (Jutila et al. 2006). High population densities hence depress food levels and thereby growth rates, which triggers an early habitat shift in different species with ontogenetic habitat shift. Although the effect of growth rates on the optimal timing of the habitat shift has been previously reported, its connection with density-dependent processes resulting from feedbacks between the population and the individual life history has been addressed only recently. In this study, we focused on the link between population structure and the optimal timing of the habitat shift through density dependence. Given the multiple and dramatic consequences that population structure and habitat shifts have independently on communities and ecosystems (de Roos and Persson 2002; Schreiber and Rudolf 2008), the implications of interactions between them need to be studied in future research.

Although we focused on a negative relation between mortality risk and body size in the rich feeding habitat, a positive relation is also common. For instance, because of fishing mortality, exploited fish populations may experience strong positive size-selective mortalities. Such size-dependent fishing mortality targeting mainly large individuals would reduce the adult biomass and thus cancel out the effects of size-dependent predation mortality on population structure that we revealed. In populations exposed to strong size-dependent predation, size-dependent fishing mortality would thus reduce density and relax competition in the "nursery" habitat, which in turn promotes a habitat shift at larger body sizes. Indeed, Atlantic salmon in the Baltic sea has experienced a drop in the fishing effort in the last decades with concurrent higher density of individuals in the "nursery" Simojoki river resulting in smaller mean sizes at the habitat shift (Jutila et al. 2006). In turn, these smaller sizes reduce survival of Simojoki river Atlantic salmon because mortality right after the habitat shift is correlated with body size at the shift. This suggests that some size-dependent fishing mortality may actually increase survival after habitat shift and perhaps enhance the fishing yield.

In this study, we have focused on the evolution of the timing of a habitat shift, but individuals may likely also evolve to mature at smaller or larger size depending on growth opportunities and mortality risk. Fish often attain sexual maturation when growth rates reduce (near the asymptotic body size) (Jonsson and Jonsson 1993). Because the habitat shift enables individuals to access a rich feeding habitat and thus rapid growth, the timing of the habitat shift may influence the optimal timing of maturation as well. Brown trout, for example, delays maturity for one or more years when moving from "nursery" streams to lakes for feeding compared to resident individuals in streams (Jonsson 1989). Given that both the timing of a habitat shift and the timing of sexual maturation influence population structure, future research focused on the joint evolution of these two traits could contribute to a deeper understanding of the linkage between life history trait evolution and density-dependent effects. Furthermore, we assumed the evolution of the body size at habitat shift to be genetically determined, but in the wild, organisms with habitat shift, such as salmonids and coral reef fish, often

show plasticity in life history traits. Previous studies have used a reaction-norm approach in which the probability to shift habitat is a function of body size and environmental conditions, for example, food availability, to asses the evolutionary outcome of different mortality sources in the absence of density-dependent effects (Thériault et al. 2008). The type of physiologically structured population model used in this study (de Roos 1997) could be used to extend such a reaction-norm approach to also account for density dependence.

Survival and growth rate have long been recognized as the traits to optimize when shifting habitats. In addition, a growing body of theoretical work and experimental evidence shows that survival and growth rate are interdependent and interact through feedbacks between the individual and its environment. Despite this, the analysis of the optimal timing of a habitat shift, as well as other life history traits, has been traditionally carried out neglecting these feedbacks. Our results demonstrate the strong influence that population structure, density dependency, and optimal timing of a habitat shift have on each other. This highlights the need for integrating ecological feedbacks in the study of life history evolution.

AUTHOR CONTRIBUTIONS

PCC-P and AMdR designed methodology. PCC-P conceived the ideas, analyzed the results, and led the writing of the chapter. AMdR contributed to later versions of the chapter.

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DATA ARCHIVING

No new data were collected and used for this study.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. A) Size-dependent mortality in habitat 2 following an exponential function (Size-dependent mortality = c * (Body size)-d; d = 0.75). B) Combinations of scaling coefficient of size-dependent (c) and size-independent mortality in habitat 2 (black solid line, left axis) that result in the same overall mortality (i.e. equal life expectancy) in this habitat for an individual shifting habitat at 19.5 cm (life expectancy in this habitat is 166.67 days for any combination).

Figure S2. Food resource density in habitat 1 in the equilibrium, as function of body size at habitat shift.

Figure S3. Evolutionary end-points (left) and resulting life expectancy in habitat 2 (right) as a function of the proportion of mortality in habitat 2 caused by size-dependent sources.

Figure S4. Evolutionary end-points (left) and resulting life expectancy in habitat 2 (right) as a function of the proportion of mortality in habitat 2 caused by size-dependent sources.

Figure S5. Optimal body size for individuals to shift habitat (color bar) as a function of the maximum size-dependent mortality (vertical axis) and the size-independent mortality (horizontal axis) in habitat 2.

Table S1. Dynamic energy budget parameter values