1 **Density-dependent effects of mortality on the optimal body size of a habitat shift:**

2 **Why smaller is better despite increased mortality risk?**

3 P. Catalina Chaparro-Pedraza, Institute for Biodiversity and Ecosystem Dynamics,
4 University of Amsterdam, 1090 GE Amsterdam, the Netherlands. Corresponding
5 author: P.C.ChaparroPedraza@uva.nl

6 André M. de Roos. Institute for Biodiversity and Ecosystem Dynamics, University of
7 Amsterdam, 1090 GE Amsterdam, the Netherlands.

8 **Abstract**

9 Many animal species across different taxa change their habitat during their
10 development. An ontogenetic habitat shift enables the development of early
11 vulnerable-to-predation stages in a safe ‘nursery’ habitat with reduced predation
12 mortality, while less vulnerable stages can exploit a more risky, rich feeding habitat.
13 Therefore, the timing of the habitat shift is crucial for individual fitness. We
14 investigate the effect that size-selectivity in mortality in the rich feeding habitat has
15 on the optimal timing of the habitat shift using a population model and the adaptive
16 dynamics approach. We show that the size-selective nature of mortality in this habitat
17 affects density-dependent body growth rate in the nursery habitat and thus the optimal
18 timing of the habitat shift. This is caused by the effect exerted by size-dependent
19 mortality on the size distribution of the population that results in strong competition in
20 the nursery habitat. We furthermore find that, as a consequence of this effect,
21 increased size-selectivity in mortality in the rich feeding habitat causes the optimal
22 body size to shift habitat to decrease. Our results reveal the interdependence between
23 population structure and life history traits, and highlight the need for integrating
24 ecological interactions in the study of the evolution of life histories.

25   **Keywords:** size-dependent mortality, size-selective mortality, optimal life history
26   trait, habitat shift.

27   **Introduction**

28   Interactions between organisms do not remain constant throughout their lives. Instead,
 29   the outcome of encounters between competitors, between prey and predator or
 30   between parasite and host depends on the developmental stage of the interacting
 31   organisms. An increase in body size is the most important ecological aspect of
 32   ontogenetic development as it determines to a large extent those interactions besides
 33   feeding, growth and reproduction (de Roos and Persson 2013). Ecological
 34   interactions, therefore, change with the increase in size during ontogeny. In particular,
 35   smaller or younger individuals of diverse fish (Sogard 1997; Krause et al. 1998;
 36   Hampton 2000), amphibian (Arendt, 2009; Rudolf, 2018; Semlitsch, 1990), reptile
 37   (Ferguson and Fox 1984; Keren-Rotem et al. 2006) and invertebrate species (Rudolf
 38   and Armstrong 2008; Boulton and Polis 1999; Keller and Ribi 1993) experience
 39   higher predation or cannibalistic risk than larger ones. To reduce the risk of injury or
 40   lethal interactions, small individuals often avoid areas with predators or larger
 41   conspecifics (Ohgushi et al. 2012) by using the same habitat differentially (Diehl and
 42   Eklov 1995) or using two different habitats for small and large individuals (Dodson et
 43   al. 2009).

44   Among vertebrate animal species, ontogenetic habitat shifts, understood as the use of
 45   different habitats in different stages of the life history, have been documented in a
 46   wide range of fish and amphibian species, as well as, in some reptiles (Werner and
 47   Gilliam 1984; Keren-Rotem et al. 2006). By switching habitat small individuals can
 48   develop in a safe ‘nursery’ habitat with reduced predation mortality, while large
 49   individuals exploit a riskier, but richer feeding habitat. For instance, salmon and other
 50   anadromous species utilize freshwater streams as breeding habitats that offer a

reduced predation mortality for embryos and larval stages compared to their marine counterparts, whilst large 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 while 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 switch 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 switch 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, physical 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 the adaptive-dynamics approach to

determine the optimal body size of the habitat shift in the context of these ecological dynamics.

Methods

The model

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

Life cycle

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 .

Habitats

Density-dependence due to competition for food is considered to be strong in habitat 1, directly influencing growth in body size like 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 a semi-chemostat growth dynamics with maximum density $R_{1\max}$ and growth rate ρ (for an explanation and justification of this type of growth dynamics, see Persson et al. (1998)). Dynamics of the resource density R_1 in habitat 1 in the absence of consumers is hence given by:

$$\frac{dR_1}{dt} = \rho(R_{1\max} - R_1)$$

(1)

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

Individual dynamics

The core part of the model is the description of the individual behavior, that is, feeding, growth, reproduction and mortality as a function of the individual state (i.e. body size) and the state of the environment (food availability). In the following sections we describe the individual level dynamics.

- *Feeding*

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) is given by:

$$f_1 = \frac{R_1}{K + R_1}$$

(2)

where K is the half-saturation resource density. In habitat 2 individuals feed at a constant feeding level f_2 .

- *Dynamic energy budget model: Individual state and fecundity*

The model follows the bioenergetics approach introduced by Kooijman and colleagues (Nisbet et al. 2000; Kooijman 2010; Kooijman and Metz 1984) in which the energy allocation to somatic and reproductive metabolism is proportional to a fraction κ and a $1-\kappa$ of the total energy assimilation rate, respectively. More specifically, we adopt the model developed and described in detail by Jager et al. (2013). Below we provide only a concise synopsis of the model.

Individuals are characterized by the state variable structural mass, W . Body length l and structural mass W are related to each other following Jager et al. (2013):

$$W = v (\delta l)^3$$

(3)

where v is the density of structural mass and δ is a shape coefficient factor. Hereafter we refer to l as body size.

The acquisition and utilization of energy are described by equations (4) to (10).

The energy assimilation rate is given by:

$$J_F = f j_a W^{2/3}$$

(4)

where f is the feeding level in either habitat, j_a is the maximum area-specific assimilation rate and the surface area for assimilation is assumed to scale with structural mass to the power of $2/3$.

156 Metabolic maintenance costs are the product of the mass-specific maintenance costs

157 j_m and the structural mass:

$$J_M = j_m W$$

158 (5)

159 Assimilates are assumed to split into two energy fluxes: the κ flux and the $1 - \kappa$ flux.

160 The κ flux is first used to cover metabolic maintenance costs, while the remaining

161 flux J_W is used to synthesize structural mass. On the other hand, the $1 - \kappa$ flux J_S is

162 allocated to reproduction when adult and to maturation when juvenile (Jager et al.

163 2013). Hence,

$$J_W = \kappa J_F - J_M$$

164 (6)

$$J_S = (1 - \kappa)J_F$$

165 (7)

166 Therefore, the dynamics of structural mass W as a function of age a is described by

167 the differential equation:

$$\frac{dW}{da} = \zeta_W \left(\kappa f j_a W^{2/3} - j_m W \right)$$

168 (8)

169 The parameter ζ_W in equation (8) represents the efficiency with which assimilates are

170 converted into structural mass. The previous equation can be rewritten as growth in

171 body size $\gamma(f, l)$ by substituting structural mass by body size (from equation 3) and

172 doing some manipulations:

$$\frac{dl}{da} = \gamma(f, l) = \xi(\kappa f j'_a - j'_m l)$$

(9)

The compound parameter ξ characterizes the growth rate in size and is defined as $\zeta_w / (3 v \delta^3)$. While the compound parameters j'_a and j'_m correspond to the assimilation and maintenance rates with respect to body length and are defined as $j_a v^{2/3} \delta^2$ and $j_m v \delta^3$ respectively.

Reproduction is assumed continuous. Adult fecundity after the substitution of structural mass by body size is described by:

$$\beta(f, l) = \begin{cases} ((1 - \kappa) f j'_a l^2) \sigma \frac{\zeta_e}{W_0} & \text{if } l > l_m \\ 0 & \text{otherwise} \end{cases}$$

(10)

The number of offspring produced per unit time is dependent on the yield for the conversion of assimilates into eggs ζ_e and the newborn structural mass $W_0 = v (\delta l_o)^3$. In addition, although we do not consider explicitly the egg stage, we do consider the egg survival σ that reduces the number of individuals effectively recruited as newborns.

We study the research question only in the ecological equilibrium state when the condition $\kappa f j'_a > j'_m l$ is fulfilled, therefore individuals do not starve and cannot shrink in size.

- *Survival*

190 Individuals in habitat 1 may die from background mortality μ_1 and in habitat 2 from
 191 either background μ_{2b} or predation mortality μ_{2p} . Background mortality is assumed
 192 to be size-independent and predation mortality is assumed size-dependent. To
 193 describe the size-dependent mortality experienced by individuals in habitat 2 we
 194 adopt a continuous piecewise-differentiable sigmoid function of body size (fig. 1):

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

195 (11)

196 where $L(l)$ is a scaled body size value, defined as $L(l) = 3l/l_v$. The sigmoid
 197 function is bounded by the maximum size-dependent mortality ε , which occurs at
 198 $l = 0$, and the maximum vulnerable-to-predation body size l_v at which size-
 199 dependent mortality vanishes. This function has been chosen because the parameters
 200 ε and l_v facilitate biological interpretation. However, size-dependent mortality has
 201 been commonly described as an exponential function of body size (fig. S1; Gislason
 202 et al. 2010; Jørgensen and Holt 2013):

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

204 (12)

205 therefore, we test the robustness of our results under this assumption (see Supp. Info.).

206 The total per capita death rate in habitat 2 μ_2 is the sum of the background and size-
 207 dependent mortality.

208 *Population dynamics*

209 Based on the individual life history described above, the size-structured population
210 model can be formulated by bookkeeping following de Roos et al. (1990) and de Roos
211 (1997). A detailed description of the size-structured population model can be found in
212 the supplementary information.

213 *Evolutionary dynamics*

214 In this study, we are interested in understanding how size-selectivity in mortality in
215 the rich feeding habitat affects the optimal timing of a habitat shift. We study the
216 evolution of the body size at the habitat shift l_s using the adaptive dynamics
217 framework, a suitable framework to analyze phenotypic evolution. Specifically, we
218 consider a population that evolves through the fixation of small and rare mutations in
219 this trait while otherwise being monomorphic (Geritz et al. 1998). A mutation is
220 eventually fixed if the population growth rate of mutant individuals in the
221 environment imposed by the resident population, which, in our model, is the food
222 resource density in the habitat 1 (R_1), is positive. This population growth rate
223 represents the mutant's invasion fitness, which for the resident equals 0 when it is at
224 an equilibrium state. The fitness of a mutant therefore depends on its own strategy and
225 on the strategy of the resident. We use the lifetime reproductive output R_0 as a
226 measure of invasion fitness following Mylius and Diekmann (1995). A mutant can
227 invade if its lifetime reproductive output $R_0(l'_s|l_s) > 1$; where l'_s is its own strategy,
228 and l_s is the strategy of the resident population. When a mutant phenotype invades, it
229 spreads and the new population reaches the ecological attractor. Then, it can be
230 invaded by another mutant that has an invasion fitness larger than one. In this way,

231 the population experiences a succession of mutations and evolves in the direction of
232 the selection gradient

$$\frac{\partial R_0(l'_s | l_s)}{\partial l'_s}$$

233 .
234 (13)

235 The point where the selection gradient becomes 0 is the Evolutionarily Singular
236 Strategy (ESS), which can be evolutionary stable or unstable. In the first case, no
237 other mutant can invade in the population and the ESS is a continuously stable
238 strategy (CSS), while in the latter evolutionary branching can occur (Geritz et al.
239 1998).

240 The lifetime reproductive output R_0 is numerically calculated and used in the
241 canonical equation of adaptive-dynamics theory (Dieckmann and Law 1996; Durinx
242 et al. 2008) to determine the resultant evolutionary trajectories. A detailed description
243 of the evolutionary model and the canonical equation can be found in the
244 supplementary information.

245 *Model parameterization*

246 All biomass densities are expressed in grams per liter, and time in the ecological
247 timescale is expressed in days. Parameters of the population with a habitat shift are
248 loosely based on the biology of Atlantic salmon (*Salmo salar*). Scaling coefficients of
249 assimilation and metabolic rate (j_a, j_m) were estimated from regressions of Koskela,
250 Pirhonen, and Jobling (1997) with the method of Jager, Martin, and Zimmer (2013)
251 and corrected for a temperature of 10 °C (although there is large variation in
252 temperature in wild populations, FishBase lists an approximate preferred temperature
253 of 9.3 °C for Atlantic salmon (Froese and Pauly 2018)). The yield parameter of

assimilates from ingested food efficiency has only a scaling effect on the population biomass equal for all stages, therefore, as it does not have any qualitative effect on the results, we assume a value of 1 g/g. The shape coefficient was estimated from regressions of Sutton, Bult, and Haedrich (2000); its estimated value is similar to shape coefficient by Pecquerie et al. (2011) for Pacific salmon species. Other parameters of the bioenergetics model are taken from Jager et al. (2013), while life history traits (i.e. body size at birth) were derived from reported data in the literature (table S1). 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. Since 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).

The model always approaches a stable ecological equilibrium for the parameterization considered. Therefore, the right-hand side of equation 9 is always positive, and possible starvation conditions of the consumers can be ignored.

Model analysis

We focus on populations in an ecological equilibrium and use the PSPManalysis software package (de Roos, 2018) to numerically compute and continue the equilibrium of the size-structured population model described above as a function of model parameters. We perform the evolutionary analysis using the PSPManalysis package as well, as it allows us to detect, classify and continue an ESS as a function

279 of ε , the scaling factor in the size-dependent mortality function μ_{2p} , and μ_{2b} , the
 280 size-independent mortality parameter in habitat 2.

281 We are interested in understanding how size-selectivity in mortality in the rich
 282 feeding habitat (habitat 2) affects the optimal timing of a habitat shift. However, a
 283 simple increase in size-dependent mortality does not only increase the size-selective
 284 nature of mortality but also the total mortality experienced in this habitat. Hence, the
 285 effect of the size-selectivity of mortality per se can only be unraveled while
 286 maintaining total mortality constant in the habitat 2. To evaluate evolutionary
 287 responses we therefore follow a specific approach, in which the contribution from
 288 size-dependent mortality sources is increased but overall mortality in habitat 2 is kept
 289 constant through a simultaneous decrease in size-independent mortality. More
 290 specifically, we find the ESS value for the body size to shift habitat when there is only
 291 size-independent mortality μ_{2b} equal to 0.006 day^{-1} in habitat 2 and adopt this as our
 292 starting, reference population (body size at habitat shift = 19.5 cm). A size-
 293 independent mortality μ_{2b} equal to 0.006 day^{-1} implies that an individual has an
 294 expected lifetime of 167 days in habitat 2 from the moment it shifts habitats.

295 Adopting this ESS body size for the resident phenotype in case it only experiences
 296 size-independent mortality μ_{2b} of 0.006 day^{-1} , we infer two combinations of size-
 297 independent mortality μ_{2b} and maximum size-dependent mortality ε that also result
 298 in a life expectancy of 167 days in habitat 2 (Pop. # 2 and 3 in table 1). To infer these
 299 combinations we numerically compute an individual's life expectancy after entering
 300 habitat 2 by integrating the following differential equation for its survival $S(\tau)$ as a
 301 function of the time τ it has spent in habitat 2:

$$\frac{dS}{d\tau} = -(\mu_{2b} + \mu_{2p}(l))S(\tau)$$

302 (14)

303 while simultaneously integrating the differential equation (9) for its growth in body
304 size l .

305 We subsequently start with a resident population that is characterized by the ESS
306 value for body size to shift habitat while experiencing a size-independent mortality
307 equal to 0.006 day^{-1} in habitat 2 and assess its evolutionary response when the
308 mortality changes to one of the inferred combinations of size-independent mortality
309 μ_{2b} and maximum size-dependent mortality ε that also lead to an average life
310 expectancy of 167 days in habitat 2 (fig. 2A). We perform an analogous analysis
311 while decreasing the size-selectivity in mortality in habitat 2: We adopt as our
312 starting, reference population, one characterized by the ESS value for the body size to
313 shift habitat when size-dependent mortality is the main source of mortality with a
314 maximum value ε of 0.015 day^{-1} in habitat 2 (body size to shift habitat = 21.4 cm; a
315 very low size-independent mortality equal to 0.0001 day^{-1} in habitat 2 is introduced to
316 avoid that individuals with a body size larger than the maximum vulnerable-to-
317 predation body size are immortal). We identify again two other combinations of
318 maximum size-dependent mortality ε and size-independent mortality μ_{2b} that result
319 in the same life expectancy in the habitat 2 (Pop. # 5 and 6 in table 1) as the size-
320 dependent mortality with maximum value ε of 0.015 day^{-1} for individuals shifting
321 habitat at the ESS body size and study the evolutionary response of the starting
322 resident population to a change to the two inferred mortality schedules (fig. 2B). This
323 procedure ensures that the evolutionary change we observe is strictly due to the
324 variation in size-selectivity in mortality and not to a change in total mortality.

For the four combinations of size-dependent and size-independent mortalities mentioned above and shown in Table 1 we use the PSPManalysis software package to compute the evolutionary trajectories of the body size at the habitat shift (evolutionary time 100 000 to 400 000 in fig. 2) as predicted by the canonical equation of adaptive dynamics. We perform an evaluation of the fitness components of the resident and a mutant individual in the environment set by the resident just before and immediately after the change in the size-selectivity of mortality (evolutionary time 100 000) to determine how various fitness components are maximized by selection under the imposed variation in size-selectivity in mortality (fig. 3). Likewise, we assess the effect of the variation in size-selectivity in mortality on the size distribution of the population (fig. 4).

Table 1. Mortality parameters used in Fig. 2, 3 and 4

Pop. #	Body size at habitat shift ^a (cm)	Life expectancy in habitat 2 (days)	Maximum size-dependent mortality ϵ	Contribution of size-dependent mortality ^b	Size-independent mortality μ_{2b}	Contribution of size-independent mortality ^b
Increasing size-selectivity in mortality in habitat 2						
1 ^c	19.5	167	0	0%	0.006	100%
2	19.5	167	0.0036	33.3%	0.004	66.7%
3	19.5	167	0.0075	66.7%	0.002	33.3%
Decreasing size-selectivity in mortality in habitat 2						
4 ^c	21.4	144	0.015	98.3%	0.0001	1.7%
5	21.4	144	0.01	70.1%	0.0021	29.9%
6	21.4	144	0.005	36.7%	0.0044	63.3%

^a Body size at habitat shift is ESS before change in size-selectivity in mortality.

^b Contribution of each source of mortality to total mortality in habitat 2.

^c Starting, reference population

A similar procedure is followed to test the robustness of the results under the assumption that size-dependent mortality is an exponential function of the body size. A detailed description can be found in the supplementary information.

Results

In the first part of this section, we show the evolutionary effects of size-selectivity in mortality in the rich feeding (habitat 2) on the optimal timing of the habitat shift.

Subsequently, we present the cause of these evolutionary responses. All evolutionary outcomes described below are continuously stable strategies (CSSs) and therefore locally evolutionarily stable (according to classification by Geritz et al., 1998).

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 body size at habitat shift decreases, so individuals have an earlier habitat shift with respect to the individuals exposed only to size-independent mortality (fig. 2A; notice the same life expectancy at the beginning of the evolutionary trajectories in fig. 2C). As a consequence of the evolution toward a smaller size at habitat shift, life expectancy in habitat 2 decreases as well (fig. 2C).

Conversely, when size-selectivity in mortality in habitat 2 decreases (i.e. the contribution of size-independent mortality sources to total mortality increases, given a constant life expectancy in habitat 2), the body size at habitat shift increases, so individuals have a delayed habitat shift with respect to the individuals exposed only to size-dependent mortality (fig. 2B; notice the same life expectancy at the beginning of the evolutionary trajectories in fig. 2D). By shifting habitat at a larger size, individuals increase their life expectancy in habitat 2 (fig. 2D).

In summary, the juveniles switch to the risky habitat 2 at a smaller body size when the risk is more size-dependent, despite that the risk would be avoidable by staying longer and growing in the nursery habitat to safety in size. This result is robust to a different size-dependent mortality function (Supp. Info. fig. S2, S3). 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

An analysis of the fitness components of the resident phenotype in the environment set by the resident population before and after the change in size-selectivity of mortality at evolutionary time 100 000 shows that an increase in size-selectivity in mortality in habitat 2 results in a slower growth rate and thus longer stay in habitat 1, higher survival and later maturation (black lines in fig. 3, left column). In contrast, this analysis reveals that a decrease in size-selectivity in mortality in habitat 2 results in a shorter stay in habitat 1 as a consequence of the increased growth rate in this habitat, lower survival and earlier maturation (black lines in fig. 3, right column).

Similarly, comparison of the resident and mutant phenotypes in the environment set by the resident after the change in size-selectivity of mortality at evolutionary time 100 000 shows that after an increase in size-selectivity in mortality, a smaller body size at habitat shift is selected for as it maximizes growth rate, leads to earlier maturation and increases fecundity at the expense of lower survival (dashed lines in fig. 3, left column). This analysis shows as well that after a decrease in size-selectivity in mortality a larger body size at the habitat shift is selected for as it maximizes survival at expense of slower growth and thus, later maturation (dashed lines in fig. 3, right column).

Fig. 3 shows that variation in size-selectivity in mortality in habitat 2 produces changes in the individual fitness components that are countered subsequently by selection. While 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

Size-selectivity in mortality in habitat 2 causes changes in the size-distribution of the population (the effects of size-dependent and size-independent mortality on habitat shift and population structure are further studied in Supp. Info. Fig. S4, S5). Because small individuals experience higher mortality rates than large individuals in habitat 2, adult density increases and juvenile density decreases in this habitat (fig. 4 left panel) when the size-selectivity in mortality is increased. This larger density of adults produces more offspring, which raises the density of juveniles in habitat 1. As a consequence of the increased density of juveniles 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 reduction in adult density the population birth rate decreases and thus the density of juveniles 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 risk would be avoidable 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. S6). 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 to changes in size-selectivity in mortality. Our naïve expectation, based on an individual-level optimization, 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. In fact, this is the case when an analysis of fitness maximization of this life history trait is carried out for an isolated individual (fig. 5). In contrast, when accounting for density dependence 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 juveniles 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. Werner and Gilliam (1984) have hypothesized before 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 introduced by 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). Furthermore, such effects have proven to shape population structure (i.e. size-distribution) and life history traits (Claessen et al. 2002; de Roos et al. 2007). In line with those findings for a population within a single habitat, we show that when there is a habitat shift, the nature of mortality in the rich feeding habitat has effects on growth, a food-dependent process, in the ‘nursery’ habitat.

Multiple studies have reported density, food availability and growth rate as determinants of the habitat shift 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); and, like Brown trout, Arctic char (Nordeng 1983) and Atlantic salmon (Lans et al. 2011)

are more likely to migrate at low food availability causing slow growth rate.

Similarly, low food availability causing slow growth rate results in smaller size at metamorphosis than that of fast growers in amphibians (Alford and Harris 1988; Beachy, Surges, and Reyes 1999). In wild populations observations are similar: a long-term study of Atlantic salmon in the Simojoki river showed that the mean body size at smolting (habitat shift) was negatively correlated with density in the previous autumn (Juttila et al. 2006). Hence, high population densities depress food levels and thereby growth rates, which triggers an early habitat shift in different species with ontogenetic habitat shift. This effect that growth rate has on the optimal timing of the habitat shift is well known from an individual-level optimization perspective, however its connection with density-dependent processes resulting from feedbacks between the population and the individual life history are only recently starting to be explored. In this study, we showed the intimate linkage between population structure and the optimal timing of the habitat shift as they have reciprocal effects on each other. 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.

In this study we have focused on size-dependent predation mortality in the rich feeding habitat whose size-selectivity implies a negative relation between mortality risk and body size. However, a positive relation between mortality risk and body size is also common. For instance, in general, fishing mortality imposed on exploited fish populations is higher for larger individuals. Fish stocks, therefore, may experience strong positive size-selective mortalities. Since adult biomass increases when the proportion of negative size-selective predation mortality increases as mortality cause,

size-dependent fishing mortality targeting mainly large individuals would reduce the adult biomass; hence, it cancels out the effect on population structure that size-dependent predation mortality has. As a consequence, in a population with size-dependent predation mortality as main source of natural mortality, size-dependent fishing mortality would reduce density and relax competition in the ‘nursery’ habitat. Consequently, the combination of negative size-selective predation mortality and positive size-selective fishing mortality would promote a habitat shift at larger body size. 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 (Juttila et al. 2006). Survival in early stages after the habitat shift are correlated with body size at the shift; therefore, Simojoki river Atlantic salmon is experiencing lower survival after the habitat shift. This suggests that some size-dependent fishing mortality may actually increase survival after habitat shift and perhaps enhance the fishing yield. Further research is necessary to determine the effects of fishing mortality on the optimal timing of the habitat shift.

For simplicity, in this study we have examined the evolution of the timing of a habitat shift while other life history traits are kept fixed. However, other life history traits are likely to evolve as well in response to changing fitness components like survival and growth. In particular, individuals may mature at smaller or larger size depending on growth opportunities and mortality risk. Fish often attain sexual maturation when growth rate reduces (near the asymptotic body size) (Jonsson and Jonsson 1993). Since 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. For instance, Brown trout moving from ‘nursery’ streams to lakes for

feeding delays maturity in comparison with stream residents for one or more years (Jonsson 1989). In future research, it would be therefore interesting to study jointly the evolution of the timing of the habitat shift and the timing of sexual maturation. Given that both traits influence population structure, such work will contribute to a deeper understanding of the linkage between life history trait evolution and density-dependent effects.

We consider the evolution of the body size at habitat shift in a monomorphic population in our model. However, in the wild, organisms with habitat shift, like salmonids and coral reef fish, often show variation in body size at habitat shift within a population. An extension of this model may include plasticity in this life history trait. For instance, adopting a reaction norm approach in which the probability to shift habitat is a function of the body size. Such an approach has been previously used to assess the evolutionary outcome of different mortality sources in the absence of density-dependent effects (Thériault et al. 2008). An extension of this approach to account for density-dependence would be possible using the Physiologically Structured Population Model framework used in this study (de Roos 1997).

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 population and the individual life history. Despite this, the analysis of the optimal timing of a habitat shift, as well as other life history traits, has been traditionally carried out in a context of individual optimization in isolation. Our results demonstrate the strong mutual influence that population structure and optimal timing of a habitat shift have on each other. This highlights the need for integrating

536 ecological interactions in the study of optimal life history traits. If we are to
537 understand the evolution of life histories the integration of ecological interactions in
538 the evolutionary analysis is certainly required.

References

- Alford, R. A., and H. R. N. 1988. Effects of larval growth history on anuran metamorphosis. *The American Naturalist* 131:91–106.
- Arendt, J. D. 2009. Influence of sprint speed and body size on predator avoidance in New Mexican spadefoot toads (*Spea multiplicata*). *Oecologia* 159:455–461.
- Beachy, C. K., T. H. Surges, and M. Reyes. 1999. Effects of developmental and growth history on metamorphosis in the gray treefrog, *Hyla versicolor* (Amphibia, Anura). *Journal of Experimental Zoology* 283:522–530.
- Boulton, A. M., and G. A. Polis. 1999. Phenology and Life History of the Desert Spider, *Diguetia mojavea* (Araneae , Diguetidae). *The Journal of Arachnology* 27:513–521.
- Claessen, D., and U. Dieckmann. 2002. Ontogenetic niche shifts and evolutionary branching in size structured populations. *Evolutionary Ecology Research* 4:189–217.
- Claessen, D., C. van Oss, A. M. de Roos, and L. Persson. 2002. The impact of size-dependent predation on population dynamics and individual life history. *Ecology* 83:1660–1675.
- Cocheret De La Morinière, E., B. J. A. Pollux, I. Nagelkerken, and G. Van Der Velde. 2002. Post-settlement life cycle migration patterns and habitat preference of coral reef fish that use seagrass and mangrove habitats as nurseries. *Estuarine, Coastal and Shelf Science* 55:309–321.
- de Roos, A. M. 1997. A Gentle Introduction to Physiologically Structured Population Models. Pp. 119–204 in S. Tuljapurkar and H. Caswell, eds. *Structured-Population Models in Marine, Terrestrial, and Freshwater Systems*. Springer Science & Business Media.

564 de Roos, A. M. 2018. PSPAnalysis: A package for numerical analysis of
565 physiologically structured population models.

566 de Roos, A. M., J. A. J. Metz, E. Evers, and A. Leipoldt. 1990. A size dependent
567 predator-prey interaction: who pursues whom? *Journal of Mathematical Biology*
568 28:609–643.

569 de Roos, A. M., and L. Persson. 2013. *Population and community ecology of*
570 *ontogenetic development*. Princeton University Press.

571 de Roos, A. M., and L. Persson. 2002. Size-dependent life-history traits promote
572 catastrophic collapses of top predators. *Proceedings of the National Academy of*
573 *Sciences* 99:12907–12912.

574 de Roos, A. M., T. Schellekens, T. van Kooten, K. van de Wolfshaar, D. Claessen,
575 and L. Persson. 2007. Food-Dependent Growth Leads to Overcompensation in
576 Stage-Specific Biomass When Mortality Increases: The Influence of Maturation
577 versus Reproduction Regulation. *The American Naturalist* 170:E59–E76.

578 Dieckmann, U., and R. Law. 1996. The dynamical theory of coevolution: a derivation
579 from stochastic ecological processes. *Journal of Mathematical Biology* 34:579–
580 612.

581 Diehl, S., and P. Eklov. 1995. Effects of piscivore-mediated habitat use on resources,
582 diet, and growth of perch. *Ecology* 76:1712–1726.

583 Dodson, J. J., J. Laroche, and F. Lecomte. 2009. Contrasting evolutionary pathways
584 of anadromy in euteleostean fishes. *American Fisheries Society Symposium*
585 69:63–77.

586 Durinx, M., J. A. J. (Hans) Metz, and G. Meszéna. 2008. Adaptive dynamics for
587 physiologically structured population models. *Journal of Mathematical Biology*
588 56:673–742.

589 Ferguson, G. W., and S. F. Fox. 1984. Annual Variation of Survival Advantage of
590 Large Juvenile Side-Blotched Lizards, *Uta stansburiana*: Its Causes and
591 Evolutionary Significance. *Evolution* 38:342–349.

592 Froese, R., and D. Pauly. 2018. Fishbase.

593 Geritz, S. A. H., E. Kisdi, G. Meszena, and J. A. J. Metz. 1998. Evolutionarily
594 Singular Strategies and the Adaptive Growth and Branching of the Evolutionary
595 Tree Evolutionarily Singular Strategies and the Adaptive Growth and Branching
596 of the Evolutionary Tree. *Evolutionary Ecology* 12:35–57.

597 Gislason, H., N. Daan, J. C. Rice, and J. G. Pope. 2010. Size, growth, temperature and
598 the natural mortality of marine fish. *Fish and Fisheries* 11:149–158.

599 Hampton, J. 2000. Natural mortality rates in tropical tunas: size really does matter.
600 *Canadian Journal of Fisheries and Aquatic Sciences* 57:1002–1010.

601 Hobson, K. A. 1999. Tracing Origins and Migration of Wildlife Using Stable
602 Isotopes : a Review. *Oecologia* 120:134–326.

603 Hutchings, J. a, and M. E. B. Jones. 1998. Life history variation and growth rate
604 thresholds for maturity in Atlantic salmon, *Salmo salar*. *Canadian Journal of*
605 *Fisheries and Aquatic Sciences* 55:22–47.

606 Jager, T., B. T. Martin, and E. I. Zimmer. 2013. DEBkiss or the quest for the simplest
607 generic model of animal life history. *Journal of Theoretical Biology* 328:9–18.
608 Elsevier.

609 Jonsson, B. 1989. Life history and habitat use of Norwegian brown trout (*Salmo*
610 *trutta*). *Freshwater Biology* 21:71–86.

611 Jonsson, B., and N. Jonsson. 1993. Partial migration: niche shift versus sexual
612 maturation in fishes. *Reviews in Fish Biology and Fisheries* 3:348–365.

613 Jonsson, N., B. Jonsson, and L. P. Hansen. 1998. The relative role of density-

614 dependent and density-independent survival in the life cycle of atlantic salmon
615 *Salmo salar*. Journal of Animal Ecology 67:751–762.

616 Jørgensen, C., and R. E. Holt. 2013. Natural mortality: Its ecology, how it shapes fish
617 life histories, and why it may be increased by fishing. Journal of Sea Research
618 75:8–18. Elsevier B.V.

619 Jutila, E., E. Jokikokko, and M. Julkunen. 2006. Long-term changes in the smolt size
620 and age of Atlantic salmon, *Salmo salar* L., in a northern Baltic river related to
621 parr density, growth opportunity and postsmolt survival. Ecology of Freshwater
622 Fish 15:321–330.

623 Keller, G., and G. Ribi. 1993. Fish predation and offspring survival in the
624 prosobranch snail *Viviparus ater*. Oecologia 93:493–500.

625 Keren-Rotem, T., A. Bouskila, and E. Geffen. 2006. Ontogenetic habitat shift and risk
626 of cannibalism in the common chameleon (*Chamaeleo chamaeleon*). Behavioral
627 Ecology and Sociobiology 59:723–731.

628 Kimirei, I. A., I. Nagelkerken, M. Trommelen, P. Blankers, N. van Hoytema, D.
629 Hoeijmakers, C. M. Huijbers, Y. D. Mgaya, and A. L. Rypel. 2013. What Drives
630 Ontogenetic Niche Shifts of Fishes in Coral Reef Ecosystems? Ecosystems
631 16:783–796.

632 Kooijman, S. a. L. M. 2010. Dynamic Energy Budget theory for metabolic
633 organisation. 3rd editio. Cambridge University Press, Cambridge, UK.

634 Kooijman, S. A. L. M., and J. A. J. Metz. 1984. On the dynamics of chemically
635 stressed populations: The deduction of population consequences from effects on
636 individuals. Hydrobiological Bulletin 17:88–89.

637 Koskela, J., J. Pirhonen, and M. Jobling. 1997. Feed intake, growth rate and body
638 composition of juvenile Baltic salmon exposed to different constant

639 temperatures. *Aquaculture international* 5:351–360.

640 Krause, J., S. P. Loader, J. McDermott, and G. D. Ruxton. 1998. Refuge use by fish
641 as a function of body length-related metabolic expenditure and predation risks.
642 *Proceedings of the Royal Society B: Biological Sciences* 265:2373–2379.

643 Lans, L., L. A. Greenberg, J. Karlsson, O. Calles, M. Schmitz, and E. Bergman. 2011.
644 The effects of ration size on migration by hatchery-raised Atlantic salmon
645 (*Salmo salar*) and brown trout (*Salmo trutta*). *Ecology of Freshwater Fish*
646 20:548–557.

647 Mylius, S. D. ., and O. Diekmann. 1995. On Evolutionarily Stable Life Histories ,
648 Optimization and the Need to Be Specific about Density Dependence. *Nordic*
649 *Society Oikos* 74:218–224.

650 Nisbet, R. M., E. B. Muller, K. Lika, and S. A. L. M. Kooijman. 2000. From
651 molecules to ecosystems through dynamic energy budget models. *Journal of*
652 *Animal Ecology* 69:913–926.

653 Nordeng, H. 1983. Solution to the “Char Problem” based on Arctic Char (*Salvelinus*
654 *alpinus*) in Norway. *Canadian Journal of Fisheries and Aquatic Sciences*
655 40:1372–1387.

656 Ohgushi, T., O. J. Schmitz, and R. D. C. N. Holt. 2012. Trait-mediated indirect
657 interactions: ecological and evolutionary perspectives. Cambridge University
658 Press, Cambridge.

659 Olsson, I. C., L. A. Greenberg, E. Bergman, and K. Wysujack. 2006. Environmentally
660 induced migration: The importance of food. *Ecology Letters* 9:645–651.

661 Pecquerie, L., L. R. Johnson, S. A. L. M. Kooijman, and R. M. Nisbet. 2011.
662 Analyzing variations in life-history traits of Pacific salmon in the context of
663 Dynamic Energy Budget (DEB) theory. *Journal of Sea Research* 66:424–433.

664 Rudolf, V. H. W. 2008. Impact of Cannibalism on Predator-Prey Dynamics : Size-
665 Structured Interactions and Apparent Mutualism. *Ecology* 89:1650–1660.

666 Rudolf, V. H. W., and J. Armstrong. 2008. Emergent impacts of cannibalism and size
667 refuges in prey on intraguild predation systems. *Oecologia* 157:675–686.

668 Schreiber, S., and V. H. W. Rudolf. 2008. Crossing habitat boundaries: Coupling
669 dynamics of ecosystems through complex life cycles. *Ecology Letters* 11:576–
670 587.

671 Semlitsch, R. D. 1990. Effects of body size, sibship, and tail injury on the
672 susceptibility of tadpoles to dragonfly predation. *Canadian Journal of Zoology*
673 68:1027–1030.

674 Sogard, S. M. 1997. Size selective mortality in the juvenile stages of teleost fishes: a
675 review. *Bulletin of Marine Science* 60:1129–1157.

676 Sutton, S. G., T. P. Bult, and R. L. Haedrich. 2000. Relationships among fat weight,
677 body weight, water weight, and condition factors in wild Atlantic salmon parr.

678 Thériault, V., E. S. Dunlop, U. Dieckmann, L. Bernatchez, and J. J. Dodson. 2008.
679 The impact of fishing-induced mortality on the evolution of alternative life-
680 history tactics in brook charr. *Evolutionary Applications* 1:409–423.

681 Walters, A. W., T. Copeland, and D. A. Venditti. 2013. The density dilemma:
682 Limitations on juvenile production in threatened salmon populations. *Ecology of*
683 *Freshwater Fish* 22:508–519.

684 Werner, E. E., and J. F. Gilliam. 1984. The Ontogenetic Niche and Species
685 Interactions in Size-Structured Populations. *Ecology* 15:393–425.

686 Wysujack, K., L. A. Greenberg, E. Bergman, and I. C. Olsson. 2009. The role of the
687 environment in partial migration: Food availability affects the adoption of a
688 migratory tactic in brown trout *Salmo trutta*. *Ecology of Freshwater Fish* 18:52–

689   59.

690

Figure 1. Size-dependent mortality in habitat 2.

Figure 2. Effects of increased (A, C) and decreased (B, D) size-selectivity in mortality in habitat 2 on the optimal timing of the habitat shift (A, B) and life expectancy in habitat 2 (C, D). When size-selectivity increases (A, C), evolutionary dynamics starts from the ESS value for the body size at habitat shift (19.5 cm) when individuals experience only size-independent mortality 0.006 day^{-1} until time 100 000. At this time, the change in size-selectivity in mortality in habitat 2 occurs (changes in size-dependent and size-independent mortality according to table 1: Pop. # 1 corresponds to the black line, 2 to the dark grey line and 3 to the light grey line). When size-selectivity decreases (B, D), evolutionary dynamics starts from the ESS value for the body size at habitat shift (21.4 cm) when individuals mainly experience a maximum size-dependent mortality 0.015 day^{-1} until time 100 000. At this time, the change in size-selectivity in mortality in habitat 2 occurs (changes in size-dependent and size-independent mortality according to table 1: Pop. # 4 corresponds to the black line, 5 to the dark grey line and 6 to the light grey line). Other parameter values as in table S1.

Figure 3. Individual growth, survival, cumulative fecundity and expected lifetime cumulative fecundity of the resident before (black solid lines) and of the resident and mutant phenotypes immediately after (black and grey dashed lines, respectively) size-selectivity in mortality increases (left column) and decreases (right column) in habitat 2. Size-dependent and size-independent mortality parameters before size-selectivity in mortality increases and decreases are as shown in table 1 for Pop. # 1 and 4, respectively; whereas after the increase and decrease they are as shown in table 1 for Pop. # 3 and 6, respectively. Resident phenotypes shift habitat at 19.5 cm (left column) and 21.4 cm (right column) and mutant phenotypes shift habitat at a body size that differs 5% from the body size at habitat shift of the resident phenotype in the direction of the selection gradient (decrease when a smaller body size at habitat shift is selected for in fig. 2, increase if a larger body size at habitat shift is selected for in fig. 2).

Figure 4. Size-distribution of the resident population just before (solid lines) and immediately after (dashed lines) size-selectivity in mortality increases (left column) and decreases (right column) in habitat 2. Individuals shift habitat at a body size of 19.5 cm (left) and 21.4 cm (right). Juveniles in habitat 1 (blue region) and in habitat 2 (yellow region), and adults (green region) are shown. Size-dependent and size-independent mortality parameters before size-selectivity in mortality increases and decreases are as shown in table 1 for Pop. # 1 and 4, respectively; whereas after the increase and decrease they are as shown in table 1 for Pop. # 3 and 6, respectively. Other parameter values as in table S1.

Figure 5. 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 for an individual in isolation (no density-dependence in habitat 1). Initial phenotype (dotted line) shifts habitat at 19.5 cm, and novel phenotypes shift habitat at a body size 5% larger than the initial phenotype (light grey dashed lines) and at a body size 5% smaller than the initial phenotype (dark grey dashed lines). Size-dependent and size-independent mortality before (after) size-selectivity in mortality increases in the habitat 2 are as shown in table 1 for Pop. # 1 (Pop. # 3). A larger fitness is achieved by a novel phenotype with larger body size at habitat shift after the increase in size-selectivity in mortality if there is no density-dependence in the habitat 1 (in all cases individuals grow at the same rate: R_1 is constant and equal to 0.457 g/L).