Food-web dynamics under climate change

Zhang, L.; Takahashi, M.; Hartvig, Martin; Andersen, Ken Haste

Published in:
Proceedings of the Royal Society B: Biological Sciences

Link to article, DOI:
10.1098/rspb.2017.1772

Publication date:
2017

Document Version
Peer reviewed version

Manuscript type: Research paper

Title: Cannibalism as a selective force on offspring size in fish

Authors

Karin H. Olsson[1,2,*], email: olsson.karin.h@gmail.com

ORCID iD, 0000-0002-1695-0989

Ken Haste Andersen[1], email: kha@aqua.dtu.dk

Affiliations

1: Center for Ocean Life, National Institute for Aquatic Resources, Technical University of Denmark, Bygning 202, DK 2800 Lyngby, Denmark

2: Cefas Laboratory, Lowestoft, Suffolk NR33 0HT, United Kingdom (present address)

* Corresponding author

Keywords: cannibalism, offspring size, life history

Abstract

Cannibalism may cause considerable mortality on juvenile fish and it has been hypothesised that it may exercise selection on offspring size in that larger offspring may enjoy a size refuge. For this to be evolutionarily advantageous the survival of individual offspring must compensate for the reduced fecundity implied by larger offspring size. We develop a model which combines standard assumptions of size-dependent mortality with adult cannibalism to investigate the potential for cannibalism to act as selective force on offspring size. We find that for this potential to be realised, the mortality due to cannibalism must exceed a threshold value that is a decreasing function of non-cannibalistic predation intensity, cannibalized size range width and the average cannibalized size. If cannibalism exceeds this threshold, the model predicts evolution of offspring size towards refuges above or below cannibalized size range depending on initial offspring size. Cannibalistic mortality cannot be so great that the population is non-viable, however, the range of parameter values describing cannibalistic intensity allowed within these boundaries is wide. On this basis, we suggest that cannibalism is a potential mechanism for offspring size selection.

Introduction

Offspring size is an important component of reproduction. Larger offspring typically enjoy greater survival (Baker 2008) but at the cost of reduced parental fecundity (Elgar 1990, Berrigan 1991). In aquatic organisms, predators are often gape-size limited and mortality therefore decreases with size (Peterson and Wroblewski 1984, Houde 1997). While the prediction that mortality declines with size may be valid when considering the average over many species, it may not be quite correct for a specific population. If, for example, the abundance or voraciousness of a segment of predators is greater than average, mortality may even increase with size for the prey in the size range targeted by predators. Such variation in size-dependent mortality may influence selection on optimal offspring size.

One candidate mechanism that may cause mortality to increase with size is cannibalism, a common phenomenon (Fox 1975, Polis 1981) that has been identified in over 100 species of fish spanning more than thirty families (Smith and Reay 1991). In many species of fish, adult cannibalism has been shown to cause considerable juvenile mortality (Sparholt 1994). The magnitude of cannibalistic mortality depends on the prey-size preference of the cannibals as well as on external factors such as availability of alternative prey. Many fish have a preferred prey size range (Ursin 1973), however, since most fish lay small eggs relative to adult body size, juveniles will at some point fall within the prey size range of the adults. The preferred prey size range is influenced by the probability of capture as well as the energetic pay-off (Christensen 1996), resulting in a predicted dome-shaped mortality curve centred on the preferred prey size (Persson 1987). This has been verified experimentally in Arctic charr *Salvelinus alpinus* (Amundsen 1994) and perch *Perca fluviatilis* (Lundvall et al. 1999), while indirect observation of guppies *Poecilia reticulata* suggested that only the smallest juveniles are in the vulnerable size range (Nilsson et al. 2011). Furthermore, stomach content analyses showed that cannibalism in Norwegian herring *Clupea harengus* (Holst

1992) and Baltic Sea cod *Gadus morhua* (Neuenfeldt and Köster 2000) was consistent with adult prey size preference. The impact of cannibalism can be high, potentially removing up to 95 % of an age class in some aquatic organisms (Fox 1975). In Baltic Sea cod, cannibalism may remove 44% of a cohort in the first two years (Neuenfeldt and Köster 2000), although the level of cannibalism varies over time and with spawning stock biomass (Jensen and Sparholt 1992). Cannibalism has also been linked to low food concentration (Folkvord 1991, Nilsson et al. 2011), though food availability and adult population size are likely to covary (Smith and Reay 1991). Due to the size-specific nature of cannibalism, in combination with the potential impact on juvenile survival, it has been put forward as a source of selection on offspring size (Weeks and Gaggiotti 1993, Nilsson et al. 2011).

To investigate the circumstances under which cannibalism can exercise selection on offspring size we build a lifetime fitness model which specifically includes cannibalistic mortality. We base our model on the assumption of a non-growing population and identify the evolutionary endpoint strategy. First, we assume that all density-dependence occurs early in life and treat cannibalistic mortality as a free parameter, recognising that the level of cannibalism may vary with many external factors. Second, we assume that cannibalism acts as the sole density-regulating mechanism, and use this criterion to determine the level of cannibalistic mortality.

Lifetime fitness model

We build an evolutionary life history model assuming a non-growing population at equilibrium to explain the selective pressure cannibalism may exercise on offspring size. We assume a two-stage life cycle consisting of a juvenile phase during which all available energy is used for growth and an adult phase where the energy is directed to reproduction, such that growth is determinate. All individuals are assumed to be affected by density-independent predation mortality, but only adults

are cannibalistic. Juveniles, therefore, also experience mortality due to adult cannibalism. We ignore the energetic benefit adults may obtain from the predation and further assume that the number of juveniles is sufficiently large so that an adult is unlikely to reduce its fitness by consuming its own offspring. To parameterise the model, we make the following assumptions of growth, mortality and reproduction (all model parameters are listed in Table 1):

1. Growth rate scales metabolically with size (body weight) w

$$g(w) = Aw^n \quad (1)$$

where A is a growth constant and n is the metabolic exponent (von Bertalanffy 1957, Day and Taylor 1997, West et al. 2001) which we set to $n = 3/4$.

2. Predation mortality rate is assumed to be a power law scaling with size as

$$\mu_p(w) = aAw^{n-1} \quad (2)$$

where a is a non-dimensional parameter giving the strength of predation. Kokkalis et al. (2017) estimated values for a for a range of fish stocks and found these to be lognormally distributed with mean 0.22 and standard deviation 0.7 in the log domain; the model will be explored for values $0.1 < a < 0.8$. Predation mortality is also assumed to be proportional to the growth constant A as higher foraging rates imply higher mortality (empirically, e.g. Sih 1982, theoretically, Werner and Anholt 1993). In fish, empirical data suggest that mortality typically scales with size as $w^{-0.25}$ (Peterson and Wroblewski 1984, McGurk 1986). The model becomes mathematically simpler if juvenile and adult mortality have the same scaling, though relaxing this assumption does not qualitatively change the outcome, as shown in Appendix A.

3. Cannibalism is assumed to increase mortality for sizes around the adult preferred predator-prey size ratio β. We model this preferred prey size using an Ursin size selection function

110 $$\theta(w/w_m) = \exp\left[\frac{-\log^2(\beta w/w_m)}{2\sigma^2}\right]$$

where w_m is the adult size and σ defines the width of the selection function. For fish, typical
112 parameter values are $\sigma = 1$ and $\beta = 100$ (Ursin 1973). We assume that cannibalistic mortality rate is proportional to the size selection $\mu_c(w) = c_c\theta(w/w_m)$. It turns out that the notation later is much
114 simplified if we define a dimensionless constant $b = c_c w_m^{1-n}/A$. With this definition, the cannibalistic mortality rate becomes

116 $$\mu_c(w) = bAw_m^{n-1}\theta(w/w_m) \quad (3)$$

where b is a non-dimensional parameter describing the strength of the cannibalistic mortality. The
118 value of b depends on the number of adults and juveniles, adult prey preference and the availability of alternative food sources. We do not know the value of b, but explore the consequences using two
120 approaches: fitness optimisation, where density-dependence occurs early in life such that b is independent of other life history parameters, and adaptive dynamics, where b is determined from
122 the population structure.

124 4. Growth is assumed to be determinate: adults direct all available energy to reproduction. The available energy at the adult stage thus scales with size as growth does at the juvenile stage, i.e.
126 Aw_m^n. Therefore, consistent with empirical studies (e.g. Charnov et al. 2001), the rate of reproduction scales with size at maturity as $R_m = \varepsilon Aw_m^n$ where ε is the reproductive efficiency
128 which accounts for energy expenditure not directly directed towards offspring production.

130 With these four assumptions, we can calculate the expected lifetime reproductive value R_0 as the probability of survival to maturation $s_{w_0 \to w_m}$ multiplied with adult reproductive output f_m: $R_0 =$
132 $s_{w_0 \to w_m} f_m$. The adult reproductive output is obtained by integrating the rate of reproduction discounted by adult survival from age at maturity, t_m, to ∞, dividing by offspring size w_0 and
134 multiplying by a factor of ½ to account for an even sex ratio:

$$f_m = \frac{1}{2w_0}\int_{t_m}^{\infty} \varepsilon A w_m^n \exp[-aAw_m^{n-1}(t-t_m)]\,\mathrm{d}t = \frac{\varepsilon w_m}{2aw_0}. \quad (4).$$

Adult reproductive output is therefore proportional to adult body size w_m, which is consistent with empirical studies (Tsoukali et al. 2016).

Survival is calculated by integrating the ratio between mortality $\mu(w)$ and growth $g(w)$ rates from size at birth to size at maturity, $s_{w_0 \to w_m} = \exp\left[-\int_{w_0}^{w_m} \mu(w)/g(w)\,\mathrm{d}w\right]$ (Thygesen et al. 2005, Kiflawi 2006). The total mortality is the sum of the predation mortality μ_p and the cannibalistic mortality μ_c and total survival is the product of the respective survival probabilities, $s_{w_0 \to w_m} = s_p s_c$. Inserting growth (Eq. 1) with predation mortality (Eq. 2) gives survival due to predation s_p as

$$s_p = \left(\frac{w_0}{w_m}\right)^a \quad (5)$$

and with cannibalistic mortality (Eq. 3) gives survival due to cannibalism s_c as

$$s_c = \exp[-bc\mathrm{H}(w_0, w_m)] \quad (6)$$

with

$$c = \beta^{n-1}\sigma\sqrt{\frac{\pi}{2}}\exp\left[\frac{\sigma^2(n-1)^2}{2}\right], \text{ and}$$

$$\mathrm{H}(w_0, w_m) = \mathrm{erf}\left[\frac{\log(\beta)+(n-1)\sigma^2}{\sigma\sqrt{2}}\right] - \mathrm{erf}\left[\frac{\log(\beta(w_0/w_m))+(n-1)\sigma^2}{\sigma\sqrt{2}}\right]$$

where erf is the error function. Multiplying survivals and adult reproductive output (Eq. 4) gives lifetime reproductive value R_0

$$R_0 = \left(\frac{w_0}{w_m}\right)^a \exp[-bc\mathrm{H}(w_0, w_m)]\varepsilon\frac{w_m}{2aw_0} \quad (7)$$

The growth parameter A disappears from the survival function and the survival depends only on the non-dimensional mortality parameters a and b and the size-selectivity parameters β and σ. Furthermore, since Eq. 7 depends on the ratio between offspring size w_0 and adult size w_m, rather than either parameter separately, we can define the non-dimensional ratio between offspring size and adult size $z = w_0/w_m$, which we will call the relative offspring size. Simple physiology

Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish
Formatted: Danish

158 requires $0 < z < 1$. Analysing the model using z provides a means of generalising offspring size strategies without referring to any specific size. Eq. 7 can thus be rewritten as

160 $$R_0 = z^{a-1} \exp[-bc\mathrm{H}(z)] \frac{\varepsilon}{2a} \quad (8)$$

If β and σ are constants, c is also constant and the fitness of a particular offspring size strategy z 162 only depends on the non-dimensional parameters reproductive efficiency ε, predation parameter a and cannibalism parameter b.

164

Optimal strategy

166 The optimal strategy is that which leads to the highest fitness, here measured as the lifetime reproductive value R_0. Evaluating the optimal strategy in a non-growing population requires 168 determining how density-dependence operates. If density-dependence occurs as a multiplicative factor early in life with no further effect on growth and survival, the optimal strategy can be 170 identified by locating the offspring size strategy which yields the highest fitness R_0 (Mylius and Diekmann 1995) for a given value of cannibalism parameter b. However, if cannibalism constitutes 172 the main density-regulating mechanism, the level of cannibalism will itself be determined as the amount of density-dependence needed to achieve a population at steady state, i.e. $R_0 = 1$. In that 174 case, we need to determine the evolutionarily stable strategy (ESS) as the strategy that cannot be invaded by other strategies (Geritz et al. 1998). Below we investigate the model under both 176 scenarios.

178 ***Early density-dependence***

As relative offspring size z increases, individual survival increases, but parental fecundity decreases. 180 Cannibalism introduces a phase of elevated mortality in the size range defined by the prey size preference parameters β and σ (Fig. 1a). The effect of offspring size on survival is then described 182 by an increasingly *S*-shaped curve, where larger offspring size offers a refuge from the cannibalistic

mortality phase (Fig. 1b). The optimal offspring size ratio z^* is found by taking the first derivative of the fitness function (Eq. 8) and setting it to 0, $dR_0/\,dz = 0$:

$$\frac{\mathrm{dR_0}}{\mathrm{dz}} = \frac{\varepsilon s_p s_c}{2z^2}\left(1 + \frac{\mathrm{bc}}{\sigma \mathrm{a}}\sqrt{\frac{2}{\pi}}\exp\left[-\left(\frac{\log(\beta z)+(n-1)\sigma^2}{\sigma\sqrt{2}}\right)^2\right] - \frac{1}{a}\right) \quad (9)$$

In the absence of cannibalism ($b = 0$), the term in the parenthesis is independent of z and Eq. 9 is negative for z in the allowed range $0 < z < 1$. Therefore, R_0 is a strictly decreasing function of z and maximum R_0 is given by the smallest possible value of z (Fig. 1c). This means that in the absence of cannibalism, the optimal strategy is to make as small eggs as possible. If cannibalism is strong enough, there may also be a local optimum for a larger-z strategy, in addition to the small-z strategy identified by Eq. 9 if $b = 0$. Whether the local, larger-z optimum will occur can be determined by analysing the expression inside the brackets. The local optimum for the large-z strategy is given by

$$z^* = \frac{1}{\beta}\exp\left[\sigma\sqrt{\log(2b^2 c^2/(\pi\sigma^2(1-a)^2))} + (1-n)\sigma^2\right] \quad (10)$$

which is only defined if

$$b \geq b_{\min} = \sqrt{\pi\sigma^2 (1-a)^2/(2c^2)} \quad (11)$$

From Eq. 10, it follows that the local maximum z^* decreases with increasing prey size preference β and increases with the strength of cannibalism b. Consequently, there are two optimal offspring size strategies: a small one and a large one, as shown in Fig. 2. For the two strategies to emerge, b must exceed a threshold value b_{min} ~~$b \geq b_{min}$~~, which decreases as the predation parameter a increases (Eq. 11, b_{min} dashed line in Fig. 2; the range of a is assumed to be $0.1 < a < 0.8$).

Density-dependent cannibalism

Next, we will assume that all density-dependence is caused by cannibalism and try to locate evolutionary stable strategies (ESS) by determining when a resident strategy z cannot be displaced

by an alternative invader strategy $\tilde{z}$ (Maynard Smith and Price 1973). In a non-growing population,
208 the resident strategy z must have fitness $R_0(z) = 1$. The level of cannibalism required to maintain
the population at equilibrium is found by rearranging Eq. 8:

210 $$b_{eq}(z) = \frac{1}{cH(z)} \log\left(\varepsilon \frac{s_p}{2az}\right) \quad (12)$$

212 The evolutionary stability of the resident strategy depends on whether there exists an alternative,
invader strategy $\tilde{z}$, which is rare enough not to affect b_{eq} but has higher fitness than the resident z.
214 This can be analysed by constructing a so-called pairwise invasibility plot (van Tienderen and de
Jong 1986, Geritz et al. 1998). This plot charts the relative success of an invader strategy (along the
216 y-axis) against a resident strategy (along the x-axis); by definition, the resident's $R_0 = 1$, so if the
invader's $R_0 > 1$, it can invade (grey areas), while if the invader's $R_0 < 1$, it canot (white
218 areas) ~~resident $R_0 = 1$, so if invader $R_0 > 1$ the invader can invade (grey areas), while if invader $R_0 < 1$ it cannot (white areas)~~. Note that this procedure is slightly different than the usual which
220 would calculate the invasion fitness as the population growth rate, with the criterion for invasion
being that the invasion fitness > 0 (e.g. Geritz et al. 1998). Nevertheless, as we are only interested in
222 whether the invader can invade or not, and not the speed of invasion, the two criteria are identical.
If the invader strategy is more successful than the resident, the trait value in the population will shift
224 towards that of the invader. For small differences in size, the direction of selection can be
determined by the sign of the first derivative of the fitness function of the invader $\tilde{z}$, evaluated for a
226 particular resident strategy z and therefore $b_{eq}(z)$ (Geritz et al. 1998). If the derivative is positive
offspring size will increase and vice versa, as indicated with black arrows in Fig 3. When the
228 derivative is 0, the strategies have equal fitness, illustrated by the intersection points along the
diagonal in Fig. 3. If the second derivative at the intersection point is negative, the point represents
230 a fitness maximum for the invader. As both smaller and larger invaders have lower fitness the
strategy is evolutionarily stable (ESS at $z = z_{eq}$, filled circle in Fig. 3). Conversely, if the second

derivative is positive, both smaller and larger invader have higher fitness and the point is referred to as an evolutionary repeller (open circle in Fig. 3).

The pairwise invasibility plot in Fig. 3 indicates the possibility of two evolutionarily stable strategies, depending on the resident strategy z. If the resident strategy z is within the size range of the adult prey size preference (indicated by the dotted line showing the Ursin selection function) and greater than the evolutionary repeller (indicated by the open circle), the gain in offspring survival increases rapidly with size, leading to selection for larger offspring as a size refuge from cannibalism. The ESS at $z = z_{eq}$ represents the evolutionary endpoint of this selection and the final trade-off between fecundity and individual offspring survival. Conversely, for resident z smaller than the evolutionary repeller, larger offspring size will reduce fecundity without a sufficient gain in survival, and selection will favour ever smaller z.

The relative offspring size at ESS and the evolutionary repeller depends on the predation parameter a and the reproductive efficiency ε (Eq. 12, Fig. 4). Cannibalism will only contribute to selection for larger offspring if the level of cannibalism at equilibrium b_{eq} is greater than b_{min} (as described above; Eq. 11 and dashed line in Fig. 2). Higher predation (large values of a) leads to lower density-dependent cannibalism (b_{eq} decreases with increasing a), so that eventually, at high a, the large offspring size ESS disappears along with the evolutionary repeller. Increasing the reproductive efficiency increases the number of surviving juveniles, which leads to higher cannibalism (consistent with the observation that cannibalism increases with larger stock size (Jensen and Sparholt 1992)). Increasing reproductive efficiency can thus compensate for increased mortality and make the ESS appear at higher mortalities.

Discussion

A key life history trade-off is that between offspring size and offspring numbers, typically representing the balance of offspring survival against parental fecundity. This constitutes the principal assumption in much theoretical work (Smith and Fretwell 1974, Kiflawi 2006) and is supported by empirical findings (Elgar1990, Blackburn 1991). Considering only this trade-off, in conjunction with metabolic scaling of growth and mortality, leads to an optimal offspring size that is infinitely small, with a correspondingly infinitely high fecundity (Thygesen et al. 2005, Kiflawi 2006). On the other hand, if mortality scales differently for juveniles and adults, optimal offspring size can be demonstrated to have a defined optimum (Kiflawi 2006, Jørgensen et al. 2011). Our model builds on this trade-off with the addition that cannibalism can also act as a driver of offspring size. The analysis of the model demonstrates that cannibalism can select for large offspring size, provided that the mortality imposed by cannibalism is strong enough to dominate other sources of mortality. It is also worth noting that in Appendix, one of the outcomes of the model is a single optimum formed from the combined effects of predation mortality and cannibalism, which suggests that cannibalism may contribute to selection on offspring size, either alone or in conjunction with other sources of mortality. Nevertheless, selection for larger offspring size may be prevented if the higher fecundity associated with producing smaller offspring compensates for the additional mortality. The conclusion from the model is therefore that two evolutionarily stable strategies may exist: small offspring size and large offspring size, where only the large offspring size strategy is driven by cannibalism.

In the case of marine animals in general and fish in particular, there is evidence for two distinct offspring size strategies: either very small compared to the adult size or roughly proportional to the adult size (Neuheimer et al. 2015, Olsson et al. 2016). The large offspring strategy, identified by Neuheimer et al. (2015) as 'proportional', approximately matches the prey size preference

determined by β and is thus only slightly lower than the large-z ESS that emerges from our model makes cannibalism a possible contributor to this offspring size strategy.

The model is kept simple to focus on size-dependent mortality and cannibalism by adults as the driving force of offspring size evolution. These simplifications ignore potentially important effects of demography, population mixing, positive effects of cannibalism and flexibility of trait evolution. For instance, compared to population demographics, evolution is typically a slow process, and for the purposes of modelling an adaptive response to evolutionary pressure we assume a population in equilibrium. Nevertheless, an underlying assumption when evaluating selection is that the magnitude of the selection factor remains reasonably constant between breeding seasons, but if cannibalism destabilises population demography (e.g. Cushing 1991, Claessen et al. 2000) or if external factors such as the availability of alternative food sources or adult mortality are highly variable, this may not be the case. Furthermore, to exercise selection on offspring size, the magnitude of cannibalism must be above a certain minimum level relative to the background mortality. Even then, selection for larger offspring would depend on the initial state of the resident population. Theoretical models have suggested that cannibalism is more likely to evolve if there is a spatial overlap between adults and juveniles (Smith and Reay 1991) and the population is well-mixed (Lion and van Baalen 2009). This suggests that the likelihood of cannibalism resulting in directional selection on offspring size may depend on habitat characteristics. For instance, Fox (1975) suggested that freshwater habitats, which are often more temporal in nature as well as ~~and~~ spatially unstructured, may be more likely to offer the required spatial overlap, which would help explain the prevalence of cannibalistic freshwater species. In the data compiled by Olsson et al. (2016), the teleost species that produced relatively large offspring tended to be small and inhabit brackish- and shallow-water habitats, especially in the breeding season, which would appear to be consistent with the observation made by Fox (~~1991~~1975).

306

Furthermore, in populations where cannibalism is substantial, juveniles may be expected to employ
308 counter-measures to mitigate the threat. For instance, juvenile perch can utilise refuges to reduce cannibalistic mortality (Lundvall et al. 1999) and fry survival in experimental guppy populations
310 improves when shelters are available (Nilsson et al. 2011). If effective, for instance if the refuges offer juveniles shelter while denying adults access, larger size may no longer confer a sufficiently
312 substantial advantage to compensate for lost fecundity and selection for larger offspring size would weaken. The model also assumes that only adults which have attained a specific size engage in
314 cannibalism, but both inter- and intracohort cannibalism has been recorded in many fish (Smith and Reay 1991). If cannibalism is a feature over a greater range of sizes, the σ of the model would be
316 expected to increase, causing the 'dome' of the mortality curve in Fig. 1a to widen and become less distinct relative to other sources of mortality, with a less prominent selection on size.

318

On the other hand, the model ignores the positive effects cannibalism may have, both on the adults
320 and surviving juveniles. For instance, if alternative food sources are scarce or inaccessible, cannibalism may provide adults with a ready supply of nutrients (van den Bosch et al. 1988), which
322 would facilitate and maintain the prevalence of cannibalism in a population (Klug et al. 2006). Furthermore, surviving juveniles would benefit from less competition for food and shelter. In this
324 model cannibalism has been assumed to be non-filial and therefore not reducing the survival of related offspring, but filial cannibalism is prevalent in fish species with parental care (Manica 2002).
326 This may exert selective pressure on offspring if parents can selectively remove low-quality offspring (Klug et al. 2006). However, most fish are broadcast spawners, and are unlikely to be able
328 to distinguish their own offspring once the eggs have hatched. Ultimately, the more benefits cannibalism confers upon the cannibals, the more likely the strategy will be and the more potential
330 the selection pressures outlined in the model will have to translate into selection on offspring size.

332

334

References

336 Amundsen, P.-A. 1994. Piscivory and cannibalism in Arctic charr. - J. Fish Biol. 45: 181–189.

Baker, J. D. 2008. Variation in the relationship between offspring size and survival provides insight
338 into causes of mortality in Hawaiian monk seals. - Endanger. Species Res. 5: 55–64.

Berrigan, D. 1991. The allometry of egg size and number in insects. - Oikos 60: 313–321.

340 Blackburn, T.M. 1991. An interspecific relationship between egg size and clutch size in birds. - Auk. 108: 973-977.

342 Brown, J. H. et al. 2004. Toward a metabolic theory of ecology. - Ecology 85: 1771–1789.

Charnov, E. L. et al. 2001. Reproductive constraints and the evolution of life histories with
344 indeterminate growth. - Proc. Natl. Acad. Sci. U. S. A. 98: 9460–9464.

Christensen, B. 1996. Predator foraging capabilities and prey antipredator behaviours: Pre- versus
346 postcapture constraints on size-dependent predator-prey interactions. - Oikos 76: 368–380.

Claessen, D. et al. 2000. Dwarfs and giants: Cannibalism and competition in size-structured
348 populations. - Am. Nat. 155: 219–237.

Cushing, J. M. 1991. A simple model of cannibalism. - Math. Biosci. 107: 47–71.

350 Day, T. and Taylor, P. D. 1997. Von Bertalanffy's growth equation should not be used to model age and size at maturity. - Am. Nat. 149: 381–393.

352 Elgar, M. A. 1990. Evolutionary compromise between a few large and many small eggs: comparative evidence in teleost fish. - Oikos 59: 283–287.

354 Folkvord, A. 1991. Growth, survival and cannibalism of cod juveniles (*Gadus morhua*): effects of feed type, starvation and fish size. - Aquaculture 97: 41–59.

356 Fox, L. R. 1975. Cannibalism in natural populations. - Annu. Rev. Ecol. Syst. 6: 87–106.

Geritz, S. A. H. et al. 1998. Evolutionarily singular strategies and the adaptive growth and
358 branching of the evolutionary tree. - Evol. Ecol. 12: 35–57.

Gislason, H. et al. 2010. Size, growth, temperature and the natural mortality of marine fish. - Fish
360 Fish. 11: 149–158.

Holst, J. C. 1992. Cannibalism as a factor regulating year-class strength in the Norwegian spring-
362 spawning herring stock. - ICES C. 1992/H14 Pelagic Fish Comm.: 1–16.

Houde, E. D. 1997. Patterns and trends in larval-stage growth and mortality of teleost fish. - J. Fish
364 Biol. 51: 52–83.

Jensen, H. and Sparholt, H. 1992. Estimation of predation mortality of cod in the Central Baltic
366 using MSVPA. - ICES C.M. 1992/J23 Balt. Fish Comm.: 1–41.

Jørgensen, C et al. 2011. A model for optimal offspring size in fish, including live-bearing and
368 parental effects. - Am. Nat. 177: E119-E135.

Kiflawi, M. 2006. On optimal propagule size and developmental time. - Oikos 113: 168–173.

370 Klug, H. et al. 2006. Parents benefit from eating offspring: Density-dependent egg survivorship compensates for filial cannibalism. - Evolution (N. Y). 60: 2087–2095.

372 Kokkalis, A. et al. 2017. Estimating uncertainty of data limited stock assessments. - ICES J. Mar. Sci. 74: 69-77.

374 Lion, S. and van Baalen, M. 2009. The evolution of juvenile-adult interactions in populations structured in age and space. - Theor. Popul. Biol. 76: 132–145.

376 Lundvall, D. et al. 1999. Size-dependent predation in piscivores: interactions between predator foraging and prey avoidance abilities. - Can. J. Fish. Aquat. Sci. 56: 1285–1292.

378 Manica, A. 2002. Filial cannibalism in teleost fish. - Biol. Rev. Camb. Philos. Soc. 77: 261–277.

Maynard Smith, J. and Price, G. R. 1973. The logic of animal conflict. - Nature 246: 15–18.

380 McGurk, M. D. 1986. Natural mortality of marine pelagic fish eggs and larvae: role of spatial patchiness. - Mar. Ecol. Prog. Ser. 34: 227–242.

382 Mylius, S. D. and Diekmann, O. 1995. On evolutionarily stable life histories, optimization and the need to be specific about density dependence. - Oikos 74: 218–224.

384 Neuenfeldt, S. and Köster, F. W. 2000. Trophodynamic control of recruitment success in Baltic cod: the influence of cannibalism. - ICES J. Mar. Sci. 57: 300–309.

386 Neuheimer, A. B. et al. 2015. Adult and offspring size in the ocean over 17 orders of magnitude follows two life history strategies. - Ecology 96: 3303–3311.

388 Nilsson, K. A. et al. 2011. Guppy populations differ in cannibalistic degree and adaptation to structural environments. - Oecologia 167: 391–400.

390 Olsson, K. H. et al. 2016. Differences in density-dependence drive dual offspring size strategies in fish. - J. Theor. Biol. 407: 118–127.

392 Persson, L. 1987. The effects of resource availability and distribution on size class interactions in perch, Perca fluviatilis. - Oikos 48: 148–160.

394 Peterson, I. and Wroblewski, J. S. 1984. Mortality rate of fishes in the pelagic ecosystem. - Can. J. Fish. Aquat. Sci. 41: 1–4.

396 Polis, G. A. 1981. The evolution and dynamics of intraspecific predation. - Annu. Rev. Ecol. Syst. 12: 225–251.

Sih, A. 1982. Foraging strategies and the avoidance of predation by an aquatic insect, *Notonecta hoffmanni*. - Ecology. 63: 786-796.

Smith, C. C. and Fretwell, S. D. 1974. The optimal balance between size and number of offspring. - Am. Nat. 108: 499-506.

Smith, C. and Reay, P. 1991. Cannibalism in teleost fish. - Rev. Fish Biol. Fish. 1: 41–64.

Sparholt, H. 1994. Fish species interactions in the Baltic Sea. - Dana 10: 131–162.

Thygesen, U. H. et al. 2005. How optimal life history changes with the community size-spectrum. - Proc. R. Soc. B Biol. Sci. 272: 1323–1331.

Tsoukali, S. et al. 2016. Adult lifetime reproductive value in fish depends on size and fecundity type. - Can. J. Fish. Aquat. Sci. 73: 1405-1412.

Ursin, E. 1973. On the prey size preferences of cod and dab. - Meddr Danm. Fisk- og Havunders. 7: 85–98.

van den Bosch, F. et al. 1988. Cannibalism as a life boat mechanism. - J. Math. Biol. 26: 619–633.

van Tienderen, P. H. and De Jong. G. 1986. Sex ratio under the haystack model: Polymorphism may occur. - J. Theor. Biol. 122: 69-81.

von Bertalanffy, L. 1957. Quantitative laws in metabolism and growth. - Q. Rev. Biol. 32: 217–231.

Weeks, S. C. and Gaggiotti, O. E. 1993. Patterns of offspring size at birth in clonal and sexual strains of Poeciliopsis (Poeciliidae). - Copeia 1993: 1003–1009.

Werner, E. E. and Anholt, B. R. 1993. Ecological consequences of the trade-off between growth and mortality rates mediated by foraging activity. - Am. Nat. 142: 242-272.

West, G. B. et al. 2001. A general model for ontogenetic growth. - Nature 413: 628–631.

Table 1. Parameters used in the model.

Parameter	Unit	Values used in model	Description
w_0	g	Variable	offspring weight
w_m	g	Variable	adult weight
z	-	$0 < z < 1$ (variable)	relative offspring size
n	-	¾ (fixed, West et al. 2001)	metabolic scaling exponent
A	g^{1-n} / time	5 $g^{1/4}yr^{-1}$ (Fig. 1, otherwise non-specified)	growth constant
ε	-	$\varepsilon = 0.1$, $\varepsilon = 0.2$, $\varepsilon = 0.4$ (fixed)	reproductive efficiency
f_m	-	variable (non-specified)	adult lifetime fecundity
a	-	$0.1 \leq a \leq 0.8$ (variable, Kokkalis et al. 2017)	predation parameter
b	-	$0 \leq b$ (variable)	cannibalism parameter
β	-	100 (fixed, Ursin 1973)	preferred predator/prey size ratio
σ	-	1 (fixed, Ursin 1973)	narrowness of the selection function
C	-	≈ 0.41 (fixed)	constant defined by β and σ (Eq. 6)

Figure legends

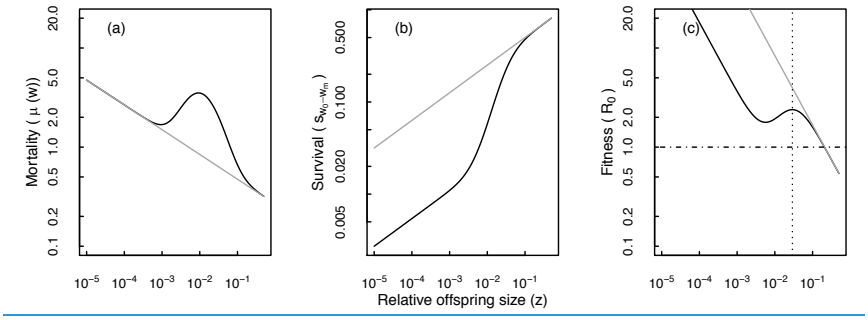


Fig 1

The effect of cannibalism according to the model on a) mortality at size ($\mu(w) = \mu_p(w) + \mu_c(w)$), b) survival to maturity ($s_{w_0 \to w_m}$), and c) lifetime fitness (R_0). Dashed vertical line shows the local large-z optimum. Parameter values are $a = 0.3$, $A = 5\text{g}^{1/4}\text{yr}^{-1}$, and $w_m = 1000\text{g}$. Grey lines correspond to $b = 0$ and black lines to $b = 3$.

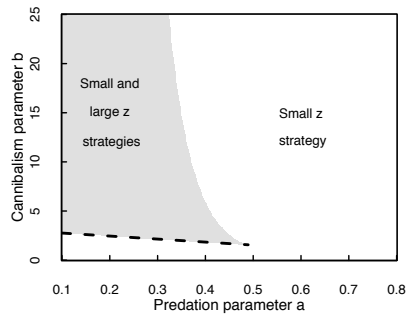
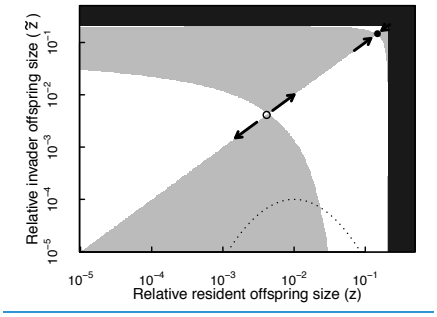


Fig 2

The combined influence of predation parameter a and cannibalism parameter b on optimal offspring size for reproductive efficiency $\varepsilon = 0.2$. Combinations of a and b resulting in selection for small offspring only is shown in white while combinations resulting in both a small offspring and a local, large offspring, optimum strategy are shown in grey. Dashed line shows $b = b_{min}$.



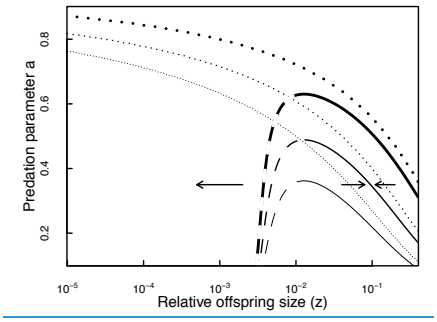
440 Fig 3

Pairwise invasibility plot showing the evolutionary outcome for density-dependent cannibalism.

442 Predation parameter $a = 0.3$ and reproductive efficiency $\varepsilon = 0.2$. Grey areas show $R_0(\tilde{z})>R_0(z)$, white areas $R_0(\tilde{z})<R_0(z)$ and black areas $R_0(z) < 1$. ESS points are identified by filled circles

444 while open circles show the evolutionary repeller and arrows the direction of selection. The dotted line shows the corresponding Ursin selection function.

446



448 Fig 4

Effect of predation parameter a on equilibrium strategies for three values of reproductive efficiency

450 ε (thin lines $\varepsilon = 0.1$, medium lines $\varepsilon = 0.2$, thick lines $\varepsilon = 0.4$). ESS and evolutionary repeller are indicated by solid and dashed lines, respectively. Arrows show the direction of selection for $\varepsilon = 0.2$.

452 Dotted lines show the corresponding limits for $R_0(z) = 1$.

454

Appendix A: The model with alternative mortality scaling

Juvenile mortality may scale differently from adult mortality, which would mean that juvenile survival decreases faster with decreasing size than has been assumed above. Formulating the model for a different scaling, juvenile predation mortality can be expressed as

$$\mu_p = aAw_m^{n-1}\left(\frac{w}{w_m}\right)^{n-1+d}$$

which approaches the expected size scaling for adults $n-1$ as size $w \to w_m$. The additional non-dimensional parameter d describes the relative difference in scaling between growth and mortality in juveniles. For $d = 0$, the model is the same as above. If $d \neq 0$, the survival function is given by

$$s_p = \exp\left[\frac{a}{d}((w_0/w_m)^d - 1)\right] \quad \text{(A.1)}$$

Using the relative offspring size $z = w_0/w_m$, fitness can be expressed as

$$R_0 = \exp\left[\frac{a}{d}(z^d - 1)\right]\exp[-bc\mathrm{H}(z)]\frac{\varepsilon}{2az} \quad \text{(A.2)}$$

and fitness of a given offspring size strategy z depends on reproductive efficiency ε, predation parameter a, the additional mortality scaling parameter d and the cannibalism parameter b. The derivative of Eq. A.2 is

$$\frac{\mathrm{d}R_0}{\mathrm{d}z} = \frac{\varepsilon s_p s_c}{2z^2}\left(z^d + \frac{bc}{\sigma a}\sqrt{\frac{2}{\pi}}\exp\left[-\left(\frac{\log(\beta z)+(n-1)\sigma^2}{\sigma\sqrt{2}}\right)^2\right] - \frac{1}{a}\right) \quad \text{(A.3)}$$

Optimal offspring size is given by setting $dR_0/\,dz = 0$. In the absence of cannibalism ($b = 0$) this becomes $z^* = a^{-1/d}$, i.e. not indefinitely small, contrary the model result above. For positive values of b, Eq. A.3 cannot be solved analytically, but there are three possible scenarios: 1) no effect of cannibalism, 2) two local optima representing a small-z and a large-z strategy with either the small-z or the large-z strategy as the global optimum and 3) a single optimum, determined by the combined values of a, d and b (Fig. A1a). For low values of a (or d close to 0), optimal offspring strategy $z^* = a^{-1/d}$ is much smaller than the size range targeted by cannibalism and two local

fitness optima will occur, provided that b is high enough, similar to when $d = 0$. Higher values of b have the potential to eliminate the viability of the small-size optimum at $z^* = a^{-1/d}$ as $R_0 < 1$, reducing the viable fitness curve to a single optimum. For higher values of a, z^* is within the target range of cannibalism, and the effect of higher cannibalism b will be to shift the optimum upwards.

As before, finding stable ESS requires calculating the first and second derivative of Eq. A.2 with respect to $\tilde{z}$. The corresponding pairwise invasibility plot (Fig. A1b) shows three equilibrium strategies z_{eq}, two of which are ESS but separated by an evolutionary repeller. The lower-z ESS corresponds to that of $z = a^{-1/d}$ (dotted line in Fig. A1b). Thus, different scaling of mortality will still allow cannibalism to select for either smaller or larger offspring size, depending on the initial conditions, where the offspring strategy is to seek refuge either above or below the targeted size range.

Figure legend

Fig. A1

Direction of selection on offspring size when juvenile mortality scales differently ($d = -0.15$). In a) the combined influence of predation parameter a and cannibalism parameter b: white indicates selection for small offspring only, light grey for two fitness optima with smaller z having higher fitness, medium grey for two fitness optima with larger z having higher fitness and dark grey for a single optimum for large z only. In b) the pairwise invasibility plot shows the evolutionary outcome of density-dependent cannibalism for $a = 0.3$ and $\varepsilon = 0.2$: grey areas show $R_0(\tilde{z})>\mathrm{R}_0(z)$ and white areas $R_0(\tilde{z})<\mathrm{R}_0(z)$, filled circles show ESS, open circles the evolutionary repeller, and arrows direction of selection. Dotted line is drawn for the smaller optimal offspring size ratio $z^* = a^{-1/d} \approx 0.00033$. In both, black areas indicate $R_0(z) < 1$.