



Cannibalism as a selective force on offspring size in fish

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18

Abstract

20
Cannibalism may cause considerable mortality on juvenile fish and it has been hypothesised that it
22 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
24 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
26 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-
28 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
30 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
32 describing cannibalistic intensity allowed within these boundaries is wide. On this basis, we suggest
that cannibalism is a potential mechanism for offspring size selection.

34

Introduction

36 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
38 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
40 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
42 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
44 size.

46 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
48 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
50 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
52 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
54 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
56 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
58 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

60 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
62 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
64 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
66 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
68 been put forward as a source of selection on offspring size (Weeks and Gaggiotti 1993, Nilsson et al.
2011).

70
To investigate the circumstances under which cannibalism can exercise selection on offspring size
72 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
74 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
76 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.

78
Lifetime fitness model

80 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
82 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
84 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
86 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
88 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):

90

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

92
$$g(w) = Aw^n \quad (1)$$

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

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

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

98 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
100 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
102 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 $w^{-0.25}$
104 (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
106 qualitatively change the outcome, as shown in Appendix A.

108 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
$$\mu_c(w) = b A w_m^{n-1} \theta(w/w_m) \quad (3)$$

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 $A w_m^n$. Therefore, consistent with empirical studies (e.g. Charnov et al. 2001), the rate of
 reproduction scales with size at maturity as $R_m = \varepsilon A w_m^n$ 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 $s_{w_0 \rightarrow w_m}$ multiplied with adult reproductive output f_m : $R_0 =$
 132 $s_{w_0 \rightarrow w_m} f_m$. 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 $1/2$ to account for an even sex ratio:

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

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

138 Survival is calculated by integrating the ratio between mortality $\mu(w)$ and growth $g(w)$ rates from size at birth to size at maturity, $s_{w_0 \rightarrow w_m} = \exp\left[-\int_{w_0}^{w_m} \mu(w)/g(w) dw\right]$ (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, $s_{w_0 \rightarrow w_m} = s_p s_c$.

142 Inserting growth (Eq. 1) with predation mortality (Eq. 2) gives survival due to predation s_p as

$$144 \quad s_p = \left(\frac{w_0}{w_m}\right)^\alpha \quad (5)$$

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

$$146 \quad s_c = \exp[-bcH(w_0, w_m)] \quad (6)$$

with

$$148 \quad c = \beta^{n-1} \sigma \sqrt{\frac{\pi}{2}} \exp\left[\frac{\sigma^2(n-1)^2}{2}\right], \text{ and}$$

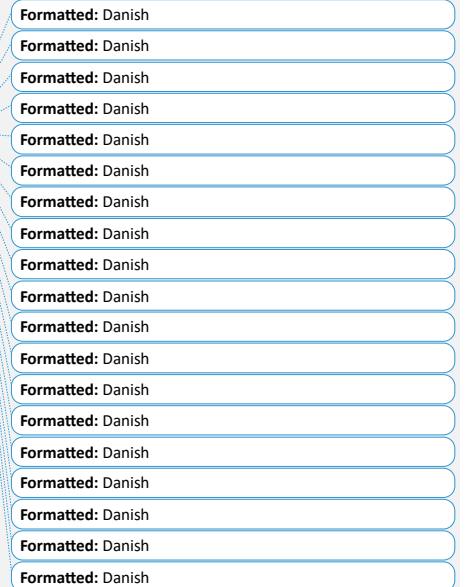
$$H(w_0, w_m) = \operatorname{erf}\left[\frac{\log(\beta) + (n-1)\sigma^2}{\sigma\sqrt{2}}\right] - \operatorname{erf}\left[\frac{\log(\beta(w_0/w_m)) + (n-1)\sigma^2}{\sigma\sqrt{2}}\right]$$

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

$$152 \quad R_0 = \left(\frac{w_0}{w_m}\right)^\alpha \exp[-bcH(w_0, w_m)] \varepsilon \frac{w_m}{2\alpha w_0} \quad (7)$$

The growth parameter A disappears from the survival function and the survival depends only on the non-dimensional mortality parameters α 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



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
$$R_0 = z^{\alpha-1} \exp[-bcH(z)] \frac{\varepsilon}{2a} \quad (8)$$

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 (Mylus 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

184 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, $dR_0/dz = 0$:

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

186 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
188 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
190 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
192 determined by analysing the expression inside the brackets. The local optimum for the large- z
strategy is given by

$$194 \quad z^* = \frac{1}{\beta} \exp \left[\sigma \sqrt{\log(2b^2 c^2 / (\pi \sigma^2 (1-a)^2)) + (1-n)\sigma^2} \right] \quad (10)$$

which is only defined if

$$196 \quad b \geq b_{\min} = \sqrt{\pi \sigma^2 (1-a)^2 / (2c^2)} \quad (11)$$

198 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
200 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
202 (Eq. 11, b_{\min} dashed line in Fig. 2; the range of a is assumed to be $0.1 < a < 0.8$).

204 **Density-dependent cannibalism**

Next, we will assume that all density-dependence is caused by cannibalism and try to locate
206 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:

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

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 cannot (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

232 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).

234

The pairwise invasibility plot in Fig. 3 indicates the possibility of two evolutionarily stable
236 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)
238 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
240 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
242 than the evolutionary repeller, larger offspring size will reduce fecundity without a sufficient gain in
survival, and selection will favour ever smaller z .

244

The relative offspring size at ESS and the evolutionary repeller depends on the predation parameter
246 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
248 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
250 large offspring size ESS disappears along with the evolutionary repeller. Increasing the
reproductive efficiency increases the number of surviving juveniles, which leads to higher
252 cannibalism (consistent with the observation that cannibalism increases with larger stock size
(Jensen and Sparholt 1992)). Increasing reproductive efficiency can thus compensate for increased
254 mortality and make the ESS appear at higher mortalities.

256 **Discussion**

A key life history trade-off is that between offspring size and offspring numbers, typically
258 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
260 supported by empirical findings (Elgar 1990, Blackburn 1991). Considering only this trade-off, in
conjunction with metabolic scaling of growth and mortality, leads to an optimal offspring size that
262 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
264 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
266 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
268 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
270 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
272 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
274 exist: small offspring size and large offspring size, where only the large offspring size strategy is
driven by cannibalism.

276
In the case of marine animals in general and fish in particular, there is evidence for two distinct
278 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
280 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
282 makes cannibalism a possible contributor to this offspring size strategy.

284 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
286 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
288 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
290 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
292 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
294 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
296 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-
298 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
300 (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
302 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
304 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

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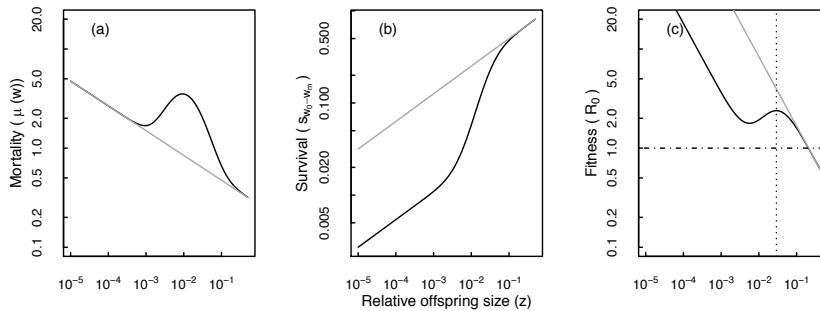
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Table 1. Parameters used in the model.

| 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 | - | $\frac{3}{4}$ (fixed, West et al. 2001) | metabolic scaling exponent |
| A | $\text{g}^{1-n} / \text{time}$ | $5 \text{ g}^{1/4} \text{yr}^{-1}$ (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) |

422

424 **Figure legends**

426 Fig 1

The effect of cannibalism according to the model on a) mortality at size ($\mu(w) = \mu_p(w) + \mu_c(w)$),
428 b) survival to maturity ($s_{w_0 \rightarrow w_m}$), 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
430 correspond to $b = 0$ and black lines to $b = 3$.

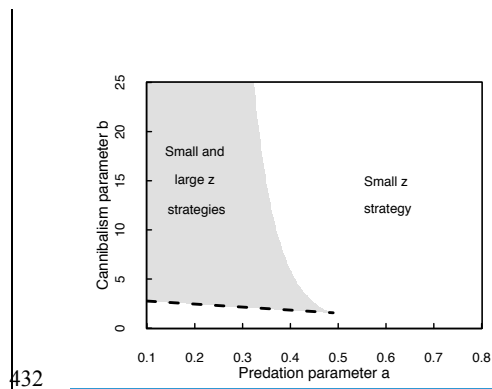
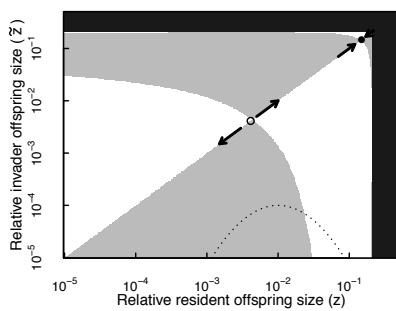


Fig 2

434 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
436 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 $b = b_{min}$.

438



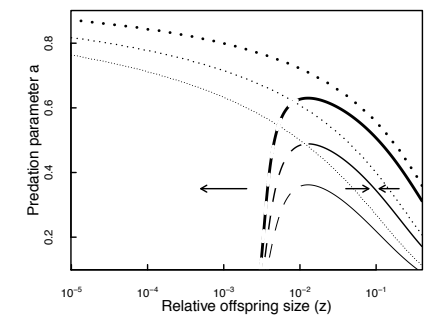
440 Fig 3

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 $R_0(\bar{z}) > R_0(z)$,
white areas $R_0(\bar{z}) < R_0(z)$ 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

456 **Appendix A: The model with alternative mortality scaling**

458 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
460 for a different scaling, juvenile predation mortality can be expressed as

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

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

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

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

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

468 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
470 derivative of Eq. A.2 is

$$\frac{dR_0}{dz} = \frac{\varepsilon s_p s_c}{2z^2} \left(z^d + \frac{bc}{\sigma a} \sqrt{\frac{2}{\pi}} \exp\left[-\left(\frac{\log(\beta z) + (n-1)\sigma^2}{\sigma\sqrt{z}}\right)^2\right] - \frac{1}{a} \right) \quad (\text{A.3})$$

472 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
474 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-
476 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
478 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
480 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
482 range of cannibalism, and the effect of higher cannibalism b will be to shift the optimum upwards.

484 As before, finding stable ESS requires calculating the first and second derivative of Eq. A.2 with
respect to \bar{z} . The corresponding pairwise invasibility plot (Fig. A1b) shows three equilibrium
486 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
488 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
490 range.

492 **Figure legend**

Fig. A1

494 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
496 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
498 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 $R_0(\bar{z}) > R_0(z)$ and
500 white areas $R_0(\bar{z}) < R_0(z)$, 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 $z^* =$
502 $a^{-1/d} \approx 0.00033$. In both, black areas indicate $R_0(z) < 1$.