

## Research

### The importance of variation in offspring body size for stability in cannibalistic populations

Jan Ohlberger, Øystein Langangen, Ian J. Winfield and Yngvild Vindenes

J. Ohlberger (<http://orcid.org/0000-0001-6795-240X>) ✉ ([janohl@uuw.edu](mailto:janohl@uuw.edu)), Ø. Langangen and Y. Vindenes (<https://orcid.org/0000-0003-1197-5818>), Centre for Ecological and Evolutionary Synthesis, Dept of Biosciences, Univ. of Oslo, PO Box 1066 Blindern, NO-0316 Oslo, Norway. JO also at: School of Aquatic and Fishery Sciences, Univ. of Washington, Seattle, WA, USA. – I. J. Winfield, Lake Ecosystems Group, Centre for Ecology and Hydrology, Lancaster Environment Centre, Bailrigg, Lancaster, Lancashire, UK.

Oikos

00: 1–11, 2019

doi: 10.1111/oik.05507

Subject Editor: Bruno Ernande

Editor-in-Chief: Dries Bonte

Accepted 13 August 2019

Animals exhibit remarkable intraspecific variation in phenotypic traits such as body size. Understanding how such trait variation affects population and ecosystem dynamics is critically important, because future environmental change and human impacts are expected to alter phenotypic trait distributions. In species with seasonal reproduction, offspring size variation within cohorts is ubiquitous, yet we know little about its implications for population stability. In addition, long-term monitoring data indicate that changes in offspring size variation occur at ecologically relevant time scales. Here, we study the consequences of changing offspring size variation by developing and analysing an integral projection model (IPM). Our model accounts for size-dependent cannibalism as well as additional density regulation occurring during the first year. The model is parameterized using literature values and long-term monitoring data for pike *Esox lucius*, a common fish predator in temperate freshwater ecosystems, but the general model structure applies to a wide range of size-structured organisms. Our analyses demonstrate that a wide size distribution of offspring promotes stable dynamics, whereas narrow distributions can be destabilizing because cannibalism increases the annual variation in mean offspring mortality. Our results indicate that the stabilizing effect of offspring size variation is likely an important property of size-structured organisms with seasonal reproduction and cannibalistic behaviour. This work highlights the importance of intracohort trait variation and describes how variation in body size can shape the dynamics of animal populations.

Keywords: integral projection model, intraspecific interactions, trait variation

#### Introduction

Most animal populations exhibit large amounts of variation in phenotypic traits, because individuals differ in their genetic makeup, behavioural strategies and experienced environmental conditions (Ebenman and Persson 1988, Bolnick et al. 2003). The role of intraspecific trait variation in shaping ecological and evolutionary processes at the species and community levels has recently received increased attention (Bolnick et al. 2011, Dall et al. 2012, de Roos and Persson 2013, Vindenes and

Langangen 2015, Hart et al. 2016). While the effects of individual variation (e.g. variation in body size, resource partitioning or variation arising from ontogenetic development) on the stability of populations have previously been investigated (Łomnicki 1988, DeAngelis et al. 1993, Bjørnstad and Hansen 1994, Claessen et al. 2000, van Kooten et al. 2010), we still have limited understanding of the population dynamical consequences of initial trait variation within cohorts, i.e. groups of similar-aged individuals (but see van Kooten et al. 2004).

Intracohort variation in offspring body size is ubiquitous in populations that exhibit discrete reproductive periods, i.e. most species in seasonal environments (Uchmanski 1985, Einum and Fleming 2002, Pfister and Stevens 2002). Moreover, long-term ecological monitoring data suggest that significant changes in offspring size distributions occur over ecologically relevant time periods. For instance, empirical data suggest decreasing variation in offspring size for well-studied freshwater (e.g. pike *Esox lucius*: Supplementary material Appendix 1) and marine (e.g. Atlantic cod *Gadus morhua*: Olsen et al. 2009) fish populations. Understanding how trait variation affects the dynamics of populations and ecosystem functioning is a fundamental challenge in ecology that is becoming increasingly important due to intensified human impacts and altered environmental conditions that may cause widespread changes in phenotypic trait distributions (Moran et al. 2015).

Various ecological processes contribute to variation in offspring size. Potential mechanisms include 1) genetic variation, 2) social structure, e.g. resource monopolization, 3) maternal effects, 4) small-scale heterogeneity in environmental conditions, 5) variation in the time of hatching or emergence and 6) random events such as disease outbreaks (Johnston and Leggett 2002, Pfister and Stevens 2002, Huss et al. 2007, Peacor et al. 2007, Rasmussen and Rudolf 2015). Producing offspring of variable size may also constitute a form of bet-hedging, i.e. an adaptation that buffers reproductive success against unpredictable environments (Philippi and Seger 1989, Einum and Fleming 2002, Marshall et al. 2008). Increased environmental variability due to climate change may indeed favour differential investment and consequently higher size variation among offspring. Prolonged or contracted reproductive periods due to climate-induced phenological change are also expected to affect offspring size distributions, which depend on the length of the reproductive season (Keast and Eadie 1984, Rasmussen and Rudolf 2015). Shorter reproductive periods may be caused by truncated parental size distributions due to size-selective removal (Wright and Trippel 2009). While many processes that contribute to variation in offspring body size have been identified, its consequences for the dynamics of populations have received less attention. Variation in offspring size could be an important driver of population dynamics, because it affects the ecological interactions among individuals such as intraspecific predation (cannibalism) and competition which depend on body size.

Understanding the broader implications of changes in size variation for population stability requires a framework that accounts for continuous size-structure and incorporates size-dependent interactions. Integral projection models (IPMs) provide such a framework, by linking individual-level trait-dependent demographic processes and ecological interactions to population-level dynamics (Easterling et al. 2000, Ellner et al. 2016). Other models such as physiologically structured population models (de Roos et al. 1992) meet these requirements and have been used to study size-based interactions within populations, including cannibalism (Claessen et al. 2000). Integral projection models are discrete time models that belong to the same class as matrix models, and therefore share their analytical advantages (Ellner and Rees 2006). The dynamics of the trait structure are determined by the main vital rate functions that describe how survival, growth, reproduction and the initial state distribution of offspring depend on the underlying state variable(s). These functional relationships can be determined from data using regression methods. IPMs provide a powerful data-driven framework for studying the ecological (and evolutionary) dynamics of populations (Coulson 2012, Vindenes and Langangen 2015, Ellner et al. 2016). In recent years several extensions have been made to increase the range of applications of IPMs, including the effects of climate change (Simmonds and Coulson 2014, Vindenes et al. 2014, 2016), yet the majority of applications so far ignore trait-based interactions among individuals (but see Bassar et al. 2016). The incorporation of such interactions thus represents a great potential for new applications of the framework both for theoretical and empirical investigations. A few IPM applications have incorporated size-based competition (Bassar et al. 2016), but intraspecific predation, i.e. cannibalism, has to our knowledge not been studied within this framework.

Cannibalism and competition are complex intraspecific interactions that affect processes such as growth and survival. Both types of interaction can alter the size distribution within cohorts (Huss et al. 2007, 2008, 2010), and their effects on population dynamics may depend on hatchling size (van Kooten et al. 2010). Cannibalism affects individual growth and size-dependent survival because cannibals and victims typically differ in body size yet may compete (at least in part) for shared resources. In particular, cannibalism often has a large impact on the survival of victims though size-dependent predation. Cannibalistic behaviour is a common phenomenon found in all major animal taxa in aquatic and terrestrial systems (e.g. protozoa, arthropods, gastropods, sharks, bony fishes, amphibians, reptiles, birds and mammals), and is known to constitute a major cause of mortality in many species, especially among early life-stages (Fox 1975, Polis 1981). Cannibalism is an inherently size-dependent interaction that has been widely studied in the theoretical literature and has been shown to affect population and community dynamics (Briggs et al. 2000, Persson et al. 2003, Claessen et al. 2004, Rudolf et al. 2007, Huss et al. 2010). However, knowledge of how population stability in

cannibalistic species depends on the size variation among offspring is lacking. We therefore developed an IPM that incorporates size-dependent cannibalism to study how size variance in offspring (here: one-year-old fish) affects population dynamics and demography. We parameterized the model for pike, a freshwater top-predator known to show cannibalistic behaviour. However, the model can easily be adapted to other size-structured organisms with other kinds of trait-based interactions (e.g. competition), and we demonstrate that our main result, the stabilizing effect of offspring size variation, is valid across a wide range of conditions.

## Methods

### Model description

#### Baseline IPM

For simplicity, we first describe a basic IPM of a population that is structured according to a continuous state variable  $x$ , here size (length in cm). In the next section we extend the model to a density-dependent model including size-dependent cannibalism. We consider a female-based model with annual time steps. The size distribution of individuals at time  $t$  is  $\mathbf{n}_t = n_t(x)$ , so that the total population size is  $N_t = \int_0^\infty n_t(x) dx$ . Without density dependence, the change in the size distribution from one year to the next is given by

$$n_{t+1}(x') = \int_0^\infty [s(x)g(x';x) + b(x)f(x';x)]n_t(x) dx$$

where for an individual of current size  $x$ ,  $s(x)$  is the annual survival probability,  $g(x';x)$  represents growth (the distribution of next year's size  $x'$ ),  $b(x)$  is the number of offspring produced that survive until next year's population (pre-reproductive) census, and  $f(x';x)$  is the distribution of offspring size  $x'$  as they enter the population next year, potentially depending on the parent's size  $x$ . Together, these four vital rates determine the projection kernel, which is equivalent to the projection matrix in matrix models (Easterling et al.

2000), and each vital rate can be decomposed further into underlying processes. We have extended the baseline IPM in two main ways, to incorporate 1) size-dependent cannibalism, which can potentially affect any vital rate, and 2) additional density-dependent feedbacks occurring in the first year of life (typical for fish life histories), regulating survival and growth during the first year. We describe these extensions below where each vital rate is defined in more detail. The sequence of annual life-history events is illustrated in Fig. 1. An overview of model functions is provided in Table 1, and model parameters and baseline values are provided in Table 2. In the following notation, density- and size-dependent functions have a subscript  $t$ .

#### Cannibalism kernel

Size-dependent cannibalism has previously been studied with continuous-time models, and we will largely follow the general processes and terminology defined by Claessen et al. (2000) although simplified and adapted to a discrete time IPM. In the following,  $y$  denotes cannibal size, while  $x$  refers to victims (note that the same individual can be both a cannibal, preying on smaller individuals and a victim if preyed upon by larger ones). We define a cannibalism kernel  $C_{cn}(x,y)$  that describes the distribution of potential prey sizes for each cannibal size  $y$ , i.e. the likelihood that an individual of size  $x$  is in the diet range of the cannibal ( $\int_0^\infty C_{cn}(x,y) dx = 1$ ). This kernel can be defined in several ways, depending on the life history and behaviour of the organism. We assume that the victim to cannibal length ratio  $x/y$  follows a lognormal distribution  $LN(x/y, \mu_{cn}, \sigma_{cn})$  with scale parameter  $\sigma_{cn}$  and location parameter  $\mu_{cn}$ . The cannibalism kernel is given by the normalized function  $C_{cn}(x,y) = LN(x/y, \mu_{cn}, \sigma_{cn})/y$  (where  $\int_0^\infty LN(x/y, \mu_{cn}, \sigma_{cn}) dx = y$ ). This implies that the range of potential victim sizes (cannibalism window) increases with cannibal size (Fig. 2a). Claessen et al. (2000) used a tent function with a similarly increasing cannibalism window with size. This kernel can be incorporated in the definition of any vital rate function to capture effects of

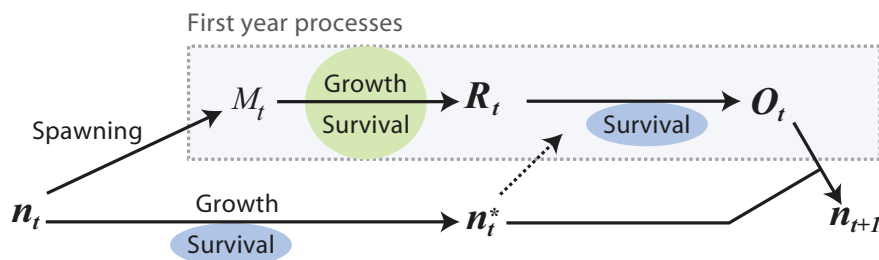


Figure 1. Timing of annual life-history events.  $\mathbf{n}_t$  is the population size distribution at time  $t$ ,  $M_t$  is the total number of eggs produced by the population,  $\mathbf{R}_t = R_t(x)$  is the population distribution of offspring before intercohort cannibalism occurs,  $\mathbf{O}_t = O_t(x)$  is the population distribution of age-1 offspring entering the next census, and  $\mathbf{n}_t^* = n_t^*(x)$  is the population distribution after density dependent survival and growth have occurred. The green colour indicates size-independent density regulation among offspring, while the blue indicates size-dependent cannibalism affecting the survival of both offspring and older individuals. Solid arrows indicate the sequence of annual events and the dashed arrow indicates an interaction.

Table 1. Overview of variables in the IPM.

Variable	Explanation
$x, y$	State variable of size at time of census, for victims and cannibals
$x'$	Next year's size (before census) or offspring size
$t$	Time
$n_t = n_t(x)$	Population size distribution at time $t$
$n_t^*(x')$	Population size distribution after growth and survival
$R_t(x')$	Offspring population size distribution after first density regulation
$O_t(x')$	Offspring distribution after intercohort cannibalism at age 1
$m(x)$	Fecundity (average egg number)
$p_m(x)$	Probability of maturity
$s_t(x)$	Survival probability from $t$ to $t+1$ , depending on $n_t(x)$
$s_t^*(x)$	Survival probability from $t$ to $t+1$ , depending on $n_t^*(x)$
$g(x'; x)$	Growth distribution (lognormal)
$\mu_g(x)$	Mean of $x'$ after growth (non-offspring), following a von Bertalanffy model
$\sigma_g^2(x)$	Conditional variance of $x'$ after growth, given current size $x$
$f_t(x')$	Offspring length distribution (lognormal) at age 1, depending on total egg number $M_t$
$\mu_{L1,t}$	Mean of $x'$ in offspring, depending on total egg number $M_t$
$\sigma_{L1}^2$	Variance in offspring size at age 1

cannibalism. Here, we assume that cannibalism mainly affects survival of the victims and that for the modelled population cannibals have alternative prey whenever smaller conspecifics are not available, i.e. growth is independent of any single food source. This assumption applies to opportunistic predators that feed on several species of alternative prey, such as pike in Windermere, UK (Winfield et al. 2012).

#### Survival including size-dependent cannibalism

In addition to the cannibalism kernel defined above, the effects of cannibalism depend on the cannibal attack rate

(Claessen et al. 2000). For a cannibal of size  $y$ , the relative attack rate on victims of size  $x$  is given by  $\beta_{cn} y^{\alpha_{cn}} C_{cn}(x, y)$ , where the parameter  $\beta_{cn}$  defines the overall cannibalism intensity, and  $\alpha_{cn}$  ( $\alpha_{cn} < 1$ ) scales this intensity to cannibal size  $y$ . Thus, the annual encounter rate of a cannibal with potential victims is given by

$$\gamma_{cn,t}(y) = \int_0^{\infty} \beta_{cn} y^{\alpha_{cn}} C_{cn}(x, y) n_t(x) dx$$

The overall mortality risk of an individual of size  $x$  due to cannibalism also depends on the size distribution of cannibals

Table 2. Overview of model parameters and baseline values.

Process	Parameter	Description	Value	Units
Background survival	$\alpha_{sb}$	size at maximum survival	80	mm
	$s_{b0}$	maximum survival	0.85	–
	$\beta_{sb}$	decline in survival at smaller and larger sizes	–0.0005	mm <sup>-2</sup>
Cannibalism survival	$\mu_{cn}$	location parameter	–1.5	mm mm <sup>-1</sup>
	$\sigma_{cn}$	scale parameter	0.3	mm mm <sup>-1</sup>
	$\beta_{cn}$	cannibalism intensity	0.01	mm <sup>–<math>\alpha_{cn}</math></sup>
	$\alpha_{cn}$	intensity size exponent	0.6	–
	$\delta_{cn}$	type of functional response	0.1	–
von Bertalanffy growth function	$L_{\infty}$	asymptotic average length	109	mm
	$K$	growth rate coefficient	0.21	–
Variance in growth	$\nu_g$	growth variance exponent	–0.015	–
	$\tau_g$	growth variance scalar	5	–
Maturation	$\mu_p$	size at 50% maturation probability	41.5	mm
	$\sigma_p$	width of maturation probability function	0.5	–
Fecundity	$\alpha_m$	fecundity constant	0.095	ind mm <sup>–<math>\beta_m</math></sup>
	$\beta_M$	fecundity exponent	3.3	–
Offspring density	$\alpha_R$	maximum per capita recruitment	$4 \times 10^{-4}$	ind ind <sup>-1</sup>
	$\beta_R$	determines carrying capacity	$1 \times 10^{-8}$	ind
Offspring size distribution	$\beta_{OL1}$	constant of mean offspring size depending on egg density	3.85	ln(mm)
	$\beta_{ML1}$	exponent of mean offspring size depending on egg density	0.04	ln(mm)

ind = individuals.

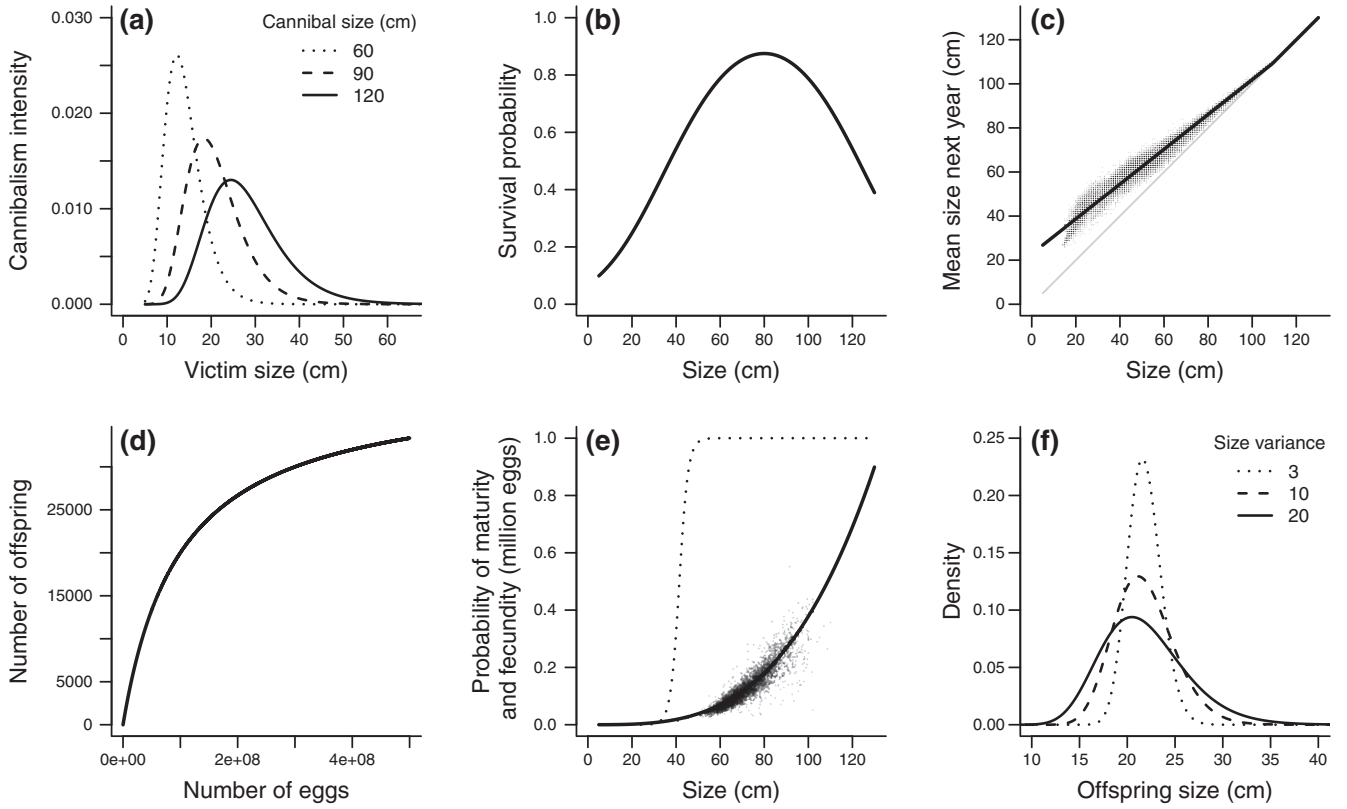


Figure 2. Basic model functions. (a) Size-dependent cannibalism kernel for different cannibal sizes, (b) background survival probability with no cannibalism, (c) mean length next year (black) and zero growth line (grey), (d) number of offspring (age 1) as a function of egg number, (e) probability of maturity (dotted line) and fecundity (solid line) as a function of size and (f) the offspring size distribution (at age 1) for different values of size variance. Back-calculated length data (c) and fecundity data (e) for Windermere pike are also shown (filled circles).

and the kind of functional response shown by the cannibals, given by

$$\omega_{cn,t}(x) = \int_0^{\infty} \frac{\beta_{cn} y^{\alpha_{cn}} C_{cn}(x, y) n_t(y)}{1 + \delta_{cn} \gamma_{cn,t}(y)} dy$$

Here, the parameter  $\delta_{cn}$  determines the functional response of the cannibal, where  $\delta_{cn} = 0$  yields a type I response (Holling 1959), i.e. victim population density does not restrict cannibals, and  $\delta_{cn} > 0$  yields a type II response, i.e. cannibalism mortality approaches a maximum at high victim densities. If cannibalism is the only source of mortality, the survival probability of a size  $x$  victim is  $s_{cn,t}(x) = \exp(-\omega_{cn,t}(x))$ . However, other sources of mortality are also likely to be present, such as predation from other species, diseases and starvation. Here we include another term capturing this background mortality, and for the pike model we assume it is density independent but non-linear so that small and very large (old) individuals have a higher background mortality than intermediate sized ones (Fig. 2b):  $s_b(x) = (2s_{b0}) / (1 + e^{\beta_{sb}(x - \alpha_{sb})^2})$ . The initial increase in background survival with size is assumed to reflect a reduction in starvation probability and interspecific predation, while the decrease for very large

individuals reflects fishing mortality and senescence related to increasing risk of infection with parasites and other diseases (Haugen et al. 2007). Including both sources of mortality, the survival function becomes:

$$s_t(x) = s_b(x) s_{cn,t}(x)$$

### Growth

Conditional on the current length  $x$ , next year's length  $x'$  follows a lognormal distribution  $g(x'; x)$ , with a mean  $\mu_g(x)$  according to a von Bertalanffy growth function, and a variance  $\sigma_g^2(x)$  in the growth increments that declines exponentially with size, i.e.  $\sigma_g^2(x) = \tau_g^2 e^{-2\nu_g x}$ . This implies that the unconditional variance in size at age increases early in life up to age-3 and thereafter decreases. We assume constant food availability and growth, though food-dependent growth is accounted for in the extended model that includes effects of competition (Supplementary material Appendix 3). Mean length next year (on log scale), given current length  $x$ , is  $\mu_g(x) = \ln[KL_{\infty} + (1 - K)x]$ . Here,  $K$  is the von Bertalanffy growth rate, and  $L_{\infty}$  is asymptotic length. We require  $\mu_g(x) > x$ , and otherwise we set  $\mu_g(x) = x$ , i.e. the

expected growth rate cannot be negative (Fig. 2c). After growth and survival, population size without offspring is given by  $n_t^*(x') = \int_0^\infty n_t(x) s_t(x) g(x'; x) dx$ .

### Reproduction and first year processes

Let  $M_t$  denote the total number of eggs the population can produce in year  $t$ . Multiple density-dependent processes may contribute to reduce this number until the resulting offspring are counted at age 1, including parental competition for reproduction sites, as well as competition, predation and disease affecting individuals during their first year of life. To capture all of these processes we included a general model for density dependence for egg production and during the first year after eggs are produced. Letting  $f_t(x')$  denote the size-distribution of offspring at age 1, the total number of offspring after this density regulation is given by

$$R_t(x') = f_t(x') \frac{\alpha_R M_t}{1 + \beta_R M_t}$$

where  $\alpha_R$  is the slope at origin (i.e. number of offspring resulting from very low egg numbers), and  $\beta_R$  is a capacity parameter such that  $\alpha_R/\beta_R$  is the maximum number of offspring (Fig. 2d). Before entering next year's population, the offspring number can be further reduced by size-dependent cannibalism by the rest of the population (Fig. 1). This inter-cohort cannibalism during the first year is assumed to occur after growth (as determined by  $f_t(x')$ ), but before the next census, thus the population of potential cannibals of the offspring is given by  $n_t^*(x')$  (Fig. 1). This model simplification is reasonable when hatchlings are too small to be effectively predated by older conspecifics, or when they are spatially segregated from later life stages (Pereira et al. 2017), for instance due to association of young fish with vegetation, as found in pike (Bry 1996). The number of offspring that enter the next census is then given by  $O_t(x') = R_t(x') s_{cn,t}^*(x')$ , where  $s_{cn,t}^*(x')$  represents the survival of offspring after cannibalism by  $n_t^*(x')$ , and next year's population distribution is  $n_{t+1}(x') = n_t^*(x') + O_t(x')$ .

Looking into each of the components of  $R_t(x')$  in more detail, the total number of eggs produced is given by  $M_t = 0.5 \int_0^\infty n_t(x) m(x) p_m(x) dx$ , where  $m(x)$  is the fecundity (mean number of eggs) of a female of length  $x$ , and  $p_m(x)$  is the probability of being mature at size  $x$  (the factor 0.5 reflects the assumption of a 1:1 sex ratio). Probability of maturity  $p_m(x)$  is assumed to follow a sigmoid function (Fig. 2e) with  $p_m(x) = 1 / (1 + e^{-\sigma_p(x - \mu_p)})$ , where  $\mu_p$  is mean size at maturation and  $\sigma_p$  determines the slope on the logit scale. Fecundity  $m(x)$  is assumed to follow a power function (Fig. 2e)  $m(x) = \alpha_m x^{\beta_m}$ , where  $\alpha_m$  is a constant and  $\beta_m$  is a size-scaling exponent.

The length distribution of offspring  $f_t(x')$  is assumed to be independent of parental length, and to follow a lognormal distribution with a constant variance on linear scale  $\sigma_{L1}^2$

(a key parameter to be varied in the model analysis) and a mean  $\mu_{L1,t}$ . This parameter depends on the total egg number:  $\ln \mu_{L1,t} = \beta_{0L1} - \beta_{ML1} \ln M_t$  (Vindenes et al. 2016), and thus captures effects of density dependence on growth during the first year (see also Supplementary material Appendix 4).

### Model analysis: changing the variance in offspring size distribution

We used this model to analyse population dynamics over a large range of variances in offspring size  $\sigma_{L1}^2$  (Fig. 2f). The effect on population stability was investigated using bifurcation analysis, which was performed by running the IPM for each discrete variance value to record the population size distribution projected over 1000 time steps. Population size for the last 100 time steps was plotted against the variance in offspring length to assess population stability (a population at equilibrium is characterized by a single population size, whereas unstable dynamics, i.e. with cyclic or chaotic behaviour, are represented by multiple population sizes).

To account for uncertainty in parameter values, we explored a broad range of values for other key parameters in the model as part of our sensitivity analysis, including mean offspring size ( $\mu_{L1}$ ) the strength of cannibalism ( $\beta_{cn}$ ), and growth variation later in life ( $\tau_p$ ). The entire analysis was repeated for a model including size-dependent competition in addition to cannibalism, to confirm the robustness of our main conclusion (Supplementary material Appendix 3). All analyses were performed in R ver. 3.3.2 (<www.r-project.org>).

### Model parameterization

As detailed below, model parameters were based on literature values and data from a long-term monitoring program for pike in the lake of Windermere, UK (Le Cren 2001, Winfield et al. 2013a, b, 2015, Vindenes et al. 2014). This dataset contains measurements of length, age, sex and body mass of individual pike collected over a period of 50 years (1946–1995), including estimates of length-at-age that were back-calculated using opercular bones, as well as estimates of the number of eggs per female. Associated diet data show that pike in Windermere predate a range of species in addition to conspecifics, including Arctic charr *Salvelinus alpinus*, brown trout *Salmo trutta*, perch *Perca fluviatilis* and roach *Rutilus rutilus* (Winfield et al. 2012).

### Survival

Parameters of the background survival function were set to  $\alpha_{sb} = 80$ ,  $\beta_{sb} = -0.0005$  and  $s_{b0} = 1.7$  (Fig. 2b). The location and scale parameters of the cannibalism kernel were set to  $\mu_{cn} = -1.5$  and  $\sigma_{cn} = 0.3$  (minimum cannibal size was 5 cm), such that the relative sizes of preferred prey in all size classes agreed with literature values (Mittelbach and Persson 1998, Persson et al. 2006, Fig. 2a). Reported lower and upper limits for the victim-to-cannibal size ratio in pike are 0.03 and 0.55 (Persson et al. 2006). The scaling parameter of the maximum cannibalistic attack rate was set

to  $\sigma_{cn} = 0.6$  (Claessen et al. 2000) and we used  $\delta_{cn} = 0.1$  for a type II functional response, the most frequently observed functional form (Begon et al. 2006). Cannibalistic voracity was set to  $\beta_{cn} = 0.01$  to reflect reasonable mortality rates. This parameter could not be estimated from our data or taken from the literature and was thus varied as part of the model analysis.

### Growth

von Bertalanffy growth parameters  $K = 0.21$  and  $L_\infty = 109$  cm were estimated from data on Windermere pike (Fig. 2c). The variance in growth, which declines exponentially with size according to the empirical data (Vindenes et al. 2014), was modelled using  $\nu_g = -0.015$  and  $\tau_g = 5$ . We considered a size range of 5–130 cm. For the numerical calculations, we used 500 mesh points for the continuous state variable, i.e. 500 size classes with a size difference of  $\sim 0.25$  cm.

### Reproduction

The maturation parameters were set to  $\mu_p = 41.5$  and  $\sigma_p = 0.5$  to match data from Windermere where female pike first spawn at  $\sim 31$ –50 cm (Fig. 2e, Frost and Kipling 1967). The size–fecundity relationship was also estimated from empirical data from Windermere. Estimates of the intercept and slope of the log–log relationship between the number of eggs and body length were  $\alpha_m = 0.095$  and  $\beta_m = 3.3$  (Fig. 2e). In the absence of robust empirical data, it is assumed that newly hatched offspring experience density regulation prior to the first census. The parameters of the asymptotic relationship were set to  $\alpha_R = 4 \times 10^{-4}$  and  $\beta_R = 1 \times 10^{-8}$  (Fig. 2d). Finally, the offspring size distribution was assumed to follow a lognormal distribution (in line with the data), where mean length depends on the total number of eggs according to an exponential decrease with parameters  $\beta_{OL1} = 3.85$  and  $\beta_{ML1} = 0.04$ .

### Data deposition

Data have been published in three datasets that are available online at: <<http://dx.doi.org/10.5285/637d60d6-1571-49af-93f7-24c1279d884d>>, <<http://dx.doi.org/10.5285/813e07dd-2135-49bc-93c6-83999e442b36>>, <<http://dx.doi.org/10.5285/4b255fc4-33d3-4beb-a238-37e1a8cf32a2>> (Winfield et al. 2013a, b and 2015).

## Results

Changes in offspring size variance have strong and consistent effects on population stability (Fig. 3). Population dynamics are stable at wide offspring size distributions, but unstable at narrow size distributions (see Fig. 2f for reference). The unstable dynamics at low variances alternate between cyclic fluctuations, as reflected by distinct recurring population densities, and irregular fluctuations in population size (Fig. 3). The range of population densities decreases with increasing size variance until the threshold is reached and the dynamics become stable. At low offspring size variance, the population

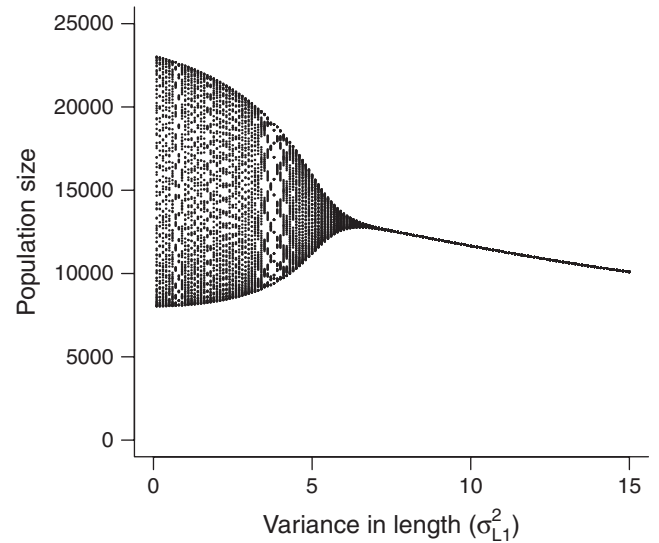


Figure 3. Bifurcation diagram. Shown is the population size as a function of the variance in offspring length ( $\sigma_{L1}^2$ ). Projections were run for 1000 time steps, and population size was sampled for the last 100 time steps.

exhibits oscillations that are not dampened over time, and a stable size distribution is not reached (Fig. 4a). Instead, the density of offspring that enter the population and consequently the densities of older cohorts both fluctuate (Fig. 4c), due to strongly varying probabilities of surviving cannibalism (Fig. 4e). In contrast, with high variance in offspring size the population reaches an equilibrium size (Fig. 4b). A stable size distribution is reached showing a size structure with distinct age-cohorts (Fig. 4d). In the stable case, survival probability of small individuals is constant and rather low due to high cannibalism, whereas survival probability of large individuals is relatively high and mostly determined by density-independent mortality (Fig. 4f; offspring survival rates over time are shown in the Supplementary material Appendix 2).

By including the effects of intraspecific competition for resources on individual growth and survival into our model, we further show that the occurrence of the stability–instability pattern across the range of offspring size variance does not critically depend on the strength of intraspecific competition, at least when cannibalism is sufficiently strong and competition is assumed to be most intense among individuals of similar body size (Supplementary material Appendix 3). Additional sensitivity tests showed that the destabilization at low values of the offspring size variance occurs for a wide range of cannibalism interaction strengths. In the sensitivity analysis, cannibalism and competition intensity were varied widely to cover a broad range of ecologically relevant interaction strengths, thus representing large variation in growth rates and survival probabilities. Importantly, the stability–instability transition disappears at 1) small mean offspring sizes, 2) large victim-to-cannibal size ratios or 3) high variances of the cannibalism kernel, which results in stable population dynamics

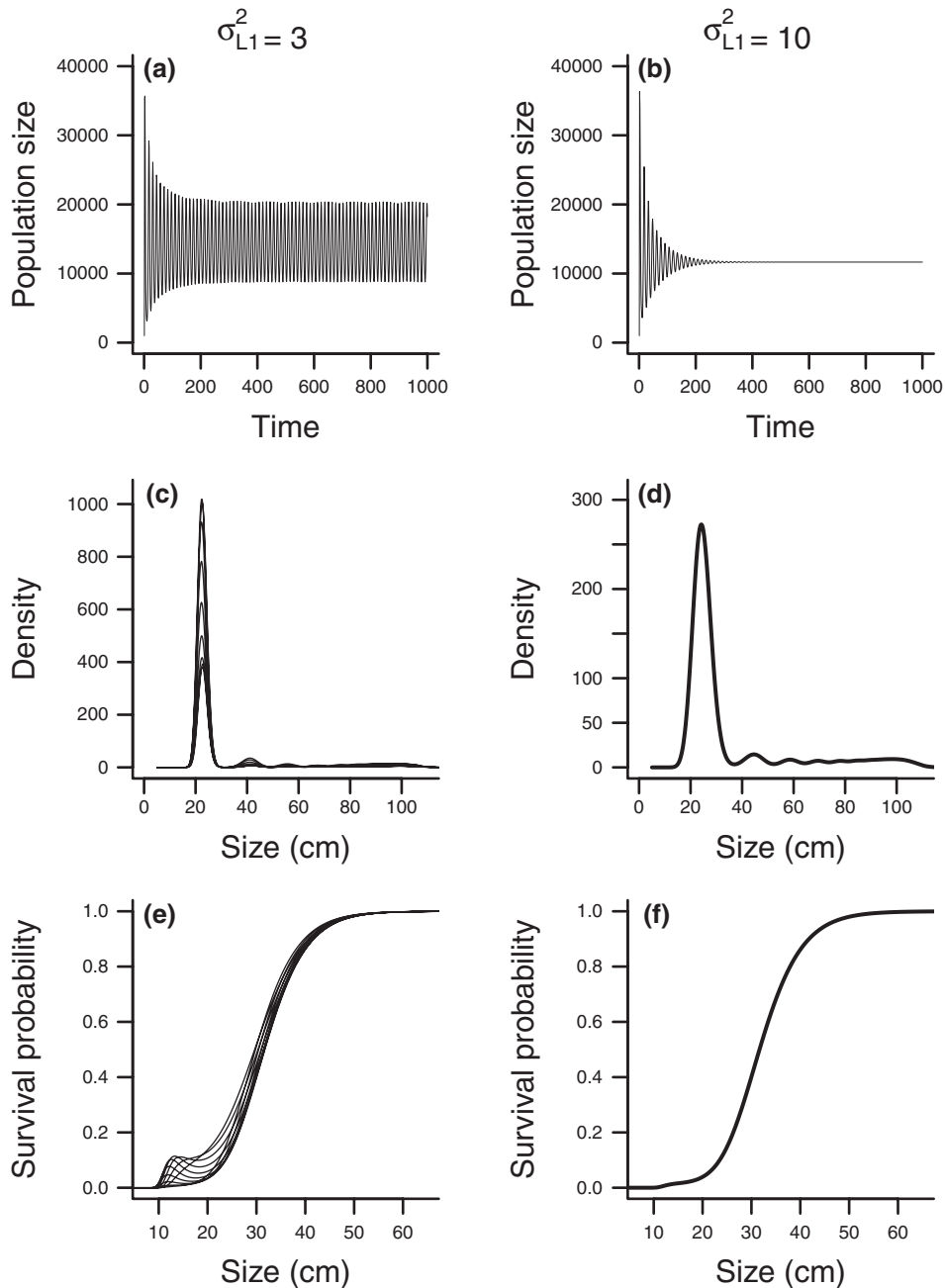


Figure 4. Unstable and stable dynamics. Shown are population densities over time (a–b), size distributions (c–d) and annual probabilities of surviving cannibalism as a function of victim size (e–f) for two values of offspring size variance representing unstable (left,  $\sigma_{L1}^2 = 3$ ) and stable dynamics (right,  $\sigma_{L1}^2 = 10$ ). Projections were run for 1000 time steps, and size distributions and survival curves were plotted for the last 10 time steps to illustrate the unstable dynamics at low offspring size variance.

irrespective of the offspring size variance (Supplementary material Appendix 4). Furthermore, the stability–instability transition is shifted to lower variance values as variation in individual growth increases. Therefore, other aspects of the ecological interaction between individuals also matter for the population dynamical response to changes in the variance in offspring size. Overall, our sensitivity analysis showed that the population dynamics are either stable throughout the range of offspring size variances or exhibit

a transition to unstable dynamics at low size variance, as presented in Fig. 3.

## Discussion

We have developed an integral projection model including size-dependent cannibalism as well as additional density regulation at the offspring stage. The main conclusion from



our analysis is that the amount of individual variation in offspring size affects population stability. In our model the population dynamics become increasingly unstable as the size variation decreases, and become more stable as the variation in offspring body size increases. Earlier work suggested that trait variation in general affects population growth and stability, and that stabilizing or destabilizing effects can be predicted from unstructured population models where the trait distribution depends only on population density (Bjørnstad and Hansen 1994). We used a more complex model with continuous size-structure and overlapping generations, which suggests that a stabilizing effect of variation in offspring body size may be generalized to size-structured organisms that are characterized by seasonal reproduction and cannibalistic behaviour. Similar life-histories may be particularly wide-spread among fish species at mid or high latitudes (Pereira et al. 2017).

The shift from stable to unstable dynamics as offspring size variance decreases is driven by a range of complex size-dependent processes. One of the key processes affecting this transition is offspring mortality, which strongly depends on intercohort cannibalism and in turn has a strong influence on the population dynamics. The offspring size distribution sets the starting point for subsequent growth and therefore influences the entire size distribution. A large offspring size variance leads to broad cohort peaks in the population size distribution, while a low size variance leads to pronounced cohort peaks. A size distribution without strong peaks implies little interannual variation in the risk of cannibalism, where offspring mortality from intercohort predation can be high but it is stable, thus preventing the occurrence of strong or weak cohorts. As the offspring size distribution is narrowed, the cohort peaks in the population size distribution become more pronounced such that more individuals of a given cohort escape cannibalism if they are outside the victim size range, or are cannibalized if they are within the victim size range. These individuals subsequently contribute to a higher (or lower) density of cannibals, thus increasing (or decreasing) the mortality among new victims. Such density-dependent feedbacks in intercohort cannibalism give rise to fluctuations in annual offspring mortality and population size. As offspring size variance is further reduced, the fluctuations increase (Fig. 3), such that at extremely low offspring size variance most of the offspring cohort either escapes cannibalism (when the number of potential cannibals is low), or is cannibalized (when preyed upon by a preceding cohort that was not heavily cannibalized). Hence, mean offspring mortality is high whenever the offspring size distribution matches the cannibalism window of preceding cohorts (Fig. 4). The population dynamics are therefore characterized by the dominance of strong cohorts. This feedback, which prevents stabilization of the population dynamics, results from the interplay between the size distributions of the interacting cohorts and intercohort predation (cannibalism and background survival rates are shown in Supplementary material Appendix 2 Fig. A2). Intercohort cannibalism on offspring thus plays a crucial

role in causing unstable dynamics. The exact quantitative pattern of where the shift occurs, or whether it occurs at all, is modified by other processes in the model, such as the growth model (mean and variance), the strength of cannibalism and the cannibalism window as determined by the cannibalism kernel, but the qualitative pattern of increased stability at higher offspring size variance remains the same across all our tested conditions (Supplementary material Appendix 4).

Previous studies have largely found destabilizing effects of cannibalism on population dynamics, yet stabilizing effects have also been reported (Cushing 1991, Hastings and Costantino 1991, Briggs et al. 2000, Claessen et al. 2000). Importantly, when cannibals are able to feed efficiently on new recruits, cannibal-driven cycles can occur due to the high mortality induced among victims (Claessen et al. 2002, Persson et al. 2006). Whether cannibals can efficiently feed on recruits also depends on the cannibalism window and initial hatchling size (Persson et al. 2004, van Kooten et al. 2010). Here we show that this is more likely to occur when the offspring size variation is low compared to the cannibalism size window. Similarly, adult-driven cohort cycles can occur when large individuals are competitively superior over small ones (Briggs et al. 2000, de Roos and Persson 2013). Both competitive superiority and cannibalism by larger conspecifics represent strongly asymmetric intraspecific interactions. In contrast, when small individuals are competitively superior, they may outcompete their larger conspecifics and induce juvenile-driven cycles. Whether or not increased offspring variation may lead to unstable dynamics in such cases remains to be explored.

The long-term monitoring data from Windermere suggest that the variance in body size of one-year-old pike has declined over a time period of 50 years (Supplementary material Appendix 1). Our model results indicate that a population experiencing such continuous declines in offspring size variance may be approaching increasingly unstable dynamics. While environmental changes have profoundly altered this freshwater ecosystem over the past few decades, including increased water temperatures (Ohlberger et al. 2013), fundamental changes in the fish community (Winfield et al. 2012), and shifts in the phenology of the fish and plankton communities (Thackeray et al. 2013), the causes of the reduction in size variance in Windermere pike are not known and merit further investigation. Our model assumes constant size variance to study the consequences of such variation; when underlying mechanisms of the size variation are identified, these could be incorporated into the model. The population does not currently show signs of instability. While the trend in offspring size variance is decreasing, size variance has generally been large, and there is considerable variation in size variance among years, both of which seem to prevent unstable dynamics. Additionally, other factors not accounted for in our model such as environmental stochasticity in survival might have a stabilizing effect. It is worth noting that the range in offspring size variance analysed in this study, which is equivalent to a coefficient of variation of up to ~20%,

encompasses size variances reported for other species. For freshwater and marine fishes, the CV in size of egg and larval stages has generally been found to range from 3 to 12% (for comparisons of multiple species see: Hutchings 1997, Einum and Fleming 2002), whereas size variation among juveniles is typically larger, with reported values of 8–23% (several fish species: van Densen et al. 1996, Nordwall et al. 2001). Most of the species examined in those studies are characterized by seasonal reproduction and cannibalistic behaviour.

The importance of phenotypic trait variation has long been recognized in evolutionary ecology, because variation in heritable traits provides the basis for natural selection. Changes in trait distributions due to altered ecological processes can facilitate adaptive evolution if reproductive fitness is increased under novel ecological conditions. One example would be increased climatic variability favouring differential maternal investment and thus higher variation in offspring body size. Similar changes could arise in response to human impacts such as harvesting. The resulting feedbacks between trait evolution and ecological processes are important to consider when evaluating potential consequences of altered trait distributions. Such eco-evolutionary feedback dynamics related to individual trait variation have recently received increasing attention (Bolnick et al. 2011, Vindenes and Langangen 2015). The model presented here provides a starting point for future investigations of eco-evolutionary dynamics, for instance by letting the offspring size distribution depend on maternal size.

This work extends the demographic modelling framework of IPMs to include cannibalism, a widespread and inherently size-dependent intraspecific interaction. Our main result demonstrates how individual size variation within cohorts can profoundly affect the dynamics of animal populations, and that increased variation in offspring body size stabilizes population dynamics under a wide range of conditions. In a broader context, our work adds to the growing evidence of the importance of early-life processes (e.g. maternal effects and cohort effects) for individuals and populations. Future developments of our modelling framework include considering species interactions and investigating the dynamical consequences of stochastic variation in offspring size distributions. Empirical studies should further investigate the potential mechanisms leading to changes in size variation and evaluate the empirical evidence for associated shifts in population dynamics.

*Acknowledgements* – We gratefully acknowledge the many researchers at the Centre for Ecology & Hydrology (Lancaster, UK) and at the Freshwater Biological Association (Ambleside, UK) who have undertaken the field and laboratory work for the long-term scientific monitoring of Windermere pike. Special thanks to Thomas Sverdrup for his help throughout the project.

*Funding* – This work was supported by the Research Council of Norway (RCN project numbers 244404 and 224738) and by the Natural Environment Research Council of the UK.

*Author contributions* – All authors designed the study, developed the model, and contributed to writing the paper. JO and YV lead the model analysis and wrote the first manuscript draft.

## References

- Bassar, R. D. et al. 2016. The effects of asymmetric competition on the life history of Trinidadian guppies. – *Ecol. Lett.* 19: 268–278.
- Begon, M. et al. 2006. *Ecology: from individuals to ecosystems*. – Blackwell Publishing.
- Bjørnstad, O. N. and Hansen, T. 1994. Individual variation and population dynamics. – *Oikos* 69: 167–171.
- Bolnick, D. I. et al. 2011. Why intraspecific trait variation matters in community ecology. – *Trends Ecol. Evol.* 26: 183–192.
- Bolnick, D. I. et al. 2003. The ecology of individuals: incidence and implications of individual specialization. – *Am. Nat.* 161: 1–28.
- Briggs, C. J. et al. 2000. What causes generation cycles in populations of stored-product moths? – *J. Anim. Ecol.* 69: 352–366.
- Bry, C. 1996. Role of vegetation in the life cycle of pike. – In: Craig, J. F. (ed.), *Pike: biology and exploitation*. Chapman & Hall, pp. 45–67.
- Claessen, D. et al. 2000. Dwarfs and giants: cannibalism and competition in size-structured populations. – *Am. Nat.* 155: 219–237.
- Claessen, D. et al. 2004. Population dynamic theory of size-dependent cannibalism. – *Proc. R. Soc. B* 271: 333–340.
- Claessen, D. et al. 2002. The impact of size-dependent predation on population dynamics and individual life history. – *Ecology* 83: 1660–1675.
- Coulson, T. 2012. Integral projections models, their construction and use in posing hypotheses in ecology. – *Oikos* 121: 1337–1350.
- Cushing, J. M. 1991. A simple model of cannibalism. – *Math. Biosci.* 107: 47–71.
- Dall, S. R. X. et al. 2012. An evolutionary ecology of individual differences. – *Ecol. Lett.* 15: 1189–1198.
- de Roos, A. et al. 1992. Studying the dynamics of structured population models: a versatile technique and its application to *Daphnia*. – *Am. Nat.* 139: 123–147.
- de Roos, A. M. and Persson, L. 2013. *Population and community ecology of ontogenetic development*. – Princeton Univ. Press.
- DeAngelis, D. L. et al. 1993. Fish cohort dynamics: application of complementary modeling approaches. – *Am. Nat.* 142: 604–622.
- Easterling, M. R. et al. 2000. Size-specific sensitivity: applying a new structured population model. – *Ecology* 81: 694–708.
- Ebenman, B. and Persson, L. 1988. *Size-structured populations*. – Springer.
- Einum, S. and Fleming, I. A. 2002. Does within-population variation in fish egg size reflect maternal influences on optimal values? – *Am. Nat.* 160: 756–765.
- Ellner, S. and Rees, M. 2006. Integral projection models for species with complex demography. – *Am. Nat.* 167: 410–428.
- Ellner, S. P. et al. 2016. *Data-driven modelling of structured populations*. – Springer.
- Fox, L. 1975. Cannibalism in natural populations. – *Annu. Rev. Ecol. Syst.* 6: 87–106.
- Frost, W. and Kipling, C. 1967. A study of reproduction, early life, weight–length relationship and growth of pike, *Esox lucius* L., in Windermere. – *J. Anim. Ecol.* 36: 651–693.

- Hart, S. P. et al. 2016. How variation between individuals affects species coexistence. – *Ecol. Lett.* 19: 825–838.
- Hastings, A. and Costantino, R. F. 1991. Oscillations in population numbers – age-dependent cannibalism. – *J. Anim. Ecol.* 60: 471–482.
- Haugen, T. et al. 2007. Density dependence and density independence in the demography and dispersal of pike over four decades. – *Ecol. Monogr.* 77: 483–502.
- Holling, C. S. 1959. Some characteristics of simple types of predation and parasitism. – *Can. Entomol.* 91: 395–398.
- Huss, M. et al. 2007. The origin and development of individual size variation in early pelagic stages of fish. – *Oecologia* 153: 57–67.
- Huss, M. et al. 2008. Resource heterogeneity, diet shifts and intra-cohort competition: effects on size divergence in YOY fish. – *Oecologia* 158: 249–257.
- Huss, M. et al. 2010. Intra-cohort cannibalism and size bimodality: a balance between hatching synchrony and resource feedbacks. – *Oikos* 119: 2000–2011.
- Hutchings, J. A. 1997. Life history responses to environmental variability in early life. – In: Chambers, R. C. and Trippel, E. A. (eds), *Early life history and recruitment in fish populations*. Chapman and Hall, pp. 139–168.
- Johnston, T. A. and Leggett, W. C. 2002. Maternal and environmental gradients in the egg size of an iteroparous fish. – *Ecology* 83: 1777–1791.
- Keast, A. and Eadie, J. 1984. Growth in the first summer of life: a comparison of nine co-occurring fish species. – *Can. J. Zool.* 62: 1242–1250.
- Le Cren, E. D. 2001. The Windermere perch and pike project: a historical review. – *Freshwater Forum* 15: 3–34.
- Lomnicki, A. 1988. *Population ecology of individuals*. – Princeton Univ. Press.
- Marshall, D. J. et al. 2008. Offspring size variation within broods as a bet-hedging strategy in unpredictable environments. – *Ecology* 89: 2506–2517.
- Mittelbach, G. G. and Persson, L. 1998. The ontogeny of piscivory and its ecological consequences. – *Can. J. Fish. Aquat. Sci.* 55: 1454–1465.
- Moran, E. V. et al. 2015. Intraspecific trait variation across scales: implications for understanding global change responses. – *Global Change Biol.* 22: 137–150.
- Nordwall, F. et al. 2001. Intercohort competition effects on survival, movement and growth of brown trout (*Salmo trutta*) in Swedish streams. – *Can. J. Fish. Aquat. Sci.* 58: 2298–2308.
- Ohlberger, J. et al. 2013. Biotic and abiotic effects on cohort size distributions in fish. – *Oikos* 122: 835–844.
- Olsen, E. M. et al. 2009. Nine decades of decreasing phenotypic variability in Atlantic cod. – *Ecol. Lett.* 12: 622–631.
- Peacor, S. D. et al. 2007. Mechanisms of nonlethal predator effect on cohort size variation: Ecological and evolutionary implications. – *Ecology* 88: 1536–1547.
- Pereira, L. S. et al. 2017. Is there a relationship between fish cannibalism and latitude or species richness? – *PLoS One* 12: e0169813–14.
- Persson, L. et al. 2006. Temporal stability in size distributions and growth rates of three *Esox lucius* L. populations. A result of cannibalism? – *J. Fish Biol.* 69: 461–472.
- Persson, L. et al. 2003. Gigantic cannibals driving a whole-lake trophic cascade. – *Proc. Natl Acad. Sci. USA* 100: 4035–4039.
- Persson, L. et al. 2004. Cannibalism in a size-structured population: energy extraction and control. – *Ecol. Monogr.* 74: 135–157.
- Pfister, C. and Stevens, F. 2002. The genesis of size variability in plants and animals. – *Ecology* 83: 59–72.
- Philippi, T. and Seger, J. 1989. Hedging one's evolutionary bets, revisited. – *Trends Ecol. Evol.* 4: 41–44.
- Polis, G. 1981. The evolution and dynamics of intraspecific predation. – *Annu. Rev. Ecol. Evol. Syst.* 12: 225–251.
- Rasmussen, N. L. and Rudolf, V. H. W. 2015. Phenological synchronization drives demographic rates of populations. – *Ecology* 96: 1754–1760.
- Rudolf, V. H. W. 2007. The interaction of cannibalism and omnivory: consequences for community dynamics. – *Ecology* 88: 2697–2705.
- Simmonds, E. G. and Coulson, T. 2014. Analysis of phenotypic change in relation to climatic drivers in a population of Soay sheep *Ovis aries*. – *Oikos* 124: 543–552.
- Thackeray, S. J. et al. 2013. Food web de-synchronisation in England's largest lake: an assessment based upon multiple phenological metrics. – *Global Change Biol.* 19: 3568–3580.
- Uchmanski, J. 1985. Differentiation and frequency-distributions of body weights in plants and animals. – *Phil. Trans. R. Soc. B* 310: 1–75.
- van Densen, W. et al. 1996. Intra-cohort variation in the individual size of juvenile pikeperch, *Stizostedion lucioperca*, and perch, *Perca fluviatilis*, in relation to the size spectrum of their food items. – *Ann. Zool. Fenn.* 33: 495–506.
- van Kooten, T. et al. 2004. Local foraging and limited mobility: dynamics of a size-structured consumer population. – *Ecology* 85: 1979–1991.
- van Kooten, T., et al. 2010. Size at hatching determines population dynamics and response to harvesting in cannibalistic fish. – *Can. J. Fish. Aquat. Sci.* 67: 401–416.
- Vindenes, Y. and Langangen, Ø. 2015. Individual heterogeneity in life histories and eco-evolutionary dynamics. – *Ecol. Lett.* 18: 417–432.
- Vindenes, Y. et al. 2014. Effects of climate change on trait-based dynamics of a top predator in freshwater ecosystems. – *Am. Nat.* 183: 243–256.
- Vindenes, Y. et al. 2016. Fitness consequences of early life conditions and maternal size effects in a freshwater top predator. – *J. Anim. Ecol.* 85: 692–704.
- Winfield, I. J. et al. 2012. Long-term changes in the diet of pike (*Esox lucius*), the top aquatic predator in a changing Windermere. – *Freshwater Biol.* 57: 373–383.
- Winfield, I. J. et al. 2013a. Pike growth data 1944–1995. – NERC Environmental Information Data Centre, <<http://dx.doi.org/10.5285/637d60d6-1571-49af-93f7-24c1279d884d>>.
- Winfield, I. J. et al. 2013b. Pike survival data 1953–1990. – NERC Environmental Information Data Centre, <<http://dx.doi.org/10.5285/813e07dd-2135-49bc-93c6-83999e442b36>>.
- Winfield, I. J. et al. 2015. Windermere pike fecundity and egg data 1963–2003. – NERC Environmental Information Data Centre, <<http://dx.doi.org/10.5285/4b255fc4-33d3-4beb-a238-37e1a8cf32a2>>.
- Wright, P. J. and Trippel, E. A. 2009. Fishery-induced demographic changes in the timing of spawning: consequences for reproductive success. – *Fish Fish.* 10: 283–304.

Supplementary material (available online as Appendix oik-05507 at <[www.oikosjournal.org/appendix/oik-05507](http://www.oikosjournal.org/appendix/oik-05507)>). Appendix 1–4.