Research paper

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

ABSTRACT

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 (e.g., 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 (i) genetic variation, (ii) social structure, e.g. resource monopolization, (iii) maternal effects, (iv) small-scale heterogeneity in environmental conditions, (v) variation in the time of hatching or emergence, and (vi) 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: 1-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 $\boldsymbol{n_t} = n_t(x)$, 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 i) size-dependent cannibalism, which can potentially affect any vital rate, and ii) 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 Figure 1. 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 $LN(\frac{x}{y},\mu_{cn},\sigma_{cn})$ with scale parameter σ_{cn} and location parameter μ_{cn}. The cannibalism kernel is given by the normalized function $C_{cn}(x,y) = LN(\frac{x}{y},\mu_{cn},\sigma_{cn})/y$ (where $\int_0^\infty LN\left(\frac{x}{y},\mu_{cn},\sigma_{cn}\right)dx = y$). This implies that the range of potential victim sizes (cannibalism window) increases with cannibal size (Figure 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 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 β_{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 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 δ_{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 $s_{cn,\,t}(\mathrm{x}) = \exp\left(-\omega_{cn,\,t}(x)\right)$. 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 (figure 2b): $s_b(x) = \frac{2s_{b0}}{1 + e^{\beta_{sb}(x - \alpha_{sb})^2}}$. 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:

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

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 (Appendix 3). Mean length next year (on log scale), given current length *x,* is $\mu_g(x) = \ln\left[KL_\infty + (1-K)\,x\right]$. Here, K is the von Bertalanffy growth rate, and L_∞ 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 (Figure 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 (described below), 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 α_R is the slope at origin (i.e. number of offspring resulting from very low egg numbers), and β_R is a capacity parameter such that α_R/β_R is the maximum number of offspring (Figure 2d). Before entering next year's population, the offspring number can be further reduced by size-dependent cannibalism by the rest of the population (Figure 1). This intercohort 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 $n_t^*(x')$ (Figure 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 (Figure 2e) with $p_m(x) = \frac{1}{1+e^{-\sigma_p(x-\mu_p)}}$, where μ_p is mean size at maturation and σ_p determines the slope on the logit scale. Fecundity $m(x)$ is assumed to follow a power function (Figure 2e) $m(x) = \alpha_m x^{\beta_m}$, where α_m is a constant and β_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 σ^2_{L1} (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 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 σ_{L1}^2 (see Figure 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 (μ_{L1}) the strength of cannibalism (β_{cn}), and growth variation later in life (τ_g). 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 (v.3.3.2, R Core Team, 2016).

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; Vindenes et al. 2014; Winfield et al. 2013a, b, 2015). 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 $s_{b0} = 1.7$ (Figure 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; Figure 2a). Reported lower and upper limits for the victim-to-cannibal size ratio in pike are 0.03 and 0.55 (Persson, Bertolo & de Roos 2006). The scaling parameter of the maximum cannibalistic attack rate was set to $\alpha_{cn} = 0.6$ (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 (Figure 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 ~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 ~31-50 cm (Figure 2e Frost & 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 $\beta_m = 3.3$ (Figure 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 $\alpha_R = 4*10^{-4}$ and $\beta_R = 1*10^{-8}$ (Figure 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 $\beta_{0L1} = 3.85$ and $\beta_{ML1} = 0.04$.

RESULTS

Changes in offspring size variance have strong and consistent effects on population stability (Figure 3). Population dynamics are stable at wide offspring size distributions, but unstable at narrow size distributions (see Figure 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 (Figure 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 exhibits oscillations that are not dampened over time, and a stable size distribution is not reached (Figure 4a). Instead, the density of offspring that enter the population and consequently the densities of older cohorts both fluctuate (Figure 4c), due to strongly varying probabilities of surviving cannibalism (Figure 4e). In contrast, with high variance in offspring size the population reaches an equilibrium size (Figure 4b). A stable size distribution is reached showing a size structure with distinct age-cohorts (Figure 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 (Figure 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 (i) small mean offspring sizes, (ii) large victim-to-cannibal size ratios, or (iii) high variances of the cannibalism kernel, which results in stable population dynamics 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 Figure 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 (Figure 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 (Figure 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 Figure A2, Supplementary Material). 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 (see 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 1-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%-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 & 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.

DECLARATIONS

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540 spawning: consequences for reproductive success. – Fish Fish. 10: 283–304.

541 **Table 1: Overview of variables in the IPM**

542

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
$\boldsymbol{n_t} = n_t(x)$	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
• σ_{L1}^2	Variance in offspring size at age 1

543 **Table 2: Overview of model parameters and baseline values.**

Process	Parameter	Description	Value	Units
Background survival	α_{sb}	Size at maximum survival	80	mm
	s_{b0}	Maximum survival	0.85	-
	β_{sb}	Decline in survival at smaller and larger sizes	-0.0005	mm^{-2}
Cannibalism survival	μ_{cn}	Location parameter	-1.5	$mm\ mm^{-1}$
	σ_{cn}	Scale parameter	0.3	$mm\ mm^{-1}$
	β_{cn}	Cannibalism intensity	0.01	$mm^{-\alpha_{cn}}$
	α_{cn}	Intensity size exponent	0.6	-
	δ_{cn}	Type of functional response	0.1	-
von Bertalanffy growth function	L_∞	Asymptotic average length	109	mm
	K	Growth rate coefficient	0.21	-
Variance in growth	ν_g	Growth variance exponent	-0.015	-
	τ_g	Growth variance scalar	5	-
Maturation	μ_p	Size at 50% maturation probability	41.5	mm
	σ_p	Width of maturation probability function	0.5	-
Fecundity	α_m	Fecundity constant	0.095	$ind\ mm^{-\beta_m}$
	β_m	Fecundity exponent	3.3	-
Offspring density	α_R	Maximum per capita recruitment	4e-4	$ind\ ind^{-1}$
	β_R	Determines carrying capacity	1e-8	ind
Offspring size distribution	β_{0L1}	Constant of mean offspring size depending on egg density	3.85	$\ln(mm)$
	β_{ML1}	Exponent of mean offspring size depending on egg density	0.04	$\ln(mm)$

544 ind = individuals

FIGURE CAPTIONS

Figure 1: Timing of annual life-history events. $\boldsymbol{n_t}$ is the population size distribution at time t, M_t is the total number of eggs produced by the population, $\boldsymbol{R_t} = R_t(x)$ is the population distribution of offspring before intercohort cannibalism occurs, $\boldsymbol{O_t} = O_t(x)$ is the population distribution of age-1 offspring entering the next census, and $\boldsymbol{n_t^*} = n_t^*(x)$ 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.

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 (dashed 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).

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

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

567 survival curves were plotted for the last 10 time steps to illustrate the unstable dynamics at low
568 offspring size variance.

SUPPLEMENTARY MATERIAL

Appendix 1: Data figure

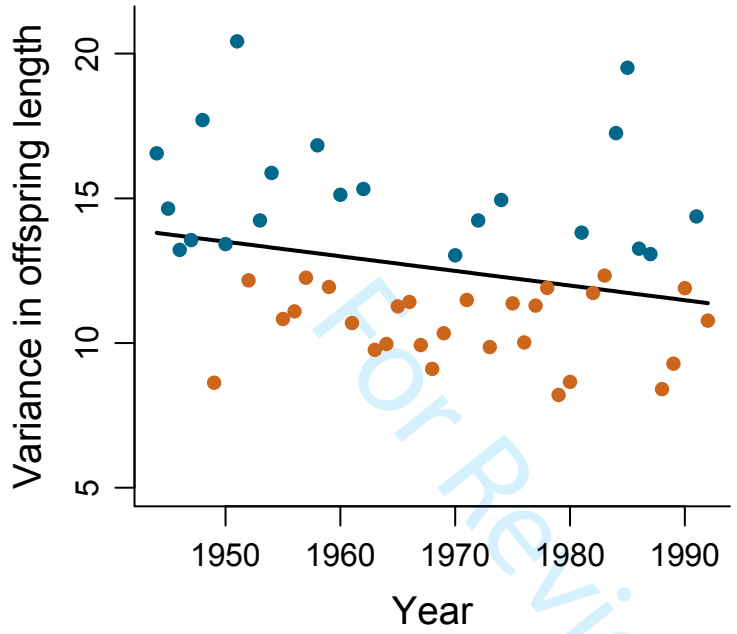


Figure A1: **Size variance in pike**. Time series of the variance in body length (cm^2) for offspring (age-1) pike from Windermere, UK, suggesting a negative trend over time (slope=-0.05; p-value=0.08). Data were based on back-calculated lengths from individuals captured at age 3 and older. Colours indicate values above (blue, dark) and below (orange, light) the long-term average.

Appendix 2: Offspring survival

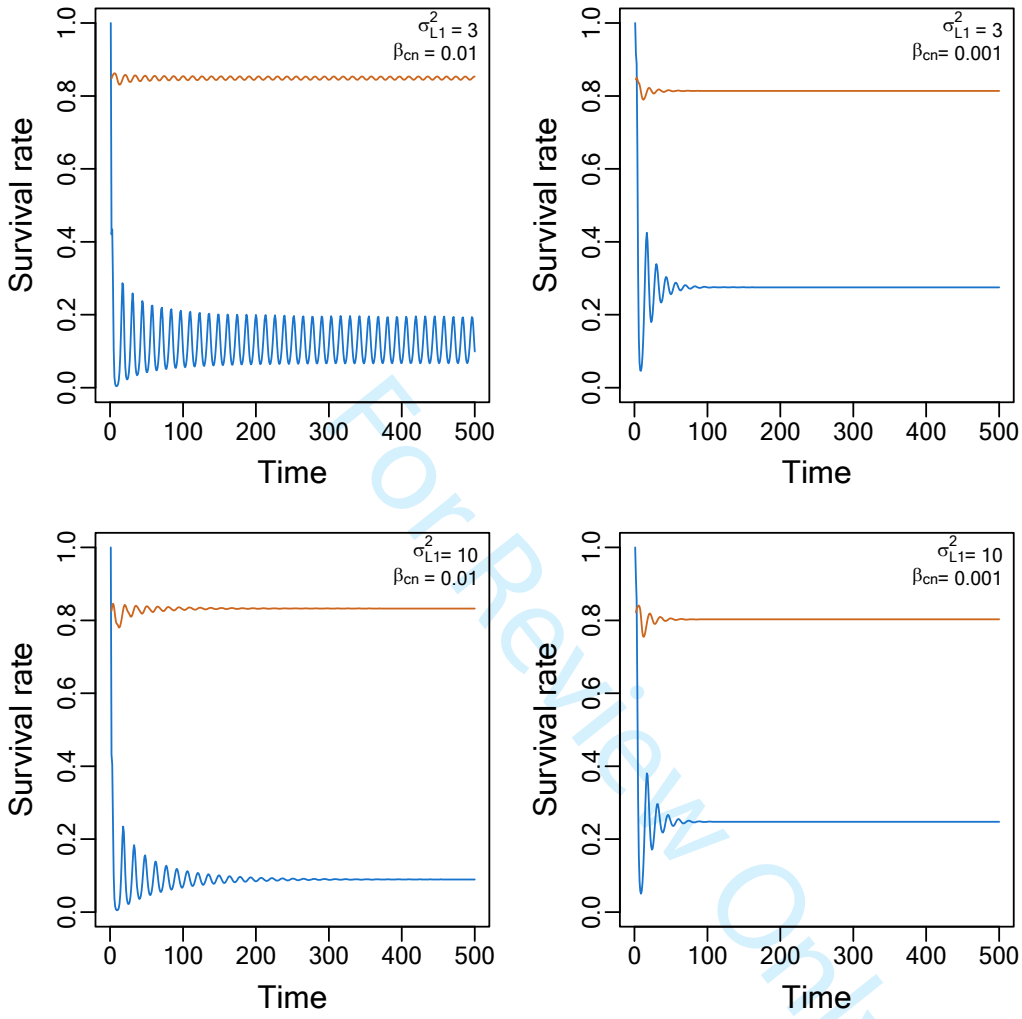


Figure A2: **Cannibalism and background survival of offspring**. Shown are offspring survival rates resulting from intercohort cannibalism (blue) and background mortality (orange) for low offspring size variance (top, $\sigma_{L1}^2 = 3$) and high offspring size variance (bottom, $\sigma_{L1}^2 = 10$) and moderate (left, $\beta_{cn} = 0.01$) and low (right, $\beta_{cn} = 0.001$) cannibalism intensity (500 time steps, all other parameters as in baseline model). Survival rates do not stabilize over time in case of low size variance and moderate cannibalism (top left) thus leading to unstable population dynamics.

Appendix 3: Incorporating competition into the model

Here we describe the methods and results for an extended version of our model that includes size-dependent intraspecific competition in addition to cannibalism. Size-dependent competition can be modelled in a number of different ways, although considering all possible effects of competition is beyond the scope of this study. We consider a scenario where size-dependent competition affects growth and survival and investigate how the main result (that offspring size variation promotes population stability) is affected.

In this model, competition is assumed to affect somatic growth and survival. Competitive interactions are governed by a competition kernel $C_{cp}(x,y)$, which describes the potential interaction strength between an individual of size x and a competitor of size y ($\int_0^\infty C_{cp}(x,y)dx = 1$). We assume that competition is most fierce between individuals of the same size, e.g. because they have similar diet or habitat preferences. Specifically, the competition intensity experienced by an individual of size x due to potential competitors of size y is given by a lognormal distribution with scale parameter σ_{cp} and location parameter $\log(x) + {\sigma_{cp}}^2$. The competition kernel describes the potential effect of an individual of a given size on other individuals across the size range through competitive interactions (Figure A3). Asymmetric competition for shared resources, for instance when juveniles and adults compete for the same prey species in the same habitat, is not reflected by this competition kernel.

Here, survival probability depends on background survival, competition, and cannibalism: $s_t(x) = s_b(x)\, s_{cn,t}(x)\, s_{cp,t}(x)$. The probability of surviving competition is given by $s_{cp,t}(x) = \exp\left(-\int_0^\infty \beta_{cpS}\, y^{\alpha_{cpS}}\, C_{cp}(x,y)\, n_t(y)dy\right)$, where β_{cpS} determines the effect size of competition on survival (higher β_{cpS} values imply stronger effects on survival), while α_{cpS} scales the competitive

intensity over competitor length y. Growth is also depending on competition in this model, through the mean length next year, given current length x and population density $n_t(x)$. This is given by $\mu_{g,t}(x) = \ln\left[KL_\infty + (1-K)x\right] - g_{cp,t}(x)$, where the last term describes the effect of competition and is scaled by the effect size β_{cpG}: $g_{cp,t}(x) = \beta_{cpG}\, x^{\alpha_{cpG}} \int_0^\infty n_t(x_{cp})\, C_{cp}(x_{cp};x)\, dx_{cp}$. The term α_{cpG} determines how the competitive intensity changes over length x, and integration is over competitor lengths x_{cp}.

Parameterization

The effect of competition on growth could not be estimated from the data and was thus varied in the model analysis. The default parameter was set to $\beta_{cpG} = 1*10^{-5}$ (scaling parameter $\alpha_{cpG} = 1$) to achieve reasonable effects of competition on the mean growth rate (i.e. biologically realistic ranges). Here, von Bertalanffy parameters were set to $K = 0.2$ and $L_\infty = 120$, so that growth patterns correspond to empirical patterns under intermediate population densities. The competition effect on survival was assumed to be an order of magnitude smaller than the effect of competition on growth ($\beta_{cpS} = 0.1\ \beta_{cpG}$, and scaling parameter $\alpha_{cpS} = 0$).

Results

The shape of the population size distribution and the stability of the population dynamics also depend on the strength of competition, in addition to the strength of cannibalism. Increased competition causes a smoother size distribution with less distinct peaks for older cohorts, whereas cannibalism causes more pronounced peaks in the size distribution due to a reduction in density and thus competition (Figure A4). Ultimately, very strong competition causes somatic growth to approach zero, which results in a unimodal size distribution. Previous work has shown that competition can lead to size convergence within cohorts due to exploitative interactions when

small individuals are competitively superior, or increased size variation, for instance when alternative prey resources are available or social dominance structures allow for resource monopolization (Huss et al. 2007, 2008, 2010). The strengths of the intraspecific interactions thus determine if and where the transition from unstable to stable dynamics occurs (Figure A5). As competition decreases and cannibalism increases, the transition appears and shifts to larger size variances. The degrees of competition and cannibalism were varied widely in order to cover a broad range of ecologically relevant interaction strengths. The lowest and highest values used for the two interactions thus represent large variation in growth rates and survival probabilities (Figure A6). For example, for offspring of 25 cm (approximately the mean length at age-1 in the baseline model), the probability of surviving cannibalism varied between about 0.004 and 0.992, while the mean annual growth rate varied between 0% and 76% across all combinations of cannibalism and competition intensities considered in the analysis.

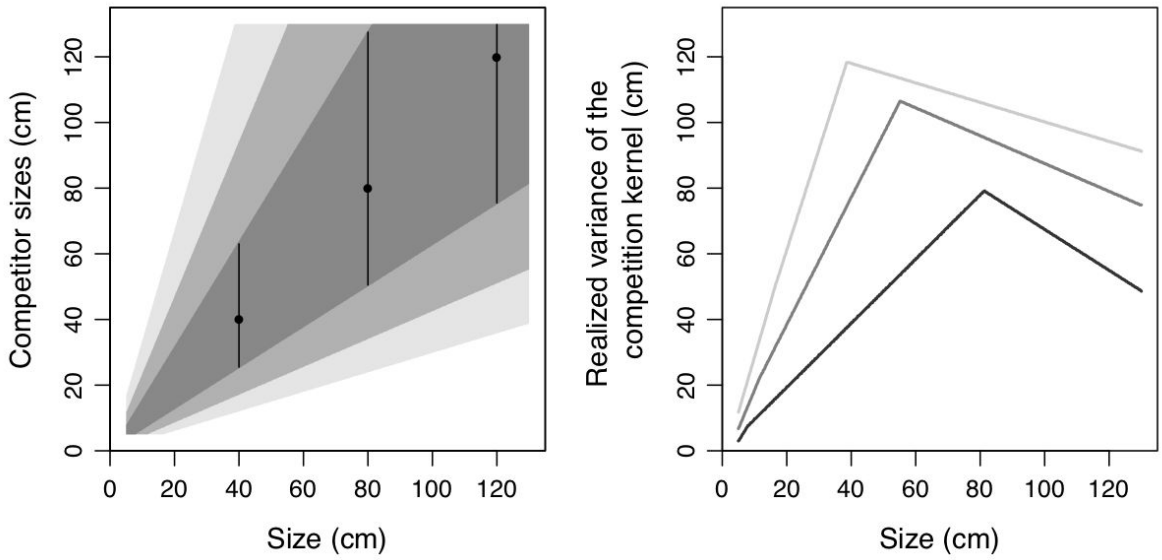


Figure A3: Illustration of the competition kernel. The left plot shows the size range of competitors as a function of the size of the focal individual (for different quantiles of the competition kernel, shaded grey areas). For three focal sizes (40, 80, 120 cm), the range (black lines) and mode (circles) of the kernel are highlighted. The right plot shows the realized variance of the competition kernel for all focal sizes (taking into account the lower and upper size limits) to illustrate that the width of the competition window reaches a maximum at intermediate sizes.

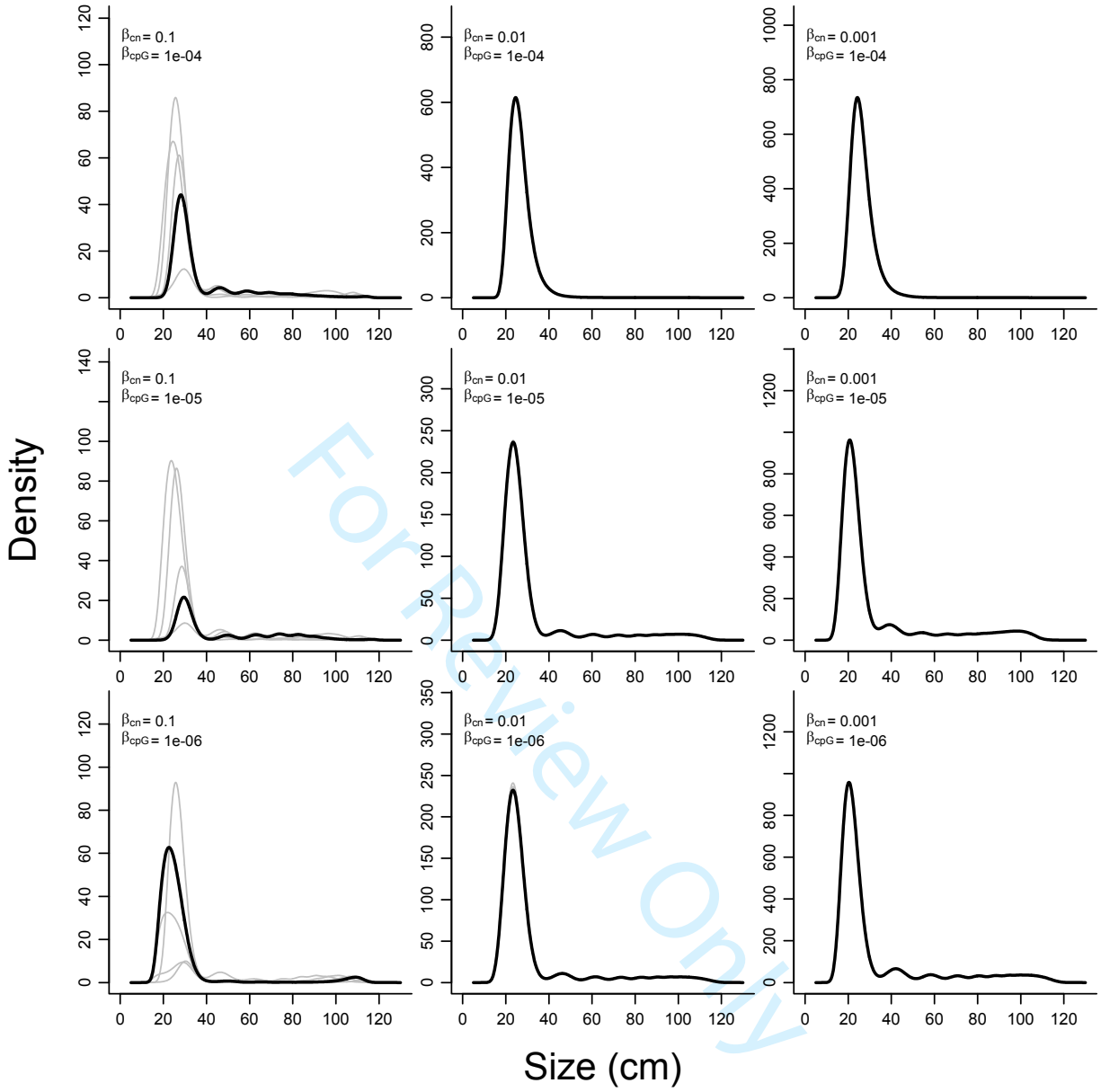


Figure A4: Stable size distribution for different degrees of competition and cannibalism. Thick black lines indicate the size distribution after the maximum number of time steps (t=1000). Thin grey lines indicate the size distribution 1, 3, 5, and 10 years earlier to indicate whether the dynamics are stable or unstable. The default value of 13 was used for the offspring size variance. Cannibalism intensity decreases from left ($\beta_{cn} = 0.1$) to right ($\beta_{cn} = 0.001$), and competition intensity increases from bottom ($\beta_{cpG} = 1e^{-6}$) to top ($\beta_{cpG} = 1e^{-4}$).

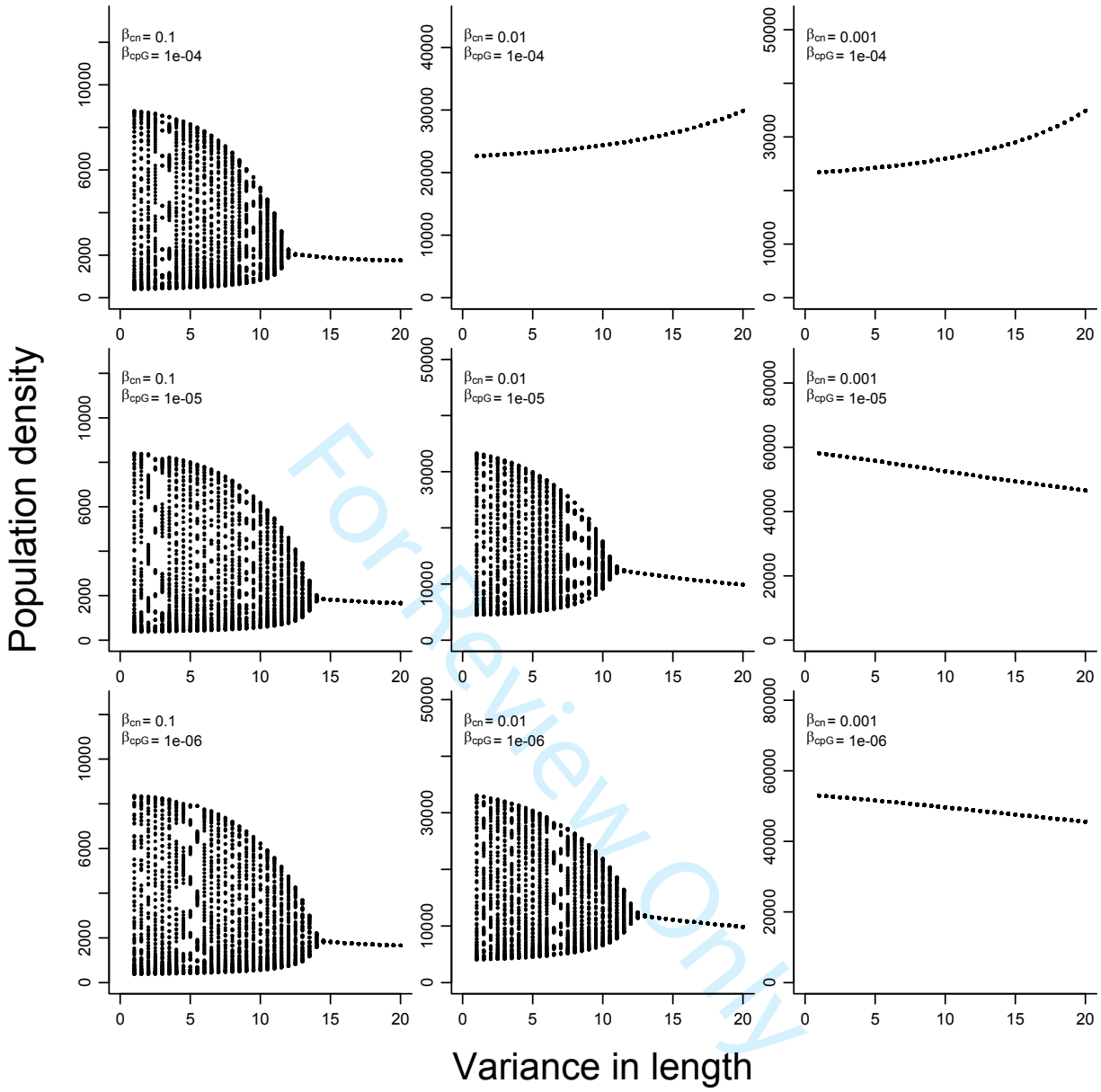
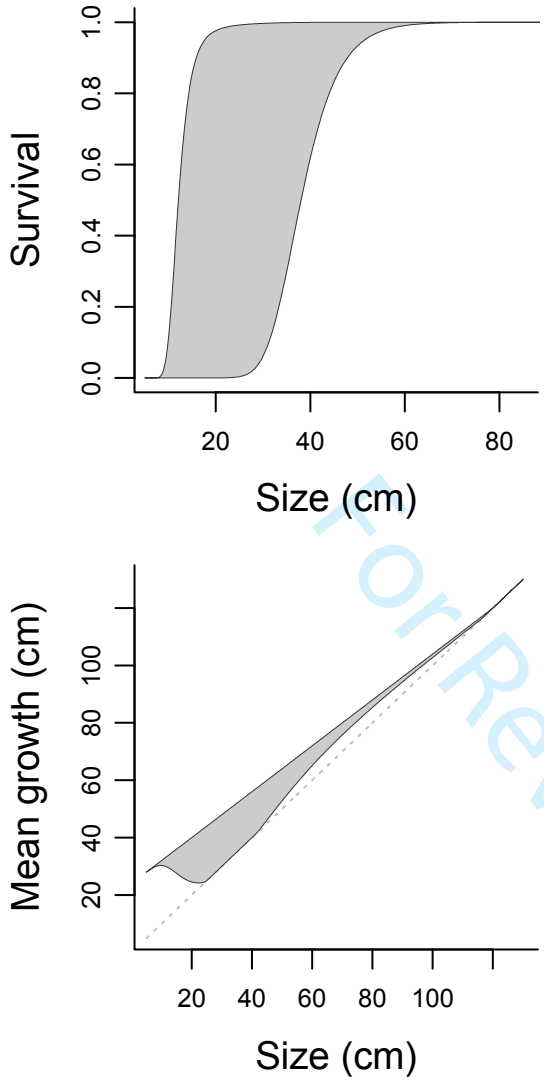


Figure A5: Bifurcation diagrams of population density against the variance in offspring length. Projections were run for 1000 time steps and population density was sampled for the last 100 time steps. Cannibalism intensity decreases from left ($\beta_{cn} = 0.1$) to right ($\beta_{cn} = 0.001$), and competition intensity increases from bottom ($\beta_{cpG} = 1e^{-6}$) to top ($\beta_{cpG} = 1e^{-4}$). These values were chosen to represent a broad range of survival probabilities and mean growth rates.



92

93 **Figure A6**: Cannibalism survival probabilities and mean growth rates for the range of
94 cannibalism and competition intensities considered in the additional model analyses. The dotted
95 line represents zero growth. The plot was produced by setting σ_{L1}^2 to 16 instead of the default
96 value, which resulted in stable dynamics for all tested combinations of cannibalism and
97 competition intensities.

Appendix 4: Additional sensitivity tests

The following sensitivity analyses were performed to further scrutinize further our results with respect to important parameters in the model. First, a bifurcation analysis for different mean values of the offspring size distribution showed that the shift to unstable dynamics at low size variance occurred for a large range of mean sizes of about 21-27 cm, but that this pattern disappeared at smaller mean sizes (Figure A7). This suggests that a transition to unstable dynamics at low size variance is expected for the range of mean offspring sizes observed in Windermere pike (~21-26 cm). Second, in addition to cannibalism intensity, the relative victim-to-cannibal size ratio and the width of the cannibalism kernel affect the size distribution and population stability. Population dynamics stabilize at a higher mean victim-to-cannibal size ratio (Figure A8), and a wider cannibalism kernel (Figure A9), because a wide diet range of cannibals contributes to a smoother size distribution through spreading the predation risk across the victim size range. Finally, we show that and the stability-instability transition occurs at lower variance values when individual growth variation is increased (Figure A10).

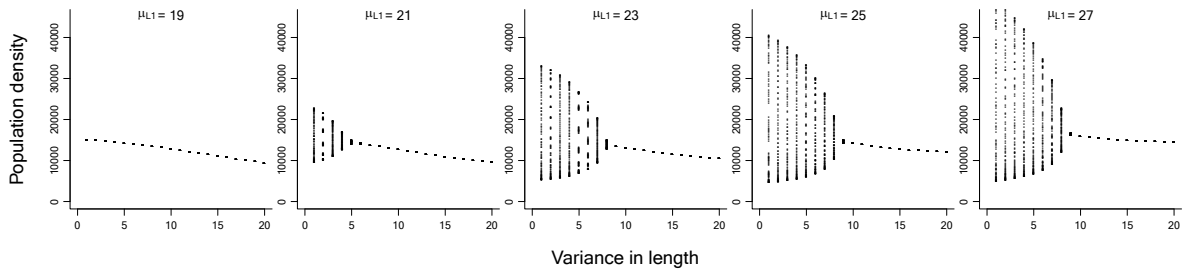
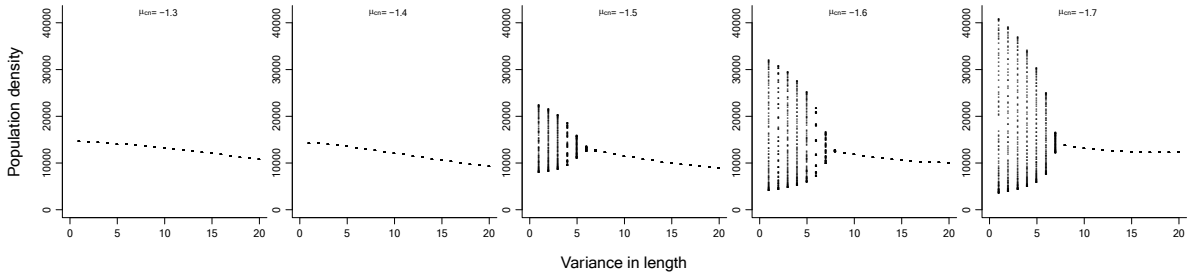


Figure A7: Bifurcation diagrams of population density over variance in offspring size for different values of mean offspring size. In order to investigate whether the shift to unstable dynamics at low size variance would occur at different values of mean offspring size (default value at ~23 cm), we fixed the mean offspring size such that it would not depend on total egg number. The range of mean offspring sizes observed in Windermere pike across years is about 21-26 cm, and the range shown here is 19 cm (left) to 27 cm (right).



121

122 **Figure A8**: Bifurcation diagrams of population density against variance in offspring

123 length for different values of the mean victim-to-cannibal size ratio (μ_{cn}). Values of μ_{cn}

124 range from -1.3 (left) to -1.7 (right). The location parameter μ_{cn} is defined on the log

125 scale.

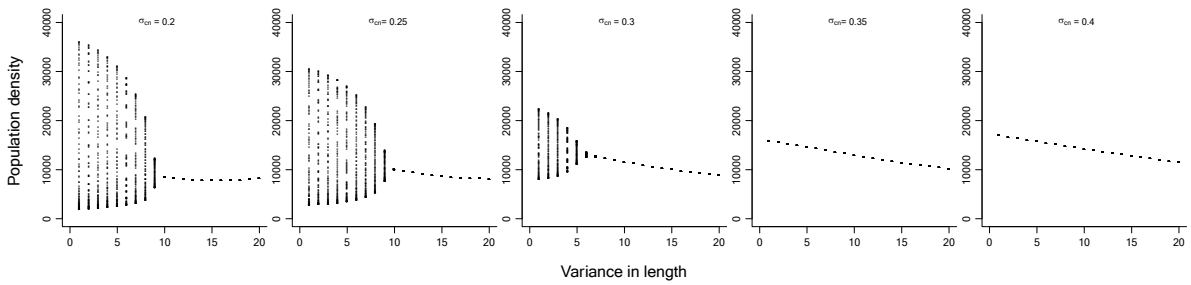
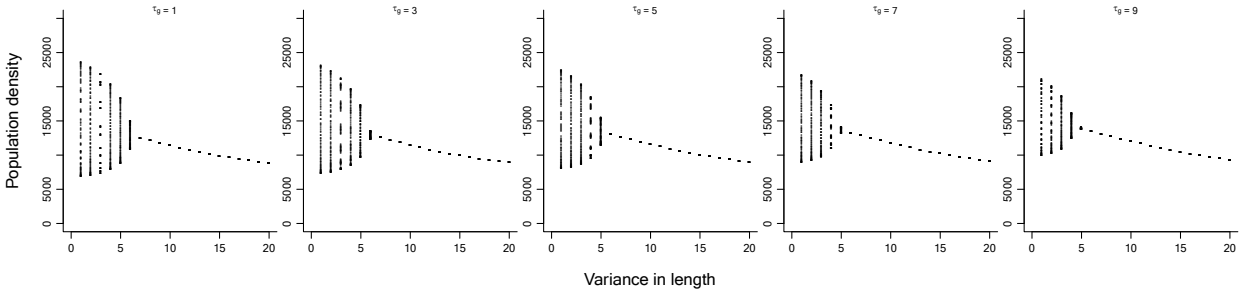


Figure A9: Bifurcation diagrams of population density against variance in offspring length for different values of the standard deviation in the victim-to-cannibal size ratio. Values of σ_{cn} range from 0.2 (left) to 0.4 (right).

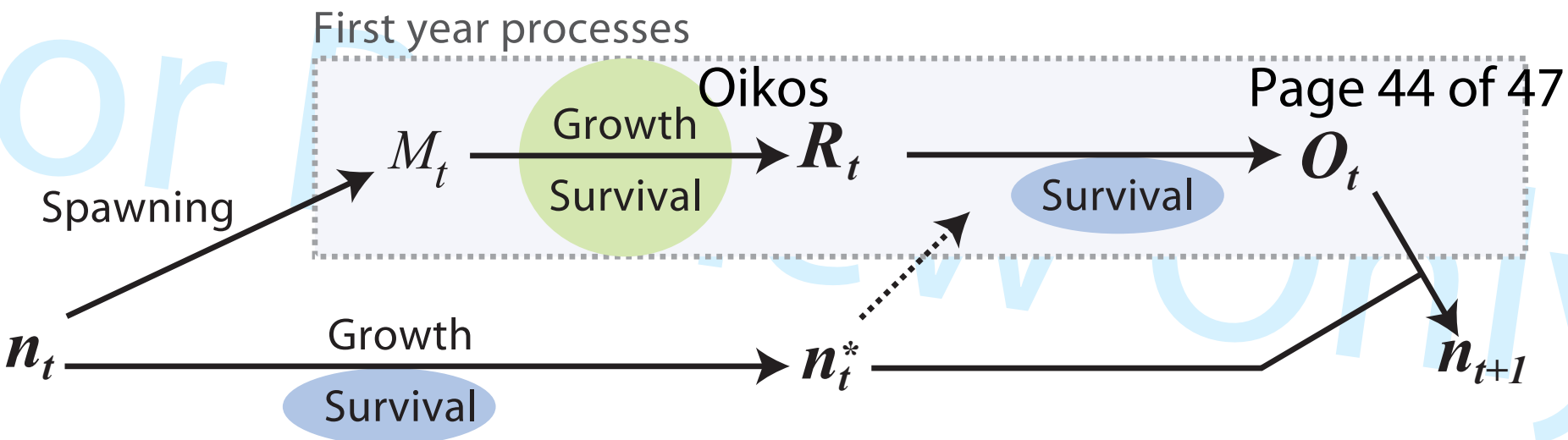


131

132 **Figure A10**: Bifurcation diagrams of population density against variance in offspring

133 length for different values of growth variation (τ_g). Values of τ_g range from 1 (left) to 9

134 (right).


First year processes
Spawning
M_t
Growth
Survival
R_t
Survival
O_t
n_t
Growth
Survival
n_t^*
n_{t+1}