Journal of Animal Ecology

Journal of Animal Ecology 2016, **85,** 356–370 doi: 10.1111/1365-2656.12477

DEMOGRAPHY BEYOND THE POPULATION

Des différences, pourquoi? Transmission, maintenance and effects of phenotypic variance

Floriane Plard[1]*, Jean-Michel Gaillard[2], Tim Coulson[3] and Shripad Tuljapurkar[1]

[1]*Department of Biology, Stanford University, Herrin Labs 454, Stanford, CA 94305, USA;* [2]*Université de Lyon F-69000 Lyon; Université Lyon 1; CNRS, UMR5558, Laboratoire de Biométrie et Biologie Évolutive, F-69622, Villeurbanne, France and* [3]*Department of Zoology, University of Oxford, Tinbergen Building, South Parks Road, Oxford OX1 3PS, UK*

Summary

1. Despite the observed distribution of variable individual phenotypes, survival and reproductive performance in wild populations, models of population dynamics often focus on mean demographic rates. Populations are constituted by individuals with different phenotypes and thus different performances. However, many models of population dynamics provide no understanding of the influence of this phenotypic variation on population dynamics.

2. In this paper, we investigate how the relationships between demographic rates and phenotype distribution influence the transmission and the upholding of phenotypic variation, and population dynamics. We used integral projection models to measure associations between differences of phenotypic trait (size or mass) among individuals and demographic rates, growth and inheritance, and then quantify the influence of phenotypic variation on population dynamics. We build an analytical and general model resulting from simplifications assuming small phenotypic variance. We illustrate our model with two case studies: a short- and a long-lived life history.

3. Population growth rate r is determined by a Lotka style equation in which survival and fertility are averaged over a phenotypic distribution that changes with age. Here, we further decomposed r to show how much it is affected by shifts in phenotypic average as well as variance. We derived the elasticities of r to the first and second derivative of each demographic rate. In particular, we show that the nonlinearity of change in selective pressure with phenotype matters more to population dynamics than the strength of this selection. In other words, the variance of a given trait will be most important when the strength of selection increases (or decreases) nonlinearly with that trait.

4. Inheritance shapes the distribution of newborn phenotypes. Even if newborns have a fixed average phenotype, the variance among newborns increases with phenotypic variance among mothers, strength of inheritance and developmental variation. We explain how the components of inheritance can influence phenotypic variance and thus the demographic rates and population dynamics. In particular, when mothers of different ages produce offspring of different mean phenotype, the inheritance function can have a large influence on both the mean and variance of the trait at different ages and thus on the population growth rate.

5. We provide new tools to understand how phenotypic variation influences population dynamics and discuss in which life histories we expect this influence to be large. For instance, in our short-lived life history, individual variability has larger effect than in our long-lived life history. We conclude by indicating future directions of analysis.

*Correspondence author. E-mail: floriane.plard@ens-lyon.fr

This article forms part of the British Ecological Society journals' Demography Beyond the Population special feature http://wileyonlinelibrary.com/BES_demography

Key-words: growth rate, heritability, individual heterogeneity, inheritance, integral projection model, phenotypic variation, population dynamics, size structure

Introduction

The evolution of a trait through natural selection requires inheritance of the trait, selection on the trait and variability of the trait within a population (Darwin 1871). Individual differences are thus indispensable to understand the mechanisms of trait evolution and how phenotype dynamics influences population dynamics. Different individuals contribute often unequal amounts to population growth (Łomnicki 1978; Kendall *et al*. 2011). Many studies have reported marked differences in fitness components among individuals within a population (Merilä & Sheldon 2000; Steiner & Tuljapurkar 2012; Plard *et al*. 2015b). Despite the strong influence of individual differences on metrics characterizing population dynamics, demographic models often neglect individual differences and focus on population average of demographic rates (see e.g. the matrix models in Caswell 2001). These models often provide a reliable description of population dynamics (Heppell, Caswell & Crowder 2000; Saether & Bakke 2000) but represent an inadequate tool to understand individual differences by answering questions like why they occur, how large they can be as a result of inheritance, ontogenetic change and selection, how they are maintained or at which extent they influence population dynamics (Huston, DeAngelis & Post 1988; Judson 1994). Despite lots of theoretical and empirical studies, how individual variation influences population dynamics is still poorly understood. While most theoretical works performed to date indicate that individual differences have the potential to influence population dynamics (Kendall & Fox 2002; Vindenes, Engen & Sæther 2008; Kendall *et al*. 2011; Vindenes & Langangen 2015) and lots of empirical studies have identified marked individual heterogeneity in demographic rates (Cam *et al*. 2002; Link, Cooch & Cam 2002; Nussey *et al*. 2011), no empirical study has demonstrated yet that individual differences have direct and important influences on population dynamics (Plard *et al*. 2015a). A main reason for this knowledge gap is the lack of models explaining how individual differences, which generally correspond to phenotypic variation, interact with demographic rates to influence population dynamics. The aim of this paper was to provide an analytical model that shows how phenotypic variation is transmitted and maintained, and to explain and quantify the influence of the interaction between phenotypic distribution and demographic rates on population dynamics and in particular on population growth rate.

We do so by analysing integral projection models (IPMs) that can incorporate functional and continuous relationships between demographic rates and one or several individual traits (Easterling, Ellner & Dixon 2000; Ellner & Rees 2006; Coulson, Tuljapurkar & Childs 2010; Coulson 2012; Rees, Childs & Ellner 2014). As a consequence, IPMs reflect the influence of phenotypic variation on demographic rates. IPMs have made possible progress on diverse questions, for example an improved understanding of eco-evolutionary dynamics (Rees *et al*. 2006; Metcalf *et al*. 2009; Smallegange & Coulson 2013; Rees & Ellner 2016), a better management of size-structured populations subject to fishing or hunting (Traill, Schindler & Coulson 2014), or a better understanding of host–parasite dynamics (Metcalf *et al*. 2016). The demographic rates in our IPMs depend on one or several continuous traits, so we can examine how different demographic rates interact to maintain and transmit phenotypic individual variability and create variability as individuals follow different growth trajectories. The sources of variation we consider are described by four components – survival, reproduction, inheritance and growth (as discussed in Coulson, Tuljapurkar & Childs 2010, and illustrated in Coulson *et al*. 2011). For the sake of simplicity, we do not consider other, potentially important, kinds of individual heterogeneity that may be due to, for example, demographic stochasticity (Kendall & Fox 2003), dynamic heterogeneity (Tuljapurkar, Steiner & Orzack 2009), unobserved differences in latent factors such as frailty (Vaupel, Manton & Stallard 1979) or variable environments (Tuljapurkar 1990; Lande, Engen & Saether 2009).

The variability of traits among individuals in a population is often first observed at birth. The distribution of birth phenotypes is determined partly by the transmission of genes (and possibly also of environments, behaviours and so on) from parents to offspring, and partly by stochasticity in the form of developmental noise (unpredictable environmental conditions). The constancy of transmission of variability from parents to offspring is often measured by genetic and non-genetic heritability (Danchin *et al*. 2011; Bonduriansky 2012). In animals, genetic heritability is typically estimated to be low for life-history traits and high for morphological traits (Mousseau & Roff 1987; Kirk *et al*. 2001). In plants, it is difficult to link seeds to parents in the field, so most IPMs for plants ignore inheritance (anonymous motherhood, Caswell 2001) and treat the birth phenotypes as being randomly distributed around (but close to) a fixed trait value. However, the growing literature on heritability in plants (Thomas & Bazzaz 1993; Sadras & Slafer 2012) points the way towards new models of inheritance in plant IPMs. One focus of our analyses is to understand how the choice of a model of inheritance influences the dynamics of traits and, as a consequence, population growth rate.

After birth, individual phenotypes and the distribution of phenotypes in a cohort are shaped by ontogenetic change in traits (via development, growth and plastic

change). Growth can be a source of variability for at least two reasons. Individuals of different birth sizes can differ in their growth rate and in their asymptotic mass/size (Kirkpatrick & Heckman 1989; Björklund 1993; Huchard *et al*. 2014). In addition, individuals of the same birth size can follow different growth trajectories because of stochastic differences in growth due to variable environmental conditions encountered. In addition to ontogenetic change, fertility and viability selection (via phenotype-dependent survival and reproduction) can also influence phenotypic variability. Early in life, directional or stabilizing viability selection can decrease phenotypic variability (Janzen 1993; McAdam & Boutin 2003), whereas disruptive selection can increase variability (Turelli & Barton 1994). Fertility selection determines the phenotypic distribution of parents that produce offspring, as opposed to all individuals who might reproduce, and so determines how much phenotypic variation is available for transmission to offspring. Our model focuses on directional selection on survival, growth and fertility. We also show how phenotypic variability and population dynamics are affected by the strength and shape of growth, fertility and viability selection, measured by the slope and curvature of demographic functions, respectively (for comparative data on these parameters, see, e.g., Kingsolver *et al*. 2001).

In this paper, we begin by introducing the general structure of IPMs and their assumptions. In the second part, we approximate the demographic rates using Taylor expansions and we derive formulas for the mean and the variance of the phenotypic distribution at each age starting from the mean and variance at birth. This enables us to analyse how phenotypic variation is transmitted and maintained and how it interacts with demographic rates. Next we turn to population equilibrium and show how the phenotypic variance at birth at equilibrium depends on the inheritance function and also on other demographic rates. These results are used in the fourth part to make a new decomposition of r that quantifies and distinguishes contributions from the mean of the phenotypic trait, from the phenotypic variance present at birth and from the accumulation of phenotypic variability generated by stochastic growth. Moreover, we obtain the elasticities of r and of these different components to two descriptors of the interaction between phenotypic distribution and demographic rates: the slope and the curvature of each demographic function. Finally, we explore how two different models for the inheritance function influence the mean and variance of the phenotype distribution at birth and population growth rate.

Model and methods

We developed a general analytical model that can be applied to any phenotypic trait linked to demographic rates for any life-history strategy. We derived general conclusions for this model. Demonstrations are presented in the Appendix S1 (Supporting information). Throughout the presentation of our model, we illustrate our formulas with two simulated case studies aiming to reveal the properties, causes and consequences of phenotypic variance across individuals on demographic rates and thereby on population dynamics. The diversity of life histories among and within species is huge (Stearns 1992), but we seek simple and robust insights that can inform more detailed analyses of particular species and/or life histories. We present the two simulated case studies here, before starting the description of our model.

The life cycle shown in the upper panel of Fig. 1 describes our short-lived case study: newborns (age 1) survive and grow to juveniles at age 2; juveniles reproduce, survive and grow to be adults at age 3; and adults reproduce and then immediately die. Such life cycles are often found in short-lived fish or amphibians (Perrone 1978) that lay large numbers of eggs but have low survival of newborns. These life cycles occur for life histories close to the fast end of the slow–fast continuum, which corresponds to the main axis of life-history variation in most taxa studied so far (e.g. Stearns 1992). In contrast, the lower panel of Fig. 1 shows a long-lived species in which newborns grow to be juveniles, which grow to be adults and then live for years as adults, note the self-loop on the adults. Juveniles constitute the 'age class' 2 and the parameters for all adults are lumped into 'age class' 3 because in long-lived species, the adulthood stage usually lasts more than 1 year. This life cycle is typical of a long-lived mammal or bird (Gaillard *et al*. 2000; Coulson 2012) with low reproduction and high survival (but here we assume no decline in adults due to senescence).

GENERAL STRUCTURE AND ASSUMPTIONS

In any population, the contribution of individuals to population growth often differs markedly according to their phenotype. For instance, individuals of high phenotypic quality (i.e. large individuals in good body condition) reproduce more and survive better than individuals of low phenotypic quality. Thus, individuals of the same age (cohort) typically differ in traits characterizing their condition or quality such as body mass or body size, and hence in survival and reproductive performance. In this paper, we consider only one phenotypic dimension, and as shown in Fig. 1, each age class has a distribution of individuals with different trait values. Probability of survival $S_a(z)$ and total reproduction $M_a(z)$ at each age a are deterministic functions of the continuous individual trait z, as sketched in Fig. 1. In contrast, in most matrix models (Caswell 2001), survival and reproductive rates are single numbers parameterized as averages within each of the life cycle age class or stage. We do not consider two-sex models in which fertility selection can differ between males and females (Andersson 1994; Coltman *et al*. 2002; Tatarenkov *et al*. 2008).

The distribution of the continuous traits is likely to change among ages. Although we used body mass as the

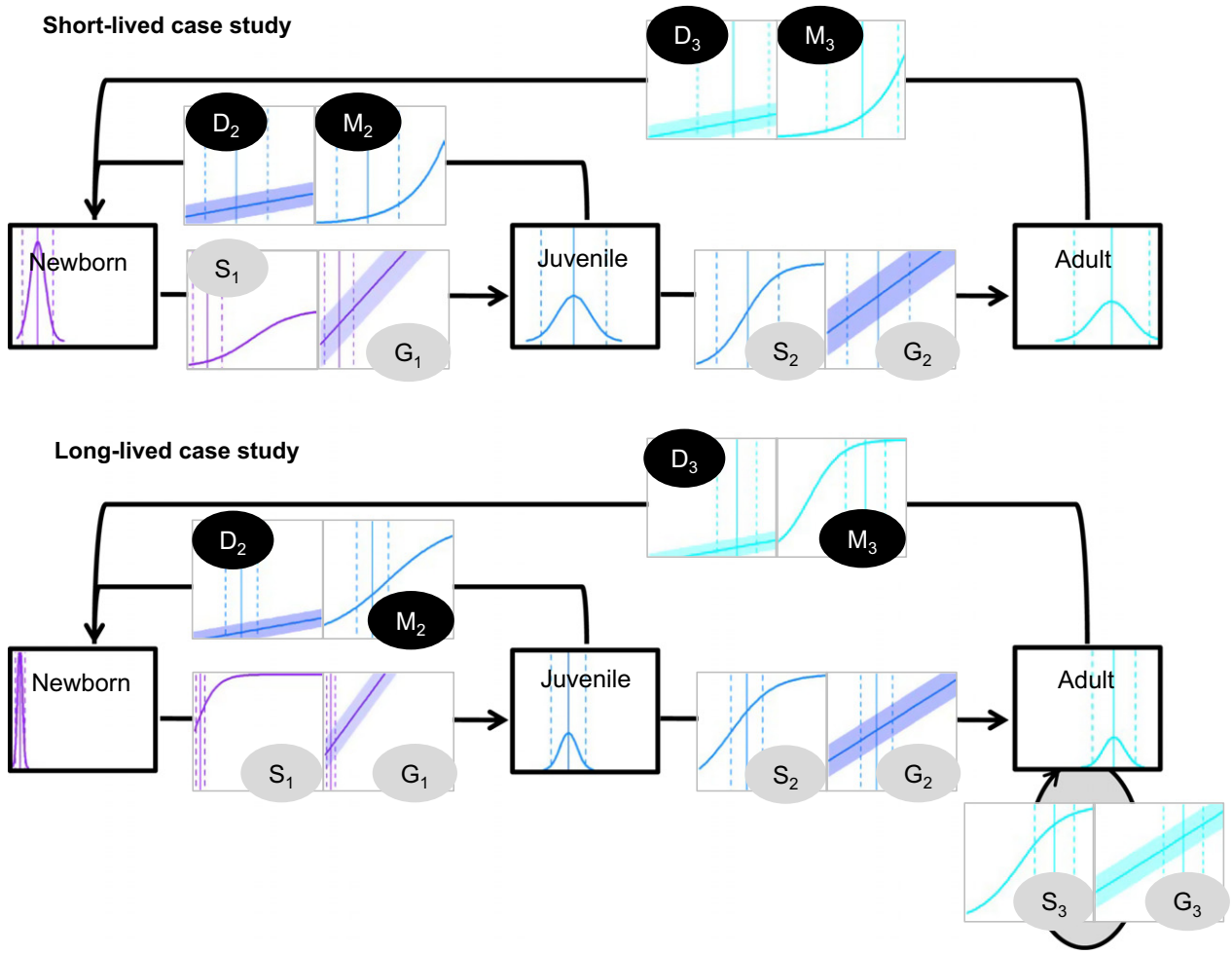

Fig. 1. Life cycles of the short- and the long-lived life histories. Survival (S), reproductive (M), growth (G) and inheritance (D) functions of the different age classes are presented as well as the phenotype distribution of the first age of each age class. The vertical axes for growth and inheritance, and horizontal axes of all functions go from 0 to 20 for the short-lived, and from 0 to 40, for the long-lived life history, respectively. We did not put units as it can be anything. The vertical axes for the reproductive functions go from 0 to 200 (resp. 0–1) for the short-lived (resp. long-lived) life history. In each plot, the solid and the dashed lines show the mean and the variance (mean +/- 2 SD) of the phenotypic distribution, respectively.

focal phenotypic trait in our illustrative case studies because its effect on demographic rates is well-established, our model can be applied to any continuous trait. In our model, ontogenetic change is just growth in body mass. Between ages a and $a + 1$, an individual's mass z changes by $G_a(z)$, but all individuals with the same trait value z at age a do not grow by the same amount, so $G_a(z)$ is really a probabilistic distribution of possible increments whose conditional distribution is the density $G_a(y|x)$ where y and x are individual phenotypes at age $a + 1$ and a, respectively. We assume that at each age, growth has a systematic part that depends on current phenotype, plus a small variance v_G that is the same for all phenotypes: $\bar{G}_a(z) + K_a$, with $\bar{G}_a(z)$ averaged over density $G_a(.|z)$, $\varepsilon\{K_a\} = 0$ and $\mathrm{Var}\{K_a\} = v_G$ for all z.

Lastly, inheritance is described by a function $D_a(y|z)$ that links the size of offspring y and parental size z according to parental age. But two parents of equal size and age can produce offspring of different sizes; hence, D is really a distribution of offspring sizes for each parental age and size. The distributions G and D are illustrated by the shaded regions in Fig. 1. Until the last part of our results, we use the following model (A) of inheritance. The average phenotype of mothers has no effect on the mean size of newborns. Offspring of mothers of different age have the same mean body mass μ_1 independent of mother age or body mass. A mother of mass X_a produces offspring whose masses X_{Oa} are randomly and normally distributed around this mean,

$$X_{Oa} = \mu_1 + \beta(X_a - \mu_{Ma}) + I \qquad \text{eqn 1}$$

$$\varepsilon\{I\} = 0 \qquad \text{eqn 2}$$

$$\mathrm{Var}\{I\} = v_I, \text{ for all mothers.} \qquad \text{eqn 3}$$

where μ_{Ma} is the mean phenotype of mothers of age a. β ($0 \leq \beta < 1$) links mother and offspring body masses, and

v_I is the variance of the inheritance function. β is the strength of the inheritance function; β is related to but not the same as heritability as commonly defined in quantitative genetics (Falconer & Mackay 1996). Indeed, β does not link parent and offspring traits at the same age but links offspring trait to parental trait at the age of reproduction (for further discussion, see Coulson, Tuljapurkar & Childs 2010).

Assembling the pieces, an individual of age a and trait value z has a probability of survival given by $S_a(z)$, the fraction of a, z individuals that survive to age $(a + 1)$. The growth of survivors is a distribution $G_a(y|z)$, the fraction of a, z individuals that changes phenotype from z to y at age $a + 1$. Reproduction by mothers of age a is described by $M_a(z)$, the total number of offspring produced by a mother of age a and phenotype z, and the fraction $D_a(y|z)$ of those offspring which has phenotype y. The dynamics are described by the functions:

$$F_a(y|z) = D_a(y|z)M_a(z), \text{ and } P_a(y|z) = G_a(y|z)S_a(z) \quad \text{eqn 4}$$

where $F_a(y|z)$ and $P_a(y|z)$ are the fertility and the survivorship functions, respectively.

Our analytical results are derived by assuming that the phenotypic distribution at each age is adequately described by its first two moments, the mean phenotype μ_a and the phenotypic variance v_a (we ignore moments of the phenotypic distribution higher than the second). This simple assumption yields useful and robust insights that we check by numerical analyses of a range of examples (for other goals such as increased numerical accuracy, more complex assumptions on moment closure may be useful, e.g. Van Kampen 1992).

For the short-lived simulated case study, the reproductive function was modelled using two parameters (an intercept and a slope with respect to body mass) and a log link because the number of newborns is a count variable. For the long-lived case study, the reproductive function was modelled using a logit link because a mother can have either 0 or 1 young at each reproductive event. For both case studies, the survival function was also modelled using two parameters, an intercept and a slope with respect to body mass, and a logit link. Growth is modelled as a linear function of body mass. Parameter values are reported in the Table S1. Our illustrative and quantitative results will be most useful for animal life histories, but the qualitative results should be useful for plant ecologists as well. Note that our analytical results are not conditioned by the functions we selected for the simulated case studies but consider all functions that are differentiable. The assumptions include a small phenotypic variance and a phenotypic trait normally distributed. Our model can be easily applied to polynomial or other nonlinear functions. The main body of the paper focuses on results, leaving mathematical details to Appendix S1.

PHENOTYPIC VARIANCE AND COHORTS

In this part, we show how phenotypic variation is maintained and transmitted by the survival and the growth functions as individuals age and grow. Consider a cohort of newborns whose phenotype (body mass) has mean μ_1 and variance v_1, as in Fig. 1. In this section, we take these quantities as known; later, we show how these quantities are determined according to the inheritance model. At later ages $a > 1$, the cohort has phenotypic mean μ_a and variance v_a. We consider that the amount of phenotypic variance at each age can affect average survival and growth to the next age; the demographic rates and phenotypic distribution at age a also determine the phenotypic distribution at age $a + 1$.

Average Vital Rates

We begin by showing how variance in the phenotypic trait influences the average vital rates at the same age, using survival rates as an example – identical results are found for growth G and total reproduction M. The survival rate for an individual of age a and phenotype x_a is a function $S(x_a)$. The average survival rate at age a for all individuals is given (approximately) by

$$s_a = S(\mu_a) + \frac{1}{2} v_a S''(\mu_a) \quad \text{eqn 5}$$

where S'' is the second derivative of the survival function evaluated at the mean phenotype. The first term on the right above, $S(\mu_a)$, is the survival rate for the average-phenotype individual (Appendix S1, part 1.2). This equation shows that when a demographic rate changes linearly with the phenotype, we may have strong selection but average rates remain similar for any phenotypic variance. The curvature (S'' for survival) of demographic rates with respect to the phenotypic trait determines how the variance in the trait influences the average demographic rate. When a demographic rate is a convex function of phenotype, for example exponential for $M(x)$ such as in the short-lived life history (Fig. 2b), the second derivative is positive and the mean demographic rate is higher than it will be if there was no phenotypic variance. The opposite is true when the demographic rate is concave in the phenotype, for example the survival function of the second and third age classes of the long-lived life history (Fig. 2e).

The dependence of demographic rates on a phenotype is always captured by the variance of rates among individuals of the same age a. The variance in survival within age class a (and similarly for other vital rates) is

$$v_s = v_a[S'(\mu_a)]^2 \quad \text{eqn 6}$$

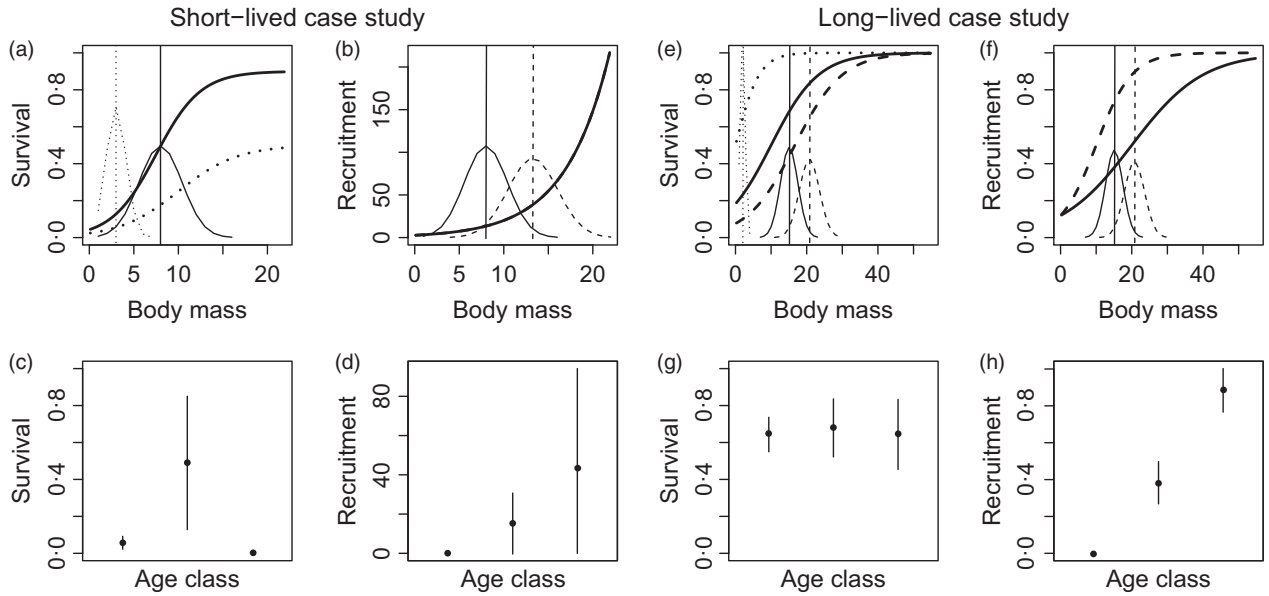

Fig. 2. Functions of survival (a, c, e, g) and reproductive (b, d, f, h) rates of the short (a–d) and long-lived (e–h) life histories. The survival (a, e) and the reproductive (b, f) functions of the different age classes are presented as well as the phenotype distribution of the first age of each age class (dotted lines: newborns, plain lines: juveniles, dashed lines: adults) for the short- and long-lived species. The mean of the survival (c, g) and the reproductive (d, h) functions are presented with segments showing the variance of these functions for each age class (mean +/- 2 standard deviations).

Figure 2 illustrates the changing mean and variance of survival S_a and total reproduction M_a for our two case studies.

Cohort Change in the Mean Phenotype

The average body mass at age $a + 1$ differs from that at age a by two terms: mean growth (g_a, ontogenetic change) of body mass and the effects of selection via survival and growth (Appendix S1, part 1.3). We can express the selective shift in terms of covariances between phenotype and selection (Price 1970; Coulson & Tuljapurkar 2008).

$$\mu_{a+1} - \mu_a = g_a + \frac{v_a S'(\mu_a)(1 + G'(\mu_a))}{s_a}, \qquad \text{eqn 7}$$

where G' and S' are the first derivatives of the growth and the survival functions, respectively, and s_a and g_a are the mean survival and growth at age a (see equation (5)).

The first contribution on the right of (7) is just the average growth increment. The last term is a product of phenotypic variance v_a with the selection gradients in survival and growth. When $G' > 0$ and $S' > 0$ (e.g. large/heavy individuals grow relatively faster and have higher survival rates than small/light individuals), the mean phenotype at age $a + 1$ increases with an increase in phenotypic variance at age a. But the last (covariance) term in (7) is also divided by the average survival so the influence of the phenotypic variance on average demographic rates is relatively low at high survival rates.

The influence of the viability selection on the mean phenotype depends on two properties of the survival function, and also on the variance (v_a) of the phenotypic distribution before survival acts. The two properties of the survival function that matter are the strength of selection ($S'(\mu_a)$) and the curvature of the survival function ($S''(\mu_a)$) (recall that the curvature affects the average survival s_a, (5)). Figure 3a illustrates three survival curves of very different curvatures that act on the phenotypic distributions at age 1 with equal mean μ_1 and small or large variance (i.e. width). The resulting phenotype distribution, with mean μ_2 and variance v_2, is shown in Fig. 3b (for the case of small v_1) and in Fig. 3c (for the case of large v_1).

Adults vs. Parents

The total number of offspring produced by an adult of phenotype z at age a is $M_a(z)$. In a model with discrete classes, the number of adults in age class a, stage class z, at time t is $n(a, z, t)$; these adults have a mean phenotype μ_a. The phenotypic distribution of actual mothers is proportional to the weighted product $M_a(z)n(a, z, t)$. Thus (in analogy with (7)), fertility selection shifts the mean phenotype of mothers aged a to

$$\mu_{Ma} - \mu_a = \frac{v_a M'_a(\mu_a)}{m_a} \qquad \text{eqn 8}$$

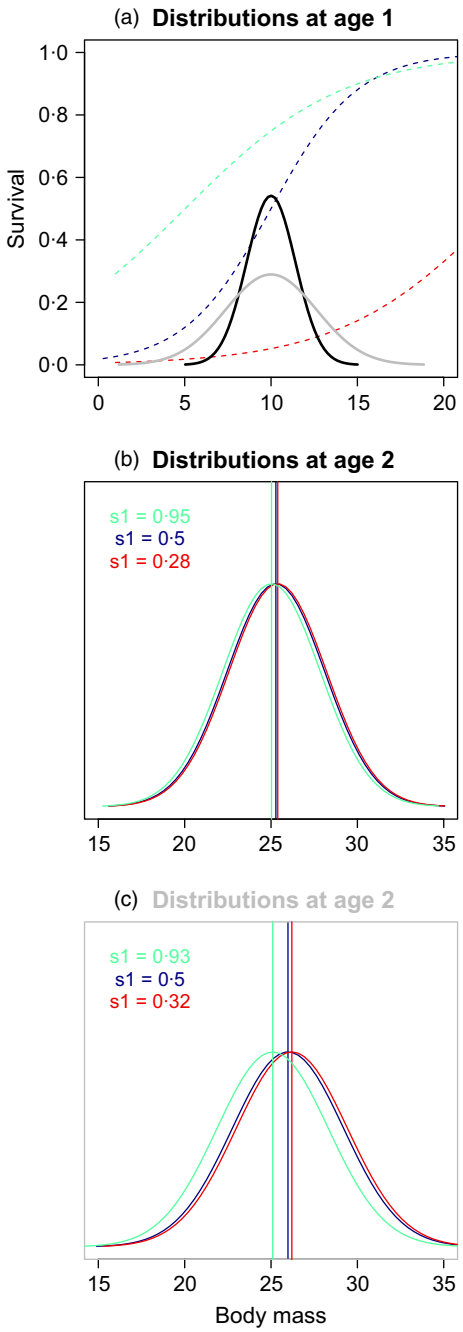

Fig. 3. Influence of the shape of the survival function on the mean and the variance of the phenotypic distribution. (a) Two phenotypic distributions at age 1 (black and grey) are presented with different variance. The functions represented with different colours show different survival functions. (b, c) The viability selection has acted on the black (b) and grey (c) distributions to give the phenotypic distributions at age 2 according to the shape of the survival functions presented in (a). s1 is the average survival rate of the individuals at age 1.

where M'_a is the first derivative of M_a and m_a is the mean recruitment at age a. In this way, fertility selection determines the phenotypes of mothers and hence of offspring.

Cohort Change in the Phenotypic Variance

A cohort starts out as newborns with phenotypic variance v_1, which may potentially change with age to v_a due to selection, and will change as the stochasticity of growth injects variance into the phenotypic distribution. Our analytical results show, surprisingly, that survival selection (including a quadratic dependence of survival on size) has little effect on phenotypic variance at each age (see Appendix S1, part 1.4). But strong survival selection on the tails of the phenotypic distribution (e.g. truncation selection) could certainly have an effect; we do not consider such effects here. So the changes in cohort phenotypic variance with age are mainly due to growth.

As age a increases, the growth uncertainty v_G accumulates. Also, between ages a and $a + 1$, selection via growth changes the phenotypic variance at a by the factor

$$\gamma_a = (1 + G'(\mu_a))^2 \qquad \text{eqn 9}$$

Thus, phenotypic variance changes with age according to

$$v_{a+1} = \gamma_a v_a + v_G \qquad \text{eqn 10}$$

Variability in growth (measured by v_G) always increases the phenotypic variance. Positive directional selection on growth ($G' > 0$) means that $\gamma_a > 1$ and so the variance among newborns expands when they get older. However, a negative directional selection that can eventually occur when the growth is defined by a nonlinear function in the case of compensatory growth or shrinkage selection, for instance, decreases the variance among ages.

Figure 4 illustrates how the growth function interacts with the phenotypic distributions at age 1 to produce a new phenotypic mean and variance at age 2. In the left-hand column, (a), two different phenotypic distributions (black and grey) at age 1 are submitted to three growth functions (coloured functions) with increasing selection (i.e. slope) but the same variability (i.e. a band of the same width v_G). The phenotypic distributions at age 1 are characterized by equal mean μ_1 and small (black) or large (grey) variance (i.e. width). The resulting phenotype distributions, with mean μ_2 and variance v_2, are shown in panels b (for the case of small variance at age 1, black) and c (for the case of large variance at age 1, grey). In the right-hand column, (d) growth has fixed slope (strength of selection) but increasing variability (three values of v_G). The rest of the right-hand column is arranged as on the left. The reader can at once see how the variance of the phenotypic distribution at age 1 influences both the mean and the variance of the phenotypic distribution at age 2 and how this depends on the growth function (see also equations (7) and (10)).

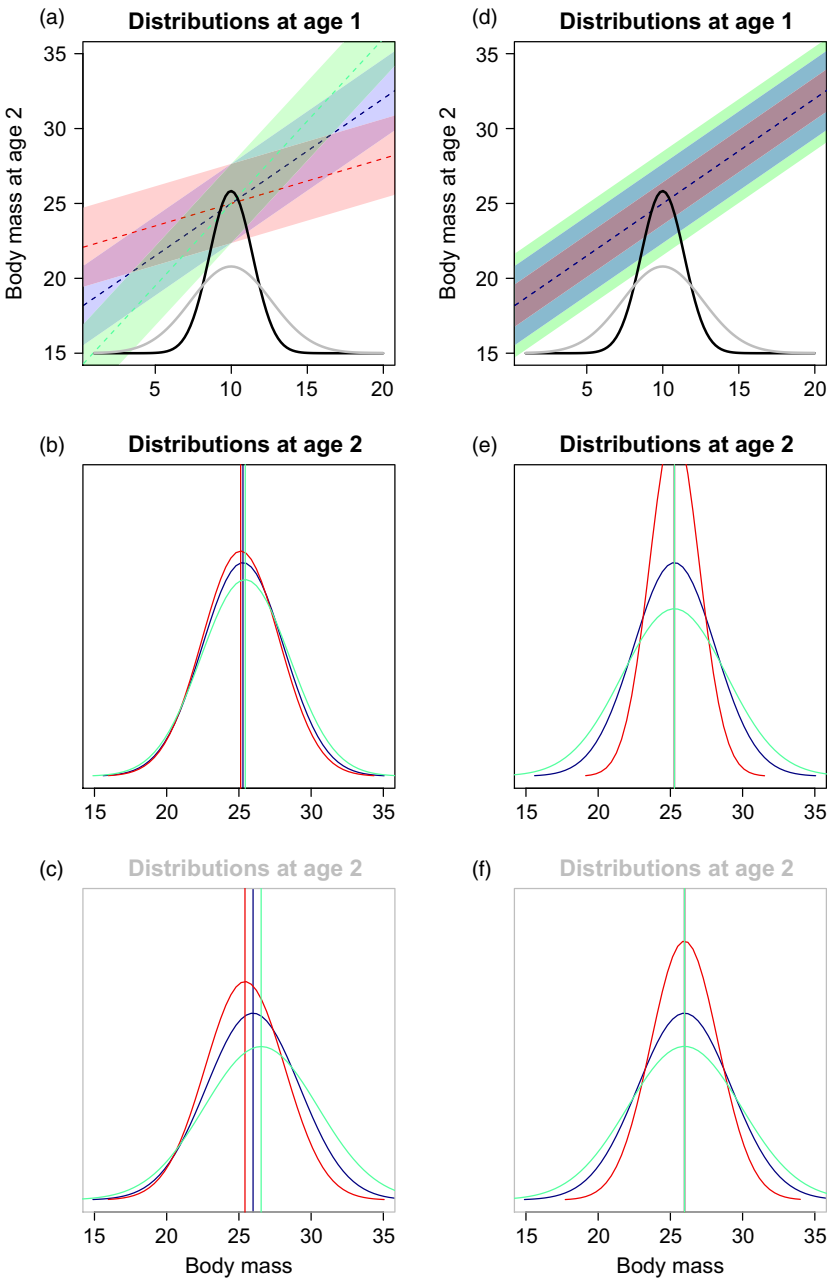

Fig. 4. Influence of the shape of the growth function on the mean and the variance of the phenotypic distribution. (a, d) Two phenotypic distributions at age 1 (black and grey) are presented with different variance. The functions represented with different colours show different growth functions. The colour shade represents the uncertainty around each growth function (v_G). (b, c, e, f) The black (b, e) and grey (c, f) distributions at age 1 have grown to give the phenotypic distributions at age 2 according to the growth functions presented in a and d.

DETERMINING PHENOTYPIC VARIANCE AT BIRTH

In the previous section, we assumed that the variance at birth v_1 is known as we have presented the dynamic of the mean and the variance of a phenotypic distribution from one age to the next, depending on its mean and its variance at birth. In this part, we can now turn to populations at equilibrium and we have thus to solve v_1 at equilibrium. Remember that μ_1 is always assumed to be known in our model A of inheritance. Assuming density independence, we consider a stable population that has stationary age-phenotype structure and growth rate r. We can apply our results immediately to a density-dependent setting where any stable population has a stationary age-phenotype structure but $r = 0$ by including density in each function describing the demographic rates. We could obtain the entire phenotypic distribution at every age, and the growth rate, by iteration of the full age-stage projections, as in Ellner & Rees (2006) and Coulson (2012). But, to reiterate, we seek analytical insights into the factors determining the phenotypic variance, and the relationship between phenotypic variation and population dynamics.

The average survival rate s_a at each age a from equation (5) is an average over the phenotypic distribution at that age. In the same way, we can define an average reproduction m_a at each a, and an average survivorship l_a at age a as

$$l_a = s_{a-1}s_{a-2}\ldots s_1, l_1 = 1 \qquad \text{eqn 11}$$

The average total number of offspring produced by the survivors of a cohort at age a is

$$\phi_a = m_a l_a, \text{ with } R_0 = \sum_a \phi_a \qquad \text{eqn 12}$$

The phenotypic variance of newborns is shaped by inheritance, which is also influenced by environmental conditions. In our model A of inheritance, the mean offspring size is fixed at μ_1, but the variance among offspring (see equation (3)) depends on the phenotypic variance among mothers (details in Appendix S1, part 3). Considering mothers of all ages, we find that

$$v_1 = \beta^2 \frac{1}{R_0} \sum_a \phi_a v_a + v_I, \qquad \text{eqn 13}$$

A strength of inheritance $\beta > 0$ means that offspring variance is a fraction β^2 of the total variance among mothers.

The variances v_a, in turn, depend on v_1 as shown by equation (10);

$$v_a = \Gamma_a v_1 + k_a v_G, \qquad \text{eqn 14}$$

where the coefficients are defined in the Appendix S1, part 1.4. Then,

$$v_1 = \frac{\beta^2 v_G \{\sum_a (\phi_a / R_0) k_a\} + v_I}{1 - \beta^2 \{\sum_a (\phi_a / R_0) \Gamma_a\}} \qquad \text{eqn 15}$$

Because ϕ_a, k_a and Γ_a depend on v_1, we must solve this equation iteratively. As the general solution is complex and thereby, not informative in the general case, we looked for what happens in special cases.

First, suppose there is no strength of inheritance $\beta = 0$ and $v_I = 0$, so that each newborn has the same size μ_1 and $v_1 = 0$. Although this case is highly unrealistic, this is the common, though rarely stated, assumption made by many structured models (e.g. many of the models presented by Caswell 2001). Secondly, make the slightly more general assumption that $\beta = 0$ but there is some random offspring variance $v_I > 0$; now variance among offspring is $v_1 = v_I$ independent of variance among parents. This assumption is used in many plant IPM models (Rees & Ellner 2009).

In practice, we expect that the strength of inheritance is positive ($\beta > 0$) and $v_I > 0$. It is obvious from (15) that v_1 increases with v_I and with v_G. In most life histories, v_1 increases also with the strength of inheritance β as illustrated for our short-lived case study in panel (e) of Fig. S3.

The strength of inheritance acts as a filter on the transmission of parental phenotypic variance into offspring variance. Parental variance is determined by the joint effects of growth uncertainty (v_G) and selection (via growth and survival). In our two simulated case studies, the strength of inheritance $\beta = 0{\cdot}2$ and the phenotypic variances v_1 is small. Figure S4 (B,D) presents a case where there is no selection (slopes of survival, reproductive and growth function equal 0) and no stochasticity in growth ($v_G = 0$). Differences between black and red points show the fertility selection. In this case, the diversity of body masses is created only by the stochasticity in the inheritance function (v_I) and this variance at birth remains the same as the cohort ages. Panels A and C present our two case studies including growth stochasticity and selection.

INFLUENCE OF PHENOTYPIC VARIATION ON *r*

We use all the previous results to propose a new decomposition of r and the elasticities of r to the slope and the curvature of each function.

Decomposition of r

Our model leads to the familiar equation for the stable growth rate r (see Appendix S1, part 1.1 for details),

$$1 = \sum_a e^{-ra} \phi_a = \sum_a e^{-ra} m_a l_a \qquad \text{eqn 16}$$

This is the Lotka equation – but the 'vital rates' here are averages over a phenotypic distribution that changes with age. For example, equation (5) shows how phenotypic mean and variance affect the average survival rate s_a. Our analyses thus far show how the phenotypic mean and variance are shaped by survival and growth, and by the (so far unknown) variance v_1 among newborns.

The population growth rate r depends on phenotypic variance at each age. To better understand this linkage, we analytically decompose r into three terms, one shaped by the mean phenotype distribution ($\widehat{r}$), a second proportional to the variance created by the stochasticity of the growth function ($\widetilde{r}$) and a third proportional to the variance at birth ($\check{r}$),

$$r = \widehat{r} + v_G \widetilde{r} + v_1 \check{r} \qquad \text{eqn 17}$$

See Appendix S1, part 2 for proofs. This analytical decomposition assumes that v_1 and v_G are small enough that we can neglect nonlinear terms in these variances.

This decomposition is illustrated with our two simulated case studies in Fig. 5. The values for the short- and long-lived life histories, respectively, are as follows: $\widehat{r}$ represents 51% and 101% of r; the term $v_G \widetilde{r}$ is 21% and –2% of r; and $v_1 \check{r}$ is 28% and 1% of r. Clearly phenotype variance matters more to the short-lived than to the long-lived life history. Part of the reason is that there is more phenotype variation in the juvenile and mainly adult age classes in short-lived vs. long-lived life histories (coefficients of variation are 0·38 vs. 0·34 at age 1 and 0·30 vs. 0·16 at age 2, respectively). More important is the difference in the curvature with respect to the phenotype z of the survival $S_a(z)$ and total reproduction $M_a(z)$ functions between the

short-lived and the long-lived life histories, as shown in Fig. 2. Over the phenotypic ranges occupied by individuals of different ages, we can see that survival (for age 2) and especially fertility are convex for the short-lived case study, but linear or concave for the long-lived case study. Recall (from (5)) that convexity (resp. concavity) means that variance increases (resp. decreases) average demographic rates. For example, the mean total reproductive rate for adults in the short-lived case study is 44 when we include phenotypic variance (as in (5) applied to M) vs. 38 excluding the contribution of phenotype variance (compared to 0·88 vs. 0·89 for the long-lived case study, compare third age class of panels d and h in Fig. 2).

Elasticities of r

The decomposition of r allowed us to look at the influence of the shape of the relationships between individual phenotypic and demographic rates on the different components of r ($\widehat{r}$, $\widetilde{r}$, v_1 and $\check{r}$). We computed the elasticities (Appendix S1, part 4) of r and its components to perturbations in the following: the survival, reproductive and growth rates and their first and second derivatives; the variance of the growth, v_G, and the inheritance, v_I functions; and β. In particular, this allowed us to compare the influence of the strength of the selection (first derivative) to the influence of the curvature (second derivative) of the different functions on r and its components.

The elasticities of r and its components are shown for the short-lived case in Fig. S1 and the long-lived case in Fig. S2. We focus on the elasticities of r, shown along the first row of each figure. Intuition from traditional demography suggests that a short life span with low newborn survival should result in a higher elasticity of r to newborn survival rate than for long life with high newborn survival (Heppell, Caswell & Crowder 2000). This is borne out by the elasticities of r to survival rates (computed at the average newborn phenotype). What is new here is that we can compute the elasticities of r to the strength of selection (i.e. to the slope) or to the curvature (i.e. second derivative) of the survival/fertility/growth functions. We find that the elasticities of r and its components to curvature of demographic rates are higher at each age than elasticities to their slopes in both case studies. For instance, the elasticities of r linked to the curvature and the slope of the survival function are 4·66 and 0·43, and 3·60 and 1·39 for the juvenile age class in the long-lived and the short-lived life histories, respectively. The elasticities of r linked to the curvature of the reproductive function are 1·45 and 0·06 for adult age class in long- and short-lived life histories, respectively. The elasticity of r linked to the slope of the reproductive function is 0 because μ_1 is fixed (assumption of the inheritance model A).

Despite the low influence of the growth function on r compared to the survival or reproductive functions (Figs S1 and S2), the growth function is the only one that directly influences the phenotype variance v_a (recall (10)) in these case studies. The elasticities show that the curvature of the growth function can have higher impact than the slope of the function (elasticities of r linked to the curvature and the slope of juvenile growth in the long-lived life history are 0·21 and 0·01, respectively). Finally, as we might expect according to the simple model of inheritance, β and v_I have only weak effects on r and its components.

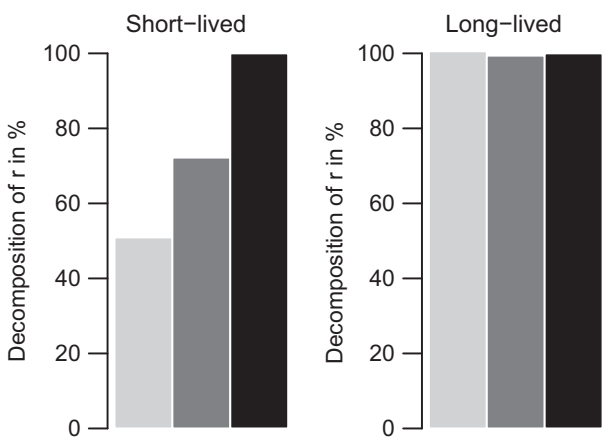

Fig. 5. Decomposition of the population growth rate r as in text equation (17) into three terms, one shaped by the mean phenotype distribution ($\widehat{r}$), a second proportional to the variance created by the stochasticity of the growth function ($v_G\widetilde{r}$) and a third proportional to the variance at birth ($v_1\check{r}$). The bars indicate values of the following: $\widehat{r}$ in light grey; $\widehat{r}+v_G\widetilde{r}$ in dark grey; $r=\widehat{r}+v_G\widetilde{r}+v_1\check{r}$ in black.

EXPLORING A DIFFERENT MODEL FOR THE INHERITANCE FUNCTION

Here, using our two simulated case studies, we explore another model for the inheritance function largely based on existing IPMs for animals such as deer (Plard *et al.* 2015a) and sheep (Coulson 2012). This second model is more flexible and thus closer to the field reality than model A. In this model B, μ_1 is variable and unknown. We must now thus solve both μ_1 and v_1. For the sake of analytical simplicity, solving is done numerically here, instead of using an analytical approach as we did for model A. We compare the model A of inheritance to a second model (B) in which we suppose that a mother's body mass has a direct effect on offspring mass, so that offspring of mothers of different age have different mean body mass,

$$X_{Oa} = \alpha_a + \beta X_a + I \qquad \text{eqn 18}$$

$$\varepsilon\{I\} = 0 \qquad \text{eqn 19}$$

$$\mathrm{Var}\{I\} = v_I, \text{ for all mothers.} \qquad \text{eqn 20}$$

where α_a is the intercept of the model, and X_{Oa} and X_a are the offspring and the mother body mass, respectively.

In model B, the mean phenotype μ_{Oa} of offspring depends on the age a of mothers,

$$\mu_{Oa} = \alpha_a + \beta\left[\mu_a + \frac{v_a M'_a}{m_a}\right], \qquad \text{eqn 21}$$

where we use (8) in the square-bracketed term to include the effects of fertility selection. The equation for v_1 has also to be modified to include the variance among offspring between mothers of different ages, so in the sum of the equation (13), we must replace v_a by $v_a + (\mu_{Oa} - \mu_1)^2$.

Inheritance model A takes the average size of newborns to be fixed, and the only effect of increasing β (or v_I) is to increase phenotypic variance (Fig. S3a–e). But inheritance has larger effect on the phenotypic distribution of newborns when the average offspring phenotype changes with the average phenotype of mothers such as in model B ((21), Fig. S3f–j). If older mothers are on average larger mothers, then the offspring of older mothers will on average be larger than the offspring of younger mothers. So model B leads to an increase in both the average μ_1 and the variance v_1 of newborn phenotypes. This matters to average newborn survival when newborn survival rate (and/or growth rates) has a high positive slope and/or high curvature (positive second derivative) with respect to phenotype.

An increase in newborn body mass from 2 to 3 produces only a 10% increase in newborn survival for the long-lived life history, but produces a 31% increase in newborn survival for the short-lived life history. Also in the long-lived as compared to short-lived life history, mean body mass of mothers does not change much with age. When inheritance follows model B, these two effects (shape of the survival function and difference among mean mass of mother of different age) make the dynamics of the short-lived life history very sensitive to the value of inheritance β. With model B, r doubles when β increases from 0·1 to 0·4 (panel H compared to panel C). It is thus critical to accurately model the inheritance function to reflect correctly the dynamics of the observed phenotype.

Discussion

Our goal here is to understand the forces that shape and maintain phenotypic variation across individuals, and to assess the effect of standing variation on population dynamics. We showed analytically how mean and variance of a phenotypic trait within a cohort change with age as a result of growth and selective disappearance acting on available phenotypic variance (via differential survival/growth of individual phenotypes). We proposed a decomposition of r that allowed quantifying the contribution of the mean and variance of a phenotypic trait and showed that the influence of phenotypic variation is much higher in our short-lived compared to our long-lived case studies. Moreover, our elasticity analysis showed that the shape (curvature) of the function linking demographic rates with the phenotypic trait has a much higher influence on the population growth rate than the strength (slope) of this function.

This model allows to understand under what mechanisms a continuous trait interacts with demographic rates to predict (i) the dynamic of the continuous trait over age, (ii) the influence of phenotypic variation on demographic rates and (iii) on population growth rate according to the demographic rates characterizing a given life history. The change in phenotypic variance with age, interestingly, is largely independent of differential survival unless we have strong selection in the tails of the phenotypic distribution due to truncation, or a nonlinear term in $(X - mu)^4$, but quadratic nonlinearities have surprisingly little effect. Instead, phenotypic variance is shaped primarily by growth. Unpredictability in growth (i.e. individuals of the same age and size grow by different amounts) injects phenotypic variance at every growth step, whereas differential growth can cause variance to grow with age or decline, depending on the direction of selection. Positive directional selection on fertility at any age makes the average size of mothers larger than the age class average, but has little effect on the phenotypic variance.

SO, WHEN DOES VARIABILITY MATTER?

Our model showed that variability can play an important role in population dynamics when the survival or the reproductive rates are low or when the curvature of these demographic rates is large. The influence of individual variability on population dynamics increases when the growth function contributes to increase the phenotype variance across ages, for example if individual phenotypes diverge as individuals grow. It can be the cases when heavy born individuals grow faster than light born individuals. Finally, the nature of inheritance shapes the phenotypic distribution of newborns. In the simplest case (our model A), the average size of newborns is fixed but the phenotypic variance among newborns increases with the phenotypic variance among mothers, with strength of inheritance and with the magnitude of developmental variation. Inheritance has a larger effect, changing both the mean and variance among newborn phenotypes when mothers of different ages produce offspring of different mean size (our model B).

The influence of individual phenotypic variation on population dynamics is thus expected to be larger in short-lived life histories (as illustrated by our case study close to the fast end of the slow–fast continuum (Stearns 1992) characterized by large clutch size/number of seedlings per capita and low early survival) than in long-lived life histories (as illustrated by our case study close to the slow end of the slow–fast continuum like ungulates, or seabirds, Prince *et al.* 1994; Coulson, Tuljapurkar & Childs 2010; Plard *et al.* 2015a). Other species display

life-history strategies that differ from the ones presented in our case studies. For instance, in primates with a long juvenile period (Dobson & Lyles 1989), the influence of individual variation is expected to be similar to our long-lived case study except if the growth rate diverges largely between light and heavy individuals. Long-lived plants such as sequoia can show long duration of the growth phase, exponential reproduction and low seed survival (Baudisch *et al*. 2013; Zambrano & Salguero-Gomez 2014). In this case, the long juvenile period combined with an exponential selection can amplify the effect of individual variability on population dynamics. This can also be the case in fish or reptiles characterized by indeterminate growth and variable reproductive performance (Felix, Vinagre & Cabral 2011). In these species with a large potential effect of individual differences, our study shows that the model of the inheritance function must reflect correctly the phenotype transmission. Until now, all IPMs on plants have modelled offspring phenotype independently of mother phenotype (Miller *et al*. 2012; Gonzalez, Rees & Martorell 2013; Zambrano & Salguero-Gomez 2014). This can be especially problematic if mothers of different ages or stages produce offspring with different phenotypes, a case that seems to be the rule in many wild populations (Venable 1992; Fox & Czesak 2000; Ericsson *et al*. 2001; Benton, St Clair & Plaistow 2008; Kindsvater, Rosenthal & Alonzo 2012). We have not analysed processes that generate a difference among mothers of different ages, but a recent study (without phenotypic differences) demonstrated that different optimal offspring size can evolve according to mother stage and reproductive value if a trade-off exists between offspring size and mother survival (Kindsvater & Otto 2014). As a consequence, more attention should be devoted to the inheritance function when building a model.

SHOULD WE STOP USING MATRIX MODELS?

In a matrix model, demographic rates are averaged over many individuals and thus implicitly include individual heterogeneity: the mean demographic rates are correct even if the model does not track differences between individuals. Taking into account, supplemental individual heterogeneity makes almost no improvement in many matrix models (Rees *et al*. 1999; Coulson 2012; Plard *et al*. 2015a). Thus, matrix models are certainly useful and valuable in many contexts. However, the mean demographic rates are influenced both by trait means and trait variances: the average demographic rate is generally different from the demographic rate value at the trait mean (because of Jensen's inequality, illustrated, e.g., by Ruel & Ayres 1999). Hence, matrix models with fixed mean rates (by stage and age class) cannot reflect changes over time in trait distributions. Our goal is not to argue against matrix models – indeed, many IPMs can be and are cast as matrix models. Rather, our goal is to understand how individual differences are maintained in a population and how they influence population dynamics in interaction with demographic rates.

LIMITATIONS OF THE MODEL

The main assumption of our analytical results – that phenotype variance is small – is reasonable for many species. For instance, coefficient of variation in our short-lived species was 0·3, whereas it is <0·1 in wild populations of cichlids and guppies (Perrone 1978; van Wijk *et al*. 2013). For long-lived life histories, the coefficient of variation at age 3 was 0·14 in our baseline case, compared with 0·12 and 0·15 in populations of Soay sheep and roe deer, respectively (Coulson, Tuljapurkar & Childs 2010; Plard *et al*. 2015a). This assumption allowed us to decompose the influence of the mean and the variance of the phenotype on each demographic rate to understand how they interact to mediate the influence of individual variability on population dynamics. However, if this interaction creates very large variance, our decomposition and so our simplification may not correctly capture the dynamics of the mean and variance of the phenotype.

In many cases, the distribution of phenotypes such as body mass or size can be modelled using normal distributions, for which our analytical results will apply. However, growth is a multiplicative process that can make the distribution of some continuous traits log-normal (Calder 1984; Graham *et al*. 2003). In this case, the present analysis may be extended to describe the dynamics of a log-normally distributed trait. Finally, the model has been derived for simple inheritance functions. But more complex inheritance functions can be dealt with using numerical methods. Moreover, we present a simple model that allows us to give first results, but this model can be expanded to include density dependence, sex differences, complex population structures or environmental stochasticity (Rees & Ellner 2009; Coulson 2012; Schindler *et al*. 2013; Childs, Sheldon & Rees 2016).

FUTURE DIRECTIONS

This model can be applied either on populations at equilibrium or to model transitory dynamics as it provides the distribution of continuous traits as individuals get older. Individual variation is often maintained or produced by variable environments (Merilä, Sheldon & Kruuk 2001; Vindenes & Langangen 2015). Using this model, one can investigate through which demographic rates, variation of environmental conditions influences individual variability and population dynamics. A first way to do it is to use different demographic functions when environment changes and a second way is to extend this model to include stochastic environment. These applications could allow understanding how individual variability is maintained and created (as individuals follow different growth trajectories): by the dynamics

of cohorts born to different environment (Kendall *et al.* 2011) or by the divergence of individuals as they get older in different environments, for instance. Using inverse integral projection models and method derived to check posterior distributions of data now also allows us to determine whether a model correctly captures the population dynamics (González, Martorell & Bolker 2016) when comparing different hypotheses. Another application would be to investigate how a population adapts to a new environment (which can even conduct to speciation; Pfennig *et al.* 2010). Even within a same species, adaptation to particular environments can lead phenotypes to differ between populations. A nice example is the difference of size between populations living on islands and on continent (Lomolino 1985). The present model allows understanding if two populations are under different selective pressures or if this is the change in phenotype under directional selection that has influenced all demographic rates. This model can also be extended by adding trade-offs between functions as they are of main importance to understand how variability is maintained in a population (Stearns 1992). Finally, this model investigates the influence of individual variability on population dynamics but does not include individual variability in their response to different selective pressures. An interesting extension of this model would be to include variable individual intercept and slope, for instance, for each function to analyse how variable individual responses can influence population dynamics.

CONCLUSION

Phenotypic variation is expected to have large influence in species characterized by large reproductive rates and demographic rates with strong curvature. This works is a first step to understand how demography works beyond the population, but many empirical studies are still needed to understand the influence of individual differences on population dynamics within the diversity of life histories and environmental conditions (Griffith *et al.* 2016).

Acknowledgements

FP and ST were partly supported by NIH R24AG039345. TC acknowledges the support of the ERC and of NERC. We thank Roberto Salguero-Gómez and two anonymous reviewers for constructive comments on a previous version of the manuscript.

Data accessibility

This paper does not use data.

References

Andersson, M.B. (1994) *Sexual Selection*. Princeton University Press, Princeton, NJ, USA.

Baudisch, A., Salguero-Gómez, R., Jones, O.R., Wrycza, T., Mbeau-Ache, C., Franco, M. & Colchero, F. (2013) The pace and shape of senescence in angiosperms. *Journal of Ecology*, **101**, 596–606.

Benton, T.G., St Clair, J.J.H. & Plaistow, S.J. (2008) Maternal effects mediated by maternal age: from life histories to population dynamics. *Journal of Animal Ecology*, **77**, 1038–1046.

Björklund, M. (1993) Phenotypic variation of growth trajectories in finches. *Evolution*, **47**, 1506–1514.

Bonduriansky, R. (2012) Rethinking heredity, again. *Trends in Ecology & Evolution*, **27**, 330–336.

Calder, W.A. (1984) *Size, Function, and Life History*. Harvard University Press, Cambridge, MA, USA.

Cam, E., Link, W.A., Cooch, E.G., Monnat, J.Y. & Danchin, E. (2002) Individual covariation in life-history traits: seeing the trees despite the forest. *The American Naturalist*, **159**, 96–105.

Caswell, H. (2001) *Matrix Population Models: Construction, Analysis, and Interpretation*, 2nd edn. Sinauer Associates, Sunderland, MA, USA.

Childs, D., Sheldon, B. & Rees, M. (2016) The evolution of labile traits in sex- and age-structured populations. *Journal of Animal Ecology*, **85**, 329–342.

Coltman, D.W., Festa-Bianchet, M., Jorgenson, J.T. & Strobeck, C. (2002) Age-dependent sexual selection in bighorn rams. *Proceedings of the Royal Society of London B: Biological Sciences*, **269**, 165–172.

Coulson, T. (2012) Integral projections models, their construction and use in posing hypotheses in ecology. *Oikos*, **121**, 1337–1350.

Coulson, T. & Tuljapurkar, S. (2008) The dynamics of a quantitative trait in an age-structured population living in a variable environment. *The American Naturalist*, **172**, 599–612.

Coulson, T.N., Tuljapurkar, S. & Childs, D.Z. (2010) Using evolutionary demography to link life history theory, quantitative genetics and population ecology. *Journal of Animal Ecology*, **79**, 1226–1240.

Coulson, T., MacNulty, D.R., Stahler, D.R., B., Wayne, vonHoldt, R.K. & Smith, D.W. (2011) Modeling effects of environmental change on wolf population dynamics, trait evolution, and life history. *Science*, **334**, 1275–1278.

Danchin, E., Charmantier, A., Champagne, F.A., Mesoudi, A., Pujol, B. & Blanchet, S. (2011) Beyond DNA: integrating inclusive inheritance into an extended theory of evolution. *Nature Reviews Genetics*, **12**, 475–486.

Darwin, C. (1871) *The Descent of Man and Selection in Relation to Sex*. John Murray, London, UK.

Dobson, A.P. & Lyles, A.M. (1989) The population dynamics and conservation of primate populations. *Conservation Biology*, **3**, 362–380.

Easterling, M.R., Ellner, S.P. & Dixon, P.M. (2000) Size-specific sensitivity: applying a new structured population model. *Ecology*, **81**, 694–708.

Ellner, S.P. & Rees, M. (2006) Integral projection models for species with complex demography. *The American Naturalist*, **167**, 410–428.

Ericsson, G., Wallin, K., Ball, J. & Broberg, M. (2001) Age-related reproductive effort and senescence in free-ranging moose, *Alces alces*. *Ecology*, **82**, 1613–1620.

Falconer, D.S. & Mackay, T.F.C. (1996) *Introduction to Quantitative Genetics*. Robert MacLehose and Company Limited, Glasgow, UK.

Felix, P.M., Vinagre, C. & Cabral, H.N. (2011) Life-history traits of flatfish in the Northeast Atlantic and Mediterranean Sea. *Journal of Applied Ichthyology*, **27**, 100–111.

Fox, C.W. & Czesak, M.E. (2000) Evolutionary ecology of progeny size in arthropods. *Annual Review of Entomology*, **45**, 341–369.

Gaillard, J.M., Festa-Bianchet, M., Yoccoz, N.G., Loison, A. & Toïgo, C. (2000) Temporal variation in fitness components and population dynamics of large herbivores. *Annual Review of Ecology and Systematics*, **31**, 367–393.

Gonzalez, E.J., Rees, M. & Martorell, C. (2013) Identifying the demographic processes relevant for species conservation in human-impacted areas: does the model matter? *Oecologia*, **171**, 347–356.

González, E.J., Martorell, C. & Bolker, B. (2016) Inverse integral projection models: using time series of population-level data to infer population dynamics. *Methods in Ecology and Evolution*, **7**, 147–156.

Graham, J.H., Shimizu, K., Emlen, J.M., Freeman, D.C. & Merkel, J. (2003) Growth models and the expected distribution of fluctuating asymmetry. *Biological Journal of the Linnean Society*, **80**, 57–65.

Griffith, A., Salguero-Gómez, R., Merow, C. & McMahon, S. (2016) Demography beyond the population. *Journal of Ecology*, **104**, 271–280.

Heppell, S., Caswell, H. & Crowder, L. (2000) Life histories and elasticity patterns: perturbation analysis for species with minimal demographic data. *Ecology*, **81**, 654–665.

Huchard, E., Charmantier, A., English, S., Bateman, A., Nielsen, J.F. & Clutton-Brock, T. (2014) Additive genetic variance and developmental

plasticity in growth trajectories in a wild cooperative mammal. *Journal of Evolutionary Biology*, **27**, 1893–1904.
Huston, M., DeAngelis, D. & Post, W. (1988) New computer-models unify ecological theory - computer-simulations show that many ecological patterns can be explained by interactions among individuals. *BioScience*, **38**, 682–691.
Janzen, F.J. (1993) An experimental-analysis of natural selection on body size of hatchling turtles. *Ecology*, **74**, 332–341.
Judson, O. (1994) The rise of the individual-based model in ecology. *Trends in Ecology & Evolution*, **9**, 9–14.
Kendall, B. & Fox, G. (2002) Variation among individuals and reduced demographic stochasticity. *Conservation Biology*, **16**, 109–116.
Kendall, B.E. & Fox, G.A. (2003) Unstructured individual variation and demographic stochasticity. *Conservation Biology*, **17**, 1170–1172.
Kendall, B.E., Fox, G.A., Fujiwara, M. & Nogeire, T.M. (2011) Demographic heterogeneity, cohort selection, and population growth. *Ecology*, **92**, 1985–1993.
Kindsvater, H.K. & Otto, S.P. (2014) The evolution of offspring size across life-history stages. *The American Naturalist*, **184**, 543–555.
Kindsvater, H.K., Rosenthal, G.G. & Alonzo, S.H. (2012) Maternal size and age shape offspring size in a live-bearing fish, *Xiphophorus birchmanni*. *PLoS One*, **7**, e48473.
Kingsolver, J.G., Hoekstra, H.E., Hoekstra, J.M., Berrigan, D., Vignieri, S.N., Hill, C.E., Hoang, A., Gibert, P. & Beerli, P. (2001) The strength of phenotypic selection in natural populations. *The American Naturalist*, **157**, 245–261.
Kirk, K.M., Blomberg, S.P., Duffy, D.L., Heath, A.C., Owens, I.P.F. & Martin, N.G. (2001) Natural selection and quantitative genetics of life-history traits in western women: a twin study. *Evolution*, **55**, 423–435.
Kirkpatrick, M. & Heckman, N. (1989) A quantitative genetic model for growth, shape, reactions norms, and other infinite-dimensional characters. *Journal of Mathematical Biology*, **27**, 429–450.
Lande, R., Engen, S. & Saether, B.E. (2009) An evolutionary maximum principle for density-dependent population dynamics in a fluctuating environment. *Philosophical Transactions of the Royal Society B: Biological Sciences*, **364**, 1511–1518.
Link, W.A., Cooch, E.G. & Cam, E. (2002) Model-based estimation of individual fitness. *Journal of Applied Statistics*, **29**, 207–224.
Łomnicki, A. (1978) Individual differences between animals and the natural regulation of their numbers. *Journal of Animal Ecology*, **47**, 461–475.
Lomolino, M.V. (1985) Body size of mammals on islands – the island rule reexamined. *The American Naturalist*, **125**, 310–316.
McAdam, A.G. & Boutin, S. (2003) Variation in viability selection among cohorts of juvenile red squirrels (*Tamiasciurus hudsonicus*). *Evolution*, **57**, 1689–1697.
Merilä, J. & Sheldon, B.C. (2000) Lifetime reproductive success and heritability in nature. *The American Naturalist*, **155**, 301–310.
Merilä, J., Sheldon, B.C. & Kruuk, L.E.B. (2001) Explaining stasis: microevolutionary studies in natural populations. *Genetica*, **112-113**, 199–222.
Metcalf, C.J.E., Rees, M., Buckley, Y.M. & Sheppard, A.W. (2009) Seed predators and the evolutionarily stable flowering strategy in the invasive plant Carduus nutans. *Evolutionary Ecology*, **23**, 893–906.
Metcalf, J., Graham, A., Martinez-Bakker, M. & Childs, D. (2016) Opportunities and challenges of integral projection models for modeling host-parasite dynamics. *Journal of Animal Ecology*, **85**, 343–355.
Miller, T.E.X., Williams, J.L., Jongejans, E., Brys, R. & Jacquemyn, H. (2012) Evolutionary demography of iteroparous plants: incorporating non-lethal costs of reproduction into integral projection models. *Proceedings of the Royal Society of London B: Biological Sciences*, **279**, 2831–2840.
Mousseau, T.A. & Roff, D.A. (1987) Natural selection and the heritability of fitness components. *Heredity*, **59**, 181–197.
Nussey, D.H., Coulson, T., Delorme, D., Clutton-Brock, T.H., Pemberton, J.M., Festa-Bianchet, M. & Gaillard, J.M. (2011) Patterns of body mass senescence and selective disappearance differ among three species of free-living ungulates. *Ecology*, **92**, 1936–1947.
Perrone, M.J. (1978) Mate size and breeding success in a monogamous cichlid fish. *Environmental Biology of Fishes*, **3**, 193–202.
Pfennig, D.W., Wund, M.A., Snell-Rood, E.C., Cruickshank, T., Schlichting, C.D. & Moczek, A.P. (2010) Phenotypic plasticity's impacts on diversification and speciation. *Trends in Ecology & Evolution*, **25**, 459–467.
Plard, F., Gaillard, J.M., Coulson, T., Delorme, D.C.W., J., Michallet, S., Tuljapurkar, S., KrishnaKumar, S. & Bonenfant, C. (2015a) Quantifying the influence of measured and unmeasured individual differences on demography. *Journal of Animal Ecology*, **84**, 1434–1445.
Plard, F., Gaillard, J.M., Coulson, T., Hewison, A.J.M., Douhard, M., Klein, F., Delorme, D., C., W. Bonenfant, C. (2015b) The influence of birth date via body mass on individual fitness in a long-lived mammal. *Ecology*, **96**, 1516–1528.
Price, G.R. (1970) Selection and covariance. *Nature*, **227**, 520–521.
Prince, P.A., Rothery, P., Croxall, J.P. & Wood, A.G. (1994) Population dynamics of Black-browed and Grey-headed Albatrosses *Diomedea melanophris* and *D. chrysostoma* at Bird Island, South Georgia. *Ibis*, **136**, 50–71.
Rees, M. & Ellner, S.P. (2009) Integral projection models for populations in temporally varying environments. *Ecological Monographs*, **79**, 575–594.
Rees, M. & Ellner, S. (2016) Evolving integral projection models: evolutionary demography meets eco-evolutionary dynamics. *Methods in Ecology and Evolution*, **7**, 157–170.
Rees, M., Sheppard, A., Briese, D. & Mangel, M. (1999) Evolution of size-dependent flowering in *Onopordum illyricum*: a quantitative assessment of the role of stochastic selection pressures. *The American Naturalist*, **154**, 628–651.
Rees, M., Childs, D.Z., Metcalf, J.C., Rose, K.E., Sheppard, A.W. & Grubb, P.J. (2006) Seed dormancy and delayed flowering in monocarpic plants: selective interactions in a stochastic environment. *The American Naturalist*, **168**, E53–E71.
Rees, M., Childs, D.Z. & Ellner, S.P. (2014) Building integral projection models: a user's guide. *Journal of Animal Ecology*, **83**, 528–545.
Ruel, J.J. & Ayres, M.P. (1999) Jensen's inequality predicts effects of environmental variation. *Trends in Ecology & Evolution*, **14**, 361–366.
Sadras, V.O. & Slafer, G.A. (2012) Environmental modulation of yield components in cereals: Heritabilities reveal a hierarchy of phenotypic plasticities. *Field Crops Research*, **127**, 215–224.
Saether, B. & Bakke, O. (2000) Avian life history variation and contribution of demographic traits to the population growth rate. *Ecology*, **81**, 642–653.
Schindler, S., Neuhaus, P., Gaillard, J.M. & Coulson, T. (2013) The influence of nonrandom mating on population growth. *The American Naturalist*, **182**, 28–41.
Smallegange, I.M. & Coulson, T. (2013) Towards a general, population-level understanding of eco-evolutionary change. *Trends in Ecology & Evolution*, **28**, 143–148.
Stearns, S.C. (1992) *The Evolution of Life Histories*. Oxford University Press, Oxford , New York, NY, USA.
Steiner, U.K. & Tuljapurkar, S. (2012) Neutral theory for life histories and individual variability in fitness components. *Proceedings of the National Academy of Sciences*, **109**, 4684–4689.
Tatarenkov, A., Healey, C.I.M., Grether, G.F. & Avise, J.C. (2008) Pronounced reproductive skew in a natural population of green swordtails, *Xiphophorus helleri*. *Molecular Ecology*, **17**, 4522–4534.
Thomas, S.C. & Bazzaz, F.A. (1993) The genetic component in plant size hierarchies: norms of reaction to density in a polygonum species. *Ecological Monographs*, **63**, 231–249.
Traill, L.W., Schindler, S. & Coulson, T. (2014) Demography, not inheritance, drives phenotypic change in hunted bighorn sheep. *Proceedings of the National Academy of Sciences*, **111**, 13223–13228.
Tuljapurkar, S. (1990) *Population Dynamics in Variable Environments*, **Vol. 85**. Springer-Verlag, New York, NY, USA.
Tuljapurkar, S., Steiner, U.K. & Orzack, S.H. (2009) Dynamics heterogeneity in life histories. *Ecology Letters*, **12**, 93–106.
Turelli, M. & Barton, N.H. (1994) Genetic and statistical-analyses of strong selection on polygenic traits - what, me normal. *Genetics*, **138**, 913–941.
Van Kampen, N.G. (1992) *Stochastic Processes in Physics and Chemistry*. Elsevier, the Netherlands.
Vaupel, J.W., Manton, K.G. & Stallard, E. (1979) The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, **16**, 439–454.
Venable, D.L. (1992) Size-number trade-offs and the variation of seed size with plant resource status. *American Naturalist*, **140**, 287–304.
Vindenes, Y. & Langangen, O. (2015) Individual heterogeneity in life histories and eco-evolutionary dynamics. *Ecology Letters*, **18**, 417–432.
Vindenes, Y., Engen, S. & Sæther, B.E. (2008) Individual heterogeneity in vital parameters and demographic stochasticity. *The American Naturalist*, **171**, 455–467.

van Wijk, S.J., Taylor, M.I., Creer, S., Dreyer, C., Rodrigues, F.M., Ramnarine, I.W., van Oosterhout, C. & Carvalho, G.R. (2013) Experimental harvesting of fish populations drives genetically based shifts in body size and maturation. *Frontiers in Ecology and the Environment*, **11**, 181–187.

Zambrano, J. & Salguero-Gomez, R. (2014) Forest fragmentation alters the population dynamics of a late-successional tropical tree. *Biotropica*, **46**, 556–564.

Received 24 June 2015; accepted 17 November 2015
Handling Editor: Dylan Childs

Supporting Information

Additional Supporting Information may be found in the online version of this article.

Appendix S1. Presentation of the model including analytical demonstrations.

Table S1. Parameters used to build the demographic rates for the short-lived and long-lived life-histories.

Figure S1. Elasticities of r, $\widehat{r}$, $\widetilde{r}$, $\check{r}$ and v_1 for the short-lived life history.

Figure S2. Elasticities of $r, \widehat{r}, \widetilde{r}, \check{r}$ and v_1 for the long-lived life history.

Figure S3. Influence of the inheritance function on the newborn phenotype distribution and on the population growth rate for the short-lived life history.

Figure S4. Influence of selection, and stochasticity in growth on the population growth trajectories.