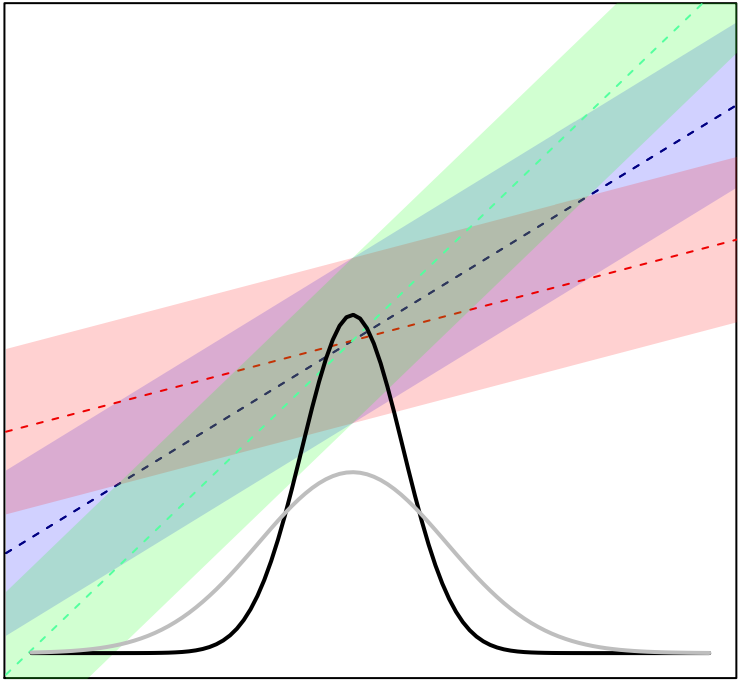

# Des différences, pourquoi? Transmission, maintenance and effects of phenotypic variance

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A manuscript in consideration as a research paper for publication in Journal of Animal Ecology as part of the Special Feature Demography beyond the Population

## 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 performance. 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

others 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 phenotype. 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 influence 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.

## Keywords

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 (Merila & Sheldon, 2000; Plard *et al.*, 2015b; Steiner & Tuljapurkar, 2012). 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 (Saether & Bakke, 2000; Heppell, Caswell & Crowder, 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; Kendall *et al.*, 2011; Vindenes, Engen & Sæther, 2008; 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 is 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 analyzing 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, e.g., an improved understanding of eco-evolutionary dynamics (Rees *et al.*, 2006; Metcalf *et al.*, 2009; Smallegange & Coulson, 2013), or a better management of size-structured populations subject to fishing or hunting (Traill, Schindler & Coulson, 2014). 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 Rees *et al.* 1999; Coulson *et al.* 2011). For the sake of simplicity, we do not consider other, potentially im-

portant, kinds of individual heterogeneity that may be due to, e.g. 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, behaviors, 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 (Bonduriansky, 2012; Danchin *et al.*, 2011). 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 models focus 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 (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 analyze 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. 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; 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 one 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 2-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 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 $G_a(.|z)$, $\mathcal{E}\{K_a\} = 0$ 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 $\mu_1$ 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,

$$X_{Oa} = \mu_1 + \beta\,(X_a - \mu_{Ma}) + I \tag{1}$$

$$\mathcal{E}\{I\} = 0 \tag{2}$$

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

where $\mu_{Ma}$ is the mean phenotype of mothers of age $a$. $\beta(0 \leq \beta < 1)$ links mother and offspring body masses, and $v_I$ is the variance of the inheritance function. $\beta$ is the strength of the inheritance function; $\beta$ is related to but not the same as heritability as commonly defined in quantitative genetics (Falconer & Mackay, 1996). Indeed, $\beta$ 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:

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

where $F_a(y|z)$ and $P_a(y|z)$ are the fertility and the survivorphip functions, respec-

tively.

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 $\mu_a$ 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 modeled 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 modeled 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 modeled using two parameters, an intercept and a slope with respect to body mass, and a logit link. Growth is modeled as a linear function of body mass. Parameter values are reported in the supplementary 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 considers all functions that are differentiable. The only assumptions include a small phenotypic variance and a phenotypic trait normally distributed. Our model can be easily applied to polynomial or other non-linear functions. The main body of the paper focuses on results, leaving mathematical details to Appendix A.

# 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 $\mu_1$ 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 $\mu_a$ 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

$$s_a = S(\mu_a) + \frac{1}{2} v_a \, S''(\mu_a). \tag{5}$$

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 A.1.2). This equation shows that when a demographic rate changes linearly with the phenotype, we may have strong selec-

tion 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, e.g., exponential for $M(x)$ such as in the short-lived life-history (Fig. 2 B), 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, e.g., the survival function of the second and third age classes of the long-lived life-history (Fig. 2 C).

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

$$v_s = v_a \left[S'(\mu_a)\right]^2. \tag{6}$$

Fig. 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 A.1.3). We can express the selective shift in terms of covariances between phenotype and selection (Price, 1970; Coulson & Tuljapurkar, 2008).

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

where $G', S'$ are the first derivatives of the growth and the survival functions, respectively, and $s_a, g_a$ are the mean survival and growth at age $a$ (see (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)). Fig. 3A illustrates three survival curves of very different curvatures that act on the phenotypic distributions at age 1 with equal mean $\mu_1$ and small or large variance (i.e., width). The resulting phenotype distribution, with mean $\mu_2$ 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 $\mu_a$. The phenotypic distribution of actual mothers is proportional to the weighted product $M(z)\, n(a, z, t)$. Thus (in analogy with (7)) fertility selection shifts the mean phenotype of mothers aged $a$ to

$$\mu_{Ma} - \mu_a = \frac{v_a\, M_a'(\mu_a)}{m_a} \tag{8}$$

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 A.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

291 $a$ and $a+1$, selection via growth changes the phenotypic variance at $a$ by the factor

$$\gamma_a = (1 + G'(\mu_a))^2. \tag{9}$$

292 Thus phenotypic variance changes with age according to

$$v_{a+1} = \gamma_a \, v_a + v_G, \tag{10}$$

$$\tag{11}$$

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

299 Fig. 4 illustrates how the growth function interacts with the phenotypic dis-
300 tributions at age 1 to produce a new phenotypic mean and variance at age 2. In
301 the left-hand column, (A), two different phenotypic distributions (black and grey)
302 at age 1 are submitted to three growth functions (colored functions) with increas-
303 ing selection (i.e., slope) but the same variability (i.e., a band of the same width
304 $v_G$). The phenotypic distributions at age 1 are characterized by equal mean $\mu_1$
305 and small (black) or large (grey) variance (i.e., width). The resulting phenotype
306 distributions, with mean $\mu_2$ and variance $v_2$, are shown in panels B (for the case
307 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)).

# 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 $\mu_1$ 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 rates $s_a$ 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

$$l_a = s_{a-1}s_{a-2}\ldots s_1,\ l_1 = 1. \tag{12}$$

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

$$\phi_a = m_a\, l_a,\ \text{with}\ R_0 = \sum_a \phi_a. \tag{13}$$

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 $\mu_1$ but the variance among offspring (see equation (3)) depends on the phenotypic variance among mothers (details in Appendix A.3). Considering mothers of all ages, we find that

$$v_1 = \beta^2\, \frac{1}{R_0} \sum_a\, \phi_a\, v_a + v_I, \tag{14}$$

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

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

$$v_a = \Gamma_a\, v_1 + k_a\, v_G,$$

where the coefficients are defined in the Appendix A.1.4. Then

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

Because $\phi_a$, $k_a$ and $\Gamma_a$ 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 $\mu_1$ 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). Second, 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 strength of inheritance $\beta > 0$ 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 $\beta$ 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 $\beta = 0.2$ and the phenotypic variances $v_1$ are small. Fig. 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 A.1.1 for details),

$$1 = \sum_a e^{-r\,a}\,\phi_a = \sum_a e^{-r\,a}\,m_a\,l_a. \tag{16}$$

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 affects the average survival rates $s_a$. 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 ($\check{r}$),

$$r = \widehat{r} + v_G\,\widetilde{r} + v_1\,\check{r} \tag{17}$$

See Appendix A.2 for proofs. This analytical decomposition assumes that $v_1, v_G$ 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 : $\widehat{r}$ 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 versus 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 A.4) of $r$ and its components to perturbations in: 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 $\beta$. 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 lifespan 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 $\mu_1$ 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 $v_1$ (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, $\beta$ and $v_I$, has only weak effects on $r$ and its components.

# 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, $\mu_1$ is variable and unknown. We must now thus solve both $\mu_1$ 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,

$$X_{Oa} = \alpha_a + \beta\, X_a + I \tag{18}$$

$$\mathcal{E}\{I\} = 0 \tag{19}$$

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

where $\alpha_a$ 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 $\mu_{Oa}$ of offspring depends on the age $a$ of mothers,

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

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 (14), 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 $\beta$ (or $v_I$) is to increase phenotypic variance (Fig. S3, A-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 ((20), Fig. S3, F-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 $\mu_1$ and the variance $v_1$ of newborn phenotypes. This matters to average newborn

survival when newborn survival rate (and/or growth rates) have a high positive slope and/or high curvature (positive second derivative) with respect to phenotype.

An increase of 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 $\beta$. With model B, $r$ doubles when $\beta$ 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 (1) the dynamic of the continuous trait over age, (2) the influence of phenotypic variation on demographic rates and (3) 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, e.g., 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 (Zambrano & Salguero-Gomez, 2014; Baudisch *et al.*, 2013). 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 modeled 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 analyzed 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 vital rate is generally different from the vital 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 less than 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 vital 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 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 lognormal (Calder, 1984; Graham *et al.*, 2003). In this case, the present analysis may be extended to describe the dynamics of a lognormally

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, or environmental stochasticity.

## 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 influences 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. 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 analyze 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.

# Acknowledgements

FP and ST were partly supported by NIH R24AG039345. TC acknowledges the support of the ERC and of NERC.

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# Figure captions

**Figure 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 goes from 0 to 200 (resp. 0 to 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

**Figure 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 function for each age-class (mean +/- 2 standard deviations).

**Figure 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 colors 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.

**Figure 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 colors show different growth functions. The color shade represents the uncertainty around each growth function. (B, C, E, F) The black (B, E) and grey (C, F) distributions at age 1 have grown to give the phenotypic distribution at age 2 according to the growth functions presented in A and D.

**Figure 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: $\widehat{r}$ 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.

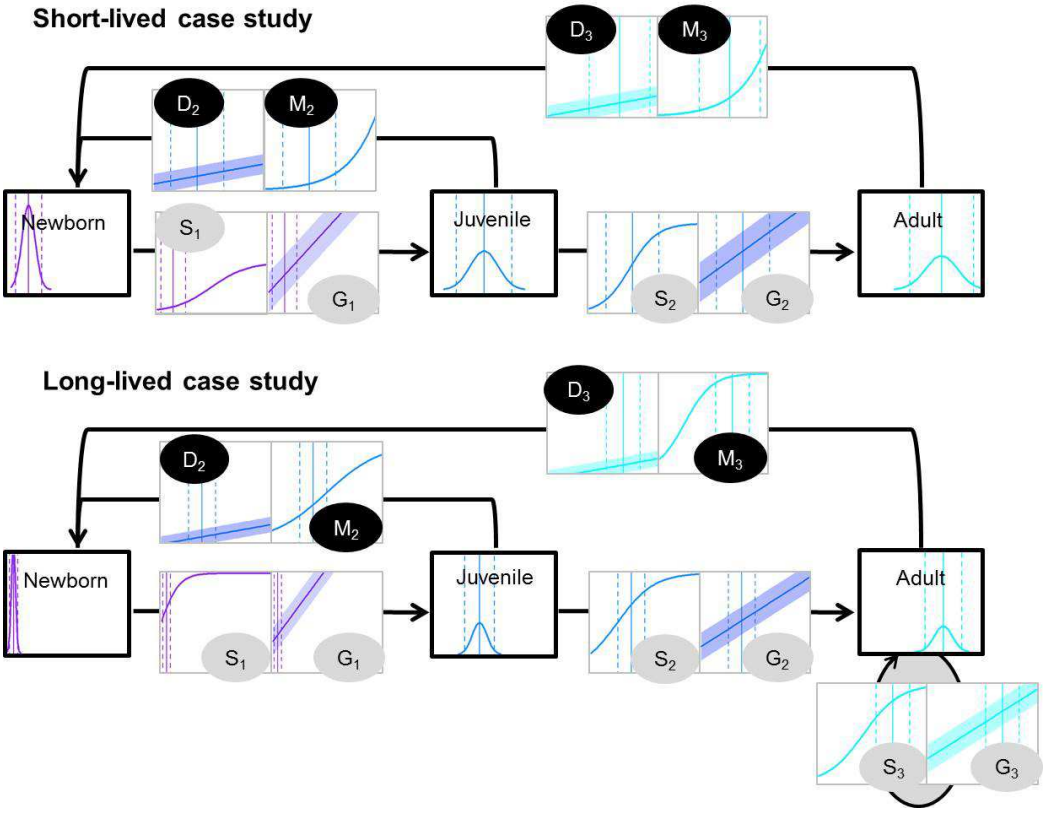
Short-lived case study
D3
M3
D2
M2
Newborn
S1
G1
Juvenile
S2
G2
Adult
Long-lived case study
D3
M3
D2
M2
Newborn
S1
G1
Juvenile
S2
G2
Adult
S3
G3

Figure 1

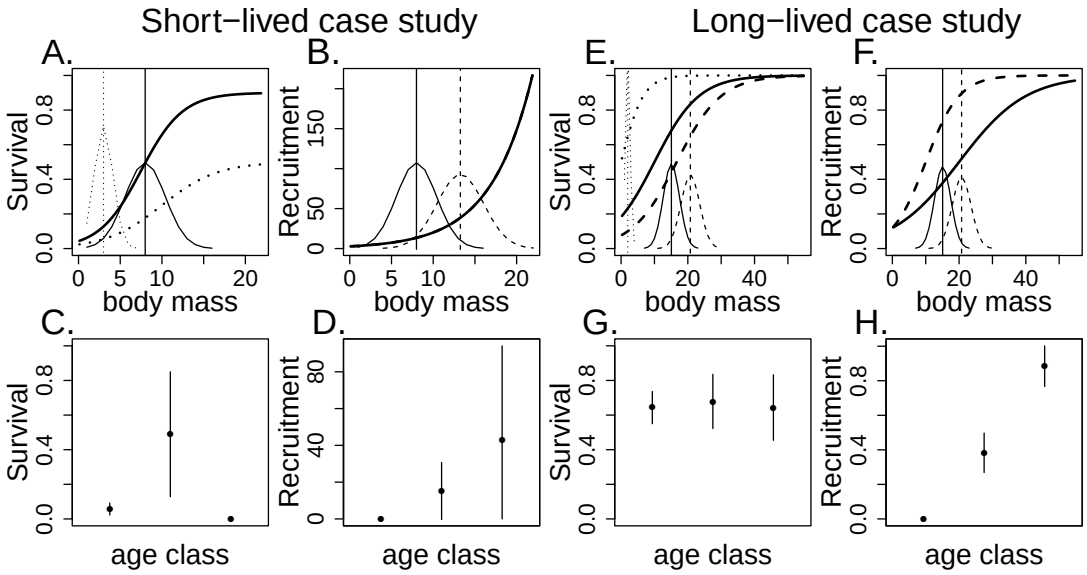
Short−lived case study
Long−lived case study
A.
B.
E.
F.
C.
D.
G.
H.
Survival
Recruitment
body mass
age class

Figure 2

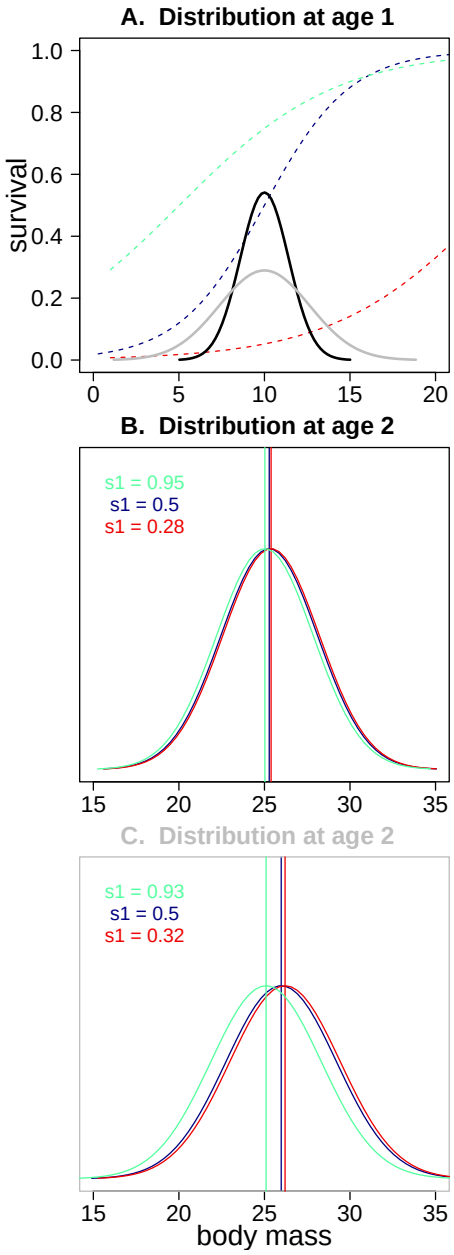
A. Distribution at age 1
survival
1.0
0.8
0.6
0.4
0.2
0.0
0
5
10
15
20
B. Distribution at age 2
s1 = 0.95
s1 = 0.5
s1 = 0.28
15
20
25
30
35
C. Distribution at age 2
s1 = 0.93
s1 = 0.5
s1 = 0.32
15
20
25
30
35
body mass

Figure 3

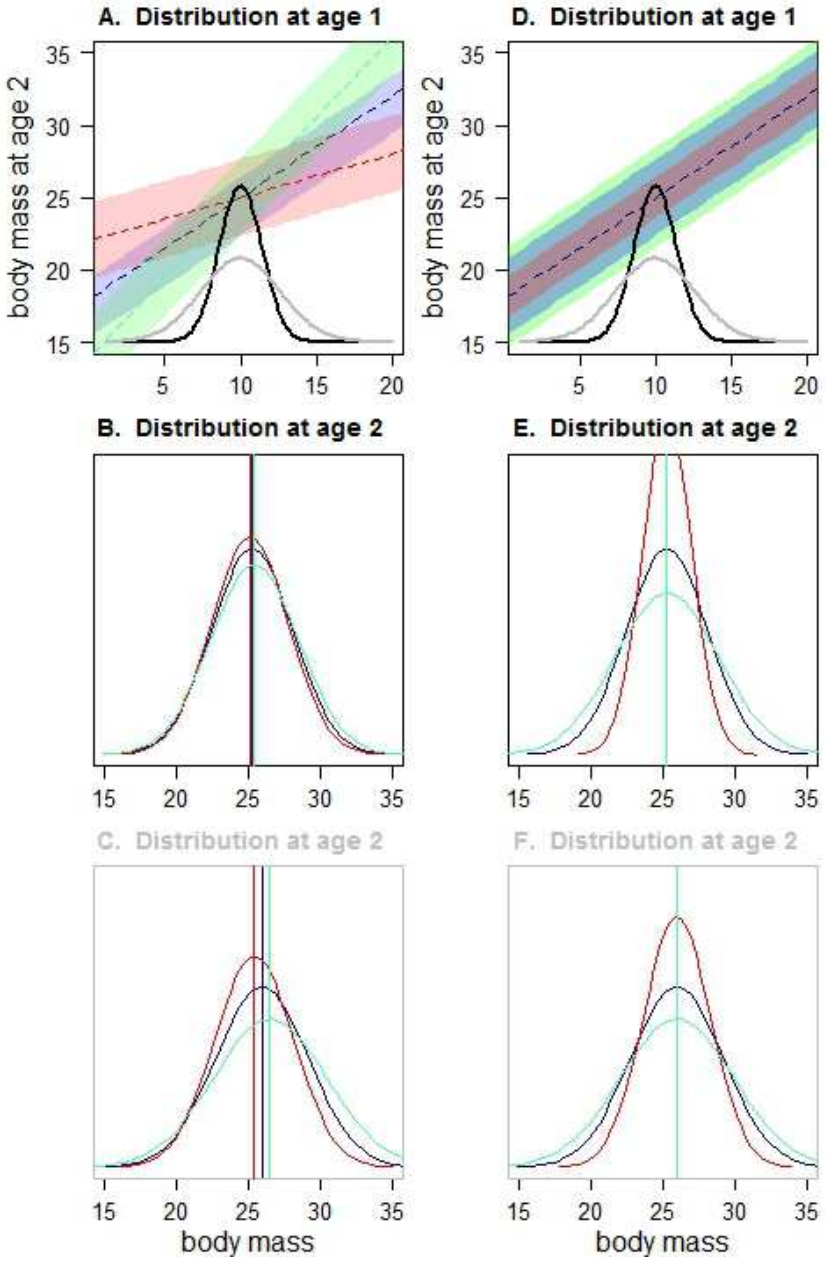
A. Distribution at age 1
D. Distribution at age 1
body mass at age 2
35
30
25
20
15
5
10
15
20
B. Distribution at age 2
E. Distribution at age 2
15
20
25
30
35
C. Distribution at age 2
F. Distribution at age 2
body mass

Figure 4

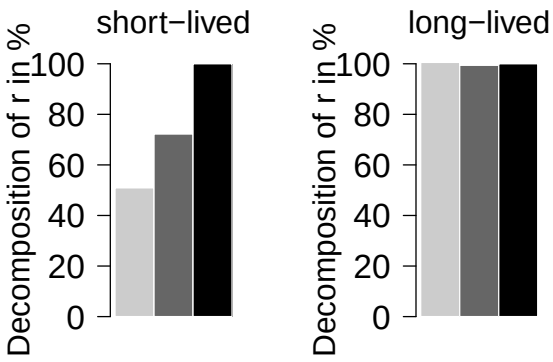


Figure 5

# Supplementary information

# A Appendix

## A.1 Successive age-dependent mean and variance of a continuous trait for a simple cohort

### A.1.1 definitions

We define the survivorship which is the cumulative survival-and-growth functions, given by

$$L_1(y|x) = \delta_{x,y} \text{ if phenotypes discrete; else Dirac delta,} \tag{A-21}$$

$$L_2(y|x) = P_1(y|x), \tag{A-22}$$

$$L_{a+1}(y|x) = \sum_z P_a(y|z)\, L_a(z|x). \tag{A-23}$$

The "net" reproduction $\mathbf{\Phi}_a$ gives the number of offspring produce by a cohort of age a and is equal to

$$\mathbf{\Phi}_a = F_a\, L_a. \tag{A-24}$$

At equilibrium, let $u_a(x)$ be the normalized phenotype distribution at age $a$, meaning that $|u_a| = 1$. Then

$$u_{a+1} = \frac{P_a\, u_a}{|P_a\, u_a|} = \frac{L_{a+1}\, u_1}{|L_{a+1}\, u_1|}. \tag{A-25}$$

Equilibrium population growth rate $r$ and the vector $u_1$ together solve the characteristic equation

$$\sum_{a\geq 1} e^{-ra}\,\mathbf{\Phi}_a\,u_1 = u_1. \tag{A-26}$$

The average survivorship at age $a$ can be written (from (A-21 – A-23) and (A-25))

$$l_a = |L_a\,u_1| = s_{a-1}s_{a-2}\ldots s_1. \tag{A-27}$$

where $s_{a-1}, s_{a-2}\ldots s_1$ are the average survival at each age. The average total number of offspring produced by a cohort at age $a$ is

$$\phi_a = m_a\,l_a. \tag{A-28}$$

Now take the norm on both sides of (A-26) to see that the characteristic equation can be written in the deceptively simple form

$$\sum_{a\geq 1} e^{-ra}\,\phi_a = 1. \tag{A-29}$$

### A.1.2 Mean demographic rates

We assume that the distribution of the phenotype $X_a$ at each age is approximately normal and can be adequately described by the first 2 moments of the distribution: the mean $\mu_a$ and the variance $v_a = \sigma_a^2$. To simplify we assume that the $\sigma_a$ are small.

For any function representing the survival rate, $S_a(x)$ can be approximated using a Taylor expansion by

$$S_a(x) \approx S(\mu_a) + (x - \mu_a)\, S'(\mu_a) + \frac{1}{2}(x - \mu_a)^2\, S''(\mu_a), \tag{A-30}$$

where $s, S', S''$ are respectively $S_a$ and its first and second derivatives. Thus the mean survival rate averaged over $X_a$ is

$$s_a = \mathcal{E}\{s_a\} = S(\mu_a) + \frac{1}{2} v_a\, S''(\mu_a). \tag{A-31}$$

This equation shows that the survival at age $a$ can be decomposed into a term dependent on the mean and a term dependent on the variance of the phenotype distribution at this age.

The growth function is defined by $H_a(x)$ whose conditional distribution is the density $G_a(h|x)$. We assume that at each age growth has a systematic part that depends on current phenotype, plus a small variance that is the same for all phenotypes,

$$H_a(x) = \bar{G}_a(x) + K_a, \tag{A-32}$$

$$\bar{G}_a(x) = \mathcal{E}\{H(x)\}, \text{ averaged over density } G_a(.|x), \tag{A-33}$$

$$\mathcal{E}\{K_a\} = 0, \text{ averaged over} X_a, \tag{A-34}$$

$$\mathrm{Var}\{K_a\} = v_G, \text{ for all x.} \tag{A-35}$$

The systematic part $G_a$ depends on the phenotype distribution at age $a$. We expand it, similarly to the survival function (A-30)

$$G_a(x) \approx G(\mu_a) + (x - \mu_a)\, G'(\mu_a) + \frac{1}{2}(x - \mu_a)^2\, G''(\mu_a), \tag{A-36}$$

where $g, G'$ and $G''$ are respectively $G_a$ and its first and second derivatives. Thus, average growth of a cohort between age $a$ and $a+1$ is

$$g_a = G(\mu_a) + \frac{1}{2} v_a \, G''(\mu_a). \tag{A-37}$$

### A.1.3 Mean of the phenotype distribution

We have an initial phenotype $X$ with mean $\mu$ and we write

$$X = \mu + \delta, \ \mathcal{E}\{\delta\} = 0, \ \mathcal{E}\{\delta^2\} = v. \tag{A-38}$$

Below we ignore terms of order $\delta^3$ and higher, and also terms of order $v^2$ and higher.

Growth (phenotype change in one time unit) is

$$H = G(X) + K, \ \mathcal{E}\{K\} = 0, \mathcal{E}\{K^2\} = v_G. \tag{A-39}$$

The term $K$ is assumed independent of $X$.

Expand

$$G(X) = G(\mu) + G'(\mu)\delta + \frac{1}{2} G''(\mu)\delta^2, \tag{A-40}$$

Survival is expanded as (A-40)

$$S(X) = S(\mu) + S'(\mu)\delta + \frac{1}{2}S''(\mu)\delta^2,$$
$$s = \mathcal{E}\{S(X)\}. \tag{A-41}$$

An individual starts with phenotype $X$ and after growth, has new phenotype $Y = (X + H)$ if it survives ($S = 1$) and new phenotype $Y = 0$ if it dies ($S = 0$). The first 2 moments of the new phenotype are

$$\mathcal{E}\{Y\} = \frac{\mathcal{E}[(X + H)\, S]}{\mathcal{E}[S]}, \tag{A-42}$$
$$\mathcal{E}\{Y^2\} = \frac{\mathcal{E}[(X + H)^2\, S]}{\mathcal{E}[S]}. \tag{A-43}$$

where $\mathcal{E}(.)$ is the expectation taken with respect to the density $X$.

Remember that we do not consider the terms of order higher than $\delta^2$ and use (A-38), (A-40) and (A-41) to have

$$\mathcal{E}\{Y\} = \frac{\mathcal{E}\{(\mu+\delta)(S(\mu)+S'(\mu)\delta+\frac{1}{2}S''(\mu)\delta^2)+(G(\mu)+G'(\mu)\delta+\frac{1}{2}G''(\mu)\delta^2)(S(\mu)+S'(\mu)\delta+\frac{1}{2}S''(\mu)\delta^2)+(G(\mu)+G'(\mu)\delta+\frac{1}{2}G''(\mu)\delta^2)(S(\mu)+S'(\mu)\delta+\frac{1}{2}S''(\mu)\delta^2)(\mu+\delta)\}}{s} \tag{A-44}$$

Simplify (A-44) to find that the mean phenotype at $a + 1$ is thus the addition of the mean phenotype at age a, the growth of this mean phenotype and a Price covariance that depends on the phenotypic variance.

$$\mu_{a+1} = \mu_a + g_a + \frac{v_a\, S'(\mu_a)\,(1 + G'(\mu_a))}{s_a} = \mu_a + g_a + v_a\, c_a, \tag{A-45}$$

We can also write a cumulative version of the mean phenotype at any age with respect to $\mu_1$, the mean birth phenotype of the cohort:

$$\mu_a = \mu_1 + \sum_{k=1}^{a-1} g_k + \sum_{k=1}^{a-1} c_k \tag{A-46}$$

### A.1.4 Variance of the phenotype distribution

Similarly for the variance, use (A-40)-(A-43) to find that

$$v_{a+1} = v_a \left(1 + G'(\mu_a)\right)^2 + v_G. \tag{A-47}$$

An amazing consequence of our assumptions is that survival selection does not change the variance of the phenotype distribution. Indeed, exponential selection on a normal variable leaves the variance unchanged and this is also true in more general cases. The exception is truncation selection, which we ignore.

To express the variance of the phenotype distribution at any age with respect to the variance at birth, we define the quantities

$$\gamma_a = (1 + G'(\mu_a))^2,\ \Gamma_1 = 1, \Gamma_{a+1} = \gamma_a \Gamma_a. \tag{A-48}$$

A cumulative version of (A-47) is

$$v_a = \Gamma_a\, v_1 + k_a\, v_G,\ a \geq 2, \tag{A-49}$$

$$k_2 = \Gamma_1, \tag{A-50}$$

$$k_a = \Gamma_a \left( \frac{1}{\Gamma_a} + \frac{1}{\Gamma_{a-1}} + \cdots + \frac{1}{\Gamma_2} \right). \tag{A-51}$$

### A.1.5 Phenotype of mothers

As did for the survival and the growth, the average reproductive rate at age $a$ can be approximated by

$$m_a = \mathcal{E}\{m_a\} = M(\mu_a) + \frac{1}{2} v_a \, M''(\mu_a). \tag{A-52}$$

where $m$ and $M''$ are respectively $M_a$ and its second derivative.

Each year, only a part of the individual of age $a$ will reproduce. The mean phenotype of mothers of age $a$ $\mathcal{E}(X_{Ma})$ must be weighted by reproductive rate and is given by

$$\mathcal{E}(X_{Ma}) = \mu_{Ma} = \frac{\mathcal{E}(X_a \, M_a)}{\mathcal{E} M_a}, \tag{A-53}$$

and, by the same kind of reasoning used to derive (A-45), we have

$$\mu_{Ma} = \mu_a + \frac{v_a \, M'(\mu_a)}{m_a} \tag{A-54}$$

where $M'$ is the first derivative of $M_a$, and $v_{Ma} = v_a$.

Now, to find the mean phenotype of all mothers, independently of the age at which they reproduce, we must weight age dependent mean mother phenotypes by the amount of reproduction at age $a$, which we call $\bar{\phi}_a$.

For a cohort,

$$\bar{\phi}_a = \phi_a, \text{ and } R_0 = \sum_a \bar{\phi}_a = \sum_a \phi_a, \tag{A-55}$$

For a stable population,

$$\bar{\phi}_a = e^{-ra}\,\phi_a, \text{ and } R_0 = \sum_a \bar{\phi}_a = 1. \tag{A-56}$$

The weighted mean phenotype of mothers is

$$\mathcal{E}(X_M) = \mu_M = \frac{\sum_a \bar{\phi}_a\,\mu_{Ma}}{R_0}, \tag{A-57}$$

.

## A.2 Decomposition of r

We show by recurrence that the parameters describing the population dynamics can be described by three components: a component generated only from the mean of the phenotypic distribution, a component linked to the variance generated from the growth function that accumulate across ages and a component generated by the transformation of the variance at birth. We ignore the terms of order $v_a\,v_G$, $v_a^2$ and $v_G^2$ or higher.

At age 1, we have $\mu_1$ and $v_1$. We take the growth function as a representative case of all demographic rates:

$$\begin{aligned} g_1 &= G(\mu_1) + v_G\,0 + v_1\,\frac{1}{2}\,G''(\mu_1) \\ &= \widehat{g}_1 + v_G\,\widetilde{g}_1 + v_1\,\breve{g}_1 \end{aligned} \tag{A-58}$$

At age 2, the mean phenotype distribution became

$$\begin{aligned}\mu_2 &= \mu_1 + \widehat{g}_1 + v_G\, 0 + v_1\,(\check{g}_1 + c_1)\\ &= \widehat{\mu}_2 + v_G\,\widetilde{\mu}_2 + v_1\,\check{\mu}_2\end{aligned} \tag{A-59}$$

with

$$c_1 = \frac{S'(\mu_1)\,(1 + G'(\mu_1))}{\widehat{s}_1} \tag{A-60}$$

at age 2, the demographic rates can be written as illustrated for the reproductive function:

$$\begin{aligned}m_2 &= M(\widehat{\mu}_2 + v_1\,\check{\mu}_2) + v_2\,\frac{1}{2}\,M''(\widehat{\mu}_2 + v_1\,\check{\mu}_2)\\ &= M(\widehat{\mu}_2 + v_1\,\check{\mu}_2) + (v_G + v_1\,(1 + G'(\mu_1))^2)\,\frac{1}{2}\,M''(\widehat{\mu}_2 + v_1\,\check{\mu}_2)\\ &\approx M(\widehat{\mu}_2) + v_G\,\frac{1}{2}\,M''(\widehat{\mu}_2) + v_1\,[\check{\mu}_2\,M'(\widehat{\mu}_2) + \frac{1}{2}\,M''(\widehat{\mu}_2)\,(1 + G'(\mu_1))^2]\\ &\approx \widehat{m}_2 + v_G\,\widetilde{m}_2 + v_1\,\check{m}_2\end{aligned} \tag{A-61}$$

So the survivorship is

$$\begin{aligned}\bar{l}_2 &= \widehat{s}_1 + v_G\, 0 + v_1\, \frac{1}{2}\, S''(\mu_1) \\ &= \widehat{l}_2 + v_G\, \widetilde{l}_2 + v_1\, \check{l}_2\end{aligned} \tag{A-62}$$

961 and the net reproductive rate:

$$\begin{aligned}\bar{\phi}_2 &= \widehat{m}_2\, \widehat{l}_2 + v_G\, \widetilde{m}_2\, \widehat{l}_2 + v_1\, (\check{m}_2\, \widehat{l}_2 + \widehat{m}_2\, \check{l}_2) \\ &= \widehat{\phi}_2 + v_G\, \widetilde{\phi}_2 + v_1\, \check{\phi}_2\end{aligned} \tag{A-63}$$

962 Now, we assume that all parameters of age $a$ can be decomposed into three
963 components and show that these parameters at age $a+1$ can also be decomposed
964 into these three components.

965 The variance at age $a$ can be simplified by $v_a = \Gamma_a\, v_1 + k_a\, v_G$ with

$$\gamma_a = (1 + G'(\widehat{\mu}_a))^2,\ \Gamma_1 = 1, \Gamma_{a+1} = \gamma_a \Gamma_a.$$

$$k_2 = \Gamma_1, \tag{A-64}$$

$$k_a = \Gamma_a \left( \frac{1}{\Gamma_a} + \frac{1}{\Gamma_{a-1}} + \cdots + \frac{1}{\Gamma_2} \right). \tag{A-65}$$

966 At age $a+1$: the mean phenotype is

$$\begin{aligned}\mu_{a+1} &= \mu_a + g_a + v_a\, c_a \\ &= \widehat{\mu}_a + \widehat{g}_a + v_G\,(\widetilde{\mu}_a + \widetilde{g}_a + k_a\, c_a) + v_1\,(\check{\mu}_a + \check{g}_a + \Gamma_a\, c_a) \\ &= \widehat{\mu}_{a+1} + v_G\,\widetilde{\mu}_{a+1} + v_1\,\check{\mu}_{a+1}\end{aligned} \tag{A-66}$$

967 Note that we can simplify $c_a$ by

$$c_a = \frac{S'(\widehat{\mu}_a)\,(1 + G'(\widehat{\mu}_a))}{\widehat{s}_a} \tag{A-67}$$

968 The demographic rates at age $a$+1 are illustrated by the case of the reproductive
969 function and can be expanded as:

$$\begin{aligned}m_{a+1} &= M(\widehat{\mu}_{a+1} + v_G\,\widetilde{\mu}_{a+1} + v_1\,\check{\mu}_{a+1}) + v_a\,\frac{1}{2}\,M''(\widehat{\mu}_{a+1} + v_G\,\widetilde{\mu}_{a+1} + v_1\,\check{\mu}_{a+1}) \\ &\approx M(\widehat{\mu}_{a+1}) + v_G\,(M'(\widehat{\mu}_{a+1})\,\widetilde{\mu}_{a+1} + \frac{1}{2}\,M''(\widehat{\mu}_{a+1})\,k_{a+1}) + v_1\,(\check{\mu}_{a+1}\,M'(\widehat{\mu}_{a+1}) + \frac{1}{2}\,M''(\widehat{\mu}_{a+1})\,\Gamma_{a+1}) \\ &\approx \widehat{m}_{a+1} + v_G\,\widetilde{m}_{a+1} + v_1\,\check{m}_{a+1}\end{aligned} \tag{A-68}$$

970 For the survivorship:

$$\begin{aligned} l_{a+1} &= \widehat{s}_a\,\widehat{l}_a + v_G\,(\widetilde{l}_a\,\widehat{s}_a + \widehat{l}_a\,\widetilde{s}_a) + v_1\,(\check{l}_a\,\widehat{s}_a + \widehat{l}_a\,\check{s}_a) \\ &= \widehat{l}_{a+1} + v_G\,\widetilde{l}_{a+1} + v_1\,\check{l}_{a+1} \end{aligned} \tag{A-69}$$

971 We can also write the net reproductive rate, $\phi_{a+1}$ according to $v_1$ and $v_G$:

$$\begin{aligned} \phi_{a+1} &= \widehat{m}_{a+1}\,\widehat{l}_{a+1} + v_G\,(\widetilde{m}_{a+1}\widehat{l}_{a+1} + \widehat{m}_{a+1}\widetilde{l}_{a+1}) + v_1\,(\check{m}_{a+1}\,\widehat{l}_{a+1} + \widehat{m}_{a+1}\,\check{l}_{a+1}) \\ &= \widehat{\phi}_{a+1} + v_G\,\widetilde{\phi}_{a+1} + v_1\,\check{\phi}_{a+1} \end{aligned} \tag{A-70}$$

972 and so

$$R_0 = \widehat{R}_0 + v_G\,\widetilde{R}_0 + v_1\,\check{R}_0 \tag{A-71}$$

973 As a consequence, all these demographic parameters can be decomposed into
974 three components. Now, we consider the net reproductive rate, the generation
975 rate and the population growth rate: $r$ is often estimated using the approximation
976 $\frac{loG(R_0)}{T_c}$ where $R_0$ is the net reproductive rate and $T_c$ is the generation time.

$$T_c = \frac{1}{K}\sum_a a\,\phi_a \tag{A-72}$$

$$\tag{A-73}$$

977 The generation rate can be decomposed like that:

$$T_c = \frac{1}{\widehat{R}_0} \sum_a a\,\widehat{\phi}_a + \frac{v_G}{\widehat{R}_0}\,(\sum_a a\,\widetilde{\phi}_a - \frac{\widetilde{R}_0}{\widehat{R}_0} \sum_a a\,\widehat{\phi}_a) + \frac{v_1}{\widehat{R}_0}\,(\sum_a a\,\check{\phi}_a - \frac{\check{R}_0}{\widehat{R}_0} \sum_a a\,\widehat{\phi}_a)$$
$$= \widehat{T}_c + v_G\,\widetilde{T}_c + v_1\,\check{T}_c \tag{A-74}$$
$$\tag{A-75}$$

978 and so the population growth rate can be expanded as

$$r = \frac{loG(\widehat{R}_0 + v_G\,\widetilde{R}_0 + v_1\,\check{R}_0)}{\widehat{T}_c + v_G\,\widetilde{T}_0 + v_1\,\check{T}_c}$$
$$\approx \frac{loG(\widehat{R}_0)}{\widehat{T}_c} + \frac{v_G}{\widehat{T}_c}\,(\frac{\widetilde{R}_0}{\widehat{R}_0} - \widetilde{T}_c\,\widehat{r}) + \frac{v_1}{\widehat{T}_c}\,(\frac{\check{R}_0}{\widehat{R}_0} - \check{T}_c\,\widehat{r})$$
$$= \widehat{r} + v_G\,\widetilde{r} + v_1\,\check{r} \tag{A-76}$$
$$\tag{A-77}$$

979 For the purpose of estimation and to avoid the errors accumulated by suc-
980 cessive approximations, we solved the characteristic equation to find the different
981 components of $r$. $\widehat{r}$ has been estimated by solving

$$\sum_{a\geq 1} e^{-\widehat{r}a}\,\widehat{\phi}_a = 1. \tag{A-78}$$

982 $v_G\,\widetilde{r}$ has been estimated by solving

$$\sum_{a\geq 1} e^{-(\widehat{r}+v_G\,\widetilde{r})a}\,(\widehat{\phi}_a + v_G\,\widetilde{\phi}_a) = 1. \tag{A-79}$$

983 Finally, $v_1\,\check{r}$ has been estimated by subtracting $r$ and $\widehat{r} + v_G\,\widetilde{r}$

## A.3 The variance of offspring

When $\mu_1$ and $v_1$ are known, we can find the mean and the variance of the phenotype distribution among all individuals and among mothers at each age, using this model. However, in practice, biologists do not know $\mu_1$ and $v_1$ but model an inheritance function to find the phenotype of offspring according to the phenotype of mothers. Thus, here we use the simple inheritance function described for the base examples where $\mu_1$ is known to find $v_1$.

In the base case, the variance of the phenotype of offspring produced by mothers of age a is

$$v_{Oa} = \beta^2 \, v_a + v_I \tag{A-80}$$

The variance of all offspring (independently of their mother age) $v_1$ can be estimated from the sum of the variance intra and inter groups of offspring produced by mothers of different ages:

$$\begin{aligned} v_1 &= \frac{1}{R_0} \sum_a \bar{\phi}_a \, v_{Oa} + \frac{1}{R_0} \sum_a \bar{\phi}_a \, (\mu_{Oa} - \mu_1)^2, \\ &= \frac{1}{R_0} \sum_a \bar{\phi}_a \, v_{Oa} \\ &= \frac{1}{R_0} \sum_a \bar{\phi}_a \, \beta^2 \, v_a + v_I \end{aligned} \tag{A-81}$$

Using (A-65) and assuming that $v_1$ and $v_G$ are small, we can use the decomposition of $\bar{\phi}_a$ (A-70) and $R_0$ (A-71) to see that

$$v_1 \approx \frac{\frac{\beta^2}{\widehat{R}_0} \sum_a \widehat{\phi}_a \widehat{k}_a v_G + v_I}{1 - \frac{\beta^2}{\widehat{R}_0} \sum_a \widehat{\phi}_a \widehat{\Gamma}_a} \tag{A-82}$$

## A.4 Elasticities

Now suppose that we change parameters with a small $0 < \epsilon \ll 1$ so that

$$\phi_a \to \phi_a + \epsilon \boldsymbol{\delta}_{\phi_a}. \tag{A-83}$$

Then we have the resulting changes

$$\begin{aligned} r &\to r + \epsilon \boldsymbol{\delta}_r, \\ \mu_a &\to \mu_a + \epsilon \boldsymbol{\delta}_{\mu_a}, \\ v_a &\to v_a + \epsilon \boldsymbol{\delta}_{v_a}, \\ v_1 &\to v_1 + \epsilon \boldsymbol{\delta}_{v_1}. \end{aligned} \tag{A-84}$$

(A-85)

Note that $\boldsymbol{\delta}$ represents a derivative.

### A.4.1 Elasticity of $\widehat{r}$, $\check{r}$, $\widetilde{r}$, and $v_1$

We use (A-75) to find:

$$
\begin{aligned}
\boldsymbol{\delta}_{\widehat{r}} &= \frac{\boldsymbol{\delta}_{\widehat{R}_0}}{\widehat{R}_0\,\widehat{T}_c} - \frac{\boldsymbol{\delta}_{\widehat{T}_c}\,\widehat{R}_0}{\widehat{T}_c^2} \\
\boldsymbol{\delta}_{\widetilde{r}} &= \frac{\boldsymbol{\delta}_{\widetilde{R}_0}\,\widehat{T}_c\,\widehat{R}_0 - \widetilde{R}_0(\boldsymbol{\delta}_{\widehat{R}_0}\,\widehat{T}_c + \widehat{R}_0\,\boldsymbol{\delta}_{\widehat{T}_c})}{\widehat{R}_0^2\,\widehat{T}_c^2} - \frac{\widehat{T}_c(\boldsymbol{\delta}_{\widehat{r}}\,\widetilde{T}_c + \widehat{r}\,\boldsymbol{\delta}_{\widetilde{T}_c}) - \boldsymbol{\delta}_{\widehat{T}_c}\,\widetilde{T}_c\,\widehat{r}}{\widehat{T}_c^2} \\
\boldsymbol{\delta}_{\check{r}} &= \frac{\boldsymbol{\delta}_{\check{R}_0}\,\widehat{T}_c\,\widehat{R}_0 - \check{R}_0(\boldsymbol{\delta}_{\widehat{R}_0}\,\widehat{T}_c + \widehat{R}_0\,\boldsymbol{\delta}_{\widehat{T}_c})}{\widehat{R}_0^2\,\widehat{T}_c^2} - \frac{\widehat{T}_c(\boldsymbol{\delta}_{\widehat{r}}\,\check{T}_c + \widehat{r}\,\boldsymbol{\delta}_{\check{T}_c}) - \boldsymbol{\delta}_{\widehat{T}_c}\,\check{T}_c\,\widehat{r}}{\widehat{T}_c^2}
\end{aligned}
\tag{A-86}
$$

with

$$
\begin{aligned}
\boldsymbol{\delta}_{\widehat{R}_0} &= \sum_a \boldsymbol{\delta}_{\widehat{\phi}_a} \\
\boldsymbol{\delta}_{\widetilde{R}_0} &= \sum_a \boldsymbol{\delta}_{\widetilde{\phi}_a} \\
\boldsymbol{\delta}_{\check{R}_0} &= \sum_a \boldsymbol{\delta}_{\check{\phi}_a} \\
\boldsymbol{\delta}_{\widehat{T}_c} &= \frac{1}{\widehat{R}_0}\sum_a a\,\boldsymbol{\delta}_{\widehat{\phi}_a} - \frac{\boldsymbol{\delta}_{\widehat{R}_0}}{\widehat{R}_0^2}\sum_a a\,\widehat{\phi}_a \\
\boldsymbol{\delta}_{\widetilde{T}_c} &= \frac{1}{\widehat{R}_0^2}(\widehat{R}_0\sum_a a\,\boldsymbol{\delta}_{\widetilde{\phi}_a} - \boldsymbol{\delta}_{\widehat{R}_0}\sum_a a\,\widetilde{\phi}_a) - \frac{1}{\widehat{R}_0^2}(\boldsymbol{\delta}_{\widetilde{R}_0}\sum_a a\,\widehat{\phi}_a + \widetilde{R}_0\sum_a a\,\boldsymbol{\delta}_{\widehat{\phi}_a}) + \frac{2\boldsymbol{\delta}_{\widehat{R}_0}}{\widehat{R}_0^3}(\widetilde{R}_0\sum_a a\,\widehat{\phi}_a) \\
\boldsymbol{\delta}_{\check{T}_c} &= \frac{1}{\widehat{R}_0^2}(\widehat{R}_0\sum_a a\,\boldsymbol{\delta}_{\check{\phi}_a} - \boldsymbol{\delta}_{\widehat{R}_0}\sum_a a\,\check{\phi}_a) - \frac{1}{\widehat{R}_0^2}(\boldsymbol{\delta}_{\check{R}_0}\sum_a a\,\widehat{\phi}_a + \check{R}_0\sum_a a\,\boldsymbol{\delta}_{\widehat{\phi}_a}) + \frac{2\boldsymbol{\delta}_{\widehat{R}_0}}{\widehat{R}_0^3}(\check{R}_0\sum_a a\,\widehat{\phi}_a)
\end{aligned}
\tag{A-87}
$$

For $v_1$, We simplify (A-82) with

$$v_1 = \frac{f}{y} \tag{A-88}$$

SO

$$\boldsymbol{\delta}_{v_1} = \frac{\boldsymbol{\delta}_f\, y - \boldsymbol{\delta}_y\, f}{y^2} \tag{A-89}$$

if a vital rate is perturbed,

$$\begin{aligned}
\boldsymbol{\delta}_f &= \frac{\beta^2\, v_G}{\widehat{R}_0} \sum_a (\boldsymbol{\delta}_{\widehat{\phi}_a}\, k_a + \widehat{\phi}_a\, \boldsymbol{\delta}_{k_a}) - \frac{\boldsymbol{\delta}_{\widehat{R}_0} \beta^2\, v_G}{\widehat{R}_0^2} \sum_a \widehat{\phi}_a\, k_a \\
\boldsymbol{\delta}_g &= \frac{\boldsymbol{\delta}_{\widehat{R}_0} \beta^2}{\widehat{R}_0^2} \sum_a \widehat{\phi}_a\, \Gamma_a - \frac{\beta^2}{\widehat{R}_0} \sum_a (\boldsymbol{\delta}_{\widehat{\phi}_a}\, \Gamma_a + \widehat{\phi}_a\, \boldsymbol{\delta}_{\Gamma_a})
\end{aligned} \tag{A-90}$$

**Perturbation of the reproductive function**

- a small perturbation in $M(\mu_a)$ will not change $\mu_a$ and $v_a$ but see from (13):

$$\begin{aligned}
\boldsymbol{\delta}_{\widehat{\phi}_a} &= \widehat{l}_a. \\
\boldsymbol{\delta}_{\widetilde{\phi}_a} &= \widetilde{l}_a. \\
\boldsymbol{\delta}_{\check{\phi}_a} &= \check{l}_a.
\end{aligned} \tag{A-91}$$

- a small perturbation in $M'(\mu_a)$ will not change $\mu_a$, $v_a$ (it can change in some

cases $\mu_1$ but not in our simplest case as $\mu_1$ is constant).

$$\begin{aligned}\boldsymbol{\delta}_{\widehat{\phi}_a} &= 0.\\ \boldsymbol{\delta}_{\widetilde{\phi}_a} &= \widetilde{\mu}_a\,\widehat{l}_a.\\ \boldsymbol{\delta}_{\check{\phi}_a} &= \check{\mu}_a\,\widehat{l}_a.\end{aligned} \tag{A-92}$$

- small perturbation in $M''(\mu_a)$ gives:

$$\begin{aligned}\boldsymbol{\delta}_{\widehat{\phi}_a} &= 0.\\ \boldsymbol{\delta}_{\widetilde{\phi}_a} &= \frac{1}{2}\,k_a\,\widehat{l}_a.\\ \boldsymbol{\delta}_{\check{\phi}_a} &= \frac{1}{2}\,\Gamma_a\,\widehat{l}_a.\end{aligned} \tag{A-93}$$

**Perturbation of the survival and the growth functions**

Say we perturb age $b$. For any $j > b$

$$
\begin{aligned}
\boldsymbol{\delta}_{\widehat{\mu}_j} &= \boldsymbol{\delta}_{\widehat{\mu}_{j-1}} + \boldsymbol{\delta}_{\widehat{g}_{j-1}} \\
\boldsymbol{\delta}_{\widetilde{\mu}_j} &= \boldsymbol{\delta}_{\widetilde{\mu}_{j-1}} + \boldsymbol{\delta}_{\widetilde{g}_{j-1}} + \boldsymbol{\delta}_{c_{j-1}}\, k_{j-1} + c_{j-1}\, \boldsymbol{\delta}_{k_{j-1}} \\
\boldsymbol{\delta}_{\check{\mu}_j} &= \boldsymbol{\delta}_{\check{\mu}_{j-1}} + \boldsymbol{\delta}_{\check{g}_{j-1}} + \boldsymbol{\delta}_{c_{j-1}}\, \Gamma_{j-1} + c_{j-1}\, \boldsymbol{\delta}_{\Gamma_{j-1}}
\end{aligned}
\tag{A-94}
$$

with

$$
\begin{aligned}
\boldsymbol{\delta}_{c_j} &= \frac{1}{\widehat{s}_j^2} \left( \boldsymbol{\delta}_{\widehat{\mu}_j}\, \widehat{s}_j \left( S''(\widehat{\mu}_j) \left(1 + G'(\widehat{\mu}_j)\right) + S'(\widehat{\mu}_j)\, G''(\widehat{\mu}_j) \right) - S'(\widehat{\mu}_j) \left(1 + G'(\widehat{\mu}_j)\right) \boldsymbol{\delta}_{\widehat{s}_j} \right) \\
\boldsymbol{\delta}_{k_j} &= \boldsymbol{\delta}_{k_{j-1}} \left(1 + G'(\widehat{\mu}_j)\right)^2 + 2k_{j-1} \left(1 + G'(\widehat{\mu}_j)\right) G''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j} \\
\boldsymbol{\delta}_{\Gamma_j} &= \boldsymbol{\delta}_{\Gamma_{j-1}} \left(1 + G'(\widehat{\mu}_j)\right)^2 + 2\Gamma_{j-1} \left(1 + G'(\widehat{\mu}_j)\right) G''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j}
\end{aligned}
\tag{A-95}
$$

For demographic rates, we show the derivative of the growth function. It is similar for the reproductive and survival functions:

$$
\begin{aligned}
\boldsymbol{\delta}_{\widehat{g}_j} &= G'(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j} \\
\boldsymbol{\delta}_{\widetilde{g}_j} &= G'(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widetilde{\mu}_j} + G''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j}\, \widetilde{\mu}_j + \frac{1}{2}\, G''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{k_j} + \frac{1}{2}\, G'''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j}\, k_j \\
\boldsymbol{\delta}_{\check{g}_j} &= G'(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\check{\mu}_j} + G''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j}\, \check{\mu}_j + \frac{1}{2}\, G''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\Gamma_j} + \frac{1}{2}\, G'''(\widehat{\mu}_j)\, \boldsymbol{\delta}_{\widehat{\mu}_j}\, \Gamma_j
\end{aligned}
\tag{A-96}
$$

Then, we use (A-69) and (A-70) to find $\boldsymbol{\delta}_{\widehat{\phi}_a}$, $\boldsymbol{\delta}_{\widetilde{\phi}_a}$ and $\boldsymbol{\delta}_{\check{\phi}_a}$

- a small perturbation in $S(\widehat{\mu}_b)$ gives

$$\begin{aligned}\boldsymbol{\delta}_{\widehat{s}_b} &= 1\\ \boldsymbol{\delta}_{c_b} &= -\frac{S'(\widehat{\mu}_a)\,(1+G'(\widehat{\mu}_a))}{\widehat{s}_a^2}\end{aligned} \tag{A-97}$$

- a small perturbation in $S'(\widehat{\mu}_b)$ gives

$$\begin{aligned}\boldsymbol{\delta}_{\widetilde{s}_b} &= \widetilde{\mu}_b\\ \boldsymbol{\delta}_{\check{s}_b} &= \check{\mu}_b\\ \boldsymbol{\delta}_{c_b} &= \frac{(1+G'(\widehat{\mu}_a))}{\widehat{s}_a}\end{aligned} \tag{A-98}$$

- a small perturbation in $S''(\widehat{\mu}_b)$ gives

$$\begin{aligned}\boldsymbol{\delta}_{\widetilde{s}_b} &= \frac{1}{2}\,k_b\\ \boldsymbol{\delta}_{\check{s}_b} &= \frac{1}{2}\,\Gamma_b\end{aligned} \tag{A-99}$$

- a small perturbation in $G(\widehat{\mu}_b)$ gives

$$\boldsymbol{\delta}_{\widehat{g}_b} = 1 \tag{A-100}$$

- a small perturbation in $G'(\widehat{\mu}_b)$ gives

$$\begin{aligned}
\boldsymbol{\delta}_{\widetilde{g}_b} &= \widetilde{\mu}_b \\
\boldsymbol{\delta}_{\check{g}_b} &= \check{\mu}_b \\
\boldsymbol{\delta}_{c_b} &= \frac{S'(\widehat{\mu}_b)}{\widehat{s}_b} \\
\boldsymbol{\delta}_{k_b} &= 2k_{b-1}\left(1 + G'(\widehat{\mu}_b)\right) \\
\boldsymbol{\delta}_{\Gamma_b} &= 2\Gamma_{b-1}\left(1 + G'(\widehat{\mu}_b)\right)
\end{aligned} \tag{A-101}$$

- a small perturbation in $G''(\widehat{\mu}_b)$ gives

$$\begin{aligned}
\boldsymbol{\delta}_{\widetilde{g}_b} &= \frac{1}{2}\, k_b \\
\boldsymbol{\delta}_{\check{g}_b} &= \frac{1}{2}\, \Gamma_b
\end{aligned} \tag{A-102}$$

**Perturbation of $v_G$, $v_I$ and $\beta$**

These perturbations will change $v_1$.

- a perturbation of $v_G$ gives

$$\boldsymbol{\delta}_f = \frac{\beta^2}{\widehat{R}_0} \sum_a \widehat{\phi}_a \, k_a \tag{A-103}$$

- a perturbation of $\beta$ gives

$$\begin{aligned} \boldsymbol{\delta}_f &= \frac{2\beta \, v_G}{\widehat{R}_0} \sum_a \widehat{\phi}_a \, k_a \\ \boldsymbol{\delta}_g &= -\frac{2\beta}{\widehat{R}_0} \sum_a \widehat{\phi}_a \, \Gamma_a \end{aligned} \tag{A-104}$$

- a perturbation of $v_I$ gives

$$\boldsymbol{\delta}_f = 1 \tag{A-105}$$

### A.4.2 Elasticity of r

We use (A-29) to find $\boldsymbol{\delta}_r$

$$\boldsymbol{\delta}_r = \frac{\sum_a e^{-ra}\,\boldsymbol{\delta}_{\phi_a}}{\sum_a a\,e^{-ra}\,\phi_a}. \tag{A-106}$$

We used a similar method as described above to find $\boldsymbol{\delta}_{\phi_a}$ in each perturbation case.

**Table S1**: Parameters used to build the demographic rates for the short-lived and long-lived life-histories. The reproductive functions have been modeled with a logit and an exponential link for the long- and short-lived life-histories, respectively. The survival function has been modeled with a logit link for both life-histories.

| | | | short | long |
|---|---|---|---|---|
| **Surv.** | newb. | $a$ | -3 | 0 |
| | | $b$ | 0.3 | 0.3 |
| | | $Sm$ | 0.5 | 1 |
| | juv. | $a$ | -3 | -1.5 |
| | | $b$ | 0.4 | 0.15 |
| | | $Sm$ | 0.9 | 1 |
| | ad. | $a$ | -1000 | -2.5 |
| | | $b$ | 0 | 0.15 |
| | | $Sm$ | 0 | 1 |
| **Repro.** | juv. | a | 1 | -2 |
| | | b | 0.2 | 0.1 |
| | adult | $a$ | 1 | -2 |
| | | $b$ | 0.2 | 0.2 |
| **Growth** | newb. | $a$ | 4 | 12 |
| | | $b$ | 1.2 | 1.5 |
| | juv. | $a$ | 6 | 10 |
| | | $b$ | 0.8 | 0.7 |
| | ad. | $a$ | 0 | 10 |
| | | $b$ | 0 | 0.7 |
| **Inher.** | | $v_G$ | 4 | 5 |
| | | $\mu_1$ | 3 | 2 |
| | | $\beta$ | 0.2 | 0.2 |
| | | $v_{inh}$ | 1 | 0.1 |

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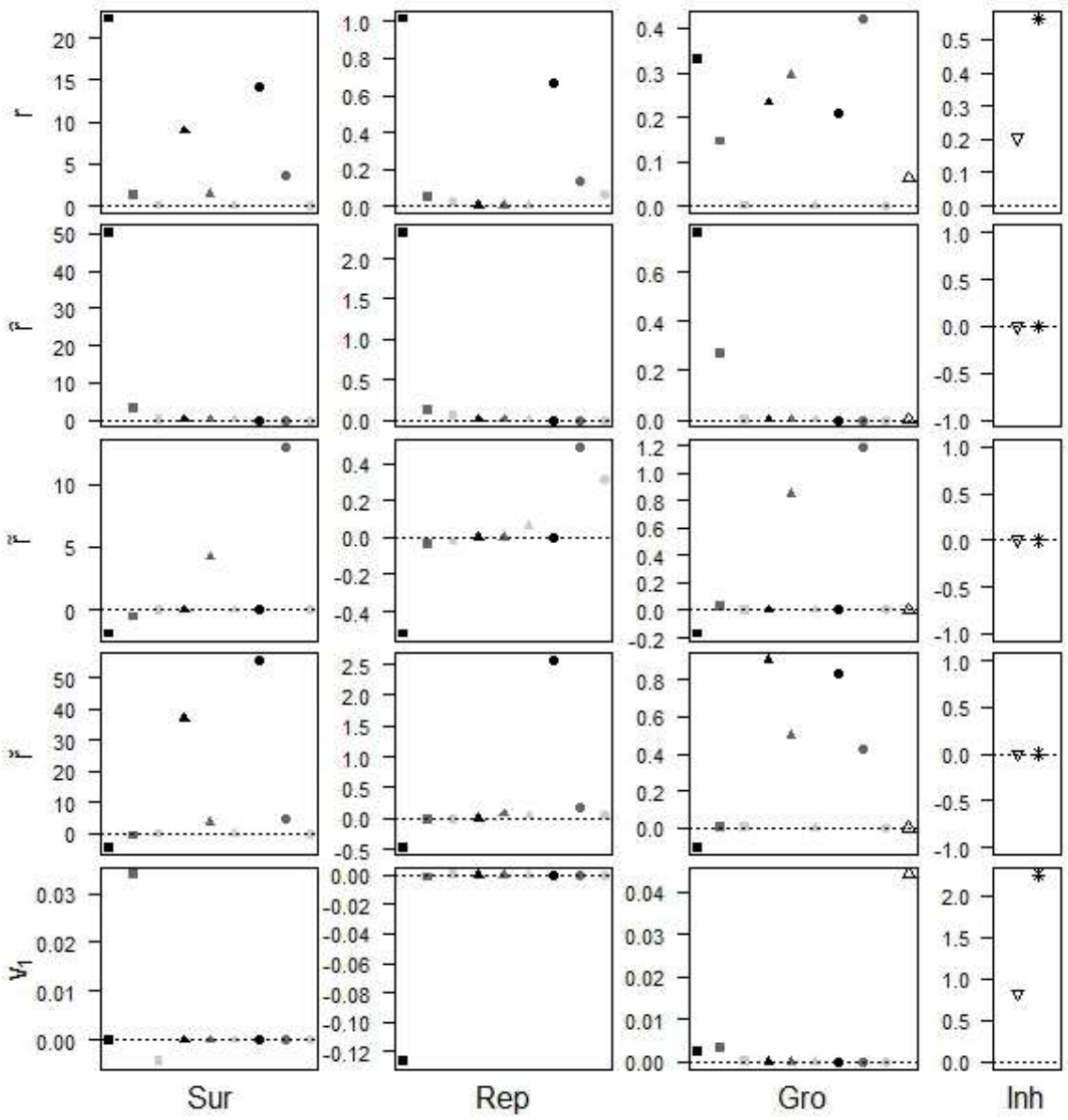

r
r̃
r̆
v1
Sur
Rep
Gro
Inh

**Figure S1**: Elasticities of $r$, $\widehat{r}$, $\widetilde{r}$, $\check{r}$ and $v_1$ for the short-lived life-history. The first 3 columns are elasticities to survival, reproduction and growth all computed at the mean phenotype of each age class. Elasticities shown are to values of each quantity (mean of each function at each age, squares), first derivatives (triangles) and second derivative (circles). The column for growth also shows elasticity to the variance of the growth $v_G$ (open triangles). The last column shows elasticities to the inheritance variance $v_I$ (open inverted triangles) and to $\beta$ (stars). Black and, dark and light grey shades are used to indicate perturbations affecting newborn, juvenile and adult age-class, respectively.

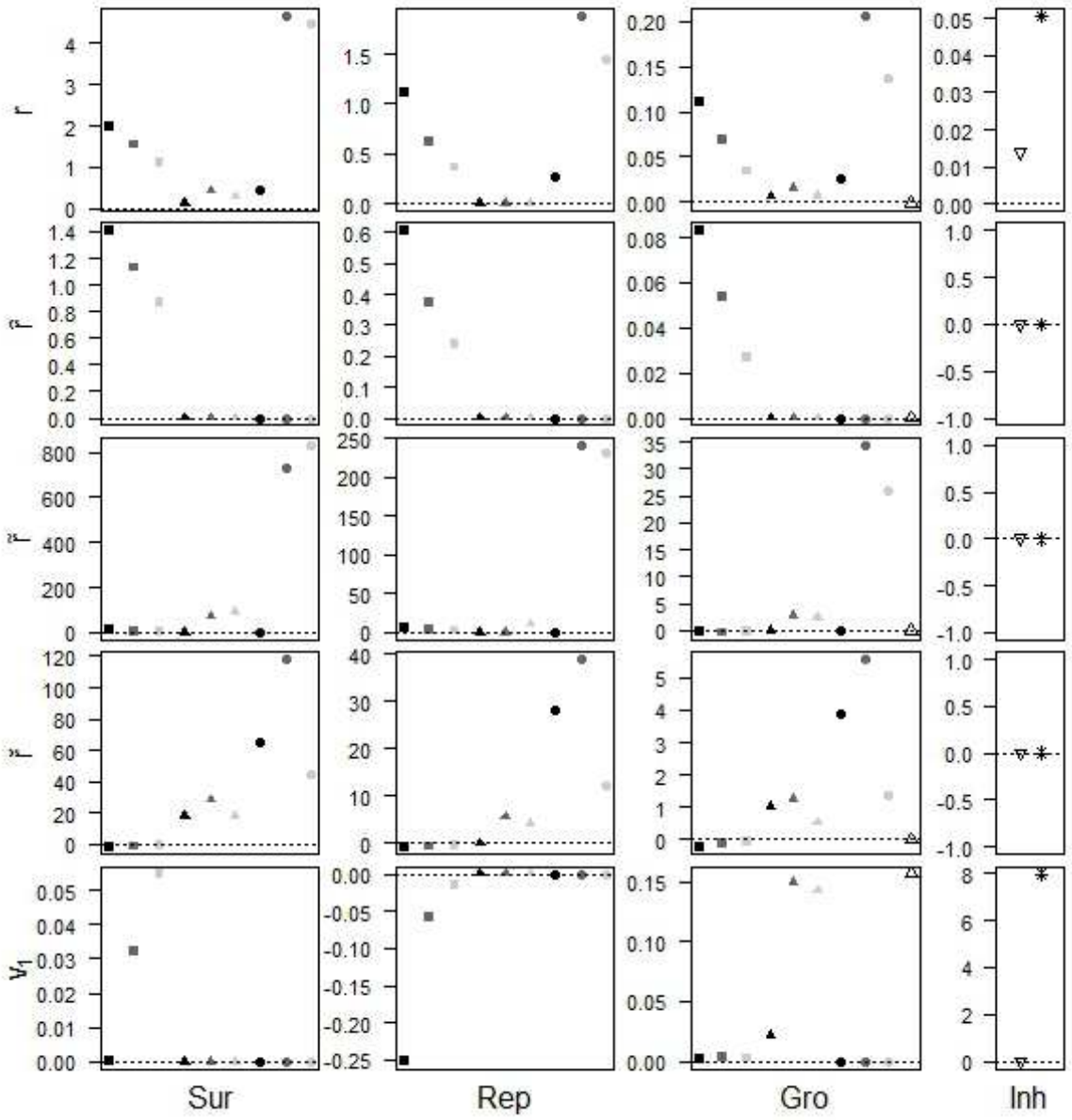

r
r̃
r̂
r̆
v1
Sur
Rep
Gro
Inh

**Figure S2**: Elasticities of $r$, $\widehat{r}$, $\widetilde{r}$, $\check{r}$ and $v_1$ for the long-lived life-history. The first 3 columns are elasticities to survival, reproduction and growth all computed at the mean phenotype of each age class. Elasticities shown are to values of each quantity (mean of each function at each age,n squares), first derivatives (triangles) and second derivative (circles). The column for growth also shows elasticity to the variance of the growth $v_G$ (open triangles). The last column shows elasticities to the inheritance variance $v_I$ (open inverted triangles) and to $\beta$ (stars). Black and, dark and light grey shades are used to indicate perturbations affecting newborn, juvenile and adult age-class, respectively.

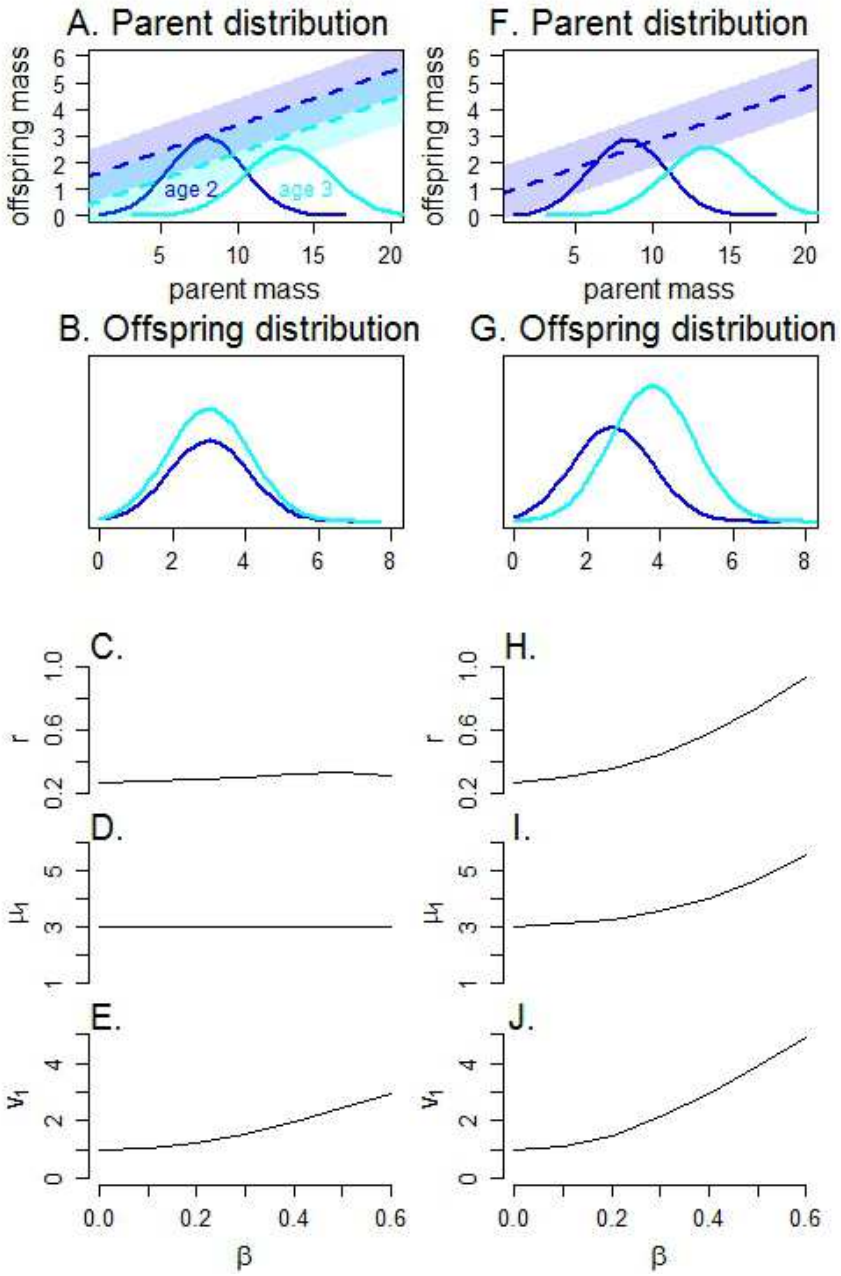

A. Parent distribution
F. Parent distribution
offspring mass
parent mass
age 2
age 3
B. Offspring distribution
G. Offspring distribution
C.
H.
r
D.
I.
μ1
E.
J.
v1
β

**Figure S3**: Influence of the inheritance function on the newborn phenotype distribution and on the population growth rate for the short-lived life-history. In the left column (A-E), inheritance follows model A, (3). In the right column (F-J), inheritance follows model B, (20), so that newborns of mothers of different ages have different mean body mass. (A,F) the inheritance function is presented along with parental phenotypic distributions. (B, G) shows the offspring distribution resulting of the parental distribution and the inheritance functions presented in A and G. The others panels show how increasing strength of inheritance affects $r$ (C, H), $\mu_1$ (D, I) and $v_1$ (E, J).

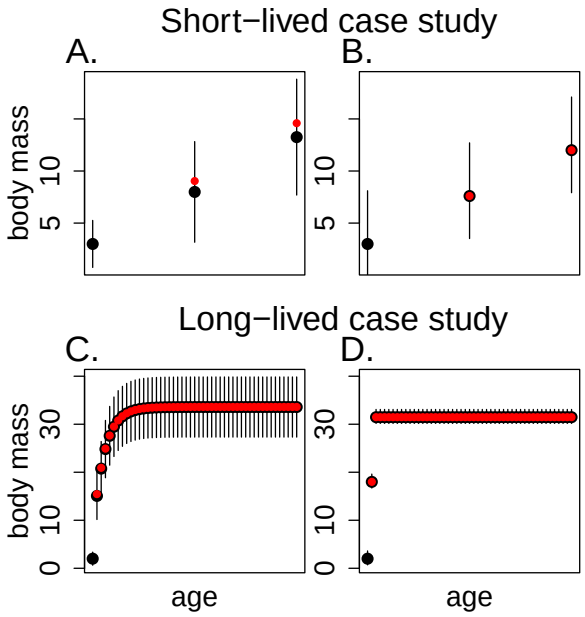
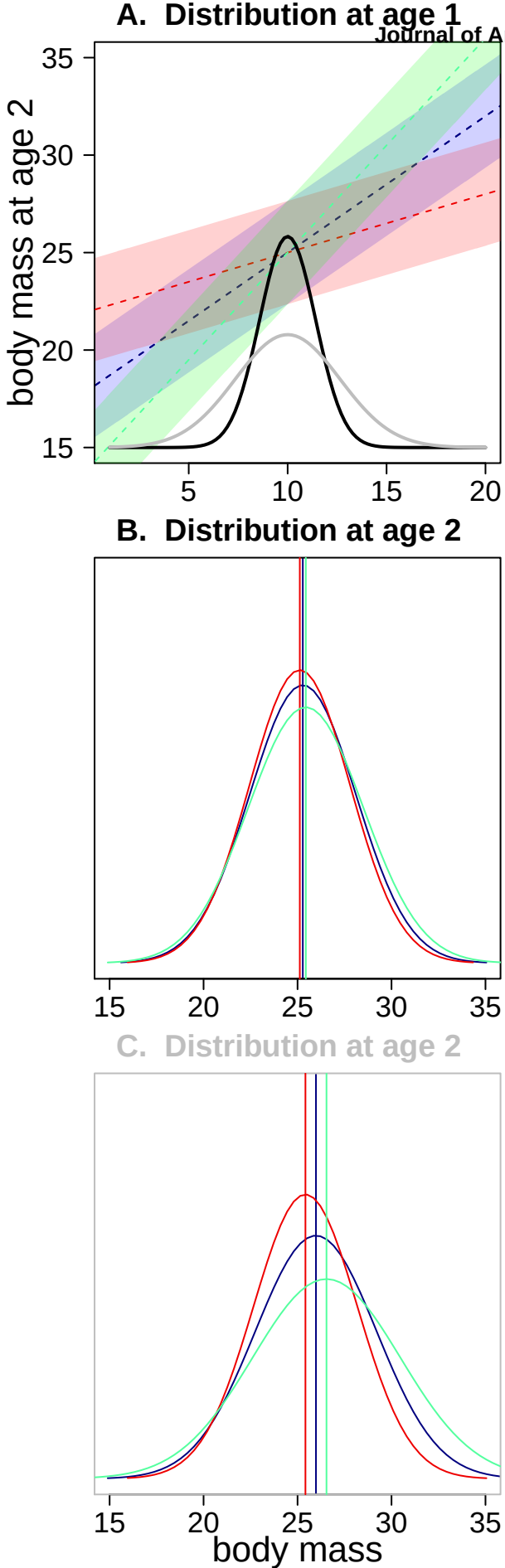
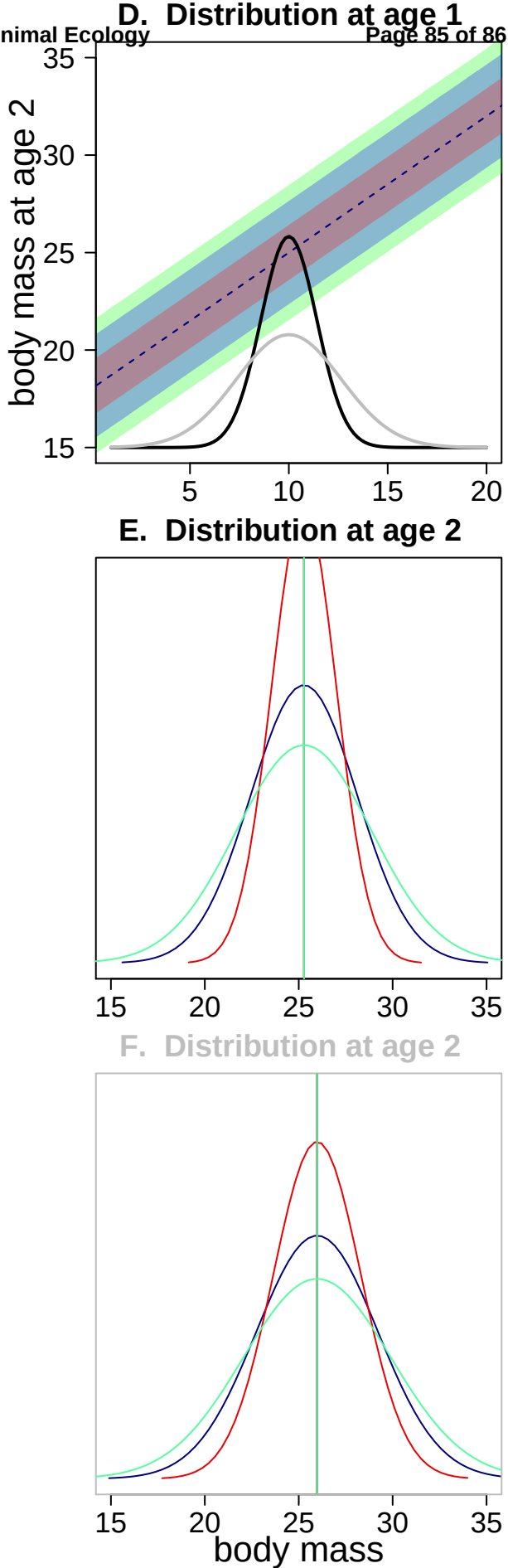
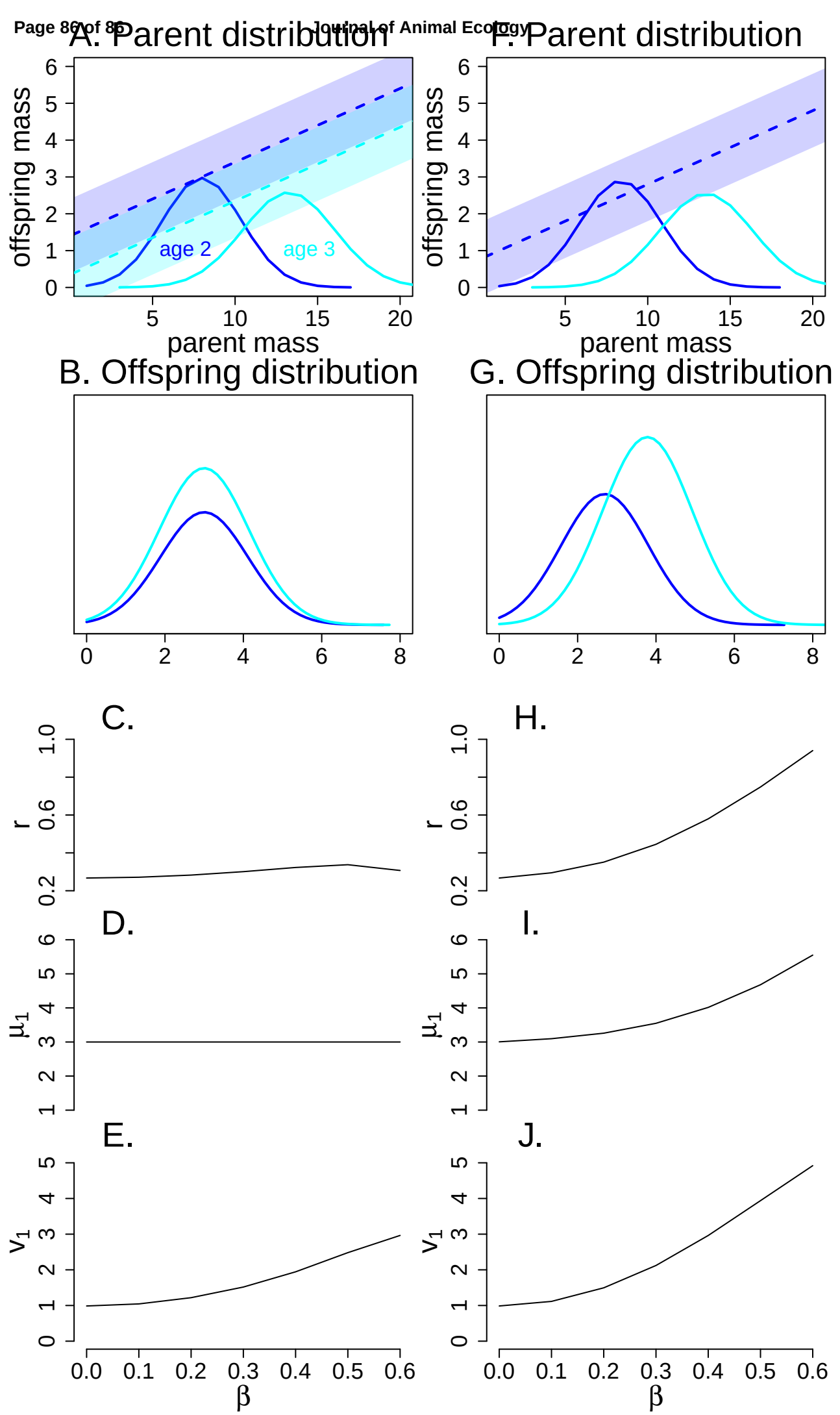



**Figure S4**: Influence of selection, and stochasticity in growth on the population growth trajectories. The two case studies including selection and stochasticity of growth are presented in the right column (A, C). In the left column (B, D), there is no selection (slopes of survival, growth and reproductive function are null) and no stochasticity in growth. The black and red points show the mean body mass of all individuals and actual mother, respectively at each age.


A. Distribution at age 1
B. Distribution at age 2
C. Distribution at age 2
body mass at age 2
body mass
15
20
25
30
35
5
10
15
20


D. Distribution at age 1
E. Distribution at age 2
F. Distribution at age 2
body mass at age 2
body mass
15
20
25
30
35
5
10
15
20


A. Parent distribution
F. Parent distribution
offspring mass
parent mass
age 2
age 3
B. Offspring distribution
G. Offspring distribution
C.
H.
r
D.
I.
μ1
E.
J.
ν1
β