A demographic perspective on trait heritability and sex differences in life history

A thesis submitted for the degree of Doctor of Philosophy

by

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Abstract

A demographic perspective on trait heritability and sex differences in life history—D.Phil. thesis by Julia A. Barthold

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Biologists have long used demographic approaches to answer questions in ecology and evolution. The utility of these approaches has meant a constant development and refinement of methods. A key milestone has been the development of phenotype structured population models that link ecology and evolution. Moreover, biostatistical research steadily improves methods to coax demographic information from scarce data. In this thesis, I build upon some of the recent advances in the field. My first three studies focus on the consequences of sex differences in life history for population dynamics. Firstly, I test whether males matter for the dynamics of African lion (*Panthera leo*) populations via a previously unquantified mechanism: the inheritance of phenotype from father to offspring. Secondly, I develop a method to estimate age-specific mortality rates for both sexes in species where one of the sexes disperses around the age of maturity. Thirdly, I apply this method to study variation in mortality between the sexes and between two populations of African lions. After these three chapters, which make contributions to the field of sex-structured population dynamics, I focus on the integration of phenotype structured modelling and quantitative genetics. I illustrate how heritability of a quantitative character that develops with age depends on (i) viability selection, (ii) fertility selection, (iii)

the development of the phenotype with age, and (iv) phenotype inheritance from parents to offspring. Our results question the adequacy of quantitative genetics methods to obtain unbiased estimates of heritability for wild populations. This thesis advances our understanding of population development over ecological time scales. This knowledge has applications in conservation and population management, but also contributes to untangling evolutionary processes in wild animals.

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Author contributions

Chapter 1

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Chapter 2

Authors: Julia A. Barthold (JB), Susanne Schindler (SS), Owen R. Jones (OJ), Craig Packer (CP), and Tim Coulson (TC)

JB, OJ, and TC conceived the research question. JB conducted the analysis and wrote the manuscript. SS advised on the construction of the model and provided MATLAB code of a similar model for translation into the statistical computing language R. CP provided data and contributed expertise on lion biology. TC gave general guidance and advised on the statistical analysis. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 3

Authors: Julia A. Barthold (JB), Craig Packer (CP), Andrew J. Loveridge (AL), David W. Macdonald (DM), and Fernando Colchero (FC)

JB conceived the research question and wrote the manuscript. Analysis was conducted by JB with support from FC. CP provided data, and AL and DM contributed expertise on lion biology. FC provided expert knowledge on hierarchical Bayesian

models for the development of the method. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 4

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JB conceived the research question, conducted the analysis with support from FC, and wrote the manuscript. AL, CP, and DM provided data and contributed expertise on lion biology. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 5

Authors: Julia A. Barthold, Floriane Plard (FP), Jean-Michel Gaillard (JMG), Tim Coulson (TC), and Shripad Tuljapurkar (ST)
JMG, TC, and ST conceived the research question. ST developed the mathematical framework. JB and FP expanded the framework, wrote the computer code, and conducted the analysis. JB adapted a published age phenotype structured integral projection model (IPM) for the analysis. FP and JMG provided a second, unpublished IPM. JMG, TC, and ST advised on the analysis. JB wrote the manuscript. All co-authors contributed to the interpretation of the results and commented on the manuscript.

Chapter 6

Author: Julia A. Barthold

Contents

CHAPTER 1

Introduction

Biologists have long delved into the secrets of life by the apt use of demographic methods (Fisher 1930; Charlesworth 1994). Demographic approaches infer the densities of populations from the fates of individuals. At the core of these approaches is the decomposition of population development into four process: two of them add individuals to populations—fertility and immigration—and two of them deduct individuals—mortality and emigration. These processes appear simple, but complex population dynamics can arise from variation among individuals. Individuals can vary in the probabilities of reproduction, mortality, and migration according to individual characteristics, environmental factors, and the density or composition of populations.

The wish to understand the temporal and spatial dynamics of population densities drove the development of a plethora of demographic models. Among these models, structured population models account for individual characteristics, stochastic models integrate environmental factors, density-dependent models allow for density feedbacks, and frequency-dependent models take care of composition feedbacks (Caswell 2001). These models have yielded valuable insights into the evolution of

life histories in plants and animals (Childs et al. 2003; Metcalf et al. 2008; Childs et al. 2011), rapid phenotypic and population responses to climate change (Coulson 2001; Pfeifer et al. 2006; Ozgul et al. 2009, 2010; Coulson et al. 2011) and to selective harvesting (Traill et al. 2014), projected population development of endangered species (Goutte et al. 2014; Jenouvrier et al. 2014), the spread of invasive species (Jongejans et al. 2011; Caplat et al. 2012), and the transient dynamics of populations after ecological disturbances (Stott et al. 2011, 2012).

But demography not only helps to understand the dynamics of animal and plant populations, demographic rates and summary statistics are also vital as measures of fitness or fitness components (Metcalf and Pavard 2007). On them hinges evolutionary hypothesis testing in behavioural ecology, sociobiology, and life history evolution. Life history theory itself is concerned with the evolution of optimal demographic trajectories (Stearns 1992), and demographic model representations of species' life histories aid understanding of this from a comparative perspective (Sæther and Bakke 2000; Jones et al. 2014). Demographic models exploiting matrix algebra have been parameterised for thousands of animal and plant species. The matrices containing the demographic parameters are now being combined to rich comparative data sets, which allow ecological questions to be addressed at the global scale (Salguero-Gómez et al. 2015, submitted). With continuously growing availability of data and new methodology, the interface between ecology, evolution, and demography is a lively field of research.

This thesis contributes to some of the current developments. The first part (Chapter 2 to 4) focusses on the consequences of dioecy for population dynamics. The second part (Chapter 5) features the integration of quantitative genetics and phenotype structured demographic modelling. Each research chapter receives here a brief introduction, before some guiding remarks send the readers on their way to unlock the remainder of the thesis.

Does sex matter for population dynamics? (Chapter 2)

Sex is ancient and virtually ubiquitous. Sexual reproduction emerged roughly 1.2 billion years ago (Butterfield 2000), and 99.9% of eukaryotes engage in it, at least occasionally (Otto and Lenormand 2002). It provides the basis for sexual selection to operate on phenotypes, and has therefore deeply contributed to shaping the natural world as we know it. Consequently, sex pervades almost everything we study in biology. Yet ecologists, when modelling population dynamics, tend to ignore it.

Despite a long-standing recognition that both sexes may matter for population dynamics (Goodman 1953), ecologists commonly revert to exclusively modelling the female part of the population. Female-only models operate with the assumption that males do not substantially influence the dynamics of the population. This can be a fair assumption for species in which the vital rates do not vary between the sexes (Caswell and Weeks 1986), but these species are rare.

Early concerns about the standard practice of excluding males from demographic models have gained traction, supported by theoretical models and empirical studies (Caswell and Weeks 1986; Lindström and Kokko 1998; Ranta et al. 1999; Mysterud et al. 2002). According to these studies, males can affect population dynamics via a number of mechanisms. Male abundance can certainly restrict population growth if there are not enough males to inseminate every fertile female in a population (Jenouvrier et al. 2010; Milner-Gulland et al. 2003). But even in species with a polygynous mating system, where the number of male mating partners is usually of no concern, males can play a role for population dynamics. Their abundance has been found or suggested to affect female survival (Chapman et al. 1995; Reale et al. 1996; Le Galliard et al. 2005), female development and migration (Mysterud et al. 2002; Rankin and Kokko 2007), offspring survival (Swenson et al. 1997; Whitman et al. 2004), and the sex ratio and size of offspring (Sæther et al. 2004; Mainguy et al. 2009).

Furthermore, in species with positive viability and fertility selection on a phe-

notype, the phenotype distribution among males may affect population growth if there is a positive relationship between paternal and offspring phenotype (Schindler et al. 2013). Let us for example consider body size as the phenotype and assume that large males have higher survival probabilities and sire more offspring than small males. If there is a positive relationship between paternal and offspring body size, then the offspring of large males will be born with larger body sizes than the offspring of small males. These larger offspring have a survival advantage over their smaller peers. If large males were removed from the population, the mean and variance of offspring body size would decrease. Since smaller offspring have lower survival probabilities, this may depress population growth.

If this mechanism were to affect population growth, I would find decreased population growth after shifting the body size distribution of males towards smaller sizes. This is what I studied in Chapter 2 by building and analysing a phenotype structured model that included both males and females. I used data from free-living African lions (*Panthera leo*) and complemented data with theoretical assumptions where they prove too scarce to inform the model. I found that the mechanism was detectable—altering the male size distributions affected the population growth rate—but the effect size was very small.

Fighting male data deficiency with statistics (Chapter 3)

One of the factors that deter ecologists from including males in their population models is that demographic data of males are scarce. In large mammals, females usually suckle their offspring and motherhood can be easily inferred from physical proximity and suckling behaviour. Males, in contrast to this, commonly only contribute sperm to offspring production, so that fatherhood is much harder to discern, particularly in species where females mate either covertly or with multiple males. Similarly, estimating survival is commonly easily accomplished for females but not for males. This holds for the many species of large vertebrates where males leave

their natal area or social group at maturity. If dispersing males leave areas that are monitored by capture-recapture field sites, they are usually lost for data collection. The possibility that males that are not re-sighted any longer may have died or dispersed renders the fate of all missing males uncertain. While missing females are likely dead, even if their bodies are not found, missing males may be dead or alive. These uncertain male records make it impossible to estimate age-specific mortality rates for males using existing methods.

For species where availability of male mortality estimates would improve inference on ecological and evolutionary processes, yet the data contain uncertain male records, new statistical methodology may provide a solution. In Chapter 3, I developed a Bayesian hierarchical model that imputes dispersal state (i.e., died or left) for uncertain male records as a latent state jointly with the coefficients of a parametric mortality model. I checked the performance of my model by simulating data and testing whether the model could accurately retrieve the mortality parameters. I applied my model to lion data that contained expert indications on whether a missing male had likely dispersed or died. I used this extra information to evaluate potential biases in the mortality parameters estimated by my model. I found that my model performed well at retrieving the mortality parameters from the simulated data. My analysis of the lion data yielded the insight that my estimates may be slightly biased, yet to an acceptable degree for this otherwise insurmountable problem.

Mortality of lions (Chapter 4)

In most large mammal species, males live shorter lives than females (Promislow 1992; Clutton-Brock and Isvaran 2007), and lions are no exception. This is common knowledge, yet the details about how mortality of lions varies between the sexes and populations remain unknown. The global lion population is plummeting and many small fragmented populations face local extinction (Riggio et al. 2012). Populations are further decimated through the common practice of shooting males for trophies

(Packer et al. 2011). Trophy hunting is an important yet controversial conservation tool and setting shooting quota at sustainable levels a vital task (Loveridge et al. 2007; Lindsey et al. 2012). Consequently, multiple demographic models evaluate consequences of male offtake on lion population growth (Whitman et al. 2004, 2007; Becker et al. 2013). But none of these models use estimates of male age-specific mortality estimates, because such estimates do not exist. Estimating lion mortality with existing survival models derived from the Cormack-Jolly-Seber framework (Cormack 1964; Jolly 1965; Seber 1965) has failed because lion data contain the type of uncertain male records described above.

In Chapter 4, I therefore used my newly-developed Bayesian hierarchical model to estimate sex- and age-specific mortality rates for two populations of African lions. I aimed to study variation in mortality between the sexes and between the different populations. One of the populations lives at high-densities and mostly undisturbed by humans, whereas the other one lives in close proximity to human settlements and is exposed to mortality through poaching, accidents, and trophy hunting. My findings confirmed that males die younger than females. In fact, they had higher mortality rates across all ages. I also found that the two populations varied consistently, and partly unexpectedly, in both male and female age-specific mortality. I trust that my mortality estimates will find use in future models to inform lion population management and identify density effects on lion survival as a research area that may need heightened attention.

Determinants of heritability in long-lived iteroparous species (Chapter 5)

With Chapter 4, this thesis concludes its contribution to the field of sex-structured population dynamics. Chapter 5 instead focuses on another recent development at the intersection of ecology, demography, and evolution—the fusion of structured population modelling and quantitative genetics. In 2010, Coulson et al. introduced how heritability, a quantity that is central to quantitive genetics, can be calculated

from age and phenotype structured integral projection models (IPMs). IPMs project the temporal dynamics of the density distribution of a quantitative character like size or body mass (Coulson et al. 2010). The mechanics behind this projection can be used to compute the phenotype density distributions of parents and their offspring at the same age, the two distributions that determine heritability.

Heritability is defined as the proportion of phenotypic variance attributable to additive genetic variance (Jacquard 1983). Developed in animal and plant breeding, it predicts the phenotypic response to directional selection on a heritable character (Falconer and Mackay 1996). It is therefore a key quantity for understanding microevolutionary processes in free-living populations (Merilä et al. 2001). Heritability is commonly statistically inferred from measurements of phenotypes at the same age among relatives (Wilson et al. 2010). While statistical methods aptly exploit the covariation in phenotype among relatives to estimate heritability, it is not clear how this covariation arises for species where the phenotype develops with age.

Let us for example assume the phenotype is body mass and the relatives under concern are parents and offspring. First a cohort of potential future parents is born and each potential parent has a birth phenotype. These individuals may survive, and if they do they grow. At various points throughout their life they may produce offspring. The probabilities of survival and reproduction may be increasing with increasing body mass, creating positive fertility and viability selection on the phenotype. Finally, the offspring recruit to the population with body masses that may be determined by the parental phenotype and age. We have therefore four potential processes that may affect the parent and offspring birth phenotype distributions and therefore heritability: viability and fertility selection, phenotype transition with age, and phenotype transition from parents to offspring. But which of these processes are most influential to the heritability of body mass at birth? This is a question that has not been widely addressed and is what I asked in Chapter 5.

In an IPM framework, the two selection processes and the two phenotype tran-

sition processes are modelled separately but jointly determine the development of the phenotype density distribution over time. Since IPMs decompose the temporal dynamics of the phenotype distribution into these four processes—differential survival, differential fertility, phenotype development with age, and parent-offspring phenotype transition—they can be used to study how each process affects heritability.

In Chapter 5, I studied the demographic determinants of heritability for two long-lived iteroparous large vertebrates, Soay sheep (*Ovis aries*) and roe deer (*Capreolus capreolus*) by analysing two age phenotype structured IPMs. I derived the analytical expressions for calculating biometric heritability and its elasticities with respect to changes in the model parameters. I found that all four processes are intricately intertwined in determining heritability, which raises the question whether current quantitative genetics methods are suited to estimate heritability in free-living animal populations.

Closing remarks

This excursion into quantitive genetics and phenotype structured demographic modelling concludes my thesis research. As an introductory note, it remains to give a few guiding remarks regarding the structure of the thesis. Every chapter is written as a stand-alone manuscript and therefore has its own supporting information section and bibliography. As a concession to this document being a thesis, and to enhance readability, all figures and tables are integrated in the text and not, as is otherwise usual, at the end of the manuscript. The research chapters (Chapters 2-5) are followed by a general summary and conclusion of the work (Chapter 6). The general introduction (this Chapter) and discussion (Chapter 6) have a joint bibliography at the end of the thesis. The appendix of the thesis contains a paper I have published on sex differences in human fertility during the time of my D.Phil., that I ultimately decided to exclude from the main body of the thesis. It overlaps to a

questionable extent with work I had done during a demographic doctoral school for which I received, in the end, a master's degree. However, in my regard it is part of my thesis research and as such included here. I furthermore added a paper, on which I am a co-author, that deals with the fundamentals of integrating quantitative genetics and structured population modelling. I make multiple references to it in Chapter 5. With this guidance, I hope, it will be possible to understand the theme that connects my thesis research, in which I took a demographic perspective on sex differences in life histories and trait heritability.

CHAPTER 2

Paternal inheritance of body size fails to challenge population biology's single-sex paradigm

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Phenotype inheritance and sex-structured population dynamics

Title

Paternal inheritance of body size fails to challenge population biology's single-sex paradigm

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Abstract

Males are usually omitted from population models with the assumption that they do not affect population dynamics. The only regularly occurring exceptions to this single-sex paradigm are models of systems where female fecundity is limited by availability of male mating partners. However, other mechanisms may also violate the assumption that males do not matter for population dynamics. Males can, for example, affect the offspring sex ratio at birth, adult female, or offspring, survival and development, or the body size distribution of offspring. Here, we test whether the inheritance of body size from fathers to offspring is a mechanism by which males affect population dynamics. We use a two-sex integral projection model (IPM) that simultaneously tracks the size distributions of both sexes and in which offspring size depends on the sizes of both parents. We parameterised this model using data from Serengeti lions (*Panthera leo*). From the model at equilibrium, we calculate several population statistics including mean body size of each sex, population growth rate, and female lifetime reproductive success. We perturb the model to evaluate how changes to the male and female size distributions affect population dynamics. These perturbations reveal that the population is mainly regulated by female growth and survival. They also show that the size distribution of the male population does not affect the dynamics of the female population, or those of the whole population. Thus, in this case, paternal inheritance does not challenge population biology's single-sex paradigm. Nevertheless, we show that two-sex IPMs are useful tools for studying nonlinear dynamics in sex-structured populations.

Key words

African lion; eco-evolutionary dynamics; inheritance; paternal effects; sex differences; sexual dimorphism; sex ratio; sex-structured population model; two-sex model

Introduction

Female-only population models are by far the most widely used kind of model in population ecology and life history evolution. Even if not explicitly stated, these models make the strong assumption that the ratio of males to females, either in total or within age, stage, or size classes, does not influence population development (hereafter referred to as intersexual frequency dependence) (Caswell and Weeks 1986; Caswell 2001). Furthermore, they also assume that the male population mirrors the female population when inferences are extrapolated to the total population. However, species that conform to these assumptions may be rare. In many species, population structure is sexually dimorphic due to sex differences in life history and morphology (Promislow 1992; Promislow et al. 1992; Owens and Bennett 1994; Clutton-Brock and Isvaran 2007).

Moreover, the relative size or structure of the male population can influence population dynamics. It is well-known that availability of males can limit female reproduction (Das Gupta 1972; Milner-Gulland et al. 2003; Jenouvrier et al. 2010). But less well-known mechanisms may pose an even greater challenge to the single-sex paradigm of population biology. Males have for example been shown or suggested to affect female survival (Chapman et al. 1995; Reale et al. 1996; Le Galliard et al. 2005), female development and migration (Mysterud et al. 2002; Rankin and Kokko 2007), offspring survival (Swenson et al. 1997; Whitman et al. 2004), and the sex ratio and size of offspring (Sæther et al. 2004; Mainguy et al. 2009). Through these mechanisms males may play a role in population development even in species where polygynous or polygynandrous mating systems are thought to render males unimportant for population growth (Mysterud et al. 2002; Rankin and Kokko 2007). Little is known about the dynamics of sex-structured populations, not least because to our knowledge no empirical two-sex population model exists that studies the significance of mechanisms other than male-limited female fecundity.

Schindler et al. (2013) have recently developed a two-sex variant of the integral projection model (IPM) that can be adapted to model different mechanisms for intersexual frequency dependence. IPMs are structured population models that track the distribution of a quantitative character like size or body mass (Easterling et al. 2000; Rees and Ellner 2009). They project the number of individuals at different trait values by using trait transition rates (i.e., development and inheritance) along with trait-specific demographic rates (i.e., survival and reproduction). From the modelled trait distribution various statistics underpinning population ecology, evolutionary biology, and life history can be derived (Coulson 2012). These statistics jointly change when model functions are perturbed. Therefore, IPMs can be used to simulate joint responses to environmental change of various ecological and evolutionary quantities like population growth rate, lifetime reproductive success, and trait heritability (Coulson et al. 2010; Smallegange and Coulson 2012).

The two-sex IPM of Schindler et al. (2013) simultaneously projects the trait distributions of both sexes by using separate male and female demographic and trait transition rates. A mating function models the formation of mating pairs (male-female pairs that reproduce with each other) according to the number of males and females at different trait values to ensure consistent population development between the sexes (i.e., every recruit must have both a father and a mother). Schindler et al. (2013) included intersexual frequency dependence in their model by assuming that, compared to larger males, smaller males sired smaller offspring with lower survival. They parameterised their framework for Columbian ground squirrels (*Urocitellus columbianus*), first assuming sexual monomorphism in size and vital rates, and found that artificially introducing sex differences in the size structure amplified the effect of size-selective mating systems on the population growth rate. These findings led us to hypothesise that, in species with substantial sexual dimorphism in size and vital rates, the relative number of males compared to females affects population dynamics due to paternal inheritance of size.

Here we tested this hypothesis using a two-sex IPM for a sexually dimorphic species. A fully empirical model would have required information on size- and sex-specific fertility, mortality, growth, inheritance, and mating probabilities from a free-living population of a sexually dimorphic species. However, such data sets are rare and often not accessible. We therefore used data from Serengeti lions (*Panthera leo*) that contained most of the relevant information and substituted missing information with reasonable assumptions. For a large free-living social carnivore the data were of high quality, but they were not sufficiently detailed to model density dependence and age effects. The model therefore approximates a population of a large sexually dimorphic carnivore but its precision in capturing the population dynamics of lions must be treated with caution.

Our IPM projected the male and female size distributions from which we calculated population growth rate, female proportion of the population, means and variances in size, female lifetime reproductive success, and female generation time. We first evaluated the performance of the model by comparing model predictions at population equilibrium with results from observations. We then conducted a perturbation analysis by changing the sex ratio at recruitment and parameters of model functions one-by-one. Perturbations revealed the relative importance of each model function to the various model predictions. They therefore showed whether the influence of the male size distribution on the female size distribution, the female life history descriptors, and the overall population development was strong enough to warrant a two-sex model for this population.

Methods

Data

The population of lions in Serengeti National Park, Tanzania, has been studied since 1966. The 2000 km^2 study area lies at the heart of the Serengeti-Mara ecosystem,

a site with mostly seasonal rainfall and a southeast to northwest gradient in vegetation from short to tall grassland to woodlands (Packer 2005; Mosser et al. 2009). Since 1984, when radio telemetry first enabled deliberate localisation of all prides, demographic censuses of each pride, recording births, deaths, and migrations of all pride members, generally occurred at least once every two weeks. Individual lions were identified from whisker-spot patterns and natural markings (Packer et al. 1991). Dates of birth were inferred from the characteristic behaviour of females around parturition (Packer et al. 2001). Female pride mates often give birth at the same time and communally rear litters (Packer et al. 1988a), and when this occurred, the size of the communal litter and the number of mothers were recorded. In total, we had demographic records on 4393 individuals up until 2010. Morphological data were collected from 42 prides between 1984 and 2009. As a proxy for body size, we used a lion's breast circumference known as heart girth (Bertram 1975). Measurements were routinely collected whenever a lion was immobilised, either for fitting radio collars or during disease surveys and genetic studies. For model parameterisation, we used a total of 291 female and 203 male heart girth measurements. Some individuals were measured twice during their lives 52 females and 32 males. Therefore, taking into account repeated measures, we had a total of 239 females and 171 males for which there were heart girth measurements. We had heart girth measurements of offspring at different ages for 82 female and 53 male offspring.

Constructing the IPM

We built a non-age-structured, deterministic, and density-independent two-sex IPM of the distributions of heart girth using the general framework of Schindler et al. (2013). Data limitations precluded a more complex model, for example one where the trait transition rates and trait-specific demographic rates varied not only with size but also with age. The model was intersexually frequency dependent and therefore nonlinear since the offspring heart girth distributions of both sexes depended

on both the maternal and paternal heart girth distributions. However, as with other lion population models (Caro et al. 2009; Becker et al. 2013), it was female-dominant (sensu Caswell 2001) in that the number of offspring depended only on the female heart girth distribution (i.e., the model was homogeneous in the number of females). The independence of the number of offspring from the number of males was assumed based on the polygynous mating system of lions and their social organisation into mixed-sex prides, both of which indicate that fertilisable females always find a mating partner. Since number of offspring was independent of males, we could attribute any changes in population statistics after perturbing the male size distribution to occur via the mechanism of paternal inheritance.

To parameterise the IPM we identified the following functions of heart girth, where subscripts f and m indicate functions for females and males, and where x denotes female and y denotes male heart girth: survival $S_f(x,t)$, $S_m(y,t)$, growth $G_f(x' \mid x,t)$, $G_m(y' \mid y,t)$, inheritance $f_f(x' \mid x,y,t)$, $f_m(y' \mid x,y,t)$, fertility $R_{f,m}(x,y,t)$, and mating function $M_{f,m}(x,y,t)$ (for details on the functional forms see Table S1 and the section *Parameterising the IPM* below).

We included two additional terms in our model: the proportion of female recruits, s, and a normalisation constant, $C_{n_f,n_m}(t)$, which we describe below. The model predicted the female and male distributions of heart girth, $n_f(x',t+1)$ and $n_m(y',t+1)$, at $t+1$ given the heart girth distributions at time t, denoted by $n_f(x,t)$ and $n_m(y,t)$.

$$\begin{aligned} n_f(x',t+1) = &\int G_f(x' \mid x,t)\, S_f(x,t)\, n_f(x,t)\, \mathrm{d}x + \\ &s\, C_{n_f,n_m}(t) \int M_{f,m}(x,y,t)\, n_f(x,t)\, n_m(y,t)\, f_f(x' \mid x,y,t)\, R_{f,m}(x,y,t)\, \mathrm{d}x\mathrm{d}y \end{aligned} \tag{2.1}$$

$$\begin{aligned} n_m(y',t+1) = &\int G_m(y' \mid y,t)\, S_m(y,t)\, n_m(y,t)\, \mathrm{d}y + \\ &(1-s)\, C_{n_f,n_m}(t) \int M_{f,m}(x,y,t)\, n_f(x,t)\, n_m(y,t)\, f_m(y' \mid x,y,t)\, R_{f,m}(x,y,t)\, \mathrm{d}x\mathrm{d}y \end{aligned}$$

(2.2)

The integral in the top part of equations 2.1 and 2.2 modelled the survival and growth of individuals already present in the population at t. The survival functions $S_f(x,t)$ and $S_m(y,t)$ described the probability that an individual of size x or y at time t survived to $t+1$ (Table S1). If an individual survived, it grew from x or y at t to x' or y' at $t+1$ with the probability described in $G_f(x' \mid x,t)$ and $G_m(y' \mid y,t)$, respectively (Table S1). Multiplying the probabilities with $n_f(x,t)$ or $n_m(y,t)$, the number of individuals of the respective sizes alive at t, and integrating over all sizes, therefore gave the number of individuals alive at $t+1$ and their new sizes.

The lower terms in equations 2.1 and 2.2 modelled the number, sex, and size of recruits to the population at $t+1$. Multiplying the mating function $M_{f,m}(x,y,t)$ for a female of size x and a male of size y at t (Table S1) with the number of females and males $n_f(x,t)$ or $n_m(y,t)$ present at t resulted in the number of matings that would have occurred if all females had mated with all males. The normalisation constant

$$C_{n_f,n_m}(t) = \frac{\int_{x_{min}}^{\infty} n_f(x,t)\,dx}{\int_0^{\infty} M_{f,m}(x,y)\,n_f(x,t)\,n_m(y,t)\,\mathrm{d}x\mathrm{d}y} \tag{2.3}$$

then scaled the number of matings to one mating per female above a size threshold for reproduction x_{min}. This constant was the number of females in the population whose heart girth was greater than the threshold, divided by the number of all theoretically possible matings. The number of one-year old recruits to the population at $t+1$ that were produced by a mating between a female of size x and a male of size y depended only on the size of the female, and was defined by $R_{f,m}(x,t)$ (Table S1, full notation of $R_{f,m}(x,y,t)$ kept in equations 2.1 and 2.2 for the sake of completeness). The sizes of offspring at recruitment were determined by the probability density distribution of offspring size at $t+1$, given parent size at t, and denoted by $f_f(x' \mid x,y,t)$ and $f_m(y' \mid x,y,t)$ (Table S1). Integrating over all parent

sizes resulted in the number and sizes of recruits. Multiplying the number of recruits with the proportion of female recruits s, or male recruits $(1-s)$, yielded the number of female and male recruits, respectively. Lionesses reproduce every two years on average (Packer et al. 1988b), but our mating function $M_{f,m}(x, y, t)$ in combination with the normalisation constant assumed a female annual mating probability of one. We therefore incorporated this aspect of lion life history into the fertility function $R_{f,m}(x, y, t)$. This quantity was a compound of the annual probability that a lioness of size x reproduces, $p_f(x, t)$, and the litter size, $m_{f,m}(x, t)$.

Parameterising the IPM

We identified functions required for the parameterisation of the two-sex IPM using individual heart girth, survival, reproduction, and offspring heart girth data (Table S1). We defined death as a permanent disappearance within one year after heart girth measurement. This practice overestimates mortality for males because some disappearances are likely to represent emigrations and not deaths. For want of alternatives to this known issue (Schaub and Royle 2013), we accepted the overestimation, but assumed that this effect is at least partially outweighed by immigrating males dying in the study area. We considered females to have reproduced if either cubs or signs of lactation were observed within one year of heart girth measurement. To determine litter size, we distributed offspring of a communal litter that survived to one year of age evenly among communally nursing mothers, and we assigned any odd ones randomly across the potential mothers.

We fitted generalised linear mixed effects models (GLMMs) with random intercepts and binomial error structures to annual survival and female reproduction, and a GLMM with random intercepts and a Poisson error structure to litter size at one year of offspring age (Table S1). For all GLMMs, we fitted pride identity at the time of measurement, and measurement year, as random effects to account for variation in territory quality, repeated measurements within prides, and temporal variation in

demographic rates (Mosser et al. 2009; Packer 2005). We did not include individual identity as a random effect due to the low number of repeated measurements (52 out of 291 for females and 32 out of 203 for males). We also considered the mean time lag between measurements of 1370 days ($\pm$130, 1 *s.e.*, $n = 52$) for females and 686 days ($\pm$96, 1 *s.e.*, $n = 32$) for males to be sufficient for statistical independence. Heart girth was fitted as a fixed effect in all models. For the logistic GLMMs fitted to annual survival and female reproduction, we excluded individuals younger than 2.5 years from the analyses due to low sample sizes for small heart girth sizes. We fitted models for males and females separately.

The resulting survival models for both sexes used to parameterise the IPM, $S_f(x,t)$ and $S_m(y,t)$, were logistic (Table S1). For small females, our survival estimates were unrealistically low. We therefore set survival of females below a size-threshold of 95.5 cm to that of males of the same size. We used this threshold to guarantee a smooth survival function. The annual number of male and female offspring born to a female of size x, $R_{f,m}(x,t)$, was the product of a logistic function describing female annual probability of reproduction, $p(x,t)$, and an exponential function describing number of offspring, $m(x,t)$ (Table S1).

Due to insufficient repeated heart girth measurements within individuals to identify size relationships between t and $t+1$, we estimated growth rates by using age as a surrogate for time as follows. We first fitted nonlinear functions of age to heart girth under Gaussian error structures. We then fitted a linear model of fitted heart girth at age x to fitted heart girth at age $x+1$. We identified growth variances by fitting intercept-only linear models of fitted heart girth to the absolute residuals of the age-size models. We constructed the growth kernels, $G_f(x' \mid x,t)$ and $G_m(y' \mid y,t)$, by using a normal distribution with the mean heart girth at $t+1$ and the variance in growth estimated from the data (Table S1). We scaled the growth kernels so that the transition rates out of a size class summed to one ($\int G(x' \mid x,t)\mathrm{d}x' = 1$ for all (x,t)).

We estimated offspring heart girth at one year of age in two steps. First, we calculated the proportion of the observed heart girth in fitted heart girth for the age at measurement to obtain a crude measure of the difference between the individual's growth trajectory and the mean growth trajectory. We then multiplied this proportion with the fitted heart girth for age $x = 1$, and used this result as offspring heart girth at one year of age. For identifying male and female offspring heart girth distributions as functions of mother heart girth, we fitted linear models of mother heart girth to female and male offspring heart girth at age $x = 1$, assuming Gaussian errors (Table S1). We identified variance in offspring size by regressing the absolute residuals around the mean offspring size function against offspring size. We lacked sufficient data to model the male and female offspring heart girth distributions as functions of father size and therefore used the same relationships as for mothers. We assumed that both parents contributed equally to offspring sizes (Table S1). We further assumed female and male offspring sizes, $f_f(x' \mid x, y, t)$ and $f_m(y' \mid x, y, t)$, to be distributed normally around the mean with the variance estimated from the data (Table S1). As with the growth kernels, we scaled the distributions of offspring sizes so that the transition rates from a single mating pair size-class summed to one, $\int f_f(x' \mid x, y, t)\mathrm{d}x' = 1$ and $\int f_m(y' \mid x, y, t)\mathrm{d}y' = 1$ for all (x, y, t).

Because we were not able to determine mating probabilities from the data, we instead modelled male mating probability as an exponentially increasing function of male heart girth, simulating size-based male reproductive skew (Table S1). We assumed that mating probabilities between females of size x and males of size y, $M_{f,m}(x, y, t)$, depended on the male size so that larger males had higher mating probabilities. We modelled this male size advantage using the function proposed by Schindler et al. (2013). We set minimum size thresholds for reproduction to the observed size of the smallest 2.5 year old female (87 cm) and male (100 cm), respectively. A normalisation, $\int M_{f,m}(x, y, t)\mathrm{d}y = 1$ for all (y, t), ensured that female mating probabilities in each size class summed to one. Finally, we calculated

the female proportion of one-year old recruits (0.45) as the proportion of females among all sexed individuals in the long-term data set that were born in the study area and survived to at least 1 year of age ($n_f = 1368$, $n_m = 1680$, $n_{unsexed} = 22$).

Numerical implementation, perturbation analyses, and empirical estimates

We approximated our IPM as a high dimensional matrix with 150 heart girth classes. Changing the number of classes to 100 or 200 had no influence on the first two decimal places of calculated quantities. The minimum and maximum heart girth were set to 0 and 160 cm respectively, while observed heart girth ranged from 48 to 148 cm. We iterated the model until heart girth distributions of both sexes were stable. At this equilibrium, we calculated the following quantities from the model: asymptotic population growth rate (λ), proportion of the population that was female, mean heart girths of both sexes, variances in heart girths of both sexes, mean female lifetime reproductive success (LRS, mean female lifetime number of female recruits), and mean female generation time as described in Coulson et al. (2010). For the perturbation analyses, we independently perturbed every parameter of the IPM by 1 % and constructed new IPMs and matrix approximations. The model parameters were the intercepts and slopes of each of the statistical functions, the proportion of females at recruitment, and the parameter ρ of the mating function that determined the degree of the male size-based reproductive skew. We conducted upwards perturbations, which meant that positive parameters became more positive and negative ones became less negative. Due to the substitution of survival probabilities of females smaller than 95.5 cm heart girth with that of same-sized males, perturbation of female survival only pertained to females larger than 95.5 cm. From the perturbed models, we calculated the proportional changes in the quantities described above.

We compared equilibrium model predictions with values directly estimated

from the demographic and heart girth data. We estimated the population growth rate from the heart girth data as the mean period survival probability plus the mean period recruitment rate within a year of heart girth measurements, assuming that 45 % of unsexed recruits were female. We calculated the female proportion of the population from the demographic data as the female proportion of the population aged one year or older and alive at end of study. We further calculated mean and variances in heart girths of both sexes as the mean and variances of all heart girth measurements by sex. We estimated female LRS from the demographic data as the mean lifetime number of female recruits that survived to their first birthday and that were born to a female recorded in the heart girth data, again assuming 45 % of unsexed recruits were female. Finally, we calculated female generation time from the observed female LRS and population growth rate. All analyses were conducted using the statistical programming language R (R Core Team 2012) (for code and model parameters see Supplementary Information).

Results

Relationships for both sexes between heart girth and survival, heart girth at t and $t+1$, and heart girth and age are displayed in Figure 2.1. Relationships between heart girth and number of matings for both sexes, between female heart girth and female probability of reproduction and litter size, and between mean mating pair heart girth and both male and female offspring heart girth are shown in Figure 2.2. The underlying functions and parameter values are given in Table S1. In the following results, we focus on the stable heart girth distributions and population statistics predicted from the model at equilibrium. We compare our model predictions to observed values, describe the impact of perturbations on model predictions, and convey the effects of intersexual frequency dependence on population dynamics.

All population statistics directly estimated from data were higher than our

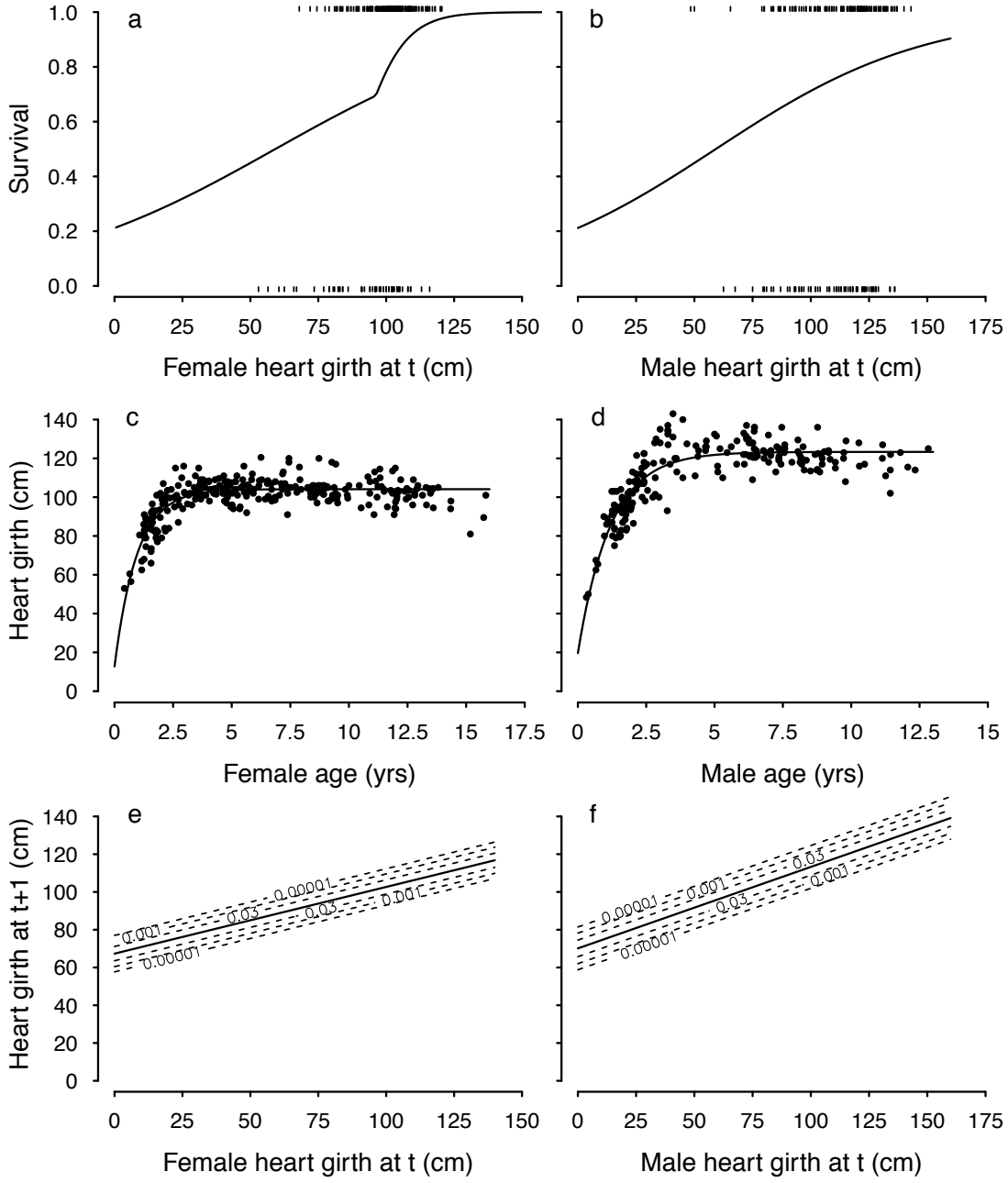

Figure 2.1. Summary of survival and growth functions used to parameterise the integral projection model. Relationships shown are between heart girth and survival, between age and heart girth, and between heart girth at t and $t+1$ for females (a, c, e; left panels) and males (b, d, f; right panels). Ticks (a, b) and dots (c, d) represent raw data; lines represent predictions from regressions and dashed contours (e, f) distributions around the mean (see Table S1 for details of statistical models).

model predictions except for variances in sizes of both sexes, for which the predictions were higher than the empirical estimates (see Table **??**). Although our model predictions of the population growth rate and mean sizes still corresponded reasonably well with observations, the predictions of the female proportion of the population, LRS, and generation time were lower than observations by a factor of between a fifth to a third. That the predictions were lower than the observations for the population growth rate, the female proportion of the population, and female LRS and generation time is consistent with female reproduction, growth and/or survival being predicted by the model to be lower than the true values. While this is plausible, it is also noteworthy that our empirical estimates are based on a mixture of period and cohort data from several years. They are therefore affected by environmental variation, which our density-independent and deterministic model did not capture. That the mean sizes of both sexes were predicted to be lower than observations, and their variances to be higher, points towards an underrepresentation of small individuals in the heart girth data. In fact, the comparison between the observed and predicted stable heart girth distributions (see Figure 2.3) reveals an obvious lack of measurements for smaller individuals, which explains the lower empirical than modelled variance. Although the observed distributions contained only a few small individuals, the predicted distributions of both sexes had three modes, one each at smaller, medium, and bigger sizes corresponding to 1-year-olds, 2-year-olds, and adults in the discrete time steps of our model. In general, the predicted heart girth distributions of both sexes resembled each other with the male one being shifted to larger sizes compared to the female one (Figure 2.3).

We used perturbation analyses to understand which demographic or trait-transition process most strongly affected model predictions. Figure 2.4 shows the effects of perturbations on the population growth rate and the female proportion of the population, and Figure 2.5 those on means and variances of sizes, female LRS, and female generation time. We first describe the effects of changing model

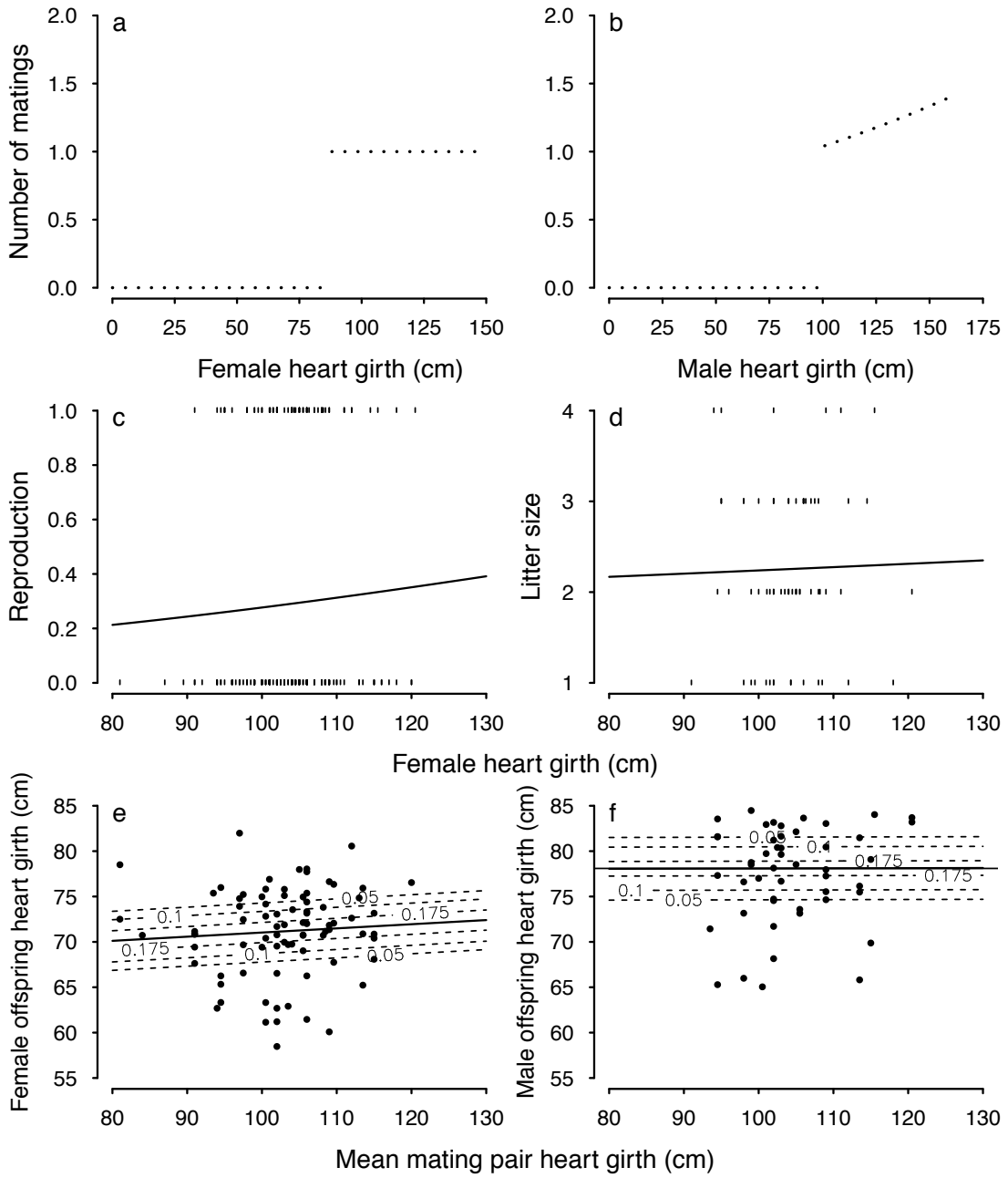

Figure 2.2. Summary of mating, reproduction, and inheritance functions used to parameterise the integral projection model. Relationships shown are between female heart girth and female number of matings (a), male heart girth and male number of matings (b), female heart girth and female probability of reproduction (c) and female litter size (d), and between mean mating pair heart girth and female and male inherited offspring sizes (e, f). Ticks (c, d) and dots (e, f) represent raw data; dotted lines (a, b) represent values at population equilibrium, solid lines (c, d, e, f) indicate predictions from regressions and dashed contours (e, f) distributions around the mean (see Table S1 for details on statistical and demographic models).

functions involved in modelling the female heart girth distribution (Figures 2.4 and 2.5, left side of figure panels). The population growth rate, female proportion of the population (Figure 2.4 a, b), female LRS and female generation time (Figure 2.5 e, f) were, unsurprisingly, most sensitive to changes in the survival and growth of females. These changes resulted in females either growing faster or transitioning with higher probability into larger and better surviving size classes, in which they also had a slightly higher reproductive output. Increasing the reproductive output itself, via increasing the annual probability of reproduction and litter size, impacted population parameters differently compared to increasing growth and survival. A higher reproductive output increased the population growth rate and female LRS less than higher growth and survival (Figures 2.4 a, 2.5 e), and it decreased both the female generation time and the female proportion of the population (Figures 2.5 f, 2.4 b). The latter effect occurred due to a male-biased sex ratio at recruitment. Accordingly, when sex ratio at recruitment was raised so that more females were born, the female proportion of the population increased (Figure 2.4 b). Finally, mean and variance of female size were most strongly affected by changes to the growth rates of females, and to a lesser degree, to the inherited female size at recruitment (Figure 2.5 a, b). An intersexual effect, where changes to the female size distribution affected the male size distribution, occurred in that increasing female growth and survival decreased mean male size, since better-surviving, larger females produced more offspring with a slightly male-biased sex ratio, thereby raising the proportion of small males in the population (Figure 2.5 b).

We now turn to the impact of perturbing functions that modelled the male size distribution (i.e., changing the male size distributions) on mean and variance in sizes, the population growth rate, the female proportion of the population, and the female life history descriptors (Figures 2.4, 2.5, right side of figure panels). As expected, mean male size increased with increasing growth and survival of males, and with greater sizes of male offspring at recruitment (Figure 2.5 b). Variance in

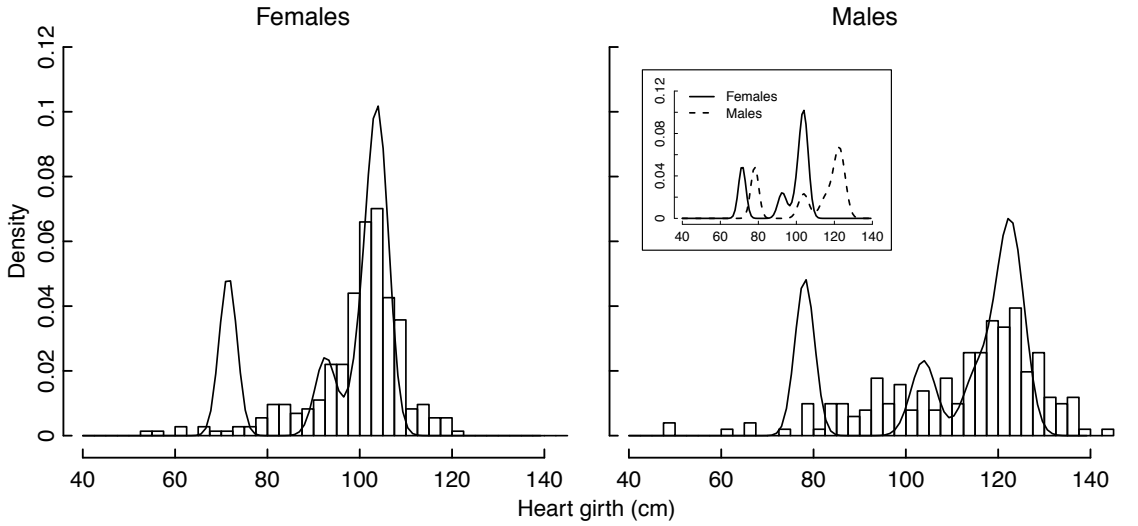

Figure 2.3. Predicted (solid line) and observed (boxes) heart girth distributions of females (left panel) and males (right panel). The inserted plot (right panel) contrasts the predicted distributions of both sexes.

male size also increased with higher growth rates but decreased with larger male offspring sizes (Figure 2.5 d). Intersexual effects, where changes to the male size distribution affected the female size distributions, female LRS and generation time, and the population growth rate, were only in the range of the fourth to fifth decimal point of model predictions, and therefore negligibly small when compared to the effects of altering female model functions on the same model predictions (Figures 2.5 a, c, e, f, 2.4 a). Among those intersexual effects, changes to the growth rates of males had the largest impacts. This occurred because the intersexual frequency dependence in our model operated via the link between male size and offspring size, with male size most strongly affected by male growth rates. Furthermore, the female proportion of the population was lowered by higher male survival, growth, and offspring size at the same scale when compared to the effects of female model functions. In summary, our results showed that the female size distributions largely determined the dynamics of both the male and female size distributions.

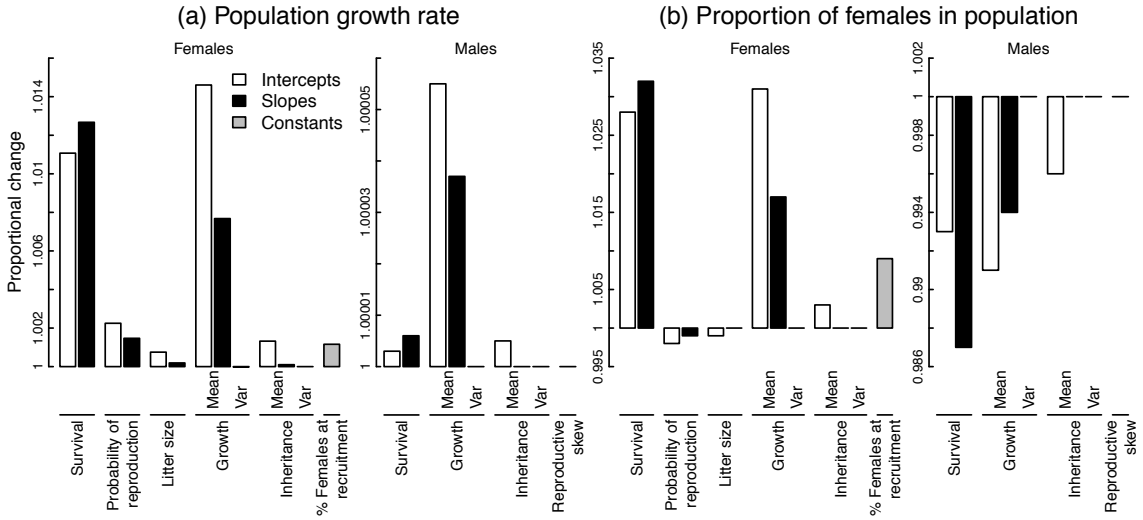

Figure 2.4. Proportional changes in population growth rate (a) and female proportion of the population (b) after perturbations. We applied a 1 % upward perturbation to intercepts (white bars), slopes (black bars), and constants (grey bars) of model functions. Functions used to model the female distribution are the female heart girth-female survival, probability of reproduction, litter size, and growth functions, the mean mating pair heart girth-inherited female heart girth function, and the % female recruits (left side of panel a and b); those that model the male distribution are the male heart girth-male survival and growth functions, the mean mating pair heart girth-inherited male offspring heart girth function, and the male reproductive skew function (right side of panel a and b).

Discussion

In this paper, we used a size- and sex-structured population model to test whether, via the mechanism of paternal size inheritance, males matter for the population dynamics of a large carnivore with sexual dimorphism in size and life history. Surprisingly, we could not find any noteworthy effects of the male size distribution on the dynamics of the population. Our study therefore suggests that paternal size inheritance as a mechanism for intersexual frequency dependence does not challenge the prevalence of female-only models in population ecology and life history biology.

In the following discussion, we first highlight the importance of intersexual frequency dependence in the context of other trait frequency dependences and population management. We then emphasise the utility of two-sex IPMs for studying complex eco-evolutionary responses of populations to anthropogenic influences in

species with sexual dimorphism in population structure and intersexual frequency dependence. We move on to discuss how, for example, two-sex IPMs for lions can model nonlinear population responses to trophy hunting. We close with a discussion of the conclusiveness of our results given the model's constraints.

A common motivation behind studies in population ecology is to understand how to manage populations. Many species undergo unwanted population declines due to anthropogenic influences that include human-induced climate change, land transformation, and harvesting (Parmesan 2006; Worm et al. 2006). Other species, such as invasive species, agricultural pests, and disease vectors, may show undesirable population growth (Patz et al. 2005; Pimentel et al. 2005). Population management measures aim to regulate the sizes of populations for conservation or damage control purposes by changing the survival and reproduction of individuals. The success of these measures relies on an understanding of how changes to demographic rates will affect the population. However, predicting population responses can be complicated by two important factors. First, individuals within a population differ in survival and remaining lifetime reproduction with respect to traits like sex, life history stage, disease load, or social status. As a result, management measures that target specific trait classes are likely to have different effects than randomly applied measures (Crouse et al. 1987). Second, changes in survival and reproduction can have consequences for other trait transition rates (e.g., the probability of moving from lower to higher social status) if ecological and evolutionary population processes are correlated (i.e., trait frequency dependences). For example, altering adult survival may affect disease infection rates (Donnelly et al. 2005), male survival may affect female survival (Le Galliard et al. 2005), or size-specific survival may affect growth (Coltman et al. 2003). Changes to trait frequency distributions through anthropogenic influences, including population management measures, may therefore affect the development of the whole population in complex, nonlinear, and potentially undesirable ways (Donnelly et al. 2005).

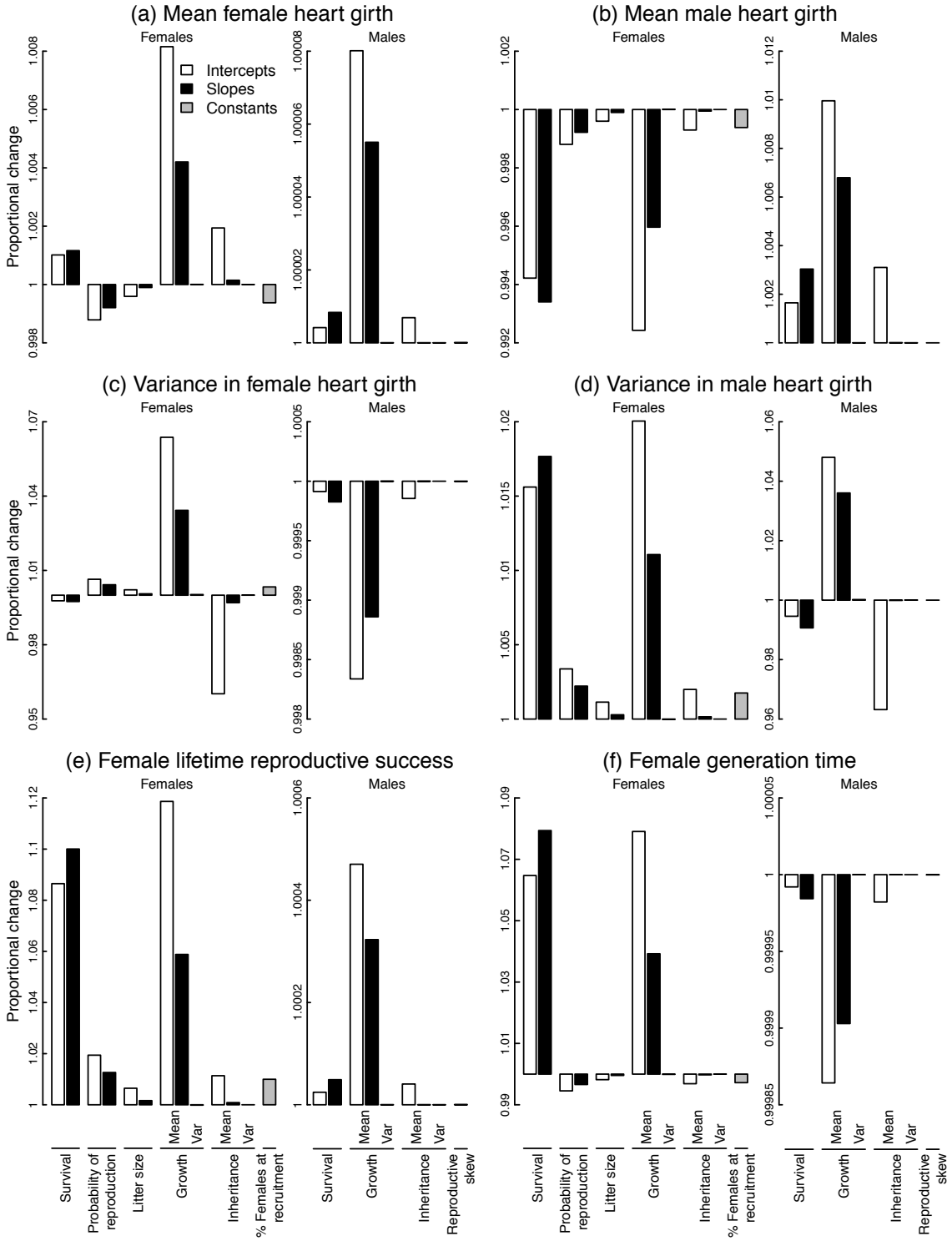

Figure 2.5. Proportional changes in mean female heart girth (a), mean male heart girth (b), variance in female heart girth (c), variance in male heart girth (d), female lifetime reproductive success (e) and female generation time (f) after perturbations. We applied a 1 % upward perturbation to intercepts (white bars), slopes (black bars), and constants (grey bars) of model functions. Functions used to model the female distribution are the female heart girth-female survival, probability of reproduction, litter size, and growth functions, the mean mating pair heart girth-inherited female heart girth function, and the % female recruits (left side of panels a-f); those that model the male distribution are the male heart girth-male survival and growth functions, the mean mating pair heart girth-inherited male offspring heart girth function, and the male reproductive skew function (right side of panels a-f).

Among the array of potential trait frequency dependencies, intersexual frequency dependence is relatively easy to investigate because, unlike traits like disease status or age, sex is time-invariant and often easily observable. Furthermore, two-sex population theory predicts, and empirical evidence supports, the occurrence of intersexual frequency dependence (Das Gupta 1972; Le Galliard et al. 2005). With the development of two-sex IPMs (Schindler et al. 2013), we now also have a tool that can model intersexual frequency dependence in age-, stage-, and trait-structured populations. Two-sex IPMs can be used to test whether the structure of the male population affects population development via mechanisms that can be included in the model as covariances between different demographic and trait transition rates. If two-sex IPMs can be parameterised as a multiple trait-structured model (e.g., size and disease load for both sexes), other trait frequency dependences can also be included in the model (e.g., between adult survival and disease load). Perturbation analyses can then reveal the effects of anthropogenic influences that are non-random with respect to different age, stage, or trait classes, under circumstances when intersexual or other trait frequency dependences affect the development of a population that is sexually dimorphic in population structure.

Lions, for example, have sexually dimorphic population structures and are often hunted for trophies (Lindsey et al. 2012). Trophy hunting tends to be age- and sex-selective with hunters preferring or being restricted to adult males (Whitman et al. 2007; Caro et al. 2009; Becker et al. 2012). Moreover, hunters may prefer larger trophies, although this awaits empirical evidence. Selective removal of adult males can decrease population size by more than just the trophy head count because it increases rates of pride takeover and infanticide (Whitman et al. 2004). Some workers have therefore suggested to target older post-reproductive males to minimise the negative impact of trophy hunting (Whitman et al. 2004, 2007; Packer et al. 2009). In their recommendations, they focused on population size (Whitman et al. 2004; Packer et al. 2009; Becker et al. 2012), but age-, sex-, and potentially size-selective

harvesting are likely to generate complex eco-evolutionary dynamics in lions, as they do in other game species (e.g., bighorn sheep, Coltman et al. 2002). These complex responses to selective hunting could be modelled using a well-parameterised two-sex IPM that includes a positive covariance between male and cub survival to simulate the effects of intersexual frequency dependence via the mechanism of infanticide. Unfortunately, data limitations precluded the inclusion of such a relationship in our model.

How conclusive is our negative result for intersexual frequency dependence via paternal inheritance? Various model constraints, mainly the approximation of size by heart girth, the lack of age in the model, and parameterisation issues, may limit the inferences that can be drawn. First, we had to approximate body size by heart girth because heart girth data can be more easily collected than body mass. While this is not optimal, the procedure is justified because heart girth is known to be well-correlated with body mass corrected for stomach content (Bertram 1975). Second, despite prior evidence for age-related declines in lion survival and reproduction (Packer et al. 1988a, 1998), data limitation unfortunately precluded the inclusion of age in our model. A more complicated model will have to await future data collection. Third, the parameterisation of the model is partly empirical and partly theoretical, which explains the mismatch between observed and predicted population statistics. While this alone is not too worrisome if we regard the model as a theoretical exploration rather than a working model of a lion population, it does raise the question of whether a misspecification of the population processes involved in determining the role of paternal inheritance may have resulted in the negative result.

A misspecification of the population processes could have occurred due to the underrepresentation of small individuals in the heart girth data (Figure 2.3). A lack of small individuals in the data most likely caused the estimated survival probabilities for small females to be very low by chance. Since the model using these low

survival probabilities predicted rapid extinction of the population, we decided to substitute the survival probabilities of small females with those estimated for small males. We abstained from making further correction to estimated model parameters because the rough comparison between population statistics estimated from the data and our model predictions indicated that our model provided a good enough abstraction of the system to allow inference. Furthermore, a lack of data also prevented the estimation of male size-based reproductive skew. We therefore instead modelled it using an exponentially increasing function (for details see Table S1). Although we argue that, in sexually dimorphic species, reproductive success is positively associated with the size of a sexually selected trait like body mass (Andersson 1994), we have no data to support or refute such a relationship in lions. Similarly, we could not accurately estimate the relationship between the father's size and offspring size and instead used the relationship estimated for the mother's size by substituting mother's size with mean mating pair size. The precise link between male size and offspring size therefore remains to be established. Moreover, we assumed offspring size to be inherited with equal contributions from both parents, which may not necessarily hold (Iyengar and Eisner 1999), and could be altered, and tested, in future models.

Despite these limitations, we are confident that the negative result for intersexual frequency dependence via paternal inheritance would hold with improved model parameterisation for two reasons. First, the impacts of changing the male size structure on our model's predictions were vanishingly small. Second, we scrutinised the credibility of our results by conducting some additional perturbations where we overinflated key parameters of functions involved in mediating the strength of the influence of paternal size inheritance (i.e., large males were assumed to have a larger share of reproduction and to sire larger offspring). This was accomplished by increasing the ρ parameter of the mating function, and the slope of the inheritance function between mean parent and female offspring size, by 250%. Since the

slope of the inheritance function between mean parent and female offspring size was much higher than that for mean parent and male offspring size, we furthermore set the slope for male offspring to be the same as this altered value for the female offspring. We then perturbed the slope of the male growth and survival function by 10 %, compared to just 1 % perturbations in the regular analysis, and even then the population growth rate reacted to the perturbations only in the third decimal point, indicating a negligible effect. Therefore, although counterintuitive, we find no evidence for a noteworthy role of paternal size inheritance in population dynamics.

In conclusion, the existence of trait frequency dependences makes predicting the responses of populations to population management measures difficult. We therefore expect that the study of these dependencies will gain traction in population ecology. Although paternal inheritance of body size as a mechanism for intersexual frequency dependence in lions has failed to challenge population ecology's single-sex paradigm, we expect two-sex IPMs to become a crucial tool for revealing other intersexual frequency dependences. We also expect to see them gain popularity as a tool for studying the impacts of anthropogenic influences on species with sexually dimorphic population structures and intersexual frequency dependence, and potentially for modelling the effects of other trait frequencies dependences on the population dynamics of such species.

Acknowledgements

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Supporting Information

Table S1 Demographic models used to parameterise the integral projection model.

Data S1 IPM parameters. Download from github.com/bartholdja/DPhil_supplements.

Code S1 R code for the two-sex IPM. Download from github.com/bartholdja/DPhil_supplements.

Table S1. Fitted demographic models for parametrisation of the IPM

Process	**Fitted GLMM/LM or mathematical model**	**Demographic model used in IPM**
Survival[†,‡]	$Y_{i,j} \sim Bin(1, \pi_{i,j,k})$, $a_j \sim N(0, \sigma_a^2)$, $b_k \sim N(0, \sigma_b^2)$	
Females	$logit(\pi_{i,j,k})_f = -10.70_{(3.51)} + 0.12_{(0.03)} x_{i,j,k} + a_j + b_k$	$S_f(x) = \begin{cases} \frac{1}{1+e^{-(-1.32+0.02x)}} & \text{if } x < 95.5\,\text{cm} \\ \frac{1}{1+e^{-(-10.70+0.12x)}} & \text{otherwise} \end{cases}$
Males	$logit(\pi_{i,j,k})_m = -1.32_{(3.24)} + 0.02_{(0.03)} y_{i,j,k} + a_j + b_k$	$S_m(x) = \frac{1}{1+e^{-(-1.32+0.02y)}}$
Growth	$Y_i \sim N(\mu_g, \sigma_g)$	
Females	$\mu_{g,f} = 67.34_{(0.00)} + 0.35_{(0.00)} x$, $\sigma_{g,f} = 4.85_{(0.26)}$	$G_f(x'\|x,t) = \frac{1}{\sqrt{2\pi}\times 4.28} e^{-\frac{(x'-[67.34+0.35x])^2}{2\times 4.85^2}}$
Males	$\mu_{g,m} = 70.11_{(0.00)} + 0.43_{(0.00)} y$, $\sigma_{g,m} = 6.71_{(0.38)}$	$G_m(y'\|y,t) = \frac{1}{\sqrt{2\pi}\times 6.71} e^{-\frac{(y'-[70.11+0.43y])^2}{2\times 6.71^2}}$
Mating function*	$M_{f,m}(x,y) =$ $\begin{cases} \frac{1}{2} e^{\frac{y}{50}\times\rho} & \text{if } x \geq x_{min} \text{ and } y \geq y_{min} \\ 0 & \text{otherwise} \end{cases}$	$M_{f,m}(x,y) =$ $\begin{cases} \frac{1}{2} e^{\frac{y}{50}\times 0.1} & \text{if } x \geq 87\,\text{cm and } y \geq 100\,\text{cm} \\ 0 & \text{otherwise} \end{cases}$
Fertility[†]		
Probability of reproduction	$Y_{i,j} \sim Bin(1, \phi_{i,j,k}), a_j \sim N(0, \sigma_a^2), b_k \sim N(0, \sigma_b^2)$ $logit(\phi_{i,j,k})_f = -2.69_{(2.64)} + 0.02_{(0.03)} x_{i,j,k} + a_j + b_k$,	$R_{f,m}(x,t) = p(x,t)m(x,t) = \frac{e^{(0.65+0.002x)}}{1+e^{-(-2.69+0.02x)}}$
Number of offspring	$Y_{i,j} \sim Poisson(\mu_{i,j,k}), a_j \sim N(0, \sigma_a^2), b_k \sim N(0, \sigma_b^2)$ $log(\eta_{i,j,k})_f = 0.65_{(1.52)} + 0.002_{(0.01)} x_{i,j,k} + a_j + b_k$,	
Offspring heart girth	$Y_i \sim N(\mu_{in}, \sigma_{in})$	
Female offspring	$\mu_{in,f} = 66.45_{(7.33)} + 0.05_{(0.07)} x$, $\sigma_{in,f} = 3.72_{(0.34)}$	$f_f(x'\|x,y,t) = \frac{1}{\sqrt{2\pi}\times 3.72} e^{-\frac{(x'-[66.45+0.05\frac{(x+y)}{2}])^2}{2\times 3.72^2}}$
Male offspring	$\mu_{in,m} = 78.00_{(9.93)} + 0.002_{(0.10)} x$, $\sigma_{in,m} = 4.49_{(0.47)}$	$f_m(y'\|x,y,t) = \frac{1}{\sqrt{2\pi}\times 4.49} e^{-\frac{(y'-[78.00+0.002\frac{(x+y)}{2}])^2}{2\times 4.49^2}}$

[†] a_j and b_k represent the random effects of measurement year and pride. [‡]Subscripts in brackets represent 1 s.e. of parameters. *Mating function could not be estimated from data. Before entering the model, the mating function was normalised so that $\int M_{f,m}(x,y,t)\mathrm{d}y = 1$ for all (y,t).

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CHAPTER 3

Dead or gone? Bayesian inference on survival for the dispersing sex

Running head

Bayesian inference on survival for the dispersing sex

Title

Dead or gone? Bayesian inference on survival for the dispersing sex

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Abstract

Estimates of age-specific survival are regularly used in ecology, evolution, and conservation research. Yet estimating survival for males from re-sighting records of marked individuals is commonly challenging for species with male natal dispersal. When males disappear from free-living study populations around the age of maturity, their fate remains uncertain; they may have died or settled outside of the study area. As an alternative to intensifying data collection on males, we present a survival model that imputes dispersal state (i.e., died or left) for uncertain male records as a latent state jointly with the coefficients of a parametric mortality model in a Bayesian hierarchical framework. The model further imputes sex as a latent state for those individuals that died before sex determination occured. To validate our model we conducted a simulation study and found that our model recovered the mortality parameters from simulated data of varying sample sizes, proportions of uncertain male records, and proportions of unsexed individuals. Subsequently, we applied our model to a long-term data set for African lions and compared the performance of the default model with that of other models that took advantage of an expert's indication on the likely dispersal state of each uncertain male record. We thus learned that our model may have the tendency to overestimate mortality in the juvenile ages, but nevertheless provides an acceptable solution to the challenge of estimating male mortality in species with data-deficiency for males due to natal dispersal.

Key words

age-specific mortality, Gompertz law of mortality, Siler model, dispersal, carnivores, African lion, sex differences in mortality, sex differences in life history, ageing, Serengeti

Introduction

Survival estimates of both sexes for wild animal populations are fundamental for testing hypotheses derived from ecological and evolutionary theory, and for predicting population size and structure for population management purposes. However, estimating survival of at least one of the sexes is commonly hindered by incomplete data on dispersing individuals. In many large vertebrate species, males leave their natal place or social group around the age of maturity. If dispersing males leave the areas monitored by field studies that collect re-sighting data on marked individuals, these migrating males impede the quality of gathered data. Following dispersing males using telemetry or GPS technology is cost and labour intensive, and therefore dispersing males are usually lost for data collection.

The possibility that missing males may have dispersed increases the uncertainty of the fate of all males that are no longer detected. Missing females are likely dead, even if their bodies are not found, since they do not disperse. Missing males, however, that were old enough for dispersal may have died or dispersed. This uncertainty in the male records prevents the estimation of male survival using existing methods.

Models to infer survival using capture-mark-recapture/re-sighting (CMRR) data derived from the Cormack-Jolly-Seber framework (CJS; after Cormack 1964; Jolly 1965; Seber 1965) can accommodate both uncensored and right-censored records (i.e., individuals known to be alive after the last observation). These approaches exploit the fact that each type of record contributes different information (White and Burnham 1999). Extensions to the initial models have been developed that accommodate species-specific life histories and data issues arising from the movement of the individuals in relation to the spatial and temporal distribution of the marking and re-sighting effort. Accordingly, these models incorporate for example incomplete and heterogeneous re-sighting probabilities, multiple states, and multiple locations

(e.g., Arnason 1973; Schwarz et al. 1993; Lebreton and Pradel 2002; Mackenzie et al. 2009; Cubaynes et al. 2010; Schaub and Royle 2013; Lagrange et al. 2014). Yet none of them can accommodate the uncertain male records that are typical for re-sighting data of species with male dispersal.

In order to address issues with missing records in CMRR data, Bayesian approaches have been developed that estimate survival probabilities and transition probabilities between states and locations while augmenting data (Dupuis 1995, 2002; King and Brooks 2002). Among these approaches, very flexible are those that estimate latent (unknown) states jointly with all other model parameters in a hierarchical framework using Markov Chain Monte Carlo (MCMC) algorithms (Clark et al. 2005; Colchero and Clark 2012; Colchero et al. 2012). Since latent states can be both finite sets of discrete states (e.g., locations or stages) or continuous variables (e.g., date of birth or death), this framework is suitable for developing a survival model that treats dispersal as a latent state, and can therefore accommodate uncertain male records.

Among mammals, males commonly form a data-deficient subpopulation. Male dispersal hinders inference on male survival, and obscured paternities hinder inference on male lifetime reproduction. The lack of data on males prevents studies that would use measures of male fitness to test evolutionary theory or would use male demographic rates to address the role of males in population dynamics. Yet, in many mammal species, males are under stronger sexual selection than females, which makes them particularly interesting to study from an evolutionary perspective (Andersson 1994). Furthermore, males have been found to influence the dynamics of populations through mechanisms such as sexual harassment and limiting female fecundity (Milner-Gulland et al. 2003; Le Galliard et al. 2005), and several more mechanisms are predicted by ecological theory (Mysterud et al. 2002; Rankin and Kokko 2007). As long as intensified data collection is out of reach, the development of statistical methods to infer male life history parameters from incomplete data is

a timely endeavour.

Here, we present a model that can estimate age-specific survival for both sexes in species with uncertain male records due to male dispersal. The model fits a parametric mortality model as a function of age and sex in a Bayesian hierarchical framework, treating potential dispersal of males with uncertain records as a latent state. Using simulated data, we first validated the model. We then applied the model to estimate age-specific survival of both sexes for Serengeti lions (*Panthera leo*) in Tanzania. Since this particular data set contains the expert opinion from the head of the study (C. Packer) on whether a missing male is likely to have dispersed or died, we used this information to gain further insights into the workings of our method.

Methods

We focus on species in which males disperse only once at around the age of maturity ('natal dispersal'). To isolate the effect of uncertainty in male records on survival estimates from other effects, we focus on data that meet the following assumptions. We assume that individuals are re-sighted with certainty if they are alive and in the study area and that individuals are only observed at one location. We further assume that survival probabilities in- and outside of the study area are equal, and that individuals born outside of the study area disperse into the study area with equal probabilities as individuals born in the study area disperse out of it. We also assume that ages of individuals whose birth was not observed (left-truncated records) can be estimated with sufficient certainty by a trained observer to allow us to not include time of birth as a latent state in the model and to model ages at death as a continuous variable. However, since the data available to us for the empirical application contained individuals that died before sexing was possible, we did construct the model to accommodate this type of record, treating the sex of

unsexed individuals as another latent state. Finally, we further make one assumption that we know is not met for data from wild animal populations: that survival probabilities only depend on age and sex and not on any other covariates. However, this assumption allows us to develop a model to estimate baseline mortality for pooled data, that can later on be easily extended to incorporate other covariates, data quality permitting

Life History Data

Data structure

The life history data used to estimate age- and sex-specific mortality included records for native-borns and immigrants. Native-borns were born in the study population, defined as all individually recognisable and constantly monitored individuals. Immigrants entered the study population some time after their birth either due to migration, or by being alive at the start of the study (Figure 3.1). The recorded types of departure from the population included death, censoring due to being alive at the end of the study, or uncertain fate (death or censoring through dispersal). Uncertain fates through dispersal were only caused by dispersals from the study population to an external population, and not by dispersals within the study population. Here, we refer to this out-migration from the study population when we use the term 'dispersal'.

Simulated data

To validate the performance of our model, we used known mortality parameters to simulate data of the described structure and checked whether our model accurately retrieved these parameters. To simulate the data, we first randomly assigned a sex for an initial number of individuals by drawing from a binomial distribution, assuming an equal probability of being born male or female. We then randomly

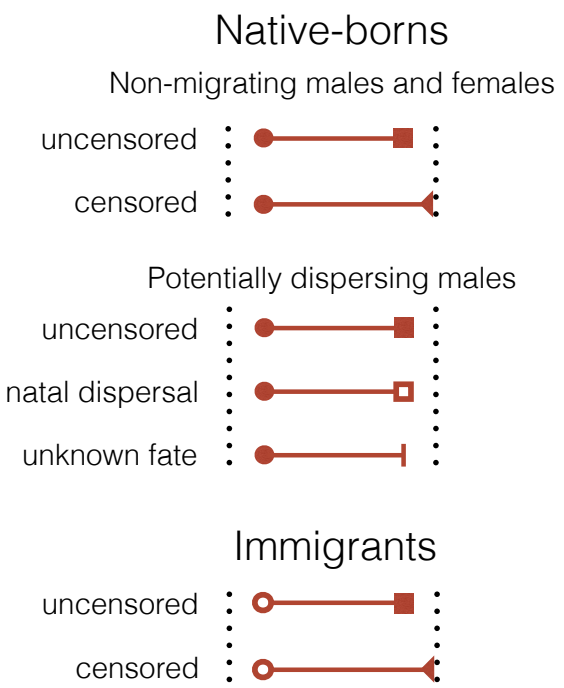

Figure 3.1. Example of types of records in the lion dataset. Circles represent times of entry (t_i^F), where the entry type for filled circles corresponds to known times of birth and open circles are entries after birth (i.e., immigration or birth before the study started). Squares are departure times (t_i^L) where filled squares are known times of death and open squares are dispersal. Filled triangles indicate individuals known to be alive at the end of the study and vertical bars indicate that the type of departure from the study population is uncertain (i.e., either death or dispersal).

drew ages at death (x_i) for each individual i by inverse sampling from a Siler CDF (see equations (3.2b) and (3.3)) with parameters $\boldsymbol{\theta}_f = \{-1.4, 0.65, 0.07, -3.8, 0.2\}$ for females and $\boldsymbol{\theta}_m = \{-1.2, 0.7, 0.16, -3.5, 0.23\}$ for males. The subscripts f and m denote females and males, respectively. We then randomly drew ages at dispersal for all males by inverse sampling from a gamma CDF with parameters $\boldsymbol{\gamma} = \{10, 3\}$ and adding the minimum age of dispersal $\alpha = 1.75$). We assigned every individual a last seen age x_i^L depending on its sex and dispersal status. For females and for those males whose ages at death were simulated to be younger than their ages at dispersal (i.e., they died before they could disperse), the last seen ages were the ages at death. For the other males, who were simulated to have died after dispersal, the last seen ages were set to be the ages at dispersal. Finally, to add immigrants to the data,

we simulated the same number of males being born in the external population. For these males, as before, we randomly drew ages at deaths and ages at dispersal, and if they were simulated to have dispersed before death, we added them to the data as immigrants with their ages at death recorded as last seen ages and their ages at dispersal recorded as first seen ages x_i^F.

We simulated data sets of two different initial numbers of native-borns (small sample size N = 500 and large sample size N = 2000). Within each sample size, we also produced further data sets where the sexes of all individuals were known, and data sets where we randomly assigned, with a probability of 0.3, the state of 'unknown sex' to all individuals that died at < 1 year of age. Finally, we simulated data that varied in the proportion of observed or 'known' deaths among individuals that were no longer re-sighted. We used three settings: 1, 5, and 10 % known deaths. In total, we thus simulated 12 data sets.

Serengeti population

The study population occupied a 2000 km^2 region of Serengeti National Park, Tanzania, that lies at the heart of the Serengeti-Mara ecosystem. The study site is characterised by seasonal rainfall and a southeast to northwest gradient in vegetation from short to tall grassland to open woodlands (Packer 2005; Mosser et al. 2009). We analysed demographic data collected between 1966 and 2013. Observations were opportunistic between 1966 and 1984, and most animals were sighted 1-3 times per month. Study prides have been monitored with radio telemetry since 1984, allowing each animal to be observed 2-6 times per month. All individuals are identified from natural markings (Packer et al. 1991), and birth dates of cubs born in the study area are deduced from lactation stains on the mothers. A large number of nomadic males enter the area, and a small proportion become resident in one or more of the resident prides. Our analyses exclude all nomadic males that never became residents in the study area (N = 548). Individuals with unknown

dates of birth were assigned an estimated age by a trained observer, using age indicators (e.g., relative body size, nose coloration, and eruption and wear of teeth) (Smuts et al. 1978; Whitman et al. 2004). The data set contained a large number of individuals of unknown sex. Since the vast majority of these unsexed individuals died within the first weeks after birth, we excluded all individuals with last seen ages younger than 0.25 years of age. The final data set contained observations on 1341 females, 1263 native-born males, 316 immigrants, and 269 unsexed native-born individuals. The proportion of females among all native-born individuals (excluding immigrants), assuming a sex ratio of 1 to 1 among individuals that died before their sex could be determined, was 0.51.

Mortality analysis

Model variables and functions

We used a Bayesian approach that allowed us to estimate not only parameters for mortality and dispersal but also the different latent states such as dispersal state and sex. We defined the random variables X for ages at death and Y for ages at natal dispersal. We also defined a dispersal state D which assigned 1 if an individual i, born at b_i and last detected at t_i^L, dispersed in its last detection age, $x_i^L = t_i^L - b_i$, and 0 if otherwise. We treated D as a latent variable for all individuals with an uncertain fate. We also defined a variable S with the assignment 1 if an individual was female and 0 if male.

We used a parametric model to infer age-specific mortality. This is expressed for continuous age x as the mortality rate, or hazard of death,

$$\mu(x|\boldsymbol{\theta}) = \lim_{\Delta x \to 0} \frac{\Pr(x \leq X < x + \Delta x \mid x \leq X, \boldsymbol{\theta})}{\Delta x}, \tag{3.1}$$

where $\boldsymbol{\theta}$ is a vector of mortality parameters to be estimated. The estimated mortality rate can be used to calculate the probability to survive from birth to age x, or

survivor function,

$$S(x|\boldsymbol{\theta}) = Pr(X \geq x) = \exp\left[-\int_0^x \mu(z|\boldsymbol{\theta})\mathrm{d}z\right], \tag{3.2a}$$

the probability that death occurs before age x, or the cumulative density function (CDF),

$$F(x|\boldsymbol{\theta}) = Pr(X < x) = 1 - S(x|\boldsymbol{\theta}), \tag{3.2b}$$

and the probability density function (PDF) for age at death

$$f(x|\boldsymbol{\theta}) = \frac{d}{dx}F(x|\boldsymbol{\theta}) = S(x|\boldsymbol{\theta})\mu(x|\boldsymbol{\theta}). \tag{3.2c}$$

To capture the bathtub-shaped mortality rate typical of large mammals, we used the Siler model (Siler 1979) in the form

$$\mu(x|\boldsymbol{\theta}) = e^{a_0 - a_1 x} + c + e^{b_0 + b_1 x}, \tag{3.3}$$

where $\boldsymbol{\theta} = \{a_0, a_1, c, b_0, b_1\}$, with $a_0, b_0 \in \mathbb{R}$ and $a_1, c, b_1 > 0$. The Siler model is a competing risk model constituted by three additive mortality hazards. The parameters capture different aspects of the shape of the age trajectory with a_0 being the initial level of mortality rate and a_1 governing the decrease in mortality over infant and juvenile ages. The c parameter scales mortality rates up or down and is usually interpreted as reflecting age-independent causes of mortality. This parameter is also dominant in capturing mortality in early adult ages when infant mortality has declined and senescence mortality not yet risen. The b_0 parameter represents the initial mortality of the age-dependent increase of mortality and b_1 determines the rate of this increase (Siler 1979).

To model the ages at dispersal, we used a gamma distribution and defined the

PDF as $g(y|\boldsymbol{\gamma})$ with CDF $G(y|\boldsymbol{\gamma})$, where $\boldsymbol{\gamma}$ is a vector of parameters to be estimated. A summary of all the functions, parameters, indicators and variables is provided in Table 3.1.

Table 3.1. Description of random variables, observed variables, and indicators.

	Modeled random variables
X	Random variable for ages at death, where x is any age element
Y	Random variable for ages at natal dispersal with elements y
D	Binary random variable for disperser or non-disperser
S	Binary random variable for sex
	Observed variables and indicators
$\mathbf{t}^F$	Vector of times of first detection
$\mathbf{t}^L$	Vector of times of last detection
$\mathbf{b}$	Vector of times of birth
$\mathbf{x}^F$	Vector of ages at first detection ($x_i^F = t_i^F - b_i$)
$\mathbf{x}^L$	Vector of ages at last detection ($x_i^L = t_i^L - b_i$)
$\mathbf{m}$	Indicator vector for immigrants ($m_i = 1$ if immigrant)
	Updated indicators
$\mathbf{d}$	Indicator vector for dispersers ($d_i = 1$ if disperser and $d_i = 0$ otherwise)
$\mathbf{s}$	Indicator vector for sex ($s_i = 1$ if female and $s_i = 0$ otherwise)
$\boldsymbol{\omega}$	Indicator for potential dispersers
	Parameters
$\boldsymbol{\theta}$	Vector of mortality parameters
$\boldsymbol{\gamma}$	Vector of natal dispersal parameters
	Functions
Mortality	
$\mu(x \mid \boldsymbol{\theta})$	Mortality (Siler model)
$S(x \mid \boldsymbol{\theta})$	Survival
$F(x \mid \boldsymbol{\theta})$	CDF for age at death ($F(x) = 1 - S(x)$)
$f(x \mid \boldsymbol{\theta})$	PDF for age at death
Dispersal	
$g(x \mid \boldsymbol{\gamma})$	PDF for age at natal dispersal (gamma)
$G(x \mid \boldsymbol{\gamma})$	CDF for age at natal dispersal

Likelihood and posterior

To construct the likelihood for our Bayesian model, we assigned a different probability to each type of record in Figure 3.1. The likelihood for females and non-migrating

native-born males was

$$p(D, x^F, x^L; \boldsymbol{\theta}) = \begin{cases} \Pr(X = x^L \mid X > x^F) & \text{if uncensored} \\ \Pr(X > x^L \mid X > x^F) & \text{if censored,} \end{cases} \tag{3.4a}$$

where x^L corresponds to the age at last detection and x^F is the age at first detection (i.e., $x^F = 0$ for individuals born in the study area and $x^F > 0$ for immigrants or individuals born before the study started), while for dispersers the likelihood was constructed as

$$p(D, x^F, x^L; \boldsymbol{\theta}) = \begin{cases} \Pr(X = x^L, Y > x^L \mid X > x^F) & \text{if uncensored} \\ \Pr(X > x^L, Y > x^L \mid X > x^F) & \text{if censored} \\ \Pr(X > x^L, Y = x^L \mid X > x^F) & \text{if dispersed.} \end{cases} \tag{3.4b}$$

As we will show below, the dispersal state D for individuals with uncertain fate was treated as a latent variable that needed to be estimated. The censored and uncensored probabilities for dispersers were used to determine how likely it was for a potential disperser (i.e., departure type of uncertain fate and age older than minimum age at dispersal) to have out-migrated (e.g., last expression in equation 3.4b) or died at the age of last detection x_L (e.g., first expression in equation 3.4b). With this, we could construct the full Bayesian model as

$$\begin{aligned} p(\mathbf{d}_u, \mathbf{s}_u, \boldsymbol{\theta}, \boldsymbol{\gamma} \mid \mathbf{d}_k, \mathbf{s}_k, \mathbf{x}^F, \mathbf{x}^L) \;\propto\; & \underbrace{p(\mathbf{d}, \mathbf{s}, \mathbf{x}^F, \mathbf{x}^L \mid \boldsymbol{\theta}, \boldsymbol{\gamma})}_{\text{likelihood}} \\ & \times \underbrace{p(\mathbf{d})p(\mathbf{s})}_{\text{priors for states}} \\ & \times \underbrace{p(\boldsymbol{\theta})p(\boldsymbol{\gamma})}_{\text{priors for parameters}}, \end{aligned} \tag{3.5}$$

where $\mathbf{d}$ was the vector of dispersal states (i.e., $d_i = 1$ if individual i dispersed and $d_i = 0$ otherwise) and $\mathbf{s}$ was the indicator vector for sex ($s_i = 1$ if female and $s_i = 0$ if male). Each of these vectors had two subsets represented by the subscripts u for

unknown and k for known.

MCMC and conditional posteriors

We used a Markov Chain Monte Carlo (MCMC) algorithm to fit the model in equation 3.5. For all implementations, we ran four parallel MCMC sequences with different randomly drawn starting values and set the number of iterations to 15,000 steps with a burn-in of 5,000 initial steps and a thinning factor of 20. We used a hierarchical framework that only needed the conditionals for posterior simulation by Metropolis-within-Gibbs sampling (Gelfand and Smith 1990; Clark 2007). This means that, for this particular case, the algorithm divided the posterior for the joint distribution of unknowns into four sections: (a) estimation of mortality parameters, (b) estimation of dispersal parameters, (c) estimation of unknown dispersal state, and (d) estimation of unknown sexes. Here we present each section, specifying the conditional posterior and the acceptance probability for the Gibbs Sampler algorithm.

Section a: Posterior for mortality parameters

The conditional posterior to estimate the mortality parameters $\boldsymbol{\theta}$ required only the ages at last detection x_i^L and the dispersal states d_i. The posterior for a given individual i was

$$p(\boldsymbol{\theta} \mid x_i^L, d_i) \propto \begin{cases} \dfrac{f(x_i^L \mid \boldsymbol{\theta})}{S(x_i^F \mid \boldsymbol{\theta})} \; p(\boldsymbol{\theta}|\boldsymbol{\theta}_p) & \text{if } d_i = 0 \\ \\ \dfrac{S(x_i^L \mid \boldsymbol{\theta})}{S(x_i^F \mid \boldsymbol{\theta})} \; p(\boldsymbol{\theta}|\boldsymbol{\theta}_p) & \text{if } d_i = 1 \; , \end{cases} \tag{3.6}$$

where $\boldsymbol{\theta}_p$ was a vector of prior hyper-parameters and x_i^F the age at first detection. If the individual was a native-born, then $x_i^F = 0$ and the denominator in both expressions was equal to 1. At every iteration and for a given parameter $\theta \in \boldsymbol{\theta}$ with conditional posterior $p(\theta \mid \ldots)$, the algorithm proposes a new parameter value for

each element of θ' and accepts it with acceptance probability

$$p(\theta, \theta') = \min \left\{ 1, \frac{\prod_{i=1}^{n} p(\theta' \mid \ldots)}{\prod_{i=1}^{n} p(\theta \mid \ldots)} \right\}. \tag{3.7}$$

Section b: Posterior for dispersal parameters

The conditional posterior to estimate the parameters $\boldsymbol{\gamma}$ for the distribution of ages at first dispersal for a given individual i was

$$p(\boldsymbol{\gamma} \mid x_i^F - \alpha, x_i^L - \alpha, d_i, \omega_i, m_i) \propto \begin{cases} g(x_i^L - \alpha \mid \boldsymbol{\gamma}) \;\; p(\boldsymbol{\gamma} \mid \boldsymbol{\gamma}_p) & \text{if } \omega_i = 1,\ m_i = 0 \ \&\ d_i = 1 \\ [1 - G(x_i^L - \alpha \mid \boldsymbol{\gamma})]\, p(\boldsymbol{\gamma} \mid \boldsymbol{\gamma}_p) & \text{if } \omega_i = 1,\ m_i = 0 \ \&\ d_i = 0 \\ \dfrac{g(x_i^F - \alpha \mid \boldsymbol{\gamma})}{S(x_i^F \mid \boldsymbol{\theta})} \;\; p(\boldsymbol{\gamma} \mid \boldsymbol{\gamma}_p) & \text{if } m_i = 1 \\ 0 & \text{otherwise,} \end{cases} \tag{3.8}$$

where $\boldsymbol{\gamma}_p$ was a vector of prior hyper-parameters for $\boldsymbol{\gamma}$, ω_i was an indicator that assigns 1 if an individual was a potential disperser (i.e., if it belonged to the dispersing sex and disappeared at an age older than the minimum age at dispersal α), and m_i was an indicator for immigrants. We set the minimum age at dispersal to $\alpha = 1.75$ years for the simulated data and $\alpha = 1.5$ for the Serengeti data. The age α corresponded to the earliest age at which immigrants could be detected and potential dispersers could be last seen. For a parameter $\gamma \in \boldsymbol{\gamma}$ with conditional posterior density $p(\gamma \mid \ldots)$ The acceptance probability for a proposed parameter of γ' was

$$p(\gamma, \gamma') = \min \left\{ 1, \frac{\prod_{i=1}^{n} p(\gamma' \mid \ldots)}{\prod_{i=1}^{n} p(\gamma \mid \ldots)} \right\}. \tag{3.9}$$

Section c: Posterior for dispersal states

Dispersal state was evaluated for individuals that were potential dispersers (i.e., $\omega_i = 1$) and estimated the joint probabilities

$$p(d_i \mid x_i^L, d_i, \omega_i, m_i) \propto \begin{cases} f(x_i^L)(1 - G(x_i^L)) \;\; p(d_i|\boldsymbol{\theta}_p, \boldsymbol{\gamma}_p) & \text{if } \omega_i = 1, m_i = 0, d_i = 0 \\ S(x_i^L)g(x_i^L) \;\; p(d_i|\boldsymbol{\theta}_p, \boldsymbol{\gamma}_p) & \text{if } \omega_i = 1, m_i = 0, d_i = 1 \\ 0 & \text{otherwise.} \end{cases} \tag{3.10}$$

The first terms on the right hand side of equation 3.10 correspond to the likelihood function as defined in equations 3.4, while the second terms are the priors for dispersal state. For this section the acceptance probability for the sampling given the last seen ages, the dispersal states, the potential disperser states, and the immigration states was

$$p(d_i, d_i') = min \left\{ 1, \frac{\prod_{i=1}^{n} p(d_i' \mid x_i^L, d_i, \omega_i, m_i)}{\prod_{i=1}^{n} p(d_i \mid x_i^L, d_i, \omega_i, m_i)} \right\}. \tag{3.11}$$

Section d: Posterior for unknown sexes

Some individuals disappeared before the minimum age at dispersal without their sex being determined. The conditional posterior for the latent state of sex was

$$p(s_i \mid x_i^L, \boldsymbol{\theta}) \propto p(x_i^L, \boldsymbol{\theta} \mid s_i) \;\; p(s_i)\,, \tag{3.12}$$

where the second term on the right-hand side is a prior for sex based on sex ratio at birth, or if the analysis was conditioned on survival to age x, based on the sex ratio

at age x.

The indicator for potential dispersers ω_i (see section c) was updated in each iteration. Individuals of undetermined sex and last seen ages older than the minimum age at dispersal were assigned 1 if imputed to be male and 0 if imputed to be female. The acceptance probability given the last seen ages and the mortality parameters was

$$p(s_i, s_i') = min \left\{ 1, \frac{\prod_{i=1}^{n} p(s_i' \mid x_i^L, \boldsymbol{\theta})}{\prod_{i=1}^{n} p(s_i \mid x_i^L, \boldsymbol{\theta})} \right\}. \tag{3.13}$$

Mortality and dispersal priors

We set the Siler parameters for the prior for females to $a_{0p} = -1.4$ ($\sigma = 0.5$), $a_{1p} = 0.65$ ($\sigma = 0.25$), $c_p = 0.07$ ($\sigma = 0.25$), $b_{0p} = -3.8$ ($\sigma = 0.5$), and $b_{1p} = 0.2$ ($\sigma = 0.25$), and for males to $a_{0p} = -1.2$ ($\sigma(a_{0p}) = 0.5$), $a_{1p} = 0.7$ ($\sigma(a_{1p}) = 0.25$), $c_p = 0.16$ ($\sigma(c_p) = 0.25$), $b_{0p} = -3.5$ ($\sigma(b_{0p}) = 0.5$), and $b_{1p} = 0.23$ ($\sigma(b_{1p}) = 0.25$). For dispersal, the Gamma parameters (shape and scale) for the prior were set to $\boldsymbol{\gamma}_p = \{8, 2\}$ with $\sigma(\boldsymbol{\gamma}_p) = \{2, 1\}$. Both the mortality and dispersal priors were uninformative.

Model application and posterior analysis

We fitted the model with sex as a fixed covariate to the Serengeti data. In order to gain deeper insights into the performance of our model, we further exploited a unique source of information that is contained in this data set. A Serengeti lion expert used the circumstances accompanying the disappearances of males to deduce whether the individuals may have dispersed. For example, since young males often leave their natal prides with brothers, a simultaneous disappearance of brothers hints that this is likely to be a dispersal event. We fitted the model with three different settings. First, all males with uncertain fates and last seen ages older than minimum age at

dispersal were assigned the state of 'potential dispersers' and entered in the model as described in 'Section c' above (Model A). Second, all males that were indicated to may have dispersed were entered as 'known dispersers' (see equation 3.4b) (Model B). And third, all males that were indicated to may have dispersed were entered as 'potential dispersers' (Model C).

To avoid problems arising from the large number of unsexed individuals that died within the first weeks after birth, we fitted the model from the start age of 0.25 years. We predicted mortality rates for each sex using the parameter estimates of every step of the MCMC after burn-in and thinning and used these predictions to calculate mean and credible intervals of mortality rates. To compare the three models, we computed the life expectancy at the model start age ρ as $e_\rho = S(\rho) \int_\rho^\omega S(x)\, dx$, with ω corresponding to the maximum age, and the Kullback-Leibler (KL) divergences of the mortality parameter posterior densities (Kullback and Leibler 1951; McCulloch 1989; Burnham and Anderson 2001).

The KL divergence calculates the difference or the amount of overlap between two distributions. To illustrate the calculation of KL, let's take a parameter θ, for which the resulting 'sub-parameters' for females and males would be θ_f and θ_m, respectively. Thus, for an individual i, we have $\theta = \theta_f I_i + \theta_m(1 - I_i)$, where I_i is an indicator function that assigns 1 if the individual is a female and 0 otherwise. For each of these parameters, our model produces a posterior distribution, say $P_f = p(\theta_f|\dots)$ and $P_m = p(\theta_m|\dots)$, respectively. The KL between these distributions is calculated as

$$K(P_f, P_m) = D_{f,m} = \int_{-\infty}^{\infty} P_f \, \log\left(\frac{P_f}{P_m}\right) \mathrm{d}\theta \, . \tag{3.14}$$

The result can be interpreted as how far off we would be if we tried to predict θ_m one from the posterior distribution of θ_f. If both distributions are identical, then $D_{f,m} = 0$, suggesting that there is no distinction between the parameters of both

covariates and hence, that both covariates have the same effect. With increasing KL values, the discrepancy becomes higher. As can be inferred from Equation 3.14, the relationship is asymmetric, namely $K(P_f, P_m) \neq K(P_m, P_f)$.

To make KL values easier to interpret, McCulloch (1989) proposed a simple calibration of the KL values that reduces the asymmetry. Let $k = K(P_f, P_m)$ and q_k be a calibration function such that

$$\begin{aligned} k &= K(P_f, P_m) \\ &= K\left[B\left(\frac{1}{2}\right), B(q_k)\right], \end{aligned}$$

where $B(\frac{1}{2})$ is a Bernouilli distribution for an event with probability 0.5 (i.e., same probability of success and failure). This calibration is then calculated as

$$q_k = \frac{\left[1 + (1 - e^{-2k})^{\frac{1}{2}}\right]}{2}. \tag{3.15}$$

Thus, q_k ranges from 0.5 to 1, where a value of 0.5 means that the distributions are identical, and 1 that there is no overlap between them.

Results

Simulation study

We used a simulation study to validate our model. For all 12 simulations, the mortality rates used to simulate the data lay within the 95 % credible intervals of the estimated mortality for both sexes (Figure 3.2). Of all the introduced variations in data quality (sample size, unsexed individuals, proportion 'known' deaths), the only one with a marked effect on the performance of the model was varying the sample size. As could be expected, smaller sample sizes resulted in wider credible intervals particularly for males and for older ages of females. Due to the wider

confidence bands for smaller sample sizes, the respective estimated mortality rates could appear to be less variable over the life span than the mortality rates used to simulate the data. This manifested as a less-pronounced U-shape of the estimated mortality rates when compared to the 'real' mortality rates (e.g., second panel in second row of Figure 3.2). The proportion of unsexed individuals dying at < 1 year of age, and the proportion of known deaths among disappearances did not discernibly affect the retrieval of the mortality parameters.

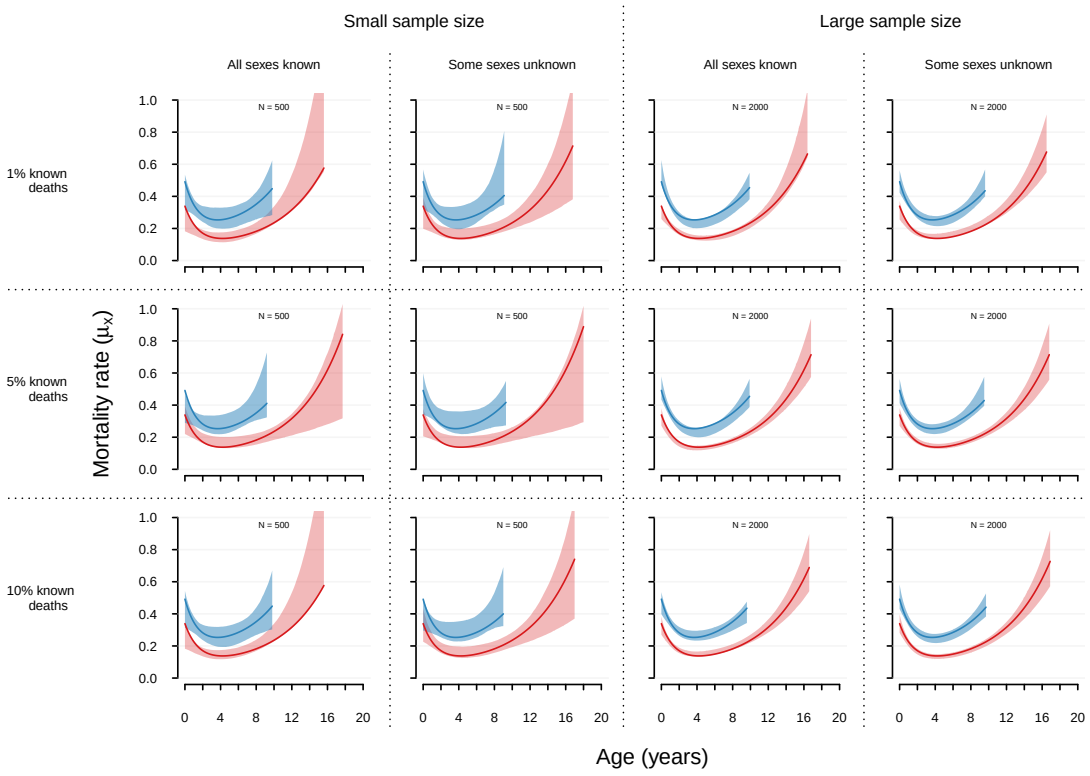

Figure 3.2. Predicted mortality rates for males (blue polygons) and females (pink polygons) compared to the mortality rates used to simulate the data (solid lines). Polygons represent 95 % credible intervals of age-specific mortality rates. Mortality rates are plotted until the ages when 95 % of a synthetic same-sex cohort would be dead. Results are given for 12 simulations varying the size of the native-born population (N = 500 or N = 2000), the proportion of known deaths among last seen ages (1%, 5%, or 10%), and whether the sex of 30% of individuals dying younger than 1 year of age remained undetermined or not.

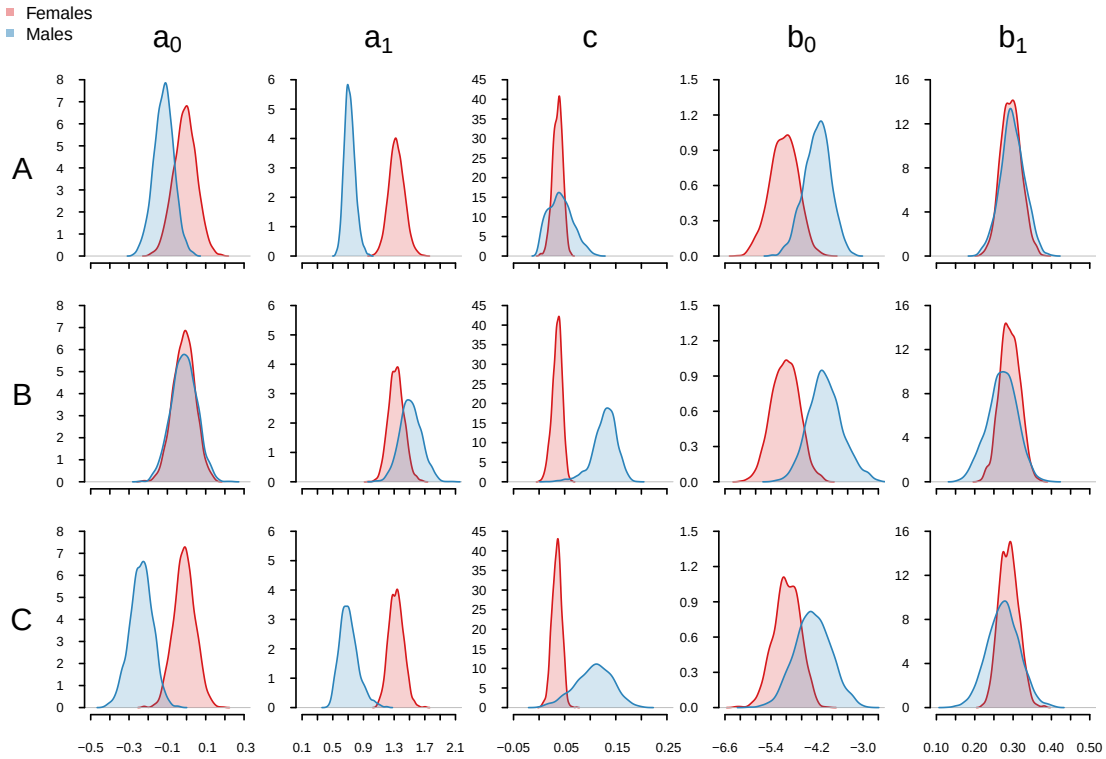

Figure 3.3. Posterior distributions of Siler parameter estimates (a_0, b_0, c, a_1, b_1) for female (pink) and male (blue) African lions of the Serengeti population. The analysis was conditioned on survival of the first 3 months of life.

Application

The empirical models for Serengeti lions converged for all estimated parameters (Figure 3.3, see also Figure S1 for traces). Overall mortality of both sexes was U-shaped with high initial cub mortality, low mortality of prime-aged adults, and an age-dependent increase in mortality during the older ages (Figure 3.4). Mortality of males was higher than mortality of females across all ages (Figure 3.4), except for very young ages, up until one year, during which confidence bands of male and female mortality overlapped. However, this may be due to the large proportion of unsexed individuals at these ages (see data description) and the imputation of sex as a latent state for these individuals, which introduced uncertainty. Due to the higher male mortality rates across most ages, female life expectancy (4.7 years at model start age) exceeded that of males by approximately 2 years.

Now we turn to the comparison between the models with varying settings for potential dispersers. Model A (Figure 3.4a) treated the data as if no further information was available on dispersal status of males with uncertain fates. This is the default setting of the model where males with uncertain fates and last seen ages older than the minimum age at dispersal are treated as potential dispersers whose dispersal state is imputed by the model as a latent state. Model B took advantage of expert knowledge on lion behaviour and treated all males that a lion expert strongly believed were dispersers, as known dispersers (Figure 3.4b). Finally, Model C treated all expert-indicated potential dispersers as potential dispersers (Figure 3.4c.) The number of potential dispersers whose dispersal state was imputed as a latent state was therefore smaller in Model C when compared to Model A.

We compare these models by examining the estimated mortality rates (Figure 3.4), the posterior density distributions (Figure 3.3), and the KL divergences (Figure 3.5). Since females were treated the same way in all three models, the posterior distributions of parameters for females were congruent among the three models (Figure 3.3). This was well-captured by the corresponding KL divergences, which were close to, or equal to, 0.5 (Figure 3.5). Consequently, female mortality rates were almost identical across all three models (Figure 3.4).

For males, the three models gave varying results. The different settings regarding potential dispersers mostly affected the estimation of the Siler parameters that describe initial mortality (a_0), the age-dependent decrease in mortality at young ages (a_1), and the age-independent mortality (c) (Figure 3.5). The initial mortality was higher in Model B, and lower in Model C, when compared to the default model A (Figure 3.3, Table S1). The age-dependent decrease in mortality was steeper in Model B compared to Model A but similar between Model A and C. The age-independent mortality was higher in both Model B and C when compared to the default Model A. The parameters governing mortality rates at older age (b_0 and b_1) were similar across all three models and the confidence bands overlapped (Figure 3.3,

Table S1).

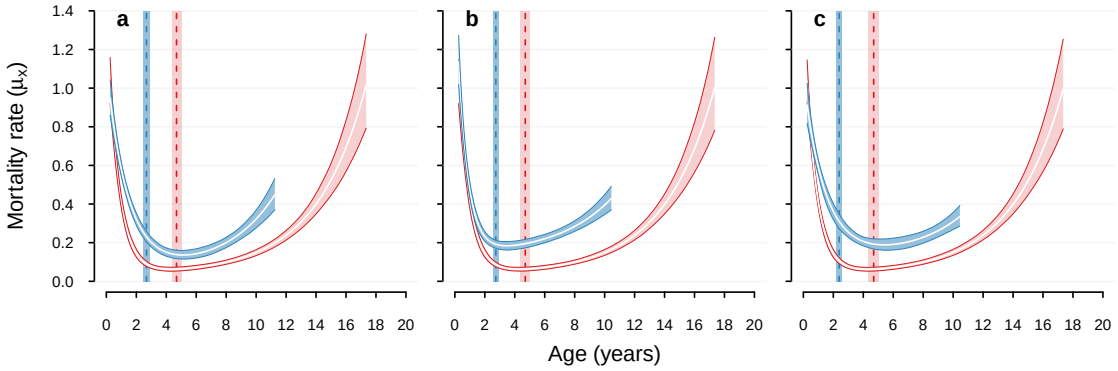

Figure 3.4. Age-specific mortality estimates for male (blue lines and polygons) and female African lions (pink lines and polygons) of the Serengeti population. Polygons represent 95 % credible intervals of age-specific mortality rates with white lines indicating the mean. Mortality rates are plotted until the ages when 95 % of a synthetic same-sex cohort would be dead. The vertical dashed lines indicate mean life expectancy at 0.25 years of age with the 95 % confident bands indicated by the rectangles. (a) Model A: all males with uncertain fate and old enough for dispersal treated as potential dispersers. (b) Model B: all males indicated by an expert as potential dispersers treated as known dispersers. (c) Model C: all males indicated by an expert as potential dispersers treated as potential dispersers.

Because the Siler mortality model is a composite of three additive mortality hazards, the differences among the three models can be more fully understood by comparing the male mortality rates predicted from the three models (Figure 3.4). Due to the steep decline in age-dependent mortality at younger ages when all expert-indicated dispersers were treated as dispersers (Model B), mortality rates during the juvenile ages up to approximately three years of age were lower in Model B when compared to both models that imputed dispersal state for potential dispersers (Model A and C). However, for the prime-adult-ages, Model B gave the highest mortality estimates, followed by Model C, and then Model A, which gave the lowest estimates. Mortality rates at older ages were highest in Model A and B. Despite these differences in the shape of the mortality rate curves, the life expectancies at 0.25 years of age were predicted to be identical by Model A and B (2.7 years), and only slightly different by Model C (2.4 years).

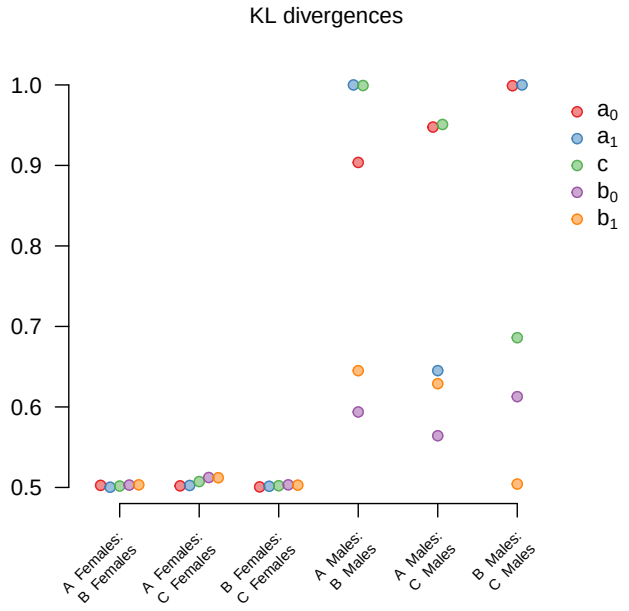

Figure 3.5. Kullback-Leibler (KL) divergences comparing same-sex Siler parameter posteriors among the three models (A, B, C) with varying settings for males with uncertain fate. Note that the KL divergence estimates are jittered in x-axis direction to improve visibility. The analysis was conditioned on survival of the first three months of life.

Discussion

Life history data of wild animals are often incomplete because animals, even though alive and well, may temporarily or permanently be absent when researchers try to observe them at a given location. This simple observation has far reaching consequences for the estimation of biological properties from these data. Accordingly, various statistical approaches have been developed that account for temporal and spatial heterogeneity in recapture probabilities. For example, multistate CPR methods have been applied to estimate survival rates while accounting for migration between locations within study sites (Arnason 1973; Schwarz et al. 1993; Lebreton and Pradel 2002; Pradel 2005; Mackenzie et al. 2009; Schaub and Royle 2013; Lagrange et al. 2014). And spatially explicit CPR methods have been developed to estimate survival probabilities and population size (Borchers and Efford 2008; Efford and

Mowat 2014).

However, none of these models can account for the extreme case of spatially structured detection probabilities that are typical for male records of highly-detectable species in which males disperse. Here, the detection probability falls from one, when animals are in the area, to zero, when they leave. Furthermore, the possibility that a non-detectable male may have dispersed contributes uncertainty to all male recapture histories that end after the age of maturity without the recovery of a body. To meet these challenges, our model does not model spatially heterogeneous detection probabilities but rather imputes the dispersal state of the uncertain male records as a latent state variable in a Bayesian hierarchical framework (Clark et al. 2005; Colchero and Clark 2012; Colchero et al. 2012).

To gauge the possibility to estimate sex- and age-specific mortality in species with male natal dispersal, we focussed on data whose incomplete records for sex and age at death arose from only one of two mechanisms. First, males dispersing from the study area created uncertain male records of age at death, and second, individuals dying before sex could be determined resulted in uncertain sex. Implicitly, the model therefore assumes that all birth dates are known and that all other types of records can be treated as complete records. Consequently, the model treated the last seen ages of immigrants, native-born females, and males that were imputed to be non-dispersers as certain ages at death. The model therefore hinges on the assumption that only males disperse, and that they disperse only once during their life. The latter is a particularly strong assumption that may not hold for many species. To relax these assumptions, the dispersal state of females in species with bi-sexual dispersal could be implemented in an analogous way to the procedure for males. Accounting for higher order dispersal, on the other hand, would be more complicated and would most likely involve setting strong priors.

Another consequence of the treatment of immigrants' last seen ages as ages at death is that the ratio of immigrants to dispersers is likely to influence the estimation

of male mortality parameters. This causes problems if the probability of dispersal out of the study area is higher than the probability of immigrating into it (see Figure S1 for a simulation). This may be the case for field sites that are established in protected areas and act as a source population for surrounding habitats of lower quality. Mortality in these habitats, and mortality during the dispersal process itself, may also be higher than mortality within the study area. Our model cannot account for this heterogeneity.

Overall, given the recovery of the mortality parameters in the simulation study, we conclude that our approach is a promising step towards providing a statistical method to infer male mortality for species with incomplete male records due to male natal dispersal. Mortality parameters were recovered even if sample sizes were low, observed deaths among potential dispersers were rare, and some individuals died before sex could be determined. Our approach is therefore the first to provide estimates of male lion mortality (Packer 2005) (see Chapter 4 for further analyses and discussion). The bathtub shape of male mortality and the higher male than female mortality rates across most ages are in line with previous findings on female mortality (Packer et al. 1998), and on sex differences in mortality in mammal species with sexual size dimorphism (Promislow 1992) and fewer years of breeding in males than in females (Clutton-Brock and Isvaran 2007).

The comparison of the different models for the lion data allows us to draw some conclusions about the biases we can expect in estimates of mortality parameters and predicted mortality rates when using our model. If all expert-indicted dispersers were in fact dispersers (Model B), then by comparing the mortality rates estimated by this model and by the one with the default treatment of dispersers (Model A), we learn that the default model may have the tendency to overestimate mortality during juvenile ages (lower a_1 in Model A than B). The default model may furthermore be biased towards slightly underestimating mortality during prime-adult ages. Since the model that treats all expert-indicated dispersers as potential dispersers (Model

C) shares properties of both Models A and B (similar c to Model B, similar a_0 and a_1 to Model A), and may come closest to reality, it seems like a promising avenue for future development to directly include expert knowledge in the Bayesian framework via priors. Nevertheless, despite the differences in mortality estimates obtained from the three models, they all predicted very similar life expectancies, and the confidence bands of predicted mortality rates overlapped across many ages. Therefore, high confidence can be put on summary statistics of mortality obtained from our model, and the obtained age-specific mortality estimates can be used, albeit with caution, where no superior mortality model or estimates exist.

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Supporting Information

Figure S1 Traces of mortality and dispersal parameter estimation for Models A to C.

Figure S2 Predicted mortality rates for simulated data if male immigration probability was set to 0.5.

Table S1 Estimated coefficients for models A to C.

Code S1 R code to simulate data. Download from github.com/bartholdja/DPhil_supplements.

Code S2 R code to run the model on simulated data. Download from github.com/bartholdja/DPhil_supplements.

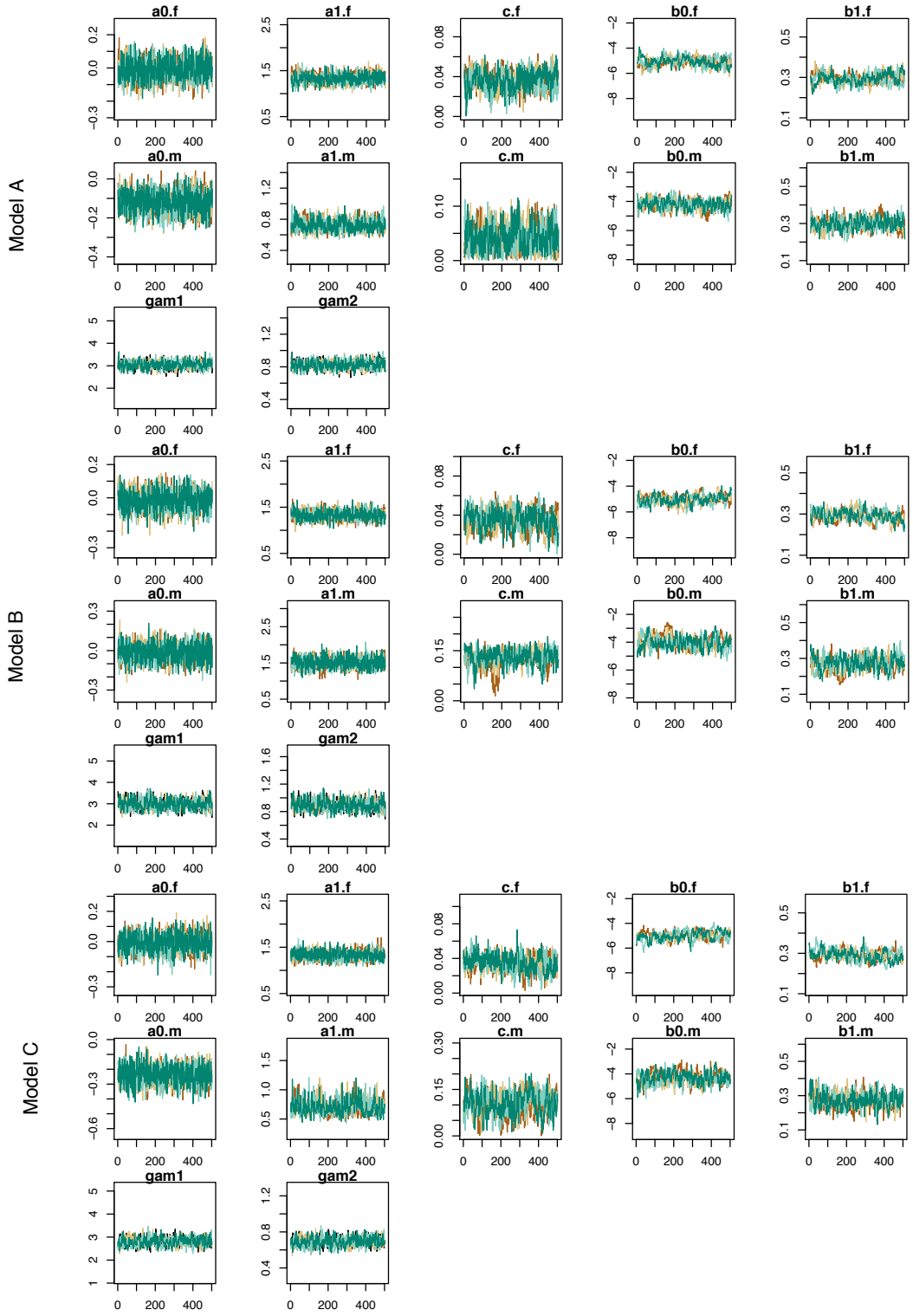

Figure S1. Trace plots for four parallel runs for the Serengeti lion mortality analysis. Estimated parameters are the Siler parameters (a_0, b_0, c, a_1, b_1; f denotes estimates for females and m for males) and Gamma parameters (shape and rate; gam1 and gam2). Model A: all males with uncertain fate and last seen ages older than minimum age at dispersal treated as potential dispersers. Model B: all males that an expert indicated as potential dispersers treated as known dispersers. Model C: All males that an expert indicated as potential dispersers treated as potential dispersers.

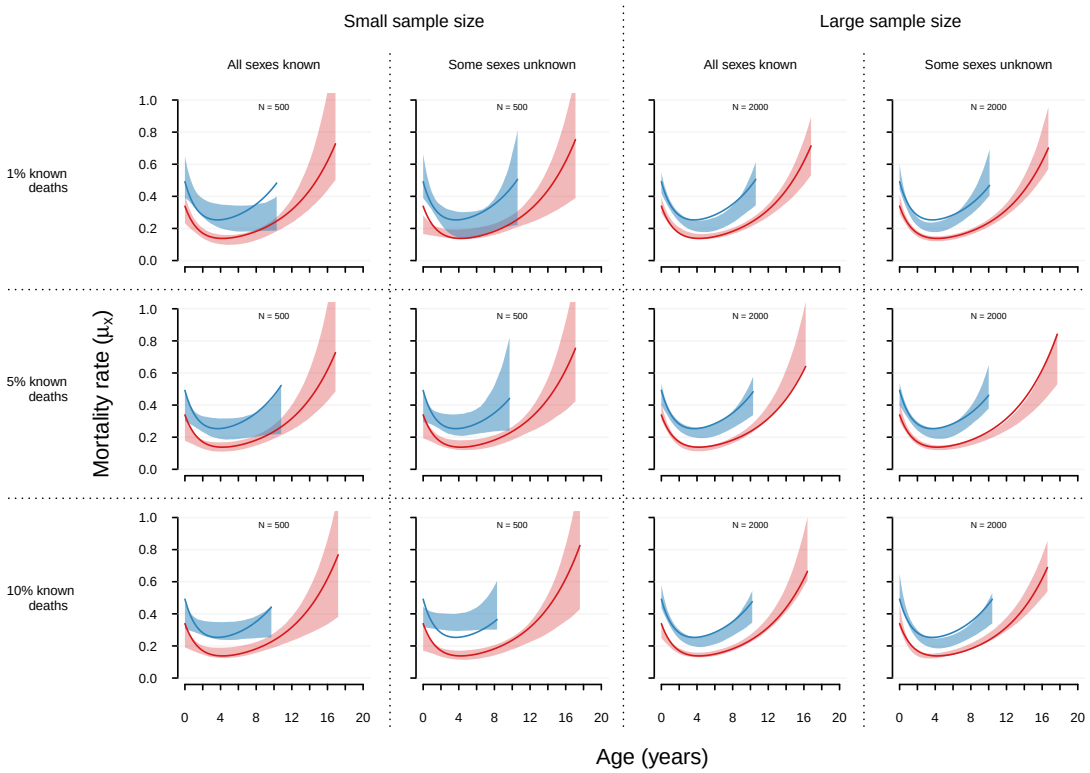

Figure S2. Predicted mortality rates for males (blue polygons) and females (pink polygons) compared to the mortality rates used to simulate the data (solid lines), if the probability to immigrate into the study area of males born outside of it was lowered from 1 to 0.5. Polygons represent 95 % credible intervals of age-specific mortality rates. Mortality rates are plotted until the ages when 95 % of a synthetic same-sex cohort would be dead. Results are given for 12 simulations varying the size of the native-born population (N = 500 or N = 2000), the proportion of known deaths among last seen ages (1%, 5%, or 10%), and whether the sex of 30% of individuals dying younger than 1 year of age remained undetermined or not.

Table S1. Estimated Siler and gamma coefficients for Model A (all males with uncertain fate and old enough for dispersal treated as potential dispersers), Model B (all males indicated by an expert as potential dispersers treated as known dispersers, and Model C (all males indicated by an expert as potential dispersers treated as potential dispersers). Given are mean, SE, and credible intervals of the parameter posterior distributions.

		Coefficient	Mean	SE	2.5 %	97.5 %
Model A	Females	a_0	-0.01	0.06	-0.12	0.11
		a_1	1.33	0.10	1.14	1.54
		c	0.04	0.01	0.02	0.05
		b_0	-5.06	0.36	-5.79	-4.38
		b_1	0.29	0.03	0.24	0.35
	Males	a_0	-0.12	0.05	-0.22	-0.01
		a_1	0.72	0.07	0.60	0.87
		c	0.04	0.02	0.00	0.09
		b_0	-4.19	0.35	-4.89	-3.55
		b_1	0.30	0.03	0.24	0.36
		gam_1	3.04	0.17	2.72	3.37
		gam_2	0.82	0.05	0.73	0.92
Model B	Females	a_0	-0.01	0.06	-0.12	0.10
		a_1	1.33	0.10	1.14	1.53
		c	0.04	0.01	0.01	0.05
		b_0	-5.02	0.36	-5.70	-4.33
		b_1	0.29	0.03	0.24	0.34
	Males	a_0	-0.01	0.07	-0.14	0.12
		a_1	1.52	0.14	1.25	1.80
		c	0.13	0.02	0.07	0.17
		b_0	-3.98	0.44	-4.81	-3.03
		b_1	0.27	0.04	0.19	0.34
		gam_1	2.97	0.22	2.56	3.45
		gam_2	0.89	0.07	0.76	1.04
Model C	Females	a_0	-0.01	0.06	-0.12	0.10
		a_1	1.33	0.09	1.15	1.51
		c	0.03	0.01	0.02	0.05
		b_0	-4.99	0.34	-5.68	-4.35
		b_1	0.29	0.03	0.24	0.34
	Males	a_0	-0.24	0.06	-0.36	-0.12
		a_1	0.71	0.12	0.51	1.00
		c	0.11	0.04	0.03	0.17
		b_0	-4.31	0.47	-5.21	-3.34
		b_1	0.27	0.04	0.19	0.36
		gam_1	2.81	0.18	2.46	3.16
		gam_2	0.69	0.05	0.60	0.80

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CHAPTER 4

Variation in male and female mortality for African lion populations under varying degrees of human impact

Running head

Sex- and age-specific lion mortality

Title

Variation in male and female mortality for African lion populations under varying degrees of human impact

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Abstract

As apex predators, flagship species, and desired trophies, the conservation of African lions is paramount for the conservation of their ecosystems. However, age-specific mortality, a fundamental descriptor of their life history, is only known for females of one population, and unknown for males due to male natal dispersal. When males disappear from monitored areas at around the age of maturity, they may have died or dispersed away from the study area. This uncertainty has hindered inferences on male mortality using standard methods for mark/re-sighting data. Here, we use a newly-developed mortality model that imputes dispersal state for uncertain male records as a latent state variable jointly with the coefficients of a parametric age-specific mortality model in a Bayesian hierarchical framework. In order to study how human impact may alter age trajectories of lion mortality, and whether mortality of both sexes responds similarly to human impact, we fitted the model to data from two wild lion populations. The first population inhabits a high-quality habitat at high densities and is largely undisturbed by humans, while the second population acts as a source population for trophy hunting and is exposed to accidents, poaching, and human-wildlife conflicts. Contrary to our expectations, we found that adult mortality was only slightly higher, and cub mortality much lower, in the disturbed population when compared to the undisturbed one. Nevertheless, higher age-independent mortality in the disturbed population indicated higher exposures to external sources of mortality; a finding that likely reflects anthropogenic mortality. We discuss these results in the light of density dependence. Our study provides estimates of age- and sex-specific mortality for two populations that may represent common types of lion populations. The estimates can therefore inform population management measures aimed to avert local extinction.

Key words

age-specific mortality, Gompertz law of mortality, Siler model, dispersal, carnivores, African lion, sex differences in mortality, sex differences in life history, ageing, Serengeti, Hwange

Introduction

Mortality varies between the sexes as an evolutionary consequence of sex-specific reproductive strategies (Clutton-Brock and Isvaran 2007), and among populations due to variation in the environment, population density, and exposure to anthropogenic factors. Estimates of mortality for wild animal populations are regularly used to test ecological and evolutionary theory, and to project future population size and structure for population management measures. Such measures are needed, for example, for many populations of African lions (*Panthera leo*) that are facing local extinction (Packer et al. 2011, Riggio et al. 2012, Packer et al. 2013). Population models for African lions use published age-specific mortality for females (Becker et al. 2013, Packer et al. 1998), but for males, no such estimates are available. This is because traditional re-sighting records of marked individuals, that readily provide estimates of female mortality, have until now been of little use in estimating male mortality due to male natal dispersal. When males disappear from monitored populations around the age of maturity, their fate remains uncertain; they may have died or settled outside of the study area. This uncertainty hinders statistical inference on male mortality.

The most common answer to male data deficiency in population ecology is to ignore males altogether and to model only the dynamics of the female population. This is a legitimate approach for a wide variety of questions and species. However, for lions it is generally recognised that male mortality may affect population dynam-

ics via the mechanistic link of infanticide (Whitman et al. 2004, Caro et al. 2009). Frequent deaths of adult males disturb the social structure of prides and male coalitions and result in a higher frequency of male takeovers with subsequent deaths of young infants, premature evictions of juveniles with low survival probability, and a risk of injuries for females trying to protect dependent young (Elliot et al. 2014). Furthermore, for many applications of demographic models of lions, males are the sex of interest, because the models aim to address issues of sustainable shooting quota in populations hunted for trophies (Whitman et al. 2007). Existing studies of lion population dynamics therefore use various approaches to overcome the lack of male age-specific mortality estimates. Some use population summary statistics without further differentiating the age- or sex-structure of the population (Packer et al. 2011), others structure their model in a way that makes a distinction of uncertain male records into deaths and dispersal superfluous (Whitman et al. 2004, 2007), or use female mortality estimates for both sexes (Becker et al. 2013). It is therefore clear that the development of a model that incorporates estimates for both sexes will fill a significant methodological gap.

Among dieocious organisms, sex differences in life history are the norm. In mammal species that exhibit a polygynous or polygynandrous mating system in which males compete for access to receptive females by physical combat, males are commonly larger than females, mature later, reproduce for fewer years, and die younger than females (Promislow 1992, Andersson 1994, Clutton-Brock and Isvaran 2007). Lions are a prominent example of this type of mating system, and a shorter male than female life span in this species is well known. Yet the degree to which mortality of males and females differ at various ages or stages of their life cycle, and whether sex differences are stable across populations, is poorly understood. Without this knowledge, evolutionary causes and ecological consequences of sex differences in mortality cannot be unveiled.

Recently, Barthold et al. (in prep.; see Chapter 3) have developed a survival

analysis method that can incorporate uncertain male records for species with male natal dispersal. In this model, dispersal state (i.e., whether a male with an uncertain fate dispersed or died) is imputed as a latent state jointly with the coefficients of a parametric mortality model in a Bayesian hierarchical framework. The mortality model decomposes mortality into age-dependent and age-independent mortality (Siler 1979). Age-independent mortality represents mortality due to external sources that kill regardless of age (Pletcher 1999). The mortality decomposition therefore allows to compare mortality between the sexes or populations not only in terms of different levels but also in terms of differences in underlying processes.

Using this model, we estimated age-specific mortality rates for males and females for two populations of African lions that varied with respect to environmental factors, densities, and human impact. The first population was hardly impacted by humans and lived in a nutrient-rich environment in high densities (the "undisturbed" population). The second population acted as a source population for trophy hunting areas, was subjected to killings in accidents and human-wildlife conflicts, and lived in a food-scarce environment at low densities (the "disturbed" population). We compared mortality between these two populations in order to study how human impact alters age trajectories of mortality, and whether males respond differently to human impact than females. If human activities affected mortality rates, we expected to find higher levels of mortality, and particularly of age-independent mortality, in the disturbed population compared to the undisturbed one. Due to the sexual size dimorphism and the polygynous mating system of lions (Clutton-Brock and Isvaran 2007), as well as previous observations (Packer et al. 1988), we expected to find sex differences in mortality at all ages in both populations. We further expected to observe an amplification of this difference in the population impacted by humans, since males were the primary target of trophy hunting and had a higher risk of being killed by famers in retaliation for raided livestock.

Methods

Data

We used life history data from two free-living lion populations that have been monitored for many years. One of the populations lives in the northern range of Hwange National Park in north-western Zimbabwe. The study area extends to 7000 km^2 and receives 600 mm rainfall seasonally. Vegetation is a mosaic of mixed deciduous woodland and scrubland with limited areas of open or bushed grassland (Rogers 1993, Loveridge et al. 2007). Water is artificially supplied during the dry season, and the prey assemblage is largely resident. The park borders on hunting concessions in the north and north-east. Human settlements occur on the north and east of the park and are mainly used for subsistence agriculture and wildlife exploitation under the Communal Areas Management Plan for Indigenous Resources (CAMPFIRE) scheme. The park shares a border with wildlife management areas in Botswana to the west. Life history data were collected between 1999 and 2013. Prides and resident male nomads and male coalitions were located by radio telemetry and censused approximately once per month. Field staff identified individual lions from markings such as whisker spot patterns, scars, and teeth characteristics (Pennycuick and Rudnai 1970, Smuts et al. 1978). A summary of the data used is given in Table S1.

The second population occupies a 2000km^2 study area in the Serengeti National Park, Tanzania. The area has a southeast to northwest gradient in vegetation from short to tall grassland to open woodlands (Packer 2005, Mosser et al. 2009). Most rainfall occurs during the wet season, when large herds of migratory herbivores pass through. In response to an increasing abundance of migratory prey, the study population has grown since the start of the study in 1966 (Packer 2005; unpublished data). We used life history data collected between 1966 and 2013. During the early

years of the study (1966-1984), observers gathered data from opportunistic sightings, about 1-3 times per month for most individuals. Since 1984, using radio telemetry, observers have sighted each animal 2-6 times per month. The observers identify individuals from natural markings (Packer et al. 1991), and deduce birth dates of cubs born in the study area from lactation stains on the mothers. A large number of nomadic males enter the area, most of them migrate through without taking up residence in the study population. Our analyses excluded all nomadic males that never became residents (n = 548). A summary of data is provided in Table S1.

In both studies, trained observers estimate age of individuals with unknown dates of birth using age indicators such as relative body size, nose coloration, and eruption and wear of teeth (Smuts et al. 1978, Whitman et al. 2004). Both data sets contain individuals that died at young ages before sex could be determined (unsexed records). For all data, we identified male records as uncertain (i.e., the male may potentially have dispersed) if missing males that were born in the study area (native-borns), and whose deaths were not observed, were older than 1.5 years at disappearance (minimum age at dispersal).

Survival analysis

The parametric mortality and dispersal models

We fitted a parametric model for age-specific mortality. With X being a random variable for ages at death, the mortality rate, or hazard of death, for continuous age x was

$$\mu(x|\boldsymbol{\theta}) = \lim_{\Delta x \to 0} \frac{\Pr(x \leq X < x + \Delta x \mid x \leq X, \boldsymbol{\theta})}{\Delta x}, \tag{4.1}$$

where $\boldsymbol{\theta}$ was a vector of mortality parameters. From the mortality rate, the probability to survive from birth to age x, or survivor function, could be calculated as

$$S(x|\boldsymbol{\theta}) = Pr(X \geq x) = \exp\left[-\int_0^x \mu(z|\boldsymbol{\theta})\mathrm{d}z\right]. \tag{4.2a}$$

And the probability that death occurred before age x, or the cumulative density function (CDF), was

$$F(x|\boldsymbol{\theta}) = Pr(X < x) = 1 - S(x|\boldsymbol{\theta}), \tag{4.2b}$$

with the probability density function (PDF) for age at death of

$$f(x|\boldsymbol{\theta}) = \frac{d}{dx}F(x|\boldsymbol{\theta}) = S(x|\boldsymbol{\theta})\mu(x|\boldsymbol{\theta}). \tag{4.2c}$$

To capture the bathtub shape of lion mortality (Packer et al. 1998), and to allow for the estimation of age-independent mortality, we used the Siler model (Siler 1979) in the form

$$\mu(x|\boldsymbol{\theta}) = e^{a_0 - a_1 x} + c + e^{b_0 + b_1 x}, \tag{4.3}$$

where $\boldsymbol{\theta} = \{a_0, a_1, c, b_0, b_1\}$, with $a_0, b_0 \in \mathbb{R}$ and $a_1, c, b_1 > 0$. The Siler model is the sum of three additive mortality hazards (Siler 1979). The first summand models the decrease in mortality rates over infant and juvenile ages, with a_0 being the initial level and a_1 modelling the rate of decrease. The middle summand is a constant hazard c, also known as a Makeham term (Makeham 1860), that captures age-independent mortality. The last summand is the Gompertz law of mortality (Gompertz 1825), which captures the exponential increase in mortality rates with age from an initial level b_0 with a rate of increase of b_1.

For modelling the ages at dispersal, we used a gamma distribution and defined the random variable Y for ages at natal dispersal. The PDF of age at dispersal was

$g(y|\boldsymbol{\gamma})$ and the CDF $G(y|\boldsymbol{\gamma})$, where $\boldsymbol{\gamma}$ is a vector of two parameters to be estimated.

The Bayesian model

The survival analysis took the ages at first x^F and last detection x^L as input. In a Bayesian hierarchical framework, the model maximised the posteriors of the mortality and dispersal model, while imputing the dispersal state D for uncertain male records (dispersed or died) and the sex S for unsexed records as latent states (Barthold et al. in prep.; see Chapter 3). Contributions to the mortality and dispersal likelihood varied according to the sex, dispersal state, and migration history of the individual. For females, immigrants, and individuals whose deaths were observed, the last seen ages entered the mortality likelihood as ages at death ($X = x^L$). Implicitly, we therefore assumed that only males dispersed and that they only dispersed once at around the age of maturity (natal dispersal).

For uncertain male records, last seen ages entered the mortality likelihood as ages at death if males were imputed to have died ($D = 0, X = x^L$) and as ages of right-censoring if males were imputed to have dispersed ($D = 0, X > x^L$). Here, with the term "dispersal" we refer to only those dispersal events during which males left the study area and which therefore resulted in a right-censored record. Right-censored records, also those due to individuals being alive at the end of the study, were incorporated in the likelihood following the Cormack-Jolly-Seber framework (Cormack 1964, Jolly 1965, Seber 1965) as the probability to have survived up to the last seen age ($X > x^L$).

The dispersal likelihood took contributions from the first seen ages of immigrants (x^F) as age of dispersal ($Y = x^F$), from the last seen ages of uncertain male records that were imputed to have dispersed as age of dispersal ($Y = x^L$), and from the last seen ages of uncertain male records that were imputed to have died, and of censored male records, as age of right-censoring ($Y > X^L$). A detailed description of the construction of the likelihoods and posteriors can be found elsewhere (Barthold

et al. in prep.; see Chapter 3).

Given the input parameters and known and latent states, the full Bayesian model was

$$\begin{aligned} p(\mathbf{d}_u, \mathbf{s}_u, \boldsymbol{\theta}, \boldsymbol{\gamma} \mid \mathbf{d}_k, \mathbf{s}_k, \mathbf{x}^F, \mathbf{x}^L) \quad \propto \quad & \underbrace{p(\mathbf{d}, \mathbf{s}, \mathbf{x}^F, \mathbf{x}^L \mid \boldsymbol{\theta}, \boldsymbol{\gamma})}_{\text{likelihood}} \\ \times \quad & \underbrace{p(\mathbf{d})p(\mathbf{s})}_{\text{priors for states}} \\ \times \quad & \underbrace{p(\boldsymbol{\theta})p(\boldsymbol{\gamma})}_{\text{priors for parameters}}, \end{aligned} \tag{4.4}$$

where $\mathbf{d}$ was the vector of dispersal states and $\mathbf{s}$ was the indicator vector for sex. Each of these vectors had two subsets represented by the subscripts u for unknown and k for known.

MCMC and conditional posteriors

We fitted the model in equation 4.4 using a Markov Chain Monte Carlo (MCMC) algorithm in 4 parallel sequences. We randomly drew starting values and set the number of iterations to 15,000 steps with a burn-in of 5,000 initial steps and a thinning factor of 20. We used a hierarchical framework that only needed the conditionals for posterior simulation by Metropolis-within-Gibbs sampling (Gelfand and Smith 1990, Clark 2007). This means that, for this particular case, the algorithm divided the posterior for the joint distribution of unknowns into four parts: (a) estimation of mortality parameters, (b) estimation of dispersal parameters, (c) imputation of unknown dispersal state, and (d) imputation of unknown sexes. We have described the different parts, specifying the conditional posteriors and the acceptance probabilities for the Gibbs sampler algorithm, elsewhere (Barthold et al. in prep.; see Chapter 3).

Mortality and dispersal priors

The Siler parameters for the prior for females were $a_{0p} = -1.4$ ($\sigma = 0.5$), $a_{1p} = 0.65$ ($\sigma = 0.25$), $c_p = 0.07$ ($\sigma = 0.25$), $b_{0p} = -3.8$ ($\sigma = 0.5$), and $b_{1p} = 0.2$ ($\sigma = 0.25$), and for males to $a_{0p} = -1.2$ ($\sigma(a_{0p}) = 0.5$), $a_{1p} = 0.7$ ($\sigma(a_{1p}) = 0.25$), $c_p = 0.16$ ($\sigma(c_p) = 0.25$), $b_{0p} = -3.5$ ($\sigma(b_{0p}) = 0.5$), and $b_{1p} = 0.23$ ($\sigma(b_{1p}) = 0.25$). For dispersal, the Gamma parameters (shape and scale) for the prior were set to $\boldsymbol{\gamma}_p = \{8, 2\}$ with $\sigma(\boldsymbol{\gamma}_p) = \{2, 1\}$. All priors for parameters were uninformative. The priors for sex as a latent state corresponded to the empirical sex ratios at model start ages (Table S1).

Model application and posterior analysis

To study the differences in mortality between the sexes and between the two populations, we fitted the model with both sex and population as covariates, and allowed for an interaction effect between sex and population. Since the negative exponential part of the Siler model may have problems in capturing the very steep decline in infant mortality after birth, the mortality at adjacent infant and juvenile ages can be overestimated by models fitted from birth. To evaluate this issue and with the goal of providing the best possible estimates, as well as estimates across the entire life span, we fitted the model from three different starting ages: birth, 0.5 years of age, and 1 year of age. Since the latter was not affected by the constraints of the Siler function we used this model for further investigations.

We predicted mortality rates for each sex and population using the parameter estimates of each MCMC iteration after burn-in and thinning. We then used these predictions to calculate the mean and credible intervals of age-specific mortality rates. Since we were *a priori* interested in the effects of population and sex on mortality, we decided against taking a model selection approach and instead calculated Kullback-Leibler (KL) divergences of the mortality parameter posterior densities (Kullback and Leibler 1951, McCulloch 1989, Burnham and Anderson 2001) as de-

scribed in detail in Barthold et al. (in prep.; see Chapter 3). The KL divergence compares two probability density distributions and can be interpreted to measure the amount of information lost when using the second probability density distribution to approximate the first one. After a simple calibration of the KL values (McCulloch 1989), the values range from 0.5 to 1, where a value of 0.5 indicates that the distributions are identical, and 1 that they do not overlap at all.

Mortality measures

We report mortality information as mortality rates, defined as the instantaneous hazard of death, and also known as the force of mortality (see Equation 4.1). From the continuous age-specific mortality rate, we can calculate the discrete age-specific probability of survival ${}_{\Delta x}\mathrm{p_x}$ (the probability to survive from age x to age $x + \Delta x$), with $\Delta x = 1$ for annual survival (Methods S1, supplementary information). Survival probabilities are a common mortality measure in capture-recapture studies, where they are also termed survival rate. The probability of dying between age x and age $x + \Delta x$ is consequently ${}_{\Delta x}q_x = 1 - {}_{\Delta x}p_x$. We also calculated life expectancy at the model start age ρ as $e_\rho = S(\rho) \int_\rho^\omega S(x)\, dx$, with ω being the highest possible age. We also computed the PDFs for age at death for males and females in the Serengeti and Hwange (see Equation 4.2c). All analyses were conducted using the statistical programming language R (R Core Team 2012).

Results

Population differences in mortality

The models converged for all estimated parameters and all starting ages (Figure 4.1, see also Figure S1 for traces). However, the posterior distributions for Hwange were wider than those for the Serengeti, which was expected due to the smaller sample size of the Hwange data (Figure 4.1, results pertain to the model fitted from 1 year of

age). This is also reflected in the wider confidence bands around the mean estimated mortality rates for Hwange compared to the Serengeti (Figure 4.2).

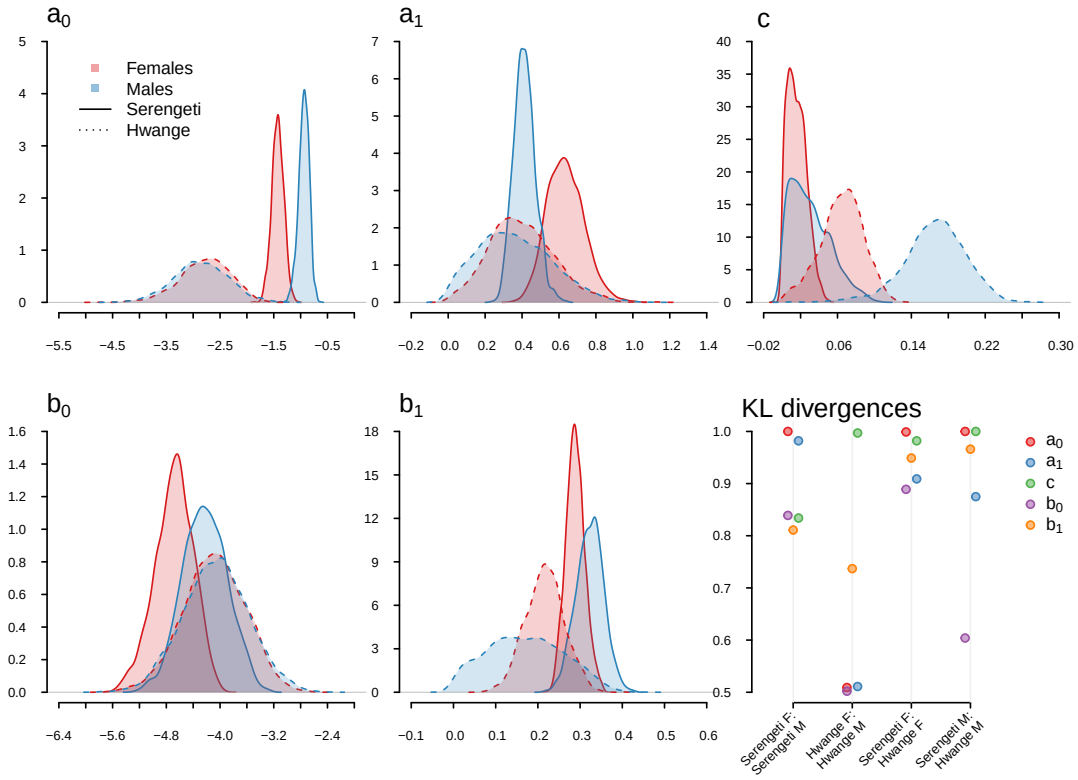

Figure 4.1. Posterior distributions of Siler parameter estimates (a_0, b_0, c, a_1, b_1) for female (pink) and male (blue) African lions of the Hwange (dashed lines) and Serengeti (solid lines) populations, and Kullback-Leibler divergences (KL divergence) comparing parameter posteriors between females (F) and males (M) within populations, and within sexes between the populations. Note that the KL divergence estimates are jittered in x-axis direction to improve visibility. The analysis was conditioned on survival to the first year of life.

Overall mortality of both sexes was U-shaped in the Serengeti with high initial cub mortality, low mortality of prime-aged adults, and an age-dependent increase in mortality during the older ages (Figure 4.2, left panels). Mortality of the Hwange lions also showed higher senescent than prime-age mortality, although the difference was less pronounced than in the Serengeti population (Figure 4.2, right panels). The main difference in overall mortality between the two populations was that, in Hwange, we could only detect moderately elevated levels of cub mortality compared to the mortality of prime-aged adults. Furthermore, this result only held for the

model fitted to the whole data set (i.e., when mortality was estimated from birth) (Figure 4.2, upper right panel).

The KL divergences comparing Serengeti and Hwange females revealed that mortality of females differed between the two populations (Figure 4.1, lower right panel). Females in the Serengeti had higher initial cub mortality (a_0) and a steeper decline in mortality over infant and juvenile ages (a_1), yet lower levels of prime-adult mortality, and higher levels of senescent mortality, when compared to females in Hwange (b_0, and b_1). Due to higher cub mortality, they also had a shorter life expectancy at birth (or 0.5 years and 1 year of age where applicable) (Figure 4.2). However, the clearly hump-shaped PDF for Serengeti females revealed that, once they reached maturity, a larger proportion of deaths occurred at older ages compared to Hwange females.

As with females, the mortality of Serengeti males differed from the mortality of Hwange males (Figure 4.1). The differences in the parameters were again in opposing directions and thus partly compensating each other. Initial cub mortality and the increase in senescent mortality (a_0, b_1) were lower in Hwange, yet age-independent mortality (c) was much higher, resulting in slightly higher mean mortality rates across the prime-adult ages for Hwange males compared to Serengeti males (Figures 4.2, 4.1). The PDFs for age at dispersal for males are illustrated in Figure S2.

Sex differences in mortality

Mortality also varied between the sexes. In the Serengeti population, mortality of males was higher than mortality of females across all ages (Figure 4.2, left panels). There was a slight overlapping of confidence bands of male and female cub mortality in the models fitted from birth and from 0.5 years of age (Figure 4.2, upper and middle right panels). However, this result may stem from the imputation of sex as a latent state for the many unsexed individuals (Table S1), which

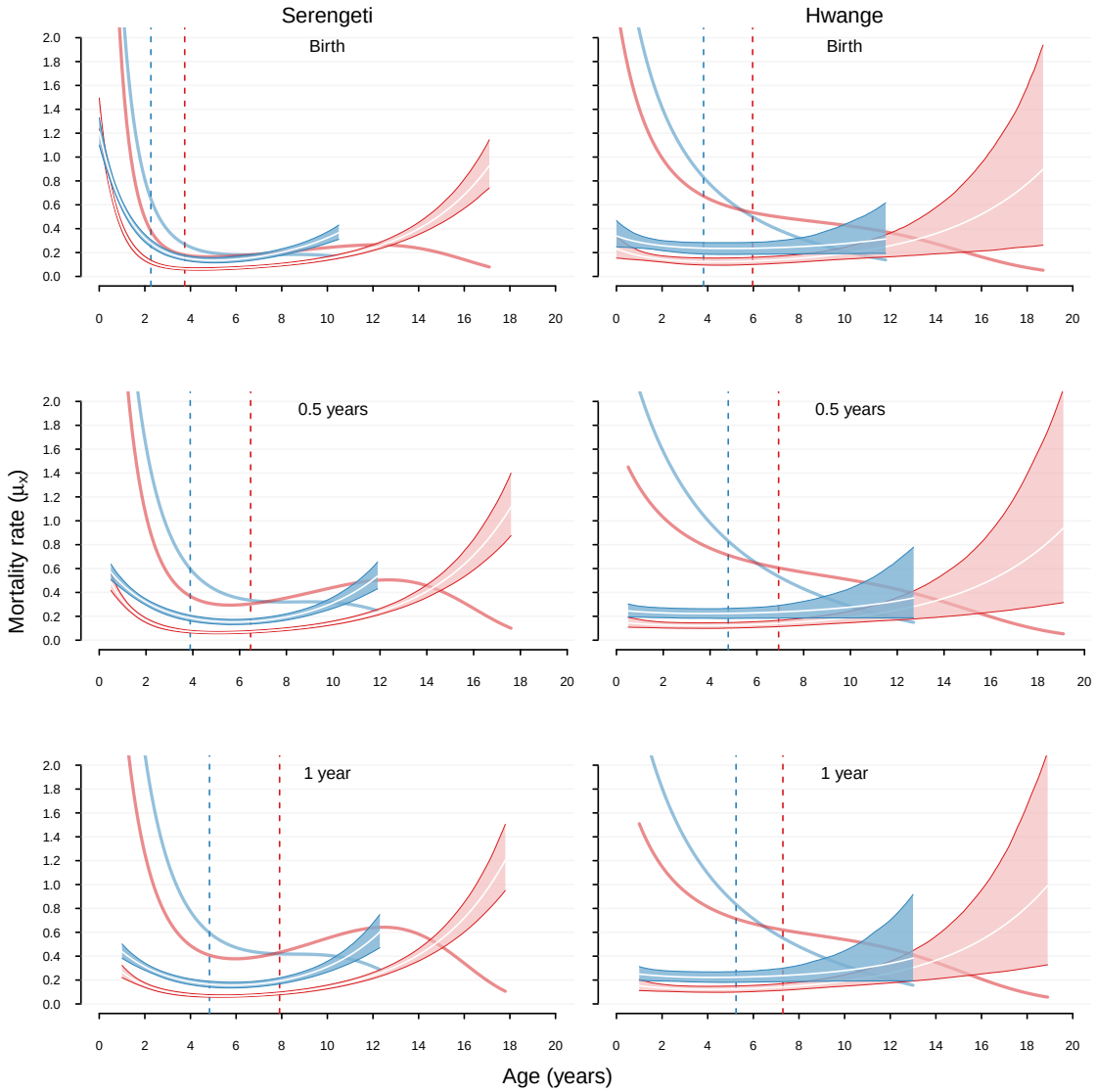

Figure 4.2. Age-specific mortality estimates for male (blue lines and polygons) and female African lions (pink lines and polygons) of the Serengeti population (left panels) and the Hwange population (right panels). Polygons represent 95 % credible intervals of age-specific mortality rates with white lines indicating the mean. Solid lines indicate the probability density function for age at death (PDF), scaled so that the areas under the curves are equal. PDFs are further multiplied with a scaling factor of 100 to improve visibility (The dashed lines indicate life expectancy at birth. Mortality rates and PDFs are plotted until the ages when 95 % of a synthetic same-sex cohort would be dead. The first row of panels shows results of the model fitted from birth. The second and third row show results of models fitted to individuals that died or disappeared at ages older than 0.5 and 1 year, respectively.

increased uncertainty. The sex difference in mortality is also reflected in the KL divergences (Figure 4.1, lower right panel). In Serengeti, males and females differed in almost all parameters, with the parameter distributions governing early-life mortality overlapping the least. In Hwange, males also had higher mortality than females. However, the difference in mortality was almost entirely due to the c parameter, which was strikingly higher for males than for females. This indicates a source of age-independent mortality that was sex-selective. Furthermore, the increase in senescent mortality was slightly less steep for males than for females (b_1), which contributed, together with the small sample size, to an overlapping of confidence bands for male and female mortality at senescent ages. Due to the higher male than female mortality in both populations, life expectancy at birth, or at 0.5 year and 1 year of age for the models fitted from these respective ages, was lower for males than for females in both populations (Figure 4.2, dashed lines). Consequently, the mean PDFs for age at death showed that more male than female deaths occurred up to the age of about 7 years of age in the Serengeti and about 6 years of age in Hwange (Figure 4.1, solid lines).

Discussion

The age trajectories of lion mortality rates, particularly for males and for populations disturbed by humans, are a missing piece of information for developing demographic models to inform population management and for advancing our knowledge on the ecology of this iconic species. Here we found that, contrary to our expectations, lions in the disturbed Hwange population outlived lions in the undisturbed Serengeti population by an average of two years, despite Hwange's history of lions being killed in accidents, for trophies, and as retaliation for raided livestock (Loveridge et al. 2007). Although adult mortality was, as predicted, higher in Hwange, the difference was too small to compensate for the much lower mortality of cubs in

Hwange compared to the Serengeti. Despite this variation in mortality between the populations, the sex difference in mortality was apparent in both populations. Conditioned on survival to 1 year of age, Serengeti females lived on average three years longer than their male counterparts, while Hwange females had a two-year survival advantage over males. In the following, we will discuss the population and sex differences in mortality in more detail.

Differences in adult mortality

Our finding that adult mortality was only slightly higher for the Hwange population when compared to the presumably undisturbed Serengeti population is surprising since Hwange lions suffered from high levels of anthropogenic mortality during parts of the study period (Loveridge et al. 2007). Mean adult life expectancy at maturity of Hwange females (defined as 2.5 years of age) was only 18 % lower than that of Serengeti females (6.07 vs. 7.39 years), and that of Hwange males was even only 5 % lower than that of Serengeti males (4.23 vs. 4.46 years). Despite these similar adult life spans, the parameters of the fitted Siler models revealed that both populations differ in the composition of adult mortality rates. The Serengeti population has low age-independent mortality (c parameters) and larger increases of mortality with age (b_1). Whereas the Hwange population experiences high levels of age-independent mortality with Hwange males having the highest levels of the study (c). This finding is in line with the Hwange population experiencing external sources of mortality, and may likely reflect, particularly for males, elevated mortality due to the population acting as a source population for surrounding hunting areas.

But why is the difference in adult mortality between the undisturbed Serengeti population and the disturbed Hwange population not larger? The seemingly trivial answer is that mortality in the Serengeti is higher, and mortality in Hwange lower than we expected without taking density dependence into account. Since the start of the Serengeti study, the population has been growing due to a long-term in-

crease in prey availability. However, growth did not occur continuously but through step-wise increases in mean population sizes, which remained stable across multiple years despite large year-to-year variation (Packer 2005). This pattern of population growth is created by the dynamics of between- and within-group competition. Only when an exceptionally large cohort of cubs recruits to a pride can a large enough fraction of the pride split off to successfully compete with other prides for space. In years in which these conditions are not met, survival may be density-dependent due to within-group competition (Packer et al. 2001, Mosser et al. 2009). Density-dependent survival has indirectly been observed for lions in Kruger National Park, where lion density was positively associated with prey biomass density. With increasing prey biomass, survival first increased and then decreased, indicating that survival declines at greater lion densities (Ferreira and Funston 2010). Over the past 15 years the Serengeti population size has remained stable (C. Packer, unpublished data), which suggests that the population may have reached carrying capacity. Therefore, density dependence, together with observed epizootic diseases, may result in mortality being relatively high across the whole study period despite the observed long-term population growth and the absence of human impact (Packer et al. 1999, Packer 2005).

In comparison, the Hwange population has, due to conservation measures, increased by 46 % since 2000, with a 200 % increase in the number of adult males since 2004 (A. Loveridge, unpublished data), when the sex ratio in the population was heavily skewed towards females through trophy hunting (Loveridge et al. 2007). Since 2004, the trophy hunting regime has changed markedly with smaller quotas now in place. However, other sources of anthropogenic mortality like poaching and conflict mortality remain unchanged. Anthropogenic mortality certainly increases the Hwange mortality rates, yet anthropogenic mortality may also keep the population at low densities and thus in a perpetual state of density-independent growth with the associated low age-dependent mortality. Therefore, the Serengeti

and Hwange population may experience similar levels of adult mortality due to different mechanisms: density dependence in the Serengeti and anthropogenic mortality in Hwange. These interpretations of the population differences in our estimates of time-invariant mortality remain speculative. They do not actually quantify the effects of particular changes in densities and hunting quotas on mortality. In future work, our model can therefore be extended to include relevant covariates, if sufficient data are available.

Differences in cub mortality

Low density is also the most likely explanation for the observed low cub mortality in Hwange. While in the Hwange study, 74 % of all native-born individuals survive to their first birthday, only 43 % do so in the Serengeti (Table S1). While both estimates fall within the range of previously reported variation in cub survival for other lion populations (Van Orsdol et al. 1985, Becker et al. 2013), the difference is remarkable and causes a considerable difference in life expectancy at birth between the two populations. Cub survival has previously been shown to decrease in the presence of juveniles and sub-adults in the pride, which in turn depends on female density (Packer et al. 2001).

An alternative explanation for the differences in cub mortality is selective appearance of individuals in the study. Cub mortality is highest in the first days of life, yet cubs are only recorded has having been born and died if they were observed after having left the den. It is therefore possible that due to the denser vegetation in Hwange, cubs are older at first sighting there than in the Serengeti, so that in Hwange a larger number of early-occurring deaths are not recorded. However, due to the large number of cubs that would have to be non-detected, this is unlikely to be the whole explanation.

Finally, both studies have the policy of adding three cubs to the data set as having been born and died, when a female shows signs of parturition but has no

cubs. It is possible that there is an interaction between litter size and cub mortality that results in a higher number of these so-called lost litters being reported in the Serengeti than in Hwange. If cub mortality is higher in the Serengeti than in Hwange due to density effects, a Serengeti female is more likely than a Hwange female to emerge from the den without any cubs, and thus to be assigned to have had three cubs. For a Hwange female that has lost cubs during the denning period but has at least one surviving offspring, no lost-litter individuals will be added to the data set. While such an interaction effect is likely to exist, this would not affect the estimates of mortally from 0.5 and 1 year of age that are both still higher in the Serengeti than in Hwange.

Sex differences in mortality

One aim of this study was to provide the first estimates for age-specific male lion mortality for future use in population projections, investigations of the effect of trophy hunting on population sizes, and studies of the evolution of sex differences in mortality. Annual survival has previously been reported to be lower, and lifespan consequently shorter, for males than for females (Orford et al. 1988, Packer et al. 1988, Whitman et al. 2004), yet no estimates of age-specific mortality for males exist. From comparative work on other species, we expected that male lions live shorter lives and have higher mortality than females across all ages because they are bigger than females and engage in costly male-male combat mating competition (Schaller 1972, Promislow 1992, Clutton-Brock and Isvaran 2007). We could confirm this for both study populations. The sex difference in life expectancy is remarkably similar in both populations despite the variation in mortality between the two populations. With increasing availability of comparative data, it will be left to future enquiry to test whether evolutionary or ecological constraints stabilise sex differences across populations.

Conclusion

Despite the fact that age-specific mortality is an important quantity for ecological and evolutionary research, mortality estimates for the dispersing sex are commonly not available. Our study has yielded mortality estimates for both sexes from two lion populations that experience varying environments, densities, and exposures to human impact. They can be used to parameterise demographic models aimed to inform population management measures, similar to the commonly used estimates for females published in Packer et al. (1998). The Sergengeti population may be taken as an archetype for a high-density population that is largely unaffected by humans, while the Hwange population may represent a population that is held at low densities by high anthropogenic mortality. The comparison between the two populations has raised the question of whether density dependent effects on mortality need to be incorporated into models that estimate sustainable male offtake quotas, because low male and overall densities may be associated with low cub mortality. Since, until it was thought that high adult male mortality increases cub mortality due to an increased rate of pride take-overs and subsequent infanticides (Whitman et al. 2004, Caro et al. 2009), a large comparative study investigating the interaction of density and infanticide effects may be called for.

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Supporting Information

Figure S1 Traces of mortality and dispersal parameter estimation (model fitted from 1 year of age).

Figure S2 PDF for age at dispersal (model fitted from 1 year of age).

Table S1 Data summary and sex ratios at model start ages.

Table S2 Estimated Siler model coefficients for the models fitted form birth, 0.5 years, and 1 year of age, respectively.

Methods S1 Method to convert continuous mortality rate into discrete probabilities of survival.

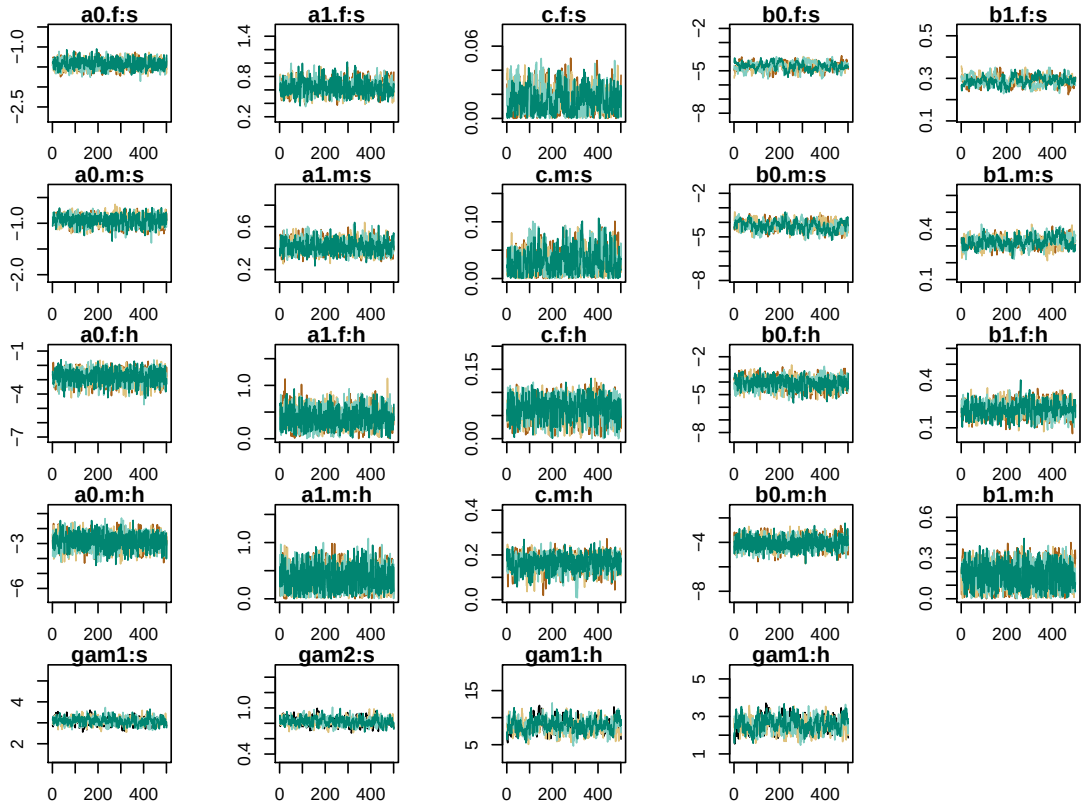

Figure S1. Trace plots for four parallel runs for the Serengeti and Hwange lion mortality analysis. Estimated parameters are the Siler parameters (a_0, b_0, c, a_1, b_1; f denotes estimates for females and m for males; s denotes Serengeti and h denotes Hwange) and Gamma parameters (shape and rate; gam1 and gam2).

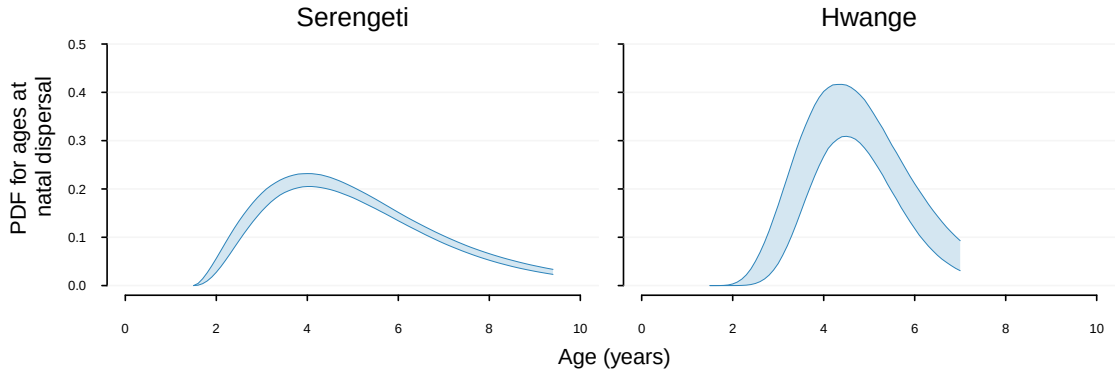

Figure S2. PDF for age at dispersal for Serengeti and Hwange male lions (model fitted from 1 year of age). Polygons represent 95 % credible intervals.

Table S1. Sample sizes for males (M), females (F), and individuals that died before sex could be determined (U).

	Serengeti			***Hwange***			***Sex ratio***
Sample	**M**	**F**	**U**	**M**	**F**	**U**	**F:M**
Birth[†]	1466(316)[*]	1507	875	179(32)	247	144	0.55[**]
0.5	988(315)	1095	62	173(32)	237	58	0.53
1	763(315)	905	4	160(32)	228	31	0.55

[†] "Birth" indicates the sample that includes all individuals. "0.5" and "1" indicate the samples that include individuals that survived to at least 0.5 years and 1 year, respectively.

[*] For males sample sizes refer to the number of native-born individuals, followed by the number of immigrants in brackets.

[**] Female to male sex ratio among all native-borns (pooled data, excluding immigrants) assuming a sex ratio of 0.5 among individuals that died before sex could be determined.

Table S1: Estimated Siler model coefficients for the models fitted form birth, 0.5 years, and 1 year of age, respectively (mean, SE, and credible intervals of the parameter posterior distributions).

			Coefficient	Mean	SE	2.5 %	97.5 %
From birth	Serengeti	Females	a_0	0.182	0.051	0.084	0.282
			a_1	1.194	0.074	1.062	1.339
			c	0.030	0.010	0.009	0.048
			b_0	-4.976	0.350	-5.611	-4.292
			b_1	0.285	0.025	0.235	0.330
		Males	a_0	0.218	0.043	0.133	0.299
			a_1	0.836	0.072	0.708	0.989
			c	0.052	0.025	0.006	0.101
			b_0	-4.219	0.391	-4.963	-3.527
			b_1	0.291	0.034	0.228	0.358
			gam_1	2.982	0.158	2.677	3.300
			gam_2	0.778	0.046	0.691	0.872
	Hwange	Females	a_0	-1.983	0.382	-2.861	-1.366
			a_1	0.507	0.163	0.223	0.858
			c	0.064	0.027	0.011	0.114
			b_0	-4.176	0.455	-5.105	-3.344
			b_1	0.204	0.044	0.115	0.289
		Males	a_0	-2.131	0.537	-3.296	-1.232
			a_1	0.49	0.182	0.158	0.884
			c	0.186	0.040	0.094	0.253
			b_0	-4.116	0.489	-5.078	-3.172
			b_1	0.133	0.084	0.007	0.306
			gam_1	7.073	0.973	5.13	8.998
			gam_2	1.739	0.262	1.225	2.238
From 0.5 years of age	Serengeti	Females	a_0	-0.786	0.080	-0.942	-0.636
			a_1	0.826	0.091	0.662	1.010
			c	0.019	0.011	0.002	0.041
			b_0	-4.774	0.320	-5.472	-4.233
			b_1	0.283	0.023	0.242	0.335
		Males	a_0	-0.643	0.072	-0.791	-0.508
			a_1	0.440	0.053	0.344	0.549
			c	0.027	0.019	0.001	0.073
			b_0	-4.337	0.347	-5.000	-3.703
			b_1	0.321	0.033	0.258	0.383
			gam_1	3.027	0.164	2.709	3.350
			gam_2	0.781	0.048	0.689	0.876
	Hwange	Females	a_0	-2.880	0.448	-3.850	-2.076
			a_1	0.359	0.166	0.065	0.705
			c	0.067	0.022	0.017	0.107
			b_0	-4.147	0.450	-5.047	-3.277
			b_1	0.208	0.045	0.116	0.293

Table S1 continued.

	Hwange	Males	a_0	-2.946	0.491	-3.897	-2.037
			a_1	0.328	0.194	0.027	0.746
			c	0.169	0.032	0.102	0.228
			b_0	-4.041	0.480	-5.009	-3.118
			b_1	0.155	0.083	0.008	0.314
			gam_1	7.020	1.069	5.077	9.279
			gam_2	1.730	0.285	1.196	2.329
From 1 year of age	Serengeti	Females	Coefficient	Mean	SE	2.5 %	97.5 %
			a_0	-1.432	0.110	-1.653	-1.227
			a_1	0.631	0.102	0.456	0.845
			c	0.017	0.010	0.001	0.040
			b_0	-4.718	0.300	-5.341	-4.184
			b_1	0.291	0.023	0.249	0.340
		Males	a_0	-0.941	0.100	-1.153	-0.760
			a_1	0.407	0.061	0.300	0.539
			c	0.031	0.022	0.001	0.083
			b_0	-4.264	0.343	-4.966	-3.639
			b_1	0.326	0.034	0.260	0.396
			gam_1	3.024	0.156	2.731	3.347
			gam_2	0.777	0.046	0.691	0.869
	Hwange	Females	a_0	-2.756	0.448	-3.714	-1.967
			a_1	0.410	0.177	0.091	0.773
			c	0.065	0.023	0.020	0.107
			b_0	-4.119	0.439	-4.956	-3.273
			b_1	0.219	0.045	0.127	0.304
		Males	a_0	-2.888	0.504	-3.911	-1.925
			a_1	0.352	0.195	0.033	0.772
			c	0.168	0.034	0.093	0.230
			b_0	-4.060	0.508	-5.055	-3.084
			b_1	0.166	0.088	0.014	0.333
			gam_1	6.884	1.019	4.989	8.905
			gam_2	1.689	0.270	1.189	2.232

Methods S1: Conversion of continuous mortality rates into discrete probabilities of survival

The probability to survive from age x to age $x+\Delta x$, with $\Delta x = 1$ for annual survival can be calculated from the mortality rate as

$$\begin{aligned} {}_{\Delta x}p_x &= \exp[-\textstyle\int_x^{\Delta x} \mu(z)\mathrm{d}z] \\ &= \exp\Big(-[H(x+\Delta x) - H(x)]\Big), \end{aligned} \tag{S-1}$$

with $H(x)$ being the cumulative hazard function $H(x) = \int_0^x \mu(z)\mathrm{d}z$. The exact expression for the Siler model is

$$\begin{aligned} {}_{\Delta x}p_x = \quad \exp\Bigg(-\Big(\Big[\tfrac{e^{a_0}}{a_1}\left(1-e^{-a_1(x+\Delta x)}\right) + c(x+\Delta x) + \tfrac{e^{b_0}}{b_1}\left(e^{b_1(x+\Delta x)}-1\right)\Big] \\ -\Big[\tfrac{e^{a_0}}{a_1}\left(1-e^{-a_1 x}\right) + cx + \tfrac{e^{b_0}}{b_1}\left(e^{b_1 x}-1\right)\Big]\Big)\Bigg). \end{aligned} \tag{S-2}$$

Alternatively, the probability to survive can be approximated as

$${}_{\Delta x}p_x \approx \frac{1}{2}\left(\exp(-\mu(x)) + \exp(-\mu(x+\Delta x))\right). \tag{S-3}$$

Evidently, the probability to die between age x and age $x+\Delta x$ is

$${}_{\Delta x}q_x = 1 - {}_{\Delta x}p_x. \tag{S-4}$$

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CHAPTER 5

The effects of phenotypic selection and phenotypic transition on biometric heritability for iteroparous long-lived species

Running head

Demographic determinants of heritability

Title

The effects of phenotypic selection and phenotypic transition on biometric heritability for iteroparous long-lived species

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Abstract

Biometric heritability measures the covariation between parents and their offspring in a phenotype, where the phenotype is measured at the same age in both generations. How this covariation arises is not clear for iteroparous species in which the phenotype develops with age. A cohort is born, varying in birth phenotype. These potential parents may survive, and if they survive their phenotype develops. At various points throughout their life they may produce offspring. Both their probability of survival and reproduction may depend on their phenotype and age. Also dependent on these characteristics may be the birth phenotype of offspring. But which of these processes then determine the phenotypic covariation between parents and offspring—differential survival, differential fertility, the development of the phenotype with age, or the transition of phenotype from parents to offspring? We aim to answer this question by studying the temporal dynamics of phenotype distributions of two iteroparous long-lived species in an IPM framework. We find that all four processes influence heritability. Generally, processes that result in potential parents surviving and successfully reproducing, regardless of their birth phenotype, reduce heritability. We conclude that identifying the demographic determinants of heritability will ultimately contribute to elucidating microevolutionary processes in free-living populations.

Key words

heritability, parent-offspring regression, phenotypic variance, parent-offspring covariance, integral projection model, IPM, stage structured population dynamics, Soay sheep, roe deer, microevolution

Introduction

The heritability of a quantitative character determines how the mean additive genetic variance of the character in a population shifts over generations in response to natural or artificial selection. Heritability is therefore a key concept in evolutionary biology, agriculture, and medicine. It is defined as the proportion of phenotypic variance attributable to additive genetic variance (Falconer and Mackay 1996, Roff 1997, Lynch and Walsh 1998). Statistical methods to estimate heritability decompose phenotypic variance into an additive genetic component and other components, based on the expected proportion of shared genes among relatives (Wilson et al. 2010). The classical method estimates heritability as the slope of the regression of offspring phenotype on mid-parent phenotype, where parent and offspring characters are measured at the same age (Kempthorne 1957). A derived method, the 'animal model', uses the same logic but increases statistical power by incorporating pedigree information in a mixed-effects model (Kruuk 2004). This statistical framework also allows unbalanced sampling designs and confounding factors such as shared environments or maternal effects (Kruuk 2004, Wilson et al. 2010). Consequently, the animal model has been used to estimate heritability of a wide variety of traits for wild populations (e.g., Charmantier et al. 2007, Nilsson et al. 2009, Morales et al. 2010). While these methods appropriately use the phenotypic covariation among relatives to estimate heritability, it is not clear how this covariation arises for phenotypes that change with age. This is not a straightforward question, and is what we ask here.

Let us consider, for example, the phenotypic covariance between parental and offspring birth weight. First, the parental cohort is born, and each individuals has a birth weight. These individuals may survive and if they do, they grow. At points throughout their lives they may reproduce. The offspring they produce have their own birth weights, which may be a function of the environment, parental age, and

parental body weight. These are factors that statistical analyses of heritability routinely correct for (Wilson et al. 2010). But which process is most likely to influence the phenotypic parent-offspring covariance—differential survival, differential fertility, variation in growth rates, or the relationship between maternal weight and offspring birth weight?

Recently, Coulson et al. (2010) showed how heritability can be calculated from an integral projection model (IPM). IPMs are discrete time structured models that describe the temporal dynamics of the density distribution of a quantitative character like size or body weight (Easterling et al. 2000). At each time step, four functions determine how the phenotype distribution changes. First, the survival function determines whether individuals of a given age and phenotype survive. Second, the development function determines how the phenotype of survivors develops. Third, the recruitment function adds offspring to the phenotype distribution according to the parental phenotype and age distributions. And fourth, the inheritance function determines the offspring birth phenotype depending on the parental phenotype and age distributions. Since IPMs decompose the temporal dynamics of the phenotype distribution into these four processes—survival, fertility, within-individual phenotypic development, and parent-offspring phenotypic transition—IPMs can be used to study how they affect heritability.

Heritability of a character can be estimated from a deterministic age phenotype structured IPM as the regression slope of the phenotypes of offspring born to one parent cohort over this cohort's lifetime regressed on the phenotypes of parents at their own birth (Coulson et al. 2010). Multiplying the slope by two yields the heritability when parents of only one sex enter the regression (Falconer and Mackay 1996). Since we can estimate heritability from these models, we can change model parameters to study how heritability is impacted by fertility and viability selection, phenotypic development, and parent-offspring phenotypic transition (Coulson et al. 2010).

Heritability is a complex function of all processes that shape the parent and offspring phenotype distributions. To understand how each process influences heritability requires the careful analysis of phenotype structured models. Such models are now routinely constructed for wild animal populations (Ozgul et al. 2010, Childs et al. 2011, Smallegange et al. 2014). They are usually analysed at equilibrium when the phenotype distribution has stable, time-invariant proportions in each age and phenotype class. This stable phenotype distribution changes if model parameters are altered. As a consequence, changes in model parameters can affect heritability at population equilibirum by affecting one of three phenotype distributions: the stable cohort birth phenotype distribution, the stable parent (at own) birth phenotype distribution, or the offspring birth phenotype distribution.

To understand why, we need to know what these distributions are and how they depend on each other. The offspring produced by the whole stable population in one time step recruits to the population at a stable phenotype distribution, the stable cohort birth phenotype distribution. It describes the cohort of potential parents, individuals that may become parents if they survive and reproduce. Which of these potential parents reproduces, and how often, during their lifetimes determines the stable parent (at own) birth phenotype distribution. It is shifted to higher phenotype values when compared to the stable cohort birth phenotype distribution, if larger individuals have higher probabilities of survival and reproduction. Finally, the offspring born to one cohort of parents over their lifetimes also recruit to the population at a stable phenotype distribution, the offspring birth phenotype distribution. While heritability is a function of the stable parent and offspring birth phenotype distributions, these two distributions, and therefore heritability, also change when the stable cohort birth phenotype distribution changes.

In order to separate this cascading effect of changing the stable cohort birth phenotype distribution from any direct effect of changing model parameters on the parent and offspring birth phenotype distributions, Coulson et al. (in prep.; see

Appendix) have recently analysed an IPM where the cohort birth phenotype distribution did not change with changes to the phenotypic transition parameters. While these simple models provided useful insights, they are biologically unrealistic and it remains open how heritability responds to changes in all model parameters in IPMs for iteroparous long-lived species with stable phenotype distributions.

To study this, we analyse two published age and body mass structured IPMs, one parameterised for Soay Sheep (*Ovis aries*) (Coulson et al. 2010) and one for roe deer (*Capreolus capreolus*) (Plard et al. in review). As an alternative to the parent-offspring regression approach as described in Coulson et al. (2010), and the classical perturbation analysis by simulations, we derive how to calculate biometric heritability from an age phenotype structured IPM and the precise expressions to compute the elasticity of heritability to changes in the model parameter. We report and compare heritability of body mass for both species and show that heritability can better be understood by taking into account those processes that influence the phenotype distributions.

Methods

The **biometric heritability** h^2 of a phenotypic trait is the slope of the regression of offspring phenotype values at age a Y ('offspring phenotype') on mid-parent phenotype values at age a X_0 ('parent phenotype') (Jacquard 1983). We work with traits at birth here, but our approach can be used for traits measured at any age. The heritability can be calculated using the formula for a regression slope

$$b = \frac{Cov(YX_0)}{Var(X_0)}. \tag{5.1}$$

If parents of only one sex are considered, the heritability h^2 equals twice the slope b, since one parent only contributes half of the offspring genome (Falconer and Mackay 1996). From (5.1), it follows that the **elasticity** of the biometric heritability, defined

as the proportional change in b, is

$$E_b = \frac{\Delta b}{b} = \frac{\Delta \mathrm{Cov}(Y X_0)}{\mathrm{Cov}(Y X_0)} - \frac{\Delta \mathrm{Var}(X_0)}{\mathrm{Var}(X_0)}. \tag{5.2}$$

The **sensitivity** of heritability S_b, or the absolute change in b, is of course $S_b = b\, E_b$. The precise forms of the elasticity and sensitivity of heritability E_b and S_b depend on which model parameters are changed. The quantities in (5.1) and (5.2) can be derived for an age phenotype structured population model, where phenotype is a quantitative character whose phenotypic values are discrete elements of the vector $\mathbf{z} = \{z_i\}$. In the following, we describe how the key quantities for calculating the elasticity of heritability can be computed. Detailed derivations of the described quantities are given in the supporting information.

Stable cohort birth phenotype distribution

The age and phenotype structure of a population that experiences time-invariant fertility and mortality rates converges to a stable age and phenotype distribution with a growth rate r (Keyfitz and Caswell 2005). Stable populations have therefore stable proportions of newborns in each phenotype class, here represented by a vector $\mathbf{u} = \{u(i)\} \equiv \{u(z_i)\}$ and referred to as the **stable cohort birth phenotype distribution**. Note that we set $(\mathbf{e}^T\, \mathbf{u}) = 1$, using the vector $\mathbf{e}^T = (1, 1, \ldots, 1)$. Furthermore, in the age phenotype structured model framework, 'birth' refers to the age of recruitment to the population (i.e., age at first census). If we define for any r a renewal matrix

$$\mathbf{A}(r) = \sum_a e^{-ra}\, \mathbf{F}_a\, \mathbf{L}_a\,, \tag{5.3}$$

where $\mathbf{F}_a$ is a matrix describing fertility for parents of age a, and $\mathbf{L}_a$ is a matrix describing survivorship from birth to age a (both matrices are described in detail in the next section), then the stable cohort birth phenotype distribution $\mathbf{u}$ and growth

rate r are together determined by

$$\mathbf{A}(r)\,\mathbf{u} = \mathbf{u}. \tag{5.4}$$

For details on the renewal matrix and the stable cohort birth phenotype distributions see Steiner et al. (2014).

Phenotype-demography matrices

Fertility of parents aged a is a matrix $\mathbf{F}_a = \mathbf{D}_a\widehat{\mathbf{M}}_a$, where $\widehat{\mathbf{M}}_a$ is a diagonal matrix whose (i,i) entry is the total number of offspring of a parent of phenotype z_i will produce at age a. The **inheritance** of the phenotype from a parent of phenotype z_i at age a to offspring is captured in the parent-offspring phenotypic transition matrix $\mathbf{D}_a$ whose (i,j) element is

$$\begin{aligned} D_a(i,z) &\equiv D_a(z_i \mid z_j) \\ &= \text{Prob.}[\text{Offspring phenotype is } z_i \mid \text{Parental phenotype is } z_j \text{ at age } a]. \end{aligned} \tag{5.5}$$

Note that $\mathbf{e}^T\,\mathbf{D}_a = \mathbf{e}^T$ so that the probabilities for the transitions out of one parental phenotype z_j sum to one.

Survival and growth together from age a to age $a+1$ are given by a matrix $\mathbf{P}_a$ whose (i,j) element is

$$\begin{aligned} P_a(i,z) &\equiv P_a(z_i \mid z_j) \\ &= \text{Prob.}[\text{Alive at age } a+1 \text{ with phenotype } z_i \mid \text{Alive at age } a \text{ with } z_j]. \end{aligned} \tag{5.6}$$

Survivorship from birth at age $a = 1$ to any age a is a matrix $\mathbf{L}_a$ whose (i, j) element is

$$L_a(i,j) \equiv L_a(z_i \mid z_j) \tag{5.7}$$
$$= \text{Prob.[Alive at age } a \text{ with phenotype } z_i \mid \text{Born age 1 with } z_j].$$

Parent-offspring covariance and parental variance

In the supplementary material (SI.5.1) we derive a formula for the covariance between offspring phenotype Y and parent birth phenotype X that is computable in terms of $\mathbf{F}_a, \mathbf{L}_a$, and $\mathbf{u}$, by first deriving the same covariance using the more familiar approach of estimating heritability from regression models. We then use the same conceptual approach to derive the covariance computable for an age phenotype structured model. In the main methods, we provide all quantities necessary to calculate the elasticities of heritability with respect to the phenotype selection and phenotype transition processes.

In a stable age phenotype structured population, the **lifetime number of offspring** produced by a birth cohort that has the stable cohort birth phenotype distribution $\mathbf{u}$ is

$$K = \sum_a (\mathbf{e}^T \mathbf{F}_a \mathbf{L}_a \, \mathbf{u}) = \sum_a (\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \, \mathbf{u}). \tag{5.8}$$

Using this, the phenotype-demography matrices $\mathbf{F}_a$, $\widehat{\mathbf{M}}_a$, and $\mathbf{L}_a$, and the stable cohort birth phenotype distribution $\mathbf{u}$ as defined in (5.4), the **parent-offspring covariance** between offspring and parent birth phenotypes is given by

$$\mathrm{Cov}(Y X_0) = \frac{1}{K} \sum_a \left\{ \mathbf{e}^T \, \widehat{\mathbf{Z}} \, \mathbf{F}_a \mathbf{L}_a \left(\mathbf{I} - \mathbf{u} \, \mathbf{e}^T \, \frac{1}{K} \sum_a \widehat{\mathbf{M}}_a \, \mathbf{L}_a \right) \widehat{\mathbf{Z}} \, \mathbf{u} \right\}, \tag{5.9}$$
$$= \mathbf{e}^T \, \widehat{\mathbf{Z}} \, \mathcal{H} \left(\mathbf{I} - \mathbf{u} \, \mathbf{e}^T \, \frac{1}{K} \sum_a \widehat{\mathbf{M}}_a \, \mathbf{L}_a \right) \widehat{\mathbf{Z}} \, \mathbf{u}, \tag{5.10}$$

where $\hat{\mathbf{Z}}$ is a diagonal matrix of the phenotypic values $\hat{\mathbf{Z}} = diag(z_i)$, and where we use, for brevity,

$$\mathcal{H} = \frac{1}{K} \sum_a \mathbf{F}_a \mathbf{L}_a. \tag{5.11}$$

Details on the derivations are given in the supplementary material (SI.5.2), particularly in the equations (S-14 to S-17).

The **variance in parent birth phenotype** is

$$\mathrm{Var}(X_0) = \frac{1}{K} \sum_a (\mathbf{e}^T \widehat{\mathbf{M}}_a \, \mathbf{L}_a \, \widehat{\mathbf{Z}} \, \widehat{\mathbf{Z}} \mathbf{u}) - \frac{1}{K^2} (\sum_a \mathbf{e}^T \widehat{\mathbf{M}}_a \, \mathbf{L}_a \, \widehat{\mathbf{Z}} \mathbf{u})^2. \tag{5.12}$$

Details on the derivation of the variance is given in the supplementary material (SI.5.1), particularly in the equations (S-18 to S-23)

Computing the elasticity of heritability

To compute the elasticity of heritability E_b via (5.2), we first compute for the baseline $\mathbf{F}_a$ and $\mathbf{L}_a$ the stable cohort birth phenotype distribution $\mathbf{u}$ and the biometric heritability b using (5.4), (5.10), and (5.12). A change in biometric heritability Δb occurs if we change the model parameters, that govern the strength of the phenotype selection and transition processes (SI.5.4, SI.5.6), by a small amount $\epsilon > 0$, since this causes changes in the phenotype-demography matrices

$$\mathbf{F}_a \, \mathbf{L}_a \to \mathbf{F}_a \, \mathbf{L}_a + \epsilon \, \mathbf{\Delta}_a. \tag{5.13}$$

The supplementary information SI.5.6, SI.5.7, and SI.5.8 give the details on the computation of the changes in the phenotype-demography matrices with respect to changes in viability selection, fertility selection, phenotype development with age, and the transition of phenotype from parents to offspring.

The changes in (5.13) change the stable population so that the stable cohort

birth phenotype distribution $\mathbf{u}$ changes by $\epsilon\,\boldsymbol{\delta}_u$ (see SI.5.3 for the computation of $\boldsymbol{\delta}_u$). From here on, we leave off the ϵ since it multiplies every change. In addition to changes in $\mathbf{u}$, the parent-offspring covariance $\mathrm{Cov}(YX_0)$ changes by $\Delta\mathrm{Cov}(YX_0)$ and the variance in parent birth phenotype $\mathrm{Var}(X_0)$ by $\Delta\mathrm{Var}(X_0)$ (see SI.5.5 for details on the perturbations of the parent offspring covariance and parent birth phenotype variance).

The covariance as given in (5.10) requires the matrix $\mathcal{H}$ defined in (5.11), which in turn needs K defined in (5.8). From (5.11) see that the change in $\mathcal{H}$ is

$$\Delta\mathcal{H} = -\left(\frac{\Delta_K}{K}\right)\mathcal{H} + \frac{[\sum_a \boldsymbol{\Delta}_a]}{K}. \tag{5.14}$$

And from (5.8) we have

$$\Delta_K = \sum_a (\mathbf{e}^T \boldsymbol{\Delta}_a \mathbf{u}) + \sum_a (\mathbf{e}^T \mathbf{F}_a\, \mathbf{L}_a)\boldsymbol{\delta}_u \tag{5.15}$$

Putting these together, the change in parent-offspring covariance using (5.10) and $\boldsymbol{\delta}_u$ (SI.5.3) is

$$\begin{aligned}
\Delta\mathrm{Cov}(YX_0) = {} & \mathbf{e}^T \widehat{\mathbf{Z}}\,[\Delta\mathcal{H}] \left(\mathbf{I} - \mathbf{u}\,\mathbf{e}^T \frac{1}{K} \sum_a \widehat{\mathbf{M}}_a\, \mathbf{L}_a\right) \widehat{\mathbf{Z}}\,\mathbf{u} \\
& + \mathbf{e}^T \widehat{\mathbf{Z}}\,\mathcal{H} \left(\mathbf{I} - \mathbf{u}\,\mathbf{e}^T \frac{1}{K} \sum_a \widehat{\mathbf{M}}_a\, \mathbf{L}_a\right) \widehat{\mathbf{Z}}\,\boldsymbol{\delta}_u \\
& - \mathbf{e}^T \widehat{\mathbf{Z}}\,\mathcal{H} \left(\frac{1}{K} \sum_a \mathbf{u}\,\mathbf{e}^T \Delta_{\widehat{\mathbf{M}}_a \mathbf{L}_a} \right. \\
& \left. + \frac{\boldsymbol{\delta}_u\, K - \Delta_K\, \mathbf{u}}{K^2} \sum_a \mathbf{e}^T \widehat{\mathbf{M}}_a\, \mathbf{L}_a\right) \widehat{\mathbf{Z}}\,\mathbf{u}.
\end{aligned} \tag{5.16}$$

Finally, the change in parent birth phenotypic variance is

$$\begin{aligned}\Delta\mathrm{Var}(X_0) = \mathbf{e}^T \sum_a \left\{ \frac{\Delta_{\widehat{\mathbf{M}}_a \mathbf{L}_a} K - \Delta_K \widehat{\mathbf{M}}_a \mathbf{L}_a}{K^2} \widehat{\mathbf{Z}} \widehat{\mathbf{Z}} \mathbf{u} \right\} \\ + \frac{1}{K} \sum_a (\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}} \widehat{\mathbf{Z}} \boldsymbol{\delta}_u) \\ - 2 \frac{\mathbf{e}^T}{K} \sum_a \left\{ \frac{\Delta_{\widehat{\mathbf{M}}_a \mathbf{L}_a} K - \Delta_K \widehat{\mathbf{M}}_a \mathbf{L}_a}{K} \widehat{\mathbf{Z}} \mathbf{u} + \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}} \boldsymbol{\delta}_u \right\} \mathbf{z}^T \mathbf{u}.\end{aligned} \tag{5.17}$$

Using (5.10), (5.12), (5.16), and (5.17), we can now compute the biometric heritability in (5.1) and its elasticity in (5.2).

To validate our analytical framework for computing the elasticities of heritability with respect to changes in the model parameters, we also numerically computed the elasticities from simulations by adding 10^{-5} to each model parameter, one at a time, dividing the resulting change in the perturbed elasticity by the unperturbed elasticity, and then scaling it back up by 10^5. This approach, which we have already briefly mentioned in the introduction, is described in detail in Coulson et al. (2010).

Application to Soay sheep and roe deer IPMs

We computed the biometric heritability of body mass in Soay sheep (*Ovis aries*) and roe deer (*Capreolus capreolus*) and the elasticities of heritability to changes in model parameters for two published age phenotype structured IPMs (Coulson et al. 2010, Plard et al. in review). Both IPMs used data from the female component of the population and categorised those ages with statistically indistinguishable functions into age-classes. The Soay sheep IPM divided individuals into four age-classes: lambs (aged 0), yearlings (aged 1-2), prime-aged adults (aged 2-7), and senescent individuals (aged 8 and older). Whereas the roe deer IPM distinguished three age classes: yearlings (aged 0), prime-aged adults (aged 1-7), and senescent individuals (aged 8 to 11 and older). The parameterisation of the IPM phenotype-demography matrices was carried out by first statistically determining the relevant phenotype-

demography functions using the life history and body mass data while controlling for confounding temporal variation in demographic rates. The functions inferred, by body weight and age class, were yearly probability of survival, yearly probability of reproduction, probability of twinning, mean and variance of annual growth, and mean and variance of parent-offspring body weight transition. Every function has an intercept and a slope per age class. The phenotype-demography matrices are then populated by predicting from the inferred functions. The total number of model parameters is the total number of coefficients of all functions. For a detailed description of the construction of IPMs see Merow et al. (2014) or Rees et al. (2014). The IPMs used here are described in Coulson et al. (2010) and Plard et al. (in review).

We now provide a brief summary of the data, the feral population of Soay sheep (*Ovis aries*) lives on the Island of Hirta in the St. Kilda archipelago, Scotland, and fluctuates as a food-limited population between 600 and 2000 individuals, of which about one-third live in the 250ha study area (Clutton-Brock and Pemberton 2004). The population has been studied in detail since 1985. During this time life history and body mass data were collected during the yearly capture and year-round censuses. A detailed description of the population and data collection protocols is provided elsewhere (Clutton-Brock and Pemberton 2004).

The roe deer population of Trois Fontaines lives in an enclosed forest of 1360 ha in North Eastern France (48°3′N, 2°61′W). The size of the population has been kept relatively constant around 250 individuals by yearly removals (mainly through exportation of captured individuals) except between 2001-2005 when an experimental manipulation of density was performed and population size peaked at 450 individuals. The population has been monitored by the Office National de la Chasse et de la Faune Sauvage since 1975. Each year, half of the population of roe deer is caught between December and March. Each individual is sexed and weighted. All individuals included in this study are of known age. The population and the study

site has been described in detail by Gaillard et al. (1993, 2013).

Results

Biometric heritability measures how similar in phenotype a generation's parents are to their offspring at the same age. This similarity is influenced by phenotypic selection and phenotypic transition. In an IPM for an iteroparous long-lived species, selection is divided into viability and fertility selection, and phenotypic transition is split into within-individual development with age and between-individual transition from parents to offspring (Coulson et al. in prep.; see Appendix). By perturbing these processes in an IPM framework, our analysis revealed how all four processes influence biometric heritability of body mass for Soay sheep and roe deer.

The estimated heritabilities were 0.20 for Soay sheep and 0.34 for roe deer. The elasticities of the heritabilities varied between -6% and 2.5%. A comparison between analytically and numerically calculated elasticities provided a check that both the analytical and numerical approaches provided equivalent results (Figures S1 and S2). In general, perturbing slopes had larger effects on heritability than perturbing intercepts (Figures 5.1 and 5.2). In the following, we will discuss the analytically calculated elasticities with respect to changes in phenotypic selection and phenotypic transition. To understand the elasticities of heritability, we also discuss the underlying elasticities in the means and variances of the three phenotype distributions that contribute towards heritability: the stable cohort birth phenotype distribution, the parent birth phenotype distribution, and the offspring birth phenotype distribution (Tables 5.1, S1, and S2).

Elasticities of heritability with respect to viability selection

Increasing the slopes of the survival function and thus increasing survival and altering viability selection resulted in large negative elasticities of heritability for both

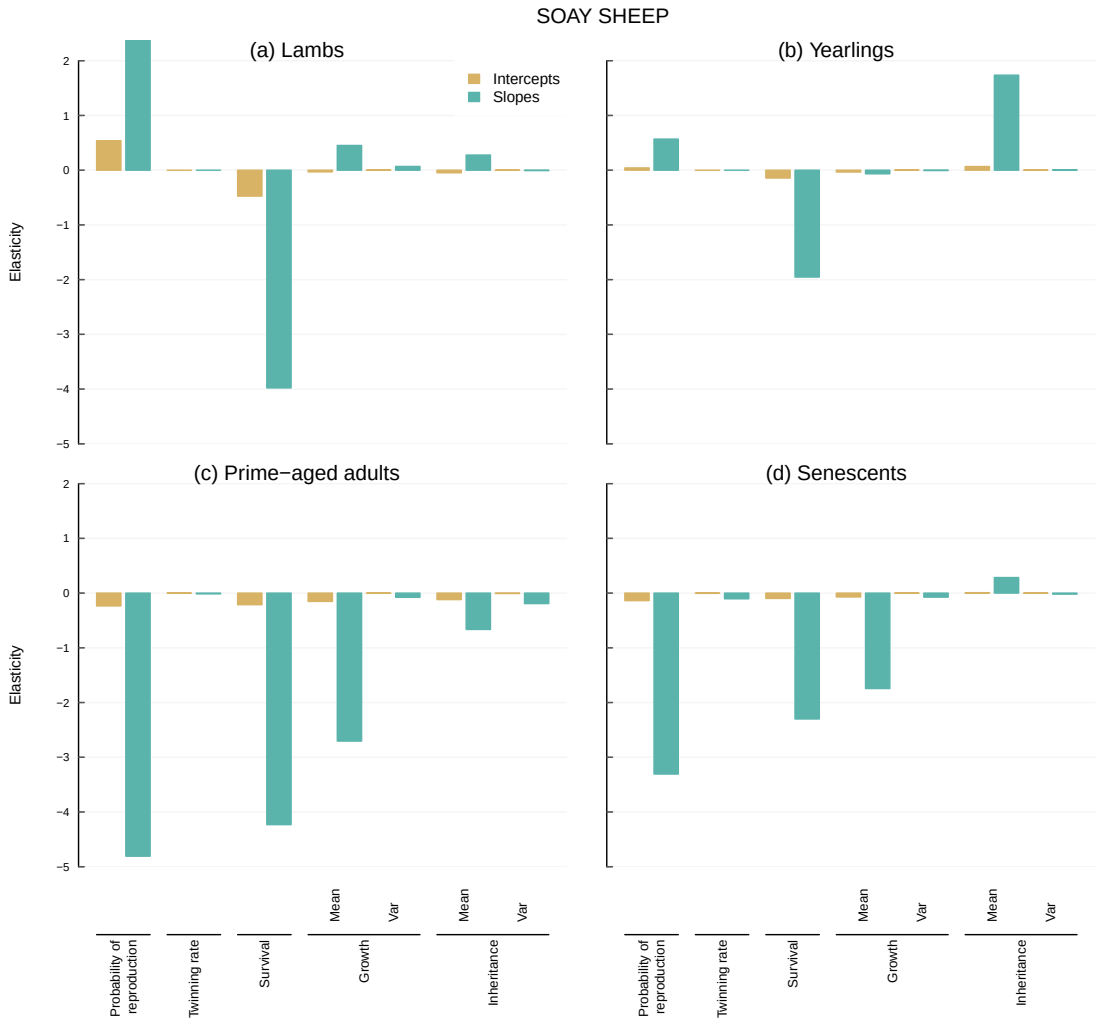

Figure 5.1. The analytically derived elasticities of heritability with respect to changes in the model parameters of the Soay sheep IPM.

species and almost all age classes (Figures 5.1 and 5.2). Changes to the slopes for the adult age classes had the most pronounced effects. Increasing these slopes increased survival across all phenotype values but increased survival of small adults more than survival of large adults. This is because the survival probabilities predicted by the unperturbed logit survival model were already approaching the boundary of one for larger adults (Figures 5.3, 5.4, also see Figure S3 for a schematic of this 'linearisation effect'). Consequently, these perturbations increased adult survival yet lowered

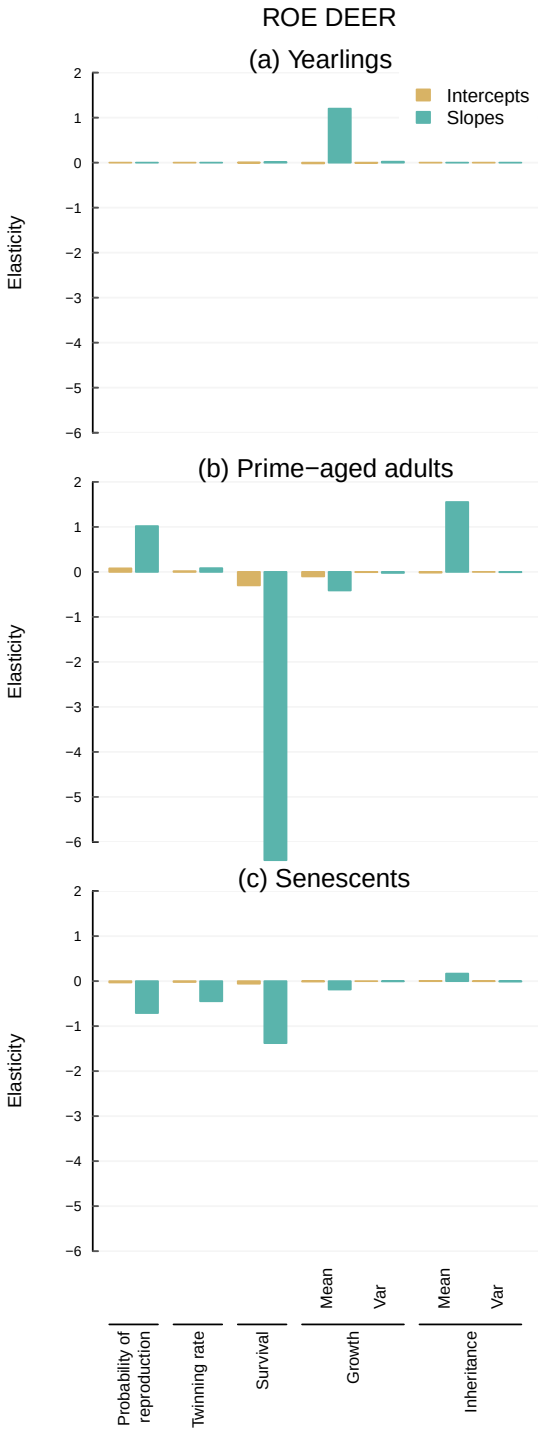

Figure 5.2. The analytically derived elasticities of heritability with respect to changes in the model parameters of the roe deer IPM.

viability selection. In contrast to this, increasing the survival slope for Soay sheep lambs, for example, increased survival and viability selection, meaning that survival increased more for larger lambs than for smaller lambs (Figures S3 and 5.3).

Regardless of the direction of the change in viability selection, increasing the adult survival slopes decreased heritability because more individuals survived and grew to reproduce at larger size. This surplus of adult individuals reproducing at large phenotype values, and producing relatively large offspring, resulted in an increase of the mean birth phenotype of the stable cohort. This meant that potential parents were larger and more similar at birth (Table 5.1). As a consequence, the variance in parent birth phenotype decreased. The surplus of large reproducing adult individuals also increased the mean offspring phenotype and decreased the variance among offspring. However, the covariance between parent and offspring birth phenotype decreased substantially, possibly because more parents with small birth phenotypes grew to large adult sizes where they gave birth to large offspring. Defined by the ratio of this covariance and the parent birth phenotypic variance, heritability overall decreased for both species as the survival slope increased (Table 5.1).

Increasing the survival slopes for Soay sheep lambs and yearlings had comparable effects (Table 5.1). However, perturbing the survival slope for roe deer yearlings had little effect because the decrease in the parent birth phenotypic variance was offset by an increase in the covariance between parent and offspring birth phenotype. This possibly occurred because if yearlings survived better, there was an increase in small individuals that survived the yearling age class and then reproduced as small adults the next year.

Elasticities of heritability with respect to fertility selection

Changing fertility and fertility selection had much larger effects on heritability for Soay sheep than for roe deer (Figures 5.1 and 5.2). For Soay sheep, fertility selection mostly operated through the probability of reproduction because the twinning rates, which determine whether individuals that reproduce give birth to twins, were very low (Figure 5.3). Another idiosyncrasy of the the Soay sheep model was that the

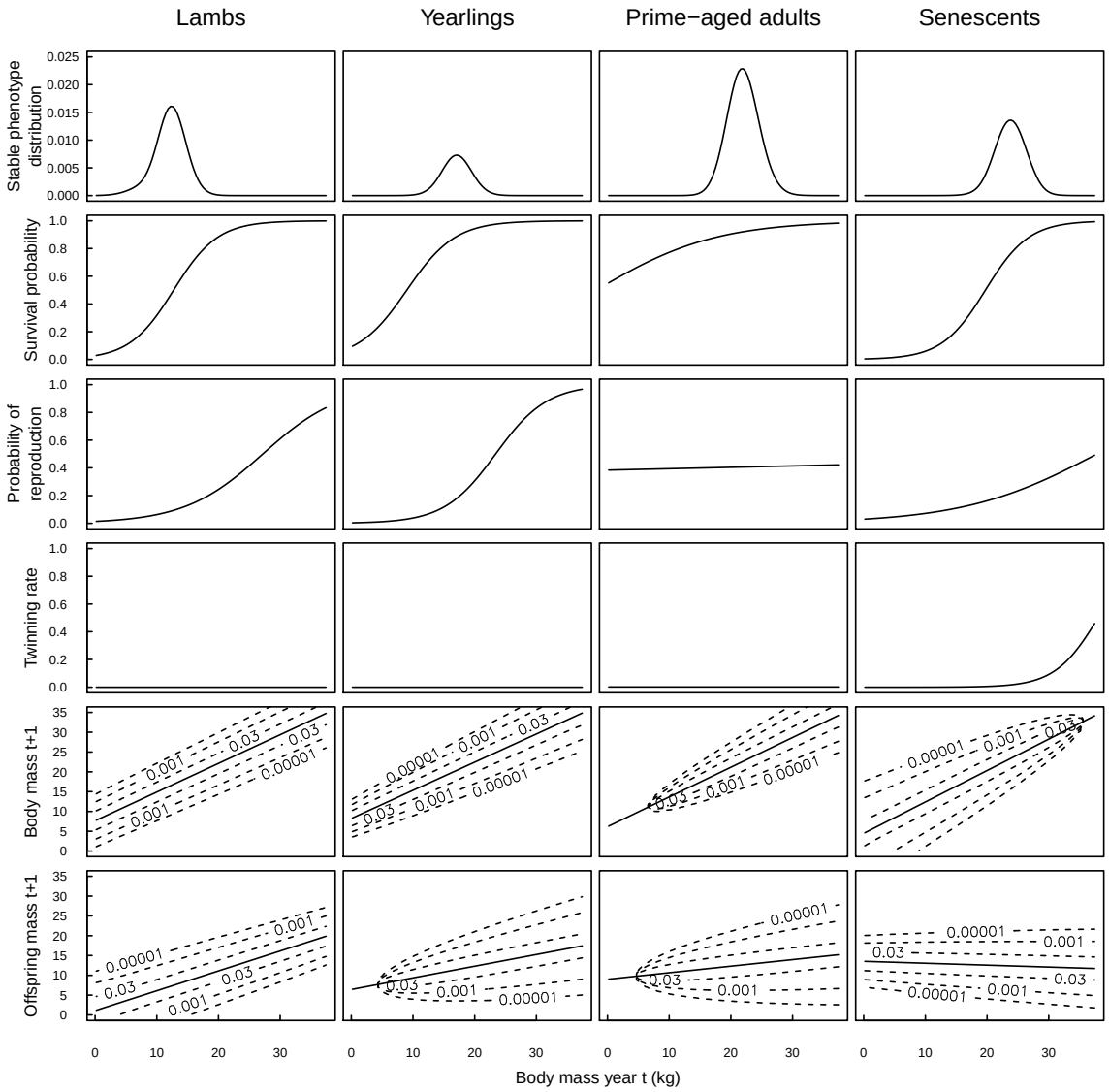

Figure 5.3. The phenotype-demography relationships used to parameterise the matrices in the IPM and the predicted stable phenotype distributions for Soay sheep. The age-class phenotype distributions together sum up to 1. For the phenotype-demography functions, lines represent predictions from regressions and dashed contours distributions around the mean.

probability of reproduction for adults was similar across all phenotypes (Figure 5.3). Therefore, fertility selection in the adult age class was low. Introducing fertility selection by increasing the slope of this function had the largest effect on heritability of all perturbations for Soay sheep (Figure S1). It decreased heritability by 5 %.

Similarly to the effect of increasing the adult survival slope, increasing fertility and positive fertility selection among adult Soay sheep resulted in more adults re-

producing at large phenotype values. This increased the mean of the stable cohort birth phenotype distribution and decreased its variance (Table 5.1). The increase in fertility selection resulted in an increase in the mean parent birth phenotype, but the overall increase in fertility also led to smaller adults having higher fertility and therefore to an increase in the variance in birth phenotype among parents. The offspring birth phenotype increased due to the increase in fertility selection, and the variance in offspring birth phenotype decreased. The parent and offspring birth phenotypic covariance therefore decreased substantially, which resulted in a large decrease in heritability (Table 5.1).

In Soay sheep, lambs have a small probability of reproduction (Figure 5.3). Increasing this probability, and simultaneously increasing fertility selection by increasing the slope of the function, increased heritability (Figure5.1). This drastically increased the parent offspring birth phenotypic covariance, because small-born parents were now more likely to give birth at small lamb sizes to small offspring. More lambs reproducing also caused a decrease in the mean and an increase in the variance of all three distributions: potential parents, parents, and offspring were on average smaller and less similar at birth. As a result, heritability increased (Table 5.1).

For roe deer, altering the probability of reproduction had less pronounced effects (Figure 5.2). Increasing fertility and decreasing fertility selection for adults, due to the linearisation effect (Figure S3), increased heritability. Supposedly, the decrease in fertility selection resulted in a decrease in the mean stable cohort birth phenotype because smaller adults had a higher probability of reproduction and a higher increase in the probability of reproduction than larger adults. At the same time, the variance in the stable cohort birth phenotype decreased; potential parents were smaller and more similar when compared to the unperturbed model. This smaller mean and variance translated into smaller means and variances for both the parent and offspring birth phenotype. The parent offspring covariance increased, because parents that were born small, and therefore took longer to grow to the

asymptotic size than large-born parents, had a higher probability of reproduction as small adults, resulting in small-born parents having a higher probability to give birth to small offspring. The increased covariance divided by a smaller parental variance resulted in an increased heritability (Table 5.1).

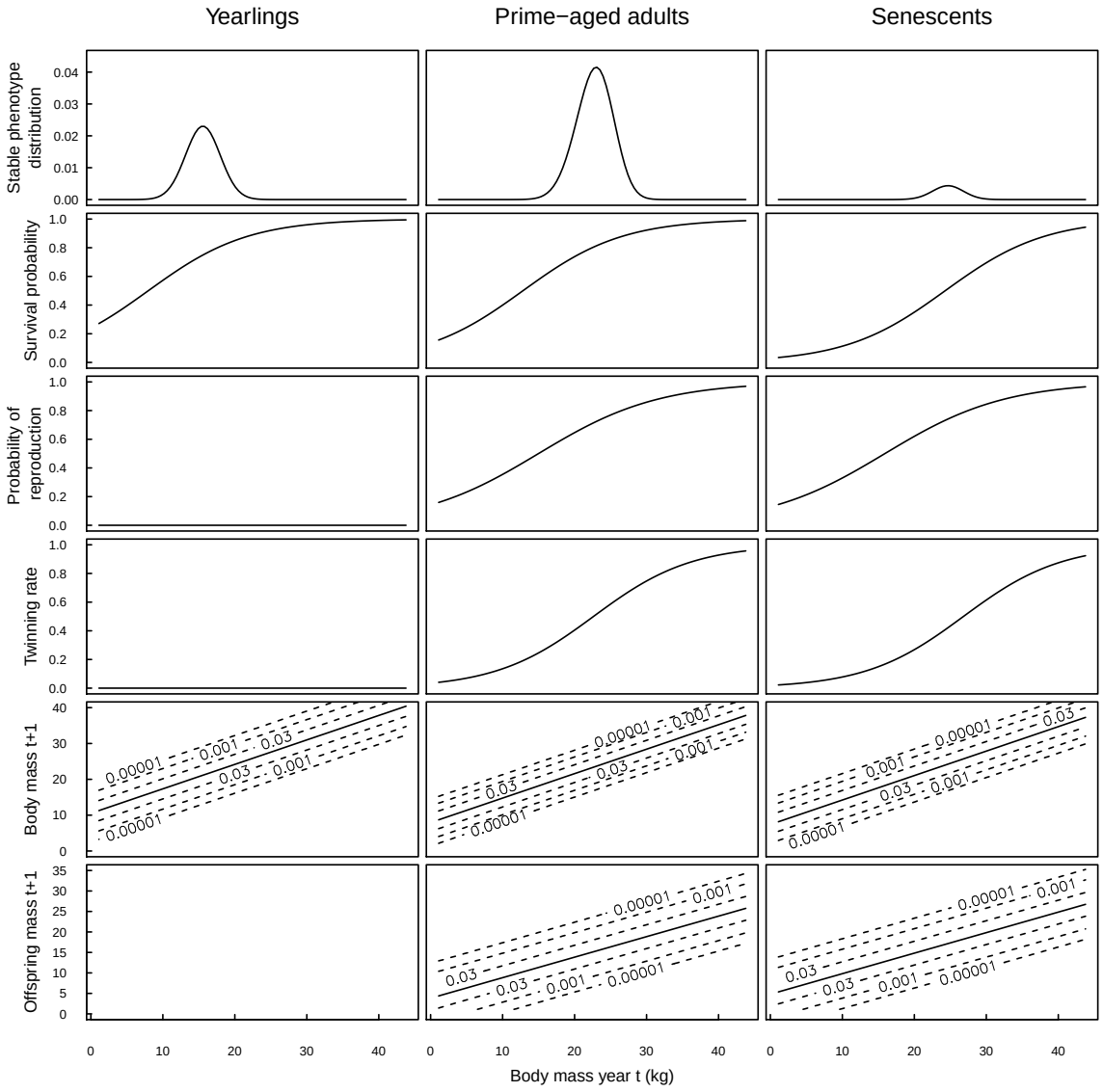

Figure 5.4. The phenotype-demography relationships used to parameterise the matrices in the IPM and the predicted stable phenotype distributions for roe deer. The age-class phenotype distributions together sum up to 1. For the phenotype-demography functions, lines represent predictions from regressions and dashed contours distributions around the mean. Roe deer yearlings do not reproduce.

Table 5.1. Change in key quantities (%) when selected model parameters are perturbed (see Tables S1 and S2 for perturbations of all parameters).

		$\mathcal{E}(X_0)$	$\mathrm{Var}(X_0)$	$\mathcal{E}(X_0)$	$\mathrm{Var}(X_0)$	$\mathcal{E}(Y)$	$\mathrm{Var}(Y)$	$\mathrm{Cov}(Y X_0)$	h^2
Soay sheep	SurvSlpL*	0.00	-0.30	-0.15	-0.71	0.15	-1.24	-4.38	-3.69
	SurvSlpY	0.01	-0.09	-0.07	0.05	0.05	-0.38	-1.73	-1.79
	SurvSlpA	0.04	-0.14	-0.01	-0.11	0.15	-0.94	-3.75	-3.65
	PrSlpL	-0.41	2.84	-0.13	1.37	-0.35	2.59	4.53	3.12
	PrSlpA	0.15	-0.03	0.14	0.11	0.40	-1.58	-4.11	-4.21
	GrMSlpA	0.30	1.55	0.39	2.01	0.38	0.90	-0.71	-2.67
	GrMSlpS	-0.04	-0.15	-0.03	-0.14	-0.01	-0.57	-2.09	-1.95
	InMSlpY	0.13	-0.10	0.09	-0.29	0.12	-0.17	1.45	1.75
	InMSlpA	1.30	2.55	1.25	2.13	1.35	1.87	1.52	-0.60
roe deer	SurvSlpY	-0.11	0.00	-0.17	-0.12	-0.03	0.07	-0.09	0.02
	SurvSlpA	0.13	-0.13	0.02	-0.16	0.36	-0.10	-6.02	-5.87
	PrSlpA	-0.27	-0.14	-0.29	-0.15	-0.16	-0.03	0.86	1.01
	GrMSlpY	0.32	-0.68	0.28	-0.69	0.30	-0.72	0.50	1.21
	InMSlpA	1.64	-0.71	1.51	-0.84	1.54	-1.33	0.70	1.56

* Model parameter labels are written in CamelCase forming compounds of the following abbreviations in order of appearance: survival (Surv), slope (Slp), lambs (L), yearlings (Y), adults (A), probability of reproduction (Pr), Growth mean (GrM), senescencts (S), inheritance mean (InM)).

Elasticities of heritability with respect to phenotypic development

Changing both intercept or slopes of the functions that determined the variances in growth had little effect on heritability for both species (Figures 5.1 and 5.2). These perturbations changed the variances in parent and offspring birth phenotype and in the parent offspring covariance to similar proportions, which then overall had no effect on heritability (Tables S1 and S2). These findings even held for Soay sheep, where the growth variances have slopes that significantly deviate from 0 (Figure 5.3).

Increasing the slope of the mean growth function for adult and senescent Soay sheep had pronounced negative effects on heritability (Figure 5.1). Increasing the slope of the mean growth function in an IPM has generally two consequences. First, all individuals of the relevant age class grow faster, and the acceleration of growth is even larger for large individuals. Second, the maximum size, to which individuals grow asymptotically ('asymptotic size'), increases because the mean growth rate function crosses the y = x line at higher phenotype values. The cross-over point

determines the size at which individuals stay on average the same size over time.

When the adult mean growth slope for Soay sheep was increased, individuals grew faster to larger phenotype values at which they had higher survival. This caused an increase in the number of adults reproducing at large phenotype values and therefore increased the mean size of potential parents, but also the variance in size among them (Table 5.1). The increase in variance may have been caused by the increase in the asymptotic size of adults, which increased the range of phenotype values over which adults reproduce. This change in mean and variance of the parent cohort cascaded through and led to similar increases in the mean and variance of parent and offspring birth phenotypes. The increase in the asymptotic size of adults, and the increase in how fast individuals grow to this size, increased the number of individuals that reproduce at large phenotype values regardless of their birth phenotype. Consequently, the covariance between parent and birth phenotype decreased, which divided by a much larger variance in birth phenotype, resulted in lower heritability (Table 5.1).

In order to understand how increasing the mean growth slope for senescent Soay sheep influenced heritability, it is important to notice that the slope for mean inheritance is negative for senescent individuals; the larger they are, the smaller is their offspring (Figure 5.3). As a result, shifting the asymptotic size for senescent individuals towards larger phenotype values, at which individuals had higher probabilities of reproduction and survival yet gave birth to smaller offspring, decreased the mean and the variance in the stable cohort birth phenotype distribution. This was again followed by similar developments in the parent and offspring birth phenotype distributions. However, the parent offspring covariance decreased even more, supposedly due to similar reasons than those for the effect of perturbing growth of adults, so that overall heritability decreased (Table 5.1).

For roe deer, only increasing the growth mean slope for yearlings had a notable effect on heritability (Figure 5.2). Increasing the slope resulted in individuals

growing faster to larger sizes at which they recruited to the adult age class. They therefore experienced higher probabilities of survival during the first time step as adults. Due to the positive slope of the mean inheritance function (Figure 5.4), this increased the mean and decreased the variance in the stable cohort birth phenotype. Since potential parents were larger and more similar at birth, plus they were exposed to higher growth rates during the yearling age class, both parents and offspring were also larger and more similar in birth phenotype. The parent offspring covariance increased, supposedly because parents grew faster as yearlings and recruited to the adult age class at larger sizes with a higher probability of reproduction. However, compared to the mean adult size, they were still small and therefore produced relatively small offspring. Therefore, this perturbation increased the fraction of small parents that made small offspring, and it thus increased the parent offspring covariance. Divided by a smaller parental variance, this led to higher heritability (Table 5.1).

Elasticities of heritability with respect to parent-offspring phenotypic transition

The only direct relationship between offspring and parent phenotype (at birth of offspring) is modelled by the inheritance function. Nevertheless, perturbing this relationship by increasing the slopes of the mean inheritance functions had only moderate effects on heritability (Figures 5.1 and 5.2). Increasing the slope of the mean inheritance function for Soay sheep yearlings and roe deer adults increased the mean stable cohort birth phenotype and decreased the associated variance. It supposedly decreased the variance because both age classes had high probabilities of reproduction for the lower part of the relevant phenotype distributions. The offspring of these comparatively small individuals at the lower parts of the phenotype distributions now recruited at larger sizes to the population, thus compressing the stable cohort birth phenotype distribution (Figures 5.3 and 5.4). Since potential

parents started out larger and more similar, the mean birth phenotype of parents was also larger and its variance smaller. Since parents were larger, they gave birth to larger and more similar offspring. The covariance between parent and offspring phenotype increased, while the variance in parent birth phenotype decreased, resulting in a higher heritabilities caused by these perturbations.

However, increasing the slope of the mean inheritance function for adult Soay sheep actually decreased heritability (Figure 5.1). The usual effect of increasing the slope of the mean inheritance function is that the mean of the offspring birth phenotype distribution shifts to larger sizes and the variance is spread out. This is because parents of all phenotypes have larger offspring but offspring of the larger parents have even a greater increase in size when compared to offspring of smaller parents. We therefore concluded above that the variance-decreasing effect of perturbing the mean inheritance slope for Soay sheep yearlings and roe deer adults on the stable birth phenotype distribution must have been mediated by high fertility for relatively small individuals. By increasing the mean inheritance slope for adult Soay sheep, we observed the predicted effect on the stable cohort birth phenotype: both the mean and the variance increased. This was likely due to the probability of reproduction having been relatively low and equal across adult phenotype values. Since potential parents were larger and more variable in birth phenotype, the mean and the variance in both parent and offspring birth phenotype also increased, which resulted in an increase in the parent offspring covariance. But as opposed to the outcome for the slope for Soay sheep yearlings and roe dear adults, the parent birth phenotypic variance had also substantially increased, which overall resulted in a reduction in heritability.

Discussion

In this study we asked which processes determine heritability in species with phenotypic development with age—differential survival, differential fertility, variation in growth rates, or the relationship between maternal size and offspring birth size? By studying this for two species of large vertebrates in an IPM framework, we have demonstrated that all four processes can contribute to determining heritability. Our work therefore recommends IPMs as tools to complement well-established methods in quantitative genetics in the quest to understand heritability and microevolution in wild animal populations.

It may appear at first glance that no simple rules for the dependency of heritability on phenotypic selection and phenotypic transition processes can be concluded from our results. The variety of ways in which heritability can respond to perturbations in the different functions seems puzzling. Nevertheless, discerning the detailed effect of each perturbation on phenotypic selection and phenotypic transition may lend some guidance to understanding the determinants of heritability.

First, perturbing intercepts mostly affects the means of phenotype distributions, while perturbing slopes affects the means and the variances of phenotype distributions by changing the degree of phenotypic selection or phenotypic transition (Coulson et al. in prep.; see Appendix). Perturbing slopes therefore generally has larger effects on heritability than perturbing intercepts. Second, perturbations of the same parameter, but for different age classes, do not always change processes in the same way. Their particular effect depends on the linear or linearised nature of the function, the unperturbed parameter values, and the stable age class phenotype distributions. Increasing the slope of the survival function, for example, can increase viability selection for lambs yet decrease it for adults due to the linearisation effect (Figure S3). Third, the different functions interact. An increase in viability selection, for example, increases the variance in offspring sizes if the inheritance func-

tion has a positive slope, but reduces offspring sizes if the slope is slightly negative (e.g., compare adult and senescent inheritance of Soay sheep, Figure 5.3). Fourth, a change in a process in one age class may actually only act through changing the phenotype distribution of the next age class. For example, the effect on heritability of an increased growth trajectory for roe deer yearlings operates through altering the size at which last-year's yearlings recruit to the adult population. The more the functions vary among the different age classes, the less predictable are their interactions. This may explain why we see more and mostly stronger responses in heritability to perturbations for Soay sheep when compared to roe deer (Figures 5.3 and 5.4).

With this information, we can distil some general insights from our results despite their variety. It appears that decreasing viability selection generally decreases heritability. To understand this we need to take the perspective of individual life history trajectories of potential parents. They are born at offspring sizes, if they survive they grow, start to reproduce, grow more, and reproduce more. If they reproduce while they are small and still growing, they give birth to small offspring. Therefore, even large-born parents at first give birth to small offspring. Over their lifetime, with increasing size, surviving parents give birth to larger offspring, regardless of their initial birth phenotype. If small-born parents die while still growing, having not given birth at all or having given birth to small offspring, then only large-born parents survive to give birth to large offspring. In this case, small-born parents give birth to small offspring and large-born parents give birth to large offspring. The parent offspring covariance is therefore high, and so too is the heritability. If however small-born parents survive to grow to large sizes and eventually, on average, give birth to large offspring, then the parent offspring covariance is lower, because small-born parents end up giving birth to large offspring. More small-born parents survive to grow to large sizes with decreasing viability selection. Decreasing viability selection therefore generally decreases heritability, as we have observed for both

Soay sheep and roe deer in the adult and senescent age classes.

Given positive viability selection, increasing fertility selection appears to decrease heritability. This is likely because fertility selection mediates the effect of viability selection on the parent offspring covariance. If positive viability selection acts in the absence of fertility selection, then small-born parents on average give birth to small offspring because small-born parents die after having given birth while still growing. However, positive fertility selection disrupts this pattern. With increasing fertility selection, small-born parents have lower probabilities of reproduction. The covariance-increasing effect of small-born parents giving birth to small offspring is therefore reduced. In support of this explanation, we have observed an heritability-decreasing effect of increasing fertility selection for Soay sheep adults. We have also observed the reversed pattern for roe deer adults, where decreasing fertility selection resulted in an increase in heritability.

The effects of both types of phenotypic selection on heritability are mediated by within-individual phenotypic development with age. The phenotype development rates determine how fast individuals grow from offspring sizes to the asymptotic size. If they grow slowly, then any effect of phenotypic selection on heritability has more time to act on the still-growing individuals. If however individuals grow fast to large sizes, then phenotypic selection has little opportunity to influence heritability. Among Soay sheep adults we observed weak fertility selection but positive viability selection on the phenotype. As we have explained above, positive viability selection increases heritability. However, by increasing the mean growth rates of Soay sheep adults, individuals grow faster to the asymptotic body size. Increasing the growth rates therefore reduces the variation in size, which is necessary for viability selection to have a positive effect on heritability. Consequently, increasing the slope of the mean growth rates for Soay sheep adults reduced heritability.

Increasing the slope of the mean inheritance function, that determines the parent-offspring phenotypic transition, generally results in increasing the variance

in phenotype among potential parents. It therefore determines the variation in phenotypes on which phenotypic selection and development can act upon to determine heritability. An increase in the phenotypic variance among potential parents amplifies the heritability-determining effects of phenotypic selection. We therefore found that increasing the slope of the mean inheritance function uniformly increased the parent offspring covariance. However, this did not uniformly result in an increase in heritability. For Soay sheep adults, the increase in phenotypic variance among potential parents also resulted in an increase in the variance in parent birth phenotype. Since this increase was greater than the increase in the parent offspring covariance, this resulted in an overall decrease in heritability.

With these insights, we now turn to the significance of our results for the use of quantitive genetic methods to understand microevolutionary processes in free-living populations. Much of this work has focussed on explaining the lack of phenotypic change in heritable characters under directional selection (Merilä et al. 2001). In this paper, we analysed IPMs at equilibrium when the phenotype distribution was stable. Therefore, the mean phenotype did not change over time and the response to selection was 0, despite the fact that we estimated considerable heritability of the character and that the phenotype was under positive selection. This is possible because phenotypic selection and phenotypic transition processes are balanced in such a way that any removals from the stable phenotype distribution through differential survival is countered by individuals shifting within the distribution due to phenotypic development with age, and individuals being added at small phenotype values due to fertility selection, phenotypic development with age, and the parent-offspring phenotypic transition. We learn from this that the phenotype of a heritable character under selection can show no response to selection, if the phenotype develops with age.

Our results may also reveal another reason why quantitative genetics has little success in predicting microevolutionary processes from estimates of heritability and

selection that are obtained from free-living populations. The statistical procedures for estimating heritability from phenotypic covariances among relatives make various assumptions about the respective phenotype distributions. These assumptions are usually that the effect of genes is additive, that there is random mating and linkage equilibrium, that survival and fertility are constant over time, and that the mean of the offspring breeding value distribution is equal to the mean of the parent breeding value distribution (i.e., there is no selection) (reviewed in Hill 2009). These assumptions are likely never met in free-living populations due to environmental stochasticity, density dependence, phenotypic development with age, and incomplete re-sighting probabilities of marked animals. This is why the animal model, that can correct for many of these factors, has been widely used to study heritability in free-living populations. Furthermore, methods have been developed to account for missing data points of those individuals that died before they could be measured in populations where viability selection acts on the trait of interest, opposing one of the key assumptions (Hadfield 2008, Nakagawa and Freckleton 2008, Steinsland et al. 2014). This process is called selective disappearance and the non-observed individuals are the invisible fraction (Rebke et al. 2010, Hadfield 2008).

In this study, we have shown how the intricate interactions among not only viability selection but also fertility selection and the phenotypic transition processes shape the parent and offspring birth phenotype distributions and heritability. Positive fertility selection causes mothers at smaller phenotype values to have fewer offspring than larger mothers. These never-born offspring of smaller mothers form another "invisible fraction", that has to our knowledge not yet been addressed in the literature. Our results also demonstrate that the effects of viability and fertility selection on the parental and offspring phenotype distributions are mediated by the development of the phenotype with age, and by the parent-offspring phenotypic transition. The processes that generate these invisible fractions, both among both parents and offspring, will therefore continue to be a significant challenge for

obtaining unbiased estimates of heritability in free-living animal populations.

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Supporting Information

Supporting methods Details of the analytical framework.

Table S1 Change in key quantities when model parameters are perturbed (Soay sheep).

Table S2 Change in key quantities when model parameters are perturbed (roe deer).

Figure S1 Comparison of the analytically and numerically computed elasticities of heritability for Soay sheep.

Figure S2 Comparison of the analytically and numerically computed elasticities of heritability for roe deer.

Figure S3 Schematic of the linearisation effect.

Code S1 R code for analytically computing heritability and its elasticities. Download from github.com/bartholdja/DPhil_supplements.

Supporting methods

SI.5.1 Parent-offspring covariance

The expected birth phenotype Y of offspring born to parents of age a and phenotype X_a can be described by fitting a model of the form

$$\mathcal{E}\left[Y|X_a\right] = \alpha_a + \beta_a\, X_a. \tag{S-1}$$

The expected offspring birth phenotype Y produced by a parent of age a and phenotype z_i can also be calculated from the parent-offspring phenotype transition matrix as

$$\mathcal{E}\left[Y|X_a = z_j\right] = \sum_i z_i\, D_a(i,j), \tag{S-2}$$

and so it follows that

$$\mathbf{z}^T\,\mathbf{D}_a = \mathbf{e}^T\,\widehat{\mathbf{Z}}\mathbf{D}_a = \alpha_a\,\mathbf{e}^T + \beta_a\,\mathbf{z}^T. \tag{S-3}$$

Since the lifetime production of offspring by a birth cohort is given by (5.8) in the main article, the probability that a newborn has a parent of age a is

$$\phi_a = \frac{\mathbf{e}^T\mathbf{F}_a\mathbf{L}_a\,\mathbf{u}}{K} = \frac{\mathbf{e}^T\widehat{\mathbf{M}}_a\mathbf{L}_a\,\mathbf{u}}{K}. \tag{S-4}$$

To derive a formula for the covariance between offspring phenotype Y and parent birth phenotype X that is computable in terms of $\mathbf{F}_a, \mathbf{L}_a$, and $\mathbf{u}$, we first derive the same covariance in terms of ϕ_a, α_a, and β_a. Consider parents of age a and use (S-1) to see that

$$E(Y \mid \text{parent is age } a) = \alpha_a + \beta_a\mathcal{E}(X_a). \tag{S-5}$$

Hence, for lifetime reproduction, writing $\overline{\alpha} = \sum_a \phi_a \alpha_a$,

$$\mathcal{E}(Y) = \overline{\alpha} + \sum_a \phi_a \beta_a \, \mathcal{E}(X_a), \tag{S-6}$$

$$\mathcal{E}(Y X_0) = \overline{\alpha} \, \mathcal{E}(X_0) + \sum_a \phi_a \beta_a \, \mathcal{E}(X_a X_0). \tag{S-7}$$

Using (S-6) and (S-7) the formula for the offspring-parent phenotype covariance in terms of the linear relationship in (S-1) is

$$\mathrm{Cov}(Y X_0) = \sum_a \phi_a \, \beta_a \, \mathrm{Cov}(X_a \, X_0). \tag{S-8}$$

From (S-8) it becomes clear that the phenotype covariance $\mathrm{Cov}(Y X_0)$ between parents at own birth and offspring at birth depends only on the regression coefficients β_a of offspring on parent trait value and on the ontogenetic development, as captured in the covariance $\mathrm{Cov}(X_a \, X_0)$ between parents birth phenotype and phenotype of parents aged a.

Next, to make the covariance computable in terms of our phenotype-demography matrices, the joint distribution of parent phenotype at age a and parent birth phenotype X_0 is

$$\text{Prob.}\,[X_a = w_2, X_0 = w_1] = \frac{M_a(w_2) \, L_a(w_2 \mid w_1) \, u(w_1)}{(\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \mathbf{u})}. \tag{S-9}$$

So given parents of age a with phenotype X_a and birth phenotype X_0,

$$\mathcal{E}(X_a) = \frac{\mathbf{e}^T \, \widehat{\mathbf{Z}} \, \widehat{\mathbf{M}}_a \, \mathbf{L}_a \, \mathbf{u}}{(\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \mathbf{u})}, \tag{S-10}$$

$$\mathcal{E}(X_a \, X_0) = \frac{\mathbf{e}^T \, \widehat{\mathbf{Z}} \, \widehat{\mathbf{M}}_a \, \mathbf{L}_a \, \widehat{\mathbf{Z}} \, \mathbf{u}}{(\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \mathbf{u})}, \tag{S-11}$$

The joint distribution of offspring birth phenotype, parental phenotype at age a,

and parent birth phenotype is

$$\text{Prob.}\,[Y = W_3, X_a = w_2, X_0 = w_1] = \frac{D_a(w_3 \mid w_2)\, M_a(w_2)\, L_a(w_2 \mid w_1)\, u(w_1)}{(\mathbf{e}^T \mathbf{F}_a \mathbf{L}_a \mathbf{u})}. \tag{S-12}$$

So we have

$$\mathcal{E}(Y) = \sum_a \phi_a \frac{\mathbf{e}^T \widehat{\mathbf{Z}}\, F_a\, \mathbf{L}_a\, \mathbf{u}}{(\mathbf{e}^T \mathbf{F}_a \mathbf{L}_a \mathbf{u})}, \tag{S-13}$$

$$= \frac{1}{K} \sum_a \{\mathbf{e}^T\, \widehat{\mathbf{Z}}\, \mathbf{F}_a\, \mathbf{L}_a\, \mathbf{u}\}. \tag{S-14}$$

Next we have

$$\mathcal{E}(YX_0) = \frac{1}{K} \sum_a \{\mathbf{e}^T\, \widehat{\mathbf{Z}}\, \mathbf{F}_a\, \mathbf{L}_a\, \widehat{\mathbf{Z}}\, \mathbf{u}\}. \tag{S-15}$$

Now we use (S-21), (S-14), and(S-15) to obtain (5.10) in the main article, which is a formula for the parent-offspring birth phenotype covariance that is computable in terms of the phenotype-demography matrices

$$\text{Cov}(YX_0) = \mathcal{E}(YX_0) - \mathcal{E}(Y)\mathcal{E}(X_0), \tag{S-16}$$

$$= \frac{1}{K} \sum_a \left\{ \mathbf{e}^T\, \widehat{\mathbf{Z}}\, \mathbf{F}_a \mathbf{L}_a\, \widehat{\mathbf{Z}}\, \mathbf{u} - \left(\mathbf{e}^T\, \widehat{\mathbf{Z}}\, \mathbf{F}_a \mathbf{L}_a\, \mathbf{u}\right) \left(\frac{1}{K} \sum_a \mathbf{e}^T\, \widehat{\mathbf{M}}_a\, \mathbf{L}_a\, \widehat{\mathbf{Z}}\, \mathbf{u} \right) \right\}, \tag{S-17}$$

$$= \frac{1}{K} \sum_a \left\{ \mathbf{e}^T\, \widehat{\mathbf{Z}}\, \mathbf{F}_a \mathbf{L}_a \left(\mathbf{I} - \mathbf{u}\, \mathbf{e}^T\, \frac{\sum_a \widehat{\mathbf{M}}_a\, \mathbf{L}_a}{K} \right) \widehat{\mathbf{Z}}\, \mathbf{u} \right\}, \tag{5.9}$$

$$= \mathbf{e}^T\, \widehat{\mathbf{Z}}\, \mathcal{H} \left(\mathbf{I} - \mathbf{u}\, \mathbf{e}^T\, \frac{\sum_a \widehat{\mathbf{M}}_a\, \mathbf{L}_a}{K} \right) \widehat{\mathbf{Z}}\, \mathbf{u}. \tag{5.10}$$

SI.5.2 Parent birth phenotype variance

In computing the variance, we need to account for the dispersion of lifetime reproduction across different ages. The parent cohort is born with the stable cohort birth phenotype distribution $\mathbf{u}$. From (S-9) it follows that for parents aged a their average birth phenotype is

$$\mathcal{E}(X_0^a) = \sum_{w_2} \sum_{w_1} w_1 \,\text{Prob.}\left[X_a = w_2, X_0 = w_1\right].$$

Note that we sum over the trait values at age a and average trait value at birth. Thus

$$\mathcal{E}(X_0^a) = \frac{\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\,\mathbf{u}}{(\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \mathbf{u})} = \frac{\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\,\mathbf{u}}{(K\,\phi_a)}, \tag{S-18}$$

where the last equality uses (5.8) in the main article. The average squared birth phenotype for parents aged a is

$$\mathcal{E}\,(X_0^a)^2 = \frac{\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\,\widehat{\mathbf{Z}}\,\mathbf{u}}{(\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \mathbf{u})} = \frac{\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\,\widehat{\mathbf{Z}}\,\mathbf{u}}{(K\,\phi_a)}. \tag{S-19}$$

The variance of birth phenotype for parents of age a is

$$V_0^a = \text{Var}(X_0^a) = \mathcal{E}\,[X_0^a]^2 - [\mathcal{E}(X_0^a)]^2\,. \tag{S-20}$$

Now the average birth phenotype for all parents (i.e., of all ages) is

$$\mathcal{E}(X_0) = \sum_a \phi_a\,\mathcal{E}(X_0^a) = \frac{1}{K}\sum_a \mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\,\mathbf{u}. \tag{S-21}$$

Finally, the variance of birth phenotype for all parents is

$$\mathrm{Var}(X_0) = \left\{ \sum_a \phi_a \left[\mathcal{E}(X_0^a)\right]^2 - \left[\mathcal{E}(X_0)\right]^2 \right\} + \left\{ \sum_a \phi_a V_0^a \right\} \tag{S-22}$$

$$= \sum_a \left\{ \phi_a \mathcal{E}\left[X_0^a\right]^2 - \left[\mathcal{E}(X_0)\right]^2 \right\} \tag{S-23}$$

$$= \frac{1}{K} \sum_a \left\{ \mathbf{e}^T \widehat{\mathbf{M}}_a \, \mathbf{L}_a \, \widehat{\mathbf{Z}} \, \widehat{\mathbf{Z}} \mathbf{u} - \frac{1}{K} \left(\mathbf{e}^T \widehat{\mathbf{M}}_a \, \mathbf{L}_a \, \widehat{\mathbf{Z}} \mathbf{u}\right)^2 \right\}. \tag{5.12}$$

SI.5.3 Perturbation of $\mathbf{u}$

For the unperturbed matrix $\mathbf{A}(r)$ we have defined the right eigenvector $\mathbf{u}$ as in (5.4) in the main article but also need the left eigenvector $\mathbf{v}$,

$$\mathbf{v}^T \, \mathbf{A}(r) = \mathbf{v}^T. \tag{S-24}$$

For convenience we normalize so that $(\mathbf{v}^T \, \mathbf{u}) = 1$. Then define the projection matrix

$$\mathcal{Z} = \mathbf{u} \, \mathbf{v}^T, \tag{S-25}$$

and the matrix

$$\mathcal{Q} = \mathbf{A}(r) - \mathcal{Z}. \tag{S-26}$$

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

$$\mathbf{F}_a \, \mathbf{L}_a \to \mathbf{F}_a \, \mathbf{L}_a + \epsilon \, \mathbf{\Delta}_a. \tag{S-27}$$

Then we have the resulting changes

$$\begin{aligned} r &\to r + \epsilon\, r_1, \\ \mathbf{A}(r) &\to \mathbf{A}(r) + \epsilon\, \Delta\mathbf{A}, \\ \mathbf{u} &\to \mathbf{u} + \epsilon\, \boldsymbol{\delta}_u. \end{aligned} \tag{S-28}$$

Note that the change $\boldsymbol{\delta}_u$ must be orthogonal to $\mathbf{u}$, so that $\mathcal{Z}\boldsymbol{\delta}_u = 0$. From (5.4) in the main article $\Delta\mathbf{A}$ is given by and abbreviated as

$$\Delta\mathbf{A} = \sum_a e^{-ra}\, \boldsymbol{\Delta}_a - r_1 \sum_a e^{-ra}\, a\, \mathbf{F}_a\, \mathbf{L}_a = \mathcal{D}_1 - r_1\, \mathcal{D}_2. \tag{S-29}$$

Clearly

$$\mathbf{A}(r)\, \boldsymbol{\delta}_u + \Delta\mathbf{A}\, \mathbf{u} = \boldsymbol{\delta}_u. \tag{S-30}$$

A standard argument (we multiply above on left by $\mathbf{v}^T$) yields the change

$$r_1 = \frac{\mathbf{v}^T\, \mathcal{D}_1\, \mathbf{u}}{\mathbf{v}^T\, \mathcal{D}_2\, \mathbf{u}}. \tag{S-31}$$

Next we observe that

$$\mathcal{Z}\, \mathbf{A}(r) = \mathbf{A}(r)\, \mathcal{Z};\ \ \mathcal{Q}\, \mathbf{A}(r) = \mathbf{A}(r)\, \mathcal{Q}. \tag{S-32}$$

Recall that $\mathcal{Z}\boldsymbol{\delta}_u = 0$ so $\boldsymbol{\delta}_u = (\mathbf{I} - \mathcal{Z})\, \boldsymbol{\delta}_u$. This is why we multiply (S-30) on the left by $(\mathbf{I} - \mathcal{Z})$ to find

$$(\mathbf{I} - \mathcal{Z})\, \mathbf{A}(r)\, \boldsymbol{\delta}_u + (\mathbf{I} - \mathcal{Z})\, \Delta\mathbf{A}\, \mathbf{u} = (\mathbf{I} - \mathcal{Z})\, \boldsymbol{\delta}_u = \boldsymbol{\delta}_u. \tag{S-33}$$

Next,

$$(\mathbf{I} - \mathcal{Z})\,\mathbf{A}(r) = \mathbf{A}(r) - \mathcal{Z}\,\mathbf{A}(r) = (\mathbf{A}(r) - \mathcal{Z}), \tag{S-34}$$

where we use (S-25). Hence (S-31) becomes

$$(\mathbf{I} - \mathcal{Z})\,\Delta\mathbf{A}\,\mathbf{u} = \boldsymbol{\delta}_u - (\mathbf{A}(r) - \mathcal{Z})\,\boldsymbol{\delta}_u = [\mathbf{I} - (\mathbf{A} - \mathcal{Z})]\;\boldsymbol{\delta}_u.$$

We now multiply across by $[\mathbf{I} - (\mathbf{A} - \mathcal{Z})]^{-1}$ on both sides to obtain

$$\boldsymbol{\delta}_u = [\mathbf{I} - (\mathbf{A} - \mathcal{Z})]^{-1}\;(\mathbf{I} - \mathcal{Z})\,\Delta\mathbf{A}\,\mathbf{u}. \tag{S-35}$$

SI.5.4 Perturbation matrices

The perturbations in (S-27) come from changes in the fertility or survival matrices, as we explain in this subsection.

SI.5.4.1 Selection on modifiers of fertility A small change in fertility results from a change in total recruitment and/or in the phenotype distribution of offspring: thus at a given age a, there is a change in the fertility matrix $\mathbf{F}_a$ to $(\mathbf{F}_a + \epsilon\,\boldsymbol{\delta}_a^F)$, with

$$\boldsymbol{\delta}_a^F = \mathbf{D}_a\,\boldsymbol{\delta}^M + \boldsymbol{\delta}^D\,\widehat{\mathbf{M}}_a. \tag{S-36}$$

SI.5.4.2 Selection on modifiers of phenotype transition rates Phenotype transition rates $\mathbf{P}_a$ are made up of age-phenotype specific survival rates ($S_a(x)$) and probabilities of changing stage ($G_a(y,x)$). A small change in one or more of these rates at age a means that we change $\mathbf{P}_a$ to $\mathbf{P}_a \to \mathbf{P}_a + \epsilon\,\boldsymbol{\delta}_a^P$ with

$$\boldsymbol{\delta}_a^P = \mathbf{G}_a\,\boldsymbol{\delta}^S + \boldsymbol{\delta}^G\,\mathbf{S}_a. \tag{S-37}$$

But notice that this change at age a in $\mathbf{P}_a$ will also change every survivorship $\mathbf{L}_b$ at ages $b > a$. The change in survivorship is and

$$\boldsymbol{\delta}_b^L = \begin{cases} 0 & \text{for ages } b \leq a, \\ \mathbf{L}_{b|(a+1)}\, \boldsymbol{\delta}_a^P\, \mathbf{L}_a & \text{for ages } b \geq (a+1). \end{cases} \tag{S-38}$$

SI.5.5 Perturbation of parent-offspring covariance and parent variance

A small change in the phenotype-demography matrices as described in (S-27) results in a change in in the covariance between offspring birth phenotype and parent birth phenotype.

Similarly, the change in the parent birth phenotype variance is

$$\Delta \mathrm{Var}(X_0) = \Delta \left(\sum_a \phi_a \mathcal{E}\, [X_0^a]^2 \right) - 2\Delta(\mathcal{E}[X_0])\, \mathcal{E}(X_0), \tag{S-39}$$

which gives with (S-19),

$$\begin{aligned} \Delta(\sum_a \phi_a \mathcal{E}\, [X_0^a]^2) = \mathbf{e}^T \sum_a \left\{ \frac{\Delta_{\widehat{\mathbf{M}}_a \mathbf{L}_a} K - \Delta_K \widehat{\mathbf{M}}_a \mathbf{L}_a}{K^2} \widehat{\mathbf{Z}}\, \widehat{\mathbf{Z}}\mathbf{u} \right\} \\ + \frac{1}{K} \sum_a (\mathbf{e}^T \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\, \widehat{\mathbf{Z}} \boldsymbol{\delta}_u), \end{aligned} \tag{S-40}$$

and

$$\Delta(\mathcal{E}(X_0)) = \frac{\mathbf{e}^T}{K} \sum_a \left\{ \frac{\Delta_{\widehat{\mathbf{M}}_a \mathbf{L}_a} K - \Delta_K \widehat{\mathbf{M}}_a \mathbf{L}_a}{K} \widehat{\mathbf{Z}}\mathbf{u} + \widehat{\mathbf{M}}_a \mathbf{L}_a \widehat{\mathbf{Z}}\, \boldsymbol{\delta}_u \right\}, \tag{S-41}$$

the equation (5.17) of the main article.

SI.5.6 Closure of terms

A general result we will use is: given a matrix $\mathcal{Q}$ and a real variable w,

$$\begin{aligned} &\text{define } \mathcal{H} = [\mathbf{I} - z\,\mathcal{Q}]^{-1}\,, \\ &\text{then } \frac{d\mathcal{H}}{dz} = \mathcal{H}\,\mathcal{Q}\,\mathcal{H}. \end{aligned} \tag{S-42}$$

For our calculations in terms of the phenotype-demography matrices, suppose there is an age m after which fertility $\mathbf{F}_a = \mathbf{F}_m$ and survival $\mathbf{P}_a = \mathbf{P}_m$. Each sum in (S-29) can be written in two parts,

$$\mathcal{D}_1 = \sum_{a=1}^{a=(m-1)} e^{-ra}\,\mathbf{\Delta}_a + \sum_{a\geq m} e^{-ra}\,\mathbf{\Delta}_a = \mathcal{D}_{11} + \mathcal{D}_{12}, \tag{S-43}$$

$$\mathcal{D}_2 = \sum_{a=1}^{a=(m-1)} e^{-ra}\,a\,\mathbf{F}_a\,\mathbf{L}_a + \sum_{a\geq m} e^{-ra}\,a\,\mathbf{F}_a\,\mathbf{L}_a = \mathcal{D}_{21} + \mathcal{D}_{22}. \tag{S-44}$$

We have to do the sums to age $(m-1)$ as written. We seek explicit expressions for the sums that start at m. In the latter we have constant fertility and survival so

$$\mathbf{F}_{m+k} = \mathbf{F}_m, \text{ and } \mathbf{L}_{m+k} = \mathbf{P}_m^k\,\mathbf{L}_m, k \geq 0. \tag{S-45}$$

SI.5.7 Explicit form of $\mathcal{D}_{22}$

First we do the easier term $\mathcal{D}_{22}$, shown in (S-44). For a real variable w, define the matrix sum

$$\mathcal{A}(w) = \sum_{a\geq m} w^a\,\mathbf{F}_a\,\mathbf{L}_a, \tag{S-46}$$

$$= \mathbf{F}_m\,w^m\,\left[\sum_{k\geq 0} w^k\,\mathbf{P}_m^k\right]\,\mathbf{L}_m, \tag{S-47}$$

$$= \mathbf{F}_m\,w^m\,[\mathbf{I} - w\mathbf{P}_m]^{-1}\,\mathbf{L}_m,. \tag{S-48}$$

We can write $\mathcal{A}(w)$ in a computable closed form in two steps:

$$\mathcal{H}_m(w) = [\mathbf{I} - w\mathbf{P}_m]^{-1}, \text{ (Step 1)} \tag{S-49}$$

$$\mathcal{A}(w) = w^m \, \mathbf{F}_m \, \mathcal{H}_m(w) \, \mathbf{L}_m. \text{ (Step 2)} \tag{S-50}$$

Next we use (S-46) to see that

$$w \, \frac{d\mathcal{A}(w)}{dw} = \sum_{a \geq m} a \, w^a \, \mathbf{F}_a \, \mathbf{L}_a. \tag{S-51}$$

From (S-42) and (S-49) we find that

$$\frac{w \, d\mathcal{A}(w)}{dw} = m \, \mathcal{A}(w) + w^{m+1} \, \mathbf{F}_m \, \mathcal{H}_m(w) \, \mathbf{P}_m \, \mathcal{H}_m(w) \, \mathbf{L}_m. \tag{S-52}$$

Hence the final expression: we use (S-49) and (S-50), and set $w = e^{-r}$, to get

$$\begin{aligned} \mathcal{D}_{22} &= \sum_{a \geq m} e^{-ra} \, a \, \mathbf{F}_a \, \mathbf{L}_a \\ &= m \, \mathcal{A}(e^{-r}) + e^{-(m+1)r} \, \mathbf{F}_m \, \mathcal{H}_m(e^{-r}) \, \mathbf{P}_m \, \mathcal{H}_m(e^{-r}) \, \mathbf{L}_m. \end{aligned} \tag{S-53}$$

SI.5.8 Explicit form of $\mathcal{D}_{12}$

Second we turn to the more involved term $\mathcal{D}_{12}$ in (S-43) which is

$$\mathcal{D}_{12} = \sum_{a \geq m} e^{-ra} \, \boldsymbol{\Delta}_a = e^{-rm} \sum_{k \geq 0} e^{-rk} \, \boldsymbol{\Delta}_{m+k}. \tag{S-54}$$

To sort this out we need to get explicit about perturbations to fertility and survival. In general we must consider the following perturbations:

fertility $\mathbf{F}_m$ at ages $\geq m$ changes to $(\mathbf{F}_m + \epsilon \, \boldsymbol{\delta}^F)$;

survival $\mathbf{P}_m$ at ages $\geq m$ changes to $(\mathbf{P}_m + \epsilon \, \boldsymbol{\delta}^P)$;

and cumulative survival $\mathbf{L}_m$ up to age m changes to $(\mathbf{L}_m + \epsilon \, \boldsymbol{\delta}^L)$. It is important

to recognise that the change $\boldsymbol{\delta}^L$ depends only on changes at ages $< m$.

Step 1: we need the $O(\epsilon)$ change in $\mathbf{P}_m^k$. This is just the coefficient of y in $(\mathbf{P}_m + y\,\boldsymbol{\delta}^P)^k$ where y is just a real variable. For the present, we write this coefficient as $\boldsymbol{\delta}_k^P$.

Step 2: we recall (S-45): for $k \geq 0$ we have $\mathbf{F}_{m+k}\,\mathbf{L}_{m+k} = \mathbf{F}_m\,\mathbf{P}_m^k\,\mathbf{L}_m$ and therefore the linear ($O(\epsilon)$) change in this product is

$$\boldsymbol{\Delta}_{m+k} = \boldsymbol{\delta}^F\,\mathbf{P}_m^k\,\mathbf{L}_m + \mathbf{F}_m\,\mathbf{P}_m^k\,\boldsymbol{\delta}^L + \mathbf{F}_m\,\boldsymbol{\delta}_k^P\,\mathbf{L}_m. \tag{S-55}$$

Step 3: we use (S-55) to rewrite the sum in (S-54) as

$$\begin{aligned}
\mathcal{D}_{12} &= e^{-rm}\sum_{k\geq 0} e^{-rk}\,\boldsymbol{\Delta}_{m+k},\\
&= e^{-rm}\,\boldsymbol{\delta}^F\left[\sum_{k\geq 0} e^{-rk}\,\mathbf{P}_m^k\right]\mathbf{L}_m\\
&+ e^{-rm}\,\mathbf{F}_m\left[\sum_{k\geq 0} e^{-rk}\,\mathbf{P}_m^k\right]\boldsymbol{\delta}^L\\
&+ e^{-rm}\,\mathbf{F}_m\left[\sum_{k\geq 0} e^{-rk}\,\boldsymbol{\delta}_k^P\right]\mathbf{L}_m
\end{aligned} \tag{S-56}$$

Step 4: we recall from (S-49) that $\mathcal{H}_m(w) = [\mathbf{I} - w\mathbf{P}_m]^{-1}$ and rewrite (S-56) as

$$\begin{aligned}
\mathcal{D}_{12} &= e^{-rm}\,\boldsymbol{\delta}^F\,\mathcal{H}_m(e^{-r})\,\mathbf{L}_m + e^{-rm}\,\mathbf{F}_m\,\mathcal{H}_m(e^{-r})\,\boldsymbol{\delta}^L\\
&+ e^{-rm}\,\mathbf{F}_m\left[\sum_{k\geq 0} e^{-rk}\,\boldsymbol{\delta}_k^P\right]\mathbf{L}_m
\end{aligned} \tag{S-57}$$

Step 5: now we just want a closed form for the sum in the second line of (S-57). Recall that $\boldsymbol{\delta}_k^P$ is just the coefficient of y in $(\mathbf{P}_m + y\,\boldsymbol{\delta}^P)^k$. So we define the function

$$\mathcal{G}(w,y) = \sum_{k\geq 0} e^{-rk}\,(\mathbf{P}_m + y\,\boldsymbol{\delta}^P)^k = \left[\mathbf{I} - w\,(\mathbf{P}_m + y\,\boldsymbol{\delta}^P)\right]^{-1}. \tag{S-58}$$

Then we must have

$$\begin{aligned}\sum_{k\geq 0} e^{-rk}\,\boldsymbol{\delta}_k^P &= \left.\frac{\partial \mathcal{G}}{\partial y}\right|_{y=0},\\ &= w\,\mathcal{G}(w,0)\,\boldsymbol{\delta}^P\,\mathcal{G}(w,0)\\ &= w\,\mathcal{H}_m(w)\,\boldsymbol{\delta}^P\,\mathcal{H}_m(w). \end{aligned} \tag{S-59}$$

Finally, using (S-59) in (S-57) we have

$$\begin{aligned}\mathcal{D}_{12} &= e^{-rm}\,\boldsymbol{\delta}^F\,\mathcal{H}_m(e^{-r})\,\mathbf{L}_m + e^{-rm}\,\mathbf{F}_m\,\mathcal{H}_m(e^{-r})\,\boldsymbol{\delta}^L\\ &\quad + e^{-r(m+1)}\,\mathbf{F}_m\,\mathcal{H}_m(e^{-r})\,\boldsymbol{\delta}^P\,\mathcal{H}_m(e^{-r})\,\mathbf{L}_m. \end{aligned} \tag{S-60}$$

For the final expressions, we use (S-43) and (S-44). For the sums up to age $(m-1)$ we use $\mathcal{D}_{11}, \mathcal{D}_{21}$ as written. Then we use (S-60) to get $\mathcal{D}_{12}$ and (S-53) to get $\mathcal{D}_{12}$.

SI.5.9 Perturbation of transition densities

Growth and parent-offspring transition involve transition densities for a variable y conditional on a known variable x, usually taken to be proportional to a standard normal f with a mean $\mu(x)$ and variance $s(x)$. The values of y are constrained to an interval $[A, B]$ and we call the constrained transition density $\hat{f}$,

$$f = \frac{1}{\sigma\sqrt{2\pi}} \exp\left[-\frac{(x-\mu)^2}{2\sigma^2}\right] \tag{S-61}$$

$$\hat{f} = \frac{f}{K}, \quad K = \int_A^B f, \tag{S-62}$$

$$\int_A^B \hat{f} = 1, \quad \int_A^B x\hat{f} = \mu_1, \quad \int_A^B (x-\mu_1)^2\,\hat{f} = \sigma_1^2 \tag{S-63}$$

The mean $\mu(x)$ and variance $s(x)$ depend on parameters that we call θ. A change in any parameter θ affects $\hat{f}$ via μ or s, so here we simply consider changes in the

latter.

$$\partial_\theta \hat{f} = \frac{\delta_\theta f}{K} - \hat{f}\frac{\delta_\theta K}{K} \tag{S-64}$$

$$\partial_\mu f = +\frac{(x-\mu)}{\sigma^2} f, \quad \delta_\sigma f = \frac{(x-\mu)^2}{\sigma^3} f - \frac{f}{\sigma} \tag{S-65}$$

$$= \left(\frac{1}{\sigma}\right)\left[\frac{(x-\mu)^2}{\sigma^2} - 1\right] f \tag{S-66}$$

$$\partial_\mu K = +\left(\frac{K}{\sigma^2}\right)\int (x-\mu)\,\hat{f} = +K\frac{(\mu_1-\mu)}{\sigma^2} \tag{S-67}$$

$$\partial_\sigma K = K\left(\frac{1}{\sigma}\right)\left[\int \frac{(x-\mu)^2}{\sigma^2}\hat{f} - 1\right] = K\left(\frac{1}{\sigma}\right)\left[\frac{\sigma_1^2 + (\mu_1-\mu)^2}{\sigma^2} - 1\right] \tag{S-68}$$

$$\partial_\mu \hat{f} = \left[+\frac{(x-\mu)}{\sigma^2} - \frac{(\mu_1-\mu)}{\sigma^2}\right]\hat{f} = +\frac{(x-\mu_1)}{\sigma^2}\hat{f} \tag{S-69}$$

$$\partial_\sigma \hat{f} = \left(\frac{\hat{f}}{\sigma}\right)\left[(x-\mu)^2 - (\mu_1-\mu)^2 - \sigma_1^2\right]\left(\frac{1}{\sigma^2}\right) \tag{S-70}$$

$$= \left(\frac{\hat{f}}{\sigma}\right)\left(\frac{1}{\sigma^2}\right)\left[(x-\mu_1)^2 - 2\,(x-\mu_1)\,(\mu_1-\mu) - \sigma_1^2\right] \tag{S-71}$$

$$= \left(\frac{1}{\sigma}\right)\left[\frac{(x-\mu_1)^2 - \sigma_1^2}{\sigma^2} + 2\frac{(x-\mu_1)\,(\mu_1-\mu)}{\sigma^2}\right] \tag{S-72}$$

The final equations may be used to construct the perturbation matrices in (S-27)/(5.13).

Supporting tables

Table S1. Change in quantities (%) when model parameters are perturbed (Soay sheep).

	$\mathcal{E}(X_0)$	$\mathrm{Var}(X_0)$	$\mathcal{E}(X_0^P)$	$\mathrm{Var}(X_0^P)$	$\mathcal{E}(Y)$	$\mathrm{Var}(Y)$	$\mathrm{Cov}(YX_0^p)$	h^2
SurvIntL*	0.00	-0.03	-0.02	0.00	0.01	-0.11	-0.46	-0.46
SurvIntY	0.00	-0.01	-0.01	0.01	0.00	-0.03	-0.13	-0.14
SurvIntA	0.00	-0.01	0.00	0.00	0.01	-0.05	-0.21	-0.20
SurvIntS	0.00	-0.01	0.00	-0.01	0.00	-0.03	-0.11	-0.10
SurvSlpL	0.00	-0.30	-0.15	-0.71	0.15	-1.24	-4.38	-3.69
SurvSlpY	0.01	-0.09	-0.07	0.05	0.05	-0.38	-1.73	-1.79
SurvSlpA	0.04	-0.14	-0.01	-0.11	0.15	-0.94	-3.75	-3.65
SurvSlpS	-0.02	-0.27	-0.03	-0.32	0.04	-0.83	-2.67	-2.36
TwIntL	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwIntY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwIntA	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwIntS	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwSlpL	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwSlpY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwSlpA	0.00	0.00	0.00	0.00	0.00	-0.01	-0.01	-0.01
TwSlpS	0.00	-0.01	0.00	-0.01	0.00	-0.04	-0.13	-0.11
PrIntL	-0.03	0.24	-0.01	0.11	-0.03	0.22	0.66	0.54
PrIntY	0.00	-0.01	0.00	0.00	0.00	-0.02	0.04	0.04
PrIntA	0.01	-0.01	0.00	0.00	0.02	-0.08	-0.22	-0.22
PrIntS	0.00	-0.02	0.00	-0.02	0.00	-0.05	-0.15	-0.13
PrSlpL	-0.41	2.84	-0.13	1.37	-0.35	2.59	4.53	3.12
PrSlpY	-0.08	-0.20	-0.04	-0.10	-0.04	-0.34	0.53	0.62
PrSlpA	0.15	-0.03	0.14	0.11	0.40	-1.58	-4.11	-4.21
PrSlpS	-0.04	-0.41	-0.05	-0.46	0.04	-1.13	-3.76	-3.31
GrMIntL	0.01	0.01	0.00	0.02	0.01	0.00	-0.01	-0.03
GrMIntY	0.01	0.02	0.01	0.03	0.01	0.02	-0.01	-0.03
GrMIntA	0.01	0.07	0.02	0.09	0.02	0.04	-0.06	-0.15
GrMIntS	0.00	-0.01	0.00	-0.01	0.00	-0.02	-0.08	-0.07
GrMSlpL	0.07	0.19	0.08	0.17	0.09	0.02	0.67	0.49
GrMSlpY	0.10	0.43	0.12	0.45	0.11	0.30	0.40	-0.05
GrMSlpA	0.30	1.55	0.39	2.01	0.38	0.90	-0.71	-2.67
GrMSlpS	-0.04	-0.15	-0.03	-0.14	-0.01	-0.57	-2.09	-1.95

Table S1. continued.

	$\mathcal{E}(X_0)$	$\mathrm{Var}(X_0)$	$\mathcal{E}(X_0^P)$	$\mathrm{Var}(X_0^P)$	$\mathcal{E}(Y)$	$\mathrm{Var}(Y)$	$\mathrm{Cov}(Y X_0^p)$	h^2
GrVIntL	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
GrVIntY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
GrVIntA	0.00	0.00	0.00	0.01	0.00	0.00	0.01	0.00
GrVIntS	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
GrVSlpL	0.01	0.03	0.01	0.04	0.01	0.03	0.11	0.07
GrVSlpY	0.00	0.03	0.00	0.05	0.00	0.02	0.05	-0.01
GrVSlpA	0.00	0.09	0.01	0.21	0.00	0.08	0.12	-0.09
GrVSlpS	0.00	0.00	0.00	0.00	0.00	-0.01	-0.07	-0.07
InMIntL	0.01	-0.11	0.00	-0.02	0.01	-0.10	-0.06	-0.04
InMIntY	0.01	-0.01	0.00	-0.02	0.01	-0.01	0.05	0.07
InMIntA	0.06	0.10	0.05	0.07	0.06	0.07	-0.04	-0.12
InMIntS	0.01	0.01	0.01	-0.01	0.02	0.00	0.00	0.01
InMSlpL	0.09	-1.35	0.00	-0.30	0.08	-1.25	0.02	0.33
InMSlpY	0.13	-0.10	0.09	-0.29	0.12	-0.17	1.45	1.75
InMSlpA	1.30	2.55	1.25	2.13	1.35	1.87	1.52	-0.60
InMSlpS	0.30	0.28	0.26	-0.13	0.38	0.16	0.19	0.32
InV1L	0.00	0.01	0.00	0.00	0.00	0.01	-0.01	0.00
InVIntY	0.00	0.01	0.00	0.01	0.00	0.01	0.01	0.00
InVIntA	0.00	0.10	0.01	0.13	0.00	0.10	0.12	-0.01
InVIntS	0.00	0.02	0.00	0.02	0.00	0.03	0.02	0.00
InVSlpL	0.00	0.15	0.00	-0.05	0.00	0.13	-0.06	-0.01
InVSlpY	0.00	0.22	0.02	0.17	0.00	0.20	0.18	0.01
InVSlpA	0.01	2.13	0.20	2.86	0.02	2.17	2.68	-0.18
InVSlpS	0.00	0.50	0.05	0.55	0.00	0.62	0.53	-0.02

*Model parameters were perturbed upwards by adding 0.01. Quantities were calculated numerically. Model parameter names are written in CamelCase forming compounds of the following abbreviations in order of appearance: survival (Surv), intercept (int), lambs (L), yearlings (Y), prime-aged adults (A), senescents (S), slope (slp), twinning rate (Tw), probability of reproduction (Pr), growth (Gr), mean (M), variance (V), inheritance (In).

Table S2. Change in quantities (%) when model parameters are perturbed (roe deer).

	$\mathcal{E}(X_0)$	$\mathrm{Var}(X_0)$	$\mathcal{E}(X_0^P)$	$\mathrm{Var}(X_0^P)$	$\mathcal{E}(Y)$	$\mathrm{Var}(Y)$	$\mathrm{Cov}(Y X_0^p)$	h^2
SurvIntY*	-0.01	0.00	-0.02	0.01	0.00	0.01	0.02	0.00
SurvIntA	0.00	-0.01	0.00	0.00	0.02	0.00	-0.30	-0.30
SurvIntS	0.00	0.01	0.00	0.01	0.01	0.01	-0.05	-0.06
SurvSlpY	-0.11	0.00	-0.17	-0.12	-0.03	0.07	-0.09	0.02
SurvSlpA	0.13	-0.13	0.02	-0.16	0.36	-0.10	-6.02	-5.87
SurvSlpS	0.07	0.20	0.08	0.21	0.16	0.31	-1.32	-1.52
TwIntY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwIntA	-0.01	-0.01	-0.01	-0.01	0.00	0.00	0.01	0.02
TwIntS	0.00	0.00	0.00	0.00	0.00	0.01	-0.01	-0.02
TwSlpY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TwSlpA	-0.12	-0.20	-0.12	-0.24	-0.06	-0.15	-0.12	0.12
TwSlpS	0.04	0.12	0.04	0.12	0.06	0.15	-0.33	-0.45
PrIntY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PrIntA	-0.02	0.00	-0.02	0.00	-0.01	0.01	0.08	0.08
PrIntS	0.00	0.00	0.00	0.00	0.00	0.01	-0.02	-0.03
PrSlpY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PrSlpA	-0.27	-0.14	-0.29	-0.15	-0.16	-0.03	0.86	1.01
PrSlpS	0.04	0.12	0.05	0.12	0.08	0.14	-0.54	-0.66
GrMIntY	0.02	-0.05	0.02	-0.05	0.02	-0.05	-0.07	-0.02
GrMIntA	0.05	0.09	0.05	0.09	0.06	0.09	-0.01	-0.10
GrMIntS	0.00	0.02	0.00	0.02	0.00	0.03	0.01	-0.01
GrMSlpY	0.32	-0.68	0.28	-0.69	0.30	-0.72	0.50	1.21
GrMSlpA	1.25	2.83	1.26	2.72	1.52	2.81	2.33	-0.38
GrMSlpS	0.07	0.44	0.08	0.50	0.13	0.80	0.31	-0.19
GrVIntY	0.00	0.02	0.00	0.01	0.00	0.01	0.01	0.00
GrVIntA	0.00	0.05	0.00	0.05	0.00	0.05	0.05	-0.01
GrVIntS	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00
GrVSlpY	0.02	0.25	0.03	0.21	0.02	0.19	0.23	0.02
GrVSlpA	0.08	1.17	0.11	1.26	0.09	1.28	1.24	-0.02
GrVSlpS	0.00	0.08	0.01	0.10	0.01	0.15	0.09	0.00
InMIntY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
InMIntA	0.07	-0.08	0.06	-0.08	0.07	-0.10	-0.10	-0.02
InMIntS	0.01	0.05	0.01	0.05	0.01	0.07	0.06	0.01
InMSlpY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
InMSlpA	1.64	-0.71	1.51	-0.84	1.54	-1.33	0.70	1.56
InMSlpS	0.16	1.28	0.19	1.50	0.25	1.95	1.67	0.17

Table S2. continued.

	$\mathcal{E}(X_0)$	$\mathrm{Var}(X_0)$	$\mathcal{E}(X_0^P)$	$\mathrm{Var}(X_0^P)$	$\mathcal{E}(Y)$	$\mathrm{Var}(Y)$	$\mathrm{Cov}(YX_0^p)$	h^2
InVIntY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
InVIntA	0.00	0.16	0.01	0.15	0.00	0.15	0.15	0.00
InVIntS	0.00	0.01	0.00	0.02	0.00	0.02	0.02	0.00
InVSlpY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
InVSlpA	0.03	3.79	0.14	3.63	0.02	3.52	3.62	-0.01
InVSlpS	0.00	0.35	0.01	0.50	0.00	0.59	0.49	-0.01

*Model parameters were perturbed upwards by adding 0.01. Quantities were calculated numerically. Model parameter names are written in CamelCase forming compounds of the following abbreviations in order of appearance: survival (Surv), intercept (Int), yearlings (Y), prime-aged adults (A), senescents (S), slope (slp), twinning rate (Tw), probability of reproduction (Pr), growth (Gr), mean (M), variance (V), inheritance (In).

Supporting figures

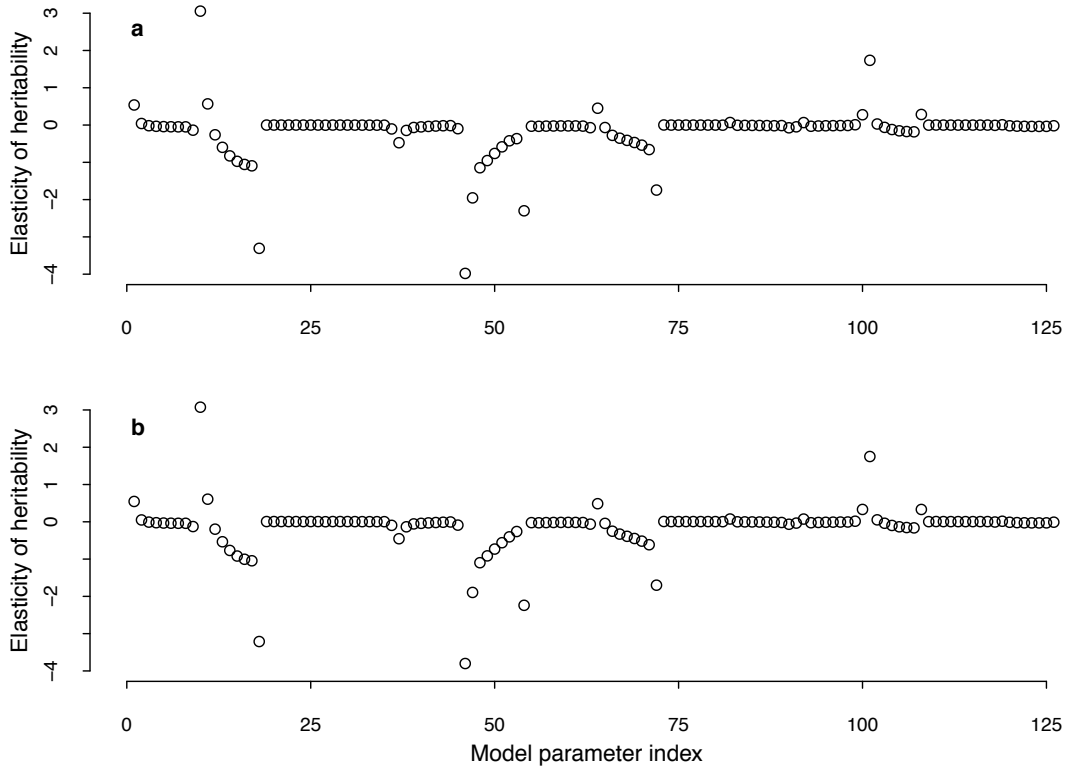

Figure S1. The analytically (a) and numerically (b) computed elasticities of heritability with respect to changes in the model parameters of the Soay sheep IPM. The two ways of computation give very similar results.

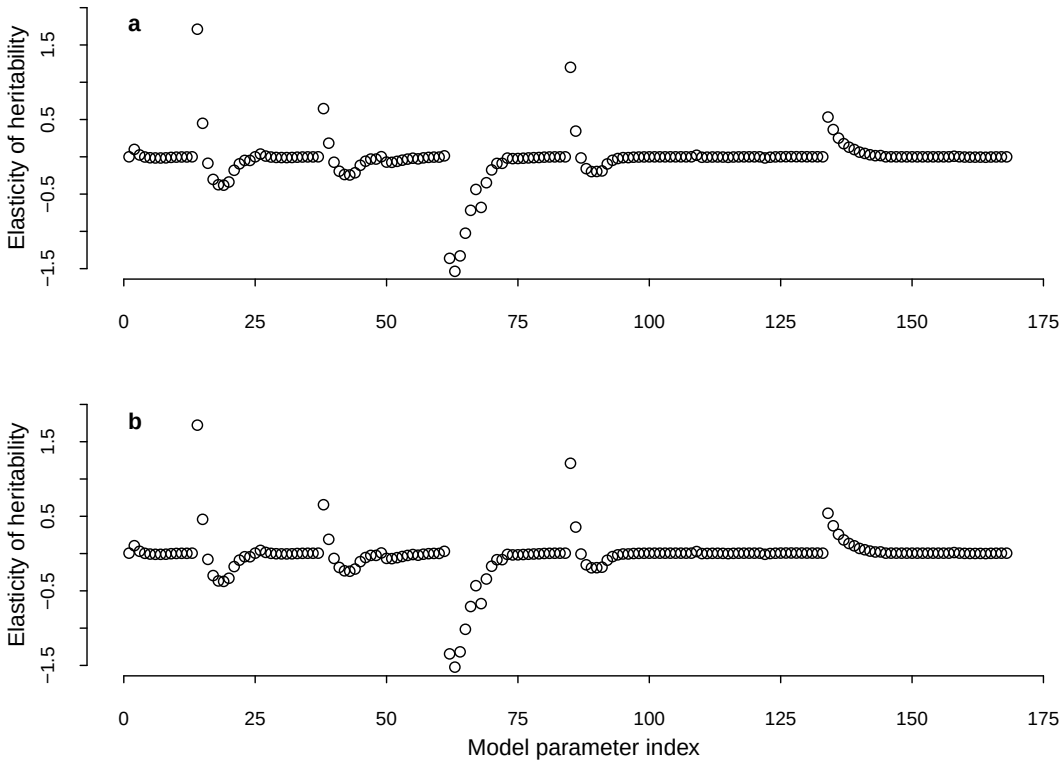

Figure S2. The analytically (a) and numerically (b) computed elasticities of heritability with respect to changes in the model parameters of the roe deer IPM. As for Soay sheep, the two ways of computation give very similar results.

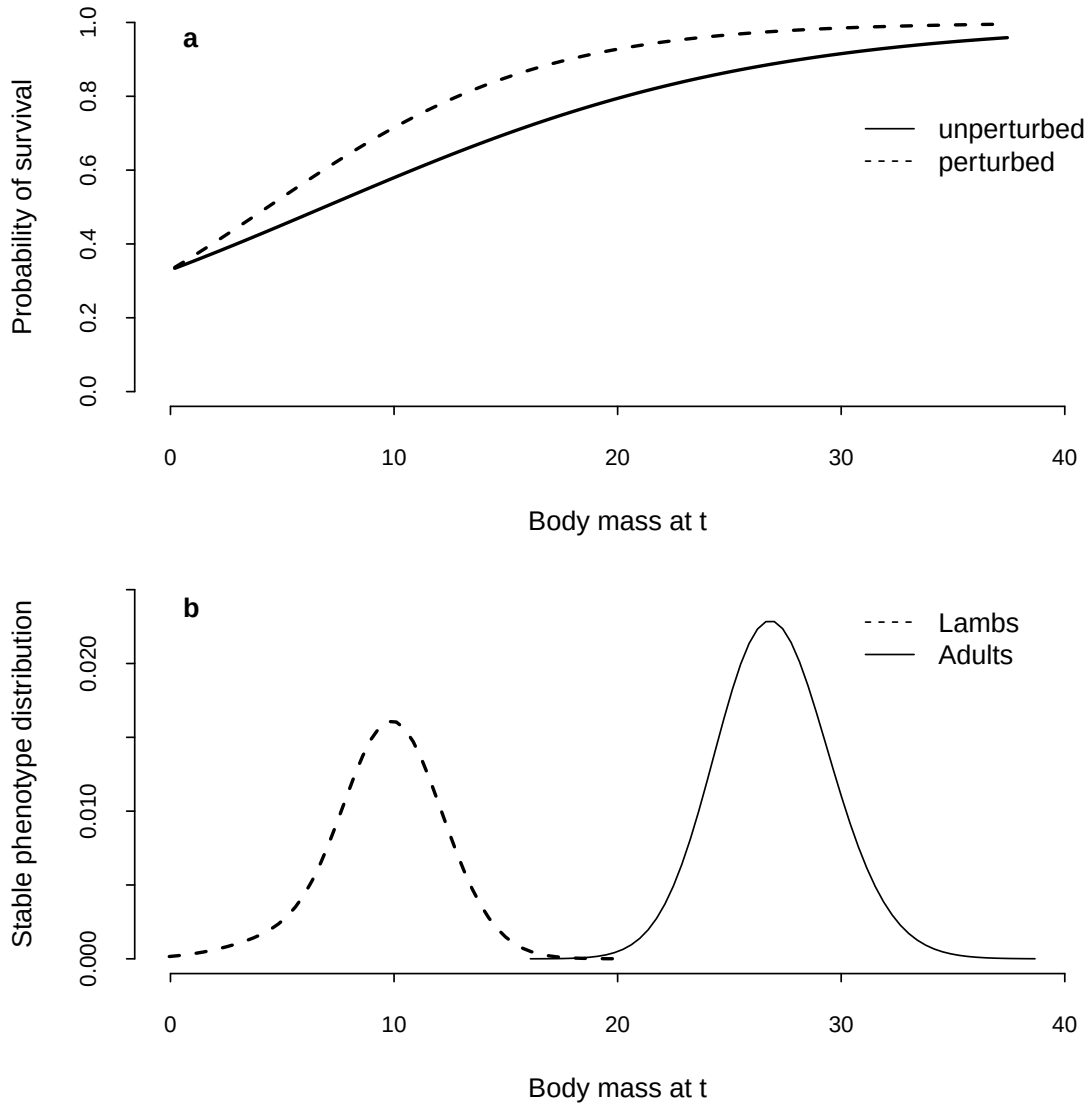

Figure S3. Schematic of the linearisation effect using a linearised survival function as an example. The effect of perturbing the slope of the survival function (a) on viability selection depends on the phenotype distribution (b). The same perturbation in (a) makes survival probabilities less equal across the lamb phenotype values and more equal across the adult phenotype values (b). Therefore the same perturbation increases viability selection for lambs and decreases viability selection for adults.

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CHAPTER 6

Discussion

In this discussion, I briefly evaluate the significance of the findings presented in this thesis. In particular, I examine my work with regard to the underlying motivations of the research, and discuss links to potential future advances in the field. In discussing the different topics, I will keep to the order in which they featured in this thesis. I will therefore first discuss the role of males in population dynamics, then the development of methods to estimate age-specific mortality, before ending with some thoughts about the use of quantitative genetics methods to study evolution in wild animal populations.

Do males need to be included in models of population dynamics?

The answer to this question is probably no, at least usually not. My initial interest in the role of males in population dynamics was driven by the perplexing observation that population ecologists routinely disregard one half of their study populations. After all, about every second vertebrate is a male, and yet males are usually omitted from population models. I have since learned that it is not only sex structure that often fails to find its way into population models. Other factors

that are also sources for potentially important, longitudinal or between-individual variation in demographic rates are also often excluded. Among these factors are population density, the composition of populations, animal movement, demographic and environmental stochasticity, age, body condition, social hierarchy or network position, habitat quality, disease status, hibernation length, and alternative morphs (Packer et al. 1999; Coulson 2001; Mosser et al. 2009; Morales et al. 2010; Ozgul et al. 2010; Smallegange and Coulson 2011; Plard et al. 2012; Vander Wal et al. 2015). With this knowledge, the question of whether males need to be included in models of population dynamics becomes part of a larger problem. Why shouldn't we include all factors known to influence population dynamics? In my opinion, the answer to this question is because including these factors is not necessarily useful for the specific purpose of a population model, which is to find the answer to a particular biological question.

To use a population model for gaining biological insight, we need to be able to construct it, parameterise it, analyse it, and draw a conclusion from it. When confronted with the question of whether to include an additional factor, the model developer therefore first needs to decide whether it is feasible to construct a more complex model. Then she has to evaluate whether data are available to parameterise it, and whether the methodology exists, or can be developed, to analyse it. But the key question is whether including the additional factor makes the conclusion drawn from the model clearer as an answer to the study question. Counter-intuitively, adding information to the model can dilute inference if added factors make the model's results harder to interpret. Therefore, the effort of overcoming the technical difficulties of including extra factors in the model is only justified if the factor is necessary to draw a conclusion.

The technical challenges associated with constructing and analysing sex structured population models are considerable. This is because including sex structure usually changes models in nature from linear, analytically analysable models to non-

linear models (Caswell 2001). Furthermore, demographic data for both sexes from free-living populations are rare. The effort to overcome these difficulties is only justified if adding sex structure to the model is necessary to answer the study question. This is the case if either the study question itself focuses on the consequences of the interactions between males and females for population dynamics (Shelton 2010; Lee et al. 2011; Schindler et al. 2013), or if males are known to heavily influence population dynamics so that not including them would alter inferences (Jenouvrier et al. 2014).

Under what circumstances do males influence population dynamics drastically enough to warrant their inclusion in population models? This has only been quantified for a few populations and for three mechanisms by which males can influence population dynamics. Firstly, males have been found to limit female fecundity if there are insufficient mates for all fertilisable females in the population. This occurs in species with a monogamous mating system (Jenouvrier et al. 2010), when sex ratios are extremely female-biased (Milner-Gulland et al. 2003), or population sizes are small (Lee et al. 2011). Secondly, males can decrease female survival by sexually harassing females when sex ratios are heavily male-biased (Le Galliard et al. 2005). Thirdly, males can affect population dynamics via infanticide (Whitman et al. 2004). In addition to these three empirically supported mechanisms, theory suggests the existence of additional mechanisms (Rankin and Kokko 2007; Mysterud et al. 2002). In Chapter 2, I have evaluated the consequences for population dynamics of a previously unquantified mechanisms by which males may play a role for population dynamics. I tested whether males affect the dynamics of populations via the inheritance of phenotype from fathers to offspring. Using data from African lions, I found that they do, but the effect size was too small to qualify males as an indispensable element of phenotype structured population models for this species.

Despite having just made a case against the general inclusion of males in population models, I am still convinced that deepening our understanding of sex

structured population dynamics is a worthwhile endeavour. I believe that well-constructed two-sex models, that model demographic interactions between the sexes, can potentially provide new perspectives on familiar study questions about the evolutionary forces that have shaped sex differences in life history and morphology (Promislow 2003; Maklakov et al. 2009; Traill et al. 2014), sex allocation (Edwards and Cameron 2014), and sex-specific parental investment (Trivers and Willard 1973). Furthermore, even if males do not affect population dynamics in undisturbed populations, studying the consequences of abnormally skewed sex ratios via human impact may be an interesting future application for two-sex models. For example, how many male mosquitos, genetically modified to father non-viable daughters, have to be released to depress population growth (de Valdez et al. 2011)? How many males can be shot for trophies if the goal is sustainable population growth (Milner-Gulland et al. 2003; Whitman et al. 2004)? How does the skewed sex ratio at the invasion front affect the spread of introduced species (Miller and Inouye 2012)? With advances in sex structured population modelling, answering these questions will become possible.

Why take the time to improve demographic inference for both sexes?

When I worked on building a two-sex model for African lions, I was perplexed to find that estimates of age-specific mortality do not exist for male lions. Lions males are hunted for trophies (Lindsey et al. 2012), sometimes to such degree that the population becomes depleted of males (Loveridge et al. 2007; Becker et al. 2013). This creates a situation where sex ratios become skewed towards dominance by females to a degree that can affect population growth. In this case, the number of males matters for population growth not by limiting female fecundity, but because high male mortality destabilises the social structure of lion populations. The frequent deaths of adult males result in a higher frequency of take-overs of female prides through coalitions among males. These take-overs are associated with dependent offspring

being killed by the incoming males (Whitman et al. 2004). To study the population consequences of trophy hunting, numerous models of lion population dynamics have been constructed (Whitman et al. 2004, 2007; Packer et al. 2011; Becker et al. 2013). However, none of them uses age-specific mortality information for males because no such estimates exist.

These mortality estimates do not exist because lion males, as with many other mammalian males, disperse around the age of maturity (Pusey and Packer 1987). If dispersing males leave the study areas that are monitored by field studies, they are usually lost for data collection. Therefore, when males go missing, their fate is unknown; they may have either died or dispersed. The estimation of mortality from incomplete data has been one of the focuses of biostatistical research in the last decade (Schaub et al. 2004; Clark et al. 2005; Colchero and Clark 2012). In Chapter 3, I have built on previous models and developed a Bayesian framework that can be used to infer mortality for both sexes for species with sex-biased natal dispersal. Estimating something from nothing, in this case mortality from unobserved deaths, is technically challenging. Nevertheless, I argue that investing the time to develop methods to provide standard life history descriptors such as age-specific mortality for both sexes is not only worthwhile to parameterise two-sex population models, it is also a necessary step towards improving our understanding of the evolution of ageing and life span.

Why gather demographic summary statistics?

In Chapter 4, I applied the newly-developed model (Chapter 3) to estimate age-specific mortality rates for both sexes in two populations of lions. This has filled the gap of the missing male mortality information. Furthermore, gathering this information for many species and populations will be crucial to determine the evolutionary forces that shape life histories.

Age-specific mortality rates are important life history descriptors. Since vi-

ability selection is a strong determinant of individual fitness, mortality rates are expected to be under natural selection (Stearns 1992). The way in which evolutionary forces have shaped age-specific mortality after the age of maturity has received considerable attention by researchers focussing on the evolution of ageing (Williams 1957; Kirkwood 1977; Monaghan et al. 2008). A decrease in mortality rates after birth, and an increase in them later in life, are now considered universal features of the life history of mammals and birds in free-living populations (Levitis 2011; Monaghan et al. 2008). Moreover, comparative evidence supports the existence of a female survival advantage in species with sexual size dimorphism and strong male mating competition (Promislow 1992; Promislow et al. 1992; Clutton-Brock and Isvaran 2007). But existing theory has little explanatory power for predicting particular shapes of age-specific mortality rates, or sex differences in them, for wild populations (Clutton-Brock and Isvaran 2007; Monaghan et al. 2008; Jones et al. 2014).

Theory predicts that the main evolutionary drivers of age-specific mortality are life history trade-offs, predictability of the environment, and the level of extrinsic mortality (Stearns 1992; Jones et al. 2008b; Williams et al. 2006). However, existing theory has not gone beyond explaining broad patterns (Jones et al. 2014). Since we generally cannot conduct mortality experiments in the field, widening the theory to explain the evolution of age-specific mortality hinges on the accumulation of comparative mortality information across species, populations, and time. Only by using these kind of data can we test for associations between age-specific mortality rates and factors such as mode of locomotion, growth patterns, diet, ecosystem, environmental stochasticity, and level of extrinsic mortality (Healy et al. 2014). Therefore, multiple demographic data repositories collect information for future analyses (Jones et al. 2008a; Salguero-Gómez et al. 2015). Estimating individual age-specific mortality rates may therefore be unrewarding in the short-term, since these studies tend to be descriptive, but this investment will pay off in the future in comparative

frameworks.

Can we obtain unbiased estimates of heritability for wild animal population?

Biologists have long applied quantitative genetics methods to predict evolution (Falconer and Mackay 1996; Lynch and Walsh 1998). Quantitative genetics methods successfully predict evolutionary change under controlled laboratory or farm conditions using the breeder's equation (Lynch and Walsh 1998). But when applying these methods to free-living populations, biologists find a frustrating mismatch between predictions of expected evolutionary change in a quantitative character and actually observed phenotypic change. Observed changes are lower than predicted from estimates of character heritability and directional selection (Merilä et al. 2001). Various hypotheses exist to explain this dilemma of 'unexplained stasis' (Merilä et al. 2001). Are we measuring one of the components of the breeder's equation incorrectly? Does the direction of selection fluctuate through time? Does selection act on the non-heritable component of the phenotype? Are traits correlated? Are effects masked by changes in the environment? Or is it a problem of insufficient statistical power? My findings in this thesis can contribute to this discussion.

In Chapter 5, I have shown that the estimates of heritability of a quantitative character for species where the phenotype develops with age is sensitive to changes in viability and fertility selection, in the development of the phenotype with age, and in the inheritance of phenotypes from parents to offspring. Under controlled conditions, these processes can be held relatively constant over the lifetime of a parent cohort. However, in wild populations, they vary considerably through time. Methods to estimate heritability therefore try to make statistical corrections of the observed phenotypes to account for these sources of variation (Wilson et al. 2010). However, the degree of interaction between the phenotypic selection and phenotypic transition processes that I have illustrated in this thesis, raises the question whether this is possible. Our findings support the hypothesis that the misspecification of the

heritability component of the breeder's equation is one of the reasons that predicting evolution for wild animal population has proven challenging. In the light of these findings, future work will have to establish whether the statistical assumptions underlying quantitative genetics methods can be met for data from wild populations. If this is not the case, then new methods to study evolution in the field need to be developed.

What remains left to say?

Demographic approaches bridge ecology and evolution. This thesis illustrates some of the types of questions that can be asked by using these approaches. It contributes towards widening the scope of these questions, particularly with the fusion of quantitative genetics and structured population modelling. However, not every demographic research topic has such potentially high impact. Expanding the theory of sex structured population dynamics, developing methods to estimate age-specific mortality from incomplete data, and estimating age-specific mortality are smaller steps towards understanding how populations develop over ecological time scales. Nevertheless, this knowledge will ultimately contribute to an understanding of how evolution has given rise to life as we know it.

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Appendix—Other work in preparation

Coulson, T., J. A. Barthold, F. Plard, A. Ozgul, and J.-M. Gaillard (in prep).

From inheritance functions to heritability: Quantitative genetics from structured models.

Title

From inheritance functions to heritability: Quantitative genetics from structured models

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Abstract

There are two approaches widely used to study phenotypic trait dynamics: quantitative genetics and integral projection models (IPMs). Application of both approaches is typically entirely phenotypic, although explicit genetic information has also been incorporated into both. Both approaches use information on phenotypic similarity between related individuals. However, the approaches use this information in different ways: the inheritance function in an IPM is different from the heritability of quantitative genetics. The aim of this paper is to better link the two approaches, and to explain their similarities and differences. We start by providing some background into both approaches. Second, we derive a generalised version of the Breeders equation in IPM form, and explain how it can be parameterised using quantitative genetic approaches. Third, we describes conditions for stasis within the IPM form of the Breeders equation. Fourth, we explain linkages between the IPM form of the Breeders equation and standard IPMs. This reveals the linkages between the survival and recruitment functions of IPMs and selection in the Breeders equation, and between the growth and inheritance functions of IPMs and the heritability of the Breeders equation. Finally, we show how IPMs can be expanded to include the additive components of the phenotype assumed by quantitative geneticists.

Introduction

There are two commonly used approaches to studying the dynamics of phenotypic traits. One approach, quantitative genetics, has grown out of animal and plant breeding (Bulmer et al. 1980). The second approach, integral projection modelling, has developed from structured population modelling (Ellner and Rees 2006). Both approaches are primarily phenotypic in that information about genes is not usually incorporated into analyses and models. Both approaches also rely on associations

between relatives, and, when the approaches are entirely phenotypic, an assumption about the additive effect of multiple genes is required to draw evolutionary conclusions from the models. However, the approaches also have various differences. These differences have generated some confusion, particularly concerning differences between the heritability of quantitative genetics and the inheritance function of integral projection models (IPMs). The primary aim of this paper is to provide further insights between the two approaches. The paper is written for the empiricist who may be wondering about the relative merits of using IPMs and quantitative genetics to study phenotypic change. Use both in tandem!

Quantitative genetics has long been used to study phenotypic change (Falconer 1960). In this approach similarities between related individuals are used to infer the additive genetic architecture underpinning the trait of interest. This means that the genotype-phenotype map is considered to be simple, with large numbers of loci making small, non-interactive, contributions to the expressed phenotype. The sum of these small contributions within an individual is the individual's breeding value, and is termed A_i. Non additive genetic processes, including environmental effects, inbreeding and epistasis, can also contribute to the genotype. These are often combined into a non additive genetic component of the phenotype, E. Consequently at the core of quantitative genetics is the decomposition of an individual's phenotype, $z_i = A_i + E_i$. Selection occurs on the phenotype and evolution proceeds until the fittest possible breeding value reaches fixation. In order to stop this process, potentially maintaining variation in breeding values within the population, some sort of "brake" is required. This can be a genetic correlation with an unmeasured character, stabilising selection or a negative covariance between A_i and E_i (Merilä et al. 2001). The additive genetic variance is the variance in breeding values across individuals. It is easier to robustly estimate the additive genetic variance. Individual breeding values are much harder to robustly estimate (Hadfield et al. 2010). The additive genetic variance can be calculated as the covariance in character val-

ues measured between parental and offspring traits measured at the same age, or, more generally by comparing trait values across individuals of known relatedness (Lynch et al. 1998). The additive genetic variance is frequently scaled by the total phenotypic variance of parents to generate a heritability (Jacquard 1983). This can be estimated as the slope of a parent-offspring regression, where parent and offspring traits are measured at the same age (Kempthorne 1957), as well by use of the animal model which provides an estimator of the parent-offspring regression slope. The quantitative genetic approach has proven extremely insightful, but empirical application to free-living species is routinely plagued by the challenge of identifying the brake stopping evolution and maintaining additive genetic variation for traits under consistent directional selection (Merilä et al. 2001).

The second approach, integral projection modelling, is considerably more recent than the quantitative genetic approach (Easterling et al. 2000). It uses discrete time structured models of continuous characters. Specifically, the approach describes the temporal dynamics of a character distribution (Coulson et al. 2010). At each time step, functions describe how mass is added to, removed from, and shuffled around within a distribution. The distribution can be uni- or multivariate (Ellner and Rees 2006, Schindler et al. 2013), and can consist of a mix of discrete and continuous traits. There are four classes of functions in an IPM. The first is a survival function that removes mass from the distribution, while the second is a development function that moves mass around within the distribution. The third and fourth functions describe how mass is added to the character distribution. The recruitment function describes how much mass is to be added as a function of parental characters, while the inheritance function describes where within the distribution the mass is to be added by providing a map from parental to offspring characters. IPMs consequently characterise the birth process, describing how mothers in a given phenotypic state produce offspring in a particular phenotypic state (Coulson 2012, Merow et al. 2014, Rees et al. 2014).

There are various differences between the quantitative genetic and structured modelling approaches, both in philosophy, but also in application. First, the quantitative genetic approaches always predicts change across generations. In contrast, integral projection models iterate forward a character distribution on a timescale appropriate to the life history of the species under study (Easterling et al. 2000). For invertebrate species, like mites, the time step may be as short as a single day (Smallegange et al. 2014), while for seasonally breeding mammals the appropriate time step is a year (Ozgul et al. 2010). IPMs consequently do not work on a per generation time step. However, the dynamics over the shorter time steps on which IPMs operate can be combined to examine dynamics per generation (Steiner et al. 2014).

A second key difference between IPMs and quantitative genetic approaches is that IPMs do not focus on just the mean (or a single moment) of the character under investigation, but instead they track the dynamics of the entire character distribution. Because they model the dynamics of the entire distribution, the dynamics of the mean, or any moment of the distribution, can be examined from model predictions (Coulson 2012).

The third difference between quantitative genetic approaches and IPMs concerns model formulation. In statistical quantitative genetics it is necessary to correct for fixed and random effects that can influence phenotypic characters (Kruuk et al. 2000). For example, parental age, environmental variation and cohort must be included as fixed effects in analyses, and it is desirable to fit parental ID into models as random effects (Wilson et al. 2010). Exactly the same fixed and random effects can be fitted into statistical functions used to parameterise IPMs (Rees and Ellner 2009). However, a choice now arises when working with IPMs that does not present itself when conducting a statistical quantitative genetic analysis: should the fixed and random effects be included in the IPM, or should the IPM simply iterate forward the distribution of the statistically corrected phenotype? Both approaches

are possible, and the choice of which route to follow will be specific to the question being asked. We point interested readers to Coulson (2012) to see the way that density can be used to statistically correct parameter values to use within an IPM, or incorporated into the model explicitly.

The final key difference we will focus on is to do with the way IPMs are analysed. IPMs are usually analysed at equilibrium. This means, by definition, that the change in the phenotypic mean between times t and $t + 1$ is zero: $\Delta\overline{z} = 0$. Evolution can be explored within an IPM in a phenomenological manner by asking how $\overline{z}$ changes when an aspect of the model is perturbed (Coulson et al. 2010). For example, to understand why a phenotypic covariance between parental and offspring characters takes a particular value, it is informative to explore how it changes when the survival, recruitment, development and inheritance functions are altered. IPMs and structured models in general, can be analysed at points other than at equilibrium (Fox and Gurevitch 2000), but relatively little work has been conducted in this area. In contrast to IPMs, empirical quantitative genetics is primarily about change in $\Delta\overline{z}$ over a single generation: $\Delta\overline{z} \neq 0$. That, in part, is because the methods were designed to identify which individuals to breed from, and to then impose strong selection to create an offspring cohort with desirable characteristics (Lush 1943). Somewhat ironically, quantitative genetics of free-living species has focused on why $\Delta\overline{z} = 0$ when it is predicted not to be so because directional selection operates on a heritable character (Merilä et al. 2001). Models analysed at equilibrium could well help solve this dilemma.

In spite of these differences between the two approaches, it is possible to calculate many key quantitative genetic parameters from IPMs including the parent-offspring phenotypic covariance (an estimate of the additive genetic variance) and the heritability (estimated as the slope of a parent-offspring regression) (Coulson et al. 2010). From a statistical standpoint there are more powerful approaches to estimating the heritability than parent-offspring regressions including the animal model

(Bulmer et al. 1980). The great power of the IPM approach is that analysis of the model helps provide insight into how different functions and parameters impact the heritability (Coulson et al. 2010). We expand on this approach in this paper.

Although heritability can be calculated from IPMs, and although the derivation of how to do so involves all functions in the model, there has been some confusion between the heritability and the inheritance function (Hedrick et al. 2014). The inheritance function and the heritability are two different things that should not be confused. The heritability is the proportion of phenotypic variance attributable to the additive effects of genes (Falconer 1960), and compares the values of parental and offspring characters at the same ages. In contrast, the inheritance function is the parental character measured at time t regressed against offspring character measured when the offspring recruits to the population at time $t+1$ (Coulson 2012). The parental and offspring characters are consequently measured at different ages. Unlike the heritability, one (generally) cannot make any inference about the inheritance mechanisms from examination of the slope of the inheritance function. The inheritance function is agnostic to mechanism – it includes additive genetic effects and all other sources of inherited variation, including cultural inheritance when applied to species with cultural transmission. It was deliberately named the inheritance function to be explicit that it should not be interpreted as reflecting any particular type of transmission. The inheritance function includes any genetic transmission from parent to offspring, as well all other forms of transmission. To claim it does not adequately capture such transmission is to misrepresent the function. The inheritance function has not previously been separated into additive genetic and non additive genetic components.

There are several objectives of this paper. First, we provide sufficient background to quantitative genetics and integral projection models to allow the reader not familiar with either approach to gain sufficient understanding to follow the remainder of the paper. Next, we write down a form of the Breeders equation in IPM form, before

discussing requirements from stasis for this model, and IPMs in general. Third, we explain how different functions in an IPM influence the slope of a parent offspring regression. To do this, we focus on model projections made over a single generation rather than analysing the model at equilibrium. We consequently initially do not explore the feedback between a change in the phenotype distribution and downstream fitness consequences. We then briefly consider differences between IPMs analysed at equilibrium, and those analysed for a single generation. Finally, we show how IPMs can be extended to model the dynamics of the $A + E$ – the additive and non additive genetic components of the phenotype.

Technical Background

We start by providing background information on the heritability h^2, IPMs, and how h^2 can be calculated from IPMs.

At the heart of quantitative genetics is the Breeders equation (equation (A-1)). It describes additive genetic change in the mean value of a phenotypic trait $\overline{z}$ from one generation to the next $\Delta\overline{\mathbf{z}}$ as a function of (i) selection on the trait $\mathbf{fi}$, (ii) the phenotypic variance of the trait amongst parents $\mathbf{P}$, and (iii) the additive genetic variance for the trait $\mathbf{G}$ (Lush 1943). Traits can be univariate or multivariate (Lande 1976). In the multivariate case $\mathbf{P}$ and $\mathbf{G}$ are square matrices with dimensions equal to the number of traits; in the univariate case they are all scalars. In the multivariate case $\mathbf{fi}$ and $\Delta\overline{\mathbf{z}}$ are single column vectors with the number of rows equal to the number of traits. The Breeders equation states,

$$\Delta\overline{\mathbf{z}} = \mathbf{P}^{-1}\mathbf{G}\mathbf{fi}. \tag{A-1}$$

In the multivariate case $\mathbf{P}$ is a variance-covariance matrix describing the variance in phenotypic trait values among parents for each trait, and the covariance between these traits; $\mathbf{G}$ is an equivalent matrix for breeding value variances and covariances;

fi is a vector measuring the strength of selection (Lande 1976). In the univariate case there is only a single variance for a sole phenotypic trait, and a single variance from the breeding value distribution; the model can be thought of as operating with matrices of dimension 1. The multivariate form of the equation is simply a higher dimensional form of the univariate case. Both forms of the equation capture selection operating on the phenotypic trait with this change resulting in a proportion of the observed phenotypic change shifting the mean of the breeding value distribution. It is assumed that non additive genetic components of the phenotype E are independent of the breeding value A.

We focus our attention on the univariate case, although extending it to the multivariate case should be straightforward. When we work with scalars, we do not embolden parameters. The scalar result of the product of the reciprocal of the phenotypic variance and the additive genetic variance is the heritability, $h^2 = P^{-1}G$ (Jacquard 1983). The heritability is consequently central to the univariate version of the breeders equation. The matrix product $\mathbf{P}^{-1}\mathbf{G}$ is the multivariate equivalent of the heritability, allowing the prediction of phenotypic change in multivariate characters. In the univariate case, the heritability can be estimated as the linear regression slope of parental trait values against offspring trait values assuming both are normally distributed with approximately equal variances. In the two sex case parental trait value is the mean of maternal and paternal values.

In order to understand how an estimate of the slope of the parent-offspring regression can be calculated from an IPM it is first necessary to introduce IPMs. A simple IPM can be written

$$n(z', t+1) = \int \left[D(z'|z,t)R(z,t) + G(z'|z,t)S(z,t)\right] n(z,t)dz. \qquad \text{(A-2)}$$

$n(z,t)$ is the distribution of the character at time t and $n(z', t+1)$ is the character distribution at time $t+1$. The remaining terms describe functions. $S(z,t)$ describes

how the character influences the probability of survival from t to $t+1$, while $R(z,t)$ describes the number of offspring an individual with character value z is expected to produce between t and $t+1$ that survive to enter the population at $t+1$. The probability density development function $G(z'|z,t)$ describes the probability that an individual that survives from t to $t+1$ that has character value z at time t will express character value z' at $t+1$. The second probability density function $D(z'|z,t)$ describes the probability that reproducing parents with character value z at time t produce offspring with character value z' at $t+1$.

It is possible to combine the survival and recruitment function into a single fitness function by adding them together: $w(z,t) = S(z,t) + R(z,t)$. Similarly, the development and inheritance functions can be combined to give a single transition function, $\theta(z'|z,t) = D(z'|z,t) + G(z'|z,t)$. The IPM can now be written,

$$n(z', t+1) = \int \theta(z'|z,t) w(z,t) n(z,t) dz. \tag{A-3}$$

The fitness function $w(z,t)$ and the transition function $\theta(z'|z,t)$ can also be combined. IPMs are frequently approximated as high dimensional matrices. When this is done it is common to combine all the functions into a single function and to write the matrix approximation as $\mathbf{n}(t+1) = \mathbf{A}(t)\mathbf{n}(t)$.

The survival and recruitment functions are usually linearised functions linking the character to expected survival and recruitment. In contrast, the development and inheritance functions require a little more explanation. The development function captures how the character develops within survivors as they age. It is a probability density function. On the x-axis is the character value at time t, and on the y-axis is the character value at time $t+1$. There are two components to the development function: a component that describes the mean response, and a component that describes the variance around the mean response. The function is usually Gaussian

in form,

$$G(z'|z,t) = \frac{1}{\sqrt{2\pi}\sigma_G(z)} e^{\frac{-(z'-E_G(z))^2}{2\sigma_G(z)^2}}. \tag{A-4}$$

The functions $E_G(z)$ and $\sigma_G(z)^2$ are typically linear functions,

$$\begin{aligned} E_G(z) &= a_G + b_G z \\ \sigma_G(z)^2 &= c_G + d_G z. \end{aligned} \tag{A-5}$$

The inheritance function typically takes the same form as the development function – a probability density function that is usually Gaussian (equation (A-4)). However, instead of describing the character value at time $t+1$ of a surviving individual that expressed character z at time t, it describes the expected character value at $t+1$ of an offspring born between t and $t+1$ to a parent with character value z at time t. We will assume it takes the same form as the development function with,

$$\begin{aligned} E_D(z) &= a_D + b_D z \\ \sigma_D(z)^2 &= c_D + d_D z. \end{aligned} \tag{A-6}$$

Parameters (a_G, A_D, B_G and b_D) for the mean components ($E_G(z)$ and $E_D(z)$) of the development and inheritance functions can be estimated from linear regression. The estimate for the intercept gives the a_Xs, while the estimate for the slope gives the b_Xs. The squared residuals from these models can then be analysed as a function of the character to give estimates of the c_Xs and the d_Xs. This logic can be applied to construct any Gaussian probability function.

In order to calculate the heritability from any IPM, one generates a cohort of newborns that will become the parental cohort: $n_p(z,t) = \int D(z'|z,t)R(z,t)n(z,t)dz$. This cohort is then iterated forwards throughout life using the IPM, with the distribution of offspring traits produced by parents that started life at each value of

z recorded. Parental and offspring traits are then regressed against one another. Coulson et al. (2010) is the only paper to use this approach to date, and in that paper they only focused on the heritability of body weight when offspring recruited to the population. It is straightforward to begin tracking the parental cohort at any age, and to track offspring development and survival to that same age, before estimating the heritability at that age.

Age-structured IPMs can easily be constructed (Ellner and Rees 2006). We will use an IPM with two age classes – juveniles and adults. The distribution of juvenile characters at time t, $n(z, 1, t)$ can be generated from the distribution of parents $n(z, 2, t)$, the parent's recruitment function $R(z'|z, 2, t)$ and the parental inheritance function $D(z, 2, t)$,

$$n(z', 1, t+1) = \int D(z, 2, t)R(z, 2, t)n(z, 2, t)dz. \tag{A-7}$$

Now assume that all adults die after reproduction. This means that the distribution of adults at time $t + 1$ is simply the result of iterating forward the distribution of one year olds at time t,

$$n(z', 2, t+1) = \int G(z'|z, 1, t)S(z, 1, t)n(z, 1, t)dz. \tag{A-8}$$

We will only consider a simple age-structured model like that described in equations (A-7) and (A-8) in this paper. A generalised age-structured model for an iteroparous life history has been developed by Coulson et al. (2010). In such models, each age-class has its own functions and parameter values.

Most structured models are analysed at equilibrium when $\Delta\overline{z} = 0$. However, some of the models we construct do not have a stationary equilibrium as $\Delta\overline{z} \neq 0$. In these cases, we select a Gaussian starting population structure and estimate heritability using offspring from this initial structure as the parental cohort. In the initial starting population structure there are 0 individuals in the largest and smallest

trait values in $n(z,t)$. In some models this prevents individuals being "evicted" from the IPM (Williams et al. 2012).

Coulson et al. (2010) showed how h^2 could be calculated from structured models, but did not explore why the heritability took specific values. In the next section, we explore factors that influence the size of the heritability in simple models.

Results

An IPM form of the Breeders equation

Dynamic structured models of ecological and evolutionary systems consist of three components: (i) the distribution to be iterated forwards, (ii) a fitness function describing the rate of replication of individuals in each part of the distribution being iterated, and (iii) a transition function describing where the fitness of each part of the distribution being iterated forwards ends up (A-3). This is true regardless of whether the structured model is iterating forward (i) the distribution of a population over a single time step as in an IPM or matrix model; (ii) the distribution of a population over a generation, or (iii) the distribution of a group of species over evolutionary time. In an IPM, the distribution being iterated forwards is $n(z,t)$; the fitness function is decomposed into a survival $S(z,t)$ and a recruitment $R(z,t)$ component; and the transition function is decomposed into a development function for survivors and an inheritance function to assign offspring character values. In the Breeders equation (equation (A-1)) the fitness function is the vector β and the transition function is the reciprocal $P^{-1}G$. The equation has been scaled such that change in the mean of the distribution being iterated forwards is predicted as a function of the selection differential that describes how the fitness function alters the mean of the parental distribution, and a heritability that describes the proportion of the selection differential that is passed from parents to offspring (A1). Given this, it should be obvious that a form of the Breeders equation that iterates the

entire character distribution forward on a per generation time step can be written in IPM notation. If we define the distribution of a phenotypic trait z in parents as $m(z,t)$ and in the offspring generation as $m(z',t+1)$ then we can iterate forward the distribution of parental traits to the offspring generation using the following equation,

$$m(z',t+1) = \int h(z'|z,t)w(z,t)m(z,t)dz. \tag{A-9}$$

Here $w(z,t)$ describes the fitness function, and specifically the association between the parental character z and parental fitness, usually measured as lifetime reproductive success (LRS). The transition function is $h(z'|z,t)$, a probability density function describing the association between parental character z at age a and offspring character z' at age a. The heritability function $h(z'|z,t)$ will likely be Gaussian (equation (A-4)). It will consist of two components (A-6) – a mean component $E_h(z) = a_h + b_h z$ where $b_h = h^2$ and a variance component $\sigma_h(z)^2 = c_h + d_h z$, both assumed to be linear. The phenotypic response to selection, $\Delta\overline{z}$, over a generation can be calculated as,

$$\Delta\overline{z} = \frac{\int z\, m(z',t+1)dz}{\int m(z',t+1)dz} - \frac{\int z\, m(z,t).dz}{\int m(z,t)dz}. \tag{A-10}$$

Equation (A-9) can be used to capture a wide range of phenotypic character dynamics, including those described by the Breeders equation. The link between equations (A-1) and (A-10) is depicted in figure (A1). We start with a distribution of characters in the parental generation. This is acted upon by a fitness function, which generates a distribution of parental phenotypes. Note that parents are a subset of the parental generation. The difference between the mean of the parental generation character distribution and the distribution of parental characters is the selection differential in the Breeders equation. This is acted upon by the slope of the mean component b_h of the heritability function in equation (A-9) to give the response

to selection $\Delta\overline{z}$ in equation (equation A-1). However, for the IPM version of the Breeders equation (equation A-9) to capture these dynamics, a particular condition is required.

The terms $\Delta\overline{z}$ in equations (A-1) and (A-10) will only be equal if the mean heritability function $E_h(z,t)$ intersects two points (Figure A1). The first of these is the point $(x = \overline{x}, y = \overline{x})$, and the second is $(\overline{w(z,t).n(z,t)}, b_h\overline{w(z,t).n(z,t)})$ (Figure A1). If $E_h(z,t)$ does not intersect these points, then the response to selection predicted by the Breeders equation will differ from the observed change in the mean trait from one generation to the next predicted by the IPM. In order for the heritability function to predict the response to selection predicted by the Breeders equation the intercept of $E_h(z,t)$ must be

$$a_h = \overline{m(z,t)} - \overline{m(z,t)}b_h = \overline{m(z,t)}(1 - b_h). \tag{A-11}$$

This insight is useful in terms of multigenerational prediction of evolutionary change using assumptions of the Breeders equation.

The Breeders equation is a static model that can only be used to predict a response to selection over a single generation. This is because if a response to selection occurs then $\overline{m(z,t)} \neq \overline{m(z',t+1)}$. Equation (A-11) allows the value of the intercept to be calculated prior to generation t being iterated forwards to $t+1$, allowing a dynamic version of the Breeders equation that can be used to iterate the phenotype distribution forwards for multiple generations by setting $E_h(z,t) = \overline{m(z,t)}(1-b_h) + b_h z$.

The value of $b_h = h^2$ does not evolve with time, remaining a constant from one generation to the next (A-9). This is because the parent-offspring covariance $cov(z',z)$ and the parental variance $\sigma(z)^2$ that determine b_h ($b_h = \frac{cov(z',z)}{\sigma(z^2)}$) evolve at the same rate, keeping it constant as a response to selection occurs. However, the variables c_h and d_h determine the response to selection and the way the population

responds to this change.

Will the parameters c_h and d_h evolve with time? These parameters describe the variation around the parent-offspring regression. This variance will likely be determined by a mixture of recombination, mutation and environmental variation independent from A. We have assumed here that they remain constant with time generating a balance between the loss of variance caused by selection and the injection of variation caused by an imperfect correlation between parent and offspring phenotype. This balance could be interpreted as selection-mutation or selection-recombination balance if environmental variation is assumed not to contribute to c_h and d_h. In future models, these parameters could be allowed to evolve in a more mechanistic setting.

The IPM version of the Breeders equation permits some interesting questions to be explored. First, the growth rate $R0(t) = \frac{m(z',t+1)}{m(z,t)}$ is mean fitness (mean LRS in most cases). The difference in growth rates between two time steps $R0(t+1) - R0(t)$ is the increase in fitness resulting from evolution of the character: if the population were at demographic equilibrium $R0(t+1) - R0(t) = 0$. At each time step, the selection differential can also easily be calculated as,

$$cov(z, w)(t) = \frac{\int z\, w(z,t) n(z,t) dz}{\int w(z,t) n(z,t) dz} - \frac{\int z\, n(z, tdz)}{\int n(z,t) dz}$$

In addition, the phenotypic variance can easily be calculated from one time step to the next.

In spite of the utility of this model it does have some undesirable properties. Because it iterates forward one generation at a time from age a at time T to age a at time $T + 1$, it cannot be used to study generation length. For iteroparous species that breed at multiple ages, the offspring of a parental cohort born at one time t will recruit at multiple different time steps meaning that any response to selection, or evolution of fitness, will be smeared across time. This may not be a

problem when choosing which individuals to breed from in an artificial setting, but it is an issue when comparing observation and model-based predictions of multi-generational evolution in free-living iteroparous species. Integral projection models as in equation (A-2) provide a way to do this. In addition, they can capture the dynamics described by equation A-9. Before we describe these in further detail, we start by considering properties of the heritability function (and the development and inheritance functions in IPMs).

Slopes and intercepts of transition functions and impacts on the phenotypic variance

At points where the mean of the transition function – be it the mean of the heritability function $E_h(z,t)$ in (A-9) or the development $E_G(z,t)$ or inheritance functions $E_D(z,t)$ in (A-2) – intersects the $y = x$ line, there is no change in trait value from one iteration to the next. At points above the $y = x$ line, the transition functions increases the character value, and at points below the $y = x$ line, the function decreases the character value (Figure A3). The intercept and slope of the transition function can consequently determine whether on average the character increases in value from one iteration to the next, remain unchanged, or shrinks.

The signs and values of b_X, c_X and d_X also determine how variance of the character distribution changes from iteration to iteration (Figure A3). If $b_X > 1$ then the variance in the character distribution increases between t and $t + 1$. In contrast, if $b_X < 1$ then the variance decreases (Figure A3).

$\sigma(z)^2$ also impacts change in the variance of the character distribution. Assuming $d_X = 0$, then as c_X increases, so too does the variance in the offspring character distribution. In other words, the larger the value of c_X the greater the increase in the phenotypic variance. The parameter d_X determines how much change in the variance of the distribution occurs at each character value z. For example, if $d_X > 0$ then more variance is injected into the character distribution at larger values of z

than at smaller values.

Readers will have noticed that, when positive, increasing the values of b_X and c_X both increases the variance in the character distribution between t and $t+1$. However, the way they increase the variance differs. If a sample of trajectories is randomly generated from different transition functions, the differences become apparent (Figure A4). Increasing $b_X > 1$ does not alter the relative ranking of individuals within the distribution, it simply spreads the distribution out (Figure A3). In the case of growth, this means large individuals get larger and small individuals get smaller but their relative ranking does not change. In the case of heritability, this would mean parents with large traits would produce offspring with even larger traits, while parents with small traits would produce offspring with ever smaller traits. In contrast to b_X increasing c_X leads to individuals potentially changing rank position within the distribution. By having different transition functions for different ages – i.e. a_X, b_X, c_X and d_X become age-specific – it is possible to generate a very wide range of trajectories, with some being more biologically realistic than others (Figure A4). Making the functions $E_X(z)$ and $\sigma_X(z)^2$ non-linear and non-Gaussian offers even more flexibility.

With these insights about the roles of different parameters in influencing the mean and variance in the phenotypic character distribution at the next iteration, we can consider why IPMs can predict $h^2 > 0$ and directional selection.

Conditions for stasis within an IPM

Both the fitness functions $w(z,t)$, $S(z,t)$ and $R(z,t)$ and the transition functions $h(z'|z,t)$, $G(z'|z,t)$ and $D(z'|z,t)$ can contribute to stationarity in models. Coulson and Tuljapurkar (2008) identified general conditions for such equilibrium using the age-structured Price equation. The Price equation that Coulson and Tuljapurkar (2008) used describes change in the mean of a character from one time step (or generation) to the next as the sum of change in the character mean due to selection

and a change in the character value due to inheritance or growth. However, these changes have not been interpreted in terms of slopes and intercepts within an IPM framework even though Coulson et al. (2010) showed how the terms in the age-structured Price equation can be calculated from an IPM.

In Coulson and Tuljapurkar (2008) contributions to changes in the mean differed across ages. However, the general processes for stability can be easily demonstrated using a non age-structured version of the Price equation,

$$\Delta\overline{z} = \frac{\int w(z,t)m(z,t)dz}{\int m(z,t)dz} - \frac{\int h(z'|z,t)w(z,t)m(z,t)dz}{\int (w(z,t)m(z,t)dz)}. \tag{A-12}$$

The first term on the right hand side of equation (A-12) describes change in the mean of the character from one generation to the next attributable to selection. The strength of selection is determined by the fitness functions. The second term of equation (A-12) describes how transmission contributes to the change in the mean character. These terms are determined by the transition functions. If the contributions cancel one another out then $\Delta\overline{z} = 0$ and the population may be at phenotypic stasis. How can different slopes and intercepts in the functions of the IPM lead to $\Delta\overline{z} = 0$ within an IPM?

The first set of conditions relate to the fitness function. If the slope of the function is 0, all phenotype values have the same fitness and the size of the trait distribution will grow exponentially regardless of the population structure or the transition functions. The population structure will converge to a stationary distribution determined entirely by the transition functions as long as the mean of the transition function .

We have so far only described a linear case for the fitness function. In contrast, if the slope of the fitness function is different from zero, the population can grow faster than exponentially: hyper-exponentially. Even in the presence of selection, it is possible for the population to remain at equilibrium if changes due to the mean

attributable to the fitness function are countered by changes attributable to the transition function (see below).

Equilibrium can also be achieved if the fitness functions are non-linear. For example, stabilising and disruptive selection can also result in $\Delta\overline{z} = 0$. Non-linear fitness functions can easily be included in IPMs (Ozgul et al. 2010). When selection is directional, it is possible for the transition function to interact with the fitness function to result in an equilibrium. If the fitness function increases (decreases) the mean of the character within a generation (i.e. parents have larger (smaller) character values than the population as a whole) then the transition function must counter these effects, with offspring having smaller (larger) trait values than their parents did at the same age. In order to understand how such processes manifest themselves within an IPM we need to understand how transition functions work in a little more detail.

The Breeders equation is simply a version of equation (A-9) for the mean of the phenotypic character distribution over a generation. However, unlike equation (A-9), it does not iterate forwards the entire character distribution from one time step to another, but rather focuses on how the mean changes over a generation. An IPM (equation (A-2)) takes a similar form as equation (A-9) above. However, it expands the fitness function $w(z'|z,t)$ into its survival and recruitment components and the heritability function into its inheritance function and a development function components. This allows the distribution to be iterated forwards from one time step to the next, rather than a generation at a time. Once this linkage is understood, it should be apparent that the Breeders equation is a simplified, special case of an IPM. This is why selection on lifetime reproductive success and the heritability can both easily be calculated from an IPM (Coulson et al. 2010).

For a heritability estimated from parent-offspring regression to be non-zero it is necessary for the parent-offspring phenotypic covariance to be non-zero. In equation (A-9) this means that $E_h(z)$ in the heritability function $h(z'|z,t)$ has a slope of

$b_h \neq 0$. Within an IPM, two requirements need to be met to achieve this. First, the inheritance function has to have a slope that is different from zero $b_D \neq 0$. Second, the development function must also have a slope that is different from zero $b_G \neq 0$ (at least for one breeding age in an age-structured model). In addition, if b_D and b_G have opposing signs, it is possible for an $h^2 = 0$. However, biologically we consider this to be unrealistic. When $b_D \neq 0$ and $b_G \neq 0$ perturbations that increase the variance in the offspring phenotype will increase the estimate of the heritability (Figure A2). We next demonstrate how this can occur.

Our first IPM is deliberately simple and has been chosen to illustrate the role the development function plays in determining the heritability. The IPM has the following functions and parameters,

$$\begin{aligned} S(z,t) &= 0.5 \\ R(z,t) &= 0.5 \\ a_G = 0\ b_G = 1 \quad &; \quad c_G = 0\ d_G = 0 \\ a_D = 0\ b_D = 1 \quad &; \quad c_D = 0\ d_D = 0. \end{aligned} \tag{A-13}$$

Because $\int [S(z,t) + R(z,t)] n(z,t) dz = 1$ the IPM has a dominant eigenvalue of unity, which means the population remains a constant size. The development function takes the value of 1 when $y = x$ and 0 otherwise, which means individuals do not change from the trait value they inherited at birth as they age. The inheritance function has an identical form, which means offspring perfectly resemble their parents. This model has a heritability of 1. The model does not tend to a stable population structure. Because (i) there is no selection, (ii) individuals do not change their trait value as they age, and (iii) individuals pass their trait values to their offspring, the initial population structure $n(z,1)$ remains unchanged with time. The size and structure of the population remains at whichever starting population structure is selected.

In spite of the biologically peculiar properties of this model it is useful for gaining insight into the behaviour of the heritability. What happens if b_G is lowered from 1 to 0.99 yet all other parameters and the initial parental cohort structure remain unchanged? This has the effect of reducing the variance in the offspring distribution (see Figure A2 for examples of how altering the development function impacts on the heritability). The offspring character distribution at birth is now compressed in relation to the parental character distribution at birth and the heritability drops below unity: $h^2 = 0.99$. A similar effect can be achieved by decreasing b_D, as this too compresses the distribution of offspring characters compared to parental characters. Changing the intercept a_G does not impact the heritability (Figure A2). Note that if we were to iterate these models forward that had slopes of $b_D < 0$ and/or $b_G < 0$, individuals would shrink towards zero and be evicted from the IPM (Williams et al. 2012). This is because the entire development function is below the $y = x$ line (see below).

If b_G is increased from 1 to 1.01 for development from age 1 to 2 (this requires an age-structured IPM) and b_D is decreased from 1 to 0.99 while all other parameters are kept the same, then the effects cancel one another out and $h^2 = 1$. The effects cancel because the new development function inflates the variance in the offspring distribution, while the new inheritance function decreases it. So even though parents and offspring do not resemble one another perfectly via the inheritance function, the lack of perfect resemblance which compresses the variance in the offspring distribution at birth can be countered by a development function that expands the variance within the offspring generation to produce a heritability of unity. Although our example uses a small perturbations to b_G and b_D, the logic holds for larger perturbations: if compression to the offspring distribution caused by $b_D < 1$ is countered by inflation to the variance caused by the development function with $b_G > 1$ – at least for one age – then the heritability can remain close to unity even if the inheritance function is nearly flat. This is a general result, but in order to understand why,

it is necessary to understand how different parameterisation of the development and inheritance functions impact the offspring phenotypic variance. We continue with the case of no selection ($S(z,t) = R(z,t) = 0.5$).

Given the effects of parameters in the development and inheritance functions on the variance in the character distribution of offspring (Figure A3), it should now be clear how each change that increases, or decreases, the offspring phenotypic variance impacts the heritability (Figure A2). These results are general, and will hold for IPMs. However, the survival and recruitment function can also impact the offspring variance.

We now let the trait be subject to positive directional viability and fertility selection by modifying the survival and recruitment functions

$$S(z,t) = R(z,t) = \frac{1}{1 + e^{-(-1+0.1z)}} : 0 \leq z \leq 1. \tag{A-14}$$

All other functions remain unchanged from our initial model – i.e. $a_D = a_G = 0$, $b_D = b_G = 1$, equation (A-14). What happens? The population grows hyper-exponentially and the breeding value distribution variance shrinks as the mean tends to the largest breeding value (Figure A5). Note that we deliberately put an upper limit on the upper most breeding value, by ensuring that the largest class in $n(1,t) = 0$. This case demonstrates the well known phenomenon that directional selection can erode phenotypic variance while increasing the phenotypic mean. It also shows how different functions can have opposing effects on the phenotypic variance in the character distribution. In IPMs of real systems the heritability will be a complex function of how each function adds, or erodes, variance from the offspring character distribution.

If mutational variance is added to the model above (by setting $c_D \neq 0$ or $d_D \neq 0$), or the development function is above the $y = x$ line, the mean of the phenotype distribution can grow indefinitely. It is important to appreciate this when modelling,

as otherwise individuals may inadvertently be "evicted" from the IPM (Williams et al. 2012). Technically, in the case we describe, the IPM occupies an infinite volume and will never achieve a stationary phenotypic equilibrium. A brake is required to prevent the phenotypic mean value growing indefinitely. We now turn to ways in which continuous phenotypic change can be prevented within an IPM.

The first way to apply a brake (in a univariate IPM – see below for a type of multivariate IPM) is to have a completely flat inheritance function $b_D = 0$. There is then no way for individuals to get larger from one generation to the next, as selection cannot be realised in any response to selection. The second way is to have flat survival and recruitment functions – i.e. no selection. In the remaining discussion of brakes, we assume the inheritance function has a slope that is greater than zero, and that at least one of the survival or recruitment functions has a slope greater than 0. The third way a brake can be applied is to have the development function cross the $y = x$ line. This can be achieved with $a_G > 0$ and $0 \geq b_G < 1$. Each cohort tends to this asymptotic size as they age. A fourth way is to have individuals shrinking once they achieve some age. The model now needs to be age-structured, and individuals in the terminal age-class need a development function that is below the $y = x$ line (Figure A4). A fifth way to apply a brake is to have opposing signs for the effect of the character on survival and recruitment – a type of stabilising selection. Non-linear selection operating on survival, recruitment, or both, is the final way a brake can be incorporated into an IPM.

In an IPM at equilibrium – i.e. one where a brake is incorporated – the heritability would typically be calculated by generating a parental cohort from the stable population structure. When a model parameter is perturbed, in addition to altering the variance of the offspring phenotype distribution, the stable population structure also changes, resulting in a change in the variance in the parental phenotype distribution. In order to understand how altering a parameter impacts the heritability in such a case, it is necessary to investigate how both the variance in the parental and

offspring character distribution is altered. This, of course, assumes that the parent-offspring phenotypic covariance is non-zero, and that requires a non-zero slope to the inheritance function. We now consider a case where the slope of the inheritance function is flat, but adult body size is heritable. In order to do this, we need to separate the phenotype into a breeding value A and non-additive-genetic component E and construct a class of multivariate IPM.

We now analyse the two age-class model (equations (A-7) and (A-8)) described in the methods with the following functions:

$$\begin{aligned} S(z,1,t) &= \frac{1}{1+e^{-(-1+0.1z)}} \\ E_G(z,1,t) &= ? \\ \sigma_G(z,1,t) &= 0 \\ R(z,2,t) &= \frac{1}{1+e^{-(-1+0.1z)}} \\ E_D(z,2,t) &= 4 \\ \sigma_D(z,2,t) &= 0.0 \leq z \leq 15 \end{aligned} \tag{A-15}$$

We wish to extend this model to allow adult body weight to have a heritability close to one, even though the inheritance function is flat $b_D = 0$. In this parameterisation all individuals are born with a character value of $z = 4$. For simplicity, we have set the variance around the inheritance and development functions to 0, but the general logic extends to cases when $c_G > 0$, $d_G > 0$, $c_D > 0$ and $d_D > 0$. The development function needs to take these offspring and distribute them across a range of parental character values. One way we can do this is to use the quantitative genetic approach of decomposing the phenotype into a breeding value A and a non additive genetic component E: $z = A + E$. We then assume that the breeding values component of the phenotype is passed from parent to offspring, but that the slope of the breeding value inheritance function is less than unity (Figure A6). In statistical quantitative genetics E can be decomposed further into contributions from inbreeding, epistasis,

the environment and maternal effects (Bulmer et al. 1980). We could extend our approach to deal with these contributions within a multivariate IPM, but we can illustrate the point we wish to make by considering just two components, A and E.

In our model we stipulate that the environmental component E contributes considerably more to the offspring phenotype than the breeding value A – in other words, the offspring phenotype is determined primarily by environmental variation and $E >> A$. Such an argument has been made for ungulate species (Wilson et al. 2005). In contrast, environmental variation contributes little to the parental phenotype, which is determined nearly entirely by the breeding value A: $A >> E$. There is variation in A among parents. Because the inheritance function for the phenotype (but not the breeding value) is completely flat, and all individuals are born with a phenotypic trait value of $z = 4$, there has to be a negative covariance between A and E in the offspring phenotype (Figure A6a), even though E is substantially larger than A. This is because $A + E = 4$, so when A is (relatively) large, E must take on a smaller value.

In order for the breeding value to dominate the adult phenotype, we require a development function that increases the variance in A and reduces the variance in E. This is achieved by having a development function for the breeding value almost entirely above the $y = x$ line, while having the development function for the environmental component almost entirely below the line (Figure A6b). Biologically what this means is that in order to have an increasing heritability with age, it is necessary to have negative covariances between A and E across ages (Figure A6).

Discussion

The aim of this paper is to explore some of the similarities and differences between the empirical quantitative genetic approach and integral projection models. In doing this, we have demonstrated the link between the heritability and the inheritance

and development functions, and between selection differentials and the survival and recruitment functions. We show that attempts to infer the heritability of a character by simply focusing on the slope of the inheritance function (Hedrick et al. 2014) are misguided. Our work also shows how flexible IPMs are in incorporating functions that allow a very wide range of growth trajectories and inheritance patterns. IPMs offer a powerful framework that is well complemented by empirical quantitative genetics, rather than an alternative, competing framework.

The primary aim of quantitative genetics when applied to free-living systems is to characterise phenotype change and evolution (Kruuk et al. 2008). The approach is primarily focused on applying powerful statistical methods to provide estimates of genetic and phenotypic variances and covariances, rather than identifying the demographic processes that caused the statistics to be particular values. Estimating quantitative genetic parameters, particularly in free-living systems where sample sizes are often small and where environmental noise is typically substantial, is statistically challenging (Hadfield et al. 2010). Robust demonstrations of evolution require large sample sizes, and correction for numerous sources of non additive genetic phenotypic variation. Nonetheless, the production of transparent, powerful, freely available *R* packages like MCMCglmm (Hadfield 2010) has aided the ability of biologists to estimate heritability and additive genetic (co)variances using these powerful statistical methods. Interestingly, appropriate application of these statistical approaches appears to be leading to a broad scale re-evaluation of the prevalence of rapid evolutionary change in the wild (Hadfield et al. 2010).

One of the reasons why estimating quantitative genetic parameters is challenging is the approach most widely used is the animal model – a mixed effect model with a relatedness covariance matrix treated as a random effect (Lynch et al. 1998). Unlike parent-offspring regression – the primary focus of this paper – the animal model uses information from all known relatives within a pedigree. From a statistical standpoint there is no doubt that the animal model has greater power, when

correctly used, to provide unbiased estimates of the heritability and additive genetic (co)variances than parent-offspring regression. Currently it is not possible to incorporate pedigree information beyond parent-offspring links into IPMs, and it is not yet entirely clear how this could be done. IPMs are consequently unlikely to become the tool of choice to estimate quantitative genetic parameters. However, this does not devalue the utility of IPMs. This is because their primary function is as a dynamic modelling framework to help understand what demographic processes most plausibly generated patterns in data (Merow et al. 2014, Rees et al. 2014, Coulson 2012). They have proven fantastically useful in doing this, and have allowed insight into rapid phenotypic change in marmots (Ozgul et al. 2010), Soay sheep (Coulson et al. 2010) and bighorn sheep (Traill et al. 2014), the evolution of life histories in plants and animals (Childs et al. 2003, Metcalf et al. 2008, Childs et al. 2011), the possible phenotypic and population responses to climate change (Coulson et al. 2011), the circumstances under which males can contribute to population growth and life history (Schindler et al. 2013), insights into optimal sex allocation and the circumstances under which ecological and evolutionary quantities are expected to simultaneously change (Smallegange and Coulson 2013). They also provide a way to understand why quantitative genetic parameters take the values they do; even the simple models we have constructed have provided novel, general, insight.

The simple models outlined here demonstrate several things. First, the way that individuals develop is critical in determining the heritability of a character. A consequence of this is that unbiased estimates of heritability require correction for factors that impact growth rates of individuals. In the high food, relatively constant environments of the barn and laboratory, most animals probably achieve close to their genetically determined growth potentials and there may be little need to correct for factors that influence growth rates throughout life. In contrast, in free-living populations, numerous factors can impact an individual's growth rate including food availability (Dunham 1978), the presence or absence of interacting species (Werner

and Gilliam 1984), disease (Spurlock 1997) and the reproductive history of the individual (Reznick 1985). Failure to adequately correct for factors that generate variation in growth rates will bias estimates of the heritability and the additive genetic variance. Second, our results show that in order to generate significant heritabilities at some ages, but not at others, it is necessary to have some potentially rather exotic correlations between the additive genetic component of the phenotype and other components both within and between different ages. These insights demonstrate that integral projection models provide a powerful tool for understanding which demographic factors influence estimates of heritability and the additive genetic variance. But our work also shows that further work is required to apply IPMs to analyse quantitative genetic parameters in real systems.

There is an alternative way to view inheritance in IPMs. The approach we have focused upon here has been motivated by the quantitative genetic framework. This has allowed us to investigate the circumstances under which phenotypic correlations between and among different ages arise. However, there is an alternative way to consider inheritance, and that is that entire state- (or trait-) dependent life history strategies are passed from parents to offspring (Childs et al. 2003). In this case there are different individuals within the population following different strategies, with each strategy defined by its own IPM, each with a different set of parameter values. There will be trade-offs between parameter values within and across functions. For example, these trade-offs could be between development rates and offspring size, between offspring number and offspring size, or between size-specific survival and recruitment. Instead of the traits themselves being heritable, it is instead the entire state-dependent life history captured by the parameter sets that are passed from parents to offspring. It is this logic that underpins the work of Childs et al. (2003) and Metcalf et al. (2008) where a trade-off between offspring number and offspring size determines parameter values among life history strategies, that then compete against one another in an evolutionary games until an evolutionarily stable life

history strategy is identified. Of course, the parameter values within an IPM could be considered as phenotypic traits themselves, and given sufficient data, perhaps quantitative genetic methods could be used to examine the heritability of these parameter values. But that's work for another day.

It should come as no great surprise that IPMs can be used in tandem with quantitative genetic approaches to understand why specific traits in particular populations have the heritability they do. This is because the heritability, and the selection differential, are simply statistics that describe aspects of the dynamics of a character distribution. The selection differential describes how the mean trait value differs between the entire population, and those that breed. This is obviously just one aspect of the dynamics of a character distribution. In contrast, the heritability is a covariance between parental and offspring characters. This describes another aspect of the dynamics of a character distribution. Given IPMs track the dynamics of character distributions over time, it is not surprising that they are a useful tool to study the dynamics of the heritability, the additive genetic variance and the selection gradient. Indeed all statistics that ecologists and evolutionary biologists use describe some aspect of a character distribution or its dynamics. If it is possible to calculate these quantities from predictions made from IPMs, it is possible to analyse the dynamics of these quantities independently, or in tandem with the analysis of other statistics of ecological or evolutionary interest (Coulson et al. 2010).

In spite of their great potential, IPMs, and structured population models in general (Caswell 2001), take some work to understand. They differ from the static Breeders equation in being dynamic. In addition, choices need to made concerning which processes to include in models. Finally, analytical, and even numerical, analyses of models, can be complicated. However, we suspect the investment in learning how to construct and analyse models will always be worthwhile.

Some quantitative geneticists have taken issue with the inheritance function (and with its name) claiming it does not capture genetic inheritance. This is a fallacious

claim. The inheritance function was appropriately named and can be constructed to capture numerous types of inheritance mechanism. We hope that in future, before launching ill-informed criticisms about a new(ish) approach, that researchers actually try the approach themselves. It would be much productive to understand the approach than to launch attacks based on anecdote and innuendo.

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Table A1. Effect of increasing parameter values in the development and inheritance functions on the mean and variance of the offspring distribution. For a model with no selection. When selection occurs, non-linear averaging can result in c_G, d_G, c_D and d_D impacting the mean.

Parameter	Effect on mean	Effect on variance
a_G	$\Uparrow$	none
b_G	$\Uparrow$	$\Uparrow$
c_G	none	$\Uparrow$
d_G	none	$\Uparrow$
a_D	$\Uparrow$	none
b_D	$\Uparrow$	$\Uparrow$
c_D	none	$\Uparrow$
d_D	none	$\Uparrow$

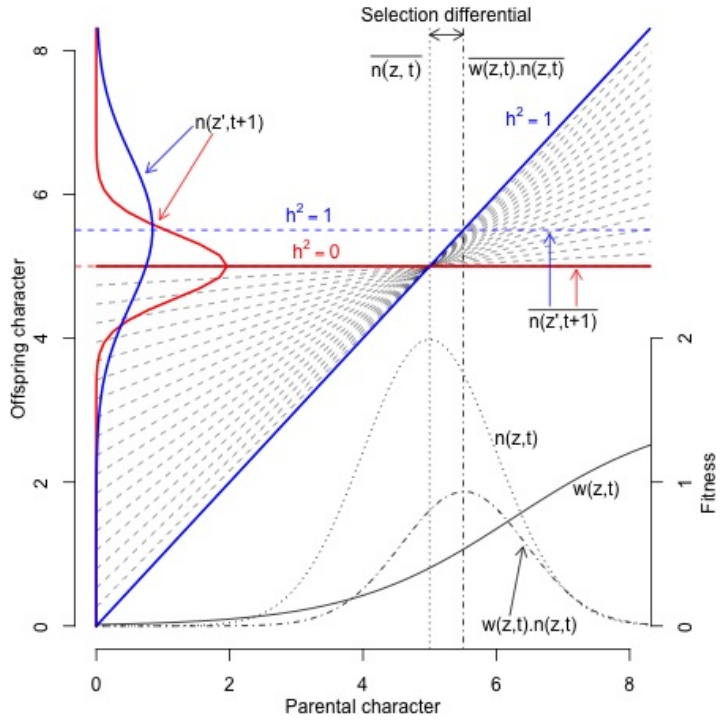

Figure A1. A graphical representation of the Breeders equation. The distribution of characters within the population $n(z,t)$ (black dotted line) is multiplied by a fitness function $w(z,t)$ (black solid line) to generate a distribution post selection $\overline{n(z,t)w(z,t)}$ (dash-dot line). The difference in the mean of these two distributions $\overline{n(z,t)w(z,t)} - \overline{n(z,t)}$ is the selection differential. The proportion of the selection differential that is realised as change in the character mean in the offspring generation $\Delta\overline{z}$ depends upon the heritability h^2 (dashed black lines). If $h^2 = 1$ all selection is realised in the offspring generation (blue dashed line, and blue distribution adjacent to the y-axis). At the other limit $h^2 = 0$, none of the selection is realised and the offspring mean is equal to the parental mean (solid red line and red distribution adjacent to the $y - axis$).

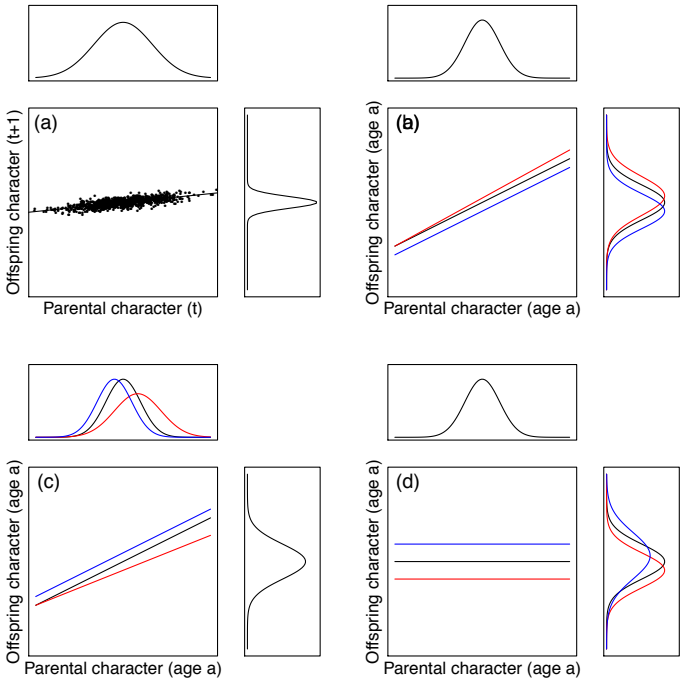

Figure A2. The inheritance function (a), the heritability (b-d), and factors affecting the heritability (red and blue lines). (a) The inheritance function describes the association between parental character measured at time t and the offspring character measured at $t+1$. There is no requirement of bivariate normality or equivalence of the variance between the offspring and parental generation that is required for parent-offspring regression to provide an estimate of the heritability. As long as there is a non-zero covariance between parent and offspring character, any process that impacts the variance in (b) the offspring character distribution or (c) the parental character distribution will impact the heritability (red lines). Solely altering the mean of (b) the offspring or (c) the parental character distribution will not impact the heritability. If the covariance between the parental and offspring character distribution is zero (d) then altering the mean (red) or variance (blue) of either distribution has no effect on the heritability.

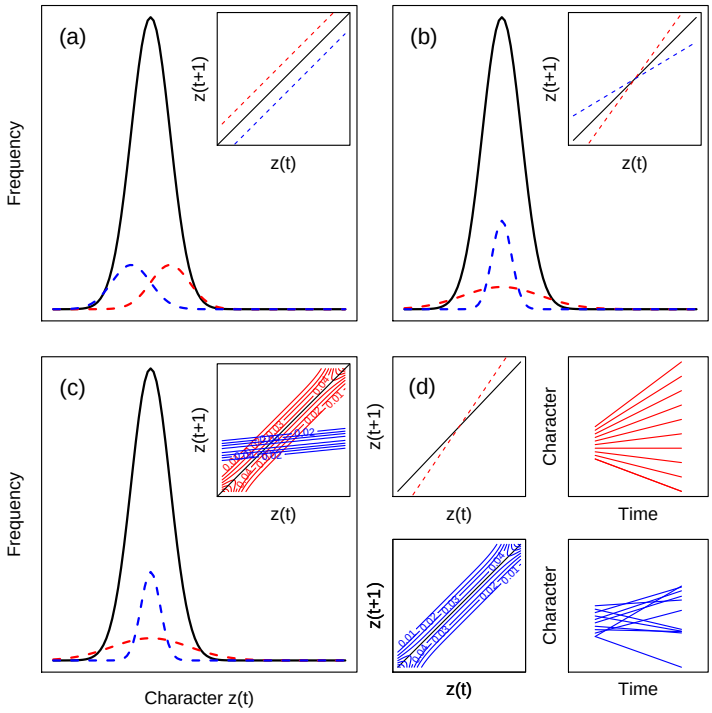

Figure A3. Ways in which the development function (insets) influences the offspring phenotypic distribution as the offspring cohort ages. (a) Points above the black $y = x$ line (the red line) increase the mean of the character distribution, while points below the $y = x$ line (the blue line) decrease it. Altering the intercept of the development function impacts the mean of the distribution (a). Altering the slope of the function impacts the variance (b). Increasing the slope increases the variance (blue lines) while reducing the slope decreases it (blue lines). (a) and (b) focus on altering parameters in the mean component of the development function ($E_G(z, t)$). The variance of the development function can also be impacted by perturbing the variance component of the development function (c). The width of the variance function and the slope of the mean function both impact the mean. Altering the slope of $E_G(z, t)$ does not impact the relative ranking of character values (upper panels in (d)). Altering the variance of the development function impacts individual ranking within the distribution (lower panels in (d)).

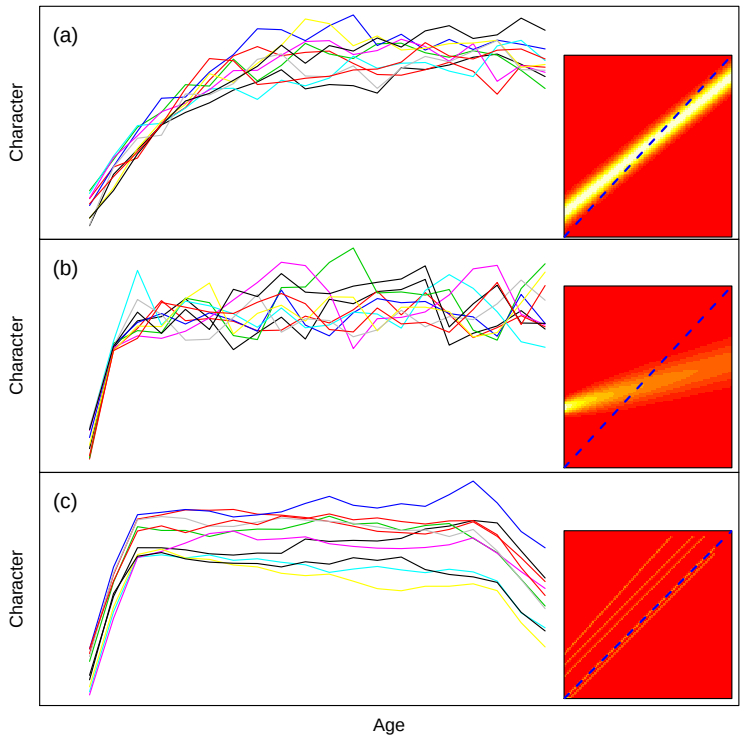

Figure A4. How different development functions generate different simulated individual growth trajectories. The closer the mean slope b_G gets to unity, the longer individuals take to reach their asymptotic size (compare (a) and (b)). The variance determines how much change in rank occurs. In (b) the variance increases with mean body size. In an age-structured model each age has its own development function (c). Depending upon the slope of these individuals can grow, or shrink, in size. In (c) individuals decline in size in the last age class (the line below $y = z$).

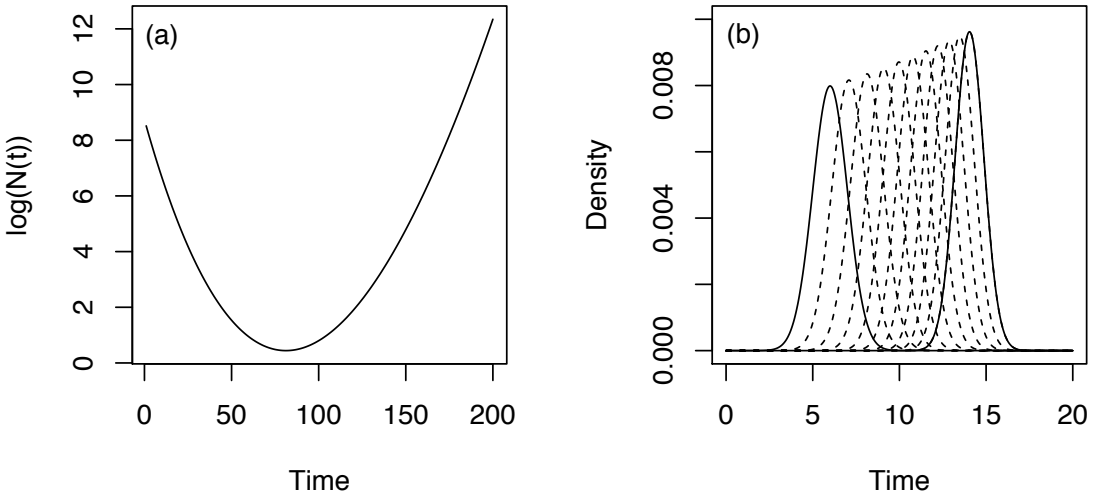

Figure A5. Effects of selection on the population growth rate (a), and the phenotypic character distribution (b) with time. (a) The population growth rate is hyper-exponential, with logged population size initially decreasing and then increasing as the character distribution moves along the x-axis. Exponential growth would be represented with a straight line on this plot. (b) the phenotypic character distribution (scaled to sum to unity) tends towards the maximum possible breeding value, and the variance reduces. Plots from a model with survival and fertility parameters from equation (A-14) and development and inheritance parameters from equation (A-14)

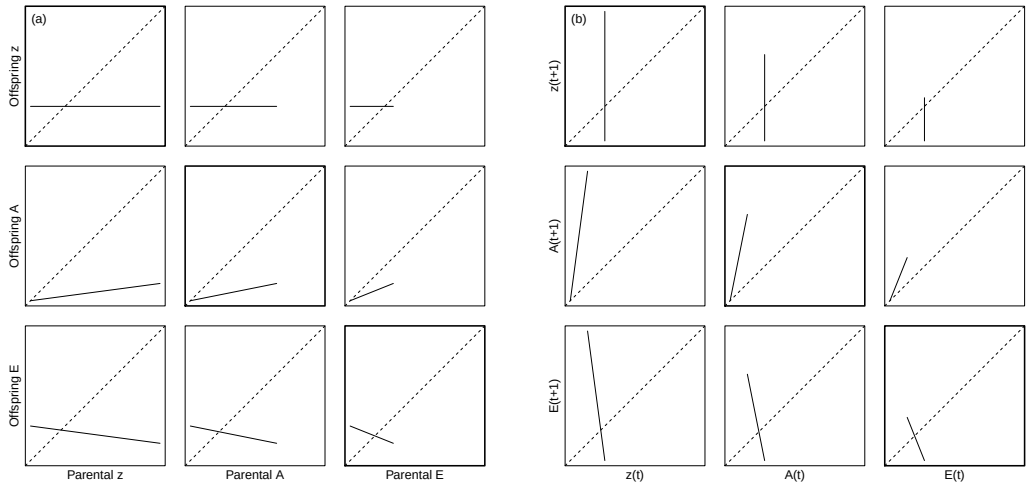

Figure A6. Covariances between the phenotype z, the breeding value A and the non-additive genetic component of the phenotype E, necessary to have a flat inheritance function for the phenotype, no heritability for the character expressed in the offspring, and a large heritability for the adult character. (a) covariances for the inheritance function; (b) covariances for the development function. The graphs on the diagonals with the emboldened boxes represent associations between the parental and offspring phenotype z, breeding value A and non additive genetic component E respectively. A horizontal or vertical line represents no covariance. An upwardly sloping line represents a positive covariance, while a downward sloping line represents a negative covariance. The steeper the line, the larger the covariance.

Appendix—Other published work

Barthold, J. A., M. Myrskylä, and O. R. Jones (2012). Childlessness drives the sex difference in the association between income and reproductive success of modern Europeans. Evolution & Human Behavior 33, 628–638.

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Evolution and Human Behavior 33 (2012) 628–638

Evolution and Human Behavior

Original Article

Childlessness drives the sex difference in the association between income and reproductive success of modern Europeans

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Abstract

The association between reproductive success and income in economically developed societies remains a controversial and understudied topic. The commonly made statement that individuals with a higher income have fewer children defies evolutionary explanation. Here we present results from an analyses of the association between lifetime reproductive success (LRS) and income for modern Europeans from 13 countries. We examine the relationships among income, partner income, sex and LRS, and the role of childlessness in driving the relationships. For women, we find a negative association between LRS and income, while for men, we find a flat or slightly positive one. The sex difference in the association appears to be driven by income's sex-specific association with childlessness; men with a low income have a relatively high risk of childlessness, while women with a low income have a low risk of childlessness. Consequently, once childless people are excluded from the analysis, LRS is negatively associated with income for both sexes. We argue that the observed LRS–income associations may be an outcome of evolved behavioural predispositions operating in modern environments and conclude that, even though humans fail to maximise LRS at all income levels in modern settings, evolutionary theory can still help to explain sex differences in LRS.

Keywords: Fertility; Reproduction; Gender differences; Earnings; Education; Socioeconomic status; Behavioural ecology

1. Introduction

Life history theory predicts that organisms allocate resources in a way that maximises their fitness (Stearns, 1992). One major component of fitness is how many offspring a parent successfully rears. Since investing in offspring is costly, resource availability should be positively correlated with lifetime number of surviving offspring (from here on reproductive success). The quantity of resources that an individual can acquire depends on many factors. In social animals, where some individuals socially dominate others, high-ranking individuals have greater control of resources. Since access to resources enhances reproductive success, a positive association between status and reproductive success emerges for many social animal species (Clutton-Brock, Albon & Guinness, 1986; Ellis, 1995). A similar positive association exists between social status, resource acquisition and reproductive success in historical and contemporary small-scale human societies (Cronk, 1991; Gurven & von Rueden, 2006; Røskaft, Wara & Viken, 1992; Skirbekk, 2008; von Rueden, Gurven & Kaplan, 2011). However, the association in modern, economically developed societies remains equivocal, particularly when researchers stratify their analyses by sex (Hopcroft, 2006).

During the demographic transition, which emanated from Western Europe at the end of the 18th century, mortality decline was followed by a decline in fertility despite a general increase in wealth, and the fertility declines first became apparent in wealthier families (Coale & Watkins, 1986; Livi-Bacci, 1986). Due to this observation, a view has emerged that, in modern humans, there is a negative association between the amount of available resources and reproductive success. This viewpoint has stimulated debate on how to reconcile observations of reproductive behaviour in post-transitional societies with evolutionary theory (e.g., Kaplan, Lancaster, Johnson, & Bock, 1995; Borgerhoff Mulder, 1998; Kaplan & Lancaster, 2000; Low, 2000; Mace, 2007). Some researchers have argued that the observed behaviour may be non-adaptive and due to a mismatch

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between evolved behavioural strategies and new environmental conditions (Kanazawa, 2003; Pérusse, 1993). Boyd and Richerson (1985) proposed that low fertility could spread through a population by means of Darwinian but non-genetic mechanisms of inheritance: people simply imitate socioeconomically successful individuals. Other researchers believe that low fertility may be adaptive, because a reduction in offspring quantity in order to enhance offspring 'quality' could increase the competitiveness of their offspring and therefore secure the long-term survival of their lineage (reviewed in Lawson & Mace, 2011; see also Boone & Kessler, 1999; Kaplan & Lancaster, 2000; Mace, 1998; compare to Becker, 1991). To date, a conclusive explanation for the demographic transition that would be consistent with evolutionary explanations of behaviour has been elusive. One step towards reaching this goal is to study the association between income or resources and reproductive success in post-transitional societies.

Between-country comparisons using population-level data indicate that there is a negative association between financial resources and reproductive success (e.g., Lee, 2003). Although this is informative for the relationship between countries, it leaves the possibility that, within subpopulations, e.g., within each country, an individual's financial resources could be positively associated with his or her reproductive success or there could also be no relationship between the two. The relationship at the individual level has only been studied a few times and, although these studies have undoubtedly generated useful insights, their informative value is, in some cases, restricted by data and study design limitations. For example, some studies did not use representative samples (e.g., Pérusse, 1993; Vining, 1986), while others did not measure individual-level lifetime number of biological children (e.g., Hopcroft, 2006; Kanazawa, 2003; Pérusse, 1993; Vining, 1986). Still more used indirect measures of social status, rather than financial resource measures such as income or wealth (Fieder et al., 2005; Goodmen & Koupil, 2009; Hopcroft, 2006; Kaptijn, Thomese, van Tilburg, Liefbroer & Deeg, 2010; Vining, 1986). For women, most results support the existence of a negative association between personal income and reproductive success (Fieder & Huber, 2007; Hopcroft, 2006; Nettle & Pollet 2008; Weeden, Abrams, Green & Sabini, 2006). However, for men, every study that has used a sample from a homogenous subpopulation, or a representative sample of the whole population, has found a positive association between income and reproductive success (Fieder et al., 2005; Fieder & Huber, 2007; Hopcroft, 2006; Nettle & Pollet, 2008; Weeden et al., 2006). Notably, from an examination of two of these studies, it seems that this positive association is driven by childlessness (Fieder & Huber, 2007; Nettle & Pollet, 2008). In these studies, when childless individuals were excluded from the analyses, the association between personal income and male reproductive success was flattened, and an increase in income decreased the likelihood of male childlessness. Collectively, these analyses challenge the existence of a universal negative resource–fertility association in post-transitional societies, demonstrate an inherent sex difference in the relationship and point towards childlessness as an important driver of the observed sex difference.

In modern hunter–gatherer societies, men provide the great majority of the caloric support for offspring (Kaplan, Hill, Hurtado & Lancaster, 2001). Even though these societies are not identical copies of our stone-age ancestors, it is reasonable to assume that during our evolutionary past males provided females with the resources necessary for reproduction. It follows that the quantity of resources that males acquired and passed on to females strongly influenced female reproductive success. Whenever males varied in how successfully they acquired resources, females would have benefited from associating with successful males and would have granted them mating privileges in return. Females who preferred successful males would have had more offspring, and if these preferences were heritable, they would have become widespread. It is likely that these preferences have persisted until the present time. In the same vein, high-status males who excelled at resource acquisition would have attracted a more fertile partner or, depending on the mating system, additional partners and, consequently, would have had a greater lifetime reproductive success. Von Rueden et al. (2011) report such a positive association between status and reproductive success for a contemporary hunter–gatherer society.

Here, we present the results of a study looking for the fingerprints of evolved partner preference on the association between lifetime reproductive success (LRS) and income in contemporary Europeans. Although wealth is perhaps a better measure of resource availability, our data set only included income measures. We therefore used these measures as indicators of resource availability. We examined the association of LRS with both personal and partner income, and we controlled for sex, country and birth year. We also controlled for education, because education is known to play a role in fertility decisions (e.g., Kravdal & Rindfuss, 2008). Although education and income are correlated, problems of multicollinearity are unlikely to be an issue because in our data education explains only 7% of the variation in income. We use interactions to study how the LRS–income association varies by sex, education and partner income. We further investigated the association between childlessness and personal income, while again controlling for sex, education and birth year.

2. Methods

2.1. Data

Our data were derived from the Survey of Health, Ageing and Retirement in Europe (SHARE), which is an interdisciplinary panel database containing individual-level data on health, socioeconomic status and social relations for

people aged 50+, and their spouses, in EU countries (Börsch-Supan & Jürges, 2005). The survey aimed to produce a representative sample of both citizens and households within countries. Households were eligible for inclusion if at least one member was older than 50, if household members were not living in an institution (e.g., prison, nursing home) or were abroad at time of fieldwork, and if the official language of the country was spoken in the household. Within households, all individuals meeting these criteria, plus their spouses and partners, independent of their age, qualified for participation in interviews. Households were selected randomly using local, regional or national citizen registries depending on availability in their respective countries. Survey responses were collected by trained interviewers, who administered questionnaires in computer-assisted, face-to-face sessions. A detailed description of the SHARE methodology can be found in Börsch-Supan and Jürges (2005). The database provides open access to researchers (http://www.share-project.org/). Here, we used data from individuals participating in the first wave, collected in 2004–2005, and from further participants in the second wave, which was collected in 2006–2007 (SHARE Survey of Health, Ageing, and Retirement in Europe, 2010). We included data for 13 EU countries (Austria, Belgium, Czech Republic, Denmark, France, Germany, Greece, Italy, Netherlands, Poland, Spain, Sweden and Switzerland), for which income information was available.

For every individual included in the final data set (N=38,940), the baseline demographic variables were sex, age and country of residence. For descriptive statistics see Table 1. This final data set was obtained by excluding some individuals from the initial data set. For example, in the initial data set, 95% of individuals who had children had their first child before the age of 42 and 46 for females and males, respectively. In order to ensure that the vast majority of individuals have completed their reproduction, spouses younger than these ages were excluded from our analysis, as were individuals with missing data for any of the response or explanatory variables — 3677 in total.

Table 1
Summary statistics for the data used in this analysis

Variable	Male	Female
Number of observations, *n*	17,233	21,707
Birth year, mean (S.D.)	1941 (10)	1941 (11)
Countries, *n*		
Austria	762	1101
Belgium	1725	2101
Czech Republic	1143	1551
Denmark	1248	1516
France	1620	2133
Germany	1691	2014
Greece	1614	2077
Italy	1586	1931
Netherlands	1539	1797
Poland	879	1195
Spain	1190	1580
Sweden	1526	1811
Switzerland	710	900
Educational level, *n*		
(1) None, primary or lower secondary	7582	11,798
(2) Upper secondary	5436	6038
(3) Post-secondary or tertiary	4215	3871
Number of children, mean (S.D.)		
Biological	2.00 (1.36)	2.06 (1.38)
Non-biological	0.11 (0.47)	0.09 (0.43)
Proportion childless	0.14	0.13
Work status, *n*		
Employed/self-employed/unemployed/homemaker	7071	12,152
Retired	10,162	9555
Personal income (purchasing power-corrected euros), mean (S.D.)		
Employed/self-employed/unemployed/homemaker	31,873 (70,289)	12,125 (20,237)
Retired	18,978 (20,435)	13,594 (19,127)
Marital status, *n*		
Single	2689	6924
Partnered	740	798
Married	13,804	13,985

2.2. Lifetime reproductive success

We inferred LRS, which we define as the lifetime number of biological children, from a series of questions posed to a single member of each family who acted as the family respondent. Collectively, the individuals included in the sample had 83,021 children, of whom 95% were biological children. Because the questionnaire only asked respondents to specify the biological status (biological offspring/non-biological offspring) of their first four children, we excluded from the dataset the 461 individuals who had more than four children when some of them were non-biological.

2.3. Personal income

In the SHARE database, income is considered to include all forms of financial inflows from various sources and is given as annual income, and where data were missing they are imputed (SHARE Survey of Health, Ageing, and Retirement in Europe, 2010). We converted these raw income data into purchasing power-corrected euros using conversion rates provided in the SHARE database. We then coerced personal income into discrete personal income quartiles (hereafter "income quartile") that were calculated separately for each combination of wave, country, sex and work status. We used income quartiles rather than raw income primarily because some of the key income variables denote gross income in the first wave, but net income in the second wave, and, therefore, coercing the income to quartiles allows the income measures to be comparable between the two waves. We chose to calculate the quartiles separately for each sex within country, because if a man's income acts as an incentive to attract partners, his perceived ranking will be in comparison to other males within the same population (i.e., males from one country do not, on the whole, compete with males from other countries, nor do

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they compete with females). Furthermore, we divided observations into the two work statuses, non-retired (i.e., employed/self-employed/unemployed/homemakers) or retired, because retired people tend to earn less than non-retired people, although their pension-based income is likely to be highly correlated with their salary from before retirement.

2.4. Education

In the SHARE database, education is coded according to the seven levels of the International Standard Classification of Education (ISCED) (UNESCO, 2006). We coerced these classifications into the following three educational levels: (1) none, primary or lower secondary education (ISCED Levels 0, 1 and 2); (2) upper secondary education (ISCED Level 3); and (3) post-secondary and tertiary education (ISCED Levels 4, 5 and 6).

2.5. Statistical analyses

We first examined LRS in relation to sex, income quartile, educational level and birth year using a generalised linear model (GLM) with a Poisson error structure (Model 1). Then, because resources of a family unit are usually shared, we constructed an additional GLM that also included the partner's income quartile (Model 2). We further employed a logistic regression (a GLM with a binomial error structure) to examine the probability of remaining childless in relation to sex, income quartile, educational level and birth year for all individuals (Model 3). In order to assess how much the

Table 2
Summary of the four minimum adequate generalised linear models explaining lifetime reproductive success (Models 1, 2 and 4) and probability of childlessness (Model 3)

	Model 1 estimate (S.E.)	Model 2 estimate (S.E.)	Model 3 estimate (S.E.)	Model 4 estimate (S.E.)
Main effect terms				
Intercept	6.77**,[a] (0.672)	6.811***,[b] (0.095)	26.076***,[a] (3.671)	8.150***,[c] (0.674)
Male	−0.005 (0.015)	−0.007 (0.026)	−45.615*** (5.613)	–
Personal income				
First quartile	0.088*** (0.013)	0.084*** (0.019)	−0.326*** (0.063)	0.054*** (0.010)
Second quartile	0.066*** (0.013)	0.085*** (0.019)	−0.104[†] (0.059)	0.044*** (0.010)
Four quartile	−0.064*** (0.014)	−0.017 (0.021)	0.302*** (0.056)	−0.012 (0.010)
Education level 1	0.133*** (0.009)	0.105*** (0.011)	−0.172*** (0.037)	0.112 (0.009)***
Education level 3	0.003 (0.011)	0.027* (0.013)	0.193*** (0.042)	0.03** (0.011)
Birth year	−0.003*** (0)	−0.003*** (0)	−0.014*** (0.002)	−0.004*** (0)
Male: birth year	–	–	0.024*** (0.003)	–
Partner's income				
First quartile	–	0.036* (0.018)	–	–
Second quartile	–	0.044* (0.018)	–	–
Fourth quartile	–	0.039* (0.018)	–	–
Interaction terms				
Male: Personal income				
First quartile	−0.08*** (0.02)	−0.053* (0.026)	0.663*** (0.087)	–
Second quartile	−0.056** (0.02)	−0.049[†] (0.026)	0.284*** (0.085)	–
Fourth quartile	0.111*** (0.021)	0.058* (0.028)	−0.688*** (0.087)	–
Male: Partner's income				
First quartile	–	0.059* (0.026)	–	–
Second quartile	–	0.051[†] (0.026)	–	–
Fourth quartile	–	−0.052[†] (0.028)	–	–
n	38,940	23,050	38,940	33,786

Models 1, 2 and 4 use a log link and Poisson error structure, while Model 3 uses a binomial error structure. Models 1, 3 and 4 include covariates of sex, income quartile, educational level and birth year. Model 2 includes these, plus union partner's income. Models 1 and 3 include data on all individuals, Model 2 uses a subset of those individuals who have a partner in the sample and Model 4 uses a subset that excludes childless individuals. Coefficient estimates are given along with their standard errors in parentheses and an indication of their p values. The residual deviance was 39,707 on 38,929 *df* for Model 1, 19,488 on 23,033 *df* for Model 2, 30,090 on 38,929 *df* for Model 3 and 17,467 on 33,779 *df* for Model 4. Personal income quartiles were calculated for each combination of country, survey wave, sex and activity status (employed/unemployed/homemaker vs. retired) separately. Educational level 1 includes individuals with no education or primary or secondary education; educational level 2 is equal to upper secondary education; and educational level 3 is equal to post-secondary or tertiary education.

[†] ≤.1.
* $p \leq .05$.
** $p \leq .01$.
*** $p \leq .001$.
[a] Intercept: Female, personal income (third quartile), educational level 2, year of birth 1900.
[b] Intercept: Female, personal income (third quartile), educational level 2, year of birth 1900, partner's personal income (third quartile).
[c] Intercept: Personal income (third quartile), educational level 2, year of birth 1900.

general patterns might be driven by childlessness, we also modelled LRS for a subset of individuals who have at least one child (Model 4). Finally, to gain an understanding of the effect of country on these relationships, we fitted a further model that modified Model 1 to include country as an additional covariate (Model 5, see Supplementary Material for results, available on the journal's website at www.ehbonline.org).

Minimum adequate models were obtained by the stepwise backwards elimination of non-significant terms. The initial model included all of the predictors described above, the two-way interactions between them and, in Model 5 only, the three-way interactions between country, sex and personal income quartile/educational level. We then eliminated terms from these models sequentially, testing the significance of the dropped terms using the X^2 approximation of the distribution of the likelihood ratio test statistic ($p \leq .05$).

Finally, we used hierarchical partitioning (HP) in order to quantify the independent and joint contribution of each predictor variable to explain the variance in the response variable (Chevan & Sutherland, 1991). The HP method avoids the problem of multi-colinearity between the explanatory variables when assessing their contribution to variation. All analyses were conducted in R version 2.11.1 (R Development Core Team, 2011).

3. Results

3.1. The association between LRS and income

LRS was significantly associated with sex, income quartile, educational level and birth year. There was also a significant interaction between sex and income quartile (Table 2, Model 1). Fig. 1 illustrates the sex differences by plotting the predicted LRS by income quartile, education and sex obtained from Model 1. For women (Fig. 1, left panel), LRS tended to decline as income increased, although the difference between the first and second income quartiles was clearly not statistically significant. This association held true for all three educational categories, because the interaction between income and education was not significant. Furthermore, women with post-secondary or tertiary education (Level 3), and upper secondary education (Level 2), tended to have a lower LRS than women with none, primary or lower secondary education (Level 1). However, there was no difference in LRS between the two higher educational levels.

Men (Fig. 1, right panel) belonging to the first three income quartiles had the same LRS and those men with the highest incomes seemed to have slightly higher LRS than the rest. However, the difference is not statistically significant when a post hoc test is applied to this subset of the data (Mann–Whitney U test between third and fourth income quartiles: U=9,075,820, p=.073). Education appeared to have the same effect for both men and women. Accordingly, highly educated men (Levels 2 and 3) tended to have a lower LRS than their less highly educated counterparts (Level 1). Furthermore, the association between birth year and LRS was negative for both sexes.

In a subset of the data containing only those individuals who had a responding partner in the sample, partner income quartile was also significantly associated with LRS (Table 2, Model 2). For women, LRS was lowest for those whose partners were in the third income quartile. For the other quartiles, effect sizes were similar. For men, those

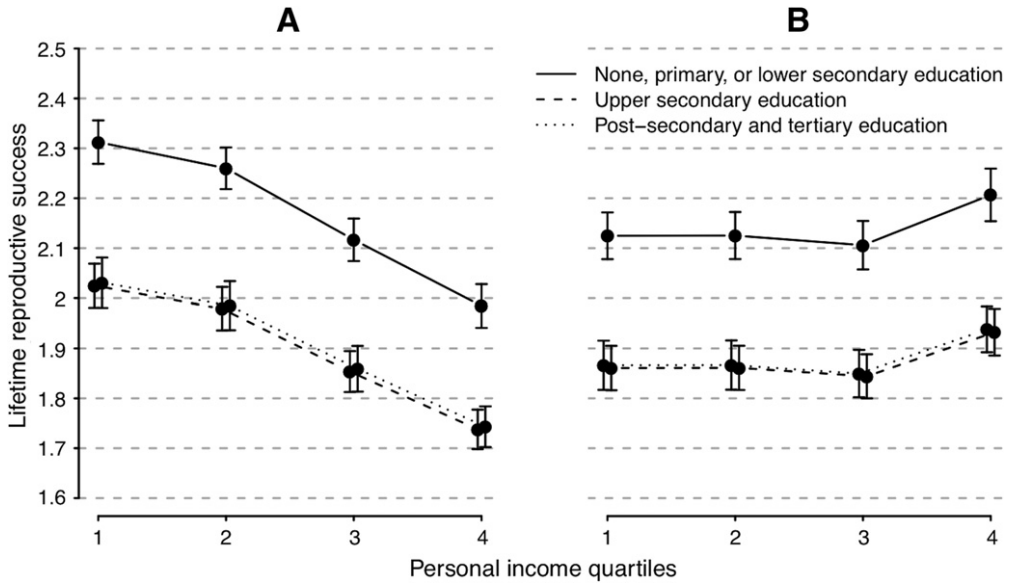

Fig. 1. The association between lifetime reproductive success (number of biological children) and income and education for (A) women and (B) men. Points represent predictions from the generalised linear model (Table 2, Model 1) for individuals born in 1942 (the median birth year in the sample), lines connect predictions for the same educational level and error bars represent 95% confidence intervals.

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who had partners in the first or second income quartile had a higher LRS than those whose partners belonged to the third income quartile. The lowest LRS was found for men whose partners were the highest earners (i.e., in the fourth income quartile). With this subset of the data, controlling for partner income in Model 2 only slightly changed the relationship between LRS and personal income for both sexes when compared to the predictions of Model 1, which used the whole sample. For women, LRS still tended to decline as income increased. However, the decline was less gradual since there was no difference in LRS between women belonging to the two lower income quartiles followed by a more pronounced difference in LRS between women belonging to the second and third income quartile. For men, LRS remained similar across all income quartiles, but men belonging to the third income quartile tended to have a slightly lower LRS than men belonging to the other income quartiles.

3.2. The association between childlessness and income

The probability of remaining childless (hereafter "childlessness") was significantly associated with sex, income quartile and educational level. There was also a significant interaction between sex and income quartile (Fig. 2 and Table 2, Model 3). The association between income quartile and childlessness differed between the sexes (Fig. 2A and B). It is clear that women's childlessness increased with income quartile (Fig. 2A). However, for men, the association was reversed, and men in higher income quartiles tended to have lower levels of childlessness (Fig. 2B). Educational level was positively associated with childlessness for both sexes. Therefore, people who had attained a post-secondary or tertiary education (Level 3) had the highest levels of childlessness. The association between birth year and childlessness is negative for women and positive for men.

3.3. The association between LRS and income among those who have at least one child

There were significant associations between LRS and income quartile, educational level and birth year in a subset of the data that only included those who had at least one child (Fig. 3 and Table 2, Model 4). In this subset, income quartile was negatively associated with LRS for both sexes. This differs from the findings for the whole sample, since, with the whole sample, the relationship between income and LRS was negative for women but flat to slightly positive for men. This analysis therefore reveals that the observed sex difference in the association between LRS and income quartile in the whole dataset is entirely driven by the sex difference in the association between childlessness and income quartile. Once individuals have entered parenthood, for both men and women, those in lower income quartiles had more children than those in higher income quartiles.

The relationship between LRS and education was not straightforward. Individuals who were in the lower educational level (none, primary or lower secondary education) had the highest LRS. Interestingly, however, people who attained a post-secondary or tertiary education had an intermediate LRS, and people who attained upper secondary education had the lowest LRS. This contrasts with the findings for the whole sample, where there was no detectible difference in LRS between the two higher educational levels. Therefore, even though highly educated individuals who chose to have children had more children than those with an

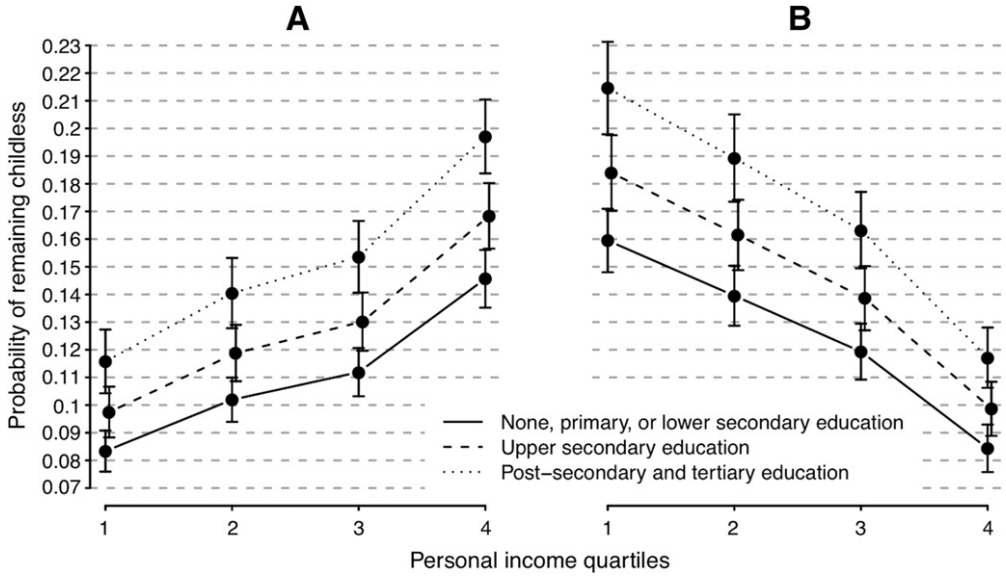

Fig. 2. The association between childlessness and income and education for (A) women and (B) men. Points represent predictions from the generalised linear model (Table 2, Model 3) for individuals born in 1942 (the median birth year in the sample), lines connect predictions for the same educational level and error bars represent 95% confidence intervals.

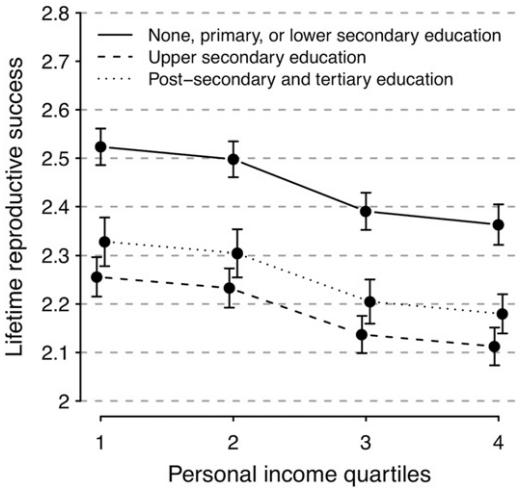

Fig. 3. The association between lifetime reproductive success (number of biological children) and income and education for men and women in a subset of data that excludes childless individuals. Sex was not a significant predictor in this model and, therefore, the points represent predictions for both sexes. Points represent predictions from the generalised linear model (Table 2, Model 4) for individuals born in 1942 (the median birth year in the sample), lines connect predictions for the same educational level and error bars represent 95% confidence intervals.

intermediate-level education, a higher proportion of highly educated individuals remained childless. These two effects cancelled each other out, leading to the potentially misleading observation that there were no differences in LRS between the two higher educational levels in the sample containing all individuals. Birth year was negatively associated with LRS for both sexes.

3.4. Differences among countries

The relationship between LRS and sex, income quartile and educational level varied among countries (Model 5, see Table S1, Supplementary Material, for full model output, available on the journal's website at www.ehbonline.org). This was due to significant interactions between income quartile and country, between educational level and country, and between sex, income quartile and country. Differences were, for the most part, quantitative, and the qualitative patterns of the different countries were generally consistent with Model 1, which did not include country as a covariate (see Section 3.1). For examples of country patterns, see Fig. S1 (Supplementary Material, available on the journal's website at www.ehbonline.org).

3.5. Amount of variance explained by the predictors

The minimum adequate model that included country as a covariate (Table S1, Model 5, available on the journal's website at www.ehbonline.org) reduced deviance only by 4% when compared to the null model. Hierarchical partitioning revealed that the variable with the highest explanatory power was country (53% of the explained variation), followed by education (25% explained variation) and birth year (11% explained variation). Income itself accounted for 8% of the explained variation and the lowest contribution to explained variation was from sex with 1%.

4. Discussion

In this study, we found clear sex differences in both the association between LRS and income and the association between the probability of remaining childless and income. The association between LRS and income was negative for women and flat to slightly positive for men. The probability of remaining childless increased with income for women, but it decreased with income for men. For individuals with at least one child, LRS was negatively associated with income for both sexes. We therefore conclude that it was the sex difference in the association between the probability of remaining childless and income that drove the observed sex difference in the association between LRS and income. There was also a sex difference in the association between LRS and the income of the partner. Women with partners who had a medium-high income had the lowest LRS after controlling for personal income. For men, those who had partners with medium-high to high income have lower LRS than those who had partners with smaller incomes. Finally, we found that the associations of LRS with income differed among countries.

Largely, our findings are in line with those of most previous studies. Our finding that the association between female LRS and income in contemporary Europe is reversed in comparison to historical and contemporary small-scale societies is congruent with several other studies in economically developed countries (e.g., Fieder & Huber, 2007; Hopcroft, 2006; Nettle & Pollet, 2008). Our finding that the association of LRS with male income is flat or slightly positive is also qualitatively consistent with observations from historical and small-scale societies (Gurven & von Rueden, 2006; Røskaft et al., 1992). However, hunting success or land ownership, which served as resource availability measures in these studies, was more clearly positively associated with LRS. This causes a quantitative difference between our findings and these results. Furthermore, our finding for men is also in accordance with most findings on the association between LRS and income for countries that have completed the demographic transition (e.g., Fieder & Huber, 2007; Hopcroft, 2006; Nettle & Pollet, 2008; Weeden et al., 2006). However, in most of these studies, the association between LRS and the measure of resource availability is again more clearly positive and therefore quantitatively different from our finding. Finally, our result contradicts some previous work, which found that men with higher income, status or wealth had fewer children than their less successful counterparts (Kanazawa, 2003; Pérusse, 1993; Vining, 1986).

In our analysis, the exclusion of childless individuals did not alter the association between LRS and income for women. For men, however, the picture was different; the

exclusion of childless individuals changed the nature of the association between LRS and income from being slightly positive to negative. This effect was similar to, but stronger than, the ones reported by Nettle and Pollet (2008) and Fieder and Huber (2007) who both found that, for men, the exclusion of childless individuals caused the relationship between LRS and income to change from positive to flat.

We propose that a likely explanation for our observed population-level patterns of the association between LRS and income is that they are an outcome of evolved behavioural predispositions interacting with modern environments. In the past, when women depended on men for resource provision, there was a close connection between the quantity of a man's resources and his partner's reproductive success. In modern secular societies, a woman's route to optimal resource access is no longer via her partner; instead, women can themselves achieve high social hierarchy positions and income levels. Women spend many years in education and face high opportunity costs when dropping out of the labour force for childbirth. Due to the resulting new trade-off between maximising access to resources through their own labour force participation and childbearing, a negative association between women's income and LRS has emerged (clearly seen in Fig. 1A).

This negative association might be relaxed by the high income of their partners. We could, however, not find support for such an effect, since women with high-income partners did not have a higher LRS than women with low-income partners, and since there was no significant interaction between partner income and personal income. Our findings seem to contradict Huber and Bookstein's (2010) report of a positive association between the LRS of US women and their husbands' incomes. However, when the authors statistically controlled for ethnicity, the effect size was small — an extra 0.004 children for every US$1000 increase in husband's annual income. Due to our large sample size, it seems unlikely that we lack sufficient power to detect an effect. Therefore the difference may be related to societal differences between the United States and Europe.

Evolution may have endowed women with a behavioural predisposition to optimise available resources per offspring, rather than to simply maximise the number of offspring (discussed for Kipsigis men in Luttbeg, Borgerhoff Mulder & Mangel, 2000). In modern market economies, there is almost no ceiling to the quantity of resources per offspring that can be accumulated. In evolutionary terms, this is a novel condition and it is conceivable that females might lack an evolved sense of what amount of resources is sufficient to successfully rear their offspring. This could lead to a runaway process of investing more and more in fewer children (Mace, 2007). Women may wish to have fewer children in order to increase per-child investment, which may improve their children's prospects (Lawson & Mace, 2011; Mace, 2007). In small families, children spend, for example, more time with parents (Lawson & Mace, 2009), perform better in educational assessments (Steelman, Powell, Werum & Carter, 2002) and receive more money (Keister, 2003). Reducing family size may therefore secure long-term survival of the lineage (Kaplan et al., 1995; Lawson & Mace, 2011; Mace, 2007).

Cross-culturally, females value cues to resource acquisition in prospective partners, which has been interpreted as an adaptive preference (Buss, 1989; Buss, Shackelford, Kirkpatrick & Larsen, 2001). Consequently, high-status men tend to have a higher copulation frequency and more sexual partners than their low-status counterparts (Hopcroft, 2006; Pérusse, 1993). Although more sex does not necessarily lead to more children (Pérusse, 1993), men still increase their LRS by breakup and re-marriage in societies characterised by serial monogamy (Jokela, Rotkirch, Rickard, Pettay & Lummaa, 2010). Attaining a high status could therefore result in a reproductive advantage, if it aids re-partnering. The re-partnering of successful men may also reduces the pool of young desirable women and thus increases competition on the marriage market. Some low-income men may therefore encounter difficulties in finding a partner to have children with. Therefore, our result that income plays an important role in determining the probability of remaining childless is not surprising: men with lower income are at a higher risk of remaining childless. Interestingly, once men have found a partner and have at least one child, men with lower income tend to have more children than those with a high income. Although increased income is associated with improved chances of being chosen as a partner for reproduction (i.e., transition from being childless to having a child), it seems to be negatively associated with having further children.

Our observation that men with high-income partners have lower LRS than men who have partners with a lower income can be explained by assortative mating. Couples often share educational backgrounds and income levels (Kalmijn, 1998). Thus, the LRS of high-earning men is likely to be constrained somewhat by the behaviour of their highly educated, high-earning wives. This is congruent with Kaplan and Lancaster's (2000) observation of the effect of wives' education on male LRS. However, by choosing a wife with a high income, men may maximise resources available per offspring instead of maximising the number of offspring, thus increasing the competitive chances of offspring (Luttbeg et al., 2000).

We found that the associations between education and LRS, and childlessness, differed from the associations between income and LRS, and childlessness, in that there was no sex difference in the first, whereas there was one in the latter. It follows therefore that studies that use income and education as predictors for reproductive behaviour of both sexes need to take these opposing associations into account. That the association between education and LRS was negative for both sexes is surprising, since men do not face the same limitation in the number of fertile years women do. This finding could indicate that unobserved factors determine the effect of education on LRS in addition to physiological constraints. However, we cannot exclude the possibility that education has a negative effect on LRS for both sexes

because highly educated women constrain the reproductive success of their likewise highly educated partners.

Although our results are revealing, our study has several data and study design limitations. First, while our analysis reveals important associations between income and LRS, it does not allow us to draw conclusions about causality or about the direction of potential causality. In particular, our study relies on cross-sectional data, which do not allow us to control for unobserved characteristics, such as physical attractiveness, career aspirations or personality traits. These unobserved characteristics may correlate with both income and fertility and bias the estimated income–LRS association away from the true causal effect of income on LRS. For example, our finding that men with low income are more likely to remain childless than their high-income counterparts could be enhanced or even caused by the fact that men who are unattractive as partners may possess attributes that also make them unproductive or undesirable employees. In addition, the chain of causation may be more complex than our models imply or could even be reversed. For example, marriage status and number of children could directly affect income, rather than income affecting the number of children. Indeed, previous workers have found that each additional child decreases female wages (Budig & England, 2001), while marriage tends to increase male earnings (Antonovics & Town, 2004). Future studies could consider using longitudinal data in which unobserved characteristics can be controlled and the time ordering of events is known. Second, data on fertility and personal income for both sexes are scarce. As a solution, we decided to use survey data, despite the known problems of non-response and underreporting of children by men (Rendall, Clarke, Peters, Ranjit & Verropoulou, 1999). Both factors may have influenced the study outcome if, as is likely, they were not consistent across all income quartiles and educational levels. Third, in our study, income was measured at various stages after the child-bearing years. However, couples form and make fertility decisions much earlier in their lives and the observed income is therefore only a proxy for available resources at point of time of fertility decisions. Fourth, fertility rates in our study populations have changed over time and it is likely that the rates of change differed between socioeconomic groups. In this analysis, we control for birth year and thus for population-wide changes in fertility over time, but we do not address differences in rates of change among socioeconomic groups. However, birth year had only small effects on LRS in our analyses. Fifth, how many children people have, and when they have them, differs among countries (Human Fertility Database). Our result that country accounted for 53% of explained variation in our model of LRS is therefore not surprising. Here we aim to show that, despite this considerable cross-country variation, sex differences in the association between LRS and income are pervasive in economically developed countries. We did not aim to explain the details of cross-country differences in the associations.

5. Conclusion

In this study, we have confirmed the existence of sex differences in the association between LRS and income in economically developed countries. We argue that the dependency on resources during the majority of human evolution may have shaped male and female reproductive strategies differently. Although both sexes strive for resources, men mostly do so to compete for partners, while women mostly do so to ensure parental investment. We also discussed how women's labour force participation hampers female LRS and argued that women's preference for high-income partners may help explain the fact that high-income men have a lower risk of childlessness than low-income men and have the same or a slightly higher LRS.

Since LRS is an important correlate of fitness, it is possible that Darwinian selection still acts on male wealth in modern populations, as claimed by Nettle and Pollet (2008). If we accept this argumentation for males, we also need to accept the reverse argument for females. Darwinian selection should act to produce future female generations that prefer to have many children while earning a low income. At the same time, males should avoid partnering with high-income females, who, as we have shown, reduce their partners' LRS. However, Europeans will continue to change their environment and behaviour much faster than any evolutionary change can occur and, therefore, applying fitness-maximising models to humans in economically developed societies will remain challenging.

Supplementary Materials

Supplementary data to this article can be found online at doi:10.1016/j.evolhumbehav.2012.03.003.

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Table S1. Summary of the minimum adequate generalised linear model for lifetime reproductive success (LRS) as a function of sex, personal income, educational level, birth year, and country (Model 5). A Poisson error structure was used. Coefficient estimates are given along with their standard errors and p–values. The residual deviance was 38,807 on 38,807 d.f.. Personal income quartiles were calculated separately for each country, survey wave, sex, and activity status (employed/ unemployed/ homemaker vs. retired) combination and are abbreviated as "qt.". Educational level 1 is equal to no education, primary, or secondary- education, educational level 2 is equal to upper secondary education, and educational level 3 is equal to post-secondary or tertiary education.

Main effect terms	**Estimate (SE)**	**Interaction terms**	**Estimate (SE)**
Intercept[a]	6.671*** (0.687)	Denmark: educational level 3	–0.016 (0.057)
Male	0.053 (0.072)	France: educational level 1	–0.089† (0.048)
Personal income (1. qt.)	0.139* (0.06)	France: educational level 3	–0.023 (0.058)
Personal income (2. qt.)	0.057 (0.066)	Germany: educational level 1	0 (0.052)
Personal income (4. qt.)	–0.086 (0.066)	Germany: educational level 3	0.028 (0.055)
Education level 1	0.173*** (0.041)	Greece: educational level 1	–0.094 (0.05)
Education level 3	–0.027	Greece:	–0.018

	(0.048)	educational level 3	(0.061)
	0.153*	Italy:	0.043
Belgium	(0.062)	educational level 1	(0.053)
	0.015	Italy:	–0.083
Czechia	(0.066)	educational level 3	(0.076)
	0.091	Netherlands:	–0.106†
Denmark	(0.065)	educational level 1	(0.05)
	0.106†	Netherlands:	–0.022
France	(0.061)	educational level 3	(0.058)
	0.045	Poland:	0.041
Germany	(0.061)	educational level 1	(0.051)
	0.099	Poland:	–0.02
Greece	(0.065)	educational level 3	(0.068)
	0.052	Spain:	0.089
Italy	(0.066)	educational level 1	(0.064)
	0.343***	Spain:	0.111
Netherlands	(0.062)	educational level 3	(0.082)
	0.26***	Sweden:	–0.241***
Poland	(0.065)	educational level 1	(0.052)
	0.17*	Sweden:	–0.026
Spain	(0.076)	educational level 3	(0.059)
	0.232***	Switzerland:	–0.089
Sweden	(0.065)	educational level 1	(0.058)
	0.205**	Switzerland:	–0.106
Switzerland	(0.073)	educational level 3	(0.067)

Birth year	–0.003***	Male: Belgium:	–0.036
	(0)	personal income (3. qt.)	(0.085)
Interaction terms		Male: Belgium:	–0.041
		personal income (1. qt.)	(0.08)
Male:	–0.17†	Male: Belgium:	0.012
personal income (1. qt.)	(0.098)	personal income (2. qt.)	(0.088)
Male:	–0.031	Male: Belgium:	–0.168*
personal income (2. qt.)	(0.101)	personal income (4. qt.)	(0.085)
Male:	0.171†	Male: Czechia:	–0.095
personal income (4. qt.)	(0.1)	personal income (3. qt.)	(0.094)
Male: educational level 1	–0.005	Male: Czechia:	0.067
	(0.019)	personal income (1. qt.)	(0.089)
Male: educational level 3	0.033	Male: Czechia:	–0.062
	(0.022)	personal income (2. qt.)	(0.093)
Belgium:	0.019	Male: Czechia:	–0.283**
personal income (1. qt.)	(0.072)	personal income (4. qt.)	(0.093)
Belgium:	–0.099	Male: Denmark:	–0.023
personal income (2. qt.)	(0.082)	personal income (3. qt.)	(0.091)
Belgium:	0.032	Male: Denmark:	–0.068
personal income (4. qt.)	(0.08)	personal income (1. qt.)	(0.088)
Czechia:	–0.051	Male: Denmark:	–0.07
personal income (1. qt.)	(0.081)	personal income (2. qt.)	(0.09)
Czechia:	–0.022	Male: Denmark:	–0.178*
personal income (2. qt.)	(0.085)	personal income (4. qt.)	(0.091)
Czechia:	0.111	Male: France:	0.03

personal income (4. qt.)	(0.086)	personal income (3. qt.)	(0.086)
Denmark:	–0.025	Male: France:	0.01
personal income (1. qt.)	(0.08)	personal income (1. qt.)	(0.08)
Denmark:	0.05	Male: France:	–0.111
personal income (2. qt.)	(0.084)	personal income (2. qt.)	(0.085)
Denmark:	0.112	Male: France:	–0.073
personal income (4. qt.)	(0.085)	personal income (4. qt.)	(0.085)
France:	0.138†	Male: Germany:	–0.113
personal income (1. qt.)	(0.073)	personal income (3. qt.)	(0.088)
France:	0.142†	Male: Germany:	0.106
personal income (2. qt.)	(0.078)	personal income (1. qt.)	(0.083)
France:	0.102	Male: Germany:	–0.064
personal income (4. qt.)	(0.08)	personal income (2. qt.)	(0.088)
Germany:	–0.067	Male: Germany:	–0.168†
personal income (1. qt.)	(0.076)	personal income (4. qt.)	(0.088)
Germany:	–0.041	Male: Greece:	–0.094
personal income (2. qt.)	(0.081)	personal income (3. qt.)	(0.088)
Germany:	–0.01	Male: Greece:	0.063
personal income (4. qt.)	(0.082)	personal income (1. qt.)	(0.081)
Greece:	–0.062	Male: Greece:	–0.04
personal income (1. qt.)	(0.074)	personal income (2. qt.)	(0.088)
Greece:	–0.032	Male: Greece:	–0.162†
personal income (2. qt.)	(0.081)	personal income (4. qt.)	(0.087)
Greece:	0.055	Male: Italy:	–0.163†
personal income (4. qt.)	(0.082)	personal income (3. qt.)	(0.088)

Italy:	–0.09	Male: Italy:	0.087
personal income (1. qt.)	(0.077)	personal income (1. qt.)	(0.085)
Italy:	0.009	Male: Italy:	–0.074
personal income (2. qt.)	(0.078)	personal income (2. qt.)	(0.084)
Italy:	0.013	Male: Italy:	–0.121
personal income (4. qt.)	(0.082)	personal income (4. qt.)	(0.087)
Netherlands:	–0.167*	Male: Netherlands:	–0.136
personal income (1. qt.)	(0.074)	personal income (3. qt.)	(0.086)
Netherlands:	–0.036	Male: Netherlands:	0.066
personal income (2. qt.)	(0.079)	personal income (1. qt.)	(0.082)
Netherlands:	–0.103	Male: Netherlands:	–0.123
personal income (4. qt.)	(0.081)	personal income (2. qt.)	(0.086)
Poland:	0.009	Male: Netherlands:	–0.015
personal income (1. qt.)	(0.078)	personal income (4. qt.)	(0.086)
Poland:	–0.017	Male: Poland:	0.025
personal income (2. qt.)	(0.085)	personal income (3. qt.)	(0.092)
Poland:	0.096	Male: Poland:	–0.076
personal income (4. qt.)	(0.085)	personal income (1. qt.)	(0.088)
Spain:	0.282**	Male: Poland:	–0.043
personal income (1. qt.)	(0.106)	personal income (2. qt.)	(0.092)
Spain:	–0.017	Male: Poland:	–0.236*
personal income (2. qt.)	(0.077)	personal income (4. qt.)	(0.093)
Spain:	–0.068	Male: Spain:	–0.088
personal income (4. qt.)	(0.082)	personal income (3. qt.)	(0.088)
Sweden:	–0.079	Male: Spain:	0.217†

personal income (1. qt.)	(0.076)	personal income (1. qt.)	(0.112)
Sweden:	–0.038	Male: Spain:	–0.123
personal income (2. qt.)	(0.081)	personal income (2. qt.)	(0.085)
Sweden:	0.039	Male: Spain:	–0.05
personal income (4. qt.)	(0.082)	personal income (4. qt.)	(0.089)
Switzerland:	–0.132	Male: Sweden:	–0.011
personal income (1. qt.)	(0.089)	personal income (3. qt.)	(0.087)
Switzerland:	0.012	Male: Sweden:	–0.01
personal income (2. qt.)	(0.092)	personal income (1. qt.)	(0.084)
Switzerland:	–0.143	Male: Sweden:	–0.023
personal income (4. qt.)	(0.097)	personal income (2. qt.)	(0.087)
Belgium:	–0.117*	Male: Sweden:	–0.17†
educational level 1	(0.049)	personal income (4. qt.)	(0.087)
Belgium:	0.077	Male: Switzerland:	–0.137
educational level 3	(0.057)	personal income (3. qt.)	(0.102)
Czechia:	–0.095†	Male: Switzerland:	0.093
educational level 1	(0.052)	personal income (1. qt.)	(0.098)
Czechia:	0.055	Male: Switzerland:	–0.183†
educational level 3	(0.068)	personal income (2. qt.)	(0.101)
Denmark:	–0.048	Male: Switzerland:	0.014
educational level 1	(0.054)	personal income (4. qt.)	(0.105)

†≤ .1, * p ≤ .05, ** p ≤ .01, *** p ≤ .001

[a] Intercept: Female, from Austria, personal income (3. quartile), educational level 2, year of birth 1900

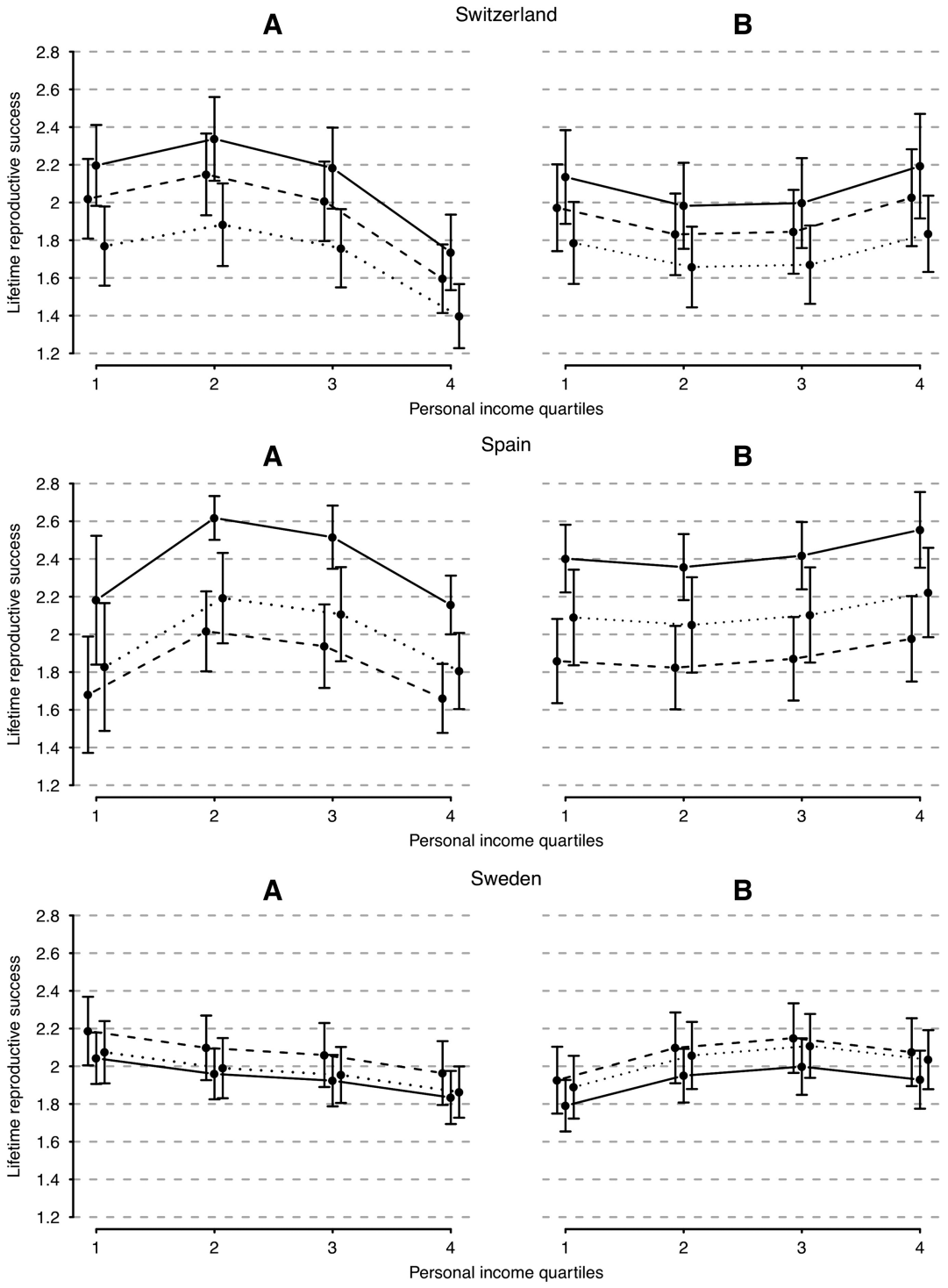

Figure S1. The association between lifetime reproductive success (number of biological children) and income and education for (A) women and (B) men for Switzerland, Spain, and Sweden. Points represent predictions from the generalised linear model (Table S1) for individuals born in 1942 (the median birth year in the sample), lines connect predictions for the same educational level and error bars represent 95% confidence intervals.