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Genetic Analysis of Life-History Constraint and Evolution in a Wild Ungulate Population

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ABSTRACT: Trade-offs among life-history traits are central to evolutionary theory. In quantitative genetic terms, trade-offs may be manifested as negative genetic covariances relative to the direction of selection on phenotypic traits. Although the expression and selection of ecologically important phenotypic variation are fundamentally multivariate phenomena, the in situ quantification of genetic covariances is challenging. Even for life-history traits, where well-developed theory exists with which to relate phenotypic variation to fitness variation, little evidence exists from in situ studies that negative genetic covariances are an important aspect of the genetic architecture of life-history traits. In fact, the majority of reported estimates of genetic covariances among life-history traits are positive. Here we apply theory of the genetics and selection of life histories in organisms with complex life cycles to provide a framework for quantifying the contribution of multivariate genetically based relationships among traits to evolutionary constraint. We use a Bayesian framework to link pedigree-based inference of the genetic basis of variation in life-history traits to evolutionary demography theory regarding how life histories are selected. Our results suggest that genetic covariances may be acting to constrain the evolution of female life-history traits in a wild population of red deer *Cervus elaphus*: genetic covariances are estimated to reduce the rate of adaptation by about 40%, relative to predicted evolutionary change in the absence of genetic covariances. Furthermore, multivariate phenotypic (rather than genetic) relationships among female life-history traits do not reveal this constraint.

Keywords: life history, quantitative genetics, natural selection, constraint, projection model, sensitivity, red deer, *Cervus elaphus*.

Introduction

Genetically based relationships among traits, especially life-history traits, are a primary determinant of the po-

tential for adaptive phenotypic evolution (Stearns 1989; Lynch and Walsh 1998; Roff 2002; Roff and Fairbairn 2007). Details of multivariate genetic architecture have been proposed as explanations for important general discordances between empirical data and naive evolutionary predictions, including the maintenance of heritable variation in populations (Walsh and Blows 2009), and stasis despite apparent directional selection of heritable traits (Merilä et al. 2001). Additionally, genetically based trade-offs among traits—that is, genetic correlations opposing the direction of multivariate selection—implicitly underlie optimality models, which are a widely used approach for understanding adaptation (Stearns 1977). However, the empirical quantitative genetic study of multivariate genetic constraints, particularly in natural populations or unmanipulated organisms, has lagged behind the available theory. What empirical data do exist are generally contrary to the contention that the manifestation of trade-offs as negative genetic correlations among selected traits is an important form of evolutionary constraint; direct evidence for constraining genetic correlations in nature is surprisingly weak (Kruuk et al. 2008). A number of in situ estimates of genetic correlations among ecologically important traits have been reported in a number of study systems (reviewed in Kruuk et al. 2008). The vast majority of these estimates are positive, including genetic correlations among life-history traits. The existence of an overall genetic constraint is not inconsistent with the occurrence of some positive correlations among selected traits (Charlesworth 1990; Houle 1991), but nonetheless, the difficulty of detecting these constraints in a quantitative genetic framework is somewhat surprising.

Major obstacles to studying the consequences of multivariate genetic architectures, especially in natural populations, are presented by the combination of the large uncertainty in typical estimates of genetic covariances and

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correlations (Lynch and Walsh 1998; Kruuk and Hill 2008) and the large number of parameters that need to be estimated if analytical models are constructed that truly reflect the multivariate nature of phenotypic evolution. Some workers have adopted a bivariate strategy, whereby hypotheses of absolute constraint—that is, of factors constraining responses to selection to nil—can be rejected if genetic correlations can be shown to be different from ± 1 (especially in studies of intersexual correlations; reviewed in Poissant et al. 2010). This bivariate approach acknowledges the importance of genetic covariances in phenotypic evolution but provides a relatively low-dimensional approach to analyses. Unfortunately, bivariate analyses are little more likely than univariate analyses to elucidate mechanisms of genetic constraint, because absolute constraints can exist in the absence of correlations of ± 1 when more than two traits determine fitness (Dickerson 1955; Roff and Fairbairn 2007; Walsh and Blows 2009), and sampling error can easily overwhelm matrix algebra-based approaches (Hill and Thompson 1978; Agrawal and Stinchcombe 2009). Dickerson (1955; see also Robertson 1955; Charlesworth 1984) considered positive directional selection of a set of k traits, all with genetic correlations of $-1/(k-1)$. Such a pattern of selection and covariance yields no adaptive evolution, because such a variance-covariance matrix has an eigenvector of length 0 (no variance) that is parallel to the direction of selection. Thus, if variation in many traits falls along relatively few composite axes in a given population (Barton 1990), multiple modest genetic correlations can generate absolute constraints. Given the large uncertainties that are inevitable in estimates of genetic correlations (Lynch and Walsh 1998), consideration of individual correlations is unlikely to provide insight into the form of genetic constraint (Roff and Fairbairn 2007; Walsh and Blows 2009). Other workers have taken to testing whether suites of traits are effectively the same or, similarly, whether matrices describing multivariate phenotypic variation have trait combinations with no variation (zero eigenvalues) and, additionally, whether primary axes of variation are largely orthogonal to the direction of selection (Blows et al. 2004; Mezey and Houle 2005; McGuigan and Blows 2007). These approaches have clearly contributed greatly to our understanding of the relationship between multivariate genetic variation and multivariate selection. However, analysis of multivariate \mathbf{G} matrices—and of mathematical constructs to describe their principal axes of variation—will generally be difficult to apply outside of lab studies, where statistical power is generally much less limiting, owing to controlled environments, manipulated pedigrees, and very large sample sizes. Lower-dimension metrics of evolutionary constraint would thus be very useful.

Several approaches are available or ripe for development

that can provide information about constraints arising from multivariate genetic relationships among traits, without necessarily requiring hypothesis testing of multiple genetic correlations in isolation and also without necessarily having to test for zero eigenvalues or to characterize mathematically derived composite axes of variation. For example, Agrawal and Stinchcombe (2009) developed the metric of evolutionary constraint R , which is the ratio of the expected increase in population mean fitness due to one generation of selection and response, accounting for genetic correlations among traits, to the expected increase in population mean fitness in the absence of correlations. This type of metric is potentially very useful, because it distills the details of genetic variance-covariance matrices, \mathbf{G} , into a single metric, rather than a problem based on the $(k^2 + k)/2$ parameters of \mathbf{G} . In this regard, the response to selection, $\Delta\bar{\mathbf{z}}$, in the multivariate breeder's equation—that is, $\Delta\bar{\mathbf{z}} = \mathbf{G}\beta$ (Lande 1979, 1982; Lande and Arnold 1983)—can be regarded as a k parameter emergent property of an analysis that otherwise has $(k^2 + k)/2 + k$ (\mathbf{G} and β) parameters. While uncertainties in emergent parameters of such analyses—that is, the direction and magnitude of responses to selection—are typically not reported, they can be calculated or at least roughly estimated, and it does not necessarily follow that uncertainties in emergent metrics (e.g., R , $\Delta\bar{\mathbf{z}}$) will be large as a consequence of uncertainties in components of \mathbf{G} . We propose that the development of approaches for the explicit joint analysis of selection and genetic architectures will allow uncertainties in these metrics to be calculated, and this will lead to more powerful inferences of evolutionary patterns and processes. For example, Agrawal and Stinchcombe's R could potentially be statistically significantly smaller than its null value of 1, even when no pairwise genetic correlations or covariances differ significantly from 0, provided that some estimates of multiple off-diagonal elements of \mathbf{G} are negative (and provided that they do not have a very strong negative sampling covariance).

In studies of the selection, evolution, and constraint of life-history traits, demographic approaches may also provide useful tools (Lande 1982; Charlesworth 1994; Caswell 2001; Coulson et al. 2003, 2010). Demographic analysis can provide model-based inference of the form of natural selection, especially of life-history traits, giving context to inferences of \mathbf{G} , and any constraint that may be represented by genetic correlations. This line of reasoning has a solid theoretical history (Lande 1982; Charlesworth 1993). There are strong theoretical foundations to demographic theory and its description of how variation in life-history traits should influence fitness variation (Lande 1982; Caswell 2001). Consequently, the form of selection of life histories can be derived from life-history data rather than by reliance on linear model-based methods of re-

lating total fitness to individual phenotype and their associated assumptions (Lande and Arnold 1983; Shaw et al. 2008; Morrissey et al. 2010). In particular, population projection matrices (Caswell 2001), such as Leslie matrices (Leslie 1945, 1948), describe temporal changes in population size and composition as a function of vital rates (survival and reproduction). Population projection models could be more widely exploited for modeling the selection, evolution, and constraint of life histories (van Tienderen 2000; Coulson et al. 2003). Vital rates are (typically annual) expected values of life-history traits, that is, survivorship and fecundity. Projection matrices can be used to obtain the population growth rate, λ , at the stable age distribution (or indeed otherwise), which provides a measure of mean absolute fitness in a population expressing a given life history. Evaluation of how λ covaries with life-history traits provides insight into the demographic effects of life-history variation. These effects are quantified through sensitivities, which are formally partial derivatives of λ with respect to the vital rates or determinants of vital rates—that is, life-history traits—and are equivalent to selection gradients (when scaled to relative fitness or when $\lambda = 1$; van Tienderen 2000; Caswell 2001; Coulson et al. 2003, 2010). Given selection gradients, evolutionary change can be predicted, conditional on \mathbf{G} (Lande 1982), and the magnitude of this change can be used to assess constraint in a manner analogous to Agrawal and Stinchcombe's calculation of R , that is, through comparison of evolutionary predictions both accounting for and discounting genetic correlations among traits.

Here we use generalized linear mixed models (Bolker et al. 2009) to estimate a \mathbf{G} matrix for life-history traits in female red deer *Cervus elaphus* from a long-term individual-based study of a wild population on the Isle of Rum, Scotland (Clutton-Brock et al. 1982). In order to test for genetic constraints on life-history evolution in this population, we obtain estimates of the degree to which covariances in \mathbf{G} reduce the rate of adaptation by combining our estimated \mathbf{G} matrices with a population projection matrix-based framework to model selection of life-history variation. Additionally, as a secondary goal, we compare inferences of the potential for multivariate correlations among life-history traits to constrain evolution, as evaluated by genetic and phenotypic variance-covariance matrices, in order to test the validity of nongenetic inference of multivariate phenotypic evolution; that is, we test the functional equivalence of \mathbf{P} and \mathbf{G} matrices for inference of evolutionary constraint. We conduct all of our analyses in a Bayesian framework and so are able to integrate over uncertainty in \mathbf{G} and in the demographic aspects of our analyses, in order to obtain metrics—and realistic quantification of associated uncertainties—of the

degree of genetic constraint on the adaptive evolution of life histories in this wild population.

Methods

We first describe the study system and phenotypic data (“Study System, Traits, and Pedigree”) and estimation of the \mathbf{G} matrix for a set of female life-history traits (“Quantitative Genetic Model of Life-History (Co)Variation”). In “Sensitivity Analysis,” we outline the use of a projection matrix to estimate sensitivities of λ to variation in the life-history traits and, hence, to infer selection, including estimates of sensitivities that incorporate either the phenotypic or the genetic covariances between the life-history traits. We then predict the evolutionary response to selection based on the estimates of \mathbf{G} and the sensitivity analyses (“Prediction of Evolutionary Trajectories”) and evaluate the evidence for evolutionary constraint by quantifying the effect of covariances on measures of the rate of adaptive evolution and phenotypic evolutionary change (“Quantification of Evolutionary Constraint”).

Study System, Traits, and Pedigree

The unmanaged population of red deer in the North Block of the Isle of Rum, Inner Hebrides, Scotland, has been intensively studied since 1971, and some pedigree and phenotypic data are available from as early as the late 1950s (Clutton-Brock et al. 1982). Very complete life-history data for many females in this population are available from intensive observation during the calving season and from multiple censuses each year. Longevity in red deer on Rum is bimodally distributed (Catchpole et al. 2004), with low mortality of deer aged one to four and relatively higher mortality of calves and reproductive and senescing adults. As a consequence of this bimodality of mortality, we were unable to tractably model (with respect to subsequent needs to evaluate \mathbf{G} and sensitivities of λ to life-history traits) age-specific survival rates across the whole lifetime. We therefore modeled (1) the longevity of individuals that lived to age five or older as a Gaussian trait. We also modeled (2) age at primiparity as a Gaussian trait. We modeled (3) fecundity and (4) offspring first-year survival as binomial traits. For fecundity, we calculated the number of years in which a female produced a calf and the number of years that she was alive post primiparity in which she did not produce a calf. For offspring survival, which we modeled solely as an aspect of maternal phenotype because of the extended period of maternal care, we calculated the number of calves of each female that did and did not survive to age one. For 3 and 4, the traits were scored as the number of successes (i.e., number of calves produced and number of calves surviving, respectively) and the

number of attempts (i.e., number of years lived post primiparity and numbers of years in which a calf was produced, respectively). Our data set consisted of 356 individuals (adult females) who had been phenotyped for at least one of these traits; occurred regularly in annual censuses (specifically, were seen in at least 10% of censuses or an average of at least five times per year, following Coulson et al. 1997); are now known to be dead and to have died natural deaths, such that we can be confident that their life-history data are complete; and were born before 1999, such that most of their contemporaries are dead, reducing censusing bias.

Pedigree information in the Rum red deer study population can be derived from a combination of observational and molecular data. The pedigree has been reconstructed using both data on social interactions and microsatellite genotypes in a Bayesian framework using MasterBayes (Hadfield et al. 2006b), with additional inclusion of inferred relationships of sibs with unsampled sires based on sibship partitions generated by colony (Wang 2004; Jones and Wang 2009). The pedigree is constructed with approximately 98% average confidence in individual parentage assignments. Full description of the procedures for generating the current pedigree is provided by Walling et al. (2010). The portion of the red deer pedigree that is informative with respect to the inference of genetic variation in one or more of the four life-history traits that we analyze here is highly complex, with a wide range of relatedness categories (fig. 1), and contains 452 individuals (the phenotyped females, described above, and additionally any of their unphenotyped, typically male parents), with 378 maternal and 217 paternal links. Of these, 356 are adult females with known longevities, of which there are 330 for which we have data for all three reproductive traits (i.e., age at primiparity, annual fecundity, and offspring survival). The reproductive trait data are based on a total of 2,755 opportunities to reproduce, during which 1,978 calves were produced, 847 of which survived their first year. We generated statistics and graphical representations of the pedigree, conditional on the availability of phenotypic data, using pedantics (Morrissey and Wilson 2010).

Quantitative Genetic Model of Life-History (Co)Variation

We estimated \mathbf{G} by fitting a generalized animal model (Lynch and Walsh 1998; Hadfield 2010) to the red deer life-history data,

$$\mathbf{y} = \boldsymbol{\mu} + \mathbf{X}\mathbf{l} + \mathbf{Z}_1\mathbf{y}\mathbf{r} + \mathbf{Z}_2\mathbf{a} + \mathbf{e}, \quad (1)$$

where \mathbf{y} is a vector of individual phenotypic observations of the life-history traits; $\boldsymbol{\mu}$ is a vector of mean phenotypes; \mathbf{X} is a design matrix relating phenotypic observations to

\mathbf{l} , a vector of the effects of (fixed) phenotypic effects of the areas within the study site in which individuals spent most of their lives; \mathbf{Z}_1 is a design matrix relating observations to cohort (i.e., year of birth) $\mathbf{y}\mathbf{r}$; \mathbf{Z}_2 is a design matrix relating observations to individual genetic effects, \mathbf{a} ; and \mathbf{e} is a vector of multivariate random normal errors. The vector \mathbf{a} contains breeding values, or the expected multivariate phenotypic values of an individual's progeny in an average environment (Falconer 1981). Vector \mathbf{a} is taken from the multivariate normal distribution $\mathbf{a} \sim N(\mathbf{0}, \mathbf{G} \otimes \mathbf{A})$, where \mathbf{G} is the additive genetic variance-covariance matrix of the four modeled life-history traits of female red deer (adult longevity, age at primiparity, fecundity, and offspring survival), \mathbf{A} is the pedigree-derived additive genetic relatedness matrix, and \otimes denotes a Kronecker product. Further information on partitioning of phenotypic variance into additive and other components of variation using general pedigrees and the animal model is provided by Henderson (1973), Lynch and Walsh (1998), Kruuk (2004), and Wilson et al. (2010). The fixed effects of area were associated with individual fidelity to five geographically convenient subsets of the study area and were included to account for spatial variation in demography throughout the study area (Coulson et al. 1997).

We fitted the model specified by equation (1) and sampled the posterior distribution by Gibbs sampling using MCMCglmm (Hadfield 2010). We ran the Markov chain Monte Carlo algorithm for 5×10^5 burn-in iterations, followed by 2×10^6 iterations with a sampling interval of 400, providing 5,000 samples of the posterior distribution of \mathbf{G} . Lacking any definitive prior information, and in order to reduce the effect of arbitrary priors on our inferences, we used priors of $0.2\mathbf{P} \times \mathbf{I}$, where \mathbf{P} is the phenotypic variance-covariance matrix, as obtained from the variance-covariance matrix of \mathbf{e} in equation (1) but where genetic and cohort effects were not estimated; \mathbf{I} is an identity matrix; and the times symbol is used to denote a Hadamard product on the variance-covariance matrices associated with the random and residual effects $\mathbf{y}\mathbf{r}$, \mathbf{a} , and \mathbf{e} . We specified these as improper priors—that is, with a low degree of belief (see Hadfield 2010)—and, as such, estimates of the genetic variances will, if anything, be biased toward values corresponding to commonly observed levels of heritability, and the magnitudes of genetic covariances should, if anything, be downwardly biased.

We calculated the phenotypic variances and covariances of the life-history traits, conditional on locations within the study area, by summing the covariance matrices associated with the \mathbf{a} , $\mathbf{y}\mathbf{r}$, and \mathbf{e} in equation (1). We evaluated the uncertainty in each covariance and correlation at the phenotypic and genetic levels by examining the posterior distribution of each parameter. We

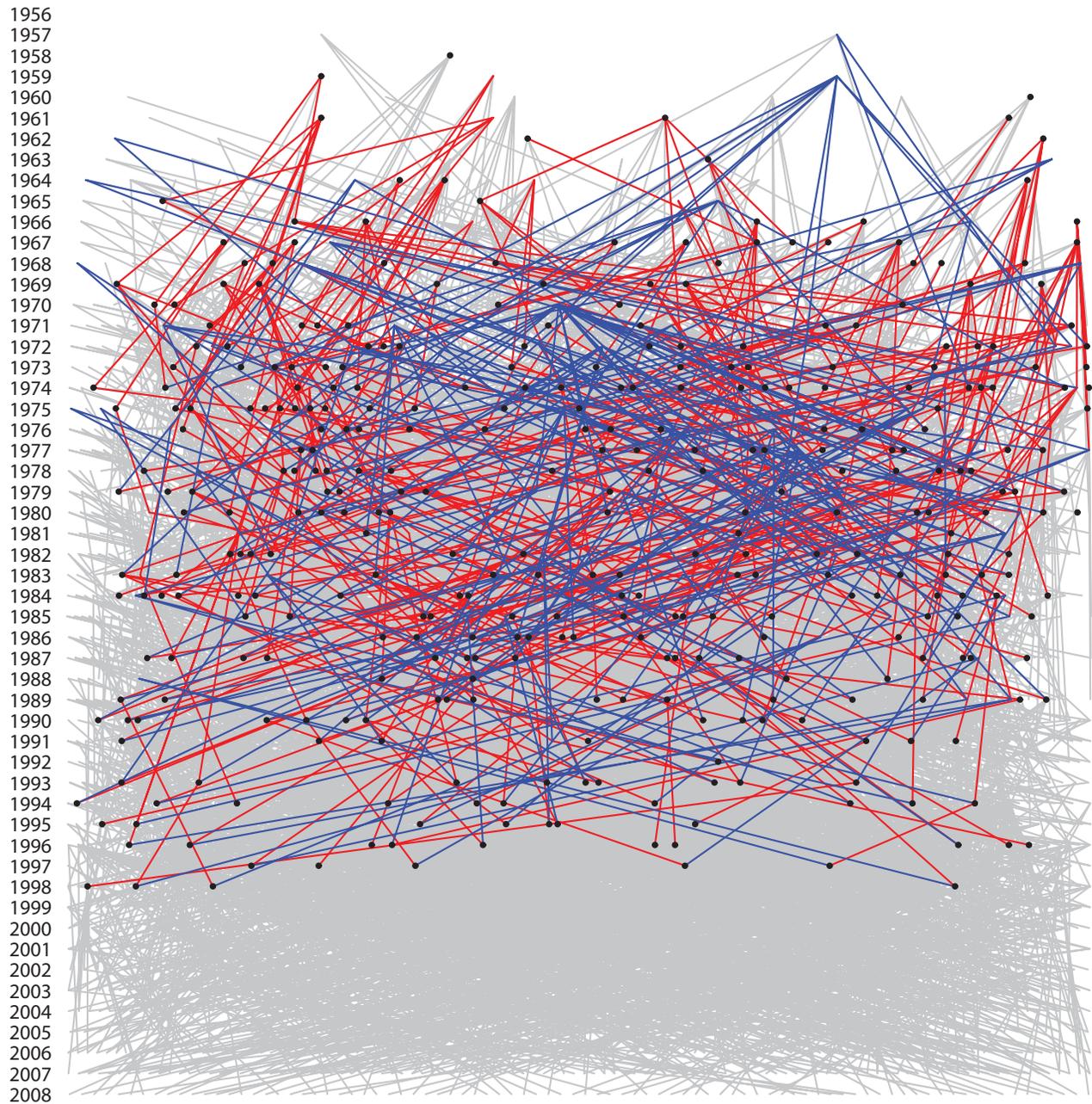


Figure 1: Structure of the portion of the Isle of Rum (North Block) red deer population’s pedigree that is informative with respect to the genetics of variation in female life-history traits. Numbers indicate cohort affinities, some of which are estimated on the basis of offspring ages and typical sex- and age-specific patterns of reproductive success. Red lines denote maternal links, and blue lines denote paternal links. Adult females born before 1999, known to be dead, and phenotyped for one or more life-history traits are denoted with black circles. Gray lines in the background indicate the density of pedigree links for the entire data set, including many links to offspring of those in the focal set, which define much of the life-history variation.

calculated the regions of 50% highest posterior density to provide a measure of uncertainty that is comparable to the standard errors that are traditionally reported. Correspondingly, we report modal values of estimated parameters as our measure of central tendency.

Inference of Selection and Constraint by Perturbation Analysis of Projection Matrices

Sensitivity Analysis. In order to derive the selective consequences of phenotypic and genetic covariances among life-history traits, we used the samples of the posterior distribution of the animal model to construct projection matrices. The matrices took the form

$$\mathbf{B} = \begin{bmatrix} 0 & m_1 s_1 & m_2 s_1 & m_3 s_1 & m_4 s_1 & m_5 s_1 & m_6 s_1 & \dots \\ s_{j,2} & 0 & 0 & 0 & 0 & 0 & 0 & \\ 0 & s_{j,3} & 0 & 0 & 0 & 0 & 0 & \\ 0 & 0 & s_{j,4} & 0 & 0 & 0 & 0 & \\ 0 & 0 & 0 & s_{j,5} & 0 & 0 & 0 & \\ 0 & 0 & 0 & 0 & s_6 & 0 & 0 & \\ 0 & 0 & 0 & 0 & 0 & s_7 & 0 & \\ 0 & 0 & 0 & 0 & 0 & 0 & s_8 & \\ \vdots & & & & & & & \ddots \end{bmatrix}. \quad (2)$$

We denote this matrix **B**, rather than the typical use of **A** (as in Caswell 2001), in order to avoid confusion with the additive genetic relatedness matrix, discussed above. Subscripts index age in years. *m* denotes annual fecundity, counted in female offspring, and *s* denotes annual survival. Entries for annual survival that are additionally subscripted by *j* denote survival during the juvenile period, for which we entered the observed population mean age-specific annual survival values. This matrix is constructed for a pre-breeding censused population, because we assess mortality in annual periods from May 1 to April 30, and breeding occurs primarily after May 1 each year. We construct **B** as a 25 × 25 matrix, which fully allows for the maximum longevity of deer (Catchpole et al. 2004). The modal values estimated projection matrix is given in the appendix.

We derived the nonzero components of **B** from estimates of the mean (μ) and the variances (σ_p^2) in the respective life-history traits, taken from the posterior distribution of the solution to the mixed model in equation (1):

$$m_i = \frac{\delta_{i, \mu_a, \sigma_{p,a}} (1 + e^{-\bar{f}})^{-1}}{2}, \text{ where}$$

$$\bar{f} = \frac{\mu_f}{\{1 + [(16 \times 3^{0.5})/2\pi]^2 \sigma_{p,f}^2\}^{1/2}}, \quad (3a)$$

$$s_i = (1 + e^{-\bar{o}})^{-1}, \text{ where}$$

$$\bar{o} = \frac{\mu_o}{\{1 + [(16 \times 3^{0.5})/2\pi]^2 \sigma_{p,o}^2\}^{1/2}}, \quad (3b)$$

$$s_i = \frac{1 - \delta_{i+1, \mu_b, \sigma_{p,l}}}{1 - \delta_{i, \mu_b, \sigma_{p,l}}}, \quad (3c)$$

where subscripts *l*, *a*, *f*, and *o* denote model parameters pertaining to adult longevity, age at primiparity, annual fecundity, and offspring survival rate, respectively; δ denotes cumulative normal functions; μ refers to model intercepts; and *P* refers to phenotypic variances conditional on location and cohort effects, obtained by summing the covariance matrices associated with the random effects. We calculated μ for each trait as a weighted average over the fixed effects in equation (1). Equation (3a) thus calculates the average fecundity of individuals aged *i* as the product of the proportion of individuals expected to have reached primiparity (the cumulative normal distribution $\delta_{i, \mu_a, \sigma_{p,a}}$) and the expected number of female offspring produced by an individual that has reached primiparity ($[(1 + e^{-\bar{f}})^{-1}]/2$), where the numerator is a logistic link function for total fecundity and division by 2 gives the expected number of female offspring. Equation (3b) calculates the survival rate of deer calves based on a logistic link function. Equation (3c) calculates the proportion of individuals entering each age class that are expected to survive to the next age class, based on cumulative normal functions describing the number of individuals surviving at the beginning and end of each age class. The second expressions in equations (3a) and (3b) obtain the latent scale population means of the binomial traits as a function of the intercepts and phenotypic variances, following Diggle et al. (2004); this is necessary because the latent scale means and intercepts are not equal when there is variance associated with random effects. Juvenile survival rates, $s_{j,b}$ were taken to be the observed juvenile annual survival rates in the population. As a consequence of using mean observed juvenile survival rates, we ignore some aspects of life-history variation and some uncertainties in our analyses below.

We calculated sensitivities of λ , the rate of population growth, to variation in each of the life-history traits by generating a perturbed matrix **B**₁ based on a small perturbation, *x*, of the value of the intercepts of the life-history traits (μ_b , μ_a , μ_f and μ_o). We used values of *x* on the order of 1% of the standard deviations of each intercept (we

also considered perturbations of 5%, which yielded results that were identical to the second decimal place in analyses we present). We obtained the proportional population growth rates associated with the original and perturbed projection matrices, λ and λ_1 , by calculating the leading eigenvalues of \mathbf{B} and \mathbf{B}_1 , respectively. We calculated sensitivities as

$$\nabla\lambda = \frac{\lambda_1 - \lambda}{x} \quad (4)$$

to obtain sensitivities for the traits modeled with Gaussian distributions—and the non-Gaussian traits on the latent scale—or by

$$\nabla\lambda = \frac{\lambda_1 - \lambda}{[1 + e^{-(\mu+x)}]^{-1} - (1 + e^{-\mu})^{-1}} \quad (5)$$

to obtain observed scale sensitivities of λ to the life-history traits with binomial distributions. We obtained λ for these calculations using standard eigenvalue-based methods (Caswell 2001), and so these numerically obtained sensitivities are calculated at the stable age distribution. These sensitivities are the partial derivatives of population growth rate with respect to population mean phenotype and, as such, are interpretable as selection gradients once divided by λ . This division by population mean fitness accomplishes the conversion to the relative fitness scale, which is required for quantitative evolutionary prediction (Lande 1982; Lande and Arnold 1983). In van Tienderen's (2000) terminology, these $\nabla\lambda$ are "integrated sensitivities." Typically, the term "sensitivity" refers to the dependence of λ on individual entries in the population projection matrix. The metrics described here are integrated because they describe the dependence of λ on life-history traits, which are defined such that they can influence multiple entries in the population projection matrix. Our use of integrated sensitivities in this way is consistent with Coulson et al.'s (2010) recommendation for the integration of evolutionary and demographic theory.

In order to evaluate the relative consequences of (co)variation among the life-history traits at the phenotypic and genetic levels, we calculated two more sets of integrated sensitivities according to both phenotypic and genetic covariances among the traits. These metrics integrate the sensitivity of λ over the effects of a given life-history trait on entries in the population projection matrix, as above, and, in addition, account for effects of correlated changes in other life-history traits, influencing λ through other entries in the projection matrix. We note here that our terminology is somewhat of mathematical convenience: life-history traits are of course determined by variation in (multiple) vital rates, not the reverse. Our terminology simply reflects and describes the methods we

implement. First, we obtained (absolute fitness-scale) selection coefficients that are equivalent in principle to selection differentials. We calculated phenotypic integrated sensitivities, $\nabla\lambda_p$, according to the formulas above, except we applied perturbations to \mathbf{B} according to both the direct effect of perturbing each trait and the correlated effects of such perturbations on the other traits. For example, for the integrated sensitivity of \mathbf{B} with respect to phenotypic change in the distribution of adult longevity, we applied the perturbation $\mathbf{P} \times [x, 0, 0, 0]^T$ to the vector of intercepts. Finally, we calculated genetic integrated sensitivities, $\nabla\lambda_G$. These are equivalent to the phenotypic integrated sensitivities but where the perturbations of \mathbf{B} are made based on \mathbf{G} rather than \mathbf{P} . These are not interpretable as selection coefficients but rather as components of the multivariate evolutionary response to selection. Genetic integrated sensitivities thus describe the change in a population's fitness as a consequence of directional selection acting only on one trait but accounting for correlated evolutionary responses in other traits. We characterized uncertainty in our estimates of all the integrated sensitivities by applying all of the above calculations to all samples of the posterior distribution of the mixed model in equation (1) and then calculating the range of the region of 50% posterior support for the sampling distribution of these parameters. This generates a range describing uncertainty that is interpretable as similar to the standard error.

Prediction of Evolutionary Trajectories. We predicted evolutionary change by combining sensitivity-based inference of the form of selection on female deer life histories with the genetic inference provided by the mixed model specified by equation (1). Specifically, we applied the Lande equation (Lande 1979, 1982; Caswell 2001) in forms that both do and do not include genetic covariances among traits, to obtain per-generation evolutionary predictions of changes in the life-history traits

$$\Delta\bar{z}|\mathbf{G} = \lambda^{-1}\mathbf{G}\nabla\lambda, \quad (6a)$$

$$\Delta\bar{z}|\mathbf{G} \times \mathbf{I} = \lambda^{-1}(\mathbf{G} \times \mathbf{I})\nabla\lambda, \quad (6b)$$

where $\Delta\bar{z}|\mathbf{G}$ and $\Delta\bar{z}|\mathbf{G} \times \mathbf{I}$ are vectors of evolutionary change, given the full genetic variance-covariance matrix and given only the genetic variances, respectively; \mathbf{G} is the additive genetic variance-covariance matrix; \mathbf{I} is an identity matrix; and $\nabla\lambda$ is a vector of sensitivities of λ to variation in the population mean life-history traits. For evolutionary prediction, the sensitivity of λ to calf survival rate, as calculated above, was divided by 2. This accounts for the fact that the covariance between maternal phenotype (where the variation is modeled) and offspring breeding values (where fitness is realized) is half the covariance between maternal phenotype and maternal breeding value (Kirk-

patrick and Lande 1989; Hadfield 2012). This division of the sensitivity of λ to calf survival rate by 2 was applied in equations (6) and thus affected the outcomes of calculations of constraint metrics, specifically, in equations (7)–(9). In order to incorporate all uncertainty in inferences of the genetics of life-history variation and of its selection (i.e., uncertainties of \mathbf{G} and $\nabla\lambda$), we made these predictions on the basis of each sample of the posterior distribution of the mixed model in equation (1). Finally, we repeated these analyses, assuming that the phenotypic variance-covariance matrix is representative of the genetic variance-covariance matrix, by substituting \mathbf{P} for \mathbf{G} in equations (6).

Quantification of Evolutionary Constraint. The methods described above allow estimation of the phenotypic means of the life-history traits after one generation of selection. We can use these to derive the projection matrix $\mathbf{B}_{\bar{z}+\Delta\bar{z}|\mathbf{G}}$ and, hence, also estimates of the population growth rate $\lambda_{\mathbf{B}_{\bar{z}+\Delta\bar{z}|\mathbf{G}}}$ in a population that has evolved for one generation. The difference between λ and $\lambda_{\mathbf{B}_{\bar{z}+\Delta\bar{z}|\mathbf{G}}}$ is the effect of evolutionary responses in the life-history traits on the population growth rate (or mean absolute fitness), that is, the per-generation expected rate of adaptation. Given this information, we can consider the extent to which the patterns of covariance among the traits generate evolutionary constraint by comparing the responses in the absence of covariances, as estimated in equation (6b). Specifically, we calculated Agrawal and Stinchcombe’s (2009) constraint metric

$$R_w = \frac{\Delta W_{\mathbf{G}}(\bar{\mathbf{z}})}{\Delta W_{\mathbf{G} \times \mathbf{I}}(\bar{\mathbf{z}})} = \frac{\lambda_{\mathbf{B}(\bar{\mathbf{z}}+\Delta\bar{\mathbf{z}}|\mathbf{G})} - \lambda_{\mathbf{B}(\bar{\mathbf{z}})}}{\lambda_{\mathbf{B}(\bar{\mathbf{z}}+\Delta\bar{\mathbf{z}}|\mathbf{G} \times \mathbf{I})} - \lambda_{\mathbf{B}(\bar{\mathbf{z}})}}, \quad (7)$$

where $\Delta W_{\mathbf{G}}(\bar{\mathbf{z}})$ is the change in mean absolute fitness due to the response to selection, based on the form of selection and the additive genetic variance-covariance matrix \mathbf{G} , and $\Delta W_{\mathbf{G} \times \mathbf{I}}(\bar{\mathbf{z}})$ is the corresponding increase in mean absolute fitness, assuming that all of the off-diagonal elements of \mathbf{G} are 0. Thus, R_w is the increase in mean absolute fitness associated with evolutionary change in mean phenotypes relative to the increase that would occur if life-history traits are genetically uncorrelated. We add the subscript W to this metric to indicate that this constraint metric is based on the change in fitness and to distinguish it from the metrics to which we extend the approach below. R_w will have a value of 1 when genetic correlations do not influence the rate of adaptation in a population. R_w will have a value <1 when genetic correlations constrain adaptive evolution and a value >1 when they facilitate adaptive evolution. The first expression for R_w is simply a repetition of Agrawal and Stinchcombe’s formulation, while the second is the formulation specific to the projection matrix–

based approaches we implement here. $\lambda_{\mathbf{B}}$ values are simply λ calculated for the three relevant projection matrices, that is, the matrix based on the observed data and the matrices based on a population that has evolved for one generation under \mathbf{G} or under just the diagonal elements of \mathbf{G} , that is, $\mathbf{G} \times \mathbf{I}$. Additionally, we calculated analogous R metrics for each trait to further characterize the influence of genetic correlations on phenotypic evolution. We denote these trait-based metrics $R_{\Delta\bar{z}}$ and obtain them as $R_{\Delta\bar{z}} = (\Delta\bar{z}|\mathbf{G})/(\Delta\bar{z}|\mathbf{G} \times \mathbf{I})$.

We further extended the R constraint metric to characterize other aspects of the influence of genetic covariances on a population’s response to selection. We calculated metrics that we denote R_e and R_r for constraint ratios based on mean-standardized evolvability and respondability (Hansen and Houle 2008). Following Hansen and Houle’s (2008) definition, evolvability is the length of the projection of the response vector $\Delta\bar{\mathbf{z}}$ on the selection vector β or, in the context of our analysis, on $\lambda^{-1}\nabla\lambda$. Evolvability thus quantifies the effect of genetic covariances on multivariate phenotypic evolution by the metric of “progress” relative to the optimum defined by the form of multivariate directional selection. Specifically, we calculated this metric as

$$R_e = \frac{(\lambda^{-1}\nabla\lambda\sigma_z)^T[\mathbf{G} \oslash (\sigma_z\sigma_z^T)](\lambda^{-1}\nabla\lambda\sigma_z)}{(\lambda^{-1}\nabla\lambda\sigma_z)^T[\mathbf{G} \oslash (\sigma_z\sigma_z^T) \times \mathbf{I}](\lambda^{-1}\nabla\lambda\sigma_z)}, \quad (8)$$

where \oslash denotes simple division, and this metric is thus based on variance-standardized evolvability. Standardization by the variance rather than by the mean leads to greater interpretability in these analyses because we model two of the traits as binomial. Similarly, we calculated the metric for respondability (Hansen and Houle 2008), or the total length of the $\Delta\bar{\mathbf{z}}$, which quantifies evolutionary change according to the metric of the total amount of phenotypic change, regardless of its direction. Accordingly,

$$R_r = \sqrt{\frac{(\lambda^{-1}\nabla\lambda\sigma_z)^T[\mathbf{G} \oslash (\sigma_z\sigma_z^T)](\lambda^{-1}\nabla\lambda\sigma_z)}{(\lambda^{-1}\nabla\lambda\sigma_z)^T[\mathbf{G} \oslash (\sigma_z\sigma_z^T) \times \mathbf{I}]^2(\lambda^{-1}\nabla\lambda\sigma_z)}}. \quad (9)$$

We present these expressions with minimal simplification, so that the interested reader may see how we obtained these expressions by substituting our equations (6) into Hansen and Houle’s (2008) equations (1) and (2) and dividing the results based on \mathbf{G} and $\mathbf{G} \times \mathbf{I}$, as in equation (7), as extensions of Agrawal and Stinchcombe’s (2009) approach. As in the calculation of sensitivities and prediction of evolutionary trajectories, we characterized the uncertainty in the R metrics over all of the uncertainty in their parameters by repeating these analyses for all samples of the posterior distribution of equation (1).

Results

Life-History Covariance

At the phenotypic level, all life-history traits in female red deer either covary positively (relative to the form of selection, i.e., early maturity, and high values of all other traits) or covary very little (table 1). At the genetic level, adult longevity is negatively related to all other life-history traits, and all other traits covary positively. The overall patterns of covariance in \mathbf{P} and \mathbf{G} contrast notably, as is evident from the visualization of the submatrices that include adult longevity (fig. 2). The 95% regions of highest posterior density of the distributions of all genetic correlations and covariances overlap 0 (values not shown but are approximately twice the reported standard error—like 50% confidence regions).

Sensitivity-Based Inference of Selection of Life Histories

Sensitivities of λ ($\nabla\lambda$) to variation in all four female life-history traits were positive (considering age at primiparity in terms of early maturity), which is an elementary result, given that each trait, in and of itself, must necessarily positively influence fitness. However, as a consequence of the negative genetic covariances of adult longevity with the other life-history traits, coordinated perturbation of the mean life histories according to \mathbf{G} resulted in a near 0 or slightly negative integrated sensitivity of λ to variation in fecundity. Sensitivities of λ to variation in each life-history trait in isolation are positive, both when the traits are considered on their own ($\nabla\lambda$) and based on phenotypic patterns of covariance among the traits ($\nabla\lambda_p$; fig. 3). As a consequence of the predominantly positive phenotypic

correlations among traits (table 1), the point estimates of the phenotypic integrated sensitivities ($\nabla\lambda_p$) are larger than the sensitivities of λ to variation in the life-history traits in isolation ($\nabla\lambda$). However, as a consequence of more negative genetic than phenotypic covariances among traits (table 1), point estimates of the integrated sensitivities based on \mathbf{G} ($\nabla\lambda_c$) are generally smaller than those based only on phenotypic patterns of variation, and the point estimate for fecundity is negative (fig. 3).

Prediction of Evolutionary Trajectories of Life-History Traits

The expected evolutionary trajectory of female life histories is for an increase in adult survival (2.80×10^{-2} years generation⁻¹; 50% credible interval [CI], 1.2×10^{-2} to 3.7×10^{-2}). There were much more modest or, indeed, essentially no predicted changes in the other traits: age at first reproduction (4.8×10^{-4} years generation⁻¹; 50% CI, -6.3×10^{-4} to 2.4×10^{-3}), fecundity (-3.6×10^{-5} logit (probability) generation⁻¹; 50% CI, -9.8×10^{-4} to 6.4×10^{-4}), and offspring survival (2.2×10^{-4} logit (probability) generation⁻¹; 50% CI, -1.4×10^{-3} to 2.2×10^{-3}). Given the very small expected change in fecundity, visualization in terms of the other three traits proves useful and provides a reasonably complete description of the expected phenotypic evolution (fig. 4). The expected evolutionary trajectories, based on the substitution of \mathbf{P} for \mathbf{G} , are very different. Under the assumption that \mathbf{P} and \mathbf{G} are proportional—that is, in a genetically uninformed analysis—an evolutionary trajectory with concurrent increases in the values of all life-history traits

Table 1: Posterior modes of phenotypic and additive genetic covariance matrices (variances on the diagonal and correlations above the diagonal) among female life-history traits

	Adult longevity	Early maturation	Annual fecundity	Offspring survival
Phenotypic:				
Adult longevity ^a	12.1 (11.615–12.982)	-.0449 (-.073–.053)	-2.158×10^{-3} (-.197–.083)	.361 (.304–.447)
Early maturation	.425 (.289–.486)	.447 (.438–.490)	.0418 (-.109–.169)	.0206 (-.100–.105)
Annual fecundity	-.286 (-.446–-.207)	.0733 (.038–.08)	.0428 (.030–.063)	.397 (.310–.600)
Offspring survival	.115 (-.059–.256)	.082 (.034–.096)	9.953×10^{-3} (-.013–.023)	.148 (.136–.228)
Additive genetic:				
Adult longevity	2.34 (1.449–3.419)	-.173 (-.468–.042)	-.767 (-.889–.452)	-.559 (-.757–.276)
Early maturation	-.0321 (-.174–.046)	.058 (.043–.089)	.583 (.287–.793)	.550 (.196–.667)
Annual fecundity	-.0227 (-.124–.013)	1.814×10^{-3} (-.003–.016)	3.135×10^{-3} (.001–.014)	.762 (.264–.874)
Offspring survival	-.167 (-.288–.016)	9.049×10^{-3} (-.003–.04)	5.624×10^{-3} (-.004–.021)	.0849 (.04–.105)

Note: Variances and covariances/correlations of traits that were formally treated as nonnormal (annual fecundity and offspring survival) are reported on the latent scale. Signs of covariances/correlations associated with early maturity are reported such that positive values indicate positive relationships among high-fitness states, that is, in terms of early maturation rather than age at primiparity. Values in parentheses are areas of 50% highest posterior density and are intended to be analogous to ± 1 SE.

^a Posterior modes of correlations are not always equal to the quotient of the covariance and the square root of the product of the associated variances. This is because the sampling error is not necessarily multivariate normal and can result in changes of sign for those covariances/correlations that are close to 0.

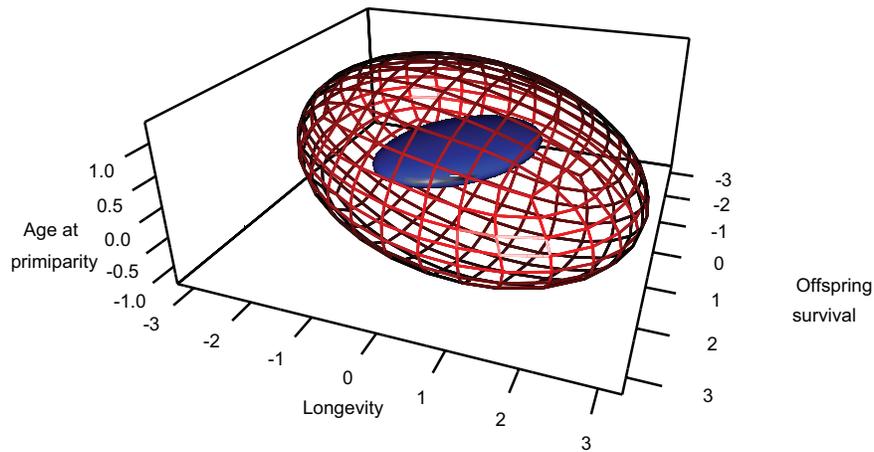


Figure 2: Visualization of patterns of phenotypic and genetic covariance among life-history traits in female red deer. The solid blue ellipsoid represents the region that contains 95% breeding values in each submatrix of \mathbf{G} , based on the posterior mode of \mathbf{G} . The ellipsoid depicted by the red lattice structure represents the region containing 95% of the space defined by \mathbf{P} , which was obtained by summing the additive genetic, cohort, and residual covariance matrices of an animal model of female life-history traits. Note that for practical purposes, the fourth analyzed trait, fecundity, was dropped for this representation. The estimated patterns of covariation of fecundity with the other three life-history traits are qualitatively similar at the phenotypic and genetic levels (table 1).

(advance of primiparity) appears much more possible (fig. 4).

Metrics of Evolutionary Constraint

$R_{\bar{w}}$ estimates the extent to which increases in the population growth rate—that is, the rate of adaptation—are dependent on genetic covariances among the life-history traits. The mode of the posterior distribution of $R_{\bar{w}}$ is 0.604 (tables 2, 3), indicating that genetic covariances among traits appear to reduce the expected rate of adaptation due to the evolution of female life-history traits by about 40%. There is, however, substantial uncertainty in this metric, and only 81.1% of the samples of the posterior distribution of $R_{\bar{w}}$ are below the null value of 1. $R_{\bar{w}}$ based on the substitution of \mathbf{P} for \mathbf{G} provides a very different inference of constraint, where correlations among traits appear to facilitate evolution, increasing the rate of change in fitness by about 33% ($R_{\bar{w}}|\mathbf{P} = 1.330$; table 3); 99.8% of the posterior distribution of $R_{\bar{w}}|\mathbf{P}$ are greater than the null value of 1.

Our metric based on Hansen and Houle’s (2008) evolvability, R_e , provided nearly identical inference of the nature of evolutionary constraints generated by covariances among life-history traits (tables 2, 3). This is not surprising, since $R_{\bar{w}}$ evaluates the question “Do covariances constrain or facilitate evolutionary increases in fitness?” while R_e evaluates the question “Do covariances constrain or

facilitate evolutionary change in the direction of maximally increasing fitness?” Clearly, while these two questions are not identical, the answers will, in practice, generally be very similar. However, our metric of respondability, R_r , did yield a qualitatively different result (tables 2, 3), since it appears that covariances among female life-history traits in the red deer system do not influence the total amount of evolution that is expected but rather act to deflect the evolutionary trajectory away from the optimum.

Genetic correlations reduced the rate of evolution of all four life-history traits and, in fact, are responsible for expected evolution of reduced annual fecundity (tables 2, 3). Note though that the absolute amount of expected evolution of annual fecundity is very small. However, in all cases, uncertainty in the degree of constraint of each trait is large, and the posterior distributions of the $R_{\Delta\bar{z}}$ statistics all substantially overlapped the null value of 1. Considering phenotypic rather than genetic covariances ($R_{\Delta\bar{z}|\mathbf{P}}$), more evolution of the life-history traits would be expected (table 3).

Discussion

We found empirical evidence that life-history evolution may be constrained by genetic correlations among traits, especially relative to the lack of trade-offs that are evident from overall phenotypic relationships among life-history

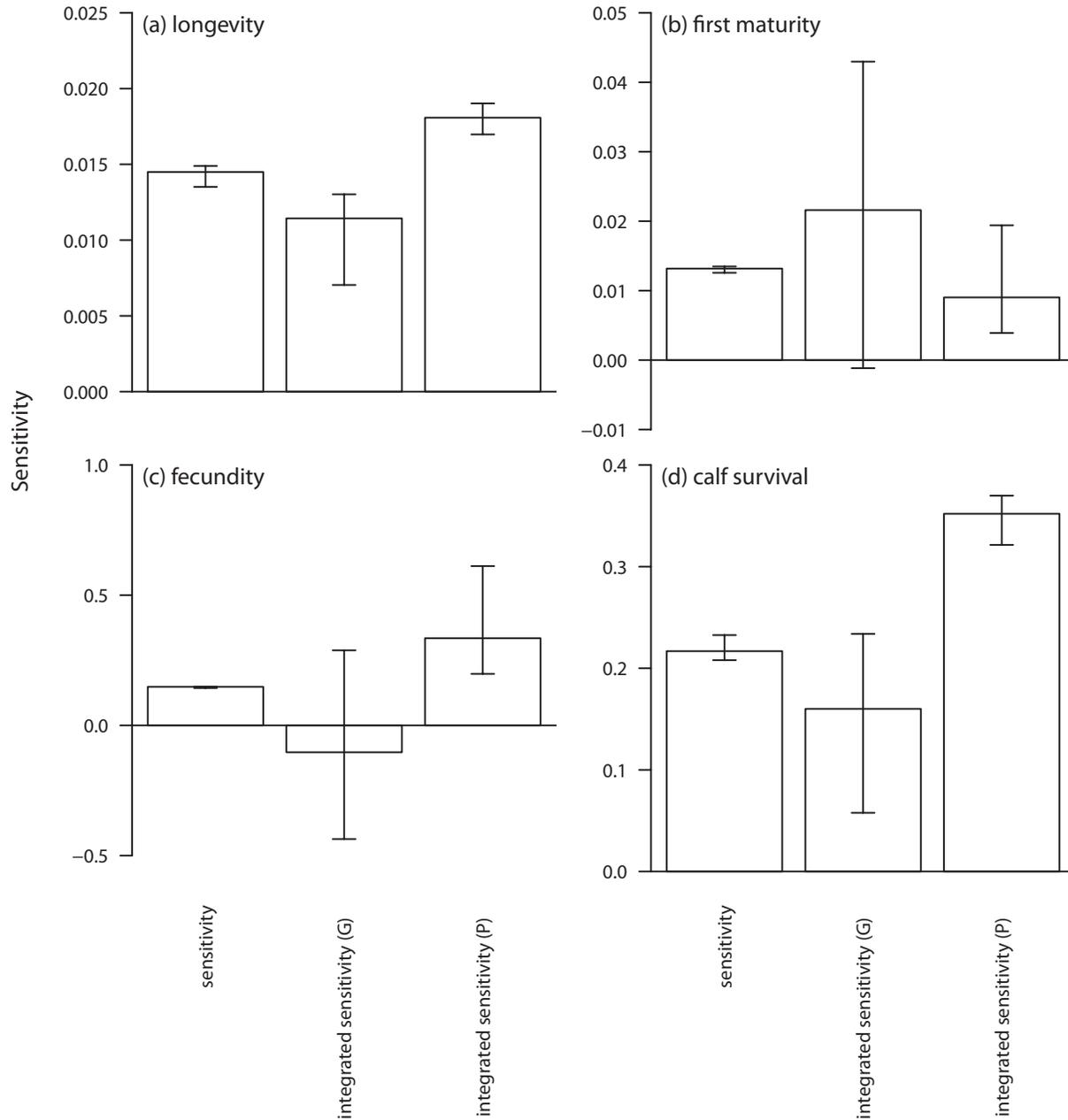


Figure 3: Sensitivity-based inference of selection of life-history traits in female red deer. Values are sensitivities, or the change in λ or mean absolute fitness, associated with a change in the mean value of the trait. Integrated sensitivities demonstrate the variation in λ associated with coordinated changes in all life-history traits resulting from variation of each focal trait in turn and associated correlated changes in the other traits, as defined by the genetic and phenotypic variance-covariance matrices, respectively. Error bars denote regions of 50% highest posterior density and are intended to be analogous to standard errors.

traits of female red deer. While all phenotypic relationships among life-history traits were positive (i.e., with respect to the high fitness state or small phenotypic values for age at first maturation) or near zero, negative relationships appear to exist in the genetic relationships among these

traits, especially with respect to correlations of longevity with the other traits (table 1). While no genetic correlations were statistically significantly different from zero in isolation, our consideration of the overall consequences of patterns of genetic variation nonetheless elucidated im-

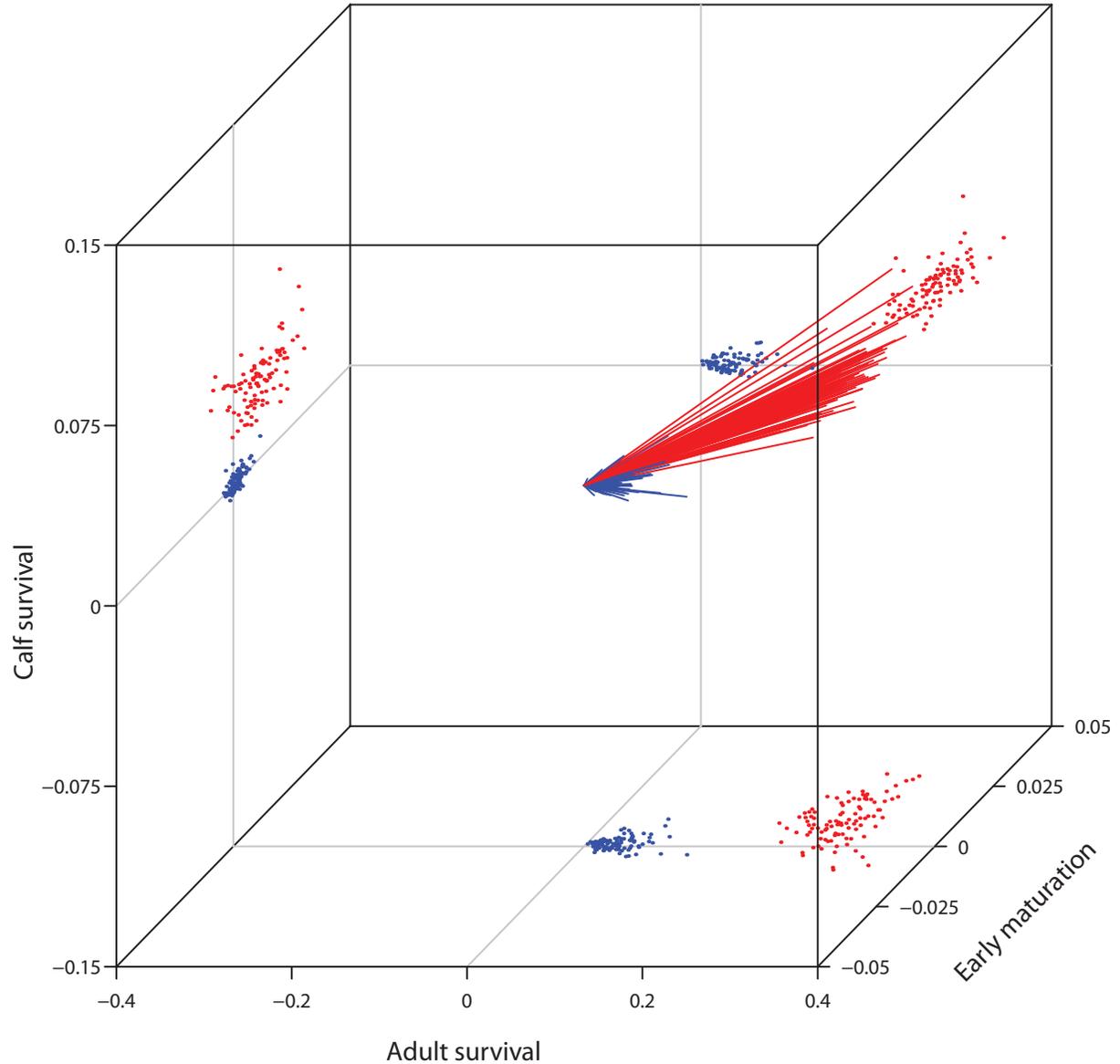


Figure 4: Vectors of predicted evolutionary change in adult survival, age at first maturity, and offspring survival in female red deer, from a starting point of 0 for each trait. Blue lines show a sample of 100 vectors of the posterior distribution of change based on the selection from model-based inference of the demographic effects of these three life-history traits and adult survival (not shown in order to make three-dimensional visualization possible) and the genetic covariances among the traits. Red lines show equivalent predicted vectors but calculated by substituting the phenotypic covariance matrix of the traits for the additive genetic covariance matrix, for comparison. Points projected on the walls of the plotting area show the ends of the vectors.

portant ways in which the genetic relationships among the life-history traits may constrain adaptive evolution of life histories. The potentially constraining genetic correlations that we did detect were largely between adult survival and reproductive traits (table 1; fig. 2), and this is one of the common axes of multivariate life-history variation that has been extensively characterized in experimental studies

(Stearns 1977). Furthermore, while adult longevity and offspring survival are significantly positively correlated at the phenotypic level, their genetic correlation is negative. Calves remain with their mothers throughout their first year of life (Clutton-Brock et al. 1982), and the effects of shared local conditions and weather (Coulson et al. 1997, 2003) are likely to generate substantial environmental co-

Table 2: Metrics of evolutionary constraint generated by covariances among traits, based on the rate of adaptation $R_{\overline{w}}$, evolvability R_e , and respondability R_r .

R metric	R	PS($R < 1$)	$R \mathbf{P}$	PS($R < R \mathbf{P}$)
$R_{\overline{w}}$.604	.811	1.329	.947
R_e	.634	.827	1.210	.940
R_r	1.017	.119	1.039	.376

Note: In tables 2 and 3, metrics subscripted by \mathbf{P} describe inferences that would be made if phenotypic relationships among traits were substituted for genetic relationships. PS describes the posterior support for each hypothesis; specifically, it describes the proportion of the posterior distribution of each constraint metric that is in qualitative agreement with the bracketed expression.

variance in mortality between females and their offspring, apparently sufficient to mask any negative genetic correlation.

The point estimate of the degree of genetic constraint on adaptive evolution, that is, $R_{\overline{w}}$, is substantial, indicating a 39.6% reduction in the rate of adaptation due to genetic correlations among life-history traits in this population. This finding is not strictly statistically significant. We note, however, that there is little tradition in evolutionary quantitative genetics of reporting statistical uncertainty in evolutionary predictions. Our approach to evaluating statistical uncertainty incorporates uncertainties throughout multiple steps of the analysis, which is also rare. The exception is that the pedigree is assumed to be known entirely without error, whereas paternal links in the pedigree have an average individual-level confidence of more than 98% (Walling et al. 2010). However, this level of pedigree error has been shown to be of little consequence for estimation of quantitative genetic parameters (Morrissey et al. 2007). Consequently, we wish to characterize this finding as quite suggestive of evolutionary constraint. Further progress on the question of whether negative genetic correlations constrain adaptive evolution to a substantial extent in nature will be attained if other such estimates and their associated uncertainties can be reported, regardless of statistical significance, especially if generated with similar statistical rigor.

The specific relationship between the direction of multivariate selection and the geometries of multivariate phenotypic and genetic relationships of the female red deer life-history traits in this population is relatively simple (statistical uncertainty aside). The direction of selection gradients of life-history traits is positive by definition (numerically negative for age at primiparity) since, in isolation, each must positively affect fitness. As a result of the predominantly positive or near zero phenotypic correlations among life-history traits (table 1, top), the first eigenvector of the phenotypic correlation matrix has positive loadings for all traits (loadings for longevity, early maturity,

annual fecundity, and offspring survival, evaluated at the posterior mode of the correlation matrix, are 0.23, 0.69, 0.51, and 0.46, respectively). Geometrically, this means that the major axis of \mathbf{P} is aligned with selection. In contrast, the first eigenvector of the genetic correlation matrix has a positive loading for longevity and negative loadings for the three reproductive life-history traits (loadings for longevity, early maturity, annual fecundity, and offspring survival are 0.54, -0.27 , -0.53 , and -0.59 , respectively). So whether assessed by spectral decomposition or by derivation of scalar metrics of constraint, as has been our main focus, it is clear that the multivariate genetic analysis is critical to assessing genetic constraint. As already extensively discussed and reviewed by Roff and Fairbairn (2007) and Walsh and Blows (2009), moving beyond bivariate genetic correlations is necessary to understand genetic constraint. For example, had we only considered some of the bivariate genetic covariances among life-history constraints, we might have been surprised to find a lack of evidence for trade-offs among different aspects of reproduction, such as between fecundity and offspring survival, that is, between offspring quantity and quality. We note that it is very difficult to interpret uncertainty in the spectral decomposition of covariance or correlation matrices by integrating over their posterior distributions, because different eigenvectors in different posterior samples can represent similar multivariate axes of variation.

We have demonstrated that \mathbf{G} and \mathbf{P} matrices for female deer life-history traits differ in their consequences for predictions of the course of adaptation. This contrasts with the common suggestion that phenotypic and genetic relationships among traits in wild organisms are similar (Cheverud 1988; but see Hadfield et al. 2006a; Kruuk et al. 2008). This is also in contrast to relations of many types of traits in laboratory or experimental settings, except some reports for covariances of life-history traits under laboratory conditions (Roff and Mousseau 1987). The emergent pattern in these various studies is that \mathbf{P} and \mathbf{G} matrices, or their component entries, are more inclined to differ for sets of traits with low heritabilities (but see

Table 3: Metrics of evolutionary constraint generated by covariances among traits, based on predicted rates of evolutionary change in each trait

Trait	$R_{\Delta\bar{z}}$	PS($R_{\Delta\bar{z}} < 1$)	$R_{\Delta\bar{z}} \mathbf{P}$	PS($R_{\Delta\bar{z}} < R_{\Delta\bar{z}} \mathbf{P}$)
Adult longevity	.832	.861	1.105	.950
Age at primiparity	.668	.482	.935	.454
Annual fecundity	-.696	.727	1.517	.761
Offspring survival	.152	.729	2.325	.944

Note: The R metrics are calculated based on predicted evolutionary change in individual female life-history traits. See table 2 note for additional information.

McGuigan and Blows 2007 for a report of lower dimensionality of \mathbf{G} than \mathbf{P} for wing shape in a laboratory study of *Drosophila melanogaster*). Relatively low heritability is a nearly universal finding for life-history traits (Mousseau and Roff 1987; Price and Schluter 1991; Merilä and Sheldon 1999, 2000; Kruuk et al. 2000; Coltman et al. 2005; Teplitsky et al. 2009). This is not surprising, because in the case of traits with high heritability, phenotypic patterns of trait covariation are in large part determined by genetic patterns. Our finding can thus be considered broadly in line with the finding in laboratory studies that genetic and phenotypic patterns of covariance in life-history traits can differ substantially, as well as the general theoretical expectation that life-history constraint is more likely to be manifested at the genetic level. This is, to our knowledge, the first description of this pattern in life histories of a free-ranging animal.

We chose to focus on female life-history traits in this population of red deer because the most data are available for these traits and their variation is known to have a detectable genetic basis (Foerster et al. 2007; C. A. Walling, M. B. Morrissey, K. Foerster, J. M. Pemberton, T. H. Clutton-Brock, and L. Kruuk, unpublished manuscript). Furthermore, the demographic approaches that we have implemented are typically implemented for the female component of a population (Caswell 2001); see Coulson et al. (2003) for application in this population to the analysis of selection of neonatal traits. Essentially, we make the assumption that equivalent traits exist in males, which are selectively and genetically equivalent to the traits in females. This assumption applies to three traits in our analysis: adult longevity, age at primiparity, and annual breeding success. It does not apply to calf survival rate, which is modeled as occurring in calves of both sexes but being determined by the phenotype of the mother. While traits that are likely to be selectively similar to the former three female traits exist for males—that is, male adult survival, male age at first (attempted or realized) reproduction, and male annual breeding success—it is not clear whether these traits are genetically equivalent in males and females. While there is evidence that the genetic correlation of female and male fitness is negative (Foerster et al. 2007), neither male fitness (Foerster et al. 2007) nor most of its component life-history traits are significantly heritable, although point estimates of genetic variances for some traits are appreciable (C. A. Walling, M. B. Morrissey, K. Foerster, J. M. Pemberton, T. H. Clutton-Brock, and L. Kruuk, unpublished manuscript). Intersexual genetic correlations among the three traits with analogues in males and females are difficult to characterize with precision; male/female adult longevity and age at first realized reproduction/age at primiparity have intersexual genetic correlations that do not differ significantly from either 0 or 1, and the genetic

correlation between annual breeding success/annual fecundity is slightly negative and significantly different from 1 (C. A. Walling, M. B. Morrissey, K. Foerster, J. M. Pemberton, T. H. Clutton-Brock, and L. Kruuk, unpublished manuscript). Thus, it is not clear whether male and female life-history traits are largely equivalent or are largely genetically uncoupled. Short of explicitly modeling selection and genetics of male traits and intersexual genetic correlations, an alternative analytical option would be to treat the female traits as sex limited. To do so, we would halve the sensitivities of λ to adult longevity, age at primiparity, and annual fecundity. This analysis leads to quantitatively stronger constraint; that is, $R_w = 0.47$, with similar statistical support to the main results that we present.

Considerations, Especially with Respect to Alternative Approaches

As a general approach to testing for genetic constraints on phenotypic evolution, our population projection model-based approach has a number of desirable features. Life-history variation is inherently probabilistic, and as such, analysis of life-history variation with generalized linear mixed models will generally be most appropriate. Additionally, the results of generalized models are more interpretable in the context of population biology than are results based on Gaussian approximation. Major difficulties in predicting the course of adaptive evolution—or, complementarily, testing for genetic constraints on such evolution—come from the technical challenges of studying the genetic basis of covariances between multivariate phenotype and fitness. A number of techniques are available to provide these inferences, but they suffer from the major drawbacks of high dimensionality and/or difficulties modeling the distribution of fitness. For example, high-dimensional analyses are necessitated by approaches advocated by Blows and Walsh (2008), in order to test for constraining multivariate genetic correlations, where fitness is modeled as a trait that potentially covaries genetically with an array of other traits, each of which is a candidate to be involved in multivariate correlations constraining evolution. These approaches can be implemented with techniques to reduce the dimensionality of the analyses (Blows et al. 2004; Mezey and Houle 2005; McGuigan and Blows 2007), but these techniques necessarily involve data loss. This data loss is explicit in approaches based on comparisons of the directions of the largest eigenvectors to the direction of selection and will also occur in applications based on rank reduction of \mathbf{G} when estimates of genetic covariances contain uncertainty. This data loss may be trivial in the large breeding experiments of typical model organisms in the lab but makes the techniques undesirable for in situ analysis of constraint, where failure

to demonstrate statistical equivalence or independence of two or more traits may result more from limited data than from the traits being effectively the same or different. In contrast, in our approach of obtaining low-dimensional metrics of constraint from the posterior distribution of \mathbf{G} , each potentially constraining component of \mathbf{G} can contribute to inference of constraint, or lack of constraint, in direct proportion to its statistical uncertainty.

A standing problem in the application of quantitative genetic approaches in empirical studies of the evolutionary dynamics of phenotypes is the fact that fitness variation can rarely be approximated by standard statistical distributions (Shaw et al. 2008). Our approach bypasses the issues pertaining to fitting models to the highly nonnormal distributions of lifetime fitness, at least insofar as our initial generalized animal model adequately accommodates the distributions of the modeled life-history traits. This differs somewhat from the aster model-based approach advocated by Shaw et al. (2008) in that fitness is not a modeled parameter but rather an emergent property of the projection matrix-based analysis of an estimated \mathbf{P} and \mathbf{G} matrix of life-history traits. Our approach therefore does not necessitate that total lifetime fitness be measured on any individual, though different components must be measured on relatives for it to be implemented in a genetically informed manner. When complete life-history data and thus lifetime fitness are known for large samples of individuals, aster models may make better use of the data for some purposes.

There are also a number of limitations of the modeling procedures that we have adopted. First, and as warned by Caswell (2001), though highly profitable, we must accept that merging explicit demographic models with quantitative genetic techniques will require that we make some major simplifications. In the context of our attempt to use projection models to obtain inference of the form of selection by transforming \mathbf{G} matrices, we have not modeled a number of processes of potential demographic importance. For example, ontogenetic variation in the reproductive biology of vertebrates is well documented, with respect to both reproductive senescence (Nussey et al. 2008, 2009) and variation in maternal performance earlier in life (Ozgul et al. 2009). However, we have not fitted models that can accommodate such patterns in either fecundity or offspring survival. An alternative treatment of the data, which might initially appear to have allowed us to include the potential for reproductive senescence to influence our results, would have been to decompose our overall binomial measures of fecundity and offspring survival into age-specific categorical traits. However, the residual variance of a categorical trait—that is, a binomial trait with one trial—is unobservable in a generalized linear mixed model and must therefore be set to an arbitrary

value to which the estimate of the additive genetic variance is not expected to be invariant. This same analytical limitation would similarly affect age-specific survival, were we to treat annual survival as a series of categorical traits, which superficially might be seen as an alternative to the rather inelegant manner in which we were forced to model the bimodal distribution of longevities in red deer.

Another decision that we made to simplify the analysis is a decision not to explicitly model maternal effects. Given the very high quality but rather modest quantity of life-history data available, we chose to avoid having to model another multivariate random effect. We note though that we have modeled the causative phenotypic variation influencing calf survival as a property of mothers. We have also modeled some common environment effects by including fixed effects of location within the study site. Finally, evolution of offspring survival rate almost certainly involves selection of variation attributable to mothers as well as to variation among individual offspring. We have simplified this greatly by attributing all selection of recruitment to age one as well as all genetic variation in recruitment to mothers. This approach provided some of the simplification necessary to merge our genetic and demographic analyses, but we note that an important future development would be to more explicitly model different aspects of selection and variation early in the life history.

Conclusions

Our explicit integration of a quantitative genetic model of the female red deer life-history variation with demographic models of the consequences of this variation allowed us to make several important advances in our understanding of this system, of the genetics of life-history constraint, and of analytical means by which these inferences can be pursued more generally. Most importantly, our results suggest that the multivariate genetic architecture of life-history traits constrains their evolution. This is in contrast to a general lack of evidence to date for genetic constraints arising from the quantitative genetic analysis of wild organisms. We note that this result is not statistically significant relative to traditional thresholds for hypothesis testing. However, this is a first attempt to evaluate uncertainty in such inferences. Furthermore, we showed that this constraint is a genetic phenomenon and is masked at the phenotypic level by patterns of environmental (co)variation among traits. Consequently, patterns of phenotypic variation and covariation are not substitutable for genetic patterns in this system. The particular approach we implemented, while fundamental to the theory of evolution in age-structured populations (Law 1991; Charlesworth 1994; Caswell 2001), has not before been implemented in an explicit quantitative genetic framework. The

Bayesian framework we used to combine the genetic and demographic methods allowed us to integrate uncertainty throughout the different levels of the analysis. This allowed us to make statistically defensible inferences of the degree of constraint, despite the unavoidable difficulties of determining what specific aspects of the multivariate genetic architecture (i.e., which correlations) are the source of constraint. The extensive life-history data available for female red deer on the Isle of Rum allowed us to capture the majority of the variation and covariation of traits that influences their fitness in our models. However, while this is highly desirable, we note that the approach is substantially more general and that the combined genetic and demographic analysis of life-history selection and constraint should provide both qualitatively and quantitatively improved inference of the evolutionary dynamics of model and nonmodel systems.

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APPENDIX

The Model-Based Projection Matrix for Red Deer

The posterior mode of the projection matrix is given in table A1. The stable age distribution (not shown) is such that fewer than 0.1% of female deer remain in the age class 25. The corresponding posterior mode of the rate of population increase at the stable age distribution is $\lambda = 0.97$ (95% credible interval, 0.94–0.99). We note that the population has actually increased during the interval studied, but nonetheless, all analyses are based on how λ varies

with life-history traits and are not dependent on the absolute value of λ . The most likely cause of the underestimation of λ is that the raw life-history data contain some missing data. Additionally, the parametric mixed model-based analysis imposes specific shapes on the trajectories of the life-history traits. Ultimately, the development of mark-recapture animal models with time-varying covariates will be useful.

Table A1: Posterior mode of the population projection matrix for red deer *Cervus elaphus*, as constructed from a mixed-model analysis of patterns of life-history variation and covariance

<i>i</i> (age class)	s_i (95% CI)	$m_i s_i$ (95% CI)
1	.76 (.76–.76)	.00 (.00–.00)
2	.93 (.93–.93)	.00 (.00–.00)
3	.98 (.97–.99)	.02 (.01–.02)
4	.97 (.96–.98)	.07 (.05–.08)
5	.96 (.94–.97)	.12 (.10–.14)
6	.94 (.92–.95)	.15 (.13–.17)
7	.92 (.90–.94)	.16 (.13–.18)
8	.90 (.87–.92)	.16 (.13–.18)
9	.87 (.84–.89)	.16 (.13–.18)
10	.84 (.80–.86)	.16 (.13–.18)
11	.80 (.77–.84)	.16 (.13–.18)
12	.78 (.73–.81)	.16 (.13–.18)
13	.74 (.69–.78)	.16 (.13–.18)
14	.71 (.65–.75)	.16 (.13–.18)
15	.67 (.62–.73)	.16 (.13–.18)
16	.64 (.58–.70)	.16 (.13–.18)
17	.60 (.55–.67)	.16 (.13–.18)
18	.58 (.51–.64)	.16 (.13–.18)
19	.55 (.48–.62)	.16 (.13–.18)
20	.52 (.45–.59)	.16 (.13–.18)
21	.49 (.42–.57)	.16 (.13–.18)
22	.46 (.39–.54)	.16 (.13–.18)
23	.43 (.37–.52)	.16 (.13–.18)
24	.41 (.34–.49)	.16 (.13–.18)
25	.39 (.32–.47)	.16 (.13–.18)

Note: The matrix is condensed such that each row represents the relevant entries of a column in the projection matrix. CI, credible interval.

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