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E-Article

Ontogenetic Patterns in Heritable Variation for Body Size: Using Random Regression Models in a Wild Ungulate Population

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abstract: Body size is an important determinant of fitness in many organisms. While size will typically change over the lifetime of an individual, heritable components of phenotypic variance may also show ontogenetic variation. We estimated genetic (additive and maternal) and environmental covariance structures for a size trait (June weight) measured over the first 5 years of life in a natural population of bighorn sheep *Ovis canadensis*. We also assessed the utility of random regression models for estimating these structures. Additive genetic variance was found for June weight, with heritability increasing over ontogeny because of declining environmental variance. This pattern, mirrored at the phenotypic level, likely reflects viability selection acting on early size traits. Maternal genetic effects were significant at ages 0 and 1, having important evolutionary implications for early weight, but declined with age being negligible by age 2. Strong positive genetic correlations between age-specific traits suggest that selection on June weight at any age will likely induce positively correlated responses across ontogeny. Random regression modeling yielded similar results to traditional methods. However, by facilitating more efficient data use where phenotypic sampling is incomplete, random regression should allow better estimation of genetic (co)variances for size and growth traits in natural populations.

Keywords: ontogeny, random regression, heritability, genetic correlation, *Ovis canadensis*, maternal effect.

Body size is considered to be a fitness-related trait in many taxa and is frequently a key determinant of both mortality and reproductive processes (Roff 2002). However, the covariance between size and fitness may differ among components of fitness defined at particular ages or ontogenetic stages (e.g., juvenile vs. adult viability) such that the selective pressure changes over the lifetime of an organism. While this has long been recognized, an evolutionary response to selection also depends on the presence of heritable variation. In this context, it is perhaps less obvious that genetic components of phenotypic variance may also change with an organism's age, particularly for those traits, including size, that are not static components of phenotype (Cheverud et al. 1983; Riska et al. 1984). Thus, to understand the evolution of body size and its ontogenetic trajectory (i.e., growth), it is therefore necessary to consider the levels of heritable phenotypic variance at different ages and also the nature of covariance between age-specific size traits. In this study, we examine this genetic (co)variance structure for body size in a natural population of bighorn sheep (*Ovis canadensis*).

The potential for a trait to evolve is usually expressed as the heritability (h^2), defined as the ratio of additive genetic variance to phenotypic variance (Falconer and Mackay 1996). Ontogenetic variation in heritability (and the components of phenotypic variance that determine it) might result from several hypothesized mechanisms. For example, variance compounding may occur with a trait expressed later in life "inheriting" variation from earlier ontogenetic stages, as well as being influenced by any new sources of variation. This compounding could occur in genetic variation, with allelic variants having cumulative effects over an individual's life (Atchley and Zhu 1997; Houle 1998), or in environmental variation (because additional sources of environmental variation may occur throughout life).

Components of phenotypic variance may also be reduced with age, for example, through viability selection (directional or stabilizing) or canalization. While differential mortality will certainly alter the distribution of a trait over time within a generation, many studies of growth

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in domestic animals have found reduced phenotypic variance for adult size traits in populations where juvenile mortality is negligible (e.g., Wilson and Réale 2005). Thus, declining variance is commonly attributed to "compensatory" or "targeted" growth (Monteiro and Falconer 1966), a form of canalization in which variable growth trajectories converge on a limited set of adult phenotypes. It is commonly assumed that compensatory growth occurs as a plastic response to environmental conditions (e.g., increased growth after a period of temporary starvation; Therkildsen et al. 2002) and will thus be reflected in reduced environmental variation for adult traits.

Estimating variance components and associated quantitative genetic parameters has historically been difficult in natural populations. However, recently it has become increasingly feasible through the use of molecular tools to infer pedigree structure (Garant and Kruuk 2005) and the application of restricted maximum likelihood approaches (notably the animal model; see Kruuk 2004 for a review). Nevertheless, determining ontogenetic patterns in (co)variance components is complicated by the need to assess phenotype at multiple ontogenetic points. In particular, while h^2 for age-specific size traits can be estimated from a single set of measurements made on individuals of different ages, genetic correlations (r_G) between these traits will also limit their potential to evolve independently of each other and thus constrain the evolution of growth trajectories (Kirkpatrick and Lofsvold 1992).

Determining the genetic covariance structure among ages requires knowledge of each individual's multivariate phenotype. Although it may sometimes be possible to infer past growth history from a single sampling event (e.g., through hard part analysis in fish; Wilson et al. 2003), repeated measurement of individuals over time (i.e., longitudinal data) will normally be required. In natural systems, these data requirements may best be met where individuals are relatively sessile, at least over the period of ontogeny examined (e.g., avian studies of fledgling growth; Badyaev and Martin 2000). In more dispersive organisms (or over longer time periods), recapture probabilities for individuals may be low, even with a high sampling effort. As a consequence, knowledge of multivariate phenotypes for individuals is likely to be incomplete, and estimating covariances between any age-specific size traits becomes limited by available sample sizes.

A partial solution to this problem may lie in the use of random regression models, which have been widely applied to genetic analyses of growth in animal breeding (e.g., Fischer et al. 2004*b*; Schaeffer 2004) but have received comparatively little attention in evolutionary studies to date (but see, e.g., Björkland 1993; Ragland and Carter 2004). Here we apply random regression methodology in an extension of the animal model. The animal model is a particular form of mixed model that utilizes pedigree information and includes an individual's additive genetic value for a trait as a random effect. The application of random regression in this situation is based on the premise that this individual genetic value will depend on when (in ontogeny) the trait is expressed and can itself be modeled as a function of time.

Growth trajectories can be viewed as "infinite dimensional traits" (Kirkpatrick et al. 1990) in that they represent an infinite number of size traits determined along the temporal axis of age. Size can therefore be treated as function of age such that, within a population, the variation in individual growth trajectories can be described using a covariance function (CF). For example, for any individual i, the additive genetic value (a_i, the deviation from the population mean phenotype caused by additive effects) at any observed age can be treated as a function of time, typically by using a regression on orthogonal polynomials (Kirkpatrick et al. 1990). By fitting this regression as a random effect, the coefficients associated with each term in the polynomial function are free to vary across individuals (allowing trajectories to differ), and it is the (co)variance associated with these coefficients that is estimated.

Polynomial functions of different orders can be used to model random effects with different functional relationships to time assumed. For example, a zero-order polynomial would describe a population of additive effect trajectories a_{iT} (for individual i at time T) that are constant with time but free to vary among individuals (such that some individuals are consistently genetically large or small across ontogeny). The random regression would then estimate a single parameter, this being the variance in the intercepts of individual trajectories a_{iT}. Added complexity can be incorporated using a first-order polynomial such that any trajectory a_{iT} is characterized by both an intercept and a slope (that may differ from 0). Thus, individual additive effects a_i change with time (assuming slope $\neq$ 0), and the variance for these effects (the additive genetic variance) is also expected to show variation with time. When using polynomials of order $\geq$1, the covariances between parameters (e.g., between slopes and intercepts) are also modeled and the full variance-covariance structure characterizes the CF. By fitting a separate CF for each source of phenotypic variation (e.g., additive, maternal genetic), the covariance attributed to that source between any two ontogenetic points can subsequently be expressed as a function of time (Meyer 1998).

Random regression models of growth can be seen as an intermediate between the more traditional approaches of either treating size as a single trait with repeated measures, under the simplifying and likely erroneous assumption that variance components are constant with age, or treating

size as a series of age-specific traits linked by a covariance structure (Meyer 1998). For example, for five age-specific size assessments, the additive genetic variance-covariance matrix contains 15 parameters to estimate (five variances and 10 covariances). However, if a_i can be adequately modeled as a first-order linear function of time using random regression, then this number of parameters is reduced to three (corresponding to the variances in intercept and slope and the covariance between). Reducing the number of parameters to estimate similarly reduces the data requirements for model parameterization. An additional advantage of the random regression methodology is that it allows interpolation between ages at which the phenotype was assessed (Kirkpatrick et al. 1990). This may be particularly useful in situations where not all individuals are assessed at the same set of ages, a likely feature of data sets from natural populations.

Here we determine the genetic (co)variance structure for size (body weight) over the first 5 years of life in a population of bighorn sheep (*O. canadensis*) resident on Ram Mountain, Alberta, Canada. This population has been the subject of long-term study, and it is known that weight traits are positively associated with multiple components of fitness including first-year survival (Festa-Bianchet et al. 1997) and reproductive success (Festa-Bianchet et al. 2000; Coltman et al. 2002). Size as a young adult is also positively correlated with female longevity (Bérubé et al. 1999). Previous studies have demonstrated heritable variation for weight traits in this population (Réale et al. 1999; Coltman 2005; Coltman et al. 2005). Variance components describing genetic and environmental effects on weight in domestic sheep are known to vary over ontogeny in domestic sheep (Wilson and Réale 2005), and prior work on this system has suggested an increasing heritability of weight with age (Réale et al. 1999). This trend was attributed, at least in part, to a decline in maternal effects on weight with increasing offspring age. However, this earlier study did not explicitly test for or estimate maternal effects.

Maternal effects occur when the phenotype of an individual is influenced by that of its mother, independently of the direct effect of inherited genes (Mousseau and Fox 1998). Maternal effects on weight traits have been extensively documented in domestic sheep, *Ovis aries* (e.g., Tosh and Kemp 1994), and may arise through both intrauterine effects and differential levels of postnatal provisioning among mothers. It has increasingly been recognized that maternal effects may themselves have a genetic basis and can thus represent an indirect genetic effect that will respond to selection (Wolf et al. 1998). Such maternal genetic effects have been demonstrated on birth weight in a feral population of domestic sheep (Wilson et al. 2005*a*) but have not otherwise been explicitly estimated in free-living vertebrate populations.

Thus, the objectives of this study are twofold. First, we estimate the phenotypic, genetic, and environmental (co)variance structures for age-specific weight traits in the Ram Mountain population of bighorn sheep. In this way, we determine the patterns of genetic (co)variance across ages that will shape the evolution of size and growth. We extend earlier work (Réale et al. 1999) by the use of a considerably larger data set and by using a pedigree comprised of both maternal and paternal links (the latter resolved using molecular pedigree analysis). Furthermore, in addition to modeling additive genetic (co)variance, we also test for maternal genetic effects as an additional source of heritable variation in weight, and we test the specific hypothesis that maternal effects on weight decline with offspring age. Second, we examine alternate methodological approaches to estimating these (co)variance structures. In particular, we present the first application of the random regression model for estimation of genetic (co)variances in a natural population and assess its utility by comparison to more conventional multiple-trait analyses.

Material and Methods

Data and Pedigree Structure

The study system is a population of bighorn sheep *Ovis canadensis* resident on Ram Mountain, Alberta, Canada (52°N, 115°W). Ram Mountain is separated from the main species range by approximately 30 km of forest, and this isolated population has been the subject of intensive individual-level monitoring since 1971. Background information on the system and more detailed description of data collection protocols are presented elsewhere (e.g., Jorgenson et al. 1993; Festa-Bianchet et al. 1996), so we limit the following description. In brief, between late May and early October of each year since 1971, sheep have been captured using a corral trap and weighed using a Detecto spring scale (± 0.125 kg). Multiple captures (between two and six per individual within a season) were used in a linear regression of weight on time to estimate individual rates of mass gain and hence allow estimation of a standardized June weight (JW; see Réale et al. 1999). For all yearlings and older sheep, JW is defined as the estimated live body mass on June 5. Because lambs were rarely caught before this date (and some animals were even born as late as the beginning of June), JW was defined as the estimated body mass on June 15 for lambs.

In order to allow estimation of maternal effects, any animals with unknown maternities were excluded from all analyses. Furthermore, we restricted our attention to JW estimated for sheep aged between 0 (lambs born that year)

and 5 years old. The reasons for this are twofold. First, average growth curves reveal that there is only limited phenotypic change after 5 years, with growth diminishing as sheep reach a comparatively stable adult weight (fig. 1). Second, although JW has been estimated for individuals as old as 18, the number of records available declines with age, and by restricting the portion of ontogeny analyzed, we therefore maximize sample sizes. In total, the data set therefore contains 590 phenotypically informative individuals born between 1972 and 2002. However, it should be noted that few individuals contribute a complete record such that at each age the number of known phenotypes is less (with n being equal to 411, 382, 335, 295, 247, and 214 for ages 0, 1, 2, 3, 4, and 5 years, respectively).

The pedigree structure of the Ram Mountain population has been reconstructed on the basis of maternal identities determined through observation of suckling behavior and paternities assigned using molecular pedigree reconstruction. Blood, hair, or ear tissue samples were collected from all individuals captured either between 1988 and 1993 or since 1997. All animals were genotyped at a panel of 32 microsatellite markers (full details of the microsatellite methodology are presented in Coltman et al. 2003, 2005). Paternity assignment was subsequently performed using the likelihood-based methodology implemented in CERVUS (Marshall et al. 1998), with those paternities assigned with greater than 95% confidence being accepted. Additional pedigree information was then recovered by identifying paternal half-sibships in the set of individuals for which no sire was assigned (see Coltman 2005 for full details) using COLONY software (Wang 2004). Members of paternal half-sibships identified in this way were assigned a common sire with unknown identity. The resultant pedigree structure contains 974 individuals (born between 1962 and 2002), with 717 maternal links and 402 paternal links (from 213 distinct dams and 90 distinct sires, respectively). While some of these animals were therefore born before the regular collection of phenotypic data, they are nevertheless included because the animal model uses all available pedigree information (i.e., including links to those individuals with unmeasured phenotype). Thus, their inclusion ensures that the relationship structure among measured individuals is correctly determined.

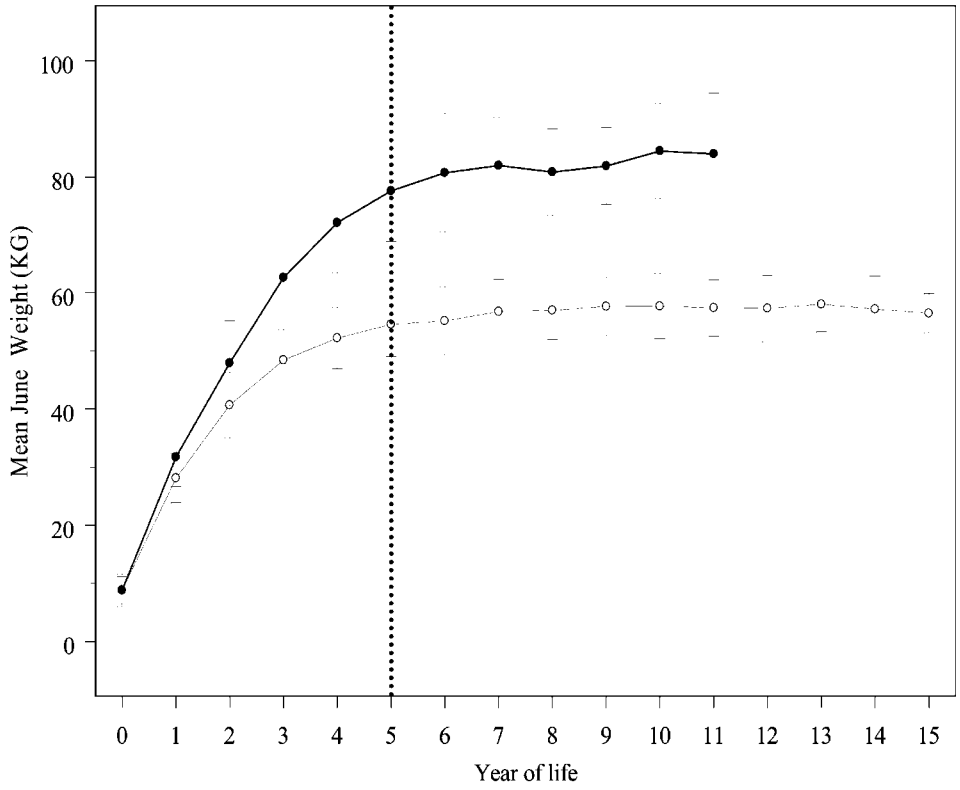

Figure 1: Mean June weight by age for male (*filled circles*) and female (*open circles*) bighorn sheep. Error bars indicate ± 1 SD around the mean. In this study, we examine phenotypic expression up to 5 years of age, thus focusing on those portions of the growth curves to the left of the dotted line.

Quantitative Genetic Analysis

Covariance components were estimated using three alternate approaches. First, we took the simplest approach of treating JW as a single trait with repeated measures; second, we treated JW as six age-specific traits JW_X (where X is the age in years taking values from 0 to 5), with each trait measured once; and third, we used a random regression methodology in which genetic and maternal influences on JW are modeled as polynomial functions of time. For each of the three approaches, variance components were estimated from animal models using restricted maximum likelihood implemented in the program ASReml (Gilmour et al. 2002). Variance was partitioned into additive genetic variance (σ^2_a), maternal genetic variance (σ^2_m), permanent environment variance (σ^2_{pe}; fitted in model 1 only), and residual variance (σ^2_r). Details of the variance component estimation for each approach are provided below. The phenotypic variance (σ^2_P) was then estimated as the sum of these variance components, and the direct heritability (h^2), permanent environment effect (pe^2), maternal genetic effect (m^2), and ratio of residual variance (r^2) were then calculated as the ratio of the relevant variance component to σ^2_P.

Under the second and third approaches, variance components and associated ratios were estimated separately for age-specific traits JW_X (X taking integer values from 0 to 5). Because weight increases with age as a consequence of growth, scale effects may prevent direct comparison of the magnitude of variance components across ages. Therefore, we also calculated phenotypic (CV_P), additive (CV_a), maternal genetic (CV_m), and residual (CV_r) coefficients of variation at each age X (where the coefficient of variation [CV] is found as 100 × (variance$^{0.5}$)/sample mean). These parameters are expected to be less sensitive to scale effects than are the unstandardized variance components (Houle 1992). Covariances between JW_X traits were also evaluated such that additive genetic, maternal genetic, and residual variance-covariance matrices (**G**, **M**, and **R**) were determined.

Finally, we performed a principal components analysis (PCA) of the variance-covariance matrices derived under the second and third approaches. This was done in order to summarize the major patterns of variation (both genetic and environmental) present for individual growth trajectories (following, e.g., Cheverud et al. 1983). If age-specific loadings associated with the most important major axes of variation (principal components) are consistent in sign, this is indicative of an integrated ontogeny (in which variation at one age affects all subsequent ages). In contrast, highly distinct and variable loadings, especially with changes in sign between ages, are associated with less constrained ontogenies and may result from genetic or environmental compensatory processes (Cheverud et al. 1983; Riska et al. 1984).

Single Trait with Repeated Measures. To account for the expected increase in an individual's mass over time, we included a fixed effect of age (as a six-level factor, with levels corresponding to years from 0 to 5) in the animal model. In this way, variation in size is modeled relative to the population average. Sex was also fitted (as a two-level factor), and because the difference in average JW between males and females varies with age (fig. 1), we also included an age by sex interaction term. Note that our decision to analyze both sexes together is supported by strong positive genetic correlations between weight in males and females (Coltman et al. 2005). Environmental conditions are known to affect body weight traits in this system (e.g., resource availability; Festa-Bianchet et al. 2004). To remove these sources of nongenetic variation as far as possible, birth year was included in the model (as a 29-level factor), as well as the interaction of this variable with age.

Random effects were then included in the animal model to partition the remaining variance for JW into additive, maternal genetic, and permanent environment components. Because each animal contributes multiple records, the latter effect is fitted to account for environmental effects that influence an individual's phenotype at all ages. In matrix notation, the model is therefore specified as

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_a\mathbf{a} + \mathbf{Z}_{pe}\mathbf{pe} + \mathbf{Z}_m\mathbf{m} + \mathbf{e}, \qquad \text{(model 1)}$$

where **y** is the vector of phenotypic observations for all individuals and **b** is the vector of fixed effects to be fitted. The random effects are related to individual phenotypic records with the corresponding incidence matrices $\mathbf{X}_1$, $\mathbf{Z}_a$, $\mathbf{Z}_{pe}$, and $\mathbf{Z}_m$.

The vector **a** contains the additive genetic effects for each individual (a_i) having mean of 0 and a variance of σ^2_a, the additive genetic variance. This is estimated from the variance-covariance matrix of additive genetic effects, which is equal to $\mathbf{A}\sigma^2_a$, where **A** is the additive numerator relationship matrix containing the individual elements $A_{ij} = 2\Theta_{ij}$ and Θ_{ij} is the coefficient of coancestry between individuals i and j obtained from the pedigree structure. Similarly, permanent environment variance (σ^2_{pe}) and maternal genetic variance (σ^2_m) were estimated by including **pe** and **m**, the vectors of permanent environment and maternal genetic effects, respectively. In all models, **e** was fitted as the vector of residual errors (corresponding to temporary environment effects) with variance of σ^2_e.

The variance-covariance matrix of maternal genetic effects is specified as $\mathbf{A}\sigma^2_m$ such that estimating σ^2_m uses the additive relationship matrix (i.e., the pedigree structure) in the same way as estimating σ^2_a. Permanent environment

effects and residual errors were assumed to be normal with means of 0 and variance-covariance matrices of $\mathbf{I}\sigma^2_{pe}$ and $\mathbf{I}\sigma^2_e$, where σ^2_{pe} and σ^2_e are the permanent environment variance and residual (temporary environmental) variances and $\mathbf{I}$ is an identity matrix with order equal to the number of maternal individuals or number of individual records as appropriate. We therefore assume that environmental errors are uncorrelated across individuals. The statistical significance of the maternal effect on JW was assessed using a likelihood ratio test (Meyer 1992) to compare model 1 with a simpler model in which the maternal genetic effect was omitted. The difference in log-likelihood scores, multiplied by -2, is distributed as χ^2 with one degree of freedom.

Multiple Traits with Single Measures. Separate analyses were then performed for each age-specific trait JW_X using the animal model, specified in matrix form as

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_a\mathbf{a} + \mathbf{Z}_m\mathbf{m} + \mathbf{e}, \qquad \text{(model 2)}$$

where $\mathbf{y}$ is the vector of phenotypic observations for all individuals at a given age X and all other terms are as described above. Note that because each animal contributes only a single record for each trait, a permanent environment effect is not fitted. The fixed effect structure for model 2 was also simplified accordingly to include only the main effects of sex and birth year. For each age X, variance components were estimated, and their associated ratios and coefficients of variation were calculated. The significance of the maternal genetic effect was assessed at each age (as described above). Bivariate models were used to estimate genetic and maternal covariances and the corresponding correlations (r_G and r_M, respectively) between each pair of age-specific traits. To reduce complexity and facilitate model convergence, the maternal genetic effect was included only for those age-specific traits where it was found to be statistically significant ($\alpha = 0.05$) in the univariate model.

Random Regression Method. JW records from all ages were then analyzed simultaneously using a random regression model in which the additive and maternal genetic effects on the phenotype of individual i are modeled by regressing on orthogonal polynomials of standardized time (T). Time is defined as the age of evaluating JW, measured in days since June 15 in the year of birth and standardized to the interval $-1 \le T \le 1$. Fixed effects were also included (identical to those described for model 1) such that, at the individual level, JW for individual i with mother j at time T is given as

$$JW_{iT} = (\text{age} + \text{sex} + \text{birth year} + \text{age : sex} + \text{age : birth year})_{iT} + f_1(a_i, n_1, T) + f_2(m_j, n_2, T) + e_{iT}, \qquad \text{(model 3)}$$

where $f_1(a_i, n_1, T)$ is the random regression function, on orthogonal polynomials of T with order n_1, of additive genetic values of individuals; and $f_2(m_j, n_1, T)$ is a random regression function with order n_2 of maternal genetic values of individuals on T; and e_{iT} is the residual error for individual i at time T. The latter term was modeled using a 6×6 unstructured matrix to permit a multivariate error structure, with e_i separately estimated at values of T corresponding to ages 0, 1, 2, 3, 4, and 5. An unstructured matrix was used to allow residual errors to be correlated across ages within individuals.

We first fitted model 3 with the random regression terms $f_1(a_i, n_1, T)$ and $f_2(m_j, n_2, T)$ omitted (model 3.0; table 1) such that all phenotypic variance is allocated to the error structure. The resultant 6×6 matrix is therefore a description of the phenotypic variance-covariance surface for JW for $-1 < T < 1$ (i.e., ages 0–5). Subsequently, using a forward model selection procedure, we fitted and compared a series of successively more complex models (models 3.1–3.9; see table 1). Models were compared using likelihood-ratio tests, with -2 times the difference in log-likelihood scores being distributed as χ^2 with one degree of freedom for each additional (co)variance component in the more complex model (Meyer 1992). Because JW is assessed at six time points (corresponding to ages 0–5), the order of each orthogonal polynomial function randomly regressed on T can, by definition, take values from 0 to 5. Here we fitted values of $n_1 = 0$ (a_i as constant

Table 1: Random regression models fitted showing the order of the polynomial function used to model additive (n_1) and maternal genetic (n_2) effects

	Polynomial order			
Model	n_1	n_2	Parameters	ln LK
3.0	NF	NF	21	−3,194.30
3.1	0	NF	22	−3,188.24
3.2	0	0	23	−3,182.61
3.3	1	NF	24	−3,183.26
3.4	1	0	25	−3,177.93
3.5	1	1	27	−3,177.40
3.6	2	NF	27	−3,178.68
3.7	2	0	28	−3,173.72
3.8	2	1	30	−3,172.87
3.9	2	2	33	−3,169.93

Note: Table also shows the number of (co)variance parameters estimated and log-likelihood score (ln LK) associated with each model. NF = effect not fitted.

with T), $n_1 = 1$ (a_i as a linear function of T), and $n_1 = 2$ (a_i as a quadratic function of T). Models were fitted omitting the maternal effect (i.e., the random regression of m_j on T) as well as with n_2 taking values 0, 1, and 2. Use of n_1 and n_2 equal to 5 should provide a "full fit" to the data (i.e., a fifth-order polynomial function can be fitted through any six values of a_{iT} for an individual), but in practice, model solutions largely failed to converge with values of n_1 and $n_2 > 2$ used (results not shown).

Following selection of the most appropriate model, the variance-covariance matrices of random regression parameters obtained for the additive genetic effect (matrix **Q** with dimensions $[n_1 + 1] \times [n_1 + 1]$) and maternal genetic effect (a matrix with dimensions $[n_2 + 1] \times [n_2 + 1]$) were used to obtain age-specific genetic parameters for comparison with those estimated under model 2. Specifically, the additive genetic variance-covariance matrix, **G**, for JW_X (at X from 0 to 5) was obtained as $\mathbf{G} = \mathbf{zQz}'$, where **z** is the vector of orthogonal polynomials evaluated at values of standardized time T that correspond to ages 0, 1, 2, 3, 4, and 5 (and $\mathbf{z}'$ is the transpose of **z**). An analogous procedure was used to obtain the maternal genetic variance-covariance matrix **M**, while the multivariate residual error structure derived from solving the random regression model represents the environmental variance-covariance matrix **R**. In this way, variance components were estimated and their associated ratios and coefficients of variation were calculated for each age, as were values of r_G and r_M between each pair of ages. Note that when using the random regression methodology, standard errors are estimated for the elements of **Q** (and the analogous variance-covariance matrix of random regression coefficients for the maternal effect), not for age-specific variance components. From these, we determined approximate standard errors for the elements of the derived **G** and **M** matrices according to the procedure recently presented by Fischer et al. (2004*a*).

Results

Model 1: Single Trait with Repeated Measures

Analysis as a single trait with repeated measures provided evidence of genetic variance for JW, with a significant heritability ($h^2 \pm$ SE of 0.126 ± 0.061; table 2). However, σ^2_m was low and accounted for <5% of the total variance ($m^2 \pm$ SE of 0.047 ± 0.033). Comparison of the full and reduced version of model 1 (with maternal effect omitted) revealed that the full model did not perform significantly better ($\chi^2_1 = 2.66$, $P = .103$). On this basis, there was little evidence for a significant maternal genetic effect on JW. Under the reduced model, heritability ($\pm$SE) was estimated as 0.164 ± 0.055 (table 2), while most of the phenotypic variance was attributable to permanent and temporary environmental effects ($pe^2 = 0.280 \pm 0.052$, $r^2 = 0.556 \pm 0.026$).

Table 2: Quantitative genetic parameters for June weight as a single trait with repeated measures (model 1)

	Maternal effect fitted	Maternal effect omitted
σ^2_P	21.25 (.948)	21.16 (.629)
σ^2_a	2.77 (1.324)	3.465 (1.213)
σ^2_m	.988 (.713)	NF
σ^2_{pe}	5.81 (1.135)	5.93 (1.111)
σ^2_r	11.76 (.473)	11.77 (.473)
h^2	.126 (.061)	.164 (.055)
m^2	.047 (.033)	NF
pe^2	.273 (.053)	.280 (.052)
r^2	.554 (.027)	.556 (.026)
ln LK	−3,452.61	−3,453.94

Note: Table shows phenotypic variance (σ^2_P) as well as the additive (σ^2_a), maternal genetic (σ^2_m), permanent environment (σ^2_{pe}), and residual (σ^2_r) components and their associated ratios to σ^2_P (denoted h^2, m^2, pe^2, and r^2, respectively). Parameters were estimated with and without a maternal genetic effect fitted, and log-likelihood scores associated with each model are shown. Standard errors are shown in parentheses.

Model 2: Multiple Traits with Single Measures

The multiple-trait analyses revealed variation in quantitative genetic parameters with age (table 3). Notably, there is little support for the presence of significant additive genetic variance for JW in lambs (JW_0), although heritable variation for this trait is present in the form of a large maternal genetic effect ($m^2 = 0.197 \pm 0.064$). This maternal effect (although reduced both in magnitude as measured by CV_M and as a proportion of phenotypic variance) persists for JW_1. Comparison of full and reduced models for each trait showed that these maternal genetic effects are significant for JW_0 ($\chi^2_1 = 12.66$, $P < .001$) and JW_1 ($\chi^2_1 = 4.31$, $P = .038$) but not for other traits (all $P > .10$). Parameters for JW_2–JW_5 were therefore estimated from reduced models with the maternal genetic effect omitted (table 3). While estimated heritability of JW is effectively 0 in the year of birth, it increased subsequently, being highest for yearlings (JW_1, $h^2 = 0.447 \pm 0.139$) and ranging from 0.251 to 0.113 in older animals (table 3). In all cases, associated standard errors are relatively large, and based on estimate standard errors, additive effects (as measured by both σ^2_a and h^2) were only significant at age 1. As a proportion of phenotypic variance, residual (environmental) effects were similar for all traits (r^2 ranging from 0.749 to 0.888) with the exception of JW_1 ($r^2 = 0.418 \pm 0.125$). The coefficients of variation show that,

Table 3: Age-specific estimates of quantitative genetic parameters for June weight

Methodology and trait	σ_a^2	σ_m^2	σ_r^2	h^2	m^2	r^2	CV_P	CV_A	CV_M	CV_R
Multiple-trait analyses (model 2):										
JW_0	8.6×10^{-5} (−)	.99 (.36)*	4.06 (.36)*	1.7×10^{-5} (−)	.197 (.064)*	.804 (.064)*	25.52	.10	11.31	22.88
JW_1	7.64 (2.70)*	2.31 (1.31)	7.14 (1.89)*	.447 (.139)*	.135 (.073)	.418 (.125)*	13.72	9.17	5.05	8.87
JW_2	5.97 (3.17)	NF	20.75 (3.08)*	.224 (.113)	NF	.777 (.113)*	11.58	5.47	NF	10.20
JW_3	7.79 (3.99)	NF	23.28 (3.80)*	.251 (.121)	NF	.749 (.121)*	10.15	5.08	NF	8.79
JW_4	4.00 (4.65)	NF	31.26 (5.14)*	.113 (.130)	NF	.887 (.130)*	9.78	3.27	NF	9.14
JW_5	6.65 (5.24)	NF	29.09 (5.40)*	.186 (.142)	NF	.814 (.142)*	9.17	3.95	NF	8.27
Random regression analysis (model 3.7):										
JW_0	.35 (1.78)	.88 (.35)*	3.80 (.54)*	.070	.175	.755	25.48	6.73	10.66	22.14
JW_1	3.61 (1.24)*	.88 (.35)*	11.01 (1.39)*	.233	.057	.710	13.06	6.30	3.12	11.01
JW_2	7.75 (2.10)*	.88 (.35)*	19.37 (2.56)*	.277	.031	.692	11.85	6.23	2.10	9.86
JW_3	10.50 (2.34)*	.88 (.35)*	21.49 (3.25)*	.319	.027	.654	10.44	5.90	1.71	8.44
JW_4	11.19 (3.55)*	.88 (.35)*	27.44 (4.12)*	.283	.022	.694	10.28	5.47	1.54	8.57
JW_5	10.77 (7.09)	.88 (.35)*	25.94 (4.87)*	.287	.023	.690	9.40	5.03	1.44	7.81

Note: Table shows heritability (h^2), maternal effect (m^2), and residual variance as a proportion of phenotypic variance (r^2), as well as coefficients of phenotypic, additive, maternal genetic, and residual variance (CV_P, CV_A, CV_M, and CV_R, respectively). For the multiple-trait analyses, maternal effects that were not significant at $P \leq .05$ were dropped and not fitted for that trait (denoted NF). Associated (approximate) standard errors are shown in parentheses where estimated. A minus sign in parentheses denotes that the standard error could not be estimated.

* Denotes significantly different from 0 at $P < .05$ based on estimated standard errors.

once scale effects are controlled for, there is a declining trend in levels of phenotypic variance from birth to age 5 (table 3). This decline is mirrored by additive, maternal, genetic, and, most notably, residual components of variance (although for CV_a this decline follows an initial increase from age 0 to 1).

Bivariate versions of model 2 indicate that age-specific JW traits generally show positive genetic covariance (table 4). The corresponding genetic correlations are strongest (close to 1) between those traits whose expression is separated by the least time (i.e., a period of 1 year) and tend to decrease with increasing time between traits. Negative values of r_G ($\pm$SE) were estimated between JW_0 and JW_4 ($r_G = -0.229 \pm 1.254$) and between JW_0 and JW_5 ($r_G = -0.756 \pm 1.302$) though neither is significantly <0. In many cases, standard errors are large (table 4) such that even when very strong genetic correlations were measured, r_G does not necessarily differ significantly from 0 (e.g., between JW_1 and JW_3; $r_G = +1.046 \pm 0.925$). Furthermore, in several cases, reliable estimates of standard errors could not be obtained. Given that the maternal genetic effect was significant only for the traits of JW_0 and JW_1, the maternal genetic correlation was estimated only between these traits and was found to be strongly positive and significantly >0 with r_M ($\pm$SE) $= 0.916 \pm 0.216$.

Model 3: Random Regression Method

On the basis of the use of log-likelihood tests to compare specific models, model 3.7 (table 1) was selected as the best model. Model 3.7 performed significantly better than models 3.0–3.6, while neither model 3.8 nor 3.9 provided a significant improvement (comparing models 3.7 and 3.8, $\chi_2^2 = 1.70$, $P = .427$; comparing models 3.7 and 3.9, $\chi_5^2 = 7.58$, $P = .181$). Thus, we estimated genetic parameters for JW from model 3.7 in which the additive effect was modeled using a second-order polynomial regression (i.e., $n_1 = 2$; a_i, the additive genetic effect on individual i, is a quadratic function of standardized time T), and the maternal genetic effect was modeled using a zero-order polynomial (i.e., $n_2 = 0$; m_j, the maternal genetic effect of mother j, is constant over T).

The phenotypic variance-covariance surface estimated from model 3.0 shows an increase in σ_P^2 with age and positive phenotypic covariance between ages that declines with time between measurements (fig. 2). By comparison, visual representation of the **G**, **M**, and **R** (residual) matrices for traits JW_0–JW_5 derived from model 3.7 (fig. 3) illustrates that the shape of the phenotypic variance-covariance surface is largely determined by the **R** matrix (fig. 3*c*). Note that the maternal genetic surface is constrained to be flat by the choice of model 3.7 (because modeling m_j as a constant with T for each mother j necessarily implies that σ_m^2 is also constant with time), while the additive genetic surface shows some increase in σ_a^2 with age.

Estimates of age-specific variance components, ratios, and coefficients of variation estimated for ages 0–5 from model 3.7 are largely similar to those from the multiple-trait analyses (model 2) and show similar patterns across ontogeny (table 3). Thus, for example, maternal effect is

Table 4: Estimates of genetic covariances (above diagonal) and correlations (below diagonal) between age-specific June weight traits (JW_X) based on bivariate model 2 and random regression model 3.7 analyses

Methodology and trait	JW_0	JW_1	JW_2	JW_3	JW_4	JW_5
Multiple-trait analyses (model 2):						
JW_0	…	.692 (.934)	1.395 (1.008)	1.632 (1.103)	−.216 (1.109)	−.756 (1.302)
JW_1	.643 (.964)	…	8.270 (2.392)*	8.126 (2.486)*	6.776 (2.690)*	4.799 (2.874)
JW_2	.698 (.153)	1.101 (.098)*	…	6.194 (−)	4.665 (2.958)	4.803 (3.163)
JW_3	.809 (.547)	1.046 (.925)	.990 (−)	…	7.839 (−)	8.858 (−)
JW_4	−.229 (1.254)	.925 (.266)*	.957 (.410)*	.990 (−)	…	4.893 (4.137)
JW_5	−.756 (1.302)	.543 (.248)*	.727 (.282)*	.990 (−)	.995 (.304)*	…
Random regression analysis (model 3.7):						
JW_0	…	1.055 (.907)	1.352 (.981)	1.214 (1.365)	.643 (2.627)	−.361 (4.976)
JW_1	.937	…	5.196 (1.625)*	5.738 (1.691)*	5.233 (2.284)*	3.682 (4.129)
JW_2	.819	.983	…	8.880 (2.161)*	8.593 (2.468)*	6.890 (3.960)
JW_3	.632	.933	.985	…	10.581 (2.789)*	9.144 (4.272)*
JW_4	.324	.824	.923	.976	…	10.434 (5.216)*
JW_5	−.186	.591	.754	.860	.950	…

Note: Associated (approximate) standard errors are shown in parentheses where estimated. A minus sign in parentheses denotes that the standard error could not be estimated.

* Denotes significantly different from 0 at $P < 0.05$ based on estimated standard errors

greatest for JW_0 (with m^2 estimated at 0.175 as compared with 0.197 under model 2) and declines with age, while r^2 (the residual variance as a proportion of σ_P^2) is again found to be relatively constant across time. Similarly, heritability is lowest in the year of birth ($h^2 = 0.07$), showing a subsequent increase. However, in contrast to model 2, h^2 is greatest for JW_3, and there is no evidence of the peak in h^2 at JW_1 estimated under model 2. With the exception of the yearling weight JW_1, age-specific heritability estimates were higher under model 3.7 than with the multiple-trait analyses. This result reflects higher estimates of the additive genetic variance obtained from random regression (except at age 1; table 3). Standard errors for variance components estimated under model 3.7 were generally smaller than the corresponding values determined from multiple-trait analyses (note that standard errors for additive genetic variances are actually approximate standard errors following Fischer et al. 2004*a*). As a consequence of these differences, estimates of σ_a^2 were significantly >0 at ages 1–4 using the random regression method (based on 95% confidence limits determined from approximate standard errors). Coefficients of variation again provide evidence of declining levels of maternal and residual variance with age. A slight decline is also seen in CV_a, which is highest for JW_0.

Genetic covariances and correlations between traits were qualitatively and quantitatively similar to those obtained from bivariate formulations of model 2 in most instances (table 4). Ninety-five-percent confidence intervals based on the standard errors of genetic covariance estimates show no significant differences between the methods.

Similarly, strong positive values of r_G were estimated under both methods, with the strength of the correlation declining as the time between trait assessment increases. The genetic correlation between JW_0 and JW_5 was again negative, though of lesser magnitude ($r_G = -0.186$). However, that between JW_0 and JW_4 was positive ($r_G = +0.324$). Comparison of standard errors associated with additive covariance components between age-specific traits shows that, where comparison is possible, values were smaller in most (but not all) cases than under model 2.

Principal Components Analyses

Because estimated σ_m^2 is only >0 at ages 0 and 1 using model 2 and was constant using model 3.7, we performed PCA only on the **G** and **R** matrices for age-specific traits. Results were qualitatively and quantitatively similar for analysis of matrices estimated under each model (fig. 4; table 5). For the **G** matrices of JW_X, the first PC explained 91% of the variance using the multiple-trait approach and 88% using the random regression method (fig. 4). Loadings were positive for all age-specific traits, indicating positive genetic covariation between all traits. The increasing loading coefficient across ages mirrors the increasing trend in σ_a^2 (fig. 3*a*). This variation described by PC1 therefore corresponds to additive variation for growth trajectories in which individual JWs tend to be either above or below the population mean at all ages. Nevertheless, a substantial proportion of additive variance for weight was also explained by PC2 (9% and 11% based on models 2 and 3.7, respectively; fig. 4). Loading coefficients for PC2 show a

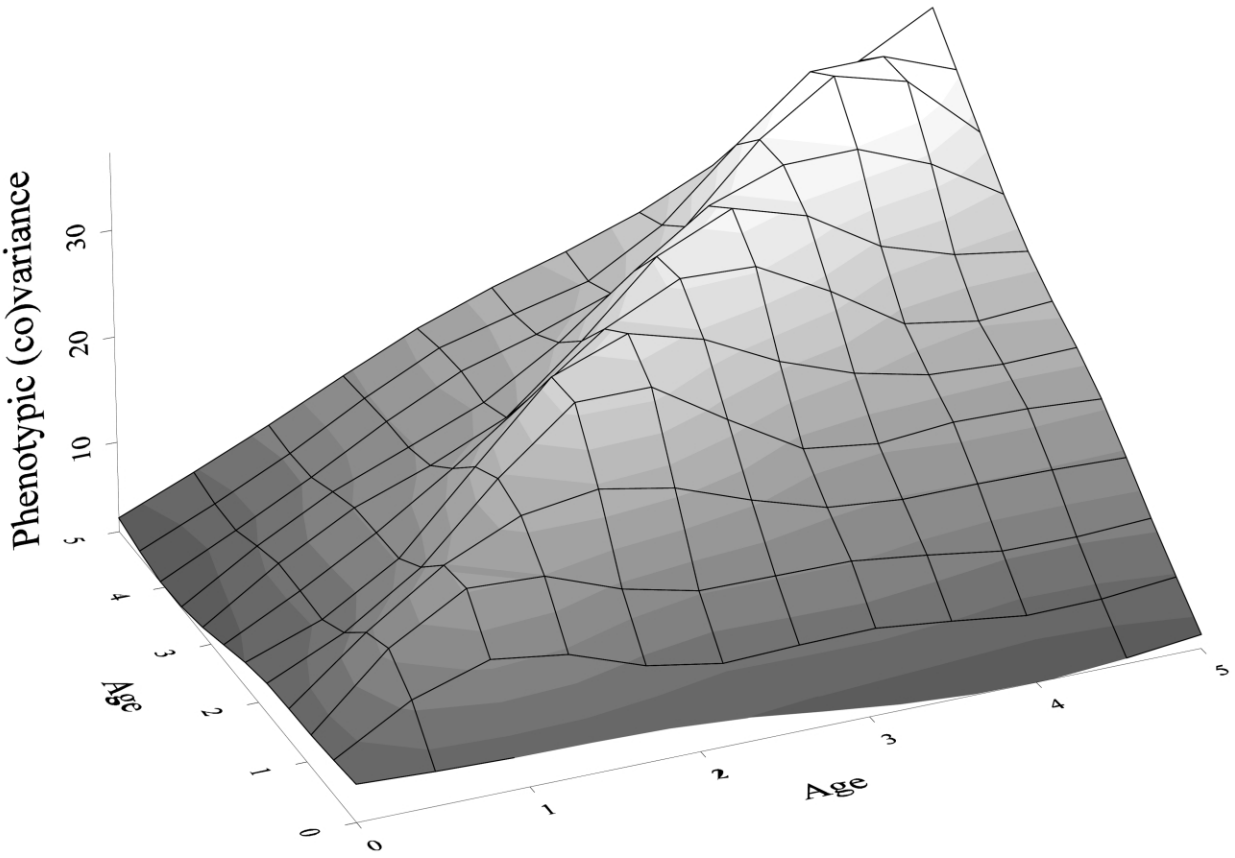

Figure 2: Phenotypic (co)variance surface for age-specific June weight in bighorn sheep (estimated using model 3.0).

switch from strong negative values at early ages to a strong positive value for weight at age 5. This pattern indicates the presence of negative additive genetic covariance between early-age and later JW traits, suggesting that some allelic variants cause individual growth trajectories to be below the average at early ages and above by age 5 (or vice versa). A generally similar pattern was seen for the **R** matrices, although the first two principal components explained less of the residual variation (58% and 16% for PC1 and PC2, respectively, under both models; fig. 4). Loadings for PC1 were consistently positive, while a sign switch was again seen between early and later ages in the coefficients for PC2. Loadings for subsequent principal components were variable in both magnitude and sign across ages (results not shown).

Discussion

Our analyses revealed that additive and maternal genetic effects contribute to heritable variation for weight traits in the Ram Mountain population of bighorn sheep. Ontogenetic changes in genetic and environmental variance components were such that estimates of heritability ranged from 0 to 0.447 across age-specific traits. Consequently, treating JW as a single trait and assuming constancy of genetic parameters across ontogeny are clearly inadequate for understanding the heritable basis of phenotypic variation. Ontogenetic patterns were similar using the two analytical approaches for estimating (co)variance structures, namely the multiple-trait and random regression methodologies. In the following discussion, we first address these patterns and their implications to the evolution of size and growth in this system. Subsequently, we discuss the alternate approaches used to estimate the covariance structures in order to consider the relative merits of the random regression method.

Evolution of Size and Growth

We found a general (though imperfect) trend of increasing h^2 for JW with age, concordant with the results of Réale et al. (1999). Additive genetic variation may increase with

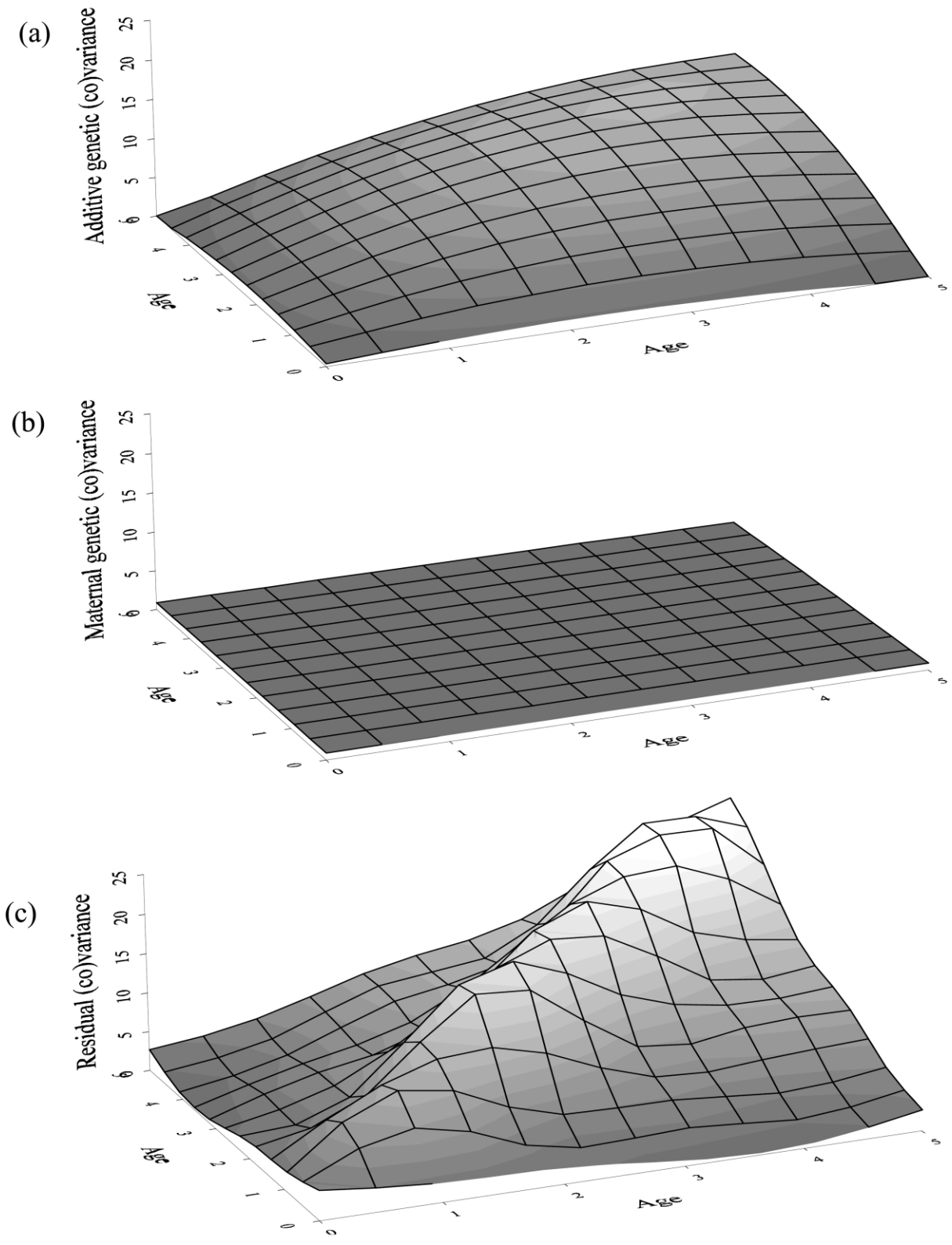

Figure 3: (Co)variance surfaces for age-specific June weight in bighorn sheep, showing additive genetic (*a*), maternal genetic (*b*), and residual (*c*) (co)variance surfaces estimated from random regression (model 3.7).

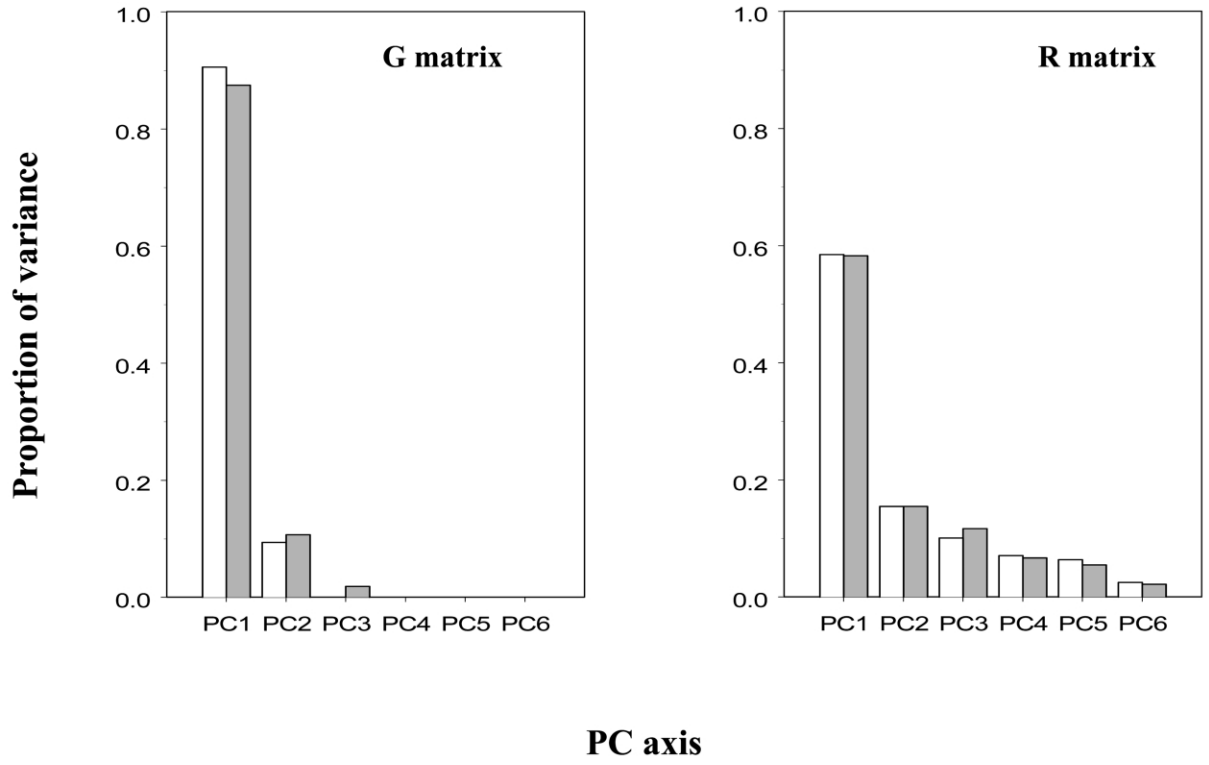

Figure 4: Percentage of total variance explained by principal components (ordered from largest to smallest) determined under model 2 (*open bars*) and model 3.7 (*gray bars*).

age through variance compounding (Houle 1998), with a trait expressed later in life inheriting variation from earlier stages as well as being influenced by any new episodes of gene expression (Atchley and Zhu 1997). However, here changes in h^2 are primarily attributable to declining levels of residual variation (measured by CV_r) as opposed to increasing additive variation (measured by CV_a). It was notable that heritability of JW_0 was low (effectively 0 under model 2), suggesting that this trait could not evolve in direct response to selection on it. On Ram Mountain, lamb weight is positively associated with survival (Festa-Bianchet et al. 1997), an effect that may largely drive the observed ontogenetic changes in variance components. Thus, the low heritability of JW_0 is consistent with expectations for fitness-related traits (Roff and Mousseau 1987; see also Coltman et al. 2005 for additional heritability estimates from this population). However, estimates of m^2 show the large influence of maternal genotype on early weight traits (e.g., 0.197 and 0.175 for JW_0 under models 2 and 3.7, respectively).

While comparable estimates are scarce, a large maternal genetic effect has also been estimated for birth weight in a feral population of Soay sheep from St. Kilda ($m^2 \pm$ SE of 0.119 ± 0.045; Wilson et al. 2005*a*). Where maternal genetic effects are present, the potential response to selection might better be expressed by total heritability (h^2_T), defined as the ratio of the sum ($\sigma^2_a + 0.5\sigma^2_m + 1.5\sigma_{am}$) to phenotypic variance (Willham 1972). Assuming that the direct maternal genetic covariance (σ_{am}) is 0, this yields estimates of total heritability that are considerably larger than the direct estimates (e.g., with estimated h^2_T for traits JW_0 and JW_1, respectively, of 0.157 and 0.261 under model 3.7). Note that expanding model 2 to explicitly test the assumption of $\sigma_{am} = 0$ indicates that this is an acceptable simplification (results not shown). Maternal genetic effects may thus facilitate a response of early weight traits to selection, although the influence of maternal genotype clearly declines with age as previously hypothesized (Réale et al.1999).

Failure to model maternal effects when they are actually present is known to cause upward bias in estimates of heritability (Clément et al. 2001), and this contributes to the finding that current estimates of h^2 for JW_0 and JW_1 are lower than previously reported (Réale et al. 1999). In fact, maternal effects may have both environmental and genetic aspects (e.g., Wilson et al. 2005*a*), while here we have fitted only the latter. Here maternal environment effects should be accounted for by the permanent envi-

Table 5: Loading coefficients for the first two principal components (PC1 and PC2) of **G** and **R** variance-covariance matrices for age-specific traits (JW_X)

Model and trait	**G** matrix		**R** matrix	
	PC1	PC2	PC1	PC2
Model 2:				
JW_0	+.013	−.425	+.091	−.050
JW_1	+.271	−.456	+.199	−.335
JW_2	+.410	−.429	+.380	−.667
JW_3	+.512	−.129	+.446	−.289
JW_4	+.521	+.145	+.547	+.505
JW_5	+.472	+.624	+.557	+.317
Model 3.7:				
JW_0	+.041	−.360	+.055	−.114
JW_1	+.262	−.452	+.184	−.409
JW_2	+.419	−.398	+.369	−.658
JW_3	+.507	−.193	+.456	−.254
JW_4	+.524	+.161	+.566	+.459
JW_5	+.471	+.666	+.546	+.330

Note: Coefficients are shown for matrices estimated using model 2 and model 3.7.

ronment effect under model 1 and by the unstructured multivariate residual error under model 3. Furthermore, additional analyses failed to support the presence of significant maternal environment variance for JW (results not shown), and this is therefore an unlikely source of bias for additive variances estimated herein.

The residual environmental variance represents the major component of phenotypic variance for all age-specific weight traits (with the single exception of JW_1 with analysis under model 2). Consequently, the ontogenetic decline in residual variance is also associated with a reduction in total phenotypic variation (as measured by the CV_P) across ontogeny. It should be noted that although σ^2_P for JW actually increases with age, this is likely a scale effect that results from dependence of the variance on the mean, which is itself increasing (Lynch and Walsh 1998). In general, reduced phenotypic variation over ontogeny may result from canalization or selection (stabilizing or directional) acting through size-selective mortality within each cohort. Heavier lambs have lower mortality in many ungulate systems (e.g., Kruuk et al. 1999; Wilson et al. 2005*b*), including this population (Festa-Bianchet et al. 1997), and the major decline in CV_R between ages 0 and 1 may therefore reflect strong viability selection for increased JW over this period. The major decline in CV_M also occurs over this period, although interestingly there is comparatively little change in CV_A. This may indicate that size-selective mortality of lambs is largely responsible for reducing variation associated with early environmental effects (and the maternal genotype). Nevertheless, this interpretation should be made cautiously because compensatory growth is common in ungulates populations (including domestic sheep; Wilson and Réale 2005) where juvenile mortality (and hence viability selection) is minimal. It is difficult to separate the potential effects of this form of phenotypic canalization from those of size-selective mortality in the current instance.

Strong genetic correlations were found between weights expressed at different ages, and selection acting on size at any age will therefore affect weight across ontogeny (i.e., the growth trajectory) as a whole. Genetic correlations were generally positive, consistent with other studies of mammalian growth (e.g., Cheverud et al. 1983; Al-Shorepy et al. 2002) such that any increase (or decrease) of weight at one age will likely result in correlated responses of the same direction at other ages. This conclusion is supported by the PCA in which consistently positive signs of the PC1 loading coefficients for the **G** matrix indicate a comparatively integrated ontogeny (Cheverud et al. 1983; Riska et al. 1984). Nevertheless, there was some evidence for antagonism of genetic effects on early versus late weight traits, a finding consistent with previous studies of domestic sheep (e.g., Fischer et al. 2004*b*). Negative genetic correlations were estimated between JW_0 and both JW_5 and JW_4 (multiple-trait approach only), and the second principal component of the **G** matrix, characterized by a sign change in the loading coefficients, explained about 10% of the additive variance. This may reflect the presence of genes having antagonistically pleiotropic effects such that, under an appropriate selection regime, a genetic response might allow contrasting directions of phenotypic change at different ages. However, size is believed to be positively associated with fitness throughout the lives of sheep in this population (Jorgenson et al. 1993; Festa-Bianchet et al. 1997, 1998; Coltman et al. 2002), and such a response is therefore unlikely.

Although we have focused primarily on the genetic aspects, similar conclusions can be made with respect to the environmental covariance structure of age-specific weight traits. Residual covariances between age-specific traits were positive, and PC1 loadings also indicated that environmental effects at one age tended to influence other (subsequent) ages in the same direction. However, PCA also suggests that some environmental influences have opposing effects on early and late weights (shown by the loading coefficients for PC2). This adds some support to the idea that compensatory growth occurs in this population, with some individuals able to attain large size by age 5 despite poor growth associated with environmental conditions experienced early in life.

Comparison of Methodologies

Analyses of age-specific traits yielded results and conclusions that were qualitatively and quantitatively similar using both the traditional multiple-trait approach and the random regression methodology. To the extent that correspondence with the more traditional methodology can be deemed a measure of success, the random regression methodology therefore performed well in this case. We found that while an inability to assess the precision of age-specific parameter estimates has been a drawback of random regression methods to date, here we found that (approximate) standard errors for estimated (co)variance components tended to be smaller using random regression (consistent with Fischer et al. 2004*a*). Although not performed in this case, it should also be possible to generate approximate standard errors for age-specific functions of variance components (e.g., h^2, r_G, CV) using a Taylor series expansion (Fischer et al. 2004*a*). Reduced standard errors can be attributed to the fact that sample sizes are less limiting than under the multiple-trait approach because phenotypic assessments at any age will contribute to parameter estimation at age *X*. This is a particularly useful feature of random regression for the analysis of incomplete data sets typical of natural populations. Here difficulties were encountered estimating standard errors under the multiple-trait approach, particularly for the genetic covariances. Because estimating genetic covariances (and correlations) with reasonable precision often requires large sample sizes (Lynch and Walsh 1998), random regression should offer a useful way forward for temporally related traits such as size.

However, the benefits of random regression do come at the cost of assuming a particular form of functional relationship between an individual's additive (or maternal) genetic effect and time. The accuracy of the parameter estimates will depend on the validity of this assumed relationship. A useful feature of the random regression framework is the ability to easily fit and compare models using different orders of orthogonal polynomials (e.g., Lewis and Brotherstone 2002). In theory, this allows model structures to range from a single-trait repeated-measures model (using zero-order polynomial functions and a univariate error structure) to a "full-fit model" in which the order of polynomial used is equal to the number of ages at which phenotype was assessed minus 1. A full-fit model becomes conceptually equivalent to the traditional multiple-trait approach but with all age-specific traits analyzed simultaneously (as opposed to the univariate and bivariate formulations of model 2 used herein).

In practice, parameterizing a full-fit random regression model will often be impossible in data sets typical of evolutionary studies (including this one). Here we used a forward model selection procedure (sequentially increasing the order of polynomials used) rather than the stepwise reduction from a full-fit model that is more typical of animal breeding studies where much larger data sets are the norm (e.g., Albuquerque and Meyer 2001). Confidence in our results is increased by the fact that fitting a third-order polynomial function for the additive genetic effect was not a significant improvement on model 3.7 (results not shown). Nevertheless, it is worth noting that without sufficient data to parameterize and compare more complex models, acceptance of a given model structure should be done cautiously.

It should also be noted that both approaches rely on a pedigree structure that likely contains errors. While pedigree errors will cause downward bias in estimated genetic variances, here paternities are assigned with high confidence such that error rates are expected to be low. Given the depth of pedigree, estimated levels of h^2, and the high confidence with which paternities and half-sibships are assigned, the limited simulation work to date would suggest that bias will be minimal in this system (Charmantier and Réale 2005).

Conclusions

In summary, we found evidence for important additive genetic effects on the covariance structure of weight traits expressed across ontogeny in the Ram Mountain population of bighorn sheep. Heritabilities for JW show ontogenetic variation, with a general increase over the first 5 years of life, a trend caused primarily by declining levels of environmental variance over the same period. This latter effect, and an associated decrease in total phenotypic variance, is consistent with compensatory growth occurring in this population. However, because the major reduction in variation occurs between ages 0 and 1, it seems likely that viability selection, known to operate on lamb weight, is a more important mechanism. We also found evidence for maternal genetic effects influencing weight in the year of birth and weight of yearlings. Thus, there is heritable variation for early weight despite the finding of low direct heritabilities. The genetic covariance structure suggests that age-specific JWs are integrated across ontogeny such that a response to selection at any age is likely to produce a positively correlated response across all ages. Finally, we found that analyses based on the use of random regression yielded results and conclusions similar to those of traditional methods of estimating genetic covariance structures. While further empirical testing is warranted, the use of random regression models seems likely to offer considerable advantages for estimating genetic covariance structures of size and growth traits in natural populations. In particular, random regression models should facilitate

more efficient use of data in situations where, because of incomplete sampling, the numbers of phenotypic assessments (and the ages at which they are made) are likely to vary among individuals.

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