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RESEARCH ARTICLE

Evolution of Adaptation and Mate Choice: Parental Relatedness Affects Expression of Phenotypic Variance in a Natural Population

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Abstract Mating between relatives generally results in reduced offspring viability or quality, suggesting that selection should favor behaviors that minimize inbreeding. However, in natural populations where searching is costly or variation among potential mates is limited, inbreeding is often common and may have important consequences for both offspring fitness and phenotypic variation. In particular, offspring morphological variation often increases with greater parental relatedness, yet the source of this variation, and thus its evolutionary significance, are poorly understood. One proposed explanation is that inbreeding influences a developing organism's sensitivity to its environment and therefore the increased phenotypic variation observed in inbred progeny is due to greater inputs from environmental and maternal sources. Alternatively, changes in phenotypic variation with inbreeding may be due to additive genetic effects alone when heterozygotes are phenotypically intermediate to homozygotes, or effects of inbreeding depression on condition, which can itself affect sensitivity to environmental variation. Here we examine the effect of parental relatedness (as inferred from neutral genetic markers) on heritable and nonheritable components of developmental variation in a wild bird population in which mate choice is often constrained, thereby leading to inbreeding. We found greater morphological variation and distinct contributions of variance components in offspring from highly related parents: inbred offspring tended to have greater environmental and lesser additive genetic variance compared to outbred progeny. The magnitude of this difference was greatest in late-maturing traits, implicating the accumulation of environmental variation as the underlying mechanism. Further, parental relatedness influenced the effect of an important maternal trait (egg size) on offspring development. These results support the hypothesis that inbreeding leads to greater sensitivity of development to environmental variation and maternal effects, suggesting that the evolutionary response to selection will depend strongly on mate choice patterns and population structure.

Keywords Heritability · Developmental stability · Genetic complementarity · Environmental variance · Maternal effects · Heterozygosity · Animal model

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Introduction

The evolution of adaptation depends on the production of new phenotypic and genetic variation, selection acting on this variation, and the inheritance of favored phenotypes (Fisher 1930). Whereas developmental variation is the proximate source of organismal diversity, it is a product of both heritable and nonheritable inputs which can each have distinct consequences for adaptive phenotypic change (e.g., West-Eberhard 2003). In particular, the degree to which phenotypes reflect additive genetic effects (i.e., narrow-sense heritability) strongly influences the rate and magnitude of the evolutionary response to selection (Roff 1997). However, the expression of additive genetic variation differs widely among populations (Hoffman and Parsons 1991), environments (Merilä 1997), and even age classes (Charmantier et al. 2006), implying that the efficacy of selection will vary accordingly. Thus, identifying the sources of developmental variance and the mechanisms determining their relative contributions is of fundamental importance for evolutionary studies.

Inbreeding leads to increased offspring homozygosity, which can have important consequences for not only fitness (Charlesworth and Charlesworth 1987; Keller and Waller 2002), but also patterns of genetic and developmental variation (Wright 1921; Lerner 1954). In particular, inbred offspring often exhibit greater morphological variation compared to outbred individuals (Yezerinac et al. 1992; David et al. 1997; Deng 1997; Réale and Roff 2003). And while theory predicts that natural selection should strongly favor behaviors that minimize inbreeding (Blouin and Blouin 1988; Pusey and Wolf 1996), matings between relatives might nevertheless occur when individual mate choice is constrained, suggesting that any effect of inbreeding on developmental variation should be particularly relevant in natural systems where mate sampling is costly (e.g., Johnsen et al. 2000; Foerster et al. 2003) and opportunities to breed are limited (e.g., Keller and Arcese 1998; Kruuk et al. 2002). However, because the increased phenotypic variation associated with inbreeding has rarely been partitioned into its heritable and nonheritable components (but see Whitlock and Fowler 1999; Kristensen et al. 2005), its source, and thus evolutionary significance, remain unclear.

One proposed explanation suggests that inbreeding might affect an organism's ability to buffer development from environmental extremes (e.g., variation in abiotic conditions, energetic demands, or resource availability; Lerner 1954). Specifically, inbred offspring are expected to have lesser diversity of allelic products important for development (e.g., metabolic enzymes) and consequently, a reduced range of environmental conditions in which stable growth can be maintained, thereby resulting in greater variation of offspring phenotypes compared to outbred progeny (reviewed in Mitton and Grant 1984). At the population level, such patterns should be reflected by reduced trait heritabilities with greater inbreeding, as additive genetic variance will be masked when developing phenotypes are strongly influenced by environmental variation (reviewed in Hoffman and Merilä 1999). Interestingly, in many taxa, environmental variation during development may also be due to maternal characters, such as allocation of resources to embryos and postnatal care (Kirkpatrick and Lande 1989; Mousseau and Fox 1998). Thus, effects of inbreeding on developmental stability might also lead to differential maternal effects, which are often implicated in rapid adaptive evolution (e.g., Badyaev 2005a; Rasanen and Kruuk 2007).

While the role of developmental stability has received the most attention (Mitton and Grant 1984; Hall 2005), the increased morphological variation observed with inbreeding might alternatively be explained by additive genetic effects alone when heterozygotes are phenotypically intermediate and thus less variable than homozygotes, which can be homozygous for different alleles (Chakraborty and Ryman 1983; Leary et al. 1983). Moreover, it is not known whether the effects of inbreeding result from action of loci directly involved in trait development, or via general effects of inbreeding depression on offspring condition, which often itself affects sensitivity to environmental variation (Merilä 1997; Hoffman and Merilä 1999). The few studies that have attempted to resolve such questions have largely relied on laboratory populations in which controlled breeding designs can be implemented (e.g., Whitlock and Fowler 1999). However, such methods involve severely inbred lines that are unlikely to be representative of mating patterns in natural populations.

As an alternative approach, comparing the effects of inbreeding on components of developmental variation in traits with distinct growth patterns might provide insight into the underlying mechanism. In particular, if greater morphological variation associated with inbreeding is due to increased sensitivity to environmental and maternal effects during development (i.e., Lerner 1954), then the greatest effect should be observed in late-maturing traits that experience a wider range of environmental and maternal inputs over a longer time period and thus accumulate greater variation from these sources compared to earlier-maturing traits ('environmental variance compounding hypothesis', Wilson et al. 2005; Wilson and Réale 2006). In contrast, if the effect of inbreeding on morphological variation is primarily due to additive effects (i.e., Chakraborty and Ryman 1983), no relationship is expected between developmental timing and the magnitude of inbreeding effect on phenotypic variance components.

Here we examine the effects of parental relatedness on morphological variation and expression of heritable and nonheritable variation in a wild population of house finches (*Carpodacus mexicanus*). In this study system, mate choice for unrelated mates is partially constrained by seasonal and social variation in availability of genetically unrelated partners (K.P. Oh and A.V. Badyaev, ms in preparation; Oh and Badyaev 2006), suggesting that effects of inbreeding on developmental variation should be particularly important. First, we show that offspring of closely related parents (as inferred from neutral genetic markers) have greater phenotypic variation compared to offspring from families with lesser parental relatedness. Second, we use a restricted maximum-likelihood 'animal model' to partition this variation into heritable and nonheritable components. Third, we investigate the mechanism by which parental relatedness might affect phenotypic variation by assessing traits' responses (i.e., change in variance components) to inbreeding in relation to growth patterns, as well as testing for general effects of inbreeding depression on offspring condition. Finally, we ask specifically how parental relatedness influences developmental sensitivity of offspring to

variation in egg size, a ubiquitous maternal effect in birds. We discuss the implications of these findings for understanding mechanisms of adaptive evolution across genetically structured populations.

Materials and Methods

Field Methods and Morphological Measurements

We studied house finches in a native population in southern Arizona during 2002–2006. Birds were trapped year-round and assigned unique combinations of one aluminum and three colored plastic bands which enabled the identification of individuals in the field. The social structure in the study population is characterized by small gregarious flocks in which the majority of courtship and pair formation occurs. In a concurrent study we have shown that constraints on mate sampling imposed by the size and composition of these flocks can lead to biased patterns of mate choice with respect to relatedness (K.P. Oh and A.V. Badyaev, ms in preparation). During the breeding season (February–July), house finches form monogamous pair affiliations and partners were identified through daily behavioral observations and nest attendance. We followed nesting attempts from nest initiation through fledging and obtained complete genetic data for 133 families (75 in 2005 and 58 in 2006). Nestlings were measured using Mitutoyo calipers to the nearest 0.01 mm (skeletal traits) and a digital balance to the nearest 0.1 g (body mass) every other day until the youngest nestling was age 7–8 days posthatching, the stage at which most traits have undergone rapid growth and have begun to approach asymptotic size (Badyaev et al. 2001a). Nests were then checked every 4–6 days until fledging at approximately day 16. For the growth analysis, we also used morphological data from older ages, as many nestlings used in this study were subsequently recaptured as free-flying juveniles and later as adults during regular population censuses (for details see Badyaev et al. 2005). We measured bill length from tip of maxilla to anterior edge of nares, bill depth in a vertical plane over both upper and lower mandibles at anterior edge of nares, bill width at anterior edge of nares, wing chord (unflattened), tarsus length, and body mass. Left and right measures were averaged for tarsus, and measurement error for all traits was estimated by calculating repeatabilities based on variance components derived from one-way ANOVA (Lessells and Boag 1987). Repeatabilities of measurements ranged from 0.753 ± 0.040 S.E. (bill width) to 0.953 ± 0.008 S.E. (tarsus length). As genotypes (see below) were analyzed outside of the breeding season, all interactions with the nests and nestling morphological measurements were conducted blind with respect to relatedness of parents.

Genetic Analyses

We collected blood samples (40–60 μl from adults, 5–15 μl from nestlings) from each individual by brachial venipuncture and genomic DNA was extracted using a commercial kit (Gentra Systems, Minneapolis, MN). DNA from blood samples were used to molecularly sex all nestlings by amplification of an intron of the CHD1 genes on the sex chromosome (Griffiths et al. 1996). All adults and offspring were genotyped at 15 highly polymorphic species-specific microsatellite loci (*Hofi53, HofiACAG07, HofiACAG25, Hofi16, Hofi29, Hofi10, Hofi70, HofiACAG01, Hofi39, Hofi19, Hofi35, Hofi69, HofiACAG15, Hofi07, Hofi26*). PCR was carried out using fluorescent-labeled primers (Applied Biosystems, USA) and product was analyzed by capillary electrophoresis in an ABI Prism 3730 DNA analyzer. Discrete microsatellite allele sizes were determined using Genotyper software (Applied Biosystems, USA).

The resulting genotypes were used to estimate relatedness (r) between mates, a value that expresses the genetic similarity between two individuals relative to random individuals within a reference population (reviewed in Blouin 2003). An important caveat to consider when interpreting values of r is that, while estimates provide a quantitative variable related to probability of identity by descent, the dependence on allele frequencies sampled from a reference population suggests that r-values are often not directly interpretable as genealogical relationships (e.g., full sibs, half sibs, cousins; Rousset 2002). In this study, pairwise relatedness estimates were calculated for all adults using MER software (Wang 2002) which implements a method of moments estimator that performs well across a range of population sizes and is particularly robust to biases due to sampling error (van de Casteele et al. 2001). Estimates of allele frequencies were calculated from the genotypes of breeding adults and associated standard errors were determined by bootstrapping over loci (30,000 iterations). Whereas r is a continuous variable, previous work has suggested that its relationship with phenotypic variation might be nonlinear, with the greatest effects observed at extreme values (David 1999). Thus, for the contrasts presented in this study, pairs were pooled (King 1985; David et al. 1997) into either 'typical' or 'high' relatedness groups by simulating all possible pairings between males and females present within the study site each year and generating a distribution of relatedness values, from which we calculated the median and associated 95% confidence interval. Actual pairings were then categorized as either 'high' (grater than upper 95% C.I. of the median) or 'typical' (less than upper 95% C.I. of the median). Following Keller and Arcese (1998), comparisons of mean and distribution of pairwise relatedness values between observed and expected mating patterns were performed using Mann–Whitney U and Kolmogorov–Smirnov two-sample tests respectively.

Recent studies have suggested that marker-derived measures of parental relatedness may be poor predictors of true inbreeding (Balloux et al. 2004; Pemberton 2004). Therefore, to further assess the validity of our classification, we calculated the inbreeding coefficient (F_{IS}) for the offspring of pairings between partners with 'high' versus 'typical' relatedness using FSTAT software (Goudet 2001).

Genotype data were also used to confirm paternity of attending parents at each nest (for details of paternity analysis see Oh and Badyaev 2006). The marker loci used yielded a combined exclusion probability of >0.999 (Jamieson and Taylor 1997). Within the families used in this study, we identified 31 offspring that were sired by extrapair males. We did not however, detect any discrepancies between offspring and maternal genotypes, confirming that intraspecific brood parasitism does not occur in this population.

Morphological Variation

To assess offspring morphological variation in relation to parental relatedness, we implemented the univariate form of the median-ratio Levene's test (Levene 1960) which constructs the variable:

$$y_i = |x_i - \mathrm{Md}(x_i)|/\mathrm{Md}(x_i)$$

where x_i is the trait value of the ith individual, and $\mathrm{Md}(x_i)$ is the sex-specific population median trait value. Compared to other measures of relative variation (e.g., coefficient of variation, F test) this metric is particularly robust to small samples sizes and departures from normality (Schultz 1985). Resultant values were compared between offspring from 'high' and 'typical' relatedness families using a general linear mixed model (PROC MIXED; SAS 9.1.3, SAS Institute, Cary, NC) with nest identity as a random effect and adjusting the denominator degrees of freedom using Satterthwaite's correction. To avoid pseudoreplication, only the first breeding attempt from each pairing was included in this analysis.

Estimation of Variance Components

Offspring phenotypic variance in each trait (V_P) was partitioned into causal components using an 'animal model' (Kruuk 2004) of the general form:

$$V_P = V_A + V_{CE} + V_R$$

where V_A represents additive genetic variance, V_{CE} is common environmental variance (e.g., common nest effects), and V_R is represents residual (i.e., unmeasured) effects. A total of 583 birds (104 sires, 99 dams, 380 offspring) with known familial relationships were used to construct a pedigree consisting of 133 full-sib and 16 half-sib groups. Data were obtained from multiple (two to five) broods for 26 of the unique pairings, along with single broods for the remaining 61 pairs. Variance component estimates and associated standard errors were calculated separately for 'typical' and 'high' relatedness families using restricted maximum-likelihood implemented in ASReml Release 2.0 software (Gilmour et al. 2006). First, we used PROC GLM to identify relevant fixed effects. In order to enable comparison of variance components among traits, any fixed effect term that was significant in at least one trait was included in models for all other traits, which resulted in the inclusion of the following terms: year, nestling sex, nestling age, presence of nest ectoparasites (Badyaev et al. 2006), and brood size, along with nest identity (nested within female identity) as a random effect (V_{CE}). We also considered a second model in which nest identity was replaced with two terms, maternal identity and paternal identity, to estimate variance due to parental effects. However, this second model resulted in comparatively large estimates of residual variance (V_R) and lower overall model log-likelihood values. Moreover, when nest identity was added back into the model, estimates of parental variance were generally reduced overall and most did not differ significantly from zero, suggesting that maternal and paternal variances were being overwhelmed by the nest environment component (Kruuk 2004; McCleery et al. 2004). Thus, here we only report variance component estimates generated from the first model, in which variance due to nest identity necessarily includes both common environmental effects and nongenetic parental effects, as well as any nonadditive genetic effects (e.g., dominance and epistasis), though the latter are generally expected to be low for morphological traits (reviewed in Roff 1998; Merilä and Sheldon 1999). To avoid negative variance components, negative values were fixed at zero. Significance of each random effect was tested using a likelihood ratio test in which two times the difference in log-likelihood scores between the full and reduced models approximates a χ^2distribution with one degree of freedom (Kruuk 2004). Similarly, significance of differences in variance components between 'typical' and 'high' relatedness families was tested by combining all individuals into one model with 'relatedness category' (REL) as an interaction term for each random effect (e.g., REL × Nest ID). A likelihood ratio test was then performed using the difference between log-likelihood of this model, and a reduced model in which a single random effect of interest was constrained to be the same across all individuals (Shaw 1991).

Offspring Relative Condition

Nestling condition was calculated as size-corrected body mass, a metric thought to reflect lipid reserves (Merilä et al.

2001), survival (Brown and Roth 2004) and offspring quality (Altwegg et al. 2000) in wild birds. Relative condition was calculated as the residuals from a least squares regression of mass on tarsus length for each sex separately and values were standardized to a mean of zero and standard deviation of one. While this approach has recently drawn criticism (e.g., Green 2001), its validity has been empirically reconfirmed as a general measure of body condition across several vertebrate taxa (Ardia 2005; Schulte-Hostedde et al. 2005) and especially appropriate for developing birds prior to onset of flying. To control for possible seasonal differences, only the first breeding attempt from each pair was included in this analysis.

Trait Developmental Timing

We used nonparametric locally weighted least squares regression (LOWESS, Cleveland and Devlin 1988) to fit growth curves to longitudinal data for each trait, separately for males and females. This approach is well-suited for unbalanced growth data, and unlike parametric methods, requires no assumption of the particular form of the curve and is thus appropriate for comparisons among traits (Moses et al. 1992). For the purposes of this study, we were primarily interested in assessing the sequence of trait maturation, which can be approximated for each trait as the period of rapid growth that occurs around the point of inflection (T_{max}) on a sigmoid curve (Ricklefs 1973; Atchley 1984). This was determined from pseudo-velocity curves that were constructed by dividing the differences in sequential predicted trait values by the differences in ages (Coellho 1985), and fitting a curve to the resulting points using LOWESS smoothing (Badyaev et al. 2001b; Setchell et al. 2001). As resulting values were not normally distributed, concordance between timing of trait maturation and effect of inbreeding (% of V_P in 'high' relatedness group − % of V_P in 'typical' relatedness group) observed for additive (V_A) and common environmental (V_{CE}) variance was tested using Kendall's τ nonparametric measure of association.

Maternal Effects

For a subset of nestlings in each group ('typical' relatedness: $N = 50$, 'high' relatedness: $N = 76$) we tested the effects on trait size of maternal allocation to eggs. In order to avoid potentially confounding effects of post-hatching environment (e.g., sibling competition; reviewed in Williams 1994), we used trait values from early in development (day 2). Female house finches in this population lay one egg per day between 0500 and 0900. During this period, we visited nests daily and photographed freshly laid eggs along with a 1 mm ruler on a specialized egg stand using a 5 megapixel full sensor digital camera mounted in a standardized position and backlit with a ring flash. This provided high-resolution digital images that were analyzed using SigmaScan Pro 5.0 software (SPSS, Inc.) to measure total egg cross-sectional area (in mm^2). In a previous study, we have verified the measurements produced by this method to accurately represent egg size (for further details of method see Badyaev et al. 2005). The effect of egg size on morphology was assessed using least squares regression (PROC REG, SAS 9.1.3) of trait size (residuals after controlling for variation due to year) on egg size.

Results

Estimation of Pairwise Relatedness

Simulations of all pairwise combinations yielded a normal distribution of relatedness values, with a slight right-hand skew (mean = −0.030, median = −0.038, range = −0.298–0.716). Comparison with distribution of relatedness values for observed pairings (mean = −0.011, median = −0.014, range = −0.203–0.228) revealed that partners were more related than expected from simulations ($Z = 2.75$, $P < 0.006$, two-sided). Shape and location of distributions of relatedness values also differed between observed and expected ($D = 0.15$, $P < 0.005$). Categorization of observed pairings based on simulated distribution resulted in a 'typical' relatedness group (mean $r = -0.069 \pm .006$ S.E.) of 67 unique pairings (186 offspring in 76 broods), and a 'high' relatedness group (mean $r = 0.056 \pm .006$ S.E.) of 52 unique pairings (131 offspring in 57 broods). Post hoc evaluation of inbreeding coefficients using offspring genotypes showed that the two groups differed in level of inbreeding ($F_{IS,\ 'typical'} = 0.006$, $P = 0.27$; $F_{IS,\ 'high'} = 0.018$, $P = 0.04$). Extrapair paternity and remating with different partners caused three sires and eight dams to appear in both 'typical' and 'high' relatedness groups.

Effect of Parental Relatedness on Morphological Variation

Analysis of relative morphological variation revealed an effect of parental relatedness across traits (Fig. 1). Compared to families with 'typical' parental relatedness, offspring of families with 'high' parental relatedness had significantly greater variation in all traits (bill length: $F_{193.5} = 4.80$, $P = 0.031$; bill width: $F_{189.7} = 8.95$, $P = 0.0036$; wing length: $F_{188.2} = 7.88$, $P = 0.0062$; tarsus length: $F_{190.1} = 8.84$, $P = 0.0038$; body mass: $F_{188.6} = 4.25$, $P = 0.042$) except bill depth ($F_{185.7} = 0.00$, $P = 0.95$).

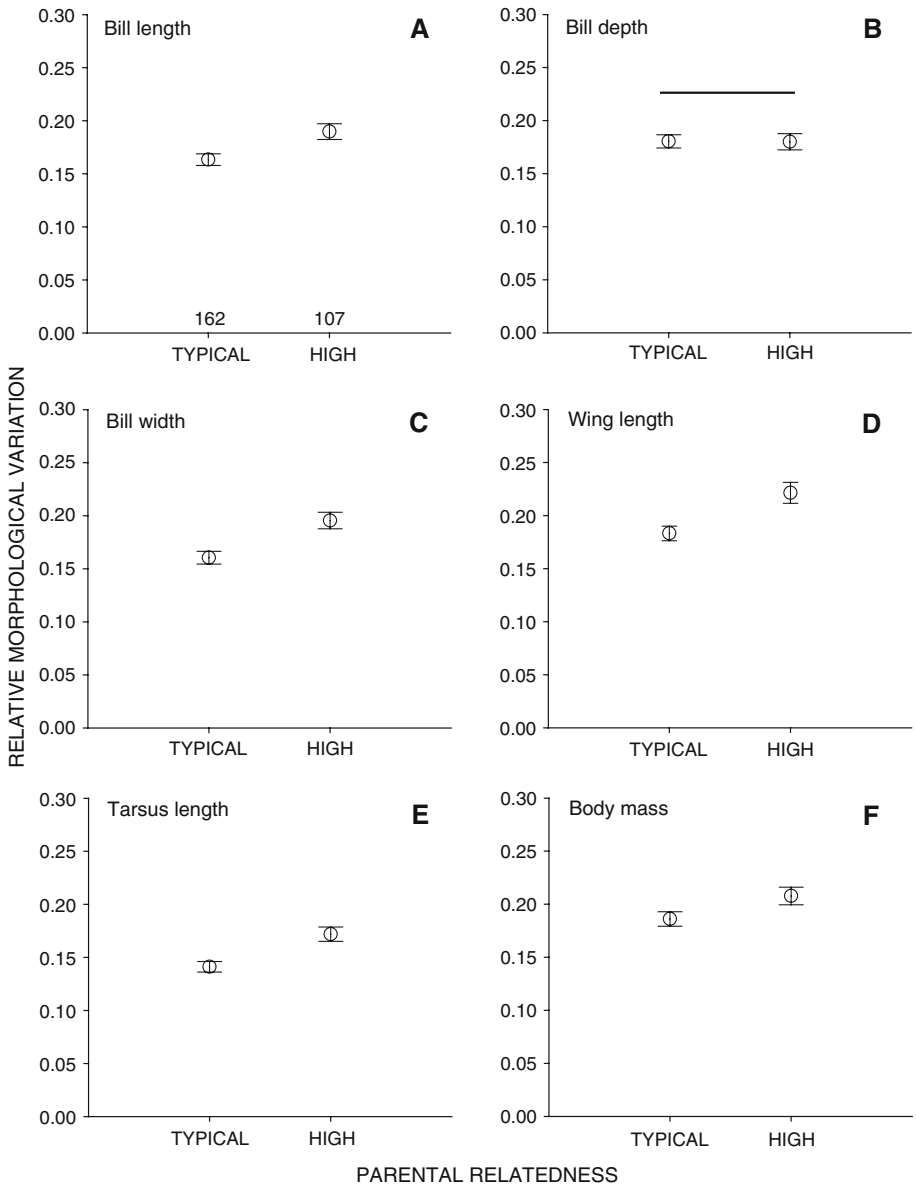

Fig. 1 The effect of parental relatedness on relative phenotypic variation in offspring. Families were categorized as having either 'typical' or 'high' parental relatedness (see Methods). Numbers above the abscissa are samples sizes. Error bars are standard errors of the mean and solid horizontal line indicates no statistical significance between group means

Variance Components

Results of mixed-model analysis revealed distinct variance component structure with respect to parental relatedness (Appendix, summarized in Fig. 2). Among offspring with 'typical' parental relatedness, phenotypic variation consisted of high and statistically significant additive genetic variance (V_A) and heritabilities for bill length, bill depth, and body mass, but only moderate values for bill width, wing length, and tarsus length (Appendix, left side). In contrast, among offspring from 'high' relatedness pairings, none of the traits had significant additive genetic components (Appendix, right side). Differences between groups with respect to common environmental variance (V_{CE}) were largely reversed; variation in the 'high' relatedness group had a relatively high and statistically significant environmental component in all traits (as indicated by a significant effect of nest identity), while only bill length, bill depth, and wing length showed significant variance due to common environment among individuals from 'typical' relatedness families. Of particular note, V_{CE} for body mass in 'typical' families was fixed at zero, suggesting a negative value. When the model was rerun with no constraints, this value converged on a small negative value ($-0.02 \times 10^{-2} \pm 0.21 \times 10^{-2}$). However, when carried through subsequent analyses, the results did not change qualitatively and thus we only report results using the constrained value. Likelihood ratio tests indicated that groups did not differ in additive genetic variance, but differed in environmental variance for wing length, tarsus length, and body mass (Appendix, far right column).

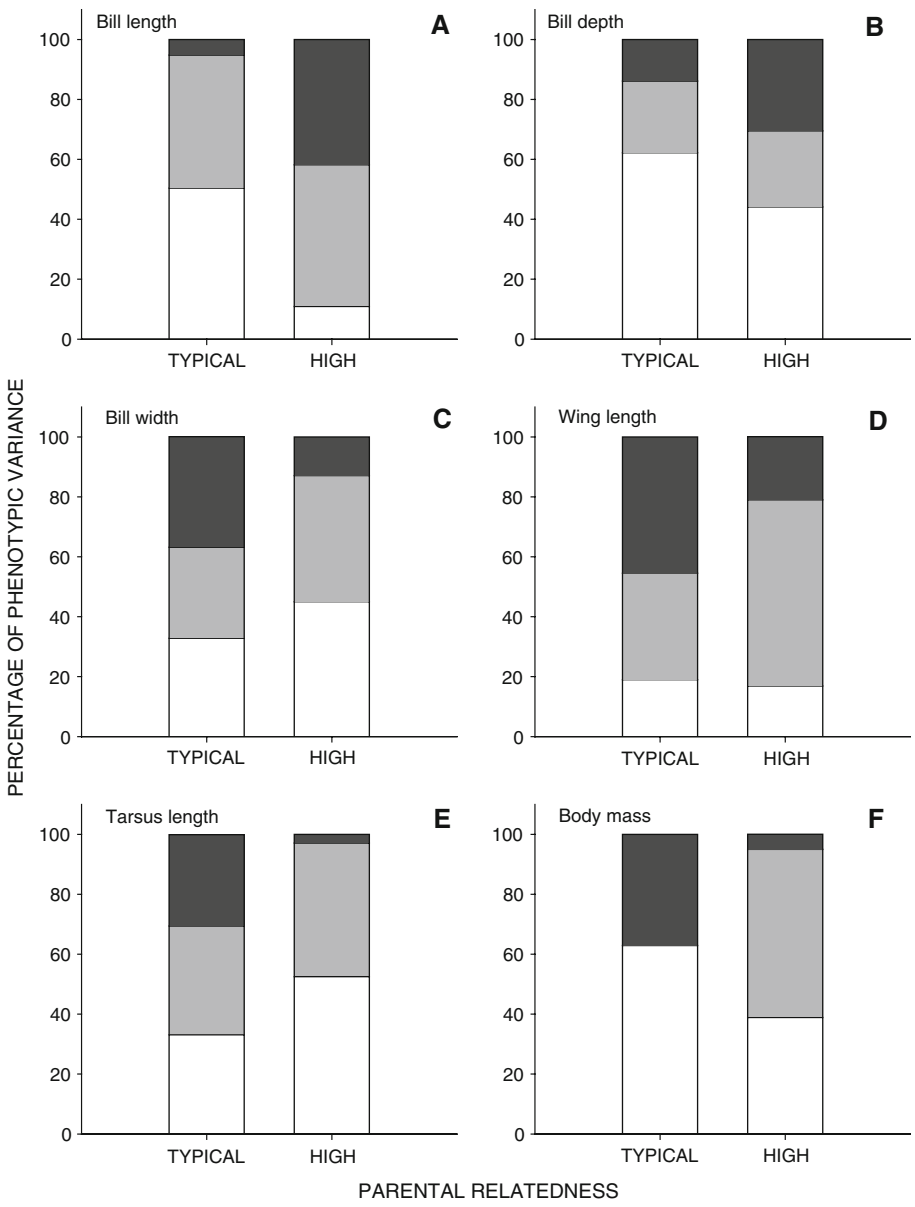

Fig. 2 Variance components of offspring phenotypic variation, expressed as percentage of total phenotypic variance (V_P) due to additive genetic ($V_{A,}$ white bars), common environment ($V_{CE,}$ light gray bars), and residual variance ($V_{R,}$ dark gray bars)

Parental Relatedness and Offspring Condition

Although offspring of more related parents had greater phenotypic variation (Fig. 1), we found no evidence of a direct effect of inbreeding on nestling condition; comparison of offspring from families with 'high' versus 'typical' relatedness revealed no significant differences in residual body mass ($F_{172} = 0.21$, $P = 0.65$).

Response to Inbreeding in Relation to Trait Developmental Timing

Growth and pseudo-velocity curves from nonparametric regression are shown in Fig. 3 (for clarity, only the period from days 1–20 is shown for each trait). As in previously studied house finch populations (e.g., Badyaev et al. 2001b), all traits exhibited growth patterns that were approximately sigmoidal. However, timing of peak velocity differed among traits, ranging from day 1 (male bill depth, Fig. 3b) to day 14 (male wing length, Fig. 3d). Additionally, the sequence in which different traits reached peak velocity differed between males ($T_{\text{max,bill depth}} < T_{\text{max,bill length}} < T_{\text{max,bill width}} < T_{\text{max,tarsus length}} < T_{\text{max,body mass}} < T_{\text{max,wing length}}$) and females ($T_{\text{max,bill width}} < T_{\text{max,tarsus length}} < T_{\text{max,bill depth}} < T_{\text{max,bill length}} < T_{\text{max,body mass}} < T_{\text{max,wing length}}$; Fig. 3). Tests of association between timing of trait maturation and response to inbreeding (i.e., % of V_P in 'high' relatedness group − % of V_P in 'typical' relatedness group) in additive

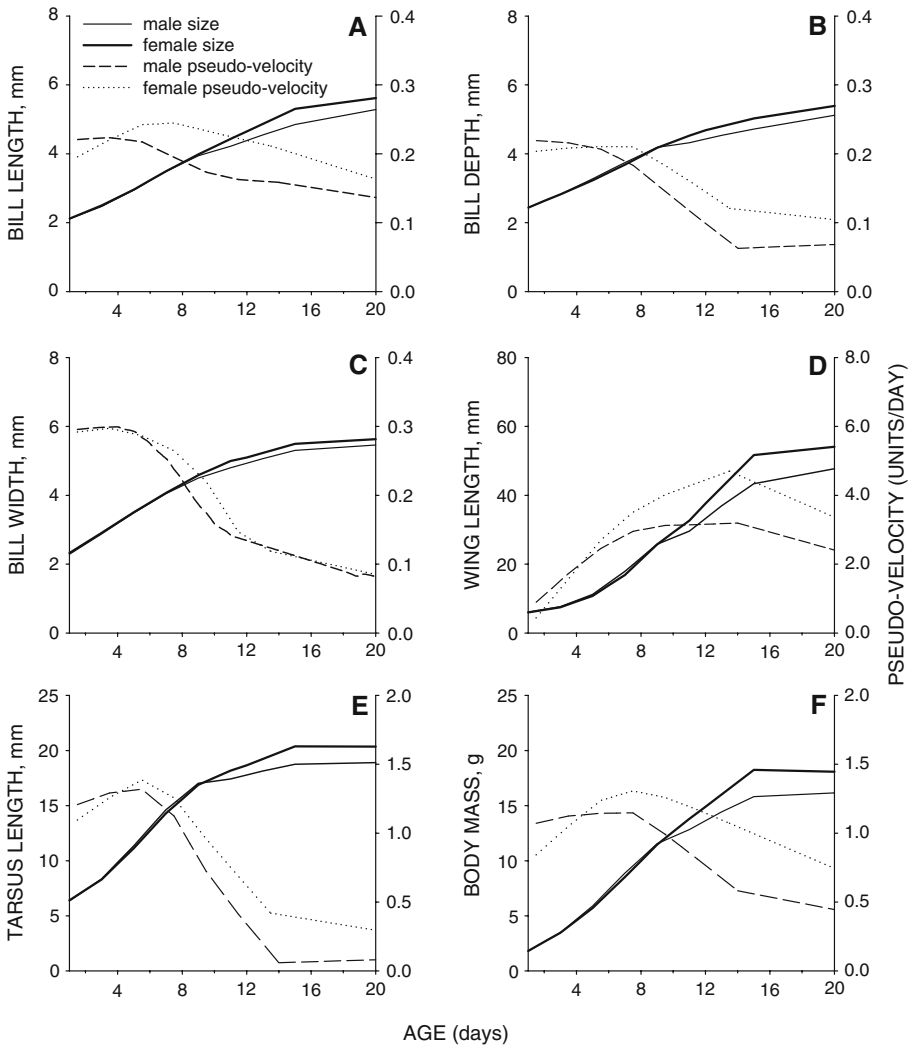

Fig. 3 Growth (solid lines) and pseudo-velocity curves (dashed lines) for male and female house finches. Curves are derived from nonparametric (LOWESS) smoothing function

genetic variance revealed no significant concordance for neither males (Fig. 4a, Kendall's $\tau = 0.20$, $P > 0.1$) nor females (Fig. 4b, $\tau = -0.33$, $P > 0.1$). However, response to inbreeding was positively correlated with timing of trait maturation with respect to common environmental variance, such that the difference in V_{CE} between 'high' and 'typical' relatedness was greatest among later-maturing traits in males (Fig. 4c, $\tau = 0.73$, $P < 0.04$) but not females (Fig. 4d, $\tau = 0.20$, $P > 0.1$).

Parental Relatedness and Sensitivity to Maternal Effects

The effect of maternal allocation to egg size on offspring morphology differed between 'typical' and 'high' relatedness groups (Table 1). Among offspring from 'high' relatedness families, larger eggs resulted in significantly larger trait size for bill length, wing length, tarsus length, and body mass (Table 1), but egg size did not covary with morphology of offspring from 'typical' relatedness families.

Discussion

The extent to which phenotypic variation reflects additive genetic effects has important implications for evolutionary response to selection (Falconer and Mackay 1996). However, because large contributions of environmental or maternal variance can overwhelm other sources of variation, the expression of additive genetic effects will depend on the sensitivity of developing organisms to variation in the biotic and abiotic environment (Schmalhausen 1949; Lerner 1954). Specifically, diversity of allelic developmental products, such as enzymes, might affect individuals' ability to buffer development from extreme environmental perturbations, suggesting that the contribution of environmental and maternal inputs to phenotypic variation may be affected by level of inbreeding. Here we tested this hypothesis by examining the influence of parental relatedness on expression of additive genetic and environmental variation in growth in a wild population of house finches.

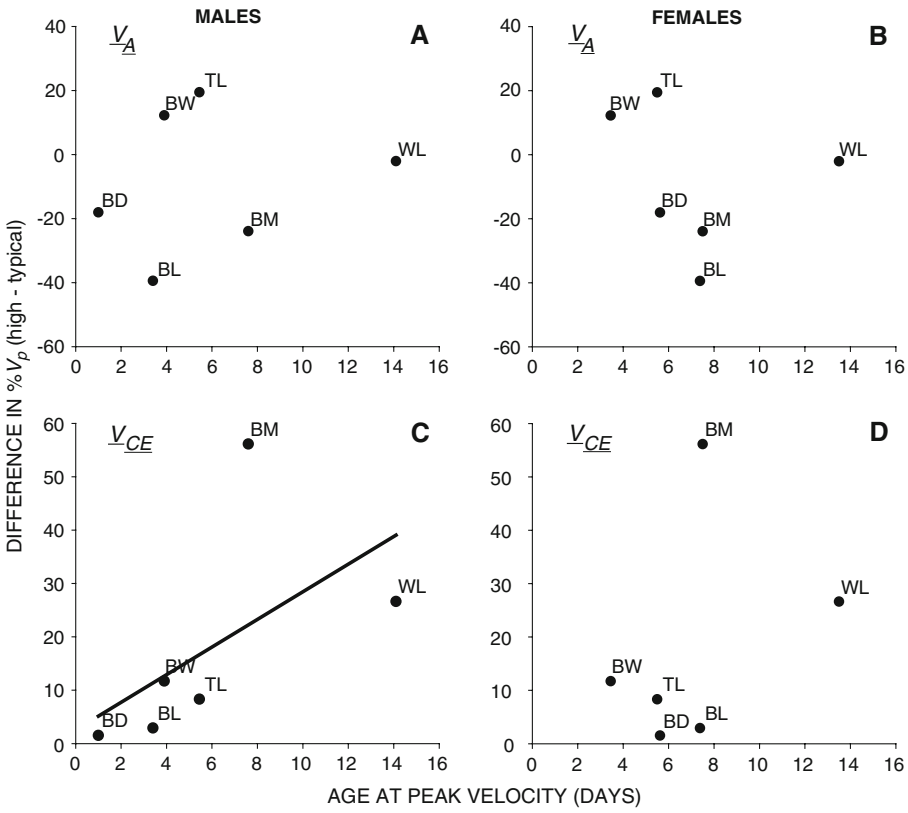

Fig. 4 The relationship between timing of trait growth (age at peak velocity) and effect of inbreeding (difference between 'high' and 'typical' relatedness families) on percentage of phenotypic variance due to (**A**, **B**) additive genetic (V_A) and (**C**, **D**) common environment (V_{CE}) components in males (left column) and females (right column). Larger values on ordinate indicate greater effect of inbreeding on variance components. Solid line represents least-squares regression shown for illustrative purposes only (see results for nonparametric tests of association). BL, bill length; BD, bill depth; BW, bill width; WL, wing length; TL, tarsus length; BM, body mass

Table 1 Maternal effects (allocation to egg size) on offspring morphology in 'typical' (left column) and 'high' (right column) relatedness families. Shown are standardized regression coefficients (b_{ST}, in standard deviations) from least squares regression of nestling trait values (at day two post-hatching) on egg size

Trait	Typical relatedness		High relatedness	
	b_{ST}	*P*	b_{ST}	*P*
Bill length	0.03	0.88	0.37	0.001
Bill depth	0.16	0.26	0.01	0.90
Bill width	−0.15	0.30	−0.08	0.49
Wing length	0.16	0.26	0.31	0.006
Tarsus length	−0.15	0.30	0.27	0.02
Body mass	0.00	0.98	0.34	0.003
N	50		76	

Our study produced four main results. First, consistent with the hypothesized effect of inbreeding on an organism's sensitivity to environmental variation, offspring of highly related parents had greater morphological variation than those from families with lower parental relatedness (Fig. 1). Second, partitioning of phenotypic variation suggested that these differences resulted from a marked increase in common environmental variance (V_{CE}) coupled with a tendency for lower additive genetic variance (V_A) among offspring of parents with relatively high relatedness compared to families with less related parents (Fig. 2, Appendix). Indeed, among 'high' relatedness families, estimates of V_A did not differ significantly from zero for any trait, while V_{CE} was significantly greater compared to 'typical' relatedness families for three of the six traits. Third, our results suggest that these patterns are due to an effect of allelic diversity on offspring sensitivity to environmental and maternal variation, as opposed to more general deleterious effects of inbreeding on offspring physiological condition, which previous studies have shown to influence expression of heritable genetic variation directly (Merilä 1997; Hoffman and Merilä 1999). This interpretation was corroborated by a positive association between timing of trait development (Fig. 3) and magnitude of response to inbreeding—later-maturing traits exhibited an increasingly greater difference between 'typical' and 'high' relatedness groups in variation due to environmental effects in males (Fig. 4c), but not in females (Fig. 4d) or with respect to differences in V_A (Fig. 4a,b). Finally, our analysis of maternal allocation to egg size demonstrated that the observed increase in morphological variation in the 'high' relatedness families was partially due to greater sensitivity to maternal effects (Table 1).

While the effect of intraindividual genetic diversity on sensitivity to environmental variation has been suggested in earlier work (Mitton and Grant 1984; Deng 1997; Réale and Roff 2003; Kristensen et al. 2005), this study provides novel insights in several respects. First, whereas the

necessity for detailed pedigrees has largely constrained previous studies to inbred laboratory lines, the use of molecular marker-derived estimates of relatedness in this study allowed us to examine naturally occurring variation in inbreeding. For such studies, within population comparisons are generally preferred to contrasts between populations, which may involve differences in environmental or genetic variation (Mitton 1993; Whitlock and Fowler 1996), or divergence in trait canalization. The few studies that have examined inbreeding effects on phenotypic variation within natural populations have typically compared morphological variation among groups of randomly sampled individuals that differ in heterozygosity at a few allozyme loci (Mitton 1978; Fleischer et al. 1983; Yezerinac et al. 1992), which is not likely to represent genome-wide diversity (Mitton and Pierce 1980) and thus, might confound the effects of inbreeding on developmental stability with additive genetic effects (Chakraborty and Ryman 1983). Recently, the precision of neutral molecular-marker derived metrics in estimating inbreeding has been questioned (e.g., Pemberton 2004), and while we cannot rule out entirely the effects of a small number of linked loci, the utilization of pairwise relatedness estimates derived from fifteen highly polymorphic microsatellite loci in this study likely captures a greater proportion of genome-wide variability and inbreeding history compared to previous studies of wild populations (Blouin 2003; see also significant F_{IS} among 'high' but not 'typical' families).

Since the effect of heterozygosity on developmental stability was first proposed (Lerner 1954), several alternative mechanisms for the relationship between inbreeding and phenotypic variation have been put forth including additivity at linked quantitative trait loci (Chakraborty and Ryman 1983) and deleterious effects of inbreeding depression on physiological condition (Hoffman and Merilä 1999). While none of these mechanisms are mutually exclusive (David 1999), elucidating their relative importance is critical for predicting traits' response to selection. In this study, we first tested whether the observed pattern of increased environmental variance with greater parental relatedness was mediated by an effect of inbreeding on physiological condition, which itself has been shown to result in larger environmental variance components (Merilä 1997; Hoffman and Merilä 1999). However, we found no differences between offspring from 'typical' and 'high' relatedness females, and while there might be aspects of viability (e.g., non-specific immune response, Saino et al. 1997) that were not directly assayed in our analysis, such characters are typically captured by overall body condition in birds (Møller and Saino 2004). Second, we capitalized on differences among traits in developmental timing (Fig. 3) to make inferences regarding sources of the increased variation observed in inbred families. Specifically, we reasoned that if inbreeding leads to increased sensitivity during development, the largest effect should be observed in late-maturing traits that are exposed to a greater range and duration of environmental inputs and are thus expected to accumulate more environmental variation compared to earlier-maturing traits. Alternatively, increased common environmental variance in late-maturing traits might result not from the direct accumulation of environmental inputs *per se*, but rather as a consequence of developmental integration (e.g., shared resources or developmental precursors) with early-maturing traits such that variation is magnified across ontogeny (Cheverud 1996). Regardless, our results (Fig. 4c) suggest that increased common environmental variance (V_{CE}) in 'high' relatedness families was at least in part due to greater input of environmental variation during development in males, though the role of nonadditive genetic effects (e.g., dominance, epistasis) cannot be excluded entirely. Interestingly, the earliest-maturing trait (bill depth) that was least-affected by parental relatedness (i.e., smallest change in V_{CE} between groups) was also the trait in which no significant difference was observed in phenotypic variance (Fig. 1b), and one of the traits unaffected by variation in maternal allocation to egg size, even among offspring from 'high' relatedness pairings (Table 1). The absence of a similar relationship between trait developmental timing and change in variance components due to inbreeding in females (Fig. 4d) may be due to differences between sons and daughters in sensitivity to maternal effects. Indeed, previous work in this species has suggested that sex-specific sensitivity to maternal effects facilitated the rapid evolutionary change of sexual dimorphism observed in newly established populations (Badyaev 2005a).

The effect of inbreeding on the expression of heritable and nonheritable variation has several important implications for evolutionary studies in natural populations (Hall 2005). At large scales these results imply that populations that differ in levels of inbreeding will have distinct responses to selection (Reed et al. 2003). And while the direct consequences of inbreeding on additive genetic variation are well known from population genetics models (Whitlock and Fowler 1999), this study suggests specifically that such effects can be mediated via an effect of inbreeding on developmental variation. Second, when the relatedness of mates varies over time or space within a population, the response to selection will be similarly heterogeneous. For example, in previous work with this species, we found pronounced seasonal patterns in availability of unrelated mates (Lindstedt et al. 2006; Oh and Badyaev 2006), suggesting that selection on heritable variation should similarly vary across the breeding season. As a corollary to this, the increase in environmental variance with parental relatedness should also influence the maintenance of genetic variation in traits under strong

selection. In populations or breeding contexts with relatively high parental relatedness, selection may act largely on nonheritable variation, thereby preserving unfit genotypes that might otherwise be removed by natural selection (Alatalo et al. 1990). Finally, the results from our analysis of trait growth patterns suggest that the effects of inbreeding on morphological variation are likely to vary in relation to trait developmental timing. In particular, a greater increase in environmental and maternal variation in late-developing traits may serve as a general prediction for future studies of inbreeding in natural populations.

Despite robust evidence of a relationship between parental relatedness and developmental stability, one unexplored question is whether the increased environmental variation can be adaptive (Badyaev 2005b). Increased phenotypic variation among offspring can be favored as a bet-hedging strategy, but only under conditions of large and unpredictable fluctuations in the adaptive landscape (Cooper and Kaplan 1982; Kaplan and Cooper 1984; McGinley et al. 1987; Young and Badyaev 2007), or when increased variation occurs exclusively in lineages that experience low fitness (Hadany and Beker 2003). In our study, V_{CE} represents not only variation due to ecological characteristics of the nest itself (e.g., proximity to food sources), but also variation due to maternal effects, which have been shown to strongly influence offspring phenotypes and fitness in this species (Badyaev 2005a). In birds, egg size is a maternal character known to have particularly pronounced effects on offspring phenotypes (Hipfner and Gaston 1999; Potti 1999; Reed 1999; Maddox and Weatherhead 2008). In addition to determining resource and energy reserves available to developing neonates, egg size may also influence thermal properties during incubation and after hatching (reviewed in Williams 1994). Interestingly, in our study we found that egg size strongly affected offspring morphology from 'high' but not 'typical' relatedness pairings (Table 1). Thus, in this natural population, parental relatedness appears to influence the expression of maternal effects by determining offspring sensitivity during development, such that phenotypes of relatively inbred offspring are more affected than outbred offspring. Such variable efficacy of maternal effects in relation to relatedness of mates is likely to have facilitated the rapid morphological adaptation of the house finch during the species' range expansion across North America over the last 70 years (Badyaev et al. 2002; Badyaev 2005a) that was characterized by the establishment of isolated and often inbred populations (Wang et al. 2003; Hawley et al. 2006).

In conclusion, this study suggests that the relative contribution of heritable genetic and environmental sources to phenotypic variation can be affected by degree of parental relatedness, thereby providing a link between mating patterns and the evolution of adaptation. A better understanding of such effects and the underlying mechanisms should be particularly important for predicting an evolutionary response to selection in natural populations.

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Appendix Components of phenotypic variance for offspring of parents with typical (left column) versus relatively high relatedness (right column)

Trait	Source	Typical relatedness		High relatedness		Difference
		Variance ± S.E.	% of V_p	Variance ± S.E.	% of V_p	
Bill length	V_A	$5.53 \times 10^{-4} \pm 1.50 \times 10^{-4}$*	50.4 ± 13.1	$1.08 \times 10^{-4} \pm 2.64 \times 10^{-4}$	10.9 ± 26.7	4.45×10^{-4}†
	V_{CE}	$4.87 \times 10^{-4} \pm 1.32 \times 10^{-4}$***	44.4 ± 10.0	$4.69 \times 10^{-4} \pm 2.01 \times 10^{-4}$***	47.3 ± 16.6	$.180 \times 10^{-4}$
	V_R	$.567 \times 10^{-4} \pm .659 \times 10^{-4}$	5.17 ± 6.23	$4.15 \times 10^{-4} \pm 1.49 \times 10^{-4}$**	41.8 ± 16.2	$.830 \times 10^{-4}$***
	V_P	$11.0 \times 10^{-4} \pm 1.50 \times 10^{-4}$		$9.92 \times 10^{-4} \pm 1.56 \times 10^{-4}$		
Bill depth	V_A	$5.45 \times 10^{-4} \pm 2.06 \times 10^{-4}$**	62.2 ± 19.8	$4.11 \times 10^{-4} \pm 2.76 \times 10^{-4}$	44.1 ± 27.3	1.34×10^{-4}
	V_{CE}	$2.09 \times 10^{-4} \pm 1.06 \times 10^{-4}$**	23.9 ± 11.1	$2.37 \times 10^{-4} \pm 1.59 \times 10^{-4}$*	25.4 ± 16.0	$.280 \times 10^{-4}$
	V_R	$1.21 \times 10^{-4} \pm .613 \times 10^{-4}$*	13.9 ± 13.2	$2.84 \times 10^{-4} \pm 1.50 \times 10^{-4}$	30.5 ± 17.8	$.830 \times 10^{-4}$
	V_P	$8.76 \times 10^{-4} \pm 1.18 \times 10^{-4}$		$9.33 \times 10^{-4} \pm 1.44 \times 10^{-4}$		
Bill width	V_A	$1.66 \times 10^{-4} \pm 1.57 \times 10^{-4}$	32.8 ± 23.1	$5.88 \times 10^{-4} \pm 2.37 \times 10^{-4}$	45.0 ± 25.2	4.22×10^{-4}
	V_{CE}	$1.37 \times 10^{-4} \pm .930 \times 10^{-4}$	30.4 ± 12.0	$5.49 \times 10^{-4} \pm 2.26 \times 10^{-4}$***	42.1 ± 14.6	4.12×10^{-4}
	V_R	$4.04 \times 10^{-4} \pm 1.07 \times 10^{-4}$***	36.9 ± 16.7	$1.68 \times 10^{-4} \pm 1.79 \times 10^{-4}$	12.9 ± 14.5	2.36×10^{-4}
	V_P	$8.71 \times 10^{-4} \pm 1.15 \times 10^{-4}$		$13.1 \times 10^{-4} \pm 2.18 \times 10^{-4}$		

Appendix continued

Trait	Source	Typical relatedness		High relatedness		Difference
		Variance ± S.E.	% of V_p	Variance ± S.E.	% of V_p	
Wing length	V_A	$.930 \times 10^{-3} \pm 1.15 \times 10^{-3}$	19.0 ± 23.1	$1.39 \times 10^{-3} \pm 2.27 \times 10^{-3}$	16.9 ± 27.2	4.60×10^{-4}
	V_{CE}	$1.73 \times 10^{-3} \pm .748 \times 10^{-3}$**	35.5 ± 13.5	$5.10 \times 10^{-3} \pm 1.70 \times 10^{-3}$***	62.1 ± 15.2	3.37×10^{-3}**
	V_R	$2.23 \times 10^{-3} \pm .694 \times 10^{-3}$**	45.5 ± 15.6	$1.73 \times 10^{-3} \pm 1.17 \times 10^{-3}$	21.1 ± 15.0	5.00×10^{-4}
	V_P	$4.89 \times 10^{-3} \pm .626 \times 10^{-3}$		$8.22 \times 10^{-3} \pm 1.41 \times 10^{-3}$		
Tarsus length	V_A	$4.46 \times 10^{-4} \pm 3.57 \times 10^{-4}$	33.1 ± 25.1	$1.28 \times 10^{-3} \pm .336 \times 10^{-3}$	52.5 ± 14.1	8.34×10^{-4}
	V_{CE}	$4.89 \times 10^{-4} \pm 2.00 \times 10^{-4}$***	36.3 ± 13.2	$1.09 \times 10^{-3} \pm .416 \times 10^{-3}$***	44.6 ± 11.9	6.01×10^{-4}**
	V_R	$4.11 \times 10^{-4} \pm 2.02 \times 10^{-4}$*	30.5 ± 16.5	$.070 \times 10^{-3} \pm .123 \times 10^{-3}$	2.88 ± 5.15	3.41×10^{-4}
	V_P	$13.5 \times 10^{-4} \pm .018 \times 10^{-4}$		$2.42 \times 10^{-3} \pm .392 \times 10^{-3}$		
Body mass	V_A	$13.3 \times 10^{-3} \pm 4.90 \times 10^{-3}$**	62.9 ± 18.0	$10.2 \times 10^{-3} \pm 3.48 \times 10^{-3}$	38.9 ± 14.1	3.10×10^{-3}
	V_{CE}	0.00 ± 0.00‡	0.00 ± 0.00	$14.7 \times 10^{-3} \pm 4.85 \times 10^{-3}$***	56.1 ± 11.5	1.47×10^{-2}*
	V_R	$7.87 \times 10^{-3} \pm 3.38 \times 10^{-3}$*	37.1 ± 18.0	$1.31 \times 10^{-3} \pm 1.40 \times 10^{-3}$	5.03 ± 5.46	6.56×10^{-3}
	V_P	$21.2 \times 10^{-3} \pm 2.66 \times 10^{-3}$		$26.1 \times 10^{-3} \pm 4.50 \times 10^{-3}$		
N (nests/nestlings)		76/186		57/131		

Underlined values are equivalent to narrow-sense heritability (h^2)

Significance based on log-likelihood ratio test: $^{\dagger}P < 0.1$; * $P < 0.05$; **$P < 0.01$; *** $P < 0.01$

Difference = absolute difference between variance component estimates

‡ Variance component fixed at boundary (see results)

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