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Original Article

Maternal and additive genetic effects contribute to variation in offspring traits in a lizard

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Evolutionary responses to selection require that traits have a heritable basis, yet maternal effects (the effect of a mother's phenotype on her offspring's phenotype) can have profound effects on evolutionary processes. It is therefore essential to understand how maternal effects contribute to phenotypic variation in offspring traits and test key assumptions of additive genetic variance in evolutionary models. We measured 5 traits linked to fitness in lizards (endurance, sprint speed, snout-vent length [SVL], mass, and growth rate) and estimated the contribution of additive genetic and maternal effects in explaining variation in these traits in the Eastern water skink (*Eulamprus quoyii*). We estimated parentage using 6 microsatellite DNA loci from lizards taking part in a mating experiment in large seminatural enclosures and used animal models to partition variance into additive genetic and maternal effects. We found that only endurance was significantly heritable (h^2 = 0.37, 95% credible interval = 0.18–0.50), whereas all other traits were either strongly influenced by maternal effects (mass, sprint speed, SVL, and captive growth rate) or were influenced by environmental variability (wild growth rate). Our study disentangles the relative contributions of additive genetic and maternal effects in contributing to variation in offspring phenotypes and suggests that little additive genetic variance exists for traits often assumed to be heritable. Although the heritability of phenotypic traits is essential in evolutionary models, our results also highlight the important role maternal effects have in explaining variation in phenotypes.

Key words: **additive genetic variance, animal model, endurance, *Eulamprus quoyii*, growth rate, heritability, physiological performance, sprint speed, water skinks.**

INTRODUCTION

Evolutionary response requires that traits have a genetic basis, and this assumption is essential for all facets of evolutionary thinking from understanding the role of "good" genes models in sexual selection (Qvarnström and Price 2001) to determining the causes of phenotypic variation in traits (Mousseau and Fox 1998). Importantly, a trait's response to selection will depend not only on its heritability and the spatial and temporal stability of selection, but also on how maternal effects, both genetic and environmental, influence trait expression (Lande and Kirkpatrick 1990; Qvarnström and Price 2001; Bonduriansky and Day 2009). In addition to the direct genetic contribution from the mother to her offspring (i.e., through additive genetic effects), mothers also can indirectly influence variance in offspring phenotypes through maternal effects (Kirkpatrick and Lande 1989; Räsänen and Kruuk 2007). For example, mothers may manipulate the offspring's developmental environment and/or differentially invest into offspring depending on their phenotype (Mousseau and Fox 1998). Theoretical work has shown that maternal effects can have important and even counterintuitive effects on the response to selection and possibly facilitate the maintenance of additive genetic variation (Kirkpatrick and Lande 1989; Bonduriansky and Day 2009).

Maternal effects are now recognized as important contributors to phenotypic variation in offspring across a wide diversity of taxa (Mousseau and Fox 1998; Qvarnström and Price 2001; Räsänen and Kruuk 2007), having persistent effects over an individual's lifetime (Kerr et al. 2007) and influencing offspring phenotypes across generations (Magiafoglou and Hoffmann 2003; Hafer et al.

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2011). A plethora of complex maternal factors are now recognized to affect offspring phenotype, including cytoplasmic and somatic factors such as hormones, proteins, RNA molecules, and nutrients that can be provided via female gametes (Mousseau and Fox 1998; Bonduriansky and Day 2009) and that are dependent on female physiological state and environment. In addition, maternal effects can manifest themselves later in development through the manipulation of the offspring environment (e.g., parturition in habitat that varies in quality) or through the influence of maternal behavior (Bonduriansky and Day 2009). The complex interrelationships between indirect environmental factors and the underlying genetic milieu make understanding the key contributors to phenotypic variation difficult, particularly in nonmodel organisms where it is not possible to control genetic background or conduct controlled mating experiments. These complexities are compounded in natural systems where environmental sources of variation can make estimates difficult to obtain. Nonetheless, determining the relative role of additive genetic and maternal effects in driving variation in offspring traits is paramount in predicting evolutionary responses, and to fully understand what contributes to phenotypic variation, it is essential that we have estimates from natural or seminatural systems in diverse taxa.

Work on understanding the proportion of phenotypic variance explained by maternal and additive genetic effects under natural settings has primarily focused on mammals and birds, which have been ideal systems given that multigenerational pedigrees can be constructed from the long-term mark recapture of individuals [see Kruuk (2004) for an overview of these studies]. The advent of sophisticated statistical tools, such as "animal models," has allowed for the partitioning of phenotypic variation into additive genetic and maternal effects variance using pedigrees (Kruuk 2004). For example, in Soay sheep on St. Kilda, maternal identity can explain as much as 5–21% of variation in morphological traits, whereas additive genetic estimates account for 12–29% (Milner et al. 2000). In red deer, additive genetic effects explained 0–60% of the variance depending on the trait, whereas maternal effects accounted for 16–28% of the variance (Kruuk et al. 2000). Work in red squirrels has estimated very low additive genetic variance in fitness but moderate maternal effects (McFarlane et al. 2014), whereas recent work in house sparrows has shown strong maternal effects (m^2 = 0.33) and low heritability (h^2 = 0.09) for annual productivity (i.e., number of recruits and broods) (Schroeder et al. 2012). Although work in mammals and birds have been influential in identifying the factors contributing to phenotypic variation, much of this work has focused on a few model systems and we know little about what proportion of variance is explained by these effects in taxonomically disparate groups that vary in parental care and levels of sociality.

The role of maternal phenotypes on offspring phenotype has been studied extensively in lizards. Maternally induced changes come in diverse forms and can be a result of differences in basking behavior (Shine and Downes 1999; While and Wapstra 2009), dietary quality (Warner et al. 2007), and physiological state (i.e., female mass, body size, or age) (Noble et al. 2013a). These maternal effects are known to influence a diversity of important offspring traits including body size and mass, performance traits, and growth rates (Shine and Harlow 1993). Although there is ample evidence that maternal effects are important contributors to phenotypic variation, estimates of additive genetic and maternal effects and their relative contribution to explaining variation in single traits have seldom been addressed in reptiles (Tsuju et al. 1989; Sorci et al. 1995; Calsbeek and Sinervo 2004; Le Galliard et al. 2004). Furthermore, few studies genetically determine paternity and thus do not adequately permit the separation of heritable from maternal effects variation because many estimates are derived from full sibling parent–offspring regressions (Sorci et al. 1995; Le Galliard et al. 2004). Although valuable, such techniques can confound heritability with maternal effects and inflate heritability estimates (Kruuk 2004).

We measured 5 traits (endurance, sprint speed, body size, mass, and growth rates) in an Australian lizard (*Eulamprus quoyii*) to understand the role of maternal and additive genetic effects in driving variation in offspring traits. We first established maternal and paternal identity of offspring from seminatural breeding experiments before using animal models to partition phenotypic variance to determine the importance of maternal and additive genetic effects in traits which are known to be under selection in many lizard species (Sinervo et al. 1992; Elphick and Shine 1998; Warner and Andrews 2002; Husak 2006; Irschick et al. 2008; Le Galliard and Ferrière 2008). Although we do not yet understand how these traits are linked to survival in *Eulamprus*, a number of other lizard studies have shown survival to increase with offspring body size (Elphick and Shine 1998; Warner and Andrews 2002), mass (Warner and Andrews 2002), sprint speed (Warner and Andrews 2002), endurance (Le Galliard and Ferrière 2008), and growth rates (Warner and Andrews 2002). The high levels of multiple paternity in *Eulamprus* (e.g., ~ 45–65% of clutches; Morrison et al. 2002; Stapley and Keogh 2005; Keogh et al. 2012; Noble et al. 2013a) provide an excellent opportunity to assess the role of heritable and nongenetic maternal effects on offspring traits because families of maternal half siblings better allow the statistical separation of additive genetic and maternal effects than full sibling families. We take a multistage modeling approach whereby we compare variance estimates from variance component models to the same models containing maternal traits to understand how variance estimates change when accounting for maternal phenotype. This allowed us to estimate maternal effect coefficients (Kirkpatrick and Lande 1989), while also informing on what proportion of maternal effect variance might be attributed to such traits.

MATERIALS AND METHODS

Experimental design

The Eastern water skink, *E. quoyii*, is a moderately sized lizard species (adult snout-vent length [SVL]: 90–118 mm) and is widespread across eastern Australia. They are viviparous, giving birth to 1–9 offspring in mid December to January and have no parental care. We collected 216 (108 males and 108 females) *E. quoyii* and allowed them to breed in seminatural enclosures as part of a mating system experiment conducted in 2010 (Noble et al. 2013a, 2013b). Experiments were conducted in six 16 × 10 m outdoor enclosures with 18 males and 18 females in each enclosure (for details on enclosures see: Noble et al. 2013a, 2013b). We collected adults at the end of the breeding season (November) and brought females back into the laboratory until parturition. At parturition (December to January), offspring SVL and mass were recorded and a small tissue sample taken for genetic analysis. Prior to release into outdoor enclosures, all offspring were housed individually in plastic containers in a temperature-controlled room (~28–30 °C).

We measured offspring sprint speed and endurance once each day (approximately 1 week after birth) over 3 consecutive days and used all 3 measurements in our analyses. Lizards were warmed to their optimal body temperature (28 °C; Law and Bradley 1990) prior

to all performance measurements using a temperature-controlled incubator. Sprint speed was measured by running lizards down a racetrack outfitted with photocells, and the time between triggering the first and the last photocell over 1 m was used to compute speed. Immediately thereafter, we measured endurance by placing lizards in a circular track and stimulated them to run until exhaustion by gently taping on the tail. Lizards were considered exhausted if, after 5 gentle taps, they no longer moved, at which point we recorded the time. Once all performance measurements were finished we recorded individual mass and SVL again prior to releasing lizards in 4 (out of 6) seminatural enclosures where they were recaptured monthly until April, prior to winter, to estimate individual growth rates. Growth rates were calculated ($SVL_{t2} - SVL_{t1}/\Delta t$) in captivity during their period of stay while being measured on performance traits and under seminatural conditions in our outdoor enclosures. We used both measures in analyses. Unfortunately, given the high incidence of natural toe loss and the low survivorship, it was not possible to use offspring survival in analyses. As a result, we were also not able to get accurate growth rate estimates for many lizards in the wild in the last month of the active season.

Parentage assignment

We used 6 polymorphic microsatellite DNA loci to identify paternal identity as outlined in Noble et al. (2013a). Briefly, polymerase chain reactions (PCRs) were carried out in 20 μL reaction volumes with 1.0 μL of DNA template, 10 μL of GoTaq® (Promega), and 0.5 μL (10 pmol/μL) of forward and reverse primers based on loci and reaction conditions from Scott et al. (2001) and Sumner et al. (2001). We labeled forward primers with different fluorescent dyes (TET, NAD, VIC, and FAM) and the final PCR products were pooled and run on an ABI 3730 DNA analyzer (Applied Biosystems) and scored by the Australian Genomic Research Facility.

Parentage was assigned using CERVUS 3.0 (Kalinowski et al. 2007) by simulating 100 000 offspring using both a strict (95%) and relaxed (80%) confidence level. Our loci were highly variable (3–34 alleles per loci; mean polymorphic information content = 0.70), and given our controlled mating system experiment, we were able to assign paternity with a high level of confidence (nonexclusion probability for parent pair = 4.46×10^{-6}). We assigned paternity conservatively and excluded possible sires if they had one or more mismatches with an offspring. We were not able to identify the father in all offspring because we were not able to amplify alleles on one or more of the parents or because some females had copulated prior to collection from the wild. Nonetheless, pedigrees in animal models can still incorporate these uncertain paternal links (Kruuk 2004).

Statistical analysis

We used mixed effect "animal models" that combine phenotypic data with pedigree information and compare the phenotypes of related individuals to estimate the additive genetic variance (σ^2_a) of each trait (Kruuk 2004). In addition, we used animal models to estimate maternal (σ^2_m) and permanent environmental effects of the traits we were interested in, where σ^2_m is the differences among individuals due to the influence of their mother (Mousseau and Fox 1998) and σ^2_{pe} is the among individual differences not accounted for by σ^2_a and σ^2_m, and can only be estimated in our performance traits which were repeatedly measured (Kruuk and Hadfield 2007). We also estimated the residual variance (σ^2_r). Thus, phenotypic variance (σ^2_p) was estimated as the sum of all variance components, including σ^2_r. Heritability (h^2) was estimated as σ^2_a/σ^2_p, maternal effects (m^2) were estimated as σ^2_m/σ^2_p, and permanent environmental effects were estimated as σ^2_{pe}/σ^2_p (Lynch and Walsh 1998). We cannot estimate maternal genetic effects because we have a single generation pedigree and thus our maternal effects consist of both maternal genetic and environmental effects.

All animal models were run in R, using the Markov Chain Monte Carlo for generalized linear mixed models (MCMCglmm) package (Hadfield 2010; R Development Core Team 2010). We took a multistep approach to our modeling by first running a variance component model that incorporated only random effects and pedigree information and then subsequently incorporating maternal and offspring traits into models. We adopted this modeling approach for a number of reasons. First, the inclusion of fixed effects can decrease the residual variance, possibly leading to inflated heritability estimates (Wilson 2008) and therefore we wanted a baseline of these estimates that were not biased by the inclusion of fixed effects. Second, we were specifically interested in understanding how maternal effect variance estimates would change with the inclusion of maternal traits because this provides a rough idea about what aspects of maternal phenotype might be contributing to maternal effects variance given that estimates will be conditioned on these female traits (McAdam et al. 2014). Lastly, we included offspring body size (SVL) and days kept in captivity prior to performance measurements because these are known to explain variance in offspring traits (Shine and Harlow 1993). The inclusion of offspring SVL in our models ensures that genetic parameters were conditioned on size to confirm that our univariate inferences for traits did not reflect multivariate consequences of size. Our variance component model for each trait was as follows:

$$T_{ijkz} = u + m_j + a_k + p_z + e_{ijkz} \quad (1)$$

Where T_{ijkz}, is the trait of interest (e.g., sprint speed); u, is the trait mean; m_j, is the variance in trait T resulting from mother j; a_k, is the additive genetic variance in trait T estimated from the pedigree; p_z, is the permanent environmental effects in trait T estimated from repeated measurements of individual z; and e_{ijkz}, is the residual variance. Equation 1 allowed us to estimate the total additive genetic and maternal effects explaining phenotypic variance in each trait. Permanent environmental effects were only estimated in sprint speed and endurance models. For traits with strong maternal effects, we ran separate GLMMs that included maternal traits, which are hypothesized to contribute to the total maternal effect variance:

$$T_{ijk} = u + \beta_{mSVL} + \beta_{mCS} + \beta_{mCond} + \beta_{OBD} + m_j + a_k + e_{ijk} \quad (2)$$

$$T_{ijkz} = u + \beta_{mSVL} + \beta_{mCS} + \beta_{mCond} + \beta_{OBD} + \beta_{OSVL} + \beta_{ODC} + m_j + a_k + p_z + e_{ijkz} \quad (3)$$

Where β_{mSVL} is the effect of mother SVL, β_{mCS} is the effect of mother clutch size, and β_{mCond} is the effect of mother body condition on the mean of trait T. In addition, β_{OBD} is the effect of offspring birth date, β_{OSVL} is the effect of offspring SVL, and β_{ODC} is the effect of days in captivity on the mean of trait T. All other parameters are the same as equation 1. Equation 2 was used to model offspring SVL, mass, and growth rates, whereas equation 3 was used to model sprint speed and endurance. To test whether variance estimates were influenced by shared enclosure effects we

also ran models that included "enclosure" as a fixed effect and assessed whether this influenced additive genetic or maternal effect variances. In all cases this did not influence estimates and we therefore present models without enclosure as a fixed effect. We used the "pedantics" package to create our pedigree (Morrissey 2010). In all models, we log-transformed endurance and sprint speed to normalize their distributions. Variance components were estimated as the mode of the posterior distribution and are reported with 95% credible intervals (CI).

We used parameter-expanded priors for our additive genetic, maternal, and permanent environmental variance estimates as we wanted to have noninformative priors (Hadfield 2010), reflecting our ignorance in making predictions (Ellison 2004). We tried a series of different priors for our residual variance (i.e., $V = 0.001$, nu = 0.002; $V = 0.01$, nu = 0.02; $V = 1$, nu = 1). Weaker priors led to increased CI's for heritability estimates of mass, SVL, and growth rates, whereas point estimates were mostly unchanged (Supplementary Tables S1 and S2), likely due to small sample sizes. The only model where estimates changed strongly was for captive growth rate and closer inspection of MCMC chains and posterior distributions suggested that a weaker prior ($V = 0.01$, nu = 0.02) was best. For all other models we set the residual variance prior to $V = 1$ and nu = 1 given that point estimates were qualitatively similar and this prior specification lead to MCMC chains mixing well with low autocorrelation between samples. To test for convergence of each model, we used a Heidelberger and Welch convergence diagnostic (Heidelberger and Welch 1983) and a Geweke convergence diagnostic (Geweke 1992). We used both diagnostics for every model, as neither is infallible (Cowles and Carlin 1996). All models had converged as measured by both diagnostics, and additionally, chains were not autocorrelated.

RESULTS

We were able to assign paternity to 226 offspring from 56 females. Offspring endurance was significantly heritable ($h^2 = 0.37$, 95% CI = 0.18–0.50; Tables 1 and 2; Figure 1), whereas heritability estimates for sprint speed, SVL, mass, and growth rates were low and the posterior mode was centered on zero (Table 1; Figure 1). Importantly, using a weaker prior for these traits ($V = 0.01$, nu = 0.02) led to wider CIs on heritability estimates, but point estimates were unchanged (Supplementary Tables S1 and S2). The estimate of heritability for endurance remained high when controlling for maternal traits (clutch size, SVL, and condition) and also offspring birth date, body size, and the time spent in captivity prior to endurance measurements (Table 1). Sprint speed, SVL, and mass showed evidence for significant maternal effects (Table 1). Female body size, condition, and clutch size were strong predictors of offspring mass and SVL and contributed significantly to explaining variation in these traits, whereas offspring birth date did not (Table 1). Inclusion of the above covariates in models decreased m^2 estimates (mass: $m^2 = 0.42$ [95% CI = 0.28–0.57]; an 11% decrease; SVL: $m^2 = 0.45$ [95% CI = 0.21–0.61]; a 12% decrease; see Table 1). Residual variance estimates increased and as a result heritability for offspring traits did not change with the inclusion of covariates in the animal model. After controlling for offspring body size and days spent in captivity, offspring birth date had a positive effect on offspring sprint speed, whereas no other covariate explained significant levels of variation (Table 1). Interestingly, additive genetic variance estimates for sprint speed were not affected by the inclusion of fixed effects; however, maternal effect variance decreased ($m^2 = 0.002$ [95% CI = 0–0.26]; a 99% decrease; Table 1). Growth rate estimates did not show evidence for significant heritability, although confidence intervals were large (Table 1; Figure 1). Significant maternal effects were evident for captive growth rates, but not wild growth rates (Table 1). The inclusion of maternal and offspring traits showed a strong effect of birth date and female clutch size on captive growth rate, decreasing maternal effect estimates by 99% (Table 1).

DISCUSSION

Understanding the causes underlying phenotypic variation in traits is fundamental for predicting evolutionary responses. We found evidence for significant heritable variation in offspring endurance, but only maternal effects contributed strongly to variation in sprint speed, body size, mass, and growth rate in captivity. In contrast, environmental factors explained variability in offspring growth rate in the wild.

In *E. quoyii*, offspring body size and mass were strongly influenced by maternal effects that are, in part, explained by maternal condition, body size, and clutch size (explaining approximately 11–12% of the m^2 variance). Life-history theory predicts a trade-off between offspring number and size given that females have finite resources to invest in producing offspring (Roff 2002). However, even with high resource availability, offspring size also may depend on abdominal space constraints in females (Uller and Olsson 2005). Our maternal effect coefficients largely support these tenants of life-history theory, with a negative effect of female clutch size (independent of female SVL) and a positive effect of female SVL (independent of clutch size) on offspring mass and SVL. Offspring size has been shown to be influenced by maternal size in a number of different species (Heath et al. 1991; Kim and Thorp 2001; Kvalnes et al. 2013) and female resource limitation can affect offspring size number trade-offs (Uller and Olsson 2005), suggesting that physiological state has important consequences for offspring phenotype. Offspring mass had low heritability and was controlled by maternal effects, consistent with findings in the lizard *Niveoscincus occelatus* (Uller et al. 2011); however, offspring body size had large confidence intervals around heritability estimates. Body size is known to have high heritability in many taxa (Hansen et al. 2011), including lizards (e.g., Calsbeek and Sinervo 2004). The low heritability estimate we found contradicts these widespread patterns. Our inability to estimate heritability precisely for this trait could be because our pedigree was only a single generation and more precise estimates would need to use a multigenerational pedigree. This would also be necessary to separate maternal genetic from maternal environmental sources of variation in these traits because our estimates encompass both these sources of variation.

Individual growth rate can have important consequences on other life-history traits such as age at maturity as well as survival and fecundity (Hutchings 1993; Roff 2002) and is predicted to exhibit lower heritability (Mousseau and Roff 1987). Both captive and wild growth rates in *E. quoyii* appeared to have low heritability (albeit with wide CIs). Maternal effects were found for captive growth rate with offspring birth date and female clutch size positively influencing growth rate (with a 99% decrease in maternal effect variance). In contrast, we did not detect maternal effects on offspring growth rate under seminatural conditions. Maternal effects have been shown to be important to offspring growth rates in red squirrels with female litter size and parturition date negatively affecting growth rate (McAdam and Boutin 2003). Life-history

Table 1

Estimates of heritability ($h^2 = \sigma^2_a/\sigma^2_p$), maternal effects ($m^2 = \sigma^2_m/\sigma^2_p$), and permanent environment effects ($P_e = \sigma^2_{pe}/\sigma^2_p$) from the posterior distribution along with their 95% CI for 5 offspring traits: SVL, mass, growth rate, endurance, and sprint speed

Model	Offspring SVL	Offspring mass	Captive growth	Wild growth	Log sprint speed	Log endurance
Model 1	$n = 301$	$n = 300$	$n = 235$	$n = 122$	$n = 675$	$n = 670$
Estimates						
h^2	0.002 (0, 0.50)	0 (0, 0.03)	0.001 (0, 0.51)	0 (0, 0.05)	0.001 (0, 0.24)	**0.374 (0.18, 0.50)**
m^2	**0.503 (0.29, 0.63)**	**0.478 (0.37, 0.61)**	**0.363 (0.17, 0.56)**	0 (0, 0.06)	**0.245 (0.13, 0.39)**	0.001 (0, 0.11)
pe^2	—	—	—	—	0.001 (0, 0.15)	0.001 (0, 0.18)
Model 2	$n = 273$	$n = 272$	$n = 208$	$n = 109$	$n = 600$	$n = 596$
Estimates						
h^2	0.002 (0, 0.59)	0 (0, 0.04)	0.003 (0, 0.64)	0 (0, 0.06)	0.001 (0, 0.30)	**0.339 (0.05, 0.45)**
m^2	**0.445 (0.21, 0.61)**	**0.424 (0.28, 0.57)**	0.001 (0, 0.26)	0.001 (0, 0.07)	0.002 (0, 0.26)	0.001 (0, 0.15)
pe^2	—	—	—	—	0.001 (0, 0.16)	0.001 (0, 0.25)
Covariate	Est. (95% CI)	Est. (95% CI)	Est. (95% CI)	Est. (95% CI)	Est. (95% CI)	Est. (95% CI)
Offspring SVL	—	—	—	—	0.02 (−0.002, 0.04)	0.007 (−0.01, 0.03)
Days in captivity	—	—	—	—	0.008 (−0.001, 0.02)	**−0.01 (−0.02, −0.002)**
Offspring birth date	0.03 (−0.02, 0.09)	0.006 (−0.003, 0.02)	**0.028 (0.021, 0.036)**[a]	−0.002 (−0.006, 0.003)	**0.02 (0.002, 0.04)**	−0.02 (−0.06, 0.02)[a]
Mother clutch size	**−0.32 (−0.57, −0.09)**	**−0.02 (−0.05, −0.002)**	**0.011 (0.0005, 0.02)**[a]	0.002 (−0.02, 0.02)	0.005 (−0.03, 0.04)	−0.002 (−0.03, 0.03)
Mother condition	0.10 (−0.02, 0.23)	**0.01 (0.001, 0.02)**	−0.004 (−0.013, 0.004)[a]	−0.006 (−0.02, 0.01)	0.003 (−0.014, 0.021)	−0.002 (−0.02, 0.01)
Mother SVL	**0.08 (0.01, 0.15)**	**0.01 (0.002, 0.02)**	−0.005 (−0.01, 0.004)[a]	0.002 (−0.003, 0.01)	0.001 (−0.008, 0.011)	0.002 (−0.006, 0.01)

σ^2p denotes the sum of all variance components (i.e., $\sigma^2_a + \sigma^2_m + \sigma^2_{pe} + \sigma^2_r$). Permanent environmental effects (P_e) were only estimated for traits with repeated measurements (i.e., sprint speed and endurance). Estimates are provided for intercept only models (Model 1) and models containing covariates predicted to explain variance in offspring traits (Model 2). Individual variance estimates for Model 1 can be found in Table 2. The number of offspring measurements used for each analysis is provided for each model and trait.

[a]Indicates that estimate was scaled (mean = 0; standard deviation = 1) to ensure model convergence. Bolded estimates indicate that CI's do not overlap zero.

Table 2

Posterior mode of variance estimates for each of the random effects in animal models without fixed effects

Estimate	SVL	Mass	Captive growth	Wild growth	Log sprint speed	Log endurance
σ^2_a	0.0071 (0, 1.348)	3.032e-06 (0, 7.152e-04)	7.0e-06 (0, 0.002)	2.546e-06 (0, 6.49e-0.4)	8.776e-05 (0, 0.021)	0.0230 (0.01, 0.032)
σ^2_m	1.12 (0.653, 1.989)	0.0102 (0.0065, 0.01659)	0.001 (0, 0.002)	2.945e-06 (0, 7.78e-04)	0.0215 (0, 0.038)	4.232e-05 (0, 0.00701)
σ^2_p	NA	NA	NA	NA	8.48e-05 (0, 0.0135)	7.038e-05 (0, 0.0101)
σ^2_r	1.058 (0.350, 1.380)	0.011 (0.0093, 0.0134)	0.002 (0.0007, 0.002)	0.012 (0.009, 0.015)	0.0501 (0.045, 0.058)	0.033 (0.029, 0.038)

Brackets below estimates are the 95% CI. Where the lower 95% CI's converge on zero, we have just assigned these a zero value because variance estimates are constrained to be above zero. Notation for the estimates are as follows: σ^2_a = additive genetic variance; σ^2_m = maternal effects variance; σ^2_{pe} = permanent environmental variance; σ^2_r = residual/environmental variance; NA = not applicable. Note that variance estimates are rounded.

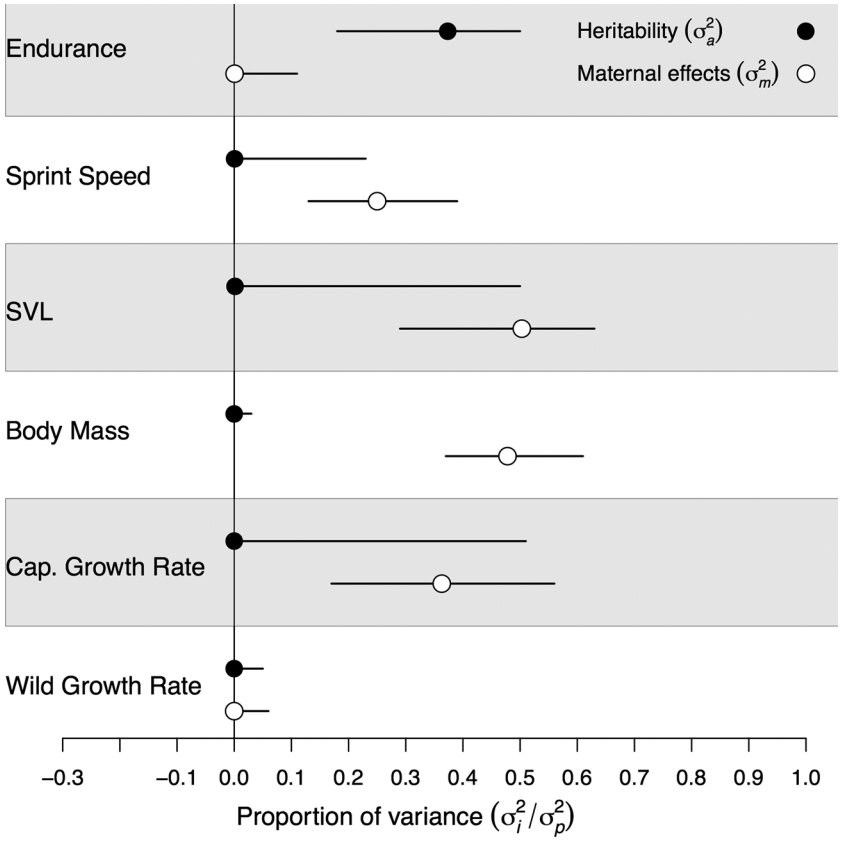

Figure 1

Estimates of heritability ($h^2 = \sigma^2_a/\sigma^2_p$) and maternal effects ($m^2 = \sigma^2_m/\sigma^2_p$) for each of the 5 offspring traits. Growth rates were split into growth in the wild and under captive conditions. Error bars around estimates are 95% Bayesian credible intervals. Note that σ^2_i indicates either σ^2_a and σ^2_m.

traits have been shown to harbor higher levels of additive genetic variation and corresponding environmental variation compared with morphological, behavioral, and physiological traits leading to lower heritabilities (Houle 1992). Our models for growth rate support the lower heritability found for life-history traits and suggest that maternal effects on growth rate might be apparent early in life and become less important as individuals develop, as has been shown in burying beetles (Rauter and Moore 2002). This might be the case if resource allocation to offspring by females is driving initial growth rate and this is then swamped by environmental sources of variation later in development. However, our small sample sizes for estimates of seminatural growth rates suggest that we must be cautious about making firm conclusions.

Whole-organism performance traits, such as sprint speed and endurance, are predicted to be more closely linked with fitness than morphological traits (Arnold 1983) and have been shown to be correlated with offspring survival in a number of lizards (Warner and Andrews 2002; Le Galliard and Ferrière 2008). Our findings of high heritability for endurance and low heritability for sprint speed are remarkably congruent with work in the lizard *Lacerta vivipara*, where full sibling comparisons showed high heritability for endurance [h^2 = 0.40; (Le Galliard et al. 2004) and h^2 = 0.46; (Sorci et al. 1995)] but low and nonsignificant heritability for sprint speed [h^2 = 0.12 (Sorci et al. 1995)]. However, sprint speed results contrast with studies in the lizard *Sceloporus occidentalis* (Tsuju et al. 1989), and the snakes *Thamnophis ordinoides* (Brodie 1989) and *Thamnophis sirtalis* (Garland 1988) in that these species exhibit significant heritability for sprint speed. The high heritability for speed in these studies may be the result of strong maternal effects, which can inflate heritability estimates in full sibling analyses (Kruuk 2004). Alternatively, the low heritability of speed we found maybe a result of strong selection on sprint speed given that selection should erode

additive genetic variation in a trait. However, offspring sprint speed in *E. quoyii* also seems to be under strong maternal control, mediated primarily through delayed birth date, given that we observed almost a 99% decrease in maternal effect variance when including maternal traits. This is congruent with other studies which have shown a strong influence of birthing date on offspring sprint speed (Shine and Harlow 1993; While et al. 2009). In contrast, the high heritability of endurance may suggest that selection is weak or that additive genetic variance is high relative to phenotypic variance because it is affected by many loci. Selection estimates for sprint speed and endurance will be necessary to fully understand the patterns we observed.

Understanding the heritability of traits linked with offspring survival has important implications for understanding the role of viability indicator models in explaining female preferences and polyandry. The weak evidence for additive genetic variation for most of the offspring traits we measured supports recent work testing "good" genes (i.e., viability indicator models) mechanisms in *Eulamprus*. Previous work has found no difference in offspring viability or traits predicted to be linked with fitness between single paternity and multiple paternity females in both observational (Noble et al. 2013a) and experimental designs (Keogh et al. 2013). Assuming that these traits are linked to offspring viability, as is the case in other lizard species (Sinervo et al. 1992; Elphick and Shine 1998; Warner and Andrews 2002; Husak 2006; Irschick et al. 2008; Le Galliard and Ferrière 2008), a lack of additive genetic variance for these traits suggests that females would not be able to choose males with high breeding values, resulting in weak selection for female choice. Although we cannot completely rule out "good" genes mechanisms, given that we lack an understanding of female mate preferences or how these traits are linked directly with offspring survival in this species, our results provide further evidence against viability indicator models by testing a key assumption of "good" genes models.

In summary, we provide estimates of heritability and maternal effects on 5 offspring traits predicted to influence fitness in an Australian lizard species, *E. quoyii*. Endurance showed significant additive genetic variance, which is congruent with a number of other studies on lizards and snakes (Sorci et al. 1995; Le Galliard et al. 2004). In contrast, we found that offspring mass, SVL, sprint speed, and captive growth rate were under strong maternal influence, whereas wild growth rate was largely determined by environmental factors. Offspring body size showed significant maternal effects with low estimates of heritability. Heritability estimates for body size and growth rates showed large confidence intervals suggesting that our pedigree was not sufficient in providing precise estimates for these traits and a multigenerational pedigree and a larger sample size will be required to resolve this uncertainty. The role of additive genetic and maternal effects in contributing to variation in offspring traits may vary across populations of a species (Uller et al. 2011) and future work will help determine how general our findings are for these traits.

SUPPLEMENTARY MATERIAL

Supplementary material can be found at http://www.beheco.oxfordjournals.org/

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Supplementary Materials

Table S1 - Estimates of heritability [$h^2 = \sigma^2_a / \sigma^2_p$], maternal effects [$m^2 = \sigma^2_m / \sigma^2_p$] and permanent environment effects [$P_e = \sigma^2_{pe} / \sigma^2_p$] from the posterior distribution using a prior of V = 0.01 and nu = 0.02, along with their 95% credible intervals (CI) for five offspring traits: snout-vent length (SVL), mass, growth rate, endurance and sprint speed. σ^2_p denotes the sum of all variance components (i.e. $\sigma^2_a + \sigma^2_m + \sigma^2_{pe} + \sigma^2_r$). Permanent environmental effects (P_e) were only estimated for traits with repeated measurements (i.e. sprint speed and endurance). Estimates are provided for intercept only models (*Model 1*) and models containing covariates predicted to explain variance in offspring traits (*Model 2*). Individual variance estimates for *Model 1* can be found in Table S2. The numbers of offspring used for each analysis are provided for each model and trait. All estimates were scaled (mean = 0, sd = 1) to ensure model convergence. Bolded estimates indicate that CI's do not overlap zero.

Model	Offspring SVL	Offspring Mass	Captive Growth	Wild Growth	Log Sprint Speed	Log Endurance
Model 1	n = 301	n = 300	n = 235	n = 122	n = 675	n = 670
Estimates						
h^2	0.004 (0, 0.76)	0.002 (0, 0.53)	0.001 (0, 0.51)	0.003 (0, 0.67)	0.001 (0, 0.25)	**0.394 (0.19, 0.52)**
m^2	**0.465 (0.21, 0.63)**	**0.58 (0.39, 0.71)**	**0.363 (0.17, 0.56)**	0.001 (0, 0.26)	**0.267 (0.13, 0.39)**	0 (0, 0.11)
pe^2	–	–	–	–	0.001 (0, 0.16)	0.001 (0, 0.19)
Model 2	n = 273	n = 272	n = 208	n = 109	n = 600	n = 596
Estimates						
h^2	0.005 (0, 0.87)	0.003 (0, 0.56)	0.003 (0, 0.64)	0.002 (0, 0.60)	0.002 (0, 0.33)	**0.342 (0.06, 0.49)**
m^2	**0.41 (0.28, 0.58)**	**0.548 (0.32, 0.67)**	0.001 (0, 0.26)	0.002 (0, 0.30)	0.001 (0, 0.23)	0.001 (0, 0.14)
pe^2	–	–	–	–	0.001 (0, 0.17)	0.001 (0, 0.27)
Covariate	**Est. (95% CI)**	**Est. (95% CI)**	**Est. (95% CI)**	**Est. (95% CI)**	**Est. (95% CI)**	**Est. (95% CI)**
Offspring SVL	–	–	–	–	0.03 (-0.002, 0.06)	0.009 (-0.02, 0.04)
Days in captivity	–	–	–	–	**0.03 (0.002, 0.067)**	**-0.03 (-0.06, -0.004)**
Offspring birthdate	0.21 (-0.11, 0.79)	**0.03 (0.002, 0.059)**	**0.028 (0.021, 0.036)**	-0.01 (-0.02, 0.004)	**0.12 (0.078, 0.17)**	-0.01 (-0.06, 0.02)*
Mother clutch size	**-0.54 (-0.92, -0.10)**	**-0.04 (-0.07, -0.001)**	**0.011 (0.0005, 0.02)**	0.003 (-0.02, 0.02)	0.009 (-0.05, 0.06)	-0.003 (-0.05, 0.04)
Mother condition	0.27 (-0.08, 0.63)	**0.03 (0.0002, 0.06)**	-0.004 (-0.013, 0.004)	**-0.017 (-0.03, -0.0001)**	0.008 (-0.036, 0.050)	-0.005 (-0.04, 0.03)
Mother SVL	**0.43 (0.06, 0.79)**	**0.04 (0.01, 0.079)**	-0.005 (-0.01, 0.004)	0.0010 (-0.008, 0.026)	0.004 (-0.047, 0.051)	0.01 (-0.03, 0.05)

Table S2 – Posterior mode of variance estimates for each of the random effects in animal models without fixed effects using a prior of V = 0.01 and nu = 0.02. Brackets below estimates are the upper 95% credible interval (CI). The 95% CI are shown, however, where the lower 95% CI's converge on zero, we have just assigned these a zero value because variance estimates are constrained to be above zero. Notation for the estimates are as follows: σ^2_a = additive genetic variance; σ^2_m = maternal effects variance; σ^2_{pe} = permanent environmental variance; σ^2_r = residual/environmental variance. Note that variance estimates are rounded.

Estimate	SVL	Mass	Captive Growth	Wild Growth	Log sprint speed	Log endurance
σ^2_a	0.0096 (1.0e-06, 2.08)	4.48e-05 (0, 0.01)	7.0e-06 (0, 0.002)	1.81e-05 (0, 0.0027)	0.0001 (0, 0.022)	0.023 (0.009, 0.031)
σ^2_m	1.18 (0.393, 1.89)	0.011 (0.006 to 0.02)	0.001 (0, 0.002)	5.20e-05 (0, 0.001)	0.021 (0.009, 0.038)	2.81e-05 (0, 0.006)
σ^2_p	NA	NA	NA	NA	9.6e-05 (0, 0.014)	6.68e-05 (0, 0.011)
σ^2_r	0.0029 (2.85e-05, 1.28)	0.006 (7.9e-05, 0.008)	0.002 (0.0007, 0.002)	0.002 (0.0009, 0.004)	0.049 (0.043, 0.056)	0.031 (0.027, 0.036)