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Quantitative genetics of costly neonatal sexual size dimorphism in squirrel monkeys (*Saimiri boliviensis*)

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Abstract

Offspring size is often an intimate link between the fitness of parents and offspring. Among mammals, neonate mass is also related to adult levels of dimorphism and intrasexual competitive mating. We describe the sex-specific genetic architecture of neonate mass in captive squirrel monkeys (*Saimiri boliviensis*), a small Neotropical primate. Best fitting quantitative genetic models show strong maternal genetic effects with little difference between sexes offering limited opportunity for neonatal dimorphism to respond to observed or hypothetical selection. Heritabilities that are approximately zero also imply it is unlikely that neonatal dimorphism can evolve as a correlated response to selection on adult size. However, male mass is also more dependent on maternal condition (age and parity) making dimorphism plastic. Finally, we hypothesize that large maternal genetic effects reflect income breeding and tightly synchronized seasonal reproduction in squirrel monkeys, both of which require strong maternal control of offspring growth and timing of birth.

Introduction

The causes of variation in offspring/propagule size and their and ecological and evolutionary consequences are important topics for understanding life history evolution (Clutton-Brock, 1991). Offspring size is necessarily a joint phenotype of parents and offspring. In all taxa, offspring size potentially influences the fitness of both parties through (1) immediate survival of offspring and potentially their later life survival and reproduction and (2) parental gain/loss of surviving offspring and investment of parental resources to produce other offspring either within a brood or in future reproductive attempts (Hoffman *et al.*, 2010; Krist, 2011). We use quantitative genetic models to explore the genetic architecture of neonatal size and dimorphism in captive squirrel monkeys (*Saimiri*), a small Neotropical primate.

Theoretical models of offspring size portray it as the result of allocation decisions by parents trading off a smaller number of large offspring or a larger number of small offspring (Smith & Fretwell, 1974). Under a fixed amount of total resources allocated to reproduction, this should result in a single optimal offspring size that maximizes parental fitness. This offspring size vs. number trade-off is strong in taxa lacking parental care and iteroparity (e.g. many arthropods, Fox & Czesak, 2000). Nevertheless, even within these taxa, there is often substantial variation in offspring size (Bernardo, 1996), much of which may be explained by variation in total investment in reproduction (Winkler & Wallin, 1987; Charnov & Ernest, 2006).

Differential parental investment among offspring based on offspring quality may also explain some aspects of size differences among offspring within populations (Haig, 1990, 1993; Crespi & Semeniuk, 2004). Offspring sex is a potential indicator of future fitness returns to parents and thus favors sex-biased investment, particularly in cases of strong sexual selection that increases variance in fitness of one sex – typically males – over the other (Trivers & Willard, 1973; Frank, 1990; Brown, 2001; Bercovitch, 2002). As such, sexual selection likely favours increase in size of both male and female offspring above the considerations of Smith & Fretwell (1974). This is because larger male offspring tend to be larger, more competitive adults and larger

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female offspring tend to be larger adults and better capable of producing large male offspring of their own (Carranza, 1996). Parental discrimination among offspring by sex has been demonstrated in many studies, but it often cannot be separated from competition among siblings for care making most primates with their widely spaced singleton broods attractive study systems (Mappes *et al.*, 1997; Wells, 2003).

Sexual size dimorphism at birth which mirrors adult dimorphism is also well documented in primates (Smith & Leigh, 1998). This trend appears to hold among other mammalian orders (Clutton-Brock, 1991) but does not apply broadly among birds (e.g. Anderson *et al.*, 1997; Cordero *et al.*, 2000). Comparisons or generalizations for other vertebrate taxa are more difficult because of more flexible sex determination and markedly reduced costs of reproduction. Neonatal dimorphism in mammals could be a byproduct of selection for increased adult male size to enhance competitive ability and mating success, or it might reflect selection on mothers to produce larger male neonates because they tend to be larger more competitive adults. Regardless of the mechanism, such neonatal size differences imply different prenatal costs for mammalian mothers rearing sons vs. daughters (Long, 2005). Maternal strategies to manage these costs may differ by offspring sex and likely vary among mothers and even within mothers based on their condition causing a large portion of the variance in offspring size in populations to be driven maternal effects (Bernardo, 1996; Fox & Czesak, 2000; Maestripieri & Mateo, 2009). Maternal condition may have strong effects on offspring size in primates because mothers have limited control over the sex of offspring carried to birth and cannot manipulate sex ratio to the degree of other taxa (West *et al.*, 2005). This likely results in greater variation in male offspring size and greater dependence of male offspring size on maternal condition (Bonduriansky, 2007). Offspring size can thus evolve through selection on this maternal genetic effect (m). Offspring genotypes, or direct genetic effects indexed by the heritability (h^2), may also explain some portion of variation and potentially be coadapted with maternal variation (Smiseth *et al.*, 2008). Such coadaptation can reduce total genetic variance and thereby limit response to selection on neonate size (negative direct-maternal genetic correlation, r_{AM}) (Cheverud & Moore, 1994).

The genetic architecture of neonatal dimorphism expands these maternal and direct genetic effects to be sex-specific and have cross-sex correlations. Neonatal dimorphism can evolve when sex-specific maternal genetic effects (m_m, m_f), heritabilities (h^2_m, h^2_f), or phenotypic standard deviations ($\mathrm{SD}_m, \mathrm{SD}_f$) differ; when cross-sex additive (r_A) or maternal genetic (r_M) correlations are less than one, and when the maternal-direct correlations differ between the sexes ($r_{AM,m}, r_{AM,f}$) or across the sexes ($r_{AM,mf}, r_{AM,fm}$). Response to selection, recorded as standardized selection differentials (i.e. intensities of selection), on either sex-specific neonate mass (i_m, i_f) is described by eqns (1) and (2) (Eisen & Hanrahan, 1972; Hanrahan & Eisen, 1973).

$$\begin{aligned}\Delta\bar{z}_m = \frac{\mathrm{SD}_m}{2}\Big[& i_m\Big(h^2_m + \frac{1}{2}m_m + \frac{3}{2}\sqrt{h^2_m m_m}\, r_{AM,m}\Big) \\ & + i_f\Big(\sqrt{h^2_f h^2_m}\, r_A + \frac{1}{2}\sqrt{m_m m_f}\, r_M \\ & + \frac{1}{2}\sqrt{h^2_m m_f}\, r_{AM,mf} + \sqrt{h^2_f m_m}\, r_{AM,fm}\Big)\Big]\end{aligned} \tag{1}$$

$$\begin{aligned}\Delta\bar{z}_f = \frac{\mathrm{SD}_f}{2}\Big[& i_f\Big(h^2_f + \frac{1}{2}m_f + \frac{3}{2}\sqrt{h^2_f m_f}\, r_{AM,f}\Big) \\ & + i_m\Big(\sqrt{h^2_f h^2_m}\, r_A + \frac{1}{2}\sqrt{m_m m_f}\, r_M \\ & + \frac{1}{2}\sqrt{h^2_f m_m}\, r_{AM,fm} + \sqrt{h^2_m m_f}\, r_{AM,mf}\Big)\Big]\end{aligned} \tag{2}$$

Equation (3) shows a useful way to express response by converting change in sex-specific neonate masses into change in neonatal dimorphism ($\Delta\tilde{\bar{z}}_{nd} = \bar{z}_m - \bar{z}_f$) and average neonate size $\Delta\tilde{\bar{z}}_{ns} = \frac{\bar{z}_m + \bar{z}_f}{2}$, Rogers & Mukherjee, 1992).

$$\begin{pmatrix}\Delta\tilde{\bar{z}}_{nd} \\ \Delta\tilde{\bar{z}}_{ns}\end{pmatrix} = \begin{pmatrix}1 & -1 \\ 1/2 & 1/2\end{pmatrix}\begin{pmatrix}\Delta\bar{z}_m \\ \Delta\bar{z}_f\end{pmatrix}. \tag{3}$$

Here, we use an extensive database of neonate mass and survival data on captive squirrel monkeys to estimate all parts of eqns (1)–(3) and interpret patterns through the ecology of wild populations (Zimbler-Delorenzo & Stone, 2011). This approach is common and has some empirical justification. Early literature reviews suggest strong correlations between lab and wild heritabilities (cf. Weigensberg & Roff, 1996), and a recent meta-analysis showed heritabilities tend to be lower in stressed wild populations but no consistent trend in maternal effects, despite reasoning that they should be larger under stressful conditions because these conditions emphasize differences among parents in their ability to buffer offspring from harsh environments (Charmantier & Garant, 2005).

Squirrel monkeys are small-bodied (700–1200 g), frugi-insectivores inhabiting secondary, lowland forests of Central and South America (Janson & Boinski, 1992; Boinski, 1999; Jack, 2011). They live in large mixed-sex social groups with a core of about 20 breeding females. Males are approximately 20% larger than females and go through a seasonal buildup of fluids in their arms, shoulders and upper back ('fattening') and compete aggressively for matings, with the largest males achieving the majority of copulations (Williams *et al.*, 1986; Boinski, 1992a, 1987b).

Unlike their closest relatives, capuchins (*Cebus*), and many other Neotropical primates, births are strongly seasonal, confined to a 1 week–2 month time span in

the wild. Gestation lasts approximately 5 months but appears to vary among females (Kerber *et al.*, 1977; Stolzenberg *et al.*, 1979), perhaps to facilitate this synchronization of births within social groups and reduce predation risk (Boinski, 1987a). Twinning is extremely rare and interbirth intervals are typically 1–2 years. Weaning ages vary from 4 to 18 months among species and populations (Zimbler-Delorenzo & Stone, 2011).

There are high costs of reproduction for squirrel monkey mothers. Neonates are very large relative to maternal body size and neonate brains are also very large relative to neonate body size (Hartwig, 1996; Garber & Leigh, 1997). Furthermore, a large portion of brain growth occurs rapidly after birth in the milk-only phase of lactation (Leigh, 2004; Langer, 2008). Male neonates have previously been reported to be substantially larger than females implying higher costs for mothers rearing male infants (112.5 g vs. 106.4 g, Rasmussen *et al.*, 1980). Males do not carry or provision infants. In *Saimiri boliviensis* and *Saimiri sciureus*, frequent low-cost allocare by adult and juvenile females is observed, consisting of brief episodes of infant handling and transport (Mitchell, 1990; Williams *et al.*, 1994; Stone, 2004).

We use the estimated quantitative genetic statistics to consider different pathways for neonatal dimorphism to develop ontogenetically and evolve under arbitrary selection regimens or those observed in captivity. For neonatal dimorphism to evolve and persist intergenerationally, there must be differences in sex-specific direct or maternal genetic effects, their correlations differ between the sexes, or the cross-sex correlations must be less than one. Differing sex-specific phenotypic standard deviations are a final route for the evolution of neonatal dimorphism (eqns 1–2). Importantly, maternal effects have been suggested as a form of phenotypic plasticity that allows for dimorphism be adjusted with maternal condition (Badyaev *et al.*, 2002; Badyaev, 2005). Moreover, we evaluate this condition-dependence of dimorphism (Bonduriansky, 2007) noting that for early life traits like neonate mass in dimorphic primates, maternal condition likely has greater influence on male rather than female neonate mass.

Materials and methods

Neonate mass and associated demographic records of captive Bolivian squirrel monkeys (*S. boliviensis*) were accessed at the Keeling Center for Comparative Medicine and Research (KCCMR) in Bastrop, Texas. Before giving birth, monkeys are housed in social groups of 15–35 animals containing one adult male and 10–15 adult females with their offspring. Current housing consists of indoor pens measuring approximately 4.3 × 1.2 × 2.0 m, with an opening to allow large groups access to multiple pens. Social groups have access to two to three pens depending on their size. Previous housing of the colony at University of South Alabama was similar (Williams *et al.*, 2002).

A commercial New World Primate diet supplemented with chopped vegetables (celery, bell peppers, squash, beans) is provided to all animals with grapes, peanuts, and mealworms fed sparingly as positive reinforcers when animals present for clinical observations. Water is available *ad libitum*. The light-dark schedule is maintained to track the local sunrise and sunset, so animals are exposed to long and short days annually. At term or immediately after giving birth, mothers and infants are moved into maternity pens with other dams and infants. Births typically occur at night. Neonate mass is collected on a scale to the nearest gram 48–72 h postpartum to avoid disrupting mother–infant bonding. Survival of neonates to 30 days after birth was used to measure phenotypic selection on mass at birth.

A data set of 1763 neonate mass records was used for quantitative genetic analysis where infant sex, subspecies membership, maternal identity and neonate survival were known. The data were $\log_e$-transformed prior to analysis to diminish mean–standard deviation relationship seen in the sex-specific values (Table 1). Log-transformation also renders the difference between sex-specific values a common measure of dimorphism used in eqns (1)–(2) above ($\bar{z}_m - \bar{z}_f = \log_e m - \log_e f = \log_e \frac{m}{f}$). This final data set excluded several outliers of both sexes identified by modified sex-specific z-scores greater than 3.6 (Iglewicz & Hoaglin, 1993). Neonate mass has a long left tail that transformation and outlier filtering do not remove. Inbreeding coefficients showed no relationship with

Table 1 Summary statistics for the *Saimiri* neonate mass (g) sample by sex and survival.

	Deaths			Survivors			Total		
	N	$\bar{x}$	SD	*N*	$\bar{x}$	SD	*N*	$\bar{x}$	SD
Female	184	94.32	13.29	700	107.18	12.59	884	104.50	13.76
Male	202	102.50	16.04	677	113.57	13.44	879	111.03	14.82
Female ($\log_e$)		4.537	0.144		4.667	0.120		4.640	0.136
Male ($\log_e$)		4.618	0.156		4.725	0.121		4.701	0.137

neonate mass in this data set. Inbreeding is rare in the pedigree used with only 72 members inbred and 32 of those having $F > 1/8$ (total of 2145 pedigree members). A smaller subset of 1132 records was used also to explore phenotypic relationships between neonate mass and maternal characteristics where they could be calculated. These characteristics were reproduction in the previous breeding season, birth date of the offspring within their cohort, number of previous offspring born to the mother (i.e. maternal parity) and maternal age.

Sex-specific phenotypic selection on neonate mass was documented as selection intensities (standardized selection differentials) – the difference between means after and before selection scaled by the initial standard deviation (Arnold & Wade, 1984). In addition, we visualized the fitness landscape with survival rates in predefined mass classes and cubic splines of individual survival data (Schluter, 1988).

For the quantitative genetic analysis, we used the 'animal model' to partition phenotypic variation in neonate mass. This is a linear mixed model containing fixed effects and random effects (Kruuk, 2004).

$$y = \mathbf{X}\beta + \sum_{i}^{n} \mathbf{Z}_i \mathbf{u}_i + \mathbf{e} \qquad (4)$$

In eqn (4), $\mathbf{y}$ is the vector of phenotypic measurements of neonate masses, $\mathbf{X}$ is an incidence matrix for fixed effects with $\boldsymbol{\beta}$ as a vector of regression coefficient estimates, $\mathbf{Z}_i$ is an incidence matrix for random effect i with $\mathbf{u}_i$ as the vector of solutions for the random effect and $\mathbf{e}$ is residual error. A maximum of three nonresidual random effects ($\mathbf{u}_i$) were fit in our analysis: direct additive genetic effect of the individual ($\mathbf{a}$), maternal genetic effect ($\mathbf{m}$) and maternal permanent environment effect ($\mathbf{c}$).

To explore potential differences in maternal investment during gestation by offspring sex we used a set of six bivariate models treating the neonate mass of males and females as separate traits. These models have only the sex-specific means for fixed effects, but differ in their random effects (Table 2). Variance components for each random effect were estimated when appropriate for each model. These were assumed to be equal to $\mathbf{A}\sigma_a^2$ (direct genetic), $\mathbf{A}\sigma_m^2$ (maternal genetic), $\mathbf{I}\sigma_c^2$ (maternal permanent environment) and $\mathbf{I}\sigma_e^2$ (residual error). $\mathbf{A}$ has elements equal to $2\theta_{ij}$ where θ_{ij} is the coefficient of coancestry between individuals i and j (Lynch & Walsh, 1998), and $\mathbf{I}$ is an identity matrix. All random effects were assumed to be uncorrelated with the exception of

Table 2 Results of bivariate quantitative genetic models for $\log_e$*Saimiri* neonate mass.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Δ_{AIC}	101.16	35.34	27.22	0	3.60	10.48
SD_f	0.1387	0.1369	0.1367	0.1369	0.1376	0.1403
SD_m	0.1404	0.1375	0.1372	0.1372	0.1374	0.1413
e_f	0.5363 (0.0685)***	0.5895(0.0410)***	0.4916 (0.0649)***	0.5919 (0.0424)***	0.5285 (0.0637)***	0.5069 (0.0808)***
e_m	0.5053 (0.0789)***	0.6727 (0.0429)***	0.5850 (0.0614)***	0.6777 (0.0433)***	0.6188 (0.0578)***	0.5634 (0.0895)***
c_f		0.4105 (0.0410)***	0.3182 (0.0494)***	0.0492 (0.0702)	0.0688 (0.0699)	0.0574 (0.0696)
c_m		0.3273 (0.0429)***	0.2562 (0.0489)***	0.0934 (0.0698)	0.0859 (0.0684)	0.0762 (0.0652)
h_f^2	0.4637 (0.0685)***		0.1901 (0.0786)**		0.0943 (0.0740)	0.0916 (0.0766)
h_m^2	0.4947 (0.0789)***		0.1588 (0.0689)*		0.0895 (0.0596)	0.1208 (0.0707)*
m_f				0.3589 (0.0770)***	0.3084 (0.0839)***	0.3441 (0.0921)***
m_m				0.2288 (0.0694)***	0.2058 (0.0707)**	0.2396 (0.0840)**
r_C		0.7727 (0.0860)***	0.7560 (0.1111)***	0.5381 (0.6501)	0.5436 (0.5527)	0.6621 (0.6394)
r_A	0.8802 (0.0836)***		0.9006 (0.2005)***		0.8336 (0.4081)*	0.8230 (0.4306)*
r_M				0.8729 (0.1437)***	0.8867 (0.1694)***	0.8589 (0.2113)***
$r_{AM,f}$						−0.2503 (0.3091)
$r_{AM,m}$						−0.3143 (0.3812)
$r_{AM,fm}$						−0.3483 (0.3843)
$r_{AM,mf}$						−0.2126 (0.4120)

*P<0.05, **P<0.01, ***P<0.001 term is different from 0 by t-test.

Δ_{AIC} change in AIC from best fitting model (model 4), smaller is better.

SD phenotypic standard deviation, subscripts are for female (f) or male (m).

e residual effect, fraction of unexplained phenotypic variance.

c maternal permanent environment effect, fraction of variance due to stable but nongenetic differences among mothers.

h^2 direct genetic heritability, fraction of variance due to variation in neonate genes.

m maternal genetic effect, fraction of variance due to variation in maternal genes.

r_C cross-sex maternal permanent environment correlation.

r_A cross-sex direct genetic correlation.

r_M cross-sex maternal genetic correlation.

r_{AM} within-sex and cross-sex direct-maternal genetic correlations.

direct-maternal genetic covariance (σ_{am}), which was estimated in model 6. Model fit is described by the difference in the Akaike information criterion from the best fitting model (Δ_{AIC}, Burnham & Anderson, 2002). Individual variance component ratios are annotated by *t*-test, *P*-values for significant difference from zero using t = estimate/SE and degrees of freedom equal to the number of records.

Results

Phenotypic selection

The intensity of selection on neonate mass is approximately equal on the sexes ($i_m = 0.1798$, $i_f = 0.1997$). For both sexes, survival rate plots show high mortality at small sizes with rapid increases in survival up to a plateau over about 110 g. Male neonates are at greater risk of mortality at all sizes (Fig. 1). This is best characterized as directional selection on neonate mass (Schluter, 1988).

Variance components

Quantitative genetic models explain about 30–50% of the phenotypic variance in sex-specific neonate mass, with male mass having larger residual variance in nearly all models (Table 2). Best fitting models by AIC include either an absence of direct additive genetic effects (model 4), or a complex mixture of direct genetic, maternal genetic and maternal permanent environment effects with the largest of these by far being maternal genetic (*m*, model 5). There is limited evidence of maternal–offspring coadaptation in the direct-maternal genetic correlations. They are all estimated to be negative (range: −0.21, −0.35), but are not significantly less than zero (model 6).

Sex-differences in the neonate variance component ratios provide some opportunity for the evolution of neonatal dimorphism particularly through differences in maternal genetics and the relatively weak maternal permanent environment cross-sex correlation. Indeed, all of the cross-sex correlations are estimated to be ≤ 0.9, though they cannot be declared significantly less than one because of their large standard errors.

Manipulation of the response equations with the model 6 values shows females to respond more to selection (Table 3). This is primarily due to their larger maternal genetic ratio. Under regimens of equal positive directional selection neonate size increases, but the level of dimorphism decreases. Using the observed selection intensities produces a nearly identical result. Increases in dimorphism can be accomplished by selecting with equal intensity on both sexes for smaller values. Neonatal dimorphism could thus result as a correlated response to neonatal size reduction.

Sex-biased investment based on changing maternal condition might decrease maternal variance component

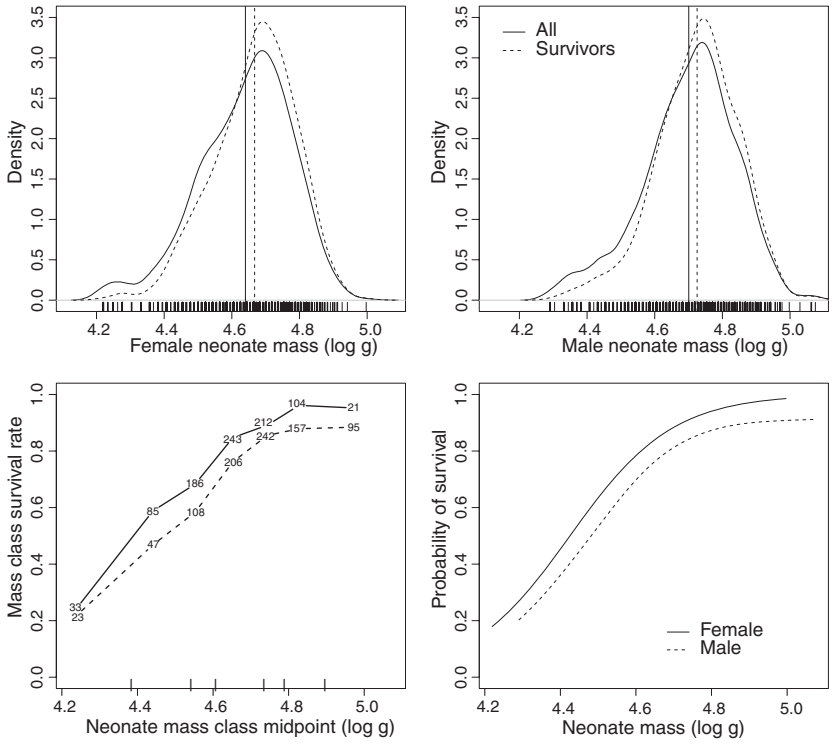

Fig. 1 Selection on $\log_e$ neonate mass in *Saimiri*. The upper panels show sex-specific distributions of all neonates (solid curves and rug plots) and survivors (dashed curves). Means are indicated by the vertical lines. Bottom panels give survival rates by neonate mass classes and cubic spline fits to the individual survival data.

Table 3 Sex-specific responses ($\Delta\bar{z}_m$ and $\Delta\bar{z}_f$) or responses in neonatal dimorphism ($\Delta\bar{\tilde{z}}_{nd}$) or average neonate size ($\Delta\bar{\tilde{z}}_{ns}$) using the observed selection intensities (first row) or other hypothetical pairings (remaining rows) from applying eqns (1)–(3) with the model 6 heritabilities, maternal genetic effects, cross-sex correlations and phenotypic standard deviations reported in Table 2.

i_m	i_f	$\Delta\bar{z}_m$	$\Delta\bar{z}_f$	$\Delta\bar{\tilde{z}}_{nd}$	$\Delta\bar{\tilde{z}}_{ns}$
0.1798	0.1997	0.0039	0.0044	−0.0005	0.0041
1	1	0.0204	0.0227	−0.0023	0.0216
−1	−1	−0.0204	−0.0227	0.0023	−0.0216
1	−1	0.0016	−0.0041	0.0057	−0.0012
−1	1	−0.0016	0.0041	−0.0057	0.0012
0	1	0.0094	0.0134	−0.0040	0.0114
1	0	0.0110	0.0093	0.0017	0.0102

ratios for male neonates. Consistent with this hypotheses, we found that phenotypic regression models with maternal age, parity, whether the mother produced an offspring in the previous year, and date of offspring birth in their cohort explained more variation in the neonate mass of males rather than females ($R^2 = 0.071$ vs. $R^2 = 0.045$, Table 4). In particular, maternal age and parity have stronger effects on male neonates.

Discussion

The inheritance of neonate mass in squirrel monkeys is likely a complex mixture of maternal and direct genetics effects. However, this is dominated by maternal genetic effects, which might be construed as justification for models of offspring size optimization through parental resource allocation in squirrel monkeys. This inference should be tempered by the fact that the genetic variance components only quantify standing variation in a population and cannot capture how fixed loci affect traits, and they can evolve themselves. Numerous embryological and developmental genetic studies in model organisms have identified pathways through which offspring genes orchestrate ontogeny (Brakefield, 2011). Direct genetic effects in offspring may be quite low because of an intense history of strong selection. This would require mutational input to be quite low or selection extremely strong to see such small heritabilties. Imprinting is also known to affect loci that influence body size in mammals (e.g. *Igf2*). However, quantitative genetic models of imprinting, particularly in the presence of maternal effects, are not yet developed (Hager *et al.*, 2008; Spencer, 2009).

Table 4 Regression model coefficients ($\beta \pm$ SE) of maternal characteristics predicting $\log_e$*Saimiri* sex-specific neonate mass.

	Female	Male
Intercept	4.6224 (0.0388)***	4.6099 (0.0427)***
Offspring age in cohort	0.0006 (0.0002)**	0.0005 (0.0002)*
Birth previous year	0.0050 (0.0133)	0.0112 (0.0139)
Maternal parity	0.0174 (0.0052)***	0.0202 (0.0052)***
Maternal age	0.0044 (0.0100)	0.0170 (0.0109)
Maternal age^2	−0.0010 (0.0006)	−0.0017 (0.0006)**
N	568	564
R^2	0.0449	0.0712
Model *P*	0.0001	<0.0001

*P<0.05, **P<0.01, ***P<0.001.

We found weak evidence of differences in the genetic architecture of male and female neonate mass in squirrel monkeys. This is consistent with theoretical arguments about the condition-dependence of sexual dimorphism (Rowe & Houle, 1996; Bonduriansky, 2007). Male neonate mass is more responsive to changing maternal condition indexed by parity or age, and large residual effects from the quantitative genetic models are also consistent with the strong dependence of neonate mass on unmeasured environmental circumstances. As such, maternal effects dependent on labile aspects of maternal condition enable neonatal dimorphism without altering the (direct or maternal) genetic architecture of neonate size. The scope of these differences may be even larger in the wild where maternal condition is likely to vary more.

Our results also provide some insights on whether neonatal dimorphism in sexually dimorphic mammals is a byproduct of sexual selection for adult size dimorphism or a direct response to selection for increased male size via maternal investment in the context of sexual selection. As heritabilities are very small for neonate mass, selection on adult mass is unlikely to have strong correlated response in neonates. This implies that neonatal dimorphism in this taxon, and perhaps other dimorphic mammals, is not simply a correlated response to adult male size increase but has been directly selected for through increased survival or mating success. Evaluating this hypothesis will require more detailed information on the genetic architecture of dimorphism at later post-natal ages, including adulthood. However, it is supported by patterns noted in recent reviews, which argue for a weak relationship between adult and neonatal dimorphism. First, maternal effects are likely to play a much larger role for early life traits, and are known to decline with age in most taxa where they have been studied (Cheverud, 1984; Wilson & Réale, 2006). More variation in neonatal dimorphism will be product of the environment provided by mothers. Second, Poissant & Coltman (2009) show that cross-sex genetic correlations typically decline strongly with age. Physiological opportunities to enhance sex differences increase with age as the sexes 'grow apart'.

Our finding of near-zero direct effect coupled with large maternal genetic effects on squirrel monkey neonate mass is intriguing, as heritabilities for neonate

mass in other taxa are usually well above zero (e.g. humans, pig-tailed macaques, and livestock, Ha *et al.*, 2002; Wilson *et al.*, 2005). Moreover, comparison of results from captive squirrel monkeys with these studies is appropriate because these are also captive or domesticated animals. Quantitative genetic statistics are population-specific because they depend on allele frequencies and local environmental conditions (Roff, 1997; Vitzthum, 2003). This demands caution when interpreting differences among species, but we cannot help but speculate that two factors – income breeding at small body size and intense reproductive seasonality – may cause this pattern of ≈0 heritability and larger maternal effects on neonate mass to typify squirrel monkeys.

Small-bodied mammals often have little capacity to physiologically store nutrients for simultaneously growing offspring and maintaining their own health. As such, they fund these activities by current energetic income rather than stored capital (Jönsson, 1997; Stephens *et al.*, 2009). In this situation, variation among mothers in foraging efficiency and resource allocation to offspring should explain large amounts of variance in offspring size at birth and be reflected in large maternal effects. Although there may be intense selection on foraging efficiency and allocation patterns, they are also likely very large mutational targets, which would maintain substantial genetic variance counteracting the variance eroding effects of selection (Houle, 1998). Squirrel monkeys are much smaller than other primates with neonate mass quantitative genetic statistics, they rear relatively very large, costly offspring and are classified as income breeders (Tardif, 1994; Hartwig, 1996; Leigh, 2004; Brockman & van Schaik, 2005). The low heritability and large maternal effect on neonate mass in other small mammals provide some limited support for this idea (El-Oksh *et al.*, 1967), but this comparison is confounded by the fact that other small mammals typically give birth to large litters, which create their own unique uterine environments, and low heritabilities and large maternal effects for neonate mass are seen in some large capital breeders (Wilson *et al.*, 2005; Kruuk & Hadfield, 2007). Other small-bodied or income breeding, singleton-bearing primates or other mammals would be better comparisons to evaluate this hypothesis.

One might also expect low heritabilities and higher maternal effects if synchronous breeding increases maternal and offspring fitness (Marshall & Uller, 2007). In this case, it is in the offspring's best interest to be born at the time indicated by climatic or social cues from the environment. As mothers are more likely to receive and have evolved mechanisms to respond to these cues, it is expected variation in their responses to those cues and how they manipulate uterine environments to accelerate or slow growth and time birth explain a large amount of the variance in neonate mass. Genes active in offspring that alter this system would be selected against because they reduce the offspring's own fitness by causing birth at inopportune times.

Squirrel monkey birth seasonality may be related to predation risk or cyclical changes in food availability (Boinski, 1987a). Birth synchrony in ungulates has been argued to be primarily caused by annual climatic cycles but predation pressure plays an important secondary role in constricting births to a narrower time window (Rutberg, 1987). Boinski *et al.* (2003) note that all squirrel monkey species face intense predation pressure, particularly from raptors, that falls heavily on infants, although small body size elevates predation risk for squirrel monkeys at all ages relative to larger primates. Large maternal genetic variance might be maintained in this system because the precise timing of birth is not indicated by a predictable climatic signal but by the birth or gestational progress of other group mates. As females conceive at different times, they face a fluctuating optimal gestation length determined by foetal maturation and their peers giving birth (Houle, 1998; Hughes & Burleson, 2000). If mothers are receiving signals from the social environment and altering their gestation accordingly it would imply there are not only maternal effects on squirrel monkey neonate mass but that these are partly social effects on neonate mass as described by evolutionary genetic models of interacting phenotypes (Moore *et al.*, 1997; Wolf *et al.*, 1999; Bleakley *et al.*, 2010).

The mechanisms that might underly this system of synchronizing birth merit further research. Boinski (1987a) suggested that the inspection of pregnant females' genitals by other females prior to birth could induce parturition either by pheromonal cues or by causing stress. Urine washing may also facilitate pheromonal communication (Boinski, 1992b). The major hormonal players in the timing of birth are known to be progesterone (inhibiting uterine contraction), oestrogen (promoting it), oxytocin (coordinating contractions once started) and probably prostaglandins as the main signals of birth timing. However, the details appear to vary substantially among mammalian species that have been investigated (Diamond *et al.*, 1987; Jenkin & Young, 2004; Norman *et al.*, 2007; Mesiano *et al.*, 2011). That oxytocin levels, both within the brain and circulating in the blood, can be influenced by social interactions suggests it could be involved in timing squirrel monkey births within groups (Uvnäs-Moberg, 2003).

In summary, we found that the environment provided by mothers, which varies genetically, explains a large portion of phenotypic variance in squirrel monkey neonate mass, while variation in offspring genes explains very little. Any genetic response to selection on neonate mass would result from changes to genes active in mothers affecting the uterine environment they provide to offspring. The absence of direct genetic

effects also implies neonatal dimorphism is unlikely to be a byproduct of sexual selection for larger adult male size, but is instead a maternal response to enhance the fitness of male offspring within the socio-ecological context of sexual selection. This pattern is surprisingly inconsistent with previous studies of neonate size in primates and other mammals. We suggest the squirrel monkey pattern reflects income breeding at small body size or tight birth synchrony, which may be influenced by maternal energetics when rearing relatively large, costly infants and high predation risk on these vulnerable neonates. There were only minor sex differences in the genetic results but male neonate mass was more dependent on maternal parity and age. This condition-dependence is a mechanism to enhance sexual dimorphism in squirrel monkey neonates without dramatically altering the sex-specific genetic architecture.

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